Alternatives to piglet castration

M. Bonneau* and A. Prunier, UMR INRA-Agrocampus Rennes SENAH, 35590 Saint Gilles, France

Introduction

About 85% of the 120 million male pigs that are slaughtered annually in the 25 countries of the European Union are castrated at a young age (EFSA, 2004). Only a handful of countries have almost totally (UK and Ireland) or partly (Spain; 60% of males left entire) abandoned castration in order to benefit from the lower production cost and leaner carcasses of entire males. Despite of these advantages, castration is still practised on the vast majority of slaughter pigs in the remaining countries, in order to prevent boar taint, an off-odour/off-flavour that affects the meat from some of entire males. The main compounds responsible for boar taint are androstenone, a testicular steroid, and skatole, a breakdown product from tryptophane, originating from the hindgut (see e.g. Babol and Squires, 1995 or Bonneau, 1998, for review).

Because castration without analgesia, as it is practised today, is a painful procedure, animal welfare concerns are putting more and more pressure on the pig production chain to abandon it. Norway has recently adopted a legislation that forbid castration without analgesia or anaesthesia and will ban castration by 2009. Switzerland is considering making a similar move. In the EU, directive 2001/93/EC states that:
- "if castration is practised after the seventh day of life, it shall only be performed under anaesthetic and additional prolonged analgesia by a veterinarian",
- castration must be performed "by other means than tearing of tissues".

The aim of the present review paper is to consider advantages and drawbacks of potential alternatives to surgical castration as it is practised today. After a presentation of the welfare consequences of surgical castration without analgesia, the extent to which pain can be relieved when analgesia is performed will be evaluated. Non surgical procedures for castration, including the use of chemicals and anti-GnRH immunisation, will then be considered. Finally, the most drastic option is to stop castration altogether and raise entire males. Advantages and disadvantages of entire male pig production will be reviewed, with a particular emphasis on boar taint, its origin and factors of variation and possible ways to control it.

I. Welfare consequences of surgical castration without analgesia

Surgical castration of male piglets is usually performed without any anaesthesia/analgesia during the first days or weeks of age. It involves cutting of the scrotum (one or two incisions), cutting and/or tearing of both spermatic cords. All theses tissues are innervated and the tissue damage associated to surgical castration is likely to generate painful stimuli. Pain can be identified by numerous physiological and behavioural indices (Mellor et al., 2000).

During castration, most piglets vocalise and demonstrate physical resistance movements (Weary et al., 1998; Taylor & Weary 2000; Marx et al., 2003). These calls are accompanied by an activation of the sympathetic nervous system, as demonstrated by an increase in heart rate (White et al., 1995). Analysis of the calls suggests that extraction of the testes and severing the spermatic cords are the most painful parts during castration (Taylor & Weary 2000). This was further supported by the observation that local anaesthesia is most effective to reduce behavioural resistance when the cords are cut (Horn et al., 1999).

During the first hours following castration, measurement of hormones in plasma clearly indicates an activation of the adrenal and sympathetic axes (Prunier et al., 2002 and 2005). The expression of the protein c-fos in

neurons of the spinal cord, which are likely to transmit the nociceptive stimuli originating from the perineal region to the brain, is increased in pigs submitted to surgical castration that are not previously treated with local anaesthetic than in those that are treated (Nyborg et al., 2000). In addition to these physiological reactions, behaviour is modified. Castrated pigs spend less time at the mammary glands, massaging and/or suckling, (McGlone & Hellman 1988; McGlone et al., 1993; Hay et al., 2003). They remain more inactive while awake, they show more pain-related behaviours (prostration, stiffness, trembling) and tail wagging. They are frequently isolated and their behaviour is more often desynchronized than in their littermates (Hay et al., 2003).

During the days following castration, measurement of corticosteroids and catecholamines in urine suggests that the adrenal and sympathetic axes are no longer stimulated (Hay et al., 2003). In contrast, behavioural observations (reduced play behaviour and overall activity, increased tail wagging and scratching the rump) show that some behavioural alterations persist beyond 24 hours and suggest that piglets experience pain for up to 5 days after castration (Wemelsfelder & van Putten 1985, Hay et al., 2003).

In addition to pain, surgical castration may have detrimental effects on the immune system and on health of the pigs (for a recent review, see EFSA 2004).

II. Surgical castration with analgesia

In order to relieve pain due to surgical castration, anaesthesia (general or local) and prolonged analgesia for at least 24 hours should be performed. However, such treatments are submitted to numerous constraints. First, the use of anaesthetics is limited to veterinarians in EU countries and Norway. Second, drugs used in animals reared for human consumption are subjected to a regulation establishing

Maximum Limits for Residues (Council Regulation No 2377/90). Due to this legislation, analgesics that can be used in pigs are: azaperone and flunixin, aspirin (i.e. acetylsalicyclic acid), ketamine, ketoprofen, paracetamol, procaine and tetracaine. The situation of the local anaesthetic lidocaine is more complex: in some European countries (as in France and Norway), it is allowed to use in pigs under the control of a veterinarian and if a delay of 28 days before slaughter is respected. Third, performing anaesthesia and prolonged analgesia is time consuming and expensive. Fourth, general anaesthesia induced by injection is usually associated with a period of sedation that affects behaviour of the piglets, preventing them from suckling after the surgery and making them more vulnerable to injury by the sow (e.g. getting laid on). Taking into account all these constraints, our view is that the two alternatives that can be considered for practical use at farm level are general anaesthesia under CO_2 and local anaesthesia under lidocaine.

