

Boar taint: interfering factors and possible ways to reduce it

Diana BILIĆ-ŠOBOT[1], Marjeta ČANDEK-POTOKAR[2], Valentina KUBALE[3] and Dejan ŠKORJANC[1]*

[1]*University of Maribor, Faculty of Agriculture and Life Sciences, Pivola 10, 2311 Hoče, Slovenia*
[2]*Agricultural Institute of Slovenia, Hacquetova ulica 17, 1000 Ljubljana, Slovenia*
[3]*University of Ljubljana, Veterinary Faculty, Gerbičeva ulica 60, 1000 Ljubljana, Slovenia*

ABSTRACT

Boar taint is an off-flavor in meat of entire male pigs caused primarily by high levels of two natural components, androstenone and skatole. Androstenone is a natural steroid produced, in parallel with anabolic testicular hormones, by the Leydig cells of the testes. It is metabolized in the adipose tissues and saliva glands, and also partly in the liver. The androstenone content in the adipose tissues depends mostly on genetic factors but also on the onset of puberty and weight of the animal. Skatole originates from the cell debris of the gut mucosa and it is produced by the intestinal bacterial metabolism of the amino acid L-tryptophan. Most of the skatole is absorbed into the blood and transported to the liver, where most of it is degraded and excreted in the urine. Skatole concentration may be influenced by genetic, feeding, and environmental factors. During the heating of pork meat, androstenone causesa urine-like odor and skatole fecal-like odor. In the present review, we discuss the metabolic (hormonal and enzymatic) factors, nutritional, and environmental factors that control the levels of skatole deposition in fatty tissuesas well as the genetic factors that controlling androstenone synthesis and deposition.

Key words: androstenone, skatole, anabolic hormone, cytochrome P450, dietary compound

INTRODUCTION

The presence of tainted meat was first described in 1936 by Lerche, an American researcher in the field of meat hygiene, and there after became the subject of numerous studies. It is primarily caused by increased concentrations of two compounds: androstenone (Patterson 1967) and skatole (Vold 1970, Walstra and Maarse 1970). Skatole and androstenone, when present in fatty tissues separately or together, increase the off-odor of pig meat (Zamaratskaia 2004). Skatole has a fecal-like odor and it is produced by the bacterial degradation of the amino acid L-tryptophan in the hindgut. Androstenone has a urine-like odor and it is a pheromone produced in parallel with testosterone as a part of the male sex hormone metabolism (Zammerini et al. 2012). Boar taint is an unpleasant odor, often perceived when cooking and eating meat from sexually mature male pigs. Thresholds for sensory perception of skatole and androstenone are considered to be between 0.20, 0.25 ppm, 0.5, and 1.0 ppm, respectively (Walstra et al. 1999). Due to its lower sensory threshold, the influence of skatole on boar taint sensory perception is higher at lower temperatures,whereas androstenone is more apparent during cooking or roasting (Agerhem and Tornberg 1996).

Factors that may affect the level of androstenone and skatole can be either animal related factors such as breed and genetics, sex, age, weight, and liver metabolism or external factors such as season of the year, rearing conditions, and feeding (Zamaratskaia 2004). The presence of androstenone and skatole in adipose tissues can be suppressed or reduced through surgical castration (with or without anesthesia), immunocastration (Batorek et al. 2012), or by supplementing the boars' diet with different compounds (Zamartaskaia and Squires 2008).

In the present review, biosynthesis, metabolism, and factors affecting skatole and androstenone levels in meat are described. Additionally, the possible mechanisms to reduce boar taint are discussed.

ANDROSTENONE

Androstenone is a steroid substance synthesized in the Leydig cells of the testes of uncastrated boars, parallel with other testicular steroids (Grower 1972, Kwan et al. 1985), and it is mainly accumulated in the fatty tissues. Androstenone is produced from the precursors pregnenolone and progesterone in a series of cascading reactions catalyzed by numerous enzymes, particularly cytochrome P450C17 (CYP17A1) and cytochrome b5 (CYB5) (Meadus et al. 1993, Davis and Squires 1999). Androstenone is a cholesterol derivative with the same structure as other steroids derived from cholesterol

**Correspondence to:*
E-mail: dejan.skorjanc@um.si

(4-ring and 17-carbon skeleton) (Chen et al. 2007). The steroid 16-androstene (C_{19}-Δ^{16}-steroid) is the naturally occurring androstenone pheromone in male mammals, which differs from sex hormones (Claus et al. 1994). In male pigs, androstenone is transported to the salivary gland where it is present together with other 16-androstene steroids. During sexual excitement, 16-androstene steroids are excreted into the boar saliva (Wysocki et al. 1989). The smell of androstenone has been described as sweaty, ammonia, dirty, parsnip, and silage like (Annor-Frempong et al. 1997).

Metabolism of androstenone is regulated by 3β- and 3α-HSD enzymes (3β and 3α-hydroxysteroid dehydrogenase enzymes) (Dufort et al. 2001, Doran et al. 2004, Sinclair et al. 2005). Moreover, conjugating enzymes from a second phase of the skatole metabolism, such as hydroxysteroid sulfotransferase (SULT2A1 and SULT2B1) and UDP-glucuronosyltransferase (UGTs) are also involved in the androstenone metabolism (Zamartsakaia and Squires 2008).

