



## Medicenna Reports Potent CD122 Blockade and Extended Survival with MDNA209 in Preclinical Models of Autoimmune Disease

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*MDNA209 is a potential first-in-class engineered Superkine designed to block CD122 and suppress overactive immune cells responsible for inflammatory and autoimmune conditions by inhibiting IL-2 and IL-15 signalling*

*Long-acting MDNA209 extended median survival by up to 4-fold in aggressive mouse models of acute graft-versus-host disease*

TORONTO and HOUSTON, Oct. 26, 2026 (GLOBE NEWSWIRE) -- Medicenna Therapeutics Corp. ("Medicenna" or the "Company") (TSX: MDNA, OTCQB: MDNAF), a clinical-stage immunotherapy company developing Superkines for cancer and autoimmune diseases, today announced new preclinical data demonstrating potent CD122 blockade and suppression of overactive immune-cell activity with MDNA209, its potential first-in-class cytokine-based CD122 superantagonist. The findings were presented by Dr. Minh D. To on October 23, 2026, in an oral presentation at The Promises of Treg and Interleukin-2 Therapies Conference, held October 21–24, 2026, in Paris, France.

The data showed that MDNA209 binds tightly to CD122 and blocks signals induced by IL-2 and IL-15, a pathway that when dysregulated plays a central role in the development and progression of autoimmune diseases. In two aggressive mouse models of acute graft-versus-host disease (aGvHD), treatment with long-acting MDNA209 improved disease symptoms and extended median survival by approximately 3- to 4-fold compared with controls. GvHD is a serious life-threatening complication of donor stem-cell transplantation in which donor immune cells attack the recipient's healthy tissues.

CD122 is attracting growing clinical and strategic interest following encouraging clinical results in celiac disease and vitiligo and argenx's acquisition of Forte Biosciences, whose lead program, FB102, is an anti-CD122 antibody. MDNA209 provides a differentiated engineered Superkine approach to blocking this target.

"These results demonstrate potent CD122 blockade with MDNA209 in human immune cells and a substantial survival benefit in two aggressive preclinical models of GvHD, further strengthening the promise of our IL-2 Superkine platform," said Fahar Merchant, PhD, President and Chief Executive Officer of Medicenna. "With clinical progress and strategic activity attracting attention to CD122 blockade, we believe MDNA209 offers a differentiated engineered Superkine approach to this validated target as we pursue potential partnership opportunities. Unlike anti-CD122 antibodies in clinical development, our IL-2 Superkine platform allows us to "fine-tune" signaling pathways across all IL-2 and IL-15 receptor sub-types rather than a single CD122 "on/off switch". This capability enables extraordinary flexibility to develop "disease-appropriate" therapeutics with MDNA209 leading the way for autoimmune diseases and MDNA11 demonstrating compelling promise for treating patients with advanced cancers not responsive to immune checkpoint inhibitors."

### Key Findings Presented

- **High-affinity CD122 blockade.** MDNA209 bound tightly to CD122 and prevented it from assembling into a receptor complex needed for IL-2 and IL-15 activity. This CD122 "clamp" mechanism blocks the signals that can sustain overactive immune cells.
- **Potent inhibition of activated T cells and natural killer cells.** Studies in human blood immune cells (PBMCs) demonstrated potent inhibition of IL-2 signaling in activated CD8+ T cells and natural killer (NK) cells, cell types implicated in autoimmune inflammation and tissue damage. In one human T-cell signaling assay, MDNA209 was shown to be approximately 150 times more potent than the anti-CD122 antibody Mikβ1 and 10 times more potent than the anti-CD25 comparator daclizumab biosimilar.
- **Suppression of immune-cell expansion and inflammatory signals.** The data indicated that MDNA209 inhibited immune-cell expansion driven by IL-2 and IL-15. It also suppressed IL-2-induced release of interferon gamma, an inflammatory protein that can contribute to tissue damage.
- **Treg viability maintained following treatment.** Four days of treatment did not reduce the viability of human regulatory T cells (Tregs), which help keep the immune system in check.
- **Activity in a laboratory model relevant to GvHD.** MDNA209 inhibited immune-cell expansion in mixed lymphocyte reactions using human PBMCs from unrelated individuals. This laboratory system models the donor–recipient immune activation involved in GvHD.
- **Proof of concept in two aggressive models of acute GvHD.** Treatment with long-acting MDNA209 improved disease symptoms and significantly extended survival in two mouse models with partially or fully mismatched donor–recipient transplants. Body weight recovery following initial loss upon transplant of donor cells was also evident. Median survival reached 23.5 and 26 days, respectively, compared with 7 days in each control group—approximately a three- to fourfold increase.

