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# Beyond Checkpoint Inhibitors: Reimagining the Future of Cancer Immunotherapy

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**Alex Shneider, Founder and CEO of CureLab Oncology, discusses DNA-based immunotherapies, inflammation-driven cancer biology, intelligent combination therapies, and the emerging science of reprogramming the tumour microenvironment.**


Cancer immunotherapy is entering a new phase—one that extends beyond targeting individual tumour cells and towards reshaping the biological systems that enable cancer to survive and evolve. Advances in DNA-based therapeutics, tumour microenvironment engineering, chronic inflammation research, and personalised neoantigen medicine are opening new avenues for treatment design. In this conversation with **BioSpectrum Asia during BIO International Convention 2026, Alex Shneider, Founder and CEO of CureLab Oncology,** shares his perspectives on the next generation of cancer immunotherapies, the growing importance of rational combination strategies, and why the future may lie in restoring healthy cellular communication rather than simply blocking disease pathways.

### How could DNA-based immunotherapies reshape the next generation of cancer treatment strategies?

It is impossible to predict every future trajectory, but the pillars of the next generation of medicine are already taking shape.

**Intracellular delivery and personalized neoantigen medicine.** In the early days of genetic medicine, DNA and RNA therapies were deployed simply to encode cancer-associated antigens — a strategy that remains fundamentally powerful. An antigen expressed natively inside a patient's own cells undergoes the precise post-translational modifications, conformational assembly, and immune presentation required to drive robust, dual B-cell and T-cell immunity. This stands in sharp contrast to traditional recombinant proteins, which elicit a predominantly antibody-restricted response. Deep sequencing has since opened the era of personalized neoantigen medicine — a frontier that, in my view, is most realistically navigated via DNA/RNA technology. Early in my career, my colleagues and I focused not merely on selecting the optimal target, but on strengthening the host's immune response to the encoding DNA itself — leveraging antigen aggregation, controlled proteasomal processing, and heterologous prime-boost regimens. I expect these threads to converge: highly personalized neoantigens paired with molecular machinery that systematically amplifies the immune response against them.

**From static dosing to smart, dynamic constructs.** A second direction is the use of plasmids and vectors to deliver localized immunostimulatory molecules, such as plasmids driving IL-12 or complementary cytokines. Looking further ahead, I believe the future belongs to genuinely dynamic constructs: vectors that do not merely express immune stimulators or silence immunosuppressive pathways at a fixed level, but modulate that expression in response to the changing homeostatic state of the host or the evolving tumor microenvironment. This is the shift from therapies that blindly execute a script to therapies that sense, adapt, and respond.

**Remodeling the asymmetric tumor microenvironment.** Third, we are seeing a meaningful shift toward using DNA/RNA vectors to remodel the tumor microenvironment itself — attacking the malignant cells directly while simultaneously stripping the tumor of its protective, immunosuppressive shield to render it susceptible to companion therapies. This work will benefit enormously from our rapidly advancing ability to map spatial transcriptomics. Tumors are highly structured, asymmetric, and compartmentalized ecosystems rather than uniform cellular masses, and our therapies must be engineered to disrupt that specific spatial defense.

These dimensions are not mutually exclusive; the most effective agents will likely combine several of these mechanisms within a single vehicle. This philosophy of deliberate convergence is the thesis behind our own platform, Elenagen, which brings several of these synergistic mechanisms into a single, multi-targeted DNA-plasmid design — aiming not just to attack a single pathway, but to alter the broader landscape of the tumor.

### What role does chronic inflammation play in tumour progression, and why has it become an increasingly important therapeutic target?

Chronic inflammation is now understood not as a bystander to cancer but as an active enabler of it. Smoldering, unresolved inflammation contributes at every stage. Early on, reactive oxygen and nitrogen species from inflammatory cells cause DNA damage and genomic instability. As a tumor grows, inflammatory signaling — through pathways such as NF-?B and IL-6/STAT3 — drives proliferation, blocks programmed cell death, promotes new blood-vessel formation, and supports invasion and metastasis.

It has become such an important target because of its central role in two of oncology's hardest problems: immune evasion and treatment resistance. Chronic inflammation recruits immunosuppressive cells — myeloid-derived suppressor cells, regulatory T cells, and pro-tumor macrophages — that switch off the cytotoxic T cells which would otherwise destroy the tumor. The result is the "cold" tumor that does not respond to checkpoint inhibitors. The same signaling confers resistance to chemotherapy by activating cell-survival programs. And systemically, chronic inflammation drives cachexia, erodes performance status, and predicts worse outcomes — which is why markers such as C-reactive protein and the neutrophil-to-lymphocyte ratio track with prognosis.

