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Driving dairy powders functionality through innovative process

Gwénaél Jan

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Driving dairy powders functionality through innovative process



STLOpen Days
19-21 March 2019



Gwénaél JAN - Microbio



Food quality & safety



Develop safe, healthy, sustainable and hedonic fermented foods



Food spoilage



Reduce health risks and food losses and waste



Driving functionality of dairy powders through innovative processes

Animal health



Offer alternatives to antibiotic treatments (probiotics)



Health & wellbeing



Develop functional and designer food against modern lifestyle diseases

Driving functionality

→ through matrices



→ later during this session

→ through processes



→ now

Powders: why do we dry beneficial bacteria?



✓ avoids contamination



✓ easy to store



✓ easy to transport



✓ easy to implement



✓ easy to pack



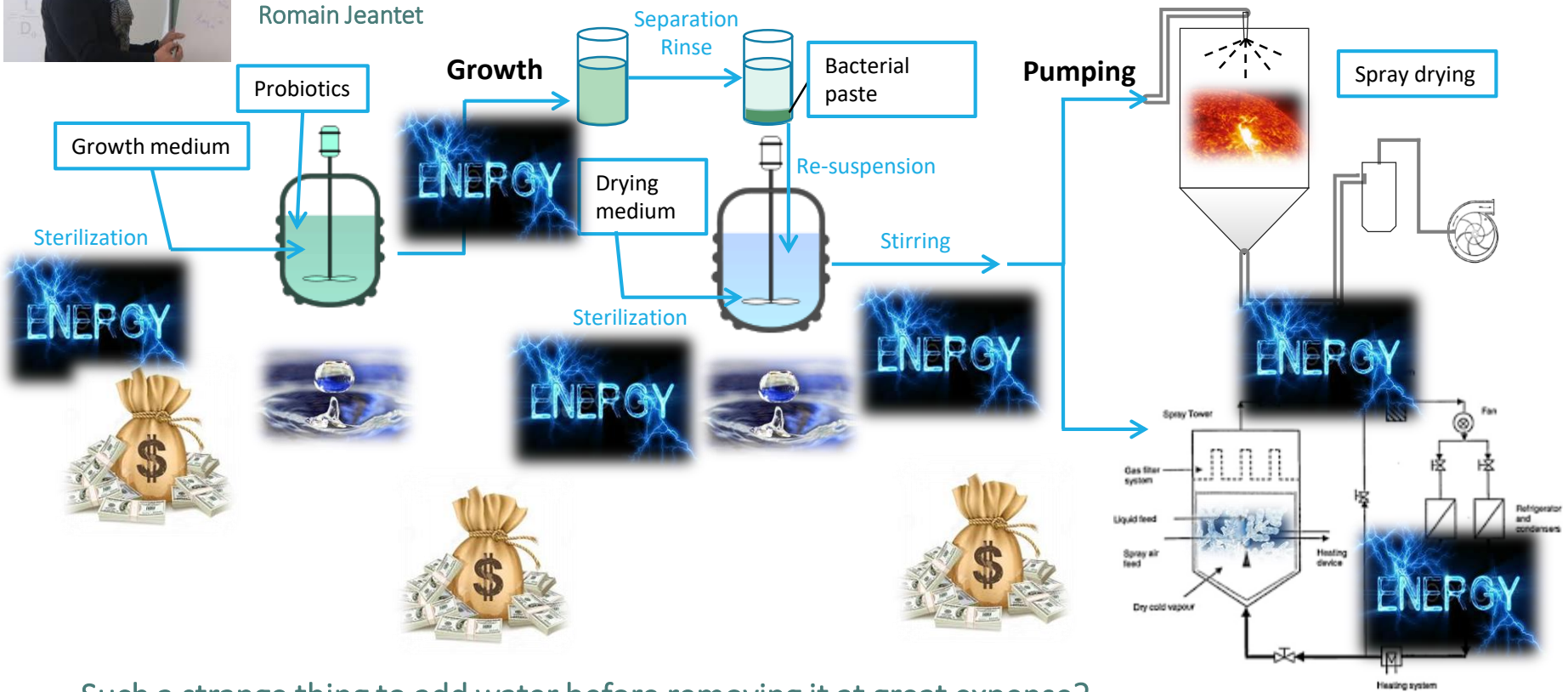
Powders: why and how do we dry beneficial bacteria?



See Poster 17 Floriane Gauher



Romain Jeantet



Such a strange thing to add water before removing it at great expense?

Freeze drying

Water is the problem: grow bacteria with less water!

