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Genetic-Microbiota Interplay in Laying Hens: Enhancing Vaccine Response and Reducing Antibiotic Reliance in Poultry Production

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Genetic-Microbiota Interplay in Laying Hens: Enhancing Vaccine Response and Reducing Antibiotic Reliance in Poultry Production

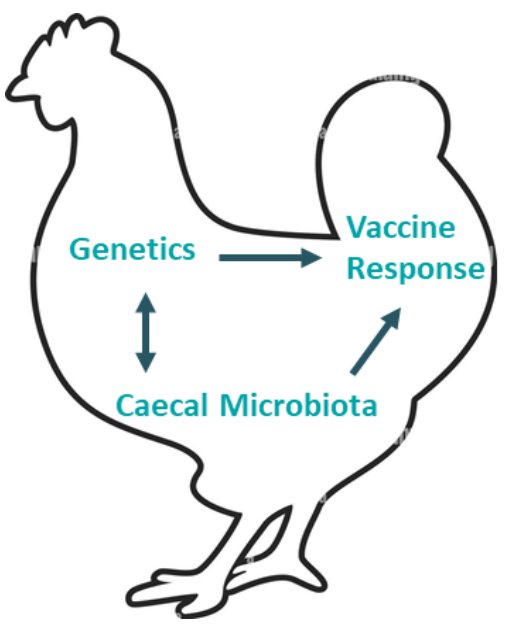
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Introduction

Vaccination stands as the primary and most effective approach to prevent infectious diseases in livestock; however, the efficacy of vaccines often varies individually. Understanding the factors influencing efficacy, including genetic variations in the host and gut microbiota composition, is crucial. We present here the first part of our project, which focuses on how the host genetic variations of 400 White Leghorn hens (LEG) is impacting the vaccine response.



Material and Method

Experimental Design

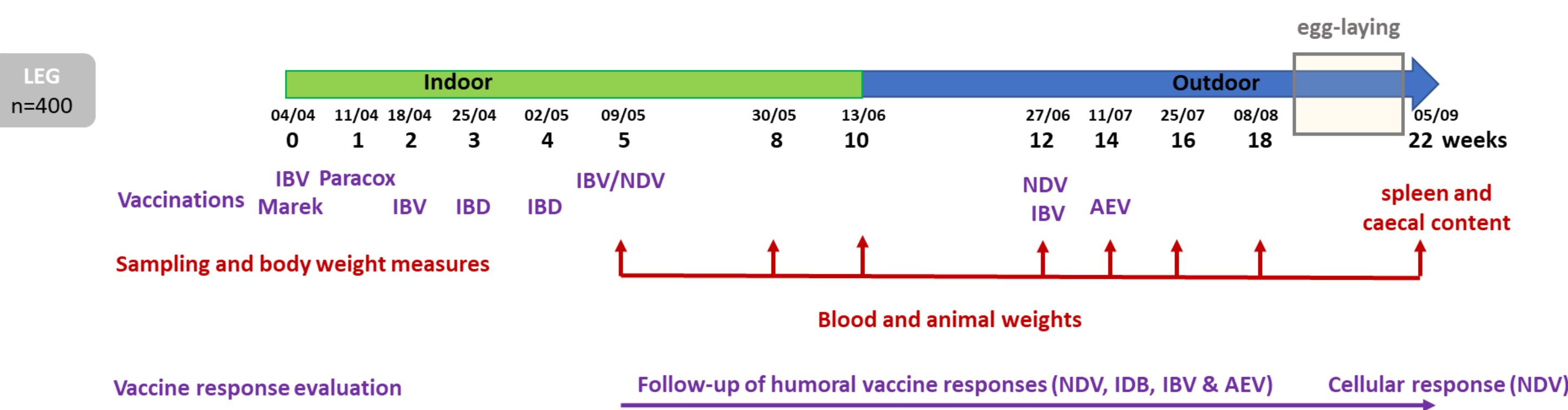


Figure 1. Timeline of the experiment: 400 LEGs indoors until week 10 and outdoor access between weeks 10 and 22. It is shown which vaccine was administered and at what time of their lives, when blood samples were collected (5, 8, 10, 12, 14, 16, 18 and 22 weeks), and spleen and caecum contents (22 weeks).

