

# Chapter 11

## Nutritional and Metabolic Disorders in Dairy Goats

João Simões and Carlos Gutiérrez

**Abstract** This chapter aims to describe significant aspects of the most common nutritional/metabolic diseases caused by insufficient or disbalanced nutrients intake, such as carbohydrates, proteins, vitamins, and macro or trace minerals, and their repercussion in goat metabolism. Goats are opportunistic feeding behavior animals, choosing the best nutrients in both hard environments or even in feed availability periods. In some conditions, e.g., poor quality forages in nutrients, and/or when energy or nutrient requirements overpasses their intake capacity and availability, goats may not keep metabolic homeostasis. Pregnant toxemia, urolithiasis, polioencephalomalacia, and selenium or vitamin E deficiency are major diseases with impact in production, reproduction and/or health in both low- and high-producing goats or their kids. In high-producing dairy goats, due to their higher nutritional demands, increased incidence of the called “production diseases” is observed. Subacute ruminal acidosis, lactational ketosis, hepatic lipidose, hypocalcemia and low milk fat syndrome are also major problem in dairy herds to require special attention. Risk factors of these disorders should be taken into account in nutritional and feed management programs. A holistic approach regarding these programs and herd health management are crucial to control or prevent nutritional and metabolic diseases in farms.

---

J. Simões (✉)

Department of Veterinary Sciences, Agricultural and Veterinary Sciences School,  
University of Trás-os-Montes and Alto Douro, 5000-801 Vila Real, Portugal  
e-mail: jsimoes@utad.pt

C. Gutiérrez

Research Institute of Biomedical and Health Sciences, University  
of Las Palmas de Gran Canaria, Trasmontana, 35413 Las Palmas, Canary Islands, Spain

© Springer International Publishing AG 2017

J. Simões and C. Gutiérrez (eds.), *Sustainable Goat Production in Adverse  
Environments: Volume I*, [https://doi.org/10.1007/978-3-319-71855-2\\_11](https://doi.org/10.1007/978-3-319-71855-2_11)

177

## 11.1 Introduction

Goats should consume feeds with energy and essential nutrients to cover basic functions (maintenance and normal activity) plus productive functions (growth, pregnancy and lactation), particularly in those breeds selected for milk or meat. To achieve both energy and nutritional requirements, this species adapted its feeding behavior and metabolism to harsh environments. Goats are very selective browsers/grazers, flexible, broad-scale and opportunistic feeding behavior animals (Mellado 2016), but previous personal dietary experiences, i.e., the personal information should also be taken into account (Morand-Fehr 2003). The feeding behavior also is affected by goats' social hierarchy. Barroso et al. (2000) reported that the most aggressive goats, apparently the large sized up and horned animals, are socially in the top; and this hierarchy affect the animal production. Additionally, the usual selection of forages when they are found abundantly decreases when their availability is scarce. Even if goats use social information, observing the feeding behavior of other individuals, the personal information seems to be prioritized when both information types are in conflict (Baciadonna et al. 2013).

Essential nutrients would be water, carbohydrates, proteins (amino acids), lipids, fat-soluble vitamins (A, D, E and K) and water-soluble vitamins (B complex and vitamin C) and minerals such as macrominerals (calcium, phosphorus, magnesium, potassium, and sodium) and microminerals (e.g., cobalt, copper, iodine, iron or selenium). Goats, as most mammals, can synthesize the needed vitamin C and thanks to ruminal and intestinal microorganisms can also synthesize vitamins K and B vitamin complex. However, when ingestion of essential nutrients is significantly lower than daily requirements for a long enough period of time, then deficiencies would occur. Nutritional and metabolic deficiencies are more commonly seen in high milk producing goats, but in kids with increased daily weight gain are also commonly at risk.

A great variety of diseases are related with energy and nutritional imbalance at digestive and other body systems exchanging animal's homeostasis, affecting several biological and biochemical systems and presenting lesional alterations in tissues and organs. Lactational ketosis, pregnancy toxemia (PT), fatty liver in adult's goats and hypoglycemia/hypothermia in kids (perinatal period) are common worldwide disorders of energy metabolism. Other diseases are caused by deficiencies or excess of vitamins and minerals (e.g., hypocalcemia and hipovitaminosis B1 or hypervitaminosis A and D), some others are (also) multifactorial and are provoked, or at least related, with nutrition, e.g., urolitiasis, and ruminal acidosis. However, it is necessary to empathizes that related nutritional disorders are not limited to these previous one. Several other nutrient-related disorders can also affect goats, such as, but not exclusively, caused by poisoning plants, fertilizers, poor feed storage (e.g., *Listeria monocytogenes* in silages, mycotoxins and botulism) and altered gut microbiome (enterotoxemia due *Clostridium perfringens* toxins production).

The present chapter supposes a review of the apparently more significant nutritional and metabolic diseases encountered in raising and managing goats around the world.

## 11.2 Lactational Ketosis and Pregnancy Toxemia

Lactational ketosis and PT (also named gestational ketosis) are manifestations of hyperketonemia in goats. In fact, both diseases are related with blood  $\beta$ -hydroxybutyrate (BHBA) levels usually greater than 0.4 mmol/L (Albay et al. 2014; Doré et al. 2015). These metabolic disorders are provoked by energy imbalance, i.e., negative energy balance (NEB) originated when outputs (to pregnancy or lactation) overpasses intakes (inputs) with detrimental energy flux. Lactational ketosis takes place in early *post-partum* and is normally related with high milk producing goats and more rarely with low-producing goats. In cows, it is considered a “production disease” with high prevalence in the first month of lactation. However, prevalence data are scarcely reported in goats, but probably a similar pattern to cows occurs (Zobel et al. 2015). PT, or the “twinning disease”, develops *ante-partum*, in the last 2–6 weeks of pregnancy due to enormous energy demands of fetuses (Edmondson et al. 2012). The prevalence of PT varies, according to farms and regions, from 1–4% to 40–60%, reaching a mortality >80%, in both does and fetuses, even when a cesarean section is made as treatment (Andrews 1997; Rook 2000; Bousquet 2005; Ismail et al. 2008; Lima et al. 2012).

*Ante-* and *post-partum* hyperketonemia can represent a major nutritional and metabolic problem in goat production relating subclinical and clinical forms of the disease with milk production (Zobel et al. 2015) and animal losses, even in low-producing does. Common clinical signs of lactational and gestational ketosis are reduced activity levels, ataxia, teeth grinding and anorexia (Brozos et al. 2011; Lima et al. 2016). In PT, anorexia with absence of ruminal motility and recumbency was even suggested as poor prognostic indicators in goats (Lima et al. 2016). Moreover, hyperketonemia also provokes immunity and inflammatory alterations with decreasing immunoglobulins serum levels of IgG, IgM and IgA (Hefnawy et al. 2010) and lymphocytes (Hefnawy et al. 2011), and increasing haptoglobins (González et al. 2011). Consequently, hyperketonemia can have a significant adverse influence in the general health and welfare of does.

