

Raising intact male pigs for meat: Detecting and preventing boar taint

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Summary

Although there are several advantages to raising intact male pigs instead of castrates, boar taint — an unpleasant odor that emanates from boar fat when it is heated — is a potential problem with rearing boars for pork. Two groups of compounds are considered primarily responsible for boar taint: 16-androstenes (mainly 5 α -androst-16-en-3-one) and skatole. The 16-androstene steroids are largely secreted in the testes and then transported to fat tissues. Skatole is produced in the intestine by bacteria and also stored in fat tissues after absorption. Chemical and sensory tests are commonly used to detect boar taint in pork. Chemical tests typically assess tissue concentrations of the compounds associated with taint. Threshold values are proposed for fat concentrations of androst-16-en-3-one and skatole, 1.0 ppm and 0.25 ppm, respectively. Sensory tests classify boar carcasses into either tainted or untainted categories according to test criteria assessed by human evaluators. Human perception of androst-16-en-3-one is under genetic control. Approximately half of adults are not sensitive to androst-16-en-3-one. Men are less sensitive than women. Human perception of androst-16-en-3-one changes with age and can be induced. Since sensory panels are sensitive, trained, and frequently exposed to taint compounds, prevalence of tainted carcasses detected by the panels is much higher than that detected by consumers. Several methods have been studied to identify and prevent tainted carcasses. Genetic selection, reducing slaughter weight, immunization of boars against gonadotropin releasing-hormone (GnRH), and injection of GnRH agonist have resulted in decreased fat concentrations of androst-16-en-3-one.

Although there are several advantages to rearing entire male pigs for meat,¹ the possibility of boar taint—an unpleasant odor that emanates from the fat when the pork is heated—is one potential difficulty with the practice. Entire male pigs can be reared for pork only when tainted carcasses are prevented from entering the fresh meat market. For this article, we review boar taint and its compounds, factors affecting boar taint, and methods to prevent taint.

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Chemical causes of boar taint

The smell and/or taste of boar-tainted meat has been described variously as an “off” or “boar” odor;² onion-like, perspiration-like, or urine-like;³ like perfume, wood, musk, or “Ivory” soap;⁴ sweet, fruity, ammonia-like, and animal-like;⁵ and fecal or bitter.⁶ Williams, et al.,⁷ found that the odor was sex-dependent. In their study, 36% of intact males possessed the odor, but only 1% of sows, 5% of gilts, and 5% of barrows did. It was not until 1968 that one of the contributory compounds, 5-androst-16-en-3-one (5-androst-16-en-3-one), was isolated and this pork characteristic was labeled “boar taint.”^{8–9}

Attempts to identify chemical compounds responsible for boar taint in pork were initiated by Lerche,¹⁰ who described the parotid gland as processing the bad odor. Prelog, et al.,¹¹ isolated

- C₁₉- Δ ¹⁶-steroid,
- 5 α -androst-16-en-3 α -ol (5 α -androst-16-en-3-one), and
- 5 α -androst-16-en-3 β -ol (5 β -androst-16-en-3-one),

and described them as possessing a musk-like odor. Their subsequent studies found that

- ketone,
- androstadienone, and
- 5 α -androst-16-en-3-one

possessed urine-like or perspiration-like smells.¹²

Craig and Pearson¹³ reported that sex odor in pork is only found in the fatty tissues. Patterson demonstrated that

- 5 α -androst-16-en-3-one is present in boar fat,⁸ and
- 3 α -hydroxy-5 α -androst-16-en-3-one is present in boar salivary glands,¹⁴

suggesting that they are responsible for the odor.

Beery, et al.¹⁵ confirmed that both the

- 3-keto, and
- 3-hydroxy steroids

are involved in the odor.

Furthermore, Thompson, et al.,¹⁶ found that

- other C₁₉ Δ ¹⁶ steroids,
- 5 β -androst-16-en-3-one (5 β -androst-16-en-3-one), and
- 5 α - and 5 β -androst-16-en-3-ols

also contribute to sex odor. Subsequently, 5 α -androstenone, 5 α -, and 5 β -androstenols were found in the testes,^{17,18} fat,^{4,19,20} submaxillary gland and saliva,^{14,18} and parotid gland²¹ of boars.

