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PATHOGENICITY AND CHARACTERISTICS OF *Blastocystis* SUBTYPES

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Abstract
Blastocystis sp., an intestinal protozoan widely distributed in the human gastrointestinal tract, exhibits a global presence, with prevalence varying by region and generally higher in developing countries compared to developed ones. In Indonesia, reported prevalence varies considerably by region and study population, ranging from approximately 34% to 72% in several regional studies. This protozoan is water-borne and primarily transmitted through cysts via the fecal-oral route. Factors such as poor hygiene, immigration, travel to tropical developing countries, contact with animals, and consumption of contaminated food or water elevate the risk of infection. The pathogenesis in humans remains unclear, as infections may either manifest symptoms or remain asymptomatic, partly due to diagnostic limitations and variation in virulence across subtypes. This review aims to elucidate the characteristics and pathogenicity of *Blastocystis* sp. subtypes, particularly ST3, which is frequently dominant in symptomatic patients, and to discuss ongoing controversies in the literature.
Keywords: *Blastocystis*; Morphological Characteristics; Pathogenicity; Subtypes

Abstrak
Blastocystis sp., protozoa usus yang tersebar luas di saluran pencernaan manusia, memiliki distribusi global, dengan prevalensi yang bervariasi antarwilayah dan cenderung lebih tinggi di negara berkembang dibandingkan negara maju. Di Indonesia, prevalensi *Blastocystis* sp. dilaporkan bervariasi menurut wilayah dan populasi penelitian, berkisar sekitar 34-72% pada beberapa studi regional. Protozoa ini bersifat water-borne dan utamanya ditularkan melalui kista melalui jalur fecal-oral. Faktor-faktor seperti kondisi higiene yang buruk, imigrasi, perjalanan ke negara tropis berkembang, kontak dengan hewan, dan konsumsi makanan atau air yang terkontaminasi meningkatkan risiko infeksi. Patogenesis pada manusia masih belum jelas karena infeksi dapat menimbulkan gejala atau tetap tanpa gejala, sebagian disebabkan oleh keterbatasan diagnostik dan variasi virulensi antarsubtipe. Review ini bertujuan untuk mengungkapkan karakteristik dan dampak patogenisitas *Blastocystis* sp., khususnya subtipe ST3 yang sering dominan pada pasien simtomatik, serta kontroversi yang masih ada dalam literatur.
Kata Kunci: *Blastocystis*; Patogenisitas; Subtipe; Karakteristik Morfologi

INTRODUCTION

Blastocystis sp. is an intestinal protozoan commonly found in the human gastrointestinal tract worldwide (Wawrzyniak et al., 2013). The occurrence of *Blastocystis* sp. varies across nations, generally showing higher rates in developing countries than developed ones. For instance, the prevalence in Japan is reported to range between 0.5-1%, while in Singapore it stands at 3.3%. In contrast, developing countries exhibit higher prevalence rates, such as 27.2% in Argentina, 40.9% in Brazil, 38.5% in Cuba, and 33.3% in Egypt (Nofita et al., 2015). In Indonesia, prevalence varies considerably by region and population rather than following a single national figure; a cross-sectional study in a rural area of Southwest Sumba, East Nusa Tenggara, reported a prevalence of 34.4% (146/424) (Sungkar et al., 2015), while a review of parasite infections in Indonesia cited rates as high as 72.4% among HIV-positive patients and 54.7% among immunocompromised children with persistent diarrhea in Jakarta (Lee & Ryu, 2019). This wide variation appears to be correlated with sanitation conditions, population studied, and diagnostic method used.

Blastocystis sp. infection is called blastocystosis. It is a water-borne disease transmitted by cysts via the fecal-oral route. Increased risk of infection can occur due to poor hygienic

conditions, immigration and travel to tropical developing countries, and contact with animals or consumption of contaminated food or water (Sadaf et al., 2013; Wawrzyniak et al., 2013).

Pathogenesis in humans is still unclear because infection can be symptomatic or asymptomatic. This parasite is considered opportunistic and may only cause clinical manifestations when host immunity declines (Wawrzyniak et al., 2013).

