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Diagnóstico de la patología respiratoria

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Importancia de las enfermedades respiratorias

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A flexible tool for the assessment of the economic cost of pig disease in growers and finishers at farm level

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Keywords:

Pig
Respiratory disease
Systems dynamics model
Financial performance
Marketing contracts

Pigmeat is the most consumed red meat globally and consumption is expected to continue to increase. The sector is faced by the risk of epidemic and endemic disease impacts and other adverse influences. The aim of this study was to develop a dynamic simulation model of pig growing and finishing that can be used to model the financial and economic impacts of a variety of scenarios both related to disease effects and other influences on production. The model consists of a physical performance module and financial performance module. The core of the physical performance module comprises three stocks to model the flow of pigs from purchase to slaughter. Mortality rates, daily live weight gain and feed conversion ratios influence the dynamics of the physical performance. Since contracts between farmers and slaughterhouses often include large price penalties for over- and underweight pigs, carcass weight distribution is an important determinant of revenues. The physical performance module, therefore, simulates slaughter weight variations. The financial performance module calculates revenue, costs and gross margins. The revenue calculations take into account price penalties for over- and underweight pigs. To demonstrate the capabilities of the model, we apply the model to assess the economic consequences of production impacts associated with respiratory disease. We use estimated production impacts associated with respiratory disease from a study of all-in-all out growing and finishing systems based on pig production data and information from slaughterhouse monitoring in the UK. Our model suggests a reduction in the gross margin of nearly 40 % as a consequence of the estimated production impacts associated with a 10% increase in respiratory disease prevalence. Due to the lack of reliable information on slaughter weight variation, we also simulate the model using different assumptions about the slaughter weight distribution. An increase in the standard deviation of carcass weights from 8 kg to 12 kg, holding average weights constant, more than halves gross margins under our scenarios. We suggest that for all-in-all-out systems, carcass weight variation is likely to be a substantial factor in reducing income in the presence of respiratory disease and the economic impact of respiratory disease may be underestimated if the effects of disease on variation in carcass weights are not included in any analysis.

Importancia económica de las enfermedades respiratorias

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Distribución de pesos en canal (aumento canales de bajo peso)

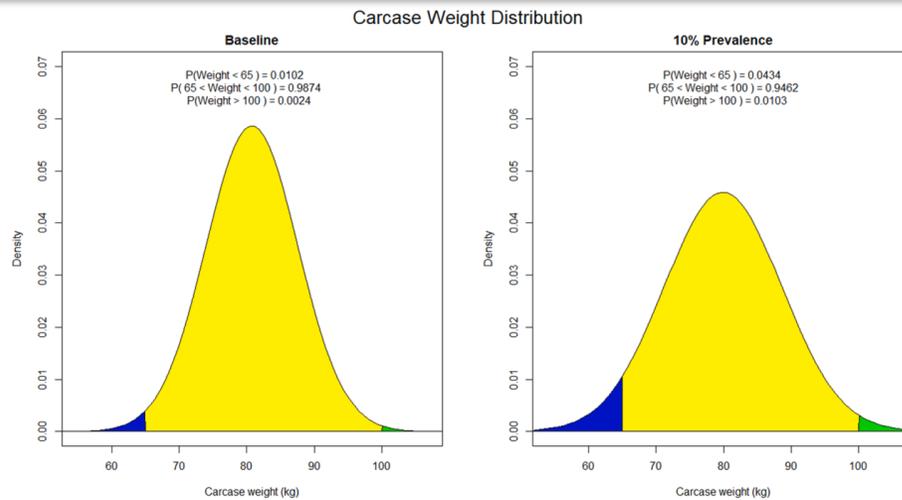


Fig. 3. Density function for carcass weight distribution in baseline and +10% prevalence scenario. Note: The area in blue (left tail) represents the probability that the carcass weight falls below the underweight threshold, the area in yellow represents the probability that the carcass weight is within specification and the area in green (right tail) represents the probability that the carcass weight falls above the overweight threshold.

Origen de las pérdidas

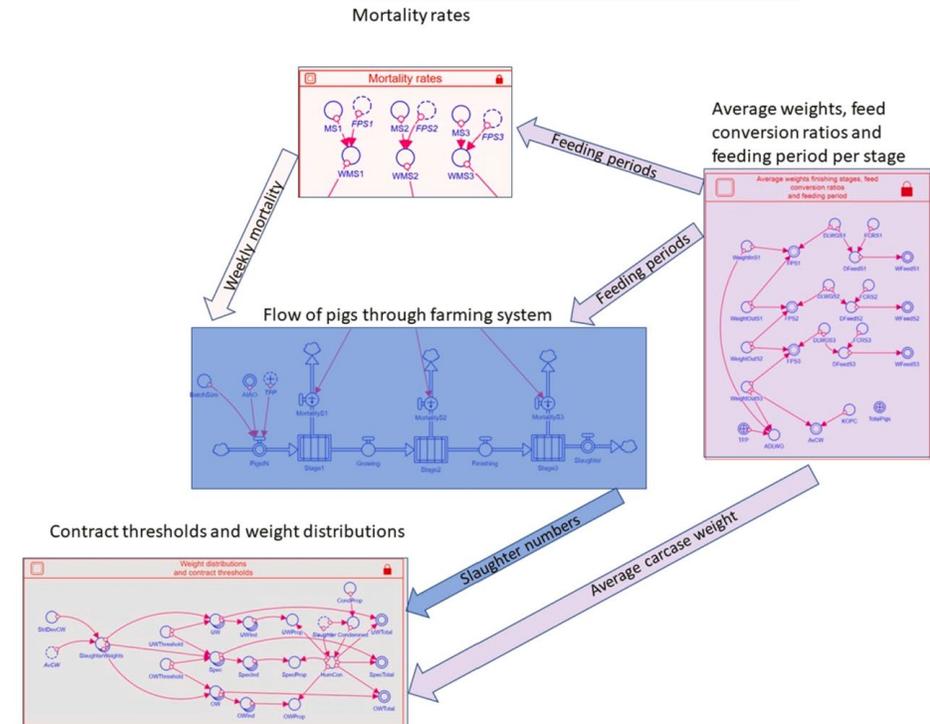


Fig. 1. Model diagram of the sectors of the physical performance module.

Importancia económica de las enfermedades respiratorias

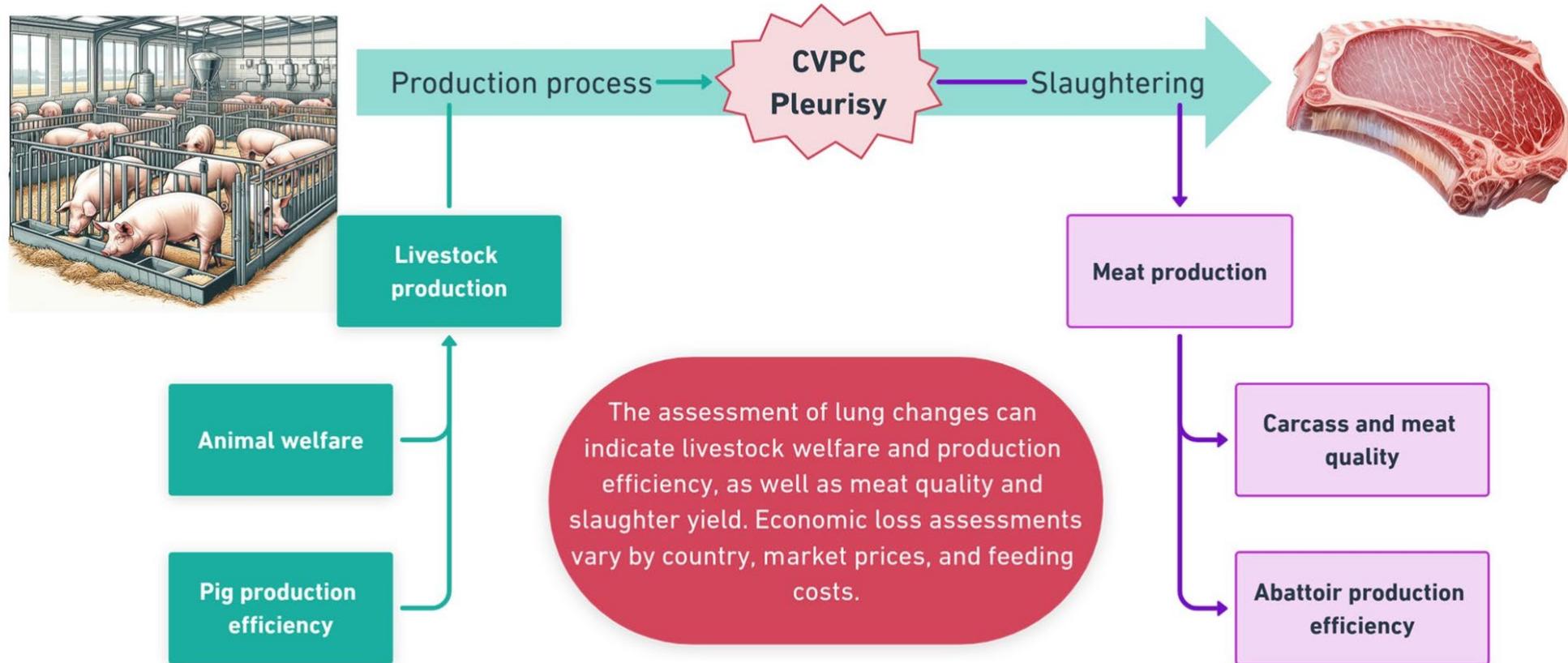


Figure 1 Impact of CVPC (cranio-ventral pulmonary consolidation) and pleurisy on abattoir productivity and economic outcomes

Importancia económica de las enfermedades respiratorias

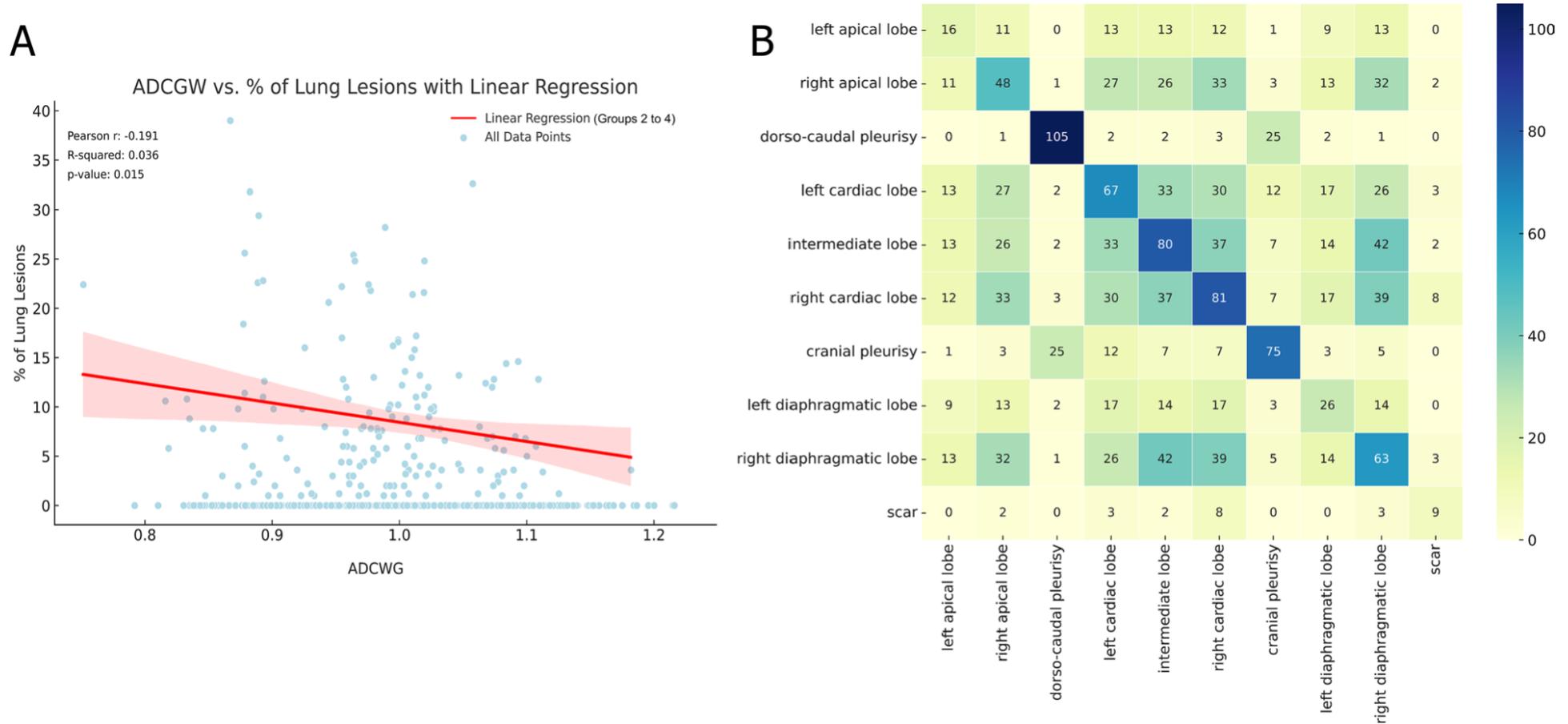


Figure 2 Relationship Between Average Daily Carcass Weight Gain (ADCGW) and Lung Lesions in slaughter-age pigs from 7 different batches. **(A)** The scatter plot shows the percentage of lung lesions against ADCGW with a linear regression line. **(B)** The co-occurrence matrix illustrates the frequency of the different types of lung lesions present together in various lobes and regions of the lungs, with a color gradient representing the count of co-occurrences

Importancia económica de las enfermedades respiratorias

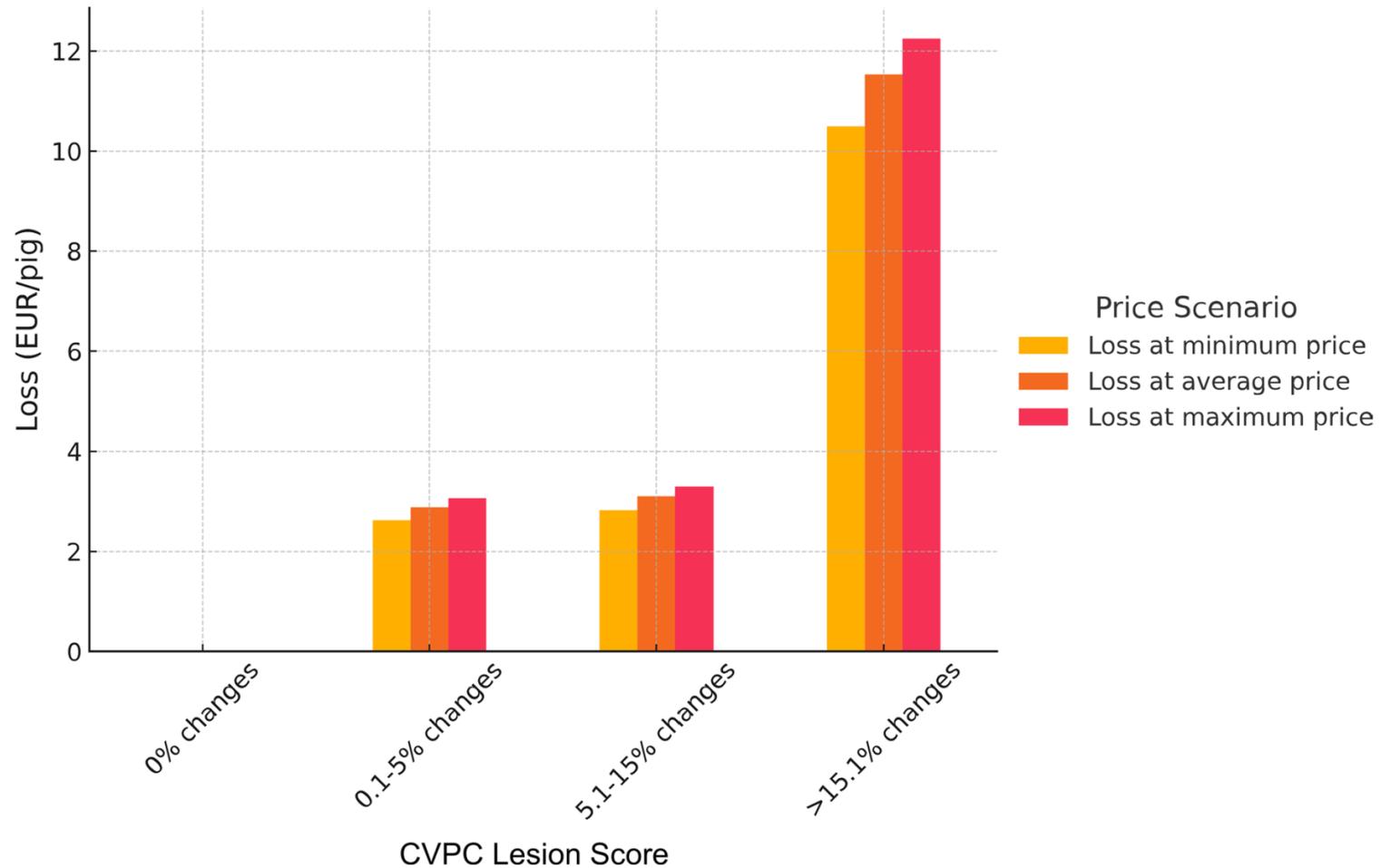


Figure 4 Economic losses depending on the degree of cranio-ventral pulmonary consolidation (CVPC) and price scenarios. The bar chart illustrates the economic losses per affected pig (in EUR) associated with varying degrees of CVPC scores under three price scenarios: minimum (201.86 EUR/100kg), average (221.77 EUR/100kg), and maximum (235.59 EUR/100kg). The x-axis categorizes the pigs into four groups based on the percentage of CVPC lung changes: 0% changes (reference), 0.1-5% changes, 5.1-15% changes, and ≥ 15.1% changes

Importancia económica de las enfermedades respiratorias

Efecto de pleuritis en la rentabilidad económica y en el retorno de la inversión

Table 7. Economic data for pigs with pleurisy scores of 2, 3, and 4, focusing on slaughter weight, cost per kilogram, total revenue, economic profit, benefit–cost ratio (BCR), and return on investment (ROI).

Score of Pleurisy (N = 156)	Slaughter Weight (kg)	Cost Per Kg (USD)	Total Revenue	Economic Profit (USD)	BCR (USD)	ROI (%)
2 (n = 30)	111.243	1.29	763.129	7.79	0.210	5.33
3 (n = 70)	110.616	1.32	758.824	6.92	0.214	4.73
4 (n = 56)	109.734	1.32	752.775	5.70	0.207	3.90

Importancia económica de las enfermedades respiratorias

Impacto de la infección por *M. hyopneumoniae* en la GMD

Table 4 Studies assessing the impact of cranioventral pulmonary consolidation (CVPC) on average daily gain (ADG) in pigs.

References	Study population	Impact on ADG	Comment
Presence of CVPC			
[59]	23 studies	10 publications: no effect 13 publications: decrease	
[51]	9 studies	-17%	Feed conversion ratio +14% High correlation between decreased ADG and feed conversion ratio
[36]	2 farms (578 pigs)	Farm A: -26 g Farm B: -26 g	
[54]	14 farms (6,997 pigs)	-38 g (-4.9%)	Large differences between farms (range: -7% to +2.6%)
[69]	39 farms	No effect	No effect on feed conversion ratio Performance data were analysed at farm level; lung lesions were assessed on one subgroup (35 pigs per farm) at slaughter
[8]	1 farm (108 pigs)	-9%	
Severity of CVPC			
[51]	5 studies	37.4 g per 10% of affected lung tissue	Five studies
[52]	21 pigs	20% CVPC throughout lifetime: 25 kg lower weight at slaughter	Radiographic monitoring of the lungs from 21 to 180 days of age 25 days extra to reach slaughter weight
[7]	2 farms (41 lungs)	41.1 g/day for 10% affected lung volume	10% affected lung leads to 16.7 days more to reach 104.5 kg
[70]	2 farms (58 lungs)	31.4 g/day for 10% affected lung volume	10% affected lung leads to 13.2-days more to reach 104.2 kg Only significant association between lung lesion and ADG in one farm
[72]	1 farm (333 pigs)	2.2 g/day for each 1% of affected lung volume	1% affected lung leads to 0.61 days more to reach slaughter weight; early infections cause more performance loss
[50]	7 farms (18 cohorts with 30-35 pigs / cohort)	6.8 g/day for 1% of affected lung	Serologic testing was more effective than slaughter evaluation in assessing the impact of subclinical infection on ADG
[73]	1 farm (138 pigs)	Score 2: -45 g/day (-6%) Score 3: -90 g/day (-11%)	Lung consolidation: score 1: 0-5%; score 2: 6-40%; score 3: > 40% Score 2: 3.13 kg less at slaughter Score 2: 10.85 kg less at slaughter
[54]	14 farms (6,997 pigs)	-7.0 g/day (0.7%) for 1 point increase in severity score (1-28)	More ADG decrease in pigs with severe lesions: score 0: 775 g; scores 1-4: 750 g; scores 5-8: 735 g; score > 8: 695 g
[71]	1 farm (500 pigs)	1.8 g/day for each 1% of affected lung surface	Financial loss of \$6.55 in pigs with more than 15% affected lung tissue compared to animals without lesions
[8]	1 farm (108 pigs)	-66.4 g/day in pigs with > 10% of affected lung surface versus pigs with no lesions:	Days to slaughter: +8 days; carcass weight: -3.6 kg

Importancia económica de las enfermedades respiratorias

Beneficio económico/cerdo en granjas libres de *M. hyopneumoniae*

Table 6

Economic benefit of eliminating Mhyo from a 5000 breed to finish operation producing 135,870 weaned pigs and 125,000 finishers per year.