### 11.2.1 Energy Metabolism and Hyperketonemia in Does

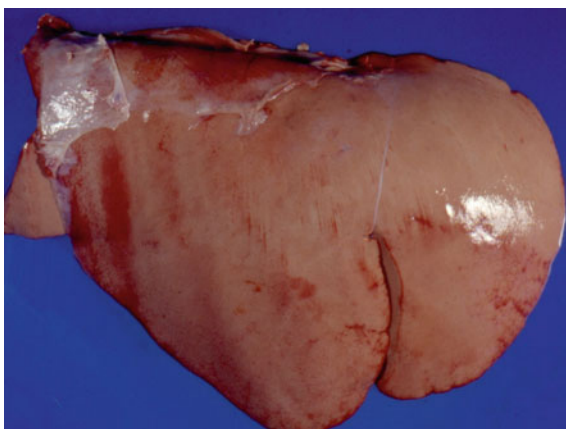
It is known that goats are well adapted to arid and semiarid environments, and show an appreciable feeding selectivity, choosing the best available forages when grazing (Mellado 2016) and consuming feed until approximately 5–6% (DM/daily) of BW. The daily DM intake increases progressively until the 2nd month of lactation,

always after the lactation peak, and progressively decreases until the next kidding mainly due to the compression of the gravid uterus to the rumen. Consequently, during the first two months of lactation, energy requirements due to milk production are higher than the energy input, and lipolysis (glucagon-mediated) occurs trying to compensate this disbalance. The loss of body condition score (BCS) is a natural consequence of this process. In a subsequent lactation phase, when the input surpasses the lactational output of energy, lipogenesis (insulin-mediated) takes place with BCS improvement. Normally, this process stop at the last month of gestation, in this last case due to fetus energy requirements. At this time a new NEB occurs and will be prolonged during early lactation phase.

Briefly, during NEB, an intense adipose tissue (triacylglycerols storage) mobilization provides high plasmatic levels of nonesterified fatty acids (NEFA) from triacylglycerol hydroxylation and are transported into liver. In the hepatocyte mitochondrial apparatus, NEFA are metabolized (ketogenesis) into three types of ketones: acetone, acetoacetate and BHBA. These ketones are a source of energy, as acetyl-Coenzyme A precursor (after Acyl group combination with the limiting factor: coenzyme A), mainly in mitochondria of skeletal muscles, mammary gland (including ketone transformation to fatty acids), heart and kidneys (Heitmann et al. 1987) with acid lactic production (causing metabolic acidosis). In normal conditions, and in pregnant and non-pregnant ruminants, all ketones are also significantly produced and absorbed from rumen to body. So, hyperketonemia occurs when the NEFA mobilization and their hepatic metabolism reach significant high levels, normally accomplished some degree of hepatic steatosis also denominated hepatic lipidosis or fatty liver disease (Fig. 11.1).

Hepatic lipidosis is due to slow exportation of lipoproteins from hepatocyte membrane to blood in ruminants (Kleppe et al. 1988), although can also be caused by cobalt or vitamins E and B<sub>12</sub> (cyanocobalamin) deficiencies (Johnson et al. 2004; Radostits et al. 2007). In consequence, a diffuse accumulation of lipids in hepatocytes occurs. This assumes special importance during a great lipid

**Fig. 11.1** Fatty liver with enlargement of the organ (hepatomegalia) and signs of rib's compression. Typical finding at *post-mortem* examination in a goat died from pregnancy toxemia (provided by the authors)



mobilization, when lipoprotein export limitation is more obvious. This lipidosis was associated with a decrease of hepatic metabolic functions (Bobbe et al. 2004). When hepatic fat concentrations reach >35% of liver weight, a poor prognosis can be established, at least in dairy cows (Herdt 1988).

### 11.2.2 Pregnancy Toxemia

PT occurs during last 4–6 weeks of pregnancy. Multiparous does with high ( $\geq 4$ ) or low ( $\leq 2$ ) BCS and multiples fetuses (twins, triplets, ...) are considered more susceptibles (Chartier 2009; Brozos et al. 2011).

A low DM/energy intake associated with a quickly fetuses growth, reaching 60–80% of total live BW during last weeks of gestation are the main etiological factor. In fact, 30–40% of glucose apported by the dam during this period is consumated by fetuses, and it is know that the fetuses have energy flux priority. At this time, pregnancies with two or more fetuses increase energy requirements until 180 and 240% of a single fetus, respectively (Ermilio and Smith 2011).

Several other risk factors are the environment, such as ventilation, thermal amplitude and poor litter box, as well as lack of exercise and stress (Chartier 2009). Additionally, during this period, hormonal alterations such as the decrease of insulin serum levels, due to an improvement of steroid hormones (gestation) and glucagon, also stimulates ketogenesis (Vernon et al. 1981; Sensenig et al. 1985).

Primary PT is related with poor quality of forages or poor nutritional management (e.g., abrupt feed changes) whereas any disease provoking a decrease of DM or energy intake causes a secondary PT (Radostits et al. 2007). High BCS seems to lead to intra-abdominal pressure due to physical presence of abdominal adipocytes reducing the rumen volume (Morand-Fehr 2005). In does with low BCS, the few body fat reserves are quickly mobilized entraining an extreme condition of subnutrition.

Blood levels of BHBA are used to improve the subclinical and clinical PT diagnosis and management. However, BHBA threshold to confirm PT is not well defined and values between 0.8 and 1.7 mmol/L have been reported (Trevisi et al. 2005; Ismail et al. 2008; Brozos et al. 2011; Sadjadian et al. 2013; Pichler et al. 2014; Zobel et al. 2015). In an attempt to minimize this problem, Doré et al. (2015) proposed a dynamic scale predicting the PT according to each one of last five weeks; in the 5th, 4th, 3rd, 2nd and 1st week *ante-partum*, the threshold value was 0.4, 0.4, 0.5, 0.6 and 0.9 mmol/L, respectively; but the values of sensitivity (61.8–73.7%) and specificity (58.4–89.7%) of this classifications were considered low. Zobel et al. (2015) suggested a cut-off of 1.7 mmol/L to define PT and lactational ketosis. Additionally, glucose tissue intolerance is well documented in some goats affected by PT (Lima et al. 2016), which present hyperglycemia but whose occurrence remains poorly understood. Even blood glyucose concentration can be used, together with BHBA level, at farm level for PT and lactational ketosis control,

although these levels represent a handicap for individual treatment of does. Further research is needed to refine this metabolic query.

### 11.2.3 Lactational and Gestational Ketosis Prevention

The prevention of both diseases are related with the minimization of risk factors reported above.

Special attention should be given to treatment and prevention of general diseases (e.g., footrot, parasitism, pneumonia, etc.) (Rook 2000). Stress minimization improving adequate facilities and welfare are two other significant aspects to take in account. However, the crucial steps are related with *ante-* and *post-partum* nutritional management (Zobel et al. 2015). Feed should apport adequate energy and nutrients, under a controlled intraruminal environment, supervising significant decrease of pH, i.e., minimizing ruminal acidosis by adequate carbohydrate/fiber ratio intake.

