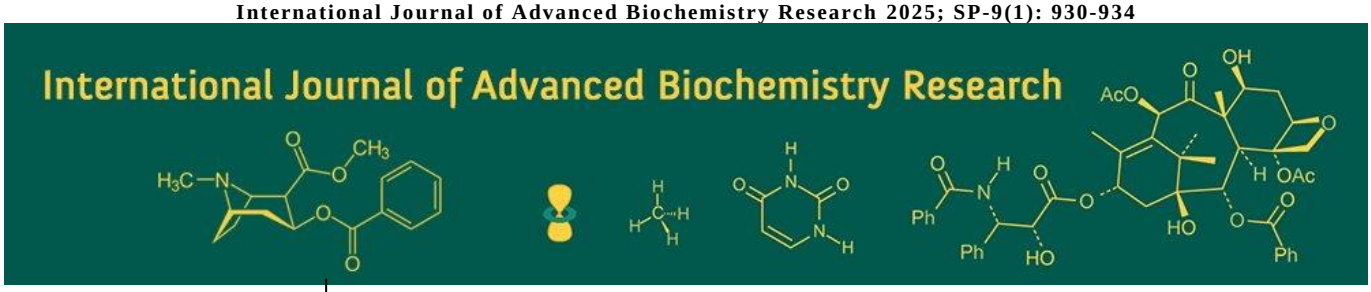

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Therapeutic approaches for managing pregnancy toxaemia in goats

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Abstract
The current study was undertaken to evaluate different therapeutic approach for pregnancy toxaemia (PT) in goats. The does after three and half months of gestation having blood β-hydroxybutyric acid (BHBA) levels ≥ 0.8 mmol/L were randomly divided into three groups. Does in Group 1 (n=7) were daily administered with 20 ml of E-Booster (p.o), Group 2 (n=6) were given daily supplementation of 100 gm commercial goat feed and does in Group 3 (n=6) does not receive any treatment or supplementation and served as control. There was a significant decline in mean blood BHBA levels noticed in the treatment groups (Group 1 and Group 2). During the course of therapy, the mean blood glucose level was not varied significantly among the groups. The decrease was almost similar in both the group supplemented with E-Booster and goat feed i.e. reached physiological levels in 3 days of treatment. However, the decline in BHBA was little quicker and sharper in does treated with E-Booster compared to the does supplemented with energy rich goat feed.

Keywords: Pregnancy toxaemia, BHBA, periparturient, E-booster

Introduction
The most frequent metabolic disorders of sheep and goats are pregnancy toxaemia (PT), hypomagnesaemia and hypocalcaemia, which occur during the periparturient phase (Brozos *et al*., 2011) [4]. PT in sheep and goats is a metabolic disorder that affects pregnant ewes or does, caused by a negative energy balance and is typically linked to rapid growth of multiple foetuses during late gestation (Navarre *et al*., 2009) [22]. It is also known as pregnancy ketosis, lambing or kidding sickness, pregnancy disease, twinning disease. It is most commonly seen in multiparous does or ewes which are carrying multiple foetuses (Brozos *et al*., 2011) [4]. It occurs because of competition between the dam and her foetuses for the glucose (Lima *et al*., 2012) [18]. Risk factors constitute multiple foetuses, poor quality of ingested feed/energy source, decreased dietary energy levels, hereditary factors, obesity, poor body condition, high parasite load, stress and multiple pregnancies. (Hefnawy *et al*., 2011) [11]. The energy or glucose requirement for foetal growth cannot be met by carbohydrates alone therefore, body reserves of fat tissue get mobilised. The mobilization of fat tissue generates non-esterified fatty acids (NEFAs) which are accumulated in the liver. NEFAs which are accumulated in the liver are partially used as energy resource, remaining is converted into ketone bodies namely acetone, acetoacetate and β-hydroxybutyric acid (B HBA). These by-products increase the concentration of ketone bodies in body fluids such as blood, milk, urine in addition to hypoglycaemia. This condition in small ruminants is referred to as pregnancy toxaemia or pregnancy ketosis. It is characterized by hypoglycaemia, low concentrations of hepatic glycogen, accelerated fat catabolism leading to high concentrations of ketone bodies (hyperketonaemia), high plasma concentrations of NEFA and high mortality rates (Van Saun, 2000) [28].
Clinical symptoms include anorexia, recumbency, a sweetish fruity odour from their breath, bloating, blindness, teeth grinding, and excessive salivation. biochemical analysis showed decreased levels of glucose and calcium, and increased levels of SGOT, SGPT, creatinine, BUN, NEFA and BHBA (Vasava *et al*., 2016) [29]. Diagnosis is usually based on history of advance gestation and clinical symptoms. Laboratory findings such as hypoglycaemia (often <2 mmol/L), increased urine ketone concentrations, high serum BHBA concentration (≥0.8

