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Nutritional Bases to Control Gastrointestinal Parasites of Livestock

AK Pathak*

[1]*Sher-e-Kashmir University of Agricultural Sciences and Technology, India*

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***Corresponding author:** AK Pathak, Division of Animal Nutrition, Faculty of Veterinary Sciences & Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology, R.S. Pura, Jammu-181 102, India, Email: dranand_pathak@yahoo.com

Abstract

This review article provides a brief description of the nutritional bases to control gastrointestinal (GI) parasites in livestock and maintained/ improved their production potential by nutritional intervention. High prevalence of GI parasites, resistance against synthetic drugs, high prices, and scarcity of good quality feedstuffs may pose the disease problem with great economic losses worldwide. The GI parasites infestation may impose progressive decline in feed intake and impaired efficient nutrient utilization, loss in body weight, diarrhea, malnutrition, sometimes tissue damage which leads to blood loss (anemia), various nutrient deficiency and sometimes death may occur. Due to infection stress, nutrient intake often compromised and available nutrients were diverted away from production purposes to a high survival priority i.e. for tissue wear and tear/ repair, synthesis of immunoglobulin's and production of immune cells. Furthermore, reduced weight gain and poor production of affected animals were the results of nutrient turnover, ultimately lead to impairment of nutrients which determine the quantum of infection, host immune response, pathogenesis and significant economic losses. Synergistic interrelationship exist between host, nutrient levels and GI parasites by providing high quality protected nutrients in balanced proportion through dietary supplementation along with fungus and some naturally occurring plants secondary metabolites especially condensed tannins (CT) having anti-parasitic properties without any side effects. Thus, nutritional intervention and strategic CT supplementation may be an effective, socioeconomic, environment friendly GI parasites control strategies.

Keywords: GI parasites control; Livestock; Nutritional bases; Performance

Introduction

Livestock are a vitally important agricultural commodity in developing countries of the world. They are reared under a wide variety of production systems ranging from large-scale intensive commercial animal farming to traditional small-holder and village production systems [1-2]. Most of the developing countries of the world lie within the limits of the production constraints caused by gastrointestinal (GI) parasites, namely the tropical and subtropical regions of the world [3]. The high prevalence of GI parasites, development of resistance against synthetic drugs (anthelmintics), high prices of medicines and scarcity of good quality feedstuffs for animals continues to pose the greatest disease problem along with great economic losses worldwide.

The GI parasites have two main features of epidemiological importance: there is no multiplication of their pre-parasitic stages and their infective stages, whether acquired directly by the host or indirectly via intermediate hosts, always give rise to a single adult male or female parasite in the definitive host [4,5]. Therefore, the number of infective stages present in the host environment at any given period is related to the number of worm eggs passed by the host and this largely determines the number of parasites potentially capable of being established in a susceptible host [4]. Heavy infestation with GI parasites may impose progressive decline in feed intake, impaired nutrient utilization, loss in body weight, diarrhea, malnutrition, tissue damage and blood loss (anaemia) and sometimes death may occur.

During infection stress, nutrient intake often compromised and available nutrients diverted away from production purposes to a high survival priority i.e. for gastrointestinal tissue repair, synthesis of plasma proteins (immunoglobulin's) and production of immune cells (macrophages and granulocytes). Moreover, reduced weight gain and poor production performance of affected animals were the results of nutrient turn over because it was obvious that GI parasitic infestation will lead to impairment of various nutrients which determine the quantum

of infection, resistance of host against disease i.e. immune status, pathogenesis, severity and significant economic losses. However, characteristics such as breed, age and nutritional status of the host as well as characteristics of the environment also have a considerable influence on the parasites and their capacity to infect and inflict damage to the host [5-10].

Hence, repeated infections are apparent in livestock throughout life. However, that viewpoint has changed and it is clear that a very complicated immune response occurs against GI parasites due to the complexity of the life cycle. In order to survive, GI parasites need to remain within the host for a relatively long period of time to ensure patency (egg production) and a balance between worm and host is maintained unless environmental conditions (population density, nutrition or climate) upset it. In general, GI parasites exist throughout the year in tropical environments as the presence of moisture and high ambient temperatures are necessary for their life cycle to proceed. For this reason parasitism in livestock is regarded as the most important animal health disease.

