Sensorion

Developing next generation solutions to address hearing disorders

Restore / Treat / Prevent

Euronext Growth: ALSEN

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Sensorion at a glance

The inner-ear specialist: building leadership across the full spectrum of hearing loss disorders.

Expertise

Inner-ear know-how

Inner-ear drug development expertise built across gene therapy and small molecule programs: disease biology, cochlear delivery, translational models, clinical endpoints and audiology trial design. Know-how that applies to any modality entering the inner ear.

Lead program

SENS-601

Gene therapy candidate designed to address GJB2-related congenital hearing loss, the most common genetic cause worldwide, across pathogenic GJB2 variants.

Second asset

SENS-401

Oral small molecule candidate designed to protect inner-ear sensory tissue. Orphan drug designation from EMA and FDA. Clinical-stage asset: Phase II trials completed across three indications. Currently pursuing partnering opportunities to fund and lead the next stage of clinical development.

Shareholder base

Blue-chip healthcare specialist investors

Invus, Sofinnova Partners, Redmile Group, Coastlands Capital and Cormorant Asset Management, joined by Sanofi as a strategic investor in the January 2026 financing. Lead program on track for multiple milestones in the near term, capitalized by that financing.

Exclusive partnerships

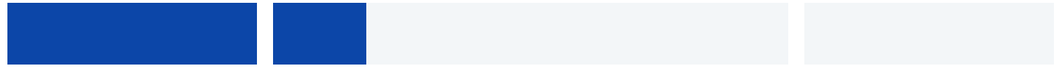
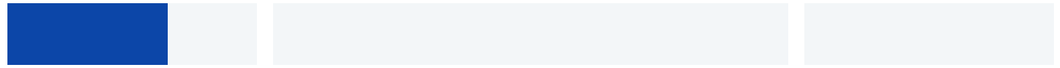
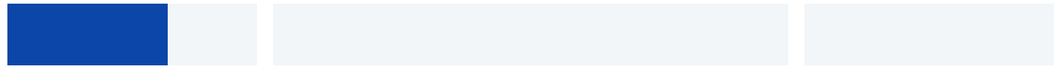
INSTITUT Pasteur

Necker ENFANTS MALADES HÔPITAL UNIVERSITAIRE

Institut reConnect

INSTITUT PASTEUR • AP-HP • INSERM • UNIVERSITÉ PARIS CITÉ • FONDATION POUR L'AUDITION

Pipeline: leading player in GJB2

Program		Preclinical	Phase I/II	Phase III	Next steps
SENS-601*	GJB2 (DFNB1A) HL: congenital onset				CTA approved in France; submitted in Canada. US IND and Australia CTA submission (YE 2026). First patient dosing in France targeted by early 2027
	GJB2 (DFNB1A) HL: pediatric progressive				Preclinical studies and patient identification ongoing
	GJB2 (DFNB1A) HL: adult onset (presbycusis)				Preclinical studies and patient identification ongoing

Program		Preclinical	Phase I	Phase II	Phase III	Next steps
SENS-401**	Hearing preservation after cochlear implantation; cisplatin-induced ototoxicity; SSNHL					Exploring partnering opportunities

* Option to license from the Institut Pasteur for SENS-601. ** 3SBio holds a right of first refusal for licensing SENS-401 in Greater China, except in combination with cochlear implants. HL: hearing loss. SSNHL: sudden sensorineural hearing loss. DFNB1A: autosomal recessive, non-syndromic sensorineural hearing loss. CTA: clinical trial application. IND: investigational new drug.

Recent highlights and upcoming milestones

✓ Achieved ○ Expected

✓ **January 2026**

€60 million financing completed

- €20m from Sanofi as strategic investor
- €40m from Redmile Group, Artal (advised by Invus) and Sofinnova Partners, existing shareholders, with Cormorant, Coastlands Capital and Sphera Healthcare

✓ **June 2026**

New Chief Executive Officer appointed

- Frédéric Chereau appointed Chief Executive Officer, effective June 1, 2026
- Amit Munshi continues as Chairman of the Board, having served as Interim CEO since February 2026

✓ **August 2026**

CTA cleared in France; submitted in Canada

- ANSM clearance received August 31, 2026 under the Fast Track assessment process
- Enables the start of HearConnex, the Phase I/II trial of SENS-601 in France
- Canada CTA under review

○ **Expected Q4 2026 → 2027**

US IND, Australia CTA and first patient dosed

- US IND and Australia CTA submissions on target for year-end 2026
- First patient dosing targeted by early 2027, with clinical data generation through 2027
- Poster to be presented at ESGCT (October 2026)

ANSM: Agence nationale de sécurité du médicament et des produits de santé. CTA: clinical trial application. IND: investigational new drug. ESGCT: European Society of Cell & Gene Therapy.

RESTORE

SENS-601 for GJB2-related congenital hearing loss

Initial market opportunity

Widespread newborn screening and advances in early genetic testing continue to improve the identification and characterization of children with GJB2-related hearing loss.

~98%

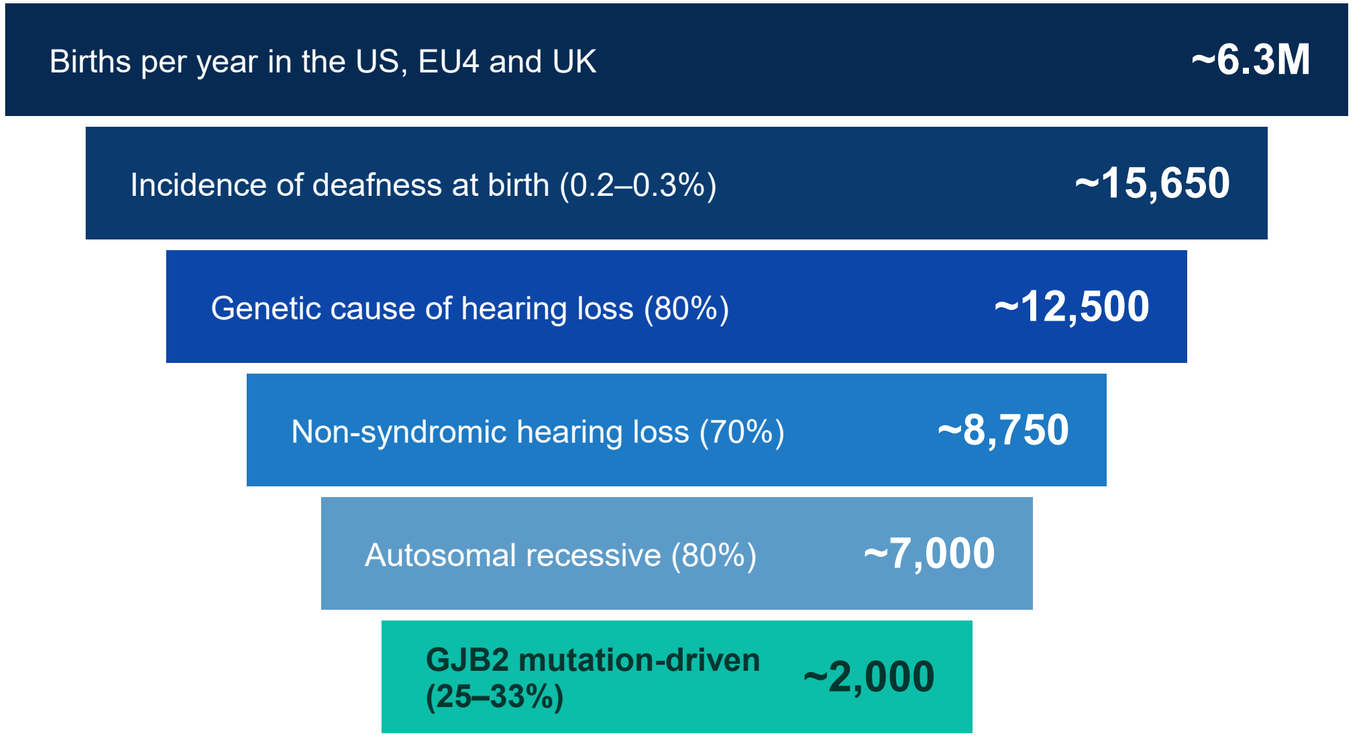
of US newborns underwent hearing screening as of 2022

~2,000

children born each year across the US, EU4 and UK with autosomal recessive GJB2-related congenital hearing loss (DFNB1A)

GJB2 (DFNB1A) HL: congenital onset

Illustrative management estimate



A GJB2 diagnosis carries lifelong consequences: prevalence rises from **~2–3 per 1,000** births to **~3–5 per 1,000** by school age, and more than **50%** of children with milder GJB2 variants progress over time, reinforcing the case for intervening at the point of genetic diagnosis.
There are no approved therapies addressing GJB2-related hearing loss.