Although the chemical compounds responsible for boar taint are under study, it is generally considered that 16-androstenes, a group of steroids in which 5-androstenone is a main component, are primarily responsible for boar taint.^{9,22,23} Skatole (3-methylindole), which has an intense fecal odor and bitter taste, also has been implicated in causing boar taint.⁶

16-androstenes and 5 α -androstenone

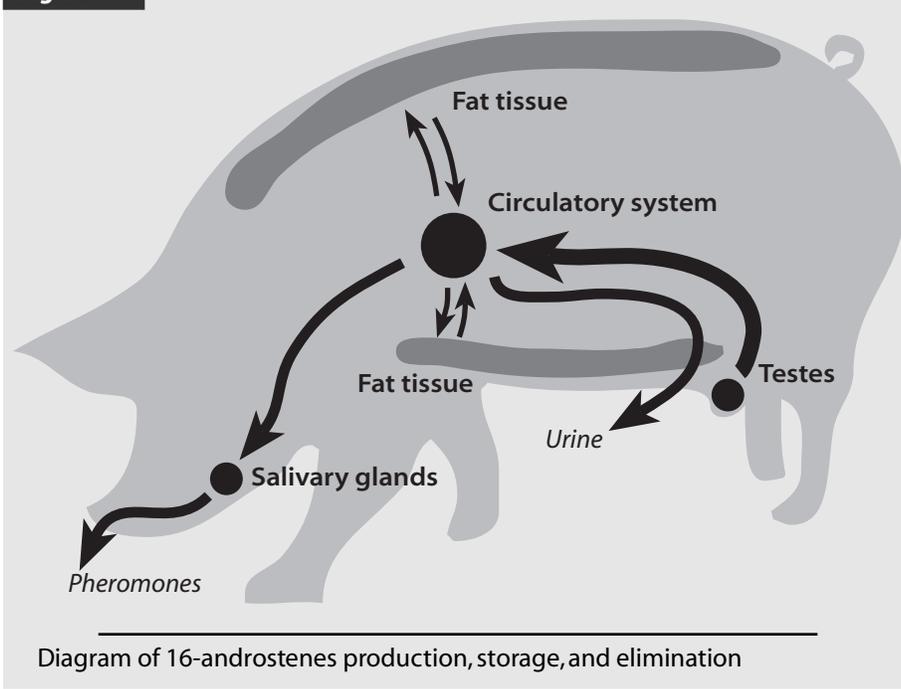
Physiology of 16-androstenes

The 16-androstenes are synthesized primarily in Leydig cells of boar testes along with other androgens and estrogens, with lesser contributions from the adrenals.^{9,24} The 16-androstenes produced in the testes are released into the systemic circulation via the spermatic vein.^{25,26} Due to their hydrophobic property, circulating 16-androstenes are then transported to fat tissue where they are stored (Figure 1).^{22,23} The apparent half-life of fat androstenone ranges from 4–14 days in boars of 100 kg (220 lb) of liveweight.^{27,28} Androstenone storage in fat is reversible. Castrating mature boars results in a progressive decline in serum and loin fat concentrations of androstenone.^{27,29,30} Serum androstenone concentrations decline more slowly than does testosterone because of androstenone release from fatty tissue.²¹

Androstenone and other 16-androstene steroids are probably catabolized in the liver.^{31,32} In young boars (≤ 100 kg, 220 lb), androstenone is eliminated mainly through the urine, in the form of 5 β -androstenol, and in trace amounts through feces.³³ In adult boars, 5 β -androstenol and, to a lesser extent, 5 α -androstenol are the only 16-androstenes that are eliminated in urine.^{26,34,35}

The 16-androstenes in the circulatory system also are transported to the salivary glands, where they function as pheromones and sexual attractants during the mating process.^{9,31} Boar salivary glands contain high concentrations of 5 α -androstenone, 5 α -, and 5 β -androstenols.¹⁴ When a mature boar is aroused by the presence of an estrous female or an unfamiliar boar, he champs copious amounts of frothy saliva. This excessive salivation provides a medium for the release of large amounts of 16-androstenes into the environment. These odorous steroids, in particular, 5 α -androstenol and 5 α -androstenone, act as signaling pheromones and trigger the mating stance in the estrous female, or indicate to the other boar that his status is being challenged.^{36,37} Exposing prepubertal gilts to these pheromones also advances the onset of puberty.³⁸