Synergistic interrelationship exists between dietary nutrient levels and GI parasitic loads, intensity in infection and disease occurrence, etc. This can be achieved by providing high quality/ high plane/ protected nutrients in balanced proportion through dietary supplementation along with some naturally occurring plants secondary metabolites especially condensed tannins (CT) having anti-parasitic properties without any side effects. These can be supported by previous studies by various workers that animal receiving low nutrient diet suffered more severe clinical disease than those fed on high nutrient diet. The GI parasite infected animals fed CT supplemented diet showed lower parasitic load, less severity of infection and maintained health and production performance compared to control (without CT) diet. Thus nutritional intervention and strategic CT supplementation minimizes the magnitude and duration of GI parasitic disease stress which was almost inevitable as a mechanism of animal survival in organic environment [11-13].

Production performance of GI parasites infected animals

The GI parasites decrease production (body weight gain, milk, meat, wool production and reproductive performance) through depression of feed intake, poor utilization of feed, blood and protein loss as well as using extra energy [3,5]. It is also important in respect of development of resistant strain of GI parasites to broad spectrum anthelmintics [11-13]. Also, the society's environmental concerns and the increasing market demand for products that are free of chemicals, have led to the search for alternative methods of parasite control [14-18]. It is perhaps more challenges for livestock production because of the more reliant on the pastures and banning the use of any chemical drugs [19]. Grazing management combined with nutritional supplementation with balanced concentrate mixture, tanniferous tree leaves, leaf meal mixture, condensed tannins fortified multi nutrient blocks [20-22] complete feed blocks [23] and/or tanniferous forage was the most frequently reported anti-helminthes strategy in livestock production [11-12,19].

As a means of reducing anthelmintic use, nutritional manipulation has been employed to enhance resilience to parasitism and maintain productive performance in livestock which is frequently lost during subclinical infections. Well-nourished animals are known to withstand the effects of worm infection much better than those given a lower plane of nutrition. Meanwhile, the resistance of animals to establishment of worm larvae can be enhanced by improved nutrition [7-12].

Nutrition is one of the biggest expenses for the animal keepers, either directly or indirectly. Most of the direct expenses arise from the purchase of supplementary feeds. Of these, protein is especially important since it forms a critical element in animal nutrition: it affect ovulation rate, embryo survival, foetal growth, colostrums and milk production, body mass, young animal survival, and growth as well as wool growth [3,5]. Several studies have shown that balanced nutrition also affects the animal's resilience and resistance to GI parasites, which can be a major constraint to production. One alternative means of controlling GI parasites in these livestock is by manipulation of their diet [7].

Nutrition-GI parasites-Host-Interrelationship

The nutritional status of the host has long been considered as an important factor in influencing the host-parasite relationship and the pathogenesis of parasite infections. While the mechanisms underlying this fact are not clear, energy balance and supply is increasingly considered to be an important factor in the immune response. Indeed, recent reports have shown that an improvement in energy supply improved resistance to helminthes infection of growing animals [8]. This nutrition-parasite interaction and the fact that long-term effect of Duddingtonia flagrans on levels of infection in animals could be affected by a differential immune response, led us to explore to what extent an enhancement in the animals immune response through energy supplementation could improve the fungus efficacy as a practical biological control agent of parasitic nematodes in pastured ruminants [24].

Infections with GI parasites in livestock do not necessarily result in disease. However, heavy infections result in clinical parasitic gastroenteritis and this consequently leads to in appetence, impaired gastrointestinal function and changes in protein, energy, mineral, vitamin metabolism and water balance [5-8]. Clinical parasitic gastroenteritis is characterized by watery diarrhea, weight loss or reduced weight gain, dull hair coat, anorexia and a general loss of condition. Subclinical infections without obvious clinical signs constitute the majority of infections and cause economic losses due to sub-optimal performance [9-10].

