The Effect of Glucogenic and Lipogenic Diets on Blood Metabolites of Baloochi Sheep

Alireza Vakili, Ali Mortezaeae and Mohsen Danesh Mesgaran

**Abstract**—The aim of present study was to assess the effect of glucogenic (G) and lipogenic (L) diets on blood metabolites in Baloochi lambs. Three rumen cannulated Baloochi sheep were used as a 3×3 Latin square design with 3 periods (28 days). Experimental diets were a glucogenic, a lipogenic and a mixture of G and L diets (50:50). The animals were fed diets consisted of 50% chopped alfalfa hay and 50% concentrate. Diets were fed once daily ad libitum. Blood samples were taken from jugular vein before the feeding, 2, 4 and 6 hour post feeding at day 27. Results indicated that β-hydroxybutyrate (BHB), glucose, insulin and aspartate aminotransferase (AST) were not affected by treatments (P > 0.05). However, lipogenic diet increased significantly activity of Alanine aminotransferase (ALT) and concentration of non-esterified fatty acid (NEFA) in blood plasma (P < 0.05)

**Keywords**—glucogenic, lipogenic, blood metabolites

I. INTRODUCTION

**INTENSE** genetic selection, improved dairy nutrition and herd management have significantly increased milk yield of dairy cows and ewes in the past decades. However, selection on high genetic merit for milk yield is only partially compensated by an increase in feed intake resulting in an ongoing increase in negative energy balance (NEB) for dairy ruminant in early lactation [6], [21]. Negative energy balance has been associated with an increase in incidence and severity of metabolic disorders, like fatty liver, ketosis [4], [11], an increase in incidence in infectious diseases [7] and ruminal acidosis [5]. Several nutritional strategies to reduce the incidence of metabolic disorders have been studied. Most studies aimed at improving the energy balance (EB) by increasing energy intake [8], [13]. A common approach is increasing the energy density of the diet by e.g. decreasing the forage to concentrate ratio [2] or by dietary supplementation of energy dense ingredients like fat [3] or non-fiber carbohydrates [14], [18]. However, increasing the dietary energy density entails a risk of compromising dry matter intake. On the other hand, decreasing the lipogenic to glucogenic nutrient ratio has been suggested to improve the EB in early lactation [20].

Lipogenic nutrients in ruminants originate from fermentation of fiber to acetate and butyrate, dietary fat or are derived from body reserves. Glucogenic nutrients originate from starch that has escaped rumen degradation or gluconeogenesis. Glucogenic feed may reduce the severity of ketosis and fatty liver, but increased incidence of (sub)clinical acidosis. Lipogenic nutrients decrease glucose and increase non-esterified fatty acid (NEFA) and β-hydroxybutyrate (BHB) plasma levels. The current study was conducted to assess the effects glucogenic or lipogenic diets on plasma glucose, insulin, NEFA, BHB concentration and activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) of Baloochi sheep.

II. MATERIALS AND METHOD

Three rumen cannulated Baloochi sheep were used as a 3×3 Latin square design with 3 periods and (28 days). The ingredients and chemical composition of concentrates are presented in Table I. Diets consisted of 50% chopped alfalfa hay and 50% concentrate and were fed once daily ad libitum. The animals were assigned to individual metabolically cages (0.5 × 1.2 × 1 m) and had free access to salt and fresh water throughout the experiment. Each period included 21 days of adaptation and 7 days of sample collection. On day 27, blood samples were taken from jugular vein before the feeding, 2, 4 and 6 hours post feeding with heparinized syringe. Plasma was obtained by centrifuge (15 min at 3500× g) and frozen at −20°C until analysis. Samples were analysed for glucose, alkaline phosphatase (ALT) and aspartate aminotransferase (AST); while concentration of NEFA, BHB and insulin were determined at pre feeding and 4 hours post feeding samples. Analyses of mentioned blood metabolites (except insulin) were performed using commercially kits on an auto-analyzer TARGA 3000, Italy (Glucose, ALT, AST, Biosystem Ltd., Spain; NEFA: FA 115 kit, Randox Laboratories Ltd., Crumlin, UK; BHB: Ranbut kit, Randox Laboratories Ltd). Insulin concentration was determined using an RIA kit (Coat-a-Count Insulin, Diagnostic Products Corporation, Los Angeles, CA). Data were applied to the mixed model of SAS (version 9.1; SAS Institute Inc., Cary, NC) with the following statistical model of: $Y_{ijklm} = \mu + A_i + B_j + C_k + D_l + (AD)_{il} + e_{ijklm}$; where $Y_{ijklm}$ was the depndent variable, $\mu$ was the overall mean, $A_i$ was the treatment effect, $B_j$ was the period effect, $C_k$ was the random effect of animal within treatments, $D_l$ was the sampling time effect, (AD) was the interaction effect of treatment and sampling time and $e_{ijklm}$ was the residual error. The sampling time was included in the model as repeated measurement by using compound symmetry. Differences between least squares means were considered significant at (p < 0.05), using PDIFF in the LSMEANS statement.

