

# A Norwegian observational study of feed conversion efficiency in Duroc and Landrace grower pigs seropositive for influenza A (H1N1) pdm09 virus

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## Abstract

**Objective:** Investigate the influence of influenza A(H1N1)pdm09 virus (pH1N1v) on feed conversion efficiency (FCE) in Norwegian Landrace and Duroc pigs.

**Materials and methods:** This observational study analyzed the growth and serological data of 1954 grower pigs collected from 43 nucleus breeding herds in eastern Norway between 2009 and 2012. Serial serological tests, enzyme-linked immunosorbent assay, and hemagglutination inhibition were used to detect pH1N1v antibodies in pigs weighing 100 kg. Statistical analyses included mixed-effects regression modelling, Cox regression, and Kaplan-Meier Failure analysis to assess the effects of breed on pH1N1v influence on growth performance.

**Results:** Duroc pigs experienced a greater reduction in FCE (5.6%; 95% CI, 5.5%-5.7%) compared to Landrace pigs (3.5%; 95% CI, 1.3%-5.6%) when exposed to pH1N1v. Seropositive pigs of both breeds maintained normal growth rates under *ad libitum* feeding conditions. To reach 100 kg body weight, seropositive Landrace pigs consumed 2.4 kg (95% CI, 0.9-3.9 kg) more feed, while Duroc pigs consumed 3.8 kg (95% CI, 3.7-4.0 kg) more feed than their seronegative counterparts.

**Implications:** Results suggest breed-specific differences in resilience to influenza even though the overall appetite of seropositive pigs was unimpaired during the growth phase (approximately 33-100 kg body weight). Study findings highlight the economic implications of selecting appropriate breeds for specific

environmental challenges. However, the study's observational nature limits the ability to infer causality and may not be generalized to other breeds or cross-breeds. By understanding breed-specific responses to influenza, producers can optimize breed selection strategies to enhance overall herd resilience and efficiency, contributing to more sustainable pork production.

**Keywords:** swine, influenza, feed conversion efficiency, breed, mixed-effects linear regression

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**Un estudio observacional Noruego de la eficiencia de conversión alimenticia en cerdos de engorde Duroc y Landrace seropositivos para el virus de la influenza A (H1N1)pdm09**

**Objetivo:** Investigar la influencia del virus de la influenza A(H1N1)pdm09 (pH1N1v) en la eficiencia de conversión alimenticia (FCA) en cerdos Landrace y Duroc Noruegos.

**Materiales y métodos:** Este estudio observacional analizó el crecimiento y los datos serológicos de 1954 cerdos de engorde recolectados de 43 granjas núcleo en el este de Noruega entre 2009 y 2012. Se utilizaron pruebas serológicas

seriadas, ensayo de inmunoabsorción enzimática e inhibición de la hemaglutinación para detectar anticuerpos pH1N1v en cerdos de 100 kg de peso. Los análisis estadísticos incluyeron modelos de regresión de efectos mixtos, regresión de Cox y análisis de falla de Kaplan-Meier para evaluar los efectos de la raza en la influencia del pH1N1v en el rendimiento del crecimiento.

**Resultados:** Los cerdos Duroc experimentaron una mayor reducción en FCE (5.6%; IC 95%, 5.5%-5.7%) en comparación con los cerdos Landrace (3.5%; IC 95%, 1.3%-5.6%) cuando se expusieron a pH1N1v. Los cerdos seropositivos de

ambas razas mantuvieron tasas de crecimiento normales en condiciones de alimentación *ad libitum*. Para alcanzar los 100 kg de peso corporal, los cerdos Landrace seropositivos consumieron 2.4 kg (IC 95%, 0.9-3.9 kg) más de alimento, mientras que los cerdos Duroc consumieron 3.8 kg (IC 95%, 3.7-4.0 kg) más de alimento que sus contrapartes seronegativas.

**Implicaciones:** Los resultados sugieren diferencias específicas de cada raza en la resistencia a la gripe, a pesar de que el apetito general de los cerdos seropositivos no se vio afectado durante la fase de crecimiento (aproximadamente

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33-100 kg de peso corporal). Los hallazgos del estudio resaltan las implicaciones económicas de seleccionar razas apropiadas para desafíos ambientales específicos. Sin embargo, la naturaleza observacional del estudio limita la capacidad de inferir causalidad y no puede generalizarse a otras razas o cruza. Al comprender las respuestas específicas de las razas a la influenza, los productores pueden optimizar las estrategias de selección de razas para mejorar la resistencia y la eficiencia general del rebaño, lo que contribuye a una producción de carne de cerdo más sostenible.

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### Étude observationnelle Norvégienne sur l'efficacité de la conversion alimentaire chez des porcs Duroc et Landrace en croissance séropositifs pour le virus de l'influenza A (H1N1)pdm09

**Objectif:** Étudier l'influence du virus de l'influenza A (H1N1)pdm09 (pH1N1v) sur l'efficacité de la conversion alimentaire (FCE) chez des porcs Landrace et Duroc Norvégiens.

**Matériels et méthodes:** Cette étude observationnelle a analysé les données de croissance et de sérologie de 1954 porcs en croissance obtenues de 43 noyaux de troupeaux de reproduction dans l'est de la Norvège entre 2009 et 2012. Des tests sérologiques en série, un essai immuno-enzymatique et l'inhibition de l'hémagglutination, ont été utilisés afin de détecter des anticorps contre pH1N1v chez des porcs pesant 100 kg. Les analyses statistiques incluaient une modélisation de régression avec effets mixtes, une régression de Cox et l'analyse de survie de Kaplan-Meier afin d'évaluer les effets de la race sur l'influence du pH1N1v sur la performance de croissance.

