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Swine inflammation and necrosis syndrome (SINS) and its association with biting behavior and production traits after weaning

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Introduction

Swine inflammation and necrosis syndrome (SINS)

Blood flow constraint of capillaries

- tissue inflammation
- loss of function

Birth to Slaughter (up to 75% of piglets)

- mostly non-severe

Should we be concerned about that?



Introduction

Damage / biting behavior

Tail, ear-biting, skin lesions

Result from animal interaction

- A performer and a victim

Could the selection on multiple heritable levels help us better select against damage?



<https://www.wattagnet.com/articles/10609-tips-for-combating-pig-tail-biting>



<https://www.alltech.com/blog/tips-reducing-tail-biting-pigs>

Objectives

Population 1:

- I. Is SINS heritable?
- II. Is that correlated with other production and behavior traits?

Population 2:

- III. Can we better model biting using social interaction models?

Material and methods

Population 1

5,960 three-way crossbred piglets
240 females and 80 sires
1 farm in the Netherlands



Topigs Norsvin

Fachhochschule
Südwestfalen
University of Applied Sciences

Pre-weaning:

SINS - tail, ear, teats, and claws (yes/no scores)
Total SINS (yes/no)
Birth and weaning weight

Post-weaning:

2,630 individuals were evaluated
Damage at 9 weeks (yes/no scores)
Carcass back fat thickness and loin depth

Material and methods

Population 1

- I. Is SINS heritable?
- II. Is that correlated with other production and behavior traits?

Three-trait models:

SINS, DAMAGE, BF/LD/BW/WW

$$y = XB + \text{litter} + \text{animal} + \text{maternal} + e$$

BLUF90 suit (Misztal et al., 2014)

Material and methods

Population 2

46,340 F1 gilts
5 farms in Germany



Post-weaning:

Damage records
145.8 \pm 13.0 days (89.5kg)
Groups of 10.7 individuals

Material and methods

Population 2

III. Can we better model biting using social interaction models?

Model 1. $y = XB + \text{hys} + \text{litter} + \text{group} + \text{animal} + e$

Model 2. $y = XB + \text{hys} + \text{litter} + \text{group} + \underline{\text{animal}_d} + \underline{\text{animal}_s} + e$

Material and methods

Population 2

Model 1:

$$BV_i = A_{D,i}$$

Effect over its
own phenotype

Model 2:

$$TBV_i = A_{D,i} + (n - 1) A_{S,i}$$

Effect over its
own phenotype

Effect over
phenotype of
others

Validation on 14,710 young F1 gilts (LR method) – Legarra and Reverter, 2018

BLUF90 suit (Misztal et al., 2014)

Results | population 1

BW

	BW_a	SINS_a	DAMAGE_a
BW_a			
SINS_a			
DAMAGE_a			

WW

	WW_a	SINS_a	DAMAGE_a
WW_a			
SINS_a			
DAMAGE_a			

BF

	BF_a	SINS_a	DAMAGE_a
BF_a			
SINS_a			
DAMAGE_a			

LD

	LD_a	SINS_a	DAMAGE_a
LOIN_a			
SINS_a			
DAMAGE_a			

Results | population 1

BW

	BW_a	SINS_a	DAMAGE_a
BW_a	0.07 (0.01)		
SINS_a		0.21 (0.02)	
DAMAGE_a			0.05 (0.01)

WW

	WW_a	SINS_a	DAMAGE_a
WW_a	0.11 (0.02)		
SINS_a		0.23 (0.03)	
DAMAGE_a			0.05 (0.01)

BF

	BF_a	SINS_a	DAMAGE_a
BF_a	0.63 (0.07)		
SINS_a		0.24 (0.03)	
DAMAGE_a			0.06 (0.01)

LD

	LD_a	SINS_a	DAMAGE_a
LOIN_a	0.42 (0.06)		
SINS_a		0.15 (0.02)	
DAMAGE_a			0.05 (0.01)

SINS heritability ranged from 0.15 to 0.24 (direct) and from 0.07 to 0.12 (maternal)

Results | population 1

BW

	BW_a	SINS_a	DAMAGE_a
BW_a		-0.30 (0.08)	-0.11 (0.09)
SINS_a			0.19 (0.08)
DAMAGE_a			

WW

	WW_a	SINS_a	DAMAGE_a
WW_a		-0.40 (0.08)	-0.61 (0.06)
SINS_a			0.23 (0.09)
DAMAGE_a			

BF

	BF_a	SINS_a	DAMAGE_a
BF_a		0.05 (0.05)	-0.84 (0.04)
SINS_a			0.43 (0.07)
DAMAGE_a			

LD

	LD_a	SINS_a	DAMAGE_a
LOIN_a		-0.16 (0.07)	0.76 (0.06)
SINS_a			0.50 (0.09)
DAMAGE_a			

SINS and damage are positively correlated
(0.19-0.50)



Results | population 1

BW

	BW_a	SINS_a	DAMAGE_a
BW_a		-0.30 (0.08)	-0.11 (0.09)
SINS_a			0.19 (0.08)
DAMAGE_a			



WW

	WW_a	SINS_a	DAMAGE_a
WW_a		-0.40 (0.08)	-0.61 (0.06)
SINS_a			0.23 (0.09)
DAMAGE_a			



BF

	BF_a	SINS_a	DAMAGE_a
BF_a		0.05 (0.05)	-0.84 (0.04)
SINS_a			0.43 (0.07)
DAMAGE_a			

LD

	LD_a	SINS_a	DAMAGE_a
LOIN_a		-0.16 (0.07)	0.76 (0.06)
SINS_a			0.50 (0.09)
DAMAGE_a			

SINS is negatively correlated with pre-weaning production traits but weakly correlated with post-weaning traits

Results | population 2

Variance components

Model	σ_{gr}^2	h_a^2 or T^2	r_{Ads}
1	72.44	0.03	-
2	69.18	0.09	-0.09

3-fold

Neutral Interaction

Validation

Model	Effect	ACC	Dispersion (b1)	Validation (N)
1	Direct	0.16	0.82	14710
2	TBV	0.27	1.16	14710

↑ ~70%

Expectation = 1.0 (no dispersion)

Conclusions

I. Is SINS heritable? Yes

- Influenced by piglets (0.15-0.20) and dams (0.07-0.12)

II. Is that correlated with other production and behavior traits? Yes

- Selection against SINS will benefit BW and WW (-0.30 to -0.40), but should not affect BF and LD
- At the piglet genetic level, SINS animals are more predisposed to being bitten (0.19-0.50) after weaning

III. Can we better model biting using social interaction models? Yes

- Genetic variance
- Accounting for social genetic effects increases accuracies in 70%



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Thank you

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