Pig digest: PCV and PEDV

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PCV2

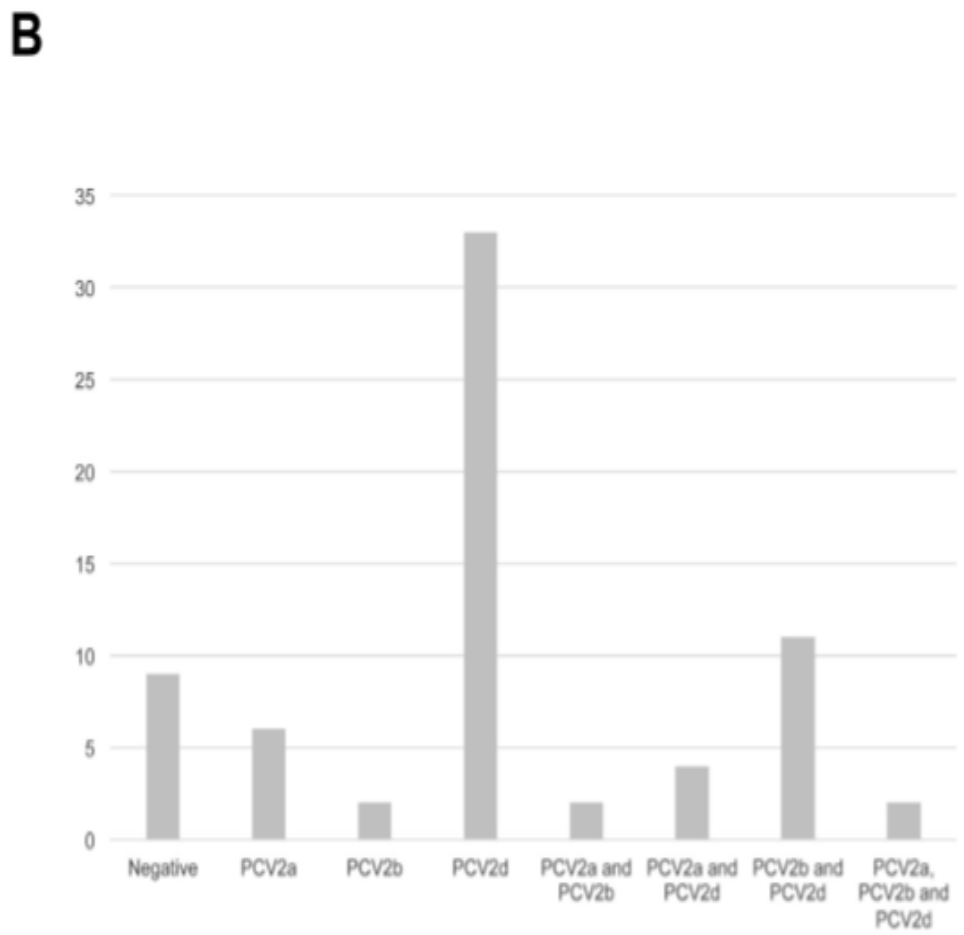
- Epidemiology change
- Genotype shift
- Former vaccine still work??

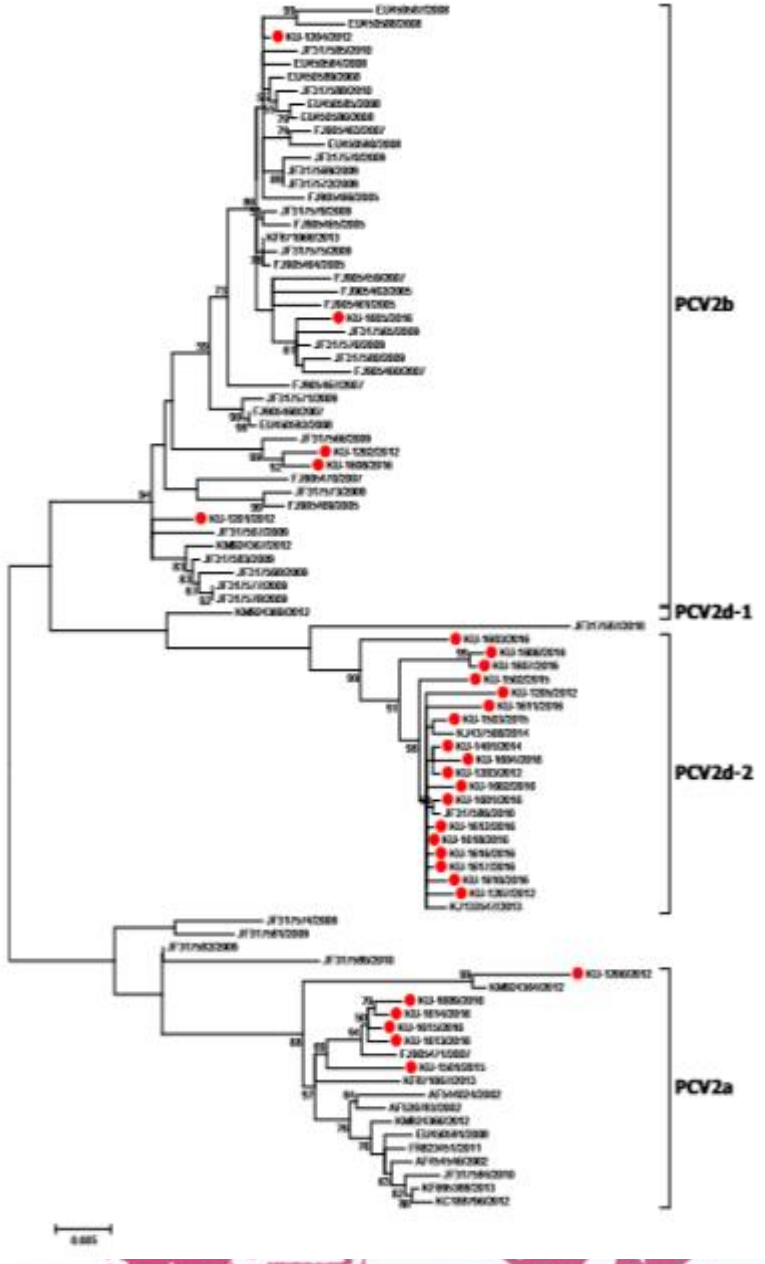
Epidemiology change

- Old epidemiology >> growth
- eliminating the effects of the virus on growth
- New epidemiology
- breeding stock
- probability of infection during gestation (mainly of gilts)
- viremic-born piglets

Genotype shift

T. Kwon et al. / Virus Research 228 (2017) 24–29





Korea

Genotype shift

Table 1
Annual report of PCV2 genotypes classified by the signature motifs and topology of ORF2 on the phylogenetic tree.

year	Positive for ORF2 specific primers	PCV2a	PCV2b	PCV2b_IM1	PCV2d
2009	6	1 (16.6%)	4 (66.6%)	–	1 (16.6%)
2010	18	5 (27.8%)	6 (33.3%)	6 (33.3%)	1 (5.6%)
2011	7	–	5 (71.4%)	1 (14.3%)	1 (14.3%)
2012	35	1 (2.8%)	16 (45.7%)	3 (8.6%)	15 (42.9%)
2013	24	–	8 (33.33%)	2 (8.33%)	14 (58.33%)
2014	23	–	1 (4.3%)	2 (8.7%)	20 (87%)
2015	22	–	–	–	22 (100%)
Total	135	7 (5.19%)	40 (29.63%)	14 (10.37%)	74 (54.81%)

