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FVB/N mouse, The Jackson Laboratory

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

Ivanova E.¹, Hue-Beauvais C.¹, Chaulot-Talmon A.^{2,3}, Castille J.¹, Aubert-Frambourg A.^{2,3}, Kiefer H.^{2,3}, Germon P.⁴, Jammes H.^{2,3}, Le Provost F.¹

¹ GABI, INRAE, AgroParisTech, Université Paris-Saclay, 78350 Jouy-en-Josas, France

² Université Paris-Saclay, UVSQ, INRAE, BREED, 78350, Jouy-en-Josas, France

³ Ecole Nationale Vétérinaire d'Alfort, BREED, 94700, Maisons-Alfort, France

⁴ INRAE, Université de Tours, ISP, F-37380, Nouzilly, France

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).

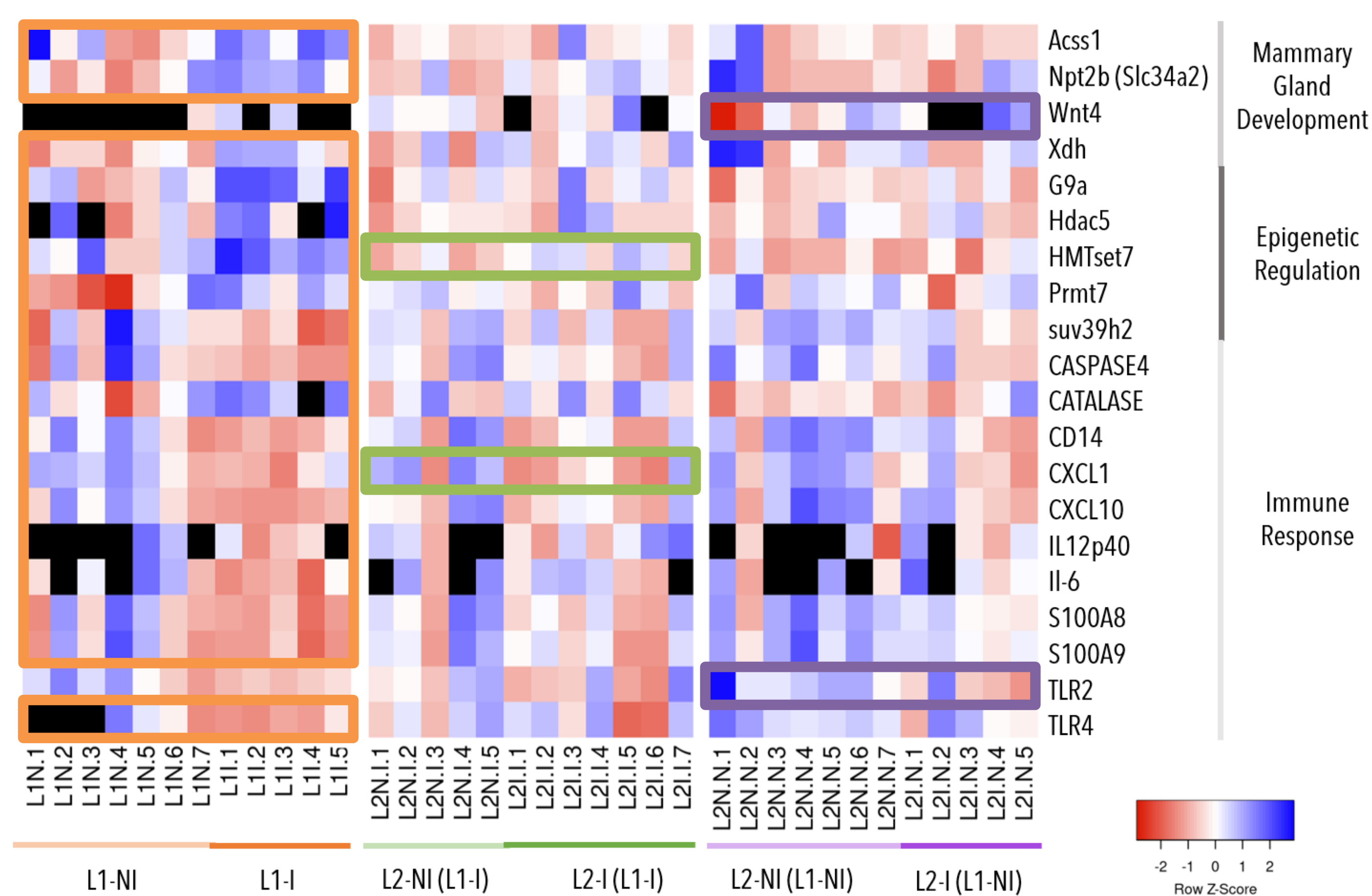
➤ **Gene expression** via RT-qPCR Fluidigm

➤ **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.

