The intestine of any host is a rich and diverse ecosystem composed of host tissues and cells, enteric pathogens, and commensal gut bacteria. These microbial bacteria exist in high density and diversity and form a complex community of bacterial genomes called the gut microbiome, which is crucial to many host biochemical processes, including homeostasis maintenance in the digestive system, metabolic functioning, stimulation of epithelial cells, regulation of both the immune system and brain, and protection against pathogens (Kinross et al. 2011; Flint et al., 2011; Partida-Rodriguez et al. 2017; Duarte et al. 2016; Hooper et al., 2012). Additionally, microbial diversity is linked to host health, where diverse gut microbial communities can provide more functional support to the host and are better equipped to buffer against disturbance (Costello et al., 2012; Zaiss and Harris 2015). However, when disturbances do occur, the gut microbiome may fall into a dysbiotic state, resulting in an imbalance in the composition, abundance, and diversity of gut bacteria populations (Partida-Rodriguez et al. 2017; Sekirov et al., 2010).

As both intestinal pathogens and commensal gut bacteria reside in overlapping niches within the intestinal ecosystem, their interactions may lead to dysbiosis of the gut microbiome. (Zaiss and Harris 2015). However, the causal relationship between pathogenic infection and microbial dysbiosis has yet to be elucidated. Microbial dysbiosis can increase a host’s susceptibility to pathogen colonization, which then allows infectious agents to further modify the microbiome. Changes in the composition and abundance of a host’s gut bacteria may also influence pathogen survival, as microbial metabolites can affect the survival and persistence of infection (Partida-Rodriguez et al. 2017). Likewise, pathogens can cause shifts in gut microbial communities through secretions and by-products that negatively alter the composition and diversity of the microbial community, leading not only to susceptibility to and colonization by future pathogens, but also potential downstream health effects (Costello et al. 2012; Partida-Rodriguez et al. 2017).

A diverse gut microbial community can either indirectly prevent pathogen colonization by creating a hostile environment to the pathogen, or resident microbes can compete directly for niche opportunities and nutrients (Costello et al. 2012; Peachey et al. 2017; Lee et al. 2014). However, many pathogens require the presence of a gut microbiota to develop an infection and colonize subsequently (Kreisinger et al. 2015). In fact, certain pathogens may promote a more diverse microbial community, resulting in expected host health benefits. For example, humans colonized with helminths had increased gut microbial diversity compared to uninfected individuals in indigenous Malaysian populations (Lee et al., 2014). Due to the long, co-evolutionary relationship between hosts and some intestinal pathogens, like helminths, pathogen-microbiota interactions might be a key factor in maintaining intestinal homeostasis and promoting host health (Zaiss and Harris 2015; Kreisinger et al. 2015; Cooper et al. 2013; Lee et al. 2014; Cantacessi et al. 2014).

Each pathogen-bacteria interaction is species-specific, creating unique host-commensal-pathogen systems (Peachey et al. 2017; Zaiss and Harris 2015). Thus far, there has been a heavy emphasis on studying the effect of helminthic infections on human and rodent gut microbiomes as well on microbial communities of captive mammals. As captivity has been demonstrated to have a strong effect on a host’s gut microbial composition and diversity, it is crucial to study how infectious pathogens influence the gut microbiomes of wild mammals (Amato 2013; Clayton et al. 2016; Peachey et al., 2017). Understanding these interactions may predict disease susceptibility and inform conservation strategies for endangered wildlife. Non-human primates (NHPs) are especially threatened with extinction as 75% of primate species are declining in population size (Estrada et al., 2017), thus highlighting the urgency to which we understand how a primate’s gut microbiome may affect and be influenced by pathogenic infection. As such, this study is one of the first to examine pathogen-gut microbiota relationships in two wild and vastly different NHPs. Here, we assessed the gastrointestinal (GI) parasite burden and gut microbial composition and diversity in the mantled howler monkey (*Alouatta palliata*) and the red-shanked douc langur (*Pygathrix nemaeus*).

Intestinal pathogenic infection has been well documented in mantled howlers, including discovery of *Parabronema* sp., *Controrchis* sp., *Trypanoxyuris* sp., *Strongyloides* sp., *Entamoeba* sp., *Isospora* sp., *Ascaris* sp., *Coccidia*, *Physaloptera* sp., and other unidentified nematodes, trematodes, and flukes (Stuart et al., 1990; Stoner 1996; Trejos-Macia and Estrada 2012; Trejos-Macia et al., 2007; Maldonado-Lopez et al., 2014; Cristobal-Azkarate et al., 2010). Contrastingly, very few studies have examined pathogen burden in red-shanked doucs. Previously, singular cases of *Echinococcus ortleppi* and *Mycobacterium avium* have been recorded in captive and semi-captive settings (Plesker et al., 2009; Plesker et al., 2010). Another study detected the presence of *Trichuris* nematodes (24%) and *Strongyloides* spp. (49%) in individuals living in Cuc Phuong National Park (Hartmann et al., 2015). Red-shanked douc langur are on the IUCN Red List of Threatened Species, so it is crucial to understand their risk for parasitic infection to shape the management and conservation of the species. Thus far, the scientific documentation of GI pathogens in red-shanked doucs contains large gaps, and so, in addition to evaluating the pathogen-gut microbiota relationship in NHPs, our study accomplishes the first comprehensive GI parasite analysis in wild and free-ranging douc langurs*.*