

Pathophysiological variability of different genotypes of human *Blastocystis hominis* Egyptian isolates in experimentally infected rats

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Abstract The genotyping of *Blastocystis hominis* clinical isolates obtained from 28 gastrointestinal symptomatic patients and 16 asymptomatic individuals were identified by polymerase chain reaction using sequenced-tagged site (STS) primers. Then, pathophysiological variability between different *B. hominis* genotypes was evaluated in experimentally infected rats. Only four *B. hominis* subtypes (1, 2, 3, and 4) were detected (18.2%, 9.1%, 54.5%, and 18.2%, respectively) in human isolates. In symptomatic isolates, subtypes 1, 3, and 4 were detected in 8 (28.6%), 16 (57.1%), and 4 (14.3%) patients, respectively. In asymptomatic isolates, subtypes 2, 3, and 4 were identified in 4 (25%), 8 (50%), and 4 (25%), respectively. Subtype 3 was the commonest in humans. Different degrees of pathological changes were found among infected rats by symptomatic subtypes compared with asymptomatic subtypes. The moderate and severe degrees of pathological changes were

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found only in symptomatic subtypes infected rats while mild degree was found only in asymptomatic subtypes infected rats. Only subtype 1 induced mortality rate with 25% among infected rats. On evaluation of the intestinal cell permeability in the Ussing chamber, a prominent increase in short circuit current (ΔI_{sc}) was found in symptomatic subtype 1 compared to symptomatic subtypes 3 and 4 infected rats. Minimal effects were found in the asymptomatic and control groups. The results proved that subtype 1 was clinically and statistically highly relevant to the pathogenicity of *B. hominis* while subtype 2 was irrelevant. Also, the results suggest the presence of pathogenic and nonpathogenic strains among subtypes 3 and 4.

Introduction

The intestinal parasite *Blastocystis hominis* is the most common parasite found in fecal examinations of humans (Windser et al. 2002), which occurs from 0.8% to 61.8% with a high rate in adults than in children (Horiki et al. 1997). The role of *B. hominis* in human intestinal disease is controversial. Several clinical and epidemiological studies implicate *B. hominis* as a pathogen (Ashford and Atkinson 1992; Sadek et al. 1997; Rossingnol et al. 2005; Tan and Suresh 2006; Kaya et al. 2007) while others dismiss it as a commensal parasite (Shlim et al. 1995; Cirioni et al. 1999; Chen et al. 2003; Leder et al. 2005). Limited information was known about the host and parasitic factors which cause symptomatic or asymptomatic infection (Tan 2004). However, Puthia et al. (2005) and Sio et al. (2006) proved that *B. hominis* is able to produce a cystine protease that breaks up IgA antibody, which allows *B. hominis* survival and colonization in the human gut. Also, disturbances on the intestinal barrier functions may be contributed to the diarrhea

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