

# >> Defining Docetaxel's antitumor effects on Calu3 lung cancer cells using high-throughput live-cell imaging

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## Omni: Kinetic cell tracking

*Automated, whole-vessel imaging and analysis*

Oncology studies cancer development and treatments, using assays like cytotoxicity and migration to assess drug toxicity. Toxicity profiles depend on assay type and drug impact, with combined assays offering more accurate results.

The Omni Pro 12 streamlines drug profiling by automating multi-assay workflows, reducing variability, and improving data reliability, advancing cancer research.



### The Omni product family

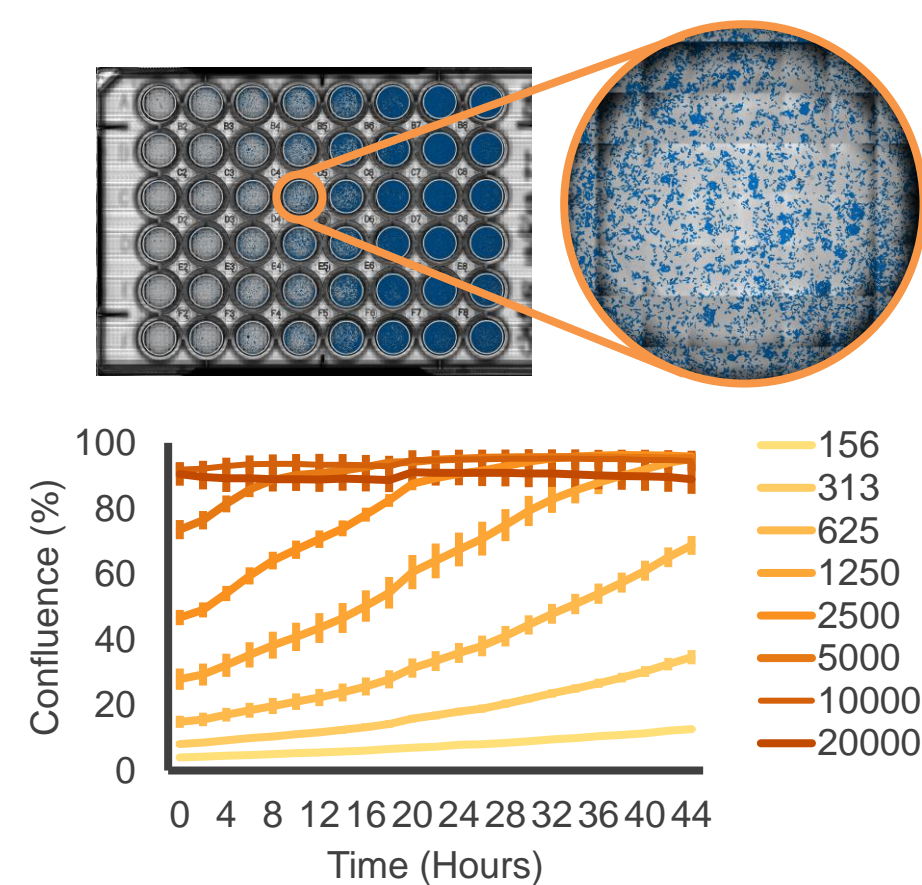
>> Assay your cells in brightfield and fluorescence

>> Track every moment, straight from your incubator

>> See every cell by movement of the camera

>> Monitor and analyze your cells remotely

>> Get started quickly



### AI-Driven imaging software for powerful, yet simple analysis

The Omni platform software modules simplify assay setup, offer real-time cellular visualization, and enable fast analysis.



