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PLASMA AMINO ACID LEVELS IN THE FAT COW SYNDROME

M. HIDIROGLOU and D.M. VEIRA

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Résumé

CONCENTRATION DES ACIDES AMINÉS PLASMATIQUES DANS LE SYNDROME DE LA VACHE GRASSE. — Le dosage d’acides aminés plasmatiques libres a été effectué chez 12 vaches atteintes du syndrome de la vache grasse (SVG) et chez 17 vaches témoins durant les six premières semaines de la lactation. Les concentrations plasmatiques de phénylalanine, d’histidine, d’acide glutamique et de lysine sont significativement supérieures chez les vaches avec SVG (P < 0,01). En contraste, les vaches avec SVG ont des concentrations de thréonine, de glutamine, d’asparagine et de citrulline plus faibles (P<0,01). Il est suggéré que les modifications rapportées dans les acides aminés plasmatiques chez les vaches avec SVG pourraient être une manifestation de l’altération métabolique du contrôle du mécanisme de la néoglucogénèse. Pour la majorité des acides aminés plasmatiques, aucune modification pouvant être associée à un moment du prélèvement n’apparaît.

The « fat cow syndrome » (FCS) is observed frequently in high producing dairy cattle which are unable to make the necessary metabolic adjustment which occurs about the time of parturition (Ford, 1958). In dairy cows that are excessively fat at calving time resulting from poor nutrition, a high incidence of metritis, mastitis, displaced abomasum and retained placenta has been observed (Emery et al., 1969). FCS is a combination of these metabolic, digestive, infectious and reproductive disorders in periparturient fat cows (Morrow, 1976; Morrow et al., 1979). Liver dysfunction in cows with FCS gives rise to very complex biochemical aspects of these diseases. Although studies on the relationship of plasma concentration of glucose, free fatty acids and triglycerides in FCS have appeared in the literature (Morrow et al., 1979; Reed et al., 1979), no data have been published on the concentration of plasma amino acids in these obese cows. Our interest in examining plasma aminograms in cows affected with FCS was stimulated by recent studies in obese humans in which examination of plasma showed some alteration in the concentration of amino acids (Holm et al., 1978). In the present experiment, the plasma concentrations of free amino acids have been investigated in normal cows and cows with FCS.
Materials and Methods

Animals

Twenty-nine dairy animals from a herd with more than 600 calvings per year were bled weekly from parturition to the sixth week of lactation. Seventeen were healthy and twelve had FCS. Clinical symptoms of cows suffering from FCS were similar to those reported by Morrow et al. (1979). These included obesity, abomasitis, mastitis, retained placenta, anorexia, progressive debilitation with markedly decreased milk production. In some of the cows from this herd, but not included in this study, which died from FCS there was diffuse fatty metamorphosis throughout each hepatic lobule (fig. 1).

The cows were fed a blended mixture of concentrate and corn silage (40:60 on a dry matter basis) with a crude protein content of 15.4%. The concentrate mix contained barley (45%), oats (25.9%), soybean meal (15.5%), molasses (8.0%), dicalcium phosphate (2.7%), limestone (1.3%), salt (1.0%), double sulphate of potassium and magnesium (0.6%) and vitamins A, D and E at $10^7$, $3 \times 10^8$ and $20 \times 10^3$ I U per tonne.

Blood samples, which were always collected at the same time of day, were collected from the jugular vein, into evacuated glass tubes containing heparin. The blood was centrifuged immediately followed by separation of the plasma.

Chemical analysis

The resulting plasma was deproteinized by the addition of 2 ml of 10% sulfosalicylic acid to 3 ml plasma. After mixing, the plasma-sulfosalicylate solution was centrifuged at 5°C for 30 min. The protein-free supernatant was stored at -20°C until analyzed. Plasma amino acid concentrations were determined on a Beckman Model 121MB amino acid analyzer with 126 data system and the 121MB instruction notes for physiological fluid analyses were followed (121MB-TB-017 April 1979).

The amino acid analysis was carried out under the following chromatographic conditions:
- resin : AA-10 (Beckman instruments),
- column size : 2.8 x 300 mm,
- resin bed height : 200 mm,
- buffer flow rate : 10.6 ml/h,
- 1st buffer : pH 2.83 (0.2N Li+) lithium citrate,
- 2nd buffer : pH 3.70 (0.2N Li+) lithium citrate,
- 3rd buffer : pH 3.75 (1.0N Li+) lithium citrate,
- column temperature : 38°C changed to 65°C 10 min into run,
- standard : 200 nmoles/ml Hamilton standard amino acid calibration mixture.

Statistical analysis

Data were analyzed statistically by the least squares method (Snedecor and Cochran, 1967).

Results and Discussion

The results (table 1) indicated some changes in free amino acid levels of the FCS cows plasma.

Fig. 1. — Lipid globules within hepatic cells of cows.
which is the major vehicle for the transport of amino acids between body tissues (Wolff and Bergman, 1972). Comparison of the concentration of individual amino acids between the two groups of cows revealed a highly significant (p < 0.01) elevation of plasma phenylalanine, histidine, glutamic acid and lysine in the FCS group; all of these amino acids are classified as glucogenic and/or ketogenic (Allen, 1977). The accumulation of the above-mentioned amino acids in the plasma of cows with FCS may indicate an interference with their utilization.