Peer-reviewed published studies; company estimates. EU4: the four largest European Union markets: France, Germany, Italy and Spain. UK: United Kingdom.

The inner ear as a gene therapy target

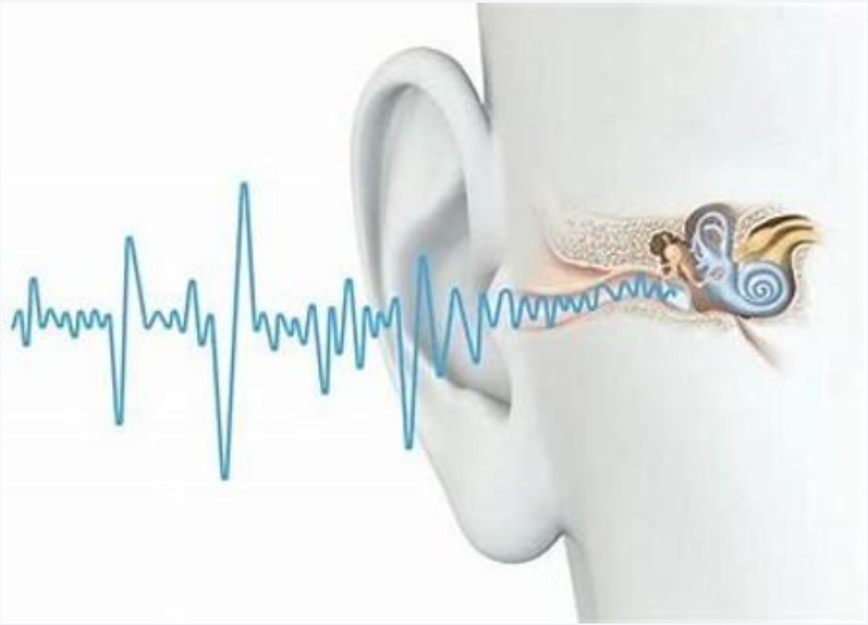
01 Direct local administration to target cells, with limited biodistribution outside the inner ear

02 Immunologically protected by the blood–labyrinth barrier

03 Low volume and low viral particle count versus systemic approaches

04 Durable activity from a single administration: cochlear cells do not normally divide

05 Primary genes involved in hearing loss are well characterized, with identified expression sites and activity

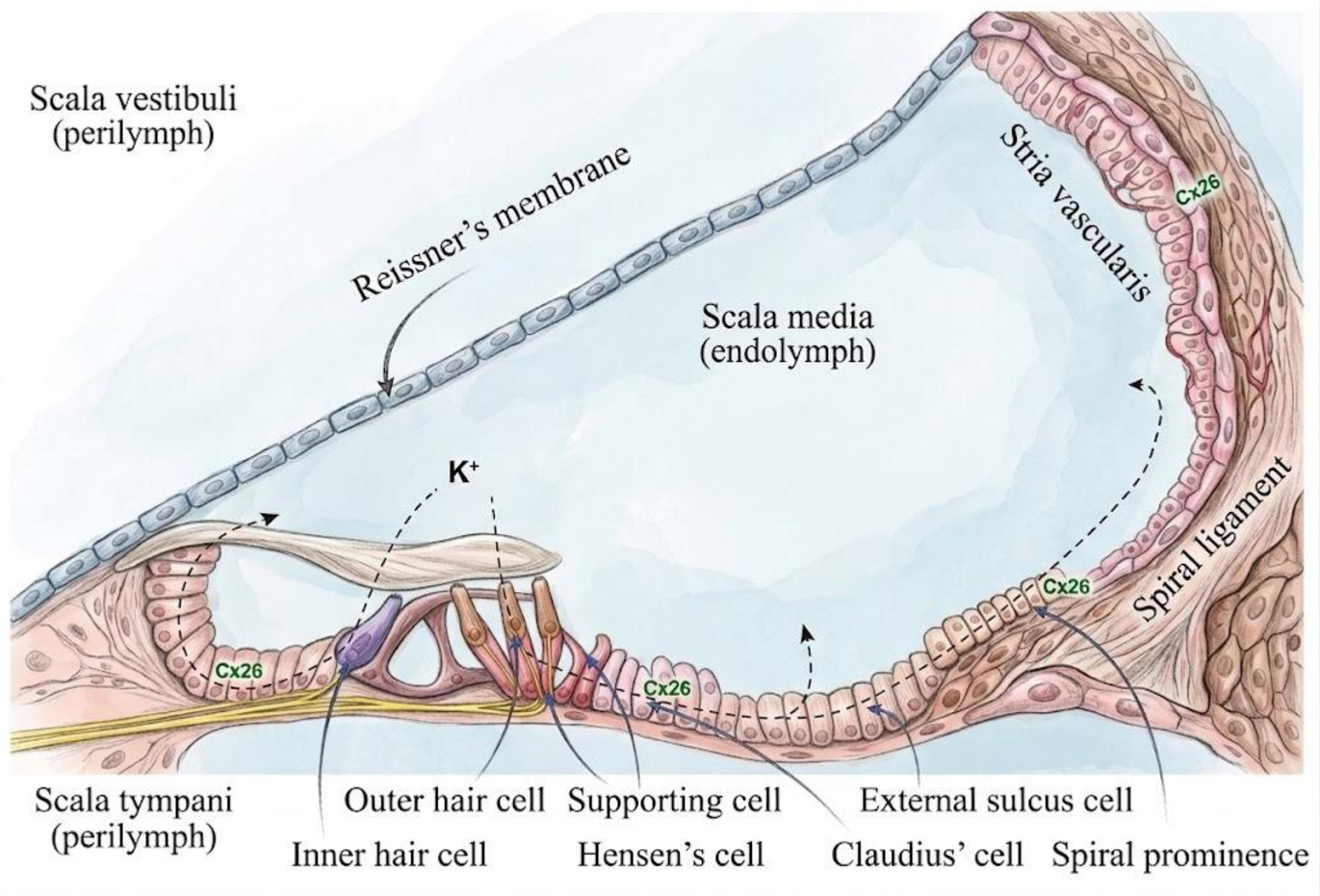


The role of Connexin 26 (Cx26) in the cochlea

Cx26, encoded by **GJB2,** is the predominant connexin in the cochlea, where it forms gap junctions between non-sensory supporting cells.

Those gap junctions enable metabolite exchange and potassium recycling that sustain the electrochemical gradient driving sound transduction.

Cx26 is **absent from sensory hair cells,** where its expression would disrupt auditory function.