Parameter	Economic benefit per pig	Annual production system economic benefit
Improvement of 36 g of average daily gain (ADG)	\$4.76	\$594,070
Improvement of 18 g on wean-to-finish feed:gain	\$0.78	\$97,143
Reduction of 1.26% in wean-to-finish mortality	\$0.008	\$945
Savings with Mhyo vaccinations in weaned pigs	\$0.25	\$33,967
Savings with antibiotic medications in growing pigs	\$1.21	\$151,250
Total economic benefit	\$7.00	\$877,375

Importancia económica de las enfermedades respiratorias

Patógenos detectados y sintomatología asociada

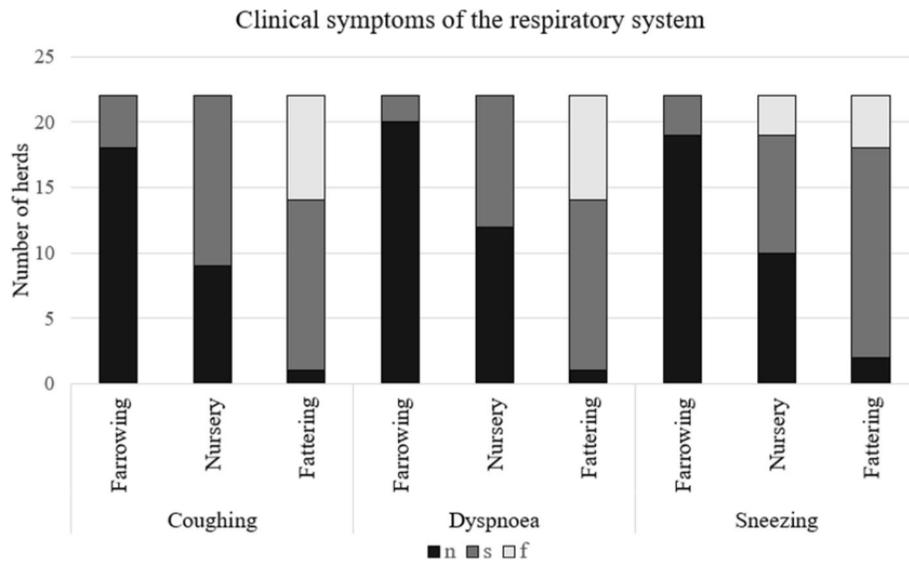


Fig. 2 The occurrence of coughing, dyspnoea or sneezing in the farrowing, nursery or fattening stages. Legend: n – not observed; s – sporadic; f – frequent

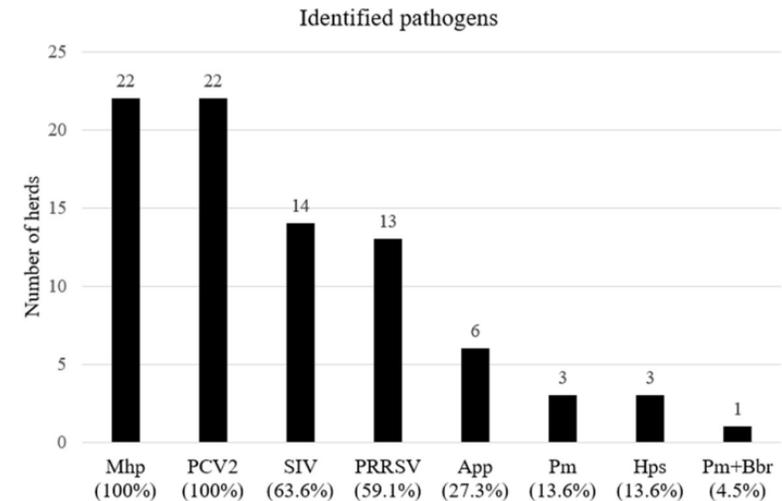


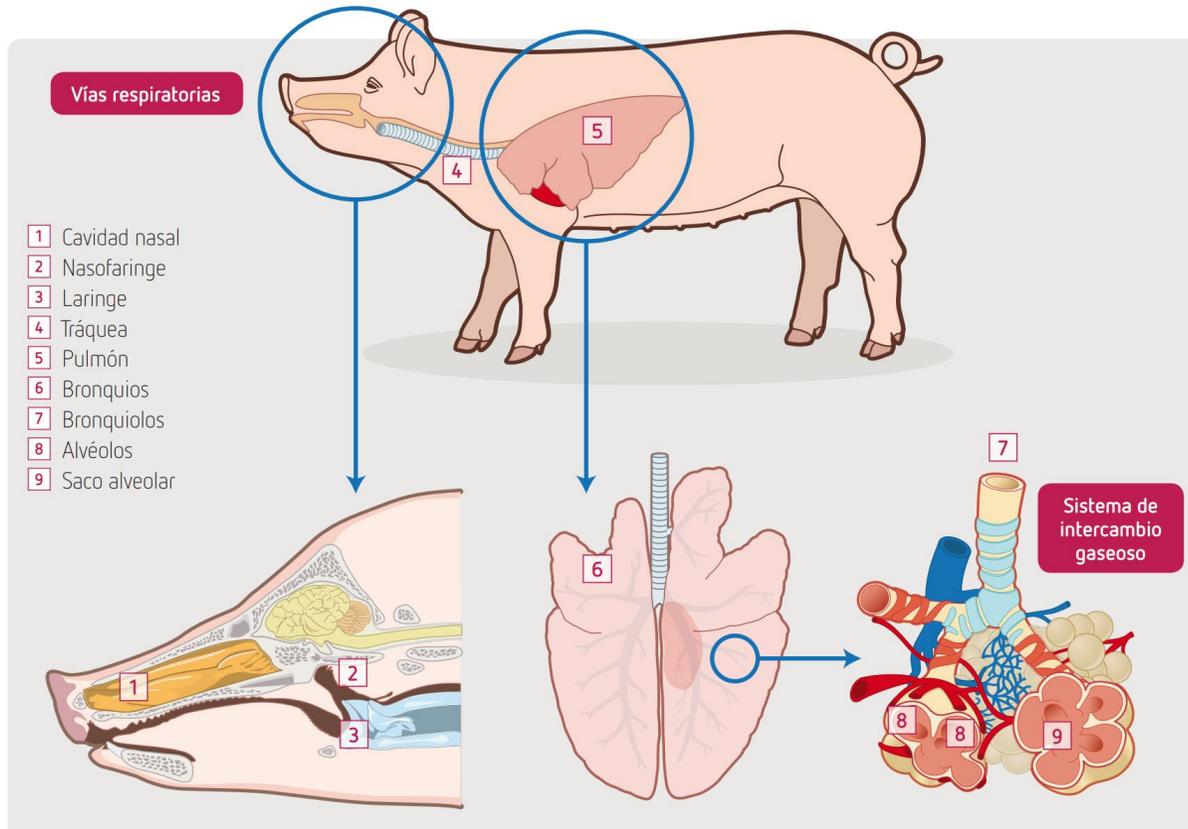
Fig. 1 Percentage of 22 pig herds with laboratory-detected respiratory pathogens (based on questionnaire responses). Legend: Mhp (Mycoplasma hyopneumoniae); PCV2 (Porcine circovirus type 2); SIV (Swine influenza virus); PRRSV (Porcine reproductive and respiratory syndrome virus); App (Actinobacillus pleuropneumoniae); Pm (Pasteurella multocida); Hps (Haemophilus parasuis); Pm + Bbr (Pasteurella multocida + Bordetella bronchiseptica)

***Estructura del aparato
respiratorio y
mecanismos de defensa***

Estructura del aparato respiratorio del cerdo

Configuración anatómica

Estructura del aparato respiratorio



Estructura de la barrera alveolocapilar

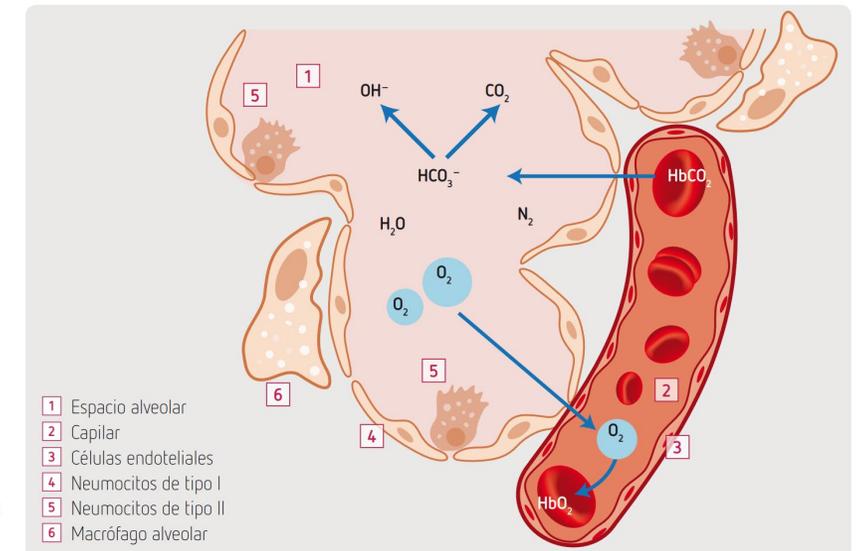


FIGURA 1. Estructura del sistema respiratorio del cerdo.

Estructura del aparato respiratorio del cerdo

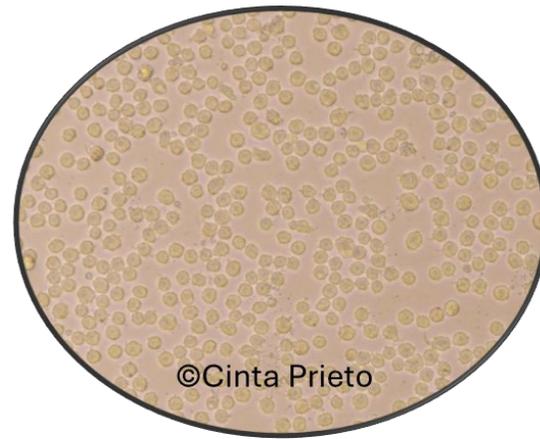
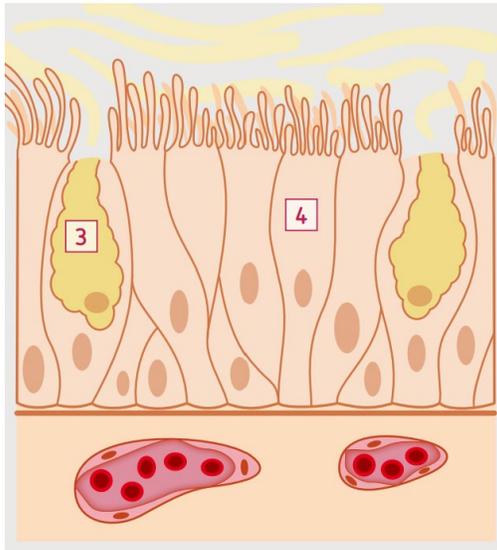
Mecanismos de defensa del aparato respiratorio

Macrófagos alveolares

Partículas menores de 5 μm que llegan a los alveolos

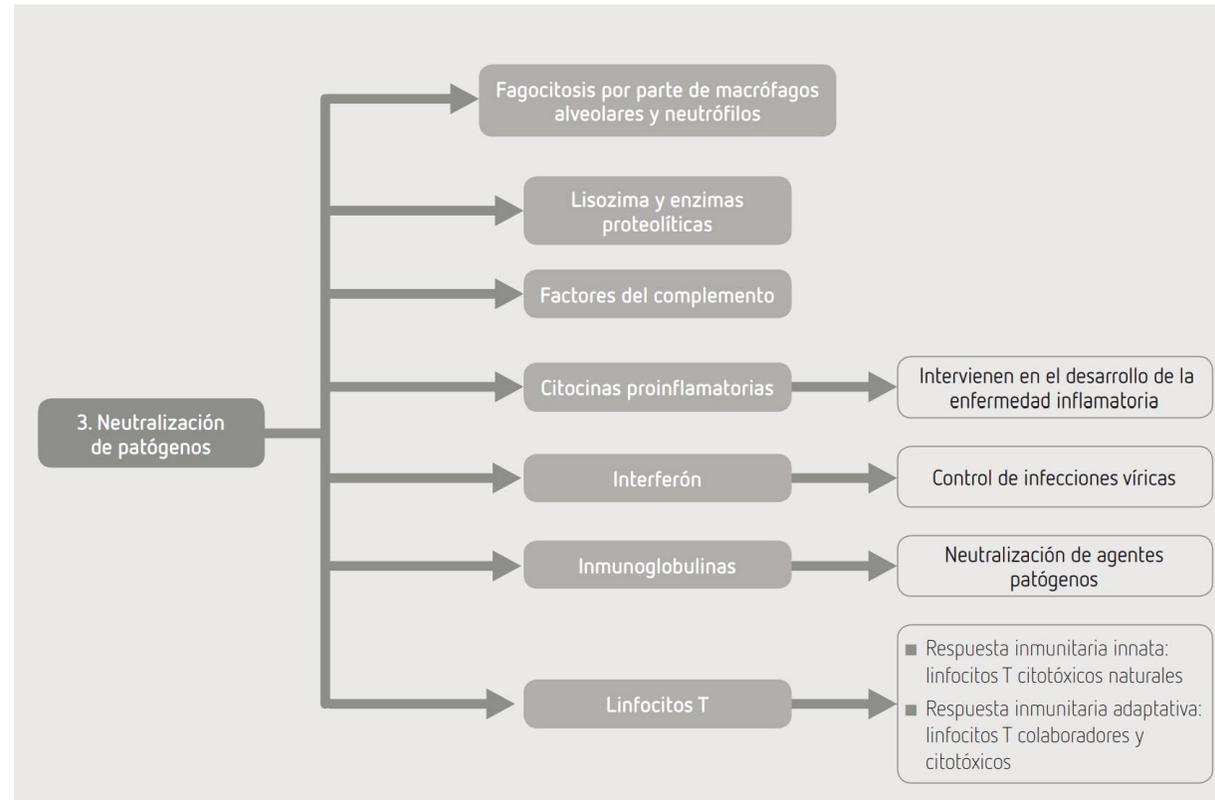
Aparato mucociliar

Partículas mayores de 5 μm



©Cinta Prieto

Otros mecanismos de defensa



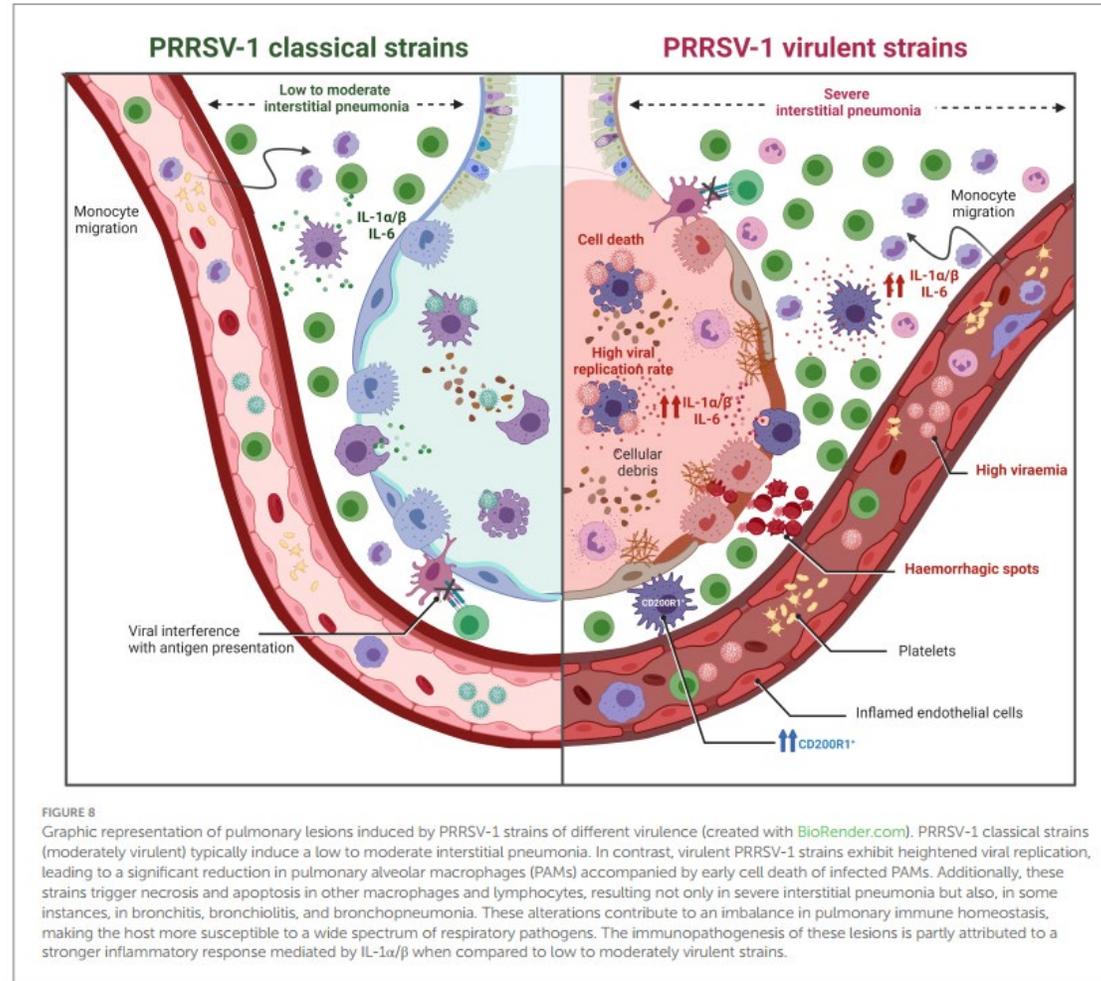
Estructura del aparato respiratorio del cerdo

Mecanismos de defensa del aparato respiratorio

Liberación de citocinas proinflamatorias



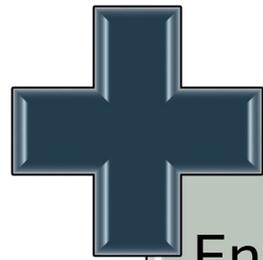
Infiltración de linfocitos y polimorfonucleares



***Patógenos respiratorios
y coinfecciones (CRP)***

Posibles causas de alteraciones en la homeostasis respiratoria

Tipos de patógenos respiratorios



Enfermedades
causadas por
patógenos
respiratorios
primarios

Complejo
respiratorio porcino
(patógenos
primarios,
patógenos
secundarios,
factores
ambientales)



Posibles causas de alteraciones en la homeostasis respiratoria

Patógenos respiratorios primarios y secundarios más importantes

Table 1. Primary and secondary pathogens in porcine respiratory disease complex (PRDC) [9].

Primary Pathogens	Secondary Pathogens
PRRSV * SIV * PRCV PCV2 * <i>Mycoplasma hyopneumoniae</i> <i>Actinobacillus pleuropneumoniae</i>	<i>Mycoplasma</i> spp. Streptococcus spp. Staphylococcus spp. <i>Escherichia coli</i> Klebsiella spp. <i>Trueperella pyogenes</i> <i>Bordetella bronchiseptica</i> <i>Glaesserella parasuis</i> <i>Pasteurella multocida</i>

* Association with swine proliferative and necrotizing pneumonia (PNP).

Complejo respiratorio porcino: resultado de las coinfecciones

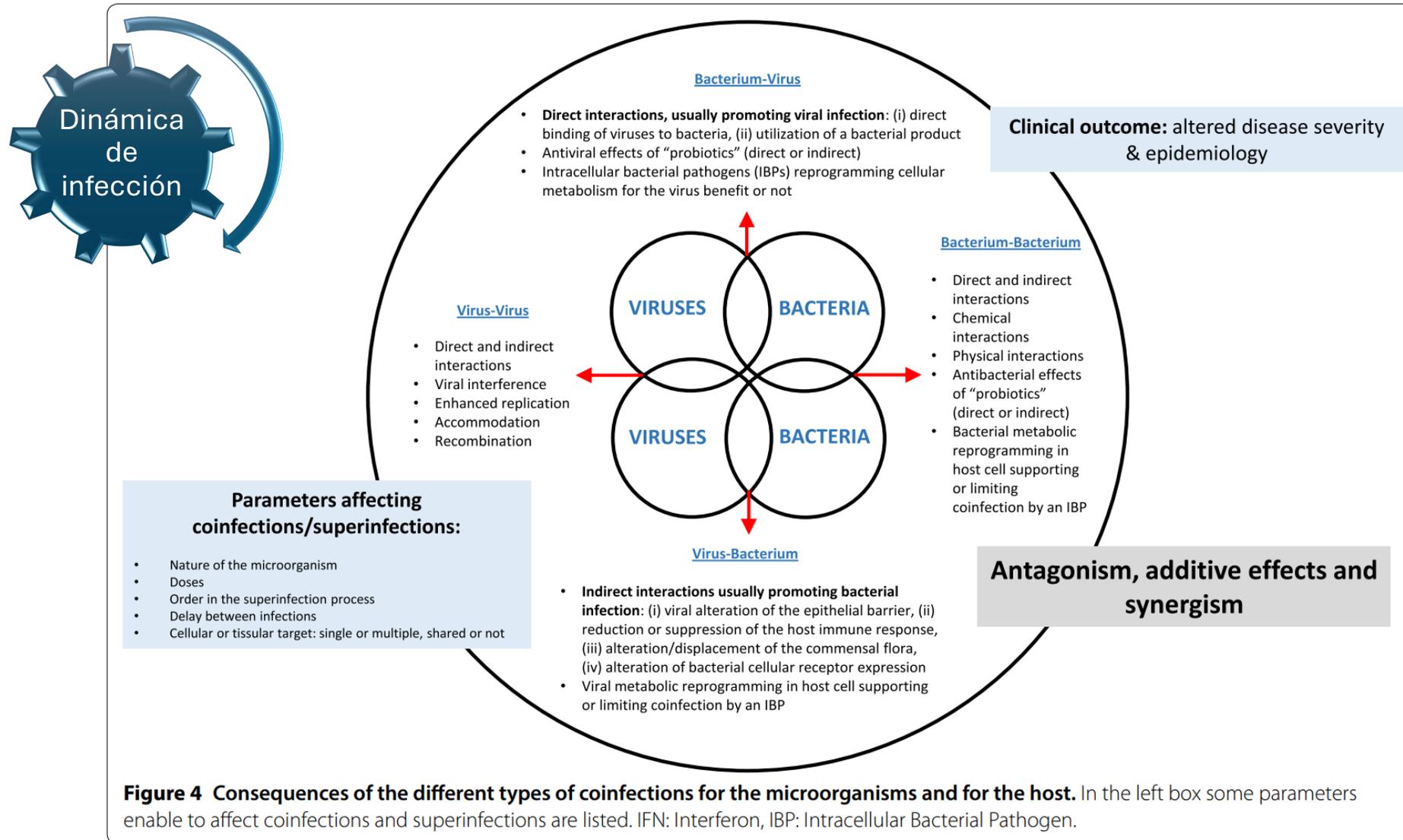


Figure 4 Consequences of the different types of coinfections for the microorganisms and for the host. In the left box some parameters enable to affect coinfections and superinfections are listed. IFN: Interferon, IBP: Intracellular Bacterial Pathogen.

Complejo respiratorio porcino: resultado de las coinfecciones



M. hyopneumoniae

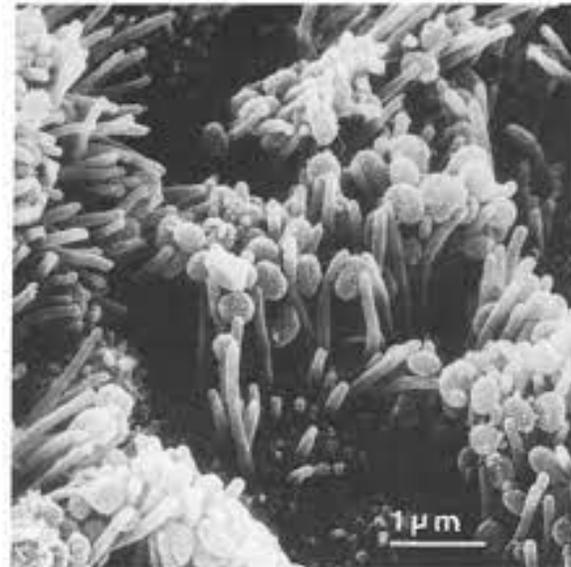
1

Adhesión al epitelio ciliado

Pérdida de funcionalidad

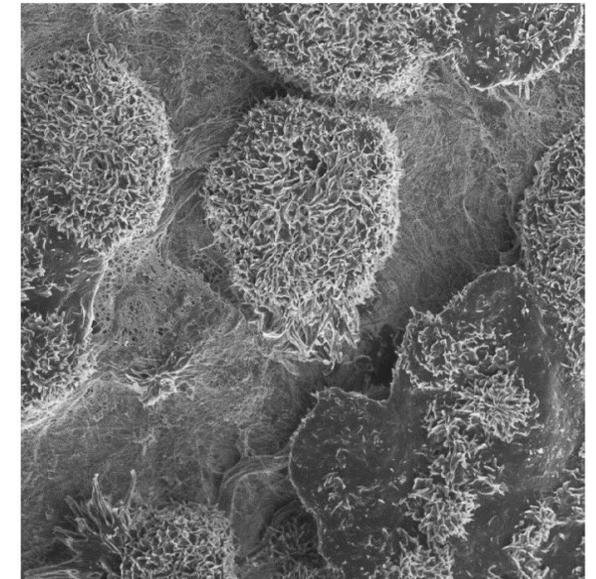
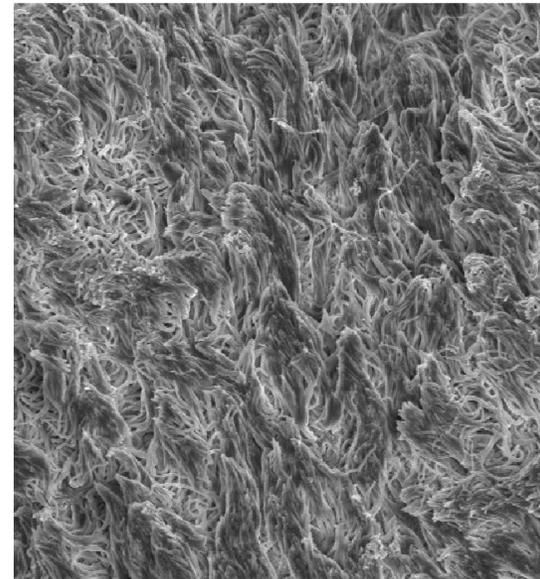
Colonización de vías respiratorias bajas por agentes secundarios

M. hyopneumoniae
B. Bronchiseptica
G. parasuis



Blanchard et al. Vet Microbiol, 1992, 30: 329-341,

Scanning electron microscope images of tracheal tissue from a vaccinated pig (left) and an unvaccinated pig (right) that demonstrates the extent of ciliary loss after infection with *Mycoplasma hyopneumoniae*: images courtesy of C. Jenkins



https://www.dpi.nsw.gov.au/__data/assets/pdf_file/0005/462056/Mycoplasmal-pneumonia-in-pigs.pdf

Complejo respiratorio porcino: resultado de las coinfecciones



G. parasuis

1

Adhesión al epitelio ciliado

Pérdida de funcionalidad

Colonización de vías respiratorias bajas por agentes secundarios

M. hyopneumoniae
B. Bronchiseptica
G. parasuis

Mucosa nasal 4 horas p.i.

