

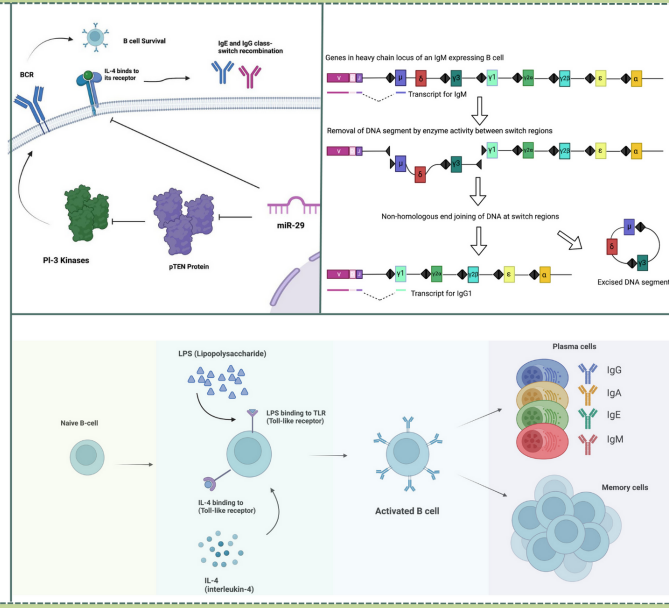
# The effect of different factors on the production of IgE and IgG by plasma cells

## INTRODUCTION

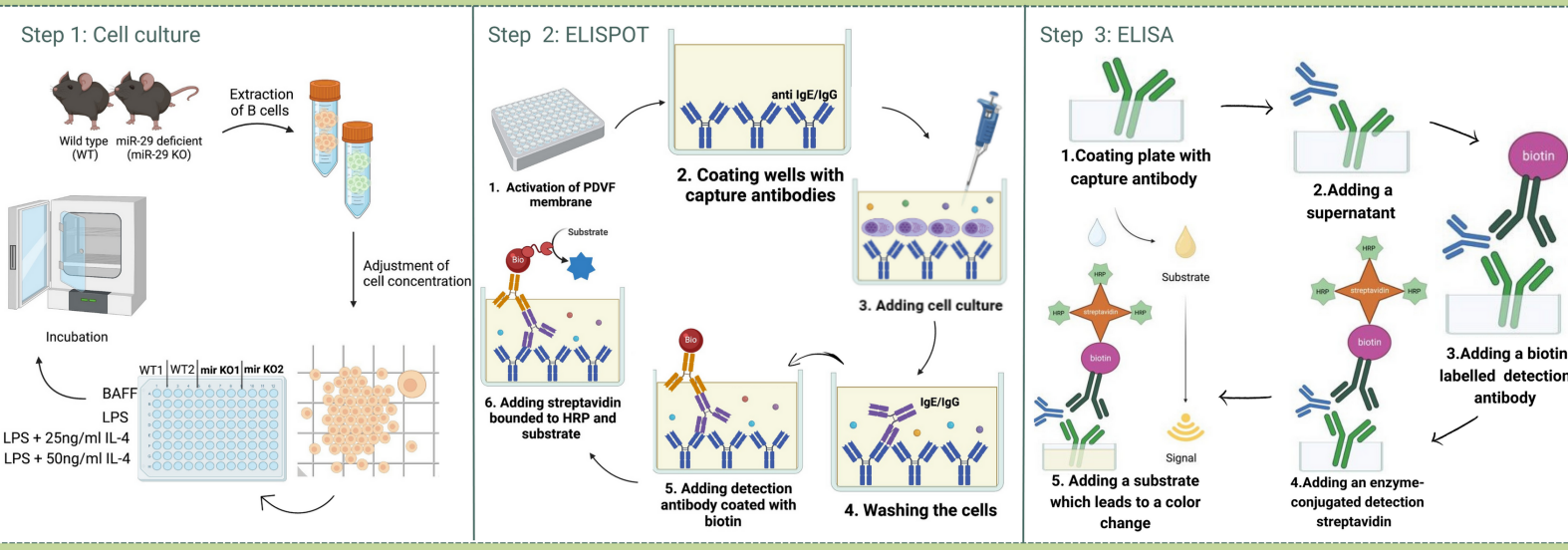
B lymphocytes play an essential role in the body's immune defense. They produce antibodies that recognize and neutralize specific pathogens. LPS (lipopolysaccharide) is the main structural component of the cell wall of bacterial pathogens. Recognition of LPS by B cells triggers the differentiation and proliferation of these lymphocytes into plasma cells that secrete antibodies. Immunoglobulin E (IgE) mediates pathogenic reactions seen in various allergic responses such as allergic asthma and food allergies. The key function of immunoglobulin G (IgG) is pathogen neutralization via target binding and halting of infection spread. Through class-switch recombination in B cells, the conversion of IgM to IgG and IgE takes place, leading to the production of different types of antibodies with different functions.