General anaesthesia under CO_2 does not need an evacuation system for excess of gas and may be easily run at the farm level but its efficacy in alleviating pain is in debate. Reactions of discomfort such as restlessness and hyperventilation were observed during induction of anaesthesia with CO_2 (Kohler et al., 1998; Schönreiter et al., 2000). Cortisol levels were higher in anaesthetized than in non-anaesthetized pigs in the four hours following castration, and beta-endorphin levels at one hour after castration were also higher in the anaesthetized pigs (Schönreiter et al., 2000). It was concluded that CO2 does little to alleviate stress at castration. New research is needed to better determine and/or to improve its efficacy in relieving pain at castration.

The efficacy of local anaesthesia by lidocaine (= lignocaine) in relieving pain at castration has been evaluated in numerous studies. Lidocaine can be injected into the testes, the spermatic cord (funiculus spermaticus) or under the skin at the site of

incision. When it is injected intratesticularly with adrenaline, it diffuses into the spermatic cords within 10 minutes (Ranheim et al., 2003). Lidocaine injection into the testes or into the testes plus the scrotal sac reduces the pain-related calls (White et al., 1995; Marx et al., 2003) as well as ACTH and cortisol responses to castration (Prunier et al., 2002). More precisely, lidocaine is efficient at reducing the number of high frequency calls and the heart rate during pulling and severing the spermatic cords (White et al., 1995). In pigs maintained under general anaesthesia with halothane, signs of nociception (increased blood pressure, decreased electroencephalography theta and alpha powers) were reduced but not fully suppressed when lidocaine was injected subcutaneously into the scrotum and either into the cords or the testes (Haga et al., 2005). In conscious pigs, it seems that sharing the dose of lidocaine into the testes and into the scrotum around the funicular area was more efficient in reducing calls during castration than injecting the entire dose into the testes (Prunier et al., 2002).

It can be objected that injecting the local anaesthesia itself may induce pain balancing the reduction in pain observed after castration. Indeed, a slight increment of ACTH was observed after injecting lidocaine but this increase was of much lower amplitude than that observed after castration with and without local anaesthesia (Prunier, unpublished). Pain-related behaviour has also been observed and was associated with the low pH of the solution (Waldmann et al., 1994).

Non steroidal anti-inflammatory drugs (NSAIDs) are the only group of "long-lasting" analgesics currently available for pigs due to the MRL regulation. Several NSAIDs are licensed for pigs but there is nearly no documentation available concerning their efficacy in relieving pain after castration and their side-effects such as bleeding. A preliminary experiment in piglets suggests that injecting the NSAID flunixine before surgical castration and the day after has very little influence on the ACTH and cortisol releases in castrated pigs receiving lidocaine (Prunier, unpublished data). Oral administration of aspirin or intravenous injection of the opioid butorphanol before castration (30 minutes) had no effect on the reduction of weight gain (50%) observed the day after castration of 8-week old pigs (McGlone et al., 1993). In 5.5-month old calves, intravenous injection of the NSAID ketoprofen before castration, reduced cortisol release after castration down to control levels (Earley & Crowe, 2002). A combination of ketoprofen with local anaesthesia (lidocaine) did not seem to be more efficient.

III. Non surgical castration procedures

There are a number of potential alternatives to surgical castration:

local destruction of testicular tissue by various chemical compounds,
Inhibition of the production of the stimulatory hormones of the hypothalamic-pituitary-gonadal axis via
o the application of exogenous hormones which down-regulate the hypothalamic-pituitary-gonadal axis,
o the neutralisation of the stimulatory hormones by specific antibodies (immunocastration).

III.A. Local destruction of testicular tissue by chemical compounds

Various substances have been investigated in different species to induce destruction of spermatogenic and hormone–producing testicular cells: formaldehyde (bovine: Gardner, 1980; sheep: Kang et al., 1993), lactic acid (bovine: Fordyce et al., 1989, Cohen et al., 1990, 1991; dog and rat: Nishumara et al., 1992; pig: Ljaz et al., 2000), acetic acid (pig: Giri et al., 2002), silver salt (pig: Ljaz et al., 2000) and zinc salt (pig: Fahim, 1994). According to the authors who have tried these substances, they are easy to administer, safe for the animals and people

who administer them, not expensive, produce no haemorrhage and only little pain, and have very few side-effects (the risk of post-operative infection is low). However, when data are carefully examined, swelling of the testes or of the scrotum has been observed (Giri et al., 2002; Cohen et al., 1990, 1991; Nishumara et al., 1992) suggesting a painful inflammatory reaction, as well as epididymitis (Gardner, 1980), necrosis and slow healing (Fordyce et al., 1989). Moreover, evaluation of pain-related reactions was very limited and insufficient to make conclusions. Most of the products that have been tested (i.e. zinc acetate, lactic acid, formaldehyde) are not subjected to the MRL legislation.