Less water in the culture



Less water to evaporate

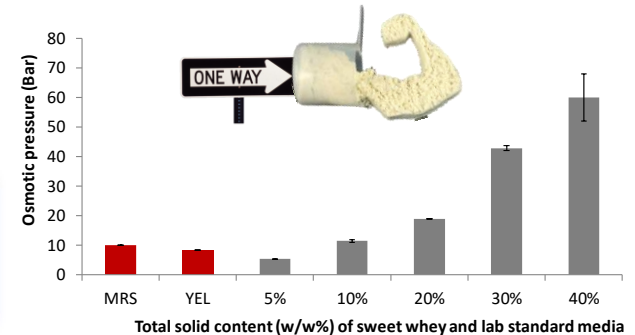


Less energy to spend

Exploit bacteria adaptation abilities



Exploit a by-product of food industry



Bugs grow and get tougher



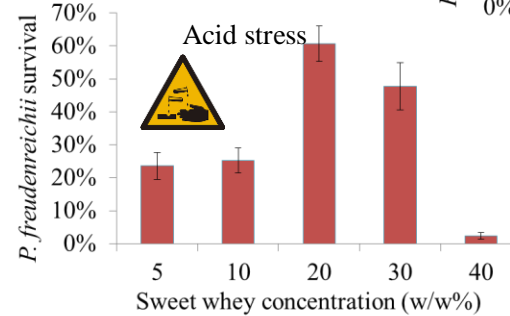
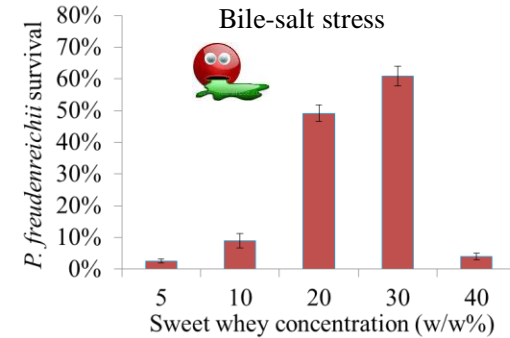
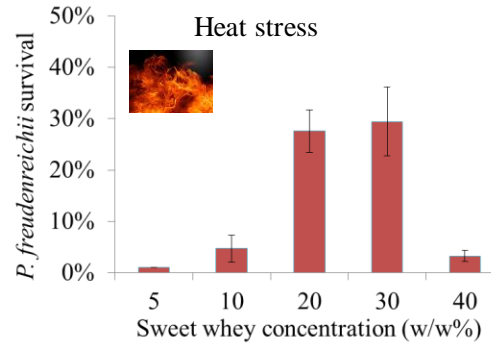
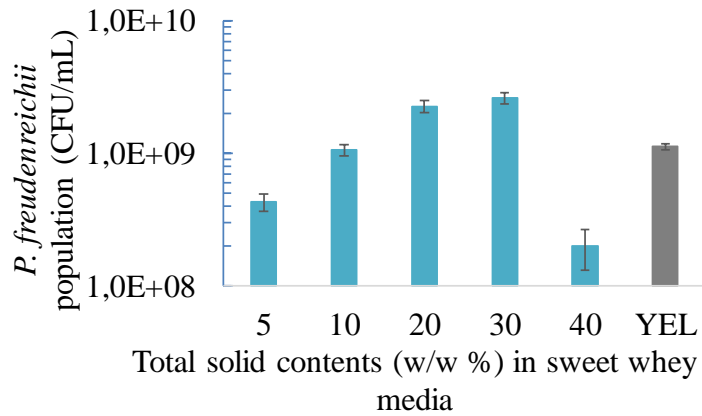
Propionibacterium



Bifidobacterium



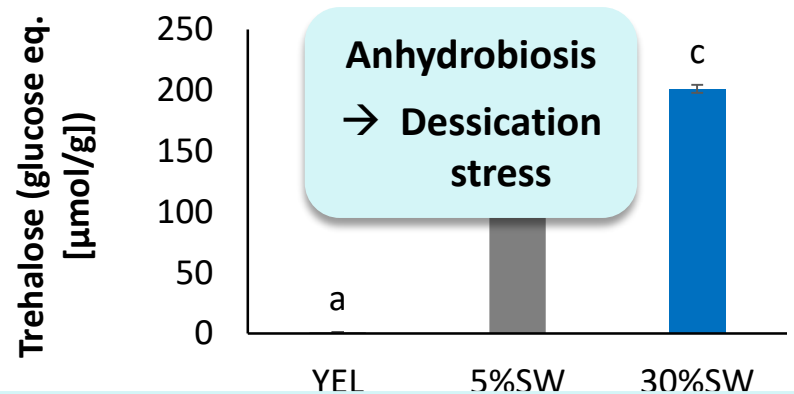
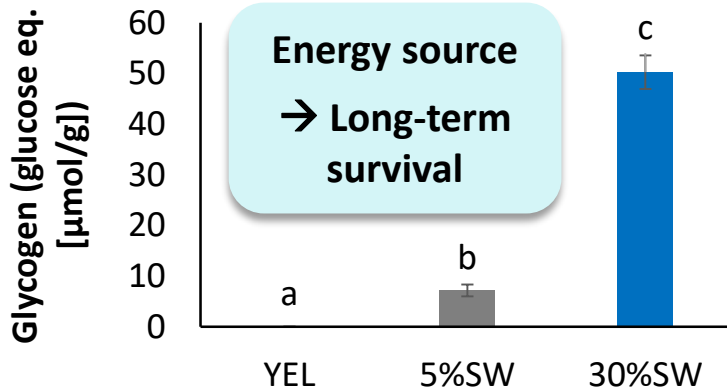
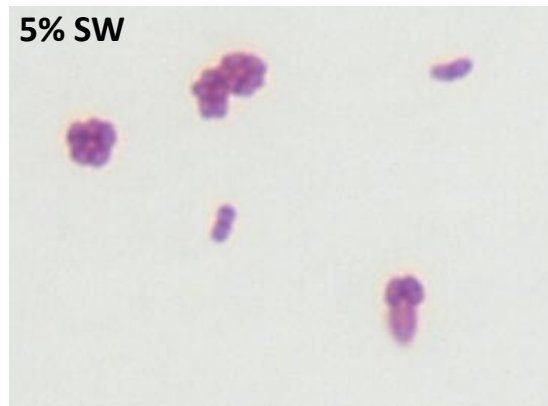
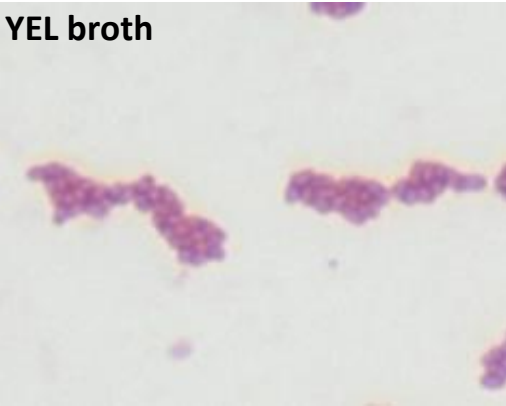
Lactobacillus



