AI-Enabled Nano-implants for Continuous Monitoring and Therapy

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Abstract

We propose a closed-loop platform in which externally generated long-range nondiffracting beams (LRNB) trap, steer and interrogate AI-controlled nano-implants inside the body, turning optical biopsy into continuous molecular surveillance and on-site therapy. The proposal builds on our analysis of LRNB for optical biopsy (SPIE Vol. 3917, 2000), which suggested that Bessel-type beams keep a narrow, non-spreading core over long distances and deliver a 1.4–4.6× fluorescence gain over Gaussian beams for fluorophores buried up to 100 mm deep, with higher contrast for small inhomogeneities.

We extend that result in four directions. First, the same non-spreading core forms an elongated optical trap (optical tweezers) that can hold and convey nano-implants along the beam axis, and its self-reconstruction lets it re-form behind scattering obstacles.

Second, the nano-implants carry molecular recognition elements (DNA probes, methylation-sensitive reporters, microRNA beacons, antibody and aptamer sensors) whose optical signature changes when early disease biomarkers appear. Third, optogenetic and photo-switchable modules let the same light that reads the implant also command it: release a drug, activate a gene circuit, or modulate excitable tissue. Fourth, an AI layer decodes the weak, scattered return signal, steers the beam, scores disease risk over time, and decides when to escalate from monitoring to therapy.

Target applications include early detection and treatment of ovarian, breast, prostate and brain cancers, Alzheimer's disease and cardiovascular disease. We set out the physics, the implant design, the molecular targets per disease, the AI architecture, the therapeutic modes, and the safety and engineering problems that must be solved before human use. The paper is a concept and research roadmap, not a report of a working system.

1. Introduction

Most serious diseases are found late because the tools that look for them sample the body rarely and from the outside. A tissue biopsy is invasive and samples a few cubic millimeters once. Imaging (MRI, CT, PET,

mammography) sees structure or bulk metabolism, usually after millions of cells have changed. Liquid biopsy (circulating tumor DNA, proteins) is sensitive but cannot say where a signal came from. Yet most pathologies begin as molecular events in a small, fixed place: a methylation shift in a fallopian-tube epithelium, a misfolded protein in the entorhinal cortex, an inflamed plaque in one coronary segment.

Optical biopsy reads tissue chemistry with light, but scattering limits it. Goldfain (2000) noted that beam divergence plus multiple scattering limit penetration depth and resolution, and that time-gated methods discard most photons in thick tissue; time-gated mammography, for example, struggles to resolve inhomogeneities below about 10 mm. His answer was a different beam: the long-range nondiffracting beam, whose narrow core does not spread with distance.

This paper asks what becomes possible if that beam is used not only to illuminate tissue but to *handle* objects in it. Light carries momentum, so a tightly confined beam can trap a particle (Ashkin, 1986); optical tweezers

have already held red blood cells inside the capillaries of living mice (Zhong et al., 2013). Combine an LRNB trap with engineered nano-implants that sense molecules and respond to light, and add AI to run the loop, and the result is a resident, remotely operated laboratory: it can be placed at a site of concern, report molecular change for months, and treat when the evidence warrants.

The guiding questions are those set for this work:

1. Can **long-range nondiffracting beams** (LNRB) reach and address sites deep in the body better than conventional beams?
2. Can **optical tweezing with LRNB** position, hold and retrieve nano-implants?
3. Can **optogenetics** and related light-gated chemistry turn the implant from a sensor into an actuator?
4. Can the system detect **early changes at the molecular level** (DNA, epigenetic marks, RNA, proteins) before disease is clinically visible?
5. What does **AI** add to assessment, monitoring and treatment?

