
Book Reviews

Fast Facts: Infection Highlights 1998–99. Ed. Mark H. Wilcox. Health Press, Oxford 1999. Pp. 102. £10.95. ISBN 1 899541 67 5.

For those doctors struggling to keep up with the pace of new developments in medicine alongside a busy clinical job, the ascendancy of electronic technology is meeting the need for accessible, concise, contemporary information. Fast Facts highlights series may represent the literary fightback. Infectious diseases and microbiology books have a shorter shelf-life than many other specialties; advances in molecular biology, changing antibiotic resistance and disease outbreaks demand regular updates.

This second edition aims to provide a concise overview of a spread of topics of particular value to infectious disease clinicians and microbiologists, but also of interest to other disciplines. Eleven topics feature new enemies (vancomycin-resistant *Staphylococcus aureus*, *E. coli* O157, TT virus) mingling with old favourites (meningococcal sepsis, community-acquired pneumonia, *Helicobacter pylori*). The current position on each subject is presented briefly enough to be read in 10–15 minutes, with a helpful summary of ‘What’s in, what’s out and what’s controversial’ and references for more detailed study. Colour-coding of pages enhances its ease-of-use.

Every top 10 (or top 11) list is notable for what is left out as well as what is included and readers wanting the latest on new-variant Creutzfeldt–Jacob Disease, for example, may have to wait until the 2000–2001 edition. Others may welcome the novelty of this ‘nvCJD-free zone’.

JOHN DAY

Hospital for Tropical Diseases, London

Microbes and Malignancy: Infection as a Cause of Human Cancers. Ed. J. Parsonnet. Oxford University Press, 1999. Pp. 465. £59.50. ISBN 0 19 510401 3.

In the popular imagination, the things that ‘cause’ cancer are cigarettes, chemical pollutants, nuclear power plants and mobile phones. Oh, and perhaps diet. A widely held image of cancer is of a sinister disease process that destroys normal tissue, that eats away part of the body. How, then, could its cause be anything other than some abnormal, environmentally aberrant, exposure?

We appreciate too little that there is rather a thin line between the complex biological processes of well-ordered cell proliferation and appropriate tissue renewal, on the one hand, and disordered, open-ended, tissue-deforming cell proliferation on the other. As is nicely pointed out in chapter 3 of this book, cancer, viewed simply, is the result of two abnormalities: faulty DNA replication during cell division, and excessive cell proliferation. There is a clear implication in several chapters that it is the latter abnormality that accounts for most of the exogenous causation of cancer. This is a refreshing perspective. True, certain reactive chemical molecules can damage DNA in ways that predispose to viable mutations in particular oncogenes; and ionizing radiation can also induce mutations. But DNA repair mechanisms have been well-honed by evolutionary pressures. Nearly all of the approximately 10000 oxidative ‘hits’ to DNA per cell per day in humans are repaired. Mutations therefore accumulate only slowly with ageing. Hence, the probability of inducing DNA damage that leads to a cancer-related mutation, and is accompanied fortuitously by other critical mutations that are prerequisite to malignancy, is not great.

A more plausible role for most ‘carcinogens’ is to increase cell proliferation. This can occur via chronic inflammation – as with long-term resident asbestos fibres in the lungs, *Schistosomiasis haematobium* in the urinary bladder, chronic hepatitis, *Helicobacter pylori* in the lower stomach and duodenum, and perhaps long-term inhalation of cigarette smoke. It can occur with sex hormones, either endogenous or exogenous, that impinge excessively on hormone-dependent tissues – breast, ovary, endometrium, or prostate. It can occur if viruses such as the near-ubiquitous Epstein–Barr virus (EBV) are released from normal host immunological controls (including via the HIV virus!) and induce a self-serving mitosis – a proliferation of lymphoid B-cells, and of their passenger viral particles, resulting in various lymphomas. It can also occur if viruses block the cell-regulating action of certain proteins – as with those strains of the human papilloma virus that vitiate the cell-cycle action of p53 protein. Indeed, when this occurs in basal-layer cells of the uterine cervix, and the normal process of cellular differentiation is impaired, proliferative squamous metaplasia, and they dysplasia, occurs.

There is much that is expert and detailed in this edited volume. The contributors are eminent laboratory scientists; the presentation is clear; the index is good. Yet there is a

more important, transcendent, property of this book – that is, a view of cancer as the result of altered patterns of cell and tissue biology caused by the presence of ‘microbes’: flukes, bacteria and viruses. The editor, Julie Parsonnet, foreshadows this perspective in her thoughtful introduction, recognizing the long coevolutionary background to parasitism.

The global epidemiological data indicate that around 20% of all cancer is due to cigarette smoking, around 30% is due to dietary imbalances, and around 20% is due to infection (with two-thirds of that being apparently due to viruses). Indeed, Parsonnet and, later, Harald Zur Hausen point out that this figure may be conservative. There are probably various infective agents involved in carcinogenesis in ways that we cannot yet imagine. One decade ago we did not know about the human herpes virus-8 as a cause of Kaposi’s sarcoma. Two decades ago we knew nothing of the role of *H. pylori* in stomach cancer. Three decades ago we were just beginning to know about hepatitis viruses and liver cancer. Four decades ago we still thought that the high rate of cervical cancer in lower socioeconomic class women, with multiple children, early age of sexual initiation, and multiple, uncircumcised partners was due to some unhygienic chemical-secretory exposure. Five decades ago we did not know that Burkitt’s lymphoma, a tumour affecting African children, had an infectious (viral) basis.