Thailand

Thangthamniyom, et al. 2017

Vaccine efficiency

Vaccine 35 (2017) 248–254

Contents lists available at ScienceDirect

Vaccine

journal homepage: www.elsevier.com/locate/vaccine

A commercial porcine circovirus (PCV) type 2a-based vaccine reduces PCV2d viremia and shedding and prevents PCV2d transmission to naïve pigs under experimental conditions

Tanja Opriessnig[a,b,*], Chao-Ting Xiao[b,c], Patrick G. Halbur[b], Priscilla F. Gerber[a], Shannon R. Matzinger[d], Xiang-Jin Meng[d]

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Vaccine efficiency

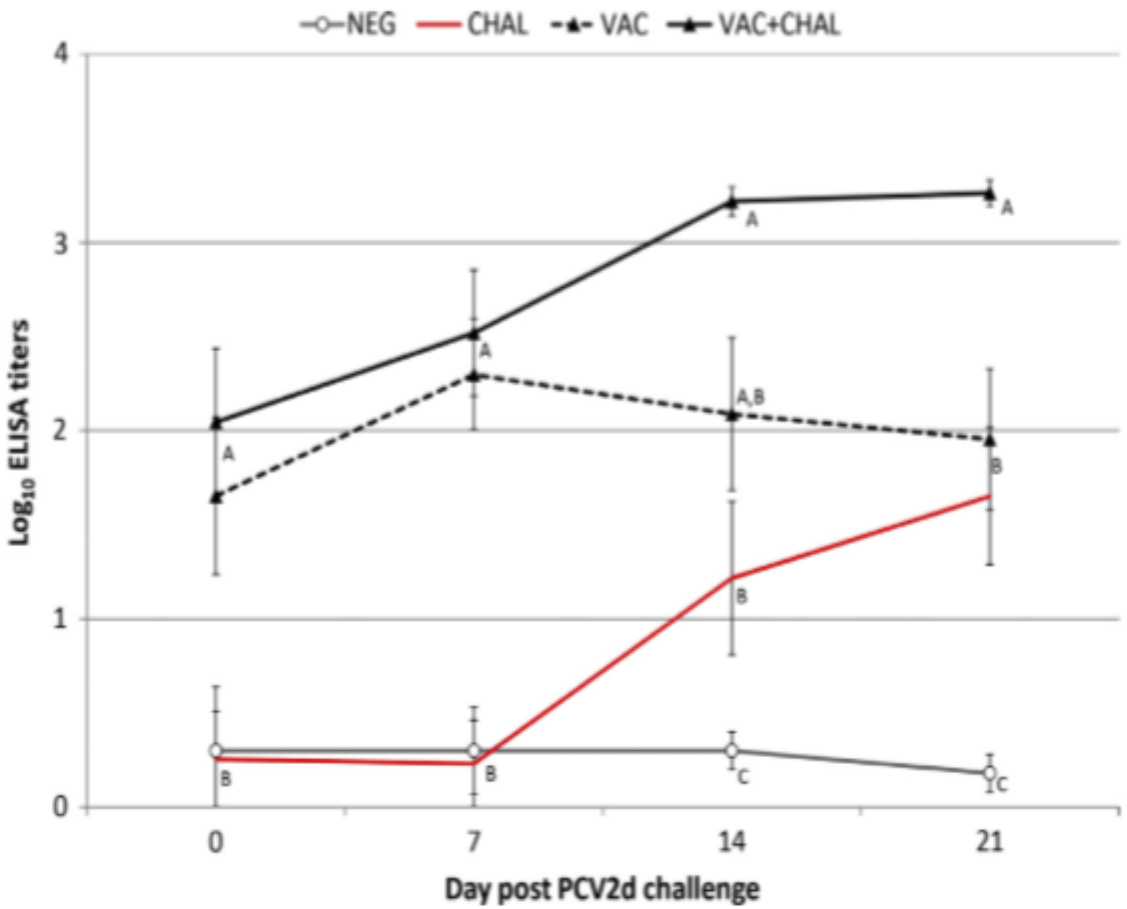
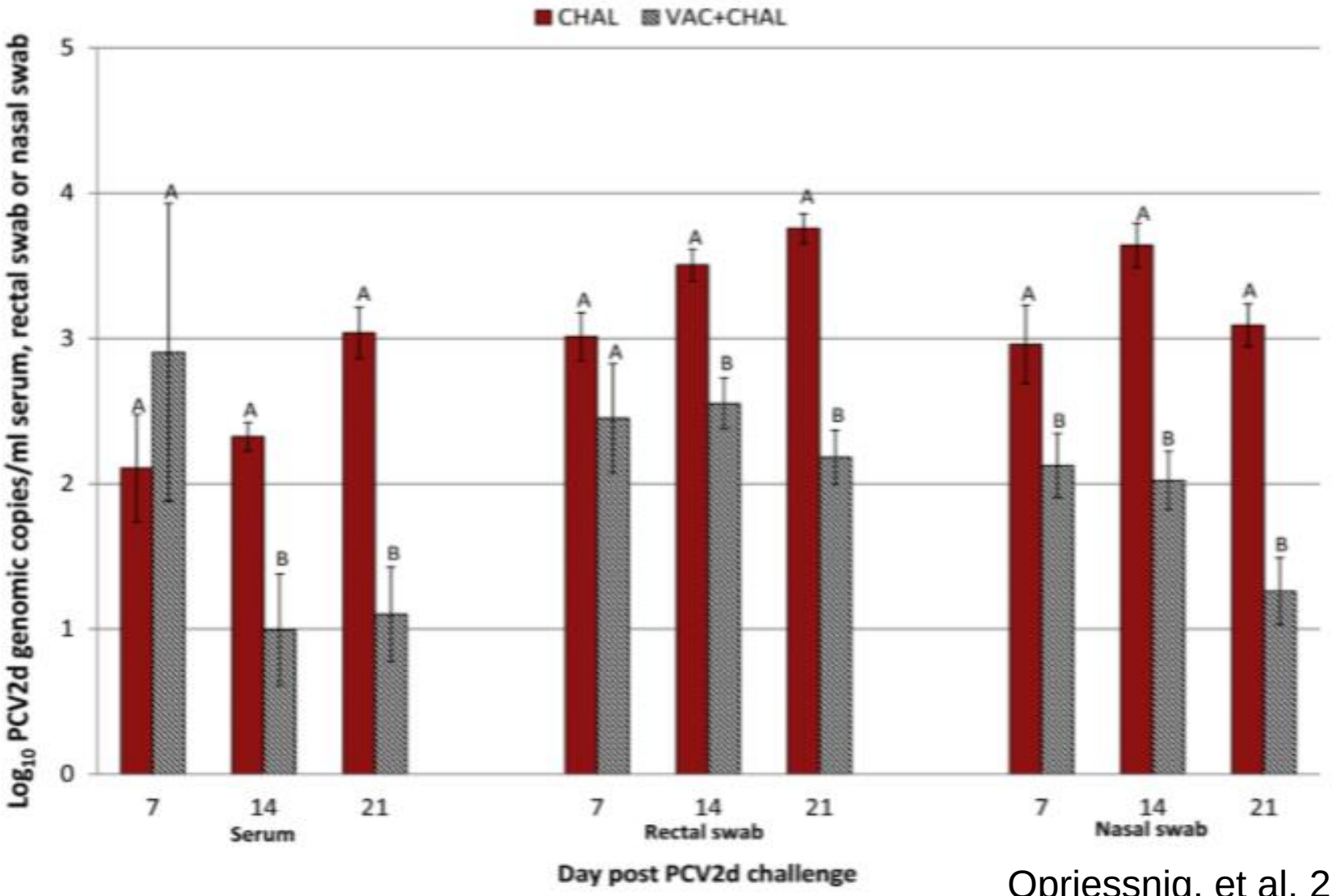


