tries with high incidence and prevalence of HCV, acute hepatitis C is frequently recognized particularly in high-risk groups since identification of acute cases is related to the incidence and prevalence of HCV in a given community rather than the total population.

To date, sustained virologic response in our study has been durable, with no subject having a late relapse. In our study and in previous studies from our group we could not find a correlation between a specific genotype and spontaneous resolution.

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The Risk of Colorectal Cancer in Ulcerative Colitis in a Population-Based Setting

Dear Sir:

It is refreshing to see the current community based data of Jess et al[1] mirror that which has been reported in 2001,[2] from the community of Orlando, Florida. The findings of this paper may be somewhat shocking to those in the academic world, but unlikely to surprise those observant clinicians who have been in practice for 10–20 years.

Jess et al[1] concluded that the risks for CRC was not increased among ulcerative colitis (UC) patients overall, and that is what we had noted as well. The 2 populations were also similar in that both were primarily Caucasian and nonselected. There were 378 UC patients in the Jess study and 319 in ours (currently up to 403, with no change in the data).

In our community data, of 319 patients with UC, 98 had pancolitis, and 54 of 98 had a duration >7 years with a mean of 18.3 years.[3] Only 1 patient developed rectal cancer, and he was 44 years old, with a 7-year history of left-sided colitis, who had a benign colonoscopy 1 year earlier.

Surveillance biopsies over a 10-year review period revealed 20 of 319 (6.2%) patients with low-grade dysplasia (LGD) that either regressed, or were never encountered again in follow-up biopsies. High-grade dysplasia (HGD) was not encountered. Of 2 patients with LGD who underwent colectomy, neither HGD or CRC were found. In this study, 19 of 319 (5.9%) patients had adenomas, none of which were cancerous or dysplastic. All were treated with polypectomy. Over the past 5 years there have not been any new cases of dysplasia or CRC.

This current article is most welcome since it emanates from an auspicious institution, and will undoubtedly be supported by the actual findings of private practitioners around the United States who have been murmuring these thoughts for over a decade.

I am only disappointed that the authors did not report on the results of colonoscopic surveillance biopsies over this time frame, and whether they had any predictive value for CRC. The practice of surveillance biopsies every 1–3 years, requiring 6–10 specimen jars, is extremely expensive, and seems to act primarily as medico-legal protection, more than as a useful tool for the patient or physician. Consideration should be given to changing the current recommendations for surveillance given the data presented above. Strong written opinions supporting not electing to do surveillance would be needed for the community practitioners, for medico-legal support and for patient reassurance. A re-evaluation of the value of the media in propagating opinions for the lay population needs to be pursued, since there are repeated instances of popularization of opinions in medicine that turn out to be incorrect, and only serve to heighten the mistrust that the general population has for the prolific new medical data that floods the information highway on a daily basis.

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Reply. We thank Dr. Sprung for his interest in our recent paper describing a surprisingly low risk of colorectal cancer in a population-based cohort of inflammatory bowel disease from a small but well-defined geographic region of the north-central United States.[1] We detected only 6 colorectal cancers among 378 ulcerative colitis patients with a median follow-up of 13 years per patient (range, 0 to 58 years). The relative risk of colorectal cancer, expressed as a standardized incidence ratio (observed cancers divided by expected), was 1.1 (95% confidence intervals, 0.4 – 2.4). After excluding the 2 cancers diagnosed within 30 days of the diagnosis of ulcerative colitis, the cumulative risk of colorectal cancer was 2% after 25 years of disease.[1] Dr. Sprung points out that our results are similar, in some respects, to work that he has published, in preliminary form, on the risk of colorectal cancer and the results of surveillance colonoscopy in a group of patients evaluated at a private gastroenterology practice from Orlando, Florida.[2–3] Among 319 ulcerative colitis patients undergo-

ing surveillance colonoscopy, only one patient developed colorectal cancer.[3] The duration of follow-up from entry into the surveillance program is unclear. In this same cohort of patients, there were 20 patients with low-grade dysplasia (LGD) of the colon or rectum, 19 who had polyps removed, and 25 who had undergone colectomy for disease activity. Dr. Sprung wonders why we did not report on our experience with surveillance biopsies, and concludes that private practitioners have realized for years that colorectal cancer risk in ulcerative colitis is low and that surveillance colonoscopy is performed primarily for medico-legal reasons.

