



## Investigation of immune response to *Eimeria maxima* in broilers

Olivier Demeure, Edin Hamzic, Herve Juin, Murielle Naciri, Helle R. Juul-Madsen, Ron Okimoto, Marie-Hélène Pinard-van Der Laan, Bertrand Bed'Hom

### ► To cite this version:

Olivier Demeure, Edin Hamzic, Herve Juin, Murielle Naciri, Helle R. Juul-Madsen, et al.. Investigation of immune response to *Eimeria maxima* in broilers. 41. Meeting and Summer School of the Scandinavian Society for Immunology Meeting, Apr 2013, Copenhagen, Denmark. Wiley-Blackwell, Scandinavian Journal of Immunology, 77 (4), 2013, Scandinavian Journal of Immunology. 10.1111/sji.12036 . hal-01191355

**HAL Id: hal-01191355**

**<https://hal.science/hal-01191355>**

Submitted on 3 Jun 2020

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Final Programme & Abstracts



# 41<sup>st</sup> Scandinavian Society for Immunology Meeting

Copenhagen · Denmark · April 14-17 2013



## Abstracts

SSI13-1097

**xylo-oligosaccharide supplemented diet modulates intestinal and systemic immunity**

S. Metzdröff<sup>1,†</sup>, A. Christensen<sup>1</sup>, C. Hansen<sup>1</sup>, A. Bergström<sup>2</sup>, T. Licht<sup>2</sup>, A. Hansen<sup>1</sup>, H. Frøkiær<sup>1</sup>

<sup>1</sup>Copenhagen University, Frederiksberg, <sup>2</sup>National Food Institute, Mørkhøj, Denmark

**Abstract Body:** Xylooligosaccharides (XOS) represent a prebiotic candidate demonstrated to increase bifidobacteria in mice feces when supplemented in the diet. As this bifidogenic effect has only been demonstrated in fecal samples, the aim of this study was to investigate if XOS changes the microbiota throughout the entire intestine and examine its effects on mucosal and systemic immune parameters in mice. A 10% XOS supplemented diet revealed a significant increase in the *Bifidobacterium* group throughout all segments of the intestine with the highest relative increase in ileum. In intestinal epithelial cells (IEC), most genes involved in innate immune responses were unaffected, while the expression of the defensin RegIIIγ, known to limit bacterial colonization of the mucosal surface, was significantly up-regulated in the small intestine. The up-regulation of RegIIIγ may explain the unaffected innate immune response seen in this study, as the homeostatic spatial relationship between the gut microbiota and the host is maintained regardless of the increase in the *Bifidobacterium* group.

We hypothesized that the XOS diet increases SCFA production, and when absorbed, this leads to increased SCFA in the blood. Here, SCFAs bind to the SCFA receptor GPR43 on neutrophils, causing a down-regulation of the pro-inflammatory cytokine IL1β. Indeed, the IL1β and also *Ifn*γ expression were significantly decreased by XOS diet, indicating that SCFAs modulate the systemic immune response. *In vitro* treatment of whole blood with propionate led to a decrease in the IL1β expression, thus supporting that increased SCFA production was involved in the IL1β down-regulation observed in the XOS fed mice. A decreased proportion of IFN-γ producing CD4 T cells were detected in mesenteric lymph nodes of XOS fed mice, but not in spleen. Together with the IL1β and *Ifn*γ reduction in blood, this supports an anti-inflammatory effect of XOS diet in both mucosal and systemic immune compartments. Taken together, our results show that XOS feeding decreases systemic and mucosal inflammation and this effect is likely to be due to an increase in bifidobacteria in the small intestine leading to up-regulation of RegIIIγ and increased SCFA production.

**Disclosure of Interest:** None Declared

SSI13-1106

**investigation of immune response to eimeria maxima in broilers**

O. Demeure<sup>1,2</sup>, E. Hamzic<sup>3,4,5,\*</sup>, H. Juin<sup>6</sup>, M. Naciri<sup>7</sup>, H. R. Juul-Madsen<sup>8</sup>, R. Okimoto<sup>9</sup>, M. H. Pinard-van der Laan<sup>3</sup>, B. Bed'Hom<sup>3</sup>

<sup>1</sup>UMR1348 PEGASE, Agrocampus OUEST, <sup>2</sup>UMR1348 PEGASE, INRA, Rennes, <sup>3</sup>UMR1313 GABI, INRA, Jouy-en-Josas, <sup>4</sup>UMR1313 GABI, AgroParisTech, Paris, France, <sup>5</sup>Department of Molecular Biology and Genetics, Aarhus University, Tjele, Denmark, <sup>6</sup>EASM, INRA, Surgères, <sup>7</sup>Laboratoire de Contrôle et Immunologie de Maladies à Protozoaires, INRA, Nouzilly, France, <sup>8</sup>Department of Animal Science, Aarhus University, Tjele, Denmark, <sup>9</sup>Cobb-Vantress Inc., Siloam Springs, United States

**Abstract Body: BACKGROUND**

Coccidiosis is a parasitic disease of the intestinal tract of animals which causes extreme economic losses. A possible approach to control coccidiosis is to select animals for enhanced immune response. Therefore, it is necessary to find the most relevant phenotypes describing the genetic variation underlying the immune responses to coccidiosis.

