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▶ To cite this version:

Florence Gondret, Sandrine Tacher, Hélène Gilbert. Modulation of redox state in pigs differeing in feed efficiency as revealed by a proteomic analysis. 65. Annual Meeting of the European Federation of Animal Science (EAAP), Aug 2014, Copenhague, Denmark. Wageningen Academic Publishers, Annual Meeting of the European Association for Animal Production, 20, 2014, Annual Meeting of the European Association for Animal Production. hal-01210669

HAL Id: hal-01210669

https://hal.science/hal-01210669

Submitted on 3 Jun 2020

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Book of Abstracts of the 65th Annual Meeting of the European Federation of Animal Science



Book of abstracts No. 20 (2014) Copenhagen, Denmark 25 - 29 August 2014

Session 13

Theatre 2

Modulation of redox state in pigs differing in feed efficiency as revealed by a proteomic analysis F. Gondret¹, S. Tacher¹ and H. Gilbert²

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Improving feed efficiency of livestock is an important goal for sustainability and profitability. The physiological bases of feed efficiency are multifaceted. The functionality of the liver, a highly specialized organ with a wide range of functions including protein synthesis, energy metabolism and detoxification may be involved in variations of feed efficiency. In this study, two lines of pigs from a divergent selection experiment on residual feed intake (RFI) were compared. Two groups (RFI-: the most efficient; RFI+: the less efficient) were fed ad libitum during the growing-finishing period. A third group composed of RFI+ pigs was feed-restricted to the level of spontaneous feed intake of RFI- pigs. At slaughter (110 kg BW), the liver was excised. Soluble hepatic proteins (n=4 pigs per group) were separated by two-dimensional gel electrophoresis (2-DE) and spots having a differential abundance between groups were submitted to MALDI-TOF/TOF identification. A total of 50 protein spots had a differential abundance between the pigs, among which 35 proteins were identified. Members of the peroxiredoxin family (PRDX2, 4, 5 and 6), the omega form of the glutathione-S transferase (GSTO1) involved in the gluthatione derivative biosynthetic process, and the superoxide dismutase (SOD)-1 responsible for destroying free superoxide radicals, had a greater abundance in RFI- than in RFI+ pigs. Feed-restriction in the RFI+ pigs resulted in an increased abundance of the peroxiredoxins and the mu form of the glutathione-S transferase. Activity levels of key enzymes in the anti-oxidant or oxidative pathways in the liver were also measured. Notably, the activities of the oxidative enzymes hydroxyacyl-CoA dehydrogenase and citrate synthase were lower in RFI- pigs than in RFI+ pigs; they did not vary with feed restriction. In conclusion, selection for feed efficiency may have modified the redox state of the animals.

Session 13

Theatre 3

The control of feed intake by metabolic oxidation in dairy cows

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High-yielding dairy cows enter a negative energy balance after calving because feed intake is too low to meet the energy requirements for maintenance and milk production. At this time, cows mobilize their fat reserves - a process that is thought to interfere with the increase of feed intake. The control of feed intake results from the integration of humoral and nerval signals at feed intake regulatory centers in the brain. Metabolic oxidation in peripheral organs, primarily in the liver, may activate nerval signaling which is integrated and translated into anorexic responses by neurons of the hypothalamus (Ht). To study the relationship between metabolic oxidation and feed intake, cows were kept in respiration chambers with simultaneous recording of gas exchange and feed intake. All feed intake events were strongly cross-correlated with carbohydrate and fat oxidation in late-lactating cows. Transition cows with higher fat mobilization capacity (H cows) exhibited the greater fat oxidation, accompanied by the greater expression of hepatic enzymes involved in various fatty acid oxidation pathways and oxidative phosphorylation, each before and after parturition. H cows revealed greater plasma acyl ghrelin concentrations and the higher acyl:total ghrelin ratio, before and after parturition. The acyl:total ghrelin ratio correlated with several aspects of fat metabolism, but not with feed intake, implying that the ghrelin system plays a prominent role in fat allocation and oxidation rather than exerting orexigenic effects. Also, H cows had less activated orexigenic neurons in the arcuate nucleus of the Ht as indicated by the lower percentage of cFOS activated agouti-related protein (AgRP) neurons. Differential AgRP neuron activation was not associated with feed intake but with decreased oxygen consumption, increased respiratory quotient, and reduced NEFA concentrations, suggesting that AgRP activation plays a pivotal role in the regulation of metabolic oxidation during early lactation.