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.

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. News1. 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. News1.). 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.

LITERATURE CITED

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.
Letters to the Editor

Association of Blastocystis hominis with Human Disease?

The paper by Qadri et al. (2) on the clinical significance of Blastocystis hominis is another classic example of guilt by association prevalent in the literature which has grown up around this organism in the past few years. In their retrospective study, these authors discuss findings in the examination of 19,252 stool specimens from 12,136 patients seen in their hospital in Saudi Arabia during a 2-year period. Of this number, 647 patients were found to have B. hominis in their stools. Specimens were examined by saline and iodine-stained wet mounts and by concentrate, with the more accurate trichrome stain reserved for “all liquid specimens and those in which identification of parasite(s) was difficult or equivocal.” It is generally recognized that stained fecal films are the single most productive means of stool examination for protozoa (4). Our own experience showed (1; E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. 26:609–610, 1988) that even with examination of trichrome-stained smears by two technologists and one of us (E.K.M.), an occasional pathogen was missed on six consecutive examinations.

When the authors eliminated the 132 patients in whom potential pathogens (bacterial, protozoal, or histoplasmonic) had been found, 515 patients remained, and only 44.4% of these were symptomatic (even including an array of nonspecific symptoms such as fatigue, headache, and depression!). Of these 239 symptomatic patients, 26 had a diagnosis of duodenal ulcer, 3 had peptic ulcer, 17 had ulcerative colitis, 2 had bleeding hemorrhoids, and 18 had leukemia. Another 22 patients had a clinically significant eosinophilia, not characteristic of Blastocystis infection despite the report of Sheehan et al. (3). No mention was made of any special types of examination for helmmin parasites (schistosomes, Strongyloides stercoralis) which might not be found on the usual stool examinations but would be a diagnostic consideration in patients with gastrointestinal symptoms and an eosinophilia of 9 to 11%, especially in the area in which these authors were working. Treatment for B. hominis infection was undertaken in 43 of these patients after three to five stool examinations had revealed only B. hominis, symptoms persisted or recurred, or eosinophilia was present. We are told that resolution of symptoms occurred in all cases after 7 to 10 days of therapy and that none had B. hominis in their stools at follow-up examination after 3 to 6 months. Again, B. hominis can best be identified on stained smears, without which it would be most difficult to be certain of its absence. Seven to ten days seems a reasonable time in which to expect resolution of symptoms from many nonspecific causes, and it would be interesting to know what percentage of patients who were not so treated had an equally good outcome.

In our series of 32 patients, 27 were eventually found to have a recognized pathogen (Entamoeba histolytica, Dienamboea fragilis, or Giardia lamblia) in addition to B. hominis and were treated appropriately with iodoquinol, metronidazole, or quinacrine. (At the time we commenced our study, iodoquinol was the drug being recommended for treatment of B. hominis infection and quinacrine or metronidazole was used only to treat those patients who had giardiasis.) On reexamination approximately 4 weeks after completion of treatment, all 27 patients were found still to be infected with B. hominis, as was the case with the 5 patients in whom we were able to find only B. hominis. None of those with recognized pathogens remained symptomatic after treatment. Again, posttreatment specimens were stained with trichrome and examined by three different persons.

In the series reported by Qadri et al. (2), 5.3% were found to have B. hominis in their stools, a smaller number than has been found in most studies. More patients were found to have Entamoeba coli (9.1%) and Endolimax nana (5.8%). We might speculate what association could have been made between either of these organisms and the symptoms presented by the affected patients if we did not have foreknowledge that they are “nonpathogens.”

LITERATURE CITED

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Author’s Reply

Markell and Udkow have raised the same points again regarding our paper (5) which were amply clarified by D. J. Sheehan in response to their earlier letter (E. K. Markell and M. P. Udkow, Letter, J. Clin. Microbiol. 26:609–610, 1988). The methodology used in our laboratory is consistent with the standard procedures (8). Stained fecal films are necessary for the identification of trophozoites in diarrheal stools, and they augment identification when it is difficult or equivocal; but anyone with reasonable laboratory experience knows that they are not the “single most productive” method. If potentially pathogenic parasites are not seen in the presence of symptoms, we do examine four to six specimens collected on alternate days, but the Kaiser Foundation Medical Center is probably the only institution where the same specimen is examined by two technologists and a physician. Laboratories handling 50 to 200 specimens a day cannot afford such luxury. During my 20 years of experience in the United States and Saudi Arabia, we did not have any difficulty in recovering Strongyloides stercoralis using the very same methods. This parasite does not seem to be endemic here, not because of inadequate methodology but because of temperatures reaching 120 to 125°F (48.9 to 51.7°C), coupled with low humidity of 15 to 20%. We do not have any difficulty in finding schistosomes, which account for 1.5 to 2.0% of all parasites, including Blastocystis hominis (6). Markell and Udkow state that eosinophilin is not
characteristic of *B. hominis* infection without any supporting reference or data.

