A complete review on propylene glycol for dairy cows

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Abstract

The use of propylene glycol in ration becoming a trend in recent years. This review unfolds the idea of metabolism in the rumen, intestine and liver. It is converted in to the propionic acid which is most glucogenic volatile fatty acid. Then it decreases the ketone bodies in the body by the influence of increased insulin level in the blood. The ketone bodies are the commonly found in increased concentration than normal, when occurrence of ketosis takes place. It can be used in the incidences of ketosis. In general, propylene glycol does not affect feed intake but due to its less palatability some precautions must be followed. It helps in milk production due to glucose enhancing effect. Also the propylene glycol have role in productive and reproductive traits of the cow if supplemented in the ration. Propylene glycol can be toxic when fed in higher dose than the recommendation.

Keywords: Complete review, propylene glycol, blood biochemistry, metabolism, milk production

1. Introduction

In recent years, the interest in using propylene glycol as a feed additive has grown among dairy livestock business. This is due to an increased supply of products from feed companies, which by virtue of their propylene glycol content, claim to increase milk production, prevent ketosis and improve reproduction. This review describes how propylene glycolis metabolised in the rumen and liver. It also gives idea about blood metabolites and hormones, liver composition, feed intake and milk production, in order to evaluate whether propylene glycol is likely to prevent excessive fat mobilisation and imbalances in carbohydrate and fat metabolism, and reduce the risk of ketosis. From transition onward upto two months of lactation, the dairy cow must accommodate a tremendous increase in energy demand by the mammary gland for milk production. This is done partly by increasing feed intake and partly by mobilisation of fat from adipose tissue. However, excessive fat mobilisation can induce an imbalance in bodily carbohydrate and fat metabolism, which may result in ketosis (Herdt and Emery, 1992; Goff and Horst, 1997) et al [44, 35].

Ketosis is a metabolic condition that primarily occurs 2–7 weeks after calving (Halse, 1978; Gillund et al., 2001) [41, 42, 33] with diagnosis occurring at a median of 24–28 days postpartum (Østergaard and Gröhn, 1999; Fleischer et al., 2001) [66, 27]. As a proportion, the lactational incidence rate of clinical ketosis has been reported to be between 0.011 and 0.092 (Erb and Gröhn, 1988; Rasmussen et al., 1999; Østergaard and Gröhn, 2000) [24, 73 67] while the prevalence of subclinical ketosis, defined by a threshold level of ketone bodies in blood or milk, or determined by semi-quantitative tests, varies between 0.12 and 0.34 (Kauppinen, 1983; Dohoo and Martin, 1984; Francois et al., 1997; Geishausser et al., 2000) [54, 18, 31, 32]. Clinical ketosis causes economic losses to the dairy farmer due to treatment costs, decreased milk production, impaired reproduction efficiency and increased involuntary culling (Gustafsson and Emanuelson, 1996; Fourichon et al., 1999; Rajala-Schultz and Gröhn, 1999; Østergaard and Gröhn, 1999; Reist et al., 2000) [40, 66, 30, 74]. It is difficult to estimate the economic loss of subclinical ketosis because it depends on the threshold values used to define subclinical ketosis (Gustafsson and Emanuelson, 1996) [40]. Clinical and subclinical ketosis are both characterized by elevated concentrations of ketone bodies in blood, milk and urine. Both clinical and subclinical ketosis are also associated with increased concentrations of non-esterified fatty acids (NEFA) and decreased levels of glucose in blood (Filar, 1979; Horber
et al., 1980; Itoh et al., 1998) [25, 47, 51]. The ketone bodies, acetacetate (AcAc), beta-hydroxybutyrate (BHB) and acetone (Ac), where AcAc is the parent ketone body, can be reduced to BHB in an enzymatic reaction or decarboxylated to Ac in a spontaneous non-enzymatic reaction (Bergman, 1971). Clinical and subclinical ketosis have also been associated with elevated fat infiltration in the liver (Gröhn et al., 1983) [37]. Propylene glycol has been used in the treatment of ketosis since the 1950s (Johnson, 1954; Maplesden, 1954) [52, 60] and is still used today (Bahaa et al., 1997; McClanahan et al., 1998) [52, 60]. Henceforth the use of propylene glycol is very important in animals.

