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Captive giraffe (*Giraffa camelopardalis*) are reported to have low linolenic acid concentrations in body tissues in comparison with free-ranging individuals. However, it is not known whether this merely reflects a different diet, or whether it impairs body functions. As linseed contains significant amounts of linolenic acid, the feeding of linseed extraction chips might be a practical way of supplementation. Captive giraffe with low linolenic acid status in their blood lipids (compared to domestic ruminants) were introduced to a diet that included linseed extraction chips. Blood lipids of animals from which samples were available after the change in dietary regime (*n* = 2) showed an increase in linolenic acid content. One of the animals had a history of skin lesions resistant to treatment. The skin lesions improved markedly during the course of linseed supplementation. While long-term effects of either linolenic acid deficiency or linolenic acid supplementation in giraffe remain to be demonstrated, these results suggest that giraffe might benefit from the addition of linseed extraction chips to their diet.

Keywords

polyunsaturated fatty acids, linolenic acid, skin lesion, peracute mortality syndrome

1. Introduction

It has been reported that captive giraffe (*Giraffa camelopardalis*) have a much lower content of polyunsaturated fatty acids (PUFA) in body tissues than

The cause of this difference is the diet. Fresh grass and leaves contain higher proportions of PUFA, predominantly linolenic acid (Harfoot 1981), than dried drying forage like hay, in which PUFA content, especially that of linolenic acid, is decreased (Harfoot 1981; Gebremeskel et al. 1991). Concentrate feeds are made from grain and seed products, in which linoleic acid is the predominant PUFA fraction (Harfoot 1981). Therefore, without access to fresh forage, giraffe and other captive herbivores probably ingest food with less PUFA than their free-ranging conspecifics, and with an inversed linoleic:linolenic acid ratio.

In ruminants, ingested unsaturated fatty acids (FA) are subjected to complete or partial hydrogenation by rumen micro-organisms before they pass on into the intestinal tract, where they are digested and absorbed (Christie 1981a). In domestic ruminants, almost all of the linolenic acid and 60–95% of the linoleic acid is hydrogenated in the rumen (Doreau and Ferlay 1994). Therefore, ruminant adipose tissue contains mainly saturated FA (Christie 1981b). Ruminants rely on the small proportion of unsaturated FA that escape ruminal degradation for the provision with essential fatty acids (EFA) (Van Soest 1994).

An important sparing mechanism for the few escaped PUFA a ruminant can absorb is that they are not allocated to adipose tissue and triglycerides (TG) but are selectively incorporated into the metabolically less active, structural lipid fractions of the body, mainly phospholipids and cholesteryl esters (Christie 1981a). Among the phospholipids, the ethanolamine phosphoglycerides (EPG) contain higher proportions of PUFA than the choline phosphoglycerides (CPG) (Crawford et al. 1976; c.f. table 2). In screening ruminants for their FA status, the total plasma FA or plasma triglycerides are therefore less revealing than plasma phospholipids.

The question remains whether a low provision of PUFA, and especially linolenic acid, in captive giraffe has pathological implications, or whether it is merely the reflection of a different diet. Except for very recent work on black rhinoceros (Diceros bicornis) (Suedmeyer and Dierenfeld 1998) there are no reports of FA deficiencies in zoo ungulates, and hardly any reports of clinical signs comparable to those of domestic animals suffering from PUFA deficiency, the best-known of which is the occurrence of skin lesions (Holman 1968; Fiennes et al. 1973). Dermatoses have rarely been reported in giraffe. Mainka and Cooper (1989) reported areas of alopecia and hyperkeratosis on the shoulders, muzzle and between the horns of a giraffe suspected to suffer from hypothyroidism. Kuschnarew and Samigin (1985) reported alopecia and hyperkeratosis of mainly unpigmented areas of the legs, the ventral abdomen and the shoulder in a group of three giraffes. Diseases due to infections or physical agents were ruled out, and the authors suspected a photosensitizing effect of prolonged alfalfa feeding; the authors considered neither a zinc nor a PUFA deficiency. The only other case reported (Flach et al. 1997) concerned a female reticulated giraffe which developed alopecia and hyperkeratosis on the hind
legs and the ventral abdomen concurrently with chronic loss of condition and persistent neutrophilia.