Their doubts about the clinical significance and effective treatment of this parasite appears to be based on their experience with 32 patients, 27 of whom had pathogenic parasites, that was published 7 years ago (4). Sheehan (Markell and Udkow, Letter) has dealt with this aspect succinctly, and Markell and Udkow’s opinion is contrary to increasing evidence of the pathogenic potential of *B. hominis* (2, 3, 5, 7, 9).

They correctly state that 55% of our patients were asymptomatic. It is possible that strains of *B. hominis* differ in virulence, which has been shown for *Giardia lamblia* (1). Since most infections with *G. lamblia, Entamoeba histolytica*, and other microorganisms are subclinical, one might speculate whether our esteemed colleagues would consider them nonpathogenic because the hosts are “asymptomatic.”

**LITERATURE CITED**


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**Typing and Nosocomial Candidiasis**

Fungi, especially *Candida* spp., are emerging as major nosocomial pathogens in both immunocompetent and immunocompromised patients. The rational development of effective measures to prevent and control nosocomial candidiasis needs a thoughtful understanding of the epidemiology of *Candida* spp., with particular reference to the reservoir and mode of transmission of the organisms.

Isenberg et al. recently provided some interesting insights into these issues, documenting the role of one individual as the source of a *Candida tropicalis* outbreak of cardiac bypass surgery wound infections involving eight patients (2). In fact, they examined fingertip and nasopharyngeal cultures of all personnel in contact with these patients, isolating *C. tropicalis* from one nurse only. Apart from this microbiological evidence, other important confirmations resulted from additional considerations: (i) the suspect nurse was the only individual involved in the surgical operations of all infected patients, (ii) the transmission of *C. tropicalis* occurred only while she was working as a scrub nurse and not while she was working as a circulating nurse, and (iii) her removal from the cardiac team terminated the cluster outbreak. The authors concluded by ascribing the cluster epidemic to the hands and oropharynx of the scrub nurse, acting as a common source.

We recently had the opportunity to investigate a nosocomial outbreak of systemic *C. albicans* infection involving eight patients who had received parenteral nutrition (3a). This epidemic had some similarities to the one reported by Isenberg et al., especially as to the possible role of one staff member as the common source of infection. In fact, the microbiological surveillance of personnel showed that two physicians and three nurses harbored *C. albicans* in the nasopharynx. We typed the isolates using DNA fingerprinting, a molecular method proposed as a helpful epidemiological tool for the study of *Candida* species (5) and successfully applied to the investigation of outbreaks of systemic *C. albicans* infections (3, 6), as well as to subspecies delineation of nosocomial isolates of *C. tropicalis* (4). DNA fingerprinting revealed that the same DNA pattern was shared by the *C. albicans* isolates from all patients and one nurse acting as a specialized nutrition nurse, whereas different DNA fingerprints were observed in the isolates recovered from the other staff members.

In the outbreak investigated by Isenberg et al., the attribution of the outbreak to the positive scrub nurse had immediate practical consequences (she was removed from the cardiac team) and has important scientific implications for both the understanding of the epidemiology of nosocomial candidiasis and the planning of relevant control interventions. In our opinion, however, the typing of *C. tropicalis* isolates would have reinforced and definitively proven the conclusions about the role of the scrub nurse as the single source of the outbreak, in particular ruling out the possibility that not all the cultured strains had the same origin. In reality, such a possibility appears to be quite unlikely in the case in question, even considering that *C. tropicalis* can be found in routine cultures from the nose, throat, skin, vagina, and gastrointestinal tract of healthy individuals (1). However, this possibility was well documented in an outbreak of systemic *C. albicans* infection which occurred in a neonatal intensive care unit (6). In that apparently single outbreak, DNA analysis revealed that two distinct strains were actually involved.
Clinical Significance of *Blastocystis hominis*

