Comparison between feeding rumen-protected choline and vitamin E on milk yield and blood metabolites in early lactation dairy cows

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Abstract. Twenty-four early-lactation primiparous and multiparous Holstein cows, beginning 5 weeks postpartum, were used for 4 weeks to investigate the effects of feeding rumen-protected choline (RPC) or vitamin E on milk yield, dry matter intake (DMI), blood metabolites and plasma enzymes. Cows were randomly assigned to one of the following treatments: no supplement (control), 90 g/day of RPC, or 4400 IU/day of vitamin E. Treatments did not affect milk yield, DMI, plasma glucose, non-esterified fatty acids, blood urea nitrogen, aspartate aminotransferase or total bilirubin, whereas feeding RPC affected cholesterol, plasma albumin, and alkaline phosphatase (ALP). Feeding vitamin E affected triglyceride, cholesterol, \( \beta \)-hydroxy butyric acid, gamma-glutamyl transferase and alanine aminotransferase, and highly affected plasma albumin and ALP. There was a tendency for vitamin E supplementation to increase plasma total protein concentration. The results of this study suggest that supplemental vitamin E may improve liver function in dairy cows in early lactation.

Additional keywords: cattle, fatty liver, ketosis, gluconeogenesis, hepatic lipidosis, metabolic parameters.

Introduction

Rapid increase in milk production in cows after calving leads to a period of reduced dry matter intake, negative energy balance, lipolysis (Goff and Horst 1997) and insulin resistance (Hayirli 2006); thus, in early-lactation cows, the concentrations of some blood metabolites and enzymes that are indicative of liver function may be altered (Kida 2002).

Choline is a vitamin-like compound that is an essential component of phospholipids, and it participates in the structure of lipoproteins that export lipids from liver in the form of very low-density lipoproteins. Hence, choline deficiency in dairy cows may be associated with hepatic lipidosis (Hartwell et al. 2000). Most dietary choline is degraded by rumen microorganisms (Sharma and Erdman 1989); therefore, choline should be in a rumen-protected form when fed. Rumen-protected choline (RPC) is fed to dairy cows to increase the supply of choline to the cows, with the goal of alleviating the development of fatty liver by enhancing export of fat from liver (Piepenbrink and Overton 2003; Cooke et al. 2004). Early-lactation cows may produce more milk when they receive RPC (Erdman and Sharma 1991; Pinotti et al. 2003), because RPC supplementation may spare methionine (Pinotti et al. 2002; Piepenbrink and Overton 2003; Overton and Waldron 2004).

Vitamin E (\( \alpha \)-tocopherol) is a powerful antioxidant that lowers oxidative stress and influences the health of dairy cows (Burton and Traber 1990), and is not degraded in the anaerobic ruminal environment (Burton and Traber 1990; Leedle et al. 1993). Vitamin E is absorbed and transported from intestine to liver following the same mechanisms as lipids. In the liver, \( \alpha \)-tocopherol is packaged in lipoproteins and distributed from the liver via plasma to the rest of the body (Herdt and Smith 1996). Vitamin E improves the free radical defence system potential and has a beneficial effect in improving glucose transport and insulin sensitivity (Paolisso et al. 1994; Faure et al. 1997). In a study on humans, vitamin E supplementation in patients with non-alcoholic fatty liver disease (NAFLD) was effective in lowering aminotransferases (aspartate and alanine aminotransferases, AST and ALT) (Patel and Babich 2010). A study on rats showed that the group supplemented with vitamin E presented the lowest average aminotransferase levels among the animals with NAFLD, but with no statistical significance (Zamin et al. 2010). In obese children with NAFLD, vitamin E has been demonstrated to improve liver function (Lavine 2000). In cows, plasma concentration of vitamin E decreases through periparturient and early lactation periods (Weiss et al. 1990).

Cows are susceptible to fatty liver and ketosis in early lactation, and most dairy farmers use choline to improve production performance. Thus, we decided to study whether vitamin E also affected production performance in dairy cows during early lactation. Therefore, the objectives of this study were...
to investigate the effects of supplementation of RPC or vitamin E, and to compare the effects of RPC with vitamin E on milk yield, dry matter intake (DMI) and metabolic parameters in dairy cows in early lactation.