Nutrition is an extremely important predisposing factor, and there is a long history of research into animal nutrition and

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Veterinary parasitological. Some researchers pointed out that even under normal seasonal conditions, animal over a large part of world may be subjected to a seasonal nutritional stress comparable to that found sufficient to break even previously acquired resistance to helminthiasis. Knox and Steel [25] reported that resilience to GI parasites, or the ability to withstand the effects of infection, can be enhanced by increased dietary intake of metabolizable protein and metabolizable energy.

Host immune response

The immune response of animals is not the same in a herd where most of the animals harbor a small proportion of the worms. Timing the immune response to the stage of infection and enriching the intestine with polyunsaturated fatty acids in the beginning of life may promote a good immune response against GI parasites; providing the protected fat in the creep-feeding of lambs in the pre-weaning stage may yield better results. The expression of immunity against GI parasites, which makes the animals resistant or resilient, can vary widely among animals of the same flock. For optimal physiological/immunological function, the quality of the diet, i.e. the chemical composition relative to the animal's requirements, and probably the ratio of nutrients, are more important than the quantity.

The amount of nutrients needed for immune-modulation in animals is higher than the required amounts suggested by the NRC for production. Resistance, or the ability of the host to prevent or eliminate infection, can also be improved by altering host nutrition, particularly increasing protein intake [7-8]. They also observed that this was an effect on acquired immunity, in particular on the rate of acquisition of immunity, rather than on initial worm establishment. However, the precise nutrients required and the mechanisms involved have not been satisfactorily identified. In the case of protein supplementation there are reports of associated changes in cellular responses to T. colubriformis and O. columbianum (eosinophils, intestinal lymphocytes and mast cells and mesenteric efferent lymph WC1+T cells) but not worm-specific serum antibody concentration.

In addition to protein and energy, dietary deficiencies or variables inhibiting the utilization of minerals and vitamins can also limit the ability of the immune system to deal with parasites. Trace elements and vitamins are components of enzymes or co-factors and therefore have pivotal roles in biochemical reactions that can have widespread repercussions in animal physiology. Changes in metabolic activity, e.g. increased cell proliferation, can induce clinical signs of mineral and vitamin deficiency in animals with sub-clinical deficiency. Cells with a short half-life such as lymphocytes are particularly sensitive to trace element deficiency. Theoretically therefore, given the range of systems involved in gut immunity to gastrointestinal parasites, deficiencies of most of the trace elements and vitamins could affect the development of protective immunity to worms.

Mucosal immunity, particularly during their development, come at some cost to the host in terms of cell and protein loss, reduced nutrient absorption and increased metabolic demand for nutrients, a cost exacerbated by the damage to gastrointestinal function due directly to the worm. The nutritional requirements for optimal mucosal immunity appear to be greater than currently recognised, and additional to those required to meet the demands of growth and production [3, 26]. The efficacy of the mucosal immune responses can also be enhanced by altering the local immunological environment, for example the degree of inflammation.

Thus gut immunity is not an isolated component of ruminant physiology, but highly integrated with the nervous, digestive and endocrine systems, and under-laid by a common cell biology regulating signaling, activation, metabolism, replication and differentiation. In such a situation, intervention which is directed towards addressing any single factor contributing to susceptibility will predictably have limited prospects of success. It is not clear what immune mechanisms are affected by protein supplementation although the function of certain lymphocyte populations may be crucial in young lambs, and circulating eosinophils and intestinal mast cell protease concentrations may be elevated due to dietary protein in older lambs [26,27]. Until recently it was thought that no effective immunity was generated by infection with GI parasites; that worms living in the gut were effectively outside the body and could neither initiate nor be affected by the immune system.

