

the development of fatty liver and limit milk production. Rumen-protected choline (RPC) have been fed to lactating cows to increase the supply of choline to the small intestine with the goal of increasing milk or alleviating the development of fatty liver syndrome [5; 12]. The objective of this study was to observe the correlation between NEFA, BHBA, TG and glucose in the blood plasma of early lactating dairy cows while feeding supplemental choline.

Eight early lactating primiparous and multiparous Holstein cows were used from October 2011 to November 2011 in our study beginning five weeks postpartum. Cows were housed in individual tie stalls and cared for under experimental procedures and protocols approved by the veterinary organization of Iran. Selection of cows was based on parity, milk yield of previous lactation (milk yield of dams for the cows in their first lactation) and body condition score (BCS). The cows received 90 g/d of rumen-protected choline (RPC) product. The RPC product (Reashure Choline, Balchem, USA) is a rumen-protected source of choline chloride. Reashure choline is produced by encapsulating choline chloride with a coating matrix able to resist rumen breakdown and release choline in intestine and contained 25% choline. Cows were fed a total mixed ration (TMR) *ad libitum*. The meal was adjusted to production intensity, and consisted of ordinary alfalfa hay, silage, beet pulp, and concentrates (including barley, corn, canola meal, cottonseed, wheat bran, cottonseed meal, wheat grain, corn gluten meal, soybean meal, sodium bicarbonate, fat meal, limestone and vitamin-mineral supplement). The RPC was top-dressed onto the TMR.

Blood samples were obtained before morning meal from the coccygeal vein on the last day and then were collected in heparinized Vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ). Blood samples were placed on ice immediately following collection. Plasma was harvested after centrifugation of the blood at 3000 g for 15 min. Plasma was stored at -20°C until subsequent analysis for NEFA, BHBA, TG and glucose. The metabolites were measured on “BT 1500 auto-analyzer” through spectrophotometer method, using kits produced by “Farasamed Co, Ltd. Tehran, Iran.

For statistical analysis, experimental data normality was verified, and then data were submitted to analysis of correlation, using SAS (2002) software package.

The data of plasma metabolites for the cows which received choline and the correlations between the metabolites are shown (in Table 1 & 2).

TG level had a positive correlation with glucose (0.393) and a negative correlation with NEFA (-0.189) and a significant negative correlation with BHBA (-0.485). Glucose had a negative correlation with NEFA (-0.338) and a significant negative correlation with BHBA (-0.543). NEFA had a significant positive correlation with BHBA (0.415).

After parturition the demand for energy is increased by the initiation of lactation. The negative energy balance is compensated by the mobilization of NEFA from adipose tissue [1,2]. Therefore, examination of plasma NEFA concentration during early lactation period provides insight into fatty liver development [13].

In the case of excessive fat mobilization, associated with marked formation of acetyl CoA, the tri-carboxylic acid cycle cannot fully metabolize fatty acids. As a consequence, acetyl CoA is converted to acetoacetate which is then reduced to BHBA by BHBA dehydrogenase or spontaneously decarboxylized to acetone [14]. BHBA concentration increased during early lactation period and NEFA provide the substrate for BHBA synthesis. Increased BHBA concentration reveals incomplete oxidation of NEFA in the tri-carboxylic acid cycle during negative energy balance [3].

Table 1

The plasma metabolites of the cows which received choline

Triglyceride	9.00 ± 1.88 (mg/dl)
Glucose	55.00 ± 4.13 (mg/dl)
NEFA	0.237 ± 0.08 (mmol/l)
BHBA	0.453 ± 0.12 (mmol/l)

Table 2

The correlation between plasma metabolites of the cows which received choline

Correlation	Triglyceride	Glucose	NEFA
Glucose	0.393	-----	-----
NEFA	-0.189	-0.338	-----
BHBA	-0.485*	-0.543*	0.415*

* P<0.05

Early lactating cows rely almost exclusively on gluconeogenesis in the liver to meet their glucose requirements. But the level of blood glucose decreases during early lactation due to high demands for lactose synthesis as well as insufficient gluconeogenesis [14, 15]. The rise in NEFA during early lactation expressed enhanced adipose tissue mobilization to cope with the high energy demand for milk synthesis, when the availability and oxidation of glucose was reduced [13].

