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**Introduction**

*B. hominis* is a chloroplastic alga that belongs to the phylum Stramenopila and is the most common parasite found in the lower gastrointestinal tract of humans (Poirier et al. 2012). *B. hominis* is distributed globally with a human infection prevalence of 1.5-10% and 30-50% for developed and developing countries respectively (Chen et al. 2014). The correlation of pathogenicity with *B. hominis* is controversial because it can be found in individuals with or without enteric symptoms. It has been surmised that this variation may be due to the aetiological effects produced by different strains of varying virulence (Fouad et al. 2011). Parasitic ingestion is via the faecal-oral route which is speculated to cause predominately gastrointestinal symptoms (Chen et al. 2014) and in the immunocompromised, links have been made to irritable bowel syndrome (IBS), and colon carcinoma (Engsbro et al. 2014, El-Gayar and Mahmoud 2014). *B. hominis* has had prolific scientific attention and yet still presents a formidable and fascinating enigma with many exciting potential research projects that beckon the ambitious scientist.

**Keywords:** Blastocystis, Blastocystis hominis, gastrointestinal diseases, parasite, immunocompromised patient, protozoa, cysts, microscopy, PCR

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The purpose of this paper is to provide a balanced and concise appraisal of the current theories of B. hominis in regards to its potential pathogenicity in humans and to promote the reader’s motivation into further study of this perplexing parasite.

**Clinical disease expression in humans**

Blastocystosis refers to the symptomatic infection with the parasite B. hominis with reported clinical presentations that range from self-limiting gastrointestinal pain to chronic ongoing diarrhoea requiring antiprotozoal medication and often in conjunction with urticaria and itching (Mirza and Tan 2012, Stenzel and Boreham 1996). Other reported symptoms include; anorexia, eosinophilia, bloating, faecal leucocytes, fatigue, fever, flatulence, hepatomegaly, rectal bleeding, and joint pain and swelling (Stenzel and Boreham 1996). The plethora of symptoms are often attributed to variations in virulence between subtypes (ST1-ST9) divided along a diverse genetic spectrum, each speculated to correlate with respective disease states (Stensvold et al 2012).

Isolated case reports implicate B. hominis as the etiologic agent of arthritis, colitis, death, diabetes, enteritis, IBS, leukaemia, severe sepsis, terminal ileitis, peritonitis and ulcerative colitis in immunocompromised patients (Stenzel and Boreham 1996). These single-case study results are controversial because many such studies failed to eliminate other potentially causative concomitant organisms, some of which are also unknown for pathogenicity and many other studies have produced contrary results (Stenzel and Boreham 1996). Additionally, an accurate presentation of true epidemiological prevalence has been thwarted due to reports failing to also include cases that show asymptomatic infections (Mirza and Tan 2012).

Studies have implicated the parasite as associated with severe sepsis (Chen et al 2014), IBS (Engsbro et al 2014) and colon cancer (El-Gayar and Mahmoud 2014) in immunocompromised patients due to the statistical prevalence of their faecal carriage of B. hominis. However other studies have disproved any such prevalence let alone an inferred causation of disease (Mirza and Tan 2012, Engsbro et al 2014).

In light of these contradicting studies, ascribing B. hominis as the etiologic agent for symptoms reported in a patient harbouring this parasite, while ignoring other potential causes such as viruses or toxins is misleading and therefore any such conclusion can only be tentative. Until B. hominis is conclusively shown to cause disease, it would be a mistake to rule out other potential or unknown pathological agents or conditions that are found to co-populate the stool sample in patients with gastrointestinal disease, even if these alternate causes in comparison to B. hominis represent a population minority (Mirza and Tan 2012, Stenzel and Boreham 1996).

**Epidemiology**

B. hominis is a strictly anaerobic protozoan parasite found globally in the lower gastrointestinal tract of humans and many other vertebrate animals. The prevalence of human infection is higher in developing countries (30-50%) compared with industrialised nations (1.5-10%), and this has been attributed to factors such as overcrowding, substandard personal and environmental hygiene, and unclean drinking and bathing water secondary to ineffecual sewerage and waste removal systems (Sohail and Fischer 2005). Distribution studies have reported that B. hominis infection is found to be more prevalent in relatively lower socioeconomic populations within industrialised nations (Stenzel and Boreham 1996). An epidemiology study performed in the Kimberley region of Western Australia to determine intestinal parasite colonisation rates between rural aboriginals and non-aboriginals found that the rural aboriginals were more likely to harbour B. hominis which was attributed to their ‘low socioeconomic status and poor living conditions’ (Brooke et al 2001).

Furthermore, another Australian study in a south east Queensland (SEQ) piggery found that 83.3% of staff were carriers of B. hominis which was attributed to zoonotic transmission due to repetitive exposure to pig faeces during the cleaning of high-density pig pens (Wang et al 2014). To summarise, B. hominis infections are not necessarily restricted to climatic conditions, socioeconomic groups, geographical areas or gender, but only with regard to the living standards or exposure to unsanitary conditions experienced by the group.

It is suggested that the different symptoms expressed between human hosts of B. hominis are analogous to the different and unique pathogenicity of B. hominis subtypes. Although B. hominis subtypes display inter-subtype variations in virulence that may convey particular symptoms, purported links with infection of particular subtypes and corresponding disease states are inconclusive. Furthermore, studies ascertaining the prevalence of B. hominis human infection can only correlate with subtypes one, three and four but cannot infer or predict unique symptomatic outcomes (Coyle et al 2011).

Statistical trends for infection implicate the very
young, elderly, those of low socioeconomic status, and the immunocompromised especially those with human immunodeficiency virus (HIV) (Chen et al 2014, Engsbro et al 2014). Other predisposing factors include the concomitant prior infection of other gastrointestinal parasites which appear to increase the chance of B. hominis infection (Stenzel and Boreham 1996).

The route of transmission of the etiologic agent is by ingestion via the faecal-oral route due to food poisoning, contaminated water, inhalation of aerosolised faeces, mechanical transmission by flies or cockroaches, zoonotic transmission, and sexual practices leading to the incidental ingestion of faecal organisms (Tille 2013).

**Diagnostic techniques**

Conventionally the detection of parasites in stool samples rely on direct smear microscopy, faecal concentrates, or permanently stained smears however problems may arise in morphological diagnosis (Stensvold et al 2006). Concentration methods are controversial because of the reports of this method causing disruption of vacuolar, multivacuolar and granular forms (Stenzel and Boreham 1996). Currently identification methods include direct microscopy of faecal smears, in vitro cultivation of stools with subculturing of forms on fresh media, genotyping by Polymerase Chain Reaction (PCR) using STS rRNA and SSU rDNA primers, and for measures of pathogenicity; histopathologic examinations of recto-sigmoidal biopsies (Fouad et al 2011, Yakoob et al 2010).

Diagnosis is routinely performed using wet mount faecal smears in saline (Figs. 1 & 2), iodine staining, and trichrome (Fig. 3) or iron haematoxylin staining (Fig. 4), however, most laboratories under report the true prevalence of the polymorphic B. hominis by only recognising the vacuolar form because it is preferentially distinguishable from other protozoa (Termmathurapoj et al 2004). In this regard, there are limited laboratory personnel with the skillset to visually detect or differentiate between the vacuolar, granular, amoebic or cystic forms (Fig. 4).