3β-Hydroxysteroid dehydrogenase/Δ^3-Δ^4-isomerase (3βHSD)

The 3βHSD (3β-hydroxysteroid dehydrogenase/Δ^3-Δ^4-isomerase) is essential for the degradation of androstenone (Payne and Hales 2004). In pigs, 3βHSD plays a key role in hepatic metabolism as it catalyzes the transformation of androstenone to β-androstenol (Doran et al. 2004, Sinclair and Squires 2005). It is also responsible for the oxidation and isomerization of Δ^5-3β-hydroxysteroid precursors into Δ^4-ketosteroids (Payne and Hales 2004). Immunohistochemical studies localized 3βHSD protein to the bile duct epithelium and its activity has been mostly associated with the microsomal fraction (Furster 1999). Porcine hepatic 3βHSD has been sequenced from a cDNA library from adipose tissue (Von Teichman et al. 2001). The enzyme is encoded by a single gene and its expression is regulated by tissue-specific mechanisms in hepatic and testicular tissues (Cue et al. 2007). The gene has been mapped to chromosome 4q16-4q21 (Von Teichman et al. 2001). As previously mentioned, 3βHSD is key for the biotransformation and metabolism of androstenone (Doran et al. 2004). In the microsomes of the liver, androstenone can be metabolized into 3β-androstenol and the metabolizing rate depends on 3βHSD protein expression (Doran et al. 2004, Sinclair and Squires 2005). Therefore, 3βHSD is considered a major candidate gene for boar taint (Payne and Hales 2004).

17β-Hydroxysteroid dehydrogenase (17βHSD)

Certain enzymes of the 17β-hydroxysteroid dehydrogenase family (17βHSDs) are important for steroidogenesis as they regulate the availability of both androgens and estrogens by interconversion of active and inactive forms of steroids (Baker et al. 2001). Eleven different 17βHSDs isozymes have been identified, some of which show a preference for oxidation, whereas others showa preference for reduction reactions. They catalyze many reactions, including interconversion between estrogen, estradiol, and testosterone; and conversion of 5α-androstanedione to DHT (5α-dihydrotestosterone). Among the several forms of 17βHSDs, at least three of them (types 1, 2, and 3) play an important role in the biosynthesis of potent gonadal steroids (Saloniemi et al. 2012). Furthermore, 17βHSDs are also involved in the biosynthesis of cholesterol and reduction of the estrogenic hormone estrone (Saloniemi et al. 2012).

Hydroxysteroid sulfotransferase (SULT2A1)

The hydroxysteroid sulfotransferase enzyme family (SULT2A1) is key for the testicular and hepatic metabolism of 16-androstene (Hobkirk 1985). SULT2A1 members are cytosolic proteins involved in catalyzing the conjugation of many steroids, bile acids, and xenobiotics. They utilize the donor molecule 3-phosphoadenosine 5-phosphosulfate (PAPS) for the transfer of a sulfate radical (SO_3) to a hydroxyl acceptor site. In steroids, hydroxyl groups are located at positions 3, 17, and 21of the steroid nucleus; thus, these are the most common locations for sulfoconjugation (Strott 2002). Steroid sulfotransferase enzymes can be found in many different organs such as liver, adrenal gland, ovary, and testis (Gasparini et al. 1976, Hobkirk 1985). SULT2A1 is a key enzyme in the Phase II of the hepatic metabolism of the 16-androstene steroids (Sinclair et al. 2005, Sinclair et al. 2006). Zamaratskaia et al. (2007) reported that the range of androstenone levels in the conjugated fraction was much lower than in the free fraction. Androstenone sulfation includes two steps: firstly, the convertion of androstenone 3-keto group to 3-enol form and, secondly, exchange of the 3-ol group with a sulfate group to form the sulfoconjugated androstenone (Sinclair et al. 2005).

Androgen and estrogen biosynthesis

Testosterone is the primary and most potent androgen in males (Chen 2007). Biosynthesis of androgen is low in younger pigs but it increases gradually together with other testicular steroids as they approach sexual maturity (Bonneau 1982, Zamaratskaia and Squires 2008). Activation of the hypothalamic-pituitary-gonadal axis at 2–4 weeks old boars results in an increase in the levels of circulating testicular steroids, including androgen (Bonneau 1982, Sinclair et al. 2001). All testicular steroid hormones, including 16-androstene, are synthesized from the common precursor pregnenolone (Brooks and Pearson 1986). Androgen biosynthesis occurs in Leydig cells (Figure 1) of the testes from the precursor pregnenolone and progesterone (Grower et al. 1972, Kwan et al. 1985).