We report here initial results from feeding linseed extraction chips to a group of three captive giraffes, including the animal with areas of alopecia described above (Flach et al. 1997). Linseed chips were chosen as a source of linolenic acid supplementation because they were part of the feeding regime of several other species at the facility and therefore readily available. We wanted to test whether the giraffe would accept the supplement, and whether any changes in blood lipid composition and in the skin lesion would occur.

2. Animals and Methods

The investigation was carried out at Whipsnade Wild Animal Park (WWAP) in 1998. The breeding group of five reticulated giraffes were fed individually with lucerne hay, two commercial concentrate pellets (“Browser Breeder”, and “Ele-Vit-E”, both from Mazuri, SDS, Essex, UK), plus a variety of fruits and vegetables, and occasional browse (usually beech, *Fagus sylvatica*). For daily intake of food items and their chemical composition see table 1. Analyses for fatty acid content of feedstuffs were not available. At the end of March 1998, linseed extraction chips (Cargill Plc, Gladstone Dock, Bootle, UK) were added to the diet of three giraffes at a daily rate of 1 kilogram per animal. All foodstuffs were weighed in and out on a daily basis. When allowed out of doors, the giraffes consumed grass in small amounts.

Plasma or serum samples, which had been collected during routine clinical examinations of five giraffes and stored frozen at -20°C, were available as presupplementation samples. Two of these animals (animals 4 and 5 in table 2) were re-sampled three months after the introduction of linseed chips. Analyses of fatty acid composition of plasma and serum samples were performed using the technique described by Williams et al. (1987). The alopecic skin lesions were monitored visually and by photography between April and August 1998. Microbiological and histological analyses of the lesions were not available.

3. Results

Every day, the entire amount of linseed chips was consumed by each animal. The results of the FA analyses are presented in table 2. The percentage of linolenic acid was increased in all lipid fractions investigated after the dietary change, while the percentage of linoleic acid did not increase. The proportions of PUFA are generally lower in the TG fraction than in the phosphoglycerides. The changes in FA profile after the dietary change are most prominent in the EPG, and least prominent in the TG fraction of plasma/serum lipids. The increase in the percentage of linolenic acid is more prominent in the younger animal.

The skin condition improved markedly during the first three months of linseed chips supplementation (figures 1 and 2). New hair growth appeared in the dry, hairless, scaly patches.
Table 1

Consumption per animal and day (in kg original substance; range of individual average intake) and chemical composition of feedstuffs; dry matter (DM) in % original substance; crude protein (CP), crude fat (CF), crude ash (CA), neutral detergent fiber (NDF), acid detergent fiber (ADF), acid detergent lignin (ADL), nitrogen-free extracts (NFE) in % of DM. (from Clauss et al., in prep.)

<table>
<thead>
<tr>
<th>Food</th>
<th>C*kg</th>
<th>DM</th>
<th>CP</th>
<th>CF</th>
<th>CA</th>
<th>NDF</th>
<th>ADF</th>
<th>ADL</th>
<th>NFE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pellet</td>
<td>3–8</td>
<td>90.18</td>
<td>19.10</td>
<td>4.52</td>
<td>8.19</td>
<td>38.24</td>
<td>20.14</td>
<td>2.69</td>
<td>48.04</td>
</tr>
<tr>
<td>Vitamin E Pellet</td>
<td>0.2</td>
<td>89.66</td>
<td>15.12</td>
<td>3.50</td>
<td>8.53</td>
<td>39.73</td>
<td>19.18</td>
<td>2.97</td>
<td>53.67</td>
</tr>
<tr>
<td>Linseed Extr. Chips</td>
<td>1.0</td>
<td>90.01</td>
<td>37.54</td>
<td>6.26</td>
<td>6.65</td>
<td>21.91</td>
<td>16.53</td>
<td>5.57</td>
<td>33.02</td>
</tr>
<tr>
<td>Lucerne stems</td>
<td>3–4</td>
<td>87.97</td>
<td>14.24</td>
<td>0.72</td>
<td>6.98</td>
<td>56.43</td>
<td>47.31</td>
<td>11.56</td>
<td>30.75</td>
</tr>
<tr>
<td>Beech browse</td>
<td>1.0</td>
<td>46.04</td>
<td>12.35</td>
<td>3.66</td>
<td>3.75</td>
<td>48.00</td>
<td>34.87</td>
<td>15.91</td>
<td>45.36</td>
</tr>
</tbody>
</table>