III.B. Down-regulation of the hypothalamic-pituitary-gonadal axis by exogenous hormones

With the exception of zeranol (Denzer et al., 1986), exogenous steroids or steroid agonists that down regulate the hypothalamic-pituitary-gonadal are effective in reducing androstenone in fat (Daxenberger et al., 2001) and boar taint levels (Hagelschuer et al., 1978; Busch et al., 1979; Lopez-Bote and Ventanas, 1988). Similarly treatment with GnRH agonists, which exert a negative feed back on LH/FSH release when administered continuously, result in a significant reduction in androstenone (Xue et al., 1994; Schneider et al., 1998) and boar taint (Reid et al., 1996). The effect is not long lasting, since treatment of young pigs does not decrease boar taint at maturity (Ziecik, Esbenshade and Britt, 1989).

Because of the negative perception by consumers of the use of hormones in meat producing animals, this option cannot be currently considered as realistic in the EU.

III.C. Immunocastration

The neutralisation of the hormones of the hypothalamic-pituitary-gonadal axis by specific antibodies is commonly referred to as "Immunocastration". Immunisation against LH is less effective than immunisation against GnRH (Falvo et al., 1986).

Although the possibility of using passive immunisation has been considered (Van der Lende et al., 1993), most experiments on immunocastration in pigs involved active immunisation against GnRH. Immunocastration schedules using Freund's adjuvant and/or repeated administration of the vaccine preparation result in an inhibition of genital tract development and a drastic reduction of plasma LH and testosterone concentrations (Figure 1). However Freund's adjuvant is unacceptable for use because it is not licensed in a commercial vaccine and repeated administrations are too laborious and expensive and can cause repeated stress to the animals. Alternative anti-GnRH immunisation methods have been developed, using an acceptable adjuvant and only two injections, according to two different schedules of immunisation (Figure 2):

early castration results in unambiguous results on testes weight, making differentiation on the slaughter line very easy (Oonk et al., 1995). However, most of the economic advantages of the entire males are lost in immunised animals (Turkstra et al., 2002; Zeng et al., 2002a).

late castration enables to maintain most of the performance advantages of intact male pigs in immunised animals (Bonneau et al., 1994; Dunshea et al., 2001; Cronin et al., 2003; Oliver et al., 2003; Jaros et al., 2005). Compared with entire males, late immunised pigs grow faster (once the castration effect is achieved), have a similar feed efficiency and exhibit higher fat contents (although lower than in surgical early castrates) in their carcass. However some measurements would have to be performed on the carcasses in order to check the effectiveness of the treatment (IV.C.).

Surgical castration affects the behaviour of the animals for a couple of weeks (see above). Later on, surgical and immunocastration have very similar effects on animal behabiour

(Cronin et al., 2003). In both cases, reduced aggressive and mounting behaviours and increased duration of feeding behaviour is obtained, in comparison with entire males.

Immunocastration results in a sharp decrease in fat androstenone and skatole levels (Figure 3), the main compounds responsible for boar taint. Both the mean levels and the variability of androstenone and skatole levels are reduced in immunocastrated pigs, compared with entire males. The levels of both compounds, as well as boar taint intensity (Falvo et al., 1986), are similar in immuno- and surgical castrates.

One concern is the variability of the immune response of the animals, resulting in some pigs not being castrated (e.g. the results of Bonneau et al., 1994 and Turkstra et al., 2002), so that the absence of boar taint cannot be totally guaranteed, unless the effectiveness of the procedure in reducing boar taint is measured in individual animals, preferably on the slaughterline. When immunocastration is performed at an early age, this can be easily achieved, using testis size as a marker of sexual development (see above). However, when it is performed only a few weeks before slaughter, the level of boar taint should be determined. Large scale studies (Dunshea et al., 2001; Jaros et al., 2005) show that the number of non-respondent pigs is low. Furthermore, because of the very high variability of androstenone and skatole levels, the non-respondent pigs are not necessarily tainted. Therefore, the proportion of non-respondent pigs that do exhibit boar taint (= percentage of non-respondents multiplied by frequency of tainted pigs in the population) is lower than the total proportion of non-respondent pigs.

The absence of boar taint in surgically castrated pigs cannot be totally guaranteed either. Indeed, in every pig population, there is a proportion of cryptorchids and intersex animals (in the range of 0.1-0.6%

for intersex and 0.3-0.8% for cryptorchids; Albertsen, 1951; Koch, 1963; Backstrom and Henricson, 1971; Krishnamurthy et al., 1971; Nador, 1990; Bellot and Vogt, 1994), which are known to exhibit high levels of boar taint. High levels of androstenone (Pailhoux et al., 1995) or 16-androstens (Booth and Polge, 1976) have been observed in such animals. It may be expected that a significant proportion of them also exhibit high skatole levels, however, to our knowledge, this has not been investigated. It may be hypothesised that they would respond to anti-GnRH immunisation in the same way as normal entire male pigs.