Nutritional Manipulation to control GI parasites

Protein supplementation: Supplementation of the diet of livestock would lead to better control of GI parasites. In general, animal need to ingest the infective larvae of GI parasites, and therefore be exposed to the developmental stages, for up to 12 weeks to develop resistance. However, this period is dependent on genetics, age and nutrition. Some research studies reported that the young lambs were better able to resist the establishment of an artificial parasite infection when their diet had been supplemented with meat and bone, and soya bean meal (20% crude protein; CP of diet), as compared to 10% CP supplemented with equivalent amounts of energy [26]. They consequently grew faster.

Many research studies have reported that protein supplementation either in the form of by-pass protein or higher dietary protein improves resilience and expression of immunity to GI parasites. Protein supplementation in the form of rumen un-degradable protein has been shown to increase the resistance of sheep to Haemonchus contortus [27,28]. When animals, which were infected with gastrointestinal nematodes (GINs) such as T. colubriformis, were fed with increased amount of rumen un-degradable protein in the form of fish meal, animals decreased less body weight than those animals that were not fed the increased level of rumen un-degradable protein. Some workers

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also reported that animals parasitized with T. colubriformis and fed with 22% protein diet achieved growth rates similar to those uninfected low protein diets as evidenced by reduced faecal egg counts.

Improved host nutrition primarily by-pass protein increases the rate of rejection of adult parasites without affecting the rate of establishment of infective larvae [26]. However, the potential of metabolisable protein to enhance resistance to helminthes infection is dependent on the requirement relative to its supply in the diet and demand for other competing physiological functions. Genetic resistance to helminthes to animals is only expressed in the presence of improved metabolizable protein supply [28].

In experiments where lambs were infused with casein into the abomasums in order to reduce the variability of protein source, resistance to T. colubriformis and O. circumcincta increased. When lambs were fed up to 20% CP as rumen bypass protein (fish meal), in an attempt to increase the amount of utilizable protein, resistance was enhanced [29]. For this reason protein supplements containing fish products have an inherent advantage over other sources. Researchers had shown that when parasitized lambs on a protein deficient diet were supplemented with methionine or a methionine-lysine mixture, they were more resilient (tolerant) and resistant to infection with T. colubriformis. Thus, supplementation with fish meal may be correcting for the deficiencies of specific amino acids. Dietary incorporation of urea molasses mineral blocks can reduce parasitic infection and pathology and enhanced productivity, because the NPN is converted into utilizable protein by the ruminal microorganisms [26].

Fats and Energy supplementation: Energy retention was declined in T. colubriformis infected sheep, while efficiency of metabolisable energy (ME) utilization for growth reduced by 30 and 37% for O. circumcincta and T. colubriformis infections respectively [30,31]. Moreover, higher heat production in infected animals might be associated with an increased maintenance energy requirement (30kJ/kg metabolic body size/day) or reduced partial efficiency of feed conversion above maintenance in infected animals [32]. A severe negative energy balance was recorded in Cooperia spp. and Ostertagia spp. infected calves at three weeks post infection which might be due to depressed appetite, intake and utilization [33].

Supplementation of different types of lipids influences the rate of acquisition of immunity. Lambs fed n-3 fatty acids during a primary infection with T. colubriformis had significantly more worms after challenge than those fed n-6 or neutral lipids. As development of protective immunity to T. colubriformis is thought to involve local inflammation, this result is consistent with the hypothesis that dietary omega-3 lipid is exerting an anti-inflammatory effect on the local immune response [34]. Although the protective effects of these relatively diverse treatments could act directly or indirectly on the gut immune responses of the host, the exact mechanisms are not resolved at present.

Research results showed greater immune response against GI parasitic infections in sheep supplemented with fish oil. Fish oil, unlike the supplements used in experiment, as a source of linoleum acid; when offered directly in the diet, it is subject to bio-hydrogenation which could diminish its beneficial effects. Supplementation with oil or protected /bypass fat sources containing n-3 and n-6 fatty acids represents an interesting GI parasite control strategy [35], as these fats can influence the regulation of the immune expression and the inflammatory response of eicosanoid-mediated immune-effectors.

