# Pôle Méca's Internal Seminar

**Date and Time:** June 12, 2026 ( 11 - 12 am )
**Venue:** Amphi 104 (Pôle Méca)

## Finite Strain Ductile Phase-Field Modelling under Chemo-Thermo-Mechanical Coupling: Application to Viscoplastic Silicon Anode Particles

Armel TENE TESSE
IMSIA

**Abstract**

The urgent need to reduce CO emissions associated with fossil-fuel consumption represents a critical challenge for all sectors. Consequently, the expansion of electric vehicles (EVs) is accelerating and driving research and development of higher performance batteries with increased energy density. Silicon based composite materials are attracting significant interest due to their lithium-storage capacity, which is nearly ten times higher than that of conventional graphite anode employed in today's EVs market. However, this significant advantage of silicon materials comes at the cost of severe volumetric expansion [2] of particles during lithiation, which induces high mechanical stress and leads to rapid electrodes degradation such as formation of cracks in both the active materials [2] and the viscoplastic binders. As result, the cell capacity is loss, and battery lifetime is reduced.
The development of next generation batteries is therefore hindered by several scientific challenges: large inelastic deformation, coupled chemo thermo-mechanical effects, and ductile damage associated with breathing and swelling of silicon active particles during lithiation–delithiation. Existing literature still lacks a comprehensive Multiphysics [3] framework capable of capturing these phenomena under finite strains.
To address these limitations, we develop a ductile phase field model formulated in finite strains and coupled with viscoplasticity and chemically driven expansion. Damage evolution is driven by both elastic and plastic contributions to the free energy [1], ensuring a thermodynamically consistent description of the progressive degradation of silicon composite materials during cycling. Representative simulations demonstrate the ability of the framework to predict the evolution of plastic dissipation and the growth of damage under chemo mechanical coupling.
The model provides a general tool for analyzing ductile damage in electrode materials undergoing finite strains and paves the way for future extensions including cyclic fatigue degradation.

**References**

[1] Hu, Tianchen, Brandon Talamini, Andrew J. Stershic, Michael R. Tupek, and John E. Dolbow. 'A Variational Phase-Field Model For Ductile Fracture with Coalescence Dissipation'. Computational Mechanics 68, no. 2 2021: 311–35. https://doi.org/10.1007/s00466-021-02033-1.
[2] McDowell, Matthew T., Seok Woo Lee, William D. Nix, and Yi Cui. '25th Anniversary Article: Understanding the Lithiation of Silicon and Other Alloying Anodes for Lithium-Ion Batteries'. Advanced Materials 25, no. 36 2013: 4966–85.
[3] Shah, Sameep Rajubhai, Luize Scalco De Vasconcelos, and Kejie Zhao. 'Computational Modeling of Electrochemomechanics of High-Capacity Composite Electrodes in Li-Ion Batteries'. Journal of Applied Mechanics 89, no. 8 2022: 081005. https://doi.org/10.1115/1.4054759.

# Crowding sensitiveness in epithelium: when biking to the lab starts amechanobiology story

Louis LAURENT
LadHyX

**Abstract**

It's a sunny day, a perfect day to ride your bike to Pôle Méca. But an unfortunately placed rock on the road causes you to fall, resulting in an epithelial wound. This wound creates a local imbalance in cell density that your epithelial cells can perceive. In humans, this perception is a fundamental property that helps maintain a constant number of cells within these tissues to ensure their proper function. Yet, we still have limited knowledge about how this process works.
Better understanding of how epithelial tissues sense cell density means better understanding of how our body reacts so quickly to an epithelial injury, but also how cancer (an abnormally high cell density) can develop. Here, we study the effect of cell density on Extracellular-signal Regulated Kinase (ERK), a protein which plays a central role in regulating physiological epithelial behaviors (migration, proliferation, cell death...) and is commonly overactivated in cancers.
Using innovative approaches combining multiplexed fluorescent molecular biosensors and quantitative microscopy, our work shows that, in a physiological context, decreasing cell density leads to an increase in ERK activity due to mechanical forces changes within cellular mechanotransduction complexes. These complexes allow cells to regulate molecular signaling pathways based on the tissular mechanical forces. We also demonstrate that owing to these complexes, ERK entry into the cell nucleus (where our DNA resides) is strongly increased at low density. This is combined with increase in both accessibility of genes and activity of other nuclear effectors, all involved in modulating epithelial behavior. This whole mechanism is established 20 minutes after density change, thus providing epithelial cells with a rapid response capacity to local density variations.

Seminar webpage link