**Résultats:** Les porcs de race Duroc ont montré une plus grande réduction de FCE (5.6%; IC 95%, 5.5%-5.7%) comparativement aux porcs Landrace (3.5%, IC 95%, 1.3-5.6%) lorsqu'exposés à pH1N1v. Les porcs séropositifs des deux races ont maintenu des taux de croissance normaux dans des conditions d'alimentation *ad libitum*. Afin d'atteindre le poids corporel de 100 kg, les porcs Landrace séropositifs ont consommés 2.4 kg plus de nourriture (IC 95%, 0.9-3.9 kg), alors que les porcs Duroc ont consommé 3.8 kg de plus (IC 95%, 3.7-4.0 kg) que leur contrepartie séronégative.

**Implications:** Les résultats suggèrent des différences spécifiques aux races quant à la résilience à l'influenza, bien

que de manière générale l'appétit des porcs séropositifs n'était pas affecté durant la période de croissance (approximativement de 33-100 kg de poids corporel). Les résultats de l'analyse mettent en évidence les conséquences économiques de sélectionner les races appropriées pour des défis environnementaux spécifiques. Toutefois, la nature observationnelle de l'étude limite la capacité à supposer une causalité et ne peut être généralisée à d'autres races ou croisements. En comprenant les réponses spécifiques à la race à l'influenza, les producteurs peuvent optimiser les stratégies de sélection de la race pour augmenter la résilience et l'efficacité globale du troupeau, contribuant ainsi à une production porcine plus durable.

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**S**wine genetics significantly influence key agricultural performance metrics, including disease resistance and growth performance. Such genetic factors are crucial for enhancing pork production efficiency and animal welfare, but also in responding to increasing global demands and environmental sustainability pressures. In Norway, a leader in pork self-sufficiency, the strategic use of crossbreeding among predominant breeds, like Landrace, Duroc, Yorkshire, and Hampshire, optimizes heterosis to balance traits, meet market demands, and bolster disease resistance cost effectively.

Building on previous research by Rowland et al<sup>1</sup> and Lunney et al<sup>2</sup> that highlight the role of breed genetics in disease resistance, our study examines the different effects of influenza A(H1N1)pdm09 virus (pH1N1v) on feed conversion efficiency (FCE) among seropositive Norwegian Landrace and Duroc pigs. Norwegian Landrace pigs exhibit superior growth performance compared to Duroc, which deviates from trends observed in other countries. This study seeks to deepen the understanding of how genetic predispositions influence resilience to influenza, aiming to enhance both the profitability and environmental sustainability of pork production.

Research into optimizing FCE focuses not just on profitability in pork production,<sup>3</sup> but also promotes responsible environmental stewardship by using less agricultural resources. To achieve this, considerable research has been dedicated to dietary influences, such as nutrition, appetite, and feed composition,<sup>4</sup>

and nondietary factors including housing conditions, genetics, and overall health.<sup>5-9</sup> Respiratory diseases caused by various pathogens are severe health and production challenges for pig producing countries.<sup>10-13</sup> Among these, the influenza A virus (IAV) stands out due to its ubiquity, multispecies hosts including humans, and impact.<sup>14,15</sup> The coexistence of multiple porcine respiratory pathogens in the same pig host, known as the porcine respiratory disease complex (PRDC), further complicates this issue, significantly impacting growth and feed efficiency by diverting energy towards immune responses.<sup>11,12,15-17</sup> The PRDC also includes other major pig respiratory pathogens such as porcine reproductive and respiratory syndrome virus (PRRSV), *Actinobacillus pleuropneumonia*, porcine circovirus-associated disease, and *Mycoplasma hyopneumonia*, which can dramatically affect pig health and pork production.<sup>18,19</sup>

The emergence of pH1N1v in 2009 was the first IAV detected in the Norwegian pig population through active serological screening of notifiable diseases absent in Norwegian pigs.<sup>20,21</sup> The virus spread quickly and became endemic in the human population first and later in Norwegian pigs, reaching approximately 800 pig herds (40% herd prevalence) in a short time.<sup>21-25</sup> Previous research by Er et al<sup>26</sup> demonstrated that pH1N1v can depress FCE in pigs even when they did not show overt clinical signs.<sup>26-28</sup> The objective of the current study is to investigate the role of breed genetics in modulating the effects of pH1N1v on FCE among Norwegian Landrace and Duroc pig breeds. These breeds represent the pinnacle of Norway's pig breeding in 46 nucleus herds in terms of biosecurity, health profile, and genetic quality, making them ideal subjects for our research on genetic modulations in response to pH1N1v.

