

Symposium:
“Cancer beyond individual cells: Microbial signals, lipid metabolism and tumor ecosystems”

Speakers:

Lorena Lobos-Gonzalez: Faculty of Medicine, Universidad de Chile, Santiago, Chile
“Introduction to symposium topics”

Alejandro Corvalan: Faculty of Medicine, Pontificia Universidad Catolica de Chile, Santiago, Chile
“Helicobacter pylori-induced senescence and non-coding RNAs in gastric cancer”

Andrew Quest: Faculty of Medicine, Universidad de Chile, Santiago, Chile
“The adipose tissue-cancer connection”

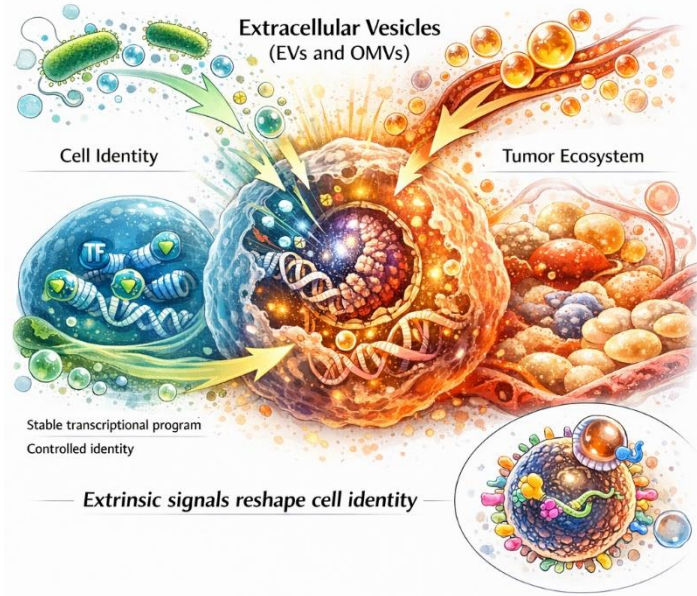
Andres Trostchansky: Faculty of Medicine, Facultad de Medicina, Universidad de la Republica, Montevideo, Uruguay
“Lipidomics analysis in cancer: EVs and lipid signaling to identify targets for intervention”

Dalhia Nurit Abramovich: Institute of Biology and Experimental Medicine (IBYME-CONICET), Buenos Aires, Argentina.
“Metformin: A Protective Tool in Gynecological Cancer Therapy”

Lorena Lobos-Gonzalez: Faculty of Medicine, Universidad de Chile, Santiago, Chile
“Closing remarks”

Cancer beyond individual cells

Microbial signals • Lipid metabolism • Tumor ecosystems



“Cancer beyond individual cells: Microbial signals, lipid metabolism and tumor ecosystems”

To function correctly, cells must maintain their identity established during development and tissue regeneration. In the first case, extrinsic and intrinsic signaling cascades define the differentiation pattern stem cells follow to become functionally distinct cells with specific functions in an organism. Following differentiation, cells must maintain their identity, and following cell division identity must be re-established. Cell identity is achieved as the consequence of an interplay between transcription factors (TFs), chromatin-associated proteins, and spatial genome organization, which in conjunction result in execution of a cell-type-specific gene expression program that must be rigorously controlled to ensure proper function of the differentiated cell types.

However, different factors challenge cell identity maintenance, emphasizing the need for proper control mechanisms at different stages of the cell cycle. First, the emergence of gene mutations or re-expression of lineage specifying genes can result in cell fate conversion thereby altering cell type definition, which often represents the driver of cancer development in the long term. Second, a perturbation of extrinsic stimuli can reshape the transcriptional program over time, driving a cell towards a different cellular state. In this symposium will discuss particularly the latter aspect and highlight how molecular crosstalk between different components in the tumor microenvironment can promote cancer development and progression and how modulating such intercellular communication can improve therapeutic outcomes.