



Press Release
For immediate release

Advanced BioDesign Reports Immune-Activating Potential of ABD-3001, Expanding the Therapeutic Promise of its First-in-Class ALDH1 Inhibitor

Frontiers in Immunology publication reveals that ABD-3001 preserves immune function while promoting immunogenic remodeling of acute myeloid leukemia cells

Lyon, France – 18th August 2026 — Advanced BioDesign today announced the publication of new preclinical findings in *Frontiers in Immunology* demonstrating that ABD-3001 (DIMATE), the company's first-in-class ALDH1 inhibitor currently being evaluated in the Phase 1/2 ODYSSEY trial, may combine potent anti-leukemic activity with immune-preserving and immune-activating properties that can now be explored in the clinical setting.

The publication, entitled ***"ALDH1 inhibition by DIMATE maintains immune responses and promotes immunogenic remodeling in AML"***, identifies a previously unrecognized immunological dimension of ABD-3001 that could significantly expand its therapeutic potential in acute myeloid leukemia (AML).

A Potentially Differentiated Approach in AML

While most AML therapies achieve therapeutic benefit at the cost of substantial immune suppression, the study demonstrates that ABD-3001 preserves key immune cell functions, including T-cell activation, natural killer (NK) cell activity, and innate immune responses, at clinically relevant concentrations.

At the same time, the compound induces molecular changes that may render leukemia cells more visible to the immune system. Investigators observed activation of inflammatory signaling pathways, increased expression of co-stimulatory molecules, and surface exposure of calreticulin, a hallmark signal associated with immunogenic cell death (ICD), a process known to stimulate anti-tumor immune responses.

Together, these findings suggest that ABD-3001 may not only eliminate leukemic cells directly but could also help recruit the patient's own immune system to participate in disease control.

Unlocking a New Therapeutic Paradigm

"This publication significantly broadens our understanding of ABD-3001's mechanism of action. Beyond its direct anti-leukemic activity, our data show that ALDH1 inhibition can preserve key immune functions while promoting immunogenic changes in leukemia cells. We believe this dual activity creates exciting opportunities for future combination strategies designed to leverage both direct tumors killing and immune-mediated disease control." **Ismail Ceylan, Founder and Chief Executive Officer of Advanced BioDesign.**

Strong Rationale for Immunotherapy Combinations

By maintaining immune competence while increasing leukemic cell immunogenicity, ABD-3001 could become an attractive partner for emerging immunotherapeutic strategies in AML, including monoclonal antibodies, cellular therapies, and other immune-engaging approaches.

These findings are particularly relevant in a disease where durable responses remain difficult to achieve and where the immune system is increasingly recognized as a critical contributor to long-term control of residual disease.

The study provides a mechanistic rationale for evaluating ABD-3001 in combination with therapies designed to amplify anti-tumor immunity while preserving direct anti-leukemic efficacy.

A New Perspective for ODYSSEY and Future Clinical Development

"AML remains one of the few major hematological malignancies where immunotherapy has not yet delivered its full potential. Importantly, the clinical efficacy of allogeneic hematopoietic stem cell transplantation demonstrates that immune-mediated graft-versus-leukemia effects can achieve profound and durable disease control in AML, while its broad application remains limited by patient eligibility, treatment-related toxicity, graft-versus-host disease, and disease relapse. The new immunological perspective opened by ABD-3001 is therefore particularly important: if the immune-preserving and immunogenic effects observed preclinically are confirmed in patients, ODYSSEY could provide a unique opportunity to investigate how ALDH1 inhibition reshapes anti-leukemic immunity in the clinical setting. Rather than replacing allogeneic immunotherapy, this approach could potentially complement or capture some of the biological principles underlying immune-mediated leukemia control by enhancing the immunogenicity of leukemic cells while preserving the functional competence of effector immune cells. These insights may help in the ongoing trial and inform the design of future clinical studies aimed at engaging the patient's own immune system in long-term disease control, potentially extending the benefits of immunological surveillance beyond the patients who are eligible for allogeneic transplantation. " **Prof. Régis Costello, MD, PhD, Head of Hematology and Cellular Therapy, AP-HM Marseille, Co-Senior Author of the Study and Principal Investigator of the ODYSSEY Trial.**

Building the Next Generation of AML Therapies

Despite recent advances in hematologic oncology, AML continues to present major therapeutic challenges, particularly in the relapsed and refractory setting. Advanced BioDesign believes that therapies capable of combining direct eradication of leukemic stem cells with immune system engagement may represent an important new direction for the treatment of AML.