Testosterone**,** under pulsatile LH stimulation**,** increases the

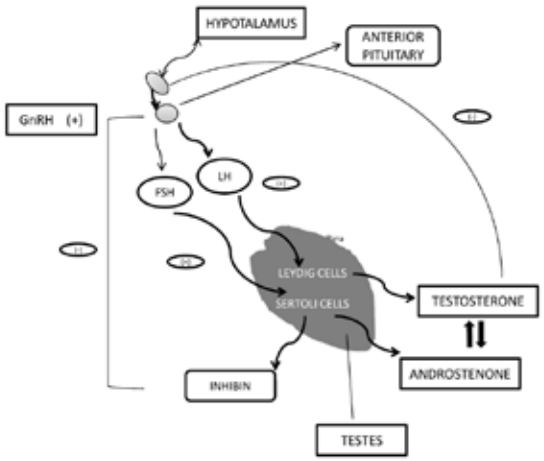

Figure 1. GnRH indirectly stimulate androstenone production by the testes

strength and muscle mass of animal (Wannenes et al. 2008). Testosterone is also involved in the regulation of testicular function while estrogens are involved in the control of GnRH and Leydig cells. These cells are important for development and maintenance of spermatozoa and spermatogenesis in some animal species (Carreau et al. 2011). Estrone sulfate (E1S) exists in large amount in the plasma of male pigs (Schwarzenberger et al. 1993, Zamaratskaia 2004). Sulfoconjugation of E1S occurs mainly in the liver. Authors have demonstrated that E1S can be converted to estradiol *in vivo* by the estrone sulfatase enzyme (Santner et al. 1990). Estradiol biosynthesis originates from cholesterol using the same metabolic pathways as testosterone, and androstenedion is a key intermediate (Payne and Hales 2004).

Biosynthesis of estrogens is controlled by the key enzyme aromatase P450 and the ubiquitous flavoprotein known as the NADPH cytochrome P450 reductase (Carreau et al. 2006). The main function of estrogens in testes is the production of spermatozoa which are under control of LH and follicle-stimulating hormone (FSH). In the testis, cytochrome P450 aromatase (P450arom) is present in the endoplasmic reticulum and it is involved in the irreversible transformation of androgens into estrogens (Carreau et al. 2003). The androgen/estrogen ratio is modified in germ cells that are important for the production of Sertoli cells, which in turn are the major source of estrogens (Papadopoulos et al. 1986.).

Suppression of androstenone accumulation

Androstenone levels in fatty tissues differ among breeds; Asian pig breeds accumulate higher amounts of androstenone than European or North American breeds (Chen et al. 2007). Among modern pig breeds, Duroc has been reported to have higher androstenone levels than Yorkshire, Landrace, or Hampshire breeds (Xue et al. 1996); whereas the Large White has higher levels than the Landrace breed (Willeke 1993). Thus, genetic selection might be a way to efficiently reduce androstenone levels. However, this approach can have a negative effect on reproductive and growth performances. For example, selection based on low levels of androstenone results in a delayed puberty in gilts (14 days) but not in boars (Sellier et al. 1989) and selection through three generations reduces the size of testes (Willeke 1993).

External factors are also important for the regulation of androstenone levels. In a study by Rasmussen et al. (2012) it was reported that pigs fed with 10% dried chicory root had a significantly lower concentration of androstenone in adipose tissues. It has also been pointed out that seasonal variations affect the concentration of androstenone and that its concentration decreases with increasing day length (Keller et al. 1997). In addition, androstenone can be influenced by social hierarchy and, thus, it is higher in dominant individuals (Giersing et al. 2000). Furthermore, production of anabolic steroids is also related to androstenone production. Its synthesis is low in younger piglets but increases during the onset of puberty along with the increased production of the testicular steroids: androgens and estrogens (Andresen 1976). The onset of puberty markedly differs between breeds as well as between individuals within each breed. Weight has also been suggested to affect the levels of androstenone. In the study of Zamaratskaia et al. (2005), androstenone levels did not differ between pigs slaughtered at 90 and 115 kg of live weight, whereas in the research of Chen et al. (2007), androstenone levels were higher in heavier pigs. These two studies were carried out on a cross between Swedish Landrace and Yorkshire pigs. In accordance with the results obtained by Chen et al. (2007), in Large White × Landrace × Duroc crossbred pig with live weight between 71 and 90 kg, androstenone levels were also higher in heavier pigs (Nicolau-Solano et al. 2007). One way to reduce androstenone levels is by using active immunization against GnRH, namely, Improvac™ vaccine (Pfizer Ltd., Parkville, Australia). Vaccination with Improvac™ is performed twice (4 weeks apart) in the growing/finishing pigs with a booster injection 4 to 6 weeks prior to slaughter (according to producer's recommendations). However, studies show that by slaughtering the pigs at a standard weight, the risk for boar taint can be reduced and the delay could be shorter than by vaccinating them (Kubale et al. 2013, Lelifano et al. 2013). Other disadvantages of the vaccine method are that the operator needs to vaccinate the pigs twice during the finishing period and that it is not positively received by consumers (see review Font-i-Furnols 2013).

SKATOLE

Skatole was first isolated from adipose tissue by Vold (1970) and Walstra and Maarse (1970), and it is perceived by most consumers as having a fecal-like odor. Skatole is formed from the amino acid L-tryptophan in the large intestine of pigs. Biosynthesis of skatole occurs in a two-step procedure; L-tryptophan is first converted to indole-3-acetic acid, which is subsequently converted to skatole (for a review on the topic, see Jensen and Jensen 1998). *Escherichia coli* and *Clostridium* spp. are responsible for the production of indole-3-acetic acid, whereas indole-3-acetic acid is converted to skatole by *Lactobacillus* sp. (Yokoyama et al. 1977, Honeyfieled and Carlson 1990, Deslandes et al. 2001). Thus, the production of skatole is primarily dependent on the availability of L-tryptophan and the activity of intestinal bacteria.