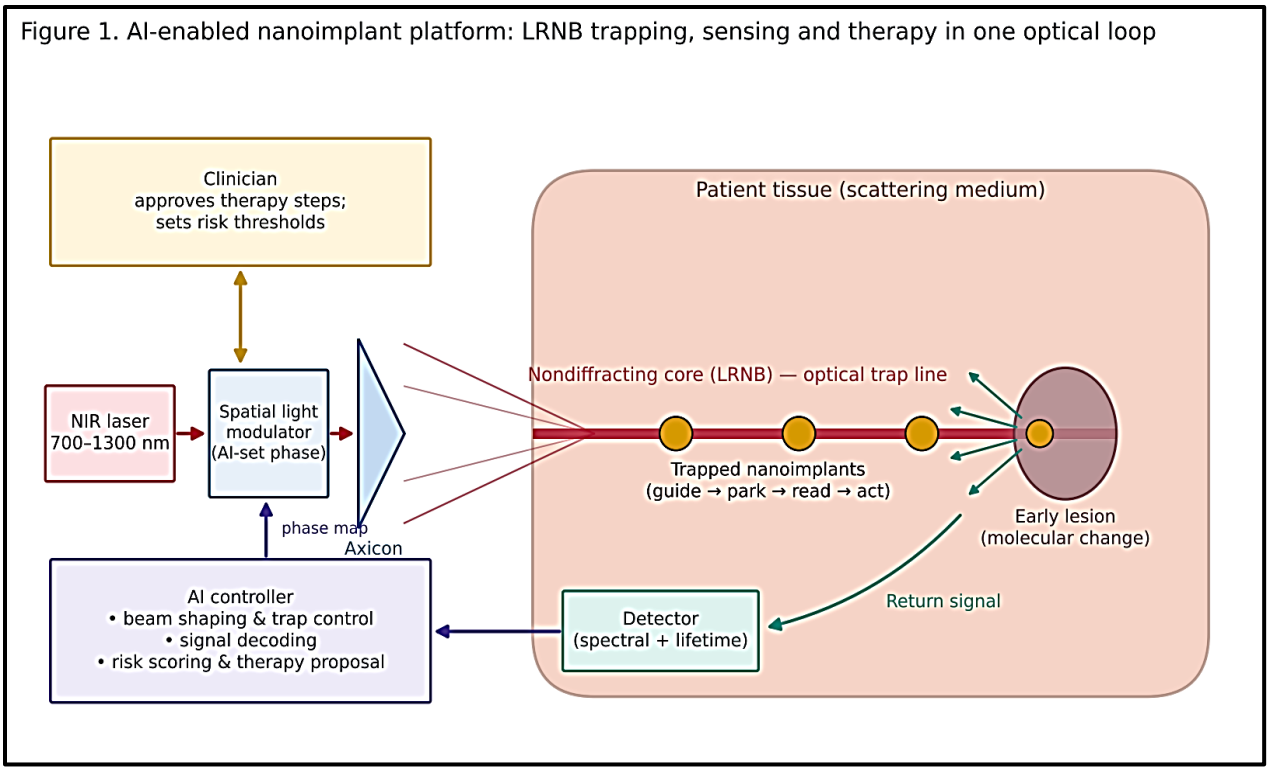


Figure 1*. An AI-shaped LRNB holds nano-implants on a trap line inside tissue; their light return is decoded by AI, which proposes therapy for clinician approval.*

2. Long-range nondiffracting beams

An LRNB keeps a narrow, bright core over a long distance because its light arrives on a cone and interferes constructively on the axis. The simplest case is the zero-order Bessel beam (Durnin, 1987), used by Goldfain:

$$E(x, y, z, t) = E_0 \cdot J_0(\alpha\rho) \cdot \exp[-i(\omega t - \beta z)], \quad \text{where } \rho = \sqrt{(x^2 + y^2)}$$

The transverse profile $J0(\alpha\varrho)^2$ does not depend on z, so the central spot keeps its width (set by α) as it travels. With custom aperture transmission functions, the axial intensity can also be made to rise or fall linearly with depth:

$$I(x, y, z) = I_0 \cdot (a + bz) \cdot [J_0(\alpha\rho)]^2, \quad with\ a + bz > 0$$

In practice the beam is made with an annular slit in the focal plane of a lens, a refractive or holographic axicon (a conical lens), a phase filter, or deliberately spherically aberrated optics. The axicon cone angle or the amount of aberration sets the depth range. Today a spatial light modulator can also generate it and reshape it in milliseconds, which matters for AI-driven steering (Section 7).

What our simulations show

We compared Gaussian and LRNB excitation of a fluorophore buried in a semi-infinite, highly scattering slab, with equal average focal-spot intensity for both beams (λ = 920 nm). Three results carry over to nano-implants:

- **Signal gain with depth.** The ratio g of LRNB to Gaussian fluorescence rose with fluorophore depth, from about 1.4 to about 4.6 over 0–100 mm, for core parameters α = 12.5–18.8 mm^{-1} and spot radii $\varrho 0$ = 0.10–0.15 mm. Smaller cores gave more gain.
- **A narrow focal line instead of a point.** In the 3D ballistic intensity plots, the Gaussian beam peaks at its waist and spreads beyond it, while the LRNB core stays narrow through and past an inhomogeneity buried 50 mm deep.
- **Better contrast for small targets.** Monte Carlo ray tracing (2×10^6 rays, 100 mm tissue, λ = 700 nm, axicon versus plano-convex lens) gave higher contrast for LRNB as the absorbing inhomogeneity shrank to a few millimeters.