The BCS evaluation, which must be improved during the second half of lactation at adequate levels, between 3 and 4 score points. Finally, both PT and lactational ketosis can be dynamically monitored *ante-* and *post-partum* using serum levels of BHBA as a tool (Doré et al. 2015; Zobel et al. 2015).

## 11.3 Hypocalcemia

Hypocalcemia is a mineral disorder with particularities between domestic ruminants. It is assumed that hypocalcemia occurs due to Ca demands, higher that it can be replaced in blood and interstitial fluids (Goff et al. 2014). According Wilkens et al. (2012, 2014), goats show efficient adaptation to Ca dietary restriction.

In goats, the total Ca levels in blood serum varies from 8.9 to 11.7 mg/dL (2.2–2.9 mmol/L) (Kaneko et al. 1997), and the cation  $\text{Ca}^{2+}$  form is the diffusive portion to tissues (blood acid-base balance can affect  $\text{Ca}^{2+}$  levels). Hypocalcemia occurs below this normal range and can present both subclinical and clinical forms. The first symptoms occur at blood serum Ca levels reach 6 mg/dL, but can decrease until 2 mg/dL, or even less, before doe reach death (Yamagishi et al. 1999). Ataxia and recumbence are two major clinical signs of hypocalcemia, other than all consequences provoked by skeletal, cardiac and smooth muscle cells relaxation (Fig. 11.2).

Similar to cows, and inversely to ewes (Oetzel 1988), with milk yield increases in high-producing goats, hypocalcemia (milk fever) is developed during early lactation. In fact, during lactation a significant flux of Ca arises from blood to milk (DeGaris and Lean 2008) and milk contents high Ca concentration, approximately 130 mg/dL (32.5 mmol/L) (Barlowska et al. 2011). However, hypocalcemia can also occurs *ante-partum* in goats, due to high fetuses Ca requirements (Härter et al.

**Fig. 11.2** Clinical form of hypocalcemia in a peri-parturient multiparous goat. Note the sagging udder in an old high milk producing goat (provided by the authors)



2017), especially from 80 days of pregnancy onward when maternal body Ca reserves, mainly in bones, are transferred for fetal development and colostrum production (Härter et al. 2015). During pregnancy, Härter et al. (2017) estimated 60.4 mg/kg daily net requirements of Ca for maintenance in goats.

The Ca homeostasis is regulated by hormonal ambience (the serum Ca level is increased by parathormone and decreased by calcitonin) but calcitriol (11,25-(OH)<sub>2</sub>D<sub>3</sub>), the active form of vitamin D, assumes an important role. In fact, dietary Ca restriction (0.22% Ca in DM) increases plasma concentrations of endogenous calcitriol, which, in turn, improves intestinal absorption of Ca in goats, but without renal excretion exchange (Wilkens et al. 2012; Herm et al. 2015). Moreover, calcitriol also is related with Ca mobilization from bone in early lactation (Wilkens et al. 2014). Herm et al. (2015) suggested that the higher calcitriol production in goats than in ewes can justify the greater ability to regulate Ca homeostasis observed in goats. Calcitriol also improves milk excretion by mammary epithelium when sufficient intracellular glucose is available (Sun et al. 2016).

Goats can efficiently adapt to dietary anion–cation balance, namely in presence of anionic diets that causes metabolic acidosis, which increases the rate of intestinal Ca absorption and a greater bone resorption rate (Liesegang et al. 2006; Liesegang 2008), but without blood Ca levels improvement.

## 11.4 Obstructive Urolithiasis

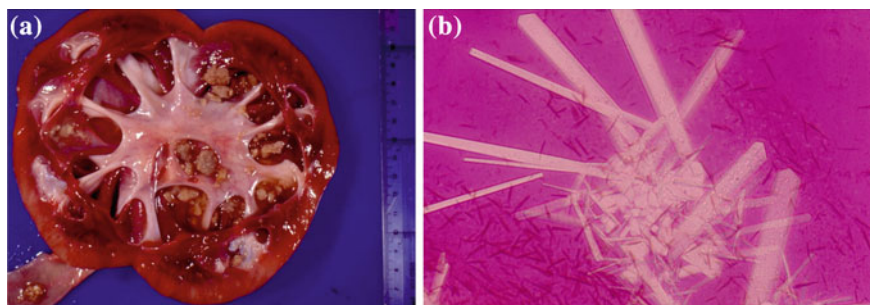
Obstructive urolithiasis is a common disorder in goats (Amarpal Kinjavdekar et al. 2013), as well as in other ruminant species, originated by cristaloid calculi formation which develops from an initial organic matrix (e.g., proteins and cells) followed by mineral deposition in the urinary tract (Osborne et al. 1985). The most frequent uroliths in goats are composed by struvite (ammonium magnesium phosphate) and silicate, followed by apatite (calcium phosphate), calcium carbonate and calcium oxalate (Gutiérrez et al. 2000; George et al. 2007; Makhdoomi and

Gazi 2013), and can be found in renal pelvis, bladder and uretra (Ewoldt et al. 2006) (Fig. 11.3).

The urethral obstruction caused by calculi, which can be partial in small ruminants (Makhdoomi and Gazi 2013), is mainly observed in males due to the long and sinuous trajet of urethra. The urethral obstruction mainly occurs in distal loop of penial sigmoid flexure, but also can be located in proximal loop and urethral process. Crystals can be observed in the prepuce hairs of some males in those farms with urolithiasis problems. Affected goats present anorexia, bloat, pain (false colic), dysuria/stranguria, restlessness and progressive ventral abdominal distention due to urine infiltration, and uremic odor when urethral rupture occurs. The rupture of urethra always needs a surgical correction which intents to open a new via for urine excretion (Van Metre and Fubini 2006).

The etiology of obstructive urolithiasis is multifactorial, even diet assumes a main role, and varies according to the system production, region and season (Nwaokorie et al. 2015), and physiologic/health status of animals. Struvite (pH: 7.2–8.8), calcium carbonate and calcium phosphate (pH: 6.6–7.8), are developed very well under alkaline urine ambience and mineral supersaturation (Jacobs et al. 2001; Straub et al. 2005; Jones et al. 2009). Table 11.1 reports the main factors involved in calculi formation.

The prevention includes the elimination or minimization of risk factors according to the respective calculi type (Ewoldt et al. 2006). The Ca:P ratio should be 2–2.5:1, and Mg should be limited to 0.2–0.6% of DM (phosphatic magnesium-based calculi). Water intake should be increased (3–5% of DM) to promote diuresis and consequently crystals elimination. Acidification of urine at pH 5.5–6.5 range can be achieved using anionic salts (e.g., sodium chloride, calcium chloride or ammonium chloride) provoking dissolution of alkaline uroliths. However, these salts can decrease the feed palatability and, thus, acidificants can present inconsistent efficacy. Jones et al. (2009) suggested a target DCAD of 0 mg/kg in DM. The ammonium chloride can be preventively added to diet at low levels (0.5–1% of DM) to prevent the feed unpalatably.