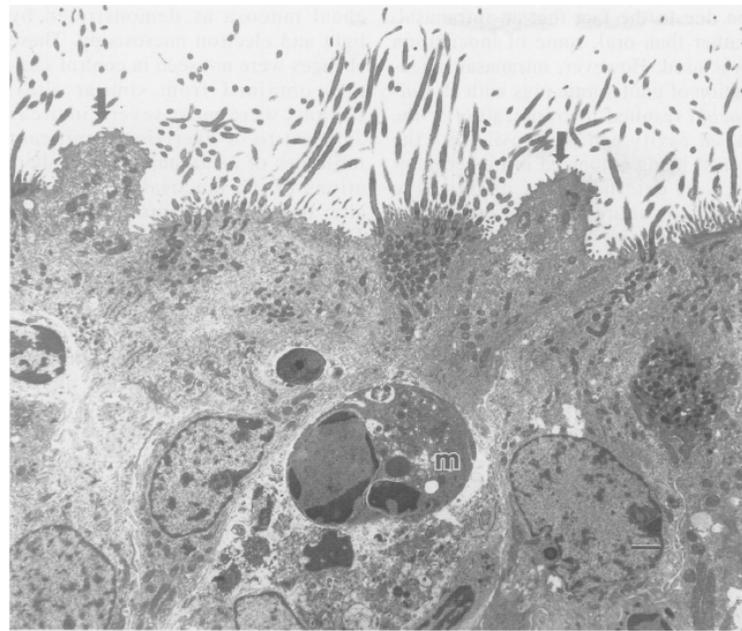


Figure 3. Nasal mucosa; CDCD pig 4 h post-inoculation. There is multifocal cell protrusion (arrows). There are decreased numbers of cilia. A cell containing phagolysosomes within the mucosa is identified as a macrophage (m). Bar = 1 micron.

Mucosa nasal 36 horas p.i.

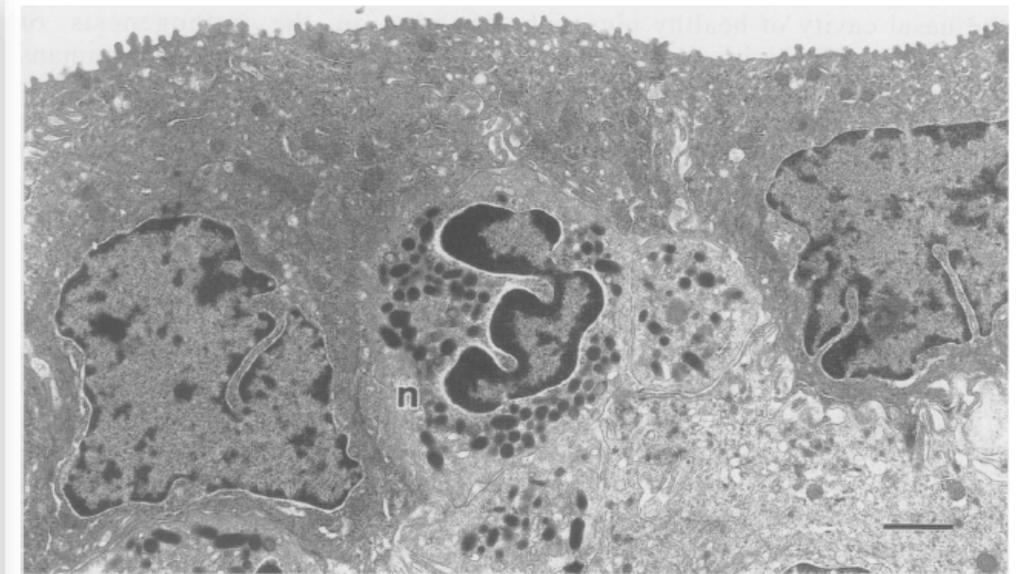


Figure 4. Nasal mucosa; CDCD pig 36 h post-inoculation. An intraepithelial neutrophil (n) is present. There is diffuse loss of cilia and basal bodies, microvilli are irregular and reduced in size, and there is dilation of the cytocavitary network within the apical cytoplasm. Bar = 1 micron.

Complejo respiratorio porcino: resultado de las coinfecciones



PRRSV
ADV
PCV-2
M. hyopneumoniae

2

Alteración de la fagocitosis

Disminución de las defensas locales

Colonización de vías respiratorias bajas por agentes secundarios

Otras infecciones secundarias

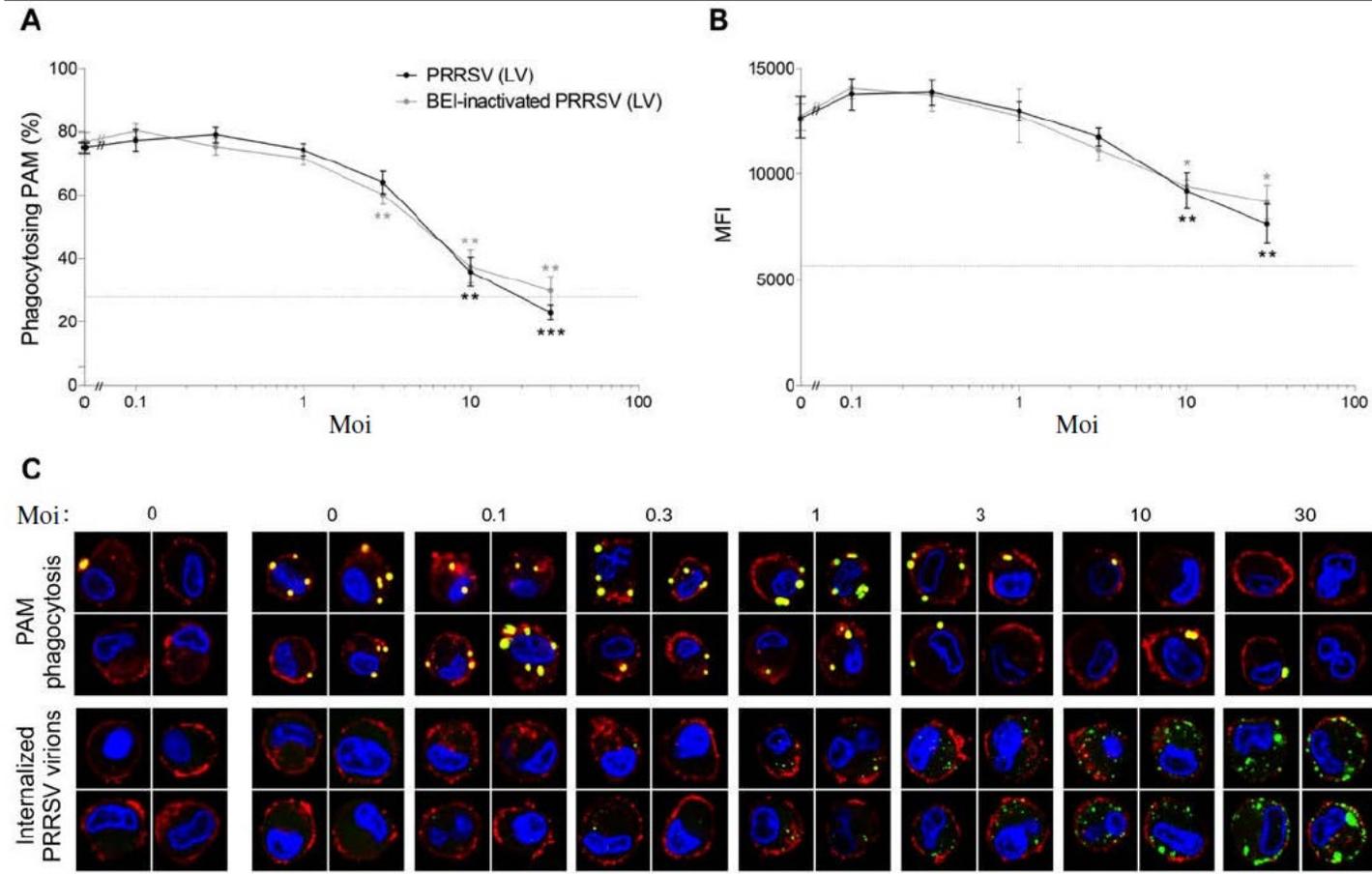


Figure 2 4 °C

37 °C

Complejo respiratorio porcino: resultado de las coinfecciones



SIV
PRRSV
A. pleuropneumoniae
Bacterias Gram -

3

Potenciación de la respuesta inflamatoria

Liberación de citoquinas + LPS

Coinfección PRRSV-SIV

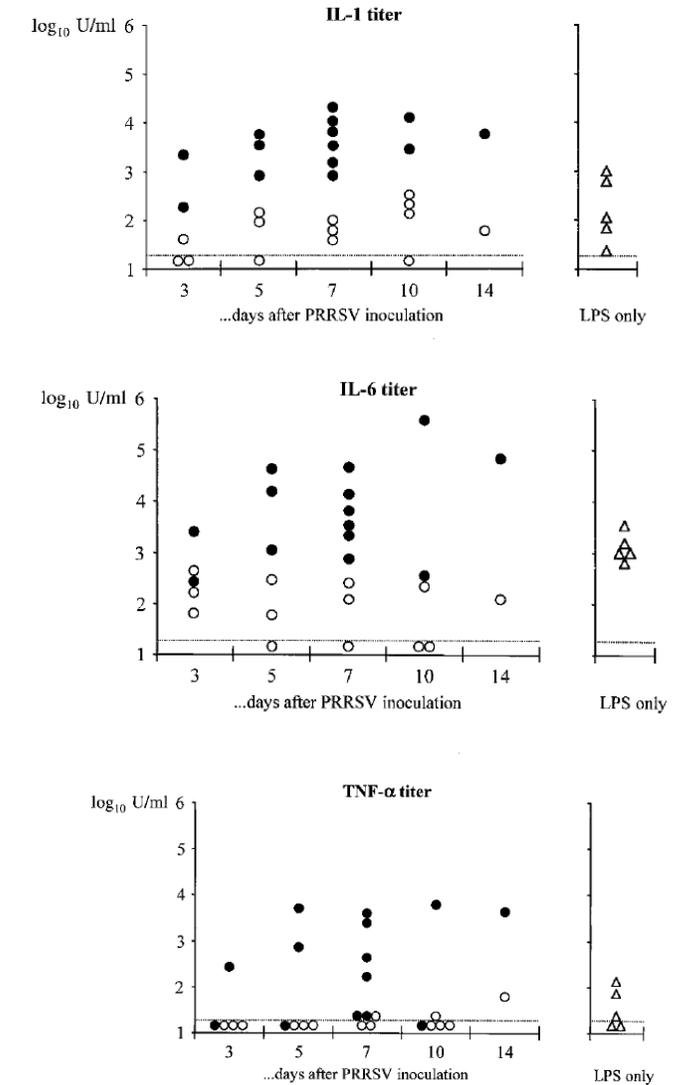
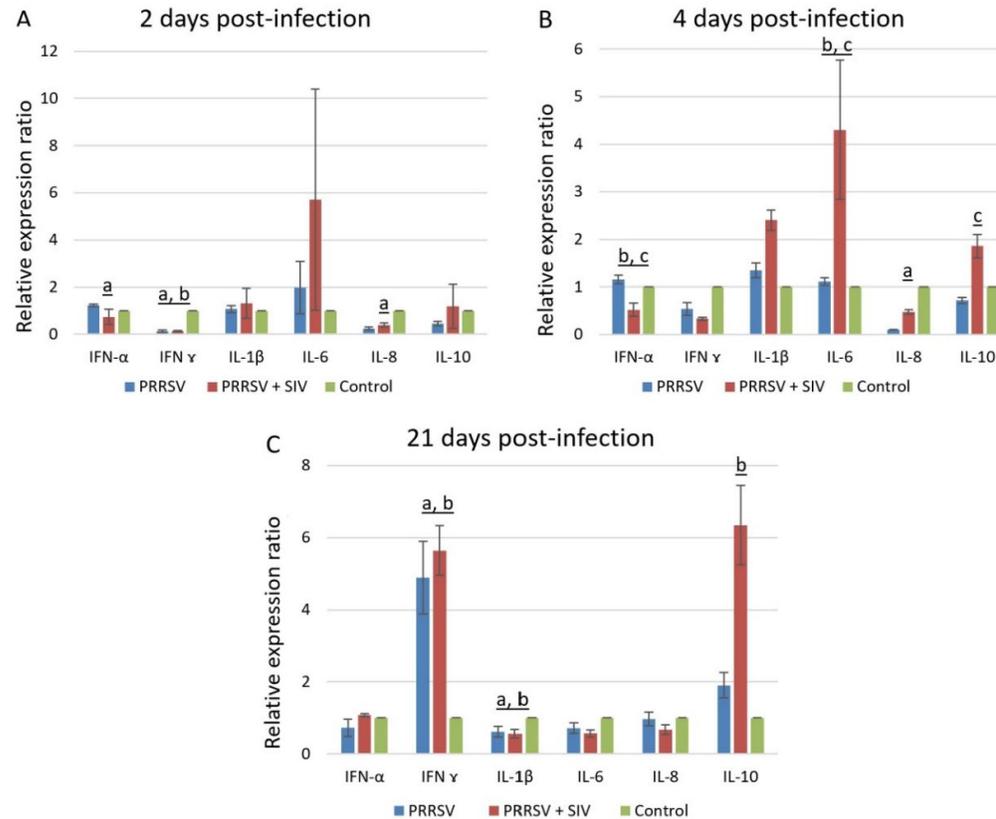


Fig. 2. Mean (\pm SEM) cytokines mRNA expression profile in BALF cells following infection with PRRSV or co-infection with PRRSV and SIV at 2 dpi (A), 4 dpi (B), and 21 dpi (C). a - $p < 0.05$ between PRRSV-infected group and control group; b - $p < 0.05$ between co-infected group and control group; c - $p < 0.05$ between PRRSV-infected group and co-infected group

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección PRRSV - *M. hyopneumoniae*

Coinfecciones virus - bacterias

Lesiones macroscópicas

M. hyopneumoniae agrava la neumonía inducida por PRRSV



Efecto más marcado en infección sincrónica o si PRRSV antecede a *M. hyopneumoniae*

Group	Infection status (day of inoculation ^a)	No. of pigs necropsied at:			Total no. of pigs necropsied
		Day 3	Day 10	Day 28	
A	PRRSV (0), <i>M. hyopneumoniae</i> (0)	6	6	8	20
B	PRRSV (0), <i>M. hyopneumoniae</i> (-21)	6	6	8	20
C	PRRSV (-10), <i>M. hyopneumoniae</i> (0)	6	6	8	20
D	<i>M. hyopneumoniae</i> (-21)	6	6	8	20
E	<i>M. hyopneumoniae</i> (0)	6	6	8	20
F	PRRSV (0)	6	6	8	20
G	Control (0)	6	6	8	20

^a Pigs were 6 weeks old at day 0.

TABLE 3. Percentage of lung with visible pneumonia lesions in pigs infected with either *M. hyopneumoniae*, PRRSV, or both

Group	% of lung exhibiting pneumonia induced by PRRSV ^a or <i>M. hyopneumoniae</i> ^b at the following necropsy date:						
	Day 3		Day 10		Day 28		
	PRRSV	<i>M. hyopneumoniae</i>	PRRSV	<i>M. hyopneumoniae</i>	PRRSV	<i>M. hyopneumoniae</i>	
Coinfecciones	A	8.2 ± 4.5 B ^c	0.2 ± 0.3	50.8 ± 15.9 A	4.1 ± 4.1 A	42.9 ± 7.5 A	7.7 ± 5.7 A
	B	12.3 ± 6.0 B	1.6 ± 1.7	48.8 ± 26.4 A	1.3 ± 2.2 B	15.6 ± 14.5 C	3.3 ± 5.9 B,C
	C ^d	42.8 ± 12.8 A	0.01 ± 0.02	29.5 ± 9.1 B	2.5 ± 3.1 A,B	28.8 ± 14.1 B	7.2 ± 8.1 A,B
Infecciones independientes	D	0 C	0.1 ± 0.1	0 C	0.9 ± 2.1 B	0 D	1.2 ± 3.0 C,D
	E	0 C	0.2 ± 0.3	0 C	0.2 ± 0.2 B	0 D	8.3 ± 4.0 A
	F	13.7 ± 5.8 B	0.1 ± 0.2	56.5 ± 12.7 A	0.05 ± 0.1 B	0.8 ± 1.59 D	0 D
	G	0 C	0.1 ± 0.3	0 C	0.02 ± 0.05 B	0 D	0 D

^a As estimated by visual observation.

^b As determined by lesion sketches and image analysis.

^c Within each column, values followed by different letters (A, B, C, or D) are significantly different from each other ($P < 0.001$).

^d Group C received PRRSV on day -10 and *M. hyopneumoniae* on day 0 and so is not matched with the other groups with respect to the number of days after inoculation with PRRSV.

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección PRRSV - *M. hyopneumoniae*

Lesiones microscópicas

Coinfecciones virus - bacterias

Group	Infection status (day of inoculation ^a)	No. of pigs necropsied at:			Total no. of pigs necropsied
		Day 3	Day 10	Day 28	
A	PRRSV (0), <i>M. hyopneumoniae</i> (0)	6	6	8	20
B	PRRSV (0), <i>M. hyopneumoniae</i> (-21)	6	6	8	20
C	PRRSV (-10), <i>M. hyopneumoniae</i> (0)	6	6	8	20
D	<i>M. hyopneumoniae</i> (-21)	6	6	8	20
E	<i>M. hyopneumoniae</i> (0)	6	6	8	20
F	PRRSV (0)	6	6	8	20
G	Control (0)	6	6	8	20

^a Pigs were 6 weeks old at day 0.

TABLE 4. Microscopic lesion scores from pigs inoculated with either PRRSV, *M. hyopneumoniae*, or both

Score for pneumonia induced by PRRSV^a or *M. hyopneumoniae*^b at the following necropsy date:

Group	Day 3		Day 10		Day 28	
	PRRSV	<i>M. hyopneumoniae</i>	PRRSV	<i>M. hyopneumoniae</i>	PRRSV	<i>M. hyopneumoniae</i>
	A	1.2 ± 1.1 B ^c	0	4.0 ± 0.9 A	1.7 ± 1.0 A	4.8 ± 0.7 A
B	1.0 ± 0 B	0.3 ± 0.5	3.3 ± 1.5 A	0.8 ± 0.4 B	1.3 ± 0.5 B	1.6 ± 1.1 C
C ^d	3.3 ± 0.5 A	0	3.3 ± 0.5 A	1.8 ± 1.0 A	4.3 ± 1.2 A	3.8 ± 0.5 A
D	0 C	0.2 ± 0.4	0 B	0.5 ± 0.6 B,C	0.1 ± 0.4 C	1.0 ± 0 C
E	0.2 ± 0.4 C	0	0 B	0.5 ± 0.6 B,C	0 C	2.5 ± 0.8 B
F	1.5 ± 0.8 B	0.3 ± 0.5	3.7 ± 0.8 A	0 C	1.4 ± 0.5 B	1.1 ± 0.6 C
G	0.2 ± 0.4 C	0	0 B	0.8 ± 0.4 B,C	0 C	0.3 ± 0.5 D

^a PRRSV scores are based on the severity of interstitial pneumonia. 0, no microscopic lesions; 1, mild multifocal pneumonia; 2, mild diffuse pneumonia; 3, moderate multifocal pneumonia; 4, moderate diffuse pneumonia; 5, severe multifocal pneumonia; 6, severe diffuse pneumonia.

^b *M. hyopneumoniae* scores are based on the severity of peribronchiolar and perivascular cuffing and lymphoid nodule formation, as follows: 1, mild; 2, moderate; 3, severe; 4, very severe. 0, no microscopic lesions.

^c Within each column, values followed by different letters (A, B, C, or D) are significantly different from each other ($P < 0.001$).

^d Group C received PRRSV on day -10 and *M. hyopneumoniae* on day 0 and so is not matched with the other groups with respect to the number of days after inoculation with PRRSV.

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección SIV - *M. hyopneumoniae*

Coinfecciones virus - bacterias

Ganancia Media Diaria

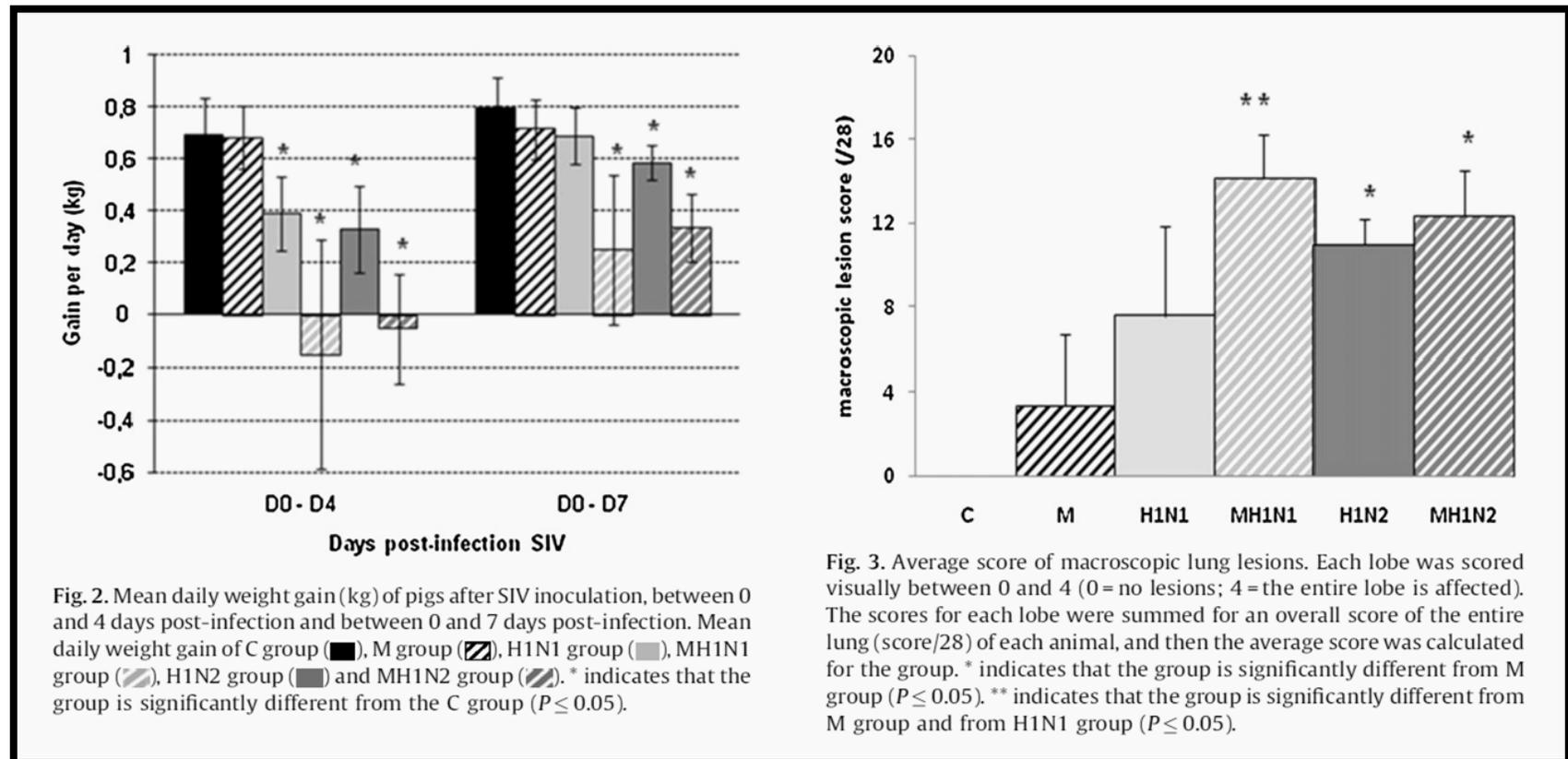


Fig. 2. Mean daily weight gain (kg) of pigs after SIV inoculation, between 0 and 4 days post-infection and between 0 and 7 days post-infection. Mean daily weight gain of C group (■), M group (▨), H1N1 group (■), MH1N1 group (▧), H1N2 group (■) and MH1N2 group (▩). * indicates that the group is significantly different from the C group ($P \leq 0.05$).