Figure 1



These compounds also contribute to dominance hierarchies in the agonistic behaviors of pigs. Aggressive boars have a higher concentration of salivary androstenone than control boars.³⁹ Spraying androstenone around newly regrouped growing pigs reduced fighting.⁴⁰

Factors influencing 16-androstenes

Genetics

Distinct breed differences in taint compounds and taint were reported. Piétrain boars had higher concentrations of androstenone than Belgian Landrace boars.⁴¹ Landrace crossbred boars had higher fat concentrations of androstenone compared to Large White crossbreeds at a liveweight of 105 kg (231 lb).⁴² Correspondingly, there was a higher proportion of tainted carcasses from Landrace crossbred boars. We found differences in concentrations of fat 16-androstenes among Duroc, Hampshire, Landrace, and Yorkshire breeds.⁴³ The Landrace breed has the lowest average salivary concentrations of 16-androstenes and fat concentrations of androstenone and skatole.

Genetic selection can effectively reduce the presence of taint compounds. Heritability of androstenone is estimated to range between 0.25–0.81 in Danish Landrace^{44,45} and 0.61–0.87 in Large White boars.^{46,47} Estimated heritability of boar taint intensity ranged from 0.13–0.54.^{48,49} The mean concentration of fat androstenone was reduced from 0.53–0.12 ppm after selecting for low androstenone for three generations.⁵⁰

Genetic selection against androstenone may adversely affect reproductive or growth performance. A single-generation selection for low androstenone resulted in delayed puberty in gilts (14 days), but not in boars.⁴⁷ Testicular size was reduced by selection for three generations.⁵¹ Selection for high or low androstenone concentrations for five generations demonstrated that growth rate was highly correlated with a

high-androstenone line in which there was more fat tissue in female carcasses.⁵²

Age or body weight

Age or liveweight are associated with fat androstenone concentrations in young boars. Testicular androstenone is undetectable at birth and then gradually increases with age and body weight. Androstenone increases dramatically in both testes and fat around the age of puberty,^{18,53} particularly during the 100- to 130-kg (220–287 lb) growth period with a concomitant increase in taint intensity.⁵⁴ An increase in androstenone with age varies among animals. Some boars do not even exhibit a significant rise in fat androstenone concentrations at puberty.^{18,50,55,56}

It is clear that fat concentrations of androstenone and 16-androstene steroids are low when animals are slaughtered before they reach sexual maturity. In Denmark and other European countries, slaughter weight is commonly under or around 100 kg (220 lb), the weight at which pigs reach puberty. Therefore, skatole might be a primary factor responsible for boar taint.⁵⁷ Relative to these countries, the slaughter weight of pigs in the United States is considerably heavier. Target market weights in the United States commonly range from 100–132 kg (220–291 lb).⁵⁸ United States producers receive the top price (lowest sort loss) when pigs are marketed in this weight range. Based upon historical trends, it is unlikely that the target market weight in the United States will decrease in the near future.

Management

The presence of sexually receptive females stimulates a rapid rise in plasma androstenone and testosterone concentrations in boars.⁵⁹ Boars mixed with gilts during rearing may have elevated concentrations of fat androstenone.^{55,60} At 80 kg (176 lb) of live weight, androstenone content in backfat is not affected by the social conditions during rearing. Between 80 and 95 kg (176 and 209 lb), the proportion of boars with an increase in androstenone content is greater in boars reared with females.⁵⁵ Mixed-sex rearing in some instances also increases the intensity of odor in boar fat.⁶¹ Therefore, split-sex rearing should be practiced if pigs are intended for slaughter at heavier market weight.

Season

A seasonal pattern of androstenone in fat, blood, and semen of boars has been reported in mature boars.^{31,62,63} Concentrations of androstenone from October through December are about five-fold higher than those in the rest of the year. Under decreasing daylength or an artificial light program that simulates the approach to winter, increased androstenone concentrations were observed.⁶²

Skatole

Formation and metabolism of skatole

Skatole is produced in the large intestine by microbial breakdown of the amino acid tryptophan originating from dietary or endogenous protein.^{64,65} The bacteria are from the genus *Lactobacillus*.^{66,67}