Materials and methods
Cows, treatments and experimental design
Twenty-four Holstein cows (9 primiparous and 15 multiparous) in early lactation (days in milk 29 ± 10.25, body condition score (BCS) 2.82 ± 0.12; mean ± s.d.) were used for 4 weeks from October 2011 to November 2011. The cows were housed in individual tie stalls and cared for under experimental procedures and protocols approved by the veterinary organisation of Iran. Selection of the cows was based on parity, milk yield of previous lactation (milk yield of dams for the cows in their first lactation) and BCS. Eight cows per treatment (average lactation number 2.56) were randomly assigned to receive one of the following treatments: no supplement (control), 90 g/day of RPC or 4400 IU/day of vitamin E. The RPC product (ReaShure; Balchem, New Hampton, NY, USA; 25% choline) was a rumen-protected source of choline chloride, and the vitamin E was the product of Roche Co. (Basel, Switzerland). The cows were fed total mixed rations (TMR) ad libitum and were milked three times daily at 8-h intervals, with no provision of water or concentrate while milking. Milk yields were measured every day.

The diet (Table 1) was formulated to meet the nutrient requirements of dairy cows (NRC 2001). The RPC and vitamin E were topdressed onto the TMR. Samples of the TMR and orts were taken once per week and frozen until the samples were processed for analysis. Weekly feed and orst samples were collected for 4 weeks. All samples were dried at 60°C for 72 h, and then weighed to determine moisture loss. Dried samples were ground through a 1-mm screen and the nutrient contents of the samples were analysed weekly. Chemical analyses of the samples were based on AOAC (AOAC 1990) method number 925.40 for DM, 923.03 for ash, 984.13 for crude protein (CP) and 972.16 for total fat. Neutral detergent fibre (NDF) and acid detergent fibre (ADF) were determined as described by Van Soest et al. (1991). The NDF was assayed with a heat-stable amylase, and both NDF and ADF were expressed inclusive of residual ash. Non-fibrous carbohydrate was calculated as the difference between 100 and the sum of CP, NDF, total fat and ash. The amounts of feed offered and refused were individually measured every day throughout the experiment, and weekly analyses of DM content of the TMR were used to calculate DMI.

Data and sample collection
Blood samples were obtained from the coccygeal vein (tail vein) before the morning meal on the last day of the experiment. Blood samples were collected in heparinised and non-heparinised Vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ, USA), and were then placed on ice immediately following collection. Plasma and serum were harvested after centrifugation of the blood at 3000g for 15 min. The plasma was stored at −20°C until subsequent analyses for glucose, albumin, triglycerides, cholesterol, non-esterified fatty acids (NEFA), β-hydroxybutyric acids (BHBA), gamma-glutamyl transferase (GGT), AST, alkaline phosphatase (ALP), ALT, total bilirubin, globulin and total protein. The serum was stored at −20°C until subsequent analysis for blood urea nitrogen (BUN). All metabolites were measured on a BT 1500 auto-analyser (Biotecnica Instruments SpA, Rome) via spectrophotometer method, using kits produced by Farasamed Co., Tehran, Iran.

Statistical analyses
Raw data were transformed to their natural logarithm to achieve a normal distribution for analysis. All transformed data were back-transformed for reporting least-squares means. Statistical analyses were performed with SAS software (SAS 2002); the GLM model was used for blood metabolites, but repeated-measurement data including milk production and DMI were analysed by ANOVA for repeated-measures using PROC MIXED of the SAS/STAT program (SAS Institute, Cary, NC, USA). The most appropriate covariance structure was selected based on the smallest Akaike information criterion. The model included the effects of treatment, day of measurement, interaction of treatment × day of measurement, and parity, with cow nested within treatment as a random effect. Significant levels were declared at $P = 0.05$.

