PREDNISONE (Group 3)

A. Evidence for carcinogenicity to humans (*inadequate*)

Many case reports of cancer include a mention of previous treatment with prednisone, as would be expected by chance alone in view of the very wide use of this drug in many different disorders. Prednisone is a common drug, prescribed for long periods in the treatment of many chronic conditions[1]. Patients treated with prednisone for rheumatoid arthritis appear to have, if anything, a lower than expected cancer risk. Over an average follow-up period of 12 years, 11% of 153 deaths that occurred in patients who had received prednisone were due to malignancies, compared to 20% of 74 deaths among patients who had not received prednisone[2]. The strong link between combination therapy for Hodgkin's disease and subsequent second malignancies (see summary of data on MOPP and other chemotherapy including alkylating agents, p. 254) is much more plausibly explained on the basis of concurrent administration of clearly carcinogenic agents than of prednisone.

A study of cancers that appeared within four years after documented use of common drugs showed that prednisone was among the 53 (of 95) drugs associated positively with cancer at least once. However, the excess consisted of 12 cases of lung cancer (31 observed, 19 expected), known to be largely related to cigarette smoking (which was not measured) and known to occur after a latent period much longer than the interval under observation.

Of more interest is the absence of those neoplasms, such as acute nonlymphocytic leukaemia and non-Hodgkin's lymphoma, which have been linked to chemotherapy and immunosuppression[3].

Thus, the evidence for a carcinogenic action of prednisone was not compelling. The evidence did not, however 'suggest lack of carcinogenicity', because there is no well-designed analytical study of prednisone alone.

B. Evidence for carcinogenicity to animals (*inadequate*)

Prednisone was tested for carcinogenicity in mice and rats by intraperitoneal administration. A significant increase in the total number of tumours was reported in female rats, but the study suffered from limitations in both design and reporting[1].

C. Other relevant data

No data were available on the genetic and related effects of prednisone in humans. It did not induce chromosomal aberrations in bone-marrow cells of rats treated *in vivo*. It was not mutagenic to bacteria[4].

References

[1]*IARC Monographs, 26*, 293-309, 1981

[2]Fries, J.F., Bloch, D., Spitz, P. & Mitchell, D.M. (1985) Cancer in rheumatoid arthritis: a prospective long-term study of mortality. *Am. J. Med., 78 (Suppl. 1A)*, 56-59

[3]Friedman, G.D. & Ury, H.K. (1980) Initial screening for carcinogenicity of commonly used drugs. *J. natl Cancer Inst., 65*, 723-733

[4]*IARC Monographs, Suppl. 6*, 472-473, 1987