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FVB/N mouse, The Jackson Laboratory

➤ Effect of inflammation on epigenetic markers in the lactating mammary gland

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Introduction

Current strategies in animal husbandry and their consequences on animal health and welfare, and on the environment, are important societal concerns in terms of both ethics and sustainability. Ruminant **longevity** depends on phenotype expression related to health and production characteristics. Phenotype expression variability depends on genetic factors, but also **epigenetic factors** that reflect environmental influences. The goal of this project is to study **epigenetic marks** involved in **mammary inflammation** during **lactation** in the hopes of discovering **biomarkers of early inflammation** that aid in achieving a compromise between animal health and milk productivity.

Methods

Proof of concept: mouse model of **early mammary inflammation** via intramammary LPS injections (tissue sampled **4 hours** post-injection).

Mammary gland samples from 1st or 2nd lactation, with or without inflammation (5 or 7 mice).

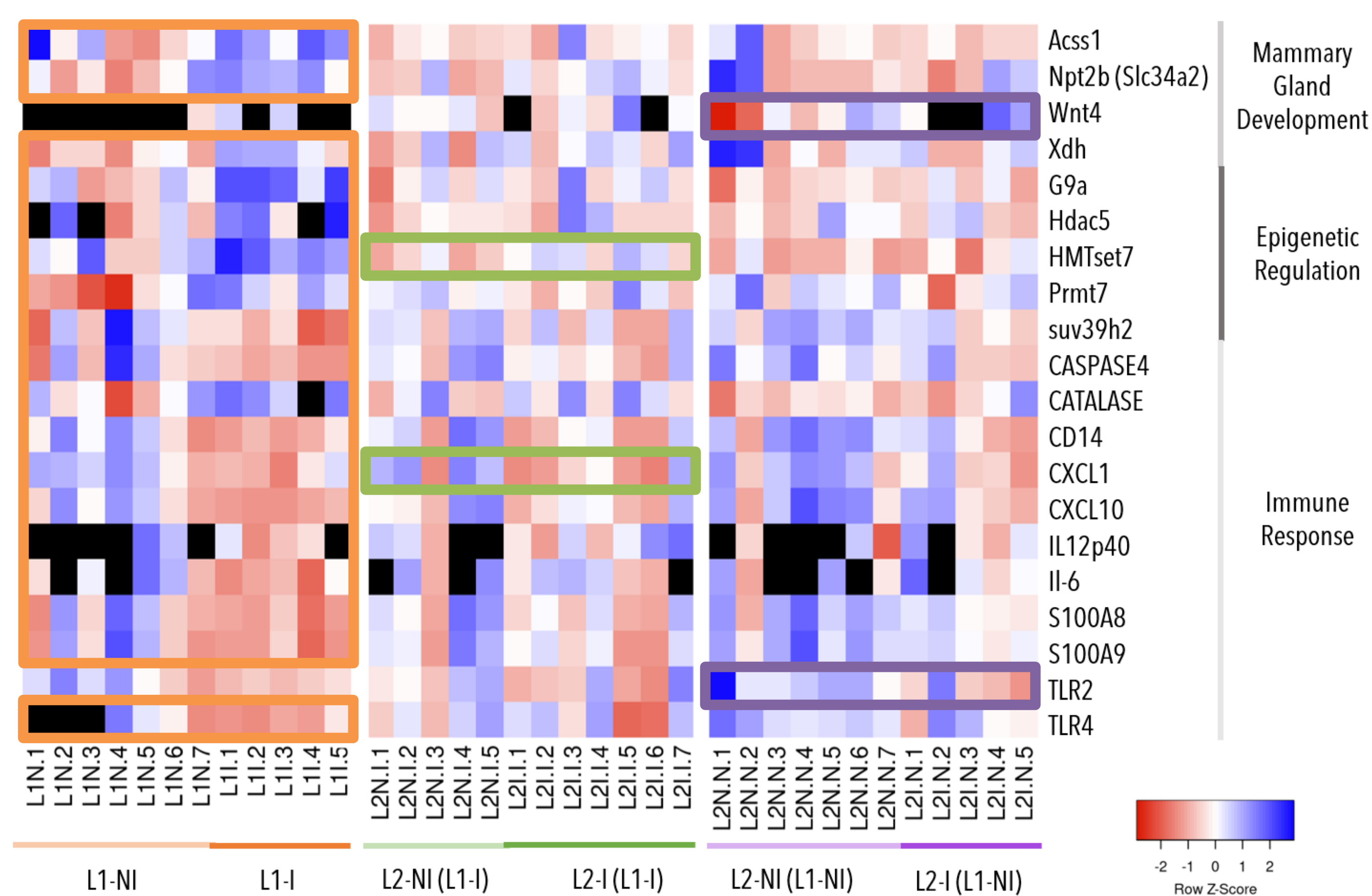
➤ **Gene expression** via RT-qPCR Fluidigm