### Animal care and use

This comparative field study was observational and conducted from 2009 to 2012 at Norsvin's commercial boar testing station in Hamar, Norway. All husbandry and housing conditions remained unchanged during the observation period. Norway has a long standing comprehensive animal welfare act that covers aquatic and terrestrial animals.<sup>29</sup>

# Materials and methods

## Study design

In this comparative study, longitudinal growth data and serological results were collected from Landrace (n = 1084) and Duroc boars (n = 870) from Norsvin's boar testing station in the Hamar municipality of eastern Norway. The indoor boar testing facility, capable of testing 1152 pigs concurrently, features 16 separate rooms housing cohorts of 72 pigs (Landrace or Duroc) divided into six pens. Batches of pigs from specific herds (n = 43 nucleus herds) arrived at the station with a mean weight of 33 kg were monitored individually using electronic feeding stations equipped with Feed Intake Recording Equipment (FIRE, Osborne Ltd). This automated system tracked individual pig feed consumption and body weight until pigs reached 100 kg. Before departure from the facility, each pig's exposure status to pH1N1v was determined by serological testing for the presence of antibodies.<sup>30</sup> Additionally, each departing pig was screened for select mandatory notifiable diseases not found in Norway including pseudorabies virus, transmissible gastroenteritis virus, porcine respiratory corona virus, PRRSV, porcine epidemic diarrhea virus, and other swine influenza viruses including pH1N1v since 2009. Influenza A specific NP antibodies were detected by enzyme-linked immunosorbent assay (ID Screen IAV Antibody Competition test, IDVET) according to manufacturer's instructions. Samples positive for IAV antibodies were tested using the hemagglutination-inhibition assay according to the method described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.<sup>31</sup> All

serological tests were performed by the Norwegian Veterinary Institute. Yearly surveillance to date (2023) has confirmed pH1N1v as the sole IAV circulating among Norwegian pigs since 2009. In our study sample, 60% of Landrace pigs and 49% of Duroc pigs were seropositive from exposure to pH1N1v (Table 1).

## Statistical analysis

Statistical tools include mixed-effects regression modelling, Cox regression with the Breslow method (CRB), and Kaplan-Meier Failure function (KMF). Comparative box plots visualized the fitted values from the regression models. The three outcome variables included FCE, overall feed intake (OFI), and age at 100 kg body weight (Age100kg), the latter being a proxy for growth rate. Key predictors (fixed effects) were breed, infection status, and each pig's birth date. Initially structured longitudinally, the data was converted into a panel format to aggregate daily growth data into the study outcomes. Mixed-effects regression techniques acknowledged the hierarchical data structure, with pig (n = 1954) nested within herd (n = 43). Data handling and analysis were conducted using SAS Enterprise Guide 4.3 (SAS Institute Inc) and STATA version 17.0 (StataCorp LP).

## Model selection and statistical approach

The selection of mixed-effects regression models was guided by causal diagrams and principles of parsimony and the Akaike Information Criterion (AIC).<sup>32</sup> The study sample of 1954 pigs originated from 43 nucleus herds. By including the herd ID as a random effects variable, we accounted for potential confounding

factors such as sanitary conditions and genetic variants unique to the herd. As the data spanned four years, pig birth date was incorporated as a fixed effect covariate in the regression model to mitigate chronological bias eg, pig genetics, feed technology, and all-time variant variables.

## Mixed-effects linear regression model formula with pig as the unit of analysis

$$Y[i,j] = \beta_0 + \beta_1 X_1[i,j] + \beta_2 X_2 [i,j] + u[i,j] + v[j] + \epsilon[i,j]$$

Where Y is one of the three outcomes in this study (OFI, FCE, Age100kg).  $Y_i$  is the value of the response for  $i$ th pig (n = 1954) nested within the  $j$ th (n = 43) herd.  $\beta$  is a vector of the 3 coefficients, constant, main predictor (breed and infection or Inf#Br), and the continuous covariate (birth date).  $X_{[i,j]}$  is the vector of 2 explanatory variables (main predictor and the covariate) for the  $i$ th pig observed value in the  $j$ th herd.  $u_{[i,j]}$  is a vector of random intercepts unique to each pig in each herd, where  $u_{ij} \sim N(0, \sigma^2_{\text{pig}})$ .  $v_j$  is a vector of random intercepts unique to each herd, where  $v_j \sim N(0, \sigma^2_{\text{herd}})$ .  $\epsilon_{[i,j]}$  is the vector of error terms where  $\epsilon_{ij} \sim N(\mu, \sigma^2)$ . The creation of the interaction term Inf#Br simplifies the comparison of pH1N1v's marginal effects on the four categories of pigs.

## Results

### Feed conversion efficiency

Seropositive Landrace pigs exhibited a decrease in FCE (kg feed/kg weight gain) by 3.5% (95% CI, 1.3%-5.6%;  $P = .002$ ), whereas seropositive Duroc pigs showed

**Table 1:** Sample size and influenza A(H1N1)pdm09 serial serology results of Landrace and Duroc pigs tested for growth performance from 2009 to 2012 at Norsvin's commercial boar testing station

Year	Influenza A(H1N1)pdm09 serology*						Total pigs
	Landrace			Duroc			
	Negative	Positive	Subtotal	Negative	Positive	Subtotal	
2009 <sup>†</sup>	140	74	214	151	30	181	395
2010	83	6	89	63	12	75	164
2011	133	524	657	148	352	500	1157
2012	82	42	124	86	28	114	238
<b>Total</b>	<b>438</b>	<b>646</b>	<b>1084</b>	<b>448</b>	<b>422</b>	<b>870</b>	<b>1954</b>

\* Serial serology was by enzyme-linked immunosorbent assay and hemagglutination-inhibition assay.

<sup>†</sup> Year of introduction of influenza A(H1N1)pdm09, the first influenza A virus, in the Norwegian pig population.



a more pronounced decrease of 5.6% (95% CI, 5.5%-5.7%;  $P < .001$ ). The continuous variable birth date indicated an improvement in FCE by 0.003% ( $P < .001$ ) for each subsequent day a pig was born. Detailed results are presented in Table 2 and Figure 1.

