Potential Danger in the Medical Use of *Trichuris suis* for the Treatment of Inflammatory Bowel Disease

To the Editor:

The commentary by Das[1] regarding the paper of Summers et al[2] on the use of the pig pathogen, *Trichuris suis,* as a treatment of inflammatory bowel disease raises some important questions. We have been aware of this treatment since 1999, when at an AGA meeting, a poster[3] prompted one of us to question the safety of it.

Summers et al[2] have repeatedly spoken of *T. suis* as noninvasive. In their 2003 paper, they state "Embryonated eggs hatch in the proximal small bowel. The larvae migrate aborally and attach to the mucosa of the distal small bowel and proximal colon. After several weeks, they mature and begin to shed eggs." The claim is made that *T. suis* "fulfills these criteria," i.e., colonizing the intestine "without invading the host."[2] Das[1] says the ova colonize the intestine.

The literature indicates otherwise. In the pig intestine, when embryonation is complete, the polar plugs of the ova dissolve away, liberating larvae bearing 5- to 7-μm-long lancet-shaped stylets with which they penetrate the mucosa. They remain for 13 to 16 days, migrating through lamina propria, undergoing 4 molts, and moving deeply to just above the muscularis mucosae and then later working their way back toward the surface. Finally as fifth stage larvae, they protrude their posteriors through to the lumen, progressively growing and extending out of the mucosa, until only esophageal portions remain embedded.[4,5]

During this "histotropic" migration, the potential exists for these larvae, with anterior stylets, to find their way into lymphatics or venules. The idea that they are noninvasive is false and all the more troublesome when one recognizes (1) that there is mucosal damage with increased vascularity and often disruption of the muscularis mucosae in inflammatory bowel disease, potentially facilitating deep penetration of larvae into the bowel wall, and (2) that larval parasites in unnatural hosts travel peculiar and often unpredictable paths. The human parasite, *Enterobius vermicularis*, which normally completes its life cycle in the intestinal lumen without even penetrating the mucosa, has on occasion been identified in human liver and lung[6]; the human pathogen, *Trichuris trichiura*, which like its porcine relative burrows in the mucosa, has been reported intraperitoneally.[7] There are numerous examples of aberrant migrations of parasites that find themselves in unfamiliar hosts: both *Toxocara canis* and *Dirofilaria immitis* of dogs migrate aberrantly when in human hosts, sometimes with significant clinical consequences. Such "lost" larvae are often stopped at the level of the liver or lung and incorporated into granulomas, but on other occasions the outcome is more serious. For example, larvae of *Baylisascaris procyonis*, the raccoon round worm, cause fatal central nervous system (CNS) disease in abnormal hosts; in humans, they cause retinal disease or encephalitis.[8] The meningeal worm of the deer, *Parelaphostrongylus tenuis*, when lost in the CNS of the moose, causes serious encephalitis and death.[9] *T. suis* have been found in the renal pelvis of wild boar.[10]

There is no predicting where *T. suis* larvae will go in humans, the abnormal host in this controversy. These larvae are invasive, and it can be reasonably expected that their paths of travel will be different in humans than they are in the domestic pig. It may only be a matter of time and numbers of larvae before retinal or CNS disease occurs in a patient "treated" with *T. suis*.

Herbert J. Van Kruiningen, MD
Department of Pathobiology and
Veterinary Science
University of Connecticut
Storrs, Connecticut

A. Brian West, MD, FRCPath
Department of Pathology
New York University School of Medicine
New York, New York

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Progressive Dysphagia Caused by Isolated Esophageal Involvement of Crohn's Disease

To the Editor:

Crohn's disease (CD) is characterized by chronic inflammation of the digestive tract, which may be localized at any level from the mouth to the anus. Involvement of the upper gastrointestinal tract is uncommon.[1,2] CD of the esophagus is very rare. The prevalence of esophageal CD ranges from 1% to 2% in