Morphological distinctions among *Blastocystis* sp. subtypes are not discernible by microscopy alone; hence molecular techniques have become essential for epidemiological, clinical, and therapeutic investigation. Numerous studies have elucidated the life cycle of *Blastocystis* sp. and its role in the digestive tract as both a commensal and potentially pathogenic organism. Advances in molecular and immunological methods have led to the identification of several distinct subtypes (Wawrzyniak et al., 2013).

METHODS

Literature for this review was searched in PubMed, Scopus, and Google Scholar using the keywords "*Blastocystis*," "subtype," "pathogenicity," "ST3," "diagnosis," and "Indonesia," combined using Boolean operators, covering publications primarily from 2000 to 2025 with priority given to studies published after 2015. Additional foundational studies published before 2000 were retained where they remain the primary reference for a specific morphological or ultrastructural finding still cited in current literature.

Eligibility Criteria

Inclusion criteria were peer-reviewed original research and review articles reporting subtype distribution, pathogenicity evidence (in vitro, in vivo, or clinical), or diagnostic methods for *Blastocystis* in humans. Exclusion criteria were studies focusing solely on non-mammalian and avian subtypes (NMASTs) without human relevance, non-English/non-Indonesian articles, and conference abstracts without full text.

RESULTS AND DISCUSSION

A total of 32 full-text articles meeting the eligibility criteria were retained for narrative synthesis, spanning taxonomic, diagnostic, and pathogenicity evidence.

Taxonomy and Morphology of *Blastocystis*

The genus *Blastocystis* is included in the single-cell Heterokonta (Stramenopiles), which has low host specificity; naming species based on the host is no longer used and is called *Blastocystis* sp. (ST). The nomenclature of *Blastocystis* sp. can employ a consensus terminology relying on SSU rRNA gene sequences to denote subtypes (ST) (Stensvold et al., 2007).

This protozoan is a round, thick-walled cyst with a size between 6-40 µm. *Blastocystis* sp. has 4 forms, namely vacuolar and granular, which are round and contain a single large vacuole. Granular cells possess numerous small granules within the cytoplasm or central vacuole. They consist of several nuclei, up to 4 in number. This represents the prevailing morphology of *Blastocystis* sp., with a diameter ranging from 2 to 200 µm (Tan, 2008). Multivacuolar and avacuolar forms are small and consist of 1-2 nuclei; amoeboid forms are rare. Pseudopodia are often attached; cysts have thick walls, consist of many vacuoles, and have 1-2 nuclei. This morphology is used to identify *Blastocystis* sp. in fecal samples with a size of 10-15 µm. *Blastocystis* sp. usually infects the human intestine in the cecum and colon and then reproduces by binary fission (Belleza et al., 2015).

Blastocystis sp. exhibits a range of subtypes identified through the analysis of SSU rRNA gene sequences. The classification encompasses 17 subtypes (STs) for mammalian and avian *Blastocystis* sp., of which nine (ST1-ST9) have been identified in human hosts. Additionally, there are eight non-mammalian and avian subtypes (NMASTs) (Belleza et al., 2015).

Diagnostic Approaches

Conventional diagnosis of *Blastocystis* sp. relies on direct microscopy of wet mounts or trichrome-stained fecal smears, and stool culture using Jones' or similar xenic media. However, microscopy has consistently shown limited sensitivity: in a prospective study of 186 patients, direct-light microscopy detected only 29% and xenic in vitro culture only 52% of cases confirmed positive by qPCR (Poirier et al., 2011). This gap is largely explained by intermittent cyst shedding and morphological overlap between *Blastocystis* sp. forms, and microscopy alone cannot distinguish subtypes. Molecular methods, particularly conventional PCR (cPCR) and quantitative real-time PCR (qPCR) targeting the SSU rRNA gene, have substantially improved detection sensitivity; a direct comparison of 288 stool samples found qPCR detected significantly more positive cases than cPCR (29% vs. 24%, $p<0.05$) (Lhotská et al., 2022). For subtype-level resolution, Sanger sequencing of PCR amplicons remains the reference method, while next-generation sequencing (NGS) is increasingly used because it can detect mixed-subtype colonization within a single host that Sanger sequencing may miss (Lhotská et al., 2022). Despite these advances, molecular diagnostics remain largely confined to research settings in Indonesia because of cost and infrastructure limitations, and microscopy remains the mainstay in routine clinical laboratories.

