Due to the incorporation of niacin into the coenzymes NAD and NADP, niacin is of great importance for the metabolism of man and animals. Apart from niacin in feed and endogenous formation, microbial niacin synthesis in the rumen is an important source for dairy cows. But the amount synthesised seems to differ greatly, which might be influenced by the ration fed. Many studies revealed a positive impact of a niacin supplementation on rumen protozoa, but microbial protein synthesis or volatile fatty acid production in the rumen showed inconsistent reactions to supplemental niacin. The amount of niacin reaching the duodenum is usually higher when niacin is fed. However, not the whole quantity supplemented reaches the duodenum, indicating degradation or absorption before the duodenal cannula. Furthermore, supplementation of niacin did not always lead to a higher niacin concentration in blood. Effects on other blood parameters have been inconsistent, but might be more obvious when cows are in a tense metabolic situation, for example, ketosis or if high amounts are infused post-ruminally, since ruminal degradation appears to be substantial. The same is valid for milk parameters. In the few studies where blood niacin and milk parameters have been investigated, enhanced niacin concentrations in blood did not necessarily affect milk production or composition. These results are discussed in the present review, gaps of knowledge of niacin’s mode of action on the metabolism of dairy cows are identified and directions for future research are suggested.

Niacin for dairy cattle: a review

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Niacin is of great importance in the metabolism due to its incorporation into the coenzymes NAD and NADP. Both forms of niacin, nicotinic acid (NA) and nicotinamide (NAM), can be converted into the coenzymes, although they contain only NAM as a reactive component. Apart from feed as a source of niacin, nearly all species are able to synthesise the vitamin from tryptophan and quinolinate. Since micro-organisms are able to produce niacin as well, ruminants have an additional supply due to their rumen microbes. Ruminal synthesis of niacin was estimated to be 1804 mg/d for a 650 kg cow producing 35 kg of 4% fat-corrected milk. This seems to cover the requirement definitely, which was assumed to be 256 mg/d for tissues and 33 mg/d for milk production, thus 289 mg/d in total. Therefore, it was concluded that a general supplementation could not be advised. But tissue requirements are estimated based on data from lactating sows and have not been experimentally determined. Furthermore, synthesis might vary, for example, when different feeding regimens are applied. Indeed, numerous studies showed positive responses to a niacin supplementation. On the other hand, a lot of research has been done where administration of niacin did not have any effect. Therefore current literature is reviewed here to distinguish the vitamin’s impact on cow performance and metabolism. The aim of the present review is to present the state of knowledge on niacin synthesis in the rumen and the amount of niacin arriving at the duodenum, niacin’s mode of action on ruminal and several blood parameters as well as its influence on milk production and composition. Where possible, conclusions are drawn from experiments and gaps of knowledge are identified. Cognition of these processes would facilitate a decision on necessity and time of a niacin supplementation.

To our knowledge, the last detailed review available on niacin (NA and NAM) in dairy cow nutrition was done in 1993. Therefore in the present review studies newer than 1990 are used to show developments. But in some cases (rumen, duodenum), older literature was included as a comparison with few new results available. Only significant effects (P<0.05) and tendencies (P<0.10) are mentioned, unless otherwise noted. In all studies, supplemental niacin was not rumen-protected.

Abbreviations: BHBA, β-hydroxybutyrate; F:C ratio, forage:concentrate ratio; NA, nicotinic acid; NAM, nicotinamide; NFC, non-fibre carbohydrate; VFA, volatile fatty acid.

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Rumen

Niacin in the rumen

In Table 1, niacin concentrations in ruminal contents from several studies are summarised. In interpretation of the results, it has to be kept in mind that different analytical methods for niacin determination exist (for example, colorimetric, microbiological and HPLC methods)\(^{(10)}\). This could lead to different results as was proven for cereal-based foods analysed by microbiological and HPLC methods\(^{(10)}\).

Niacin concentration in the rumen was enhanced if pure NA or NAM was supplemented\(^{(11,12)}\), while the highest intake via feed components did not necessarily force the highest concentration in the rumen\(^{(8,12)}\). Santschi et al.\(^{(18)}\) found no difference in total niacin content in the rumen when comparing rations with a forage:concentrate ratio (F:C ratio) of 60:40 or 40:60. However, they noticed an effect on the concentrations of each vitamer. Although no NAM was present in the feed, it was found in the rumen. Furthermore, NAM was significantly increased with the low-forage ration. NA decreased numerically and hence total niacin content was not affected. Earlier work showed an effect of the F:C ratio on ruminal niacin concentrations, which was highest in the all-concentrate ration\(^{(14)}\) (data not shown). Thus, there is evidence that ruminal niacin concentrations and/or the concentrations of each vitamer are influenced by niacin supplementation and the F:C ratio.

Some studies have been conducted to measure the ruminal synthesis of niacin. Micro-organisms use aspartate and dihydroxyacetone phosphate for niacin production\(^{(4)}\). It is extremely difficult to measure real synthesis; therefore apparent synthesis is calculated by subtracting the intake from the amount reaching the duodenum. Some data are given in Table 2. It can be assumed that there is an influence of type of feed. Zinn et al.\(^{(15)}\) mentioned a stimulating effect of starch on the ruminal synthesis of all B vitamins. Schwab et al.\(^{(16)}\) found a significant effect of the non-fibre carbohydrate (NFC) content of feed on niacin synthesis, while the F:C ratio had no effect. But the effect of NFC might also reflect large differences in niacin intake (Table 2). In the above-mentioned studies where an effect of the F:C ratio on ruminal niacin concentrations was found\(^{(8,14)}\), duodenal niacin flow was not measured, therefore it was not possible to calculate apparent synthesis to compare these values.

In all studies listed in Table 2, the ration with the highest niacin content within a study resulted in the lowest apparent niacin synthesis. It was stated that there seems to be an optimal concentration. Synthesis will occur below this level and above it, excess niacin is degraded by the bacteria\(^{(17)}\). This might be the reason why in two studies with cows and feedlot calves where 6 or 2 g NA/d were supplemented\(^{(11,15)}\), only 2 and 20 %, respectively, of the amount added reached the duodenum. Santschi et al.\(^{(18)}\) reported a ruminal disappearance rate for niacin of 98.5 % as well. The fate of niacin that disappeared from the rumen is not clear. Zinn et al.\(^{(15)}\) suggested either degradation or absorption. It is not completely clarified if absorption of vitamins could take place in the rumen. Erickson et al.\(^{(19)}\) found free NAM to be absorbed at 0.98 g/h from a dilution in a washed rumen of cows. NA was not absorbed, because it is ionised under a physiological pH. But usually, most of the niacin is bound in the bacterial fraction\(^{(18,19,26)}\). Therefore, under normal circumstances, no absorption should take place from the rumen\(^{(18)}\). Yet it has to be kept in mind that with niacin supplementation, a high amount of usually free niacin reaches the rumen. Thus, some absorption might occur. However, in the work of Campbell et al.\(^{(12)}\), supplementation of NAM gave significantly higher duodenal values of niacin than NA. If only NAM is absorbed from the rumen at normal ruminal pH values\(^{(19)}\), the opposite would be expected. Consequently, ruminal degradation might be the reason for the high disappearance rate of supplemented niacin from the rumen. Another possible explanation could be that niacin is absorbed in the proximal duodenum, before the duodenal cannula. In man, niacin is absorbable from the stomach as well\(^{(21)}\). To our knowledge, no studies concerning absorption from the abomasum are available.

