



HAL
open science

Porcine respiratory cell and tissue coinfections and superinfections with porcine reproductive and respiratory syndrome and swine influenza viruses

George Saade, Caroline Hervet, Déborah Ménard, Patricia Renson, Juliette Bougon, Céline Deblanc, Gaëlle Simon, Olivier Bourry, Nicolas Bertho, François Meurens

► To cite this version:

George Saade, Caroline Hervet, Déborah Ménard, Patricia Renson, Juliette Bougon, et al.. Porcine respiratory cell and tissue coinfections and superinfections with porcine reproductive and respiratory syndrome and swine influenza viruses. *Viruses* 2020 - Novel Concepts in Virology, Feb 2020, Barcelone, Spain. , 2020. hal-04557502

HAL Id: hal-04557502

<https://hal.inrae.fr/hal-04557502v1>

Submitted on 6 Jun 2024

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.

G. Saade¹, C. Herve¹, D. Ménard¹, P. Renson², J. Bougon², C. Deblanc², G. Simon², O. Bourry², N. Bertho¹, F. Meurens¹

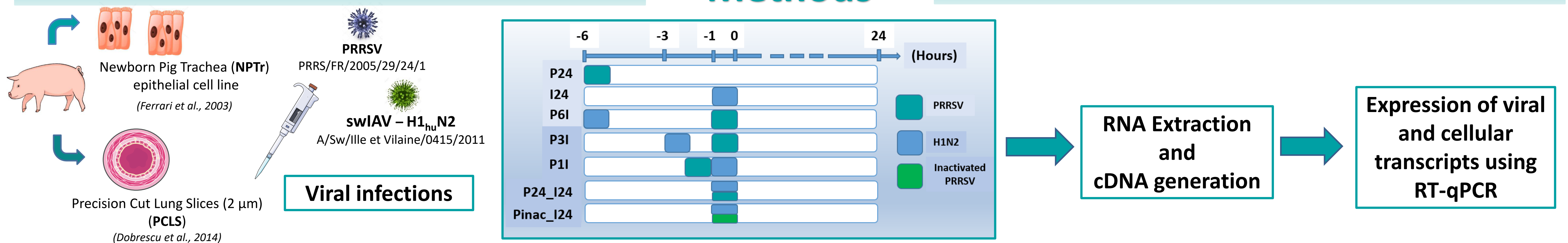
¹INRAE, Oniris, BIOEPAR, 44300, Nantes, France

²Unité de Virologie Immunologie Porcines ANSES, Laboratoire de Ploufragan-Plouzané-Niort, Zoopôle Les Croix BP 53, 22440 Ploufragan, France