**Fig. 11.3** Hidronefrosis (a) in a goat died from urinary obstruction (struvite calculi). Urianalysis (b) at microscope ( $\times 40$ ) showing a cluster of struvite crystals in another goat with a disbalanced diet in favor of concentrate (provided by the authors)



**Table 11.1** Main risk factors involved in urolithiasis occurrence in goats and sheep

Factors	Effects	References
Hot and dry climates	Water restriction/metabolism	Sahinduran et al. (2007)
Low water intake	Concentrate urine and decrease diuresis	Stratton-Phelps and House (2004)
Water hardness	Excessive minerals	Medina-Escobedo et al. (2002) and Sahinduran et al. (2007)
Castrated animals	Low diameter of uretra	Kannan and Lawrence (2010)
Urinary pH > 7	Improves apatite, calcium carbonate, and struvite uroliths formation	Hesse and Heimbach (1999)
Urinary pH < 7	Improves silicate uroliths formation	Wagner and Mohebbi (2010)
Fed high grain rations	Improves struvite uroliths formation	Corbera et al. (2007)
Pelleted rations	Reduces salivary excretion of phosphorous (struvite or apatite)	Corbera et al. (2007)
High content of P and Mg in diet	Crystals (struvite and apatite) increase in alkaline urine	Stewart et al. (1990)
Calcium and oxalate-rich forages with low phosphorus and magnesium at grassland	Improves calcium carbonate uroliths	Halland et al. (2002)
Vitamin A deficiency	Urinary tract infection and metaplasia of uroepithelium	Packett and Coburn (1965)
High (phyto)estrogens intake	Uretral mucosa alteration	Jones et al. (2009)
Urinary tract infection	Inflammation process by: <i>Ureaplasma</i> spp. (also increase urine pH); <i>Streptococcus</i> spp.; <i>Escherichia coli</i>	Cornelius et al. (1959) and Livingston et al. (1984)
Urine mucoproteins and proteins with polyelectrolytes	Matrix formation	Packett and Coburn (1965) and Osborne et al. (1985)

Both water intake improvement (sodium chloride) and urine acidification (ammonium chloride) procedures are also used at farm level to treat all animals when alkaline uroliths occur. In unsurveilled urine pH individuals, uretral rupture in fast grown males (high DM intake) are an indicator of urolithiasis problem in the herd. However, Freeman et al. (2010) suggested that urolithogenic crystals by microscopic observation is an easy test to predict uroliths formation.

## 11.5 Polioencephalomalacia

Polioencephalomalacia is a metabolic disorder associated with thiamine deficiency in both adults and young ruminants including goats (Thomas et al. 1987; Lima et al. 2005; Chigerwe and Aleman 2016). Thiamine or vitamin B<sub>1</sub> is a cofactor in the metabolism of carbohydrates necessary for their supply to the neurons in the brain as exclusive source of energy (Kevelam et al. 2013). Consequently, alterations following the death of nerve cells occur promoting a cerebrocortical necrosis, the synonymous denomination of polioencephalomalacia (Gould 1998). However, the specific biochemical mechanisms of these degenerative lesions are not well understood. Interactions between sulfur based metabolism and reduced activity of thiamine pyrophosphate, the active form of thiamine, seem to play a major role (Olkowski 1997; Amat et al. 2013a, b).

Thiamine is synthesized by ruminal bacteria in normal conditions (Alves de Oliveira et al. 1996). Alterations in ruminal fauna reduce the production of thiamine, accelerate the thiamine degradation or inactivation leading to a lack of absorption of this vitamin. Thiamine degradation occurs by Thiaminase I enzymatic catabolism (Kraft et al. 2014). Thiaminase II seems not have a significant role, but it is under reappraisal by some researchers (Murata 1982; Amat et al. 2013b). Thiaminase I can be produced by bacteria (e.g., *Bacillus* spp. and *C. sporogenes*) (Brent and Bartley 1984; Cebra and Cebra 2004), whose population increases in rumen when goats ingest highly digestible carbohydrates and low roughage, provoking subclinical or clinical ruminal acidosis with dysbiosis in ruminal flora and fauna. Thiaminases I (and II) replace the thiazole moiety by organic nucleophiles (Costello et al. 1996; Toms et al. 2005). In dependence of a cofactor (cosubstrates present in ruminal fluid, e.g., proline, pyridoxine and imidazol), thiaminase I produces thiamine analogues which inhibit thiamine dependent reactions. Levamisole and promazines substances seem to act as cofactor under specific ruminal conditions (Cebra and Cebra 2004; Amat et al. 2013a, b). Treatment (Vitamin B<sub>1</sub> and corticosteroids) can be effective in early phases and polioencephalomalacia prevention is related with elimination or control of main causes of non absorption of vitamin B<sub>1</sub>.

## 11.6 Hypomagnesemia

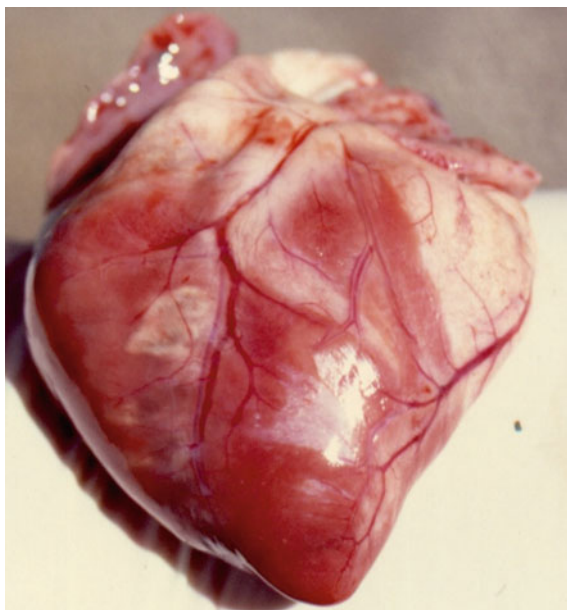
Adult's goats, mainly when grazing growing pastures (less Mg contents), in final Winter/Spring, or when fertilized (high levels of potassium and nitrogen) or also in kids with fast growth fed with milk replacers can develop hypomagnesemia (blood Mg levels <1.1 mg/dL; <0.6 mmol/L). The lack of Mg exchanges neuromuscular transmission and, consequently, the muscle excitability. Goats show mainly periodic tetanic contractions and convulsions, especially when stimulated, leading to death during one of these episodes (Stelletta et al. 2008).

Mg is mainly absorbed in rumen and any cause decreasing persistently its absorption or the high milk production, increase hypomagnesemia incidence. So, this disorder was also called grass tetany, stall tetany, lactation tetany, milk tetany or transport tetany. Prevention is based in (unpalatable) Mg salts (e.g., magnesium oxide) dietary supplementation until 0.6% of DM, mainly during risk periods (Martens and Schweigel 2000; Stelletta et al. 2008).

