5th International Conference on Adjuvant Therapy of Primary Breast Cancer

This conference will be held in St Gallen, Switzerland between 1–4 March 1995. The deadline for abstracts is 31 October 1994. For more information, contact Mrs B Nair, c/o Professor H J Senn, Department of Medicine C, Kantonsspital, CH-9007 St Gallen, Switzerland. Tel. 41 (0) 71 62 10 97; Fax 41 (0) 71 25 68 05.

19th LH Gray Conference: Quantitative Imaging in Oncology

This meeting will be held between 3–4 April 1995 in Newcastle, U.K. Papers and posters are invited on diagnosis and quantitation of disease, techniques for treatment volume definition, uses of various imaging modalities in treatment planning, treatment planning systems—the state of the art and its current potential value, treatment verification and quantitative assessment of tumour response. It is anticipated that the meeting's proceedings will be published as a peer-reviewed supplement of a leading scientific journal. For further information, contact Dr K Faulkner, Regional Medical Physics Department, Newcastle General Hospital, Westgate Road, Newcastle-Upon-Tyne NE4 6BE, U.K. Tel. (091) 273 8811, ext. 22400; Fax (091) 226 0970.

Third Central European Lung Cancer Conference

This meeting on recent developments and controversies in lung cancer will be held in Prague, Czech Republic on 28–31 May 1995. For further information, contact the Conference Secretariat, Czech Medical Association, J E Purkyne PB 88, Sokolská 31, 120 26 Prague 2, Czech Republic. Tel: (42) 2 296889/297271; Fax: (42) 2 294610/24216836.

European Cancer Centre/NDDO Postgraduate Course on Basic and Current Topics in Anticancer Drug Development

This will be held on 7–8 September 1995 at Vrije Universiteit, Amsterdam, The Netherlands. For further information contact the European Cancer Centre, c/o AZVU, PO Box 7057, 1007 MB Amsterdam, The Netherlands. Tel. 31 20 644 4500/4550; Fax 31 20 644 4551.

The Sixth International Conference on Topoisomerases: From Molecular Aspects to Clinical Application

This conference, organised by the European Cancer Centre and NDDO, will be held on 1–4 October 1995 at Vrije Universiteit, Amsterdam, The Netherlands. For further information contact the European Cancer Centre, c/o AZVU, PO Box 7057, 1007 MB Amsterdam, The Netherlands. Tel. 31 20 644 4500/4550; Fax 31 20 644 4551.

European Journal of Cancer Vol. 30A, No. 7, pp. 1039–1040, 1994.

0959-8049/94 $7.00 + 0.00

Letters

Vasectomy and Testicular Cancer

K. P. Dieckmann

IN AN ISSUE of this journal (Vol. 29A, No. 7), much space was devoted to the question of whether vasectomy can increase the risk of testicular germ cell cancer (GCT). While the epidemiological data presented [1] are hardly misleading, the take-home message for the reader remained somewhat less clear-cut.

Our own data clearly support the contention that there is no increased risk for GCT after vasectomy: in a case–control study, 4 patients among 538 patients (0.74%) treated for GCT during the period 1969–1990 in five Berlin-based urology departments were found to have had previous vasectomy. In the control group which consisted of 531 otherwise healthy age-matched males treated for various injuries in the same hospitals, 4 men also reported a previous vasectomy (0.75%). The relative risk (RR) thus amounts to 0.98 with 95% confidence intervals 0.24–3.96, indicating that there is no significant association between the two conditions ($P = 0.736$, χ^2 test).

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Received 27 May 1993; accepted 9 Dec. 1993.

Whether or not these data and similar data of Morris Brown and colleagues [2] that were not mentioned in the article [1], are added to the meta-analysis of Lynge and colleagues [1], there was no need to formulate the conclusions as cautiously as the authors did. In fact, there is overwhelming epidemiological evidence that vasectomy does not have a bearing upon the pathogenesis of testicular GCT.

In addition, the biological explanation of why vasectomy could at least accelerate the occurrence of GCT as offered by Jorgensen and colleagues [3] is a rather mechanical view on the pathogenesis of GCT. While it is undisputed that all GCT are derived from testicular intraepithelial neoplasia (TIN; carcinoma *in situ*), the factors influencing the transition of TIN to frank GCT are unknown [4]. However, it is much more probable that molecular genetic or immunological [5], or endocrinological factors [6] are involved rather than simple increase of fluid pressure within the rete testis and seminiferous tubules. Even if this hypothesis was true, it would not account for an overall increase of GCT in the group of vasectomised men.

In summary, epidemiological studies have failed to document an association of GCT and vasectomy [7]. The only study that indicated an increased risk showed a relative risk of 4.2 which is not a convincingly high factor, as demonstrated by wide 95% confidence intervals of 1.8–8.2. Thus, a selection bias might

possibly account for this result. In the light of a missing biological link for the putative association of the two conditions, the conclusion must clearly be that there is no increased risk for testicular germ cell tumours after vasectomy.