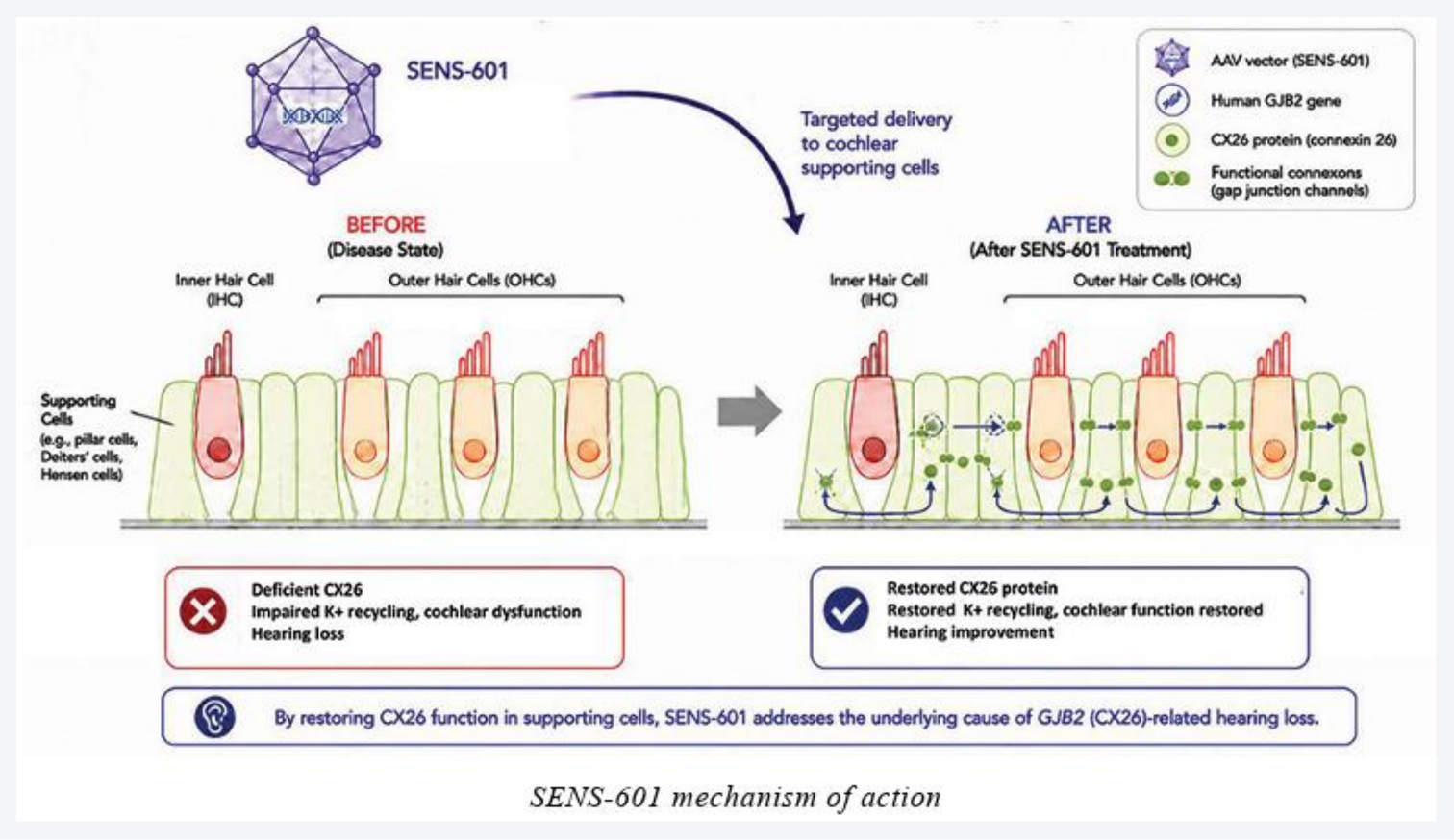


GJB2 mutations and non-syndromic hearing loss

>100

GJB2 recessive mutations described to date, with SENS-601 designed as a single-construct approach across GJB2 variants

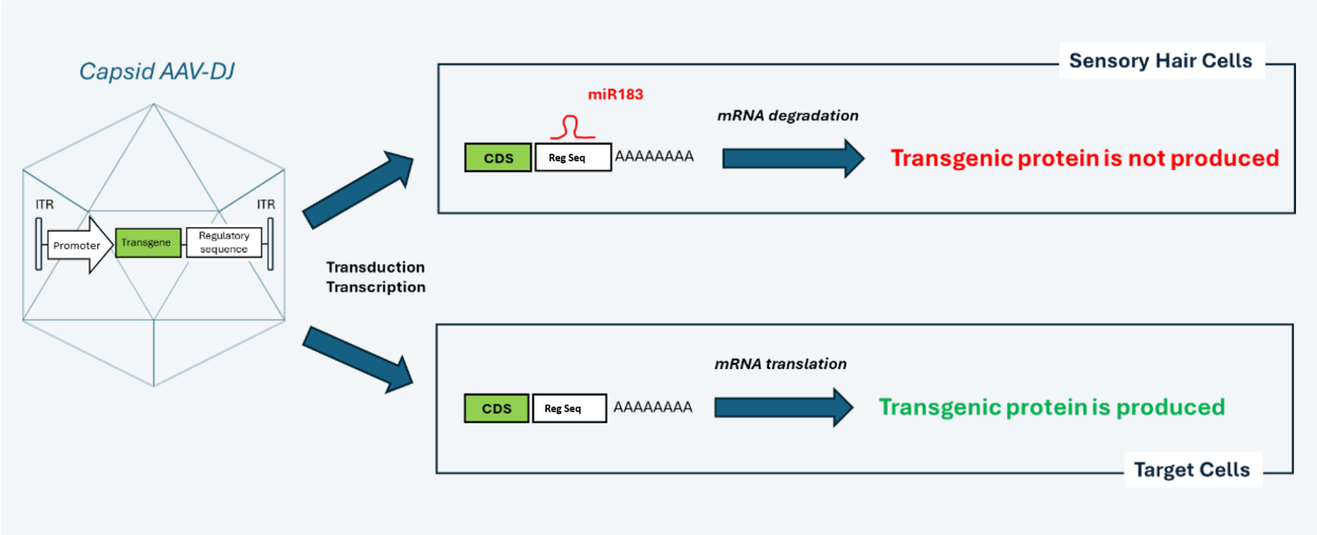
> Severity of hearing loss correlates with the degree of GJB2 loss of function.



SENS-601 mechanism of action

Construct design

- **AAV-DJ capsid** with broad tropism for cochlear supporting cells.
- Cassette combining a constitutive 5' promoter, the CX26 coding sequence and a **proprietary regulatory sequence** in the 3' UTR, yielding functional **26 kDa** CX26 proteins.
- **Selective.** Sequence complementary to **miR-183**, present in sensory hair cells but not in supporting cells: transgene off in hair cells, on in target cells.
- **Sustained.** The miR-183 sequence stays expressed in the aged cochlea, including in humans.
- **Durable.** miR-183-mediated transgene silencing in hair cells holds for at least 12 months after treatment.



Left: the AAV-DJ capsid and its cassette. Right: after transduction, transgene mRNA is degraded in sensory hair cells and translated in target cells.

Result: Cx26 functional protein translated in all supporting cells of the inner ear, but not in the sensory hair cells.

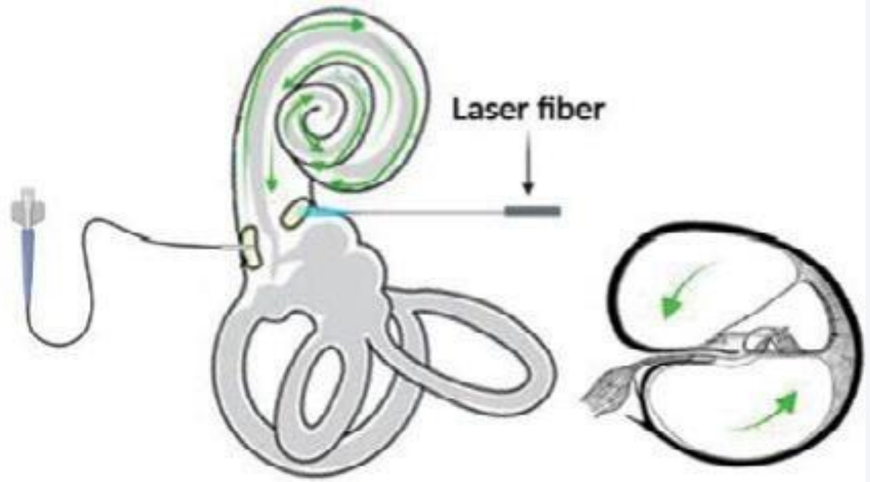
UTR: untranslated region.

Surgical delivery approach

Dual-fenestration technique combines two familiar procedures, **cochlear implantation and stapedotomy**, and is designed to minimize overpressure and 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 the multiple administrations performed to date.

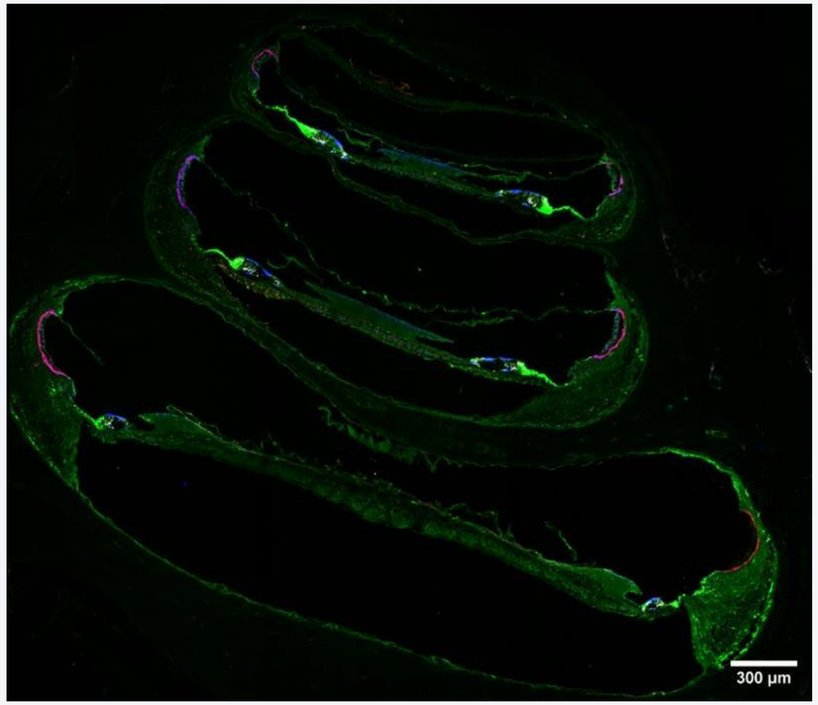


Proprietary injection system combining cochlear implantation and stapedotomy approaches