2. The metabolism in the rumen
Experiments regarding the metabolism of propylene glycol were done showed that half of the infused propylene glycol (range 100–910 g) disappears from the rumen within 1–2 hours. The portion excluded within 3 hours (Emery et al., 1964, 1967; Clapperton and Czerkawski, 1972) [21, 3, 14]. Thus, propylene glycol was having its metabolism by absorption, fermentation or direct passage to the intestine and rumen and then in liver. It seems that propylene glycol is completely digestible as very low traces were detected in the faces (Emery et al., 1964, 1967) [21, 23]. Thus, the most important ways in which propylene glycol can be cleared is by absorption and fermentation.

2.1 Effect on volatile fatty acid
Normally the volatile fatty acids are in a particular ratio in the rumen like 7:3:1 and they become a change in their concentration when the diet gets changed. Propylene glycol has a significant effect on rumen fermentation pattern. It is very difficult to relate rumen VFA responses to dietary components used in the different studies, like propylene glycol as suggested by Czerkawski and Breckenridge (1973) [16] but when the in vitro, analysis did the direct conversion of the propylene glycol is in lactate was observed while when it ferment it was metabolized to propionate in liver Emery et al., (1967) [23]. Propylene glycol was predominantly absorbed from the rumen without alteration. An in vitro trial with rumen fluid from sheep suggests that feeds with a high forage/concentrate ratio result in less conversion of propylene glycol to propionate compared with feeds with a low forage/concentrate ratio (Czerkawski and Breckenridge, 1972, 1973) [16]. Kristensen et al. (2002) [57] suggested that under normal rumen conditions propylene glycol is predominantly metabolised in the rumen and not in the liver, both cows and heifers receiving propylene glycol are known to have a significantly higher proportion of propionate in rumen volatile fatty acids (VFA) that is indicative of substantial intraruminal metabolism of propylene glycol. Like molassed sugar beet pulp, in particular, induced the conversion of propylene glycol to propionate, probably because the highly digestible fibre in this feed stimulated the growth of certain microbes with the ability to rapidly ferment propylene glycol to propionate (Czerkawski and Breckenridge, 1973) [16]. Christensen et al. (1997) [12] reported that propylene glycol administered as an oral drench or mixed with the concentrate gave a more pronounced effect on the acetate/propionate ratio than feeding propylene glycol as part of a total mixed ration.

2.2 Rumen pH
Propylene glycol was seemed to increase in propionate as seen in the previous discussion, the consequence may be decreased in pH but it was observed that there is no change in significant pH scale of the rumen liquor when fed with 200 to 688 g per day. (Dhiman et al., 1993; Cozzi et al., 1996; Shingfield et al., 2002) [17, 15, 79].

3. The metabolism in the liver
As we have discussed priorly that the direct conversion of the propylene glycol in the rumen is lactate which is the direct substrate in the gluconeogenesis which enters in Krebs cycle and converted into oxaloacetate which directly has a role in propionic acid synthesis in ruminants. While propionic acid is the VFA having a role in energy production and milk yield synthesis in the cow. (Nielsen, 2004) [65], an experiment with radioactive labelled C14 has indicated that propylene glycol is metabolised to glucose via carboxylation of pyruvate to oxaloacetate in dairy cows (Emery et al., 1967) [23]. As already mentioned, Kristensen et al. (2002) [57] reported that a proportion of 0.1 of absorbed propylene glycol was converted to lactate in cows which were infused with 463 g propylene glycol. Huff (1961) [48] has shown that propylene glycol can be converted into lactuldehyde in the liver of rabbits, which can be converted into lactate. Lactate is a common substrate in gluconeogenesis in ruminants and enters via pyruvate, which is converted to oxaloacetate (Fig. 1). Different experiments with other animal species have also shown that propylene glycol is primarily oxidized to lactate and pyruvate (Ruddick, 1972) [76]. Further, propylene glycol that is metabolised to propionate in the rumen can also be converted to glucose via gluconeogenesis in the liver (Fig 1).