The therapeutic art lies in a distinction that is easy to miss: we want to promote productive, acute anti-tumor immunity while damping the chronic, immunosuppressive inflammation that protects the tumor. These are biologically distinct, and the goal is to convert one into the other — to turn cold tumors hot — rather than to suppress immunity wholesale.

This is the thesis behind our own program. In a randomized Phase II trial in platinum-resistant ovarian cancer, adding our inflammation-modulating plasmid to chemotherapy more than doubled median overall survival, with zero treatment-related serious adverse events. We have also seen the anti-inflammatory mechanism produce a published efficacy and safety signal beyond oncology — in a peer-reviewed study of chronic, inflammation-driven pain — while preclinical work on the p62 target has shown reductions in neuronal inflammatory processes. Together, these reinforce a broader point: controlling the inflammatory context is emerging as a foundation on which other therapies work better. The field still needs better biomarkers and deeper mechanistic understanding, but the direction of travel is clear.

### How do you see combination therapies evolving within the immuno-oncology landscape?

To my mind, this is one of the central questions—and one of the primary hurdles—in modern oncology. Cancer is an extraordinarily complex, evolving biological phenomenon. Just as no conventional war has ever been won with a single type of weapon, however refined, I do not believe the war on malignancy can be won with monotherapy. The future is indisputably combinatorial.

Overcoming institutional and corporate inertia: For decades, combination therapy ran against two powerful human forces: the ego of the inventor, who sought exclusive credit for a breakthrough, and the commercial instinct of the corporation, which sought unshared revenue. The encouraging news is that this protective era is ending. The industry has finally come to understand that it is far better to win together than to lose alone—and that collective realization may shift clinical outcomes as much as any single blockbuster molecule.

The mathematical impossibility of brute force: The core operational difficulty is that we cannot simply test every combination blindly; the mathematics strictly forbids it. Five candidate agents yield more than 30 possible combinations; 10 yield over 1,000; 20 yield more than a million; and 30 yield over a billion. This is even before accounting for variables like dosing, scheduling sequence, and timing. Because a linear increase in therapeutic components produces an explosive, exponential increase in combinations, brute-force empiricism is a statistical dead end. The real mandate of the next decade is rational, biology-guided selection.

Compensating for intrinsic limitations: While there is no exhaustive catalog of principles for this selection, several core strategies stand out. The most familiar is combining agents with entirely distinct mechanisms of action—such as pairing a platinum agent with paclitaxel—to strike the disease through independent pathways. However, a more sophisticated approach is designing combinations where one agent actively compensates for another's structural limitation. Many advanced immunotherapies excel at expanding the population of cytotoxic immune cells, yet they share a single, fatal bottleneck: those cells cannot physically penetrate "cold" solid tumors. An agent that converts a cold tumor into a hot one—as we believe Elenagen does by remodeling the tumor's microenvironmental architecture—can unlock the locked-out activity of companion therapies that would otherwise stall at the tumor's edge.

Disabling treatment-induced resistance loops: A closely related principle is disabling the tumor's capacity to neutralize treatment in real time. Gemcitabine, for example, activates NF-B, and NF-B in turn drives cellular resistance to gemcitabine. Because Elenagen suppresses NF-B activation, combining the two is highly rational, breaking a vicious feedback loop and pointing toward a broader class of synergistic combinations with agents whose efficacy is otherwise blunted by inflammatory signaling.

Looking further ahead, I expect scheduling to matter immensely: chronotherapy, in which therapeutic administration is tightly coordinated with the body's circadian rhythms, represents a highly promising, underutilized dimension of trial design. Finally, we must pair these regimens more thoughtfully with evidence-based supportive care and nutrition to ease the cumulative toxicity patients bear.

The direction of travel is unmistakable: the industry is moving decisively away from the romanticized search for a single, silver-bullet drug, and toward intelligent, collaborative, biologically reasoned combinations.

**What opportunities do emerging biotech ecosystems outside traditional pharmaceutical hubs bring to global innovation?**

Over the past four years, the world has once again fractured into competing geopolitical axes, and this trend will likely persist — and may well deepen. Yet there is one arena into which we must never allow these divisions to extend: the development of medicines. Disease is the common adversary of all humanity, and the search for cures must remain a shared endeavor even as other forms of cooperation fray.