➤ **DNA methylation** via RRBS

	Lactation 1 (L1)	Lactation 2 (L2)
Lot 1	NI	NI
Lot 2	I	NI
Lot 3	I	I
Lot 4	NI	I

NI: non-inflamed
I: inflamed

Mammary inflammation affects gene expression



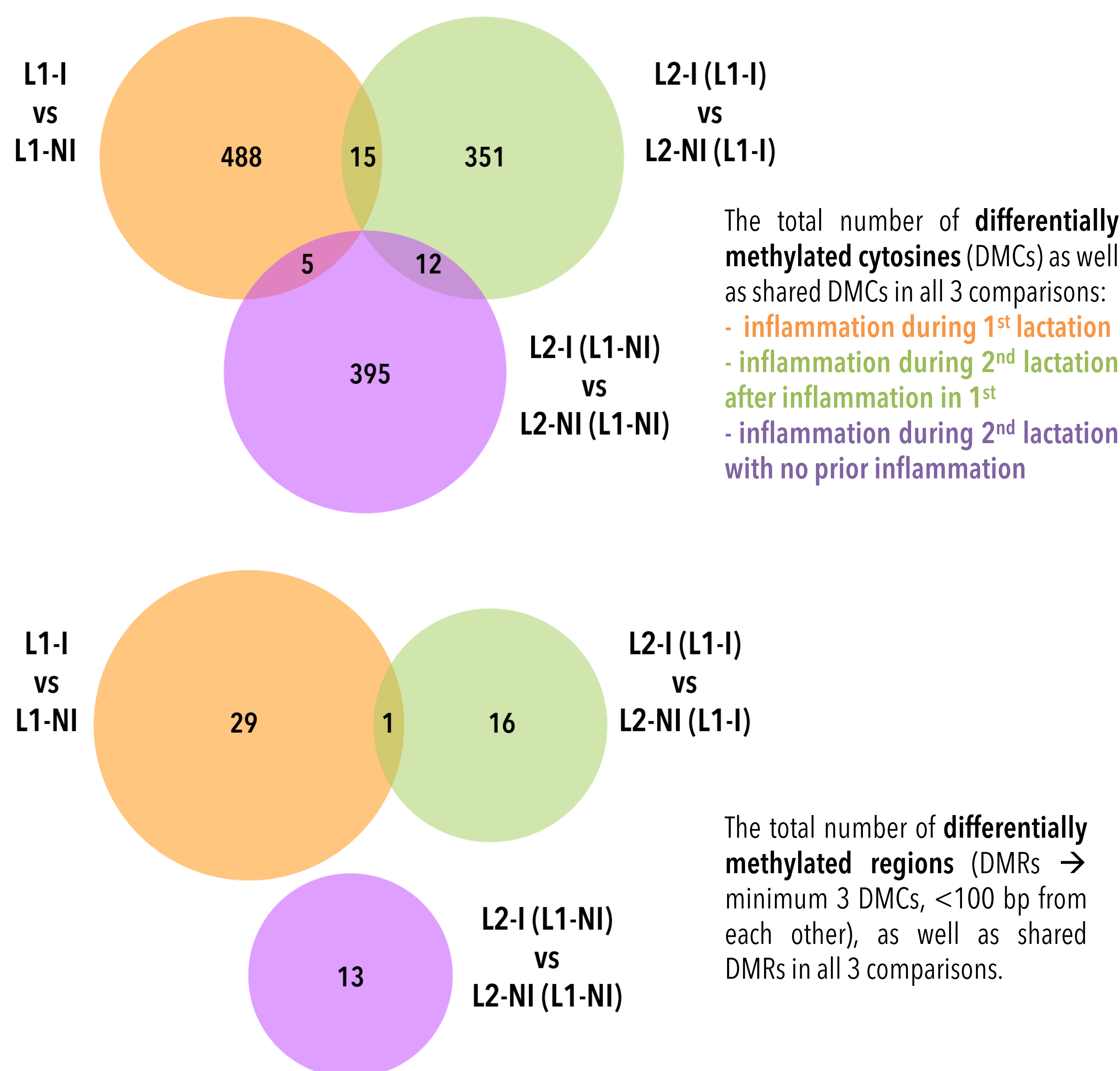
Gene expression ($\Delta\Delta C_t$) in 3 comparisons: **inflammation during 1st lactation**, **inflammation during 2nd lactation after inflammation in 1st**, and **inflammation during 2nd lactation with no prior inflammation**. The genes listed are differentially expressed ($p < 0.05$) in at least one comparison (color-coded circles). Each column represents one individual. Lowest gene expression is represented in blue, highest – in red.

Inflammation provokes **different gene expression changes** depending on lactation rank, with the **most** changes observed during **1st lactation**.

Discussion

Our results show that **differentially expressed genes** were **not differentially methylated**, suggesting other pathways for transcription regulation are involved. We found that **lactation rank** and **inflammation history** are important variables for determining markers of early mammary inflammation. Next, we will study whether differentially methylated genes in different conditions intervene in **similar signaling pathways**. Results were acquired in a mouse model with the objective of **transferring this knowledge to farmed species**. From this work, new leads will emanate which could lead to proposals for **new strategies** to be used in **breeding**.

DNA methylation changes caused by mammary inflammation



Inflammation provokes **different DNA methylation changes** depending on lactation rank and inflammation history.