Fig. 2. Anti-PCV2 IgG response. Pigs were vaccinated against PCV2 at 3 weeks of age (dpv 0 or dpc −28) and challenged with PCV2d at 7 weeks of age (dpv 28 or dpc 0). Data presented as mean group $\log_{10}$ ELISA titer ± SEM. Group means include positive and negative pigs. Significantly different values for a dpc are indicated by different superscripts. The significance level was set to $P > 0.05$.

Opriessnig, et al. 2017

Vaccine efficiency



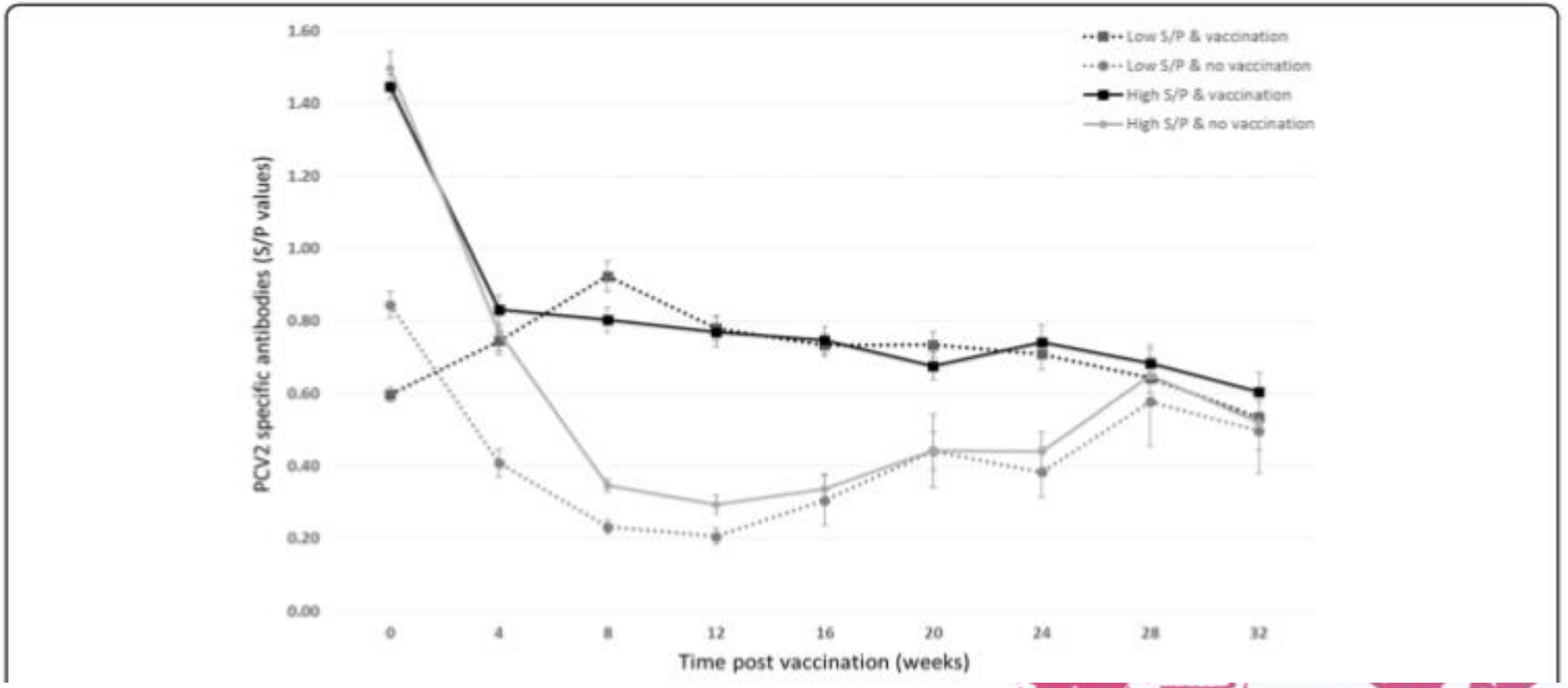
Opriessnig, et al. 2017

Vaccination timing

RESEARCH ARTICLE Open Access

Impact of maternally derived immunity on piglets' immune response and protection against porcine circovirus type 2 (PCV2) after vaccination against PCV2 at different age

Paolo Martelli[1*], Roberta Saleri[1], Giulia Ferrarini[1], Elena De Angelis[1], Valeria Cavalli[1], Michele Benetti[1], Luca Ferrari[1], Elena Canelli[1], Paolo Bonilauri[2], Elena Arioli[3], Antonio Caleffi[3], Heiko Nathues[4] and Paolo Borghetti[1]



