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Designing specific cheese-ripening ecosystems to shape the immune effects of dairy products?

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ABSTRACT

Although large numbers of viable microorganisms are ingested in ripened cheese, little is known about the microbial ecosystems' influence on the host's immune responses. We designed experimental smear-ripened cheeses with bacteria and yeasts that have opposite immune effects and evaluated their impact in the dextran sulphate sodium (DSS) and trinitrobenzene sulphonic acid (TNBS) colitis mouse models. Mice were fed with a control diet, a milk matrix or with lab-designed, 28-day-ripened prototype soft cheeses A and B (CheA and CheB) from cow milk that respectively hosted consortia of immuno-enhancing and immuno-modulatory microbial strains. Inflammatory markers and transcriptional signatures were evaluated in healthy mice colitic mice. In the DSS colitis model, there were no differences between CheA and CheB in terms of the inflammatory read-outs. In contrast, CheA (but not CheB) exacerbated weight loss and colon lesions in the TNBS model suggesting that designer cheeses may provide opportunities for diet management.

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1. Introduction

The role of dietary habits as an environmental risk factor (Gentschew & Ferguson, 2012; Ng et al., 2013) or a factor able to prolong remission in inflammatory bowel disease (IBD) (Rajendran & Kumar, 2010) has not received a great deal of research attention. Diet may influence gut inflammation through several biologically plausible mechanisms, including antigenic responses to food and alteration of the host microbiota (Clarke & Mullin, 2008; Viladomiu, Hontecillas, Yuan, Lu, & Bassaganya-Riera, 2013). However, the association between diet and IBD has not been clearly demonstrated – mostly because the etiology of both Crohn's disease (CD) and ulcerative colitis

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Abbreviations: CD, Crohn's disease; DSS, dextran sulphate sodium; IBD, inflammatory bowel disease; MPO, myeloperoxidase; PBMC, peripheral blood mononuclear cell; SAA, serum amyloid A; TNBS, trinitrobenzene sulphonic acid; TNF-α, tumor necrosis factor alpha; UC, ulcerative colitis

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(UC) are not fully understood. The impacts of genetic factors, microbiome diversity and environmental factors have been well documented. Some risk factors and modulators, however, may vary in their influence; for example, smoking cessation improves CD but worsens UC (Birrenbach & Bocker, 2004; Cosnes, 2004). Identification of the food types or components responsible for beneficial effects in IBD is tricky, since a range of doses and frequencies of macronutrients and micronutrients (including trace elements and pollutants) have to be considered. Although discriminating between causes and effects is challenging, there is a clear association between the incidence of IBD and loss of diversity within gut microbiota. This association fits with the hygiene hypothesis (Bach, 2002; Khosravi et al., 2014), in which the use of antibiotics and the increased consumption of refined food in our diet depletes the amount and activity of raw and fermented foods (Petrof, Claud, Gloor, & Allen-Vercoe, 2013). Thus, both nutritional food supplements and/or diets containing "healthy" food microbes are recommended.