In summary, niacin concentrations and apparent synthesis in the rumen are affected by niacin supplementation and the ration fed. But it is not known which feed component most influences niacin in the rumen. If niacin is supplemented, only a small part reaches the duodenum. Ruminal absorption might occur, but does not seem to make a large contribution. Ruminal degradation or absorption in the abomasum or before the duodenal cannula seems more likely.

Effect of niacin on rumen metabolism

In contrast to ruminal bacteria it is assumed that protozoa are not able to synthesise niacin and need to cover their requirements from feed or bacterial synthesis\(^{(22)}\). Doreau & Ottou\(^{(22)}\) observed no effect of 6 g NA on bacteria, but an increase of protozoa\(^{(22)}\). This especially concerned *Opbrysoscolecidae*, but *Isotrichidae* were not affected. Increasing protozoal numbers, especially *Entodinia* (family *Opbrysoscolecidae*), may increase bacterial numbers as well, because *Entodinia* are able to regulate the ruminal environment by consuming starch\(^{(19)}\).

Others also found a significant increase in total protozoa in the rumen fluid due to niacin feeding\(^{(23–25)}\), which was once primarily attributable to increases in numbers of *Entodinia*\(^{(25)}\). Therefore, an effect of niacin on the microbial population is likely, but might be mainly on protozoa.

As a result of this probable effect of niacin on microbial population, ruminal N metabolism could also be affected. A stimulating effect of niacin on microbial protein synthesis has been observed *in vitro*\(^{(26)}\) and *in vivo*\(^{(23,24)}\). In contrast, in some *in vivo* studies no influence was seen on microbial protein production, either on the total amount or on the efficiency\(^{(12,15)}\).

Whereas some *in vivo* trials\(^{(22,27,28)}\) showed no niacin effect on ammonia concentration in the rumen, other *in vitro*\(^{(26)}\) and *in vivo*\(^{(23,24)}\) experiments showed a decreasing effect of niacin on rumen NH\(_3\)-N. An interaction of fat and niacin towards increasing ammonia concentrations in the high-fat, and decreasing values in the low-fat, diet after niacin feeding was also found *in vivo*\(^{(27)}\). It is known that ammonia fixation of the rumen bacteria and fungi occurs largely via NADP- or NAD-linked glutamic dehydrogenase, and possible assimilation of ammonia via NAD”-dependent glutamic dehydrogenase was also shown for protozoa\(^{(29)}\). This might be favoured by a niacin supplementation.

The fermentation pattern of carbohydrates might also be altered due to a possible niacin effect on the microbial
<table>
<thead>
<tr>
<th>Reference</th>
<th>Feeding ration</th>
<th>Niacin content of feed (mg/kg DM)</th>
<th>Niacin intake (mg/d)</th>
<th>Niacin concentration in the rumen</th>
<th>Vitamer</th>
<th>Studied fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Riddell et al. (1985)(11)</td>
<td>55 % wheat straw, 45 % concentrate (corn starch, dextrose, soyabean meal)</td>
<td>Without niacin: 6 mg/kg DM, 6060 mg/d</td>
<td>102–114 mg/kg DM**</td>
<td>NA and NAM†</td>
<td>Whole rumen content</td>
<td></td>
</tr>
<tr>
<td></td>
<td>With 6 g NA:</td>
<td>697 mg/kg DM, 6060 mg/d</td>
<td>119–155 mg/kg DM**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdouli &amp; Schaefer (1986)(13)</td>
<td>27 % lucerne hay; 73 % barley</td>
<td>Without niacin: 64 mg/kg DM, 868 mg/d</td>
<td>0·48 mg/l fluid + 2·32 mg NAD/l†</td>
<td>NA and NAM†</td>
<td>Rumen fluid</td>
<td></td>
</tr>
<tr>
<td></td>
<td>29 % lucerne hay; 71 % oats</td>
<td>19 mg/kg DM, 166 mg/d</td>
<td>0·32 mg/l fluid + 1·51 mg NAD/l†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Campbell et al. (1994)(12)</td>
<td>60 % forage (lucerne haylage, maize silage), 40 % concentrate (corn, soyabean hulls and meal)</td>
<td>Without niacin: n.d., –, 0 mg/l fluid</td>
<td>NA</td>
<td>NAD</td>
<td>Rumen fluid</td>
<td></td>
</tr>
<tr>
<td></td>
<td>With 12 g NA:</td>
<td>+ 12 000 NA, 14 mg/l fluid</td>
<td>NA</td>
<td>NAM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>With 12 g NAM:</td>
<td>+ 12 000 NAM, 14 mg/l fluid</td>
<td>NA</td>
<td>NAM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>With 6 g NA and 6 g NAM:</td>
<td>+ 6000 NA, 12 mg/l fluid</td>
<td>NA</td>
<td>NAM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Santschi et al. (2005)(8)</td>
<td>60 % forage (mixed silage, maize silage), 40 % concentrate (corn, soyabean meal)</td>
<td>26 mg/kg DM, 520 mg/l fluid</td>
<td>143 mg/kg DM</td>
<td>NA</td>
<td>Solid-associated bacteria</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 % forage (mixed silage, maize silage), 60 % concentrate (corn, soyabean meal)</td>
<td>23 mg/kg DM, 453 mg/l fluid</td>
<td>137 mg/kg DM</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>60 % forage (mixed silage, maize silage), 60 % concentrate (corn, soyabean meal)</td>
<td>26 mg/kg DM, 520 mg/l fluid</td>
<td>173 mg/kg DM</td>
<td>NA</td>
<td>Liquid-associated bacteria</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 % forage (mixed silage, maize silage), 60 % concentrate (corn, soyabean meal)</td>
<td>23 mg/kg DM, 453 mg/l fluid</td>
<td>161 mg/kg DM</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>60 % forage (mixed silage, maize silage), 40 % concentrate (corn, soyabean meal)</td>
<td>26 mg/kg DM, 520 mg/l fluid</td>
<td>0·08 mg/l fluid</td>
<td>NA</td>
<td>Particle-free fluid</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 % forage (mixed silage, maize silage), 60 % concentrate (corn, soyabean meal)</td>
<td>23 mg/kg DM, 453 mg/l fluid</td>
<td>0·09 mg/l fluid</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NA, nicotinic acid; NAM, nicotinamide; n.d., not determined.

** Values with unlike superscript letters within a study were significantly different (P < 0.05).

* Depending on different sampling times after feeding (0 to 8 h), means were significantly different at 4 and 6 h after feeding.