SENS-601 Efficiently Transduces NHP Cochlea

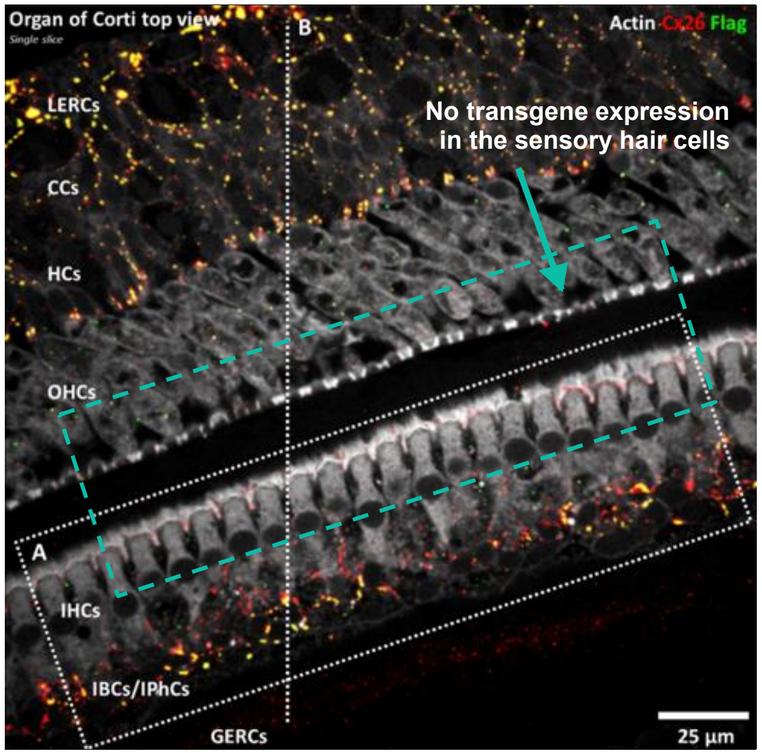
- Injection of SENS-601 (Flag surrogate), at the NHP equivalent effective dose, leads to strong transgene detection in the cochlea of NHP from base to apex
- Flag signal within the CX26 network was quantified across the different compartments of interest and tonotopic axis
- Transgene expression pattern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

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



CLAUDIN11 SOX2 GFP Actin

Tropism and selectivity to CX26-expressing target cells in NHP



Maximum intensity projection of whole-mount organ of Corti, three weeks after a single intracochlear injection of GJB2-Flag-GT, a SENS-601 surrogate.

Three weeks after a single intracochlear injection: extensive transduction of the supporting cells that naturally express GJB2 observed along the tonotopic axis of the cochlea.

No transgene expression detected in inner or outer hair cells, which were devoid of both CX26 and Flag signal.

Well tolerated at three and nine weeks post injection.

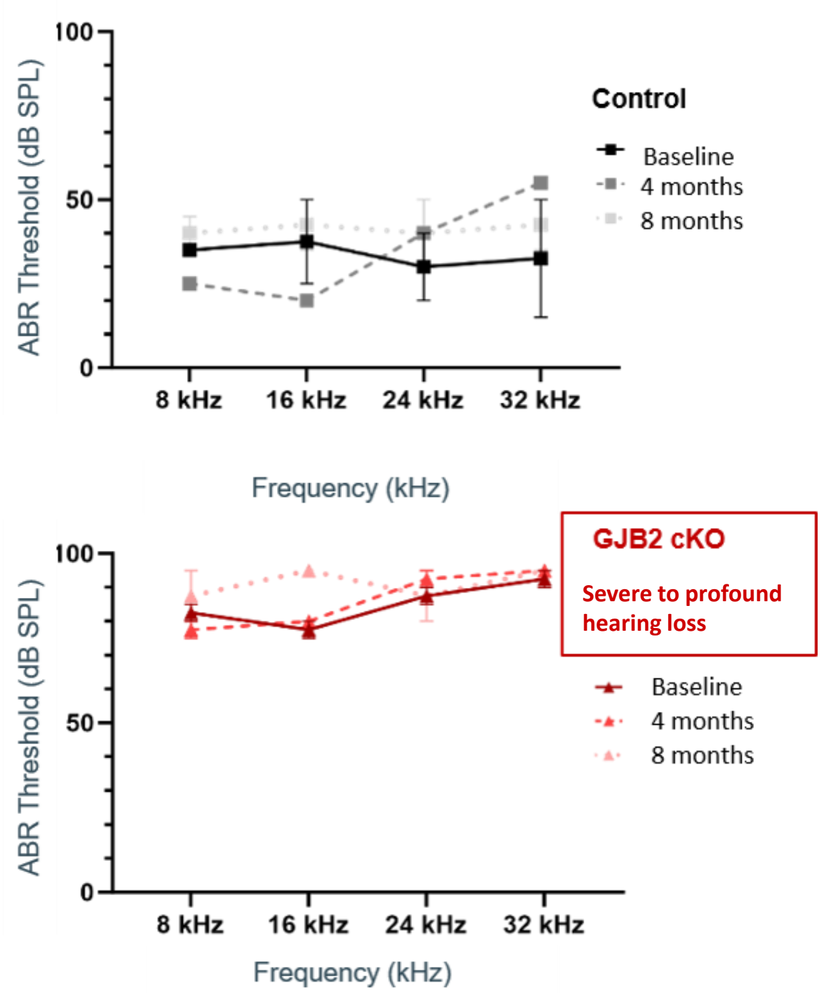
NHP: non-human primate.

Cx26 knock-out mouse model

Exclusive conditional knock-out model; conditional design circumvents the embryonic lethality of complete biallelic GJB2 loss.

Early inactivation in utero, while the inner ear develops, reproduces the pathophysiology of **DFNB1A**, the most common form of GJB2 hearing loss.

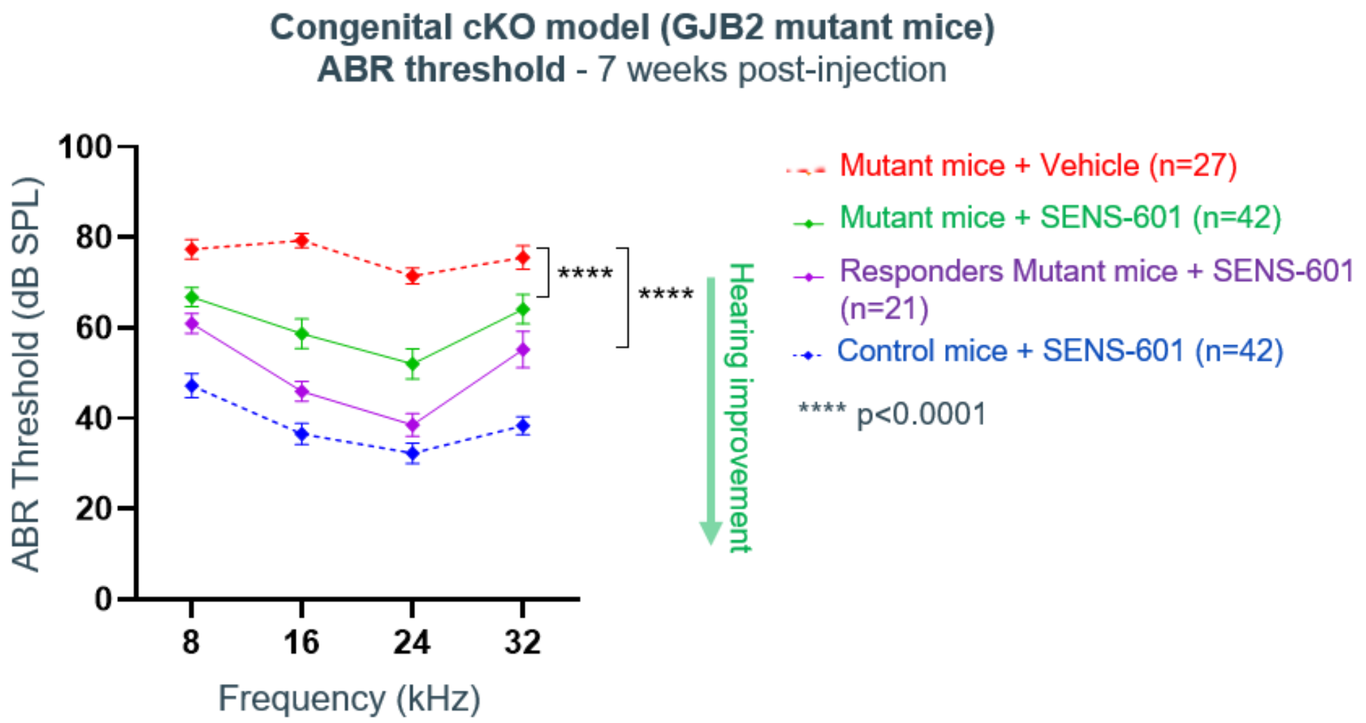
Complete Cx26 depletion confirmed, with severe-to-profound hearing loss measured by ABR



ABR thresholds (dB SPL) by frequency (kHz) in control and GJB2 cKO animals. Giese et al., ARO 2026.