mmol/L), and occasionally hypocalcaemia (George, 2022) [8]. Body condition score (BCS) less than 2 out of 5 scale, blood BHBA > 0.8 mg/dL and blood glucose < 40 mg/dL (Murugeswari *et al*., 2022) [21], presence of ketone bodies in urine (Vijayanand *et al*., 2021b) [30, 31] were considered as reliable indicators for diagnosing pregnancy toxaemia.

Materials and Methods

The current study was conducted in pregnant does presented to the Department of Veterinary Gynaecology & Obstetrics, Veterinary college, Hebbal, Bengaluru and of goat farmers in surrounding areas of Bengaluru, Karnataka, India during March, 2024 to August, 2024. Pregnant does aged between 1 to 8 years with parity of 0 to 7 which were in more than three and half months of gestation period were subjected to Abdominal palpation or Ultrasonography or combination of both for pregnancy confirmation. The does were subjected to blood glucose and BHBA estimation using BeatO Curv Glucometer and Freestyle Optium Neo-H blood ketone monitoring system respectively with appropriate compatible strips. The does with subclinical pregnancy toxaemia having blood BHBA levels ≥ 0.8 mmol/L were randomly divided into three groups.

Group 1 (n=7): Each doe in this group were daily administered with high energy supplement (E-booster™ from Intas Pharmaceuticals Limited, Ahmedabad, India) containing gluconeogenic precursor fortified with Nicotinamide and Cyanocobalamin, 20 ml P/O until BHBA level reached the physiological range.

Group 2 (n=6): Each doe in this group received daily supplementation of 100 g commercial goat feed manufactured by Higain Feeds & Farms Private Limited (Bengaluru, Karnataka) containing Energy 45%, Fiber 35%, Protein 15%, Mineral mixture & essential salts 5% until BHBA level reached the physiological range.

Group 3 (n=6): Does with pregnancy toxaemia was not received any treatment or supplementation and served as the control group.

Blood BHBA and glucose levels were estimated daily by collecting the blood sample from the study does in the morning before grazing for consecutive 4 days or until their levels reached back to normal physiological range in the treated groups (Group 1 and 2).

Statistical Analysis

Blood glucose, blood BHBA were analyzed by two-way ANOVA (Analysis of variance) following Tukey's multiple comparisons test. Values were denoted by means ± standard error (SE) and the differences were considered as statistically significant at $P<0.05$.

Results and Discussion

Blood beta hydroxy butyric acid levels (BHBA levels)

The mean blood BHBA levels on 0th day (day of diagnosis) in Group 1, Group 2, and Group 3 was 1.12±0.11, 1.08±0.08, and 0.90±0.03 mmol/L. The mean blood BHBA level was 0.98±0.06, 0.96±0.06 and 0.90±0.03 mmol/L after first day of treatment, 0.82±0.02, 0.81±0.03 and 0.88±0.03 mmol/L after second day of treatment, 0.68±0.01, 0.70±0.00 and 0.91±0.03 mmol/L, respectively after 3rd day of treatment indicated that the treatment resulted in decrease in BHBA level. Similar BHBA levels have been reported by Marutsova and Marutsova (2018) [19] during last two weeks prior to lambing in Lacaune and Mouton-charollais sheep with SCK (subclinical ketosis) as 1.11 mmol/L and Vijayanand *et al*. (2021) [30, 31] as 1.3±0.05 mmol/L in small ruminants with PT. Henze *et al.* (1998) [12] observed higher mean blood BHBA levels (3.47±0.22 mmol/L) in ketotic sheep, whereas Vasava *et al*. (2016) [29] reported 4.82±0.27 mmol/L in pregnancy toxaemic goats.