Vaccination & vaccine response

- 4 vaccines administrated to 400 LEG: Infectious Bursal Disease (IBD), Newcastle Disease Virus (NDV), Infection Bronchitis Virus (IBV) and Encephalomyelitis Virus (AEV).
- Blood samples collected at different times (see **Figure 1.**) to perform ELISA (enzyme-linked immunosorbent assay) tests for humoral response.

Genotyping & GWAS analyses

- Animals were genotyped (Labogena, Jouy-en-Josas) with a 57k SNP chip.
- The individual genotypes were used with the individual phenotypes (vaccine responses) to perform:
 - ✓ genome wide association analyses (GWAS) with the GCTA software;
 - ✓ heritability analyses with the R package BGLR.
- Analyses of humoral responses included only 'positive' hens, i.e. only those with a titre above the threshold indicated by the kit.

Trend of Vaccine Response

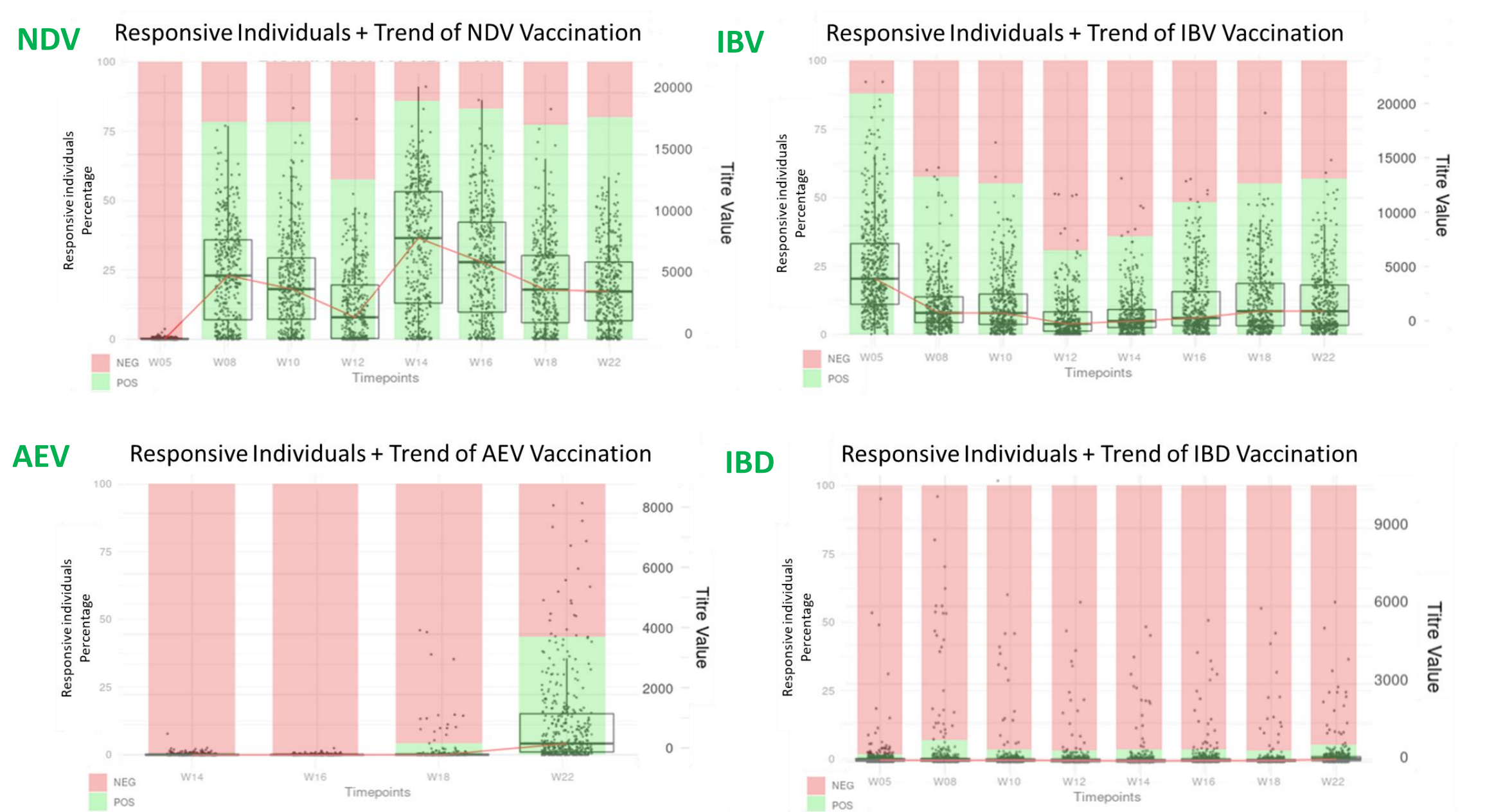


Figure 2. Vaccination trend and percentage of responders and non-responders to the four vaccines: For each vaccine, the following is represented: by using boxplots, the vaccine trend during the experiments; by using barplots, the 'positive' hens, i.e. only those with a titre above the threshold indicated by the kit (green colour) and the 'negative' hens, i.e. only those with a titre below the threshold indicated by the kit (red colour) are represented.

Vaccine response trends (**Figure 2.**) over time for the four vaccination: for IBD and AEV a very poor response has been seen, whereas for IBV and NDV there is a greater response, especially for NDV. Thus for the following analyses the IBD will not be taken into account.

Results

GWAS and Heritability

	W5	W8	W10	W12	W14	W16	W18	W22
NDV - h ²		0.23 ± 0.06	0.27 ± 0.07	0.39 ± 0.10	0.19 ± 0.04	0.32 ± 0.08	0.26 ± 0.06	0.21 ± 0.05
AEV - h ²								0.35 ± 0.09
IBV - h ²	0.19 ± 0.04	0.25 ± 0.06	0.29 ± 0.08	0.33 ± 0.10	0.29 ± 0.08	0.34 ± 0.09	0.30 ± 0.08	0.34 ± 0.09

Table 1. Heritability analysis: At each time point (columns) for the three vaccines (rows) heritability was calculated, with a particular look at IBV at week 18 (in bold).

The heritability range measured for the three vaccines at the different time points was 0.19-0.39 (**Table 1.**), implying a low-to intermediate heritability. GWAS analysis (**Figure 3.**) revealed a genomic region associated with the humoral response of the IBV vaccine at week 18.