Permanent staining, although more sensitive to wet mount techniques, incorporate polyvinyl alcohol (PVA) and Schaudinn fixative which are potentially hazardous chemicals to laboratory staff and disposing these chemicals is problematic (Zhang et al 2012). Couturier et al (2015) developed a single-vial stool collecting device that uses an alcohol based fixative (Alcorfix) rather than the PVA-formalin fixed method. Their reports indicate that the mini Parasep solvent-free (SF) tubes are used for both collection and concentration, which saves time, and has comparable filtration performance and morphology detection to the PVA-formalin fixed method (Couturier et al 2015). Leelayoova et al (2002) showed that short-term in vitro cultivation of B. hominis in Jones medium is more sensitive than both wet-prep smears and trichrome permanent staining reporting a detection rate six times and two times more respectively. PCR was found to be suitable for genotypic characterisation though insensitive for detection of B. hominis in stool samples (Leelayoova et al 2002). Termmathurapoj et al (2004) proposed a concomitant increase in specificity and sensitivity utilising in vitro cultivation to isolate and extract B. hominis DNA which is then used to facilitate an increased PCR detection upon subsequent application.

A recent study showed that recto-sigmoidal biopsies of those infected with B. hominis displayed a statistical correlation with degree of inflammation and B. hominis genotype I which was proposed as the etiologic agent causing the inflammation (Fouad et al 2011). B. hominis pathogenicity is still largely regarded a scientific enigma thus utilisation of recto-sigmoidal biopsies as a determinant of high-specificity genotype I detection is currently of little value.

Several studies have shown that the cultured growth of B. hominis in various commercially available liquid media (RPMI 1640, Medium 199, and DMEM) resulted in marked improvements in specificity and sensitivity over traditional methods (Zhang et al 2012, Stensvold et al 2012). Furthermore, additional attributes relating to environment safety, preparation, storage and disposal convenience, in congruence with the facility for morphologic differentiation, earmarks short-term in vitro culturing as a superior alternative for clinical diagnosis and field studies over current methods (Leelayoova et al 2002, Stensvold et al 2012, Zhang et al 2012).

Dogruman-Al et al (2015) designed a novel enzyme linked immunosorbent assay (ELISA) for the detection of Blastocystis antigens in stool samples, to be used as a high-throughput automated screening test. This method reported a 92% sensitivity over the 18% sensitivity achieved by direct microscopy and represents a viable alternative to conventional microscopy methods, predominately used in most medical diagnostic laboratories.

**Laboratory diagnosis**

Until an alternative diagnostic method is incorporated into mainstream medical pathology, it is likely that the inherent problems associated with direct microscopy relating to a limitation in morphology experience, will continue to cause high false-negative reports (Elghareeb
et al 2015).

In an effort to promote morphology experience the Centers for Disease Control and Prevention (CDC) provide complementary diagnostic reference resources and training through the Division of Parasitic Diseases (DPDx) website (www.cdc.gov/dpdx). Basic diagnostic guidelines include testing multiple stool samples before reporting negative results due to intermittent shedding, concentrating fixed stool samples to maximise recovery of “cyst-like forms” (Figs. 1-4), and the utilisation of suitable diagnostic techniques based on the available resources of the laboratory (McHardy et al 2014, CDC 2016). The Spontaneous Sedimentation Tube Technique (SSTT) is an inexpensive, albeit time consuming (45 mins) method for separating B. hominis organisms from faecal matter, and reported as achieving higher recovery than direct smear (6% vs. 3%), ether-formalin concentration method (EFCM) (55% vs. 41%), and sulphate zinc flotation technique (FAUST) (34% vs. 3%) (Tello et al 2012).

The CDC precaution against washing specimens in water during concentration procedures, which will lyse the trophozoite forms and result in false negatives (CDC 2016). Additionally, the CDC recommend permanently stained smears (Figs. 3 & 4) over wet mount preparations (Figs. 1 & 2) to avoid a false positive census, by confusing faecal matter, fat globules or yeasts with cysts (CDC 2016). Comparative studies show that trichrome staining enables a higher detection rate (12.3%) over iodine staining (6%) and formal ether concentration technique (10%) (Elghareeb et al 2015). Trichrome staining (Fig. 3) produces a green to grey central vacuole with cytoplasmic inclusion bodies staining bright to dark red (CDC 2016).

Differentiating between faecal matter and the small B. hominis cyst forms is difficult, especially in concentrated wet mount preparations because iodine may also stain vegetative matter, of incidental size (approx. 3-5 µm) and shape (generally round). Studies indicate that cysts are the predominant form of B. hominis recoverable.

Table 1. Morphological features of various B. hominis forms in faeces using trichrome staining

<table>
<thead>
<tr>
<th>Form</th>
<th>Size (µm)</th>
<th>Central vacuole</th>
<th># Nuclei</th>
<th>% recover</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vacuolar</td>
<td>5 - 15</td>
<td>Present</td>
<td>1 - 4</td>
<td>83.1</td>
<td>Large central vacuole</td>
</tr>
<tr>
<td>Cyst</td>
<td>3 - 5</td>
<td>Absent</td>
<td>1 - 4</td>
<td>12.8</td>
<td>Thick cell wall</td>
</tr>
<tr>
<td>Granular</td>
<td>6 - 8</td>
<td>Present</td>
<td>1 - 4</td>
<td>4.2</td>
<td>Granules seen in vacuole</td>
</tr>
<tr>
<td>Amoeboid</td>
<td>3 - 8</td>
<td>Either</td>
<td>1 - 2</td>
<td>4*</td>
<td>Conflicting morphology</td>
</tr>
<tr>
<td>Multivacuolar</td>
<td>5 - 8</td>
<td>Either</td>
<td>1 - 2</td>
<td>-</td>
<td>Rare</td>
</tr>
<tr>
<td>Avacuolar</td>
<td>- 5</td>
<td>Absent</td>
<td>1 - 2</td>
<td>-</td>
<td>Rare</td>
</tr>
</tbody>
</table>

Source: adapted from (Tan 2008, Elghareeb et al 2015) * Rare and exclusive to culture.

Figure 1. Trophozoites of B. hominis in saline wet prep (x400). Note considerable size variation (Photograph: Richard Bradbury)
Figure 2. Trophozoite of *B. hominis* in saline wet prep (x400). Note distinctive refractile nuclei (Photograph: Richard Bradbury)

Figure 3. Trophozoite (bottom) and cyst (middle left) of *B. hominis* with a cyst of *Giardia intestinalis* (top right) in trichrome stain (x1000) (Photograph: Richard Bradbury)
from faecal samples however the vacuolar trophozoite (Fig. 3) is more commonly reported (Table 1), because this form is more easily recognisable (Stenzel and Boreham 1996). Additional factors that contribute to the diagnostic challenge involve morphological alterations due to osmotic changes, time of unixed storage, chosen stain, and culturing (Stenzel and Boreham 1996).

Furthermore, there is conflicting information of structural and ultra-structure morphology differentiating *B. hominis* forms (Stenzel and Boreham 1996).