The values of BHBA decreased in both treated groups (G1 and G2) within 24h of treatment, and this decline was significant, quicker or earlier in the group supplemented with E-Booster® (G1) (day 0: 1.12±0.11 vs. day 1: 0.98±0.06 vs. day 2: 0.82±0.02 vs. day 3: 0.68±0.01 mmol/L) compared to group supplemented with energy rich goat feed (G2) (day 0: 1.08±0.08 vs. day 1: 0.96±0.06 vs. day 2: 0.81±0.03 vs. day 3: 0.70±0.00 mmol/L) and reached to normalcy within three days of treatment in both the groups. Cal-Pereyra *et al*. (2015) [5] found a similar pattern of drop in blood BHBA levels in propylene glycol + glycerol (2.6 v/s 1.1 v/s 0.7 mmol/L) when supplemented with two daily intakes of cracked maize (2.5 v/s 1.7 v/s 1.5). Sathish, (2023) found similar trend in decline with GLYCOW® gluconeogenic precursors like propylene glycol, liquid glucose and cobalt (0.88 v/s 0.60 v/s 0.54 mmol/L) and broken maize (0.80 v/s 0.74 v/s 0.69 mmol/L).

A lack of dietary energy intake results in insufficient substrate for ruminal synthesis of the glucose precursor, that is propionate, resulting in a negative energy balance (NEB). The synthesis of NEFAs leads to increased BHBA due to lipid mobilization and β-oxidation, resulting in excess ketone bodies (Ismail *et al*., 2008 and Hefnawy *et al.*, 2010) [15, 10]. Furthermore, while the liver produces ketone bodies, they are used up by other tissues, and ketosis could be induced by either underuse or overproduction. Harmeyer and Schlumbohm (2006) [9] reported that, this increase is due to a decline in pregnant females' ability to use BHBA, which has a range of detrimental effects on energy balance and glucose metabolism, favoring the development of pregnancy toxaemia, particularly with multiple/twin pregnancies.

Propylene glycol (Gluconeogenic precursor), which is absorbed intact from the rumen at a rate of 40% per hour, reaches its maximal blood level within 30 minutes of intake and its peak blood glucose conversion approximately 4 hours later. Propylene glycol is most likely converted to pyruvate before being transformed into glucose (Herdt and Emery, 1992) [13]. Whereas maize (major ingredient in energy rich goat feed) must be broken down by ruminal microbes and turned into volatile fatty acids, specifically propionic acid (Warner, 1964; Fahey and Berger, 1993) [32, 6]. Around 18-42% of maize starch may escape rumen digestion and undergo digestion in the small intestine (Orskov, 1986) [23].

According to Aiello *et al*. (1984) [1] and Jesse *et al*. (1986) [16], propylene glycol increases the concentration of pyruvate, which enhances the formation of oxaloacetate via pyruvate carboxylase. The level of intra-mitochondrial citrate is predicted to rise as available oxaloacetate increases, forming malonyl-CoA, a potent fatty acid transformation suppressor in mitochondria. This leads to lower ketone body production. Furthermore, Herdt and Emery (1992) [13] mentioned that glucose given by propylene glycol increases the insulin: glucagon ratio, which affects ketosis.