Martelli, et al., 2016

Vaccination timing

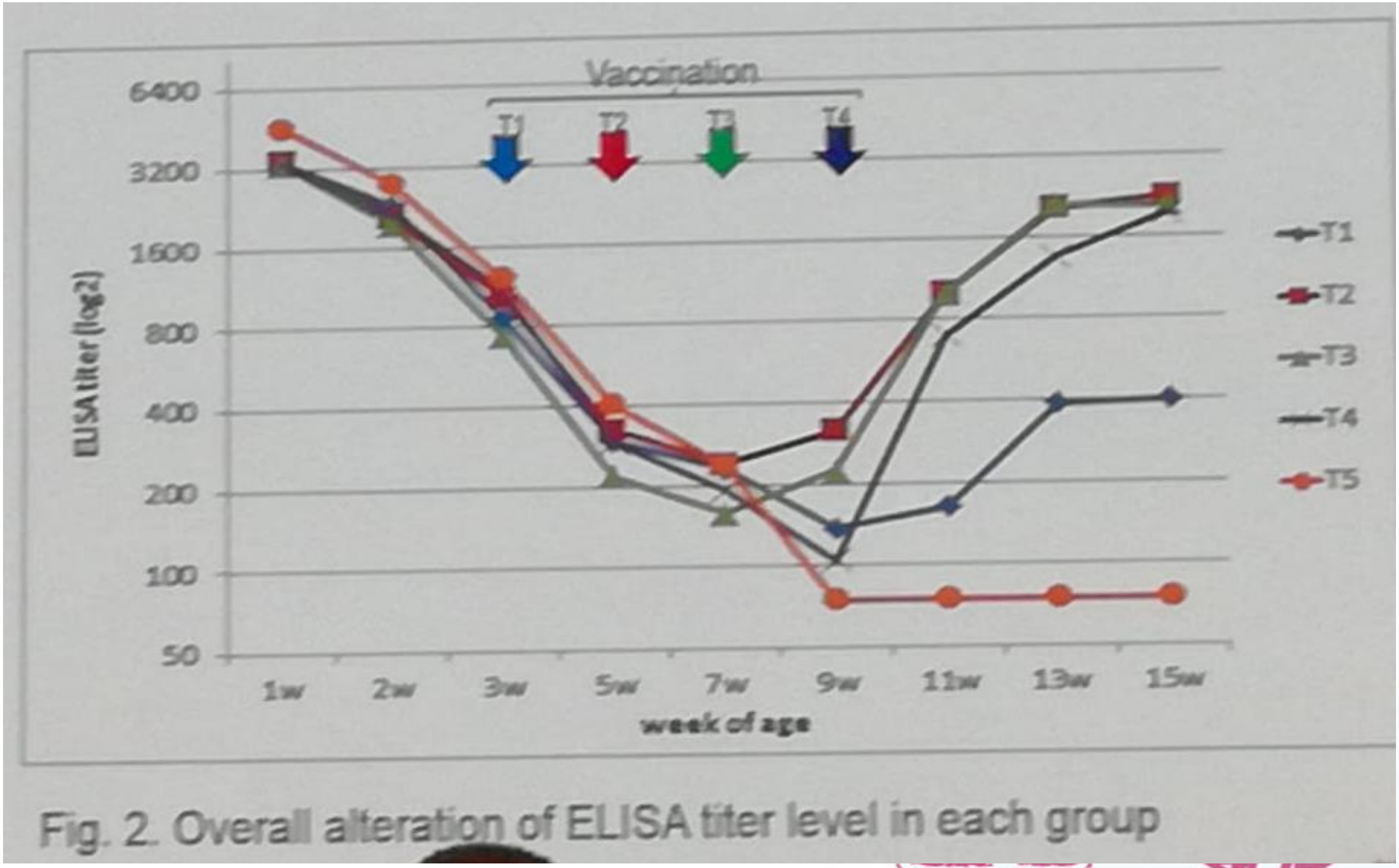


Fig. 2. Overall alteration of ELISA titer level in each group

Vaccination timing

Table 1 Morbidity (parenteral injections) and mortality in the four groups under study for each replicate

	REPLICATE		GROUP			
			A	B	C	D
MORBIDITY*	1st		6.2[a]	5.4[a]	14.7[b]	18.9[b]
	2nd		22.7[a]	20.7[a]	21.3[a]	22.4[a]
	3rd		10.3[a]	0[b]	8.3[a]	18.2[a]
MORTALITY*	1st	from weaning to 12 weeks of age	3.1[a]	1.6[b]	3.1[a]	3.6[a]
		from 12 weeks to slaughter	3.9[a]	1.6[b]	4.7[a]	3.1[a]
	2nd	from weaning to 12 weeks of age	5.3[a]	4.0[a]	2.7[b]	4.3[a]
		from 12 weeks to slaughter	2.7[a]	2.7[a]	4.0[b]	5.4[b]
	3rd	from weaning to 12 weeks of age	5.7[a]	1.9[b]	2.0[b]	3.8[a]
		from 12 weeks to slaughter	14.0[a]	7.7[b]	18.0[a]	20.0[a]

Legend: * proportion of pigs; Different superscript letters indicate statistically significant differences ($p < 0.05$)

Group A: pigs vaccinated at 4 weeks; group B: pigs vaccinated at 6 weeks; group C: pigs vaccinated at 8 weeks; group D: non-vaccinated placebo/controls

vaccination in sows at mating, befor farrowing and in piglets at 6 weeks of age was more effective for controlling PCV2 natural infection

Martelli et al., 2016

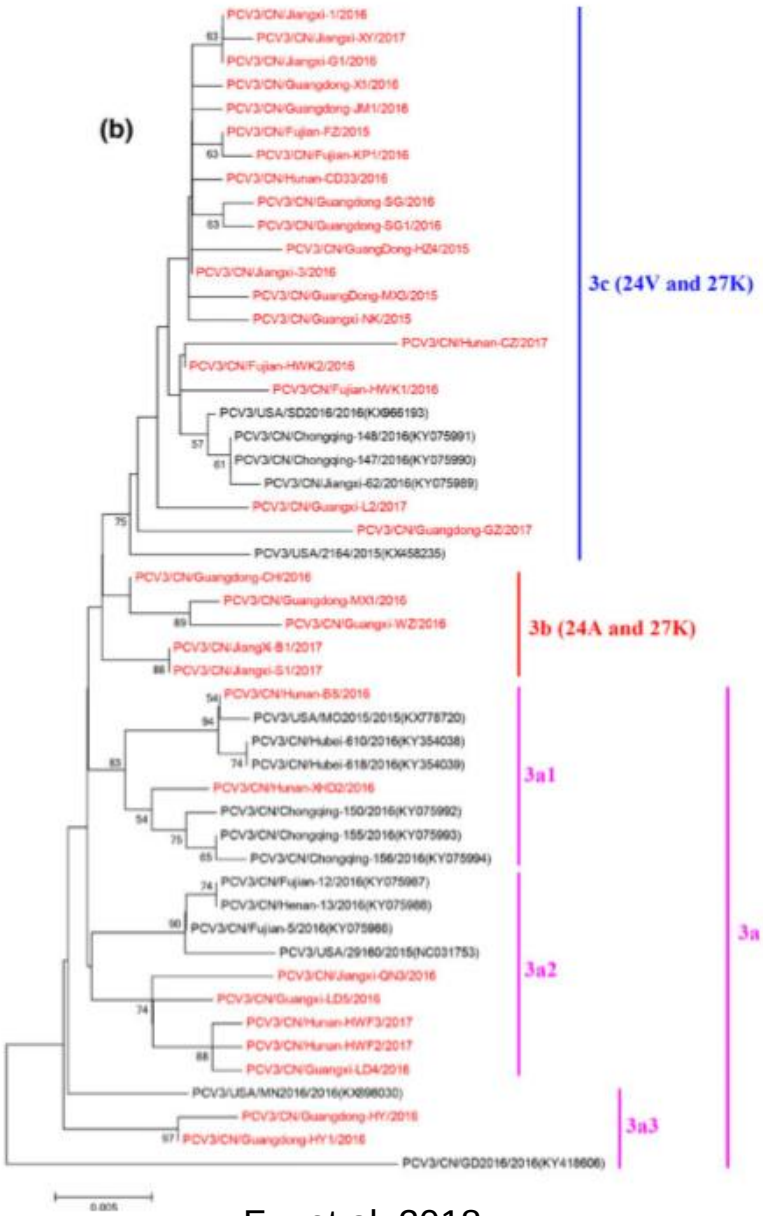
Take home message

- PCV2 vaccine work very well
- Vaccination will change epidemiology of virus >> prepare monitoring PCV2 infection >> adapt vaccinated protocol
- Balance vaccination with level of MDA
- Genotype shift

