Correspondence

Safer staining method for *Helicobacter pylori*

Kuipers et al.1 recently reported a very low seroconversion for *Helicobacter pylori* infection in an adult population. They measured *H. pylori* immunoglobulin G (IgG) antibodies in two serum samples taken from each of 115 patients, obtained with a mean interval of 11-15 years, and found that only two patients became infected during follow up. From their data, the authors suggested that the age related increase in *H. pylori* prevalence was due to a dominant infection rate in childhood. Data on seroconversion in an untreated population are quite scarce. We report our data on 207 asymptomatic Italian children (aged 4-15 years) and 1010 blood donors (aged 18-65 years) who have been assessed serologically for both IgG and IgM (by in-house enzyme linked immunosorbent assay (ELISA), with a specificity and sensitivity of 93%).

Our results show that the prevalence of *H. pylori* IgG antibodies increases with age, both in children and in adult blood donors, but that the prevalence of *H. pylori* IgM antibodies is highest in the 18-25 year age group and that it decreases with age (fig. 1). Concentrations of IgG or IgM antibodies in *H. pylori* positive patients (measured by optical density at 470 nm) did not change with age. Our data strongly support their hypothesis of an age-cohort effect, with the acquisition of most *H. pylori* infection during youth (below the age of 20 years).

High IgM titres consistent with a first contact with the infection associated with low IgG titres, that consistently correlate with active *H. pylori* gastritis, may support the hypothesis of a spontaneous elimination of the infection in young patients. A spontaneous elimination of the first infection was shown in 33 out of 134 Gambian children aged 1-15 months by measuring serum antibodies and performing a 13C urea breath test every month over a period of 2 years. Most contact with *H. pylori* infection occurs in childhood, but the majority of younger subjects will spontaneously eliminate it. In Italians this occurs mostly during the second or third decade and in Gambians in the first 5 years of life; the difference is probably related to either hygiene conditions or the nutritional status of the population.

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Tissue artefacts caused by sponges

Following the recent correspondence by Platt and Newman regarding the use of tea-bags or synthetic Shandon bags in the processing of small biopsy specimens, we wish to draw attention to a tissue artefact which may occur when such specimens are processed in synthetic bags.1 Following the discovery that triangular shaped defects in renal and liver biopsy specimens were due to the use of foam sponges in embedding cassettes,2 we changed our procedure and processed all such specimens wrapped in perm paper. Recently, however, our laboratory ran out of perm paper and for a few weeks we processed renal biopsy specimens in Shandon bags. We soon noticed a triangular elliptical defect (fig 1) was occurring in tissue sections. Close inspection of the bag

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**Figure 1** Elliptical effect in tissue sections.

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It is caused
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abnormal cytokine produc-
tion. Recent evidence indicates that,
as in the mouse, human T cells of the T,3 subset
produce interleukin-2
(IL-2) and γ-interferon
(IFN), whereas
the T,2 subset produce
interleukin-4
(IL-4) and IL-5 but not IL-2 and γ-IFN.4 Further studies
suggest a functional relationship
between these two T cell subsets
and the production of Th1 and Th2
subsets.4 In the mouse, Th1 cells
produce IL-2, IFN-γ, and TNF-α,
while Th2 cells produce IL-4, IL-5,
and IL-10.5

Dr Metz comments:
We thank Dr Slater for his comments, and
agree that the prominence of eosinophils in
conditions like HES has distracted attention
from the possible cell of origin: a T cell.
We would also agree that the T cells
in our patient were probably of the Th2
type, and that the cytokine profile
seen in our patient was consistent
with Th2 cell activity. The presence
of eosinophils in the bone marrow
suggests a possible role in the
clinical manifestations of the disease.

Book reviews


The title promises much, but in fact this volume in the Cancer Surveys series aims to illustrate a series of models for selected cancers, and comprises a mixture of short articles. This review is in three sections: clinical, experimental, and molecular pathology. Interpretation of "model" includes animal, cell culture, and theoretical systems, so that the collection is a little heterogeneous. Of the 12 chapters, four are concerned with breast cancer (the article on human breast cancer by Drs Walker and Varley is particularly well presented) and two with colorectal cancer. The other half of the book deals with some molecular aspects of thyroid and pancreatic cancers, lymphomas, tumour metastasis and, slightly curiously, prospects for cervical cancer vaccines. There is also a brief review of oncogenes, growth factors, and control of the cell cycle, and a short biography of each of the 26 authors, many of whom are from the Imperial Cancer Research Fund laboratories. There is a certain amount of repetition and progress has been made. The nomenclature for p53 has been changed to TP53 throughout, except in the references. References are mostly up to 1991, with a few from 1992. The book is well produced but sparsely illustrated, mostly with line diagrams and a few photographs of cells or gels. It is difficult to know who buys this sort of book, but the reference lists could be useful to workers in the relevant fields.


It is with mixed feelings that I review this book. Having had the privilege of being trained by Dr Stokes, I cut my microbiological teeth on the fourth edition, and have frequently referred to it and subsequent editions. The changes in microbiology in the 90s and the role of the routine clinical laboratory have been addressed in the seventh edition.

The details of a busy routine microbiology laboratory are helpful, particularly in the prevailing climate. A clearer understanding of "value for money" is now

T cell lymphoid aggregates in idiopathic hypereosinophilic syndrome

Dr Metz and colleagues presented fascinating
information which suggests that some
of the hypereosinophilic syndrome
(UES) may result from occult T cell prolifera-
tion and interleukin-5
(IL-5) secretion. Unfortunately, in the absence of
bone marrow genotypic studies, it must
remain uncertain as to whether the pro-
liferation in Dr Metz's case had a poly- or
monoclonal origin. Interestingly, a relation-
ship between HES and cutaneous T cell lym-
phoma (CTCL) has also been highlighted
in French publications, together with
evidence that a interferon may be of
therapeutic benefit.

Dr Metz briefly discusses the relation-
ship between eosinophils and cutaneous T cell
disease, but sadly makes no mention of
helper T cell subdivision based on cytokine
production. Recent evidence indicates that,
as in the mouse, human T cells of the T,3 subset
produce interleukin-2 (IL-2) and γ-interferon
(IFN), whereas those of the T,2 subset produce
interleukin-4 (IL-4) and IL-5 but not IL-2 and γ-IFN. Further studies, such as the one
Metz presents, are likely to provide new
insights into the pathogenesis of eosinophilic
sarcoidosis.

Dr Metz comments:
We thank Dr Slater for his comments, and
agree that the prominence of eosinophils in
conditions like HES has distracted attention
from the possible cell of origin: a T cell.
We would also agree that the T cells
in our patient were probably of the Th2
type, and we in fact demonstrated
detectable concentrations of IL-5. However,
although many Th2 cell cytokines
are upregulated in this disease, the clinical
manifestations might also be due to
the presence of other cytokines.

We were surprised to find that the eosinophilic
infiltrate in the bone marrow
was not infiltrating the bone
marrow, as we reported, failed to
demonstrate any detectable
erythrophagocytosis or other
changes in bone marrow,