† The vitamin content was determined via microbiological assay, where it is not possible to distinguish between the vitamers.
population, resulting in a change in volatile fatty acid (VFA) production in the rumen. Results for in vivo experiments are presented in Table 3. Butyrate was the VFA which was mostly but inconsistently affected, but there were also influences on acetic and propionic acid; in some surveys, no effect was seen at all. The effect of niacin on butyrate might be induced by the effect on rumen protozoa, since the presence of some protozoa species led to more butyrate production\(^{30}\). This would match with the work of Doreau & Ottou\(^{22}\), who observed higher protozoal counts and an increase in the molar proportion of butyrate. But it is contrary to Samanta et al.\(^{24}\), who observed higher protozoal counts and a decrease in the molar proportion of butyrate. Thus, the effect of niacin on protozoa might not be the main reason for its effect on VFA.

In total, the responses of ruminal parameters to niacin feeding vary greatly. Ottou & Doreau\(^{31}\) concluded that response differences could be due to the level of niacin supplementation, but this was not obvious here, since niacin concentrations varied in an equal range in all studies.

### Table 2. Apparent synthesis of niacin in the rumen of cattle and flow at the duodenum

<table>
<thead>
<tr>
<th>Reference</th>
<th>Feeding ration</th>
<th>Niacin supplement (g/d)</th>
<th>Niacin intake with feed (mg/d)</th>
<th>DM intake (kg/d)</th>
<th>Duodenal flow (mg/d)</th>
<th>Apparent synthesis (mg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Riddell et al. (1985)(^{11})(^{<em>,</em>})</td>
<td>55% forage ( wheat straw), 45% concentrate (corn starch, dextrose, soyabean meal)</td>
<td>6 NA</td>
<td>6 - 7</td>
<td>5.7</td>
<td>50</td>
<td>5.7</td>
</tr>
<tr>
<td>Miller et al. (1986)(^{12})(^{<em>,</em>})</td>
<td>12% lucerne meal, 88% maize grain, urea</td>
<td>0</td>
<td>0.7</td>
<td>60.6</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Zinn et al. (1987)(^{13})(^{<em>,</em>})</td>
<td>55% concentrates (corn, molasses, fat)</td>
<td>0</td>
<td>4.1</td>
<td>3.8</td>
<td>314</td>
<td>314</td>
</tr>
<tr>
<td>Riddell et al. (1994)(^{12,12})</td>
<td>45% forage (lucerne hay, Sudan grass), 55% concentrates (corn, soyabean hulls and meal)</td>
<td>12 NA</td>
<td>19.9</td>
<td>19.9</td>
<td>&gt;12 000 NA</td>
<td>&gt;12 000 NA</td>
</tr>
<tr>
<td>Santschi et al. (2005)(^{14})</td>
<td>58% forage (grass-legume silage, maize silage), 42% concentrate (corn, soyabean meal, protein supplement)</td>
<td>0</td>
<td>19.8</td>
<td>21.3</td>
<td>465</td>
<td>465</td>
</tr>
<tr>
<td>Schwab et al. (2006)(^{15})</td>
<td>35% forage (corn silage, Lucerne and grass hay), 65% concentrate (soyabeans hulls and meal, beet pulp), total 30% NFC</td>
<td>0</td>
<td>22.2</td>
<td>21.3</td>
<td>1399 NAM(\dagger\dagger)</td>
<td>1399 NAM(\dagger\dagger)</td>
</tr>
<tr>
<td>Schwab et al. (2006)(^{15})</td>
<td>35% forage (corn silage, Lucerne and grass hay), 65% concentrate (corn, barley, soyabeans hulls and meal, beet pulp), total 40% NFC</td>
<td>0</td>
<td>22.2</td>
<td>21.3</td>
<td>469 NAM(\dagger\dagger)</td>
<td>469 NAM(\dagger\dagger)</td>
</tr>
<tr>
<td>Schwab et al. (2006)(^{15})</td>
<td>60% forage (corn silage, Lucerne and grass hay), 40% concentrate (soyabeans hulls and meal, beet pulp, blood meal, fat), total 30% NFC</td>
<td>0</td>
<td>18.1</td>
<td>21.3</td>
<td>727 NAM(\dagger\dagger)</td>
<td>727 NAM(\dagger\dagger)</td>
</tr>
<tr>
<td>Schwab et al. (2006)(^{15})</td>
<td>60% forage (corn silage, Lucerne and grass hay), 40% concentrate (corn, barley, soyabeans hulls and meal, beet pulp, blood meal, fat), total 40% NFC</td>
<td>0</td>
<td>19.8</td>
<td>21.3</td>
<td>363 NAM(\dagger\dagger)</td>
<td>363 NAM(\dagger\dagger)</td>
</tr>
</tbody>
</table>

NA, nicotinic acid; NAM, nicotinamide; n.d., not determined; NFC, non-fibre carbohydrates.

\* Significant differences (P<0.05) between control and niacin groups. In the paper of Santschi et al.\(^{14}\), the level of significance was not declared; furthermore, Zinn et al.\(^{12}\) and Miller et al.\(^{12}\) did not calculate the apparent synthesis. Therefore it was not possible to characterise significances in these studies.

\†† Apparent synthesis = duodenal flow – intake.

\‡‡ In these studies, apparent ruminal synthesis was not calculated by the authors, but daily intake and duodenal flows were given, therefore apparent synthesis was calculated by us.

\# The vitamin content was determined via microbiological assay, where it is not possible to distinguish between the vitamers.

\‡ In these studies, the vitamer applied was not named. It was just stated that niacin was supplemented. But since the term niacin is occasionally also used as a synonym for NA\(^{60}\), it is assumed that NA was fed in this survey.

\‡‡‡ Significant differences between control v. niacin and NA v. NAM (P<0.05).

\§§ Significant effects of forage (P<0.05).

\††† Significant effects of NFC (P<0.05).
Duodenum

The amount of niacin reaching the duodenum varies less than does the concentration in the rumen. Duodenal flow values for niacin are given in Table 2. From these data it can be concluded that a niacin supplementation led to higher niacin values reaching the duodenum, but to a lower extent. This indicates abomasal or duodenal absorption before the duodenal cannula. Niacin flow at the duodenum was higher than daily niacin intake after post-ruminal niacin supplementation, even if the total amount given did not reach the duodenum. This was not the case when niacin was added to the ration in cows. Therefore, it is likely that an oral niacin supplementation is highly degraded in the rumen and might also suppress niacin synthesis. A higher amount seems to reach the duodenum when it is infused post-ruminally.

The type of feed might modify the amount of niacin reaching the duodenum. Schwab et al. found an effect of the F:C ratio. The high-forage ration decreased NAM content in duodenal fluid significantly, and tended to decrease NAM content. The NFC content had no effect. Apparent synthesis of niacin in the rumen was affected by NFC, but not by the F:C ratio. This further indicates that the NFC effect on apparent synthesis might be due to different niacin intake, and that the F:C ratio could be important. But more information is lacking.

Even if given post-ruminally, NAM seems to convert to NA. After NFC supplementation only the amount of NA was enhanced at the duodenum, while NAM was even lower than in the control group. The authors concluded that this was due to the acidic environment in the abomasum which may transform NAM to NA. Additionally, supplementation of NFC in feed enhanced the amount of niacin arriving at the duodenum to a higher extent than did NA.