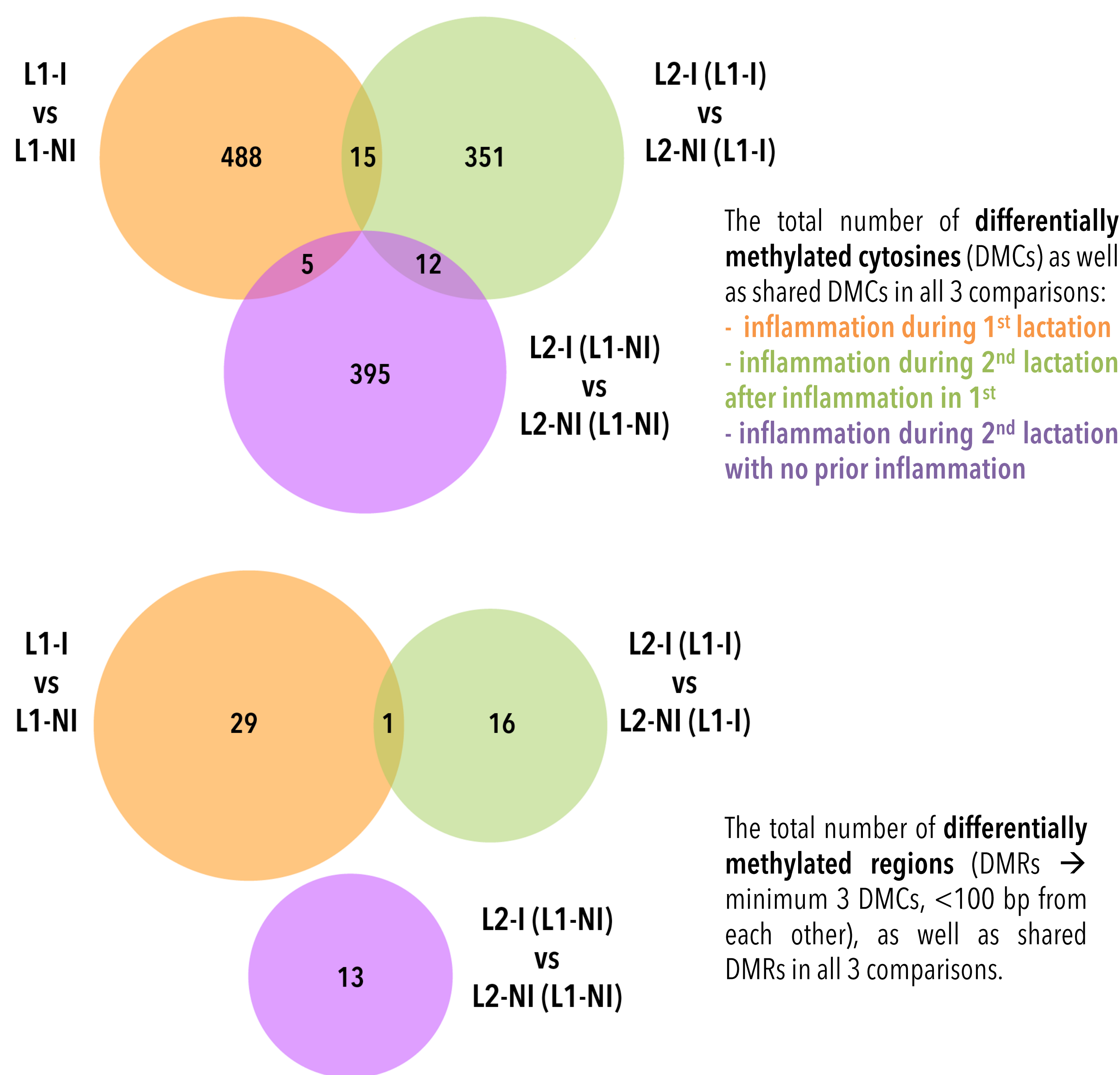
Inflammation provokes **different gene expression changes** depending on lactation rank, with the **most** changes observed during **1st lactation**.

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.