Although it is widely acknowledged that the intake of the so-called probiotic microorganisms is able to influence the host's immune responses (Foligné, Daniel, & Pot, 2013; Marteau et al., 2004; van Baarlen, Wells, & Kleerebezem, 2013) epidemiological data on IBD and fermented foods (especially cheeses) are scarce. Some case-control studies have indicated that a high cheese intake is associated with CD (Maconi et al., 2010), whereas others have shown that patients with IBD eat cheese less frequently (Zvirbliene, Kiudelis, Zalinkevicius, & Kupcinskas, 2006). Meta-analyses of the impact of dairy products and cheese on diseases are inconsistent, since no particular cheese types or cheese-derived core microbial ecosystems have been clarified (Aune et al., 2012; Labonte, Couture, Richard, Desroches, & Lamarche, 2013). Although cheese-enriched diet was found to enhance anti-inflammatory and immune regulatory responses in normal mice and in a dextran sulfate sodium (DSS) colitis mouse model (Hosoya, Ogawa, Sakai, & Kadooka, 2012), the beneficial outcomes were not attributed to specific microorganisms (whether as live forms or inactivated). In addition to potentially bioactive peptides (Beermann & Hartung, 2013; Korhonen, 2009), fatty acids and amines in milk, dominant cheese microbiota should also be considered (Stanton, Ross, Fitzgerald, & Van Sinderen, 2005). Indeed, foodborne bacteria and fungi from cheese are detectable in the human distal colon, where they have been shown to be metabolically active and capable of altering the resident microbiota (David et al., 2014). Moreover, many microbial-derived antigens, secreted compounds, surface molecules and cell-wall components (such as peptidoglycan, exopolysaccharides, teichoic acids, and mannans) have immunomodulatory properties (Lebeer, Vanderleyden, & De Keersmaecker, 2010). Whereas the type of immune-related response clearly depends on the intrinsic characteristics of each microbe from defined genera and species (*e.g.* Gram-positive or Gram-negative bacteria, yeasts and fungi), precise immune tuning will be strain-specific (as demonstrated *in vitro* for probiotic strains of lactic acid bacteria (LAB) such as lactobacilli (Foligne et al., 2007a, 2007b; Nova et al., 2007) and bifidobacteria (Hoarau et al., 2008; López, Gueimonde, Margolles, & Suárez, 2010; Riedel et al., 2006) and yeasts (Foligné, Dewulf, Vandekerckove, Pignède, & Pot, 2010; Maccaferri, Klinder, Brigidi, Cavina, & Costabile, 2012; Romanin et al., 2010). Previous research has demonstrated the major role of bacterially induced IL-10 *in vitro* and the associated potential for relieving experimental colitis *in vivo* (Foligné et al., 2010; Foligne et al., 2007a, 2007b; Hoarau et al., 2008; López et al., 2010; Maccaferri et al., 2012; Peran et al., 2005; Riedel et al., 2006; Romanin et al., 2010). However, attempts to characterize the immune patterns induced by strains from cheese-ripening ecosystems are rare (Rahman et al., 2013) and have been restricted to non-starter LAB.

We have previously studied the diverse immunomodulatory properties of various microbial strains used in the preparation of dairy products (Adouard, unpublished data). By considering neutral, immuno-enhancing and immune-regulatory bacteria and yeasts, we defined distinct starter mixtures with opposite properties and used them to produce experimental cheeses likely to have opposite immune effects. Given that the health effects of probiotics are strain-specific, we postulated that multiple-strain preparations would be more effective than single-strain preparations (Timmerman, Koning, Mulder, Rombouts, & Beynen, 2004). In the context of gut inflammation, multiple-strain preparations have never been assessed in pre-clinical or clinical trials. There is increasing evidence to suggest that murine colitis may be prevented by administering several mixtures (Drouault-Holowacz et al., 2006; Gionchetti et al., 2012; Hart et al., 2004; Rachmilewitz et al., 2004; Roselli et al., 2009), although the efficacy varies with the composition of the mixture (Roselli et al., 2009). Similarly, the immune and health properties of multiple-strain and multiple-species probiotic mixes in a cheese ecosystem cannot be deduced from their respective components (Chapman, Gibson, & Rowland, 2011; Timmerman et al., 2007). Thus, we decided to screen various mixtures for their immuno-regulatory potential, with the objective of selecting optimal strains as tools for further cheese-making experiments (*i.e.* custom ripened dairy products with either neutral, anti- or pro-inflammatory-like dominant ecosystems). Indeed, prototype cheeses may be useful in evaluating the effects of immune intervention in pre-clinical models of immune-dysregulated diseases (such as colitis).

This type of tool would be of great interest for better dietary management in immune-related disorders and could open up new perspectives in cheese-making. For example, a custom-designed cheese could form part of an appropriate diet in a specific at-risk population or patient population.

The objective of the present study was thus to evaluate the potential impact of fermented dairy products (based on the immune determinants of their microbial components), rather than to design a "probiotic cheese" *per se*.

2. Material and methods

2.1. *Microorganisms, growth conditions and mixtures*

In the present study, 14 strains (10 bacteria, 3 yeasts and 1 fungus isolated from a dairy environment) were considered. Furthermore, five reference strains (*Bifidobacterium longum* Bb536, *Escherichia coli* TG1, *Lactobacillus acidophilus* NCFM, *Lactobacillus salivarius* Ls33 and *Lactococcus lactis* MG1363, Table S1) were used for immune cell stimulation, as previously described (Foligné et al., 2010). All strains used in this study were

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