Apparent absorption of niacin in the duodenum was not influenced by the type of feed and accounted for 67%, 79% and 84% (73% of the NA and 94% of the NAM) of the amount reaching the duodenum. When supplemental niacin was fed, Riddell et al. observed a higher amount of niacin reaching the duodenum, but excretion with faeces was equal. Therefore, the authors concluded that absorption in the duodenum must have been higher in the supplemented group. But no measurements were taken in the large intestine, thus results could also be due to a higher degradation or absorption in the large intestine. In other studies, a B vitamin blend was supplemented, either in the feed or post-ruminally, but did not influence absorption in the duodenum (18).

Little knowledge is available concerning the mechanism of absorption. New research in human subjects suggests that the mechanism for NA absorptions in physiological amounts is dependent on an acidic pH and a specialised Na+ independent carrier-mediated system (33). In higher concentrations,
diffusion was observed to be the main mechanism in rats\(^{(34)}\). For NAM, absorption was suggested to occur via diffusion at twice the rate of NA\(^{(35)}\), but new research on NAM absorption is not available. Furthermore, it is not known if the same mechanisms take place in ruminants.

Briefly, niacin feeding enhances the amount reaching the duodenum. But not the whole quantity supplemented reaches the duodenum, even after post-ruminal infusion. This provides evidence for abomasal or duodenal absorption before the duodenal cannula. Furthermore, there might be influences of the type of feed and vitamin given. Apparent absorption in the duodenum seems to be high, but the mechanism of absorption has not yet been studied in ruminants.

**Blood**

**Niacin in blood**

Data concerning blood niacin concentrations are given in Table 4. Obviously, concentrations vary in a wide range. A reason for this might lie in difficulties of vitamin analysis and/or in different blood fractions examined.

There is disagreement about the existence of NA in blood. Whereas Campbell et al.\(^{(12)}\) found both vitamins, Kollenkirchen et al.\(^{(36)}\) stated that only NAM was present in the blood of sheep. In two studies, only values for NAM were named\(^{(37,38)}\). It was not stated whether only NAM was found, or if only NAM was analysed. The metabolism of niacin in the body might provide an explanation for this discrepancy. There appears to be no direct conversion of NA to NAM. NA is first converted to NAD, and NAM is then produced from the hydrolysis of excess NAD\(^{(39)}\). Part of the NAM formed is reutilised to NAD, but NAM is produced in excess to supply extra-hepatic organs with niacin\(^{(40)}\). Therefore, NAM seems to be the main transport form of niacin in blood\(^{(44)}\), although the NA that escaped liver metabolism is also transported to various cell types in the body\(^{(41)}\).

The difference in niacin content of the analysed blood fractions between control and niacin-supplemented groups was significant in three studies\(^{(37,38,42)}\), but not in the others\(^{(12,43,44)}\). Campbell et al.\(^{(12)}\) found a significant difference between the vitamers. The addition of NA enhanced both NA and NAM, while feeding NAM had a decreasing impact on blood NA and NAM concentrations. This was not expected, since the NAM-supplemented group had the highest duodenal values of niacin; at this point it is not explainable why this should result in the lowest niacin content of plasma. For rats, it was demonstrated that NAM is also able to pass from the bloodstream back to the lumen\(^{(34)}\). This could explain the previously mentioned results in the NAM group\(^{(12)}\), should it occur in ruminants as well. But the reasons for and physiological role of such a process remain unclear\(^{(34)}\).

In sheep, the NAM concentration of whole blood was not influenced by NA or NAM supplementation\(^{(36)}\). Hence the conclusion was drawn that concentrations in blood appeared to be unaffected by supplementation, even though the amount reaching the duodenum was increased. In contrast, Ottou et al.\(^{(42)}\) infused 6 g niacin into the proximal duodenum and observed an increase in the niacin content of whole blood. The results of this study also lead to the conclusion that ruminal absorption could be excluded as a reason for observed differences in blood niacin content, because changes occurred after post-ruminal infusion. In other studies as well there was no obvious relationship between ruminal and blood niacin concentrations\(^{(12,36)}\).

In humans, there seems to be a kind of homeostasis of niacin in blood\(^{(45)}\). Excess niacin gets converted into a storage form of NAD in the liver. Pires & Grummer\(^{(46)}\) conducted an experiment with different amounts of NA infused in the abomasum and concluded from effects on blood metabolites that some build-up of NA in blood or adipose tissue might have occurred. If some homeostasis system exists also in ruminants, it would explain studies without an effect on blood niacin, but would fail to elucidate observed differences in the others.

**Effect on blood metabolites**

The effect of niacin on several blood parameters (glucose, NEFA and β-hydroxybutyrate (BHBA) as the main ketone body) has been studied extensively in dairy cattle (Table 5). Only surveys including glucose, NEFA and BHBA are incorporated in this Table. One study mentioned separate results for several lactation weeks\(^{(47)}\), and so values for week 2 were included in Table 5 as the earliest sampling time.

**Non-esterified fatty acids**

In Table 5, the only significant effect of a niacin supplementation was an increase of NEFA in the niacin group\(^{(43)}\). This was not expected, since niacin is thought to be anti-lipolytic, which would result in a lower NEFA concentration. The authors proposed that this was due to increased lipoprotein lipase activity, which is stimulated by NA, thus resulting in decreased plasma TAG content and increases in NEFA. Apart from this effect, significant interactions between niacin and fat supplementation were observed, resulting in an increase in NEFA when niacin was supplemented, while NEFA decreased when niacin and fat were given\(^{(48)}\). If only studies are considered where niacin was given to periparturient cows (treatment started 2 weeks before or within 2 weeks after calving), there was no effect of a niacin supplementation (Table 5) as was described by Chamberlain & French\(^{(49)}\) as well. Jaster & Ward\(^{(47)}\) also analysed influences in other lactation weeks (not included in Table 5), where a decreasing effect of niacin on NEFA in week 4 was observed. Therefore, if given orally, it is not clear that niacin acts more on NEFA in periparturient than other cows.

NA was used as a lipid-lowering agent in humans for decades, but, until recently, cellular mechanisms have not been well understood\(^{(50)}\). In 2003, the receptor HM74A was identified in adipose tissue, to which NA is a high-affinity ligand\(^{(51)}\). Activation of the receptor starts an inhibitory G-protein signal that reduces adipocyte CAMP concentrations by repressing adeny1 cyclase activity, which inhibits lipolysis. The endogenous ligand of HM74A is not known\(^{(50)}\). But NAM acted only as a very weak agonist on HM74A and seems therefore not to affect plasma lipid profiles\(^{(51)}\). For humans it was concluded that the endogenous level of NA is too low to impact on receptor activity\(^{(52)}\), but supplementation might enhance this level.
et al. (1990)(46)*\# $\# P<0.05$) between the control and niacin groups have been observed for these parameters.