There are some possible drawbacks of immunocastration. The cost of the treatment has to be compared with the economic gains obtained from discontinuing castration of male pigs (in Australia, the cost of the treatment with the locally available commercial vaccine is about 3 € per pig; Moore, 2000). Control on the slaughter line of the effectiveness of the procedure would be necessary in late castrated animals (see above and IV.C.). Consumers may be reluctant to accept immunocastration, because they are hormonal vaccines (residues issue). There are also some safety concerns in case of accidental self-injection to the person who is vaccinating the pigs. Finally, the issue of possible pain for the treated animal has not been directly investigated. With aqueous vaccine preparations, there is little reaction on the site of injection (Dunshea et al., 2001). However, because the GnRH vaccines are directed against hormones produced by the animals, they may induce cellular damages away from the injection site. Molenaar et al. (1993) found that anti-GnRH immunisation in the pig resulted in lesions of the hypothalamus. Whether or not this effect has deleterious consequences on the animals (behaviour, neuroendocrine regulations) remains unknown. In any case, these consequences should be compared with those due to surgical castration.

IV. Production of entire male pigs

IV.A. Advantages and disadvantages of entire male pig production

Further to the avoidance of castration and associated pain for the animal, the main advantages are the following:

Lower production costs:
o labour costs involved in performing castration are eliminated,
o animal losses and temporary decrease in performance following castration are avoided,
o entire males need less feed to grow and may sometimes grow faster than castrates (e.g. Walstra and Kroeske, 1968; Fowler et al., 1981; Andersson et al., 1997).

Reduced output of nitrogen resulting from better efficiency for nitrogen retention (e.g. Desmoulin et al., 1974).

Leaner carcasses (e.g. Prescott and Lamming, 1967; Hansson et al., 1975; Fortin et al., 1983; Hansen and Lewis, 1993).

Lower lipid content and higher content of unsaturated fatty acids in adipose tissues of entire males may be regarded as favourable from the nutritional point of view (e.g. Malmfors and Nilsson, 1978; Wood and Enser, 1982; Ellis et al., 1983; Desmoulin et al., 1983; Barton-Gade, 1987).

There are also a number of disadvantages associated with entire male pig production, including:

Deteriorated welfare for the dominated animals, resulting from increased aggression and "riding" or mounting by the dominant ones (Lundström et al., 1987; Giersing,1998; Cronin et al., 2003).

Elevated incidence of carcass damages resulting from increased fighting. Carcass damages range from superficial skin blemishes (Sather et al., 1995) to major carcass bruising (Warriss, 1984; Moss & Trimble, 1988).

Lower dressing percentage, in relation with the presence of a fully developed genital tract (e.g. Prescott and Lamming, 1967; Fortin et al., 1983; Hansen and Lewis, 1993).

Excessively lean carcasses and a lack of cohesion between backfat and the underlying muscle (Wood, 1984) may occur in lean genotypes.

Increased incidence of DFD meat, because entire males are more active (Moss and Robb, 1978; Ellis et al., 1983).

Less tender meat may sometimes be observed, in relation with the reduced intramuscular lipid content (Martin et al., 1968; Bonneau et al., 1979; Barton-Gade, 1987).

Lower quality of adipose tissue, resulting from higher water content and increased content of unsaturated fatty acids, the processing quality of fat is worst in entire males. Entire male fat is softer and less resistant to oxidation, especially in lean genotypes.

Boar taint, which is the most important issue associated with entire male pig production. Boar taint is an offensive off-odour/flavour resulting from the tissue deposition of the compounds androstenone, a testicular steroid, and skatole, a breakdown product from tryptophane, (for review see e;g. Babol and Squires, 1995 or Bonneau, 1998).

IV.B. Control of boar taint

Skatole levels can be reduced by modulating nutrition and husbandry conditions, whereas genetic selection is more efficient at lowering androstenone content. The most recent review of these aspects is available in EFSA (2004).

Fat skatole levels can be limited by rearing pigs on slatted floors rather than on concrete, by using wet instead of dry feeding, by keeping pigs clean, by allowing them unlimited access to drinking water (Kjeldsen, 1993; Hansen et al., 1994). Claus et al. (1994) demonstrated that feeding pigs a mixture of inulin and bicarbonate during a few days before slaughter results in a sharp reduction in fat skatole levels. A number of feedstuffs containing high amounts of fermentable carbohydrates can be incorporated in the diets,

resulting in a decrease in skatole levels (Jensen et al., 1995, 1997; Andersson et al., 1997). The addition of zeolite (Baltic et al., 1997) to the diet is effective in reducing fat skatole levels. Finally, withholding feed on the evening prior to slaughter has been shown to reduce fat skatole levels (Maribo, 1992; Kjeldsen, 1993). The control of nutrition or husbandry conditions does not however guarantee the absence of skatole-related taint.

There are strong indications of a genetic control of skatole levels (Squires and Lou, 1995; Xue et al., 1996; Pedersen, 1998; Hortos et al., 2000; Doran et al., 2002; Pedersen, 1998; Lundström et al.,1994), probably in relation with a polymorphism in some of the enzymes involved in the liver metabolism of skatole (Friis, 1995; Squires and Lundström, 1997; Babol et al., 1998a,b, 1999; Squires and Lundström, 1997; Diaz and Squires, 2000a; Diaz and Squires, 2000b; Diaz and Squires, 2003; Lin, Lou and Squires, 2003, 2004). The availability of genetic markers identifying those animals that can efficiently metabolise skatole in the liver would make it possible to select pigs against skatole. Selection of pigs directly on fat skatole levels may be not realistic, since a high environmental load of skatole is required before those animals with insufficient liver metabolism will accumulate high levels of skatole in fat.