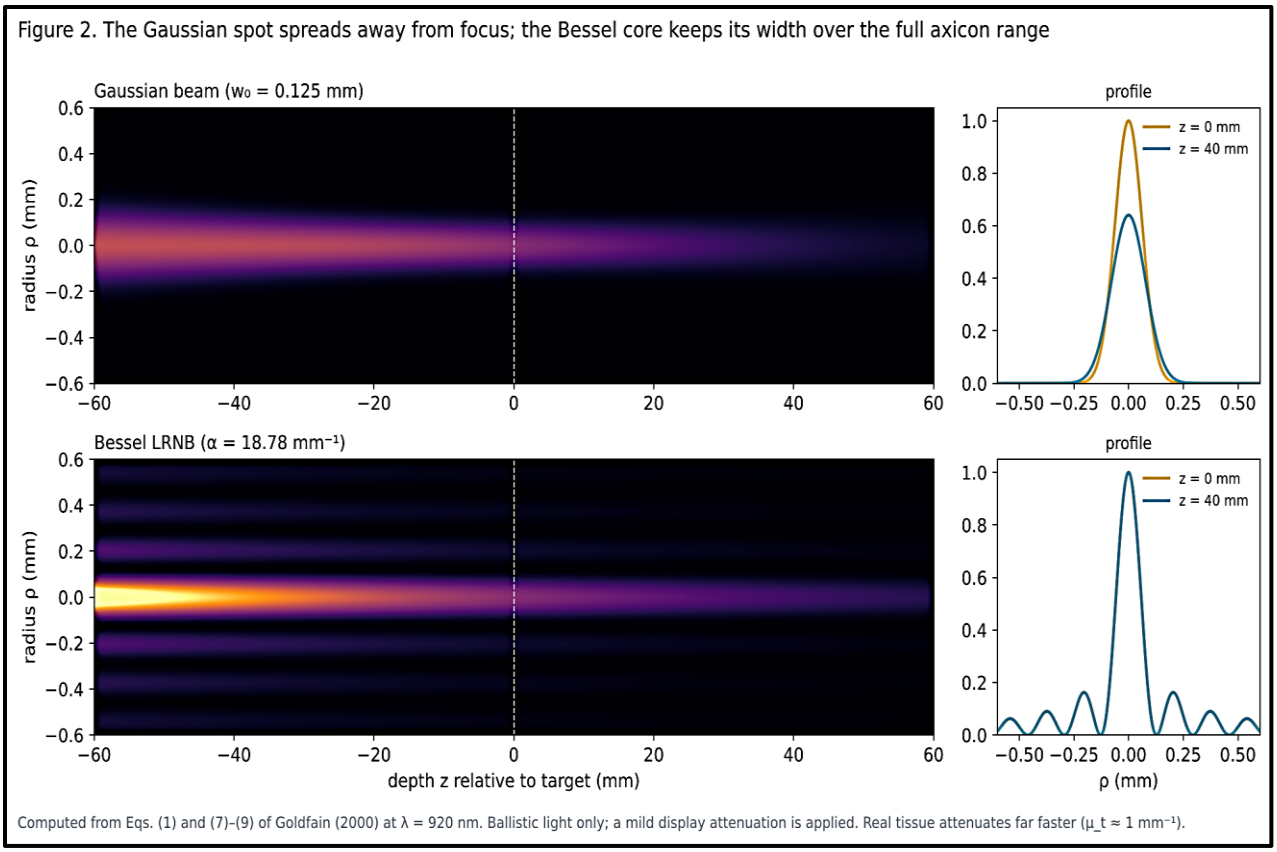


Figure 2*. Computed ballistic intensity for a Gaussian beam and a Bessel LRNB using our parameters (λ = 920 nm). The Bessel core keeps the same profile at 0 and 40 mm; the Gaussian peak drops and widens.*

We've listed diagnosis, laser surgery and photodynamic therapy as clinical uses and called for experimental validation. Later work supports the core idea: Bessel beams **self-reconstruct** after an obstacle, because the cone of light refills the axis behind it (Garcés-Chávez et al., 2002), and self-

reconstructing Bessel beams give sharper light-sheet images deep in scattering samples (Fahrbach et al., 2010; Planchon et al., 2011).

Limits to keep in view

The gains are for the ballistic component. In real tissue that component falls by roughly e^(−μ_t z); our Monte Carlo used μ_t ≈ 1.04 mm^{-1}. Ballistic light therefore runs out within millimeters to a few centimeters, depending on wavelength. Three choices push this back: working in the near-infrared windows (650–950 nm and 1000–1700 nm, where absorption and scattering are lowest; Weissleder, 2001), delivering the beam closer to the target (endoscope, catheter, laparoscope or a fiber placed by needle), and adaptive wavefront shaping that pre-corrects scattering. A realistic design uses external LRNB for shallow organs (breast, prostate via transrectal probe, superficial vessels) and minimally invasive delivery for deep ones (ovary, brain, coronary arteries).

3. Optical tweezing with LRNB

An LRNB core is a long, thin optical trap: it holds a nano-implant sideways with a gradient force and can push or pull it along the axis, so one beam can deliver, park and retrieve implants. Two forces act on a particle in a beam:

- **Gradient force** pulls a particle whose refractive index exceeds the surroundings toward the brightest region. In a Bessel beam this is the central core (and, more weakly, each ring), so the particle is confined radially.
- **Scattering force** (radiation pressure) pushes the particle along the direction of propagation.

In a conventional tweezer, a high-numerical-aperture lens makes the gradient force win in all three directions, giving a point trap at the focus. That needs a tight focus, which scattering tissue destroys within a fraction of a millimeter. A Bessel core instead confines radially over its whole

length, while the scattering force acts as a conveyor along it. Garcés-Chávez et al. (2002) used this to trap and stack particles in several separate sample chambers along one Bessel beam, with the beam re-forming behind each trapped particle.