It must be kept in mind that after supplementation in the usual range for dairy cows, NAM seems to be the dominating form of niacin in blood. Apart from relatively low NEFA values in some surveys, this could also explain the absence of a niacin effect on NEFA in most studies, even in those where an effect on blood niacin concentrations was shown(37,38,42)*. In two of those studies, the increase in blood niacin was an increase of NAM (37,38), which would not be expected to act on lipolysis. Reduction of plasma NEFA was achieved in fasting cows after one single abomasal infusion of 6 mg NA/kg body weight (approximately 5 g/cow)(46)*, but not after continuous duodenal infusion of 6 g NA/cow per d(53). Maybe if higher amounts of NA were to reach the duodenum, concentrations of NA in blood would be enhanced, possibly due to an increase in absorption via passive diffusion of NA at higher concentrations. Therefore, lipolysis would be affected, while physiological amounts due to an oral supplementation are converted in the liver into NAM and have therefore no effect.

In human subjects, it was often observed that after the effect of NA decays there was a major rebound of NEFA plasma concentrations(50). The same result was achieved in dairy cows, where mechanisms are not known. Karpe & Frayn (50) suggested that NA interferes with the ability of adipose tissue to normally regulate its lipolysis, but mechanisms are not known. Pires & Grummer(46)* concluded that the magnitude of the rebound depends on the dose of NA or duration of time with decreased NEFA. Karpe & Frayn(50) suggested that NA interferes with the ability of adipose tissue to normally regulate lipolysis, but mechanisms are not known. Pires & Grummer(46)* state that if NA is continuously delivered.
Table 5. Impact of niacin on several blood metabolites

<table>
<thead>
<tr>
<th>Reference</th>
<th>Feeding ration</th>
<th>Niacin supplement (g/d)</th>
<th>NEFA (μmol/l)</th>
<th>BHBA (mg/l)</th>
<th>Glucose (mg/l)</th>
<th>Blood fraction</th>
<th>Lactation week†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Driver et al. (1990)‡</td>
<td>45 % forage (lucerne hay and silage), 55 % concentrate (ground corn, ground oats, heat-treated soyabean meal)</td>
<td>0</td>
<td>69 mg/l</td>
<td>106</td>
<td>495</td>
<td>Plasma</td>
<td>–1 until 15</td>
</tr>
<tr>
<td></td>
<td>45 % forage (lucerne hay and silage), 55 % concentrate (ground corn, ground oats, heat-treated whole soyabean meal)</td>
<td>6</td>
<td>82 mg/l</td>
<td>97</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jaster &amp; Ward (1990)‡</td>
<td>50 % maize silage, 50 % concentrate (ground shelled corn, soyabean meal)</td>
<td>0</td>
<td>250</td>
<td>29</td>
<td>556</td>
<td>Plasma</td>
<td>2</td>
</tr>
<tr>
<td>Martinez et al. (1991)‡</td>
<td>40 % chopped lucerne hay, 60 % concentrate (beet pulp, whole cottonseed and -meal, corn, wheat, molasses), total 2 % fat</td>
<td>0</td>
<td>367</td>
<td>n.d.</td>
<td>711</td>
<td>Plasma</td>
<td>Average 84 DIM</td>
</tr>
<tr>
<td>Erickson et al. (1992)‡</td>
<td>45 % forage (lucerne grass haylage, maize silage), 55 % concentrate (high-moisture shelled corn, soyabean meal) With 3 % Ca salts of long-chain fatty acids and niacin</td>
<td>0</td>
<td>265</td>
<td>65</td>
<td>554</td>
<td>Plasma</td>
<td>2 until 14</td>
</tr>
<tr>
<td>Chilliard &amp; Ottou (1995)‡</td>
<td>79 % forage (corn silage, hay), 20 % concentrate (beet pulp, wheat, barley, rapeseed meal, soyabean meal, molasses) With niacin infused into the proximal duodenum</td>
<td>0</td>
<td>130</td>
<td>46</td>
<td>725</td>
<td>Plasma</td>
<td>Average 110 DIM</td>
</tr>
<tr>
<td></td>
<td>77 % forage (corn silage, hay), 18 % concentrate (rapeseed meal, soyabean meal) With 3.5 % rapeseed oil infused into the proximal duodenum With 3.5 % rapeseed oil and niacin infused into the proximal duodenum</td>
<td>6 NA</td>
<td>93</td>
<td>40</td>
<td>733</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervantes et al. (1996)‡</td>
<td>Eight different forage:concentrate ratios; lucerne hay or haylage and maize silage were used as forage, maize and soyabean meal as concentrate With 400 g Ca salts of fatty acids With 400 g Ca salts of fatty acids and nicotinamide</td>
<td>0</td>
<td>120</td>
<td>39</td>
<td>588</td>
<td>Plasma</td>
<td>Average 112 DIM</td>
</tr>
<tr>
<td></td>
<td>40 % forage (lucerne haylage, maize silage), 60 % concentrate (corn, soyabean hulls and meal), total 2.8 % fatty acids</td>
<td>12 NA</td>
<td>126</td>
<td>38</td>
<td>589</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 % forage (lucerne haylage, maize silage), 60 % concentrate (corn, soyabean meal, whole raw soyabean tallow), total 5-9 % fatty acids</td>
<td>0</td>
<td>157</td>
<td>64</td>
<td>607</td>
<td>Plasma</td>
<td>Average 30 DIM</td>
</tr>
<tr>
<td></td>
<td>49–60 % forage (lucerne and maize silage), 51–40 % concentrate (cracked com, soyabean meal, roasted soyabean meal, whole cottonseed)</td>
<td>0</td>
<td>378</td>
<td>114</td>
<td>594</td>
<td>Plasma</td>
<td>–19 d until 40</td>
</tr>
<tr>
<td></td>
<td>40–50 % forage (lucerne and maize silage), 60–40 % concentrate (ground corn, starch, soyabean meal, roasted soyabean meal, whole cottonseed)</td>
<td>12</td>
<td>389</td>
<td>110</td>
<td>610</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drackley et al. (1998)‡</td>
<td>40–50 % forage (lucerne haylage, maize silage), 60–50 % concentrate (soyabean meal and hulls, shelled corn)</td>
<td>0</td>
<td>98</td>
<td>50</td>
<td>692</td>
<td>Plasma</td>
<td>4 until 43</td>
</tr>
<tr>
<td></td>
<td>40–50 % forage (lucerne haylage, maize silage), 60–50 % concentrate (soyabean meal and hulls, shelled corn, fat)</td>
<td>12 NA</td>
<td>117</td>
<td>48</td>
<td>693</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BHBA, β-hydroxybutyrate; NA, nicotinic acid; NAM, nicotinamide; n.d., not determined; DIM, days in milk.

* Significant differences (P ≤ 0.05) between the control and niacin groups have been observed for these parameters.
† Blood values given in this Table derive from that lactation week or are a mean of the given time span, where 0 is calving; therefore negative numbers are weeks prepartum and positive values post-partum.
‡ In these studies, the vitamer applied was not named. It was just stated that niacin was supplemented. But since the term niacin is occasionally also used as a synonym for NA(45), it is assumed that NA was fed in these surveys.
§ Several diets postpartum were given; therefore, the forage:concentrate ratio differs.
in sufficient quantities, it will limit lipolysis in adipose tissue and therefore reduce plasma NEFA.