![Fig 1: Metabolism of propylene glycol and its interaction with the ketogenesis in the liver of cattle. Gluconeogenic pathways are shown by the solid lines. (Krebs, 1966)](image)

3.1 The effect on physiological parameters in relation to ketosis
The effects of propylene glycol on physiological parameters in the blood where propylene glycol has been used prophylactically and as a single component propylene glycol increases glucose and insulin, while NEFA and BHB are decreased, i.e. not in the treatment of ketotic cows and not in a mixture including their feed additives. (Miyoshi et al., 2001) [62]

3.2 Effect on Blood
Blood samples were taken and checked for glucose and insulin after supplementation of propylene glycol. It was observed that glucose and insulin were peaked by averagely 30 min post supplementation of propylene glycol Grummer et al. (1994) [39]. Oral administration of propylene glycol indicates that propylene glycol is readily available for the cow.
and that most of the propylene glycol is relatively quickly absorbed from the rumen and utilised in the liver for gluconeogenesis Studer et al. (1993) [82] Grummer et al. (1994) [89] Christensen et al. (1997) [112]. The peak, was seen at different dose and at different times especially those for insulin where the concentration increased by 200–400% within 30 min using dosages ranging from 307 to 1036 g (Studer et al., 1993; Grummer et al., 1994; Christensen et al., 1997) [82, 12, 39] the time of blood sampling in relation to the time of propylene glycol allocation is of great importance with respect to measured glucose and insulin responses. In some experiments the peak was observed (Grummer et al., 1994; Christensen et al., 1997; Miyoshi et al., 2001) [12, 62, 11] within the first 3 h after administering propylene glycol, while others (Cozzi et al., 1996; Shingfield et al., 2002a) [15, 78] later than the expected time. Consequently, the long interval between blood sampling relative to administration of propylene glycol may explain the minor effects of propylene glycol on insulin observed in these studies (Cozzi et al., 1996; Shingfield et al., 2002) [15, 79] showed that the glucose response is rather limited compared with insulin, even in experiments where blood samples were collected shortly after propylene glycol allocation (Christensen et al., 1997; Miyoshi et al., 2001) [62, 12]. An explanation for the limited effect of propylene glycol on glucose, although significant in some trials, is probably the large increase in insulin that maintains plasma glucose concentrations (Brockman and Laarveld, 1986) [100]. This is also in accordance with the concentration of insulin peaking before that of glucose (Studer et al., 1993; Grummer et al., 1994) [39, 82]. Ruminal conversion of propylene glycol to propionate may also explain the earlier peak in insulin since propionate stimulates pancreatic insulin secretion (Brockman, 1982) [100]. Further, propylene glycol itself or intermediates of propylene glycol metabolism, e.g. lactate and pyruvate, may also stimulate insulin secretion.

4. Physiological state of the animal
The trials were conducted in early lactation (Sauer et al., 1973; Formigoni et al., 1996; Fonseca et al., 1998; Miyoshi et al., 2001; Pickett et al., 2003) [62, 26, 29, 28, 69], mid and late lactation (Cozzi et al., 1996; Shingfield et al., 2002) [15, 79], heifers (Christensen et al., 1997) [12] in the lactating cow. If the plasma concentration of NEFA is high, so is the possibility of hepatic production of ketone bodies (Heitmann et al., 1987) [43]. Normally NEFA and BHBA get increased after early lactation and if balance feeds not given the concentration gets increased to peak level which can lead to mobilization of body reserve lead to ketosis. Feeding 60–150% more energy than required during the last 8 weeks before calving resulted in fatter cows at calving and higher levels of plasma NEFA in early lactation compared to cows fed to requirements (Nachtoni et al., 1986; Rukkwamsuk et al., 1998; Krup et al., 1999) [63, 77, 58]. It is therefore likely that the glucogenic effect of propylene glycol will reduce plasma NEFA, especially in cows that are fat at calving. It was observed that the concentration of NEFA and BHBA becomes decreased with supplementation of propylene glycol. Also, it was seen that insulin and glucose concentration was seemed to be elevated Grummer et al. (1994) [39] Cozzi et al. (1996) [15] and Shingfield et al. (2002) [79].

5. Dose
On the basis of Physiological state and the articles regarding comparison of the dose of propylene glycol can be studied Grummer et al. (1994) [39] stated the direct effect of the propylene glycol dose (ranging from 0 to 919 g per heifer per day) on the glucose on plasma glucose, insulin, NEFA and BHb levels. Sauer et al. (1973) [26] determined the effect of propylene glycol -dose in the range of 0–495 g per day on NEFA and BHb. This could be explained by due to differences in the physiological state of the animals between trials and possibly Cozzi et al. (1996) [15] did not find any significant effect of propylene glycol -dose in the range of 0–400 g per day on glucose, insulin and NEFA also less dose range used by Cozzi et al. (1996) [15] compared to Sauer et al. (1973) [26] and Grummer et al. (1994) [38]. Most significant studies are given above in a tabular form.