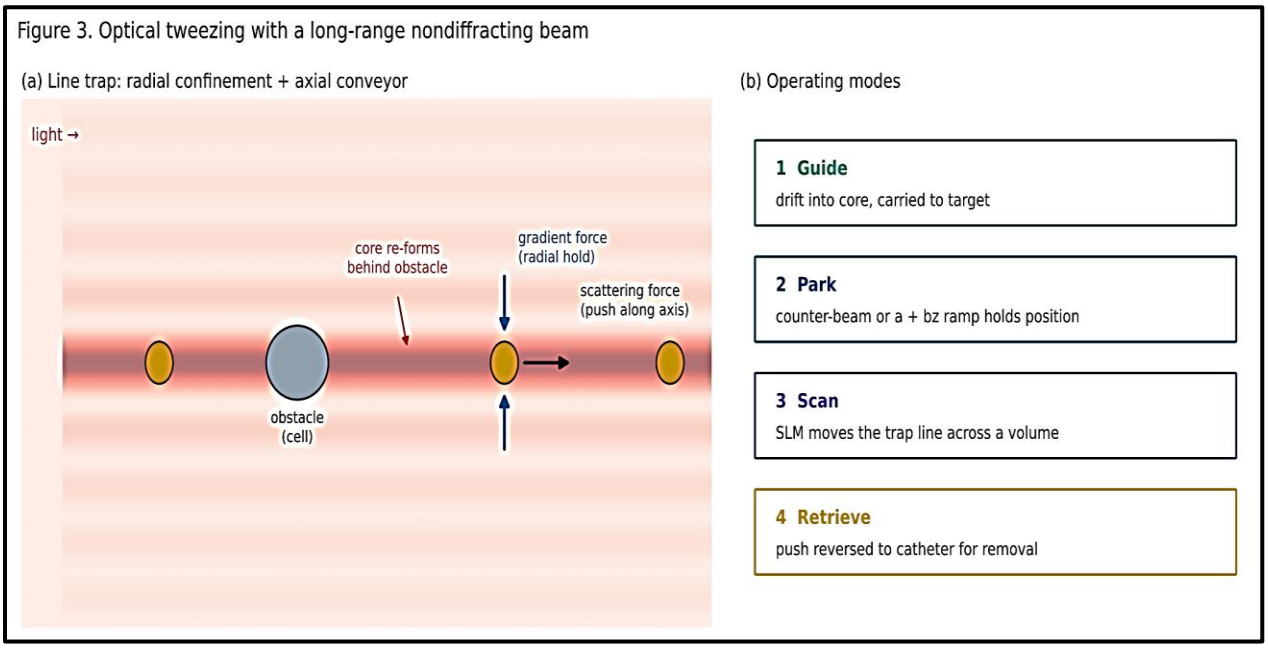


***Figure 3**. (a) The Bessel core confines implants radially and pushes them along the axis and re-forms behind an obstacle. (b) The four operating modes.*

Operating modes for nano-implants

1. **Guide.** Implants injected into blood, lymph, cerebrospinal or peritoneal fluid drift until they enter the core, then are held on the beam line and pushed toward the target.
2. **Park.** Axial position is held by balancing the forward push with a counter-propagating LRNB, a standing-wave pattern, or the a + bz intensity ramp from Section 2, which creates an axial force gradient. Implants can also be fixed by a binding chemistry once in place.
3. **Scan.** Steering the cone angle or the beam's lateral offset (by spatial light modulator) moves the trap through a volume, letting one implant sample several sites.
4. **Retrieve.** Reversing the push moves implants back to a collection point, such as a catheter tip, for removal or ex vivo reading.

Why LRNB and not a focused beam

Property	Focused Gaussian tweezer	LRNB tweezer
Trap shape	Point near focus	Line, mm to cm long
Behaviour after an obstacle	Focus degraded	Core re-forms (self-reconstruction)
Depth range per setting	~ Rayleigh range (μm)	Set by axicon angle (mm–cm)
Many implants on one beam	Needs multiple foci	Several along the same line
Axial control	Strong 3D trap	Conveyor; needs counter-beam or ramp to hold

Physical limits

Trap force scales with optical power reaching the particle and the particle's polarizability. In tissue, only the ballistic and weakly scattered light builds

the core, so usable force falls rapidly with depth. Typical optical-tweezer forces are piconewtons; blood-flow drag on a 200 nm particle in a capillary is in the same range, but in arteries it is far larger. Practical guidance therefore means either low-flow compartments (interstitial fluid, lymph, CSF, peritoneal fluid, capillary beds), short-guided paths from a nearby catheter or fiber, or implants given magnetic or acoustic handles so that light does the fine positioning and other fields do the bulk transport. Heating limits power: tissue exposure must stay within laser-safety limits, which favors the 1000–1300 nm window, where water absorption is still modest.

4. Nano-implant architecture

Each nano-implant is a 50–500 nm particle built in layers: a core that the beam can grab and that converts light, a recognition shell that binds a biomarker, a reporter that changes the light sent back, and an optional payload the light can release. The intelligence sits outside the body; the implant is kept passive so it needs no battery or electronics.