Blood glucose levels

In the current study, overall mean blood glucose values in does with pregnancy toxaemia before treatment (day of diagnosis) was 51.47±4.10 mg/dL. The mean blood glucose levels recorded on 0th day (before treatment) in Group 1, Group 2, and Group 3 was 53.57±4.73, 49.66±4.61 and 50.83±3.36 mg/dL, respectively. The mean blood glucose values were 58.14±4.45, 54.16±4.27 and 50.66±3.14 mg/dL after 1st day of treatment, 61.71±4.45, 60.16±3.92 and 51.16±3.36 mg/dL after 2nd day of treatment, 65.85±4.64, 65.16±3.82 and 51.66±3.39 mg/dL after 3rd day of treatment, respectively. Non-significant increase in the mean blood glucose level was noticed in the treated groups from day 1 and till day 3 of treatment when compared to untreated group (control).

Similar mean plasma glucose levels of 47.16±0.62 mg/dL and 57.0±11.57mg/dL in PT/ketotic does on the day of diagnosis was recorded by Akraiem *et al*. (2020) and Vijayanand *et al*. (2021) [30, 31].

Henze *et al*. (1998) [12] found that the average blood glucose level in ketotic sheep before therapy was 52.02±3.12 mg/dL. Furthermore, hypoglycemia was detected in just 40, hyperglycemia in 20, and normoglycemia in 40 per cent of ewes, and plasma glucose levels varied, with affected animals having higher values than healthy animals. In similar way Souto *et al*. (2019) [26] found that 82.9 percent of Dorper, Santa Ines, and mixed breed ewes with pregnancy toxaemia had hyperglycemia (132.12±30.96 mg/dL) and normoglycemia (68.94±7.2 mg/dL), while only 17.10 percent had hypoglycemia (43.74±5.22 mg/dL). The average glucose value in pregnancy toxaemia affected sheep was 97±2.9 mg/dL.

George (2022) [8] reported that the hypoglycaemia is not a reliable indicator, with up to 40% of cases having normal glucose levels and up to 20% having hyperglycemia. If the diagnosis requires additional confirmation, CSF glucose concentrations may be much more accurate than blood glucose levels. Uma Rani (2015) [27] recorded mean blood glucose value as 36.50±1.73 mg/dL in pregnancy toxaemic does and 117.50±5.20 mg/dL in healthy goats. Lima *et al.* (2012) [18] recorded 31.68±9 mg/dL of blood glucose level in pregnancy toxaemia of small ruminants on the day of diagnosis.

Cal-Pereyra *et al*. (2015) [5] investigated the blood glucose levels during experimentally induced pregnancy toxaemia in Corriedale ewes. Before therapy, the study groups had blood glucose levels of 26.82±9.72 and 29.52±9.9 mg/dL, respectively. Furthermore, they investigated various treatment protocols, like oral administration of propylene glycol with glycerol (Group 1) and twice-daily feeding of cracked corn (Group 2) and found a similar increase in blood glucose levels after treatment, with a mean of 58.68±8.64 mg/dL and 36.00±16.02 mg/dL on 1st day, 51.12±8.64 mg/dL and 41.04±19.26 mg/dL on 2nd day, and 57.06±5.22 mg/dL and 55.08±19.26 mg/dL on 3rd day of treatment. Sathish (2023) recorded blood glucose levels in Hassan ewes with pregnancy toxaemia. Before treatment, the study groups had blood glucose levels of 61.00±6.98 and 60.75±4.88 mg/dL respectively. They checked two treatment protocols, such as GLYCOW® containing propylene glycol, liquid glucose, cobalt and broken maize respectively. They found a similar fashion of increase in blood glucose levels after treatment, with a mean of 70.78±5.52 mg/dL and 65.50±5.14 mg/dL on 1st day, 75.44±4.13 mg/dL and 71.25±4.09 mg/dL on 2nd day, and 79.56±2.91 mg/dL and 74.38±3.51 mg/dL on 3rd day of treatment.

According to Henze *et al*. (1998) [12] and Pereira *et al*. (2010) [24], sheep have a reduced capacity to metabolize glucose during advance gestation, with rising glycemic levels most likely caused by increased insulin resistance in the peripheral tissues. Thus, in addition to a small number of sheep with hypoglycemia, another proportion of affected animals with normoglycemia also suffer from pregnancy toxaemia and the similar fashion of blood glucose level in does with pregnancy toxaemia has been recorded in the current study.