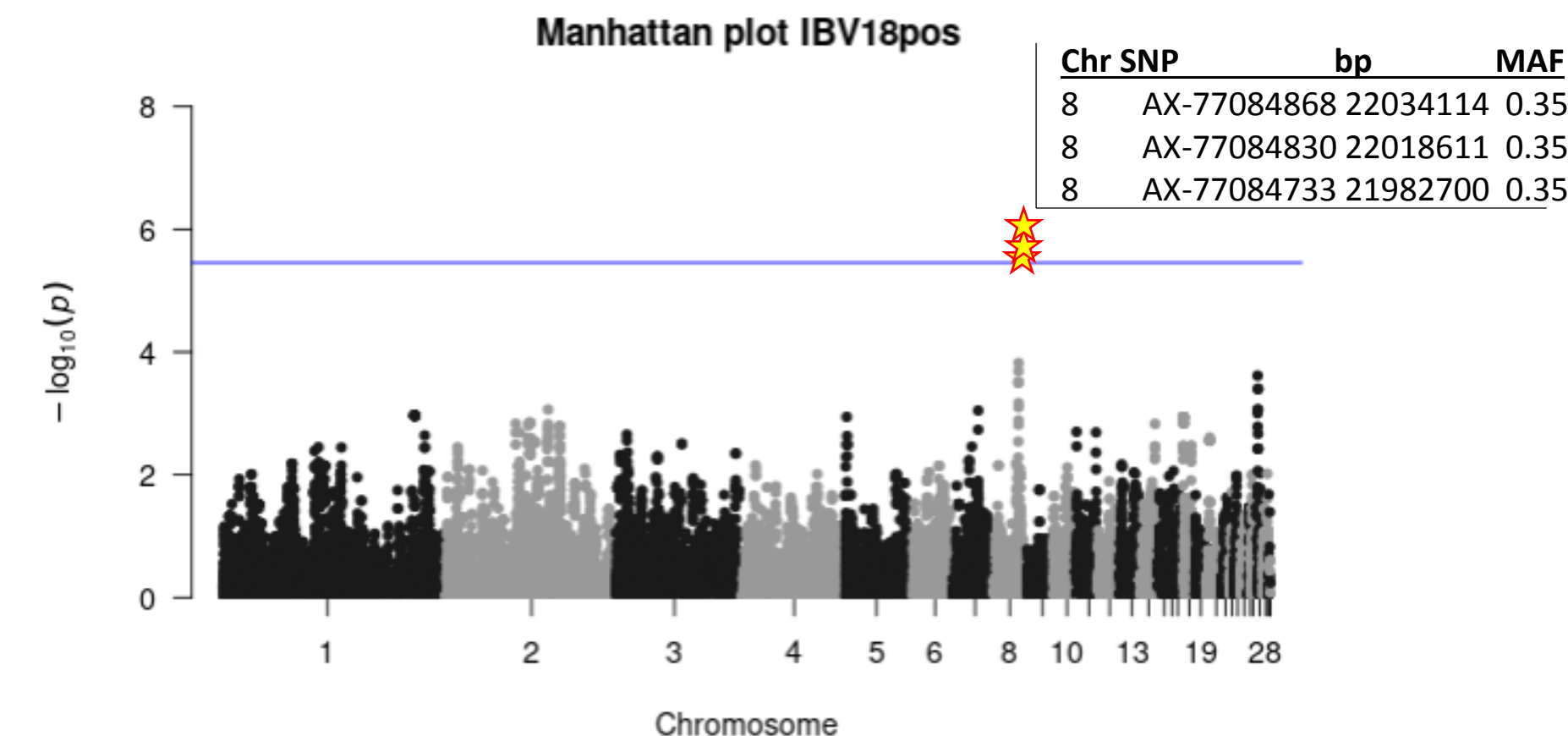


Figure 3. Manhattan plot of IBV vaccine at week 18: The blue line represents the threshold calculated by the False Discovery Rate (FDR) correction (3.49593e-06), the stars highlight the three identified significant single nucleotide polymorphisms (SNPs) and in the panel the three SNPs are made explicit by reporting the chromosome (Chr), the SNP name, the position in terms of base pair (bp) and the Minor Allele Frequencies (MAF).

Conclusions

The heritability analysis (**Table 1.**) showed a low- to intermediate heritability, thus confirming previous results obtained on NDV (Pinard van der Laan *et al.*) and broadening it to AEV and IBV. Only one QTL region was highlighted after GWAS analyses, which might be due to an insufficient population size for detecting many genes with weak individual effects (polygenic determinism; **Figure 2.**). In the second part of this project, the association between the microbiota and the vaccine response will be analyzed, which could explain a larger part of the variability found.