Blastocystis Pathogen

In Zulfa et al. (2017) research, the *Blastocystis* sp. subtype associated with diarrhea in children in Jakarta indicated that ST3 emerged as the predominant subtype, exhibiting an overall prevalence of 63.8%, followed by ST1 at 27.5%, and ST2 at 7.2%. These three subtypes were detected in both symptomatic and asymptomatic groups. Within the symptomatic group, four STs were identified, with ST3 as the most prevalent subtype at 72.2%, followed by ST1 at 22.2%, ST2 at 2.8%, and ST4 at 2.8%.

In terms of age groups, the prevalence rate among children aged 6-9 years was observed to be over 66.6% (46 out of 69), while for those aged 10-13 years it was 33.3% (23 out of 69). These findings, in contrast to research conducted in Papua New Guinea, demonstrate variation in prevalence rates between age groups and emphasize that young adults exhibited higher prevalence rates than children in that study (Pramestuti & Saroh, 2017).

Various *Blastocystis* sp. isolates exhibit differences in the outer cell membrane layer known as the surface coat. This component is believed to serve a protective function against the host's innate immune response and to significantly contribute to adhesion during colonization. An early investigation into the ultrastructure of *Blastocystis* sp., which elucidated the structure of the surface coat, was conducted using cell culture. The study identified amoebic, granular, and vacuolated forms of the organism. The amoeba form is characterized by a filamentous layer serving as an outer structure without distinct cells or membranes (Dunn et al., 1989). Conversely, the granular and vacuolated forms exhibit distinct membranes, and their filamentous layers appear denser and thinner compared to the amoeba form. Additionally, pockets along the surface coat have been observed in these forms (Tan, 2008).

Using an electron microscope, Matsumoto et al. (1987) researched fecal isolates from culture samples. The results showed no differences in the filamentous layer and identified a capsule consisting of a filamentous layer outside the cell membrane. Dunn et al. (1989) used stock cultures of *Blastocystis* sp. and found variations in surface coat structure. There was no clear association between surface coat thickness and mean cell size. Two cells displayed a thin surface coat, and one was amebic. The surface coat was indistinguishable in isolates from colonoscopy samples; however, in culture isolates, cells were found with a thick surface coat. Cassidy et al. (1994) observed differences in the structure of the surface coat among isolates obtained from various animal hosts.

In symptomatic patients, the surface coat of ST3 exhibits a rougher and thicker appearance. To investigate the biochemical properties of the *Blastocystis* sp. surface coat, a periodic acid-based treatment test was conducted to detect carbohydrates. Positive research findings indicated the presence of carbohydrates in the filamentous layer of *Blastocystis* sp., as well as in organelles such as the central vacuole, vesicles, and Golgi bodies (Lanuza et al., 1996). Subsequent research by Lanuza et al. (1996) employed a lectin probe to identify specific carbohydrates in the surface layer of *Blastocystis* sp. Their results revealed the inclusion of α-D-mannose, α-D-glucose, N-acetyl-α-D-glucosamine (GlcNAc), α-L-fucose, chitin, and sialic acid in the carbohydrate structure of the surface coat. Some of these components are also present on the surface of other protozoan parasites as glycoconjugates. Beyond serving as a membrane barrier, this component plays a crucial role in host adhesion and invasion, as well as in evading the host's immune response. For instance, the GlcNAc polymer of chitin identified in Trichomonas is considered essential for the parasite's adhesion to host cell lectins.

A study of variation, morphology, and surface coat properties of *Blastocystis* sp. using axenic cultures from identified subtypes carried out flow cytometry tests to describe morphology. It was found that most of the irregularly shaped amoebae showed positive propidium-iodide (PI) staining, mostly in ST1 isolates and a few in ST4 and ST7 isolates (Yason & Tan, 2018).

