# A Summary of Current and Future Research on Boar Taint, a Meat Quality Issue

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**Introduction**

Boar taint is a meat quality issue that is characterized by an off-odour and off-flavour in meat products from uncastrated male pigs. This issue arises from excessive accumulation of the sex pheromone androstenone, as well as skatole, which is produced from the breakdown of tryptophan by the gut microflora (Zamaratskaia & Squires, 2009). The production of androstenone and skatole increases as the boar nears sexual maturity; therefore, in the swine industry, males are typically castrated shortly after birth to prevent the later development of boar taint. Castration has been recognized as an animal welfare concern, which has put pressure on pork producers to cease this procedure, but controlling boar taint without castration would also improve growth, increase lean yield, improve feed utilization and decrease feed consumption, which would improve profitability and enhance sustainability of swine production (EFSA, 2004). Due to these benefits, discovering a solution to this meat quality issue that eliminates the need to castrate is highly sought after, and as such many approaches have been taken. Below we highlight a few of our key contributions to this area of research, as well as the future directions of our research program.

**Key Contributions that Advanced our Understanding of Boar Taint**

The development of boar taint is dependent on the rate of synthesis and metabolism of androstenone and skatole, which is influenced by the genetics of the animal (Zamaratskaia & Squires, 2009). To characterize the metabolism of androstenone and skatole, we cloned key enzymes and expressed them in a cell line. When treated with androstenone or skatole, the cells expressing the key enzymes produced metabolites of these compounds that possessed a greater ability to be excreted than the parent compounds, which suggested these enzymes assist in reducing accumulation in the fat (Laderoute et al., 2018; Wiercinska et al., 2012). From this we have been able to identify key genes in the metabolic pathways of androstenone and skatole, and investigate their role in the development of boar taint.

Following metabolism in the liver, androstenone presumably enters the gastrointestinal tract, like many other sex steroids, where it may be excreted or acted on by the gut microflora, which allows it to be absorbed by enterocytes into the venous blood (Roberts et al., 2002). Our research group proposed that this process, known as the enterohepatic circulation, can be manipulated to increase the excretion of androstenone, which would reduce the amount that is available for accumulation in fat. We tested various non-nutritive binding agents and found that some, such as activated charcoal, could significantly reduce plasma androstenone concentrations, and following a recovery period where the additive was removed from the feed, initial plasma concentrations of androstenone could be restored (Fig. 1) (Jen & Squires, 2011). However, the reduction of plasma androstenone concentrations was more pronounced in some individuals than others, suggesting that genetics may influence the clearance of androstenone by the enterohepatic circulation amongst individuals.

Our lab and many others have contributed to the identification of key genes involved the metabolism of boar taint (Leung et al., 2010; Moe et al., 2008), which we have used to develop a set of approximately 1000 single nucleotide polymorphism (SNP) markers. We are currently working to gain a

better understanding of physiological processes and metabolic pathways that result in boar taint, which will allow us to identify additional candidate genes. From this, we can expand our SNP set by looking for SNPs within these key genes and assessing the function of these SNPs through functional mutation studies to determine their role in the development of boar taint. Defining the genetic predisposition of an animal to develop boar taint is important since the amount of boar taint varies across breeds and individuals.

**Future Directions**

Our future directions are aimed at identifying the causes of variation in boar taint among different breeds or individuals to develop individualized treatments to prevent boar taint. One key physiological process that we intend to fully characterize is the transport of sulfated androstenone in the plasma, as well as the subsequent uptake of this compound into the fat. Androstenone was previously found to bind non-specifically to the plasma protein albumin (Bone et al., 2018), but androstenone primarily exists in a sulfated state in the plasma. Therefore, characterizing the transport of sulfated androstenone will provide a greater understanding of how this compound is delivered to the fat where it accumulates and causes boar taint. Another area we wish to further research is factors affecting the production of skatole in the gut.

