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To cite this version:

HAL Id: hal-01865604
https://hal.science/hal-01865604
Submitted on 2 Jun 2020

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Hepatic molecular changes induced by a high-fat high-fibre diet in growing pigs
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The introduction of more fibre to cereal-based diets in pigs gained interest due to new economic considerations and to potential health and welfare benefits. The addition of fat to a fibre-rich diet is required to maintain dietary energy value for performance. Feeding pigs a high-fat high-fibre diet, however, changes the energy source and nutrients as compared to a low-fat high-starch diet. The liver plays a central role in energy metabolism. This study was undertaken to investigate hepatic molecular pathways in pigs fed diets with contrasting sources of energy and nutrients. From 74 d of age onwards, 48 Large White castrated male pigs were fed a high-fat high-fibre diet (HF, n=24) or a low-fat high-starch diet (LF, n=24). Diets were formulated to be isoenergetic and isoproteic. Starch derived from cereal grains (wheat and barley) in the LF diet was partially replaced by rapeseed and soybean oils in the HF diet and crushed wheat straw (insoluble fibre) was included as a diluent of dietary energy in this diet. At 132 d of age, the liver was excised, weighed and processed for biochemical and molecular analyses. Transcriptomics analysis was performed using porcine microarrays (Agilent, GPL16524, 8 × 60K). Functional pathways were deduced from genes declared as differentially expressed (P<0.01) using DAVID Bioinformatics Resources and Ingenuity Pathway Analysis. Compared with LF pigs, HF pigs had a lower ADG and ADFI (P<0.01) during the test period. At slaughter, the liver was lighter (-7%, P=0.03) in HF than in LF pigs. In liver, the protein content was unaffected but the glycogen content and glucokinase activity were reduced in HF pigs compared with LF pigs (P<0.05). A total of 802 annotated genes were differentially expressed between the two diets. In HF pigs, genes involved in glycogen and hexose metabolism and genes participating to oxidative phosphorylation and ATP synthesis were down-regulated. Conversely, genes contributing to cell growth, cell cycle phase, cell death and cell adhesion were up-regulated. Liver hyper-proliferation, hepatic fibrosis, and liver necrosis were suggested as top-toxicity functions responding to diet. In conclusion, pig liver functions can be affected by dietary components such as fibres and lipids.