**Pathogenesis**

The pathogenicity of *B. hominis* is unclear due to many conflicting case study reports. To date studies have shown that *B. hominis* is predominately found as a commensal in asymptomatic carriers and circumspectly present in patients reporting gastrointestinal pathology such as acute or chronic diarrhoea, vomiting, flatulence, bloating, IBS, colon carcinoma, and appendicular peritonitis (Roberts *et al* 2014, Sandoval *et al* 2015, Fréalle *et al* 2015, Mirza and Tan 2012, Stenzel and Boreham 1996). *B. hominis* infection is correlated with the abased living conditions endemic to developing nations and Stenzel and Boreham (1996) astutely postulated that *B. hominis* may become pathogenic "under specific host conditions, such as immunosuppression, poor nutrition, or concomitant infections". Moreover, some studies showed that *B. hominis* were frequently found in stools of IBS patients although no correlation between genotypes and degree of inflammation was present (Fouad *et al* 2011, Poirier *et al* 2012).

A potential link with *B. hominis* and pathogenicity was suggestive in randomised, placebo-controlled trials where gastrointestinal symptoms improved following eradication of *Blastocystis* (Engsbro *et al* 2014). However, symptom improvement may have resulted from the eradication of other cohabiting microorganisms which were not accounted for.

**Clarifying pathogenicity**

A potential way forward to establish *B. hominis* pathogenicity is to collate previous research results and design a project to inclusively substantiate and link past elucidations. This may require the design of a laboratory setting which accounts for the conflicts reported between past case study reports whilst enabling clinical conditions to create and predict pathogenicity.

The problems associated with proving *B. hominis* pathogenicity predominately relate to the apparent inconsistencies between test subjects of different case studies. Any number of the aforementioned correlations may present with or without infection and infection may be symptomatic or asymptomatic. Additionally, infection may comprise any number of the various *B. hominis* genotypes, which in isolation have shown some statistical correlations to pathogenicity and degree.

**Figure 4.** Trophozoite of *B. hominis* in modified iron-haematoxylin stain (x1000). Note size variation and thin cell walls (Photograph: Richard Bradbury)

A step wise approach to proving *B. hominis* pathogenicity is to establish an animal model, whose infection correlates with clinical symptoms and pathogenicity reported in human subjects. Hussein et al. (2008) injected rats with respective *B. hominis* genotypes extracted from symptomatic and asymptomatic human subjects with veritable degrees of gastrointestinal pathology.

The samples taken were exclusively populated with *B. hominis* (testing negative for other common pathogenic organisms), thus ruling out other potential causations. The respective rat reactions were close reproductions of the clinical conditions seen in the corresponding human subjects. Hussein et al. (2008) established subtype 1 as clinically and statistically highly proportional to pathogenicity, subtype 2 as irrelevant, and the presence of pathogenic and non-pathogenic strains among subtypes 3 and 4. All of the rats injected with genotype 1 from symptomatic isolates developed a severe degree of histopathological change. The suitability of rats, as an appropriate animal model, is further emphasised by the results obtained by Fouad et al. (2001), who performed a very similar experiment to Hussein et al. (2008), analysing *B. hominis* genotypes derived from patients with IBS, with near identical results and subsequent conclusions for symptom causality.

Hussein et al. (2008) did not reveal whether the symptomatic human subjects were immunodeficient however it is likely because immunosuppression is indicated as a primary factor in *B. hominis* pathogenicity (Cirioni et al. 1999). Since the rats reproduced corresponding symptoms to the human donors, despite having an assumed healthy immune system, correlations with *B. hominis* and immunodeficiency likely describe a prior condition that predisposes infection rather than an auxiliary condition caused by infection. This concept, drawn from the logical inference of separate studies, may provide an interesting research project. This would involve monitoring the immunological status of rats exposed to various *B. hominis* genotypes, over time, and alternatively, the analysis of symptomatic rats, conditioned for various immunodeficient states, upon exposure to *B. hominis* of different genotypes.

Such results may for instance, prove that immunodeficient individuals are metabolically conditioned to exhibit gastrointestinal disease following *B. hominis* exposure whilst asymptomatic carriers are unlikely to be immunodeficient. Studies show that *B. hominis* promotes its own survival and colonisation in the gut by producing a cysteine protease that disassembles IgA antibodies (Sio et al. 2006, Abdel-Hameed and Hassanin 2011). Its stands to reason, therefore, that an immunodeficient state may be a precursor to *B. hominis* proliferation, leading to microbiome predominance with potentially subsequent and effectual gastrointestinal symptoms.

A similar potential research project may involve the promotion of disease from an asymptomatic carrier of *B. hominis* by creating within the animal model an immunocompromised state. The question might be, does the degree of microbiome predominance, controlled by immunocompromising, correlate with gastrointestinal pathology and subsequent symptom severity? If we are to assume that the rats in the Hussein et al. (2008) experiment were dosed equally and had equivocal immunological states, then the near identical replication of symptoms between human and rat subjects may not only be ascribed to the specific genotype carried through but also to the proportionality of *B. hominis* genotypes to other commensal organisms, within the sample.

A measurement of parasitic population density may well predict symptomatic severity and account for case study disparities relating to the supposition that *B. hominis* represents the aetiological agent responsible for gastrointestinal disease (Tan 2008). Tan (2008) cites many studies for and against a correlation between infection density and symptoms, theorising the discrepancy relates to “genotype differences among *Blastocystis* isolates or to host factors such as age and genetic background variations in the populations studied.” Correlations, therefore, between genotype-specific density and symptoms may resolve current conflicts between case study reports so as to increase the validity for the tenure that *B. hominis* can be pathogenic.

Studies have shown that the constitution of the human intestinal microbiome is highly variable, driven by genetic and environmental factors, and that low microbiome diversity is associated with disease (Eckburg et al. 2005, Wu and Lewis 2013, Putignani 2012). Morton et al. (2015) proved that colonisation of *Entamoeba* species is highly predictable following the detection of a characteristic microbiome profile. There are very few studies investigating intestinal micro-eukaryotic diversity and, to date, no metagenomics studies profiling intestinal microbiomes with *B. hominis* genotypes (Scanlan and Marchesi 2008, Pandey et al. 2012, Morton et al. 2015, Lukeš et al. 2015). Such studies may determine if colonisation of a particular *B. hominis* genotype is predicated, or becomes pathogenic,
by a characteristic microbiome. A greater understanding of the relationship between an intestinal microbiome and its corresponding micro-eukaryotic diversity, may explain the dichotomous nature of B. hominis, and potentially lead to novel treatments with improved efficacy.

**Treatment**

Metronidazole (Nigro et al 2003) and nitazoxanide (Rossignol et al 2005) were found to eliminate Blastocystis with subsequent improvement in gastrointestinal symptoms. Urticaria circumstances associated with B. hominis has been successfully treated with paromomycin (Armentia et al 1992).

Moghaddam et al (2005), Ghadirian and Azami (2005) evaluated treatment with metronidazole and Trimethoprim/Sulfamethoxazole on patients infected with B. hominis and reported a 33% and 22% clearance respectively. However, clinical improvement may be due to the treatment of other coinhabitant species (Coyle et al 2011). In vitro studies have shown B. hominis growth inhibition by emetine dihydrochloride, iodoquinol, furazolidone, metronidazole and trimethoprim/sulfamethoxazole (Moghaddam et al 2005).