Layer	Function	Candidate materials
Core	Optical handle (high refractive index for trapping); energy converter	Silica, polystyrene, gold or silver nanoshells, lanthanide up-conversion nanoparticles (UCNP), magnetic iron-oxide inclusion for coarse transport
Stealth coat	Avoid immune clearance, protein fouling	PEG, zwitterionic polymers, cell-membrane cloaking
Recognition shell	Bind a target molecule selectively	DNA/RNA hybridisation probes, molecular beacons, CRISPR–Cas12/13 reporters, aptamers, antibodies or nanobodies, methyl-CpG-binding domains
Reporter	Turn binding	FRET dye pairs, quantum dots,

Layer	Function	Candidate materials
	into an optical signal	plasmonic SERS tags, lifetime-encoded fluorophores, NIR-II emitters
Payload / actuator	Act when commanded by light	Photo-cleavable drug linkers, photosensitisers (PDT), photothermal gold, light-triggered siRNA, or upconversion particles that drive optogenetic proteins (Section 5)

Figure 4. Layered nanoimplant: sense, report and act on command by light

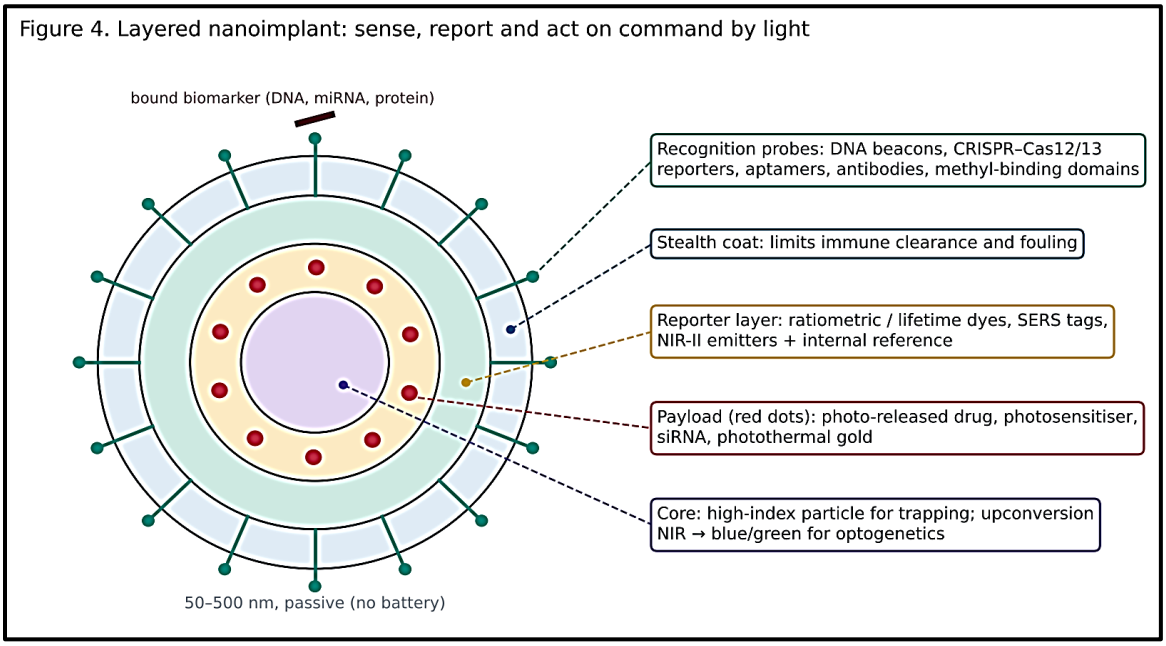


***Figure 4**. Layered, passive nano-implant: core, reporter with payload, recognition probes and stealth coat.*

How an implant talks back

The beam does three jobs at once: it holds the implant, it excites the reporter, and it collects the return. The read-out is a spectral and temporal fingerprint, not an image:

- **Ratiometric fluorescence.** Two emission bands, one fixed reference and one target-sensitive, so the ratio is robust to how much light reaches the implant.
- **Fluorescence lifetime.** Lifetime does not depend on intensity, so it survives scattering; binding events shift it measurably.
- **Surface-enhanced Raman (SERS).** Gold or silver tags give sharp vibrational spectra that can be multiplexed, so one implant can report many targets.

- **Barcoding.** Each implant carries a unique spectral or lifetime code, so many implants at different sites, or with different targets, are read at once and told apart.

Our equation for the surface signal, $F(0) = I_s \int E(0, z_f) K(z_f) \mu_{a,f}(z_f) dz_f$, still governs detection: the return must cross the same tissue. This is why the reporter design favors bright, long-wavelength, lifetime- or ratio-coded emission, and why the AI decoder in Section 7 is central rather than an add-on.

Placement routes

Implants reach a site by intravenous injection with targeting ligands, then LRNB guidance for the last centimeters; by local injection (intraperitoneal for ovary, intraductal for breast, intrathecal for brain); or by catheter or fiber delivery with on-site optical release. Most implants would be designed for slow biodegradation (silica, calcium phosphate,

biodegradable polymers) and retrieval or clearance after a set monitoring period.

5. Optogenetics: light that commands cells

Optogenetics gives cells light-sensitive proteins so that light switches their activity on or off; here it turns the nano-implant from a sensor into a remote control. Channelrhodopsin-2 made neurons fire on demand with millisecond blue-light pulses (Boyden et al., 2005). Since then the toolkit has grown to include inhibitory pumps, red-shifted opsins, and light-switchable gene expression (for example, blue-light CRY2/CIB1 and red/far-red phytochrome systems).

The obstacle is depth: most opsins respond to blue or green light, which tissue absorbs within a millimeter. Up conversion nanoparticles solve this. They absorb near-infrared light, which penetrates well, and emit blue or green light locally. Chen et al. (2018) used this to stimulate deep-brain neurons in mice with near-infrared light delivered from outside the skull.