### Table 1. Ingredients and nutrient composition of the diet

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>(g/kg DM)</th>
<th>Chemical composition (in DM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lucerne hay (medium chopped)</td>
<td>204.7</td>
<td>DM (g/kg) 590</td>
</tr>
<tr>
<td>Corn silage</td>
<td>175.8</td>
<td>CP (g/kg) 171.7</td>
</tr>
<tr>
<td>Beet pulp</td>
<td>41.3</td>
<td>Ash (g/kg) 57.6</td>
</tr>
<tr>
<td>Ground barley grain</td>
<td>198.8</td>
<td>Total fat (g/kg) 43.8</td>
</tr>
<tr>
<td>Ground corn grain</td>
<td>58.7</td>
<td>NDF (g/kg) 308.4</td>
</tr>
<tr>
<td>Ground wheat grain</td>
<td>28.5</td>
<td>ADF (g/kg) 183.8</td>
</tr>
<tr>
<td>Solvent-extracted soybean meal</td>
<td>79.9</td>
<td>NFC (g/kg) 382.1</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>7.1</td>
<td>Ca (g/kg) 8.1</td>
</tr>
<tr>
<td>High-limit whole cottonseed</td>
<td>29.5</td>
<td>P (g/kg) 5.0</td>
</tr>
<tr>
<td>Canola meal</td>
<td>100.2</td>
<td>NEL (MJ/kg) 6.95</td>
</tr>
<tr>
<td>Corn gluten meal</td>
<td>11.5</td>
<td>RUP (g/kg of CP) 314.5</td>
</tr>
<tr>
<td>Fat supplement (energy booster)</td>
<td>15.9</td>
<td>RDP (g/kg of CP) 685.5</td>
</tr>
<tr>
<td>Mineral and vitamin supplement&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.6</td>
<td>Met (g/kg MP) 22.1</td>
</tr>
<tr>
<td>Salt</td>
<td>2.5</td>
<td>Lys (g/kg MP) 76.9</td>
</tr>
<tr>
<td>Calcium carbonate</td>
<td>19.0</td>
<td></td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>10.2</td>
<td></td>
</tr>
<tr>
<td>Di-calcium phosphate</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>Magnesium oxide</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td>Mycosorb&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td>Biotin premix</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td>Zeolite</td>
<td>19.0</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Containing: (g/kg) 190 Ca, 90 P, 30 Mg, 4 Fe, 0.5 Cu, 5 Mn, 4 Zn, 0-1 Co, 0.1 I, 0.03 Se, 0-4 antioxidant; (IU/kg): 5 × 10<sup>5</sup> vitamin A, 10<sup>5</sup> vitamin D, 3 × 10<sup>3</sup> vitamin E.

<sup>b</sup>Mycotoxin binder (Alltech, Nicholasville, KY, USA).
Results

The effects of the supplemental choline and vitamin E on milk yield, DMI and blood metabolites of the experimental cows are shown in Table 2. Feeding either RPC or vitamin E did not affect milk yield (P = 0.65), DMI (P = 0.75), or the concentrations of plasma glucose (P = 0.13), NEFA (P = 0.16), BUN (P = 0.28), AST (P = 0.43), total bilirubin (P = 0.55), globulin (P = 0.85) and total protein (P = 0.07). Compared with unsupplemented animals, those that received choline had differences in blood cholesterol, and those that received vitamin E had differences in blood triglyceride, cholesterol, BHBA, GGT and ALT (P < 0.05). For both choline and vitamin E groups, there were differences (P < 0.01) in plasma albumin and ALP compared with the control group.

Discussion

Milk production and intake

In the present study, there were no differences in milk production between cows receiving supplemental RPC or vitamin E or those unsupplemented. Conflicting results have been reported with respect to the effects of RPC supplementation. Zahra et al. (2006) reported that Holstein cows receiving 56 g/day of RPC from 2 weeks prepartum to 4 weeks postpartum produced, on average, 1.2 kg/day more milk than the unsupplemented group. In agreement, Piepenbrink and Overton (2003) reported no differences in milk yields when cows were supplemented with different amounts (0, 45, 60 and 75 g/day) of RPC from 21 days before expected calving to 63 days postpartum (Piepenbrink and Overton 2003). However, Pinotti et al. (2002) reported that cows consuming 20 g/day of RPC from 14 days before the expected calving date and to 30 days postpartum produced 3.5 kg/day more milk than unsupplemented cows. Guretzky et al. (2006) fed Holstein and Jersey cows diets that were topdressed once daily with 60 g of RPC from 21 days prepartum to 21 days postpartum and found that daily yield of milk increased by 2 kg/day compared with the control group. The differences in response to RPC might be related to the quality of the products used and the protection method against rumen degradation, because differences in the degree of rumen-protection have been shown for different products (Kung et al. 2003). In addition, differences among studies could be due to differences in the nutritive value of the diets being fed and the production of choline by rumen bacteria.