Grado de lesión macroscópica

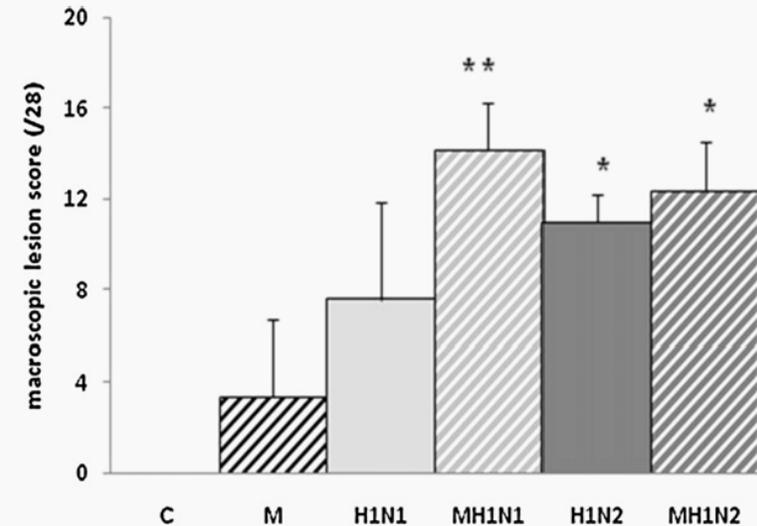


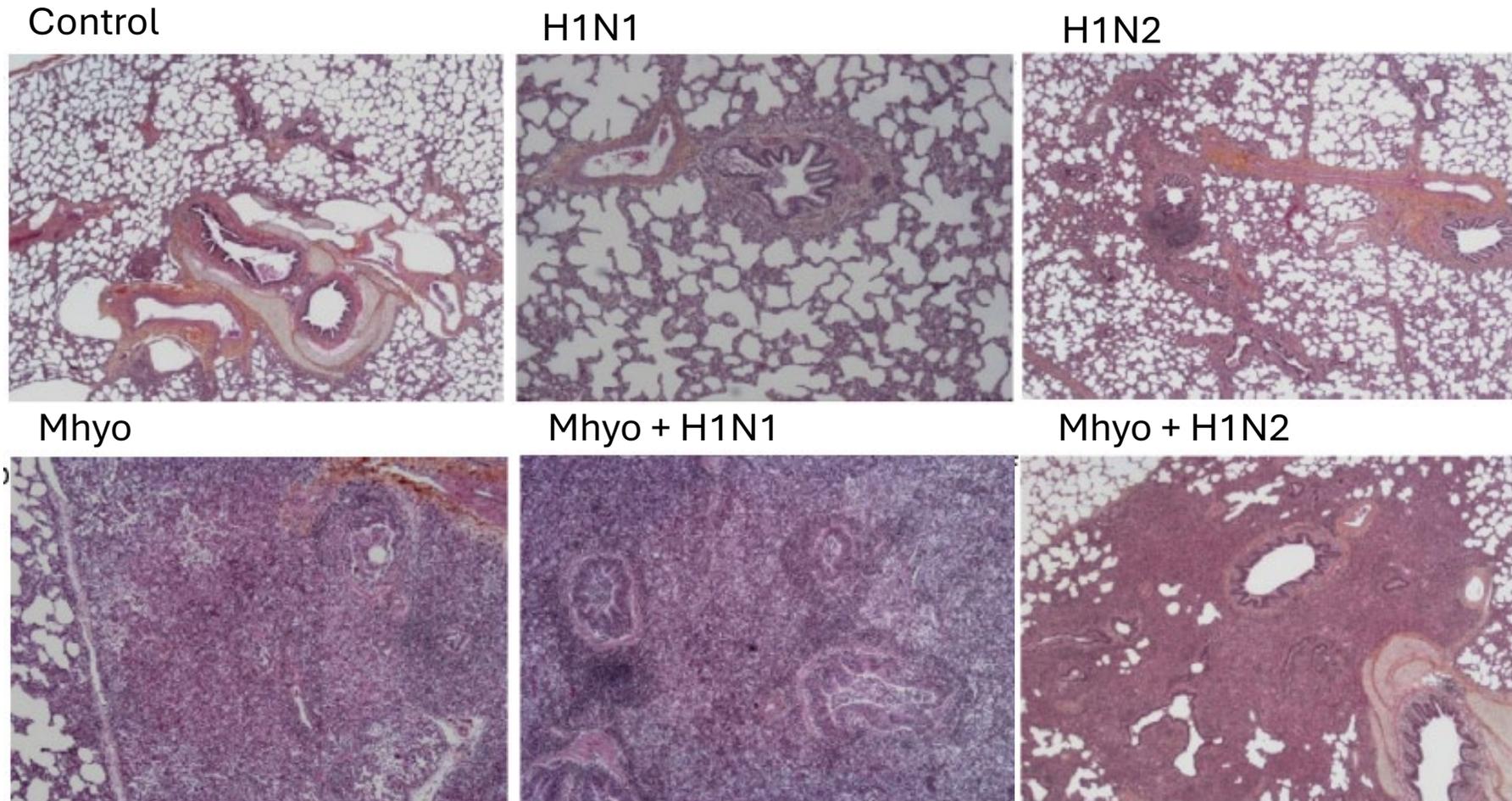
Fig. 3. Average score of macroscopic lung lesions. Each lobe was scored visually between 0 and 4 (0 = no lesions; 4 = the entire lobe is affected). The scores for each lobe were summed for an overall score of the entire lung (score/28) of each animal, and then the average score was calculated for the group. * indicates that the group is significantly different from M group ($P \leq 0.05$). ** indicates that the group is significantly different from M group and from H1N1 group ($P \leq 0.05$).

La coinfección SIV- *M. hyopneumoniae* produce lesiones más graves y mayor alteración de los parámetros productivos que las infecciones independientes

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección SIV - *M. hyopneumoniae*

Coinfecciones virus - bacterias



Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección SIV – *G. parasuis*

Signos clínicos y lesiones

Temperatura rectal

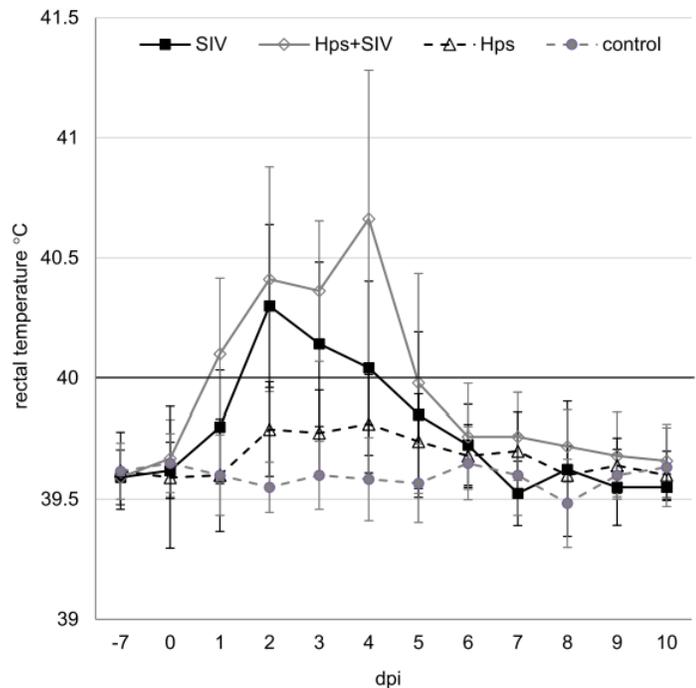


Fig. 1 Rectal temperature (mean ±SD) in pigs single or dual inoculated with swine influenza virus (SIV) and/or *Haemophilus parasuis* (Hps). Number of pigs affected: Hps + SIV 11/11; SIV 7/11; Hps 3/11

Puntuación clínica

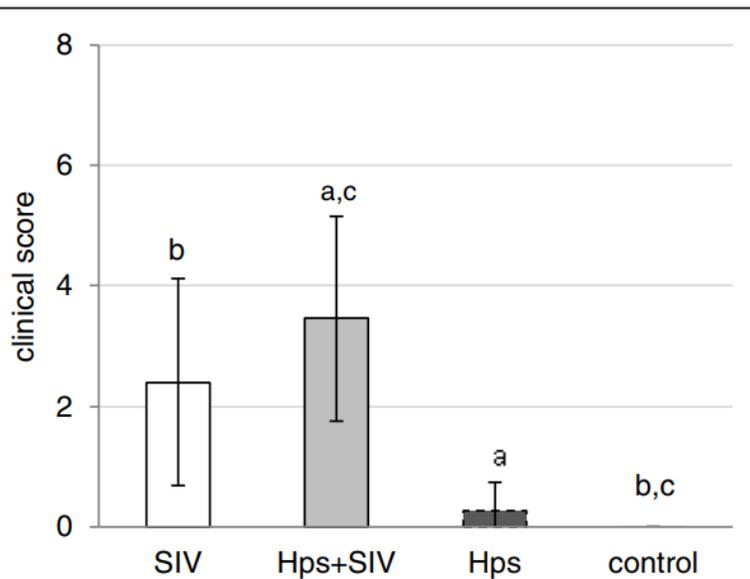


Fig. 2 Clinical score (mean±SD) of pigs single or dual inoculated with swine influenza virus (SIV) and/or *Haemophilus parasuis* (Hps). The significant differences between groups are indicated with the same superscripts

Lesión pulmonar

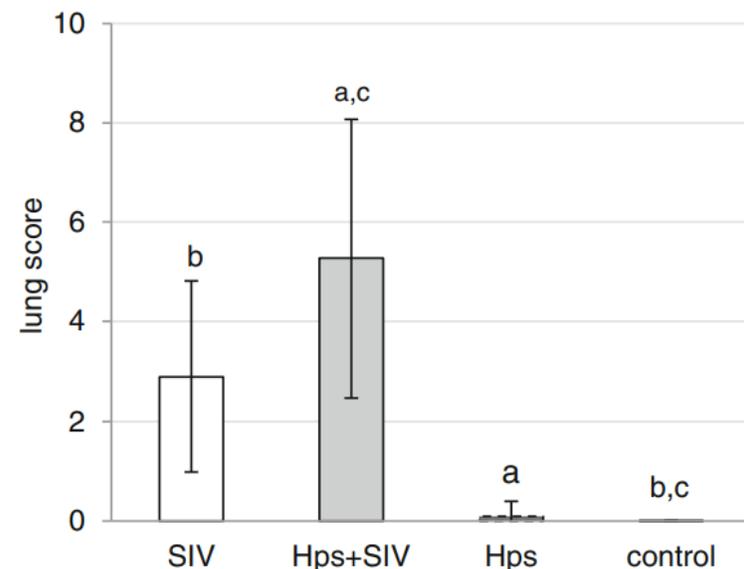


Fig. 3 Lung score (mean±SD) of pigs single or dual inoculated with swine influenza virus (SIV) and/or *Haemophilus parasuis* (Hps). The significant differences between groups are indicated with the same superscripts

Coinfecciones virus - bacterias

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección SIV – *G. parasuis*

Coinfecciones virus - bacterias

Excreción SIV- *G. parasuis*

La coinfección SIV- *G. parasuis* aumenta la excreción del virus de la gripe

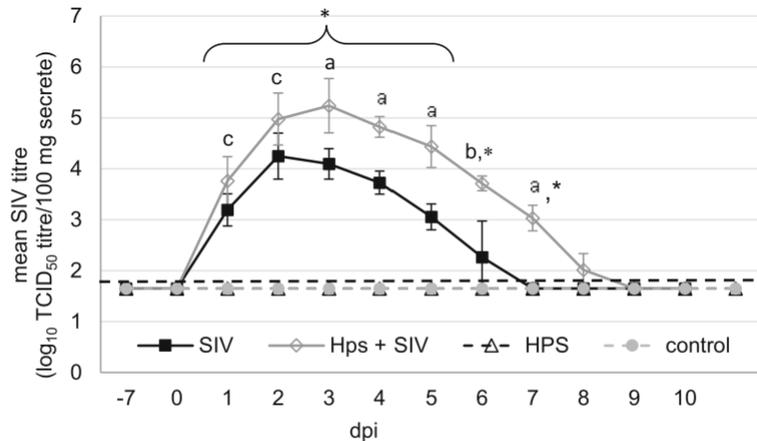


Fig. 4 Nasal virus shedding (mean ±SD) after inoculation of pigs with swine influenza virus (A/Poland/Swine/14131/2014) and or *Haemophilus parasuis*. Mean virus titres (determined by cell culture) in nasal swabs collected during study period. The dashed line represents the detection limit. * - the significant differences compared to control group; a - the significant differences between inoculated groups; b - the significant differences between SIV and *Hps* + SIV inoculated groups; c - the significant differences between SIV, *Hps* + SIV and *Hps*

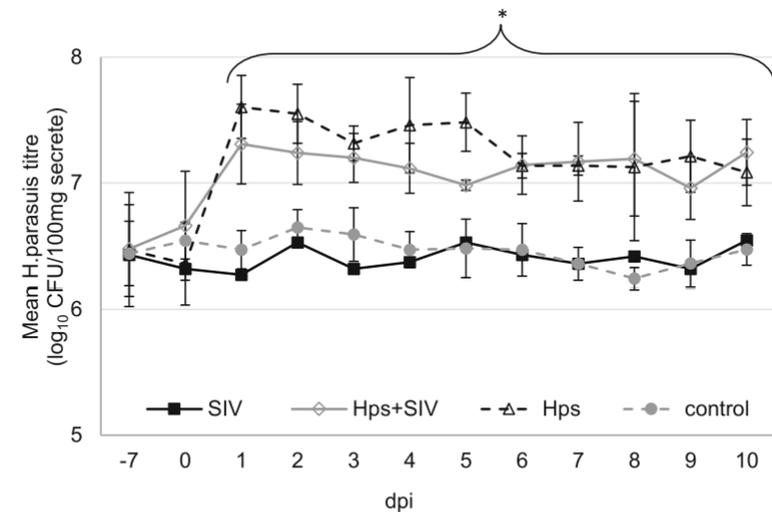


Fig. 5 Nasal bacteria shedding (mean ±SD) after inoculation of pigs with *Haemophilus parasuis*. Mean colony forming units (CFU) (determined by quantitative PCR) in nasal swabs collected during study period. * - the significant differences compared to control and SIV group

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfecciones virus - bacterias

Coinfección SIV – *G. parasuis*

Título en pulmón SIV- *G. parasuis*

La coinfección SIV- *G. parasuis* aumenta la replicación de ambos patógenos

Replicación del virus de la gripe

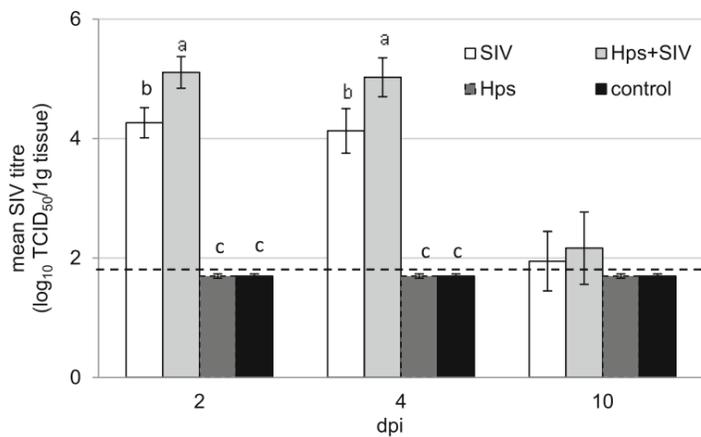


Fig. 6 Swine influenza virus titre (mean \pm SD) (determined by cell culture) in the lung at 2, 4 and 10 days after single or dual inoculation of pigs with swine influenza virus and/or *Haemophilus parasuis*. The dashed line represents the detection limit. Columns with various superscripts within the same day of study differ significantly

Replicación de *G. parasuis*

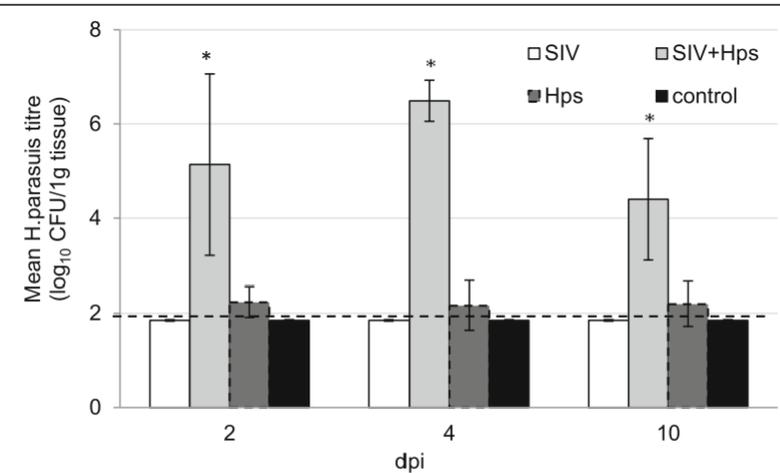


Fig. 7 *H. parasuis* titres (mean \pm SD) (determined by quantitative PCR) in the lung at 2, 4 and 10 days after single or dual inoculation of pigs with swine influenza virus and/or *Haemophilus parasuis*. The dashed line represents the detection limit. * - significant differences compared to remaining groups

Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección SIV – *S. suis*

Carga vírica

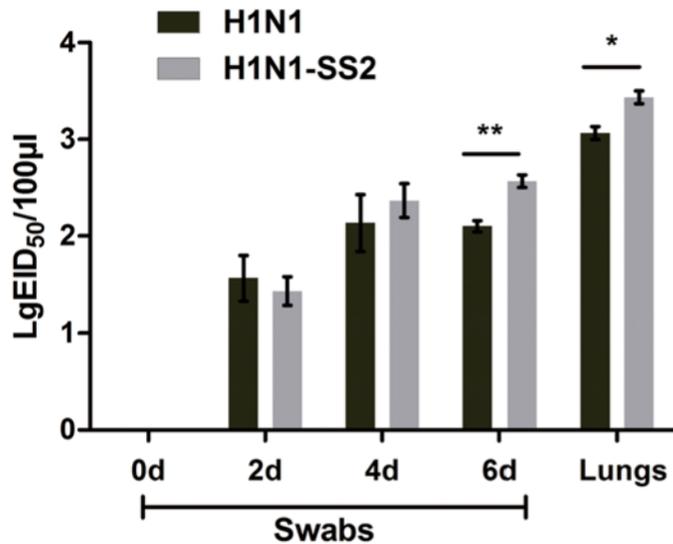


Fig 2. Virus isolation of nasal swabs and lungs in each group. Swabs were collected from the right nostrils of pigs from each group every other day. The obtained nasal swabs were inserted into vials containing 1.5 mL of sterile PBS. Supernatants were collected and viruses were qualified as LgEID₅₀. At the day 6 of the experiment, all pigs were humanely euthanized and lungs were collected and homogenized in sterile PBS and then centrifuged to collect the supernatant. Virus titer was quantified as LgEID₅₀. Data were showed as mean ± SEM by Student's t-test. *P*-value less than 0.05 was noted with a single-asterisk and *p*-value less than 0.01 was noted with a double-asterisk.

Lesiones

Coinfecciones virus - bacterias

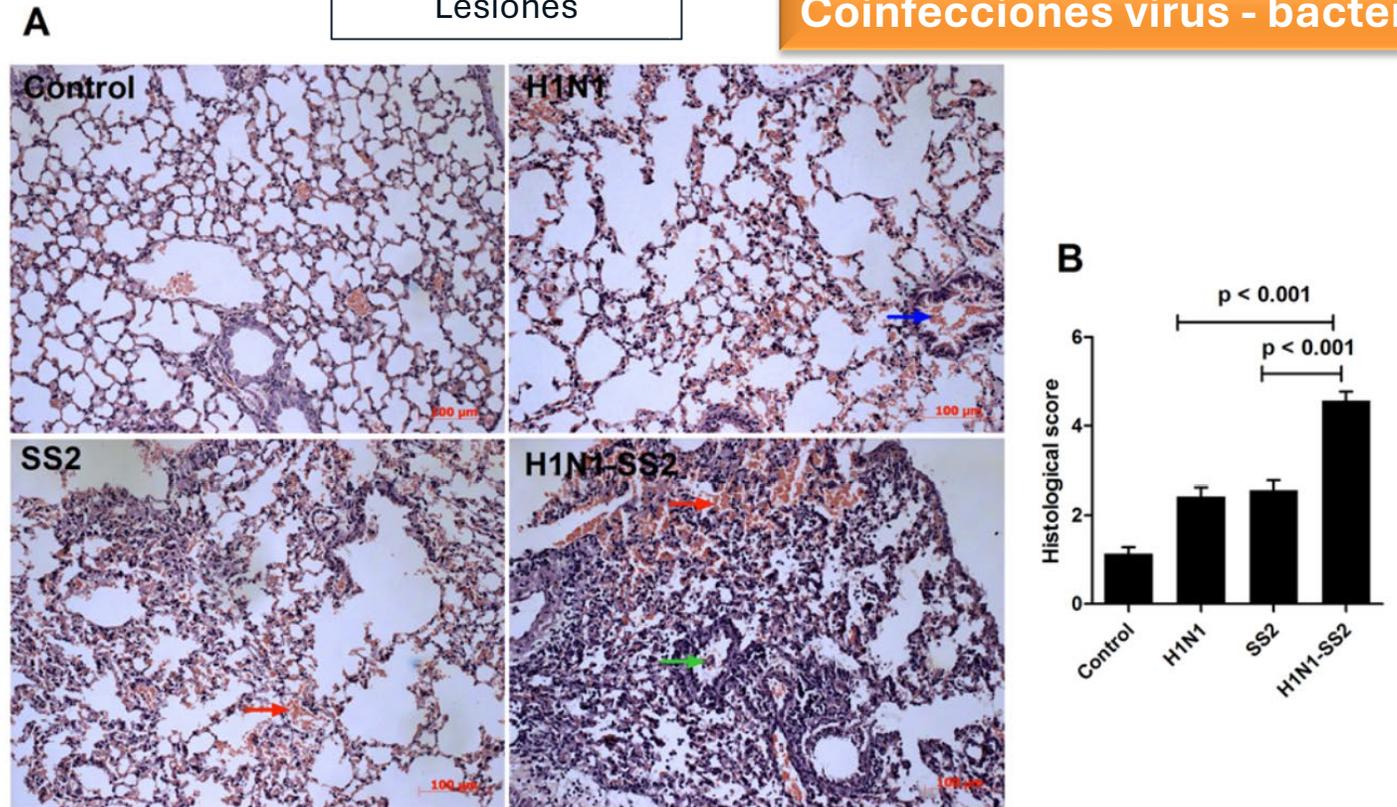


Fig 1. Histopathologic changes in lungs of infected pigs. Pigs were PBS-control infected or infected with H1N1, SS2 and H1N1-SS2 as described in methods. On day 6 of the experiment, lungs were removed and underwent Hematoxylin and eosin stain. (A) The microscopic lesions of lung tissues from each group showed different extent of acute pneumonia with pathological changes: alveolar wall thickening, bleeding (red arrow), debris in the lumen (green arrow), erythrocyte effusion (blue arrow), and the accumulation of inflammatory cells. (B) Histological score of sections of lungs in pigs from each group were showed as mean ± SEM by two-tailed Student's t-test. *P*-value less than 0.05 was considered to represent a statistically significant difference.