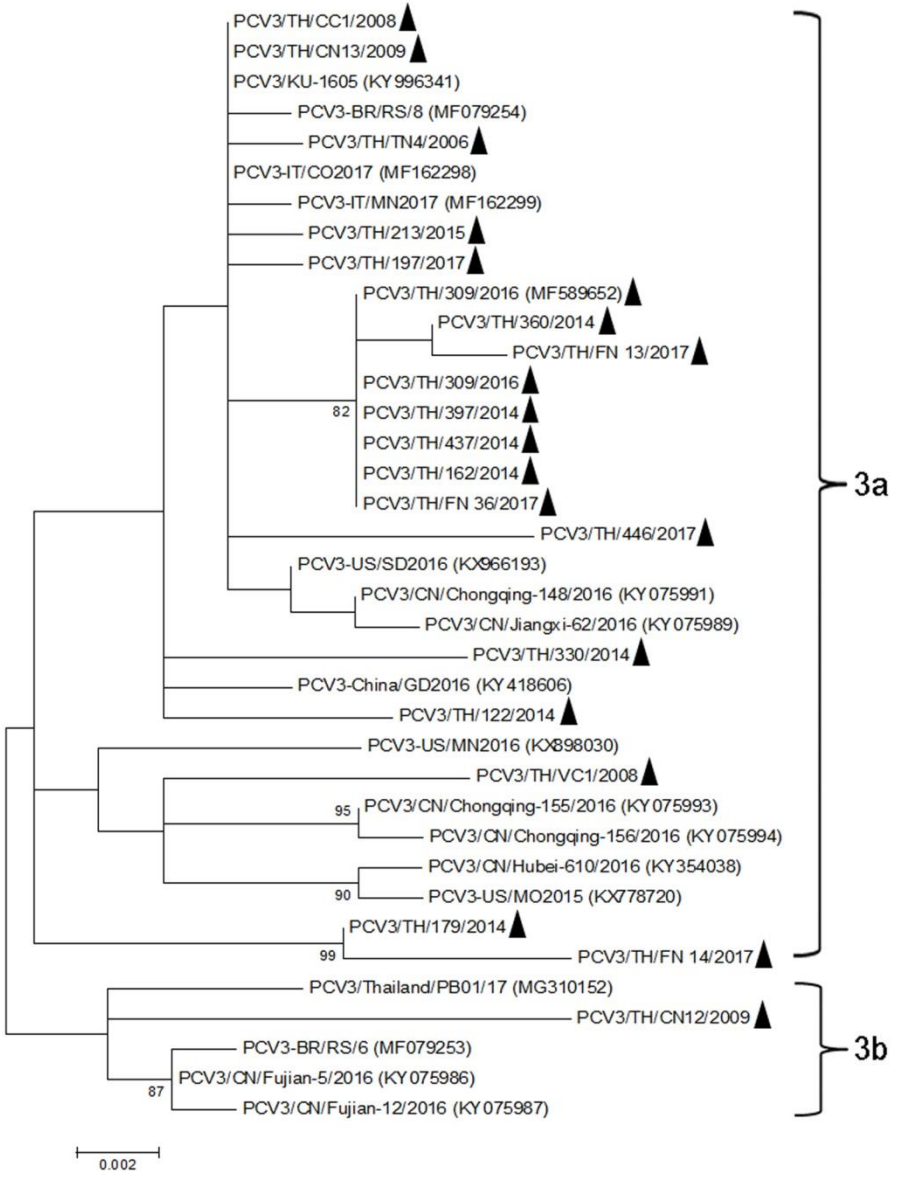
PCV3

- Genetic diversity of PCV3 ???
- Origin of outbreak ???
- Pathogenicity ???
- PCV2 vaccine against PCV3 ???

Low diversity of PCV3



Fu, et al. 2018

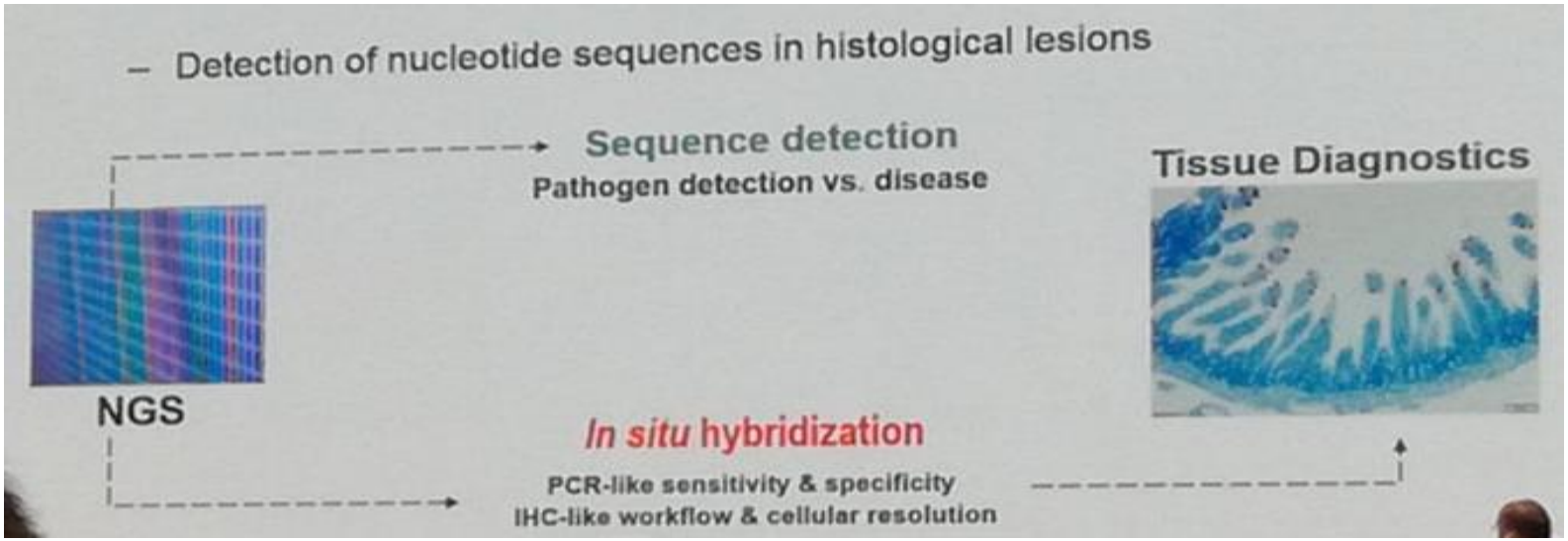


Sukmak, et al. 2018

Low diversity of PCV3

- Genetic diversity = 3.2% worldwide
- PCV3 >> recent outbreak
- divergence time of PCV1 and PCV2 approximately 84 years ago
- PCV3 >> approximately 50 years ago
- No geographical relationship

PCV3 pathogenicity



- NGS + in situ hybridization
- PCV2 and PCV3 were predominantly found in lymph nodes, spleen, heart, etc
- Replicate in same tissue
- However, pathogenicity **could not conclude**

Vaccinated farms	Age group	Number of serum pools tested	Number of positive serum pools			Proportion of positive serum pools		
			PCV3	PCV2	PCV3/PCV2	PCV3	PCV2	PCV3/PCV2
	Piglets	8	1	0	0	12.5%	0.0%	0.0%
	Weaners	22	2	0	0	9.1%	0.0%	0.0%
	Fatteners	48	14	2	0	29.2%	4.2%	0.0%
	Sows	14	3	1	1	21.4%	7.1%	7.1%
	Total	92	20	3	1	21.7%	3.3%	1.1%

Non-vaccinated farms	Age group	Number of serum pools tested	Number of positive serum pools			Proportion of positive serum pools		
			PCV3	PCV2	PCV3/PCV2	PCV3	PCV2	PCV3/PCV2
	Piglets	8	0	1	0	0.0%	12.5%	0.0%
	Weaners	10	1	4	0	10.0%	40.0%	0.0%
	Fatteners	34	10	28	4	29.4%	82.4%	11.8%
	Sows	8	1	2	1	12.5%	25.0%	12.5%
	Total	60	12	35	5	20.0%	58.3%	8.3%

Detection of PCV3, PCV2 and PCV3/PCV2 co-infections in pools of serum from different age groups of pigs from 6 farms vaccinated and 4 farms non-vaccinated against PCV2. A group was considered positive if at least one pool reacted positive in Real Time PCR.