Studies of dietary management have shown that infected patients on lactose-free and/or high fibre diets reported proportionately higher clinical improvement than those treated with metronidazole (Stenzel and Boreham 1996). Furthermore, studies have suggested that B. hominis infection treatment with clinical improvement is achieved by diet-mediated alterations of intestinal nutrient levels, redox potential, or the microbiome (Miller and Minshew 1988, Kain et al 1987). These findings suggest a correlation between diet-mediated microbiome and subsequent micro-eukaryotic diversity, parasite proliferation, and symptom severity.

Eukaryotes in the gut act both in consort with the microbiome community and in collaboration or response to the host immune system (Parfrey et al 2011). High through-put sequencing technology (culture independent) has shown that the microbiome bacterial community is a significant determinant of health and disease (Parfrey et al 2011). Future application of this technology into micro-eukaryotic diversity will establish the ecological relationship of B. hominis with other gut microorganisms and the host immune system to be used as a predictive model for pathology (Parfrey et al 2011).

Treatment for B. hominis infection is controversial because there is no unequivocal data confirming pathogenesis, treatment specificity, or that clinical improvement is relative to B. hominis eradication (Coyle et al 2011). High through-put sequencing technology applied to the micro-eukaryotic-microbiome ecology will aid to clarify the relevance of and approach to B. hominis treatment.

**Prevention**

Since infection of B. hominis is linked to poor sanitary living conditions associated with a lower socioeconomic status and the occupational handling of faecal organisms and immunocompetence, control measures include good hygiene, upkeep of personal health and maintenance of sanitary facilities, and community education to prevent environmental contamination and the ingestion of water infiltrated with sewage runoff (Tan 2008, Parkar et al 2010, Tille 2013). The cyst form of B. hominis is not sterilised in chlorinated water because of a strong resistance to it (Zaki et al 1996), however, boiling drinking water or using carbon block filters and ultraviolet light has been correlated with lower infection rates (Leelayoova et al 2004).

Occupationally induced zoonotic transmission may be prevented by interrupting the faecal-oral spread by improved cleaning and animal handling procedures using Personal protective equipment (PPE) such as gloves and a mask. Wang et al (2014) recorded a significantly increased prevalence of B. hominis infection among SEQ piggery workers, compared to Cambodian pig handlers, attributed to the differences in pig pen densities. During the cleaning of high density pig pens SEQ workers use high-pressure hoses which aerosolise the faeces leading to increased zoonotic transmission via oral ingestion (Wang et al 2014). Another study demonstrated the prevalence of B. hominis infection in Perth and Western Australian zookeepers (63%) attributing the use of high-pressure hose cleaning of animal enclosures as incidentally causing the oral ingestion of contaminated water (Parkar et al 2010).

Interestingly all of the Perth Zoo and Western Australian zoo workers that had B. hominis infection did not have other parasites and reported gastrointestinal symptoms. The use of appropriate PPE, such as a mask, would aid in the prevention of zoonotic transmission during the cleaning of animal enclosures.

**Discussion**

The evaluation of the involvement and clinical relevance of B. hominis in human disease is still an enigma particularly because the vast number of studies to date have drawn conclusions from correlations
in isolation to multiple other unconsidered factors, such as the ecological relationship between micro-eukaryotic diversity, microbiome profiles, and immune interactions. Bacterial microbiome analysis by high-throughput sequencing technologies have mapped characteristic microbial communities with predictive correlations to health and disease but these applications into eukaryotic relationships are in relative infancy.

Despite the inherent problems involved in inconsistencies between study conclusions, high false-negative detections that affects epidemiological accuracy, and unpredictable treatment outcomes, the scientific community consensus is skewed towards the probability of *B. hominis* clinical relevancy. The sheer number of *B. hominis* studies performed is becoming burdensome to wade through and until the collaborative primer is found, manuscripts such as this provide a means to conceptualise the many fronts of discovery. Additionally, the author has sought to stimulate interest by hypothesis into potential means of discovery by linking conclusions between studies and by providing the current direction of research.

It is the author’s opinion that an establishment of the micro-eukaryotic ecological interrelationship between a characteristic microbiome and subsequent host immune condition will determine treatment. The author predicts the medical laboratory may shift in part from the current model of diagnosing disease that identifies the aetiological pathogen in isolation to a more holistic characterisation of the hosts microbial ecology with treatment designed to recalibrate the ecology rather than the target approach that produces side effects.

**Conclusion**

*Blastocystis* is currently shrouded in controversy whereby its role in human disease remains unsubstantiated. Isolated case studies corroborating infection with gastrointestinal symptoms are of little value because of the difficulty of excluding causations from other potentially symptomatic etiologic agents. Although molecular tests utilising STS rRNA primers have elucidated subtype differentiation between genotypic strains, no conclusive correlations between *B. hominis* genotype and disease phenotypes have been made.

A statistical relationship exists between infections and abased living conditions endemic to low socioeconomic populations but the definitive causation of subsequent diseases in such populations, and including gastrointestinal symptomology of developed populations, remains unclear. Purported links to clinical conditions such as gastrointestinal disease, IBS, colon carcinoma, and peritonitis are only tentatively based on the inconclusive supposition that *B. hominis* is a contributing factor to pathology. The improvement of clinical symptoms pertaining to the eradication of *B. hominis* by chemotherapy is often reported in clandestine to other coinfections that may contribute to the clinical effect, which reveals that the involvement of *B. hominis* in pathology is only circumstantial.

In order to evaluate the clinical relevance for diagnosis, treatment, and control, the determination of a detailed understanding of biology must be known, which could be achieved using a conglomerate of large randomised, placebo-controlled trials differentiating for age, gender, race, geographical location, socioeconomic status, and clinical condition. Furthermore, the establishment of genotype-specific *B. hominis* pathogenicity may be revealed through comprehensive studies into the interrelationship between intestinal micro-eukaryotic diversity, microbiome ecology, and host immune states. The determination of the *B. hominis* micro-eukaryotic relationship to a characteristic microbiome by high-throughput sequencing technologies is in infancy and may reveal fascinating insights into this enigmatic parasite.

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What are the potential outcomes of allowing Australian consumers direct access to community pathology services? A review of patient initiated testing.

Susan Drummond

School of Health, University of New England, Armidale NSW

Abstract
Pathology differs from many other health and medical services, both in Australia and internationally, in that it is a referred service. The testing is not initiated by the patient (the consumer) themselves but is requested on their behalf by a practitioner who controls what diagnostic or therapeutic laboratory tests will be performed on the patient. With an emerging number of medically sophisticated informed consumers, there has been a major paradigm shift in healthcare, moving from doctor focus to consumer focus. Internationally, governments have enacted policy changes driving healthcare initiatives, including direct access to pathology testing (DAT), for those consumers seeking to take responsibility for their own healthcare outcomes. This review provides a brief overview and discussion of the potential outcomes associated with DAT.

Keywords: direct access testing, self-management, self-requested pathology, consumer-ordered testing, patient initiated testing.

Introduction
Direct access testing, self-requested or consumer-ordered testing as it is sometimes known, allows consumers to take control of their own health care by requesting pathology tests directly from providers independently without the need for a request from their doctor (CHF 2009). Although the notion of direct access to pathology testing originated in the United States of America (USA) over 40 years ago (Soloway 1995), an increasing interest in consumer engagement and greater empowerment of consumers has seen DAT become increasingly popular internationally for those consumers seeking to manage their own healthcare.