This phenomenon might also be an explanation for the increase in NEFA in the work of Martinez et al. (43), if blood measurements were done in the rebound phase, but authors only named the day of blood sampling, not time after feeding. The time of measurement might be another explanation for studies without a niacin effect on NEFA. NEFA returned to starting values 4–6h after one abomasal infusion of 6mg NA/kg body weight (approximately 5g/cow)(46). This might take longer with an oral supplementation, since niacin has to pass the reticulo-rumen, but in some studies blood concentration of NEFA was measured before morning feeding(37,38,42), no effect lowered due to niacin feeding. Even in studies where niacin lactation was detected once (48), since niacin feeding enhanced ketones was not clearly explained. Erickson et al. (37) found more in negative energy balance, despite lower insulin concentrations in blood after niacin supplementation (28,44,57,58). Enhanced glucose elimination or there was an increase(59) in the niacin-supplemented group.

In the studies cited in Table 5, no significant effect of niacin on BHBA in blood glucose can be seen, even in studies with enhanced blood niacin concentrations. An impact of time after parturition is possible, since Jaster & Ward(47) found no effect in lactation weeks 2 and 8 to 12; however, in lactation weeks 4 and 6, the NAM group exhibited enhanced glucose concentrations, while the NA group was not different from control.

In other studies not included in Table 5, glucose concentrations were equal in control and treatment groups (28,44,57,58) or there was an increase(59) in the niacin-supplemented group.

For dairy cows it was assumed that increased glucose and insulin concentrations occurred in blood after niacin supplementation due to greater gluconeogenic activity (59). Others concluded that it is not clear if this is due to increased glucose production with a negative feedback by decreasing serum level of fatty acid precursors for hepatic ketogenesis. If an effect was seen, the mode of action of niacin on ketones might be traced back to either the amount of NA arriving in blood or to a time effect.

**Glucose**

In conclusion, NEFA have been shown to be lowered by NA under certain conditions, but not by NAM. After the effect of NA disappears, a rebound above basal values occurs, which afterwards returns to normal. Apparently, to induce these effects, the amounts of niacin arriving at the duodenum have to be high, which might not be the case in feeding trials with an oral, not rumen-protected supplementation. However, there were effects after oral supplementation as well. Based on data available, it is not possible to conclude if the presence or absence of an effect after oral supplementation is based on sampling time or the amount of NA arriving in blood.

**β-Hydroxybutyrate**

The only significant effect of niacin on BHBA in Table 5 was found in the work of Erickson et al. (55), where BHBA was lowered due to niacin feeding. Even in studies where niacin concentrations in blood have been enhanced (37,38,42), no effect was found. But an interaction between niacin, fat and week of lactation was detected once (48), since niacin feeding enhanced ketones during fat supplementation and decreased ketones when no fat was added throughout the study. But in lactation weeks 1 to 3, almost the opposite was seen. Jaster & Ward (47) also observed a time effect towards a significant reduction of BHBA in both NA- and NAM-supplemented groups in week 4, but not in lactation weeks 2 and 6 to 12.

An absence of an effect of niacin on BHBA was attributed to the low level of BHBA (27), because supplementation was started later in lactation, after the period with the highest incidence of ketonaemia (12,48,53). Driver et al. (58) found more NAM in the blood of treatment groups, but assumed this is only beneficial if the cows are in state of abnormal carbohydrate metabolism. The authors assumed that increases in blood ketone-body levels following the administration of NA are mainly and perhaps entirely due to changes in plasma NEFA levels, which was also observed in other surveys (56). But this is not obvious in several studies in Table 5. BHBA concentrations in the niacin-supplemented group were significantly lower in the study of Erickson et al. (55). This could not be seen in the NEFA level, at least not in the fat-supplemented rations. Others also observed differences in responses of NEFA and BHBA concentrations in blood to a niacin supplementation (48). Erickson et al. (55) concluded that NA impeded ketogenesis, but had no influence on lipolysis. As another mechanism they mentioned that mobilised fatty acids are stored in the liver of niacin-supplemented cows. However, in general it was deduced that the mechanism by which niacin reduces ketones is not known (55).

In the studies cited in Table 5, no significant effect of niacin on BHBA in blood glucose can be seen, even in studies with enhanced blood niacin concentrations. An impact of time after parturition as possible, since Jaster & Ward (47) found no effect in lactation weeks 2 and 8 to 12; however, in lactation weeks 4 and 6, the NAM group exhibited enhanced glucose concentrations, while the NA group was not different from control.

In other studies not included in Table 5, glucose concentrations were equal in control and treatment groups (28,44,57,58) or there was an increase (59) in the niacin-supplemented group.

For dairy cows it was assumed that increased glucose and insulin concentrations occurred in blood after niacin supplementation due to greater gluconeogenic activity (59). Others concluded that it is not clear if this is due to increased glucose production or decreased removal of glucose (57). Chilliard & Ottou (53) observed a decreased slope of glucose elimination after an intravenous injection of glucose when niacin was infused into the duodenum of cows in mid-lactation. Furthermore, the decrease in plasma glucose following an insulin challenge was less in the niacin group. In humans, NA was assumed to lower insulin sensitivity, but this was not observed in 20% of subjects studied (60). Enhanced glucose elimination after an intravenous glucose tolerance test was found in cows in negative energy balance, despite lower insulin concentration, which suggests an increased response to endogenous insulin (61). It was proposed that the decreasing impact of sufficient amounts of NA on NEFA is the cause for the observed results, rather than a direct effect of NA, since high NEFA concentrations have been shown to induce insulin resistance (61). But results seem to be contradictory, which may in part be explained by different levels of energy supply and thus lipolysis. Other explanations cannot be given; it can only be concluded that insulin is involved in reactions of blood glucose to niacin.

**Milk**

To our knowledge, only two research groups measured the niacin content of milk of dairy cows (14,62). Values ranged from 0.46 to 0.87mg/l (14,62). Wagner et al. (62) found only...
NAM, while Nilson et al. (14) did not distinguish between vitamers. NAM content of milk was enhanced after NA supplementation (62), but the highest niacin content resulted in the lowest milk niacin content in the other study (14). Ruminal niacin concentrations have also been measured, and no relationship was apparent between ruminal and milk niacin concentrations (14). But other information is lacking; therefore no statement for the carryover of niacin into milk can be made.

The influence of niacin supplementation on other milk parameters is shown in Table 6, where only studies measuring at least milk yield, fat, and protein content are included.

**Milk yield**

In two studies, milk yield was increased after niacin supplementation (37,48), while it was not influenced in the others mentioned in Table 6. The absence of a niacin effect was explained in that cows were too far into lactation and thus not in a negative energy balance (42). But this would not match with the work of Cervantes et al. (37) where an effect was seen even though cows were in mid-lactation and probably not in a negative energy balance. In other studies not presented in Table 6, milk yield was either not affected (62) or was increased due to niacin feeding (47). But these authors did not observe differences until lactation week 9. In addition, values in the NA group did not differ from control; only the NAM group did (47).

The increase in microbial protein production after niacin feeding was made responsible for enhanced milk production (47). Furthermore, these authors suggested that the function of niacin in lipid and energy metabolism might play a role. Even if the niacin content of plasma was enhanced after niacin supplementation, this had no impact on milk yield (38,42). But in one study NAM in plasma and milk yield were enhanced in supplemented animals (57). Therefore, exact mechanisms remain unclear.

