

## Sprint Bioscience's DISA program further validated in two recent scientific publications

**Two articles have been published recently that strengthen the validation of the protein TREX1 as a target for cancer therapy. These results strengthen the commercial potential of Sprint Bioscience's DISA program, consisting of potent, drug-like inhibitors of the TREX1 protein.**

Two independent groups have investigated the therapeutic potential of targeting TREX1 in cancer treatment. Taken together, these two publications lend further support to the therapeutic potential in Sprint Bioscience's DISA program which aims to develop inhibitors of the TREX1 protein, thereby activating the immune response in tumors and improving the response to treatment with various types of immunotherapies. The studies also deepen the mechanistic understanding of immune stimulation using TREX1 inhibitors and will support the development of cancer therapies targeting TREX1, aiding the development of combination treatments, biomarker development, and patient selection for upcoming clinical trials.

By using gene-editing techniques (knockdowns), the researchers showed that inactivation of TREX1 alone in cancer cells led to significant tumor growth inhibition in mouse models. The effect on tumor growth could be further enhanced by combining TREX1 inactivation with checkpoint inhibition. This suggests that targeting TREX1 could potentially be a promising strategy for cancer therapy.

The study by Tani *et al* [1] shows that the immunostimulatory effect of TREX1 knockdown can be achieved by blocking the catalytic activity of the TREX1 protein, further supporting the potential therapeutic use of small-molecule inhibitors of this target. They also show that TREX1 inactivation sensitizes tumor cells to killing by immune cells, expanding the potential combination treatments to also include cell therapies.

In the study by Zhang *et al* [2], patient datasets were analyzed and revealed that low TREX1 expression correlated with improved survival outcomes in certain types of cancer, thereby providing human genetic evidence of the importance of TREX1 in cancer progression and response to treatment.

### References:

[1] Tani et al, TREX1 inactivation unleashes cancer cell STING-interferon signaling and promotes anti-tumor immunity, *Cancer Discovery*, 2024: <https://doi.org/10.1158/2159-8290.cd-23-0700>

[2] Zhang et al, Cytosolic DNA accumulation promotes breast cancer immunogenicity via a STING-independent pathway, *Journal for Immunotherapy of Cancer*, 2023: <https://doi.org/10.1136/jitc-2023-007560>

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**About Sprint Bioscience AB (publ)**

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Sprint Bioscience develops small molecule first-in-class drug programs with a focus on oncology. With a fragment-based drug development method, the company develops drug programs in a time- and resource-efficient manner. The programs are out-licensed to global pharmaceutical companies during the pre-clinical phase and the company has successfully entered into several license agreements. The Sprint Bioscience share is listed on the Nasdaq First North Premier Growth Market and trades under the ticker symbol SPRINT. The company is based in Stockholm with operations located in Huddinge. Further information is available on the company's website; [www.sprintbioscience.com](http://www.sprintbioscience.com). Certified Advisor is FNCA Sweden AB, [www.fnca.se](http://www.fnca.se).

**Attachments**

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