Bugs grow and get tougher



Periodic acid-Schiff staining

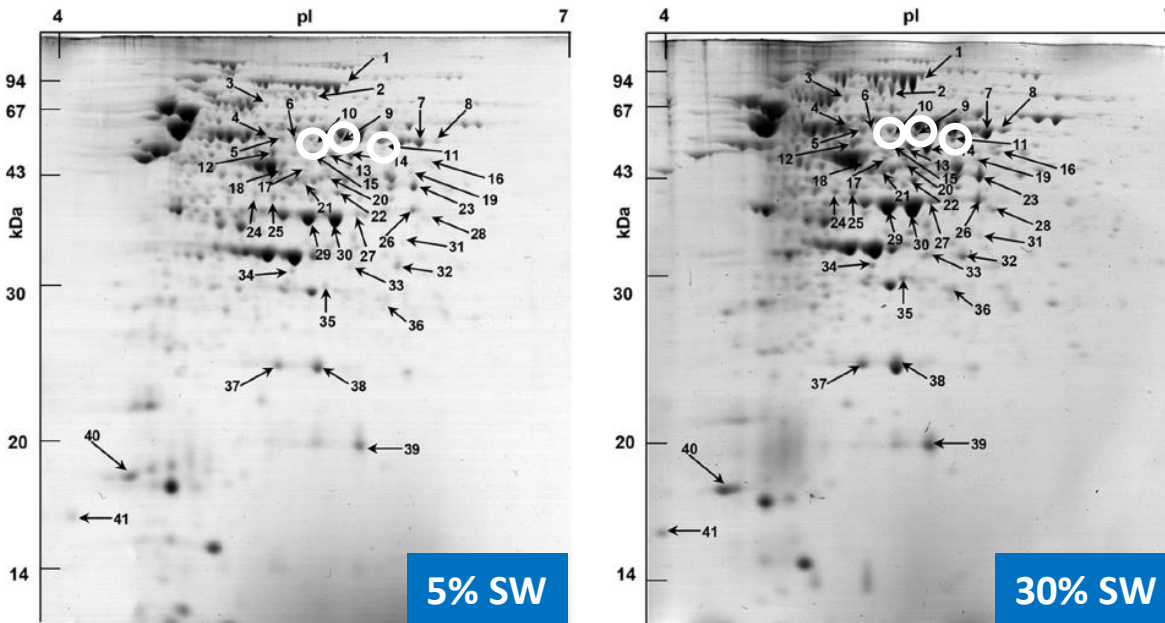


30% sweet whey induces osmoadaptation and sugar accumulation

Bugs grow and get tougher



2-D gel proteomic analysis



Osmoregulation proteins

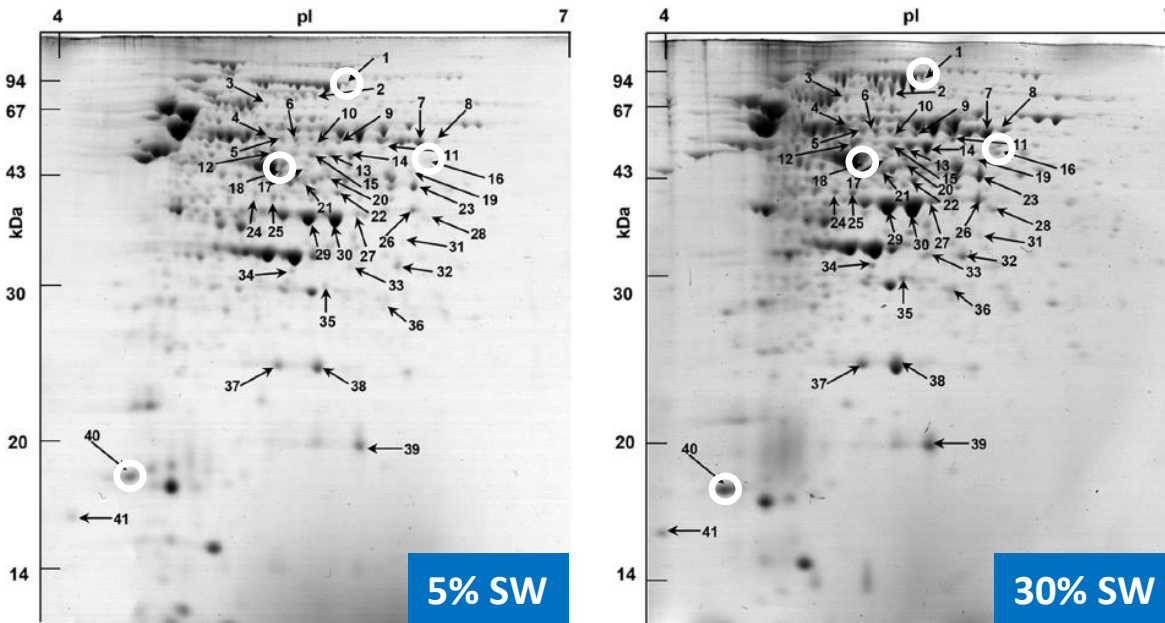
- **Glycine betaine transporter**
ATP-binding OpuCA of
osmoprotectant ABC transporter
- **Choline synthesis**
Glycine hydroxymethyltransferase
- **Proline synthesis**
Xaa-Pro aminopeptidase