Specific immunisation against androstenone or its immediate precursors would result in a reduction of androstenone (responsible for boar taint) without any negative effect on androgens and oestrogens, which promote muscle growth and feed efficiency. The results obtained so far have been however quite inconsistent and, even in the best cases, the obtained reduction in fat androstenone was not sufficient. (Shenoy et al., 1982; Williamson and Patterson, 1982; Williamson et al., 1985; Brooks et al., 1986).

The heritability of fat androstenone content is very high, with estimates ranging from 0.25 to 0.87 (review by Willeke, 1993). However, a selection against androstenone results in a decrease in the production of androgens and oestrogens and, thus, have a negative effect on performance and sexual maturation (Willeke et al., 1987). This problem can be partly alleviated, using a selection index associating androstenone and either testis (Sellier and Bonneau, 1988) or bulbo-urethral gland (Sellier et al., 2000) development. However, selection against androstenone is probably not realistic in practice, using the above-mentioned selection criteria. Genetic markers for low androstenone may be developed in the future based on the expression of cytochrome b5 (Davis and Squires, 1999) or other major genes controlling fat androstenone level (Fouilloux et al., 1997) and of a genetic linkage with markers on chromosome 7 (Quintanilla et al., 2003).

IV.C. Assessment of boar taint on the slaughter line

There is currently no practical procedure that can guarantee the absence of boar taint in meat from immunocastrated (III.C.) or entire male (IV.B) pigs. Therefore, the production of such animals cannot be envisaged in most countries, unless boar taint is assessed on line in order to sort out tainted carcasses and direct them to processing. To achieve that, it is necessary to:

    define cut-off levels for the sorting of tainted carcasses,
    develop quick, cheap and reliable procedures, usable in industrial conditions.

On the basis of the results of sensory assessments by trained panels, cut-off levels for skatole are considered to be 0.20 or 0.25 ppm (e.g. Hansen-Møller and Godt, 1995). Due to systematic differences between the various methods used for the measurement of fat androstenone, cut-off levels for androstenone range between 0.5 and 1 ppm. However, trained panels are not representative of the real consumers. The definition of cut-off levels from consumer results is usually not

possible, because of the high variability and background "noise" associated with such data. A more realistic approach could be to model the expected proportion of unsatisfied consumers according to androstenone /skatole concentrations (Bonneau et al., 2000b).

Regarding the procedures to be used on slaughter lines, sensory assessment by one expert, as proposed by Jarmoluk et al. (1970) is not realistic in industrial conditions, because of the very poor repeatability of the assessment. A number of attempts have been made to develop objective methods based on the measurement of androstenone and/or skatole concentrations that can be related to taint intensity (for a recent review, see EFSA 2004).

In the present state of knowledge, there is no satisfactory method for the sorting of carcasses on the slaughter-line. More efforts are needed to develop quick, cheap and reliable methods to measure or estimate malodorous compounds and relate them to the level of consumer dissatisfaction.

Conclusions

Because it is a painful procedure, surgical castration without any analgesia or anaesthesia is likely to be banned in EU countries, although the time when this will occur cannot be predicted. With the exception of the few countries where entire male pig production is presently performed, the industry has currently no practical solution to face the consequences of such a ban. In "entire male pig countries", dissatisfaction is growing because the tendency to ever increasing slaughter weights results in elevated incidence of boar taint.

The first possible way is to continue to castrate pigs, using less painful procedures:

> Surgical castration with local anaesthesia and prolonged analgesia may be an alternative provided that the

lack of negative side-effects is fully established, that farmers are allowed to use these products for castration and that cost (additional work + products) is reasonable. Immunocastration seems quite promising as it has been proved to be very effective. There are however some uncertainties regarding non-respondent pigs, cost of the vaccine, possible deleterious consequences on the animals, and the public acceptance of the procedure.

Discontinuing castration cannot be envisaged unless a clear solution has been found to the boar taint problem. Controlling nutrition and husbandry conditions can reduce skatole but has no sensible effect on androstenone-related boar-taint. Genetic control of boar taint will be feasible only with marker assisted selection. There are good candidates for such markers but their usefulness still needs further investigation.

Banning surgical castration is not yet practically feasible. There are a number of good potential options to solve the problem, which need further investigations.

References

Albertsen K., 1951. Nordisk Veterinaermedicin, 3, 849-868.

Andersson K. et al., 1997. Livest. Prod. Sci., 51, 131-140.

Awoniyi C.A. et al., 1988. J. Reprod. Fertil., 84, 295-302.

Babol J. & Squires E.J., 1995. Food Research International 28, 210-212.

Babol J. et al., 1998a. J. anim. Sci., 76, 822-828.

Babol J. et al., 1998b. J. anim. Sci., 76, 829-838.

Babol J. et al., 1999. J.anim. Sci., 77, 84-92.

Backstrom L. & Henricson B., 1971. Acta Vet. Scand., 12.

Baltic M. et al., 1997. In Boar taint in entire male pigs, Eds. M. Bonneau, K. Lundström & B. Malmfors. EAAP Publication 92, 97-99.

Barton-Gade P., 1987. Livest. Prod. Sci. 16, 187-196.

