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 a diagnostic 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 because possible solutions 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. 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.

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. 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 detected 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 = 0.540). 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.

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.

Inflammatory pseudotumour and Rosai-Dorfman disease of soft tissue

We read the recent article by Govender and Chetty 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 significant 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