Approximately 87% of the available skatole produced in the intestine is absorbed across the intestinal wall and transported in the blood to the liver, where the majority is degraded. The feces only contain 13% of the total microbial gut production of skatole.^{68,69} The half-life of skatole in blood is approximately 60 minutes and the half-life in muscles and fat tissue is 11 hours.⁶⁹ The degraded products are then excreted in the urine.⁷⁰ The nondegraded skatole is deposited in fat and muscle. High concentrations of skatole in fat tissue can give rise to an unpleasant odor or taste.⁷¹ The contribution of indole to the unpleasant odor or taste is lower even though fat concentrations of indole may occasionally exceed those of skatole.⁷²

Factors affecting fat concentrations of skatole

Since skatole is formed primarily from a dietary component in the animal intestine, a number of dietary components have been studied for their effect on skatole production. Although skatole is produced from tryptophan, addition of tryptophan to a normal pig diet does not increase fat skatole concentrations⁷³ or taint intensity.⁷⁴

Dietary and intestinal skatole concentrations do not influence fat skatole concentrations.^{75,76} In fact, infusion of skatole into the terminal ileum of pigs does not increase skatole concentrations in subcutaneous fat.⁷⁷ Diets with high fiber contents stimulate the fermentative process in the hind gut⁷⁸ and increase daily elimination of skatole and indole in feces.⁷⁵ Because high-fiber diets cause large fecal bulk, concentrations of skatole in feces are not affected by the fiber content of a diet.⁷⁵ Diets supplemented with antibiotics or antibiotic feed additives (such as tylosin) do not affect fat concentrations of skatole.^{75,79}

Several studies demonstrated that some dietary ingredients affect fat concentrations of skatole. Yeast slurry from breweries elevates skatole concentrations in the hind gut⁸⁰ and backfat.^{73,80} Sugar-beet pulp containing a high concentration of nondigested and nonstarch polysaccharides decreases concentrations of indole and skatole in subcutaneous fat and increases fecal output of skatole and indole.^{81,82}

Fat concentrations of skatole may be reduced by management practices. Appetite feeding elicits a higher concentration of skatole in fat compared with restricted feeding.⁸³ Pigs with free access to water or wet feeding have reduced skatole concentrations in their fat tissue relative to dry feeding systems.⁸⁴ Pigs at higher stocking rates have higher fat concentrations of skatole than pigs kept clean at a lower stocking rate.⁸⁵ The influence of stocking rate is more significant in summer than in winter.

Detecting boar taint

Prevalence of boar taint

Prevalence of boar taint is reported to vary from 1%–30% in different studies based on subjective sensory tests by laboratory panels.^{3,7,43,86,87} In an early United States study of the prevalence of sex odor in pork, 25% of the boars showed at least a trace of sex odor.³ In another study,⁷ a small proportion of barrows, sows, and gilts had a taint problem. In a recent United States study,⁸⁷ scores of boar meat

for taint were quite low, although odor panel scores were higher for boars (102 kg, 225 lb bodyweight) than barrows (108 kg, 238 lb bodyweight). We found 15% of boars at 100 kg (220 lb) of liveweight to be tainted.⁴³ Taint in sow and gilt carcasses has been postulated to be related to the period of the estrous cycle at the time of slaughter.⁴ Plasma androstenone concentrations decline in the period preceding estrus, with comparatively low concentrations at estrus and an increase post-estrus.⁸⁸ Fecal skatole concentrations are low during the estrous period and high during the luteal phase.⁸⁹

Taint associated with androstenone and skatole

Although androstenone and skatole are the main compounds considered responsible for boar taint, disagreement exists among researchers in different countries. In general, skatole is considered a primary compound for taint in Denmark.⁹⁰ Androstenone is believed to have a higher contribution than skatole to boar taint in most other countries.^{57,91,92} Our studies indicate that androstenone is more important than skatole for taint.^{43,93,94,95}

Skatole is formed in all male, female, and castrated pigs. It is still not known why only intact male pigs deposit skatole in fat tissues in amounts that can cause problems with taint. Skatole and androstenone seem to have synergetic effects: unpleasant odor associated with androstenone can be strengthened when high skatole concentrations are found simultaneously. Skatole may be a problem if pigs are slaughtered at a light weight, at which androstenone concentrations are low.

Threshold values for taint compounds

Chemical and sensory tests are widely used to detect boar taint in meat from entire male pigs. Chemical tests typically assess tissue concentrations of the compounds associated with taint. Sensory tests classify boar carcasses into either tainted or untainted categories according to test criteria assessed by human evaluators. The threshold concentrations of taint compounds are typically indicated according to the results of chemical tests.