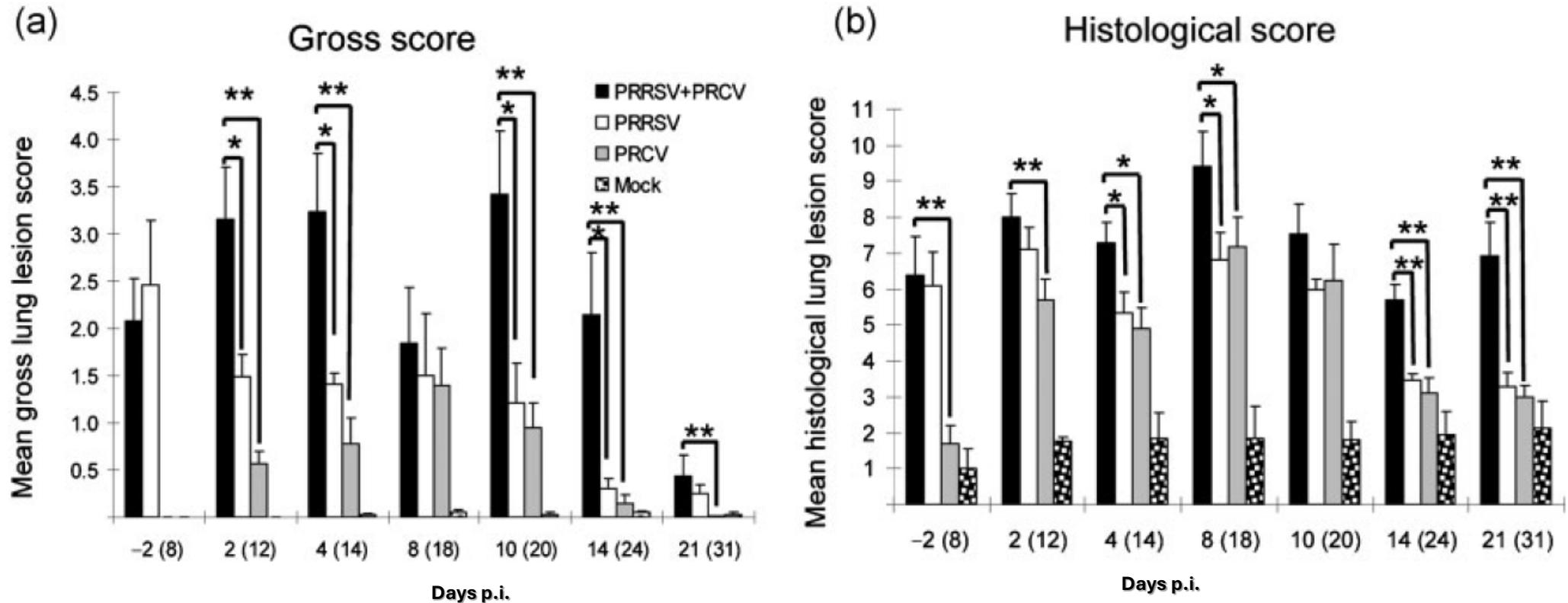
Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección PRRSV - PRCV

Coinfecciones virus - virus

Inoculación PRRSV: D0
Inoculación PRCV: D10

La coinfección PRRSV-PRCV produce lesiones más graves y mayor alteración de los parámetros productivos que las infecciones independientes



Complejo respiratorio porcino: resultado de las coinfecciones

Coinfección PRRSV – PCV-2

Coinfecciones virus - virus

Table 1
Experimental design and group designations.

Group designation	Batch ^a	Number of pigs	PCV2 inoculum	PRRSV inoculum
Negative controls	1	8	None	None
Col-92-2a	1	9	PCV2a	VR-2385
Col-92-2b	1	9	PCV2b	VR-2385
Col-06-2a	1	9	PCV2a	NC16845b
Col-06-2b	1	9	PCV2b	NC16845b
PRRSV-I-92	2	4	None	VR-2385
PRRSV-I-06	2	5	None	NC16845b
B3-PRRSV-I-92	3	5	None	VR-2385
B3-PRRSV-I-06	3	4	None	NC16845b

^a Batch 1 and 2 pigs were derived from the same source herd free of PRRSV and PCV2 whereas batch 3 pigs were derived from a different source herd seropositive for PCV2.

Table 7

Mean group macroscopic (percentage of lung surface affected by lesions) and microscopic (interstitial pneumonia ranging from 0 = normal to 6 = severe, diffuse) lung lesions (mean group amount ± SE). Data obtained from B3-PRRSV-I-92 and B3-PRRSV-I-06 pigs (gray shaded area) were not included in the analysis. Significant ($P < 0.05$) differences between groups are indicated by different superscripts (A, B, C).

Group	Macroscopic lung lesions (0–100%)	Microscopic lung lesions (0–6)
Negative controls	0.1 ± 0.1 ^A	0.75 ± 0.25 ^A
Col-92-2a	54.8 ± 4.3 ^B	4.44 ± 0.24 ^B
Col-92-2b	56.3 ± 4.5 ^B	4.67 ± 0.17 ^B
Col-06-2a	52.8 ± 6.4 ^B	4.78 ± 0.32 ^B
Col-06-2b	48.7 ± 4.7 ^{B,C}	4.44 ± 0.24 ^B
PRRSV-I-92	31.8 ± 8.3 ^{B,C}	2.50 ± 0.87 ^A
PRRSV-I-06	4.3 ± 2.0 ^{A,C}	1.80 ± 0.80 ^A
B3-PRRSV-I-92	43.8 ± 5.7	4.60 ± 0.24
B3-PRRSV-I-06	25.8 ± 10.2	4.00 ± 0.71

PRRSV y
PCV-2

Solo
PRRSV

***Diagnóstico de las
enfermedades
respiratorias***

Aproximaciones al diagnóstico de las alteraciones respiratorias

¿Cómo abordar el diagnóstico de las enfermedades respiratorias?

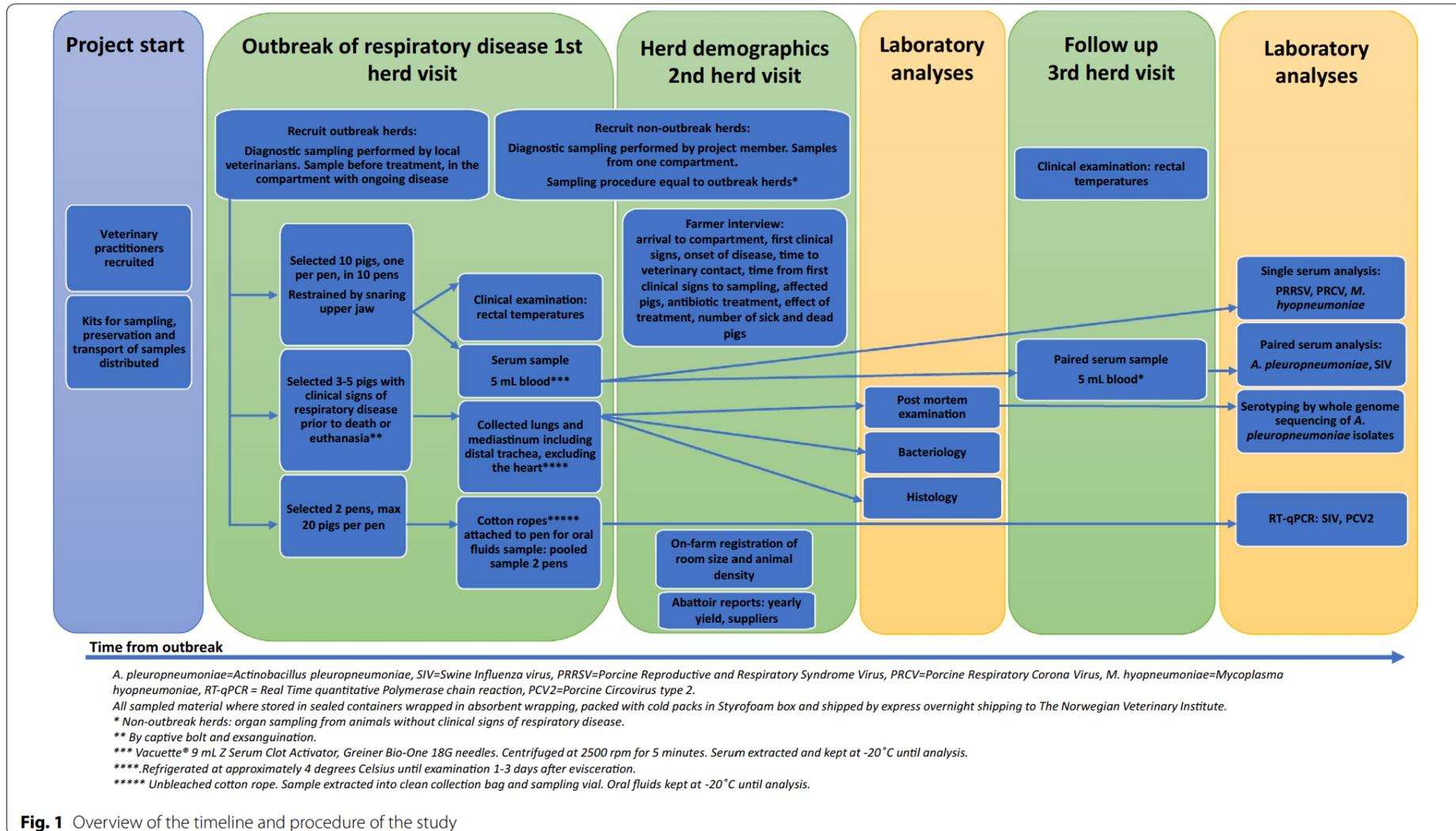


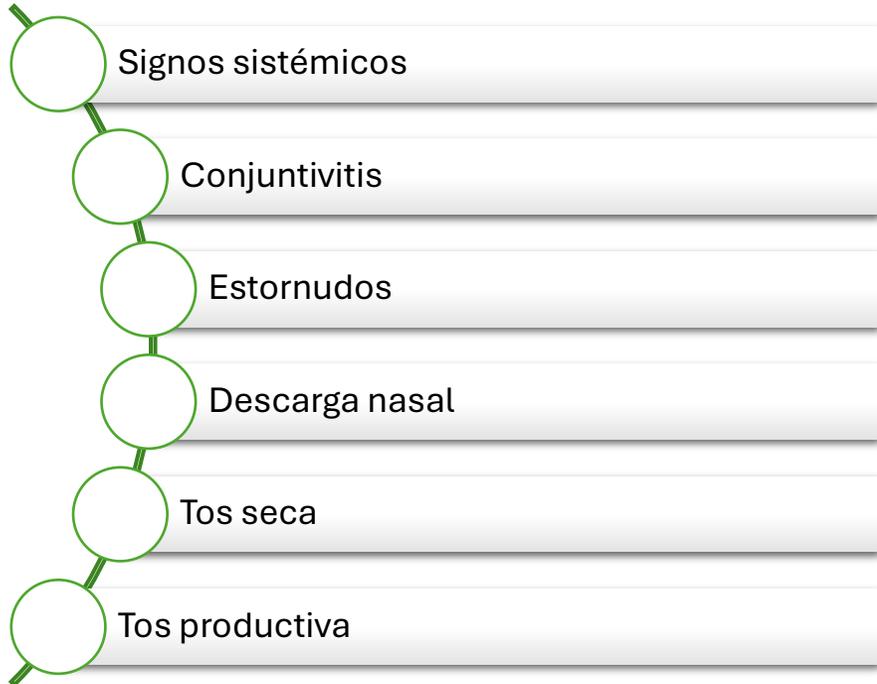
Fig. 1 Overview of the timeline and procedure of the study

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

1. Edad de presentación
2. Morbilidad y mortalidad
3. Signos clínicos

Observación de los animales



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Signos clínicos



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Signos clínicos



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Signos clínicos

Índice de tos: toses o estornudos/cerdo/minuto

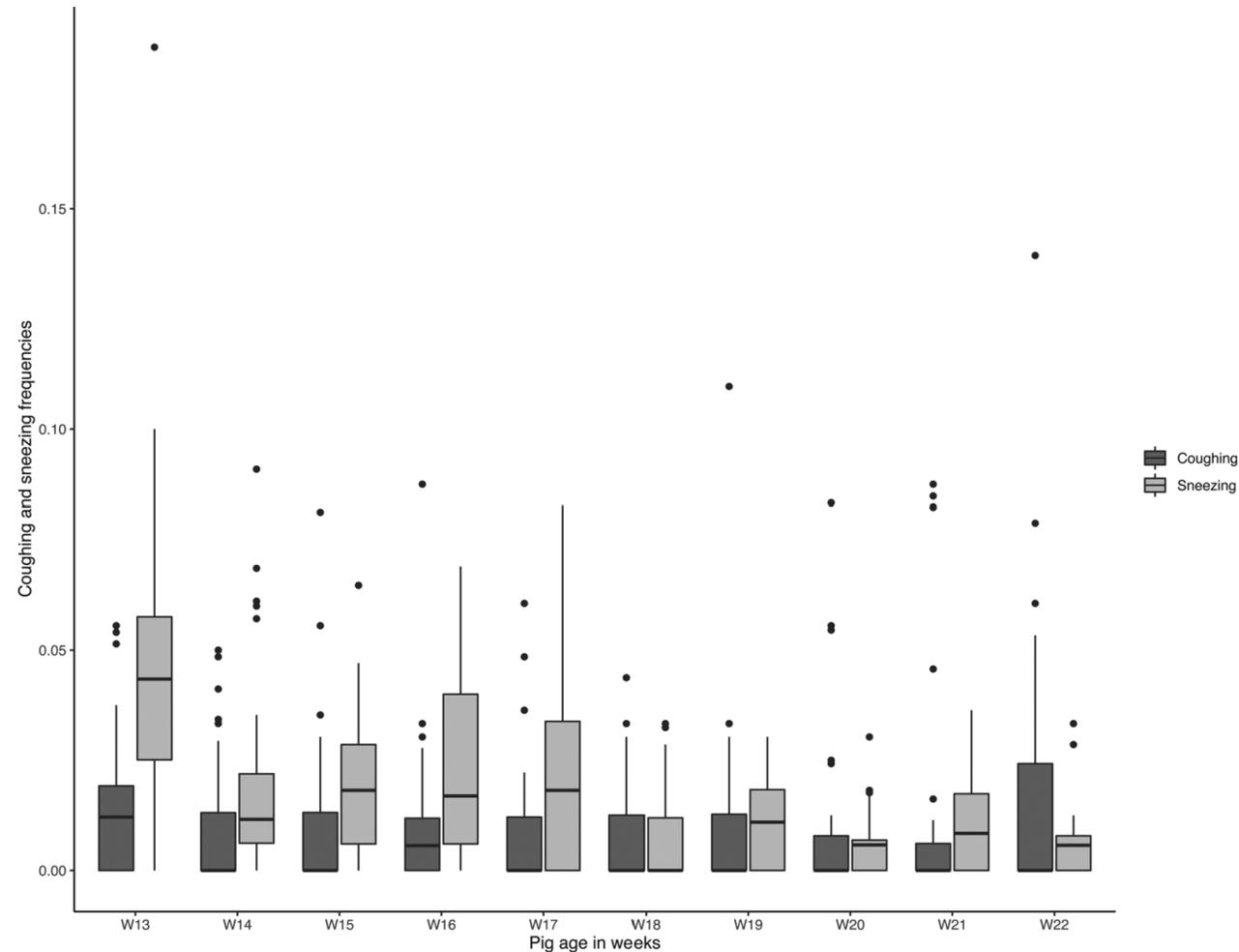


Fig. 1. Coughing and sneezing frequencies (average number of coughs/sneezes per pig per minute) measured in 48 pens of pigs aged 13 weeks ($n = 33 \pm 2$ pigs/pen) for ten consecutive weeks, on an Irish finisher pig farm in 2018 (box: median and interquartile range (IQR); whiskers: $1.5 \times$ IQR).

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Signos clínicos

Índice de tos: N° total de episodios de tos / [n° de cerdos x tiempo de observación (min)] x 100

Monitorización con ayuda de la IA (ReHS: Respiratory Health Status)



Figure 1. Left picture: SoundTalks® monitor in science mode (blue led). Right picture: Plugged into a power outlet (blue led).

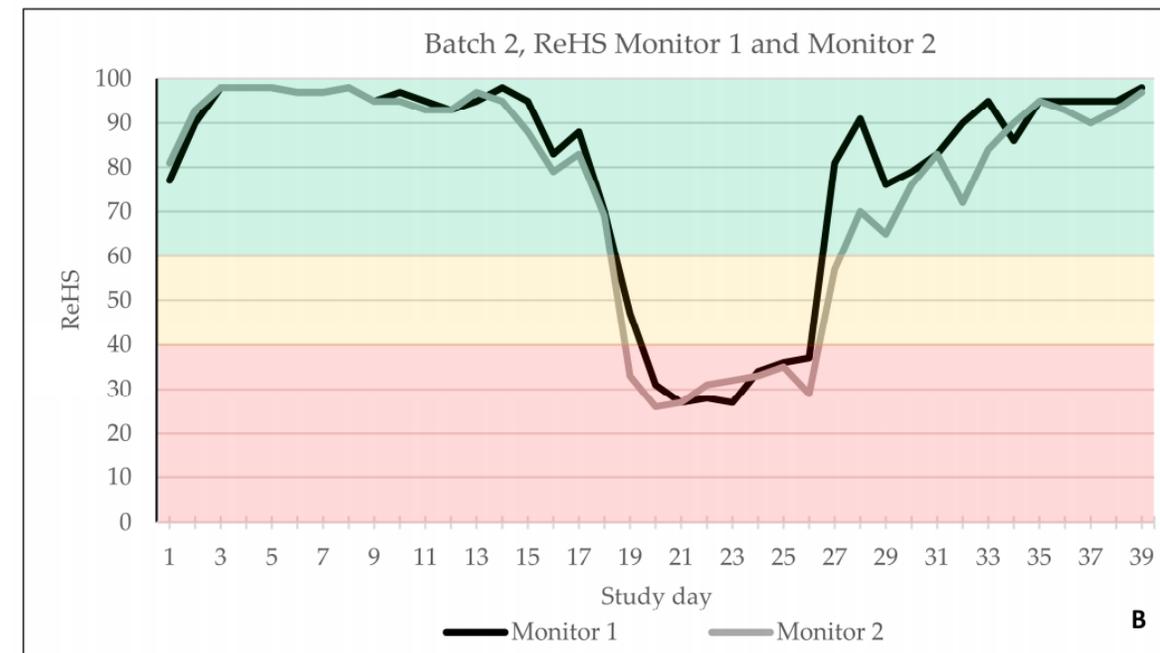
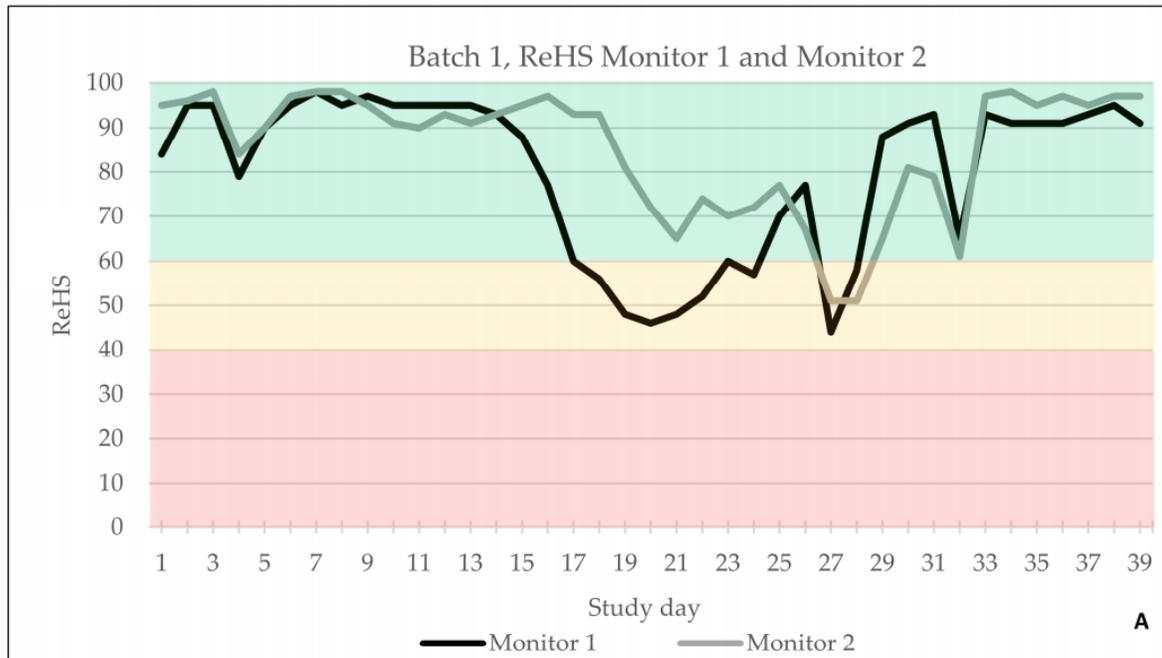


Figure 3. ReHS of batch 1 (A) and batch 2 (B) obtained by monitor 1 and monitor 2 over the entire study period. Green indicated a normal ReHS, which ranges from 60 to 100. Yellow indicated a potential risk of a respiratory problem, equivalent to 40–59 ReHS and red a high risk of a respiratory problem, equivalent to 0–39 ReHS.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Bronconeumonía catarral (purulenta)



Vía de entrada: aerógena

Observaciones macroscópicas:

