In our primary care–based study in the United Kingdom, we observed that the prevalence of Rome II criteria IBS was 10.25% (123/1200) and that the prevalence of undiagnosed celiac disease in this cohort was 3.3% (4/123),[3] a prevalence similar to that observed by Locke et al.

Fasano et al[4] recently showed that case finding for celiac disease in high-risk groups in the United States reveals a hidden iceberg—the disease appears to be more common than previously recognized, as are delays in diagnosis. In addition, patients clearly benefit symptomatically from a gluten-free diet.[5] Finally, given the sample size in the study by Locke et al (n=50), their study had only a 20% power to predict a 3-fold relative risk (assuming a .05 significance level).

In view of these observations, we believe that it is imperative that we are more circumspect about dismissing a possible link between IBS and celiac disease. Larger population-based studies are required to help clarify whether this relationship exists in the United States.

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1. Locke GR III, Murray JA, Zinsmeister AR, Melton LJ III, Talley NJ. Celiac disease serology in irritable bowel syndrome and dyspepsia: a population-based case-control study. *Mayo Clin Proc*. 2004;79:476-482.
2. Dickey W, McMillan SA, Hughes DF. Sensitivity of serum tissue transglutaminase antibodies for endomysial antibody positive and negative coeliac disease. *Scand J Gastroenterol*. 2001;36:511-514.
3. Sanders DS, Patel D, Stephenson TJ, et al. A primary care cross-sectional study of undiagnosed adult coeliac disease. *Eur J Gastroenterol Hepatol*. 2003;15:407-413.
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In reply: We appreciate Sanders and Azmy's comments and their interest in our article.[1] We wish to clarify that our study design was different than inferred by Sanders and Azmy. The patients in our study were identified by the symptoms they reported on a survey mailed to a random sample of the community. Hence, ours was a population-based sample, not a sample of primary care patients. The study consisted of minimal-risk interventions, and thus the patients did not undergo small bowel biopsy as part of the study protocol. The results of celiac serologic testing were unknown at the time of the physician interview and did not play a role in any decisions about patient care.

Sanders and Azmy discuss the possibility of endomysial antibody–negative celiac disease. We recognize that it can occur and may be more common than has been appreciated previously.[2,3] However, we believe that it is important to highlight that false-negatives are a lesser issue in populations with a low prevalence of disease. Even if all the patients who were TTg-positive and endomysial antibody–negative in our study were considered to have celiac disease, the differences between the cases and controls are minimal.

Although Sanders and Azmy were also concerned about the power of our study to detect clinically relevant differences, we believe that the order of magnitude of the prevalence rates must be emphasized. Some may argue that the difference of 4% in patients with IBS and 2.6% in controls is clinically important. However, a study of 2700 subjects per group would be needed to detect this size difference (with 80% power at an α level of .05). Our goal was to estimate the importance of celiac disease in explaining why IBS symptoms are so common in the community. We did not mean to imply that there is no association between celiac disease and IBS or dyspepsia in the community. Our findings, however, support the conclusion that the vast majority of people with functional bowel symptoms in the community do not have occult celiac disease.

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1. Locke GR III, Murray JA, Zinsmeister AR, Melton LJ III, Talley NJ. Celiac disease serology in irritable bowel syndrome and dyspepsia: a population-based case-control study. *Mayo Clin Proc*. 2004;79:476-482.
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Unenabled Embryo Use

To the Editor: In the commentary by Guenin[1] in the June 2004 issue of the *Mayo Clinic Proceedings*, the author explained cogently why 6 arguments in support of using human embryos in research and therapy do not work. Fortunately, his "argument from nonenablement" also does not work.

Guenin advances the curious argument that the parents' conceptual intent regarding the embryo's future and the accident of where the embryo is located (petri dish vs uterus) actually determine the embryo's ontological status, ie, *what* it is. According to him, the decision to suspend the embryo's development at an early stage and donate the embryo for experimentation transforms it into an "epidosembryo," to which no "possible person" corresponds. This is transubstantiation by mental fiat and physical location—a truly miraculous occurrence!

The real issue is the ontological status of the *zygote* because all subsequent development consists simply of growth. Growth is *accidental* change, whereas conception is *substantial* change. A gamete implanted in a woman's uterus will never grow into a baby.