Letters to the Editor

Blastocystis hominis

Dr. Qadri's reply to Markell and Udkow's letter (E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. **28**:1085–1086, 1990), as well as his article on the clinical significance of *Blastocystis hominis* (2), should leave clinical and laboratory scientists with an uneasy feeling. He states that ". . . Markell and Udkow's opinion is contrary to increasing evidence of the pathogenic potential of *B. hominis* . . . ." In fact, all the information and references provided by Qadri do nothing more than indicate that there is an association between gastroenteritis and finding *B. hominis* in the stool. Some patients with diarrhea have the organism in their stool and some do not; some asymptomatic patients have it in their stool and some do not. Real evidence of pathogenicity is lacking.

Markell and Udkow comment on the "guilt by association" phenomenon which has grown up around this organism in the recent literature. Stated another way, simply repeating continuously the statement that *B. hominis* is a pathogen will not make it so. In addition to the fact that there is no clear segregation of this organism between symptomatic and asymptomatic persons, we need to remember the following: (i) Koch's postulates have never been satisfied (there is no reproducible model of experimental infection due to *B. hominis* only), (ii) no pathologic evidence of or immunologic response to "infection" has been demonstrated in humans, (iii) no mechanisms of pathogenicity, such as toxin elaboration, attachment to intestinal mucosa, or invasiveness, in humans have been described, (iv) no antimicrobial agent which is uniquely active against *B. hominis* has been shown to reliably eradicate both the organism and the diarrhea, and (v) there has never been a point-source outbreak of gastroenteritis in which epidemiologic evidence suggested that *B. hominis* was the cause.

Does propagation of an unsubstantiated claim do harm? You bet it does! At least one popular handbook of antimicrobial therapy now lists a "treatment" for *B. hominis* infections (3). The implication is that there is a disease here worth treating. If *B. hominis* is not a pathogen, then many patients will be unnecessarily treated and the real cause of their disease may be obscured. In addition, there was recently published an article stating that *B. hominis* was the cause of infective arthritis (1). Apparently, the authors and journal editors felt that since this was such a well-accepted pathogen, no scientific documentation was necessary. The article included no illustrations or cultural results to confirm that what was observed was actually *B. hominis*. Now this article is forever embedded in published and computerized reference sources (that is how I found it) and will undoubtedly be quoted as further evidence for the pathogenicity of *B. hominis*.

The scientific literature, including the *Journal of Clinical Microbiology*, must share some responsibility for this situation. *B. hominis* may, indeed, someday be proven to be pathogenic. However, it is time to stop publishing articles which provide no new information on this subject and to insist that scientific documentation replace speculation concerning its pathogenicity.

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Author's Reply

Realizing the limitations of a retrospective study, we concluded that *Blastocystis hominis* should be considered as a causative agent in patients with "recurrent symptoms, especially when the parasite is present in large numbers in fecal specimens in the absence of other known pathogens" (7). As Dr. Rosenblatt points out, we had referred to the pathogenic "potential" of this organism in response to an earlier letter (E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. **28**:1085–1086, 1990). Although none of these statements explicitly establishes *B. hominis* as a pathogen, both imply its association with gastroenteritis.

I agree that there is no segregation of this organism between symptomatic and asymptomatic persons. As Kain et al. (4) stated, "this is not dissimilar to *Entamoeba histolytica* and *Giardia lamblia* which are frequently shed in low numbers and found in a considerable percentage of asymptomatic individuals." To this list one can add many other microbes.

Dr. Rosenblatt has raised some other important and interesting points. Koch's postulates were laid down in the early days of bacteriology and were effective in establishing the causative agents of most bacterial diseases, but they are not always fulfilled for all microbes. Experimental infection of diarrhea in guinea pigs and *B. hominis*-associated diarrhea in nonhuman primates has been described (6, 13; C. H. Zierdt, Clin. Microbiol. Newsl. **5**:57–59, 1983). Cohen (2) and Russo et al. (8) reported cases of *B. hominis* enteric disease in which colonoscopy and mucosal biopsy revealed sigmoid diverticulosis, friability, and acute nonspecific colitis without tissue invasion.