30% sweet whey upregulates bacterial osmotic stress proteins
→ osmoadaptation

Bugs grow and get tougher



2-D gel proteomic analysis



Stress proteins

- **Multi-stress tolerance**
ClpB 2 chaperone
Elongation factor EF-Tu
- **Heat tolerance**
Heat shock protein 20
- **Oxidative tolerance**
Cysteine synthase

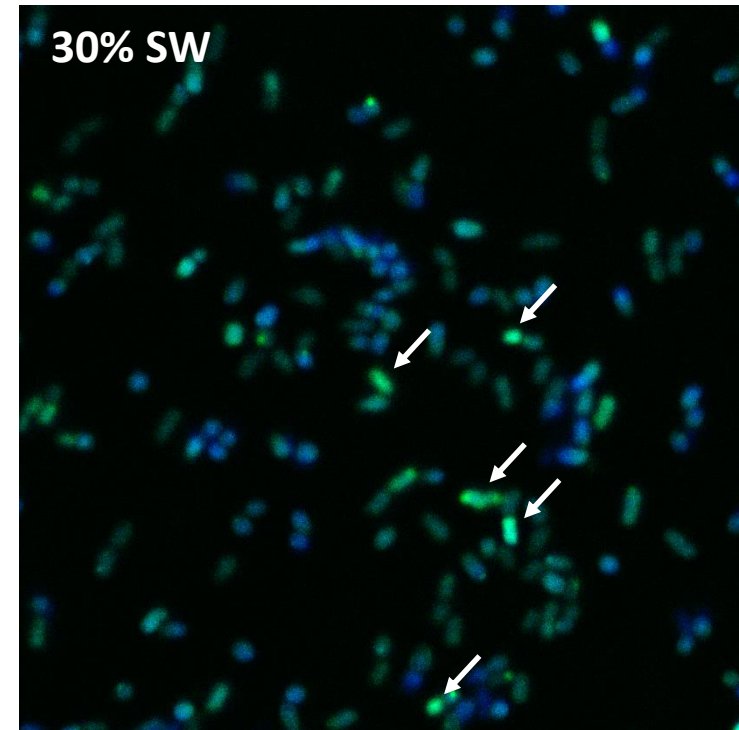
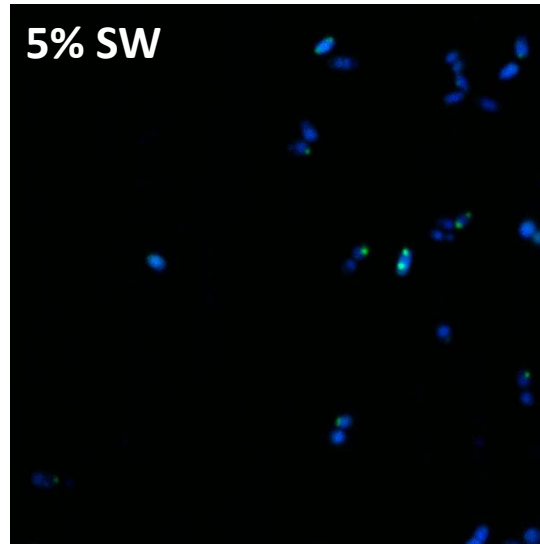
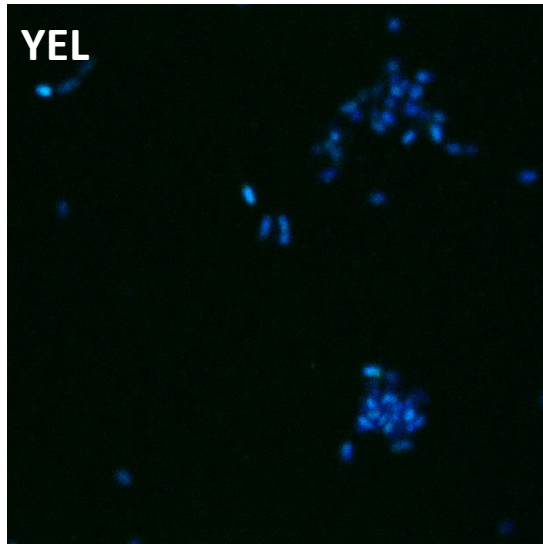
30% sweet whey upregulates bacterial multi-stress proteins
→ cross protection