In ruminants, skatole causes an acute lung disease called acute bovine pulmonary edema and emphysema (Deslandes et al. 2001). Pigs, as non-ruminants, can tolerate high amounts of skatole without any effects to their health.

Absorption of skatole is very fast. The half-life of skatole in blood is 1h (Annor-Frempong et al. 1997), and 11h in muscle and adipose tissues (Agergaard and Laue 1993). Skatole is absorbed from the intestines into the blood stream and through *V. Porte* transferred to the liver (Jensen 2006). The liver metabolizes most of the skatole at a rate of 1L/h (Friis 1993) through the action of the hepatic cytochrome P450 family and sulfotransferase enzymes. The non degraded part accumulates in adipose tissues (Squires and Lundstrøm 1997, Babol et al. 1998, Zamaratskaia and Squires 2008), and only a small proportion is excreted in the urine (Frris 1993).

Skatole is metabolized in the liver to a variety of metabolites (Baek et al. 1997, Diaz et al. 1999). There are two phases for the metabolism of skatole in the liver (for a review on the topic, see Wesoly and Weiler 2012). Phase I involves the

addition of a hydroxyl group that can be used to attach a conjugate in Phase II. The main enzymes involved in this phase are CYP2E1 and CYP2A, which have been isolated from cytochrome P450 systems (Doran et al. 2002). These two enzymes together with aldehyde oxidases are thought to be the primary enzymes responsible for the oxidative metabolism of skatole (Babol et al. 1998, Diaz and Squires 2000). Identified metabolites from phase I include 3-OH-3-methylindolenine, which together with 3-methyloxindole is the most common skatole metabolite *in vitro* (Diaz et al. 1999). Some of the metabolites from phase I serve as substrates for conjugation with either sulfate or glucuronic acids in phase II (Babol et al. 1998, Diaz and Squires 2000). The enzymes involved in phase II are SULT1A1 (sulfotransferase) and UGT (urdine-di-phosphate-glucuronosyltransferase). Baek et al. (1997) reported that a high amount of 6-sulfatoxyskatole was found in the plasma of entire male pigs, which suggested a rapid metabolism and clearance of skatole *in vivo*. SULT1A1 enzyme is involved in this metabolism. In phase II, the water solubility of the skatole metabolites is increased, facilitating excretion via urine (Zamaratskaia et al. 2011).

Hepatic metabolism plays an essential role in the accumulation of skatole in fatty tissues (Zamaratskaia and Squires 2008). CYP2E1 and CYP2A enzymes, which are responsible for the production of skatole, are located in the liver, but can also be found in other tissues such as kidneys, gonads and brain (Rasmussen and Zamaratskaia). In male pigs, a high concentration of skatole in fatty tissues is associated with low enzymatic activity of both CYP2E1 and CYP2A (Squires and Lundstrøm 1997, Zamaratskaia et al. 2005).

ANDROSTENONE AND SKATOLE INTERACTION

In male pigs, increased androstenone accumulation in fatty tissues after puberty causes an increase in skatole concentration (Zamaratskaia 2004). Steroid levels increase after puberty and have a positive association with regulation of the rate of skatole metabolism (Babol et al. 1996). This suggests a relationship between androstenone and skatole at the level of hepatic metabolism, perhaps by sharing a common metabolic pathway. Other authors reported a relationship between androstenone and skatole through regulation of the expression of cytochrome P450 (Doran et al. 2002). Androstenone is a potential inhibitor of the expression of CYP2E1 (Tambyrajah et al. 2004) and CYP2A (Chen 2007) isozymes. Moreover, 17β-estradiol and testosterone demonstrated a direct inhibition of CYP2E1 and CYP2A activities on liver microsomes of male and females pigs (Zamaratskaia et al. 2007). Stimulation of pigs with hCG as administrator to increase the level of testicular steroids was shown to have impacts on skatole metabolism *in vivo* (Andresen 1975, Bonneau 1982, Chen et al. 2006). Zamaratskaia et al. (2006) reported that individual response of pigs to the effects hCG stimulation did not affect level of skatole.

POSSIBLE WAYS TO REDUCE SKATOLE LEVELS

Modification of intestinal apoptosis

The concentration of skatole in adipose tissues can be very variable and depends on several factors, such as the formation, absorption, metabolism, and accretion of skatole in adipose tissue (Wesoly and Weiler 2012). It has been shown that L-tryptophan derived from intestinal mucosa apoptotic cells because of dietary supplementation does not increase skatole production in the large intestine (Claus et al.1996). According to Claus et al. (1994), gut mucosa cell debris from the distal part of the gastrointestinal tract is the source of L-tryptophan for microbial skatole formation. Changing the availability of L-tryptophan in the large intestine may influence skatole accumulation (Claus et al. 1994). It has also been shown that the mitotic rate in the mucosa of the small intestine is elevated when feeding pigs with high-energy content diets (Raab et al. 1998). High-energy rations increase the rate of mucosa cell proliferation and counteract the apoptosis that leads to lower levels of skatole formation in the colon, and thus to higher concentrations in faces, peripheral blood plasma, and adipose tissues (Claus et al. 1996). Skatole formation is also influenced by endogenous anabolic hormones. Testosterone regulates cell renewal and apoptosis in the intestine when the food intake is lowered and, therefore, contributes to reduce skatole formation (Claus et al. 1994). In addition, there are other systematic intestinal hormones, such as IGF-I, that may lead to gut mucosal cell degradation by increasing the mitotic and apoptotic index in the gut mucosa (Calus et al. 1996).