### Overall feed intake

The study demonstrated a clear inverse correlation between OFI and FCE, where a decrease in FCE led to increased feed consumption necessary for weight gain. Our data indicated that compared to their uninfected counterparts at 100 kg, seropositive Landrace pigs consumed more than 2.4 kg (95% CI, 0.9-3.9 kg;  $P = .002$ ) of compensatory feed while Duroc pigs consumed 3.8 kg (95% CI, 3.7-4.0kg;  $P < .001$ ). Furthermore, the birth date coefficient revealed a daily decrease in OFI of 17 g starting from the earliest born pig (Table 3). Figure 2 is a visual presentation of predicted OFI values differentiated by pig breed, infection status, and chronology.

### Growth rate and compensatory feeding

Despite the observed decline in FCE, infected pigs maintained normal growth rates, a phenomenon attributed to compensatory feeding under *ad libitum* conditions. The CRB, boxplots and KMF curves, shown in Table 4 and Figures 3 and 4, respectively, indicated minimal differences in growth rates between infected and uninfected pigs across both breeds. Even with depressed FCE in seropositive pigs, their growth rates were comparable to seronegative pigs, facilitated by unimpaired appetite and an *ad libitum* feeding system. Some seropositive pigs, because of greater appetite, had slightly faster growth rates than their seronegative counterparts.

### Discussion

Our comprehensive observational study of 1954 pigs uncovered breed-specific responses to pH1N1v infection by regression analysis focusing on infection status and the breed. Landrace pigs exhibited a smaller decline in FCE compared to Duroc pigs, underscoring inherent differences in disease resilience and

growth efficiency between breeds. In seropositive pigs, the FCE reduction was 6% for Duroc and 3% for Landrace, highlighting Landrace's superior resilience. At 100 kg, the seropositive Landrace pigs consumed an additional 2.4 kg (95% CI, 0.9-3.9 kg) of feed, while seropositive Duroc pigs consumed 3.8 kg (95% CI, 3.7-4.0 kg). Compensatory feed consumption that occurred from unrestricted feeding allowed seropositive pigs to achieve similar growth rates as their seronegative counterparts. In comparison, Duroc pigs exhibited greater compensatory feeding, which carries economic implications in terms of feed cost to the farmer.

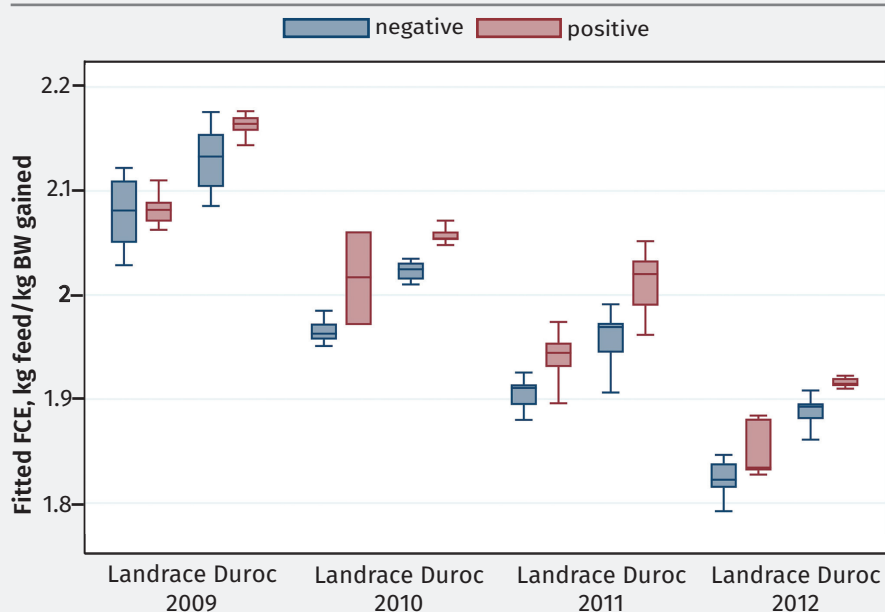
Despite its observational nature, the controlled environment provided by the boar testing station ensured uniform conditions for husbandry, housing, ventilation, and feeding for every cohort of pigs. This consistency allowed for a simplified analysis of variance components, enabling the mixed-regression techniques to effectively concentrate on the interactions between breed genetics and pH1N1v infection, thereby enhancing the study's validity. Additionally, the inclusion of birth date as the continuous

**Table 2:** Mixed-effects linear regression\* comparing the feed conversion efficiency (FCE) between Landrace and Duroc pigs (n = 1954) serologically positive for influenza A(H1N1)pdm09 virus

Feed conversion efficiency of a pig growing from 33-100 kg				
Predictors	Coefficient <sup>†</sup>	SE	P	95% CI
<b>Breed#Infection status</b>				
Landrace#negative	0	-	-	-
Landrace#positive	0.035	0.0111	.002	0.013 to 0.056
Duroc#negative	0.058	0.0225	.01	0.014 to 0.102
Duroc#positive	0.113	0.0227	< .001	0.068 to 0.157
Birth date	-0.0003	0.00001	< .001	-0.00032 to -0.00028
Constant ( $\beta_0$ ) <sup>‡</sup>	6.527	0.239	< .001	6.059 to 6.996
<b>Breed Margins<sup>§</sup></b>				
Landrace	1.95	0.0106	< .001	1.93 to 1.97
Duroc	2.02	0.0178	< .001	1.99 to 2.06
<b>Infection status</b>				
Negative	1.96	0.0109	< .001	1.94 to 1.98
Positive	2.00	0.0106	< .001	1.98 to 2.02

\* Data is hierarchical with 1954 pigs nested in 43 breeding herds where the 43 unique herd IDs represented the random effects in the regression model (values not shown).  
<sup>†</sup> The coefficients and standard errors of predictors were the parameters for Gaussian curves describing the variability between pigs.  
<sup>‡</sup> Constant represents the FCE of a seronegative Landrace pig born on October 3, 2008.  
<sup>§</sup> Least squares means.

