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## COLLEGE OF NATURAL SCIENCES

### SCHOOL OF PHYSICAL SCIENCES

### DEPARTMENT OF MATHEMATICS

SEMINAR ANNOUNCEMENT

DATE: Wednesday 16th September 2026 TIME: 3:00PM – 4:00PM

VENUE: Maths Seminar 203- Maths Department

**Presenter: Dr. Fouedji Chenceline**

# From Spatiotemporal Cell Dynamics to Tumor Dynamics: A Multiscale Mathematical Modeling Approach to MARCKS Protein in Cancer

The myristoylated alanine-rich C-kinase substrate (MARCKS) protein plays a fundamental role in intracellular signal transduction, cell motility, and cancer progression across various solid malignancies. In this talk, we present a unified multiscale mathematical framework tracing the dynamics of MARCKS from subcellular spatiotemporal interactions to macro-level tumor-immune interactions.

We first review the reaction-diffusion dynamics governing MARCKS shuttling between the cytosol and the cytoplasmic membrane. By mapping the governing transport equations to a cubic complex Ginzburg¬ Landau equation, we perform modulational instability (MI) analysis to establish the criteria for localized structure formation. Our analytical predictions demonstrate that phosphorylation and membrane binding rates exert opposing effects on MARCKS pattern formation, while self-diffusion amplifies structural localization, driving cyclic transport via multisoliton-like patterns.

Building upon these biophysical foundations, we transition to a systemic macroscopic model incorporating tumor cells, effector immune cells, and MARCKS concentration during immunotherapy. Using a novel nonlinear system of differential equations, we derive the basic reproduction number (R0) via the next-generation matrix method and identify a transcritical bifurcation at R0 = 1 separating tumor clearance from persistent growth. Extending this framework to delay differential equations (DDEs) accounts for intracellular signal delays and time lags in immune recruitment. Hopf bifurcation analysis reveals that while critical delays ($\tau c \approx 0.5073$ days) destabilize the tumor-free state, targeted MARCKS inhibition combined with adequate immunotherapy robustly stabilizes the tumor-free regime, rendering the system resilient to delay-induced oscillations.

Together, these findings highlight a direct analytical continuity between intracellular biophysical mechanisms and clinical outcome control, establishing MARCKS pathway inhibition as a pivotal target for suppressing metastatic dynamics and preventing tumor recurrence.

COME ONE COME ALL