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# Effect of Individualized Feeding Strategy on Plasma Indicators of Energy Balance of Holstein Cows in the 12 First Weeks of Lactation

C. Gaillard, M. Vestergaard, M.R. Weisbjerg, J. Sehested

Aarhus University, Foulum, 8830 Tjele, Denmark

In early lactation dairy cows are in negative energy balance (NEB), the extent and duration depends partly on the feeding strategy (Friggens et al 2007). Studies showed an increased lactation milk yield by use of an individualized feeding strategy with a high energy diet before, and a low energy diet after, live weight nadir, comparing to a standardized diet throughout lactation (Bossen and Weisbjerg 2009). The objective of the present study was to examine how such an individualized feeding strategy affects plasma indicators of energy balance in the 12 first weeks of lactation. It was hypothesized that an individualized feeding strategy with a high energy diet until live weight nadir will reduce the magnitude of NEB in that period, and that a reduction in diet energy concentration starting at live weight nadir will extend the NEB period further, as detected by the levels of metabolites in blood. Plasma glucose, beta-hydroxybutyrate (BHBA), and non-esterified fatty acids (NEFA) are relevant indicators of energy balance (Duffield et al 2009).

Sixty-two Holstein cows, managed for 16 months lactation, were randomly allocated to one of two feeding strategies (NOR or EXP) at calving. On the NOR strategy, cows were fed a diet with 60:40 forage:concentrate ratio throughout lactation, whereas on the EXP strategy cows were fed a diet with a higher energy concentration (50:50 forage:concentrate ratio) until they reached  $\geq 42$  DIM and a live weight gain  $\geq 0$  kg/d based on a five-day average, and were then shifted to the diet of the NOR group. Weekly blood samples were drawn for analysis of plasma glucose, BHBA and NEFA until 12 weeks after calving.

Weekly data per cow within two periods were evaluated using a linear mixed-effects model using the software R with the lme function to estimate LSMmeans. Treatment, parity and weeks were included as fixed effects. A random function indicated that the cows were attributed to a treatment at random. A covariance structure of order 1 (AR1) by animal was used to take into consideration the correlation between weeks on the same cows. There were no interactions between treatment, parity and week. Data were analyzed for two periods, before and after the shift in diet. From weeks 1 to 5 after parturition, the EXP group had higher levels of glucose and lower levels of BHBA and NEFA than the NOR group. In second period (week 10 to 12), all the EXP cows had shifted to NOR, and metabolites concentrations were then similar for EXP and NOR cows (Table 1).

**Table 1. Metabolites concentrations (LSMmeans) before and after the shift in diet**

|              | Concentrations before the shift |     |      |         | Concentrations after the shift |     |      |         |
|--------------|---------------------------------|-----|------|---------|--------------------------------|-----|------|---------|
|              | EXP                             | NOR | SE   | P-value | EXP                            | NOR | SE   | P-value |
| Glucose (mM) | 3.8                             | 3.6 | 0.05 | 0.02    | 3.7                            | 3.8 | 0.05 | 0.7     |
| BHBA (mM)    | 0.6                             | 0.8 | 0.06 | 0.04    | 0.7                            | 0.6 | 0.05 | 0.1     |
| NEFA (eqv/L) | 387                             | 483 | 28.1 | 0.02    | 151                            | 135 | 13.1 | 0.4     |

To conclude, the individualized feeding strategy with a high energy diet until live weight nadir reduced the magnitude of NEB in that period as described by increased plasma glucose, and decreased NEFA and BHBA concentrations. The metabolites concentrations, for the second period, were similar for both groups. Cows on EXP were more stable, during the 12 first weeks of lactation, compared to the NOR cows. This last group had largest changes in metabolites concentrations from the first period to the second, particularly regarding NEFA's concentrations, that can induce more metabolic disorders.

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Email: charlotte.gaillard@agrsci.dk