* Consumption

4. Discussion

The percentages of linoleic acid in plasma/serum phospholipids in the investigated giraffes were higher than reported in domestic ruminants (Christie 1981b; c.f. table 3), which might reflect a higher dietary intake in this FA via the concentrates, or could be due to differences in forestomach physiology (Meyer et al. 1998); the relatively fast passage rate of giraffes (Clauss et al. 1998) could result in more unsaturated fatty acids leaving the rumen before they are hydrogenated. In contrast, linolenic acid percentages in the plasma/serum before supplementation were below those seen in domestic ruminants, supporting the view that captive giraffe are low in this fatty acid.

Linseed supplementation did not increase the average linoleic acid proportions, but those of linolenic acid in plasma/serum phospholipids increased. The increase was more prominent in the younger animal, which received, relative to its body weight, a higher dose of linseed chips. Unfortunately, there is no reported data on blood lipids in free-ranging giraffe to use for comparison. This data should be collected in the near future.

Long-term effects of the feeding of linseed extraction chips to giraffes remain to be investigated. The improvement in the skin lesion of one giraffe coincided with the first three months of linseed supplementation, suggesting that linolenic acid deficiency may have been the cause, or a major factor. To our knowledge, there are no reports of linolenic acid-responsive skin lesions in ruminants. The improvement of the lesions occurred at the onset of summer when the giraffes spent increasing amounts of time out of doors. Therefore, the hypothesis of Kuschnarew and Samigin (1985) that lucerne causes photosensi-
Table 2

The percentages of linoleic (LE) and linolenic (LN) acid in the total fatty acid content of ethanolamine phosphoglycerides (EPG), choline phosphoglycerides (CPG) and triglycerides (TG) in giraffe plasma and serum before and after the start of linseed extraction chips supplementation in March 1998.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Sample date</th>
<th>Plasma/Serum</th>
<th>Condition</th>
<th>EPG LE</th>
<th>EPG LN</th>
<th>CPG LE</th>
<th>CPG LN</th>
<th>TG LE</th>
<th>TG LN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>male</td>
<td>adult</td>
<td>25.01.96</td>
<td>s</td>
<td>clinically healthy</td>
<td>64.13</td>
<td>1.60</td>
<td>36.88</td>
<td>1.08</td>
<td>7.36</td>
<td>0.85</td>
</tr>
<tr>
<td>2a</td>
<td>male</td>
<td>1</td>
<td>21.11.95</td>
<td>s</td>
<td>clinically healthy</td>
<td>59.47</td>
<td>1.60</td>
<td>33.59</td>
<td>1.05</td>
<td>5.69</td>
<td>0.40</td>
</tr>
<tr>
<td>2b</td>
<td>male</td>
<td>1.5</td>
<td>24.03.96</td>
<td>p</td>
<td>just prior to death</td>
<td>53.89</td>
<td>1.69</td>
<td>32.92</td>
<td>1.17</td>
<td>6.52</td>
<td>0.74</td>
</tr>
<tr>
<td>3</td>
<td>male</td>
<td>1</td>
<td>26.02.98</td>
<td>s</td>
<td>euthanized</td>
<td>61.72</td>
<td>2.52</td>
<td>31.63</td>
<td>1.38</td>
<td>7.63</td>
<td>0.74</td>
</tr>
<tr>
<td>4a</td>
<td>female</td>
<td>7</td>
<td>03.09.93</td>
<td>p</td>
<td>onset of weakening</td>
<td>41.46</td>
<td>1.13</td>
<td>33.58</td>
<td>1.25</td>
<td>6.00</td>
<td>0.71</td>
</tr>
<tr>
<td>5a</td>
<td>female</td>
<td>2</td>
<td>17.03.98</td>
<td>p</td>
<td>clinically healthy</td>
<td>53.66</td>
<td>1.79</td>
<td>31.81</td>
<td>1.30</td>
<td>5.79</td>
<td>0.70</td>
</tr>
<tr>
<td>mean (n=6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>55.72</td>
<td>1.72</td>
<td>33.40</td>
<td>1.21</td>
<td>6.50</td>
<td>0.69</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8.15</td>
<td>0.45</td>
<td>1.90</td>
<td>0.13</td>
<td>0.83</td>
<td>0.15</td>
</tr>
</tbody>
</table>