Threshold values, above which a carcass can be considered to be tainted, are proposed by European researchers for both androstenone and skatole. They are 1.0 ppm for fat androstenone and 0.25 ppm for fat skatole.⁹⁶ In Denmark, 0.20 ppm of fat skatole was used as a threshold.⁹⁰ Recently, the Danish have increased the threshold of fat skatole to 0.25 ppm (1997).⁹⁷ Threshold values for both androstenone and skatole in cooked ham are higher than those in fresh meat due to the volatile property of the compounds.⁹⁸ They are 1.5 and 0.5 ppm for androstenone and skatole, respectively.⁹⁹

Sensory test for taint

The threshold concentrations used in chemical analysis are based upon panel sensory test findings. The findings of these panels are potentially influenced by several factors.

- Human perception of androstenone is under genetic control. Approximately 50% of adults cannot detect the odor of androstenone,

even at high concentrations.¹⁰⁰ In contrast, approximately 15% of adults detect a subtle odor, but are not offended by it, and may even find it pleasant. The remaining 35% are exquisitely sensitive to androstenone, detecting less than 200 parts per trillion in air.¹⁰¹

- Human sensitivity to androstenone also is influenced by gender. Women tend to be more sensitive to boar taint than men. Kloek¹⁰² investigated 100 men and 100 women for their ability to detect 5 α -androstenone and found that 46% of the males were unable to detect this compound, while only 24% of the females could not detect it. In addition, among panelists, females rated the odor higher in intensity than males. Griffiths and Patterson⁵ had similar results. Analysis of the olfactory responses of 50 men and 50 women to a pure sample of 5 α -androstenone showed that only 7.0% of women were unable to detect the odor compared with 44.3% of men.⁵
- Human perception of androstenone changes with age. The threshold for the perception of androstenone tends to increase with age in men but decrease in woman.¹⁰³
- Human ability to perceive androstenone can be induced. Ten of 20 initially insensitive subjects became sensitive after systematic exposure to androstenone for 6 weeks.¹⁰⁴ Some subjects became sensitive to exposure within 1 week after the initiation of exposure. Laboratory panels are often selected based on their sensitivity to taint compounds. They are then trained to detect odor using relatively low concentrations of compounds. It is likely that the sensory threshold will decrease after repeated exposure to compounds during training and testing.¹⁰⁴

Consumers' response to taint

It has been reported that acceptability of boar meat varies greatly between laboratory panels and consumers. A trained sensory panel in a laboratory can easily detect "taint" in heated fat samples because they are sensitive to taint and trained. Also, no masking agents such as spices are used in panel tests. When preparing and eating boar meat in the normal domestic environment, consumers' responses to boar meat may differ from panelists' responses. In a Canadian study,¹⁰⁵ a potential problem with boar taint was detected by highly trained sensory panel, but was not perceived by a consumer survey. Cowan and Joseph¹⁰⁶ reported that laboratory panels found 58% of boars and 31% of barrows contaminated with taint. Consumers, however, were unable to distinguish between tainted and untainted pork sorted by the panels. In fact, consumers were not even able to identify taint in boar meat that had fat concentrations of androstenone above 2.0 ppm.¹⁰⁷

It has also been reported that acceptability of boar meat varies differently among different populations. Several studies of the reaction of consumers to boar meat have been carried out in a number of European countries and Canada. In general, British,^{108,109} Irish,¹¹⁰ Spanish,¹¹¹ and Canadian¹¹² consumers are not sensitive to boar taint, Dutch and Swedish consumers are sensitive, and French consumers are most sensitive.¹¹⁰ It should be realized that in the French study, the consumers were not randomly selected and were also informed that the study involved testing for boar meat.