After characterizing these physiological processes, and identifying areas where variation exists between breeds and individuals, we will be able to identify additional key genes that are involved with the development of boar taint. Using the process described above, we will investigate SNPs that affect the expression of key enzymes, transporters or receptors involved in the development of boar taint, which we will then use to develop effective, individualized solutions to reduce androstenone and skatole accumulation in the fat based on the genotype of a particular animal.

Currently, there is a significant desire for animal production systems to function in a sustainable and welfare friendly manner. Our research aims to provide solutions to boar taint that do not require castration, allowing the swine industry to achieve these higher standards for sustainability and animal welfare without compromising meat quality.

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**Fig 1**

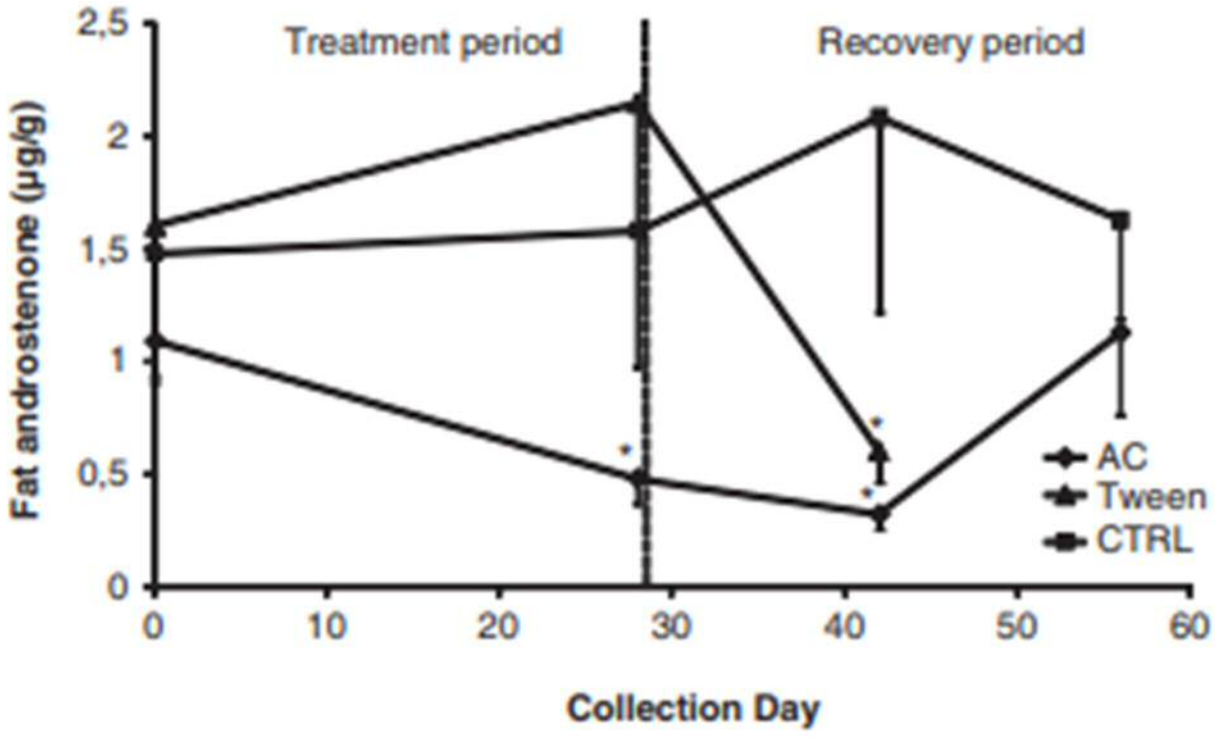


**Fig 1.** Profile of androstenone concentrations in fat of activated charcoal (AC ), Tween-60 – polyoxyethylene sorbitan monosterate (Tween) and a control (CTRL). Boars were treated from day 0 to 56 in the trial. Values are plotted as mean ± SE; * indicates values significantly different ($p < 0.05$) from values at day 0.