**Figure 1:** Box plots for the predicted FCE of pigs growing from 33 to 100 kg categorized by breed, infection status, and testing cohort. The differences in 2009 were less obvious because of the smaller positive pig sample size given the introduction of influenza A(H1N1)pdm09 to Norwegian pigs occurred in September 2009. Boxes indicate the 25th percentile, median, and 75th percentile. Whiskers show the 10th and 90th percentiles. FCE = feed conversion efficiency; BW = body weight.



**Table 3:** Mixed-effects linear regression comparing overall feed intake (OFI) of Duroc and Landrace grower pigs (n = 1954) when infected with influenza A(H1N1)pdm09 virus\*

Overall feed intake of a pig growing from 33-100 kg				
Predictors	Coefficient	SE	P > z	95% CI
<b>Breed#Infection status</b>				
Landrace#negative	0	-	-	-
Landrace#positive	2.42	0.775	.002	0.9 to 3.93
Duroc#negative	4.05	1.572	.01	0.97 to 7.13
Duroc#positive	7.9	1.592	< .001	4.78 to 11.02
Birth date	-0.02	0.001	< .001	-0.019 to -0.016
Constant ( $\beta_0$ ) <sup>†</sup>	456.91	16.74	< .001	424.1 to 489.72
<b>Breed</b>				
	<b>Margin<sup>‡</sup></b>			
Landrace	136.57	0.745	< .001	135.11 to 138.03
Duroc	141.40	1.248	< .001	138.96 to 143.85
<b>Infection status</b>				
Negative	137.05	0.762	< .001	135.56 to 138.55
Positive	140.10	0.741	< .001	138.66 to 141.56

\* Data is hierarchical with 1954 pigs nested in 43 breeding herds where the 43 unique herd IDs represented the random effects in the regression model (values not shown). The coefficients and standard errors of predictors were the parameters for Gaussian curves describing the variability between pigs.

<sup>†</sup> Constant represents the OFI of a seronegative Landrace pig born on October 3, 2008.

<sup>‡</sup> Least squares means.

variable served as a proxy to account for time-variant biases among the pigs studied over the four years.

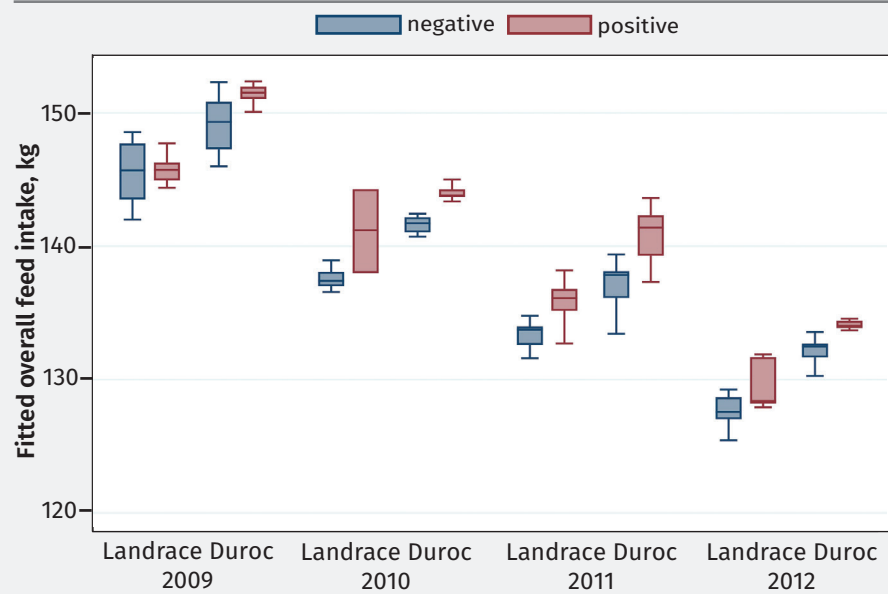
Although this study demonstrates that Landrace pigs possess genetic advantages over Duroc pigs in reducing pH1N1v impact on growth performance, the majority of growing pigs raised for slaughter in Norway are derived from the crossbreeding of Landrace, Duroc, Yorkshire, and Hampshire. Consequently, the impact of pH1N1v on these crossbreeds, as well as on the other 300 pig breeds and their resulting crossbreeds raised in other countries, is likely to vary. While our findings affirm that breed genetics can influence the effects of pH1N1v on growth performance, the ability to quantify the external validity of these negative effects remains limited both in Norway and internationally.

The parallel patterns in pH1N1v pig herd prevalence and human pH1N1v variant persistent trends in Norway hint at ongoing human-to-pig transmission, affecting pork production efficiency under the current nonintervention policy.<sup>33,34</sup> This interspecies transmission underlines a crucial one health perspective, necessitating a holistic approach to managing public and animal health.

The global diversity of over 300 pig breeds, each with distinct growth and disease resilience traits, presents opportunities to optimize farm economics and national strategies by capitalizing on breed-specific characteristics. The global persistence of pH1N1v in both humans and pigs, along with the prevalence of other porcine respiratory diseases, necessitates a broader consideration of the compounded effects of concurrent infections on growth performance and their economic impact.