Over the years there has been much debate regarding the outcomes of DAT with some expressions of concern raised in the body of literature as well as in position statements from key stakeholders and professional societies. This paper seeks to clarify what the potential outcomes may be of allowing Australian consumers direct access to community pathology services and categorises them into desirable and undesirable outcomes.

Methods
The primary sources of relevant articles for the review were through the following electronic databases: Emerald Fulltext, Science Direct (Elsevier), Pubmed and MEDLINE (MEDical Literature Analysis and Retrieval System online) using search engines Google and Google Scholar as well as published literature reviews, with no literature published after June 2015 being included in the review.

To assist in the process of successfully identifying and locating the appropriate research literature a number of key terms and words were employed to help define the search. These included direct access testing, consumer direct access, self-management, patient-centred healthcare and self-requested pathology. Only those publications with the objective of identifying potential desirable and undesirable outcomes of DAT were included in the review.

Results
Due to the limited number of peer reviewed publications, consumer group and key stakeholder perspectives were included in the review a total of 10 citations were included in the study, nine of which originated from the USA where consumers have embraced direct access to laboratory testing, bypassing the traditional healthcare model of practitioner initiated testing. The objective of the study was to identify and quantify potential desirable and undesirable outcomes of DAT (Table 1).
### Table 1. Summary of findings

<table>
<thead>
<tr>
<th>Origin</th>
<th>Citation</th>
<th>Year</th>
<th>Desirable outcomes</th>
<th>Undesirable outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>American Society for Clinical Pathology</td>
<td>2015</td>
<td>Accessibility privacy and confidentiality</td>
<td>Inappropriate testing&lt;br&gt;Liability of reporting abnormal results&lt;br&gt;Reimbursement of costs&lt;br&gt;Negative impact of health status&lt;br&gt;Lack of follow up on findings&lt;br&gt;Increase in false positive/negative results</td>
</tr>
<tr>
<td>Australia</td>
<td>Commonwealth Department of Health and Ageing</td>
<td>2013</td>
<td></td>
<td>Lack of evidence based testing&lt;br&gt;Inappropriate testing&lt;br&gt;Inappropriate result interpretation</td>
</tr>
<tr>
<td>USA</td>
<td>Plebani, Laposata &amp; Lundberg</td>
<td>2011</td>
<td>Time savings&lt;br&gt;Monetary savings&lt;br&gt;Privacy and confidentiality&lt;br&gt;Consumer empowerment</td>
<td>Inappropriate result interpretation&lt;br&gt;Psychological consequences&lt;br&gt;Emotional consequences&lt;br&gt;Lack of follow up of findings&lt;br&gt;Lack of treatment</td>
</tr>
<tr>
<td>USA</td>
<td>Lippi, Favaloro &amp; Plebani</td>
<td>2011</td>
<td>Increase knowledge&lt;br&gt;Time savings&lt;br&gt;Monetary savings&lt;br&gt;Privacy and confidentiality</td>
<td>Inappropriate testing&lt;br&gt;Lack of result interpretation</td>
</tr>
<tr>
<td>USA</td>
<td>Davies</td>
<td>2008</td>
<td>Accessibility&lt;br&gt;Consumer empowerment&lt;br&gt;Time savings&lt;br&gt;Monetary savings&lt;br&gt;Privacy and confidentiality</td>
<td>Inappropriate testing&lt;br&gt;Lack of follow up of findings&lt;br&gt;Liability of reporting abnormal results&lt;br&gt;Increase in false positive/negative results</td>
</tr>
<tr>
<td>USA</td>
<td>Wilkinson &amp; Pontius</td>
<td>2003</td>
<td>Monetary savings&lt;br&gt;Convenience&lt;br&gt;Time savings&lt;br&gt;Privacy and confidentiality&lt;br&gt;Promotion of self control</td>
<td>Restrictions to test availability&lt;br&gt;Inappropriate result interpretation&lt;br&gt;Promotes pathology as a commodity rather than a service</td>
</tr>
<tr>
<td>USA</td>
<td>Schulze</td>
<td>2001</td>
<td></td>
<td>Liability of reporting abnormal results&lt;br&gt;Lack of result interpretation&lt;br&gt;Restrictions to test availability</td>
</tr>
<tr>
<td>USA</td>
<td>Halsey</td>
<td>2000</td>
<td>Increase knowledge&lt;br&gt;Time savings&lt;br&gt;Privacy and confidentiality&lt;br&gt;Accessibility&lt;br&gt;Consumers take an active role</td>
<td></td>
</tr>
<tr>
<td>USA</td>
<td>Soloway</td>
<td>1995</td>
<td>Privacy and confidentiality</td>
<td>Monetary cost&lt;br&gt;Counseling unavailable&lt;br&gt;Communication of results&lt;br&gt;Liability of reporting abnormal results&lt;br&gt;Inappropriate result interpretation</td>
</tr>
<tr>
<td>USA</td>
<td>Soloway</td>
<td>1990</td>
<td>Convenience&lt;br&gt;Accessibility&lt;br&gt;Proactive health management&lt;br&gt;Promotion of doctor/patient partnership&lt;br&gt;Abandonement of doctor gatekeeper role</td>
<td>Inappropriate testing&lt;br&gt;Accuracy and safety of testing</td>
</tr>
</tbody>
</table>
Determination of the frequency and relative frequency (%) of the occurrence of desirable and undesirable outcomes in the studied literature were also tabulated to identify possible themes (Table 2). Themes included inappropriate testing, lack of result interpretation, liability of reporting of abnormal results and the absence or presence of legal disclaimers indicating test results are not for diagnostic or prognostic use but rather are provided for informational purposes only.

**Discussion**

**Desirable outcomes**

Privacy and confidentiality were the most frequently documented desirable outcome. This may be attributed to the majority of literature studied emanating from the USA which has a vastly different healthcare system to that seen in Australia. Healthcare consumers may not wish to have tests results appear in their personal health records if they have a predisposition to a chronic disease or cancer, as health insurance premiums may be affected by the health status of the individuals covered by the policy. Similarly monetary savings were described as being desirable with large out-of-pocket costs associated for medical treatment in the USA (Halsey 2000; Davies 2008).

Consumer empowerment was an outcome frequently mentioned. This was also seen in studies by Zikmund-Fisher et al (2010) and Kerns et al (2013) who indicate consumers are increasingly seeking more meaningful involvement in their medical decision making. Time savings and convenience or accessibility were seen as being highly desirable with these attributes mentioned frequently.

