DISIT Seminar Series

2026

Tuesday 24th
NOVEMBER

14.30 - 15.30

Room 203

Exploring Inflammation through Bioactive Peptides, Melatonin and NAMPT in Metabolic Disease and Multiple Sclerosis

Prof. Ignacio Bejarano

University of Sevilla

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Exploring Inflammation through Bioactive Peptides, Melatonin and NAMPT in Metabolic Disease and Multiple Sclerosis

Prof. Ignacio Bejarano

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Our research examines how plant-derived bioactive compounds and endogenous regulators influence inflammation, oxidative stress and metabolism in disease.

Our bench-to-table approach combines cellular studies, animal models and nutritional interventions. Lupin protein hydrolysates show anti-inflammatory, antioxidant and lipid-lowering effects, reducing cholesterol, visceral adiposity and fatty liver changes. This work extends to fermented beverages and hydrolysates obtained from food-production by-products, targeting obesity, dyslipidaemia, gut dysbiosis and metabolic dysfunction-associated steatotic liver disease.

This work led us to examine whether bioactive peptides could act together with melatonin. Although best known for regulating sleep and circadian rhythms, melatonin also influences immune responses, oxidative balance and cellular protection. We currently study the combined effects of lupin peptides and melatonin on obesity, metabolic dysfunction and fatty liver disease through animal models and clinical research.

This connection extends to multiple sclerosis. In experimental autoimmune encephalomyelitis, melatonin reduces immune-cell infiltration into the central nervous system, limits Th1 and Th17 responses, promotes regulatory immunity and enhances corticosteroid effects. These results supported MELATOMS-1, a clinical trial of melatonin with ocrelizumab in primary progressive multiple sclerosis.

Our newest collaboration with Valentina Audrito's group investigates NAMPT, a molecule that connects metabolism and inflammation. Inside cells, NAMPT maintains NAD production; outside cells, it can act as an inflammatory signal. By selectively inhibiting its intracellular activity or neutralizing its extracellular actions, we aim to determine how each function contributes to immune activation, neurological disability and central nervous system damage in EAE.

Together, these lines trace a continuous path from nutrition to immunometabolism and neuroinflammation, opening opportunities for collaboration across biology, chemistry, data science and quantitative modelling.

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