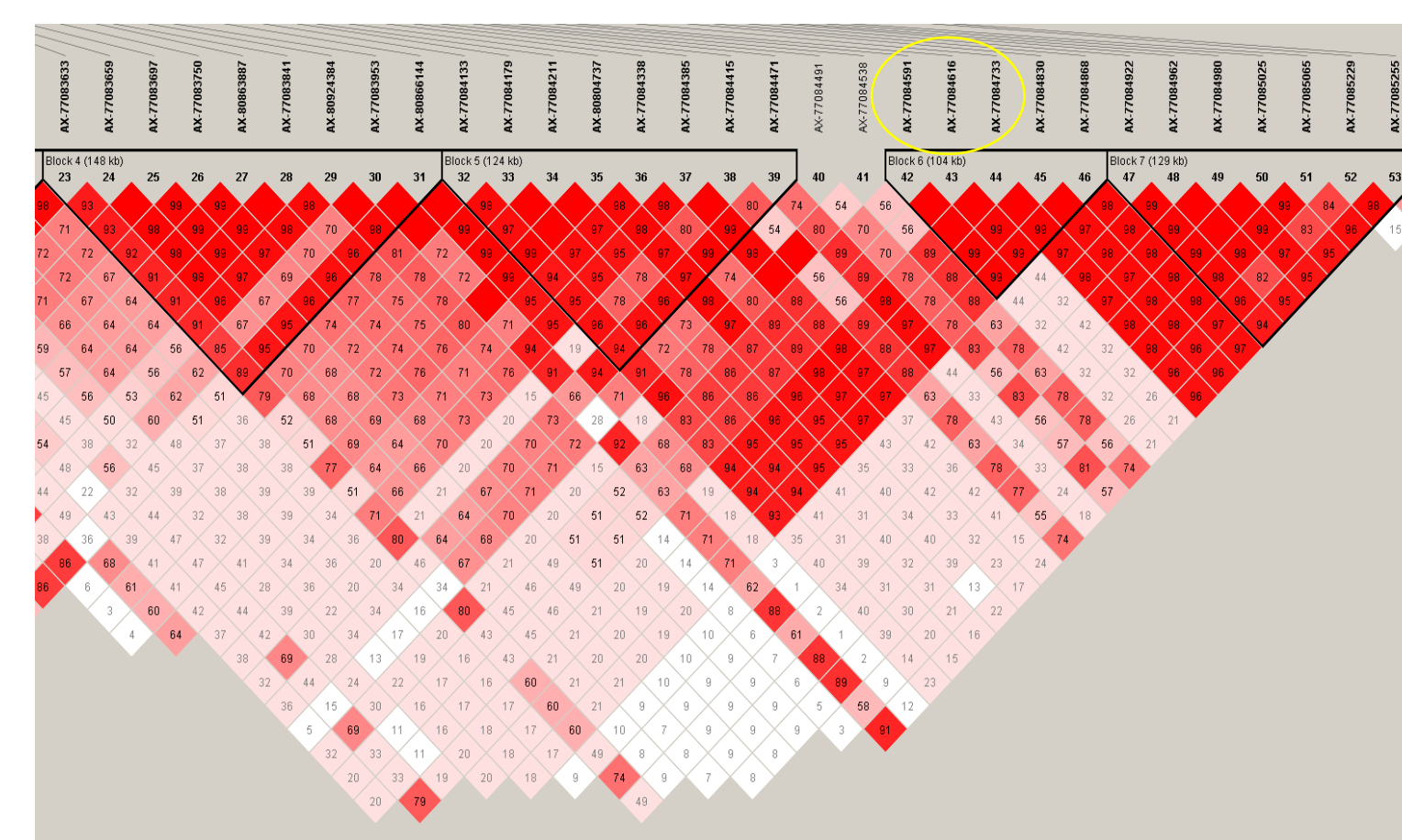


Figure 4. Linkage Disequilibrium (LD) visualization via Haploview: the three SNPs are highlighted by the yellow circle.

A Linkage Disequilibrium (LD) analysis (**Figure 4.**) was performed to define the quantitative trait locus (QTL) taking $R > 0.1$ into account. A VEP analysis was then performed to investigate potential genes underlying this area of interest. The genes identified are: long non coding RNA (lncRNA), CYP4B7, PDZK1IP1, CMPK1, FOXD2 and TRABD2B (where the SNPs identified are located).