Bugs grow and get tougher



DAPI staining, confocal microscopy

- Green fluorescence indicates poly(P)
- Blue indicates DNA



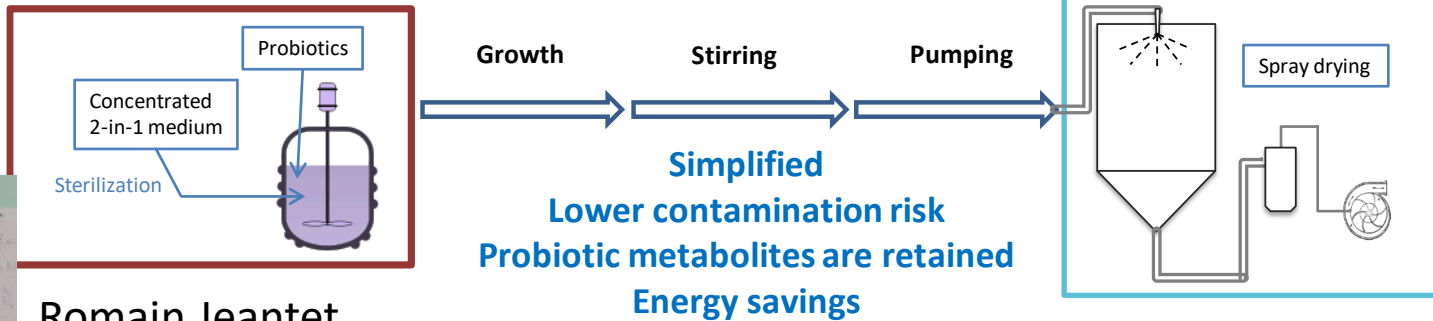
30% sweet whey induces bacterial polyP accumulation
→ cross protection / ATP homeostasis and repairing of damaged proteins