The impact of pH1N1v on growth performance could be exacerbated by concurrent infections with other respiratory

**Figure 2:** Fitted (predicted) values of overall feed intake from the fitted regression models. Boxplots are categorized on three levels by breed, infection status, and testing cohort by year. Boxes indicate the 25th percentile, median, and 75th percentile. Whiskers show the 10th and 90th percentiles.



pathogens,<sup>16,35-37</sup> potentially amplifying the economic losses beyond those caused by uncomplicated pH1N1v. This consideration is crucial for understanding the full scope of economic and health implications in pig farming, both in Norway and globally.

## Implications

Under the Norwegian conditions of this observational study:

- Breed-specific influenza resilience can guide breeding strategies for improved FCE.
- Breed predisposition affects economics by modulating OFI during influenza outbreaks.
- Genetic selection can mitigate the economic impacts of respiratory diseases.

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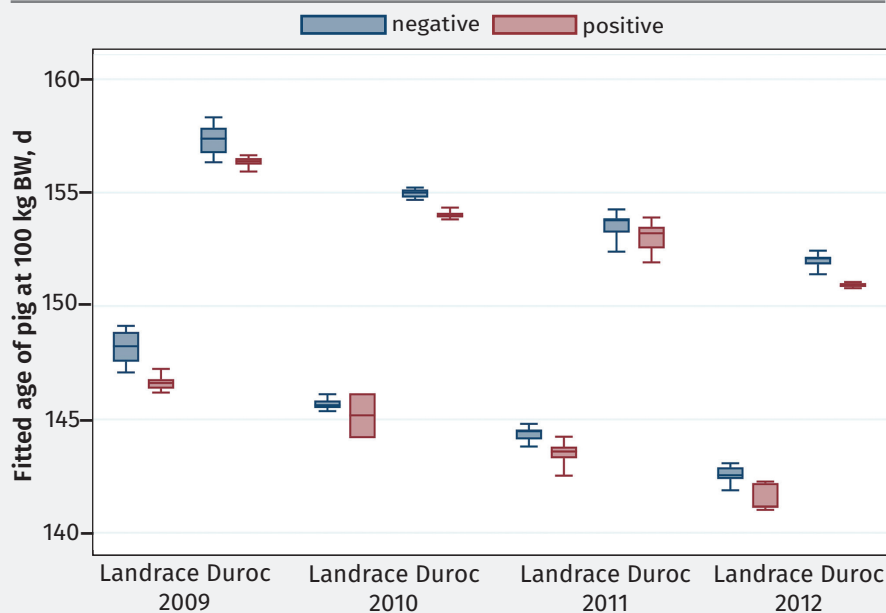
## Conflict of interest

None reported.

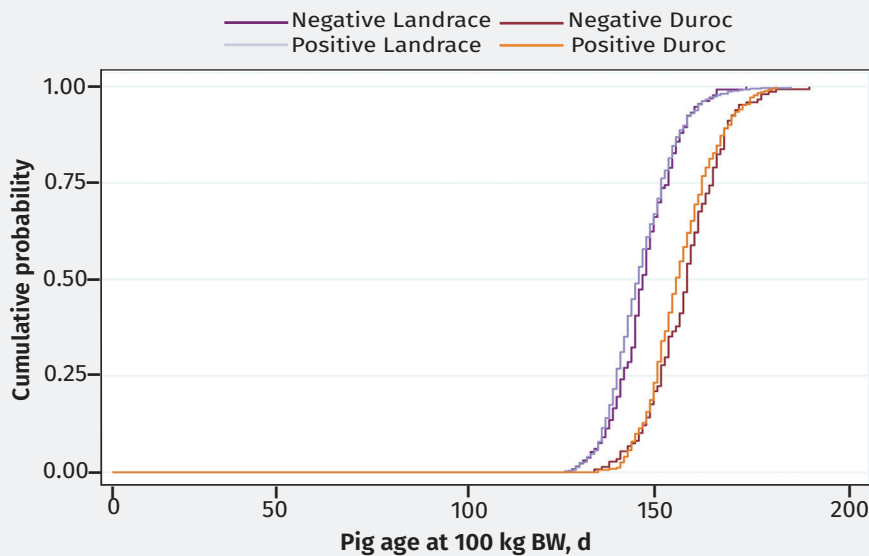
**Table 4:** Cox Regression Analysis Breslow for growth rate comparative analysis at 100 kg body weight

Predictors	Hazards ratio	SE	P	95% CI
<b>Breed#Infection status</b>				
Landrace#negative	1	-	-	-
Landrace#positive	1.136	0.072	.04	1.003 - 1.286
Duroc#negative	0.490	0.033	< .001	0.429 - 0.560
Duroc#positive	0.523	0.037	< .001	0.455 - 0.601
Birth date	1.0004	0.0001	< .001	1.0002 - 1.0005

**Figure 3:** Boxplots of predicted pig age at 100 kg body weight (BW) categorized by breed, infection status, and testing cohort by year. Boxes indicate the 25th percentile, median, and 75th percentile. Whiskers show the 10th and 90th percentiles.



**Figure 4:** Four distinct Kaplan-Meier Failure Curves for pig age at 100 kg body weight (BW) categorized by breed and infection status.