6. The effect on liver
It was showed that supplementation of the propylene glycol decreases the triacylglycerol (TG) content by the Administration of 1036 g propylene glycol daily from approximately 10 days before calving by 66 and 58% on days 1 and 21 postpartum, respectively (Studer et al., 1993) [82]. The similar results were seen by (Pickett et al. (2003) [69] he gave 518 propylene glycol per day orally for the first 3 days after calving and found a decrease in TG-content by 44% on day 7 postpartum compared to control cows. While some of the results were contrasted to this fact, propylene glycol supplementation of dry cows, fed to requirements, had no effect on liver TG (Bremm et al., 2000) that might be with some other results. The reducing effect of propylene glycol on liver TG is in accordance with propylene glycol decreasing plasma NEFA around calving, which subsequently causes lower fat accumulation in the liver (Grummer, 1993; Drackley et al., 2001) [38]. The effect of propylene glycol on the risk of ketosis In Table 1, only plasma BHB was used to evaluate the effect of propylene glycol on ketone bodies. However, quite a few investigations have used other ketone bodies, i.e. Ac and AcAc, to evaluate the effect of propylene glycol. Sauer et al. (1973) [26] and Halse and Møller (1978) [41, 42] in both these studies it was concluded that Acetic Acid concentration in blood plasma from early lactating cows, was lowered down when propylene glycol -doses were 178–495 g per day. It has also been showed that 350–500 g propylene glycol per day significantly decreases the Acetate content in milk when to cows with a milk concentration (Piatkowski et al., 1974; Girschewski et al., 1977) similar results were seen by Hűnnger and Staufenbiel, 1999 with a lower dose of propylene glycol. From these studies, it is concluded that propylene glycol has anti-ketogenic properties. The plasma NEFA and liver TG-content get significantly reduced when propylene glycol is supplemented to the diet. So we can suggest propylene glycol can help in reducing the risk of the ketosis in dairy cows.
**Table 1:** The effect of propylene glycol on the fermentation pattern and pH in the rumen of cows and heifers

<table>
<thead>
<tr>
<th>Reference propylene glycol – dose (g per day)</th>
<th>Allocated methoda</th>
<th>Number of cows</th>
<th>Days in milk</th>
<th>Time of samplingb</th>
<th>Acetate (mol/100 mol)</th>
<th>Propionate (mol/100 mol)</th>
<th>Butyrate (mol/100 mol)</th>
<th>Acetate/propionate</th>
<th>Rumen pH</th>
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<tr>
<td><strong>Shingfield et al. (2002a)</strong> [79]</td>
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<td>0</td>
<td>32</td>
<td>61</td>
<td>4–5 h post-PROPYLENE GLYCOL</td>
<td>67.4 a (100)</td>
<td>17.0 a (100)</td>
<td>11.9 (100)</td>
<td>4.0 a (100)</td>
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<td>0</td>
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<td>Dry cows and</td>
<td>4 h post-PROPYLENE GLYCOL</td>
<td>67.0 a (100)</td>
<td>18.3 a (100)</td>
<td>8.5 (100)</td>
<td>3.8 a (100)</td>
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<td>336c</td>
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<td><strong>Cozzi et al. (1996)</strong> [15]</td>
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<td>3</td>
<td>125</td>
<td>0, 2, 4, 6 and</td>
<td>8 h post-PROPYLENE GLYCOL</td>
<td>65.3 (97)</td>
<td>19.5 (110)</td>
<td>11.3 (105)</td>
<td>3.4 (89)</td>
<td>6.52 (100)</td>
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<td><strong>Grummer et al. (1994)</strong> [39]</td>
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**Dhiman et al. (1993)** [17]

| 0                                           | 20                | 119            | 3 h post-PROPYLENE GLYCOL | 66.8 a (100) | 18.3 a (100) | – | 3.6 a (100) | 6.83 (100) |
| 688e                                        | TMR               | 20             |              |                   |                       |                         |                        |                   |         |
| **Dhiman et al. (1993)** [17]               |                   |                |              |                   |                       |                         |                        |                   |         |
| 0                                           | 20                | 119            | 5 h post-PROPYLENE GLYCOL | 66.1 a (100) | 18.1 a (100) | – | 3.7 a (100) | 6.72 (100) |
| 688e                                        | TMR               | 20             |              |                   |                       |                         |                        |                   |         |
| **Emery et al. (1964)** [21] f              |                   |                |              |                   |                       |                         |                        |                   |         |
| 0                                           | 1                 | –              |              |                   |                       |                         |                        |                   |         |
| 2347                                        | P                 | 1              |              |                   |                       |                         |                        |                   |         |