Bellot R. & Vogt D., 1994. Archivos Latinoamericanos Produc. Anim., 2.

Bonneau M., 1998. Meat Sci., 49 (suppl. 1), S257-S272.

Bonneau M. et al., 1979. Ann. Zootech., 28, 53-72.

Bonneau M. et al., 1994. J. anim. Sci., 72, 14-20.

Booth W. & Polge C., 1976. J. Reprod. Fertil., 46.

Brooks R.I. & Pearson A.M., 1986. J. anim. Sci., 62, 632-645.

Busch W. et al., 1979. Arch. Experim. Veterinarmed., 33, 99-109.

Caraty A. & Bonneau M., 1986. Comptes Rendus des Séances de l'Académie des Sciences de Paris, Série D 303, 673-676.

Claus R. et al., 1994. Meat Sci. 38, 289-305.

Cohen R.D.H. et al. 1991. Canad. J. Anim. Sci., 71, 1-11.

Cohen R.D.H. et al., 1990. Canad. J. Anim. Sci., 70, 1063-1072.

Cronin G.M. et al., 2003. App. Anim. Behav. Sci., 81, 111-126.

Davis S.M. & Squires E.J., 1999. J. Anim. Sci., 77, 1230-1235.

Daxenberger A. et al., 2001. Livest. Prod. Sci., 69, 139-144.

Denzer L. et al., 1986. J. anim. Sci., 62, 1164-1171.

Desmoulin B. et al., 1974. Journées Rech. Porcine en France, 6, 247-255.

Desmoulin B. et al., 1983. Journées Rech. Porcine France 8, 89-98.

Diaz G.J. & Squires E.J., 2000a. Toxicol. Sci., 55, 284-292.

Diaz G.J. & Squires E.J., 2000b. J. Agric. Food Chem., 48, 833-837.

Diaz G.J. & Squires E.J., 2003. Xenobiotica, May issue.

Directive 2001/93/EC. ECOJ n° L316 of 1/12/2001.

Doran E. et al., 2002. Anim. Sci., 74, 461-468.

Dunshea F.R. et al., 2001. J. anim. Sci., 79, 2524-2535.

Earley B. & Crowe M.A., 2002. J. Anim. Sci., 80,1044-1052.

EFSA, 2004. Welfare aspects of the castration of piglets. Scientific Report of the Scientific Panel for Animal Health and Welfare on a request from the Commission related to welfare aspects of the castration of Piglets. European Food Safety Authority- AHAW/04- 087.

Ellis M. et al., 1983. Anim. Prod., 37, 1-9.

Fahim M.S., 1994. United States Patent and Trademark Office. Application N° 206469.

Falvo R.E. et al., 1986. J. Anim. Sci., 63, 986-994.

Fordyce G. et al., 1989. Austr. Vet. J. 66, 272-276.

Fortin A. et al., 1983. Canad. J. anim. Sci., 63, 711-714.

Fouilloux M.N. et al., 1997. Genet. Select. Evol. 29, 357-366.

Fowler V.R. et al., R., 1981. Anim. Prod., 32, 357.

Friis C., 1995. Proceedings EAAP Working Group on Production and Utilisation of Meat from Entire Male Pigs, Milton Keynes, England, INRA and MLC. 4p.

Gardner I.A., 1980. New South Wales Veterinary Proceedings 16, 60-61.

Giersing M., 1998. Social dominance, Competitive aggression and social stress in the domestic pig with particular reference to boar taint. PhD thesis, Royal Veterinary and Agricultural University, Copenhagen.

Giri S.C. et al., 2002. Chemical castration in pigs. Indian J. Anim. Sci., 72, 451-453.

Haga H.A. & Ranheim B., 2005. Vet. Anaesth. Analg. 32, 1-9.

Hagelschuer H. et al., 1978. Monatsh. Veterinarmed., 33, 779-784.

Hagen G. et al., 1988. Proceedings 11th Congress on Animal Reproduction and Artificial Insemination, Dublin, 493-495.

Hansen B.C. & Lewis, A.J.; 1993. J. anim. Sci. 71, 2122-2132.

Hansen L.L. et al., 1994. Anim. Prod. 59, 99-110.

Hansen L.L. et al., 1997. In Boar taint in entire male pigs, Eds. M. Bonneau, K. Lundström & B. Malmfors. EAAP Publication 92, 92-96.

Hansson I. et al., 1975. Swed. J. Agr. Res., 5, 69-80.

Hay M. et al., 2003. Appl. Anim. Behav. Sci., 82, 201-218.

Horn T. et al., 1999. Deutsche Tierarztliche Wochenschrift, 106, 271-274.

Hortos M. et al., 2000. Proceedings 46th International Congress of Food Science and Technology, (Vol.1) (p. 98-99), 27 August - 1 September 2000, Buenos Aires, Argentina.

Jaros P. et al., 2005. Livest. Prod. Sci., 92, 31-38.

Jensen M.T. et al., 1995. Anim. Sci., 61, 293-304.

Jensen M.T. et al., 1997. In Boar taint in entire male pigs, Eds. M. Bonneau, K. Lundström & B. Malmfors. EAAP Publication 92, 80-83.

Kang Y.S. et al., 1993. Korean J. Anim. Sci., 35, 463-469.