Mineral supplementation: Emerging demand of organic food products, day by day good quality feed and fodders crises, day by day decline in pasture land, repeated grazing practices, dependence on home grown feeds and forages and high prevalence of GI parasites are the basic rationale of minerals deficiency in animals and/or it could be more prevalent in organic animal farming. Young sheep infected with T. colubriformis utilized Ca and P inefficiently. It has been reported that the GI parasitic infection in abomasums and intestine adversely affected the calcium, phosphorus and magnesium deposition in growing animal, which affects bone mineralization [36].

Experimental diet containing phosphorus at a level of 0.28% DM increased weight gain and decreased worm burden and FECs in T. vitrinus infected lambs over those lambs fed a low phosphorus (0.19%) level diet. Zinc plays an important role to build up a successful immune response against GI parasites [36]. Iron had presumably no direct effect on GI parasites control; however, iron supplementation improves host performance because it restores iron status in the body which is lost through blood during GI parasitic infections.

Copper (Cu) acts as both anti-parasitic and host immunity boosting to some nematodes in goat, sheep and chicken. Supplementation of Cu oxides particle (5g) decreased the establishment rate of T. circumcinacta and H. contortus by 56 and 96%, respectively; but not of T. colubriformis in lamb. Excess manganese (Mn) may increase more infection as GI parasites need it and perhaps toxic to immunity response. Cobalt deficiency has also been reported to decrease the ability of ruminants to resist GI parasitic infection and previous workers anticipated that the gut immune response will prove to have requirements for other trace elements too. However, dietary selenium (Se) is an essential micronutrient that affects various aspects of animal health, including optimal immune responses. Through its incorporation into seleno-proteins, Se is involved in regulating oxidative stress, redox, and other crucial cellular processes in nearly all tissues and cell types, including those involved in innate and adaptive immune responses.

Deficiency of molybdenum (Mo) in soil and pasture of many areas may prone to GI parasitic infection while dietary Mo

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supplementation (@4-8mg/kg DM) in sheep may reduce worm burden. Some workers reported 78% and 23% reduction of H. contortus and T. vitrinus population in sheep supplemented 0.05mmol Mo/kg DM of feed. However, extreme of these both limits can increase the infection. Diets low in Mo impedes the ability of sheep to reject a challenge infection of H. contortus and T. colubriformis. Development of resistance to T. colubriformis given as a single bolus in lambs was assisted by a dietary intake of Mo in the order of 0.15-0.30mg Mo/kg body weight, induced by feeding diets containing approximately 6-10mg Mo/kg DM.

This appears to enhance the immune response to initial exposure to T. colubriformis and was associated with 3-5 fold decreases in FECs and worm counts after challenge [37,38]. Similarly, lowered FECs and worm counts observed in young lambs when received the challenge as a smaller dose over several weeks as would occur in the field. Thus, Mo intake is an important consideration in the nutrition of lambing ewes and lambs at weaning in the presence of pasture contaminated with GI parasitic larvae. For that reason Mo concentration found to give optimal protective immunity which was significantly higher than usually recommended. The quality of diet is particularly important in ruminants, whose digestive physiology results in some nutrients being habitually available at only marginal concentrations.

The effect of a deficiency is probably further compounded in the case of gastrointestinal infection because of the resulting interference with nutrient uptake, the loss of nutrients into the intestinal lumen and the involvement of the extensive gastrointestinal nervous and immune systems, both of which are metabolically active. This large-scale metabolic activity is likely to place an unusually heavy demand on nutrients. As a result the quality of the diet at the time of first exposure to infection is critical, particularly in the early post-weaning period when the developing immune response is competing with growth for a reduced amount of available nutrients.

Vitamin supplementation: The vitamins have diverse functions and are significant for animal health and production performance. Vitamin A is necessary for cell division and differentiation, plays a key role in regulation of keratinisation and immune cell function. On the other hand, vitamin E is a biological antioxidant in mammalian cell membranes, providing protection against free radicals. Several studies have documented the beneficial effects of vitamins A and E in general health, disease resistance and performance. Growing animals are more prone to GI parasitic infections thus their feeding practices during growth are very important for performance and general health. Major nutrients, such as energy and protein, play an important role in the susceptibility of animals to disease and particularly to infection with GI parasites. Balanced supplies of micronutrients (vitamins A and E) are also important, as deficiencies of these micronutrients have been associated with increased incidence of other diseases. Risk of specific clinical signs and general health disorders in growing lambs deficient in vitamin A and/or vitamin E are well documented. One of the major actions of vitamin A is increasing resistance to infection, which is very important in the case of GI parasitic infections.