Hypoglycemia was caused by a lack of net energy in the diet, as well as an increased need for energy in the late stages of pregnancy due to twin or triplets. NEB produces hypoglycemia, which leads to decreased food intake and glucose turnover, resulting in pregnancy toxaemia (Vasava, 2016) [29]. In the clinical or final stages of pregnancy toxemia, hyperglycemia can often be associated with death of the foetus. (Souto *et al*., 2013; Lima *et al*., 2012) [26, 18].

The most typical finding in pregnant ewes is a high level of blood glucose concentration, which can be attributed to a state of stress that causes a high amount of cortisol (Ford *et al.*, 1990) [7]. Cortisol levels rise during normal pregnancy; nevertheless, subsequent pathologic increases in cortisol are linked to fetal and maternal morbidities; also, cortisol has a negative impact on maternal glucose homeostasis. In the study conducted, cortisol infusion raised maternal plasma glucose levels in pregnant sheep compared to control non-pregnant ewes (Keller-Wood *et al*., 2014) [17].

Propylene glycol (gluconeogenic precursor) administration increases blood glucose levels within 12 hours. Propylene glycol, which is absorbed intact from the rumen at a rate of 40% per hour, reaches its maximal blood level within 30 minutes of consumption and its peak blood glucose conversion approximately 4 hours later. Propylene glycol is most likely converted to pyruvate before being transformed into glucose (Herdt and Emery, 1992) [13]. In the does of Group 2 supplemented with energy-rich goat feed (majorly maize), there was an increase in blood glucose levels in a similar fashion to Group 1 because the broken maize must be digested by ruminal microbes and transformed into the volatile fatty acids, in particular propionic acid (Warner, 1964; Fahey and Berger, 1993) [32, 6]. Propionic acid is then absorbed at the ruminal papillae before being partly converted into lactate in the rumen wall and finally transformed into glucose in the liver by gluconeogenesis. Approximately 18-42% of maize starch might escape rumen breakdown and can be digested in the small intestine (Orskov, 1986) [23], while only 30-35% of the glucose produced by intestinal starch digestion may be noticed in the portal vein (Huntington and Reynolds, 1986) [14].

Table 1: Mean blood BHBA level (mmol/L) in does with pregnancy toxaemia before and after treatment

Days of treatment	Group 1 (n=7)	Group 2 (n=6)	Group 3 (n=6)
0th day	1.12±0.11^{ax}	1.08±0.08^{ax}	0.90±0.03^{ax}
1st day	0.98±0.06^{axz}	0.96±0.06^{axw}	0.90±0.03^{ax}
2nd day	0.82±0.02^{ayz}	0.81±0.03^{axw}	0.88±0.03^{ax}
3rd day	0.68±0.01^{ay}	0.70±0.00^{ayw}	0.91±0.03^{ax}

Note: Common superscript in row: a Common superscript in column: w, x, y, z

Means bearing common Superscripts with in a row or with in a column do not differ significantly with each other (P>0.05).

Table 2: Mean ± SE blood glucose level (mg/dL) in does with pregnancy toxaemia before and after treatment

Days of Treatment	Group 1 (n=7)	Group 2 (n=6)	Group 3 (n=6)
0th day	53.57±4.73	49.66±4.61	50.83±3.36
1st day	58.14±4.45	54.16±4.27	50.66±3.14
2nd day	61.71±4.45	60.16±3.92	51.16±3.36
3rd day	65.85±4.64	65.16±3.82	51.66±3.39

Conclusion

Efficacy of both the treatment protocols *viz* E-Booster containing Gluconeogenic precursor and Energy rich feed was good in bringing back the increased BHBA levels to the physiological range. The pregnancy toxaemia often commonly occurs during advance gestation because of negative energy balance due to rapid growth of multiple foetuses should be diagnosed at the earliest with hand held devices and treated immediately with gluconeogenic precursors or energy rich feed supplements to avoid further complications in terms of kid mortality, dam mortality and does infertility problems to minimize the economic loss to the shepherds or goat farmers.

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