Several investigators have made observations regarding the association of *B. hominis* with epidemics of diarrhea in the tropics and subtropics, recent travel to or immigration from the tropics, and consumption of untreated water (1, 2, 9–12).

Few antimicrobial agents are uniquely active against a particular organism. The recent literature describes the eradication of *B. hominis* from stools with resolution of gastrointestinal symptoms by metronidazole in over 120 patients (3, 7, 8; Zierdt, Clin. Microbiol. Newsl.). During the presentation of our paper at the 1989 annual meeting of the American Society for Microbiology, several people personally related their similar experiences to me.

With regard to the report on infective arthritis due to *B.*

hominis (5), contrary to Dr. Rosenblatt's allegation, the authors appear to have performed normal laboratory tests, including routine aerobic and anaerobic cultures of blood, synovial fluid, urine, stool, and endocervix (all negative). Had the authors included a photomicrograph of the organism as "scientific documentation" or "illustration," most referees probably would have suggested deletion because the parasite is so well described and easy to identify. Dr. Rosenblatt appears to question the integrity and competence of the investigators. Is it because of their affiliation with an institution in Jamaica? Probably they see more parasites in a month than a comparably sized hospital in the United States in 1 year.

Neither I nor some 20 other investigators, reporting over 500 cases of *B. hominis*-associated gastroenteritis, and 2 with opposing views (16 cases) have any vested interest in the organism or its pathogenicity. All have reported their observations and experiences in peer-reviewed journals. Editors and referees of scientific and medical periodicals judge manuscripts on their merit, scientific documentation, and proper interpretation. Expecting them to be narrow-minded is defeating their purpose.

I concur with Dr. Rosenblatt and several previous investigators that more information is needed regarding the epidemiology, mechanism of pathogenicity, immunologic response, radiologic and endoscopic findings, and management of *B. hominis* infections and hope that the controversy surrounding this parasite's role in disease will stimulate research to elucidate these parameters.

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Selective Staphylococcal Broth

We compliment Dr. Cookson et al. on their extremely informative article on staff carriage of epidemic methicillin-resistant *Staphylococcus aureus* (MRSA) (3). The authors make an extremely pertinent statement: "we used broth enrichment culturing to increase the sensitivity of detection of EMRSA [epidemic MRSA] carriage; in fact, without it carriage would have been entirely missed in eight of our nurses." The use of this most sensitive culture method for the detection of MRSA in epidemiological surveillance cannot be overemphasized. In a recent review of MRSA (1), each of the epidemiological investigations cited used direct plating methods and showed that the carriage rate for MRSA was low. It is our contention that the use of insensitive culture methods for MRSA in epidemiological surveillance is one possible reason for the low carriage rates for MRSA reported in the literature.

In a comparison of plated media, including Baird-Parker, Trypticase soy with 5% sheep blood (BAP), and chocolate agar with staphylococcal broth, we have been able to demonstrate the isolation of almost three times as many positive *S. aureus* cultures as by any plated agar method (unpublished data). Recently, one of us reported that a selective staphylococcal broth (Difco Laboratories, Detroit, Mich.) proved superior to direct plating for the recovery of *S. aureus*, resulting in improved recovery rates of 20% (nares) and 66% (vaginal vestibule) (4). Referring to these data, Campos (2) suggested the use of a broth enrichment for detecting MRSA in surveillance cultures. We have also compared staphylococcal broth with direct plating for the recovery of MRSA on BAP. In an attempt to identify carriers of epidemic MRSA, 124 intensive-care-unit and 14 operating-room employees had cultures of the nares. Four employees (2.9%) carried MRSA. All four MRSA isolates were found in the selective broth cultures, while only two of the four (50%) were detected by direct plating. MRSA could be detected only on repeat cultures of specimens from these four employees by the selective culture technique.

Although the use of an enrichment broth for cultures in the laboratory is routine, in practice most laboratories do not use broth enrichment for epidemiological surveillance. The fact that 62% of positive individuals in the Cookson evaluation and 50% in our evaluation would not have been detected without the use of an enrichment broth culture technique strongly suggests the need to use this culture method for epidemiological surveillance.