Introduction

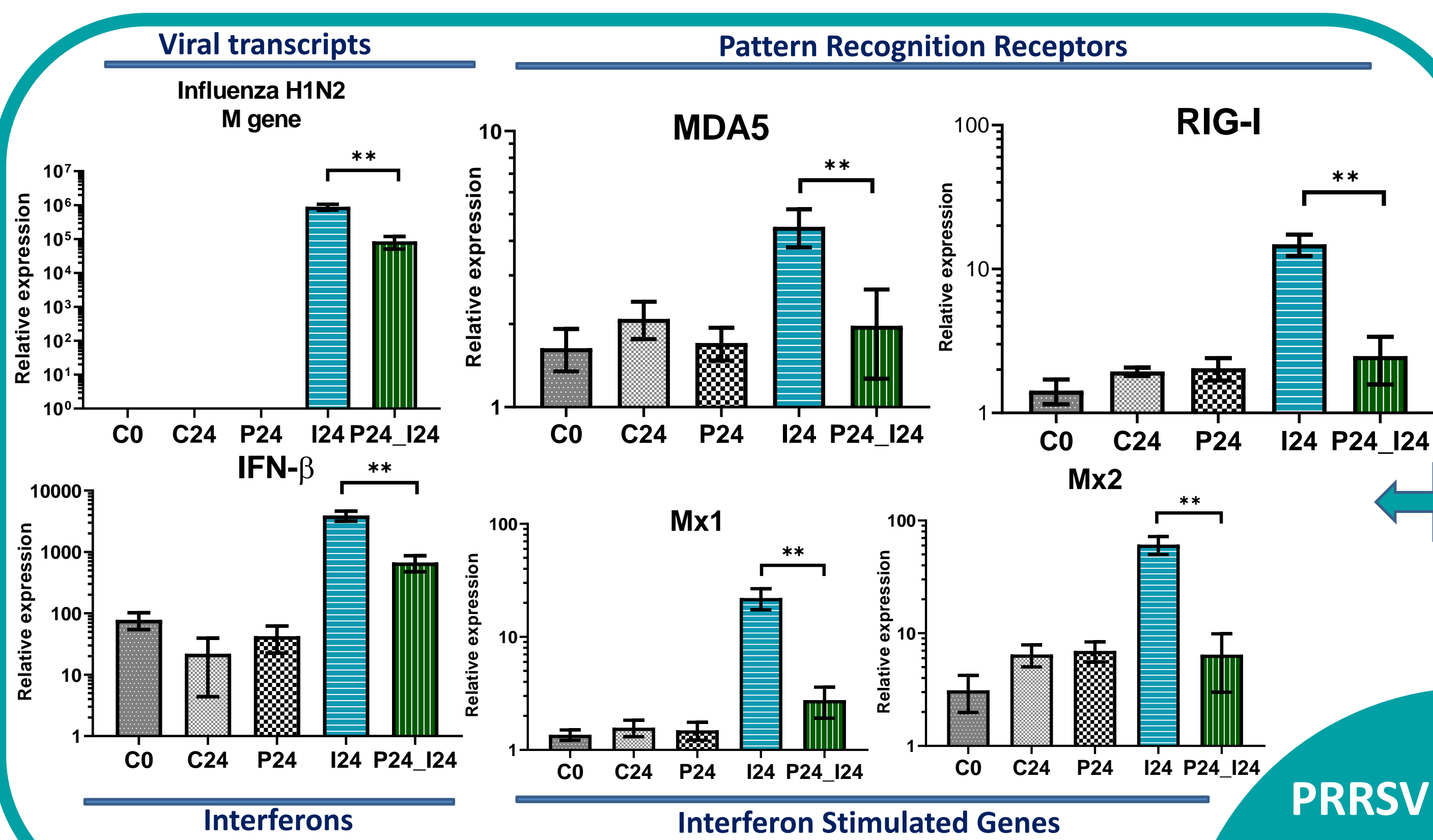
Porcine reproductive and respiratory syndrome virus (PRRSV) and swine influenza type A virus (swIAV) are major contributors to the porcine respiratory disease complex that is still threatening porcine farming around the world. Understanding the interactions between these two viruses and the effect of their coinfections on the porcine immune response will lead to better preventive and therapeutic measures. *In vitro* evaluation of these interactions is still very limited since the two viruses do not share the same host cells. swIAV infects mainly epithelial cells, while PRRSV infects only cells expressing CD163 such as alveolar macrophages. In this study we tried to evaluate the effect of PRRSV infection on the replication of swIAV, the host antiviral response and the possible induction of trained immunity in trachea epithelial cells and precision cut lung slices, using activated and inactivated PRRSV and comparing different intervals between the infections.