DFNB1A: autosomal recessive non-syndromic hearing loss caused by GJB2 mutations. ABR: auditory brainstem response.

Hearing restoration in Cx26 knock-out mice



Hearing improvement observed **as early as three weeks** after injection, across all tested frequencies.

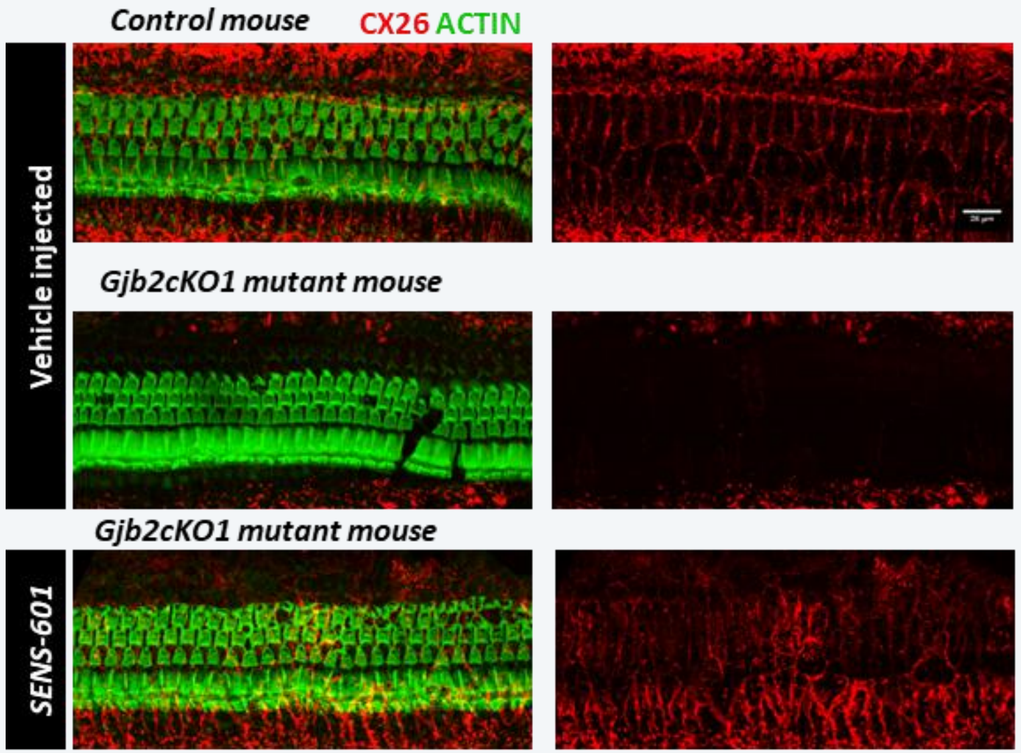
Demonstrated across multiple dose levels, with a therapeutic dose selected for further development.

Replicated across multiple research and development manufacturing batches, alongside restoration of the Cx26 network in the cochlea.

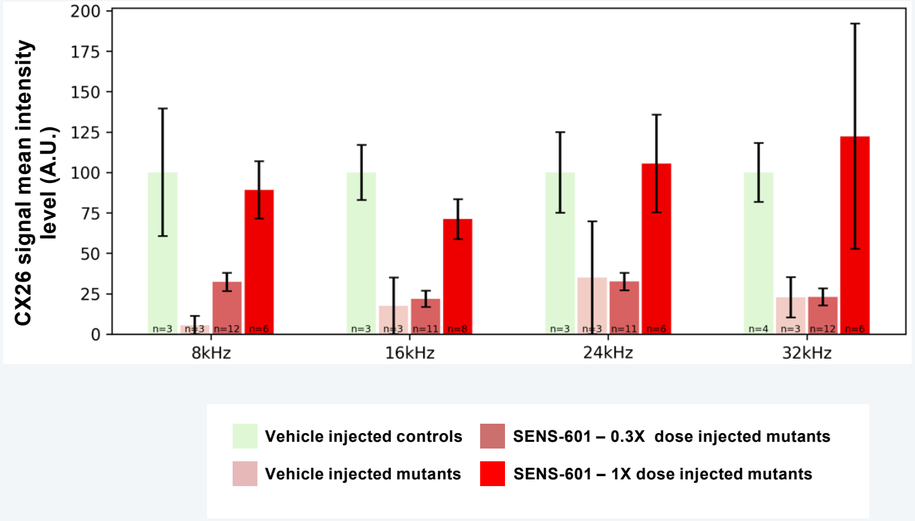
Pooled ABR data from efficacy and dose-ranging studies in the cKO1 model. Control + SENS-601 (n=42); mutant + vehicle (n=27); mutant + SENS-601 (n=42); responders (n=21, frequencies restored ≤60 dB). Giese et al., ARO 2026.

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

CX26 network immunostaining



CX26 levels quantification

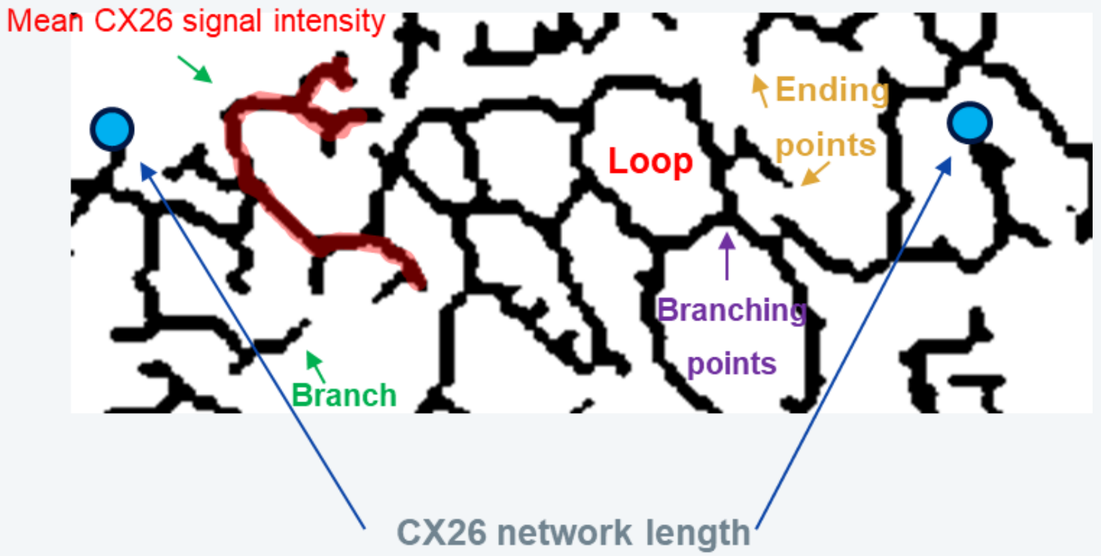


Tonotopic and dose-dependent improvement of CX26 levels, evaluated by AI-based image analysis of the cochlear CX26 network.

