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Book Review

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Genetic Predisposition to Cancer Editors: R.A. Eeles, B.A.J. Ponder, D.F. Easton and A. Horwich Publishers: Chapman and Hall Medical, London (1996)

MUCH HAS been learned in recent years about genetic predisposition to cancer. Genes causally related to inherited cancer syndromes have been isolated and characterised. Genes have been cloned which predispose to familial common cancers, such as breast, ovarian and colon, and it seems safe to predict that additional cancer predisposition genes will be discovered in the near future. Genetic screening of individuals considered at high risk for developing cancer has become a distinct possibility. Genetic screening is associated with a host of potential psychological and ethical problems, and the consequences for clinical management, the decision concerning having children and various other issues can be profound.

The book offers a comprehensive overview of this exciting field, covering all topics of interest. In Part I, the reader is introduced to the concepts and strategies of gene mapping and cloning and relevant statistical techniques such as the LOD score calculation. Furthermore, the standard terminology is defined and explained. Parts 2 and 3 give a comprehensive account of the current knowledge on inherited cancer syndromes and chromosome fragility syndromes, respectively. Part 4 deals with the genetics of familial common cancers, the clinical management of individuals harbouring mutations in particular genes and the potential psychological sequelae. The chapters in the final part discuss the concept of cancer family clinics, the strengths and shortcomings of the major laboratory techniques available for screening for gene mutations, and the ethics of genetic testing.

Considering the complexity of, and rapid development in, this area of medicine, the thoroughness and comprehensiveness of the book is remarkable. All topics are discussed critically, providing the reader with a well-balanced view of contentious issues. Importantly, the authors have succeeded in presenting the material in a way which is easy to comprehend even for readers with little knowledge in the field. Therefore, the book can be highly recommended not only to experts in the field but to every healthcare professional with an interest in the cancer problem.

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Letters

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Letter to the Editor: Comments on *Health-related Quality of Life of Adults Surviving Malignancies in Childhood*, Apajasalo *et al.*, *Eur J Cancer*, 32A, No. 8, pp. 1354–1358, 1996

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THE REPORT by Apajasalo and associates [1] is an important contribution to the expanding body of knowledge concerning health-related quality of life (HRQL) in survivors of cancer in childhood. However, the results should not have come as a surprise to the investigators, nor to the author of the accompanying editorial [2]. More than 40% of the patients had acute lymphoblastic leukaemia (ALL) and the majority of these individuals would have had low or standard risk disease. We have reported that the HRQL of survivors of standard risk ALL is similar to the HRQL in a comparable sample of the general population [3]. Using the

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multi-attribute utility function for the Health Utilities Index Mark 2 system [4], the mean global utility score for a control group [5] is 0.95 and for survivors of standard risk ALL is 0.96, but for survivors of high-risk disease it is 0.90.

Furthermore, the remarkable (and unexplained) absence of survivors of brain tumours in the Finnish cohort avoids the inclusion of a group of individuals who bear a heavy burden of morbidity, as we have also reported [6, 7]. These reports [3, 6, 7] were published before Apajasalo and associates resubmitted their manuscript.

The Finnish study provides an overoptimistic assessment of HRQL in survivors of cancer in childhood, and is not generalisable to that population. It can be argued that 'the tale is in the tail'—the subjects with the important levels of morbidity are often to be found in the right-hand tail of the distribution of HRQL scores, and measures of central tendency (e.g. mean score) can often obscure important characteristics of the sample.

Finally, the statement that 'the patient is known to be the most appropriate source of this information' is overly simplistic, and the concept of 'multiple truths' is an important and valid basis for interpreting results from various perspectives [3].

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Breast Cancer and HIV Infection: Two Case Reports

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BREAST CANCER is, as yet, not thought of as an HIV associated cancer. However, several cases of this association have been reported [1–4] and an epidemiological link may exist. Above all, HIV infection may influence the course of breast cancer [1]. We describe here two cases of breast cancer in HIV infected women.

Patient 1 was diagnosed HIV seropositive in 1985 when she was 36 years of age. Because of a suicide attempt, she received a blood transfusion in 1982. In 1993, as the HIV infection was still asymptomatic, she presented with a right breast mass classified as T2N1b. Radical modified mastectomy was performed. The histological examination showed an infiltrating ductal carcinoma grade 3 and 5 positive nodes out of 8. Oestrogen and progestin receptors were positive. Chest radiograph, bone scan and liver ultrasound were negative. The patient then received locoregional radiotherapy and six cycles of FEC (5-fluorouracil, epirubicin, cyclophosphamide) chemotherapy without any major toxicity. The CD4 count before and after chemotherapy was 700 and 234 mm^3, respectively. Seven months after completion of chemotherapy, the patient developed an isolated histologically proven bone metastasis. Treatment with 5-fluorouracil (5-FU) and vinorelbine (FUN) was initiated in August 1994 with a CD4 count of 230. The first course was complicated by grade IV stomatitis and leucopenia, so subsequent courses were administered at a reduced dose (30%) and were well tolerated.

In February 1995, the disease was evaluated as stable, the CD4 count was 223 and the patient was given an LHRH (luteinising hormone releasing hormone) analogue until September 1995, when she progressed (bone, skin, nodes). She is now receiving semi-intensive chemotherapy with epirubicin and cyclosphosphamide without unexpected toxicity.

Patient 2 was diagnosed HIV seropositive in 1990 (heterosexual transmission). She was still asymptomatic in 1993 when zidovudine was started because of a low CD4

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