This is reflective of the American culture where independence and an expectation that personal needs, including healthcare needs, will be satisfied quickly, without necessitating third party involvement or approval. According to Wilkinson and Pontius (2003), more and more Americans are demanding an active role in managing their healthcare.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Frequency (n)</th>
<th>Relative frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Desirable</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Privacy and confidentiality</td>
<td>7</td>
<td>25.5</td>
</tr>
<tr>
<td>Accessibility and convenience</td>
<td>6</td>
<td>19.3</td>
</tr>
<tr>
<td>Time savings</td>
<td>5</td>
<td>16.1</td>
</tr>
<tr>
<td>Monetary savings</td>
<td>4</td>
<td>12.9</td>
</tr>
<tr>
<td>Consumer empowerment</td>
<td>4</td>
<td>12.9</td>
</tr>
<tr>
<td>Increase knowledge</td>
<td>2</td>
<td>6.5</td>
</tr>
<tr>
<td>Proactive health management</td>
<td>1</td>
<td>3.2</td>
</tr>
<tr>
<td>Promotion of doctor/patient partnership</td>
<td>1</td>
<td>3.2</td>
</tr>
<tr>
<td>Abandonment of doctor gatekeeper role</td>
<td>1</td>
<td>3.2</td>
</tr>
<tr>
<td><strong>Undesirable</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate or lack of result interpretation</td>
<td>7</td>
<td>21.2</td>
</tr>
<tr>
<td>Inappropriate or lack of evidence based testing</td>
<td>6</td>
<td>18.1</td>
</tr>
<tr>
<td>Liability/ communication of abnormal results</td>
<td>5</td>
<td>15.2</td>
</tr>
<tr>
<td>Lack of follow up of findings/treatment</td>
<td>4</td>
<td>12.1</td>
</tr>
<tr>
<td>Increase in false positive/negative results</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Counseling unavailable</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Restrictions to test availability</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Reimbursement/monetary costs</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Psychological/emotional consequences</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Accuracy and safety of testing</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

*Table 2.* The frequency and relative frequency (%) of desirable and undesirable outcomes in the studied literature.
A further five desirable outcomes were identified although their relative frequency in the literature was low. Increased knowledge and proactive management of health were identified infrequently as desirable outcomes yet they stand to have a big impact on healthcare budgets in the future. Evidence suggests effective self-management of chronic diseases such as heart disease and diabetes results in a reduction in the frequency and duration of hospitalisation thus reducing the burden and costs associated with hospitalisations (Hamar et al 2013). With chronic disease now contributing to over 70% of disease burden in Australia, a figure that is expected to increase to 80% by 2020 (Jordan and Osborne 2007), proactive self-management of health has been identified as a key action area.

Evidence suggests that patients with effective self-management skills have improved health and make better use of health professionals’ time, utilize less services and effectively reduce costs (Barlow, Turner and Wright 2000; Lorig et al 2001). Similarly, research by Hibbard and Greene (2013) finds evidence for improved health outcomes as individuals gain skills and confidence in managing their own healthcare, whilst ASCP (2015) suggest DAT has the potential to benefit some patients and enhance the doctor-patient relationship.

A review prepared for the Australian Government Department of Health and Ageing (2013), stated that 75% of healthcare providers participating in their survey did not think there were any potential risks to the health of individuals or the community associated with non-Medicare pathology services, such as DAT.

Undesirable outcomes

Inappropriate result interpretation and testing were the most frequently recorded undesirable outcomes identified in the literature with a relative frequency of 21.2% and 18.1% respectively. This was followed by liability and communication of abnormal results. Central to this, is research evidence suggesting there are at least three groups of proactive consumers who seek DAT.

The so called “worried well” who may initiate testing to confirm their health status, the risk-assessment testers whose lifestyle or professions place them at increased risk of diseases and the health monitors, those who test because they have a certain disease or illness requiring monitoring (Halsey 2000). The literature surrounding inappropriate testing discusses the risk of obtaining abnormal results which do not necessarily reflect an underlying pathology (Lippi et al 2011).

This lack of evidence based testing may result in false positive or negative results as reference ranges are established on a presumably normal, healthy population, which may in turn prompt further unjustified testing and additional costs. The chance of an individual having a given disease or illness is dependent on the sensitivity of the test, i.e. the portion of the population who test positive, and the specificity of the test, i.e. those without disease who test negative.

A perfect test is never positive in a patient who is disease free and is never negative in a patient who is in fact diseased, however many diagnostic tests fall short of this ideal. Sensitivity and specificity are population measures and do not provide diagnostic information for any one individual. Positive and negative predictive values are therefore used to aid in interpretation of test results to help determine what the probability is that the individual truly has a disease (Artsob 1993).

This measure is dependent on the population chosen and the prevalence of the disease. An example of this is the large number of false positive Lyme disease western blot results returned from American testing laboratories on Australian patients in the absence of the recommended positive ELISA more sensitive screening test and the probable absence of Lyme disease in Australia. Because of this, regulations have been placed in some countries and states restricting the types of tests available.

Inappropriate result interpretation was a factor identified with high frequency which has been addressed in some states, such as California in the USA, which has determined results should be conveyed in plain language in oral, written or electronic form. Laboratories may also provide recommendations for the consumer to seek professional medical advice from their healthcare provider.

Whilst a number of other undesirable outcomes were identified in lesser frequency, many have been easily overcome in recent years with greater legislative controls and regulations placed on laboratories. The ASCP (2015) recognises that whilst direct access testing is becoming increasingly popular, it recommends patients attend a regulated or certified laboratory and discuss results with their physician in order to ensure optimal patient health outcomes.

Many people only seek advice from a doctor after they exhibit symptoms of illness or become ill. Testing initiated by a doctor may require a number of consultations with the patient at great cost to both the patient and or Medicare with the patient requiring an initial visit often when clinical symptoms of illness are already
being experienced by the patient, a consultation to obtain test results as well as follow up consultations if further monitoring is required.

With DAT costs may be reduced with the patient only requiring one consultation for which they may be better educated and prepared to discuss possible concerns and treatment options, saving both time and money. Furthermore, a recent report by the American College of Physicians (ACP 2014) recommends mandating of direct access to laboratory testing with anticipated benefits including patients having increased control over their personal health information, facilitating their ability to receive this information in a timely manner and reinforcing patients’ active participation in their healthcare.

Conclusion
To date DAT has demonstrated few problems, none of which are unable to be addressed through education and regulation. With the highly regulated pathology industry in Australia, the risk of undesirable health outcomes is minimal. Whether Australian consumers want DAT remains a topic for further investigation.

References


Schulze M 2001. 25 percent more states allow direct access testing. Lab Med 32: 661-664.


The AIMS Fellowship is an attractive and highly competitive option to academic post graduate degrees.

The Fellowship is recognised by the Department of Health and Ageing as meeting the requirements for the supervision of GX and GY laboratories.