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

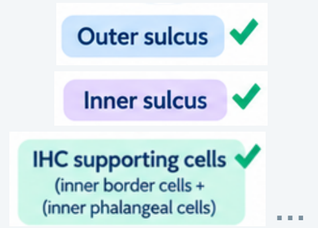
The CX26 network itself is rescued, not only the level of expression

Main CX26 network metrics

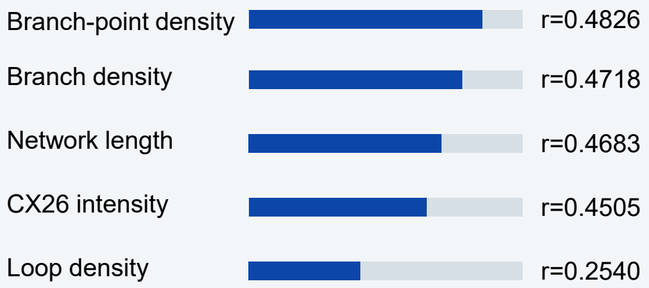


150 CX26 network features measured per sample

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

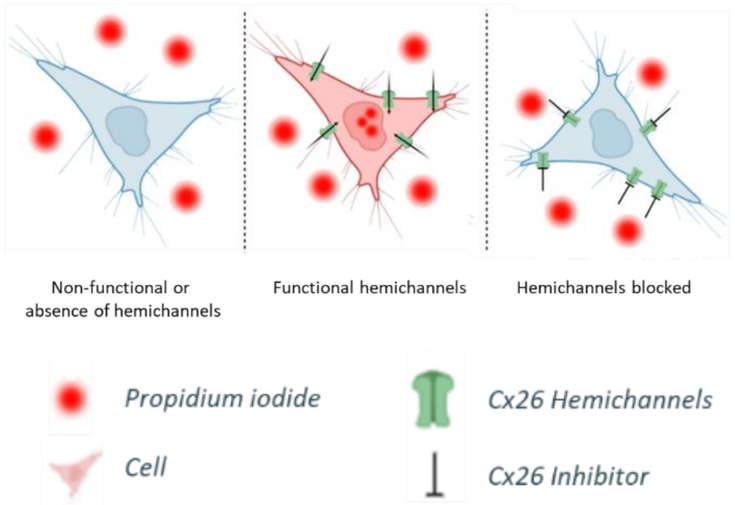
Analyses performed 18 weeks post-administration at P0/P2.

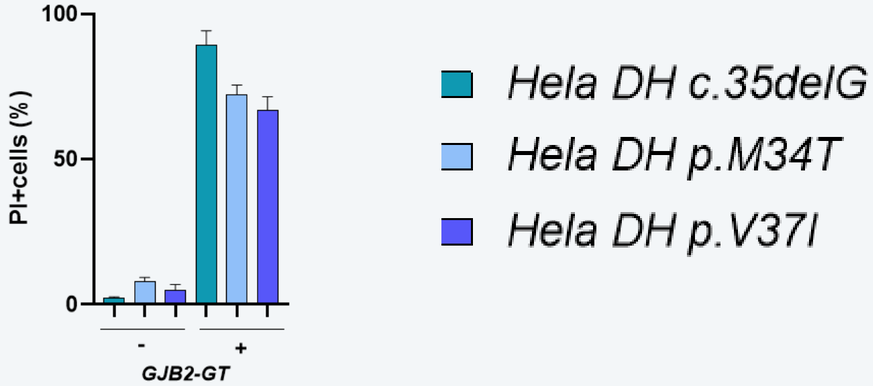
Restoration of functional CX26 gap junctions in vitro

Propidium iodide, in red, is a fluorescent dye that cannot cross intact cell membranes, so dye uptake is a direct measure of functional hemichannel assembly.

HeLa DH cells lacking CX26, or carrying a mutated CX26, showed no dye transfer.

Cells transduced with SENS-601 took up dye across all three variants tested: the truncating *c.35delG* and the missense *p.M34T* and *p.V37I*.





Functional gap junction permeability assay. Giese et al., ARO 2026.

Toxicology findings in NHP and mice

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.

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

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

Status

Regulatory

France: CTA approved 31 August 2026

Canada: CTA under review

United States: IND submission expected by YE 2026

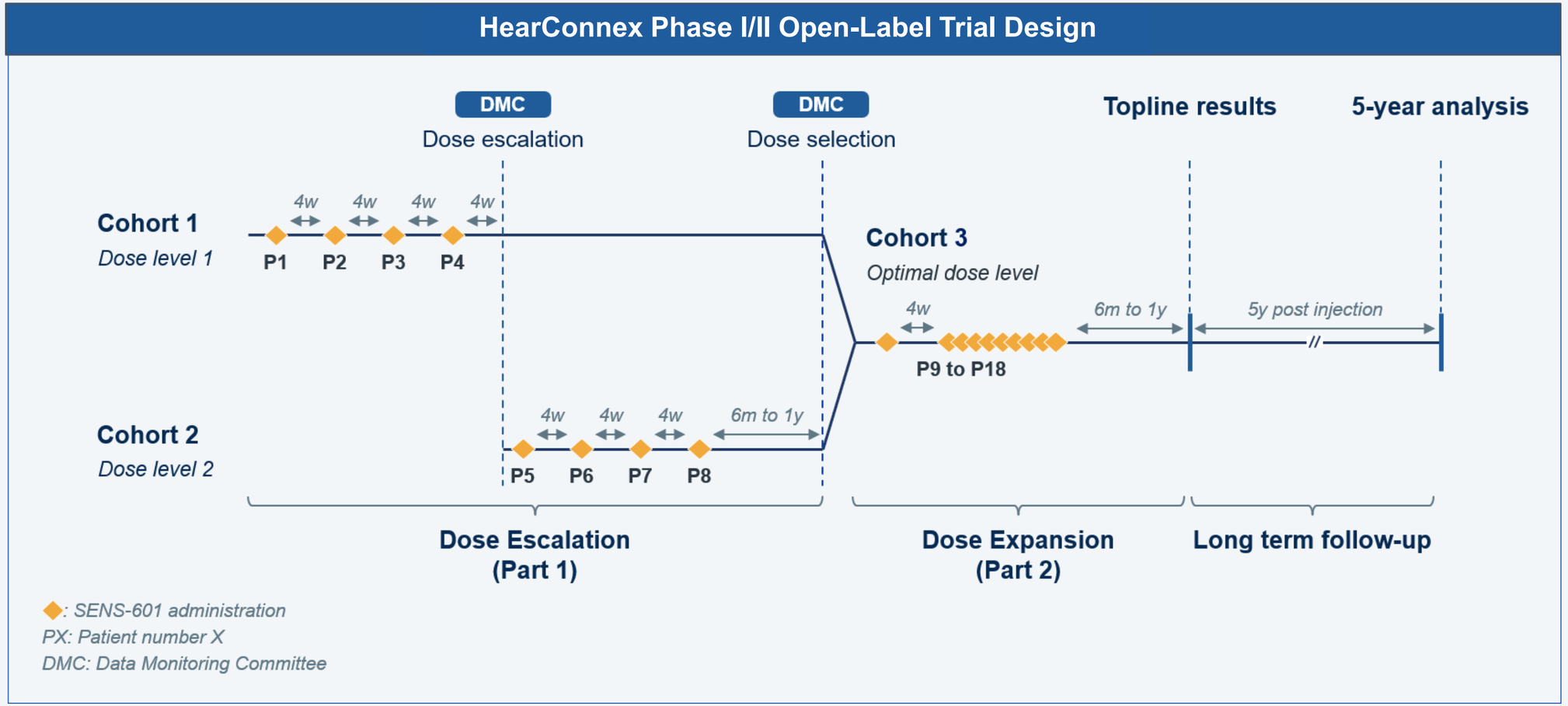
Australia: CTA submission expected by YE 2026

Enrollment

First patient dosing anticipated by early 2027

Clinical data generation expected throughout 2027

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.
Patients age:
Part 1 (Dose escalation): ≥ 6 months to ≤ 31 months, cochlear implant-naïve, ≥ 6 months to ≤ 6 years, already unilaterally implanted within 18 months ; Part 2 (Dose expansion): ≥ 6 months to ≤ 31 months, cochlear implant-naïve

Summary

Activity to date

CTA cleared in France, submitted in Canada

Congenital onset; Fast Track granted in France

Natural history study running

OTOCONEX in pediatrics in France and in other European countries

Near-term milestones

US IND and Australia CTA submission

Expected by end of 2026

Phase I/II trial initiation

Dosing of the first patient targeted by early 2027, with clinical data generation expected throughout 2027

Sensorion core development capabilities for SENS-601

01

Differentiated biological approach

- **Pan-GJB2 approach,** designed to address GJB2-related hearing loss across pathogenic GJB2 variants.
- **Proprietary regulatory sequence** drives expression in supporting cells while sparing sensory hair cells.

02

Validated surgical technique

- **Dual-fenestration approach** familiar to ENT surgeons, with a proprietary injection device delivering a controlled volume and flow rate.
- **Well tolerated** in the prior SENS-501 clinical program

03

Manufacturing

- **CDMO partnership** for clinical- and commercial-grade GMP supply.
- **Process development and analytics** in-house.