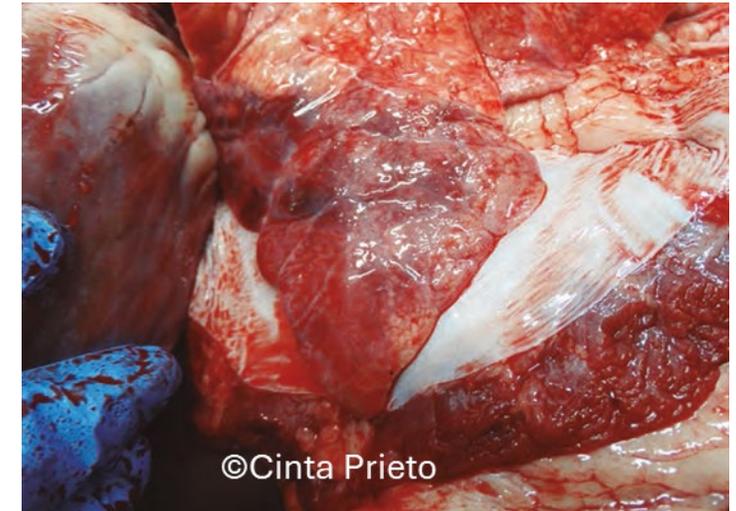
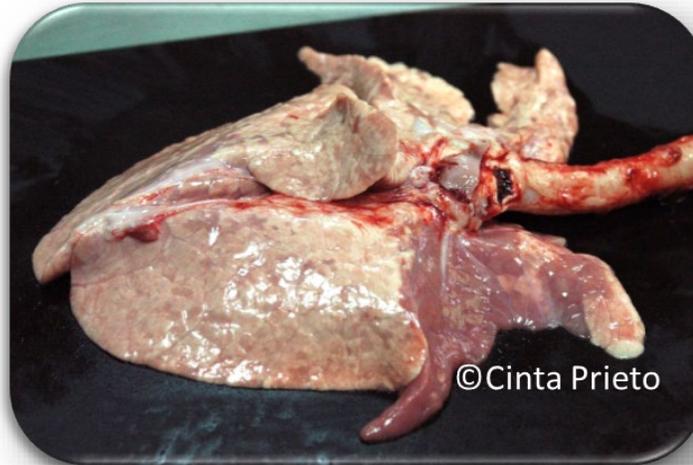
- Distribución cráneo-ventral
- Elevado grado de consolidación
- Clara demarcación entre tejido afectado y tejido normal
- Exudado purulento en vías respiratorias

Lesiones microscópicas:

- Inflamación de los espacios broncoalveolares
- Infiltración de neutrófilos
- Abundante exudado en vías respiratorias y alveolos

Agentes causales más habituales:

- *Bordetella bronchiseptica*
- *Pasteurella multocida*
- *Mesomycoplasma hyopneumoniae* (neumonía enzoótica)



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Pleuroneumonía fibrinosa o fibrinonecrotizante

Lesiones



Vía de entrada: aerógena

- Observaciones macroscópicas:**
- Distribución cráneo-ventral o dorsocaudal (APP)
 - Daño tisular extenso. Con frecuencia necrosis del tejido pulmonar
 - Exudación y/o fibrina en la superficie pleural

- Lesiones microscópicas:**
- Engrosamiento de los tabiques interlobulillares,
 - Presencia de fibrina y neutrófilos en bronquios, bronquiolos y alvéolos
 - Áreas de necrosis coagulativa,
 - Pleuritis fibrinosa.
 - Casos crónicos:
 - Secuestros pulmonares,
 - Adherencias pleurales



- Agentes causales más habituales:**
- *Actinobacillus pleuropneumoniae*
 - *Bordetella bronchiseptica* (bronconeumonía necrótico-hemorrágica)



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Pleuroneumonía fibrinosa o fibrinonecrotizante

Lesiones

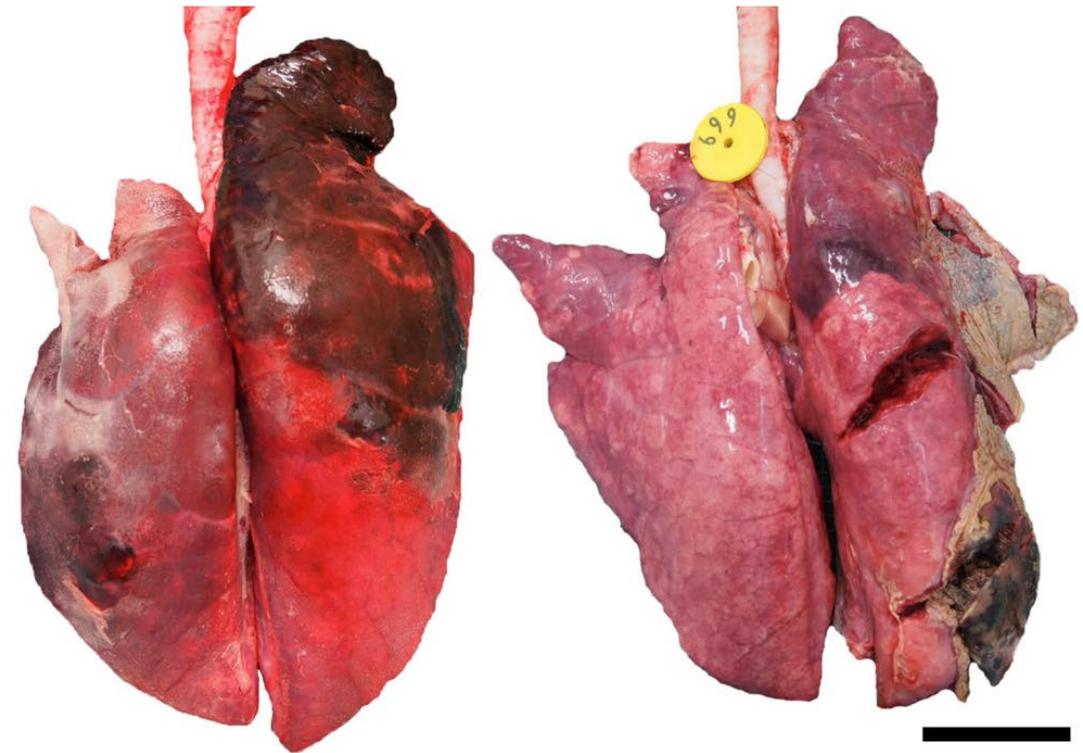
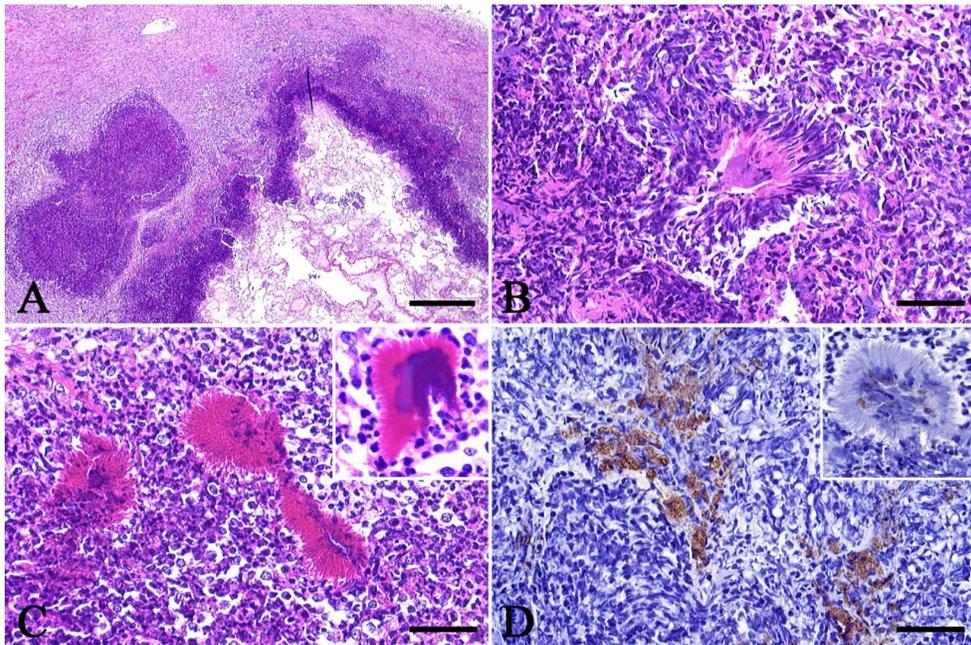


Fig. 1. Gross lesions of lungs. In the pig No. 695 (left side), the lung is swollen, and severe hemorrhages are observed in the right anterior and middle lobes and left caudal lobe. The surfaces of the left lobes are partially covered with fibrin. In the pig No. 699 (right side), severe hemorrhages and fibrin deposition are observed in the right middle and caudal lobes. Bar=5 cm.

Fig. 2. Histopathologic features of pneumonia. Pig No. 699. (A) Multifocal abscesses with central necrosis are encapsulated with granulation tissue. Hematoxylin and eosin (HE). Bar=400 μ m. (B) Numerous neutrophils infiltrate around the necrotic area. Oat cells surround the bacterial cells. HE. (C) Asteroid bodies in the inflammatory foci. Eosinophilic amorphous material with radiating club-shaped configurations is deposited around the bacterial cells (inset, higher magnification). HE. (D) The bacterial cells are immunolabeled with antiserum raised against *Actinobacillus pleuropneumoniae* serovar 15 (inset, higher magnification of the asteroid body). Immunohistochemistry. Bars (B–D)=40 μ m.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Pleuroneumonía fibrinosa o fibrinonecrotizante

Lesiones



Figure 3. Acute (A) and chronic (B) pleuropneumonia by *Actinobacillus pleuropneumoniae*: in (A), an acute, locally extensive, and protruding nodule coexists with associated pleuritis localized in the craniodorsal side of the left lung lobe, referable to *A. pleuropneumoniae* and cranioventral pneumonia. In (B), multifocal nodules of the chronic form.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Distribución cráneo-ventral
- Demarcación clara y distribución lobulillar
- Áreas firmes al tacto y purpúreas o grisáceas
- Más frecuente en lóbulos apicales e intermedios

Lesiones microscópicas:

- Necrosis del epitelio bronquial, bronquiolar y alveolar
- Oclusión de las vías respiratorias por células inflamatorias y material necrótico,
- Infiltración linfocítica peribronquial y perivascular
- Hiperplasia del epitelio de las vías respiratorias y los alvéolos

Neumonía bronquiolo-intersticial



Vía de entrada: aerógena



Agentes causales más habituales:

- *Mesomycoplasma hyopneumoniae* (micoplasmosis)
- Virus de la gripe



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Distribución cráneo-ventral
- Demarcación clara y distribución lobulillar
- Áreas firmes al tacto y purpúreas o grisáceas
- Más frecuente en lóbulos apicales e intermedios

Lesiones microscópicas:

- Necrosis del epitelio bronquial, bronquiolar y alveolar
- Oclusión de las vías respiratorias por células inflamatorias y material necrótico,
- Infiltración linfocítica peribronquial y perivascular
- Hiperplasia del epitelio de las vías respiratorias y los alvéolos

Neumonía bronquiolo-intersticial

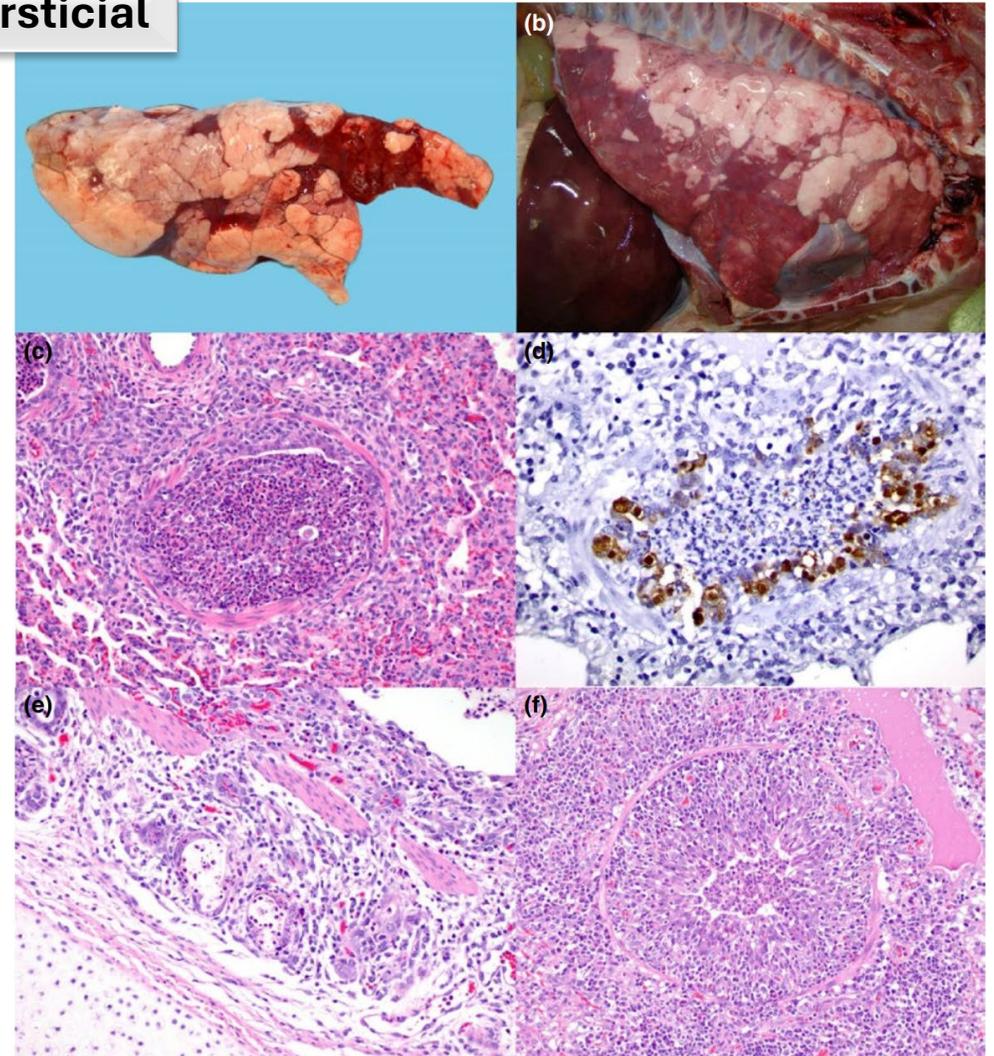


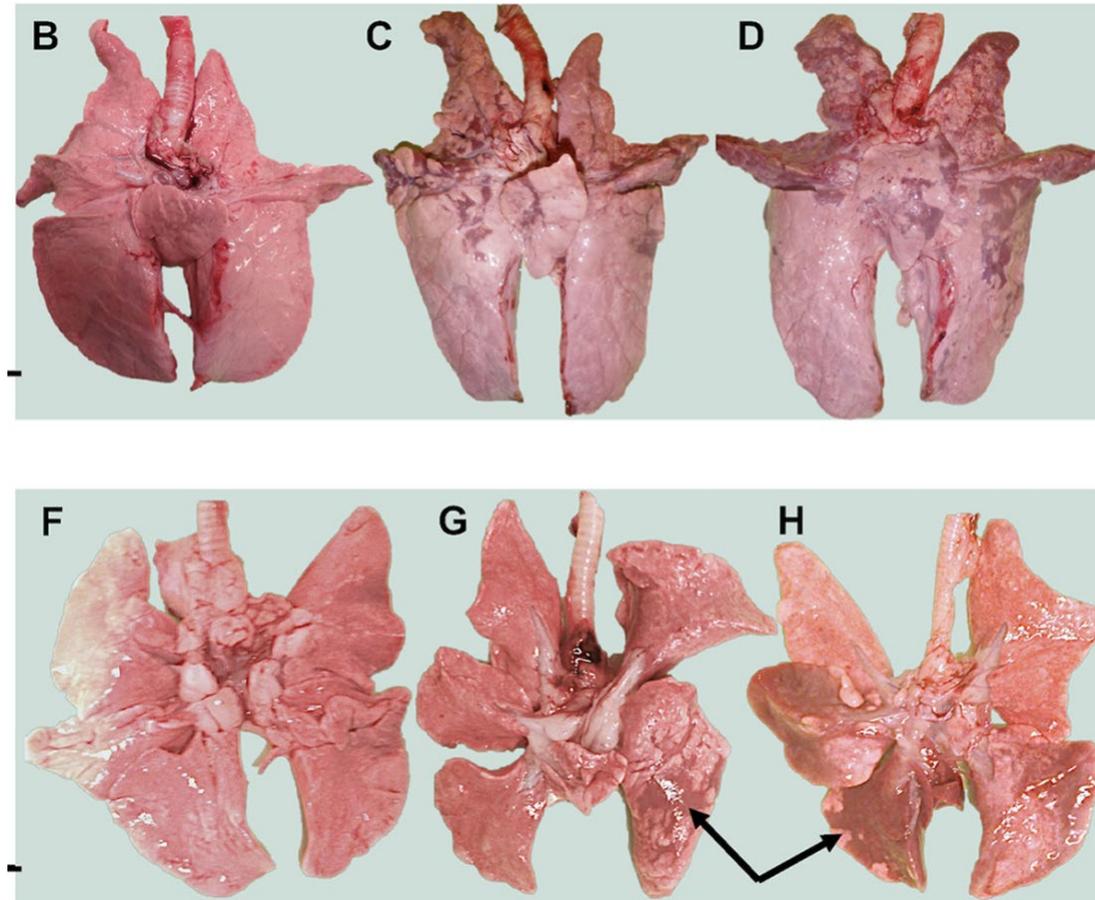
FIGURE 1 Lungs, pigs. (a) Multifocal, dark red depressed areas resembling a checkboard, mostly in the cranioventral areas of the lungs. Acute influenza A virus (IAV) infection. (b) Dark red consolidation of the ventral lung lobes. Subacute IAV infection with *Mycoplasma hyopneumoniae* (Mhyo) coinfection. (c) Acute necrotizing bronchiolitis with obliteration of the lumen by neutrophils and adjacent alveolar atelectasis, influenza A virus, H&E. (d) Positive signal for IAV nucleoprotein (NP) within nucleus and cytoplasm of epithelial cells of bronchioles in acute IAV infection. Immunohistochemistry for anti-8251 IAV antibody. (e) Necrotizing bronchial adenitis and bronchial epithelial necrosis with lumina partially filled with necrotic debris and degenerate neutrophils in IAV infection. H&E. (f) Subacute bronchiointerstitial pneumonia with lymphocytic peribronchiolitis and bronchiolar epithelial hyperplasia, influenza A virus, H&E

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Neumonía bronquiolo-intersticial



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Distribución generalizada
- Lesiones macroscópicas poco claras
- Falta de colapso pulmonar
- Patrón lobulillar
- Potencial edema intersticial

Lesiones microscópicas:

- Engrosamiento de septos alveolares
- Exudado inflamatorio
- Ocasionalmente proliferación de neumocitos de tipo II

Neumonía intersticial



Diseminación sistémica



Agentes causales más habituales:

- PRRSV
- ADV
- Otros virus sistémicos



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Neumonía intersticial

Lesiones

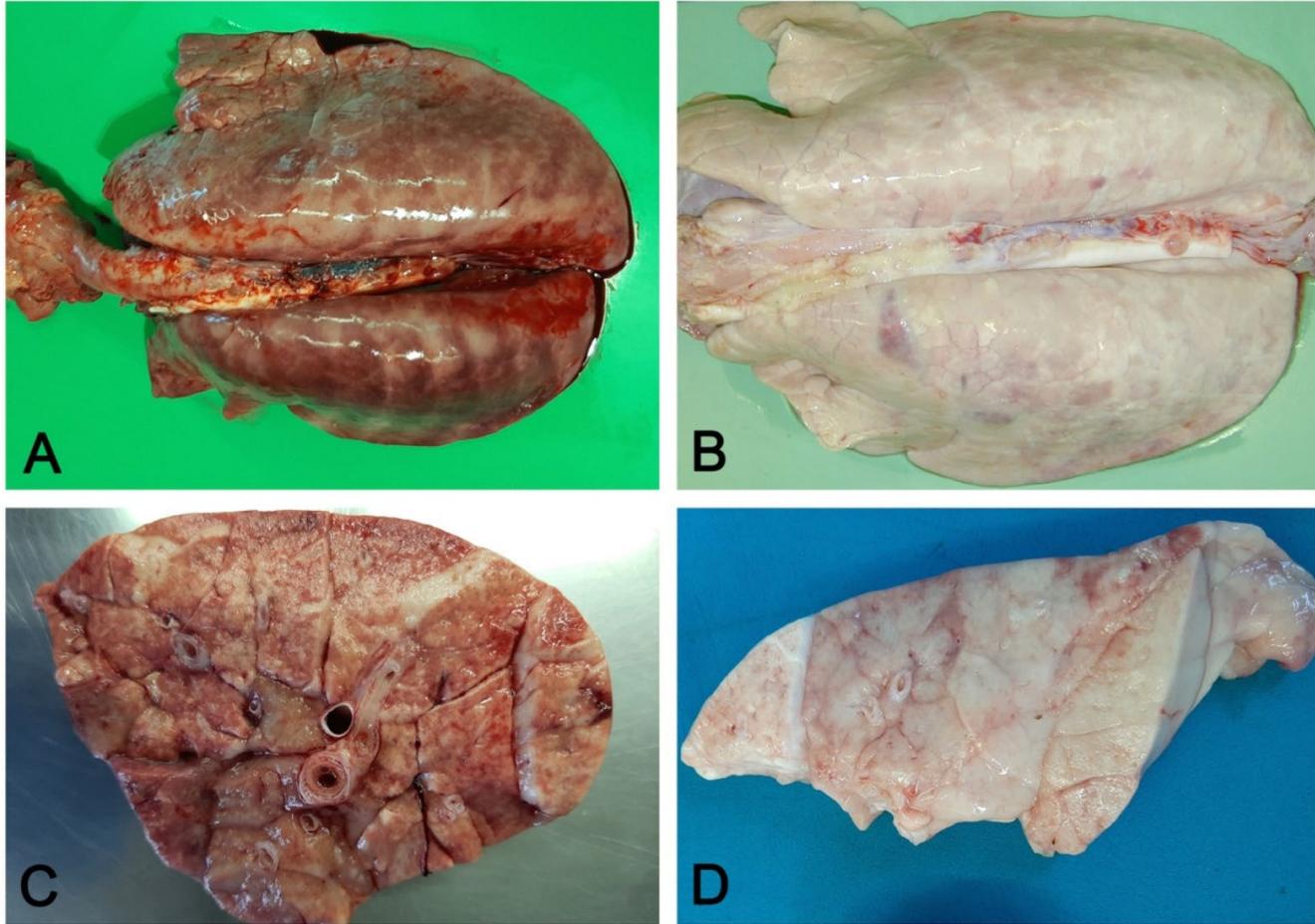


Figure 5. Interstitial pneumonia: non-collapsed lungs, with rib impressions (A), in acute (A) and chronic (B) interstitial pneumonia. Acute changes include diffuse hyperemia and edema of peribronchovascular connective tissue (C). In chronic stages, the lung is slightly increased in consistency and colored whitish due to fibrosis (D).

