sonable doubt. Personally, I find it hard to believe that the invasive potential of melanoma is reliably predicted by the width of dermal nests of melanocytes.²

Third, I realise that Dr Cook has not proposed a subdivision of MIN that also incorporates low grade changes.² To many histopathologists, however, this will be a natural temptation in view of similar usage in other types of intraepithelial neoplasia. The incidence of MIN would then increase dramatically and stand the chance of losing credibility by becoming a depositary for many minor abnormalities.

Finally, as the epidemiological aspects of melanoma are of crucial public health importance, reports will have to contain both old and new terminology into the foreseeable future.

As emphasised by Professor Mooi, the CRC Panel's study is an important analysis of observer variation in the diagnosis of thin cutaneous melanoma.³ One wonders, however, what results would have been achieved by a control group of general histopathologists using traditional atypical (dysplastic) melanoma terminology and enforced reading of an acknowledged reference source, such as the Armed Forces Institute of Pathology, *Melanocytic tumours of the skin fascicle*.⁴

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- 1 Mooi WJ. Diagnosis of thin melanoma [editorial]. *J Clin Pathol* 1997;50:179-80.
- 2 Cook MG, Clarke TJ, Humphreys S, et al. The evaluation of diagnostic and prognostic criteria and the terminology of thin cutaneous malignant melanoma by the CRC Melanoma Pathology Panel. *Histopathology* 1996;28:497-512.
- 3 CRC Melanoma Pathology Panel. A nationwide survey of observer variation in the diagnosis of thin cutaneous malignant melanoma including the MIN terminology. *J Clin Pathol* 1997;50:202-5.
- 4 Elder DE, Murphy GF. *Atlas of tumour pathology. Melanocytic tumours of the skin*. 3rd series. Fascicle 2. Washington: Armed Forces Institute of Pathology, 1991.

Dr Cook and colleagues comment:

Dr Slater makes several points in his letter about our paper and the use of the term melanocytic intraepidermal neoplasia. It may be worthwhile reiterating the processes by which the CRC Melanoma Pathology Panel came to suggest MIN for consideration. The CRC study of melanoma in pigmented lesions identified a wide discrepancy between the centres participating in the diagnosis of thin melanomas. The CRC Panel tried to identify the causes and solutions to this problem but only succeeded in providing a modest improvement in concordance. The possible implication suggested that the difference between dysplastic melanocytic lesions and melanoma in situ was not sharp and that they were not biologically distinct. Furthermore, as they were not managed differently the panel considered the need to continue to recognise them as separate entities. A term such as "melanocytic dysplasia-melanoma in situ spectrum" might have been used but an alternative—MIN—was considered more usable, and analogies with CIN were emphasised. Professor Mooi pointed out that "intraepithelial" would be more precise than "intraepidermal" and we would not argue with that. He also considered the term neoplasia to be too inclusive but we would

argue that it can have a more specific meaning when used in the context as we have defined it.

Professor Mooi has indicated that he does not personally value MIN highly because he avoids the diagnostic dilemma by hardly ever making the diagnosis of melanoma in situ, and he therefore does not have a problem in distinguishing it from dysplastic lesions (personal communication, Professor Mooi, 1997). His idea that the origin of some melanomas may be from the dermal component is interesting but not in conflict with MIN. However, it does question the use of microinvasion as an appropriate term and clearly there is much work to be done in validating either theory.

As far as Dr Slater's comments are concerned, we would like to emphasise that we put forward the MIN terminology for consideration as a possible solution to a demonstrable problem. We have tested its acceptability with a national survey but accept that further discussion and agreement is necessary before it could be a formal recommendation.

Dr Slater is also broadening his aim to include the validity of the growth phase concept of Clark *et al.*¹ We agree that this interesting theory does need more published support before it can become firmly accepted. His suggestion of the inevitable subdivision of MIN is completely against the rationale for introducing the term and we cannot accept this argument.