In studies of the susceptibility of *Blastocystis* sp. to the colonic antimicrobial peptide (AMP) LL-37, the surface coat is thought to act as a barrier preventing AMP from reaching the membrane, so that AMP cannot form pores in the organism. Using fluorescent-labeled antibodies against LL-37, it was found that in *Blastocystis* sp. ST7-B isolates, AMP could enter the cell surface, lowering survival rates compared to ST4 isolates. Using light microscopy with negative Indian ink staining, the most susceptible *Blastocystis* sp. isolates (ST1) were found to have an invisible surface coat, whereas the ST7-B isolate had the thickest and densest surface coat (Yason & Tan, 2018).

Scanning electron micrographs (SEM) of isolates ST1, ST4, and ST7 were used to analyze the surface coat of these subtypes. The surface of ST1 was found to be more uniform than isolates ST4 and ST7. Meanwhile, ST4 showed uneven surfaces in several cells, and ST7 had repeated surface layers connected to surrounding cells, believed to establish inter-cell relationships and enhance survival (Yason & Tan, 2018). In a prior investigation, Tan et al. (2000) used SEM of ST7 to demonstrate colony development on agar with interconnected and uneven surface layers of cells, providing evidence for the cytoadherence properties of this structure and clarifying the increased difficulty in homogenizing cells that form solid masses in culture when in suspension.

In an initial study characterizing monoclonal antibodies (MAb) for identification of *Blastocystis* sp. ST7 using immunogold labeling and biochemical tests, MAb was found to be specific for carbohydrates in the surface coat and plasma membrane (Tan, 2008).

Based on SSU-rRNA gene analysis, *Blastocystis* sp. is found in various hosts, including humans and animals, mammalian and non-mammalian. Nine of the 17 STs have been identified in humans (Alfellani et al., 2013; Clark et al., 2013; Stensvold et al., 2007). Kaneda et al. (2000) found that ST1, ST2, and ST4 were related to gastrointestinal symptoms. In 2012, Poirier et al. (2012) reported ST7 to be linked to IBS, and Wawrzyniak et al. (2013) found that ST7 may use hydrolases to destroy host tissue. The presence of intestinal microbiota influences the occurrence of the *Blastocystis* sp. pathogen. ST10 and ST14, meanwhile, were found only in livestock, not humans, even after direct contact.

Certain *Blastocystis* sp. subtypes display host specificity and varying geographic distributions. In the Americas, there is a heightened prevalence of ST1 and ST2, while in Australia, Europe, and Southeast Asia, ST1 and ST3 predominate, and in Europe ST4 prevails

(Stensvold et al., 2007). Recent research in South America has identified ST4 in humans, with ST1, ST2, and ST3 being the most frequently detected subtypes (Jiménez et al., 2019). However, globally, ST3 stands out as the most prevalent subtype among humans and is consistently found in ST distribution analyses, irrespective of geographic origin (Meloni et al., 2011; Nagel et al., 2012). Furthermore, *Blastocystis* sp. carriers are observed to be three times more prevalent in individuals with irritable bowel syndrome (IBS) and immunosuppressed patients compared to asymptomatic carrier controls (Nagel et al., 2015).

Blastocystis subtype 3 (ST3) is present in the gastrointestinal tract of both symptomatic and asymptomatic individuals globally, with substantial prevalence across human populations. However, the actual geographic distribution and comprehensive genetic diversity of ST3 remain undisclosed, creating gaps in knowledge that impede a thorough understanding of its transmission mechanisms, epidemiology, and pathogenicity within human populations (Clark et al., 2013).

In research by Abdel-Hameed and Hassanin (2011), tests on the protease activity of *Blastocystis* sp. in both symptomatic and asymptomatic individuals revealed the presence of *Blastocystis* sp. protease in 94.4% of patients. Notably, symptomatic patients with ST3 exhibited elevated protease levels, which, combined with a 32 kDa low molecular weight protein, constitutes a virulence factor responsible for protein degradation. This mechanism enables the parasite to evade the host immune system or modulate it through degradation of host immune molecules.