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Multifocal
- Focos de necrosis encapsulados
- Evolución a abscesos o granulomas

Lesiones microscópicas:

- Engrosamiento de septos alveolares
- Exudado inflamatorio
- Ocasionalmente proliferación de neumocitos de tipo II

Neumonía tromboembólica



Diseminación hematógena

Agentes causales más habituales:

- Bacterias piógenas
- Parásitos



Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Neumonía tromboembólica

Lesiones

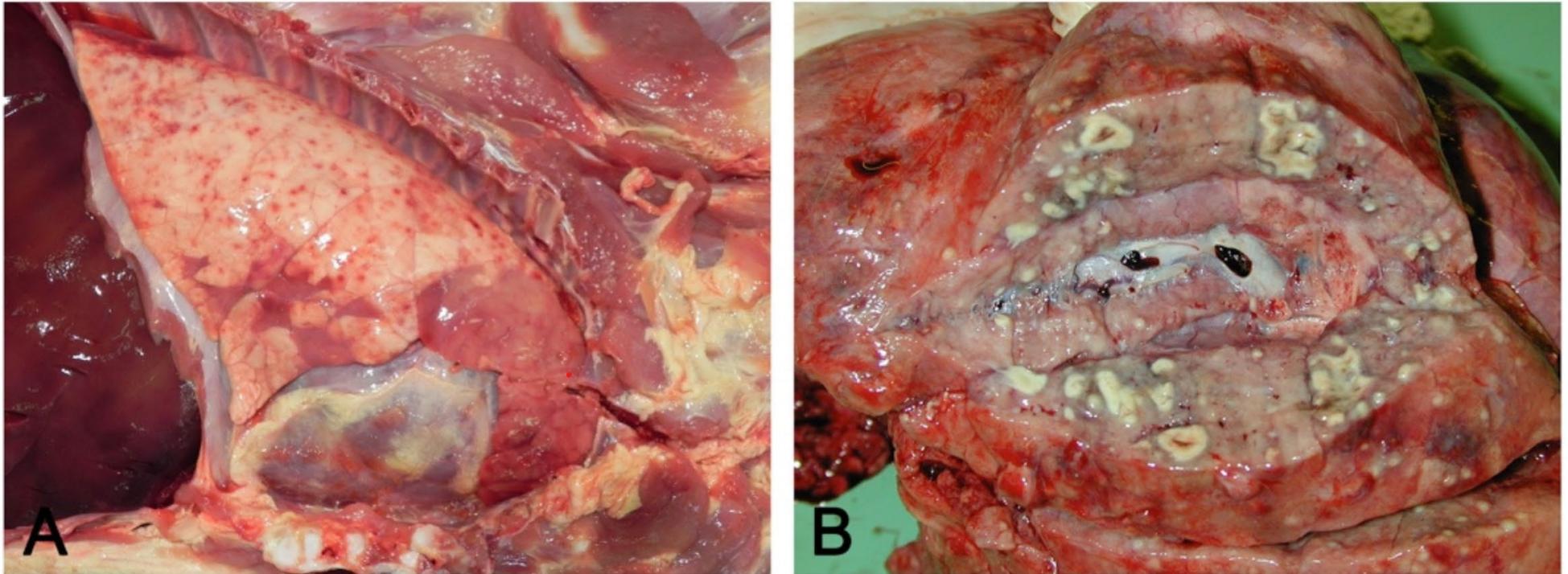


Figure 4. Hematogenous lung spread: in (A), together with a cranioventral pneumonia, the right basal lobe shows multiple acute and recent red foci of hyperemia/hemorrhage referable to bacteremia often of intestinal origin. In (B), multiple foci of suppuration surrounded by normal lung parenchyma, due to lung arrest of microthrombi originating from septic phlebitis outside the lungs.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Multifocal
- Módulos o granulomas
- En ocasiones también en otros órganos

Lesiones microscópicas:

- Microscópicamente, los nódulos contienen un centro necrótico infiltrado de macrófagos y células inflamatorias rodeado de tejido conjuntivo

Neumonía granulomatosa



Diseminación aerógena o hematógena



La lesión afecta a todos los lóbulos pulmonares y los linfonodos mediastínicos.

Agentes causales más habituales:

- **Micobacterias**
- **Parásitos**

<https://sesc.cat/es/neumonia-granulomatosa-de-causa-desconocida-en-un-cerdo/>

Aproximaciones al diagnóstico de las alteraciones respiratorias

Sospecha clínica

Lesiones

Observaciones macroscópicas:

- Variables

Lesiones microscópicas:

- Variables

Complejo Respiratorio Porcino



Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico



Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

In vivo

Hisopos nasales

- Gripe (momento toma de muestra)
- Rinitis atrófica

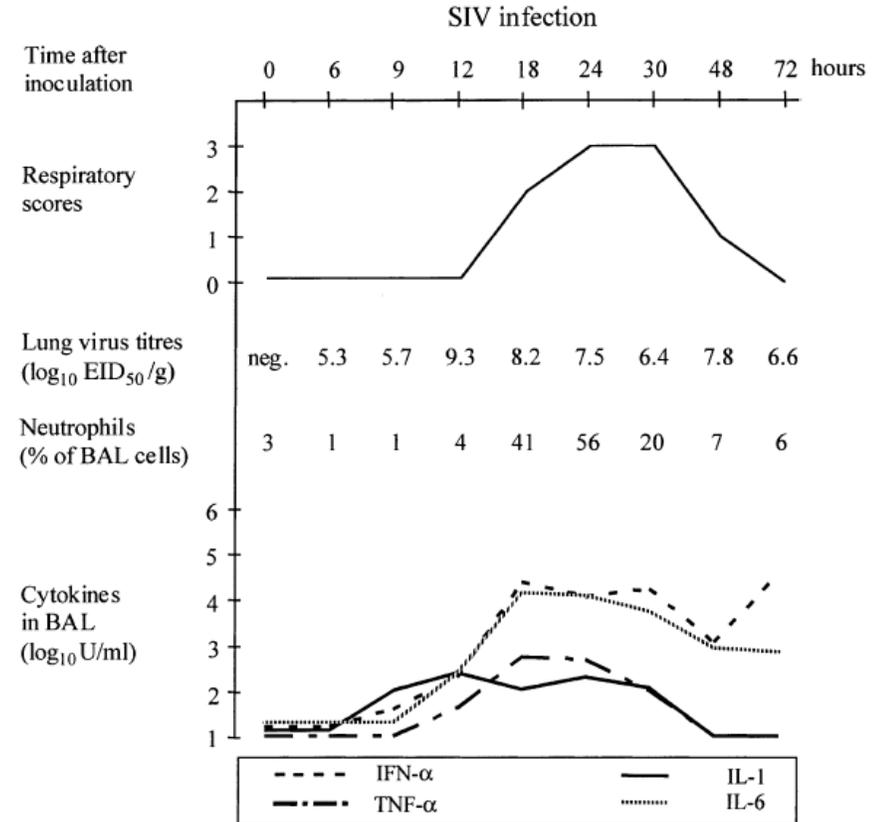
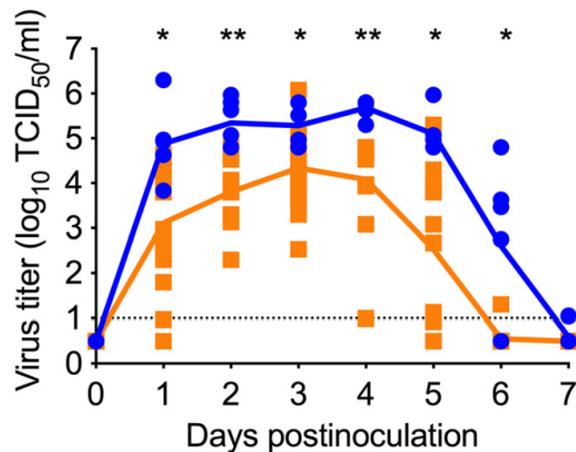


FIG 2 wtTX98 and lvTX98 excretion by intranasally inoculated pigs. Conventional influenza-naive pigs were intranasally inoculated with 6.3 log₁₀ TCID₅₀ of wtTX98 (blue circles, *n* = 6) or lvTX98 (orange squares, *n* = 16) in 3 mL of PBS, and nasal swabs were taken daily to evaluate virus titers in a CPE assay on MDCK cells. Log₁₀-transformed virus titers of individual pigs are shown by dots; lines indicate mean values of each group. Virus titers were compared between groups using 2-sided Mann-Whitney U tests. The black dotted line represents the detection limit. *, *P* < 0.05; **, *P* < 0.001.

Van Reeth et al. (2002). *Vet. Immunol Immunopathol.*, 87:161-168

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

In vivo

Hisopos o lavados traqueobronquiales o laríngeos



• *M. hyopneumoniae*

Aislamiento muy difícil

Table 1

Description of current *M. hyopneumoniae* diagnostic assays and relative diagnostic sensitivity by sample type. Relative diagnostic sensitivity is based on comparisons between sample types within the same assay type, from very low (-) to very high (++++). Only clinical specimens that have been compared in the literature are included, although more sample types may exist per assay type.

Diagnostic category	Assay	Principle	Sample	Relative diagnostic sensitivity	References
Detection of viable bacteria	Bacterial culture	Isolation of <i>M. hyopneumoniae</i>	Nasal swab	+	(Marois et al., 2007)
			Tonsillar swab	++	
			Lung tissue	+ / ++	
			Tracheobronchial swab	+++	
			Tracheobronchial lavage	+++	
Detection of bacterial antigens	Immunofluorescence	Detection of <i>M. hyopneumoniae</i> antigens using antibodies in tissue	Frozen lung tissue	No comparison available	
	Immunohistochemistry	Detection of <i>M. hyopneumoniae</i> antigens using antibodies in tissue	Fixed lung tissue		

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

• *M. hyopneumoniae*

In vivo

Table 1

Description of current *M. hyopneumoniae* diagnostic assays and relative diagnostic sensitivity by sample type. Relative diagnostic sensitivity is based on comparisons between sample types within the same assay type, from very low (-) to very high (++++). Only clinical specimens that have been compared in the literature are included, although more sample types may exist per assay type.

Diagnostic category	Assay	Principle	Sample	Relative diagnostic sensitivity	References	
Detection of bacterial nucleic acid	In situ hybridization	Detection of <i>M. hyopneumoniae</i> -specific genome regions using complementary probes in tissue	Fixed lung tissue	No comparison available		
			Standard PCR	Multiple sample type	No comparison available	
				Nasal swab	+	(Kurth et al., 2002; Sibila et al., 2004; Marois et al., 2007, 2010; Fablet et al., 2010)
	Nested PCR	Two primer specific amplifications of <i>M. hyopneumoniae</i> nucleic acid, using internal specific primers complementary to the first amplification nucleotide sequence	Oro-pharyngeal swab	++	(Fablet et al., 2010)	
			Tonsillar swab	++	(Sibila et al., 2004; Marois et al., 2007, 2010)	
			Lung tissue	++	(Kurth et al., 2002)	
			Tracheobronchial swab	+++	(Kurth et al., 2002; Marois et al., 2007, 2010; Fablet et al., 2010)	
			Tracheobronchial lavage	+++	(Kurth et al., 2002; Marois et al., 2007; Fablet et al., 2010)	
			Bronchial swab	+++	(Sibila et al., 2004)	
			Oral fluids	-/+	(Pieters et al., 2017)	
			Nasal swab	+	(Marois et al., 2010; Pieters et al., 2017)	
			Tonsillar swab	++	(Marois et al., 2010)	
			Tracheobronchial lavage	++	(Pieters et al., 2017)	
			Lung tissue	++	(Marois et al., 2010)	
			Laryngeal swab	+++	(Sievers et al., 2015; Pieters et al., 2017; Betlach et al., 2020; Sponheim et al., 2020)	
			Tracheobronchial swab	+ / ++++	(Marois et al., 2010; Sievers et al., 2015)	
			Deep-tracheal catheter	+ / ++++	(Betlach et al., 2020; Sponheim et al., 2020)	
			Bronchial swab	++++	(Sievers et al., 2015; Betlach et al., 2020)	

Hisopos o lavados traqueobronquiales o laríngeos



Técnicas moleculares

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

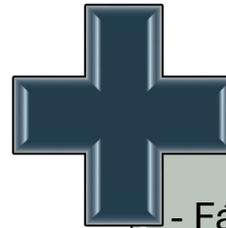
In vivo

Sangre

Fluidos orales

Patógenos
sistémicos

Serología



- Fáciles de obtener en animales en crecimiento
- Permite realizar el seguimiento en poblaciones
- Menor coste

- Peor sensibilidad para algunos patógenos
- Muestra agregada, no individual (no válida para aislamiento)
- Carga del patógeno generalmente baja (no válida para secuenciación)
- Pueden no representar tipos más patógenos (APP)

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

In vivo

Caracterización de serotipos de APP

Fluidos orales vs pulmón

Table 5 Frequencies of detection of *A. pleuropneumoniae* serotypes found in clinical and subclinical animals

Serotype	Lungs (n = 712)			Oral fluids (n = 171)		
	n	Percentage (%)	CI 95%	n	%	CI 95%
1	4	0.6	0.15–1.43	29	16.96	11.66–23.44
2	157	22	19.06–25.28	58	33.92	26.87–41.54
3	1	0.1	0–0.78	6	3.51	1.30–7.48
4	80	11.2	9.01–13.79	41	23.98	17.79–31.09
5	56	7.9	6.0–10.09	9	5.26	2.43–9.76
6	11	1.5	0.77–2.75	10	5.85	2.84–10.49
7	15	2.1	1.18–3.45	73	42.69	35.17–50.47
8	26	3.6	2.40–5.31	19	11.11	6.82–16.81
9/11	200	28.1	24.81–31.55	29	16.96	11.66–23.44
10	3	0.4	0.09–1.23	12	7.02	3.68–11.94
12	5	0.7	0.23–1.63	42	24.56	18.31–31.72
13	90	12.6	10.29–15.31	39	22.81	16.75–29.83
14	0	0	0–0.52	19	11.11	6.82–16.81
15	0	0	0–0.52	1	0.58	0.01–3.21
16	0	0	0–0.52	0	0	0–2.13
17	84	11.8	9.52–14.40	29	16.96	11.66–23.44
18	13	1.8	0.98–3.10	21	12.28	7.77–18.16
19	0	0	0–0.52	16	9.36	5.44–14.75
NT ^a	2	0.3	0.03–1.01	2	1.16	0.14–4.16

^a Non-typeable.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

Toma de muestras

In vivo

Serología



- Permite realizar el seguimiento en poblaciones
- Permite determinar posibles interferencias con anticuerpos maternos en programas de vacunación
- Con algunos patógenos permite diferenciar vacunación y exposición (APP)
- Permite garantizar el estatus sanitario de las cerdas de renovación

- Detección más tardía
- No permite caracterizar al agente etiológico (antibiograma)
- A veces pueden existir reacciones cruzadas (ej. subtipos del virus de la gripe)
- La seroconversión es variable

Aproximaciones al diagnóstico de las alteraciones respiratorias

Diagnóstico etiológico

In vivo

Serología

Dinámica infección *M. hyopneumoniae*

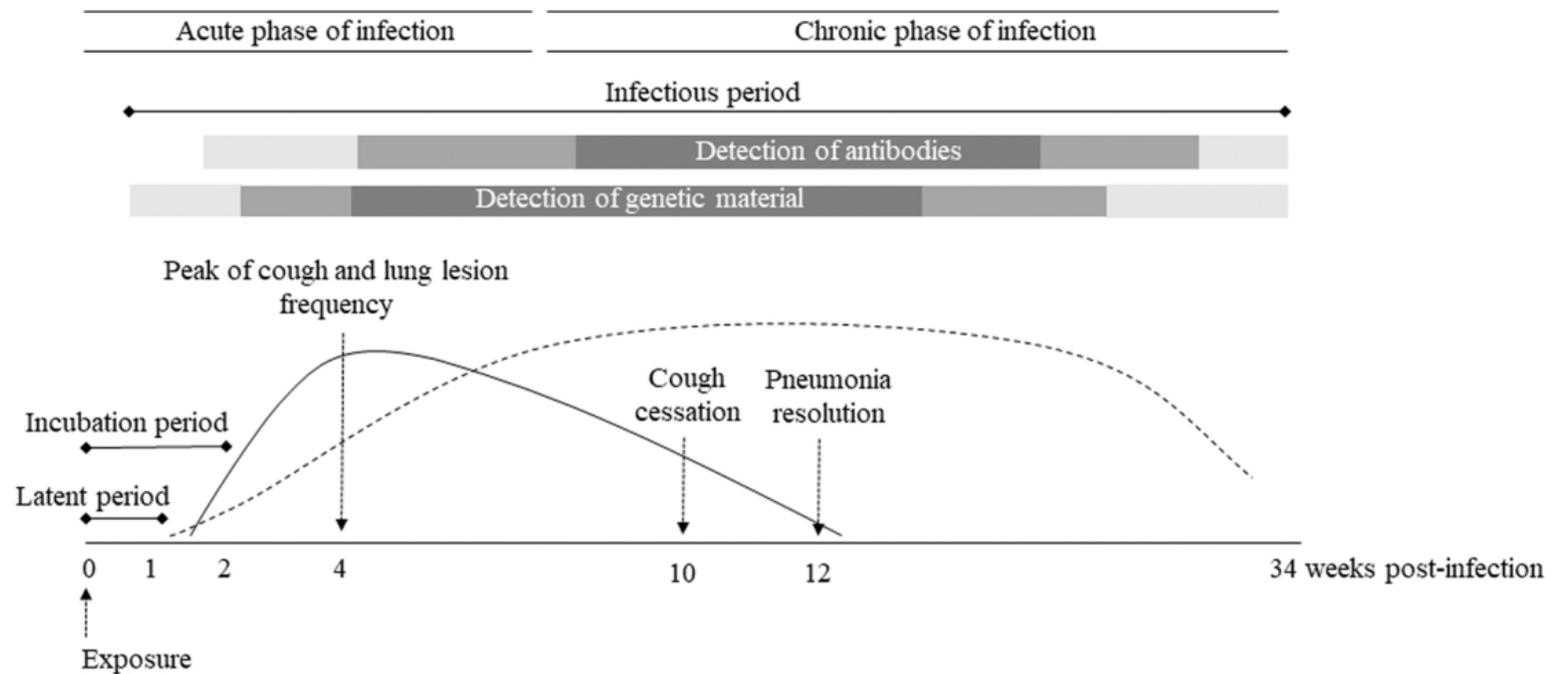


Fig. 1. Schematic overview of coughing, gross lung lesions and antibody dynamics, and detection of *M. hyopneumoniae* by PCR in clinical samples of pigs after experimental infection. Latent period: time interval between infection and infectiousness (to transmission/infection of other pigs). Incubation period: time elapsed between infection and appearance of clinical signs (onset of disease). The solid line represents coughing and lung lesion dynamics, whereas the dotted line depicts *M. hyopneumoniae*-specific antibody dynamics. Gray shading symbolizes the likelihood of genetic material or antibody detection for *M. hyopneumoniae*, with darkest shade having the highest likelihood. Onset and duration of each parameter is proposed based on summarized information described in the literature.

Aproximaciones al diagnóstico de las alteraciones respiratorias

Vigilancia epidemiológica *M. hyopneumoniae* cerdas de renovación

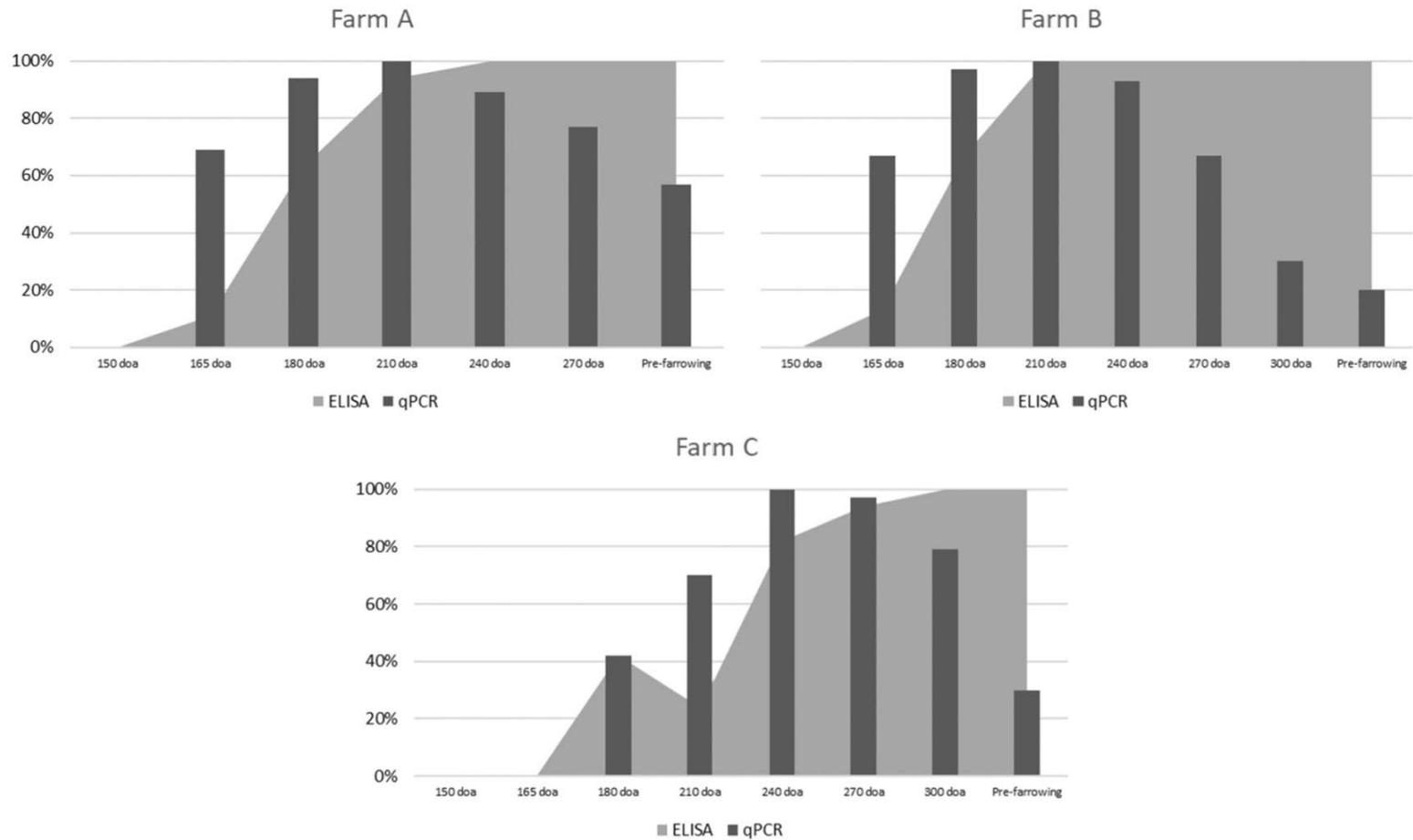


Fig. 1. Percentage of *M. hyopneumoniae* positivity in the ELISA and PCR assays of negative replacement gilts housed in three *M. hyopneumoniae* positive farms (A, B, and C). DOA = days of age.

Diagnóstico gripe: caracterización de subtipos

Caracterización molecular de subtipos o serotipos

Diagnóstico gripe: caracterización de subtipos

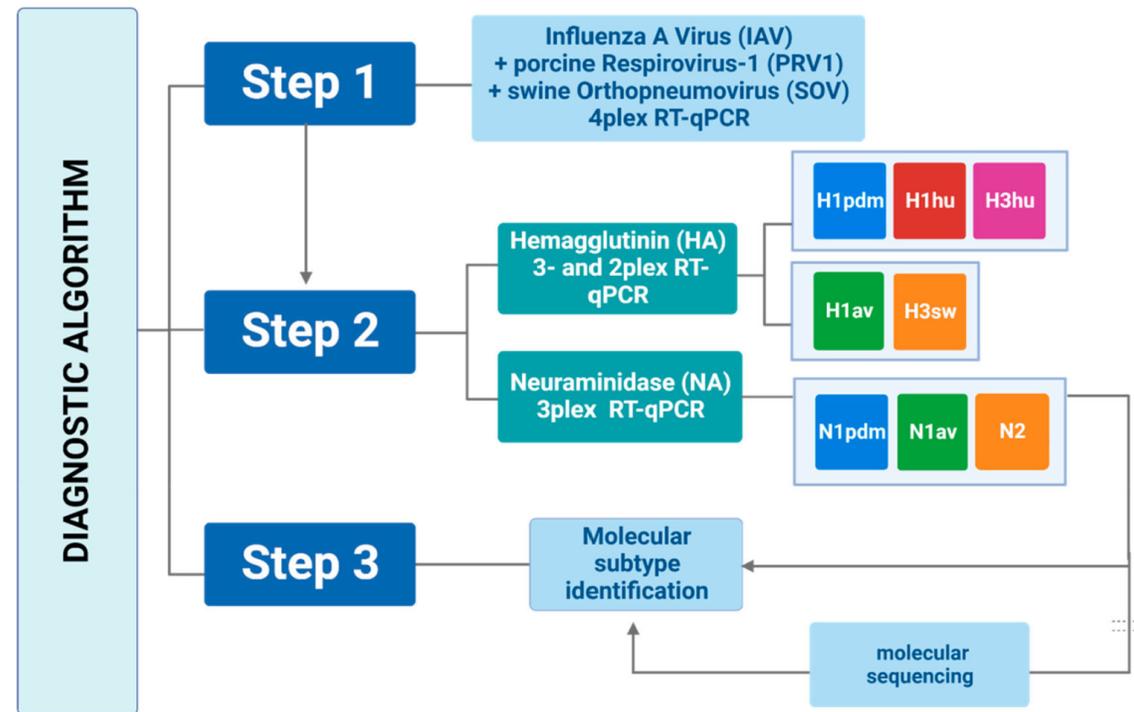


Figure 1. Diagnostic algorithm based on one-step multiplex RT-qPCRs for detection and subtyping of swine influenza A viruses (swIAV) as well as the detection of porcine respirovirus 1 and swine orthopneumovirus circulating in European pig populations. Step 1 depicts a tetraplex RT-qPCR, targeting the M-gene segment of (sw)IAV, the F-gene of PRV1 and the NP-gene of SOV; an internal control (IC2) is essentially included in this tetraplex RT-qPCR (not shown). In step 2, subtyping for IAV RNA-positive samples is attempted employing the one-step duplex- and triplex HA- and the triplex NA-specific RT-qPCRs developed in this study. Step 3 is only required in case if HA or NA subtype/lineage cannot be assigned by the shown RT-qPCRs: HA and/or NA amplicons need to be generated by conventional one-step RT-PCR for Sanger amplicon or minION sequencing and BLAST searches or swine H1 clade classification by Anderson, Macken [57] on the Influenza Research Database (IRD) to finalize subtyping of swIAV.

