

RELATIONSHIP BETWEEN ANDROSTENONE ACCUMULATION IN PLASMA AND FAT OF INTACT MALE PIGS

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Key words: Boar Taint, Androstenone, Estradiol, Testosterone, backfat, development**Background**

Meat from a small percentage of intact male pigs contains boar taint due to high concentrations of the testicular steroid androstenone (Patterson, 1968) or skatole (Vold, 1970) in fat. Most male pigs are castrated shortly after birth to prevent boar taint. However, due to the superior carcass characteristics and feed conversion of intact males, it would be advantageous to find methods other than castration, to prevent boar taint.

During early testicular development, the Leydig cells of the domestic pig are very active in steroidogenesis, with a peak in plasma steroids at 2 to 4 wk of age (Schwarzenberger et al., 1993; Sinclair et al., 2001). However, the importance of this increase in steroidogenesis on steroid production at puberty is unknown. Testicular steroid production is influenced by the physiological development of the animal and at market weight, boars will vary in their sexual maturity (Bonneau, 1982). Boars with low fat androstenone at slaughter may be individuals with delayed pubertal onset or animals with a reduced capacity for androstenone production (Bonneau and Russeil, 1985; Sellier and Bonneau 1988).

Carcass characteristics may also influence the concentration of androstenone in the carcass. Generally, a high level of androgen and estrogen production coincides with a leaner carcass (Wood and Enser, 1982), due to the anabolic effects of testicular steroids. However, the effect of carcass leanness on androstenone accumulation in fat is relatively unknown.

Objective

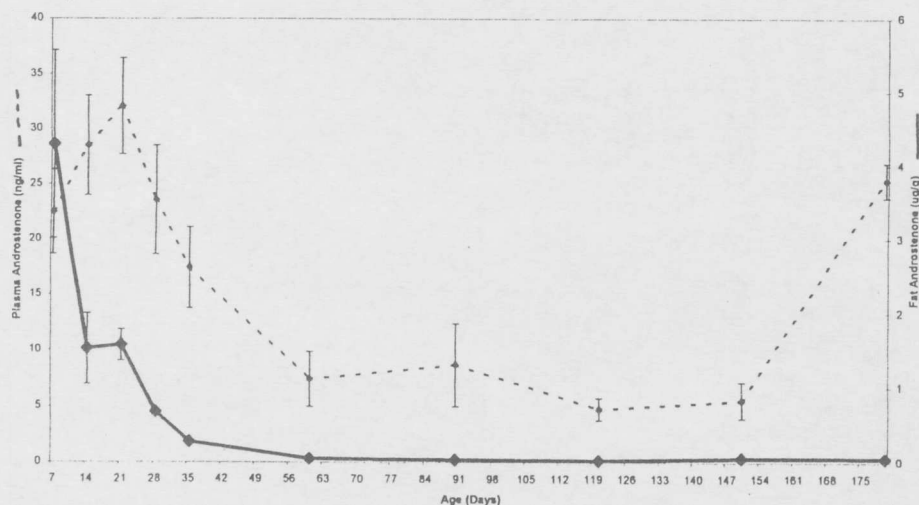
The objective of this work was to examine the relationship between androstenone levels in plasma and fat of intact male pigs. The effect of factors such as stage of development and back fat thickness were considered.

Methods

Yorkshire boars from the Arkell Swine Research Station, University of Guelph were used. Blood samples were taken from the orbital sinus at weekly intervals until 35 days of age and then monthly until slaughter. Samples were centrifuged at 4°C to collect plasma and stored at -20°C until they were assayed for steroid hormone concentrations. Animals were slaughtered at 110 ± 5.2 kg. Back fat thickness was measured at the point of the 11th rib and a back fat sample was removed and frozen at -20°C until assayed for androstenone. Plasma and fat samples were assayed for androstenone with an enzyme-linked immunosorbent assay (ELISA) method, modified after Claus et al. (1988) as described previously by Squires and Lundström (1997). Radioimmunoassays were used for measurements of testosterone (Raeside and Middleton, 1979) and estrone sulfate (EIS) (Schwarzenberger et al. 1993) in plasma samples.