Figures in parentheses are relative values compared to control groups. Values with different letters (a, b, c) within column and reference differ significantly (P < 0.05).

a P: propylene glycol poured on feed; C: propylene glycol mixed with concentrates; O: propylene glycol administered orally; TMR: propylene glycol mixed with TMR.
b Time of rumen sample collection compared to time of propylene glycol - allocation. c Mean dose of all animals, but dose was 2.5 ml/kg of body weight0.75. d Linear effect of propylene glycol (P < 0.10).
e Mean dose of all animals, but dose was 5% of individual forage intake.
f The same cow served as treatment and control cow, i.e. rumen samples were collected during and after allocation of propylene glycol in this cow.

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7. The effect on milk production, feed intake, energy balance, and reproduction

Milk yield and milk composition

Generally, we consider the milk is composed of fat and solid not fat wherein solid not fat has many more components which is also very much important like protein carbohydrate mainly lactose, minerals and vitamins (FAO,2001). The main component which gets change due to propylene glycol supplementation is lactose. An increase in milk yield could be expected when increasing the energy concentration of the feed ration (Dhiman et al., 1993; Andersen et al., 2002) [17, 3]. Which is directly proportional to the lactose content of milk. Propylene glycol is an additive with a high energy content of 23.7 MJ gross energy/kg DM (Miyoshi et al., 2001) [62]. When early lactating cows were fed 495 g of propylene glycol per day. Milk lactose content increased significantly by 0.2 percentage units but the dosage of 178–360 g per day had no effect on milk lactose percentage (Fisher et al., 1973) [20]. In mid-lactating cows, no effect was observed on milk lactose percentage when adding 200–400 g of propylene glycol to the diet (Cozzi et al., 1996; Shingfield et al., 2002b) [15, 30]. The decrease in milk fat % has been seen in milk with lactation of cow supplemented with propylene glycol. The reason for the tendency to a lower milk fat content could be that propylene glycol leads to a lower proportion of acetate in the rumin. This might reduce the amount of acetate available for de novo fatty acid synthesis in the mammary gland. The tendency towards reduced milk fat content could be due to the decrease in plasma NEFA. Since lowered NEFA concentrations lead to decreased NEFA-uptake by the mammary gland (Emery and Herdt, 1991; Nielsen and Riis, 1993) [23]. Early lactating cows had a tendency to decreased milk fat content and in fact, Fisher et al. (1973) [20] observed a significant reduction in the fat percentage when allocating 360 g of propylene glycol per day (Table 1). While other component like protein, one could expect an increased milk protein percentage when supplying propylene glycol, under the assumption that propylene glycol decreases amino acid requirements for gluconeogenesis, and that the spared amino acids would be limiting for increased protein synthesis in the mammary gland (Griniari et al., 1997; Knowlton et al., 1998) [36, 55]. Also, an increase in the energy content of the feed may stimulate an increase in milk protein percentage (Sutton, 1989) [75]. However, propylene glycol does not affect milk protein in early lactating cows, probably because the relatively small doses of propylene glycol have not been able to increase the energy content of the diet sufficiently and there has not been a shortage of the spared amino acids for the milk protein synthesis. Thus, it has been argued that reductions in the catabolism of glutamine, glutamate and aspartate will not directly increase the supply of limiting amino acids (Reynolds et al., 1997) [82]

7.1 Feed intake

Propylene glycol is an unpalatable feed additive which is having a taste not acceptable to the animal (Johnson, 1954; Girschewski et al., 1977) [52]. This certainly decreases the dry matter intake of the cow. So it is very much important to mix the propylene glycol into the concentrate or drench directly to the animal Miyoshi et al. (2001) [62]. No direct effect of propylene glycol was seen in the cow in early lactation regarding palatability. An explanation for this is most likely that the dosages used do not increase the energy density of the feed sufficiently to induce an increase in feed intake. Furthermore, feed intake in early lactation is also influenced by metabolic factors and the increase in insulin triggered by propylene glycol (Fisher et al., 1973; Miyoshi et al., 2001; Pickett et al., 2003) [62, 69, 26]. While when it was seen for a mid-lactating cow the significant reduction in DMI was observed Dhiman et al. (1993) [17].