<table>
<thead>
<tr>
<th>TRANSFUSION SCIENCE</th>
<th>Qualification for the Fellowship is by EXAMINATION in one of eight disciplines</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLINICAL BIOCHEMISTRY</td>
<td>Candidates for the Fellowship must have been AIMS members for a minimum of two years and must meet certain other criteria</td>
</tr>
<tr>
<td>CYTOLOGY</td>
<td>The Fellowship Program is modular – candidates must complete:</td>
</tr>
<tr>
<td></td>
<td>★ Two compulsory modules</td>
</tr>
<tr>
<td></td>
<td>★ Two elective modules</td>
</tr>
<tr>
<td></td>
<td>★ A viva voce examination</td>
</tr>
<tr>
<td></td>
<td>★ A scientific dissertation</td>
</tr>
<tr>
<td>HAEMATOLOGY</td>
<td>Candidates have up to five years to complete the Fellowship Program</td>
</tr>
<tr>
<td>ANATOMICAL PATHOLOGY</td>
<td>To enrol in the Fellowship Program or for further information please contact the AIMS National Programs Manager:</td>
</tr>
<tr>
<td>IMMUNOLOGY</td>
<td>Phone: +61 7 3876 2988.</td>
</tr>
<tr>
<td>MICROBIOLOGY</td>
<td>Email: <a href="mailto:programs@aims.org.au">programs@aims.org.au</a></td>
</tr>
<tr>
<td>GENERAL (including Core Laboratory)</td>
<td></td>
</tr>
</tbody>
</table>

Australian Journal of Medical Science | February 2016 | Vol. 37 | No. 1
A 39-year-old male presented at the Emergency Department with a history of lassitude and pallor for the last six months. A full blood count was performed with the following results:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>39 g/L</td>
<td>130-180 g/L</td>
</tr>
<tr>
<td>MCV</td>
<td>107.0 fL</td>
<td>80-100 fL</td>
</tr>
<tr>
<td>MCH</td>
<td>34.2 pg</td>
<td>26.5-33.0 pg</td>
</tr>
<tr>
<td>WBC</td>
<td>9.17 x 10^9/L</td>
<td>3.5-11.0 x 10^9/L</td>
</tr>
<tr>
<td>Platelet</td>
<td>30 x 10^9/L</td>
<td>150-400 x 10^9/L</td>
</tr>
<tr>
<td>Neutrophil</td>
<td>5 %</td>
<td></td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>95 %</td>
<td></td>
</tr>
<tr>
<td>Monocyte</td>
<td>0.0 %</td>
<td></td>
</tr>
<tr>
<td>Eosinophil</td>
<td>0.0 %</td>
<td></td>
</tr>
<tr>
<td>Basophil</td>
<td>0.0 %</td>
<td></td>
</tr>
<tr>
<td>Neutrophil</td>
<td>0.5 x 10^9/L</td>
<td>1.7-7.0 x 10^9/L</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>8.7 x 10^9/L</td>
<td>1.5-4.0 x 10^9/L</td>
</tr>
<tr>
<td>Monocyte</td>
<td>0.00 x 10^9/L</td>
<td>0.1-0.8 x 10^9/L</td>
</tr>
<tr>
<td>Eosinophil</td>
<td>0.00 x 10^9/L</td>
<td>0.04-0.44 x 10^9/L</td>
</tr>
<tr>
<td>Basophil</td>
<td>0.00 x 10^9/L</td>
<td>0.0-0.2 x 10^9/L</td>
</tr>
</tbody>
</table>

The full blood count showed anaemia and thrombocytopenia. The red cells were macrocytic and rouleaux was markedly increased.

The white cells showed a neutropenia and monocytopenia and there was a lymphocytosis present. The lymphocyte count included 52% abnormal lymphoid cells with eccentric, oval shaped nuclei, basophilic cytoplasm with numerous cytoplasmic villi. These cells resembled ‘hairy cells’. The blood film was consistent with hairy cell leukaemia.

A bone marrow aspirate and trephine were performed and examined.

The bone marrow aspirate showed erythroid precursors with normal maturation. Granulopoiesis was predominantly normal as was megakaryopoiesis. A large proportion of the lymphocytes were abnormal in appearance with similar morphological features to those seen in the peripheral blood.

The bone marrow trephine showed a heavy infiltrate of widely spaced lymphoid cells with oval nuclei surrounded by halos of pale staining cytoplasm. Erythropoiesis was markedly reduced. Granulopoiesis was left shifted while megakaryocytes were present in patchy distribution. Flow cytometry was performed on the peripheral blood with the following immunophenotype:

CD45+/HLA-DR+/CD19+/CD20+/CD22+/CD5-/CD10-/CD23-/CD25+/FMC7+/CD00c+/CD103+/CD123+/CD27+/Lambda+

CD25 = 72%++
CD27 = 66%+
CD123 = 73%++

Flow cytometry identified a monoclonal B-cell population of lymphoid cells. Approximately 64% of the lymphocytes had the above hairy cell phenotype. This was a case of hairy cell leukaemia, classical form. Cytogenetic studies were not performed on this patient.

Patients with hairy cell leukaemia usually present with a splenomegaly, pancytopenia and monocytopenia. Hairy cell leukaemia is an indolent neoplasm occurring more often in males than females with a ratio of 5:1 and a median age of 50 years. There are two forms of hairy cell leukaemia; the classical form and the variant form.

The classical form is characterised by cells which vary in size from 10-20μm in diameter. The nucleus is eccentric and round to oval in shape with basophilic cytoplasm with many fine hair-like projections around the entire circumference. The variant form is charac-
characterised by smaller cells with a diameter of 10-15\(\mu\)m. The nucleus is generally centrally placed rather than eccentrically placed. The white cell count is much higher compared with the classical form; it can be as high as 100 x 10⁹/L.

The bone marrow usually shows a patchy or diffuse infiltrate and may result in a ‘dry tap’. The trephine typically shows an infiltrate of lymphocyte nuclei surrounded by halos of pale-staining cytoplasm.

The immunophenotype of the classical form is CD25+ while the variant is CD25-.

The patient in this case study was treated with the purine nucleoside analogue cladribine. The patient became severely neutropenic as a result of this treatment and remained so for a period of four weeks. Patients treated with cladribine usually achieve a high rate of remission.
Small bowel diverticulum

Piero Nelva, Hock Kua, Ann Niap, Stephen Bare

Anatomical Pathology, Monash Medical Centre, Clayton, Victoria

Diverticulum in the small intestine

A diverticulum is a circumscribed pouch or sac occurring normally or created by herniation of the lining mucous membrane through a defect in the muscular coat of a tubular organ. A diverticulum may be present in the stomach, the small intestine or colon. Most diverticula are asymptomatic until becoming inflamed and are typically detected by radiography after the ingestion of a radiopaque substance (Ferreira-Aparicio et al 2012).

Most cases of diverticulosis occur in patients over the age of 40 years and clinical evidence is seen in slightly over 10% of individuals in this age group. The incidence of diverticulosis is likely to be higher however, with an Australian study finding evidence at autopsy of diverticulosis in 45% of 200 examined large bowels. Diverticula preferentially involve the left side of the colon and the majority of duodenal diverticula are thought to be acquired as a result of herniation through a defect in the muscularis externa, caused by the entrance of large vessels.

The disease is common in North America, Europe and Australia, but unusual in Asia and Africa. The main protective factor seems to be a high-fibre diet, which is thought to act by diminishing the degree of colonic segmentation which is the mechanism responsible for mucosal herniation.

Symptoms of diverticulosis include abdominal cramping and spasms of unexplained cause, diarrhoea and altered bowel habits. Faecal material trapped in the diverticulum can cause bleeding, inflammation and infection giving rise to diverticulitis.

In cases of small bowel diverticuli, a diagnosis of Meckel’s diverticulum (MD) must be excluded. Meckel’s diverticulum is a congenital defect related to the failure of the vitelline duct (which joins the intestine to the yolk sac in utero) to completely atrophy and be subsequently resorbed. The presence of a MD can be associated with other congenital and structural abnormalities and certain malignancies.

Macroscopically diverticuli have a flask-like shape and may be filled with faeces, mucin or other colonic contents. Microscopically the diverticulum lacks a muscle layer except for the residual bundles of muscularis mucosae (Rosai 2004).

