

classes of binding sites for Hp which are probably located in the apoprotein matrix and the lipid core, respectively. Lipoproteins play an important role in the transport of haematoporphyrins (Hp and HpD) in the bloodstream, it has been suggested that tumor localising property of the porphyrin photosensitisers used in photodynamic therapy (PDT) is related to such binding, particularly to low lipoprotein.

LDL binds to specific receptors on cellular membranes by arginyl and lysyl residues. Chemical modification of these residues abolishes the interaction between lipoproteins and receptors both *in vitro* and *in vivo*. *In vitro* studies have also demonstrated that tumors have a higher affinity binding for LDL because of an increase of the number of membrane LDL-receptors. To a greater understanding of transport mechanism of porphyrins we have studied the affinity of HpD to proteic amino acid by thin layer chromatography and nuclear magnetic resonance (NMR), using a standard mixture. Our results indicate that the tryptophane is the aminoacid mainly involved in HpD binding. Whereas other aromatic aminoacids are at secondary levels. Furthermore a study on the binding sites of human serum albumin to HpD by NMR is also presented. A better knowledge of these binding sites will allow to improve photodynamic therapy and to transfer selectively antitlastic chemo therapeutic drugs in neoplastic cells.

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PUBLICATION

ONCE A DAY (I.E. 24 HOURLY) KAPANOL™, A NEW SUSTAINED RELEASE MORPHINE FORMULATION, IN THE TREATMENT OF CANCER PAIN: PHARMACOKINETIC ASPECTS

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Sustained release morphine formulations are now generally accepted as the formulations of choice in the treatment of severe cancer pain. Kapanol™/Kadian™ (Glaxo/Faulding), a new sustained release formulation consisting of polymer coated pellets in a capsule, has recently

been shown to have a superior morphine release profile compared to the standard tablet, MS Contin. If fact, the release characteristics of morphine from Kapanol suggested the possibility that one oral dose of Kapanol per 24 hours could effectively treat severe cancer pain. Twenty four patients completed a randomised, double-blind, two period, crossover study comparing 24-hourly Kapanol to 12-hourly MS Contin. The morphine dose per 24 hours was identical for the two formulations and morphine pharmacokinetics were determined over 24 hours at steady state for each formulation (i.e. after 7 days). The Cmax, Cmin, AUC and fluctuations in plasma morphine concentrations were not significantly different between the two formulations ($P > 0.05$, repeated measures ANOVA). Kapanol had significantly longer Tmax values ($P < 0.001$) than MS Contin. These results indicate that one Kapanol dog per 24 hours should at least be as efficacious as 12 hourly MS Contin in the treatment of severe cancer pain from the pharmacokinetic perspective.

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PUBLICATION

PALLIATIVE SINGLE-DOSE THIRD-BODY IRRADIATION OF MULTIPLE METASTASES IN TERMINALLY ILL PATIENTS

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In the group of 34 patients (pt) with multiple bone, lymph-nodes and visceral metastases 44 third-body (ThB) areas were irradiated using 600 cGy to the upper ThB and 1000 cGy to the middle ThB. The most of pt were terminally ill. Within the group 27 pt (80%) were III° or IV° of physical activity WHO (Zubrod) scale.

The result of the treatment was either fast relief of pain (40% doing so within 24–48 h.) as well as change to more active physical state. WHO scale III° or IV° were 17 pt (50%) according to 27 (80%) before ThB irradiation. All pt were hospitalised and were discharged typically one day after ThB irradiation. No deaths were seen immediately.

The method seemed to improve quality of life in about 40% of pt, not duration of life.

Testicular tumours

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ORAL

VARIATIONS IN CANCER SURVIVAL: A QUALITY ASSURANCE CHALLENGE

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Published survival figures for cancer indicate significant variations across Europe. Much of this variation may be due to different cancer registration practises and it is notable that two European countries, Denmark and Scotland, which have effective population based cancer registries, apparently low five year survivals for a range cancer.

Recent changes in the way health care is administered within the United Kingdom have made it possible to examine in detail variations in clinical practice. It is clear that much of the variation in outcome is related to differences in standards of clinical care. Clinician and centre specific outcome following treatment for testicular teratoma, melanoma, breast cancer and colon cancer have been reviewed. For these cancers some hospitals are no longer recognised as providing effective treatment and for the commoner cancers, individual clinicians have been accredited within hospitals.

Further work on the cost-effectiveness of various interventions has been done which has resulted in the Health Authority restricting the development of new services such as a screening for prostate cancer. Progress towards the delivery of cancer care on sound research evidence will be described.

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ORAL

ESTROGEN RELATED CANCER RISK IN MOTHERS OF TESTICULAR CANCER PATIENTS

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The belief that estrogens are centrally involved in the pathogenesis of both testicular cancer in young men and cancers in the endometrium and female breast has become widespread. In order to provide further insight into the possible hormonal links between these cancers, we investigated the cancer pattern in a large cohort of women who had given birth to sons who developed testicular cancer. Particular focus was given to estrogen-related cancers.

Material: Mothers of 2,204 testicular cancer patients were identified, with a total of 70,063 person years of follow up.

Results: No increased risk was found with regard to breast, endometrial or ovarian cancers among mothers of testicular cancer patients. Brothers of testicular cancer patients have a significantly increased relative risk of developing testicular cancer ($P < 0.001$).

Conclusion: On the basis of this large study no clues of common hormonal etiology of testicular cancer in young men and cancers of the endometrium and breast could be identified. Some kind of hereditary predisposition of testicular cancer seems likely.