Indeed, during the first half of the twentieth century, there was general disdain for the idea that infectious agents could cause cancer. This disdain was one of the confining legacies of the germ theory. Koch’s postulates prescribed that every case of the (infectious) disease be associated with the infective agent, that the agent be identified and isolated, and that it induce the disease when administered in pure culture to another individual. Cancer did not behave that way. It was manifestly not a contagious disease. It did not measure up to the standards of the germ theorists.

Hence, the seminal findings of Peyton Rous, in 1911, of a transmissible subcellular agent (a virus) that caused sarcomas in chickens were dismissed, or ignored, for nigh on a half-century. At best it was an interesting curiosity, largely irrelevant because chickens were too-distant relatives of humans. Only when the paradigm shifted, and the role of infectious agents in cancer became a respectable idea, did Rous win a Nobel Prize for that early work – in 1966! It was in the 1960s that viral oncology got on the move. Baruch Blumberg discovered the hepatitis B surface antigen (the ‘Australia antigen’) which, subsequently was shown to predict the occurrence of hepatocellular carcinoma.

The sixteen chapters in this book are of good, even, quality. They nearly all dip briefly into the history of their area, so that the sense of an unfolding story is maintained. The first five chapters deal with mechanisms of infection-induced malignancy. The next six deal with viruses in cancer, and the final five review the role of parasites (flukes and worms) and bacteria in cancer. It is a pity there is no concluding chapter, drawing the lines of research together and contemplating the future. A recurring implication in this book is that cancer is an occasional, incidental, consequence of microbes colonizing human bodies as they

pursue their ancient, self-interested, ecologically legitimate search for nourishment and reproductive opportunity. This is an interesting metaphor for humankind’s rapidly intensifying colonization of the biosphere.

A. J. McMICHAEL,

*Department of Epidemiology and Population Health,
London School of Hygiene and Tropical Medicine,
London, UK*

Salmonella enterica Serovar Enteritidis in Humans and Animals: Epidemiology, Pathogenesis and Control. Eds. A. M. Saeed, R. K. Gast, M. Potter and P. G. Wall. Iowa State University Press, 1999.

Salmonella enteritidis, as we would more usually write it in the United Kingdom, is, in numeric terms at least, the foodborne disease of the last decade. The rise in *S. enteritidis* has driven the annual total for salmonellae which has been a major contributor to the rise in statutory notifications of food poisoning. On the way it has spawned numerous official committees and reports, the demise of a government health minister and was the first of a number of well publicized food scares which have disproportionately afflicted Britain to the point of attracting transatlantic notice [1]. What this valuable book makes clear is that, with the possible exception of the instructive example of Sweden, this epidemic was a worldwide phenomenon. As Angulo and Swerdlow repeat, in their chapter on epidemiology in humans in the US, *S. enteritidis* ‘is the prototypic emerging pathogen’. Why this should be so is the subject of a comprehensive epidemiological overview by Poppe, of Health Canada, and a number of country reports which show the fascinating conjunction of similarities and differences, not just in the *S. enteritidis* story, but also in public health organization. The chapter by Gerner-Smidt and Wegener on Denmark shows the value that can be added to surveillance by good organization within a clearly thought out epidemiological and statistical framework.

Control is also well covered and the means used to address it in different countries vindicates Tauxe’s assertion in his foreword that the ‘hallmark of the efforts to control these new foodborne disease challenges’ will be ‘a multi-disciplinary approach’. However it does seem that there may be something more akin to a ‘silver bullet’ than he envisaged when writing. It is an oft repeated truism that text books are out of date by the time they are published and during the last year in the UK, *S. enteritidis* has started to fall dramatically. The reasons for this are yet to be evaluated but slaughter and restocking of primary breeders and vaccination of flocks lower down the production chain may be anticipated to have played an important part. Does this vitiate the book’s usefulness? Not at all; the multidisciplinary understanding it displays is a *sine qua non*, whatever means would be required to control *S. enteritidis* not to mention other contemporary foodborne epidemics of equally complicated genesis. In an age when we can access more information than we can assimilate, at the press of a

computer key, this book illustrates why there is still a place for multi-authored textbooks. It collects together authoritative information in an accessible format. My only criticism might be of the one or two contributions that effectively write up scientific projects which should more properly have been placed in peer reviewed journals. Overall, however, the book could usefully find a place on the shelf of

any who have to consider critically food safety issues. It is likely to be frequently consulted.

R. L. SALMON

*Public Health Laboratory Service Communicable Disease
Surveillance Centre (Wales)*

1. Firth P. Consuming fears. *Sci Am* 1999; **280**: 24–5.