**Milk protein**

In contrast to most studies in Table 6, Erickson et al. (55) observed a significant increase, and Drackley et al. (48) a significant decrease in milk protein concentration, after niacin supplementation. Furthermore, an interaction between niacin and type of soyabean processing (38), or niacin and fat supplementation (28), was demonstrated. For protein yield, tendencies for an increase due to niacin supplementation have been detected (37,48,55). There were also tendencies for interactions between niacin, fat, and week of lactation (48). In the other studies in Table 6, no effect of a niacin supplementation was seen. Even in surveys where niacin concentration in blood was significantly enhanced in the supplemented group, differences in the response of milk protein to niacin supplementation occurred (37,38,42). In one study, no effect was observed (42), while an increase in protein yield was found in another (37). Furthermore, an interaction between niacin and type of soyabean processing was also observed for protein concentration of milk (38).

Erickson et al. (55) assumed that amino acid uptake of the mammary gland might be enhanced due to the effect of niacin on insulin. Intravenous insulin has been shown to increase milk protein and the percentage of casein in milk (63). Several studies also measured casein concentrations in milk. No effects of niacin on casein content or yield in milk were observed (47), there even was a tendency for lowered casein content and yield after niacin supplementation (48). However, in another study (55), the decrease in percentage casein-N of total N due to niacin feeding was significant for only one of two rations. It is therefore not possible to conclude if niacin acts via insulin on casein and/or protein synthesis.

Especially in the case of protein yield, changes in milk yield might also play a role or were probably the reason for observed differences (48). A theory for occasionally observed effects of niacin on milk protein content was an increased microbial protein synthesis in the rumen (55). Other authors stated that mechanisms of niacin to increase protein content of milk still need to be clarified (38). Thus, it cannot be concluded if effects are rather systemic or ruminal.

**Milk fat**

Except for Belibasakis & Tsirgogianni (57), who observed increased milk fat concentrations and yield after niacin was given, there were no significant effects of niacin on milk fat in studies in Table 6. Cervantes et al. (37) observed a tendency for decreasing milk fat content in NAM groups. Nevertheless, there have been several interactions. Interactions were found between niacin and fat (28) as well as between niacin, fat, and week of lactation (48). Bernard et al. (64) showed an interaction for niacin and processing of soyabean. In surveys not mentioned in Table 6 no effect was seen (62), whereas other authors found increased milk fat content in lactation weeks 1 and 4 after NAM but not after NA supplementation (47).

If only studies are considered where niacin supplementation had an impact on blood niacin content, then there was no effect on milk fat (38,42) or a trend towards lower milk fat contents in the niacin-supplemented groups (37). Therefore, changes following niacin supplementation might rather lie at the ruminal level. But since most research on the effects of niacin in the rumen was focused only on the rumen, and no milk measurements were done, it is difficult to accept or to reject this thesis. Three studies measured ruminal and milk parameters in the same trial (12,27,28) and all came to different results. One observed no effect of niacin on ruminal VFA concentration, but an interaction between niacin and fat on milk fat content (28). Another detected a tendency toward a decreased molar proportion of acetate and an interaction between fat and niacin for molar proportion of butyrate, which did not lead to changes in milk fat content or yield (27). Campbell et al. (12) found no effect on ruminal VFA concentrations or molar proportions, or on milk fat. Hence, other mechanisms might be involved as well.

**Future research directions**

Considering the number of metabolic reactions where NAD(H) and NADP(H) are involved, the importance of niacin is obvious. However, animal trials with niacin supplementation did not lead to consistent results; therefore it is still not possible to determine the exact conditions or doses for niacin supplementation. But there are several
### Table 6. Impact of niacin on several milk parameters

<table>
<thead>
<tr>
<th>Reference</th>
<th>Feeding ration</th>
<th>Niacin supplement</th>
<th>Milk (kg/d)</th>
<th>Protein (%)</th>
<th>Protein (kg/d)</th>
<th>Fat (%)</th>
<th>Fat (kg/d)</th>
<th>Lactation week†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Driver et al. (1990) (38)‡</td>
<td>45% forage (lucerne hay and silage), 55% concentrate (ground maize and oats, heat-treated soyabean meal)</td>
<td>0</td>
<td>38·5</td>
<td>2·84</td>
<td>1·09</td>
<td>3·53</td>
<td>1·34</td>
<td>– 1 till +15</td>
</tr>
<tr>
<td>Erickson et al. (1990) (25)</td>
<td>60% forage (corn silage, lucerne-grass silage), 40% concentrate (shelled corn, soyabean meal)</td>
<td>12 NA</td>
<td>23·2</td>
<td>3·31</td>
<td>1·11*</td>
<td>3·26</td>
<td>0·94</td>
<td>110 DIM</td>
</tr>
<tr>
<td>Martinez et al. (1991) (43)‡</td>
<td>40% chopped lucerne hay, 60% concentrate (beet pulp, whole cottonseed and -meal, corn, wheat, molasses), total 2% fat</td>
<td>0</td>
<td>30·8</td>
<td>2·97</td>
<td>1·03</td>
<td>3·45</td>
<td>1·25</td>
<td></td>
</tr>
<tr>
<td>Lanham et al. (1992) (44)‡</td>
<td>40% forage (corn silage, Bermuda grass hay), 60% concentrate (corn, soyabean meal)</td>
<td>6 NA</td>
<td>23·7</td>
<td>3·02</td>
<td>1·10</td>
<td>3·82</td>
<td>1·00</td>
<td></td>
</tr>
<tr>
<td>Bernard et al. (1995) (64)‡</td>
<td>54% forage (corn silage, lucerne hay), 46% concentrate (whole soyabean, soyabean meal and hulks, corn, wheat middlings)</td>
<td>0</td>
<td>25·5</td>
<td>4·90</td>
<td>1·27</td>
<td>3·24</td>
<td>0·75</td>
<td></td>
</tr>
<tr>
<td>Ottou et al. (1995) (42)</td>
<td>79% forage (corn silage, hay), 21% concentrate (beet pulp, wheat, barley, rapeseed meal, soyabean meal, molasses)</td>
<td>0</td>
<td>22·5</td>
<td>3·11</td>
<td>0·70</td>
<td>4·34</td>
<td>0·98</td>
<td>On average 110 DIM</td>
</tr>
<tr>
<td>Cervantes et al. (1996) (37)</td>
<td>Eight different forage:concentrate ratios; lucerne hay or haylage and maize silage were used as forage, maize and soyabean meal as concentrate</td>
<td>0</td>
<td>30·7</td>
<td>3·21</td>
<td>0·98</td>
<td>3·45</td>
<td>1·07</td>
<td>On average 112 DIM</td>
</tr>
<tr>
<td>Christensen et al. (1996) (57)</td>
<td>60% forage (lucerne haylage, maize silage), 30% concentrate (corn, soyabean hulks and meal)</td>
<td>0</td>
<td>36·1</td>
<td>3·04</td>
<td>1·09</td>
<td>3·89</td>
<td>0·95</td>
<td>On average 30 DIM</td>
</tr>
<tr>
<td>Belibasakis &amp; Tsirgogianni (1996) (57)‡</td>
<td>50% forage (corn silage), 50% concentrate (corn, soyabean meal, wheat bran)</td>
<td>0</td>
<td>32·3</td>
<td>3·23</td>
<td>0·75</td>
<td>3·46</td>
<td>0·81</td>
<td>On average 90 DIM</td>
</tr>
</tbody>
</table>
Table 6. Continued