It has also been suggested that high nutrient/energy levels increase androstenone and skatole levels by accelerating the sexual maturation of boars (Claus et al. 1994). Various compounds in the boars' diet have different effects on the reduction of skatole concentration (Table 1). Diets with a high content of fermentable carbohydrates escape digestion in the small intestine and, consequently, reduce the production of skatole (Jensen et al. 1997). It has been reported that other sugars, such as lactose, raffinose, and stachyose, as well as certain sugar alcohols are effective in reducing skatole levels in fat in ewes both *in vitro* and *in vivo* (Cummings and Englyst 1995). On the other hand, a diet with potato starch increases the production of butyrate in the colon and leads to a reduction in the formation of skatole (Claus et al. 2003). This effect was probably due to butyrate-dependent inhibition of apoptosis in the colon, which reduced the availability of cell debris to form microbial skatole (Claus et al. 1994, Claus et al. 2003). Butyric acid is the main energy source for the epithelial cells of the colon and represents 70% of the total energy for this type of cell (Smith et al. 1998). Claus et al. (2003) reported that feeding raw potato starch resulted in decreased colon crypt cell apoptosis (from 2.06 to 0.90), while the mitosis rate did not changed. In addition to the compounds mentioned, plant extracts such as Chinese medicinal herbs, feed additives derived from *Sanguinary Canadensis*, essential oil components from herbs, and species of tannin-rich preparations may offer a strategy to reduce skatole formation (Michiels et al. 2009, Blank et al. 2010).

Effect of hygiene, environmental temperature, slaughter weight, and type of feed on skatole reduction

Factors such as the temperatures in the pig pens and floor areas, the type of flooring, and stocking density, are important for the development of skatole. In astudy by Hansen et al. (1994), it was reported that high temperatures in the pen contribute to a decrease in the concentration of skatole. It has been demonstrated that skatole can be re-absorbed through pig skin and that approx. 40% of skatole is absorbed through the belly region of the pigs (Friss 1993).This problem mostly occurs in warm summer periods when the temperature of the feces and urine on the piggery floor may be higher than the ambient temperature, particularly when the pigs are lying in the excreta (Hansen et al. 1994). Other studies show that pigs on heavily soiled concrete floors with a mixture of feces and urine, compared with pigs reared on clean concrete or clean slatted floors, have increased levels of skatole in fatty tissues (Kjeldsen, 1998, Hansen et al. 1994, Hansen et al. 1995).

The age and the weight of the pigs also affect skatole levels. During the weaning period, the concentration of androstenone is low but increases steadily throughout puberty (Andresen 1976), concomitantly with the increase in the production of other testicular steroids (Claus 1997). After the weaning period, the fecal microflora in pigs is variable. Nevertheless, microbial populations stabilizes between 5 and 6 weeks post weaning (Zamartskaia 2004). The adaptation of the intestinal microflora increases probably after the weaning period due the intestinal cell turnover (Lanthier et al. 2006). In addition, the relationship between androstenone and skatole in the regulation of enzymes involved in skatole metabolism suggests that skatole is associated with puberty in pigs. Zamaratskaia et al. (2005) found a positive correlation between the testicular hormones testosterone and estrone sulfate, and the level of skatole suggesting that these compounds have an influence on the metabolism of skatole. In addition to age, slaughter of male pigs at a weight below 100 kg could be used to avoid high concentrations of skatole in the fatty tissues.

The type of feeding can also affectively reduce the level of skatole. The use of wet feed in boars diminished the level of skatole in back fat in comparison to dry feed (Andresson et al. 1997, Kjeldsen and Udesen 1998). In contrast, other studies showed that liquid feed had a weak or no effect on the concentrations of skatole in the fatty tissues of boars (Jensen at al. 1998, Hansen et al. 2000). Twelve hours fasting before slaughter also decreased the concentration of skatole in males (Ambrosen 1993).

POSSIBLE WAYS TO REDUCE SKATOLE LEVELS USING DIFFERENT FEED COMPONENTS IN THE DIET

Starch and resistant starch amounts

Raw potato starch (RPS), like inulin and fructooligosaccharide, is not digestible in the small intestine, and thus it is fermented by intestinal bacteria to yield short chain fatty acids (Claus et al. 2003) which might causean analogous effect to L-tryptophan biotransformation (Chen et al. 2007). Lösel and Claus (2005) investigated three groups of pigs fed with 20%, 30%, and 40% of RPS in their diet. They found that 40% of RPS in the diet significantly reduces the level of skatole formation and accumulation in the intestine and adipose tissues, respectively. The same authors also demonstrated a positive effect when feeding pigs with 30% RPS. In these pigs, skatole formation in the large intestine was also reduced and, consequently, its concentration in the boar tissues and gilts (Lösel et al. 2006). These results are in contrast with those reported by Øverland et al. (2011), where 25% of pelleted RPS in the diet did not have any effect on skatole formation in the hind-gut of pigs. This lack of effect can be explained by the high temperatures used in the pelleting process (83 °C), which cause the gelatinization of potato starch (RPS is fully gelatinized at 69°C) and led to less starch being available for the fermentation in the hind-gut (Øverland at al. 2011). A significant reduction in skatole levels was achieved in the hind-gut and adipose tissues even with boar diets with lower amounts of RPS (20%) (Andersson et al. 2005, Zamaratskaia et al. 2005).

