



PRESS RELEASE

Lund, October 8th, 2026

Alpha1H builds its scientific case as Phase III gets under way

New insights into the mechanism of action of the anti-cancer drug Alpha1H are published today in *Molecular Therapy*, a Cell Press journal. The study maps how Alpha1H disrupts the adhesive structures that hold malignant cells together in the tumor and anchor the tumor to the underlying tissue. By targeting adhesion, Alpha1H disrupts the tumor structure, in addition to killing the tumor cells. The findings explain the massive tumor cell shedding observed in treated patients and strengthen the scientific basis for Hamlet BioPharma's bladder cancer program, with patients now being treated in Phase III.

Earlier clinical studies showed that Alpha1H, administered directly into the bladder, triggered rapid shedding of tumor cells and tumor fragments into the urine, alongside reductions in tumor size and number. The new paper examines the molecular mechanisms responsible for these potent effects, combining analysis of patient samples with experiments in cancer cell models.

The study identifies a broad effect across several adhesion molecule systems that maintain tumor structure. Alpha1H disrupted both the connections between neighboring tumor cells and the structures that anchor them to adjacent tissues. These effects support cell detachment as a contributor to the anti-tumor activity, alongside Alpha1H's previously reported ability to kill tumor cells. By weakening both intercellular junctions and the deeper cell–matrix attachments, Alpha1H facilitates the shedding of individual tumor cells as well as larger tumor fragments, providing a mechanistic basis for the anti-tumor effects observed clinically.

The effects on tumor-cell detachment were rapid, as shown by changes in adhesion-related gene activity detected in urine samples collected within two hours of treatment. Suppression was also evident in tumor biopsies taken after the month-long treatment course. In the higher-dose group, 350 of the 444 adhesion-associated genes were inhibited compared to placebo. This connects the rapid response after acute treatment with longer-term changes, detected at surgery after the end of treatment.

These findings are particularly relevant since cell adhesion remains a challenging therapeutic target in oncology. Although adhesion molecules, particularly integrins, have long been recognized as important drivers of tumor growth, invasion and metastasis, relatively few adhesion-targeted therapies have achieved clinical success in cancer. One challenge is the

complexity and redundancy of adhesion networks, which can limit the efficacy of therapies directed against individual targets. Alpha1H differs from this conventional approach by broadly disrupting multiple interconnected adhesion systems, resulting in rapid physical detachment of tumor cells. This mechanism may therefore represent a distinct therapeutic strategy in which tumor architecture itself becomes a target for general cancer treatment, as shown here for bladder cancer.

For investors, the findings broaden the therapeutic profile and gives prospective partners a detailed, peer-reviewed account of the molecular mechanisms underlying the clinical findings.

Publication and company updates

Chaudhuri A et al. Broad targeting of the cell adhesion machinery - cancer cell detachment triggered by the alpha1-oleate complex. Molecular Therapy (2026), article in press. [doi:10.1016/j.ymthe.2026.09.029](https://doi.org/10.1016/j.ymthe.2026.09.029)

For further information, please contact

Catharina Svanborg, Chairman, Hamlet BioPharma AB, +46 709 42 65 49,

catharina.svanborg@hamletpharma.com

Jakob Testad, CEO, Hamlet BioPharma AB, +46 708 48 42 10,

jakob.testad@hamletbiopharma.com