The company's ongoing development strategy aims to fully explore the therapeutic potential of ALDH1 inhibition, including its emerging role at the interface between cancer metabolism and anti-tumor immunity.

This emerging immune-metabolic profile may create opportunities for strategic collaboration with pharmaceutical partners seeking differentiated AML assets, combination-ready mechanisms, and translational strategies supported by both preclinical immune biology and ongoing clinical evaluation.

Key Highlights from the Publication

- Demonstrates preservation of T-cell, NK-cell, and innate immune functions following exposure to DIMATE.
- Shows enhanced NK-cell cytotoxic activity against leukemia target cells.
- Identifies activation of immune-related inflammatory pathways in AML cells.
- Reveals induction of molecular features associated with immunogenic cell death, including calreticulin surface exposure.
- Provides a mechanistic rationale for future immune-based combination strategies.
- Supports the integration of immune biomarker monitoring into the ongoing ODYSSEY clinical trial.
- Suggests that ABD-3001 may bridge direct anti-leukemic activity and immune-mediated control of residual disease.

Link to the publication: [Frontiers | ALDH1 inhibition by DIMATE maintains immune responses and promotes immunogenic remodeling in AML](#)

About ABD-3001

ABD-3001 is the pharmaceutical formulation of DIMATE, a first-in-class "suicide" inhibitor of class I aldehyde dehydrogenases (ALDH1). This innovative compound was developed to target a critical survival pathway in cancer cells. Its mechanism of action involves inhibition of ALDH enzymes, which

are frequently exploited by tumor cells to mitigate oxidative stress. ALDH inhibition induces intracellular redox imbalance, ultimately leading to cancer cell death while largely sparing normal cells. This therapeutic strategy is designed to overcome resistance mechanisms associated with standard chemotherapy and currently available targeted therapies.

DIMATE is currently being evaluated in a Phase 1 clinical trial (ODYSSEY) and may represent a novel therapeutic option for patients with limited or no remaining treatment alternatives.

About the ODYSSEY clinical trial

ODYSSEY is a Phase I/II clinical trial for the treatment of relapse and refractory acute myeloid leukemia (AML). It is a multicenter study, with centers in Paris, Lyon and Marseille, designed to assess the safety and tolerability of the drug candidate ABD-3001.

Following an adaptive design, the study integrates an ascending single-dose first part, on six patient cohorts, followed by a second part, during which three patient cohorts will receive full four-week treatment cycles, enabling initial efficacy results to be obtained.

Fully funded by Advanced BioDesign, the ODYSSEY clinical trial is coordinated by Professor Régis COSTELLO (Hôpital de la Conception, Marseille), in collaboration with Professor Lina BENAÏBA (Hôpital Saint-Louis, Paris), Doctor Maël HEIBLIG (Hôpital Lyon Sud, Lyon), Doctor Ludovic GABELLIER (Hôpital Saint Eloi, Montpellier) and Professor Thomas CLUZEAU (Centre Hospitalier Universitaire de Nice, Nice).

About Acute Myeloid Leukemia

Acute myeloid leukemia (AML) represents an approximately USD 1.5 billion global market with persistent unmet medical need, particularly in relapsed/refractory and high-risk patient populations. Despite recent progress with targeted agents and azacitidine–venetoclax-based regimens, resistance, relapse, and limited durability of response remain major clinical and commercial challenges. By targeting ALDH1, a resistance-associated metabolic pathway, and by potentially preserving immune function while increasing leukemia cell immunogenicity, ABD-3001 could support differentiated positioning in AML, rational combination development, and future partnering opportunities across both salvage and high-risk treatment settings.

About Advanced BioDesign

Advanced BioDesign is a clinical-stage biotechnology company focused on overcoming treatment resistance and relapse in oncology. The company has developed a first-in-class therapeutic approach targeting ALDH1, a key enzyme used by cancer cells to detoxify harmful metabolic by-products and evade standard treatments. Its lead candidate, ABD-3001, selectively disrupts cancer cell metabolism by inhibiting ALDH1, triggering cancer cell death while largely sparing healthy cells. Supported by ongoing clinical evaluation in AML, translational immune-biology data, and a mechanism relevant across multiple tumor types, Advanced BioDesign is advancing a differentiated platform designed to address resistance-driven cancers and create future opportunities for clinical expansion, combination development, and strategic partnering.

For more information: <https://www.a-biodesign.com> ; LinkedIn [@Advanced BioDesign](#)

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