Results and Discussion

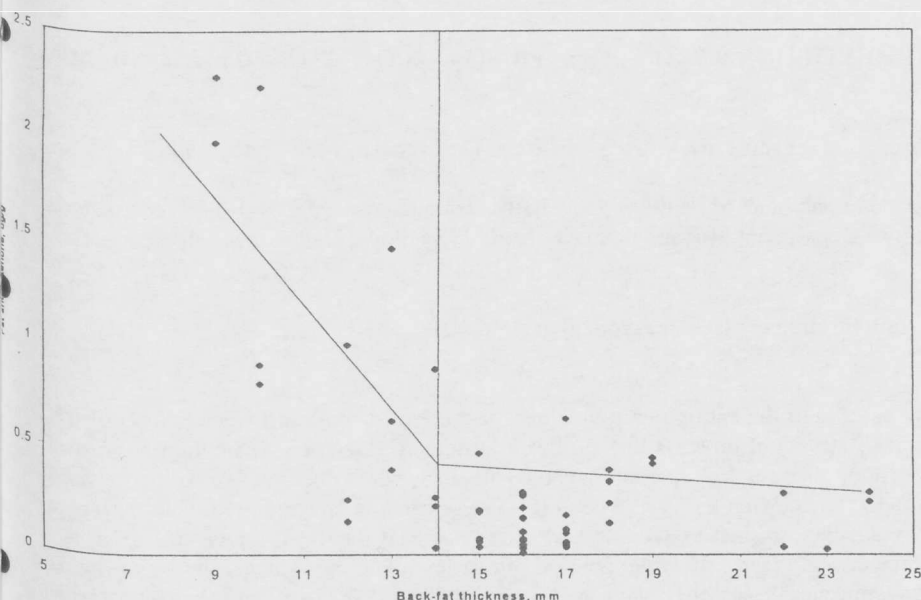
The profile of androstenone concentrations in plasma is given in Figure 1. An initial peak in plasma androstenone concentrations occurred between 2 and 4 weeks of age. A rapid increase in androstenone levels in the plasma was observed after 150 days, reaching about 25 ng/ml by day 180. Concentrations of androstenone in fat in animals from 7 days of age to 180 days of age, are also shown in Figure 1. The mean concentration of androstenone in fat at day 7, 14, 21, and 28 ranged from 4.2 ug/g to 0.6 ug/g. Levels of androstenone in fat from day 7 to 28 were correlated ($r = 0.68$, $P < 0.05$) with the levels of androstenone present in plasma. However fat androstenone concentrations declined from day 7 to 21 whereas plasma levels increased during this time period. From day 36 to 180, levels of androstenone in fat were low (0.02 ug/g to 0.1 ug/g), while a marked increase in plasma androstenone occurs from day 150.



High concentrations of plasma androstenone in market weight pigs were not always associated with high concentrations of androstenone in fat. However, animals with concentrations of plasma androstenone below 15 ng/ml had less than 1 ug/g of androstenone in fat. Animals with plasma androstenone concentrations greater than 15 ng/ml had androstenone concentrations in fat which ranged from 0.01 ug/g to 2.3 ug/g.

There was a significant negative correlation between backfat thickness measured at slaughter and androstenone concentrations in fat ($r = -0.57$; $P < 0.01$; Figure 2). With the exception of one animal, all pigs with more than 0.5 ug/g of androstenone in fat had 14 mm of backfat or less. The leanest animals, with backfat thickness of 9 mm, had the highest concentrations

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of androstenone in fat (1.9 ug/g - 2.3 ug/g) whereas animals with greater backfat thickness had much lower concentrations of androstenone in fat. The data were divided so that two regression lines were fitted with a calculated break-point backfat thickness of 14 mm ($P < 0.05$).

Animals were then separated into a group of fat boars with backfat measurements higher than 14 mm and a group of lean boars with backfat measurements lower than 14 mm. Plasma concentrations of androstenone, EIS and testosterone were significantly higher ($P < 0.05$) in the lean group than in the fat group. Androstenone concentrations in fat were 0.2 ug/g for the fat group and 0.9 ug/g for the lean group. This difference can also be seen in the correlation coefficients between backfat thickness and fat

androstenone concentrations in the two groups. A strong negative relationship between backfat thickness and androstenone concentration in fat was found in the lean group ($r = -0.80$; $P < 0.01$), whereas no significant correlation was present in the fat group. Plasma androstenone at market weight and concentrations of androstenone in fat were also significantly correlated in the lean group ($r = 0.42$; $P < 0.05$), but not in the fat group of animals.

Accumulation of androstenone in fat may be indirectly affected by factors other than strictly the hydrophobic properties of androstenone. The results of this study show that leanness of the carcass is indirectly related to the amount of androstenone accumulation in fat. Lean market weight animals as well as neonatal animals had high levels of androstenone in fat, indicating that androstenone concentrations are highly dependant on the storage capacity of the fat. This finding may explain the discrepancies within the literature on the correlation between plasma androstenone and fat androstenone at market weight. This would suggest that animals with very little fat have a much smaller volume/space for dispersing androstenone and therefore possess a higher value per gram of fat. This view; however, implies that androstenone uptake into fat tissue is solely dependant on levels in peripheral plasma.

Differences in fat composition could also be a factor in androstenone accumulation. Animals of different ages or different leanness may possess fatty tissue differing in composition. Backfat from intact males has a higher water content, protein content (Barton-Grade, 1987) and higher percentage of unsaturated fats (reviewed in Babol and Squires, 1995) in comparison to gilts and castrates. It may be that a higher water content of the fatty tissue will decrease androstenone accumulation in fat.

Conclusions

These results suggest that androstenone concentrations in fat are not likely to be fully dependent on the levels of androstenone in plasma. These findings indicate that androstenone accumulation in fat may be a more complex process, influenced by a variety of factors other than the hydrophobicity of androstenone.

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