Current Opinion in Microbiology, 2015, 24:1–6

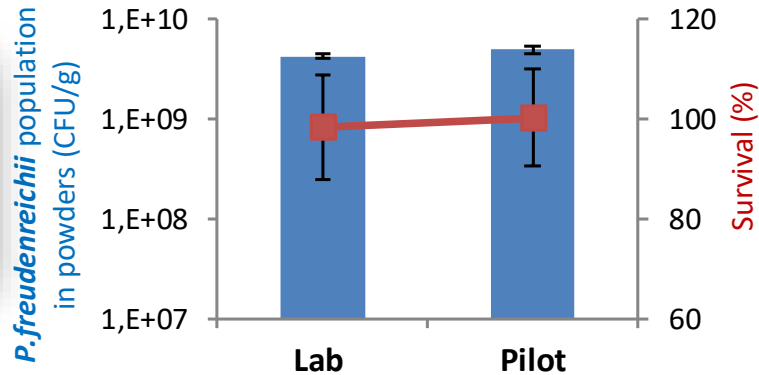
Bugs grow and get tougher



2-in-1 process



Romain Jeantet



Dairy Platform

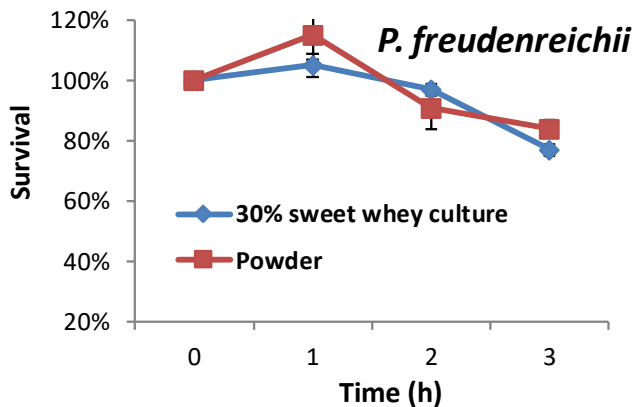


It's whey and it's a probiotic / a starter
 → functionalized whey powder

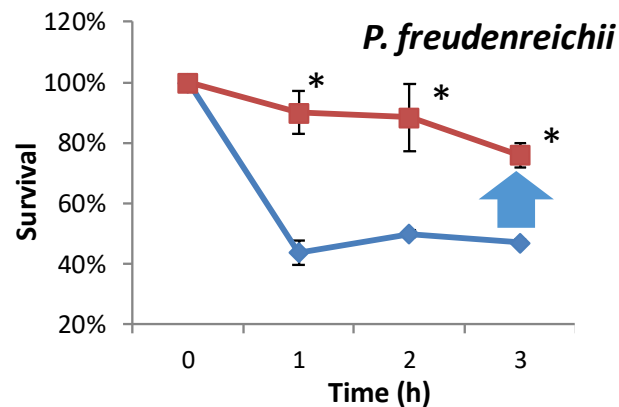
Functionalized sweet whey

In vitro digestion behavior: Fresh culture vs. Rehydrated powder

Simulated gastric fluid



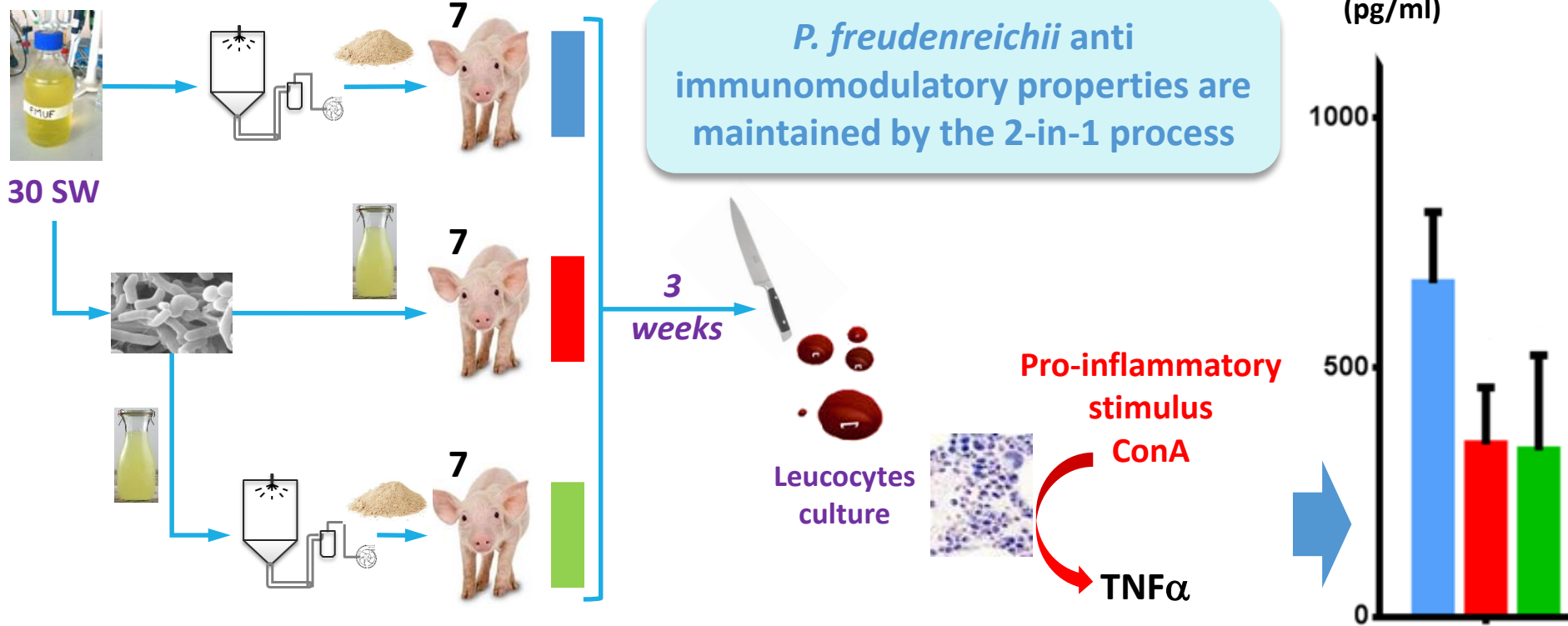
Simulated intestinal fluid



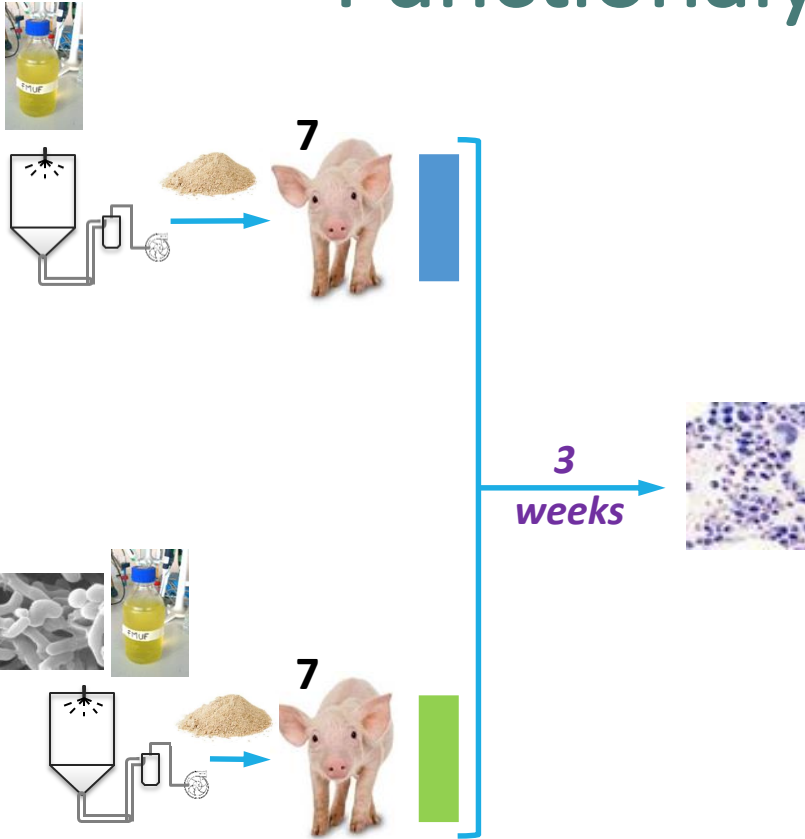
- Culture ≈ Powder in gastric survival
- Induced intestinal resistance, depending on the specie