Vitamin D3 induces a significant increase in circulating lymphocytes and the percentage of eosinophil vacuolization, a condition favoring a Th2 immune response, a hallmark of GI parasitic infection. Acquired resistance to Cooperia oncophora infection in cattle develops rapidly as a result of prior infections. Compared to primary infection, re-infection resulted in a marked reduction in worm establishment. In order to understand molecular mechanisms underlying the development of acquired resistance, researchers characterized the trans-criptomic responses of the bovine small intestine to a primary infection and re-infection. Vitamin D receptor activation was strongly induced only during re-infection, suggesting that this pathway may play an important role in the development of acquired resistance via its potential roles in immune regulation and intestinal mucosal integrity maintenance.

Deficiency of vitamin A, B12 (or cobalt), E (or selenium) have shown to delay the adult worm expulsion, more parasitic eggs in feces and increased fecundity due to changes in host intestinal physiology that promote host protection. Research study noted that vitamin B12 deficient lambs had higher FECs than vitamin B12 supplemented one after natural infection with GINs [39].

Tanniferous tree leaves as supplement: Various forages, plants or tree leaves with natural anti-parasitic properties may play an important role in future helminthes control strategies for livestock. Several tannin-containing forages, shrubs, tree leaves in particular those with condensed tannins (CT), showed anti-helminthes (AH) activity against GINs of large and small ruminants. Many studies have shown that use of tree leaves as ingredient of concentrate mixtures in the diets of ruminant could be particularly important in small ruminants for improved utilization of poor quality cereal straws [9]. This is also beneficial for control of helminthes parasites as tree leaves contain many polyphenolic compounds which are inhibitory to GINs. In a study, inclusion of dried leaves of Acacia karoo in their basal diet at a rate of 40% dry matter and infected with H. contortus significantly decreased in the faecal egg counts, and worm burdens were reduced by 34%, which is attributed the presence of tannins in tree leaves.

Forages and tree leaves which contain CT, notably sulla (Hedysarum coronarium), Lotus pedunculatus, Psidium guajava, Ficus infectoria, Acacia nilotica and Acacia karoo have been shown to significantly increase the growth rate of parasitized lambs and kids relative to non-CT containing forages, in the absence of anthelmintics [9,40-43]. Infected lambs and deer exhibited significantly lower FECs and lower abomasal and intestinal worm burdens when fed sulla compared to their counterparts fed conventional forages without CT. Low concentrations of CT have

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been shown to protect plant proteins against rumen degradation and to increase protein flow to the small intestine where they can improve the supply and absorption of amino acids.

Dietary supplementation of CT may enhance resistance of GIN through increases in tissue protein supply, which are prioritized for repair and immune response [44]. The CT could complex with nutrients and inhibits nutrient availability for larval growth or decrease GI parasites metabolism directly through inhibition of oxidative phosphorylation, causing larval death [11,12,19]. Inhibition of the electron transport system by CT was observed with Photobacterium phosphoreum. The CT extracted from various tree leaves and forages can disrupt the life cycle of GINs by preventing their eggs from hatching and by preventing larval development to the infective stages. Since CT is not absorbed in the digestive tract, they become concentrated in the faeces and may be chemically active in the feces. The CT may react directly with parasite larvae in the faecal pellet by binding to the cuticle of larvae, which is high in glycoprotein, inhibiting larval development [19,45].

