considered among the strains from endocervical and conjunctival origins.

C trachomatis infections cannot be eradicated without effective disease-control programmes that focus on early diagnosis, targeted screening, and effective treatment. Such an approach will lead to an eventual decline in the incidence of ocular and urogenital chlamydial infections.

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1 Dolin PJ, Faal H, Johnson GJ, et al. Reduction of trachoma in a sub-Saharan village in absence of a disease control programme. *Lancet* 1997; **349:** 1511–12.
2 Aoki K, Sato C, Hashimoto N, Chiba S, Numazaki K. Clinical and etiological studies of chlamydial conjunctivitis in Sapporo, Japan. *Jpn J Ophthalmol* 1998; **32:** 444–49.
3 Hayes LJ, Bailey RL, Mabey DCW, et al. Genotyping of *Chlamydia trachomatis* from a trachoma-endemic village in the Gambia by a nested polymerase chain reaction: identification of strain variants. *J Infect Dis* 1992; **166:** 1173–77.
4 Wagenvoort JHT, Suchland RJ, Stamm WE. Serovar distribution of urogenital *Chlamydia trachomatis* strains in the Netherlands. *Genitourin Med* 1988; **64:** 159–61.

Diagnosis of gastric carcinoma in Japan and western countries

SIR—Junichi Sakomoto and Mitsunori Yasue's June 14 commentary[1] leaves the reader with the impression that there is a fundamental difference in the diagnostic criteria applied by Japanese and western pathologists to well-differentiated adenocarcinoma of the stomach. However, in Schlemper and colleagues' study (June 14, p 1725),[2] the diagnostic approach to forceps biopsy material obtained from lesions of the gastric mucosa was not uniform among the western pathologists. I was the western pathologist whose diagnosis was identical with that of the Japanese pathologists, and who was therefore assigned to the Japanese viewpoint in the tables and summary. Since I receive much material from other German pathologists for consultation purposes, I can say with confidence that most pathologists in Germany would have interpreted the preparations investigated in Schlemper's study in the same way as did our Japanese colleagues and myself. I believe that there are probably no basic differences between Japanese and western pathologists, and that, apart from myself, many other western pathologists would have diagnosed the cases exemplified by figures 1 and 2 in Schlemper's study unequivocally as an adenocarcinoma.

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1 Sakamoto J, Yasue M. Do Japanese statistics on gastric carcinoma need to be revised? *Lancet* 1997; **349:** 1711–12.
2 Schlemper RJ, Itabashi M, Kato Y, et al. Differences in diagnostic criteria for gastric carcinoma between Japanese and Western pathologists. *Lancet* 1997; **349:** 1725–29.

SIR—With respect to Ronald Schlemper and colleagues' report[1] we would like to consider several issues that are closely related to our own experience. First, as a general rule of oncology, when the intraepithelial location of the cancer is not explicitly specified, invasiveness must be regarded as intrinsic to any epithelial malignant disease. It is noteworthy, however, that cytological atypia (another cancer attribute) may be undetectable even in metastatic lesions,[2] which strongly contradicts the sensitivity of cellular/nuclear abnormality in the histological diagnosis of cancer, whatever its location.[1]

Second, it is accepted that invasive cancers represent the most advanced stage of a multistep sequential process and, in the gastrointestinal tract, this has been validated by clinical and molecular studies. Hence a spectrum of preinvasive neoplastic lesions theoretically also recognisable in the morphogenesis of intestinal-type gastric cancer and the terms gastric epithelial dysplasia or adenoma have been adopted to define such flat or raised lesions. A clear-cut border between dysplasia and invasive lesions may be hard to distinguish, especially in high-grade preinvasive lesions. According to Schlemper and colleagues, these are the cases in which the Japanese pathologists favour the diagnosis of full-blown cancer (by attaching particular importance to nuclear atypia). As a result of this practical Japanese approach (i) the diagnosis of cancer is consistently made on biopsy and endoscopic mucosal resection (EMR) samples (from the Japanese viewpoint, cases e,f,i,j,l,m,n,o,p,q,r); (ii) advanced gastric carcinoma is successfully prevented; but (iii) evidence of any invasiveness is consistently lacking from either the biopsy or the EMR specimens (cases h,k,m,n). Although this clinicopathological behaviour, which might be amply justified by several considerations (especially in high-risk areas); what we would highlight is that inconsistent diagnostic classifications lead to inconsistent guidelines for the clinical management of such lesions.

The fallout on patient management concerns whether and when it is justified to surgically treat neoplastic lesions with no histological evidence of invasion. Leaving aside the EMR procedure, the main point is the timing of gastric resection in the absence of any invasive carcinomatous pattern.[1,3] Unquestionably, high-grade gastric epithelial dysplasia, detected as initial diagnosis or during the follow-up of low-grade dysplasia, must be removed. For low-grade lesions (ranging from mild to moderate gastric epithelial dysplasia), the high percentage of cases in whom no further dysplasia is detected (possibly regression), either after *Helicobacter pylori* eradication or with no therapy (73/123 of our patients with a mean follow-up of 46 months, range 12–160, unpublished data) would advise against any over-hasty surgical treatment. We emphasise that such a wait and monitor approach is ethical only with strict follow-up (in terms of endoscopic schedule and sampling method).[4] It is worth mentioning that, judging from our prospective follow-up study of gastric epithelial dysplasia (130 patients with follow-up longer than 12 months, table), most patients did not develop gastric cancer after a mean follow-up of 40 months, and the prevalence of gastric cancer detected in the early stage was 78%.

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1 Schlemper RJ, Itabashhi M, Kato Y, et al. Differences in diagnostic criteria for gastric carcinoma between Japanese and Western pathologists. *Lancet* 1997; **349:** 1725–29.
2 Riddell RH. Premalignant and early malignant lesions in the gastrointestinal tract: definition terminology and problems. *Am J Gastroenterol* 1996; **91:** 864–72.
3 Sakamoto J, Yasue M. Do Japanese statistics on gastric carcinoma need to be revised? *Lancet* 1997; **349:** 1711.
4 Rugge M, Leandro G, Farinati F, et al. Gastric epithelial dysplasia: how clinico-pathologic background relates to management. *Cancer* 1995; **76:** 37.
5 Rugge M, Farinati F, Di Mario F. Can we justify resecting all gastric epithelial dysplastic lesions? *Gastroenterology* 1995; **108:** 1955–56.

initial diagnosis	No of patients	EGC	AGC	GC-nos	Mean time elapsed from the initial diagnosis to cancer detection (range)
Mild Ged	89	3	0	0	45 months (15–80)
Moderate GED	34	6	2	0	36 months (21–72)
Severe GED	7	5	0	2	25 months (12–36)

EGC=early gastric cancer, AGC=advanced gastric cancer, GC-nos=Gastric cancer of unknown pTNM stage.

Gastric cancers detected during prospective follow-up of gastric epithelial dysplasia (GED)