## 11.7 Selenium and Vitamin E Deficiencies

Selenium and vitamin E deficiencies cause a nutritional/metabolic disease named white muscle disease or nutritional muscular dystrophy, which mainly affects to kids with fast growth in arid and semiarid regions, where soil/forage present low levels of the mineral/vitamin. In goats presenting this disorder, serum values of Se and Vitamin E are under 0.08 mg/L (0.9 mmol/L) and 1.0 mg/L, respectively (Bickhardt et al. 1999; McComb et al. 2010), and are due to low intact of both nutrients. Se and Vitamina E are responsive for antioxidant processes in tissues, limiting muscular skeletal and cardiac dystrophy (Fig. 11.4), and promoting integrity and production of muscle fibers. Kids appears to be more sensitive than lambs to vitamin E deficiency under low SE intake ambiance (Liesegang et al. 2008), and both nutrients improve immune status (Malá et al. 2009; Shokrollahi et al. 2013).

**Fig. 11.4** Macroscopic aspect of a young male kid's heart affected by nutritional muscular dystrophy showing typical pale and discolored areas. The kid suffered sudden death after moderate exercise (provided by the authors)



Se supplementation (combined with iodine) can improve kids' growth and inclusive Se (Aghwan et al. 2016) and NEFA (Aghwan et al. 2014) meat concentration (source for human population), but selenosis risk should be avoided. A positive effect of Se and vitamin E on milk yield and composition in lactating goats has also been reported (Tufarelli and Laudadio 2011). Moreover, Se supplementation seems to improve the fetal development in pregnant goats (Sevcikova et al. 2011) and also affects spermatogens in young bucks (Ganabadi et al. 2010).

A major question regarding Se and Vit. E management in farms would be that the supplementation of these nutrients are more valuable in Se deficient areas, i.e., the response to dietary supplementation is more significant when goats are in effective Se homeostatic unbalancement; and can vitamin E interact with Se more deeply in Se deficiency environment. Some studies supporting this hypothesis has been reported (Sánchez et al. 2007; Liesegang et al. 2008).

Regarding all aspects reported above, these trace elements joint to I vitamin E play an important role in the normal metabolism of kids and goats, not only promote their welfare and resistance to disease, but also can contribute for an adequate production performance.

## 11.8 Production Diseases in High-Producing Dairy Goat

Under pastoralism, the ruminal pH fluid in low-producing dairy goats is usually higher than 6 due to low concentrate/fiber ratio in feed intake. So, subacute ruminal acidosis and low milk fat syndrome are not expected to occur in these goats. However, the increase in milk yield production is because of genetic selection programs that needs to be accomplished with nutrients and energy intakes, not only to maximize lactation potential, but also to minimize lactational ketosis and hepatic lipidosis. This can be achieved improving the density energy in terms of DM, i.e., increasing the concentrate/fiber ratio (1.5; 60% concentrate) in diet.

A major problem of this nutritional management is the decrease of ruminal pH for levels <5.8 for longer than 5–6 h per day, i.e., subacute ruminal acidosis (Jia et al. 2014; Huo et al. 2014), which alters local fauna (Liu et al. 2015), volatile fatty acid production (Huo et al. 2013; Liu et al. 2015), local bacterial digestion with toxic protein formation, e.g., histidine (Hollmann et al. 2013), as well as chemical (pH) inflammation of rumen wall (rumenitis). This process is very well know in dairy cows and is related with ruminal parakeratosis (ruminal *papillae* atrophy), laminitis, liver abscesses and pulmonary bacterial emboli (superior vena cava syndrome), as well as ketosis (in a more advanced phase) and low milk fat syndrome (Kleen et al. 2013; Abdela 2016).

On one hand, diets with high carbohydrates content (energy) are needed to cover lactational requirements, avoiding ketosis and its consequences. On the other hand, these high energetic density diets can provoke disorders previously reported. To achieve a balance of these two sides, several procedures should be taken into account also regarding feed management practices, e.g., grouping goats by

production level, improving fresh diet accessibility, using total mixed ration (minimizing feed selectivity behavior) or pelleted diets (due to goat selectivity for small particle size).

Low milk fat syndrome is classically related with ruminal acidosis and volatile fatty acid profile alteration, i.e., ↑propionate:↓acetate:↓butyrate ratio (Bauman and Griinari 2003; Urrutia and Harvatine 2017). In fact, ruminal acidosis decreases fat and protein milk contents at long-term in goats (Dong et al. 2013). Even, the etiopathophysiology of this syndrome remains poor understandable. Ruminal biohydroxylation of polyunsaturated fatty acids to intermediate fatty acids, e.g., at least trans-10 and Cis-12 conjugated linoleic acids (Ventto et al. 2017), seems to play a significant role. In goats, this conjugated linoleic acids methyl esters seems to be dose-effect dependent in reduction of milk fat secretion (Fernandes et al. 2014) but improving energy balance (Baldin et al. 2014). However, probably the regulation of fat synthesis in mammary gland is also involved in the low milk fat syndrome occurrence (Suárez-Vega et al. 2017; Ventto et al. 2017).

No effective prevention has been found until today to limit conjugated linoleic acids methyl esters produced in rumen, keeping the production levels. Best practices for nutritional, feed and health managements evaluating feed intakes, milk yield and contents, BCS, metabolic profiles, ruminal and urinary pH and the health status of individuals are strongly recommended.

## 11.9 Concluding Remarks

Nutritional and metabolic disorders are closely related modifying the homeostasis with negative impact in production, reproduction, health and welfare of goats. These animals are exceptional selective browsers/grazers, which allow the best use of biomass and nutrients intake in harsh environments. In these conditions, the plants diversity seems to be the best option to minimize the potential lack of energy, mineral and vitamins. As prevention of nutritional deficiencies, diet should be supplemented regarding epidemiology disorders in each region of the world.

Goat genetic improvement for milk yield and composition need to be complemented by nutritional and feed management programs. Similar to dairy cows, but with several particularities mainly related with feeding behavior, metabolism and diseases' ethiopathophysiology, goats present several disorders called "production diseases". Even a major factor can be originally involved, the interrelationship of several body systems, which can be affected by several risk factors. Consequently, a holistic approach is needed to control or even prevent nutritional and metabolic disorders.

