

# Periweaning Failure to Thrive Syndrome (PFTS): Is There a Genetic Component?

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## Summary and Implications

Periweaning Failure to Thrive Syndrome (PFTS) is a serious and potentially fatal disorder with variable morbidity and mortality rates that have been reported in US and Canadian farms. A genetic basis has been hypothesized. To investigate what regions of the genome could be linked to that, a total of 70 affected and 37 non-affected piglets were genotyped with over 60,000 genetic markers to investigate genetic differences between the two groups. This allows for the identification of genomic regions that could be linked to resistance to the disease providing new insights and knowledge on the genetic basis of this syndrome.

## Introduction

Periweaning Failure to Thrive Syndrome (pronounced P-Fits), formerly known as 'Post-weaning Cachexic (or Catabolic) Syndrome is a disorder of unknown etiology, recognized in the United States and Canada since 2008. This clinical condition is characterized by anorexia, lethargy, and progressive debilitation of pigs within 2 to 3 weeks after weaning with morbidity and mortality rates variable between batches. Abnormal and compulsive oral behaviors (e.g. licking and chewing) normally accompanied these symptoms. Since no pathogens have been detected to date that explain causality, and the disease has not been successfully experimentally reproduced, it has been suggested that genetics may play an important role in PFTS. Moreover, the existence of a genetic predisposition has been detected at two studies and a preliminary genome analysis has been performed in Brazil.

## Materials and Methods

In this research, 107 pigs were collected in North America within 2-3 weeks after weaning from commercial farms. A total of 70 met the PFTS case definition (cases) while 37 were aged-matched pen mates (control). Cases and controls were balanced across the farms using a 2:1 ratio. Piglets were genotyped with the 80K SNP chip and 53,810 filtered autosomal genetic markers were used. Population

structure analysis was performed, showing that the samples collected have a similar genetic background between cases and controls. Then, three analyses looking at frequency of genetic marker differences were performed comparing cases and controls.

## Results and Discussions

Only moderate suggestive associations were reported with logistic regression analysis (Table 1), while genetic marker differences are represented in Fig. 1. A total of four regions not identified previously on chromosomes 1, 3 and 11 were concordant for at least two types of analyses. Genes included in these regions were investigated through a literature search and using the Human - Mouse: Disease Connection (HMDC) database. The search revealed that some of the genes are involved in energy homeostasis, caloric intake, and growth rate. With these limited analyses, we could not confirm major regions of difference for PFTS but we identified the presence of novel genomic regions that may be moderately associated with this syndrome.

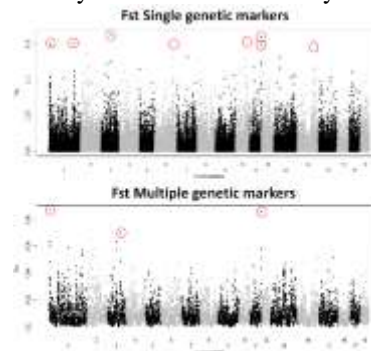


Fig. 1: Plot of the normalized  $F_{st}$  values, represented by dots. Each autosome has some different colors. Red dots represent the highest values.

CHR	BP	P
1	267892798	0.000197
14	135811847	0.0002326
17	18259899	0.0002466
1	4313782	0.0002653
11	86552038	0.000277
11	86552276	0.000277
3	74543589	0.000371

Table1: Chromosome and position of the SNPs with P value <0.0004 by logistic regression.

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