Methods

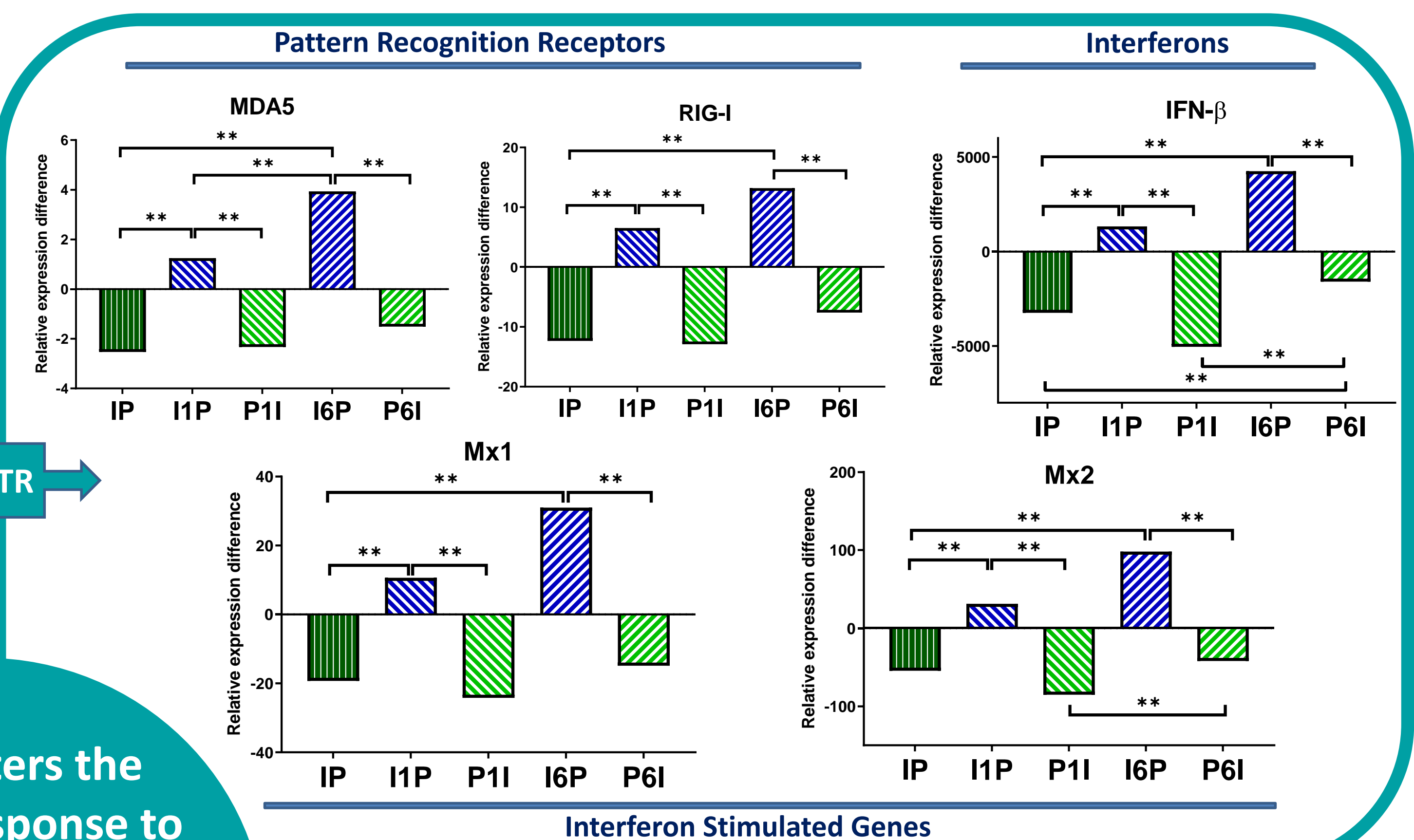


Results