An LRNB-trapped UCNP is therefore a natural optogenetic relay: the beam that positions it also powers it, and its narrow core limits stimulation to the implant's neighborhood.

Uses in this platform

Goal	Mechanism	Example
Neuromodulation	Opsin-expressing neurons near a UCNP implant	Restore gamma rhythms in Alzheimer's models; suppress seizure foci near brain tumors
Cardiac rhythm control	Opsins in cardiomyocytes or pacing cells	Optical pacing or termination of arrhythmias (shown in animal hearts)
Gene-circuit therapy	Light-switchable promoters in engineered or	Express an anti-tumor cytokine only at the tumour, only when lit

Goal	Mechanism	Example
	resident cells	
Immune activation	Light-gated CAR-T or calcium channels in T cells	Switch engineered T cells on at the lesion, limiting systemic toxicity
Sensing (reverse direction)	Genetically encoded calcium or voltage reporters	Implant reads local cell activity as a functional biomarker

Optogenetics requires gene delivery (usually a viral vector) and has reached humans only in narrow settings (for example, a vision-restoration trial in retinitis pigmentosa; Sahel et al., 2021). For near-term work, the same light-command principle can be applied without genetic change: photo-cleavable drug cages, photosensitizers for photodynamic therapy, and photothermal particles respond to light directly. We treat optogenetics as the long-range option and photochemistry as the near-term one.

6. Probing early changes at the molecular level

Disease starts as a local molecular change long before a lump, a symptom or an image appears, and an implant parked at the right site can read that change where it happens. Implants sample the local microenvironment: cell-free DNA and RNA shed by nearby cells, extracellular vesicles (exosomes), secreted proteins, metabolites and pH. Implants designed for cell uptake can also read intracellular RNA. Because the signal is measured at its source, concentrations are far higher than in blood, and a positive reading comes with a location.

What the implant can measure

- **DNA sequence changes.** Point mutations and fusions in shed DNA, read by hybridization beacons or CRISPR–Cas12 reporters that light up only on a matching sequence.

- **Epigenetic marks.** Promoter hypermethylation is often among the earliest changes in cancer; methyl-binding probes or methylation-sensitive beacons can report it.
- **RNA and microRNA.** Fusion transcripts and disease-linked microRNAs, read by molecular beacons or Cas13 reporters.
- **Proteins and aggregates.** Antibody, nanobody or aptamer sensors for secreted proteins, protein fragments and misfolded aggregates.
- **Metabolites and microenvironment.** Oncometabolites, reactive oxygen species, protease activity and pH, read by activatable dyes and Raman spectra.

Target panel by condition

Condition	Placement site	Early molecular targets	Why early detection matters
Ovarian	Fallopian-tube	TP53 mutations	Most cases are

Condition	Placement site	Early molecular targets	Why early detection matters
cancer (high-grade serous)	fimbria and ovarian surface, by intraperitoneal or transvaginal route	(present in nearly all cases and in precursor STIC lesions); BRCA1/2 status; promoter methylation markers; CA-125 and HE4 proteins	found at stage III–IV; precursors start in the tube
Breast cancer	Ducts and lobules of high-risk breast, by intraductal or percutaneous route	PIK3CA, TP53, BRCA1/2 variants; HER2 amplification; RASSF1A and other methylation; miR-21, miR-155	Dense breasts hide small lesions on mammography ; BRCA carriers need frequent

Condition	Placement site	Early molecular targets	Why early detection matters
			surveillance
Prostate cancer	Peripheral zone, by transrectal or trans-perineal route	TMPRSS2–ERG fusion (about half of cases); GSTP1 hypermethylation (most cases); PCA3 RNA; PSMA expression	Separates aggressive from indolent disease; reduces repeat biopsies and over-treatment
Brain tumors (glioma)	Resection cavity or high-risk region, intrathecal or at surgery	IDH1 R132H mutation and its metabolite 2-hydroxyglutarate; MGMT promoter methylation;	Early recurrence detection after surgery; tracks treatment

Condition	Placement site	Early molecular targets	Why early detection matters
		EGFRvIII; TERT promoter mutations	response without repeat MRI or biopsy
Alzheimer's disease	Cerebrospinal fluid spaces or near hippocampus/entorhinal cortex	Amyloid-β 42 oligomers, phosphorylated tau (p-tau217, p-tau181), GFAP and neurofilament light	Molecular changes start 15–20 years before dementia; new anti-amyloid drugs work best early
Cardiovascular disease	Coronary or carotid plaque,	Oxidised LDL, matrix metalloproteinase-9	Flags a plaque likely to

Condition	Placement site	Early molecular targets	Why early detection matters
	by catheter; myocardium	and myeloperoxidase activity (plaque instability), IL-6 and inflammation markers, cardiac microRNAs (miR-1, miR-133, miR-208a)	rupture before a heart attack or stroke; detects early heart-muscle injury

Monitoring logic

One reading is rarely decisive. The value lies in a time series from the same site: a stable baseline, then a sustained rise in a mutation fraction, a methylation index or an aggregate signal. Each implant carries an internal reference reporter so drift, fouling and changes in light delivery can be separated from true biology. Multiplexed panels (for example, mutation +

methylation + protein) reduce false positives, because a real early lesion moves several markers together.