## References

- Abdela N (2016) Sub-acute ruminal acidosis (SARA) and its consequence in dairy cattle: a review of past and recent research at global prospective. *Achievem Life Sci* 10(2):187–196
- Aghwan ZA, Alimon AR, Goh YM et al (2014) Fatty acid profiles of supraspinatus, longissimus lumborum and semitendinosus muscles and serum in Kacang goats supplemented with inorganic selenium and iodine. *Asian-Australas J Anim Sci* 27(4):543–550
- Aghwan ZA, Sazili AQ, Kadhim KK et al (2016) Effects of dietary supplementation of selenium and iodine on growth performance, carcass characteristics and histology of thyroid gland in goats. *Anim Sci* 87(5):69–690
- Albay MK, Karakurum MC, Sahinduran S et al (2014) Selected serum biochemical parameters and acute phase protein levels in a herd of Saanen goats showing signs of pregnancy toxemia. *Vet Med* 59(7):336–342
- Alves de Oliveira L, Jean-Blain C, Durix A et al (1996) Use of a semi-continuous culture system (RUSITEC) to study the effect of pH on microbial metabolism of thiamin (Vitamin B1). *Arch Tierernahr* 49(3):193–202
- Amarpal Kinjavdekar P, Aithal HP, Pawde AM et al (2013) A retrospective study on the prevalence of obstructive urolithiasis in domestic animals during a period of 10 years. *Adv Anim Vet Sci* 1(3):88–92
- Amat S, McKinnon JJ, Olkowski AA et al (2013a) Understanding the role of sulfur-thiamine interaction in the pathogenesis of sulfur-induced polioencephalomalacia in beef cattle. *Res Vet Sci* 95(3):1081–1087
- Amat S, Olkowski AA, Atila M et al (2013b) A review of polioencephalomalacia in ruminants: is the development of malacic lesions associated with excess sulfur intake independent of thiamine deficiency? *Vet Med Anim Sci* 1(1):1. <https://doi.org/10.7243/2054-3425-1-1>
- Andrews A (1997) Pregnancy toxemia in the ewe. *Practice* 19(6):306–314
- Baciadonna L, McElligott AG, Briefe EF (2013) Goats favour personal over social information in an experimental foraging task. *PeerJ* 1:e172. <https://doi.org/10.7717/peerj.172>
- Baldin M, Dresch R, Souza J et al (2014) CLA induced milk fat depression reduced dry matter intake and improved energy balance in dairy goats. *Small Rumin Res* 116(1):44–50
- Barłowska J, Sz wajowska M, Litwinczuk Z et al (2011) Nutritional value and technological suitability of milk from various animal species used for dairy production. *Compr Rev Food Sci Food Saf* 10(6):291–302
- Barroso FG, Alados CL, Boza J (2000) Social hierarchy in the domestic goat: effect on food habits and production. *Appl Anim Behav Sci* 69(1):35–53
- Bauman DE, Griinari JM (2003) Nutritional regulation of milk fat synthesis. *Annu Rev Nutr* 23:203–227
- Bickhardt K, Ganter M, Sallmann P et al (1999) Investigations on manifestations of vitamin E and selenium deficiency in sheep and goats. *Dtsch Tierarztl Wochenschr* 106(6):242–247
- Bobe G, Young JW, Beitz DC (2004) Invited review: pathology, etiology, prevention, and treatment of fatty liver in dairy cows. *J Dairy Sci* 87(10):3105–3124
- Bousquet CA (2005) Pathologie caprine en deux-sèvres: état des lieux et impact sur les niveaux de réforme et de mortalité. Ph.D. thesis, University of Paul-Sabatier de Toulouse, Toulouse, France
- Brent BE, Bartley EE (1984) Thiamin and niacin in the rumen. *J Anim Sci* 59:813–822
- Brozos C, Mavrogianni VS, Fthenakis GC (2011) Treatment and control of periparturient metabolic diseases: pregnancy toxemia, hypocalcemia, hypomagnesemia. *Vet Clin North Am Food Anim Pract* 27(1):105–113
- Cebra CK, Cebra ML (2004) Altered mentation caused by polioencephalomalacia, hypernatremia, and lead poisoning. *Vet Clin North Am Food Anim Pract* 20:287–302
- Chartier C (2009) Pathologie caprine: du diagnostic à la prévention. *Les Éd. du Point Vétérinaire, Rueil-Malmaison, France*

- Chigerwe M, Aleman M (2016) Seizure disorders in goats and sheep. *Vet Intern Med* 30(5):1752–1757
- Corbera JA, Morales M, Doreste F et al (2007) Experimental struvite urolithiasis in goats. *J Appl Anim Res* 32:191–194
- Cornelius CE, Moulton JE, McGowan B (1959) Ruminant urolithiasis: I. Preliminary observations in experimental ovine calculosis. *Am J Vet Res* 20:863–871
- Costello CA, Kelleher NL, Abe M et al (1996) Mechanistic studies on thiaminase I. Overexpression and identification of the active site nucleophile. *J Biol Chem* 271:3445–3452
- DeGaris PJ, Lean IJ (2008) Milk fever in dairy cows: a review of pathophysiology and control principles. *Vet J* 176:58–69
- Dong H, Wang S, Jia Y et al (2013) Long-term effects of subacute ruminal acidosis (SARA) on milk quality and hepatic gene expression in lactating goats fed a high-concentrate diet. *PLoS ONE* 8(12):e82850. <https://doi.org/10.1371/journal.pone.0082850>
- Doré V, Dubuc J, Bélanger AM et al (2015) Definition of prepartum hyperketonemia in dairy goats. *J Dairy Sci* 98(7):4535–4543
- Edmondson MA, Roberts JF, Baird AN et al (2012) Theriogenology of sheep and goats. In: Pugh DG, Baird AN (eds) *Sheep and goat medicine*, 2nd edn. Elsevier-Saunders, Maryland Heights, MO, pp 150–231
- Ermilio EM, Smith MC (2011) Treatment of emergency conditions in sheep and goats. *Vet Clin North Am Food Anim Pract* 27:33–45
- Ewoldt JM, Anderson DE, Miesner MD et al (2006) Short- and long-term outcome and factors predicting survival after surgical tube cystostomy for treatment of obstructive urolithiasis in small ruminants. *Vet Surg* 35(5):417–422
- Fernandes D, Gama MA, Ribeiro CV et al (2014) Milk fat depression and energy balance in stall-fed dairy goats supplemented with increasing doses of conjugated linoleic acid methyl esters. *Animal* 8(4):587–595
- Freeman SR, Poore MH, Young GA et al (2010) Influence of calcium (0.6 or 1.2%) and phosphorus (0.3 or 0.6%) content and ratio on the formation of urolithogenic compounds in the urine of Boer-cross goats fed high-concentrate diets. *Small Rum Res* 93(2):94–102
- Ganabadi S Jr, Halimatun Y, Amelia Choong KL et al (2010) Effect of selenium supplementation on spermatogenic cells of goats. *Malays J Nutr* 16(1):187–193
- George JW, Hird DW, George LW (2007) Serum biochemical abnormalities in goats with uroliths: 107 cases (1992–2003). *J Am Vet Med Assoc* 230(1):101–106
- Goff JP, Liesegang A, Horst RL (2014) Diet-induced pseudohypoparathyroidism: a hypocalcemia and milk fever risk factor. *J Dairy Sci* 97(3):1520–1528
- González FHD, Hernandez F, Madrid J et al (2011) Acute phase proteins in experimentally induced pregnancy toxemia in goats. *J Vet Diagn Invest* 23(1):57–62
- Gould DH (1998) Polioencephalomalacia. *J Anim Sci* 76(1):309–314
- Gutiérrez C, Escolar E, Juste MC et al (2000) Severe urolithiasis due to trimagnesium orthophosphate calculi in a goat. *Vet Rec* 146(18):534
- Halland SK, House JK, George LW (2002) Urethroscopy and laser lithotripsy for the diagnosis and treatment of obstructive urolithiasis in goats and pot-bellied pigs. *J Am Vet Med Assoc* 220:1831–1834
- Härter CJ, Castagnino DS, Rivera AR et al (2015) Mineral metabolism in singleton and twin-pregnant dairy goats. *Asian-Aust J Anim Sci* 28(1):37–49
- Härter CJ, Lima LD, Castagnino DS et al (2017) Net mineral requirements of dairy goats during pregnancy. *Animal* 13:1–9
- Hefnawy AE, Youssef S, Shousha S (2010) Some immunohormonal changes in experimentally pregnant toxemic goats. *Vet Med Int* 2010:768438. <https://doi.org/10.4061/2010/768438>
- Hefnawy AE, Shousha S, Youssef S (2011) Hematobiochemical profile of pregnant and experimentally pregnancy toxemic goats. *J Basic Appl Chem* 1(8):65–69
- Heitmann RN, Dawes DJ, Sensenig SC (1987) Hepatic ketogenesis and peripheral ketone body utilization in the ruminant. *J Nutr* 117(6):1174–1180
- Herdth TH (1988) Fatty liver in dairy cows. *Vet Clin North Am Food Anim Pract* 4(2):269–287