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A total of 19,252 stool specimens from 12,136 patients were examined by direct microscopy and the ethyl acetate-Formalin concentration method during the last 2 years. All liquid specimens and those in which parasite identification was difficult or equivocal were also examined in trichrome-stained preparations. A total of 3,070 intestinal parasites were seen in 2,889 patients. *Blastocystis hominis* was found in fecal material from 647 patients (17.5%). A total of 132 cases (25.6%) were observed to be in association with other enteric pathogens. *B. hominis* in large numbers was present as the only parasite or with other commensals in 515 specimens from patients (79.6%). Of these patients, 239 (46.4%) had symptoms, the most common being abdominal pain (87.9%), constipation (32.2%), diarrhea (23.4%), alternating diarrhea and constipation (14.5%), vomiting (12.5%), and fatigue (10.5%). Forty-three (18%) of the patients were treated with metronidazole (0.5 to 1.0 g/day) because of recurrent symptoms and the presence of large numbers of *B. hominis* cells in repeated stool specimens. After 7 to 10 days of treatment, all patients became asymptomatic with negative stools on follow-up examinations for *B. hominis*.

Although the pathogenic potential of *Blastocystis hominis* in human enteric disease had been reported in the older literature (2, 3, 9, 15), most of the recent textbooks describe it as a commensal found frequently in fecal specimens (7, 10, 17). Despite occasional case reports since 1976 regarding its association with human disease (4, 6, 13, 18), many physicians and clinical microbiologists consider its presence in stool specimens to be of questionable significance (8, 11). Recently, Sheehan et al. (16) and Babcock et al. (1) described the association of human disease with the presence of *B. hominis* in stool specimens of several patients and emphasized the need for further studies to elucidate its role and effective therapies. Since we have observed *B. hominis* in stool specimens of 647 patients in the last 2 years, we decided to undertake a retrospective chart review of these patients. Our findings are presented in this report.


**MATERIALS AND METHODS**

A total of 19,252 fresh stool specimens from 12,136 patients at the King Faisal Specialist Hospital and Research Centre (KFSH&RC), Riyadh, Saudi Arabia, were examined for the presence of intestinal parasites. The fecal specimens were grossly examined for stool consistency. Watery or diarrheal specimens were examined microscopically within 10 to 20 min of their receipt in the laboratory by using saline and iodine-stained wet-mount preparations. Soft or formed stool samples were examined by these procedures within 6 h of receipt. All specimens were subsequently concentrated by the ethyl acetate-Formalin method. The laboratory report sent to the clinicians included information about numbers of *B. hominis* cells and other parasites as few, moderate, or many. Wheatley's modification of Gomori's trichrome stain was also performed on all liquid specimens and those in which identification of parasite(s) was difficult or equivocal.

**RESULTS**

Over a 2-year period, 19,252 consecutive stool specimens from 12,136 patients were examined at KFSH&RC for the presence of intestinal parasites. KFSH&RC is a 500-bed tertiary-care referral hospital with admissions of 27,842 patients and an outpatient volume of 698,469 during the period of this study. Of the 2,889 patients with intestinal parasites, 647 had *B. hominis*. The medical charts of these patients were reviewed for the presence of other pathogens, pertinent laboratory findings, symptoms, underlying disease, therapy, and follow-up. *B. hominis* was found in association with other organisms in 359 patients (Table 1); 132 had potentially pathogenic protozoan and metazoan parasites, *Campylobacter jejuni*, and *Shigella flexneri*; 227 had commensals. The former group was excluded from further follow-up for obvious reasons of difficulty in interpreting the data in terms of attributing any symptoms to *B. hominis*.