04

Clinical execution experience

- Operational experience in genetically defined hearing loss, built through the **Audiogene trial**.
- **OTOCONEX natural history study** feeding patient identification and recruitment.

05

HL strategic connectivity

- **Global network of clinical sites and hearing-loss key opinion leaders,** including a strategic collaboration with the Hearing Institute and Institut Pasteur.

ENT: ear, nose and throat. GMP: good manufacturing practice. CDMO: contract development and manufacturing organization. HL: hearing loss.

CORPORATE OVERVIEW

Team, governance and financial position

Executive team

Fred Chereau

Chief Executive Officer

At Sensorion since 2026. Previously President and CEO of LogicBio and SVP Strategy and Business Development at Alexion.

Laurent Desire

Head of Preclinical

At Sensorion since 2020. Previously Head of the Cellular and Molecular Biology Unit at Yposkesi.

Stephanie Filipe

Head of Business Ops and Portfolio Management

At Sensorion since 2020. Previously Program Leader and Preclinical Manager at Cellectis.

Bernd Schmidt

Chief Technical Officer

At Sensorion since 2024. Previously SVP Product Delivery at Quell Therapeutics.

Board of directors

Amit Munshi	Chairman of the Board, USA
Federico Mingozzi*	USA
Aniz Girach*	UK
Health Opportunities*	Represented by Eric de la Fortelle, Switzerland
Khalil Barrage	USA
Julien Miara	France
Sofinnova Partners	Represented by Cédric Moreau, France
Redmile Group	Represented by Florian Reinaud, France

*Independent Director, as determined under the French Middlenext Code.

Scientific advisory board

Pr Christine Petit

Chair of the SAB. Professor, Institut Pasteur, Hearing Institute/ Institut reConnect, France

Pr Alain Fischer

Professor, Collège de France, France

Dr Robert Dow

Chief Development Officer, Scendea, UK

Pr Paul Avan

Head of Center for Research and Innovation in Human Audioglogy, , Institut Pasteur, Hearing Institute/ Institut reConnect, France

Dr Diane Lazard

Head of ENT and Maxillofacial Surgery, Princess Grace Hospital, Monaco; Institut Pasteur, Hearing Institute, Institut reConnect, France

Dr Hernán López-Schier

Research Director, Research Innovation, NYU Abu Dhabi; Helmholtz Center Munich, Germany

Financial position

Cash and equivalents & short term deposits

€88 million

As of 30 June 2026

Cash runway

Year-end 2027

Shareholder base

Blue-chip healthcare specialist investors, alongside strategic investor Sanofi

Including Invus, Sofinnova Partners, Redmile Group, Cormorant Asset Management and Coastlands Capital.
Listed on Euronext Growth Paris.

Reported under IFRS.

Current status and expected milestones

Program	H2 2026	2027
SENS-601: congenital onset	CTA approved in France Canadian CTA submitted US IND and Australia CTA submission	Dosing of the first patient targeted by early 2027 Clinical data generation expected throughout 2027
SENS-601: pediatric progressive	Preclinical studies and patient identification advancing	
SENS-601: adult onset	Preclinical studies and patient identification advancing	

Sensorion at a glance

On a mission to enable people with inner ear hearing disorders to live life with unlimited connections.

SENS-601 is in the clinic, on a global development path

CTA approved in France submitted in Canada; US IND and Australia CTA submission planned by year-end 2026

Potential to disrupt a treatment landscape with no approved therapies

SENS-601 is designed to address GJB2-related hearing loss across pathogenic GJB2 variants

Deep domain expertise and key partners to strengthen our science and pipeline

Our ecosystem includes Institut Pasteur, Necker-Enfants Malades Hospital (AP-HP) and industry leaders

APPENDIX

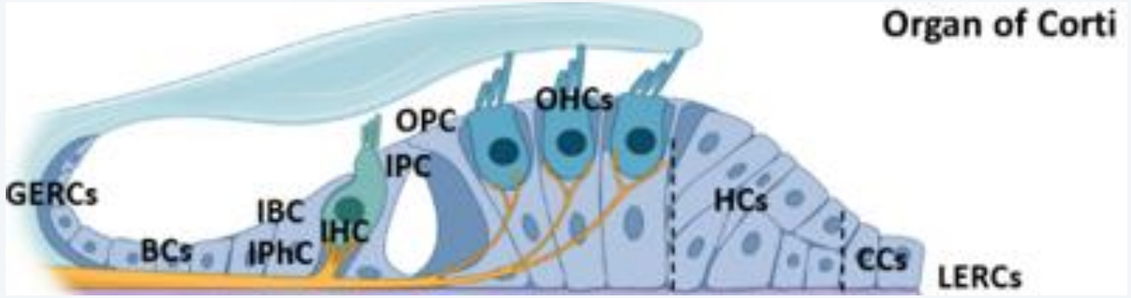
CX26 biology in detail

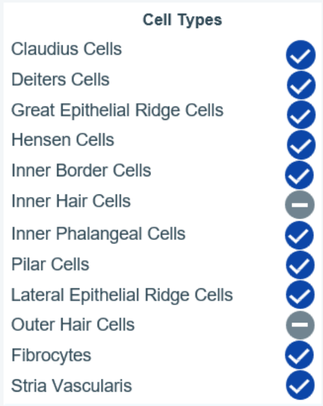
GJB2, which encodes CX26, is naturally expressed in the non-sensory supporting cells of the cochlea.

CX26 is the predominant connexin in the cochlea; its heteromeric or heterotypic hexamers form gap junctions.

GJB2 is absent in sensory hair cells, where functional gap junction channels would interfere with their specialized auditory signal transduction.

Cx26 gap junctions carry the intercellular exchange of metabolites needed for auditory tissue homeostasis, including ions such as K^+ required to maintain the steep electrochemical gradient driving sound transduction.





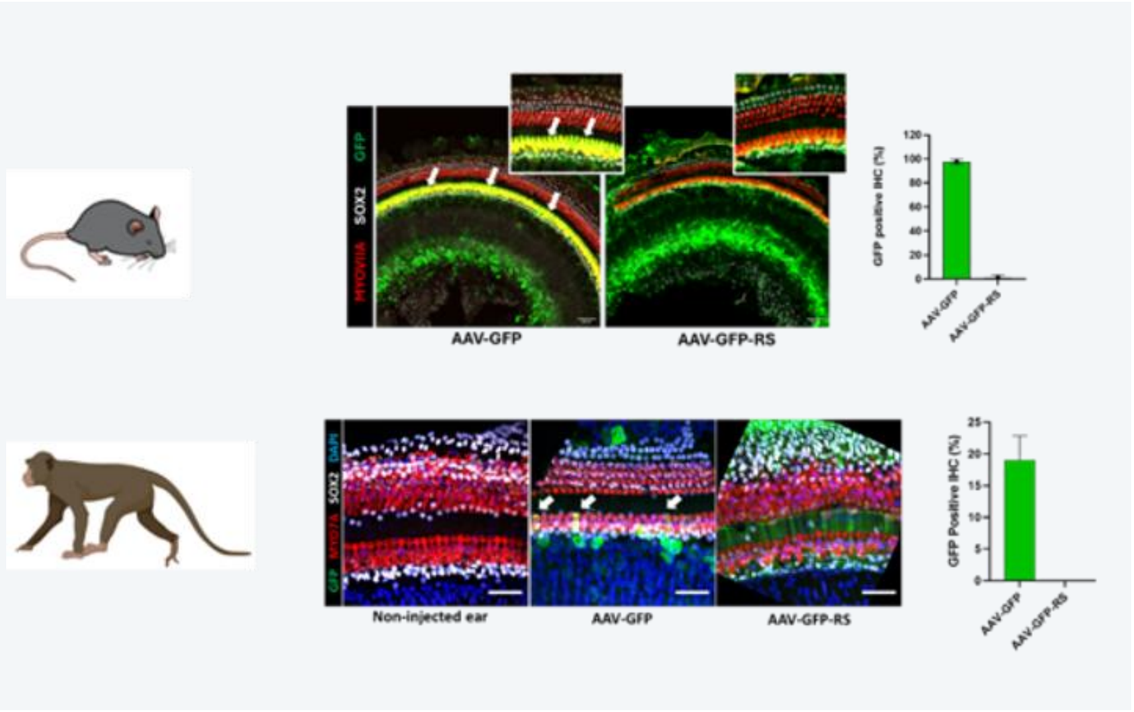
Kemperman, Hoefsloot and Cremers, J R Soc Med 2002;95:171–177. Chen et al., Front. Mol. Neurosci. 2022;15.

