results originate in two independent centres. Leary *et al* suggest that as HPV DNA is not always present in glandular neoplasia[1] that HPV might be a cofactor rather than an initiator of endocervical glandular neoplasia. If this is so then HPV DNA need not necessarily be detected, possibly explaining the discrepant results from the United Kingdom and elsewhere.

To summarise, results from the United Kingdom[3 7] suggest that infection with HPV types 6, 11, 16, 18 and 31 does not necessarily have a major role in cervical glandular neoplasia.

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4 Tase T, Okagaki T, Clark BA, *et al*. Human papillomavirus types and localization in adenocarcinoma and adenosquamous carcinoma of the uterine cervix: A study by in situ DNA hybridization. *Cancer Res* 1988;48: 993–8.
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Radiation colitis is another mimic of chronic inflammatory bowel disease

We read with great interest the article written by Shepherd.[1] This informative review will be of great use to practising histopathologists when they face an avalanche of colorectal biopsy specimens with relatively little clinical information. The article should persuade both pathologists and physicians that clinical information is of great importance in reaching a histological diagnosis. The colorectal mucosa has limited ways of expressing itself in response to injury—a single brick from the Berlin Wall may look identical to one from the longstanding Wall of China.

The article does not define the histological features of chronic inflammatory bowel disease, but this diagnosis must be based on a combination of several morphological features such as crypt distortion, metaplasia (Paneth cells or pseudopyloric), fibrosis of the lamina propria associated with loss of crypts and/or significant increase in chronic inflammatory cells. On this basis we believe that radiation colitis is an important addition to the diagnostic possibilities. Radiotherapy is a common form of treatment for many pelvic carcinomas and the clinical features of radiation enteropathy may appear after many years when the inheriting surgeon may be unaware that the patient has been irradiated. Radiation colitis in the chronic phase demonstrates a very significant crypt distortion, vascular telangiectasia, and fibrosis of the lamina propria, which can easily be misinterpreted as healed or quiescent chronic inflammatory bowel disease, unless the relevant information is available.[2]

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1 Shepherd NA. Pathological mimics of chronic inflammatory bowel disease. *J Clin Pathol* 1991;44:726–33.
2 Haboubi NY, Hasleton PS. *The causation and clinical management of pelvic radiation disease.* Berlin: Springer-Verlag, 19–35.

Oestrogen receptors in conjunctival malignant melanoma

Paridaens *et al* claim to have demonstrated oestrogen receptors in paraffin wax sections of formalin fixed conjunctival malignant melanomas.[1] It is not unreasonable to expect that these lesions may be susceptible to endocrine factors, but the authors' results do not support their conclusions.

I have two reservations. First, the cytoplasmic staining they observed conflicts with the known nuclear location of oestrogen receptors.[2] Second, although the antibody to ER-D5 recognises an epitope on an oestrogen receptor related protein, several studies have shown that immunostaining with this reagent correlates poorly with the results of ligand binding assays for oestrogen receptors.[3-5] Furthermore, the authors are mistaken to believe that ER-D5 is ". . . present only in oestrogen receptor positive tissues."[6]

Finally, the statement that ". . . a nuclear binding assay, which identifies *non-functional* receptors, may be more appropriate" makes no sense. Surely it is more appropriate to identify *functional* receptors by, for example, seeking oestrogen regulated proteins, such as progesterone receptor and cathepsin D.

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Dr Paridaens et al comment:

We thank Professor Underwood for his comments on our paper. We disagree, however, with his statement that "the cytoplasmic staining they observed conflicts with the known nuclear location of oestrogen receptors". An alternative immunochemical approach in the detection of the receptor moiety of steroid-hormone receptor complexes or unliganded receptors is the use of antibodies directed against receptor proteins. We used a monoclonal antibody which has been shown to be specific to D5 antigen, a non-hormone-binding component related to cytosolic oestrogen receptors, and which does not recognise classic type 1 nuclear oestrogen receptor.[1] The cytoplasmic staining we observed therefore reflected recognition of the ER-D5 antigen which has been shown to be closely related to oestrogen receptors.[2]

Secondly, a study by Coffer *et al* showed a significant correlation ($p < 0{\cdot}001$) between D5 immunoradiometric assay (IRMA) value and oestrogen receptor sites in breast tumours assayed by [3H]oestradiol binding sites.[2] However, the correlation between ER-D5 immunohistochemistry and ligand-binding assays for oestrogen receptors has been in dispute[3-5] and as a result the predictive value of the immunocytochemical method using anti-ER-D5 should be interpreted with caution.

Thirdly, the distributors of the antibody (Amersham) indicate that the antigen ER-D5 is present only in oestrogen receptor positive tissues, a finding which was confirmed by King *et al*.[6]

Finally, the aim of our concluding statement was to highlight the importance of identifying the hormone receptors that are biologically active (functional as opposed to non-functional receptors) to predict response to hormonal treatment, because this cannot be assessed by immunocytochemistry alone.

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Secretarial services to consultant microbiologists

A questionnaire on the use of secretarial services sent to 21 consultant microbiologists in Yorkshire in July 1991 produced a