Seen in that light, the rise of new innovation centers is doubly welcome. Innovation in our industry is no longer the exclusive province of Boston, San Francisco, and Basel. Emerging ecosystems across Asia-Pacific, Latin America, the Middle East, and Eastern Europe are contributing scientific talent, clinical-trial capacity, manufacturing capability, and capital — and that diffusion is good for global innovation and for patients.

The opportunities are concrete. Rigorous clinical trials can often be conducted in these regions efficiently and with access to diverse, well-characterized patient populations, generating high-quality data that can then be brought to the FDA and EMA. Regional manufacturing, including for newer modalities, is expanding capacity and resilience. And strategic, sometimes non-dilutive, capital from regional companies and conglomerates can fund development in ways that complement traditional venture financing. Much of our own randomized clinical evidence was generated outside the United States, which allowed a small company to produce a meaningful dataset before entering the larger and more expensive Western trial and regulatory process.

For the Asia-Pacific region in particular, the strengths are striking: deep pools of capital, world-class manufacturing, fast-growing oncology markets, and integrated pharmaceutical companies in Japan, Korea, China, and elsewhere that are increasingly eager to co-develop and commercialize innovative assets rather than simply in-license late-stage products. Forums such as the BIO International Convention exist precisely to connect originators of science with the regional partners who can develop, manufacture, and commercialize it. The most effective model I see is one in which science and capital flow across borders — a discovery made anywhere can be advanced through partnerships spanning several regions.

There are caveats worth naming honestly: the need for regulatory harmonization, ensuring data quality and cross-agency acceptance, and attention to intellectual property and geopolitical considerations. But these are manageable, and the upside is substantial. If the geopolitical map is fragmenting, the map of disease is not — illness respects no border, ideology, or alliance. That is precisely why the shared fight against disease can, and should, become a unifying ground on which nations belonging to different axes continue to collaborate. A more distributed innovation landscape means more shots on goal against our common enemies — and faster, more affordable routes to bringing therapies to patients everywhere, whichever side of any line they happen to live on.

## Which scientific advances are most likely to define the future of cancer immunotherapy?

One cannot predict the future, least of all in science. However, there are a few fundamental shifts in biological thinking that I believe are rapidly hardening into definitive drug-development strategies.

From silencing single words to reshaping the dialect: The cells of a tissue—and immune cells above all—communicate in a complex, fluid language whose words are interleukins, cytokines, and other signaling molecules. For most of recent history, the industry's approach has been to intercept a single word we judged to be a malicious command: an antibody that blocks one specific cytokine, or VEGF, or another discrete signal. The structural trouble is that biological language, like any natural language, is highly redundant. Silence one word—say, "hit"—and the system soon restores the malicious meaning with a biological synonym: "punch." Bispecific antibodies, which engage two targets at once, are a welcome step, but to my mind they represent only an incremental improvement; they still operate at the level of individual words. The deeper, transformative advance will come from changing the entire dialect—shifting a tissue systemically out of a pathological mode of communication, such as chronic inflammation or intratumoral immunosuppression, and back toward a healthy, homeostatic dialogue.

From attacking the cancer cell to reprogramming its neighbors: The second profound shift is moving away from acting directly on the tumor cell and toward acting on it indirectly, through the cells that surround it. Our own work with Elenagen illustrates this exact principle. The plasmid acts selectively on mesenchymal stromal cells (MSCs), in effect making them behave younger: they cease secreting a pro-inflammatory secretome—itself a chorus of many molecular words that together sustain chronic inflammation—and begin secreting an anti-inflammatory one instead.

What I find most striking is the secondary cascade. Although only a limited number of cells take up the plasmid directly, the altered secretome those cells release travels dynamically through the body, reprogramming distant stromal cells much like the plasmid itself would. The organism thus propagates and amplifies the effect of a small, localized amount of the original drug, carrying its comprehensive therapeutic effect far beyond the cells originally treated. A therapy that successfully recruits the body to spread its own corrective signal is a fundamentally different proposition from a conventional agent that must physically reach every single target cell itself.

Decoding the grammar of cellular networks: These are incredibly complex, highly networked systems, and as an industry, we are only beginning to truly read them. I expect artificial intelligence to become an indispensable partner in this specific effort—not merely predicting structures, but actively helping us decode the intricate grammar of cellular communication. AI will teach us how to efficiently shift a diseased dialect back toward a healthy conversation. If the last era of immunotherapy was defined by aggressively blocking the wrong signals, the next will belong to platforms capable of restoring the right conversation.