Construct design in detail

The transgene cassette comprises a promoter, a CX26 coding sequence and a proprietary regulatory sequence in the 3’ untranslated region.

The regulatory sequence is transcribed into mRNA complementary to miR-183, a small non-coding RNA occurring robustly in sensory inner hair cells but not in surrounding supporting cells.

Binding of miR-183 represses translation or triggers degradation of the Cx26 transgene mRNA, sparing CX26 expression in the sensory hair cells.



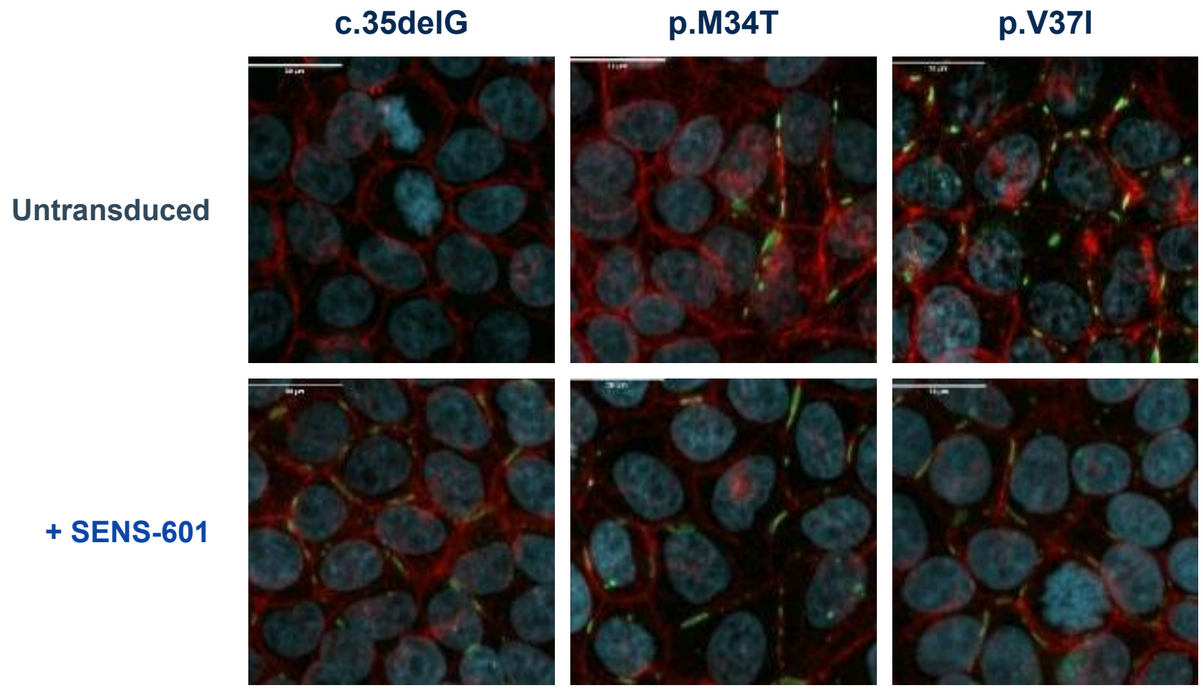
Regulatory-sequence selectivity in mouse (top) and non-human primate (bottom) cochlea: GFP-positive hair cells are strongly reduced with the regulatory sequence present.

Giese et al., ARO 2026

Gap junction permeability assay

HeLa DH cells are highly stable during electrophysiological testing, making them the industry standard for gap junction assays.

c.35delG is a truncating mutation associated with severe-to-profound hearing loss; p.M34T and p.V37I are missense variants associated with mild-to-moderate hearing loss.



Propidium iodide uptake in HeLa DH cells with and without SENS-601 transduction.

Giese et al., ARO 2026

SENS-401: an oral otoprotectant candidate

Orally available small molecule, the (R)-enantiomer of azasetron, with a dual mechanism: **5-HT$_3$ receptor antagonism with calcineurin-inhibitory activity.**

Designed to protect inner-ear sensory tissue.

Evaluated across three indications, each having completed proof-of-concept clinical testing.

Orphan drug designation
EMA[1] and FDA[2]

Pediatric investigation plan
Approved by EMA[3]

Status
Available for partnership

EMA: European Medicines Agency. FDA: U.S. Food and Drug Administration.
1: SSNHL indication ; 2: CIO indication ; 3: SSNHL and CIO indications

SENS-401 data package across three indications

Hearing preservation after cochlear implantation

Positive Phase IIa: 25 of 28 randomized patients implanted

Complete preservation of residual hearing observed in 40% of treated patients versus 0% of controls at six weeks post-implantation, maintained eight weeks after treatment stopped. SENS-401 detected in the perilymph of all treated patients.

Cisplatin-induced ototoxicity

Phase IIa completed: 47 randomized

Subgroup and post-hoc analyses suggested a potential otoprotective benefit in patients exposed to the highest cumulative cisplatin doses, above 300 mg/m².

Sudden sensorineural hearing loss

Phase IIb (AUDIBLE-S): 115 randomized

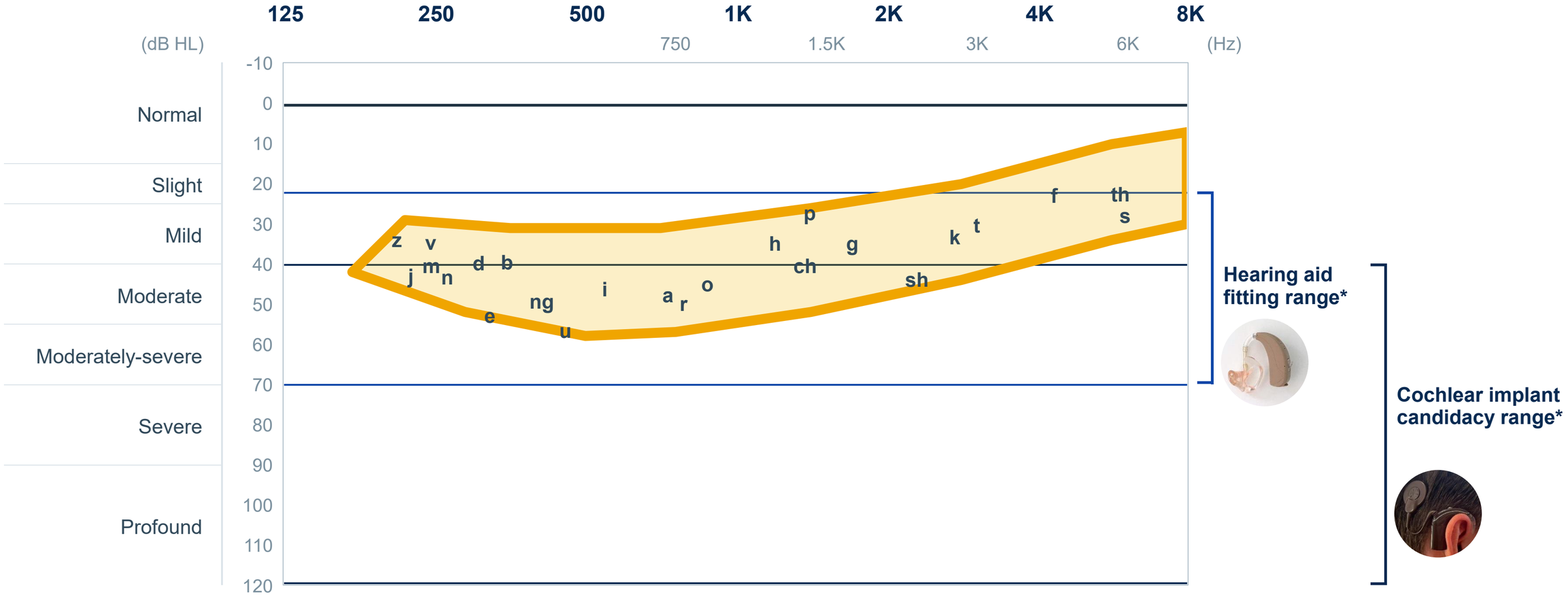
In AUDIBLE-S, a randomized, double-blind, placebo-controlled Phase IIb trial of 115 patients. Exploratory analyses suggested a potential treatment effect on secondary endpoints.

The hearing loss spectrum and standard of care

Hearing aids amplify sound, but do not restore the clarity lost when the cochlear supporting cell network fails.

Cochlear implants bypass the sensory epithelium entirely, and are indicated only toward the severe-to-profound end of the spectrum.

Access and clarity are both required for a good outcome. No approved therapy addresses the underlying cause.



*Guideline criteria may vary slightly by manufacturer, device and country.