Of the remaining 515 patients harboring *B. hominis*, the male-to-female ratio was about 1:1 and 8.9% of the infestations were in children and 19.3% were in patients over 50 years old (Table 2). No enteric symptoms were found in 55.6% of the patients. Large numbers (more than three to five organisms per 40 × field) of *B. hominis* cells were found as the only pathogen in 107 of the 239 patients with symptoms and in association with nonpathogenic parasites in 132 of these patients. The most common symptom was found to be abdominal pain, followed by constipation, diarrhea, and others (Table 3). Medical chart review showed that eosinophilia (9 to 11%) was present in 22 of these patients. A total of 71 patients had an underlying disease or condition, including duodenal ulcer (26 patients), leukemia (18 patients), ulcerative colitis (17 patients), kidney transplant (3 patients), peptic ulcer (3 patients), breast cancer (2 patients), and bleeding hemorrhoids (2 patients). Endoscopy in a 26-year-old female patient was suggestive of *Campylobacter* infection or giardiasis. However, *B. hominis* was the only organism detected in five different specimens.

The presence of large numbers of *B. hominis* cells alone in three to five repeated stool specimens, persistence or recurrence of symptoms, and eosinophilia in 43 of the symptomatic patients led to the administration of metronidazole.

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(250 to 500 mg twice a day for 7 to 10 days). This resulted in the resolution of symptoms, with the stools becoming negative for *B. hominis* on follow-up examination in all 43 patients over a period of 3 to 6 months.

**DISCUSSION**

Like *Giardia lamblia* of 30 years ago, *B. hominis* has been traditionally regarded as a harmless parasite of humans and has not found it easy to have itself recognized as a human pathogen. Recent interest in its clinical significance has resulted in sporadic reports over the last 12 years (4, 6, 13, 18). Phillips and Zierdt (12) in 1976 reported the pathogenic potential of *B. hominis* in humans and gnotobiotics, and this was followed by a report of two cases of mild persistent diarrhea in Italy (13). Although incidence rates of 12.2% in Southern California (6), 16% in New York (16), 10% in Nepal (1), and 14.1% in Yugoslavia (Z. P. Pikula, Letter, J. Clin. Microbiol. 25:1581, 1987) have been found for *B. hominis*, only 1 of the 2,000 specimens examined in North Wales (4) was positive for *B. hominis*. Of the 12,136 patients examined in this series, 647 (5.3%) had *B. hominis*, surpassed only by *Entamoeba coli* (9.1%) and *Endolimax nana* (5.8%).

Garcia et al. (6) reported that 61% of their positive specimens had few cells of *B. hominis*, 29% had moderate numbers, and 10% had many organisms. At KFSH&RC, we found that 21, 24, and 55% of specimens had few, moderate, and many organisms, respectively. Sheehan et al. (16) enumerated *B. hominis* cells and found that 43 (69%) of 62 patients had five or more cells per 40× field. Although our method of enumeration was semiquantitative owing to this being a retrospective study, we found that 89% of our patients with signs and symptoms of enteric disease had "many" organisms, compared with 82.6% in the series of Sheehan et al. (16).

Garcia et al. (6) observed that 24 (66.6%) of 36 patients had enteric symptoms, consisting mainly of diarrhea (79%), abdominal pain (58%), nausea (29%), and cramps (29%). Of the 23 patients with high *B. hominis* counts in feces in another series, 19 (82.6%) had symptoms (16), with abdominal discomfort (79%) being the most frequent complaint, followed by anorexia (53%), diarrhea (52%), and flatus (52%). Excluding the specimens containing known human pathogens, 239 (46.4%) of the 515 patients in our study had signs and symptoms of human disease. Eighty-nine percent of these patients had large numbers of *B. hominis* cells in the stools. As in the study of Sheehan et al. (16), the most frequent symptom was abdominal pain (88%). However, in our patients the second most common complaint was constipation (32%), followed by diarrhea and constipation (15%) and others.

Despite the presence of symptoms in 239 patients with *B. hominis* alone or in association with commensals, only 43 patients were treated with metronidazole. Resolution of symptoms accompanied by the absence of *B. hominis* cells in stool on follow-up examinations in all cases resulted after 7 to 10 days of therapy. Similar responses to metronidazole in four patients were described in the recent literature (5, 6, 14, 18).

Our study supports the emerging view that *B. hominis* should be considered as a causative agent of human disease in patients with recurrent symptoms, especially when the parasite is present in large numbers in fecal specimens in the absence of other known pathogens. Until recently, our laboratory reports sent to clinicians of harmless intestinal parasites, such as *Entamoeba coli*, *Chilomastix mesnili*, and others, included the statement that "the organism(s) reported are generally considered as non-pathogenic." We have discontinued this practice for *B. hominis*.

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LITERATURE CITED