Cutaneous Lymphoma Project Group (Burg et al, 1998) involving 827 patients CTCL, which includes MF and SS, was four times as frequent as cutaneous B-cell (CBCL) lymphoma. This indicates that in the EORTC data non-MF CL comprises about 20% of all CLs. Over 700 patients with CL have been registered at the University of California, San Francisco CL, USA, clinic since 1971. Approximately 5–6% were of the non-MF type (unpublished data). Therefore, the comparison of Iscovich et al's (1998) data with that of Weinstock and Horm (1988) requires qualification.

The authors note that the incidence of CL in the USA rose from 1973 to 1984, '... and may have continued to rise (Weinstock, 1994; Koh et al, 1995).' However, I could find no data or statement in either of those two articles to support the suggestion of a continued increase in the incidence of CL since 1984. In fact, Koh et al (1995) state, 'However, preliminary analyses did not find that the crude incidence of MF continued to increase during the subsequent 6 years (MA Weinstock, unpublished data).'

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Possible relationship of the apolipoprotein E (ApoE) ε4 allele to prostate cancer

Sir,

Mantzoros et al (1997) report that increased insulin-like growth factor 1 levels are a risk factor for prostate cancer. Another molecular marker, the apolipoprotein E (ApoE) ε4 allele, is a risk factor for Alzheimer's disease (Poirier et al, 1993) and might be a risk factor for prostate cancer as well.

Alzheimer's disease and prostate cancer share a common incidence pattern. Onset of Alzheimer's disease before age 60 is infrequent and caused by specific gene abnormalities. Prostate cancer is also rare in men before age 60, and there is generally a strong genetic component in these cases. As men get older, the prostate cancer incidence continues to increase, and the older cases do not generally have a family history (Stephenson, 1996). Like Alzheimer's disease, the supposition is that if men get old enough most will develop prostate cancer.

Among the three ApoE alleles, the ε4 allele confers the highest Alzheimer's disease risk. The ε3 allele is associated with less risk, and the ε2 allele appears to be protective. The ε4–ε4 genotype (i.e. two ε4 alleles) confers the highest risk of all (Strittmatter and Roses, 1995).

The antioxidant activity of ApoE alleles protects cells in culture from oxidative damage. The ε2 allele is most protective, the ε3 allele less so, and the ε4 allele least protective of all (Miyata and Smith, 1996). The decreased antioxidant activity of ε4 could contribute to its association with Alzheimer's disease. Because antioxidants also protect against cancer (Duthie et al, 1996), the ε4 allele might predispose to the development of malignant disease.

In 35 men with prostate cancer, ApoE genotype was determined by polymerase chain reaction with a standard method (Slooter et al, 1997). The frequency of the ε4 allele was 0.24. This may be compared with a control ε4 allele frequency of 0.135 or 0.138, reported by Slooter et al (1997). The increased frequency of the ε4 allele in the prostate cancer cases resembles its increased frequency in dementia of 0.22 (Slooter et al, 1997).

Furthermore, the two prostate cancer patients who were homozygous ε4–ε4 were age 52 and 58 years, significantly younger than the average age (67 ± 5.7 years, mean ± s.d.) of the 33 other patients (P = 0.0248, Mann–Whitney U test and Wilcoxon rank sum W-test corrected for ties). In Alzheimer's disease, the patients who are homozygous ε4–ε4 also have the earliest disease onset (Blacker et al, 1997). Thus, further investigation of the possible relationship of ApoE to prostate cancer, and perhaps other forms of cancer, might be worthwhile.

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