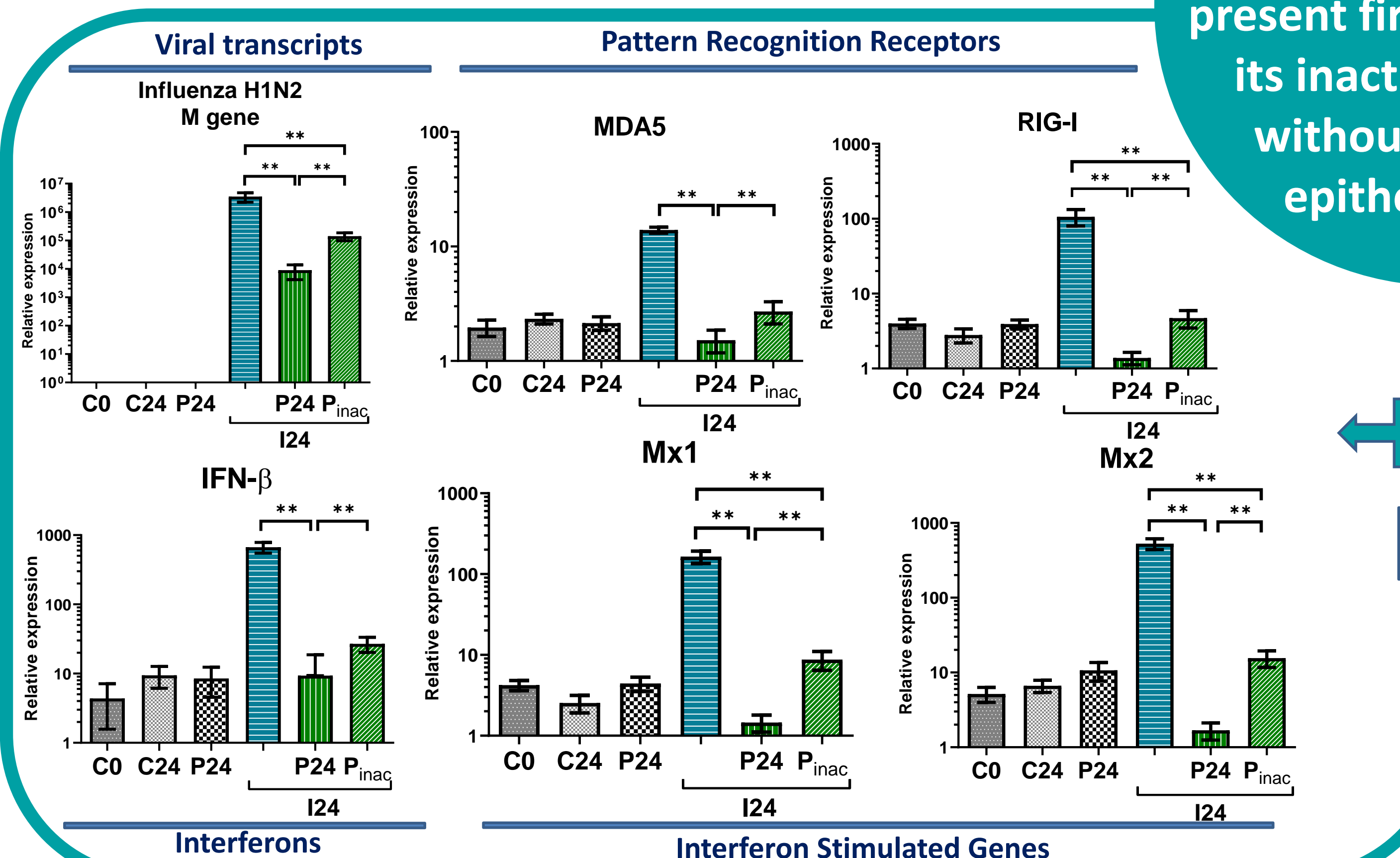
Impact of PRRSV on response to Influenza virus