Take home message

- PCV3+PCV2 co-infection are common but more frequent in non-PCV2-vaccinated farm
- PCV3 infection does not seem to affect efficiency of vaccination against PCV2 and severity of PCV2 infection
- PCV2 infection in non-PCV2-vaccinated farm does not facilitate PCV3 infection

PEDV

- New strain of PEDV ????
- Role of spike (S) gene

Emergence of mutants of porcine epidemic diarrhea viruses (PEDV) in Korea and application of nanobiotechnology for vaccine adjuvant against PEDV

Daesub Song[1], Minjoo Yeom[1], Woonsung Na[1], Jong-Woo Lim[2], Hyoung Hwa Jeong[3], Seungjoo Haam[2], BongKyun Park[4]
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[4] *Animal Virology Lab., College of Veterinary Medicine, Seoul National University, Gwanak-ro 1, Gwanak-gu, Seoul 08*

Report on large deletion of spike gene !!!

I -070

Identification of porcine epidemic diarrhea virus variant with a large spike gene deletion from a clinical swine sample in the USA

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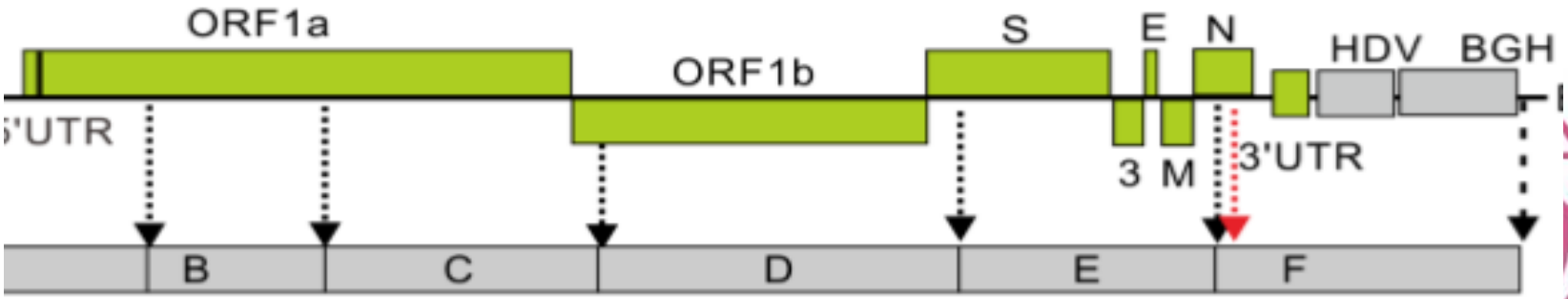
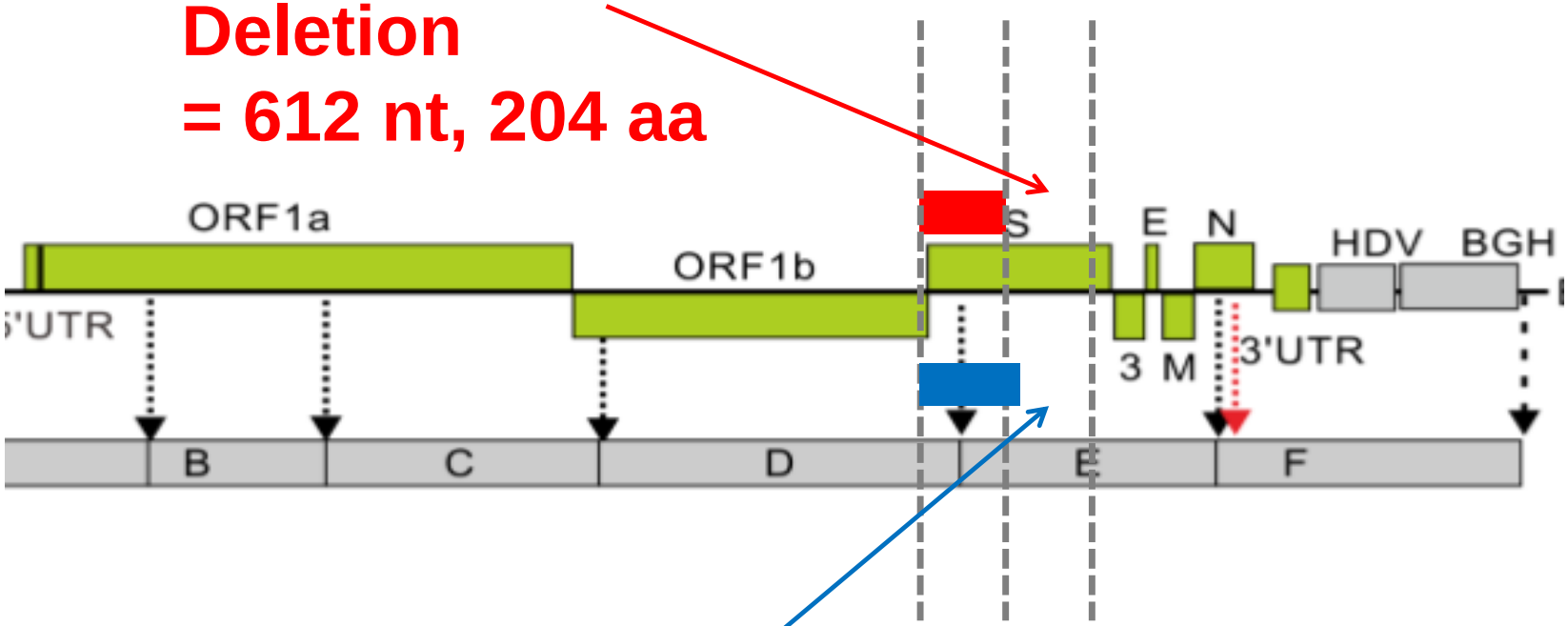
Role of spike (S) gene

- S protein mediates PEDV invasion into host cell
- receptor-binding subunit = S1
- membrane-fusion subunit = S2
- main target of neutralizing antibodies
- High variation



Korea
Deletion
= 612 nt, 204 aa



USA
Deletion
= 600 nt, 200 aa

Take home message

- Low prevalence
- Viral culture for 3 passage show positive results to RT-PCR (Korea)
- Viral isolation were unsuccessful (USA)
- Inoculation = no infection (USA)
- del-PEDV-Korea similar to PEDV korean strain
- del-PEDV-USA similar to USA strain

III-045

The S gene is necessary but not sufficient for the virulence of epidemic PEDV strains