Diagnóstico gripe: caracterización de subtipos

Caracterización molecular de subtipos o serotipos

Caracterización de serotipos de APP

Schuwerk et al. *Vet Res* (2021) 52:10
<https://doi.org/10.1186/s13567-020-00890-x>



RESEARCH ARTICLE

Open Access



Sero- and *apx*-typing of German *Actinobacillus pleuropneumoniae* field isolates from 2010 to 2019 reveals a predominance of serovar 2 with regular *apx*-profile

Lukas Schuwerk^{1,2*}, Doris Hoeltig³, Karl-Heinz Waldmann³, Peter Valentin-Weigand¹ and Judith Rohde¹

Abstract

Serotyping is the most common method to characterize field isolates of *Actinobacillus (A.) pleuropneumoniae*, the etiological agent of porcine pleuropneumonia. Based on serology, many farms seem to be infected and antibodies against a wide variety of serovars are detectable, but, so far it is unknown to what degree respective serovars contribute to outbreaks of clinical manifest disease. In this study, 213 German *A. pleuropneumoniae* field isolates retrieved for diagnostic purposes from outbreaks of porcine pleuropneumonia between 2010 and 2019 were genetically serotyped and analyzed regarding their *apx*-toxin gene profile using molecular methods. Serotyping revealed a prominent role of serovar 2 in clinical cases (64% of all isolates) and an increase in the detection of this serovar since 2010 in German isolates. Serovar 9/11 followed as the second most frequent serovar with about 15% of the isolates. Furthermore, very recently described serovars 16 (n = 2) and 18 (n = 8) were detected. Most isolates (93.4%) showed *apx*-profiles typical for the respective serovar. However, this does not hold true for isolates of serovar 18, as 75% (n = 6) of all isolates of this serovar deviated uniformly from the "typical" *apx*-gene profile of the reference strain 7311555. Notably, isolates from systemic lesions such as joints or meninges did not harbor the complete *apx/CABD* operon which is considered typical for highly virulent strains. Furthermore, the extremely low occurrence (n = 1) of NAD independent (biovar II) isolates in German *A. pleuropneumoniae* was evident in our collection of clinical isolates.

Keywords: *Actinobacillus pleuropneumoniae*, Serotyping, *apx*, Virulence, Atypical, Biovar, Systemic

Diagnóstico gripe: caracterización de subtipos

Caracterización molecular de subtipos o serotipos

Caracterización de serotipos de APP

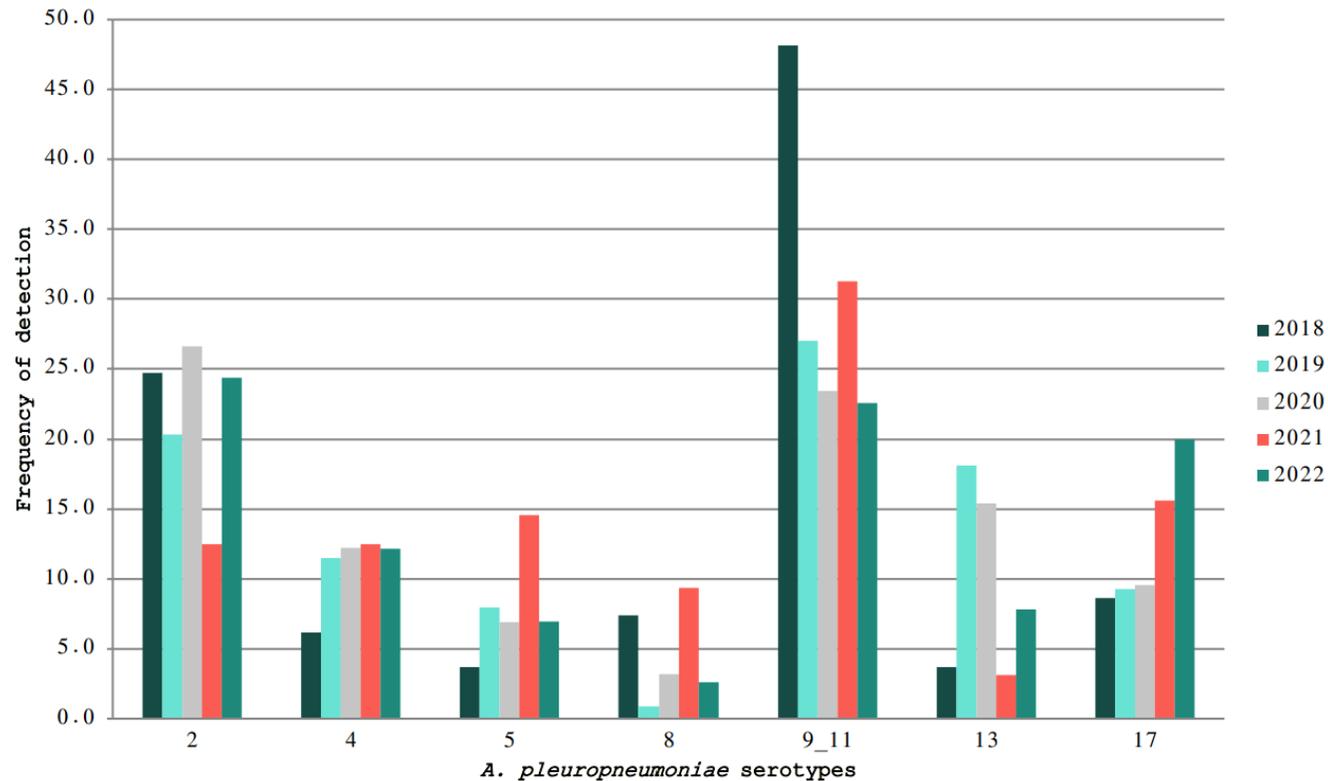
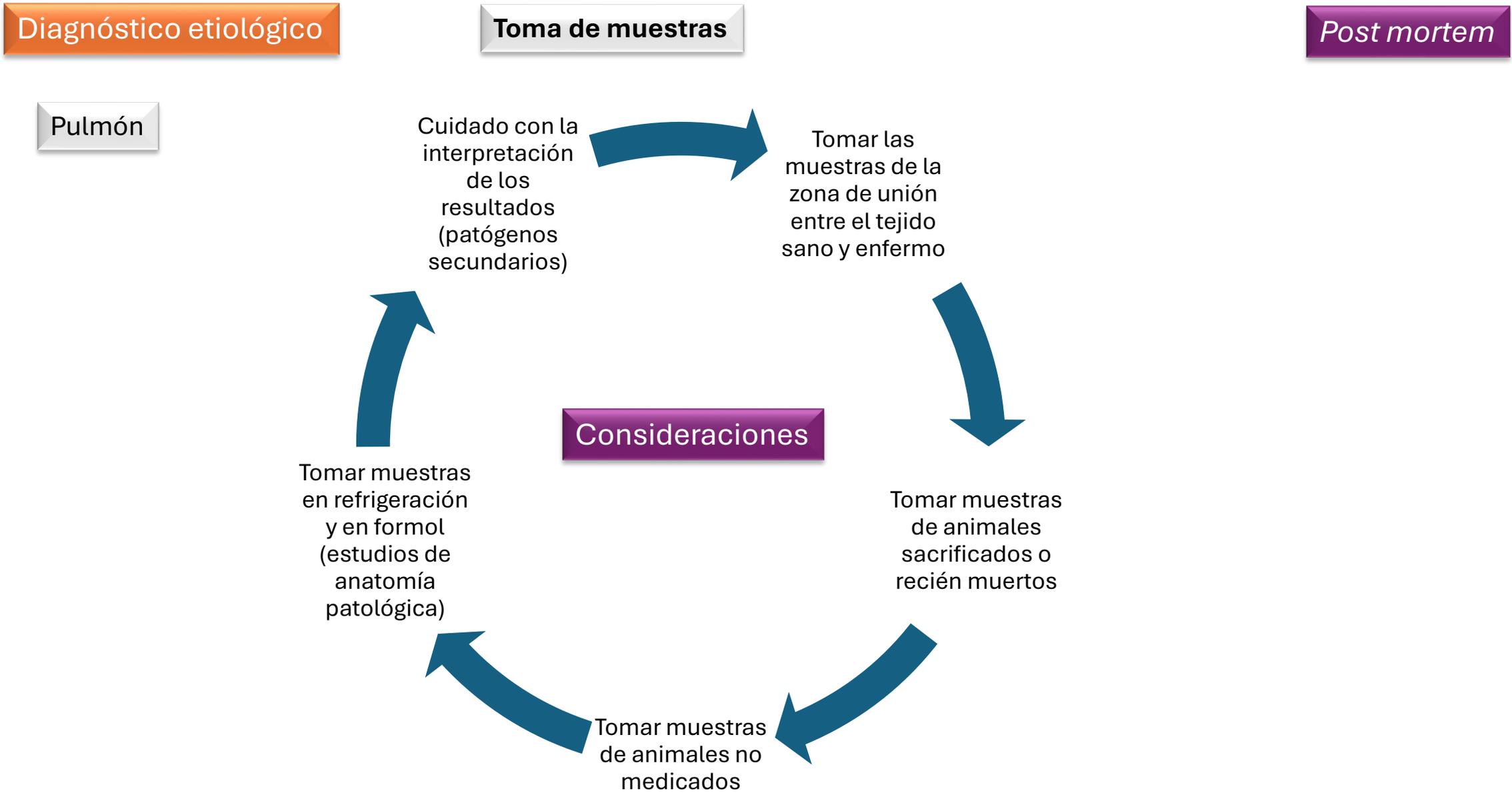


Figure 1 Evolution of detection rates of the most frequently detected *A. pleuropneumoniae* serotypes during 2018–2022.

Aproximaciones al diagnóstico de las alteraciones respiratorias



Vigilancia epidemiológica en el matadero

Valoración de distintos lotes de animales de cebo

Neumonía

Distintos métodos

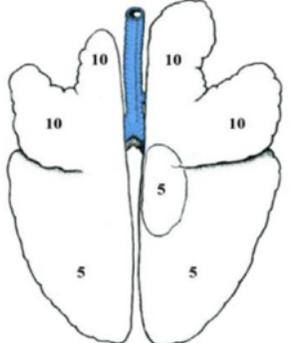
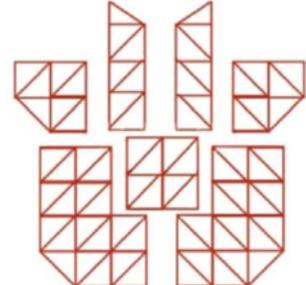
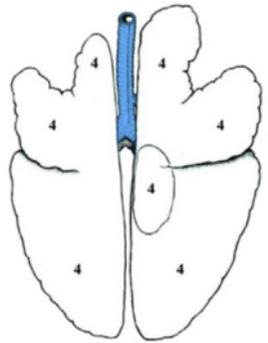
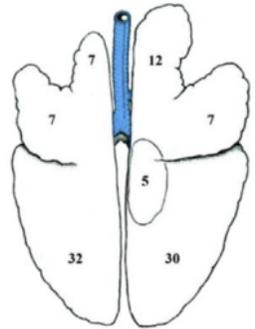
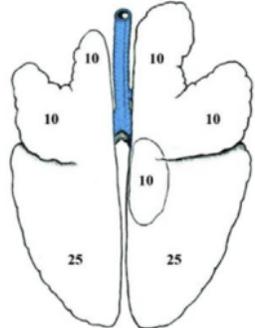
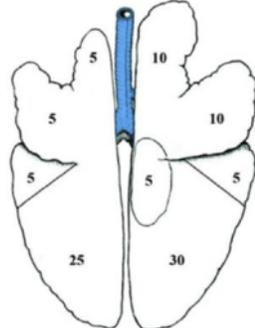
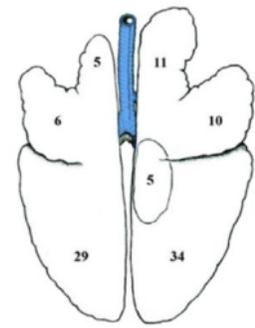
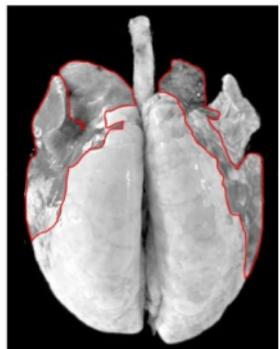
Goodwin et al. [55]	Hannan et al. [56]	Madec and Kobisch [57]	Morrison et al. [59]
Two-dimensional Score: 0-55	Two-dimensional Score: 0-35	Two-dimensional Score: 0-28	Three-dimensional Score: 0-100
			
Straw et al. [58]	Christensen et al. [60]	Reference method [61]	Sibila et al. [27]
Two-dimensional Score: 0-100	Three-dimensional Score: 0-100	Three-dimensional Score: 0-100	Two-dimensional Score: 0-100
			

Figure 1 Commonly used methods to score cranioventral pulmonary consolidation in slaughter pigs (adapted from [49]).

Vigilancia epidemiológica en el matadero

Valoración de distintos lotes de animales de cebo

Pleuritis

SPES

Table 3 Overview of pleurisy scoring systems in slaughter pigs^a

Score	Scoring system				
	Madec and Kobisch [57]	CTPA ^b	Pointon et al. [66]	SPES ^c	
0	No pleurisy				
1	Interlobular pleurisy (visceral pleurisy)	Fibrinous pleurisy	Interlobular pleurisy (adhesions between lung lobes)	1 N: pleurisy with normal lungs 1P: pleurisy with pneumonic lungs	Cranioventral lesion: interlobar pleurisy or at ventral border of caudal lobes
2	Localized pleurisy < 2 cm diameter	Extended pleurisy: lungs cannot be removed from the carcass	Pleurisy (adhesions of lungs to chest wall)	2 N: pleurisy with normal lungs 2P: pleurisy with pneumonic lungs	Dorsocaudal monolateral focal lesion
3	Extensive pleurisy > 2 cm diameter with adhesions to ribcage	–	–	–	Bilateral lesion of type 2 or extended monolateral lesion (at least one third of one diaphragmatic lobe)
4	Partial or total ribcage condemnation	–	–	–	Severely extended bilateral lesion (at least one third of both diaphragmatic lobes)

^a The Danish system [41] is explained in the text but not listed in the table, as it differs significantly from the other systems

^b CTPA System by the Centre Technique de Productions Animales[54]

^c SPES Slaughterhouse Pleurisy Evaluation System[67]

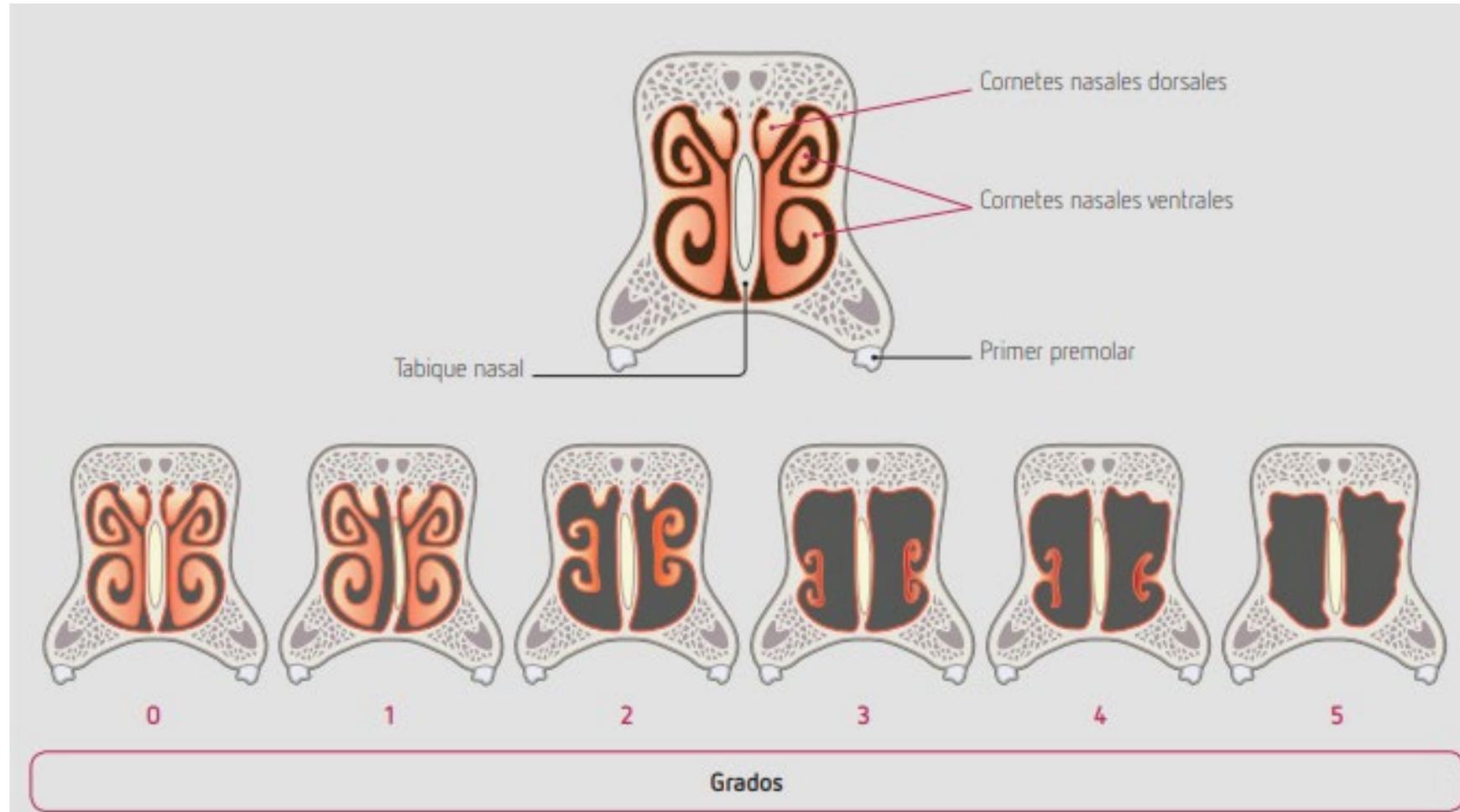
Vigilancia epidemiológica en el matadero

Valoración de distintos lotes de animales de cebo

Rinitis atrófica

Valoración de cornetes nasales

Corte a la altura del 2º premolar (comisura labial)



Vigilancia epidemiológica en el matadero

Valoración de distintos lotes de animales de cebo

Rinitis atrófica

Valoración de
cornetes nasales

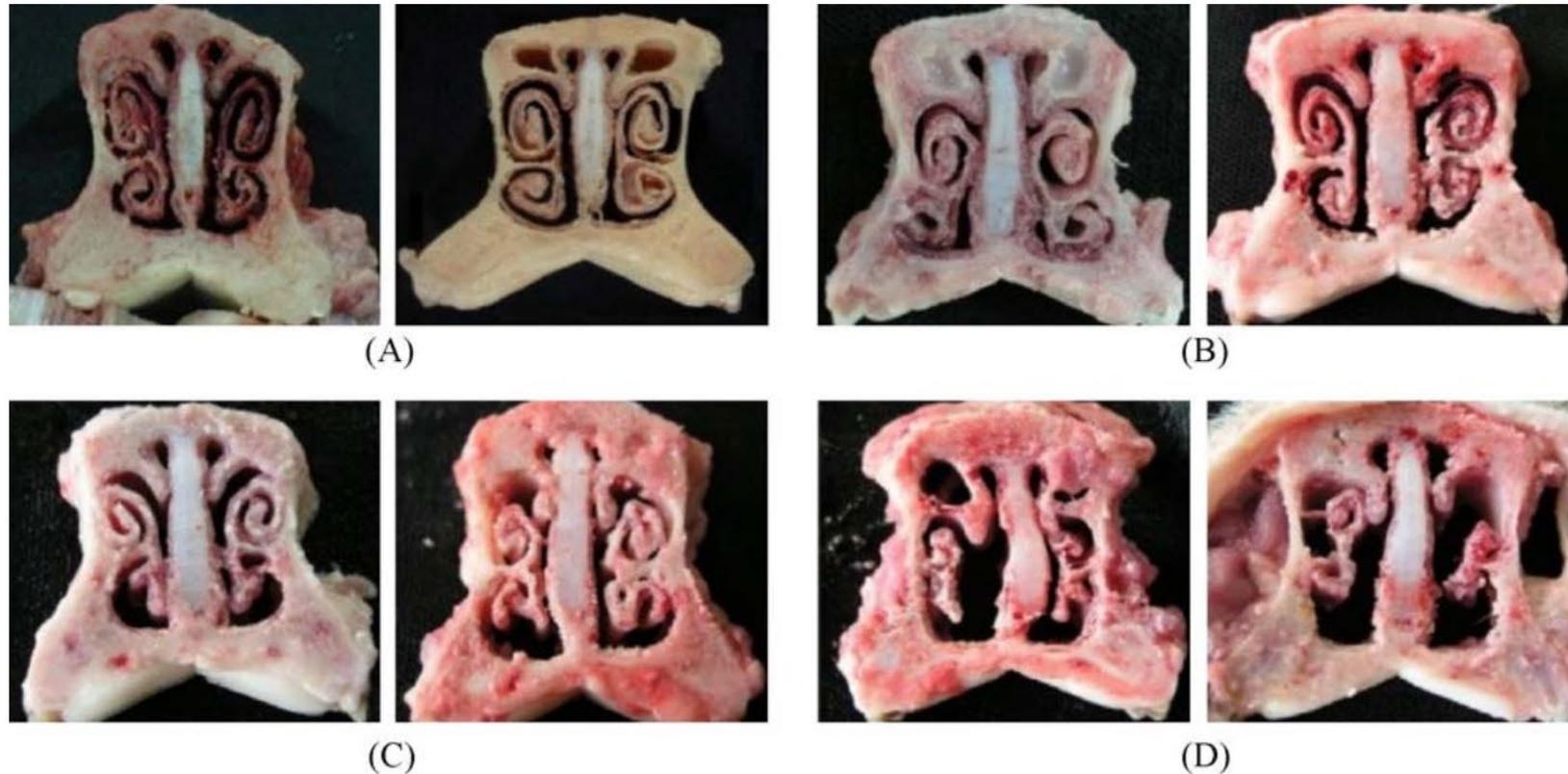


Fig. 3. Representative photographs of the turbinate conchae of experimental pigs in groups vaccinated with rsPMTs vaccine (B), conventional AR-toxoid vaccine (C), and unvaccinated (D), at 2-weeks after authentic PMT challenge. The unvaccinated and unchallenged pigs were served as the negative control (A).

***Muchas gracias por su
atención***