A study has found that fructooligosaccharides at concentrations of 0.5%, 1.0%, and 1.5% can reduce the conversion of L-tryptophan *in vitro* (Xu et al. 2002). In another study, Jensen et al. (1997) investigated the effect of 10% sugar beet pulp, coconut cake, palm cake, lupines, barley hull meal, and fructooligosaccharides on the concentration of skatole in blood. They found that lupines and fructooligosaccharides caused a significant reduction. However, yeast from breweries was found to be a feeding component that elevates skatole concentration in the hindgut and backfat (Jensen et al. 1995).

Non-digestible carbohydrates

Chicory (*Cichorium intybus* L.*)* is a bioactive crop that may have a positive effect on the reduction of the skatol concentration in blood and back fat of boars (Hansen et al. 2008). Several studies have shown that fermentable carbohydrates, such as the inulin obtained from chicory, at dietary inclusion levels of 5% (Rideout et al. 2004) or 14% were effective in reducing the concentration of skatole in the hindgut. Hansen et al. (2006) found that 25% of crude or dried chicory in boars' diet reduces the skatole level in blood and backfat in a period of 1week. Feeding of boars with lower amounts of chicory fora longer period is likely to have the same effect. Similarly, Zammerini et al. (2012) demonstrated that feeding boars with 9% chicory 2 weeks before slaughter reduces skatole on the backfat, particularly on the level as castrated boars. Feeding male pigs with even lower amounts (3%, 6%, and 9%) of chicory inulin effectively reduced skatole levels in the digestive organs and adipose tissues (Kjos et al. (2010). A possible explanation is that chicory inulin reduces the number of *Enterococcus* spp. which are important for the production of amino acid L-tryptophan in the colon.

Table 1. Feed components used to influence skatole formation in the blood, small intestine and adipose tissue.

					Reduction of				
% ratio	*Feed component*	*Weeks*	*Pig breed*	*BW, kg*	*skatole in blood*	*skatole in small intestine*	*skatole in adipose tissue*	*indole*	*References*
58	Row potato starch	2°	LN × (Yorkshire × Duroc)	100.3	n.d.	+	+	n.d.	Claus et al. 2003
20,30,40	Raw potato starch	2-3 ◆	(Pietrain × (LW × Danish LN)	108.8	n.d.	+	+	n.d.	Losel and Calus. 2005
30	Resistant potato starch	4◆	Pietrain (LW × German LN)	105-115	+	+	n.d.	n.d.	Losel et al. 2006
60	Raw potato starch	2*	LN × Swedish Yorkshire	115	+	+	+	n.d.	Chen et al. 2007
30	Row potato starch	1*	Large White	100.5	+	n.d.	+	-	Pauly et al. 2008
10 + 5	Raw potato starch + wheat bran	6•	Pietrain × Rattlerow Seghers sows	100.7	n.d.	n.d.	-	n.d.	Aluwé et al. 2009
10	Raw potato starch	6•	Pietrain × Rattlerow Seghers sows	100.7	n.d.	n.d.	-	n.d.	Aluwé et al. 2009
5,10,15,20	Raw potato starch	4*	(Norwegian LN × Yorkshire) × Norwegian LN	100.5	n.d.	+	+	-	Øverland et al. 2011
20	Raw potato starch	2*	Swedish Yorkshire × LN	115	n.d.	+	+	n.d.	Zamaratskaia et al. 2005
25	Chicory dried	3-9*	Danish (Duroc × zigzag) × (LN × LW)	110-124	+	n.d.	+	+	Hansen et al. 2006
10-13.3	Chicory dried	1*	Danish (LN × LW) × (Duroc LN)	100.5	-	n.d.	-	n.d.	Hansen et al. 2008
9	Chicory dried	1*	(Norwegian LN × Yorkshire) × (Norwegian × Duroc)	111.7	-	-	n.d.	-	Vhile et al. 2012
10	Chicory dried	2*	LN × (Yorkshire × Duroc)	130	n.d.	-	-	n.d.	Rasmussen et al. 2012