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TABLE I  
INGREDIENT AND CHEMICAL COMPOSITION (%) OF GLUCOGENIC, LIPOGENIC CONCENTRATES

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Lipogenic (L:50:50 G)</th>
<th>Glucogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn</td>
<td>-</td>
<td>23.8</td>
</tr>
<tr>
<td>Barely</td>
<td>-</td>
<td>20.2</td>
</tr>
<tr>
<td>Canola meal</td>
<td>6.7</td>
<td>13.8</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>24</td>
<td>13.8</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>10.2</td>
<td>-</td>
</tr>
<tr>
<td>Wheat pulp</td>
<td>29.2</td>
<td>-</td>
</tr>
<tr>
<td>Sunflower meal</td>
<td>20.4</td>
<td>-</td>
</tr>
<tr>
<td>Bergafat T-300(^1)</td>
<td>8.5</td>
<td>-</td>
</tr>
<tr>
<td>CaCO(_3)</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Mineral and vitamin premix(^2)</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Salt</td>
<td>0.2</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Fractionated palm fatty acids and as a triglyceride it contains the natural percentage of glycerol (Berg+Schmidt, Hamburg, Germany).

\(^1\)Composition of vitamin-mineral mix: Ca, 196.0 g/kg; P, 96.0 g/kg; Mg, 19.0 g/kg; Fe, 3.0 g/kg; Na, 71.0 g/kg; Cu, 0.3 g/kg; Mn, 2.0 g/kg; Zn, 3.0 g/kg; Co, 0.1 g/kg; I, 0.1 g/kg; Se, 0.01 g/kg and Vit A, 500000 IU/kg; Vit D, 100000 IU/kg; Vit E, 100 IU/kg.

Despite of higher ALT activity in plasma of sheep fed lipogenic diet compared with other diets, liver integrity and function during experiment was not affected by dietary treatments and activities of ALT and AST were in normal (healthy) reference range for all treatment [1].

III. RESULTS AND DISCUSSION

Data of plasma metabolites concentration are shown in table II. There were no differences among treatments in activity of aspartate aminotransferase (AST) or plasma BHBA concentrations (p > 0.05; Fig. 4). However, sheep fed the mixture of both diets during the experiment numerically had higher values of plasma BHBA than those fed the lipogenic or glucogenic diet (0.566 mmol l\(^{-1}\) vs. 0.442, 0.386 mmol l\(^{-1}\), respectively). These data agree with previous research that in which concentrations of BHBA in sheep was not affected when supplemental fat was fed to lactating dairy cows [12]. Plasma NEFA was greater (P < 0.05) in sheep fed the lipogenic diet compared with the other diets (Fig. 3). Generally, plasma concentrations of NEFA increased with fat supplementation [12], [17], [9], [15]. Plasma insulin and glucose concentration were greater (P < 0.05) in sheep fed the mixture of both diets than other treatments. The increase of plasma glucose and insulin concentration in sheep consumed glucogenic diet is in accordance with results of Van Knegsel et al., 2007 who indicated that starch resource has an efficacious glucogenic effect that favours the increase of gluconeogenesis, glycogenolysis, or both. An increment in insulin and glucose concentration was observed post feeding in all treatments where the highest value was related to the mix of both diets treatment. These trend curve were shown as figure 1 and 2. Considering the lipogenic effect of insulin, the low insulin concentration in sheep fed the lipogenic diet corresponds with a tendency for higher plasma NEFA concentration. An increase in NEFA concentrations for cows fed the lipogenic diet compared with the glucogenic diet corresponds with other studies that increased the dietary fat content and also found an increase in plasma NEFA concentration, as in [10], [16].

**TABLE II** BLOOD METABOLITES IN SHEEP RECEIVING GLUCOGENIC, LIPOGENIC OR BOTH DIETS

<table>
<thead>
<tr>
<th>Treatments</th>
<th>Glucose, mg/dl</th>
<th>Insulin, µIU/mL</th>
<th>ALT, U/l</th>
<th>AST, U/l</th>
<th>NEFA, mmol/L</th>
<th>BHBA, mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lipogenic (L:50:50 G)</td>
<td>70.83(^a)</td>
<td>53.30(^a)</td>
<td>16.06(^b)</td>
<td>55.55</td>
<td>0.208 (^a)</td>
<td>0.442 (^b)</td>
</tr>
<tr>
<td>Glucogenic</td>
<td>76.21(^b)</td>
<td>81.76(^b)</td>
<td>15.33(^c)</td>
<td>55.60</td>
<td>0.187 (^a)</td>
<td>0.566 (^c)</td>
</tr>
<tr>
<td>S.E.M</td>
<td>1.69</td>
<td>5.45</td>
<td>0.64</td>
<td>2.39</td>
<td>0.02</td>
<td>0.04</td>
</tr>
<tr>
<td>P-value</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.04</td>
<td>0.99</td>
<td>0.05</td>
<td>0.22</td>
</tr>
</tbody>
</table>

\(^1\)Means within a row with different superscripts differ (P<0.05).

**Fig. 1** Plasma glucose concentration in sheep fed glucogenic (G), lipogenic (L) or mix of both diets

**Fig. 2** Plasma insulin concentration in sheep fed glucogenic (G), lipogenic (L) or mix of both diets

**Fig. 3** Plasma NEFA concentration in sheep fed glucogenic (G), lipogenic (L) or mix of both diets
Present study demonstrated that feeding the mixture of G and L diets (50:50) compared with each of them have a potential to improve negative energy balance through affecting on plasma glucose. Furthermore, feeding sheep a high glucogenic nutrients diets may be resulted in a less negative NEB due to anabolic effects of a high insulin concentration.

IV. CONCLUSION

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REFERENCES