Nevertheless, several investigations have identified ST4 as a potentially pathogenic strain due to its high prevalence in patients experiencing severe diarrhea. Another subtype, ST8, has also been suggested as potentially pathogenic; while infrequently observed in humans, it has been linked to severe symptoms in two separate studies (Alfellani et al., 2013). Despite ST3 being the most prevalent subtype among humans, there is a weak correlation between the presence of a given ST and reported symptoms. Findings from mouse studies indicate that ST1 is statistically associated with pathogenicity, suggesting the existence of both pathogenic and nonpathogenic strains within ST3 and ST4 (Ajjampur & Tan, 2016).

One study suggested that the 32 kDa protease of ST3 could be a virulence factor influencing protein degradation (Abdel-Hameed & Hassanin, 2011). In comparison, other studies show that the *Blastocystis* sp. antigen at 29 kDa can be used as a marker of pathogenicity, differentiating symptomatic from asymptomatic infections (Abou Gamra et al., 2011).

Recent studies exploring the impact of *Blastocystis* sp. on interferon-gamma expression and proinflammatory cytokines in the cecal mucosa of mice revealed a significant increase in transcription of type 1 genes and proinflammatory cytokines, including IFN-γ, IL-12, and TNFα. These findings indicate that *Blastocystis* sp. infection, when tested in mice, induces specific local host responses involving T cells, monocytes/macrophages, and NK cells in response to antigens. Moreover, an examination of the effects of *Blastocystis* sp. on infected mice demonstrated its influence on weight loss and diarrhea in animals inoculated with high doses (Ajjampur & Tan, 2016).

An additional investigation demonstrated that *Blastocystis* sp. has the ability to infiltrate the lamina propria, submucosa, and muscle layer (Elwakil & Hewedi, 2010). Conversely, a separate study identified heightened urinary hyaluronidase levels in *Blastocystis*-infected mice, suggesting potential invasion of the colonic epithelium (Chandramathi et al., 2010). Further research involving human subjects is necessary to validate these findings. In laboratory settings, mice have been used as an effective animal model for studying *Blastocystis* sp. infection: mice infected with ST1 displayed histopathological alterations across all administered doses, underscoring the pathogenic potential of ST1 with individual variation. This underscores the

utility of animal models for assessing pathogenicity, particularly given that mice are not natural carriers of *Blastocystis* sp., unlike other species commonly found with the parasite (Ajjampur & Tan, 2016).

According to Souppart et al. (2009), *Blastocystis* sp. infection is not specifically associated with a particular subtype but is rather influenced by various risk factors related to transmission — environmental factors such as transmission routes and sources of contamination, parasitic factors including pathogenic and zoonotic potential, and host factors such as genotype, immunity, and age.

It is important to note that the association between ST3 and symptomatic infection remains correlational rather than causal. Koch's postulates have not been fulfilled for any *Blastocystis* sp. subtype, and several cross-sectional and animal studies have found no consistent subtype-symptom relationship, with intra-subtype variation in virulence potentially outweighing between-subtype differences — for example, experimental infection of mice with ST1-ST4 isolates showed that ST3 and ST4 were each represented by both pathogenic and non-pathogenic strains (Ajjampur & Tan, 2016). The clinical relevance of *Blastocystis* sp. as a whole continues to be debated, and host factors such as immune status, gut microbiota composition, and coinfection may better explain symptomatic presentation than subtype identity alone.

CONCLUSION

Current evidence indicates that pathogenicity in *Blastocystis* sp. is not determined by subtype alone but likely results from an interaction between subtype-specific virulence factors (such as surface coat composition and protease activity), host immune status, and gut microbiota context. ST3 remains the most frequently detected subtype in symptomatic populations, including in Indonesia, but its correlation with clinical symptoms is inconsistent across studies, and Koch's postulates remain unfulfilled for any subtype. Three research gaps warrant priority attention: first, nationally representative, molecularly confirmed subtype-prevalence data for Indonesia are lacking, as most existing studies are regionally limited; second, standardized diagnostic protocols combining microscopy with molecular subtyping are not yet routinely implemented outside research settings; and third, prospective studies linking subtype identity with quantified symptom severity and host immune markers are needed to clarify causality. Clinically, these findings suggest that a positive microscopic finding of *Blastocystis* sp. alone should not automatically justify treatment in asymptomatic individuals, and molecular subtyping should be considered when persistent gastrointestinal symptoms remain unexplained after excluding other pathogens.

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