Introduction
Diarrheal diseases represent a huge public health problem worldwide and are the second-leading cause of death particularly in children younger than 5 years of age with mortality rate of 1.5 million children every year (WHO fact sheet, 2009). Amongst various microbial agents causing diarrheal diseases, *Giardia intestinalis* (syn. *duodenalis* or *lamblia*) has been reported to be the most common. *Giardia intestinalis* is a microaerophilic flagellated eukaryotic protist causing waterborne intestinal infections in humans and more than 40 animal species (Rossignol 2010). Recently, giardiasis has been included in the Neglected Diseases Initiative by the World Health Organization (Savioli 2008). It is frequent in both developed and developing countries and is estimated to inflict 280 million symptomatic human infections annually (Nkruah and Nguah 2011). The highest prevalence of *Giardia* infection occurs in the tropics and subtropics where there is inadequate sanitation and inadequate treatment of drinking water (Ratanapo et al., 2008). However prevalence rates of human giardiasis range from 2-7% in developed countries and 20-30% in developing countries (Berkaman et al., 2002; Lindquist et al., 2006, Rossignol 2010). In India, prevalence rates of *Giardia* infection in patients with diarrhea range from 0.4% to 70%, and asymptomatic cyst passage has been found to be as high as 50% in rural southern India (Laishram, 2012). Giardiasis is a disease of main concern as it affects school going children, adults, hypogammaglobulinemic, malnourished and immunocompromised hosts and individuals carrying HLA antigens A1, A2, B8 and B12.

The parasite has a biphasic developmental cycle with two morphologically different forms: the cyst and trophozoite. Transmission of *Giardia* cysts to humans occurs mainly by consuming contaminated food and water and is facilitated by the relative resistance of cysts to chlorination and its ability to survive in cold and fresh water for several months (Huang and White 2006). Although the outbreaks have been linked to contaminated water but fecal oral route is regarded as the major source of infection, particularly in countries where the water is warm or where there is little
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drainage and no reticulated water supply is there such as child care centers and in nursing homes (Upcroft et al., 2001). Giardiasis begins with the ingestion of cysts followed by the colonization of excysted trophozoites in the lumen of small intestine. The protist Giardia colonizes the small intestine mainly by adhering to the brush border membrane of enterocytes without invading the epithelium or mucosal layers, a process which seems to be the prerequisite for Giardia induced enterocyte dysfunction (Gardner and Hill 2001). Adherence of Giardia trophozoites to the host intestinal epithelium is considered to be an important step in the pathogenesis of giardiasis, although the precise mechanism is still not known (Inge et al. 1988; Katelaris, 1995; Daniel and Belosevic, 1995). The interaction of Giardia trophozoites to brush border causes diffuse loss of microvillus brush border with or without villus atrophy, resulting into intestinal disaccharidases insufficiency and malabsorption of electrolytes and nutrients mainly carbohydrates, fats and water leading to diarrheal symptoms (Khanna et al., 1988; Buret, 2007). In addition, it has been found that inflamed intestines are deficient in antioxidants making it more susceptible to tissue damage (Kruideneir et al., 2003). Moreover, the clinical manifestations of giardiasis depend on the duration and severity of infection, age, nutritional and immune status of the host (Isolauri et al., 2001). A striking feature of giardiasis is the uneven presentation of clinical symptoms, ranging from asymptomatic carriage to chronic disease with associated diarrhea, malabsorption, recurrent abdominal pain, growth retardation, duodenitis, jejunitis, cholecystitis and weight loss (Laishram et al., 2012).

Giardiasis is a self-limiting disease, with spontaneous resolution of the acute phase, in individuals with fully developed immune system (Belosevic et al., 1989). However, in certain cases where acute infection develops into a chronic stage, recurrence of symptoms for short periods occurs (Wolfe, 1992). Host defenses against Giardia infection may be classified in two broad categories-innate and acquired immune responses. Amongst innate responses intestinal mucin makes the first line of defense against Giardia. Mucins may protect the intestinal epithelium by binding Giardia and restricting it to the mucus layer thus eliminating them via peristalsis and impeding microbial-epithelial interactions (Hecht, 1999).
Langford et al., in 2002 documented the role of nitrogen oxide and intestinal defensins in innate immune response against *Giardia* infection. Amongst acquired immune responses both humoral and cellular branches of the immune system plays a vital role in controlling *Giardia* infection (Heyaman and Menard 2002; Harish and Varghese, 2006). Singer and Nash, 2000 reported the importance of T cells as well as their cytokines in the control of *Giardia* infection as T cell cytokines may also induce the production and release of anti-giardial defensins into the intestinal lumen. However, secretory IgA play a central role in the host defense against *Giardia* infection, suggesting the possible role of B cells in anti-giardial host defense (Vinayak et al., 1989; Walterspeil et al., 1994; Langford et al., in 2002).