We disagree with Dr. Sprung's conclusion that our results support his view that surveillance colonoscopy is unnecessary. Similar to Dr. Rubin's conclusion in the editorial accompanying our paper,[4] we believe that our results represent a success and the product of a number of factors, including a compliant population with a high prevalence of surveillance colonoscopy and maintenance therapy, and a relatively high colectomy rate (cumulative probability of 21% after 25 years of disease).[1]

The original version of our paper included data on the incidence and progression of colorectal dysplasia and polyps, but during the peer review process it was suggested that this information should be published separately. These data have been published in preliminary form,[5] and the full manuscript is in press.[6] The cumulative incidence of colorectal dysplasia (including flat dysplasia, polypoid dysplasia, and adenomas) among these patients was 1.9% (95% CI, 0.4%–3.5%) at 5 years, 5.1% (95% CI, 2.1%–8.0%) at 15 years, and 9.2% (4.5%–14.3%) at 25 years.[6] To our surprise, none of the 6 patients diagnosed with LGD progressed to high-grade dysplasia or cancer; however, when polypoid dysplasia and adenomas within the field of colitis were included in the analysis, the actuarial progression/recurrence rate was 8.2% (95% CI, 0%–18.8%) at 5 years, 26.6% (95% CI, 5.8%–44.8%) at 10 years, and 47.1% (95% CI, 8.8%–71.3%) at 20 years.[6]

We agree with Dr. Sprung that the evidence of benefit for surveillance colonoscopy is not definite, and that it is expensive. Nevertheless, there are several retrospective case-control studies suggesting that surveillance may reduce colorectal cancer[7,8] or cancer-related mortality[9] in ulcerative colitis, and at this juncture it is the best tool we have to detect dysplasia or cancer at a treatable stage. Few studies have examined the number of biopsies necessary during surveillance, but the more the better.[10] A recent consensus statement from a panel convened by the Crohn's and Colitis Foundation of America concluded that a minimum of 33 biopsies should be obtained.[11] At Mayo Clinic Rochester, in an effort to reduce costs somewhat, we have opted to place a minimum of 32 surveillance biopsies in 4 separate bottles (cecum/ascending, transverse, descending/proximal sigmoid, rectosigmoid), and polyps are placed in separate bottles.

In conclusion, we hope that our study is not interpreted as justification for relaxing our vigilance in detecting colorectal neoplasia in inflammatory bowel disease.

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Hepatitis C Infection and Lymphomas: Is There any Benefit in Viral Treatment?

Dear Sir:

Pal et al[1] reported that the hepatitis C virus (HCV) has the capacity to infect and replicate in vitro and in peripheral lymph nodes. Their work adds to the evidence that HCV has lymphoproliferative activity that can play a role in lymphomagenesis.

Acceptance of this association could have major therapeutic implications for patients with HCV positive lymphomas. The authors indicate that the hepatic lymph node infection could represent an immune alteration that could lead to an impaired response to HCV treatment. However, in spite of lymphatic involvement, some studies have shown that antiviral treatment has been successful in HCV-positive patients with non-Hodgkin lymphoma (NHL) and that a successful response to anti-viral treatment resulted in remission of lymphoma. Do these observations imply that treatment success may be more plausible in lymphomas or histologies that are less likely to affect the perihepatic lymph nodes?

How do the authors explain the success rate among HCV-related lymphomas observed in published series?[2] In HCV carriers with lymphoproliferative disorders, is this an issue to take into account to plan antiviral treatment strategies?

To date, the association between lymphomas and HCV has been based on observational studies. However, no clear mechanism of