**OBJECTIVES**

The aims of this study were i/ to investigate the response of Cobb500 broilers to *Eimeria maxima* challenge and test possible individual differences that could be due to genetic diversity and ii/ to find which phenotypes are good predictors of the sanitary status of the animals and could be measured at a large scale for a further genome-wide association study.

**METHODS**

The experiment lasted 23 days with 200 Cobb500 animals being challenged at day 16 while 40 animals were used as controls. Body weight was measured at 1, 8, 16 and 23 days. After the slaughter of the animals at day 23, the following phenotypes were measured: weight loss between day 16 and 23, rectal temperature, intestinal lesion score, hematocrit level, oocysts count and plasma coloration measured by absorbance at 405, 450, 490, 550, 590 and 650 nm.

## RESULTS

Difference in weight gain between infected and control animals was highly significant ( $p < 0.0001$ ). Further, rectal temperature was lower in infected animals ( $p < 0.05$ ), while animals with the highest lesion score had higher temperature. Lesion scores in jejunum were higher than those in duodenum. Hematocrit level was significantly lower in challenged animals compared to controls ( $p < 0.05$ ). There was highly significant difference in plasma coloration ( $p < 0.0001$ ) between infected and control animals for all wave lengths measured. Weight loss had the highest correlation with duodenum lesion score ( $r = -0.307$ ) and hematocrit level ( $r = -0.205$ ). The highest correlation with lesion scores ( $r = -0.4$ ) was obtained with plasma coloration measured at 490 nm. Finally, the variation coefficients were larger in challenged animals as compared to controls for traits measured.

## CONCLUSION

Significant differences in the response to an *Eimeria maxima* challenge between animals indicate that the individual variations found are likely caused by genetic differences. Weight loss is considered as the most important trait for explaining the response to an *Eimeria maxima* challenge. Finally, hematocrit level and plasma coloration can be used as predictors for growth variation and intestinal lesion score, respectively.

**Disclosure of Interest:** None Declared

## Innate Immunity

SSI13-1116

### Induction of IFN- $\beta$ by *Lactobacillus acidophilus* is dependent on phagocytosis and is abrogated by TLR ligand-induced macropinocytosis

L. Henningsen<sup>1</sup>\*, D. F. M. Silva<sup>1</sup>, S. B. Metzendorf<sup>1</sup>, H. Frøkiær<sup>1</sup>

<sup>1</sup>Department of Veterinary Disease Biology, University of Copenhagen, Faculty of Health and Medical Sciences, Frederiksberg, Denmark

**Abstract Body:** Background: Immature dendritic cells (DC) engulf large volumes of extracellular fluid through macropinocytosis. This may be increased further upon ligation of single TLR ligands such as LPS and Pam3CSK4 to TLR on DC (West et al. 2004). In contrast, phagocytosis of bacteria is dependent on specific sequential interactions between the surface of the bacteria and receptors on the DC. How these two different modes of uptake affect the immune response is not well understood. *Lactobacillus acidophilus* induces a strong IFN- $\beta$  response in bone-marrow derived DC (BMDC). The induction is dependent on internalization as well as on spleen tyrosine kinase (Syk) signalling (Weiss et al. 2010). Syk is also known to play a role in different types of endocytosis (Y. Tohyama and H. Yamamura 2009). We hypothesise that *L. acidophilus* uptake via phagocytosis is a prerequisite for IFN- $\beta$  induction.

**Objective:** In the present work the aim is to study the nature of endocytosis involved in uptake of *L. acidophilus* by DC and how this affects the induction of IFN- $\beta$ .

**Methods:** Endocytosis in DC was quantified by measuring the uptake of fluorescence-labelled dextran or bacteria by flow cytometry and visualised by confocal microscopy. Induction of IFN- $\beta$  and other cytokines in BMDC was measured by qPCR and ELISA.

**Results:** Increased macropinocytosis was induced upon stimulation with the TLR ligands LPS, Pam3CSK4 and the Gram negative *E. coli*, but not by *L. acidophilus*. Addition of LPS, Pam3CSK4, or *E. coli* to the BMDC 30 minutes prior to *L. acidophilus* increased the uptake of *L. acidophilus* and inhibited the IFN- $\beta$  induction by *L. acidophilus*. Inhibition of Syk decreased the number of cells phagocytosing *L. acidophilus*, but did not affect the TLR-ligand induced macropinocytosis of *L. acidophilus*.

**Conclusion:** The strong IFN- $\beta$  induction by *L. acidophilus* is based on Syk-dependent phagocytosis as opposed to endocytosis through macropinocytosis. Induction of macropinocytosis by TLR ligands or *E. coli* prior to *L. acidophilus* stimulation abrogates phagocytosis and IFN- $\beta$  induction by *L. acidophilus*.

**References:** West M.A. et al. (2004); *Science*, 305:1153-1157

Weiss, G. M. et al. (2010); *Immunology*, 131: 268-281