Tanniferous leaf meal mixture containing multi-nutrient blocks (MNB-CT) maintain nutrient intake, their digestibility and nutrient utilization in H. contortus infected goats fed on roughage-based diets [20-22]. The body weights and better body condition scores of the MNB-CT supplemented animals indicate efficient digestibility of the feeds taken by these animals. MNB-CT can be used to deliver compounds other than essential or deficient nutrients. Previous work has established that MNB-CT can successfully deliver natural anthelmintic and assist in the control of GINs. Recent research study reported that good nutrition through MNB-CT supplementation had maintained the body weights and general health status of animals [46]. The benefits of MNB-CT supplementation are generally related to better appetite, improved body condition and reduction in FECs, resulting in lesser H. contortus infection in goats maintained on MNB-CT. Feeding of CT fortified leaf meal mixture incorporated densities complete feed (CT-DCF) indicates positive impact on nutrient intake and utilization, protein bioavailability, health status and better immune response and overall performance of H. contortus infected goats [23,47].

Nematophagous Fungi as Feed additive: Predatory fungi can kill varieties of GI parasitic species and substantially reduce the intensity of parasitic loads in livestock. With regard to the effect of nematophagous fungi against GI parasites, it is possible to reduce the number of infective larvae on herbage and subsequently reduce the number of nematodes establishing in the grazing animals. Challenges to fungal control are a requirement for daily administering of fungi to the host and achieving the required fungal density inside the dung. However, a nematode-killing fungus, Duddingtonia flagrans, showed a trapping efficiency rate of 78% and activity for up to 90 days on pasture, providing a viable alternative to reduce animal mortality from GI parasitic infections [24,48]. Dietary incorporation of nematophagous fungi as feed additives especially D. flagrans, Harposporrium anguillulae and Arthrobotrys spp. at the time when GI parasitic infestations are expected to high had the potential to control GI parasites in the pasture. Feeding of these fungi which survived in the gastro-intestinal tract (GIT) have reported to reduce the infectivity of herbage and also reduced worm burdens in grazing animals [49]. Feeding of different doses of D. flagrans spores mixed with complete diet reduced the infective larvae with the increased doses of spores, and feeding daily was more effective than intermittent feeding.

Economic losses

The economic losses caused by GI parasitic infestation are more important. They vary from country to country and region to region depending on climatic conditions and intensiveness of animal farming. Thus, losses occurred due to GI parasitic infections are greater in tropical countries than in temperate countries. Because favorable temperatures in the tropics may causes rapid multiplication and propagation of GI parasites.

Animals grazing on pasture are most susceptible to GI parasitic infection than stall fed animals due to exposure to faecal contamination [1]. The survey on cattle grazing on pasture indicated that 59% suffered from anemia and 79% anemic cases suffered from GI parasitism [50]. Malnourished animals are more susceptible to GI parasitic infection [2] and are more inclined to carry heavy parasitic loads because of their failure to throw off infestations quickly [51,52]. They cause's great economic losses in terms of reduction in meat, milk, wool, and hide production, body weight loss, high treatment cost of affected animal, ineffective anthelmintic treatment due to development of drug resistance and additional labor required for handling such animals [3,5-7]. The manifestation of GI parasitic infection may occurs in the form of poor weight gain, anemia, emaciation and high mortality in young ones. Direct economic losses due to GI Parasitic infections are the losses in weight gain (27% reduction) and it is estimated as 170 million rupees per year. However, economic losses due to mortality in kids are estimated as 59 million rupees per year [53].

Conclusion

In summary, nutrition play an important role against GI parasites, but much more specific research work is needed. Animal infected with GI parasites frequently also suffer from malnutrition and the impact that nutrition has on the patho-physiology of parasitic infection is recognized as a key area of research where the outcomes may afford practical nutritional strategies to counter the detrimental effects of GI parasites. Current evidence not only would suggest that each nutrient and non-nutrient substance behaves differently with regards to its impact on each type of GI parasite, but that nutrients and non-nutrient substance may impact directly on intestinal morphology, and on the GI parasite itself. This review has attempted to collate relevant recent work specifically designed to determine

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the direct and compounded effect of nutrition on GI parasites control, health status and productive performance of livestock.

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