

# Effect of *Lactobacillus* strains on carriage of vancomycin-resistant *Enterococcus faecalis*

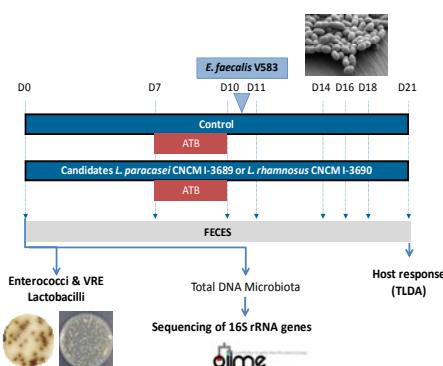
CROUZET L.<sup>1</sup>, DERRIEN M.<sup>2</sup>, CHERBUY C.<sup>1</sup>, CHALIN B.<sup>1</sup>, LANGELLA P.<sup>1</sup>, GROMPONE G.<sup>2</sup>, RIGOTTIER-GOIS L.<sup>1</sup>, SERROR P.<sup>1</sup>

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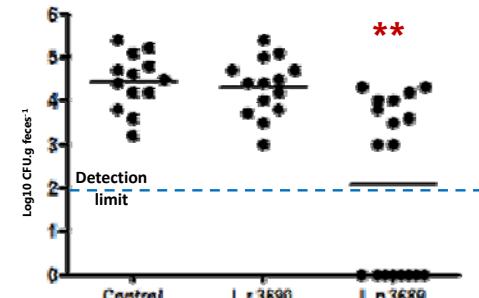
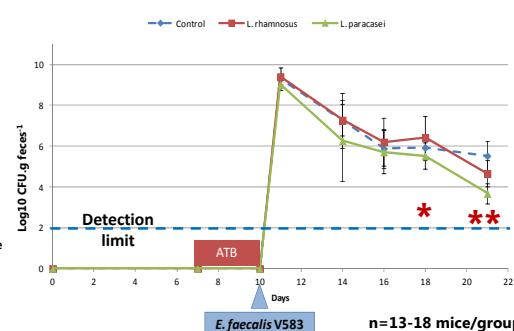
The gastrointestinal (GI) tract is a reservoir for bacterial opportunistic pathogens, which benefit from the disruption of the intestinal microbiota homeostasis (dysbiosis) to infect critically ill elderly and immuno-compromised patients. Enterococci are natural inhabitants of the subdominant gut microbiota and become dominant upon antibiotic treatments. Among them **vancomycin-resistant enterococci (VRE)** can cause systemic infections responsible for health-care associated pathologies with life threatening issues and increased hospital costs. Reduction of intestinal colonization or carriage after antibiotic treatment could limit the risks of VRE infections and dissemination.

We investigated the effects of two **potential probiotic lactobacilli** (*Lactobacillus rhamnosus* CNCM I-3690 and *Lactobacillus paracasei* CNCM I-3689) on **intestinal VRE carriage and clearance after antibiotic treatment**.

## Experimental strategy

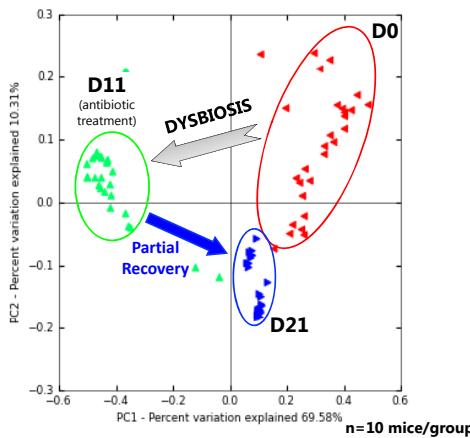


## *L. paracasei* CNCM I-3689 strain reduces *E. faecalis* V583 carriage



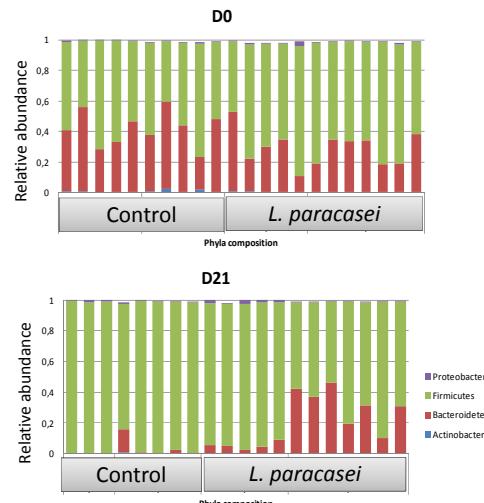
## Model of antibiotic-induced dysbiosis in the presence of *E. faecalis*

## *L. paracasei* CNCM I-3689 strain does not impact antibiotic-dependent dysbiosis

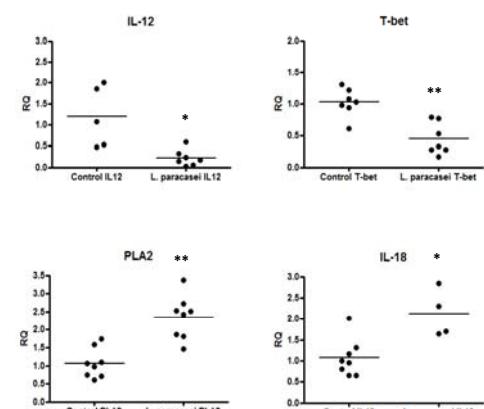


## *E. faecalis* V583 colonisation kinetics

## *L. paracasei* CNCM I-3689 strain improves Bacteroidetes recovery



## *L. paracasei* CNCM I-3689 supplementation impacts RNA expression of IL-12, T-bet, PLA2 and IL-18



## OTUs analysis of the microbiota composition

## Phyla analysis of the microbiota composition

## Host response analysis (Preliminary data)

Our results identify the *L. paracasei* CNCM I-3689 strain as a promising candidate to decrease VRE in the GI tract.

Sequencing of 16S rDNA of the microbiota showed that *L. paracasei* CNCM I-3689 increases recovery of Bacteroidetes phylum compared to control group.

Our findings suggest that *L. paracasei* CNCM I-3689 effect could be mediated by the gut microbiota and by the host response. A better understanding of the underlying mechanisms will contribute to propose non-antibiotic strategies to prevent enterococcal infections.