7. The AI layer

AI makes the system usable: the physical signal from a deep implant is faint, scattered and confounded, and no fixed rule can steer the beam, decode the reporters and judge disease risk at once. We assign AI four jobs, each with its own model and its own checks.

1. Beam formation and steering. Scattering tissue scrambles any beam differently in each patient and over time. A controller learns the tissue's transmission behaviour from feedback (the implant's own return signal is a "guide star") and sets the spatial light modulator to rebuild the Bessel core at the implant. Reinforcement learning or model-predictive control then holds the trap against breathing, heartbeat and flow, and plans guide-park-scan-retrieve paths. Hard limits on power and dwell time are enforced outside the learned controller.

2. Signal decoding. Deep networks trained on simulated and phantom data (Monte Carlo light transport of the kind Goldfain used, now with full tissue models) separate the implant's spectral and lifetime fingerprint from tissue autofluorescence and background. They un-mix barcodes from many implants, correct for depth and fouling using the internal reference reporter, and output calibrated concentrations with uncertainty.

3. Longitudinal risk assessment. For each patient the system builds a baseline, then tracks change. Time-series models (state-space or transformer models) flag sustained, multi-marker shifts and combine them with clinical data (age, family history, BRCA status, imaging, PSA, lipid profile) into a risk score per site and condition. The output is a probability with its evidence, not a diagnosis.

4. Therapy decision support. When risk crosses a threshold the clinician has set, the system proposes an action (repeat reading, confirmatory imaging or biopsy, or local light-triggered treatment) and the dose. A human clinician approves any therapeutic step; the system can act

autonomously only within a pre-approved, low-risk envelope (for example, adjusting monitoring frequency).

Design principles

- **Physics-informed models.** Embed the diffusion and Bessel-beam equations so models stay plausible outside their training data.
- **Calibrated uncertainty.** Every reading carries confidence; low confidence triggers re-measurement, not action.
- **Explainability.** Each alert shows which markers moved, by how much, and against which baseline.
- **Federated learning.** Models improve across hospitals without moving raw patient data.
- **Safety interlocks.** Laser power, thermal dose and therapy release are bounded by hardware interlocks that the AI cannot override.

8. From monitoring to therapy

The implant that detects a change can treat it on the spot, because the same beam that reads it can trigger its payload, and the narrow LRNB core confines the effect to a sub-millimeter region. Goldfain already named laser surgery and photodynamic therapy as LRNB applications; implants make these molecularly targeted.

Mode	Trigger	Effect	Best suited to
Photo-released drug	Light cleaves a linker or opens a shell	Chemotherapy, PARP inhibitor, anti-inflammatory or anti-thrombotic released only at the site	Early ovarian, breast and prostate lesions; inflamed plaques
Photodynamic therapy (PDT)	Light activates a photosensitizer	Reactive oxygen species kill nearby cells	Small tumor foci, glioma margins

Mode	Trigger	Effect	Best suited to
Photothermal therapy	Gold nanoshells absorb near-infrared light	Local heating (about 42–50 °C) kills cells or sensitizes them to drugs	Prostate and breast lesions
Gene silencing	Light releases siRNA or antisense oligonucleotides	Knocks down an oncogene or inflammatory gene	Mutation-driven early cancers; plaque inflammation
Optogenetic modulation	UCNP converts NIR to visible light for opsins	Neural, cardiac or immune cell control (Section 5)	Alzheimer's circuits, arrhythmia, immunotherapy
Mechanical action	Tweezer-driven implant	Micro-disruption of a clot or local mixing of	Microvascular thrombosis

Mode	Trigger	Effect	Best suited to
	motion	a drug	(exploratory)

The closed loop

1. **Sense:** the implant reports its marker panel at set intervals.
2. **Decode:** AI converts the return light into calibrated marker levels with uncertainty.
3. **Assess:** AI updates the risk score against the patient's baseline.
4. **Decide:** above a threshold, AI proposes an action; the clinician approves.
5. **Act:** the beam delivers a measured light dose to release or activate the payload.
6. **Verify:** the implant reads the same markers again; the dose is adjusted or stopped.