9	Chicory inulin	4◦	(LN × Yorkshire) × LN	100.5	+	+	+	n.s.	Kjos et al. 2010
3,6,9	Chicory inulin	4◦	(Norwegian LN × Yorkshire) × Norwegian LN	100.5	+	+	+	n.s.	Øverland et al. 2011
5	Chicory inulin	2◦	Yorkshire	100.5	+	n.d.	n.d.	n.d.	Riduet et al. 2004
4,8,12	Chicory dried root extract	2◦	(White LN × White Duroc) × Pietrain	98.0	n.d.	+	+	n.d.	Zammerini et al. 2012
25	Blue lupine	1◦	Danish (LN × LW) × (Duroc LN)	100.5	+	n.d.	+	n.d.	Hansen et al. 2008
10	Blue lupines	6•	Pietrain × Rattlerow Seghers sows	100.7	n.d.	n.d.	+	n.d.	Aluwé et al. 2009
12.2, 8.1	Jerusalem artichoke	1◦	(Norwegian LN × Yorkshire) × (Norwegian × Duroc)	111.7	+	+	n.d.	+	Vhile et al. 2012
30	Wheat bran	Fattening◦ period	Pietrain × Seghers hybrid	100.1	n.d.	n.d.	-	n.d	Van Oeckel et al. 1998
15	Soybean hulls	Fattening ◦period	Pietrain × Seghers hybrid	100.1	n.d.	n.d.	-	n.d	Van Oeckel et al. 1998
15	Sugar bleep plump	Fattening◦ period	Pietrain × Seghers hybrid	100.1	n.d.	n.d.	-	n.d	Van Oeckel et al. 1998
5	Beneo	6•	Pietrain × Rattlerow Seghers sows	100.7	n.d.	n.d.	-	n.d.	Aluwé et al. 2009
1	Clinoptilolite	6•	Pietrain × Rattlerow Seghers sows	100.7	n.d.	n.d.	-	n.d.	Aluwé et al. 2009
0.85	Formic acid	During the ◦whole experiment	(Norwegian LN × Yorkshire) × Norwegian LN × Duroc)	113.2	+	n.d.	-	n.d	Øverland et. al. 2008
0.85	Benzoic acid	During the ◦whole experiment	(Norwegian LN × Yorkshire) × Norwegian LN × Duroc)	113.2	+	n.d.	-	n.d.	Øverland et. al. 2008
0.85	Sorbic acid	During the◦ whole experiment	(Norwegian LN × Yorkshire) × Norwegian LN ×Duroc)	113.2	-	n.d.	-.	n.d.	Øverland et. al. 2008

+ = effect of feed component detected (skatole concentration decreased); - = no effect of feed component; n.d.= not defined; BW = final body weight; LN = Landrace; LW = Large White; ⬥ = Barrows and gilts; • = Entire male pigs and barrows; ◦ = Entire male pigs; ∘ = Castrated male pig

Organic acids

Supplementing diets with different organic acids reduces the number of coliforms, enterococci, and lactic acid-producing bacteria in the gastrointestinal tract (Wesoly and Weiler 2012). It has been demonstrated that organic acids with weak antibacterial properties (1.0% formic, 0.85% benzoic, 0.85% sorbic and 0.85% pure acids) decrease the concentration of skatole (Øverland et al. 2008).

GENETIC EFFECTS

QTLs and candidate genes for androstenone

Genetic variation exists among breeds and within breeds for fat androstenone level. Quantitative trait loci (QLT) are described by the position of markers that are in linkage disequilibrium and most closely associated with the differences in the trait phenotype under investigation. The mapping data of QTL involved in boar taint has demonstrated its multifactorial nature (Robic et al. 2008). Previous studies have reported a high heritability (h^2) for androstenone level, i.e., from 0.25 to 0.88 with an average value of 0.56 (Sellier et al. 1989). The first QTLs analysis of androstenone levels in fatty tissues was performed on a three-generation experimental crossing between Large White and Meishan breeds (Quintanilla et al. 2003). One major candidate gene for a QTL was selected on the SSC7 pig chromosome but no polymorphism responsible for androstenone variability was found (Quintanilla et al. 2003). In this study, another two QTLs on SSC3 and SSC14 were also linked to androstenone. Other authors have reported QTLs for androstenone and boar flavor on SSC6 (Lee et al. 2005). Interestingly, Varona et al. (2005) failed to detect any QTLs for androstenone. Gregersen et al. (2012) estimated around 30.000 SNPs segregating in 923 boars from three Danish breeds: Duroc, Landrace and Yorkshire. SNPs were used for detection of boar taint compounds (androstenone, skatole and indole). A total of 46 QTLs were identified for all three compounds, where only 8 of them were significant for androstenone on SSC 2, 3, 5, 6, 7, 11, 12 and 14. Some single-nucleotide polymorphisms (SNP) in candidate genes encoding several key enzymes involved in the metabolic pathway of boar taint have also been investigated (Skinner et al. 2005, Peacock et al. 2007). It was found that SNPs in the 5'-untranslated region of. *CYB5* were associated with a decrease in androstenone synthesis (Lin et al. 2004).