<table>
<thead>
<tr>
<th>Reference</th>
<th>Feeding ration</th>
<th>Niacin supplement (g/d)</th>
<th>Milk (kg/d)</th>
<th>Protein (%)</th>
<th>Protein (kg/d)</th>
<th>Fat (%)</th>
<th>Fat (kg/d)</th>
<th>Lactation week†</th>
</tr>
</thead>
<tbody>
<tr>
<td>DiCostanzo et al. (1997)**</td>
<td>50 % forage (lucerne haylage, maize silage, earlage), 50 % concentrate (cracked corn, whole cottonseed meal and meal, soyabean meal and hulls, blood meal, wheat middlings)</td>
<td>0</td>
<td>28-0</td>
<td>2-90</td>
<td>3-40</td>
<td>On average</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>12 NA</td>
<td>29-0</td>
<td>2-91</td>
<td>3-33</td>
<td>90 DIM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>24 NA</td>
<td>25-9</td>
<td>2-91</td>
<td>3-38</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>36 NA</td>
<td>28-7</td>
<td>3-17</td>
<td>3-35</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Madison-Anderson et al. (1997)(28)</td>
<td>50 % forage (lucerne hay, maize silage), 50 % concentrate (rolled maize and barley, soyabean meal, molasses)</td>
<td>0</td>
<td>31-9</td>
<td>3-03</td>
<td>0-96</td>
<td>3-11</td>
<td>0-99</td>
<td>On average</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12 NA</td>
<td>32-2</td>
<td>3-11</td>
<td>1-00</td>
<td>3-32</td>
<td>1-05</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>35-1</td>
<td>2-96</td>
<td>1-04</td>
<td>3-33</td>
<td>1-15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minor et al. (1998)†§</td>
<td>49–60 % forage (lucerne and maize silage), 51–40 % concentrate (rolled maize, soyabean meal, extruded soyabeans, whole cottonseeds)</td>
<td>0</td>
<td>32-0</td>
<td>3-01</td>
<td>0-94</td>
<td>3-65</td>
<td>1-13</td>
<td>0 till 40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12 NA</td>
<td>35-5</td>
<td>2-92</td>
<td>1-04</td>
<td>3-22</td>
<td>1-14</td>
<td></td>
</tr>
<tr>
<td>Drackley et al. (1998)††</td>
<td>40–50 % forage (lucerne haylage, maize silage), 60–50 % concentrate (soyabean meal and hulls, shelled corn)</td>
<td>0</td>
<td>30-5</td>
<td>3-29</td>
<td>0-99</td>
<td>3-56</td>
<td>1-06</td>
<td>4 till 43</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12 NA</td>
<td>33-2*</td>
<td>3-16*</td>
<td>1-04</td>
<td>3-50</td>
<td>1-15</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>31-8</td>
<td>3-16</td>
<td>0-98</td>
<td>3-68</td>
<td>1-16</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>33-6*</td>
<td>3-13*</td>
<td>1-05</td>
<td>3-60</td>
<td>1-21</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NA, nicotinic acid; NAM, nicotinamide; DIM, days in milk.

* Significant differences (P<0.05) between the control and niacin groups have been observed for these parameters.
† Values given in this Table derive from that lactation week or are a mean of the given time span, where 0 is calving; therefore negative numbers are weeks prepurpartum and positive values post-partum.
‡ In these studies, the vitamer applied was not named. It was just stated that niacin was supplemented. But since the term niacin is occasionally also used as a synonym for NA[45], it is assumed that NA was fed in these surveys.
§ Niacin was mixed in the concentrate; the goal was to reach an intake of 6 g niacin/cow per d.
‖ In this study, there was no influence of niacin supplementation on milk yield; therefore, the authors gave only average milk yield for all groups.
* In this study, 2 years were analysed, the mean of both years was taken; furthermore, values for each group have not been given, only for the main effects (processing of soyabeans, niacin supplementation), which are presented here.
** This study was also designed to test the effect of different heat-stress-exposure; therefore different lines not only represent different niacin levels, but also different climatic conditions. Each niacin level had its own control group, but only values for the first one are presented here.
†† Several diets post-partum were given; therefore the forage:concentrate ratio differs.
gaps of knowledge, which could, once resolved, answer this question. First, cognition of the effect of feeding on ruminal fermentation, niacin degradation and synthesis is insufficient. Furthermore, ruminal samples were taken at varying times after feeding and after niacin supplementation, which surely has an impact on the observed results. In addition, it is not known if absorption can occur in the abomasum or before the duodenal cannula and the mechanism of absorption is unspecified for ruminants. Niacin concentrations in blood also vary, which might be due to the different blood fractions analysed or vitamers examined. Different methods for niacin determination may lead to different results as well. It is also unknown whether some type of homeostatic system exists, as was suggested for man. NEFA concentrations in blood seem to be lowered by NA, but not by NAM, and it is uncertain if NA acts on ketone bodies via this effect on NEFA or if other mechanisms are involved. Furthermore, the effect on NEFA might also have an impact on glucose metabolism, which is mediated through insulin, even though mechanisms are not clear. The vitamin’s mode of action on milk parameters is uncertain and might be systemic or ruminal or a combination of both. If effects are rather systemic, feeding trials with oral, not rumen-protected supplemnations will have limits. This seems to be at least the case for blood parameters, since disappearance before the duodenum is high.

Considering these points, we would suggest the following directions for future research:

1. Different feeding regimes should be compared that characterise the impact of feed on niacin metabolism. Niacin content of the feed should be determined, as well as tryptophan, aspartate and quinolinate contents, since these are precursors of niacin synthesis.
2. Simultaneous determination of ruminal, duodenal, blood and milk parameters would be useful to detect potential conjunctions.
3. The time of sampling to investigate ruminal, duodenal and blood parameters should be standardised in relation to time of niacin feeding to avoid confusion between niacin and time effects.
4. Experiments should be conducted with niacin infused in the abomasum and simultaneous duodenal and blood niacin measurements to study absorption site and extent.
5. Studies on the mechanism of absorption for both vitamers would be useful.
6. Surveys on possible metabolic storage, for example, liver or tissues (such as ruminal or duodenal walls) seem to be favourable, where NAD(H) and NADP(H) concentrations are measured as well.
7. In general, research concerning niacin flow in the body is advisable.
8. To investigate if effects of niacin on milk parameters are rather systemic or ruminal, surveys with or without post-ruminal niacin infusion are desirable.
9. Studies on the influence of niacin on insulin in ruminants should be performed.
10. Distinctions should be made between both vitamers. In addition, the conditions and locations of conversion of one vitamer to another should be better investigated.

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References