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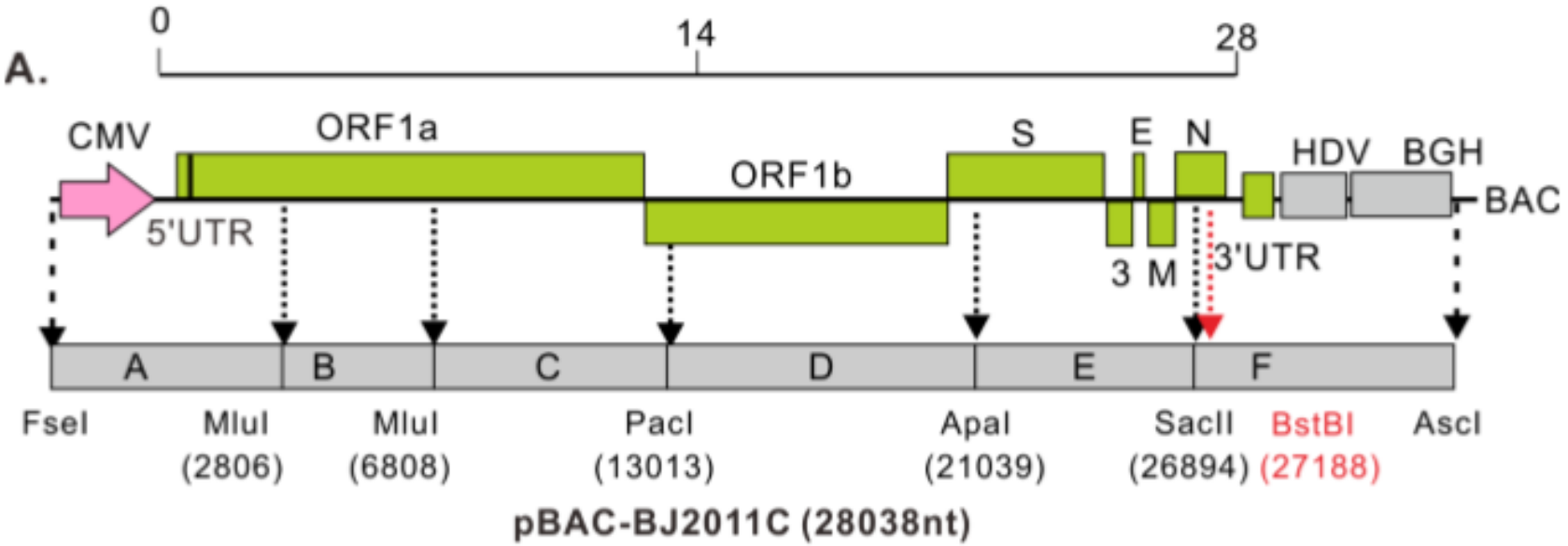
*Corresponding Author: hanx0158@cau.edu.cn

Introduction:
The recently emerged highly virulent variants of porcine epidemic diarrhea virus (PEDV) are the major cause of the global PED pandemic and have caused enormous economic losses to the worldwide swine industry. Remarkably, deletions, insertions or amino acid substitutions have been found in the spike protein (S) of the novel strains as compared to the classical strains such as CV777. The objective of this study is to determine whether the mutations within S gene are associated with the increased virulence.

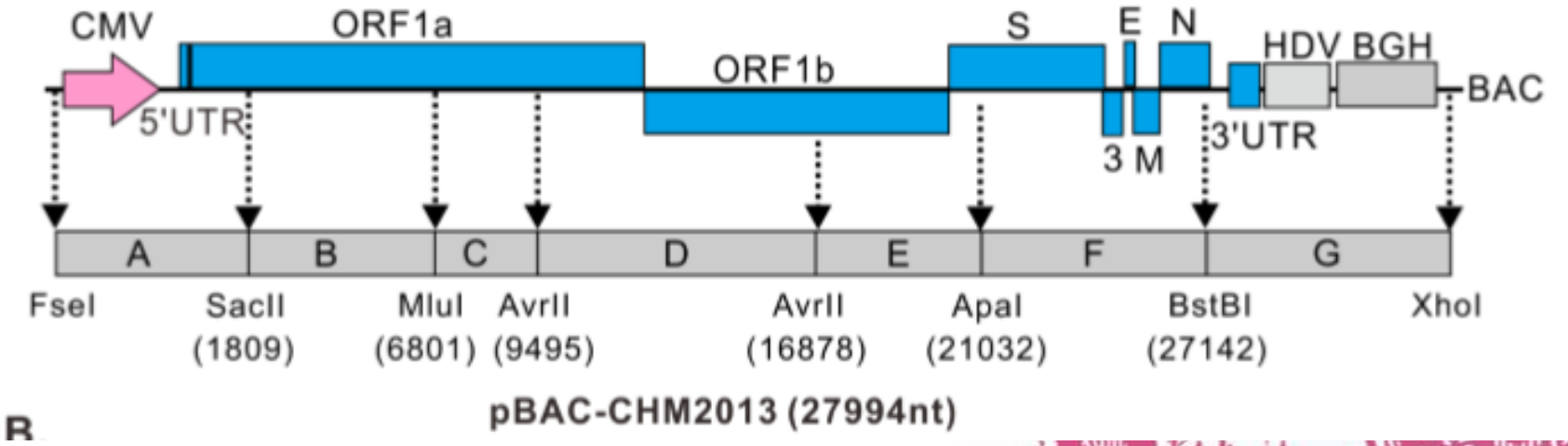
Materials and Methods:
By using reverse genetics, we generated two full-length chimeric infectious cDNA clones by swapping the S genes between the highly pathogenic strain BJ2011c and low pathogenic strain CHM2013. The viruses were rescued by transfection of recombinant BAC plasmids into Vero CCL81 cells and the virulence was tested in 2-day-old piglets.

Results:
The animal studies showed that WT BJ2011C caused death of the piglets within 48 hours whereas the chimeric virus BJ2011C-$S_{CHM2013}$ carrying the S gene from strain CHM2013 showed very mild virulence and did not caused death of the piglets. On the other side, both CHM2013 and the chimeric virus CHM2013-$S_{BJ2011C}$ carrying BJ2011C S gene showed no virulence to piglets.

Conclusion:
Thus, we conclude that the S gene is necessary but not sufficient to confer the enhanced virulence.