Reduction in gluconeogenesis by the liver, due to accumulation of fat in the liver, may lower blood glucose levels and decrease insulin secretion, which would support greater lipid mobilization and increase rate of fatty acid uptake

by the liver and increase ketogenesis [3]. An increase in plasma glucose during early lactation will likely increase insulin and decrease NEFA concentration.

The results of our study show that feeding choline improves liver function due probably to increasing formation of VLDL and its export from liver into the blood. In result the amount of TG in blood increases and the level of NEFA decreases.

Due to increase of glucose in the blood, the secretion of insulin increases. Since the increase of insulin suppress fat mobilization from adipose tissue, the amount of NEFA decrease in the blood and as a result, the influx of NEFA into the liver decreases, then the liver has fewer problems for oxidation of NEFA, in consequence, the level of BHBA decreases in the blood.

As a conclusion, we can recommend that choline can be useful in the nutrition of early lactating dairy cows due to increasing formation of VLDL to export fat from liver which results in improvement of liver function.

Armenian State Agrarian University, M.Rahmani@live.com

**M. G. Rahmani, R. G. Kamalyan, M. J. Dehghan-Banadaky,
G. J. Eskandari**

The Correlation Between Energy Carriers in the Blood Plasma of Early Lactating Dairy Cows Receiving Supplemental Choline

In an experiment on early lactating Holstein cows it was shown the presence of correlation between non-esterified fatty acids (NEFA), β -hydroxyl butyric acid (BHBA), triglyceride (TG) and glucose in plasma while feeding diet with choline. TG had a positive correlation with glucose. TG and glucose shown negative correlations with NEFA and BHBA. NEFA had a positive correlation with BHBA. This finding indicates that feeding choline can be useful to divert fatty acids to phospholipid synthesis to export fat from liver as well as improving gluconeogenesis and reduction of fat mobilization from adipose tissue which results in reducing production of BHBA.

**Մ. Գ. Ռահմանի, Ռ. Գ. Քամալյան, Մ. Զ. Դեհջան-Բանադակի,
Գ. Զ. Եսկանդարի**

Էներգակրիչների կորելյացիան կովերի արյան պլազմայում վաղ կաթնատվության շրջանում կերի հետ հավելյալ խոլինի կերակրման պայմաններում

Հոլսթեյն ցեղի կովերի կաթնատվության վաղ շրջանում կերի հետ խոլինի կերակրման պայմաններում գրանցվում է հավաստի կորելյացիա արյան պլազմայի չեթերիֆիկացված ճարպաթթուների, β -հիդրօքսիկարագաթթվի, եռացիլգլիցերիդների և գլյուկոզի միջև: Եռացիլգլիցերիդների և գլյուկոզի միջև դիտվում է դրական կորելյացիա: Եռացիլգլիցերիդները և գլյուկոզը դրսևորում են բացասական կորելյացիա չեթերիֆիկացված ճարպաթթուների և β -հիդրօքսիկարագաթթվի համեմատ: Վերջիններս միջև դիտվում է դրական կորելյացիա: Տվյալները վկայում են, որ խոլինի կերային հավելումը նպաստում է ճարպաթթուների օգտագործմանը լյարդի ֆոսֆոլիպիդների սինթեզում, ճարպերի հեռացմանը և գլյուկոզի սինթեզին այդ

օրգանում, ինչը նվազեցնում է ճարպերի մոբիլիզացումը ճարպային հյուսվածքից և իջեցնում β -հիդրօքսիկարապաթթվի խտությունը արյան պլազմայում:

**М. Г. Рахмани, Р. Г. Камалян, М. Дж. Дехджан-Банадаки,
Г. Дж. Ескандари**

Корреляция между энергоносителями в плазме крови в ранний период лактации коров, получавших в качестве добавки холин

В опытах на коровах породы Голштейн показано наличие корреляции в плазме крови между неэстерифицированными жирными кислотами (НЖК), β -гидроксибутиратом, триглицеридами (ТГ) и глюкозой в ранний период лактации при скармливании холина. ТГ имели положительную корреляцию с глюкозой. ТГ и глюкоза показывали отрицательную корреляцию с НЖК и β -гидроксибутиратом. НЖК находилась в положительной корреляции с β -гидроксибутиратом. Данные указывают на то, что скармливание коровам холина может способствовать направлению обмена НЖК на синтез фосфолипидов и экспорту жиров из печени, а также глюконеогенезу и уменьшению мобилизации жиров из жировой ткани, что приводит к уменьшению продукции β -гидроксибутирата.

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