

18.9) and also weakly associated with development of bloody diarrhea (OR = 2.5) (2). Avery uses the term endogeneity as it is used in econometric analyses; however, the term “intermediary variable,” i.e., a factor in the causal pathway leading from exposure to disease, is more frequently used in epidemiology. In this context, we chose to consider bloody diarrhea as a potential confounder (3). A confounder is a risk factor but is also independently associated with the exposure variable of interest and is not regarded as part of the causal pathway (see online Figure at <http://www.cdc.gov/ncidod/EID/vol11no03/05-0071-G.htm>). Bloody diarrhea may act as a confounder if patients with bloody stools are treated differently by the examining physicians or if, for instance, unknown virulence factors contribute to the risk of having bloody stools.

A second line of critique of our methods apparently develops from the idea that virulence factors determine the serogroup. This idea, however, is a biological misconception. In fact, virulence genes and serogroup are independent at the genetic level, and an important point of our article is that HUS is determined by the virulence gene composition of the strain rather

than the serogroup.

Regardless of the status of the bloody diarrhea variable, excluding it from the model doesn't change the conclusions of the article. A revised model contains only the significant variables age and *stx*₂ (Table). Serotype O157 is still not an independent predictor of HUS, and this result is robust.

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Table. Risk factors for HUS among 343 STEC patients, Denmark 1997–2003, comparison of models with and without bloody diarrhea as a variable*

Determinant	No. of patients	No. (%) with HUS	Original model, OR (95% CI)	New model, OR (95% CI)
<i>eae</i>				
Negative	111	0 (0.0)		
Positive	232	21 (9.1)	NI	NI
<i>stx</i> ₂				
Negative	159	1 (0.6)	1	1
Positive	184	20 (10.9)	18.9 (2.4–146)	24.6 (3.2–187)
Age				
≥8 y	178	3 (1.7)	1	1
≤7 y	165	18 (10.9)	11.4 (3.2–41.3)	9.7 (2.7–34.1)
Bloody diarrhea				
No	218	6 (2.8)		
Yes	125	15 (12.0)	4.5 (1.6–12.7)	EX
O157				
No	262	10 (3.8)		
Yes	81	11 (13.6)	NS	NS

*HUS, hemolytic uremic syndrome; STEC, Shiga toxin–producing *Escherichia coli*; OR, odds ratio; CI, confidence interval; NI, not included (test not appropriate); NS, not significant; EX, excluded from model.

Rectal Lymphogranuloma Venereum, France

To the Editor: Lymphogranuloma venereum (LGV), a sexually transmitted disease (STD) caused by *Chlamydia trachomatis* serovars L1, L2, or L3, is prevalent in tropical areas but occurs sporadically in the western world, where most cases are imported (1). LGV commonly causes inflammation and swelling of the inguinal lymph nodes, but it can also involve the rectum and cause acute proctitis, particularly among men who have sex with men. However, LGV serovars of *C. trachomatis* remain a rare cause of acute proctitis, which is most frequently caused by *Neisseria gonorrhoeae* or by non-LGV *C. trachomatis* (2). In 1981, in a group of 96 men who have sex with men with symptoms suggestive of proctitis in the United States, Quinn et al. found that 3 of 14 *C. trachomatis* infections were caused by LGV serovar L2 (3). In France, 2 cases of rectal LGV were reported in an STD clinic in Paris from 1981 to 1986 (4). In 2003, an outbreak of 15 rectal LGV cases was reported among men who have sex with men in Rotterdam; 13 were HIV-infected, and all reported unprotected sex in neighboring countries, including Belgium, France, and the United Kingdom (5). At the same time, a rise in *C. trachomatis* proctitis (diagnosed by using polymerase chain reaction [PCR]; [Cobas Amplicor Roche Diagnostic System, Meylan, France]) was detected in 3 laboratories in Paris and in the *C. trachomatis* national reference center located in Bordeaux. To identify the serovars of these *C. trachomatis* spp., all stored rectal specimens were analyzed by using a nested *omp1* PCR-restriction fragment length polymorphism assay. The amplified DNA product was digested by restriction enzymes. Analysis of digested DNA was performed by elec-

trophoresis. Patterns were compared visually with reference patterns (6).

From January 1, 2003, to March 31, 2004, a total of 44 of 124 male rectal swabs were positive for *C. trachomatis*. Of those, 38 were identified as belonging to the L2 serotype, which confirms the diagnosis of rectal LGV. Epidemiologic information was retrospectively obtained by clinicians through review of medical records, telephone interview, or both. A complete history was available for 14 of the 38 cases. All 14 men reported unprotected anal sex with anonymous male sex partners in France, and none reported a stay in an LGV-endemic area. Their mean age was 40 years (31–50); 8 were HIV-infected, and 9 had another concomitant STD. The mean duration of symptoms before LGV diagnosis was 50 days (range 11–120 days). All 14 patients had symptoms of acute proctitis, including rectal pain, discharge, and tenesmus, and 3 (all HIV-infected) had fever. Deep, extended rectal ulcerations were reported in 8 patients, 3 of whom were HIV-infected and had lesions suggestive of rectal carcinoma. In 1 patient in whom a late diagnosis was made 4 months after the onset of symptoms, a rectal tumorlike stricture was observed. All 14 patients were treated with tetracycline for a mean duration of 16 days (range 10–60 days).

An information campaign among microbiologists and clinicians and a

sentinel LGV surveillance system were launched in April 2004. Subsequently, LGV was diagnosed in 65 additional male patients, some retrospectively. In total, rectal LGV was diagnosed in 103 patients from July 2002 to August 2004 (Figure).

Prompt diagnosis and treatment is indeed paramount to prevention and control. Diagnosis may be further hampered because rectal LGV may mimic other conditions such as rectal carcinoma or Crohn disease. Treatment duration should be no shorter than 21 days, and follow-up examinations should be conducted until all signs and symptoms have resolved (7,8). If left untreated, rectal LGV could lead to serious complications such as rectal stricture (1). If recently exposed to infection, sexual contacts should receive prophylactic treatment to prevent reinfection and to eliminate a potential reservoir. The emergence of rectal LGV, characterized by deep mucosal ulcerations and frequently occurring in HIV-infected men who have sex with men, is a serious concern for the gay community in Europe.

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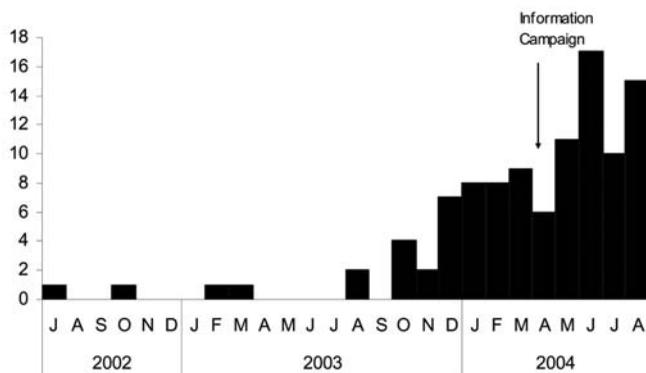


Figure. Number of rectal lymphogranuloma venereum cases diagnosed in men in France, July 2002–August 2004.

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Pertussis in Soldiers, Israel

To the Editor: The role of adults as reservoirs of pertussis was previously well established (1–7). Young army recruits undergoing basic train-