The lack of effects of dietary vitamin E on DMI and milk yield agree with the study of Zhao et al. (2008), in which dairy cows, 94 days in milk, were supplemented with vitamin E at 5000 and 10 000 IU/cow.day for 6 weeks. In another experiment, milk yield was also unaffected in animals were assigned to either a basal diet (containing 1000 IU/day of vitamin E) or a diet with an extra 1000 IU/day of vitamin E (total 2000 IU), from 14 days before expected calving to 7 days postpartum (Baldi et al. 2000).

Administration of RPC or vitamin E did not affect DMI (P = 0.75; Table 2). In agreement, several studies have reported that RPC supplementation had no effect on DMI in dairy cows (Piepenbrink and Overton 2003; Guretzky et al. 2006; Zahra et al. 2006; Davidson et al. 2008). In accordance with our research, Erdman and Sharma (1991) indicated that supplementing early-lactation cows with 0, 15, 30 or 45 g/day of RPC from week 5 to week 21 postpartum did not affect DM intake. Similarly, Piepenbrink and Overton (2003) fed cows 0, 45, 60, or 75 g/day of RPC from 21 days before expected calving to 63 days postpartum and found no effect of RPC supplementation on DMI. Likewise, Pinotti et al. (2003) found

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>Treatment group Choline</th>
<th>Treatment group Vitamin E</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk (kg/day)</td>
<td>34.70 ± 0.98</td>
<td>35.01 ± 0.73</td>
<td>36.75 ± 0.90</td>
<td>0.6550</td>
</tr>
<tr>
<td>DMI (kg/day)</td>
<td>21.43 ± 0.68</td>
<td>22.68 ± 0.60</td>
<td>23.26 ± 0.70</td>
<td>0.7520</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>48.25 ± 3.39</td>
<td>55.00 ± 1.92</td>
<td>54.86 ± 1.81</td>
<td>0.1310</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>3.21 ± 0.06b</td>
<td>3.61 ± 0.07 a</td>
<td>3.69 ± 0.10 a</td>
<td>0.0011</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>9.50 ± 0.46a</td>
<td>9.00 ± 0.38ab</td>
<td>7.57 ± 0.56 b</td>
<td>0.0371</td>
</tr>
<tr>
<td>Cholesterol (mg/dL)</td>
<td>181.37 ± 8.91a</td>
<td>157.37 ± 3.55b</td>
<td>154.14 ± 9.06b</td>
<td>0.0489</td>
</tr>
<tr>
<td>NEFA (mmol/L)</td>
<td>0.32 ± 0.04</td>
<td>0.24 ± 0.02</td>
<td>0.25 ± 0.02</td>
<td>0.1579</td>
</tr>
<tr>
<td>BHBA (mmol/L)</td>
<td>0.48 ± 0.03a</td>
<td>0.45 ± 0.03a</td>
<td>0.37 ± 0.02 b</td>
<td>0.0153</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>14.40 ± 0.65</td>
<td>15.11 ± 0.60</td>
<td>15.89 ± 0.54</td>
<td>0.2793</td>
</tr>
<tr>
<td>GGT (units/L)</td>
<td>29.00 ± 1.58a</td>
<td>26.50 ± 0.96ab</td>
<td>23.86 ± 0.83 b</td>
<td>0.0284</td>
</tr>
<tr>
<td>ALP (units/L)</td>
<td>74.00 ± 2.80</td>
<td>71.50 ± 4.62</td>
<td>66.85 ± 3.18</td>
<td>0.4298</td>
</tr>
<tr>
<td>ALT (units/L)</td>
<td>60.62 ± 2.68a</td>
<td>42.87 ± 3.68b</td>
<td>42.43 ± 2.06b</td>
<td>0.0004</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.25 ± 0.06</td>
<td>0.20 ± 0.05</td>
<td>0.17 ± 0.02</td>
<td>0.5542</td>
</tr>
<tr>
<td>Globulin (g/dL)</td>
<td>3.60 ± 0.14</td>
<td>3.61 ± 0.12</td>
<td>3.70 ± 0.12</td>
<td>0.8499</td>
</tr>
<tr>
<td>Total protein (g/dL)</td>
<td>6.84 ± 0.17</td>
<td>7.21 ± 0.16</td>
<td>7.39 ± 0.13</td>
<td>0.0746</td>
</tr>
</tbody>
</table>
no influence of RPC supplementation on DMI when transition cows were fed 20 g/day of RPC from 14 days prepartum to 30 days postpartum.