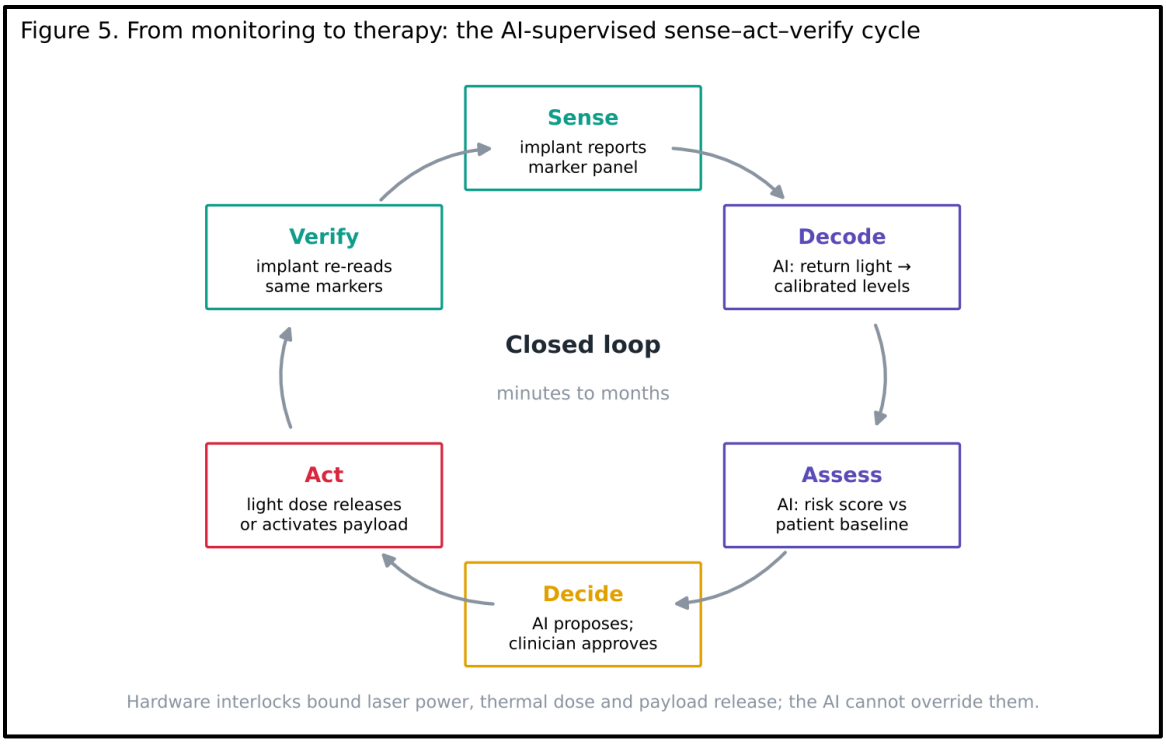


Figure 5*. The AI-supervised sense–decode–assess–decide–act–verify cycle, bounded by hardware interlocks.*

The verify step is what distinguishes this from conventional targeted therapy: the response is measured at the lesion within minutes to days, rather than inferred months later from imaging.

9. Safety, open challenges and roadmap

The concept is physically sound, but depth, trapping force in flowing fluid, and long-term biocompatibility are the hard problems, and each needs experimental proof before clinical claims.

Main challenges

- **Depth of the ballistic core.** Our gains assume the ballistic component survives. Beyond a few centimeters it largely does not, so deep targets need catheter, endoscopic or fiber delivery, or wavefront shaping that has yet to be shown at clinical depth and speed.
- **Trap stiffness versus flow and motion.** Piconewton optical forces are easily overwhelmed in large vessels and moving organs; hybrid magnetic or acoustic transport is likely needed.
- **Thermal and photochemical safety.** Continuous trapping deposits heat. Power must stay within laser exposure limits for skin and tissue, and photosensitizers must not activate off-target.

- **Biocompatibility and fate.** Immune clearance, protein fouling, inflammation, toxicity of lanthanide or gold cores, and long-term accumulation must be characterized; biodegradable designs are preferred.
- **Sensor drift and specificity.** Fouling and probe degradation cause drift over weeks; local biomarker levels may vary with benign inflammation. Reference reporters, multiplexing and AI calibration mitigate but do not remove this.
- **Optogenetic gene delivery.** Viral vectors add immunological and regulatory risk; non-genetic photochemistry is the near-term path.
- **AI validation.** Models trained largely on simulations must be validated on phantoms, animals and then patients; bias across skin tones and tissue types (which change light transport) must be measured.
- **Regulation and ethics.** The system is a drug–device–software combination product. Continuous molecular surveillance raises consent, data ownership and incidental-finding questions.

Roadmap

Phase	Goal	Success criterion
1. Bench	LRNB trapping and read-out of reporter nanoparticles in tissue phantoms and ex vivo tissue	Stable trap and ratiometric read-out at 5–20 mm depth
2. Small animal	Guided placement, multi-week sensing, light-triggered release in mouse tumor, Alzheimer's and atherosclerosis models	Detection earlier than imaging; local therapy response verified by implant
3. Large animal	Catheter/endoscope-assisted delivery at human-scale depths; safety and clearance	No significant toxicity; read-out reproducible across animals
4. First-in-human	Accessible sites first: prostate (transrectal), breast, superficial vessels, resection cavities	Safety; agreement with biopsy and standard biomarkers

10. Conclusion

Long-range nondiffracting beams (LNRB) can do more than illuminate tissue: their narrow, self-reconstructing core can carry, hold and read nano-implants, and an AI controller can turn those implants into a resident system for molecular assessment, monitoring and therapy. Our 2000 analysis supplies the optical foundation, showing deeper reach and better contrast than Gaussian beams for small, buried targets. Optical tweezing, up-conversion-driven optogenetics and molecular reporters supply the handling, actuation and sensing. AI supplies beam control, signal decoding, longitudinal risk scoring and supervised therapy decisions.

The payoff would be detection of cancer, neurodegeneration and vascular disease at the stage of a mutation, a methylation shift or a misfolded protein, with a location attached, followed by treatment confined to that location and verified by the same implant. The obstacles (ballistic depth, trap force in flow, biocompatibility and validation) are real, and the

roadmap starts where physics is most favorable: shallow and catheter-accessible sites.

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Figures 1–5 are original schematics prepared for this paper; Figure 2 is computed from the beam equations in Section 2.