Cell Confluence



Scratch Assay



Fluorescence Analysis



Clonogenic Assay



Organoid Analysis



iPSC Monitoring

## Real-time analysis of cell behavior

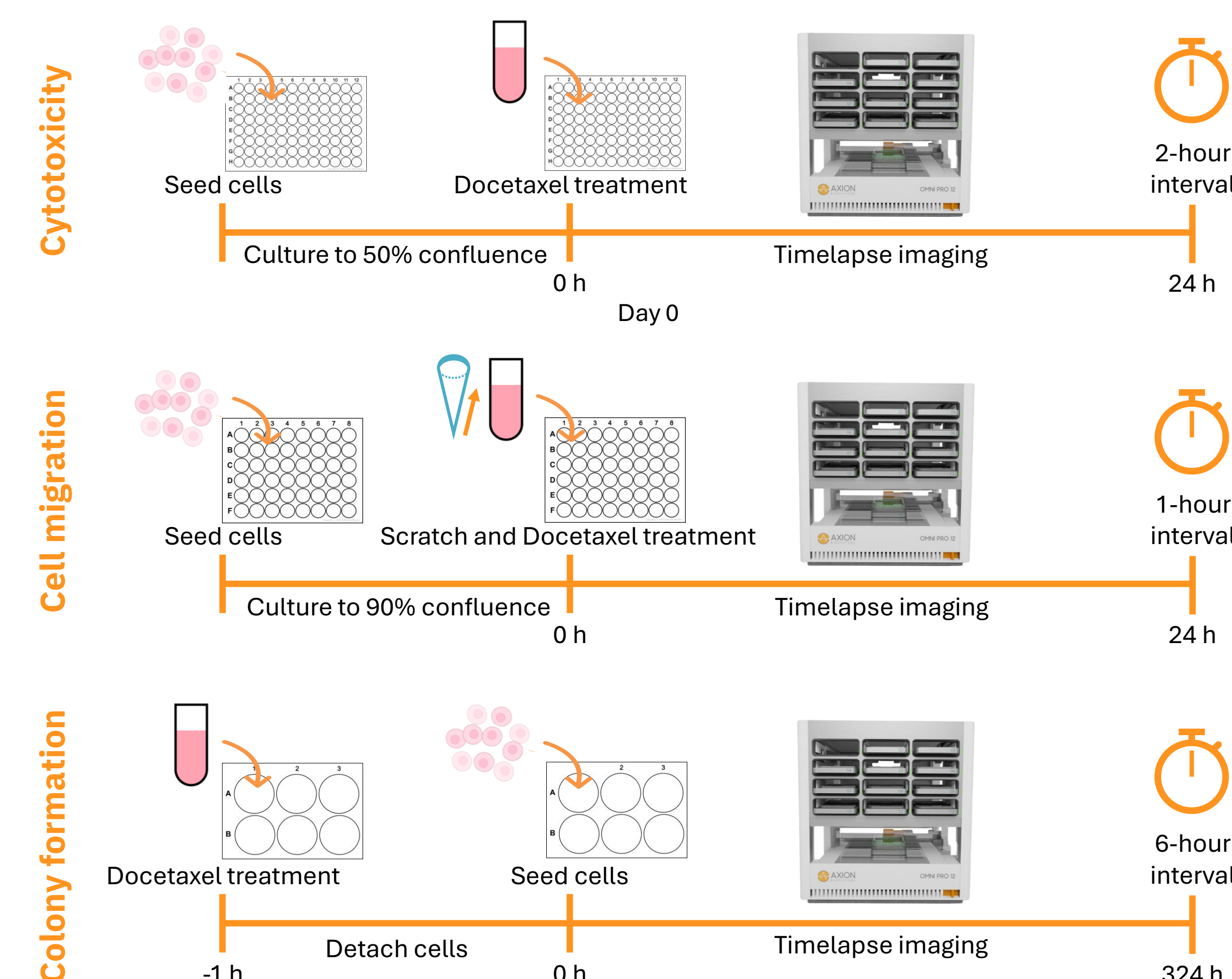
*The effect of assay type on drug toxicity profiles*

### Introduction

To assess drug effectiveness and resistance for the treatment of cancer, many different assays are used. Docetaxel, a chemotherapy drug that inhibits microtubule depolymerization, is commonly studied for its effects on cell proliferation and migration. Assay results often vary but combining them offers a more complete drug profile.

This study used the Omni Pro 12 live-cell imaging system to assess Docetaxel's impact on cell viability, migration, and colony formation in Calu3 lung cancer cells. The system minimized variability by standardizing experimental conditions.