**Blood metabolites**

Feeding RPC and vitamin E did not affect plasma glucose concentration ($P = 0.13$). The lack of an effect of RPC on plasma glucose concentration is in agreement with other studies (Guretzky et al. 2006; Cooke et al. 2007). However, Hartwell et al. (2000) reported that RPC supplementation in dairy cows from 28 days before expected calving to 120 days of lactation tended to increase average plasma glucose concentration. In another study, RPC-treated cows recorded higher blood serum glucose concentration (Soltan et al. 2012). The role of choline and vitamin E in glucose metabolism is not evident; however, reducing the severity of lipid accumulation in the liver would favour hepatic gluconeogenesis (Cadorna-Galindo et al. 1997). Reduction in gluconeogenesis, due to accumulation of fat in the liver, lowers blood glucose levels and decreases insulin secretion, which would support greater lipid mobilisation (Grummer 1993).

The effects of feeding RPC and vitamin E on plasma albumin were highly significant ($P < 0.01$). In a study on ewes, the data demonstrated that vitamin E supplementation from 2 weeks before mating through pregnancy until lambing improved levels of albumin, globulin and total serum protein in treated ewes (El-Shahat and Monem 2011). A similar finding was obtained in buffaloes (Helal et al. 2009). Part of the effect of choline and vitamin E might be the result of improvement in liver function, or might be due to increased concentration of insulin (because of increase in gluconeogenesis), which lowers plasma NEFA concentrations through reduced lipolysis. However, insulin was not measured in the current study, and thus, we cannot assess whether the latter hypothesis holds. Albumin is associated with postpartum diseases and can be used to predict disease risks in early lactation (Saun 2004). In early lactation, cows with blood albumin concentrations $\geq 3.5$ g/dL are less likely to have postpartum diseases, whereas when albumin concentrations were $< 3.25$ g/dL, cows present a 3-fold greater risk of diseases, and when cows have low albumin concentrations ($< 3.0$ g/dL), they fail to respond properly to disease challenge (Saun 2004). Therefore, in our study, because of increases in albumin concentration of the treated cows, we could speculate that the treated cows might be more resistant to various diseases than the control group.

The effect of choline on plasma triglyceride concentrations was not significant ($P > 0.05$), but the effect of vitamin E was significant ($P < 0.05$). Davidson et al. (2008) reported that RPC-treated cows, from 21 to 91 days in milk, exhibited lower serum triglyceride concentrations than unsupplemented cows, and Soltan et al. (2012) reported a similar result for RPC-treated cows, from the first day to week 12 postpartum. These findings are not in agreement with the results of our research. In an experiment on lambs, feeding vitamin E for 42 days did not affect the content of plasma triglyceride (Gabryszuk et al. 2007).

In our study, the decrease in plasma triglyceride level in the vitamin E group might be due to increase in insulin activity, because vitamin E supplementation can improve insulin activity (Cinz et al. 1999), but since insulin was not measured in the current study, this hypothesis cannot be proven.

Supplementation of cows with RPC and vitamin E decreased the concentration of cholesterol ($P < 0.05$). For choline, our results disagree with Soltan et al. (2012), who reported that supplementing early-lactation Holstein dairy cows with 30 g/day of RPC for 12 weeks increased cholesterol concentration. Pinotti et al. (2003) reported that supplementing transition dairy cows with 20 g/day of RPC, from 14 days prepartum to 30 days postpartum, had no effect on plasma cholesterol concentrations. The smaller sample size and the lower dose of RPC in the study of Pinotti et al. (2003) may explain the difference. In agreement with our study, Zahra et al. (2006) and Guretzky et al. (2006) reported that cows receiving RPC had lower serum cholesterol concentration than cows not receiving RPC. For vitamin E, our finding disagrees with that of Weiss et al. (1994), who determined that feeding 890 IU/cow.day of supplemental vitamin E from 2 weeks pre-calving to 2 weeks post-calving did not affect plasma cholesterol concentrations. In our study, the decrease in plasma cholesterol might be due to incorporation of cholesterol in very low-density lipoprotein synthesis, because cholesterol is a component of lipoproteins (Kaneene et al. 1997).