Enhanced digestive stress tolerance
→ functionalized probiotic

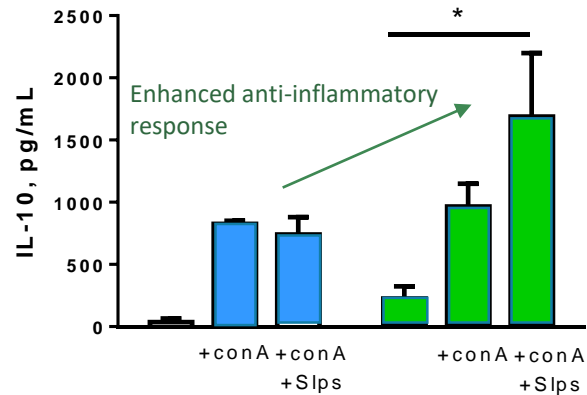
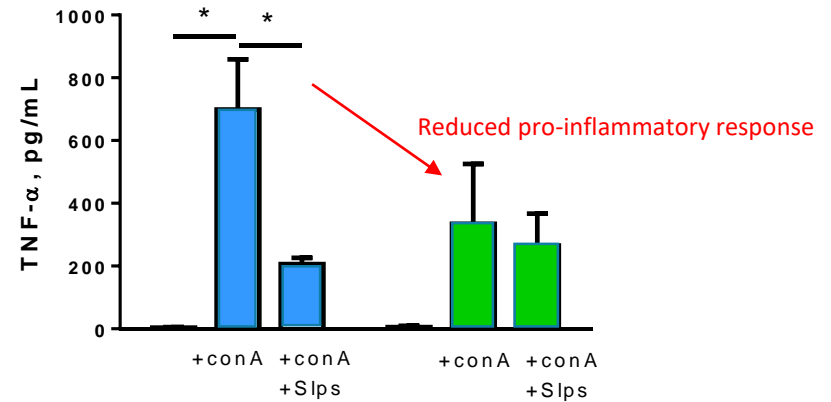
Functionalized sweet whey



Functionalized sweet whey



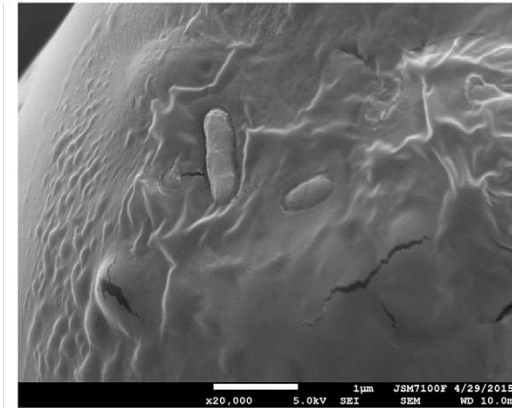
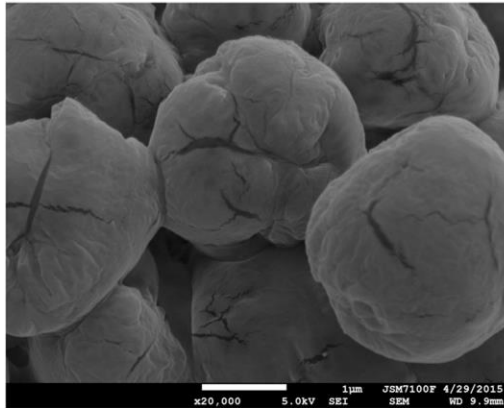
P. freudenreichii anti inflammatory properties are maintained by the 2-in-1 process



Functionalyzed sweet whey



Provide the missing bugs in a functionalized infant formula?



nature publishing group

Review

Bifidobacterium longum subspecies *infantis*: champion colonizer of the infant gut

Mark A. Underwood^{1,2}, J. Bruce German^{2,3}, Carlito B. Lebrilla^{2,4} and David A. Mills^{2,5}

Oligosaccharides are abundant in human milk. Production of these highly diverse structures requires significant energy expenditure by the mother and yet these human milk oligosaccharides offer no direct nutritive value to her infant. A primary function of human milk oligosaccharides is to shape the infant's intestinal microbiota with life-long consequences. *Bifidobacterium longum* subspecies *infantis* (*B. infantis*) is unique among gut bacteria in its prodigious capacity to digest and consume any human milk oligosaccharide structure, the result of a large repertoire of bacterial genes encoding an array of glycosidases and oligosaccharide transporters not found in other bacterial species. *In vitro*, *B. infantis* grows better than other bacterial strains in the presence of human milk oligosaccharides, displays anti-inflammatory activity in premature intestinal cells, and decreases intestinal permeability. In premature infants, *B. infantis* given in combination with human milk increases *B. infantis* and decreases Enterobacteriaceae in the

feces. Probiotics containing *B. infantis* decrease the risk of necrotizing enterocolitis in premature infants. Colonization with *B. infantis* is also associated with increased vaccine responses.