### Methods



### Results

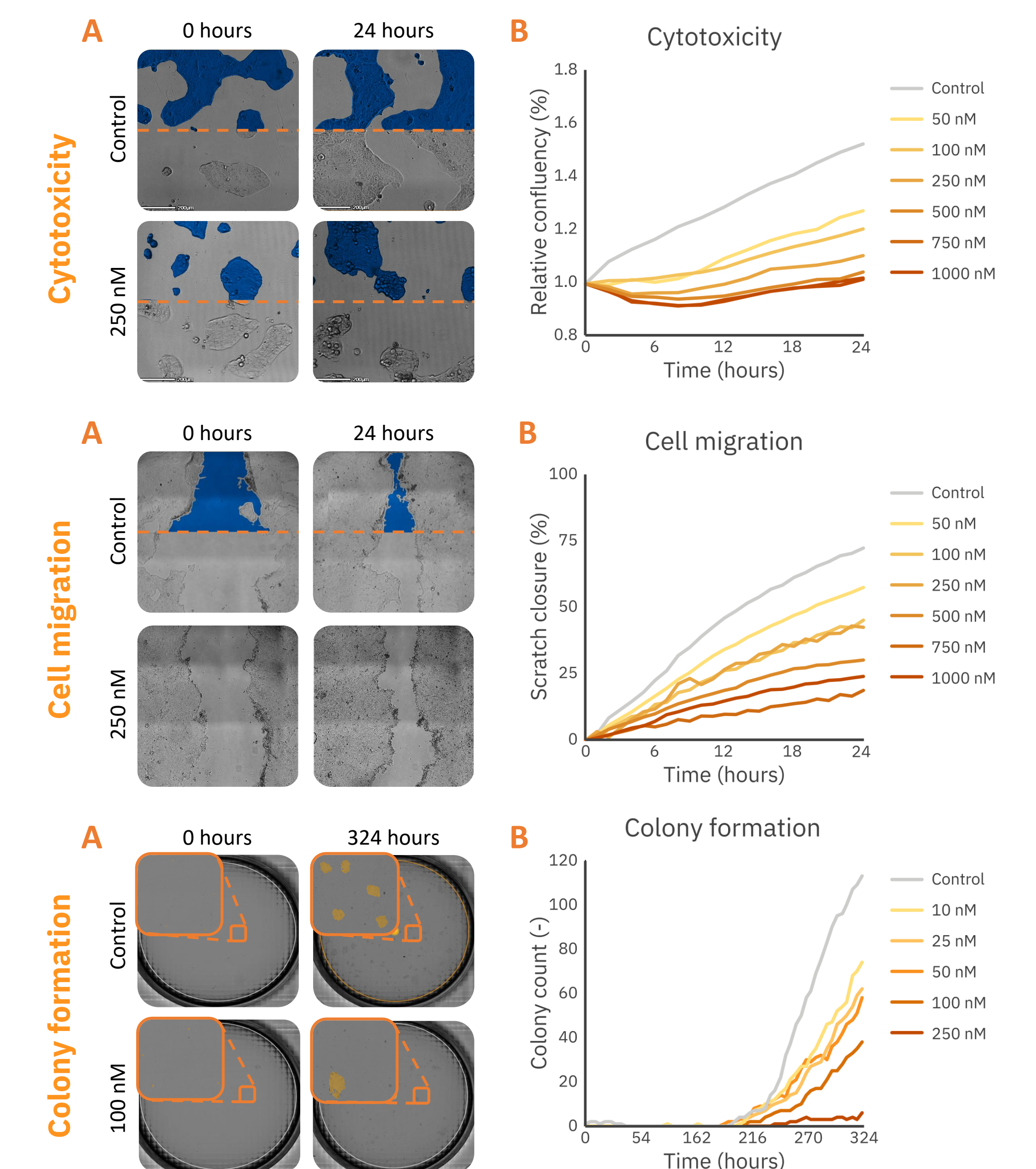
**Cytotoxicity:** Docetaxel (DCT) inhibits Calu3 proliferation in a dose-dependent manner, with peripheral cells being more affected than central cells in clusters. Higher concentrations of DCT lead to a reduced proliferation recovery after 24 h.

**Cell migration:** DCT slowed Calu3 migration in a dose-dependent manner, albeit in a more gradual manner compared to proliferation inhibition.

## Reducing between-assay variability

*Differential effective concentrations of docetaxel per assay type*

**Colony formation:** DCT reduced the long-term colony-forming ability of single Calu3 cells in a dose-dependent manner, with 250 nM nearly eliminating colony formation after 324 hours. Long-term monitoring showed that Calu3 cells need at least 180 hours to form detectable colonies.



### Conclusion

This study demonstrated the dose-dependent inhibition of DCT on Calu3 cell viability, proliferation, and migration, with varying effectiveness across assays. Simultaneous *in vitro* assays using the Omni Pro 12 provided a comprehensive toxicity profile, highlighting its potential therapeutic efficacy in lung cancer. This study emphasizes integrating multiple assays under consistent conditions to improve predictions of drug responses.