after linseed supplementation

| 4b     | female | 12        | 06.06.98    | p             | clinically healthy, skin condition | 53.88  | 3.16   | 32.95  | 1.66   | 5.31  | 0.76  |
| 5b     | female | 2         | 17.06.98    | p             | clinically healthy                 | 55.67  | 3.46   | 33.18  | 2.45   | 5.99  | 1.16  |
| mean (n=2) |     |           |             |               |             | 54.78  | 3.31   | 33.07  | 2.06   | 5.65  | 0.96  |
| SD     |                |             |               |               |             | 1.27   | 0.21   | 0.16   | 0.56   | 0.48  | 0.28  |
tization seems doubtful in this case. A general improvement due to seasonal influences, on the other hand, cannot be excluded.

**Table 3**
The percentages of linoleic (LE) and linolenic (LN) acid in the total fatty acid content of phospholipids (PL) and triglycerides (TG) in the plasma of domestic ruminants (from Christie 1981b).

<table>
<thead>
<tr>
<th>Animal species</th>
<th>PL LE</th>
<th>PL LN</th>
<th>TG LE</th>
<th>TG LN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>10.7</td>
<td>2.0</td>
<td>1.7</td>
<td>1.0</td>
</tr>
<tr>
<td>Sheep</td>
<td>15.8</td>
<td>2.5</td>
<td>4.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Goat</td>
<td>14.6</td>
<td>3.9</td>
<td>3.2</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Zinc (Zn) deficiency can cause typical skin lesions in ruminant animals (Puls 1994). Zinc content was not determined in the course of this investigation. Although a diet of solely lucerne hay, which contains about 25–33 mg Zn/kg DM (NRC 1989), would not meet reported Zn requirements of domestic ruminants of 40–50 mg/kg DM (Kamphues et al. 1999), a diet that consists of large proportions of a complete feed like the commercial pellet used for these giraffe (with a Zn content of 44 mg/kg DM claimed by the manufacturer) seems unlikely to cause Zn deficiency.

Linseed chips are palatable and high in protein and fat (see table 1). Therefore they are energy-dense, but will not induce rumen acidosis because their energy density is not due to soluble carbohydrate content (c.f. NfE-values of different feedstuffs in table 1). However, due to the presence of cyanogenic compounds and linatin, linseed products should not be fed ad libitum (Kamphues et al. 1999). The ration of 1 kg per animal was based on Benbow’s recommendation (1988) for the supplementation of “thin giraffes”. Kamphues et al. (1999) recommend that no more than 1 kg of linseed extraction chips be fed to beef cattle per animal per day, and no more than 2 kg for dairy cows. As with all dietary changes, the new supplement should be introduced gradually, and it should be offered individually to avoid over-feeding of a dominant animal. In this case, the giraffes accepted the linseed chips readily and generally consumed them before turning to other diet items. As PUFA in body tissues are especially susceptible to oxidation, a vitamin E supplement should be fed together with the linseed chips, as was done routinely at WWAP.

In captive giraffe, there is a high incidence of the so-called peracute mortality syndrome, a condition in which the animals are found to have widespread serous fat atrophy at necropsy (Fowler 1978; Junge and Bradley 1993). The additional provision of an energy-dense food, like linseed extraction chips, could counteract tendencies to mobilise body fat stores. Additionally, a deficiency in EFA, and especially in linolenic acid, has been reported to increase metabolic rate, diminish fat storage capacity, and reduce both insulin secretion...
and resposisveness in certain animals (Holman 1968; Vernon 1992; Chillard 1993; Ashes et al. 1995). This theory might also be applicable to giraffes.

5. Conclusions

1. Data from the literature suggest that captive giraffes, unless especially supplemented or fed large amounts of browse, have a low supply of linolenic acid.

2. Whether this deficiency is a cause of clinical problems remains to be demonstrated.

3. It has been observed that supplementing the diet of two captive giraffes with linseed extraction chips increased the linolenic acid percentage of plasma phospholipids.

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References


J. Nijboer, J.-M. Hatt, W. Kaumanns
A. Beijnen, U. Gansloßer (eds.)

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