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## References

- Rowland RR, Lunney J, Dekkers J. Control of porcine reproductive and respiratory syndrome (PRRS) through genetic improvements in disease resistance and tolerance. *Front Genet.* 2012;3:260. <https://doi.org/10.3389/fgene.2012.00260>
- Lunney JK, Steibel JP, Reecy JM, Fritz E, Rothschild MF, Kerrigan M, Tribble B, Rowland RR. Probing genetic control of swine responses to PRRSV infection: Current progress of the PRRS host genetics consortium. *BMC Proc.* 2011;5(Suppl 4):S30. <https://doi.org/10.1186/1753-6561-5-S4-S30>
- Guan R, Wu J, Wang Y, Li X. Comparative analysis of productive performance and fattening efficiency of commercial pigs in China for two consecutive years. *Sci Rep.* 2023;13:8154. <https://doi.org/10.1038/s41598-023-35430-y>
- Rodrigues LA, Koo B, Nyachoti M, Columbus DA. Formulating diets for improved health status of pigs: Current knowledge and perspectives. *Animals (Basel).* 2022;12(20):2877. <https://doi.org/10.3390/ani12202877>
- Núñez P, Gol S, Reixach J, Casto-Rebollo C, Ibáñez-Escriche N. Incorporation of feeding behaviour traits to increase the genetic gain of feed efficiency in Pietrain pigs. *J Anim Breed Genet.* 2023;140:485-495. <https://doi.org/10.1111/jbg.12773>
- Pierozan CR, Agostini PS, Gasa J, Novais AK, Dias CP, Santos RS, Pereira Jr M, Nagi JG, Alves JB, Silva CA. Factors affecting the daily feed intake and feed conversion ratio of pigs in grow-finishing units: The case of a company. *Porcine Health Manag.* 2016;2:7. <https://doi.org/10.1186/s40813-016-0023-4>
- Soleimani T, Gilbert H. An approach to achieve overall farm feed efficiency in pig production: Environmental evaluation through individual life cycle assessment. *Int J Life Cycle Assess.* 2021;26:455-469. <https://doi.org/10.1007/s11367-020-01860-3>
- Godinho RM, Bergsma R, Silva FF, Sevellano CA, Knol EF, Lopes MS, Lopes PS, Bastiaansen JWM, Guimarães. Genetic correlations between feed efficiency traits, and growth performance and carcass traits in purebred and crossbred pigs. *J Anim Sci.* 2018;96:817-829. <https://doi.org/10.1093/jas/skx011>



