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Letter to the Editor

Chen, G., Zamaratskaia, G., Andersson, H. K., & Lundström, K., 2007. Effects of raw potato starch and live weight on fat and plasma skatole, indole and androstenone levels measured by different methods in entire male pigs. *Food Chemistry, 101*, 439–448

For decades, it was accepted and useful to criticize scientific papers in a non-insulting way. I will try to phrase my criticism as scientifically as possible, even if the background affects political and economical interests. Prof. Lundstöm and her colleagues gave us an example of a paper (*Food Chemistry 101*, 439–448, 2007) which indeed needs some criticism for its study design, analytical methods and physiological discussion.

Their experiment are based on two groups of entire male pigs fed different diets. Within each group, pigs were slaughtered on two occasions, not randomly but depending on the weight, meaning that the first group to be slaughtered had a faster growth than the other pigs. Since growth is linked to maturity and therefore steroid concentrations, the whole experimental design is questionable because it has been known for decades that androstenone, a steroid, is linked to maturity and, furthermore, skatole concentrations increase with weight. To finish my comments on the study design, the authors additionally fed 600 g of starch without compensation in the control group. From a scientific point of view this is highly questionable and should be accepted with extreme care.

Prof. Lundström stated that cross-validation of methods to measure boar taint (androstenone and, for a less part, skatole) is missing. A real cross-validation needs different assays with different detection principles from different laboratories. So, Prof. Lundström's claim is somewhat too optimistic; particularly some of her analytical approaches are not validated (e.g. cross-reactivities of the antibody). The ELISA procedures are not repeatable since the spiking of standards is not described and the evaporation step is not checked for losses due to the volatility of androstenone. I suppose that the authors used an antibody similar to that of Squires and Lundström (1997) with cross-reactivities below 3% for related steroids. Unfortunately, no cross-reactivities are given for 5α-androsten-3α-ol or 5α-androsten-3β-ol since they are the most related to the supposed but never proven sulphated androstenone. There is no example in the literature that an antibody will react with a free and sulphated steroid in a comparable way unless it is a very unsuitable one.

Some of the figures and tables seem to be interpreted in a wrong way. Fig. 2 reveals that the colorimetric determination underestimates skatole values compared with HPLC, as shown by the equation, which is puzzling because indole also contributes to equivalents. Therefore one of the "validated" methods seems to be wrong. Without ulterior motive, it would be interesting to see the correlation without the five or ten highest values. Overall, colorimetric determinations are old-fashioned and should not be used in relevant analytical problems. After serious thought, I believe that the equation in Fig. 3 is wrong because the slope of 1.003 does not fit to the figure at all. Table 4 gives percentages of skatole and androstenone-tainted samples and shows decreasing values for skatole and increasing values for androstenone with increasing weight. How could there be a positive correlation between these parameters, as described earlier, several times by the authors themselves?

As Prof. Lundström will agree, androstenone is a pheromone in the pig, together with the above-mentioned androstenols. Why should the intestine be an active part of sulphoconjugation of a pheromone? Androstenone sulphate, as assumed but not proven by Sinclair and Squires (2005), includes the formation of an intermediate and highly unstable enol steroid (Hawkinson, Eames, & Pollack, 1991), followed by subsequent sulphation. No other keto steroid has been shown to be sulphated, in that way to a great extent, previously. The supposed sulphated androstenone should be isolated and analyzed directly by LC–MS, including a synthesis of the substance, which is state-of-the-art knowledge. This should be done before a prolonged and unneccesary discussion about this substance.

Finally, even if we accept 1 μg/g for androstenone as the threshold level, there remains the question of how to deal with 22% (weight 90 kg) or even 44% (115 kg) of tainted carcasses. Maybe swedish consumers will accept tained carcasses but the overwhelming rest of Europe will not. Careful analytical chemists (as I hope Prof. Lundström and collegues are) should always consider that their statements may influence political decisions. The only solution is not to use entire male pigs for fattening at all and to castrate or immunocastrate male pigs.

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References

Hawkinson, D. C., Eames, T. C., & Pollack, R. M. (1991). Kinetic competence of an externally generated dienol intermediate with steroid isomerase. *Biochemistry, 30*, 6956–6964.

Sinclair, P. A., & Squires, E. J. (2005). Testicular sulphoconjugation of the 16-androstenone steroids by hydroxysteroid sulfotransferase: Its effect on the concentration of 5α-androstenone in plasma and fat of the mature domestic boar. *Journal of Animal Science, 83*, 358–365.

Squires, E. J., & Lundström, K. (1997). Relationship between cytochrom P450IIE1 in liver and levels of skatole and its metabolites in intact male pigs. *Journal of Animal Science, 75*, 2506–2511.

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