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3. Jörgensen N, Giwercman A, Hansen SW, Skakkebaek NE. Testicular cancer after vasectomy: origin from carcinoma *in situ* of the testis. *Eur J Cancer* 1993, **29A**, 1062–1064.
4. Dieckmann K-P, Loy V, Huland H. Das Carcinoma in situ des Hodens: klinische Bedeutung, Diagnose und Therapie. *Urologe A* 1989, **28**, 271–280.
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European Journal of Cancer Vol. 30A, No. 7, pp. 1040–1041, 1994.

0959–8049/94 $7.00 + 0.00

Unusual Clonal Evolution During Blast Crisis of Chronic Myeloid Leukaemia (CML)

F. Christodoulidou, R.T. Silver, M.J. Macera and R.S. Verma

THE PROGRESSION of chronic myelogenous leukaemia (CML) to blast crisis is generally associated with additional chromosomal abnormalities. The four most frequent changes observed are trisomy 8, isochromosome 17q, trisomy 19 and/or an additional Ph chromosome. In addition to these changes, more than 300 chromosomal abnormalities, involving so-called unusual or variant translocations, have been reported [1, 2]. We present a previously unreported chromosomal abnormality within a multiclonal marrow population in a patient with blast phase CML, where the clone containing the aberrant chromosome became the dominant clone.

The patient, a 49-year-old Caucasian male with a diagnosis of CML, was treated initially with γ-interferon (IFNγ, 9.8×10^6U) for 2 years without any significant response, and then recombinant interferon-α (rIFN α-2b, 6×10^6U) three times a week. He was maintained on rIFN α-2b, 6×10^6U. After 17 months of treatment, his disease entered complete clinical remission.

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Revised 3 May 1993; accepted 11 Oct. 1993.

Initial cytogenetic evaluation of our patient revealed the typical 46,XY,t(9;22)(q34;q11) karyotype of CML, and a positive *bcr* rearrangement. Two years later, although clinically in chronic phase, the dividing marrow cells began to display blast crisis abnormalities, with up to three additional clones, each clone containing one extra chromosome 17 and up to two extra chromosome 8s. Two years after detection of the additional clones, the patient was clinically in blast crisis. At that time, 88% of his dividing marrow cells contained a highly unusual aberration. A duplication of the long arm of chromosome 1 (q11→qter) had translocated to one chromosome 10(p12) with loss of the 10 (p12→pter) (Figure 1). A cell line with 48 chromosomes, including a +8 and +17 in addition to the der(10), became the dominant clone (49%). Five separate cell lines were present.

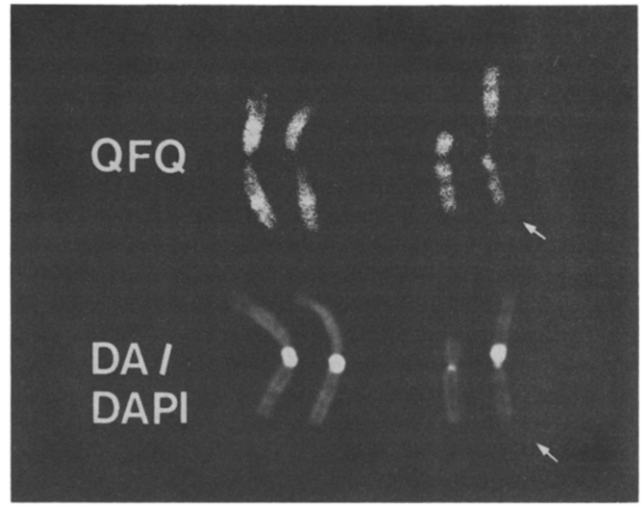

Figure 1. Aberrant chromosome exhibiting an unusual translocation involving chromosomes 1q and 10p. The centromere of chromosome 10 is retained in the translocation. QFQ, Q bands by fluorescence using G binding; DA/DAPI, binding technique.

A perplexing question surrounding the onset of blast crisis is whether additional cytogenetic aberrations initiate it or whether they are epiphenoma. In our case, the additional +8 and +17 chromosomal abnormalities proceeded the clinical onset of blast crisis. The presence of additional chromosomal abnormalities and, in a few cases, amplifications or mutations of protooncogenes in acute phase are suggestive of a possible role for additional genetic events in the evolution of CML. [3,4].

Recently, Ahuja and colleagues [5] demonstrated that transcripts of the p53 gene (a 53 kDa nuclear protein) were uniformly detected in chronic phase cells from 38 CML patients; however, in 10 out of 16 patients in blast crisis, p53 transcripts were either reduced or undetected. They also showed that heterogeneous anomalies in the structure and expression of the gene appear to be common for blast crisis but are generally not observed in other human malignancies. Numerous studies have suggested a role for the p53 gene located on chromosome 17p in cellular proliferation, regulation of the cell cycle and oncogenesis [6], and in several mouse erythroleukaemia cell lines, it appears to be acting as a negative regulator of cell growth [5]. It is of interest to note that the aberrant 48, XY,t(9;22), der (10),+8,+17 clone not only became the dominant clone, but further transformed by adding a duplicated 1 (q11→qter) translocated to the 10(p12).