Probiotic organisms have historically been selected based on ease of production and stability. The advantages of *B. infantis*, selected through coevolution with human milk glycans, pres-

hypothesis that human milk contained "bifidogenic factors" that stimulated the growth of these bifidobacteria (1).

Probiotics are dietary supplements that promote health benefits by stimulating the growth and/or activity in the gut lumen of commensal microbes (ideally without stimulating potential pathogens); they do not contain live organisms. Probiotics are dietary supplements that do contain live organisms and are intended to promote health benefits through a variety of mechanisms. This article will focus on the coevolution of a collection of complex prebiotic oligosaccharides found in abundance in human milk and a single bacterial subspecies, *Bifidobacterium longum* subsp. *infantis* (*B. infantis*) unique in its capacity to consume these oligosaccharides. Note that the species *B. longum* has two subspecies: *B. longum* subsp. *infantis* and *B. longum* subsp. *longum*. These subspecies will be abbreviated in this review as *B. infantis* and *B. longum*, respectively.

HUMAN MILK OLIGOSACCHARIDES SHAPE THE INFANT INTESTINAL MICROBIOTA

Humans stand at the end of the long evolution of mammals, with differences in milk conspicuous for the volume, number of structures, and complexity of milk oligosaccharides (Figure 1) (2–4). Human milk oligosaccharides (HMOs) are

RESEARCH ARTICLE

The Journal of Clinical Investigation

Commensal *Propionibacterium* strain UF1 mitigates intestinal inflammation via Th17 cell regulation

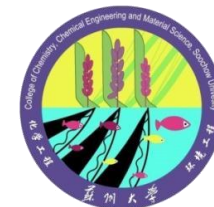
Natacha Colliou,^{1,2} Yong Ge,^{1,2} Bikash Sahay,¹ Minghao Gong,^{1,2} Mojgan Zadeh,^{1,2} Jennifer L. Owen,³ Josef Neu,⁴ William G. Farmerie,⁵ Francis Alonzo III,⁶ Ken Liu,⁷ Dean P. Jones,⁷ Shuzhao Li,⁷ and Mansour Mohamadzadeh^{1,2}

¹Department of Infectious Diseases and Immunology, ²Division of Gastroenterology, Hepatology & Nutrition, Department of Medicine, ³Department of Physiological Sciences, ⁴Division of Neonatology, Department of Pediatrics, and ⁵Interdisciplinary Center for Biotechnology Research, University of Florida, Gainesville, Florida, USA. ⁶Department of Microbiology and Immunology, Loyola University Chicago, Maywood, Illinois, USA. ⁷Division of Pulmonary, Allergy, Critical Care and Sleep Medicine, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia, USA.

Consumption of human breast milk (HBM) attenuates the incidence of necrotizing enterocolitis (NEC), which remains a leading and intractable cause of mortality in preterm infants. Here, we report that this diminution correlates with alterations in the gut microbiota, particularly enrichment of *Propionibacterium* species. Transfaunation of microbiota from HBM-fed preterm infants or a newly identified and cultured *Propionibacterium* strain, P. UF1, to germfree mice conferred protection against pathogen infection and correlated with profound increases in intestinal Th17 cells. The induction of Th17 cells was dependent on bacterial dihydrolipoamide acetyltransferase (DlaT), a major protein expressed on the P. UF1 surface layer (S-layer). Binding of P. UF1 to its cognate receptor, SIGIRR1, on dendritic cells resulted in the regulation of intestinal phagocytes. Importantly, transfer of P. UF1 profoundly mitigated induced NEC-like injury in neonatal mice. Together, these results mechanistically elucidate the protective effects of HBM and P. UF1-induced immunoregulation, which safeguard against proinflammatory diseases, including NEC.

More information

- Huang et al., 2016, *J Funct Foods* **23**, 455-463
- Huang et al., 2016. *Applied Env Microbiol* **82**, 4641-4651
- Yang et al., 2017. *Lett Appl Microbiol* **64**, 283-288
- Huang et al., 2017, *J Food Eng* **196**, 11-17
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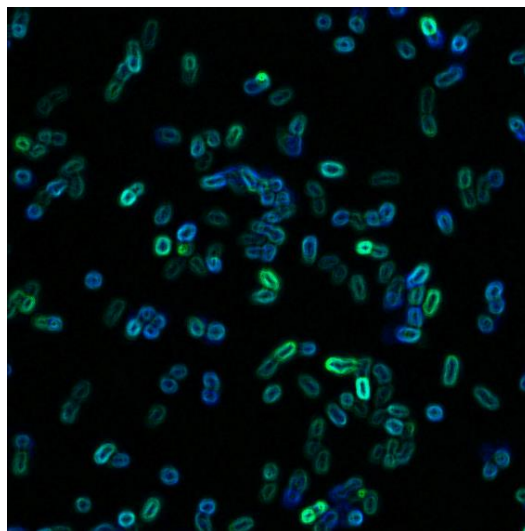
谢谢

**THANK YOU FOR
YOUR ATTENTION**



MERCI

Song Huang



Romain Jeantet