In ruminant animals, it is well known that the major site for the transport of amino acids between body tissues (Wolff and Bergman, 1972). Comparison of the concentration of individual amino acids between the two groups of cows revealed a highly significant (P < 0.01) elevation of plasma phenylalanine, histidine, glutamic acid and lysine in the FCS group; all of these amino acids are classified as glucogenic and/or ketogenic (Allen, 1977). The accumulation of the above-mentioned amino acids in the plasma of cows with FCS may indicate an interference with their utilization.

In ruminant animals, it is well known that the contribution of carbon for glucose synthesis from amino acids via the pathways of gluconeogenesis is of vital importance (Jarrett, 1968). In cows with FCS, these organs, in which marked fat infiltration occurs, are of major concern to the clinician. White et al. (1974) observed lower levels of valine, leucine and isoleucine in the plasma of humans with fatty metamorphosis of the liver. Lowering of these plasma branched-chain amino acids in plasma is an indication of liver damage.

This, as was reported by Leng (1970), depends on the nutrition and physiological condition of the ruminant animal. Insulin and glucagon play central roles in maintaining normal blood glucose concentrations. A major role of insulin through its hypoglycemic effect, is to regulate gluconeogenesis in the ruminant (Brockman, 1978). In humans, obesity is usually associated with hyperinsulinemia and it would be of interest to ascertain how this mechanism for control of gluconeogenesis could be affected in cows with FCS.

It is well known that, in the mammalian body, the main sites of gluconeogenesis are the liver and kidney (Jarrett, 1968). In cows with FCS, these organs, in which marked fat infiltration occurs, are of major concern to the clinician. White et al. (1974) observed lower levels of valine, leucine and isoleucine in the plasma of humans with fatty metamorphosis of the liver. Lowering of these plasma branched-chain amino acids in plasma is an indication of liver damage.

### Table 1. Concentration of free amino acids in plasma of healthy dairy cows and dairy cows with the fat cow syndrome

<table>
<thead>
<tr>
<th>Amino acid</th>
<th>Healthy (μmol/l)</th>
<th>FCS (μmol/l)</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Threonine</td>
<td>58.8 ± 1.76</td>
<td>51.3 ± 2.08</td>
<td>**</td>
</tr>
<tr>
<td>Valine</td>
<td>141.7 ± 4.56</td>
<td>128.2 ± 5.39</td>
<td>...</td>
</tr>
<tr>
<td>Methionine</td>
<td>16.0 ± 0.46</td>
<td>17.1 ± 0.54</td>
<td>...</td>
</tr>
<tr>
<td>Isoleucine</td>
<td>66.4 ± 2.36</td>
<td>62.5 ± 2.79</td>
<td>...</td>
</tr>
<tr>
<td>Leucine</td>
<td>91.6 ± 3.02</td>
<td>100.7 ± 3.58</td>
<td>...</td>
</tr>
<tr>
<td>Phenylalanine</td>
<td>31.4 ± 0.87</td>
<td>36.1 ± 1.03</td>
<td>**</td>
</tr>
<tr>
<td>Histidine</td>
<td>34.1 ± 0.96</td>
<td>38.9 ± 1.13</td>
<td>**</td>
</tr>
<tr>
<td>Lysine</td>
<td>62.6 ± 1.95</td>
<td>70.3 ± 2.31</td>
<td>**</td>
</tr>
<tr>
<td>Tryptophan</td>
<td>18.0 ± 0.60</td>
<td>16.0 ± 0.73</td>
<td>*</td>
</tr>
<tr>
<td>Aspartic acid</td>
<td>19.4 ± 0.56</td>
<td>18.9 ± 0.66</td>
<td>...</td>
</tr>
<tr>
<td>Serine</td>
<td>79.2 ± 2.30</td>
<td>71.8 ± 2.72</td>
<td>*</td>
</tr>
<tr>
<td>Glutamine</td>
<td>93.9 ± 6.79</td>
<td>59.3 ± 8.03</td>
<td>**</td>
</tr>
<tr>
<td>Proline</td>
<td>55.9 ± 1.23</td>
<td>59.0 ± 1.46</td>
<td>...</td>
</tr>
<tr>
<td>Alanine</td>
<td>155.9 ± 4.17</td>
<td>151.0 ± 4.93</td>
<td>...</td>
</tr>
<tr>
<td>Tyrosine</td>
<td>27.8 ± 0.91</td>
<td>28.4 ± 1.08</td>
<td>...</td>
</tr>
<tr>
<td>Arginine</td>
<td>90.3 ± 2.60</td>
<td>92.4 ± 3.08</td>
<td>...</td>
</tr>
<tr>
<td>Taurine</td>
<td>59.2 ± 2.11</td>
<td>54.3 ± 2.50</td>
<td>...</td>
</tr>
<tr>
<td>Glutamic acid</td>
<td>127.3 ± 3.41</td>
<td>141.7 ± 4.03</td>
<td>**</td>
</tr>
<tr>
<td>Ornithine</td>
<td>33.9 ± 0.85</td>
<td>33.8 ± 1.13</td>
<td>...</td>
</tr>
<tr>
<td>Asparagin</td>
<td>23.0 ± 1.22</td>
<td>17.5 ± 1.48</td>
<td>**</td>
</tr>
<tr>
<td>Glycine</td>
<td>355.8 ± 14.72</td>
<td>386.0 ± 17.36</td>
<td>...</td>
</tr>
<tr>
<td>Aminoadipic acid</td>
<td>7.7 ± 0.58</td>
<td>8.1 ± 0.90</td>
<td>...</td>
</tr>
<tr>
<td>Aminobutyric acid</td>
<td>10.9 ± 0.79</td>
<td>13.7 ± 1.04</td>
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</tr>
<tr>
<td>Cystathionine</td>
<td>0.9 ± 0.008</td>
<td>1.1 ± 0.08</td>
<td>*</td>
</tr>
<tr>
<td>1-Methylhistidine</td>
<td>1.9 ± 0.27</td>
<td>2.7 ± 0.41</td>
<td>...</td>
</tr>
<tr>
<td>2-Methylhistidine</td>
<td>3.6 ± 0.31</td>
<td>4.6 ± 0.46</td>
<td>...</td>
</tr>
<tr>
<td>Citrulline</td>
<td>43.2 ± 1.42</td>
<td>30.7 ± 1.68</td>
<td>**</td>
</tr>
</tbody>
</table>