The existing epidemiological data for melanoma is based on current terminology and is therefore flawed, hence the need for the CRC Pathology Panel study in the first place. It is accepted that clarification of that data will take many years but it will be necessary whatever terminology is adopted.

The most important results from the nationwide survey highlight the need for more reliability in the recognition of melanoma and related lesions. This problem needs to be urgently addressed, as the detection of thin melanomas and borderline lesions has been rising rapidly.

- 1 Clark WH Jr, Elder DE, Guerry D IV, Braitman LE, Tock BJ, Shultz D, et al. Model predicting survival in stage I melanoma based on tumor progression. *J Natl Cancer Inst* 1989;81:1893-904.

Serum CagA antibodies in asymptomatic subjects and patients with peptic ulcer

We read with great interest the paper by Graham *et al.* in which, although different methodologies were used, the authors failed to find a significant association between CagA seropositivity and peptic ulcer disease or increased antral inflammation in a North American population.

We would briefly like to mention our previous experience about the patterns of antibody response to *Helicobacter pylori* infection in patients with chronic renal failure.² A group of 51 patients with different degrees of chronic renal failure was studied: histology of gastric specimens was carried out according to the Sydney's system, and serum antibodies to CagA were investigated by western blotting. The prevalence of infection was 76.4% and patients infected by *H pylori* had a greater activity of gastritis than non-infected patients ($p < 0.0001$). However, patients infected by CagA positive strains of *H pylori* did not have

a greater activity of gastritis than those infected with CagA negative strains ($p = 0.540$). No significant correlations were found with other histological features such as gastric atrophy, chronic inflammation, and intestinal metaplasia.

These results raise some questions about the pathogenetic mechanism of *H pylori* infection. Finding a specific or a unique virulence factor of infection should lead us to select patients for more specific treatment and, among the virulence factors considered, CagA has been suggested as one of the most important.³

However, the work of Graham *et al.* seems to exclude this, at least for the North American population. Moreover, our findings, obtained in patients with chronic renal failure, seem to confirm that CagA seropositivity is not necessarily related to increased antral inflammation, suggesting that the gastric microenvironment present in the stomach of these patients could interfere with the pathogenetic mechanism of *H pylori* infection.

These findings underline that clinical infectious disease usually represents an interplay between the bacteria and the host and that the "variable host" should receive more attention. We believe that further studies in a well defined population could help clarify the exact pathogenetic mechanism of *H pylori*.

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- 1 Graham DY, Genta RM, Graham DP, Crabtree JE. Serum CagA antibodies in asymptomatic subjects and patients with peptic ulcer: lack of correlation of IgG antibody in patients with peptic ulcer or asymptomatic *Helicobacter pylori* gastritis. *J Clin Pathol* 1996;49:829-32.
- 2 Rapisarda S, Figura N, Granata A, Vasquez E, Fraggetta F, Randone S, et al. Degrees of gastritis and patterns of antibody response to *H. pylori* infection in patients with chronic renal failure [abstract]. *Gut* 1996;38(suppl 2):A53.
- 3 Peek RM, Miller GG, Tham KT, Perez-Perez GI, Zhao X, Atherton JC, et al. Heightened inflammatory response and cytokine expression in vivo to cagA+ *Helicobacter pylori* strains. *Lab Invest* 1995;73:760-70.

Inflammatory pseudotumour and Rosai-Dorfman disease of soft tissue

We read the recent article by Govender and Chetty¹ with great interest. The authors report the presence of a soft tissue lesion showing combined histological and immunophenotypic features of Rosai-Dorfman disease (RDD), and an inflammatory pseudotumour. They conclude that there may be a continuum between the two entities, with RDD representing the early histiocytic lesion and inflammatory pseudotumour the later (myo-) fibroblastic lesion. The alternative hypothesis is that aberrant cytokine expression in an inflammatory pseudotumour results in transformation of histiocytes.

The authors did not mention whether investigations for mycobacteria were performed. We have recently observed the