7.2 Energy balance and reproduction

It is normally seen that with an increase in milk production the milk yield decreases. The same phenomenon can be seen after the time of supplementation of propylene glycol. Milk fat gets decrease after supplementation of propylene glycol Miyoshi et al. (2001) [62] found with 518 g propylene glycol daily from 7 to 42 days postpartum compared to control cows had a decrease in milk fat %. It was observed that. Excessive negative energy balance and high levels of ketone bodies have been associated with decreased reproduction efficiency in dairy cows (Reist et al., 2000; Vries and Veerkamp, 2000; Jorritsma et al., 2003; Taylor et al., 2003) [74, 85, 64, 53]. Therefore, the ability of propylene glycol to reduce fat mobilisation and hepatic ketogenesis, especially in cows with high levels of NEFA might have beneficial effects on reproduction. Lucci et al. (1998) [80] used “days to first oestrus” to assess the effect of propylene glycol on reproduction, but found no significant difference between the propylene glycol -group (40.2 days) and the control group (45.2 days). Daily drenching of 518 g propylene glycol per cow from 7 to 42 days after calving did not affect days to the first service, days open, and services per conception (classical reproduction traits) compared to control cows (Miyoshi et al., 2001) [62]. However, first ovulation occurred significantly earlier (32.3 days versus 44.5 days) and the first luteal phase was significantly longer (13.1 days versus 7.3 days) indicating a better quality of the follicles and improved ovarian function in propylene glycol -fed than control cows. The lack of significant differences in the classical reproduction traits could be explained by the lack of difference in energy balance between propylene glycol -cows and control cows. In the study of Miyoshi et al. (2001) [62], cows were inseminated at first oestrus and observed 45 days postpartum, which might have been a disadvantage for propylene glycol -cows in relation to days to first service and days open, since they already had their first ovulation 32.3 days postpartum, while control cows did not have theirs until 44.5 days postpartum. More studies with more animals are needed to confirm the effects of propylene glycol on reproduction.

8. Toxicity and side effects of propylene glycol

Farmers and veterinarians in Denmark have experienced that some cows expressed rapid shallow breathing, ataxia, salivation, somnolence and depression when adding propylene glycol to the feed ration of horses. (Dorman and Haschek, 1991; McClanahan et al., 1998) [19, 61]. The most extreme cases are for horses and cows that died after accidental allocation of 3–4 kg propylene glycol (Dorman and Haschek, 1991) [19]. Hyperventilation could be explained by propylene glycol -induced destruction of red blood cells (RBC) and thereby a shortage of oxygen for the cow. In sheep, it has been shown that injection of an aqueous solution containing 39 ml of propylene glycol, induced haemolysis (Potter, 1958). The median toxic dose (TD50) of propylene glycol to cows has been reported to be 2.6 g/kg body weight (Pintchuk et al., 1993) [70]. This corresponds to a TD50-dose of 1.5 kg propylene glycol for cows weighing 600 kg. Clinical signs used to determine the TD 50 of propylene glycol were: ataxia depression and involuntary recumbency (Pintchuk et al., 1993) [70]. LD50-values have not been reported for cattle,
but using an LD50-value of 18 g/kg body weight 0.75 (assuming a body weight of 0.3 kg for rats and an LD50-value of 25 ml/kg body weight) leads to an LD50-dose of 2.2 kg propylene glycol for a cow weighing 600 kg. The above mentioned indicates that propylene glycol may have some side effects, especially at dosages above 500 g per day. However, it is noteworthy that some cows have shown symptoms even at doses lower than 150 g per day (Hindhede, 1976) [43] when using propylene glycol, whether it is mixed with the concentrates, the TMR or drenched, it is important to pay attention to the symptoms described above, especially during the first days after administering propylene glycol.

9. Conclusions

From the discussion, it can be concluded that propylene glycol is the glucogenic chemical substance, which was used extensively in the livestock despite its less palatability. Feeding resulted in reduced serum NEFA and BUN levels, implying reduced catabolism of body tissue, increased plasma glucose concentration, BCS and milk yield, and did not affect dry matter intake. It was strongly indicated that it was helpful in lowering the incidences of ketosis in cattle. Propylene glycol tends to increase milk yield and reduce milk fat percentage.

10. References