The first line of treatment for giardiasis is antibiotics namely nitroimidazoles and nitrofurans. However, the use of antibiotics is now being questioned due to the emergence of multi-drug resistant strains, low compliance with patients, and unpleasant side effects (Upcroft et al., 2001; Gardner and Hill, 2001; Lemee et al., 2000). The most serious adverse reactions reported in patients treated with flagyl (metronidazole) are convulsive seizures, encephalopathy, aseptic meningitis, optic and peripheral neuropathy, the latter characterized mainly by numbness or paresthesia of an extremity (Gardener and Hill, 2001). All these adverse effects have shifted the interest of scientists towards the use of safer, effective and cheap biointerventions which includes phytomedicines such as use of plant extracts and products derived from bees and nutritional interventions like probiotics that are safe, effective and inexpensive (Upcroft et al., 2001; Gardner and Hill, 2001; Lemee et al., 2000).

The term probiotic was initially used in the 1960s and is derived from a Greek word meaning "for life." Probiotics have emerged as the potential form of live therapy for prevention and treatment of gastrointestinal diseases and are known as microbial interference therapy. The probiotics are defined as "live microorganisms which when administered in adequate amounts confer a health benefit to the host (WHO report 2001) and are composed of yeast or bacteria. These are generally available as capsules, tablets, packets, or powders. Most
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frequently used probiotics includes mainly lactic acid bacteria belonging to genera *Lactobacillus* and *Bifidobacterium* species and the yeast *Saccharomyces boulardii*. The main characteristics of probiotics are resistance to gastric acid, alkalinity, bile salts, digestive enzymes and ability to adhere and colonize the intestinal tract. The rationale for using probiotics for intestinal diseases involves restoration of microbial balance in the gut, secreting various toxic substances such as hydrogen peroxide, organic acids, bacteriocins and biosurfactants competing with pathogens for nutrients and preventing their adhesion to the intestinal enterocytes, antioxidative ability and immunomodulatory potentials (Nagpal et al., 2012; Hempel et al., 2012). Probiotic strains have been reported to augment the immune response by stimulating the phagocytic activity of lymphocytes and macrophages (Reid et al., 2001).

Several animal and human studies have demonstrated that some probiotic strains can successfully modify the mucosal immune response to modulate the levels of secretory IgA and cytokine expression but their immunomodulatory potentials of probiotic microbes are species and strain specific (Matsumoto et al., 2005; Kaur and Bhatia 2012). It follows therefore, that probiotic bacteria may be an alternate live bacteriotherapy that can be a part of healthy diet for humans, providing a barrier against microbial infections (Sarrela et al., 2000; Benyacoub et al., 2005). Experimentally, it has been shown that probiotics offer a natural approach in the management of various gastrointestinal diseases caused by various pathogens, e.g. *Salmonella* and *Giardia*, Rotavirus and *Clostridium difficile* (Sullivan, 2005; Szajewska, 2005; Shukla et al., 2008; Rishi et al., 2011). It has also been shown that probiotics in conjunction with antibiotics have synergistic effect in the management of *Helicobacter pylori* infection (Zou et al., 2009), salmonellosis (Preet et al., 2010) and giardiasis (Shukla et al., 2013).

The interactions between probiotic and inflamed intestinal mucosa have been best highlighted in animal models of inflammatory bowel diseases. Since giardiasis too leads to inflammation of the small intestine in humans, making experimental giardiasis a useful model to study the underlying mechanism of pathogenicity, the effect of treatment and host parasite relationships (Carmen et al., 2011). Earlier
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reports have shown that probiotic *Lactobacilli casei* has the potential to reduce both the intensity and duration of murine giardiasis (Benyacoub et al., 2005; Shukla and Sidhu 2011; Shukla et al., 2012). However, evidence for an effective probiotic against giardiasis with special reference to their antioxidative and immunomodulatory activity both *in vitro* and *in vivo* is lacking and warrants further study.