QTLs and candidate genes for skatole

The heritability values for skatole varies widely among breeds (from 0.34 to 0.19). For the breeds Landrace and Duroc h^2 is estimated to be 0.23 and 0.36, respectively (Tajet et al. 2006). A QTL for skatole has been mapped to SSC14 at 61 cM, spanning the markers between S0007 and SW761 (Lee et al. 2005). The same authors also detected another QTL for skatole mapped to SSC6 at position 70/80 cM. In the study of Gregersen et al. (2012) two QTLs regions for skatole were indentified on SSC 3 and 9, while in the study of Grindflek et al. (2011) QTLs were found on SSC 1, 2, 3, 7 and 15. There are few studies regarding the SNPs in genes and metabolic enzymes that might affect skatole level in fatty tissues (Zamarastakaia and Squires 2008). In porcine liver, CYP2E1 plays an additional important role in catalyzing the first step of skatole degradation and low skatole in fatty tissues has been associated with high levels of CYP2E1 in animals obtained from an F_4 wild pig cross (Doran et al. 2002, 2004). To date, an SNP in the promoter region of CYP2E1 (Skinner et al. 2005) and other SNPs in the coding region of CYP2E1 and SULT1A1 enzymes (Lin et al. 2005) have been related to the skatole deposition in fatty tissues (Lin et al. 2005). Polymorphisms in candidate genes for boar taint vary among the hybrid breeds of Landrace, Yorkshire, and Duroc (Quintanilla et al. 2003, Lee et al. 2005, Peacocak et al. 2007, Zamarastakaia et al. 2008). Further research on the genetics of boar taint should consider the effects of the SNPs and QTLs of the candidate genes involved in the relevant metabolic pathways.

OTHER BOAR TAINT COMPOUNDS

Indole is produced by bacteria in the colon from the breakdown of the amino-acid L-tryptophan (Jensen et al. 1994). Many bacteria are able to metabolize L-tryptophan to indole and indole acetic acid (IAA), whereas only a few specialized gut bacteria (mainly from the genera *Clostridium*and *Bactericides*) can catalyze the steps from IAA to skatole. It has been demonstrated that pH is important for the formation of indole and skatole. Skatole-producing bacteria are favored under acidic conditions at pHs below 5.0, while indole-forming bacteria predominate at pH 8.0 (Yokoyama and Carlson 1979, Jensen et al. 1995). Environmental/hygiene factors in the pens are very important for controlling skatole levels. Skatole can be transferred among pigs in the pen by feces. A dirty penis can also be an indicator of indole accumulation in the fatty tissues (Hansen et al. 1995). Other indolic compounds such as indole-3-methanol, indole-3-propionic acid, indole-3-acetonitril, and indole-3-ethanol have also been found in the fatty tissues of boars (Hansen-Moller et al. 1998) and can also be candidates for boar taint. Other potential boar taint substances are 4-methylphenole (p-cresol) (Patterson, 1967) and 4-phenyl-3-buten-2-one (Rius and García-Regueiro, 2001), which are also found in the fatty tissues of boars. There are also several androstenone steroids, found in the fat of pigs, that influnce boar odor with a sensory perception threshold: 5α-androst-16-en-3α-ol (0.9 μg/g); 5α-androst-16-en-3β-ol (1.2 μg/g); 5α-androst-16-en-3-one (0.6 μg/g); 5,16-androstadien-3β-ol (8.9 μg/g) and 4,16-androstadien-3-one (7.8 μg/g) (Rius et al. 2005). The same authors also reported that aldehydes and short chain fatty acids can also contribute to the development of boar taint.

CONCLUSIONS

Dietary manipulation by adding different supplements to the boar's diet, such as row potato starch, chicory inulin, jerusalem artichoke, and blue lupine, can reduce the risk of high skatole levels in blood, small intestine, and adipose tissues by reducing skatole absorption through the

intestinal walls. In the liver, cytochrome P450 is responsible for the interaction between skatole and androstenone. The relationship between the testicular synthesis of androstenone and the liver metabolism of skatole seems to be responsible for the levels of both compounds in the fatty tissues. Although a number of candidate genes for boar taint have been identified, new candidate gene identification as well as new marker development is required to reduce the presence of boar taint in meat. Similarly, future research is also still necessary in the field of feed additives in diets.

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Vplivi na vonj po merjascu in možnosti kako ga zmanjšati

IZVLEČEK

Vonj po merjascu je povzročen zaradi visoke ravni dveh naravnih substratov: androstenona in skatola. Androstenon je naravni steroid, ki se proizvaja vzporedno s testikularnimi hormoni v Leydigovih celicah testisov. Androstenon se metabolizira v maščobnem tkivu in žlezah slinavkah ter deloma tudi v jetrih. Vsebnost androstenona v maščobnem tkivu je odvisna predvsem od genetskih faktorjev, kot tudi od vstopa v puberteto in telesne mase merjasca. Skatol izvira iz ostankov celic črevesne sluznice in je proizveden s črevesno bakterijsko presnovo amino kisline L-triptofana. Večina skatola se absorbira v kri in je prenesen v jetra, kjer se v njih razgradi, preostanek pa se izloči z urinom. Na vsebnost skatola lahko vplivajo genetskifaktorji, krmljenje in okoljski dejavniki. Med segrevanjem svinjskega mesa tvori androstenon specifičen vonj po urinu, med tem ko skatol tvori vonj po blatu. V preglednem članku smo predstavili presnovne (hormonske in encimske), prehranske in okoljske dejavnike, ki nadzorujejo raven skatola shranjenega v maščobnih tkivih, kot tudi o genetskih dejavnikih, ki nadzorujejo sintezo in nalaganje androstenona.

Ključne besede: androstenon, skatol, anabolični hormon, citokrom, P450, prehranski dodatki