MicroRNAs are short noncoding RNAs that can regulate a variety of mRNAs' by negatively influencing their stability and/or translation. In B cells, micro-RNA miR29 was observed to inhibit the translation of the PTEN protein. This PTEN protein is essential for downregulating the PI3 kinase, a key signaling component of the B cell antigen receptor, thereby playing a central role in regulating B cells' survival and facilitating class switch recombination. Furthermore, the target scan database has shown that miR-29 has a predicted binding site of the IL-4 receptor transcript to negatively regulate the expression of the IL-4 receptor. The cytokine IL-4 has been shown to highly promote class-switching of antibodies to IgE. Mice that are miR-29 deficient usually have rashes and allergic reactions and their life span is shorter.

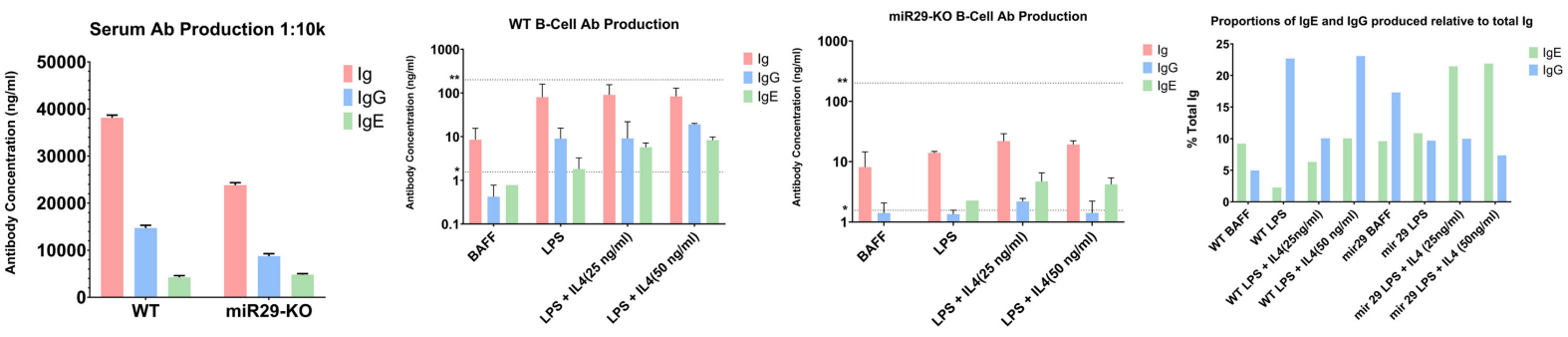
In our study, we investigated the influence of LPS and IL-4 on class switch recombination to IgE and IgG in B cells from wild-type mice and miR-29 knock-out (KO) mice.  
**Hypothesis:** Because we hypothesize that miR-29 negatively regulates the IL-4 receptor expression, we suspect that B cells with a miR-29 KO will have a higher class-switch to IgE in the presence of high concentrations of IL-4.



## METHODS



## RESULTS



## CONCLUSIONS

- In conclusion, the data analysis of ELISA demonstrates that the knockout of the miR29 gene results in a significant decrease in antibody production.
- T-cell dependent stimulation (with anti-CD40) of class-switching (see Poster 2.) is more efficient than stimulation by LPS.
- Additionally, IL-4 stimulation promotes IgG clas-switching in WT-B cells, whereas it promotes class-switching to IgE in miR-29 KO B cells, thus our hypothesis has been proved.

### Future Directions

Understanding the precise molecular mechanisms underlying miR29-mediated regulation may provide valuable insights into immune modulation and potential therapeutic strategies for allergic disorders, e.g. atopic dermatitis.