* Least squares means with standard error. The mean of samples taken at weekly intervals for six weeks after calving.
(Morgan et al., 1977). However, in human obesity, Adibi (1968) observed elevated plasma levels of the branched-chain amino acids (valine, isoleucine, leucine), tyrosine and phenylalanine. It has been postulated that this increase could be a manifestation of peripheral ineffectiveness of insulin (Felig et al., 1969, 1974). This discrepancy needs to be resolved.

In the present experiment the plasma branched-chain amino acid patterns in FCS were not significantly different (P > 0.05) from those of healthy cows. In the FCS group certain plasma amino acids tended to be depressed, particularly threonine, glutamine and asparagine with a most striking decrease occurring in plasma citrulline. Some of these changes may result from decreased deamination of other amino acids. Although plasma free amino acid level changes in ruminants are difficult to interpret, it would appear that, in a metabolic defect such as FCS, a modification of the plasma amino acid pattern can occur.

After calving the cows were group fed a complete mixed ration on an ad libitum basis which prevented collection of individual feed intake. Clinical observations indicated that appetite was depressed in the cows exhibiting FCS. It has been suggested that the amount of food eaten is one of the factors which affect plasma amino acid levels in ruminants (Champredon and Pion, 1972; Foldager et al., 1980). In studies in which ruminants were starved for short periods an increase in plasma glycine has been observed (Brown et al., 1981; Cross et al., 1975); in the present experiment there was no difference between healthy and FCS cows. It was also reported by Cross et al. (1975) that plasma glutamic acid was reduced during a short period without food. In this study plasma glutamic acid increased in the FCS cows. These trends suggest that the differences observed between the healthy cows and those with the FCS were unlikely to have been due to severe undernutrition.

During the blood sampling period, the concentration of a number of plasma amino acids showed a significant increase from parturition to six weeks of lactation. Taurine, ornithine and cystathione increased from 52.47, 31.43, 0.67 nM/ml of plasma at parturition to 64.40, 39.69 and 1.22 nM/ml of plasma respectively. For other plasma amino acid concentrations, no clear pattern of change emerged which could be associated with time of sampling. These observations in dairy cows agree with those of Halpenny et al. (1969) who showed a very limited variation in the concentration of plasma amino acids during lactation.

Conclusions

Although this study did not directly address the clinical significance of the observed plasma free amino acids changes, the question arose as to whether the above-mentioned increased levels of some amino acids, associated with FCS, contribute to the physiopathology of FCS itself. It is suggested, that, since in FCS there is not a single biochemical parameter for the accurate evaluation of this condition, the reported changes in the plasma amino acid profile of the cows with FCS may serve as a useful guide for the diagnosis of this morbidity.

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Acknowledgements

Thanks are due to Mr. D. Tute from the Chemistry and Biology Research Institute for carrying out the analytical work and to Dr. K. Hartin for clinical assistance.

Summary

Assays of free amino acids in the plasma of 12 cows with the fat cow syndrome (FCS) and 17 healthy cows were carried out from parturition to 6 weeks of lactation. Plasma concentrations of phenylalanine, histidine, glutamic acid and lysine in the cows with FCS were significantly higher (P < 0.01) than in the healthy cows. In contrast, the cows with FCS exhibited a (P < 0.01) lower concentration of threonine, glutamine, asparagine and citrulline. It is suggested that the reported changes in the plasma amino acids of cows with FCS could be a reflection of the metabolic alteration in the control mechanism of gluconeogenesis. For the majority of plasma amino acids, no clear pattern emerged which could be associated with the time of sampling.
References