Treatment of the condition is initially by withholding solid foods, often accompanied with IV fluids the introduction and maintenance of a high fibre diet. As a last resort surgical resection of the colon segment containing the diverticulum may be required.

Laboratory investigations

The histology laboratory received a 120mm long segment of small bowel. In the mid-section was a 20 mm diverticulum containing faecal material and an embedded red chilli. There was an abscess where the diverticulum abuts the serosa.

Microscopically, the sections showed small bowel mucosa lining a diverticulum. Vegetable matter is contained within the diverticulum. The mucosa is poorly preserved. There is a peri-diverticular abscess with a collection of neutrophils and fibrin, and foci of fat necrosis.

The patient was discharged from hospital after a short stay. Outpatient follow up recorded that they are well, surgical wounds have healed and bowel habits have returned to normal.

Figure 1. Segment of small bowel. Normal colonic mucosa (black arrows) and area of ulceration and abscess (green arrow).
References


Figure 2. (left) Colonic mucosa (purple arrow) lining the segment of bowel. Normal muscularis propria (black arrows) at the edges of the diverticulum becomes attenuated and disappears at the bottom of the diverticulum. Vegetable matter in diverticulum is indicated by the red arrow. Whole mount x1.

Figure 3. (below) Ulcerated bowel wall. Note loss of villous architecture amid inflammatory cell infiltrate. H & E x20.
Updated Review: Molecular Virology and Control of Flaviviruses
Edited by Pei-Yong Shi
Caister Academic Press 2012
Hard Cover  X + 358 pages
RRP: US $360

The genus Flavivirus includes some of the most important emerging arboviruses (i.e. ARthropod BOrne viruses) known to cause human diseases. Dengue virus (DENV), West Nile virus (WNV), Japanese encephalitis virus (JEV), and yellow fever virus (YFV) are globally important flaviviruses. Broadly, fever-arthralgia-rash, viral haemorrhagic fever with or without hepatitis, and central nervous system disease are clinical syndromes caused by flaviviruses.

Chapters in the book are written by well-known arbovirologist and review a comprehensive coverage of virion structure and function, transmission and vector control, diagnosis, vaccine and therapeutics. The book begins with the chapter “flaviviruses: past, present and future”, where the chapter provides a detailed history, summarises the current situation and predicts future trends in regard to flavivirus infections. It also gives a clear understanding of how mosquitoes were spread from one continent to another as a result of the trade. Subsequent chapters describe viron structure, structural and non-structural proteins, replication, assembly, fitness and transmission, immunity, vaccine development and progress, and vector control strategies. Each chapter is interesting and presented in depth, and also comprehensively referenced.

The ‘Flavivirus Fitness and Transmission’ chapter describes quasispecies theory very well and explains how it has been used to predict evolution and adaptation of the viruses. Although the book does not have a separate epidemiology chapter for each virus, the ‘Flavivirus Vaccine’ chapter covers this topic. Flaviviruses are transmitted by an array of mosquito species, whose description in the book is inclusive and thorough. The chapter illustrates the strategies of vector control from conventional control methods through to currently undergoing research (use of Wolbachia – bacteria that infects arthropods).

Chapter ‘Flavivirus Diagnostics’ has discussed each and every aspects of diagnostic measures (virus isolation, nucleic acid detection, serological test and neutralization test) in brief. However, the review could have been discussed in depth.

In summary, the book provides a valuable and absorbing window to one who is in early phase of entry to flavivirus research; reading this book thoroughly contributes a strong base in the field. It should also be very useful for graduate students and experts.

Mr Narayan Gyawali
PhD Scholar, Arbovirology
Central Queensland University, North Rockhampton
Australian Professional Acknowledgement of Continuing Education (APACE)

3 APACE credits per set of questions will be awarded if at least 8 out of 10 questions are answered correctly. 24 credits maximum per accreditation period claim.

Journal-based CPD No. 47
Page 1 of 1

Questions relating to the article ‘Blastocystis hominis: the fascinating enigma’ (refer page 2 of this journal).

1. Genotypically B. hominis comprises 15 subtypes. True/False

2. Studies indicate that cysts are the predominant form of B. hominis recoverable from faecal samples. True/False

3. Metronidazole and nitazoxanide were found to eliminate Blastocystis with subsequent improvement in gastrointestinal symptoms. True/False

4. B. hominis is thought to cause irritable bowel syndrome (IBS), and colon carcinoma in the immunocompromised patient True/False

5. It is the most common parasite found in the lower gastrointestinal tract of humans. True/False

6. B. hominis is a chloroplastic algae that belongs to the phylum Stramenopila. True/False

7. PCR was found to be suitable for genotypic characterisation and sensitive for detection of B. hominis True/False

8. Occupationally induced zoonotic transmission may be prevented by improved cleaning and animal handling procedures and using PPE. True/False

9. The Spontaneous Sedimentation Tube Technique for separating B. hominis organisms from faecal matter is reported as achieving lower recovery than direct smear, ether-formalin concentration method and sulphate zinc flotation technique True/False

10. B. hominis is a strictly aerobic protozoan parasite found globally in the lower gastrointestinal tract of humans and many other vertebrate animals True/False

Name: __________________________________________

Email: __________________________________________

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AJMS APACE Questions, AIMS National Office, PO Box 1911, Milton Qld 4064. Facsimile: 61 7 3876 2999
Australian Professional Acknowledgement of Continuing Education (APACE)

3 APACE credits per set of questions will be awarded if at least 8 out of 10 questions are answered correctly. 24 credits maximum per accreditation period claim.

Journal-based CPD No. 47
Page 1 of 1

Questions relating to the article ‘What are the potential outcomes of allowing Australian consumers direct access to community pathology services?’ (refer page 14 of this journal).

1. In the USA consumers have embraced direct access to laboratory testing, bypassing the traditional healthcare model of practitioner initiated testing. True/False

2. Inappropriate result interpretation was not a factor identified with high frequency in the USA. True/False

3. Direct access testing allows consumers to request pathology tests directly from providers independently without the need for a request from their doctor. True/False

4. Privacy and confidentiality were the most frequently documented desirable outcome. True/False

5. 85% of healthcare providers participating in a Department of Health and Ageing survey did not think there were any potential risks to the health of individuals or the community associated with non-Medicare pathology services, such as DAT. True/False

6. With DAT costs may be reduced with the patient only requiring one consultation for which they may be better educated and prepared to discuss possible concerns and treatment options, saving both time and money. True/False

7. Positive and negative predictive values are used to aid in interpretation of test results to help determine what the probability is that the individual truly has a disease. True/False

8. Consumers are increasingly seeking more meaningful involvement in their medical decision making. True/False

9. Self-management of chronic diseases such as heart disease and diabetes does not result in a reduction in the frequency and duration of hospitalisation. True/False

10. Ten papers were used for this study, five of which originated from the USA. True/False

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Acknowledgements

Acknowledge individuals who have made substantial contributions to the study including technical work and financial support. Authors are responsible for obtaining consent from all the individuals acknowledged by name as inclusion may be interpreted as an endorsement of the article's contents.

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Style manual for authors, editors and printers 2002. 6th ed. John Wiley & Sons Australia Ltd.


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