

# SCIENTIFIC SEMINAR



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## *A journey from the cytoplasm to the nucleus in hormone-related cancers*

Genetic, environmental, and metabolic insults disrupt chromatin homeostasis leading to abnormal epigenetic restriction or plasticity. Such aberrant chromatin states confer oncogenic properties through repression of tumor suppressors or activation of oncogenes. Moreover, these chromatin states are often dynamic, unlocking phenotypic plasticity in cancer cells, a notion now considered an emerging hallmark of cancer. In this updated scenario, perturbation of epigenetic homeostasis acquires a dual role: i) as a hallmark of cancer in oncogenic processes driven by mutation of epigenetic regulators and ii) as an enabling capability upon non-mutational epigenetic reprogramming. In the complex landscape of chromatin dynamics, metabolic intermediates represent crucial substrates for chromatin-modifying enzymes, creating an interdependent communication by reflecting microenvironmental perturbations onto the chromatin status. However, how metabolic deregulation driven epigenomic conditioning impacts cell fate remains elusive. This fact emphasizes the importance of understanding this two-way road, the metabolism-epigenetics axis, as a priority in cancer research. In our lab, we are interested in understanding the interplay between nuclear methylation metabolism, epigenetic regulation and cellular plasticity, with the ultimate goal of uncovering new therapeutic avenues for cancer patients.

**CICbioGUNE**

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Basque Foundation for Science

**Thursday**  
**June 26**  
**Atrio 800**  
**12.00H**



**BRTA**

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