Kjeldsen N., 1993. In Measurement and prevention of boar taint in entire male pigs, Ed. M. Bonneau, INRA Edition, Paris, 137-144.

Koch W., 1963. In Intersexuality. C. Overzier (ed), Academic Press, New York, 563 p.

Kohler I. et al., 1998. J. Vet. Med. Series A, 45, 625-633.

Krishnamurthy S. et al., 1971. Canad. J. Am. Sci., 51.

Lin Z. et al., 2003. EAAP Working Group on Production and Utilisation of Meat from Entire Male Pigs, 13-14 November 2003, Teagasc, National Food Centre, Ashtown, Dublin 15, Ireland.

Lin Z. et al., 2004. Mammalian Genome 15, 218-226.

Liu C.Y. et al., 2001. J. Anim. Sci., 79, Suppl. 1

Ljaz A. et al., 2000. Pakistan Vet. J., 20, 129-134.

Lopez-Bote C. & Ventanas J., 1988. Meat Sci., 22, 163-171.

Lundström K. et al., 1987. Swed. J. Agr. Res., 17, 51-56.

Lundström K. et al., 1994. Livest. Prod. Sci., 38, 125-132.

Malmfors B. & Nilsson R., 1978. Swed. J. Agric. Res., 8, 209-217.

Manns J.G. & Robbins S.R., 1997. In Boar taint in entire male pigs, Eds. M. Bonneau, K. Lundström and B. Malmfors. EAAP Publication 92, 137-140.

Maribo H., 1992. 38th International Congress of Meat Science and Technology, Clermont-Ferrand, France, 201-204.

Martin A.H. et al., 1968. Canad. J. Anim. Sci., 48, 171-179.

Marx G. et al., 2003. J. Sound Vibr. 266, 687-698.

Mc Cauley I. et al., 2003. Austral. J. Agric. Res., 54, 11-20.

McGlone J.J. & Hellman J.M., 1988. J. Anim. Sci., 66, 3049-3058.

McGlone J.J. et al., 1993. J. Anim. Sci., 71, 1441-1446.

Mellor D.J. et al., 2000. In: Moberg GP and Mench JA (eds) The Biology of Animal Stress pp 173-198. CAB International, Amsterdam.

Meloen R.H. et al., 1994. Vaccine 12, 741-746.

Metz C. et al., 2002. Livest. Prod. Sci., 74, 147-157.

Molenaar G.J. et al., 1993. J. Neuroimmunology, 48, 1-11.

Moss B.W. & Robb J.D., 1978. J. Sci. Food Agric., 29, 689-696.

Moss B.W. & Trimble D., 1988. Rec. Agric. Res. (NI), 36, 101-7.

Nador, A., 1990. Magyar Allatorvosok Lapja, 45.

Nishumara N. et al., 1992. J. Reprod. Dev., 38, 263-266.

Nyborg P.Y. et al., 2000. Dansk Veterinaertidsskrift, 83, 16-17.

Oliver W.T. et al., 2003. J. anim. Sci., 81, 1959-1966.

Oonk H.B. et al., 1995. Livest.Prod. Sci. 42, 63-71.

Pailhoux E. et al., 1995. Journées Recherche Porcine en France, 27, 81-86.

Pedersen B., 1998. In: Skatole and boar taint (p. 129-136). Ed W. K. Jensen. Danish Meat Research Institute, Roskilde, Denmark.

Prescott J.H.D. & Lamming G.E., 1967. Anim. Prod., 9, 535-545.

Prunier A. et al., 2002. Journées Rech. Porc. Fr., 34, 257-268.

Prunier A. et al., 2005. Journal of Animal Science 83: 216-222.

Quintanilla R. et al., 2003. J. anim. Sci., 81, 385-394.

Ranheim B. et al., 2003. 8th Word Congress of Veterinary Anesthesia, Knoxville TN, p 209.

Reid J. et al., 1996. Reprod. Nutr. Develop., 36, 321-332.

Sather A.P., 1995. Canad. J. anim. Sci., 75:45-56.

Schneider F. et al., 1998. Anim. Reprod. Sci., 50, 69-80.

Schonreiter S. et al., 2000. KTBL-Schrif, 391: 137-145.

Sellier P. & Bonneau M., 1988. J. anim. Breed. Genet., 105, 11-20.

Sellier P. et al., 2000. Livest. Prod. Sci., 63, 265-274.

Shenoy E.V.B. et al., 1982. Acta Endocrinol., 100, 131-136.

Squires E.J. & Lou Y., 1995. Ontario Swine Research Review. OAC Publication, University of Guelph, Guelph, Canada.

Squires E.J. &Lundström K., 1997. J. anim. Sci., 69, 1092-1100.

Taylor A.A. & Weary D.M., 2000. Appl. Anim. Behav. Sci., 70, 17-26.

Turkstra J.A. et al., 2002. J. anim. Sci., 80, 2953-2959.

Van der Lende T. et al., 1993. In: M Bonneau (ed) Measurement and Prevention of Boar Taint pp 201-206. INRA Editions, Paris, Fr.

Waldmann K.H. et al., 1994. Deutsche Tierarztliche Wochenschrift 101, 105-109.

Walstra P. & Kroeske D., 1968. World Rev. anim. Prod., 4, 59-64.