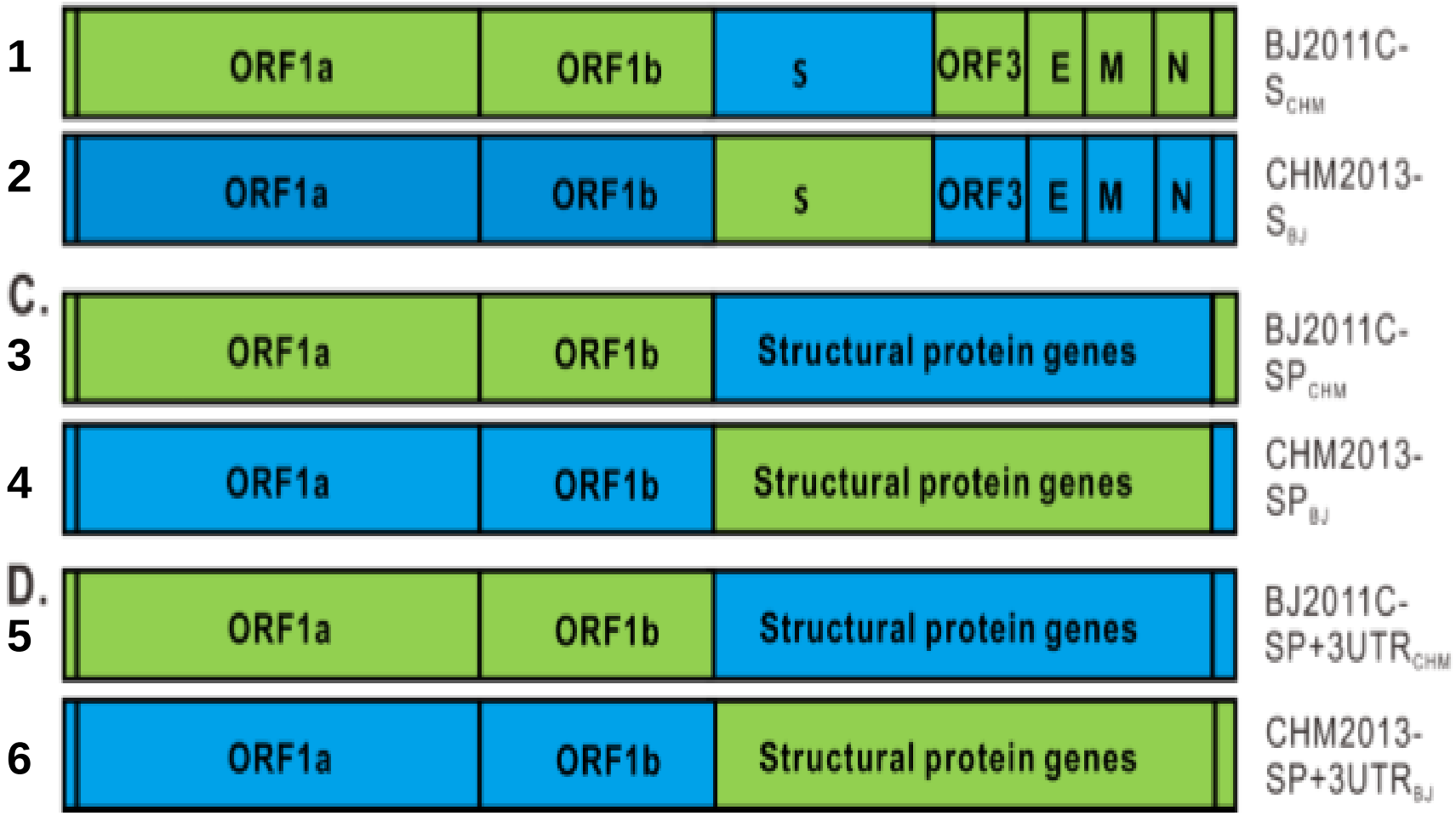


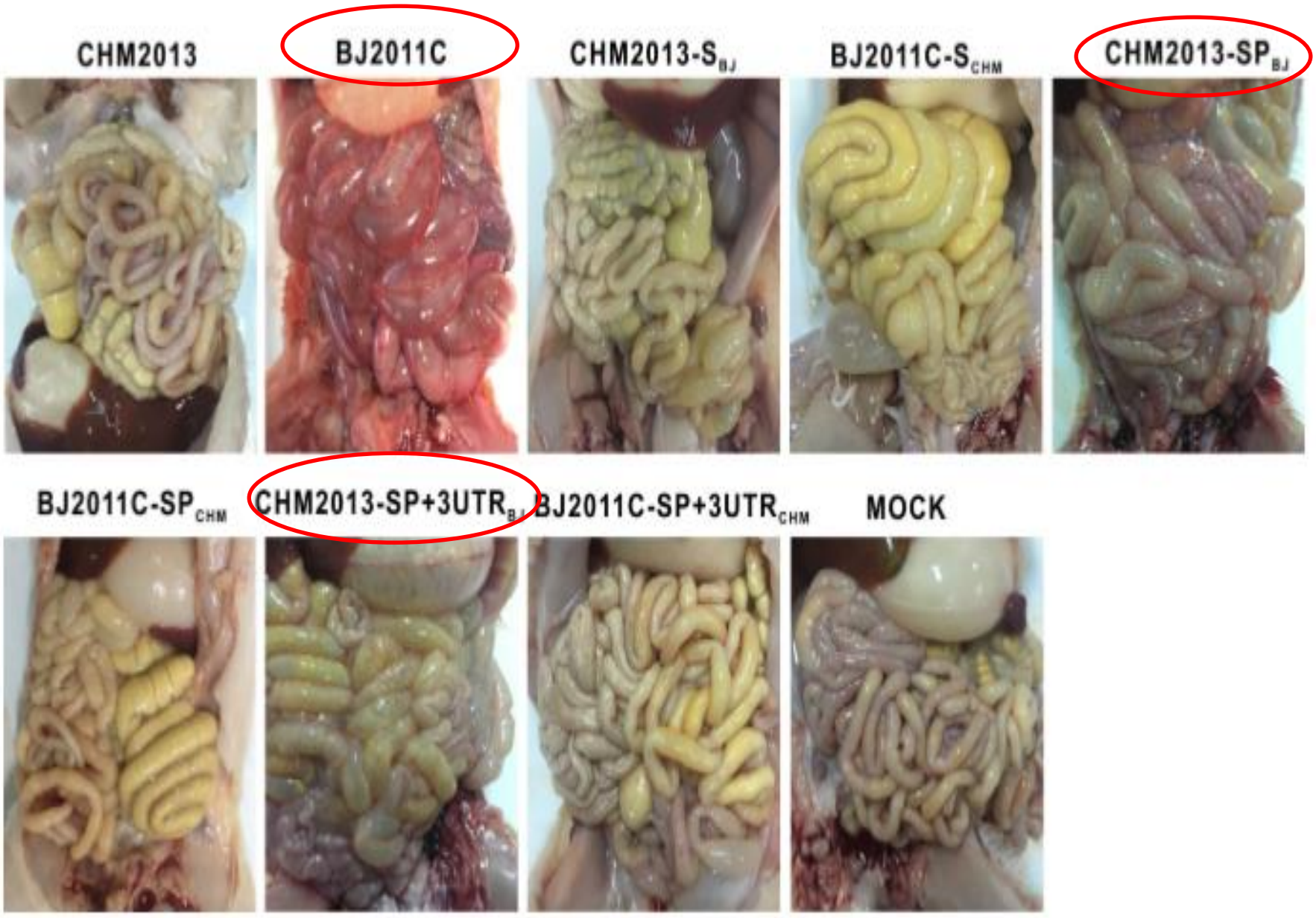
Virulent !!!



Non-Virulent !!!

Reverse genetic technology create new virus!!!




CHM2013
BJ2011C
CHM2013-S$_{BJ}$
BJ2011C-S$_{CHM}$
CHM2013-SP$_{BJ}$
BJ2011C-SP$_{CHM}$
CHM2013-SP+3UTR$_{BJ}$
BJ2011C-SP+3UTR$_{CHM}$
MOCK

Take home message

- S gene of the highly virulent PEDV strain BJ2011C is necessary but not sufficient to confer the fatal virulence to two-day-old piglets
- SP region and 3’UTR promote the efficiency of viral colonization of intestinal tract and also contribute critically to the post-colonization pathogenicity

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