Abstract Summary

Title: Rumen-protected choline (RPC) influences hepatic metabolism during induction of fatty liver

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Objective: Determine the effect of sources of RPC with a low (28.8%) or high concentration (60.0%) of choline chloride (CC) on hepatic metabolism.

Treatments:

1. Holstein cows (n=110) were blocked by body condition and fed the following treatments for 14 days:
    - 0 g/d RPC (**CON**)
    - 12.9 g/d of choline ion from RPC containing 28.8% CC
    - 25.8 g/d of choline ion from RPC containing 28.8% CC
    - 12.9 g/d of choline ion from RPC containing 60.0% CC
    - 25.8 g/d of choline ion from RPC containing 60.0% CC
2. Feed was restricted to 50% of the net energy for lactation in the last 9 days
3. Intake of metabolizable methionine was maintained at 20 g/d for the 14-day experiment

Results:

- Cows fed RPC had:
    1. reduced hepatic triacylglycerol (greater effect with larger dose of RPC)
    2. increased hepatic glycogen (greater effect with larger dose of RPC)
    3. tended to have greater concentration of TAG in lymph 6 h after fat feeding and increasing the amount of RPC tended to increase serum TAG.
    4. altered mRNA expression of genes related to gluconeogenesis, hepatic uptake of fatty acids, oxidation and reesterification of fatty acids, export of TAG, synthesis of antioxidants, autophagy, and lipophagy.

Take Home Messages: This research provides further evidence that feeding RPC during negative nutrient balance promotes lipotropic effects that reduces the risk of fatty liver in dairy cows.

Full Abstract

Rumen-protected choline (RPC) influences hepatic metabolism during induction of fatty liver

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Objectives were to determine the effect of sources of RPC with a low (**L**, 28.8%) or high concentration (**H**, 60.0%) of choline chloride (CC) on hepatic metabolism. Holstein cows (n=110) at 232±3.9 d of gestation were blocked by body condition (4.02±0.50) and fed 0 (**CON**), 12.9 or 25.8 g/d of choline ion either as **L12.9, L25.8, H12.9,** or **H25.8** for 14 days. Feed was restricted to 50% of the net energy for lactation in the last 9 d, whereas intake of metabolizable methionine was maintained at 20 g/d for the 14-d experiment. Hepatic tissue was collected on d 13. On d 14, 450 g fatty acids (**FA**) were fed, and blood sampled for 21 h. Lymph was sampled at 6 h in cows fed CON, L25.8, and H25.8. Data were analyzed using mixed-effects models, and orthogonal contrasts were used to evaluate the effect of supplementing RPC [CON vs. ¼·(L12.9+L25.8+H12.9+H25.8)], source [½·(L12.9+L25.8) vs. ½·(H12.9+H25.8)], amount [½·(L12.9+H12.9) vs. ½·(L25.8+H25.8)], and interaction [½·(L12.9+H25.8) vs. ½·(H12.9+L25.8)]. Feeding RPC reduced hepatic triacylglycerol (**TAG**) and increased glycogen, and the effect was greater with the larger dose of RPC. Cows supplemented with RPC tended to have greater concentration of TAG in lymph after 6 h after fat feeding and increasing the amount of RPC tended to increase serum TAG. Supplementation of RPC altered the mRNA expression of genes related to gluconeogenesis, hepatic uptake of FA, oxidation and reesterification of FA, export of TAG, synthesis of antioxidants, autophagy, and lipophagy. Feeding RPC during negative nutrient balance promotes lipotropic effects that reduces the risk of fatty liver in dairy cows.

Item	CON	L12.9	L25.8	H12.9	H25.8	SE	RPC	Source	Amount
Liver, as-is %									
TAG	9.32	6.59	5.05	6.61	6.00	0.55	0.01	0.28	0.02
Glycogen	1.83	2.59	3.55	3.13	4.07	0.18	0.01	0.01	0.01
Serum TAG, mg/dL	15.5	15.3	16.8	16.8	20.0	1.6	0.22	0.08	0.07
Lymph TAG, mg/dL	11.4	-	15.7	-	15.9	3.4	0.08	0.98	-
Hepatic mRNA, fold change									
PC	1	1.06	0.91	1.18	0.99	-	0.76	0.21	0.05
CD36	1	1.12	1.22	1.16	1.06	-	0.05	0.39	0.94
ACADM	1	1.08	0.95	1.09	0.99	-	0.66	0.62	0.02
FASN	1	0.90	1.09	0.67	0.99	-	0.51	0.19	0.06
APOB100	1	1.08	1.15	0.92	1.21	-	0.34	0.46	0.04
SOD1	1	1.08	0.99	1.10	0.99	-	0.24	0.68	0.01
ATG3	1	1.18	1.12	1.09	1.11	-	0.01	0.31	0.64
RAB18	1	1.03	0.90	1.01	0.93	-	0.53	0.87	0.06

Key Words: choline, fatty liver, mRNA