Warriss P.D., 1984. In Proc. 30th Eur. Mtg. of Meat Res. Workers, Bristol, p. 1:8.

Weary D.M. et al., 1998. Appl. Anim. Behav. Sci. 56, 161-172.

Wemelsfelder F. & van Putten G., 1985. B-260 Zeist: Instituut voor Veeteeltkundig Onderzoek 'Schoonoord'.

White R.G. et al., 1995. J. Anim. Sci. 73, 381-386.

Willeke H., 1993. Pig News Inform., 14, 31N-33N.

Willeke H. et al., 1987. J. anim. Breed. Genet., 104, 64-73.

Williamson E.D. & Patterson R.L.S., 1982. Anim. Prod., 35, 353-360.

Williamson E.D. et al., 1985. Livest. Prod. Sci., 12, 251-264.

Wood J.D., 1984. In: Fat quality in lean pigs. Meat Research Institute Special report N° 2, 9-14.

Wood J.D. & Enser M., 1982. Anim. Prod., 35, 65-74.

Xue J. et al., 1996. J. anim. Sci., 74, 2170-2177.

Xue J.L. et al., 1994. J. anim. Sci., 72,1290-1298.

Zeng X. et al., 2002c. Anim. Reprod. Sci., 70, 223-233.

Zeng X.Y. et al., 2002a. Livest. Prod. Sci., 77, 1-11.

Zeng X.Y. et al., 2002b. Theriogenology, 58: 1315-1326.

Ziecik A.J. et al., 1989. J. Reprod. Fertil., 87, 281.

Figure 1. The effect of immunocastration on hormone levels and testes development: studies involving Freund's adjuvant and/or numerous administrations of the immunogen. Drawn from the results published in Caraty and Bonneau (1986), Falvo et al. (1986), Awoniyi et al. (1988), Hagen et al. (1988), Meloen et al. (1994), Manns and Robbins (1997), Liu et al. (2001), Metz et al. (2002), Zeng et al. (2002c), Mc Cauley et al. (2003).
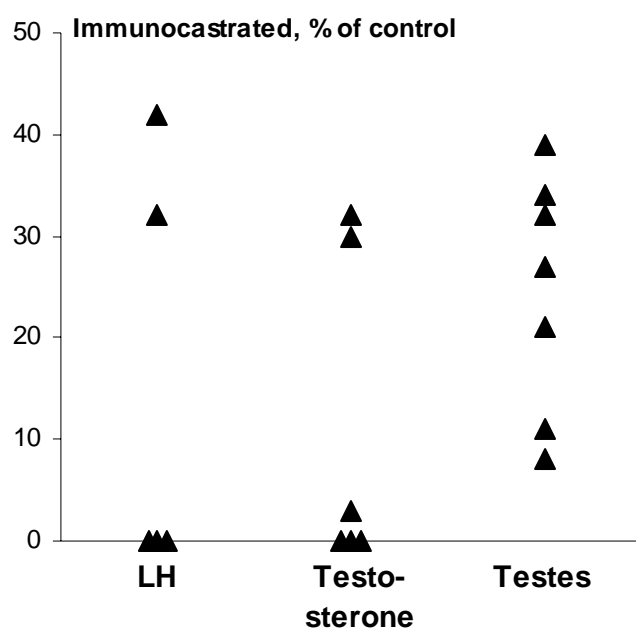
Figure 2. The effect of early (O) or late (◆) immunocastration on testosterone, testes development and performance: larger scale studies involving two injections and potentially acceptable adjuvants. Drawn from the results published in Bonneau et al. (1994), Dunshea et al. (2001), Turkstra et al. (2002), Zeng et al. (2002a), Cronin et al. (2003), Oliver et al. (2003), Jaros et al. (2005).
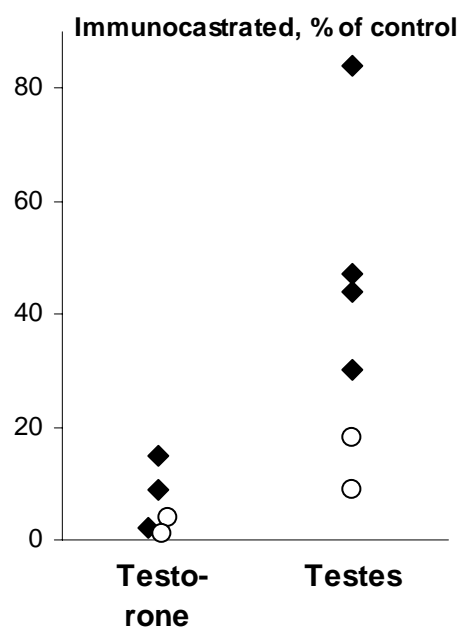

Figure 3. Androstenone and skatole levels observed in entire males (■), immunocastrates (◇) and surgical castrates (-) in studies where immunisation schedules aimed at late (study 1-3) or early (study 4-5) castration. Surgical castration was performed at an early age in all studies. Vertical bars indicate standard deviation (not available for studies 4-5). Drawn from the results published in Bonneau et al., 1994 (1); Dunshea et al., 2001 (2); Jaros et al., 2005 (3); Turkstra et al., 2002 (4); Zeng et al., 2002b (5).