Together, these findings support the potential therapeutic utility of directly targeting disease-driving immune cells with MDNA209 through CD122 blockade, with possible applications across autoimmune and inflammatory diseases.

#### **About CD122**

CD122, also known as IL-2 receptor beta, is a shared signaling component of the receptors for IL-2 and IL-15. These interleukins help regulate the activation, growth and survival of immune cells. In certain autoimmune diseases, these pathways can sustain hyperactive immune cells that attack healthy tissues. Blocking CD122 offers an approach to inhibit IL-2 and IL-15 pathways through a single target. Recent clinical results reported with investigational CD122-blocking antibodies have supported growing interest in this mechanism as a potential treatment strategy for autoimmune diseases.

#### **About MDNA209**

MDNA209 is an investigational, long-acting Superkine in preclinical development as a potential first-in-class cytokine-based CD122 superantagonist. MDNA209 is engineered for high-affinity binding to CD122 (>100x higher than native IL-2) and is designed to potentially inhibit signaling by both IL-2 and IL-15, thereby suppressing overactive immune cells that can contribute to autoimmune and inflammatory diseases.

#### **About Medicenna Therapeutics**

Medicenna is a clinical-stage immunotherapy company developing engineered cytokine therapies designed to selectively engage the immune system to treat cancer and autoimmune diseases. The Company's most advanced program, bizaxofusp (formerly MDNA55), is a targeted IL-4 Empowered Superkine that has been evaluated in more than 130 patients across five clinical trials, including a Phase 2b study in recurrent glioblastoma. Bizaxofusp has received Fast Track designation from the FDA and Orphan Drug designation in the United States and Europe. Medicenna is also advancing MDNA11, a long-acting IL-2 Superkine designed to selectively activate cancer-fighting immune cells, which is currently being evaluated in the Phase 1/2 ABILITY-1 study and the Phase 1b NEO-CYT study. With its proprietary BiSKIT and T-MASK platforms, the Company is also developing MDNA113, a targeted PD-1 x IL-2 bifunctional immunotherapy for solid tumors, and MDNA209, an IL-2/IL-15 superantagonist effectively blocking the overactive CD122/CD132 pathway responsible for autoimmune diseases.

For more information, please visit [www.medicenna.com](http://www.medicenna.com), and follow us on [X](#) and [LinkedIn](#).

#### **Forward-Looking Statements**

This news release contains forward-looking statements within the meaning of applicable securities laws. Forward-looking statements include, but are not limited to, statements regarding the therapeutic potential, possible applications and continued preclinical development of MDNA209, and the Company's future operations, plans, objectives and expectations. These statements assume that preclinical findings support further development and that necessary resources and regulatory permissions can be obtained. There can be no assurance that such statements will prove to be accurate and actual results and future events could differ materially from those anticipated in such statements. Important factors that could cause actual results to differ materially include uncertainties in preclinical research, the risk that preclinical results do not translate into clinical benefit, development, regulatory and financing risks, and the risks detailed in the latest annual information form of the Company and in other filings made by the Company with the applicable securities regulators from time to time in Canada.

The reader is cautioned that assumptions used in the preparation of any forward-looking information may prove to be incorrect. Events or circumstances may cause actual results to differ materially from those predicted, as a result of numerous known and unknown risks, uncertainties, and other factors, many of which are beyond the control of the Company. The reader is cautioned not to place undue reliance on any forward-looking information. Such information, although considered reasonable by management, may prove to be incorrect and actual results may differ materially from those anticipated. Forward-looking statements contained in this news release are expressly qualified by this cautionary statement. The forward-looking statements contained in this news release are made as of the date hereof and except as required by law, we do not intend and do not assume any obligation to update or revise publicly any of the included forward-looking statements.

This news release contains hyperlinks to information that is not deemed to be incorporated by reference in this news release.

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