There were no effects of either RPC or vitamin E supplementation on NEFA concentrations ($P = 0.16$). Conflicting results have been reported with respect to the effects of RPC supplementation on NEFA. The lack of an effect of RPC supplementation on plasma NEFA concentration is in agreement with others (Hartwell et al. 2000; Piepenbrink and Overton 2003; Guretzky et al. 2006; Zahra et al. 2006; Davidson et al. 2008). However, some studies have reported decreased concentrations of plasma NEFA in response to RPC supplementation (Pinotti et al. 2003; Cooke et al. 2007). Plasma concentrations of NEFA were decreased in Chinese Holstein dairy cows supplemented with 30 g/day of RPC from 15 days before expected calving to 15 days postpartum (Xu et al. 2006). Soltan et al. (2012) reported that RPC supplementation reduced blood serum NEFA, and Pinotti et al. (2003) reported that when cows were supplemented with 20 g/day of RPC, from 14 days prepartum, the concentrations of NEFA were reduced on the day of calving. In our study, blood samples were not obtained on the day of parturition; therefore, it is difficult to make a direct comparison with the study of Pinotti et al. (2003).

There was no effect of supplementing the experimental cows with RPC on plasma BHBA concentration, suggesting that choline supplementation did not affect hepatic ketogenesis. However, supplemental vitamin E decreased plasma BHBA concentrations. The lack of an effect of RPC supplementation on plasma BHBA concentration is in agreement with observations elsewhere, in which choline was either fed or infused to dairy cows (Piepenbrink and Overton 2003; Pinotti et al. 2003; Guretzky et al. 2006; Cooke et al. 2007; Davidson et al. 2008). However, one study reported a tendency for decreased plasma concentrations of BHBA when RPC was supplemented from 14 days prepartum to 30 days postpartum (Pinotti et al. 2004).

The concentrations of urea-N in blood were not affected ($P = 0.28$) by the supplementation of either RPC or vitamin E. For choline, this result is consistent with those obtained by other researchers (Zahra et al. 2006; Davidson et al. 2008). Blood
urea level may reflect liver function, because fat infiltration impairs the ability of the liver to detoxify ammonia to urea, and thus it lowers serum urea concentration (Strang et al. 1998), and ammonia decreases the ability of the liver to convert propionate to glucose (Overton et al. 1999), thus potentially linking fat accumulation to impaired gluconeogenesis in the liver (Drackley et al. 2001).

Administration of RPC or vitamin E did not decrease total bilirubin ($P = 0.55$). Bilirubin is a breakdown product of heme catabolism, and is bound to albumin to be sent to the liver for excretion. Generally, increased concentrations of bilirubin in plasma of cows indicate a decrease in bile flow (Furll et al. 1993).

In this study, there was a tendency ($P = 0.07$) to increase the concentration of plasma total protein in the group supplemented with vitamin E compared with the other groups. Total protein concentration in fresh cows <6.0 g/dL is indicative of disease risk (J. Ferguson, pers. comm.).

Some researchers have reported that blood metabolites such as glucose, total protein, albumin, globulin and urea-N concentrations and the activities of AST and ALT in cows and goats were not affected by choline supplementation (Bindel et al. 2005; Zahra et al. 2006; D’Ambrosio et al. 2007; Toghdory et al. 2007; Mohsen et al. 2011).

In early-lactation cows, due to triglyceride infiltration into hepatocytes, increased levels of some plasma enzymes including GGT, AST, ALT and ALP are indicative of disease and thus it lowers serum GGT concentration (Kida 2002). In the present study, the activities of all tested enzymes for the choline and vitamin E groups were indicated an improvement in liver function. According to other studies, the concentrations and activities of AST, ALT and GGT in cows and goats were not affected by choline supplementation (Bindel et al. 2005; Zahra et al. 2006; Toghdory et al. 2007).

**Conclusion**

From the results of the present study, it can be concluded that vitamin E supplementation to early-lactation dairy cows may improve liver function.

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**References**