Prevention of boar taint

It is imperative to prevent tainted carcasses from entering the fresh food market. Castration is one of the methods to remove testicular synthesis of 16-androstenes, but it also removes the anabolic effect of androgenic steroids.^{29,113}

Preventing tainted carcasses from entering the fresh food market

To prevent tainted carcasses from entering the fresh food market, they must be identified before they are distributed to food markets. An online screening method in slaughterhouse had been used to detect tainted carcasses related to skatole in all Danish slaughterhouses since September 1993.⁹⁰

A method for assessing boar taint on the slaughter line must satisfy the following criteria, proposed by Bonneau and Russeil:¹¹

- It must be easy to perform, because of the large number of carcasses to be assessed.
- It must provide a result quickly to avoid prolonged storage of carcasses. It is particularly important in United States slaughterhouses, where the speed of the slaughter line is very fast. The equipment installed in Danish slaughterhouse may not be suitable for use in the United States because only 80 carcasses can be tested per hour.⁹⁰
- It must be inexpensive; otherwise, the economical benefit from rearing boars would be lost.
- It must be accurate to avoid consumer complaints.

Preventing the production of tainted carcasses

Immunological techniques

Immunological methods have been studied to solve the problem of boar taint. Immunization against 5 α -androstene has only been partially successful.^{115–117} The antibodies bind to 5 α -androstene and block its biological actions. Meanwhile, the steroid-antibody complex delays the metabolic clearance of the steroid. Consequently, in the immunized pigs the concentration of 5 α -androstene in the circulation is much higher than in normal animals,¹¹⁵ or is not different between immunized and control animals.¹¹⁷

Anti-C₁₉ Δ^{16} -steroids reduce mean fat tissue concentrations of the C₁₉ Δ^{16} -steroids and mean sensory scores for taint intensity.¹¹⁸ However, the results vary dependent on animals. Three of 15 immunized boars (20%) had similar concentrations of 5 α -androstene to those of untreated control boars.¹¹⁸

Active immunization against gonadotropin releasing-hormone (GnRH) has been attempted to block the formation of the steroids that cause boar taint. Immunization reduces the weight of testes and accessory sex glands.^{119–121} Immunization against GnRH impairs pituitary and Leydig cell function in boars. Plasma concentrations of luteinizing hormone (LH) and testosterone, pituitary LH content, testicular LH receptor content, testis, and sex accessory organ weights are

significantly reduced in GnRH-immunized boars compared with controls.¹²² Concentrations of fat 5 α -androstene also are reduced after active immunization against GnRH.¹²⁰

Chemical castration

Androstene synthesis can be effectively blocked by injecting young mature boars with a GnRH agonist (leuprolide), a human drug approved by the FDA for use in prostate cancer.⁹³ A single injection of the compound initially stimulates LH and testosterone secretion, and then depresses androgen hormone production in the testis for at least 30 days. All treated animals had fat concentrations of androstene below the threshold concentrations 1 month after injection. Thus, it is plausible to give an injection late in the growth period to entire males, thus disrupting testicular production of taint androgens and allowing them to be eliminated from the pig's body prior to slaughter.

Meat processing

Androstene and skatole are volatile and can be evaporated during processing or heating.¹²³ Acceptability of boar meat from tainted carcasses was improved after processing.^{98,99,123} The intensity of boar taint was reduced in cooked boar meat.¹²⁴ It appears that taint can be reduced or eliminated through heating. Developing a precooked product is a feasible method to find a niche market for boar meat.

Other methods

Feeding level is reported to correlate negatively with strength of taint.¹²⁵ Boars fed ad libitum have higher concentrations of skatole and androstene in fat⁸³ than their littermates on restricted feed (85%) at 100 kg (220 lb) of liveweight.¹¹⁸ High energy intake stimulates testicular development and androstene production, and thus, androstene accumulation in fat.¹²⁶ Similar results with pigs of 130 kg (287 lb) of slaughter weight (70% restriction) were reported by Brennan, et al.⁵⁴

Administering growth hormone (GH) by daily injection from 65–105 kg (143–231 lb) liveweight reduces fat androstene concentrations in boars.¹²⁷ Administering GH with sustained-release implants for 6 weeks before slaughter at 87 kg (192 lb) (171 days of age) results in reduced concentrations of boar taint in the loin chops from boars as assessed by a trained sensory panel.¹²⁸ Boars given GH for 18 weeks have improved boar taint scores compared with untreated boars.¹²⁹

Implications

- Due to boar taint, male pigs cannot be reared for meat at current slaughter weights in the United States even though there are several advantages to rearing intact males instead of castration.
- Vaccination of GnRH or injection of the GnRH agonist to intact males may be effective methods to prevent production of tainted animals.
- If potentially tainted animals can be identified before vaccination or injection, the number of animals to be vaccinated or injected will be significantly reduced.

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