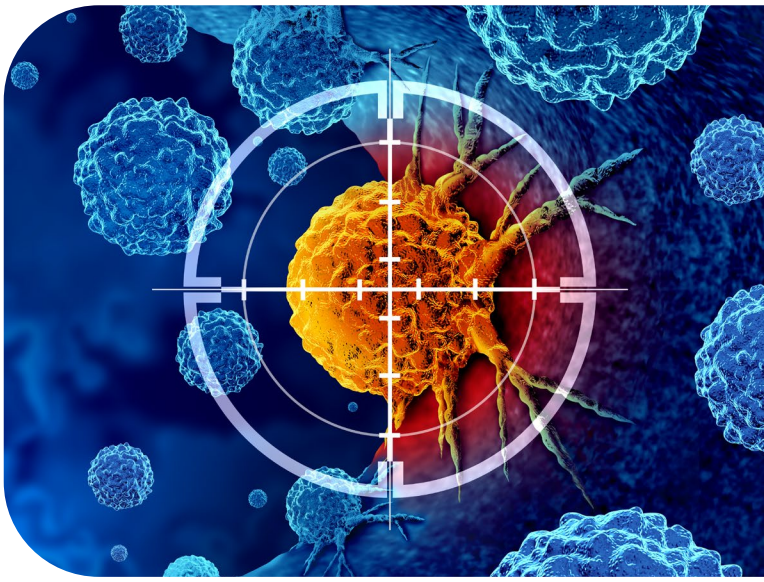
>> Oncology Research

Oncology research is critical for understanding the mechanisms of cancer and developing novel therapeutics. Automated, AI-powered **Omni and Lux live-cell imagers** can advance oncology research by enabling scientists to monitor cancer cell behavior in real time in **2D and 3D *in vitro* cell models** straight from the incubator, allowing for continuous, noninvasive tracking of tumor growth, morphology changes, and drug responses in a more physiologically relevant context.



Learn how Omni and Lux can support your oncology research with **these selected publications:**

Harnessing virus flexibility to selectively capture and profile rare circulating target cells for precise cancer subtyping

Li H, Chen Y, et al. Nature Communications. (2024)

The absence of a capturing surface with strong target-binding affinity and resistance to non-target cells makes the effective isolation of rare target cells from whole blood challenging. In this study, the authors propose a solution using the versatile properties of bacterial virus (phage) nanofibers.

Highlights:

- The mechanical properties of virus-modified solid surfaces play a significant role in the isolation of rare cells, as revealed by this study.
- The solid surface gains a self-adjusting ability due to the inherent flexibility and deformability of virus nanofibers.
- By drawing upon a deeper mechanistic understanding of target-ligand interactions, the phage-based strategy introduces an affinity-based solid bioassay.

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APR-246 as a radiosensitization strategy for mutant p53 cancers treated with alpha-particles-based radiotherapy

Micheali O, Luz I, et al. Cell Death & Disease. (2024)

Radiation therapy (RT) continues to be a widely used treatment for cancer patients globally. This study explores the use of a combination therapy involving alpha-emitting Radium-224 (224Ra) for internal radiation and systemic APR-246, a compound that reactivates p53, to target tumors with mutant p53.

Highlights:

- Radiosensitivity to alpha-particle-based radiotherapy is exhibited by cancer cells with mutant p53, as demonstrated in the study.
- The study provides evidence that APR-246 significantly boosts the sensitivity of cancer cells and tumors with mutant p53 to alpha-particle-based radiation therapy, making them more responsive to treatment.
- A promising therapeutic approach for improving treatment outcomes in patients with mutant p53 tumors is offered by these findings.

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Targeting initial tumour–osteoclast spatiotemporal interaction to prevent bone metastasis

Gu C, Chen P, et al. Nature Nanotechnology. (2024)

Bone is the most frequent site for metastasis, and the low proliferation and immunoediting during the early stages make current treatment options less effective. In response, the authors of this study propose an *in situ* decoupling-killing strategy.

Highlights:

- By identifying the essential tumasteoclast-inducing behavior and using drug-free physical killing through crystal formation, this strategy bypasses pharmacological and biochemical resistance.
- This strategy can be applied to treat metastases in various organs, as the tumor-microenvironment-inducing behavior is conserved and common in tumor metastasis.
- A proof-of-concept for a behavior-target strategy and a research model for exploring tumor cell behavior in detail are provided by this study.

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Casein kinase 2 phosphorylates and induces the SALL2 tumor suppressor degradation in colon cancer cells

Hermosilla VE, Gyenis L, et al. Cell Death & Disease. (2024)

SALL2 plays a crucial role in the development of the brain and eyes. It is downregulated in cancer, where it functions as a tumor suppressor by promoting cell cycle arrest and cell death. However, there is limited information available regarding the regulation of SALL2.

Highlights:

- A methuosis-like phenotype and cell death were induced in SW480 cells by silmitasertib.
- SALL2 sensitizes cancer cells to CK2 inhibition, as confirmed by the fact that Sall2-deficient tumor organoids exhibited greater resistance to silmitasertib-induced cell death.
- A new mechanism for reducing SALL2 levels in cancer cells is proposed in this report, which could have significant implications for cancer therapy using CK2 inhibitors.

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Preclinical evaluation of Zn(II) self-assemblies with selective cytotoxic activity against cancer cells *in vitro* and *in ovo*

Allison SJ, Wordsworth DA. Chemistry - A European Journal. (2024)

Metallosupramolecular chemistry focuses on the design of poly-dentate ligands that, upon binding with metal ions, spontaneously assemble into well-defined and predictable structures. In this study, researchers explore how ligands L1 and L2 interact with Zn2+ to form dinuclear complexes. These complexes exhibit strong cytotoxic effects and demonstrate selective toxicity toward cancer cell lines, both in laboratory cultures (*in vitro*) and within living organisms (*in ovo*).

Highlights:

- Self-assemblies are formed by ligands L1 and L2 with Zn2+ and Cu2+, and they have the ability to bind to various anions.
- Depending on the ligand, metal, and anion used, these show varying selectivity profiles.
- The findings also demonstrate that [(L2)2Zn2]4+ is effective *in ovo* at doses that do not cause toxicity, highlighting its promising potential as a therapeutic agent.

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Development of *in vitro* assays for advancing radioimmunotherapy against brain tumors

Walter Y, Hubbard A, et al. Biomedicines. (2022)

Glioblastoma (GBM) is the most prevalent type of primary brain tumor. The current standard treatment involves a combination of surgery, radiation therapy, and chemotherapy using temozolomide (TMZ). Radioimmunotherapy (RIT) is being explored as a new treatment option in clinical trials, aiming to merge the benefits of immunotherapy with radiotherapy. Here, the authors create *in vitro* assays to facilitate the swift evaluation of RIT approaches, helping to assess their potential effectiveness in treating GBM.

Highlights:

- In the first 20-40 hours after treatment, irradiated T98G and U87 GBM cells show significantly more migration compared to untreated cells.
- The migration rates of T98G cells increase with the addition of temozolomide.
- Cell survival does not change significantly 21 days after treatment with either temozolomide or durvalumab. However, durvalumab eliminates the increased migration effect, suggesting it may have potential in combating local invasion.

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