CHAPTER 1

INTRODUCTION
Dientamoeba fragilis is derived from the trichomonad group and has been responsible for causing gastrointestinal symptoms in persons infected globally (Nagata et al., 2012). The prevalence of the infection range between 0.5% to 16% (Nagata et al., 2012). *D.fragilis* was first described as an amoeba by Jepps and Dobell a century ago (Jepps & Dobell et al., 1918) It has been more than 87 years since the discovery of *D.fragilis*, however information on its biology, life cycle, mode of transmission as well as the pathogenesis remain an enigma when compared to other parasite (Stark et al., 2006).

The understanding of the parasite’s life cycle remains still in its infancy (Banik et al., 2012) and information pertaining to the biology of this parasite is still uncertain with many more aspects of the parasite remaining to be explored (Johnson et al., 2004). In addition, the understanding of the host distribution and zoonotic potential of this organism still remains uncertain (Johnson et al., 2004).

*D.fragilis* got its name due to the presence of its two nucleus and also due to the fragile nature of the parasite where it has been shown to degrade easily when exposed in the environment (Johnson et al., 2004). Initially this parasite was classified as an amoeba for 50 years, but due to the existence of centrodesmus and resemblance to *Histomonas meleagridis*, its taxanomy was questioned (Johnson et al., 2004). It was believed that *D.fragilis* possessed a flagella which can disintegrate when cultured or when the parasite invades a tissue (Johnson et al., 2004). Then for the next 20 years since the classification of *D.fragilis* was not confirmed till the DNA studies proved that *D.fragilis* belongs to Trichomonad (Johnson et al., 2004).
Although animals play a small role in transmitting this parasite, humans however remain the main host in transferring *D. fragilis* (Barratt et al., 2011b). The mode of transmission of *D. fragilis* remains uncertain (Clark et al., 2014). The parasite is either transmitted by human, animals or other parasites. Most studies reports that *D. fragilis* is transmitted via the fecal oral route (Barratt et al., 2011a). The possibility of *D. fragilis* transmission via the egg of an intestinal nematode have been proposed (Dobell et al., 1940).

The presence of pseudocystic, precyctic or cystic stage of this parasite have yet to be proven since the parasite is known to be fragile (Greenway, 1928; Knoll & Howell, 1946; Wenrich, 1936). The survival period of this parasite is between 6 to 24 hours (Johnson et al. 2004).

It has been reported that the prevalence of *D. fragilis* is between 0.3% and 52% (Barratt et al., 2011b). This parasite has always been known to be non-pathogenic although there are evidence to support its pathogenicity. Co-infection with other parasites such as *Entamoeba histolytica, Giardia* and *Cryptosporidium* have been reported (Barratt et al., 2011b). The reported prevalence of *D. fragilis* have been based mostly using light microscopy which may not accurately provide the information when compared to polymerase chain reaction (PCR) or the use of the *in vitro* culture method (Barratt et al., 2011b).

More studies need to be carried out to enhance the detection methods for *D. fragilis*. Till date, there is only one prevalence data on this organism in Malaysia. Therefore there is a need to assess if this parasite does occur in the stools of Malaysians especially in Orang Asli and school children. Furthermore, studies *in vitro* and *in vivo*
animal models need to be carried out to ensure maintenance of the parasite. This will ensure a constant supply for more molecular, biochemical, biology and mode of transmission studies so that more information of the parasite can be elucidated. With a steady supply of parasite material there will be a greater opportunity to improve the detection methods including staining and molecular methods as well as explore other fundamental aspects of this interesting organism.
Chapter 1: Introduction

1.1 Objectives of the study

1.1.1 To assess the prevalence of *D.fragilis* in Orang Asli (aborigines) villages and school children in the state of Selangor and

   a) To assess its associative presence with other parasites including *Blastocystis* sp.

   b) To assess the associated risk factors

1.1.2 To assess the biological features of the life cycle stages of *D.fragilis* seen in *in vitro* cultures.

1.1.3 To assess the susceptibility of mice and rats towards experimental infection with inoculation of cyst-like stages of *D.fragilis*

1.2 Research hypothesis

1.2.1 Orang asli and school children are easily infected with *D.fragilis* due to being lesser aware of good hygiene practices.

1.2.2 *D.fragilis* has another mode of reproduction apart from binary fission which possibly could account for the high parasite numbers seen in *in vitro* cultures within a short frame of time.

1.2.3 Rats and mice can be infected with cyst–like stages of *D.fragilis*. 