- Herm G, Muscher-Banse AS, Breves G et al (2015) Renal mechanisms of calcium homeostasis in sheep and goats. *J Anim Sci* 93(4):1608–1621
- Hesse A, Heimbach D (1999) Causes of phosphate stone formation and the importance of metaphylaxis by urinary acidification: a review. *World J Urol* 17(5):308–315
- Hollmann M, Miller I, Hummel K et al (2013) Downregulation of cellular protective factors of rumen epithelium in goats fed high energy diet. *PLoS ONE* 8(12):e81602. <https://doi.org/10.1371/journal.pone.0081602>
- Huo W, Zhu W, Mao S (2013) Effects of feeding increasing proportions of corn grain on concentration of lipopolysaccharide in the rumen fluid and the subsequent alterations in immune responses in goats. *Asian-Aust J Anim Sci* 26(10):1437–1445
- Huo W, Zhu W, Mao S (2014) Impact of subacute ruminal acidosis on the diversity of liquid and solid-associated bacteria in the rumen of goats. *World J Microbiol Biotechnol* 30(2):669–680
- Ismail ZAB, Al-Majali AM, AMireh F et al (2008) Metabolic profiles in goat does in late pregnancy with and without subclinical pregnancy toxemia. *Vet Clin Pathol* (37)4:434–437
- Jacobs D, Heimbach D, Hesse A (2001) Chemolysis of struvite stones by acidification of artificial urine. *Scand J Urol Nephrol* 35:345–349
- Jia YY, Wang SQ, Ni YD et al (2014) High concentrate-induced subacute ruminal acidosis (SARA) increases plasma acute phase proteins (APPs) and cortisol in goats. *Animal* 8(9):1433–1438
- Johnson EH, Al-Habsi K, Kaplan E et al (2004) Caprine hepatic lipidosis induced through the intake of low levels of dietary cobalt. *Vet J* 168(2):174–179
- Jones ML, Streeter RN, Goad CL (2009) Use of dietary cation anion difference for control of urolithiasis risk factors in goats. *Am J Vet Res* 70(1):149–155
- Kaneko J, Harvey JW, Bruss ML (1997) *Clinical biochemistry of domestic animals*, 5th edn. Academic Press, New York, USA
- Kannan KVA, Lawrence KE (2010) Obstructive urolithiasis in a Saanen goat in New Zealand, resulting in a ruptured bladder. *N Z Vet J* 58(5):269–271
- Kevelam SH, Bugiani M, Salomons GS et al (2013) Exome sequencing reveals mutated SLC19A3 in patients with an early-infantile, lethal encephalopathy. *Brain* 136:1534–1543
- Kleen JL, Uppang L, Rehage J (2013) Prevalence and consequences of subacute ruminal acidosis in German dairy herds. *Acta Vet Scand* 55(1):48
- Kleppe BB, Aiello RJ, Grummer RR et al (1988) Triglyceride accumulation and very low-density lipoprotein secretion by rat and goat hepatocytes in vitro. *J Dairy Sci* 71:1813–1822
- Kraft CE, Gordon ERL, Angert ER (2014) A rapid method for assaying thiaminase I activity in diverse biological samples. *PLoS ONE* 9(3):e92688. <https://doi.org/10.1371/journal.pone.0092688>
- Liesegang A (2008) Influence of anionic salts on bone metabolism in periparturient dairy goats and sheep. *J Dairy Sci* 91(6):2449–2460
- Liesegang A, Risteli J, Wanner M (2006) The effects of first gestation and lactation on bone metabolism in dairy goats and milk sheep. *Bone* 38(6):794–802
- Liesegang A, Staub T, Wichert B et al (2008) Effect of vitamin E supplementation of sheep and goats fed diets supplemented with polyunsaturated fatty acids and low in Se. *J Anim Physiol Anim Nutr (Berl)* 92(3):292–302
- Lima EF, Riet-Correa FT, Ivon M (2005) Polioencephalomalacia in goats and sheep in the semiarid region of north-eastern Brazil. *Pesq Vet Bras* 25:9–14
- Lima MS, Pascoal RA, Stilwell GT et al (2012) Clinical findings, blood chemistry values, and epidemiologic data obtained from dairy goats with pregnancy toxemia (PT). *Bov Pract* 46(2):102–110
- Lima MS, Silveira JM, Carolino N et al (2016) Usefulness of clinical observations and blood chemistry values for predicting clinical outcomes in dairy goats with pregnancy toxemia. *Ir Vet J* 69:16. <https://doi.org/10.1186/s13620-016-0075-4>
- Liu J-H, Bian G-R, Zhu W-Y et al (2015) High-grain feeding causes strong shifts in ruminal epithelial bacterial community and expression of Toll-like receptor genes in goats. *Front Microbiol* 6:167. <https://doi.org/10.3389/fmicb.2015.00167>