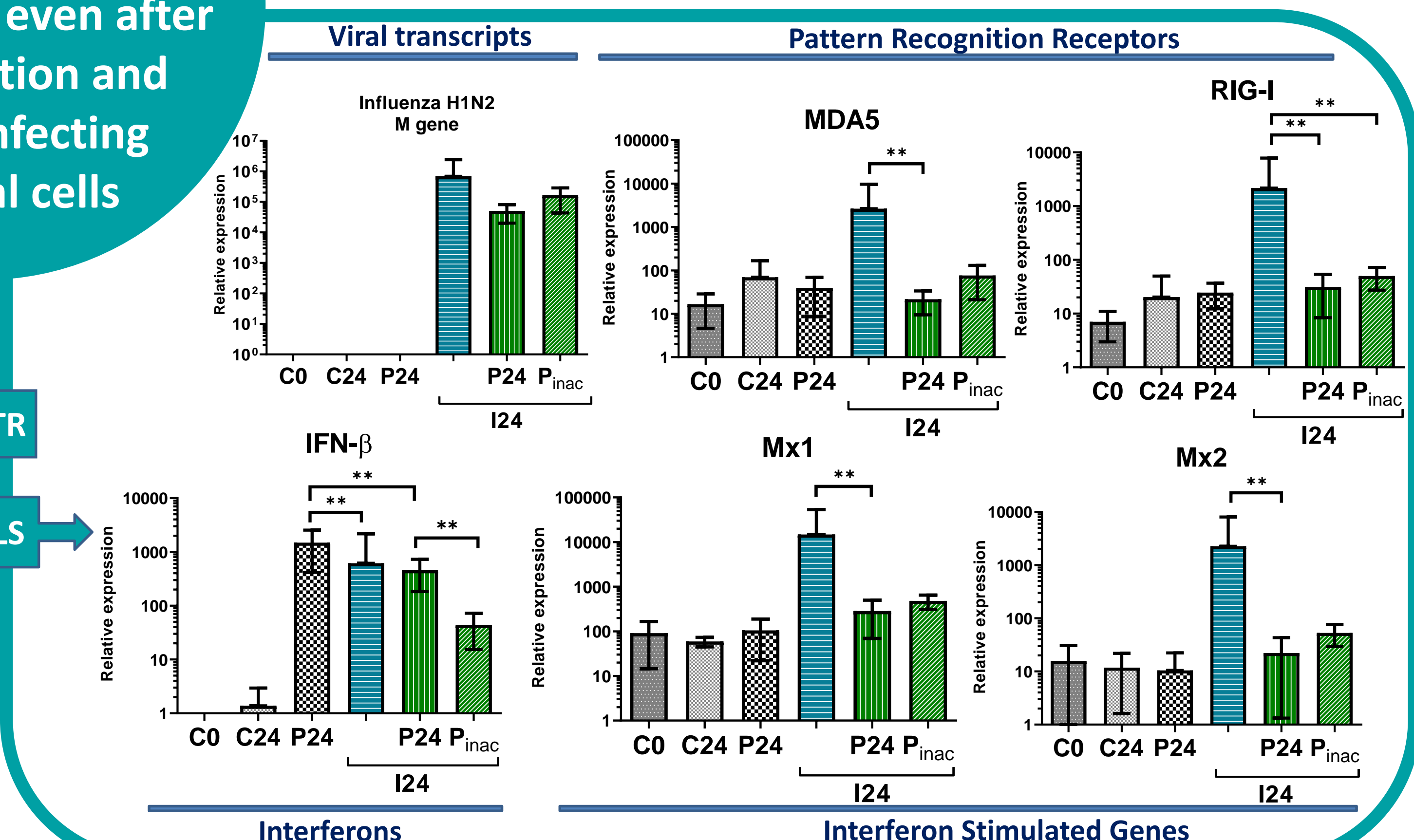
Impact of infection order and delays between infections



Impact of inactivated PRRSV (NPT)



Impact of inactivated PRRSV (PCLS)



PRRSV alters the antiviral response to swIAV infection only if present first, even after its inactivation and without infecting epithelial cells

C0: Contol 0h, C24: Control 24h, P24: PRRSV 24h, I24: Influenza 24h, P24_I24: PRRSV_Influenza, Pinac: Inactivated PRRSV 24h, IP: PRRSV_Influenza, I1P: linfluenza_1h_PRRSV, P1I: PRRSV_1h_Influenza, I6P: Influenza_6h_PRRSV, P6I: PRRSV_6h_Influenza. MOI: 3, n = 6, Mean values, Standard deviations, **: P < 0,01 * : P < 0,05. Data were sorted by rank, then One-Way ANOVA was used. Means of the ranks among the groups were compared using Tukey's test.

Conclusion

PRRSV has no tropism to epithelial cells, however, our results showed that the virus was able to interfere with swIAV infection by affecting swIAV replication and altering the response of the hosting cell. Inactivation of the virus reduced its inhibiting effect without obliterating it completely, which means that the presence of the virus is sufficient to alter the capacity of the cells to replicate swIAV. PRRSV impact tends to decrease when the delay between the viruses increases and is completely reversed when the PRRSV is inoculated after swIAV. The inhibition effect of the active virus was also confirmed on PCLS combining different cell types, especially alveolar macrophages, the target of PRRSV, and epithelial cells, the target of swIAV. PRRSV again clearly affects the expression of ISGs however, its effect on swIAV replication is less clear. The mechanisms of this viral interference are still unclear and will be investigated later to ultimately apply better preventive and therapeutic approaches.

References

- Dobrescu I, Levast B, Lai K, et al (2014) In vitro and ex vivo analyses of co-infections with swine influenza and porcine reproductive and respiratory syndrome viruses. *Vet Microbiol* 169:18–32.
- Ferrari, M., A. Scalvini, M. N. Losio, A. Corradi, M. Soncini, E. Bignotti, E. Milanese, P. Ajmone-Marsan, S. Barlati, and D. Bellotti. 2003. "Establishment and Characterization of Two New Pig Cell Lines for Use in Virological Diagnostic Laboratories." *Journal of Virological Methods* 107 (2): 205–12.