9. Reyer H, Shirali M, Ponsuksili S, Murani E, Varley PF, Jensen J, Wimmers K. Exploring the genetics of feed efficiency and feeding behaviour traits in a pig line highly selected for performance characteristics. *Mol Genet Genomics*. 2017;292:1001-1011. <https://doi.org/10.1007/s00438-017-1325-1>
10. Wellenberg G, Bouwkamp F, Wolf P, Swart W, Mombarg M, de Gee A. A study on the severity and relevance of porcine circovirus type 2 infections in Dutch fattening pigs with respiratory diseases. *Vet Microbiol*. 2010;142:217-224. <https://doi.org/10.1016/j.vetmic.2009.10.003>
11. Van Alstine WG. Respiratory system. In: Zimmerman J, Karriker L, Ramirez A, Schwartz KJ, Stevenson GW, eds. *Diseases of Swine*. 10th ed. Wiley-Blackwell; 2012:348-362.
12. Torremorell M, Allerson M, Corzo C, Diaz A, Gramer M. Transmission of influenza A virus in pigs. *Transbound Emerg Dis*. 2012;59 (Suppl 1):68-84. <https://doi.org/10.1111/j.1865-1682.2011.01300.x>
13. Done SH. Environmental factors affecting the severity of pneumonia in pigs. *Vet Rec*. 1991;128:582-586. <https://doi.org/10.1136/vr.128.25.582>
14. Chauhan RP, Gordon ML. A systematic review analyzing the prevalence and circulation of influenza viruses in swine population worldwide. *Pathogens*. 2020;9:355. <https://doi.org/10.3390/pathogens9050355>
15. Deblanc C, Simon G. Involvement of swine influenza A viruses in the porcine respiratory disease complex. *Virologie (Montrouge)*. 2017;21:225-238. <https://doi.org/10.1684/vir.2017.0709>
16. Brockmeier SL, Loving CL, Nicholson TL, Palmer MV. Coinfection of pigs with porcine respiratory coronavirus and *Bordetella bronchiseptica*. *Vet Microbiol*. 2008;128:36-47. <https://doi.org/10.1016/j.vetmic.2007.09.025>
17. Euzéby JP. The immune system of swine respiratory tract - a review. *Rev Med Vet (Toulouse)*. 1993;144:665-681.
18. Boeters M, Garcia-Morante B, van Schaik G, Segalés J, Rushton J, Steeneveld W. The economic impact of endemic respiratory disease in pigs and related interventions - a systematic review. *Porcine Health Manag*. 2023;9:45. <https://doi.org/10.1186/s40813-023-00342-w>
19. Lee IK, Kye YC, Kim G, Kim HW, Gu MJ, Umboh J, Maaruf K, Kim SW, Yun C-H. Stress, nutrition, and intestinal immune responses in pigs - a review. *Asian-Australas J Anim Sci*. 2016;29:1075-1082. <https://doi.org/10.5713/ajas.16.0118>
20. Grontvedt CA, Er C, Gjerset B, Germundsson A, Brun E, Jorgensen A, Lium B, Framstad T. Influenza A(H1N1)pdm09 virus infection in Norwegian swine herds 2009/10: The risk of human to swine transmission. *Prev Vet Med*. 2013;110:429-434. <https://doi.org/10.1016/j.prevetmed.2013.02.016>
21. Hofshagen M, Gjerset B, Er C, Tarpai A, Brun E, Dannevig B, Bruheim T, Fostad IG, Iversen B, Hungnes O, Lium B. Pandemic influenza A(H1N1)v: Human to pig transmission in Norway? *Euro Surveill*. 2009;14:19406. <https://doi.org/10.2807/ese.14.45.19406-en>
22. Gjerset B, Er C, Lotvedt S, Jorgensen A, Hungnes O, Lium B, Germundsson A. Experiences after twenty months with pandemic influenza A (H1N1) 2009 infection in the naive Norwegian pig population. *Influenza Res Treat*. 2011;2011:206975. <https://doi.org/10.1155/2011/206975>
23. Er C, Skjerve E, Brun E, Framstad T, Lium B. Occurrence and spread of influenza A(H1N1)pdm09 virus infection in Norwegian pig herds based on active serosurveillance from 2010 to 2014. *Epidemiol Infect*. 2016;144:3148-3165. <https://doi.org/10.1017/S0950268816001424>
24. Britton AP, Trapp M, Sabaiduc S, Hsiao W, Joseph T, Schwantje H. Probable reverse zoonosis of influenza A(H1N1)pdm09 in a striped skunk (*Mephitis mephitis*). *Zoonoses Public Health*. 2019;66(5):422-427. <https://doi.org/10.1111/zph.12553>
25. Lium B, Zerihun A, Er C. The surveillance programme for specific virus infections in swine herds in Norway 2013. *Surveillance programmes for terrestrial and aquatic animals in Norway. Annual report 2013*. Norwegian Veterinary Institute; 2014.
26. Er C, Lium B, Tavornpanich S, Hofmo PO, Forberg H, Germundsson Hauge A, Grontvedt CA, Framstad T, Brun E. Adverse effects of influenza A(H1N1)pdm09 virus infection on growth performance of Norwegian pigs - a longitudinal study at a boar testing station. *BMC Vet Res*. 2014;10:284. <https://doi.org/10.1186/s12917-014-0284-6>
27. Grontvedt CA, Er C, Gjerset B, Germundsson A, Framstad T, Brun E, Jorgensen A, Lium B. Clinical impact of infection with pandemic influenza (H1N1) 2009 virus in naive nucleus and multiplier pig herds in Norway. *Influenza Res Treat*. 2011;2011:163745. <https://doi.org/10.1155/2011/163745>
28. Er C, Skjerve E, Brun E, Hofmo PE, Framstad T, Lium B. Production impact of influenza A(H1N1)pdm09 virus infection on fattening pigs in Norway. *J Anim Sci*. 2016;94:751-759. <https://doi.org/10.2527/jas.2015-9251>
29. Animal Welfare Act. Government Norway; 2009. <https://www.regjeringen.no/en/dokumenter/animal-welfare-act/id571188>
30. Er JC, Lium B, Framstad T. Antibodies of influenza A(H1N1)pdm09 virus in pigs' sera cross-react with other influenza A virus subtypes. A retrospective epidemiological interpretation of Norway's serosurveillance data from 2009-2017. *Epidemiol Infect*. 2020;148:e73. <https://doi.org/10.1017/S0950268820000643>
31. World Organisation for Animal Health. *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. 13th ed. World Organisation for Animal Health; 2024. <https://www.woah.org/en/what-we-do/standards/codes-and-manuals/terrestrial-manual-online-access>. Accessed September 29, 2024.
32. Burnham KP, Anderson DR, Huyvaert KP. AIC model selection and multimodel inference in behavioral ecology: some background, observations, and comparisons. *Behav Ecol Sociobiol*. 2011;65:23-35. <https://doi.org/10.1007/s00265-010-1029-6>
33. Hennig C, Graaf A, Petric PP, Graf L, Schwemmler M, Beer M, Harder T. Are pigs overestimated as a source of zoonotic influenza viruses? *Porcine Health Manag*. 2022;8:30. <https://doi.org/10.1186/s40813-022-00274-x>
34. Norwegian Institute of Public Health. Influenzasesongen 2022-2023. Ukerapport Influenza - Uke 40, 2022. [Influenza Season 2022-2023. Weekly Influenza Report - Week 40, 2022]. Accessed November 11, 2024. <https://www.fhi.no/contentassets/8916b2449e4c4ed8bb33fabeb980ff4/vedlegg/2022-40-influensaovervaking-2022-2023-uke-40.pdf>
35. Nicholson TL, Brockmeier SL, Loving CL. Contribution of *Bordetella bronchiseptica* filamentous hemagglutinin and pertactin to respiratory disease in swine. *Infect Immun*. 2009;77:2136-2146. <https://doi.org/10.1128/IAI.01379-08>
36. Loving CL, Brockmeier SL, Vincent AL, Palmer MV, Sacco RE, Nicholson TL. Influenza virus coinfection *Bordetella bronchiseptica* enhances bacterial colonization and host responses exacerbating pulmonary lesions. *Microb Pathog*. 2010;49:237-245. <https://doi.org/10.1016/j.micpath.2010.06.004>
37. Van Reeth K, Gregory V, Hay A, Pensaert M. Protection against a European H1N2 swine influenza virus in pigs previously infected with H1N1 and/or H3N2 subtypes. *Vaccine*. 2003;21:1375-1381. [https://doi.org/10.1016/S0264-410X\(02\)00688-6](https://doi.org/10.1016/S0264-410X(02)00688-6)