- Livingston CW, Calhoun MC, Gauer BB et al (1984) Effect of experimental infection with ovine ureaplasma upon the development of uroliths in feedlot lambs. *Israel J Med Sci* 20:958–961
- Makhdoomi DM, Gazi MA (2013) Obstructive urolithiasis in ruminants—a review. *Vet World* 6(4):233–238
- Malá S, Kovář F, Misurová L et al (2009) Influence of selenium on innate immune response in kids. *Folia Microbiol (Praha)* 54(6):545–548
- Martens H, Schweigel M (2000) Pathophysiology of grass tetany and other hypomagnesemias. Implications for clinical management. *Vet Clin North Am Food Anim Pract* 16(2):339–368
- McComb T, Bischoff K, Thompson B et al (2010) An investigation of blood selenium concentrations of goats in New York State. *J Vet Diagn Invest* 22(5):696–701
- Medina-Escobedo M, Zaidi M, Real-de Leon E et al (2002) Prevalence and risk factors of urinary lithiasis in Yucatan, Mexico. *Salud Pública de México* 44(6):541–545
- Mellado M (2016) Dietary selection by goats and the implications for range management in the Chihuahuan desert: a review. *Rangeland J* 38(4):331–341
- Morand-Fehr P (2003) Dietary choices of goats at the trough. *Small Rumin Res* 49(3):231–239
- Morand-Fehr P (2005) Recent developments in goat nutrition and application: a review. *Small Rumin Res* 60(1–2):25–43
- Murata K (1982) Actions of two types of thiaminase on thiamin and its analogues. *Ann N Y Acad Sci* 378:146–156
- Nwaokorie EE, Osborne CA, Lulich JP et al (2015) Risk factors for calcium carbonate urolithiasis in goats. *J Am Vet Med Assoc* 247(3):293–299
- Oetzel GR (1988) Parturient paresis and hypocalcemia in ruminant livestock. *Vet Clin North Am Food Anim Pract* 4:351–364
- Olkowski AA (1997) Neurotoxicity and secondary metabolic problems associated with low to moderate levels of exposure to excess dietary sulphur in ruminants: a review. *Vet Hum Toxicol* 39:355–360
- Osborne CA, Polzin DJ, Abdullahi SU et al (1985) Struvite urolithiasis in animals and man: formation, detection and dissolution. *Adv Vet Sci Comp Med* 29:1–45
- Packett LV, Coburn SP (1965) Urine proteins in nutritionally induced ovine urolithiasis. *Am J Vet Res* 26(10):112–119
- Pichler M, Damberger A, Arnholdt T et al (2014) Evaluation of two electronic handheld devices for diagnosis of ketonemia and glycemia in dairy goats. *J Dairy Sci* 97:7538–7546
- Radostits OM, Gay CC, Hinchcliff KW et al (2007) Pregnancy toxemia in sheep. In: Radostits OM, Gay CC, Hinchcliff KW et al (eds) *Veterinary medicine: a textbook of the diseases of cattle, sheep, pigs, goats and horses*, 10th edn, Saunders Elsevier USA, Philadelphia, pp 1668–1671
- Rook JS (2000) Pregnancy toxemia of ewes, does, and beef cows. *Vet Clin North Am Food Anim Pract* 16(2):293–317
- Sadjadian R, Seifi HA, Mohri M et al (2013) Variations of energy biochemical metabolites in periparturient dairy Saanen goats. *Comp Clin Pathol* 22:449–456
- Sahinduran S, Buyukoglu T, Gulay MS et al (2007) Increased water hardness and magnesium levels may increase occurrence of urolithiasis in cows from the Burdur region (Turkey). *Vet Res Commun* 31(6):665–671
- Sánchez J, Montes P, Jiménez A et al (2007) Prevention of clinical mastitis with barium selenate in dairy goats from a selenium-deficient area. *J Dairy Sci* 90(5):2350–2354
- Sensenig SC, Dawes DJ, Heitmann RN (1985) Energy metabolite concentrations and net fluxes across splanchnic and peripheral tissues in pregnant ewes. *J Anim Sci* 61(Suppl. 1):454
- Sevcikova L, Pechova A, Pavlata L et al (2011) The effect of various forms of selenium supplied to pregnant goats on the levels of selenium in the body of their kids at the time of weaning. *Biol Trace Elem Res* 143(2):882–892
- Shokrollahi B, Mansouri M, Amanlou H (2013) The effect of enriched milk with selenium and vitamin E on growth rate, hematology, some blood biochemical factors, and immunoglobulins of newborn goat kids. *Biol Trace Elem Res* 153(1–3):184–190

- Stelletta C, Giancesella M, Morgante M (2008) Metabolic and nutritional diseases. In: Cannas A, Pulina G (eds) Dairy goats feeding and nutrition, CAB International, Bologna, Italy
- Stewart SR, Emerick RJ, Pritchard RH (1990) High dietary calcium to phosphorus ratio and alkali-forming potential as factors promoting silica urolithiasis in sheep. *J Anim Sci* 68: 498–503
- Stratton-Phelps M, House JK (2004) Effect of a commercial anion dietary supplement on acid–base balance, urine volume, and urinary ion excretion in male goats fed oat or grass hay diets. *Am J Vet Res* 65:1391–1397. Erratum in: *Am J Vet Res* 65:1700
- Straub M, Hautmann RE, Hesse A et al (2005) Calcium oxalate stones and hyperoxaluria. What is certain? What is new? *Der Urologe* 44(11):1315–1323
- Sun F, Cao Y, Yu C et al (2016) 1,25-dihydroxyvitamin D3 modulates calcium transport in goat mammary epithelial cells in a dose- and energy-dependent manner. *J Anim Sci Biotechnol* 7:41. <https://doi.org/10.1186/s40104-016-0101-0>
- Suárez-Vega A, Toral PG, Gutiérrez-Gil B et al (2017) Elucidating fish oil-induced milk fat depression in dairy sheep: milk somatic cell transcriptome analysis. *Sci Rep* 7:45905. <https://doi.org/10.1038/srep45905>
- Thomas KW, Turner DL, Spicer EM (1987) Thiamine, thiaminase and transketolase levels in goats with and without polioencephalomalacia. *Aust Vet J* 64:126–127
- Toms AV, Haas AL, Park JH et al (2005) Structural characterization of the regulatory proteins TenA and TenI from *Bacillus subtilis* and identification of TenA as a thiaminase II. *Biochemistry* 44:2319–2329
- Trevisi E, D’Angelo A, Gaviraghi A et al (2005) Blood inflammatory indices in goats around kidding. *Ital J Anim Sci* 4(Suppl. 2):404–405
- Tufarelli V, Laudadio V (2011) Dietary supplementation with selenium and vitamin E improves milk yield, composition and rheological properties of dairy Jonica goats. *J Dairy Res* 78 (2):144–148
- Urrutia NL, Harvatine KJ (2017) Acetate dose-dependently stimulates milk fat synthesis in lactating dairy cows. *J Nutr* 147(5):763–769
- Van Metre DC, Fubini SL (2006) Ovine and caprine urolithiasis: another piece of the puzzle. *Vet Surg* 35(5):413–416
- Ventto L, Leskinen H, Kairenius P et al (2017) Diet-induced milk fat depression is associated with alterations in ruminal biohydrogenation pathways and formation of novel fatty acid intermediates in lactating cows. *Br J Nutr* 117(3):364–376
- Vernon RG, Clegg RA, Flint DJ (1981) Metabolism of sheep adipose tissue during pregnancy and lactation. Adaptation and regulation. *Biochem J* 200(2):307–314
- Wagner CA, Mohebbi N (2010) Urinary pH and stone formation. *J Nephrol* 23(Suppl. 16):165–169
- Wilkens MR, Richter J, Fraser DR et al (2012) In contrast to sheep, goats adapt to dietary calcium restriction by increasing intestinal absorption of calcium. *Comp Biochem Physiol A Mol Integr Physiol* 163:396–406
- Wilkens MR, Liesegang A, Richter J et al (2014) Differences in peripartal plasma parameters related to calcium homeostasis of dairy sheep and goats in comparison with cows. *J Dairy Res* 81(3):325–332
- Yamagishi N, Oishi A, Sato J, Sato R, Naito Y (1999) Experimental hypocalcemia induced by hemodialysis in goats. *J Vet Med Sci* 61:1271–1275
- Zobel G, Leslie K, Weary DM et al (2015) Ketonemia in dairy goats: effect of dry period length and effect on lying behavior. *J Dairy Sci* 98(9):6128–6138