Table 1. Summary of the typing results for HLA-A and DR loci identified on HCC cell lines

Cell line	Ethnic group*	Amplified alleles A	Amplified alleles DR
HA22T/VGH	O	2,—	12,—
HCC-M	O	11,—	1,—
HCC-T	O	28,—	1,—
HuH-7	O	2,—	8,—
huH-1	O	9,29	8,—
huH-4	O	9,26	9,—
HuH-6	O	2,9	9,12
KMCH-1	O	11,31	8,17
Sk-Hep1	C	2,9	10,11
Tong/HCC	C	9,29	7,16
HepG2	C	2,9	16,18
Mahlavu	C	2,11	9,8
Hep3B	B	28,29	7,18
PLC/PRF/5	B	10,33	8,12

*O, oriental; C, caucasian; B, black. The numbers under A and DR represent the equivalent of serologically defined HLA specificities.

is approximately 10%. Due to the highly polymorphic characteristics in the HLA region, it is rare to observe homozygosity in two HLA loci at the same time. Yet in this study, a 29% (4/14) of HCC cell lines were homozygous at both HLA-A and DR. Our results therefore suggest that the high frequency of homozygosity of HLA alleles seen in HCC is a result of deletion of the genomic HLA locus. Since there is no selective advantage for the HCC cells *in vitro* to lose HLA antigens, it is most likely that the deletions occurred in the primary tumour, which is under selection pressure by the host immune system. As the human HLA gene complex is located on chromosome 6p21, deletions may occur at the short arm of chromosome 6 or possibly the entire chromosome. Whichever the mechanism, the effect of HLA loss means that the HCC cells have effectively reduced their antigenic exposure to the host T cells immune surveillance.

In conclusion, in this preliminary study, we have observed loss of heterozygosity/deletion of HLA-A and DR loci in 29% of HCC cell lines studied. Although some caution is required in evaluating these results, our findings are in agreement with those reported for other types of cancers [3–7]. Further work needs to be done to study liver cancer cells from primary tumours to determine the precise genetic alterations in HLA genes in greater detail. These findings may serve as a guide to evaluate possible mechanisms used by HCC cells to escape immune recognition *in vivo*.

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Adenocarcinoma of the Eccrine Sweat Gland: Response to Both Combination Chemotherapy and Local Field Irradiation

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ADENOCARCINOMAS OF the eccrine sweat gland are rare malignancies with a tendency to invade locally and recur; occasional

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patients with metastatic disease have been reported [1]. Very little information has accumulated on the role of chemotherapy and radiation therapy in the palliation of patients with extensive disease. We describe the case of a patient who demonstrated evidence of response to both treatment modalities.

A 57-year-old caucasian man was initially diagnosed with adenocarcinoma of the eccrine sweat gland involving the left axilla in 1983, and was treated with left axillary dissecton as well as resection of the skin malignancy. He remained well until 1990 when a mass developed in his lower left neck. A left modified neck dissection was performed in June 1990; histopathology and electron microscopy confirmed the recurrence of the original malignancy. Subsequently, a number of left neck masses developed and three further surgical excisions were performed. He was then referred for external beam radiation, but was found to have very extensive involvement with infiltration of the skin from the left pre-auricular area down through the left neck, and the anterior chest wall to the level of the nipple and extending medially toward the midline. The patient had also developed lymphoedema of the left arm, as well as generalised weakness and continuous diaphoresis. Chemotherapy was initiated with CAP (cyclophosphamide 500 mg/m^2, doxorubicin 50 mg/m^2 and cisplatin 50 mg/m^2) every 3 weeks commencing in October 1992. The mass located anterior to the left pinna resolved completely, along with the erythema and induration of the skin of the anterior chest and neck; the lymphoedema of his arm also diminished considerably. The patient completed a total of five cycles of CAP chemotherapy, with no clinical or radiographic evidence of residual malignancy.

The patient remained well for a year after the completion of chemotherapy. New dermal lesions were then noted in the skin of his left neck, as well as lymphadenopathy in his right axilla, the latter measuring 3 cm in size. As a result of increasing pain in his neck, he received further chemotherapy with CAP. After one course of chemotherapy, the skin lesions disappeared, and the right axillary nodes shrank to 1.5 cm (Figure 1). One further course of CAP was administered, with further shrinkage of his right axillary lymphadenopathy.

Seven months after completing the final course of chemotherapy, he developed a recurrence of his right axillary lymphadenopathy as well as a mass in his lower left anterior neck. As a result of the patient's reluctance to have further chemotherapy, external beam radiation (30 Gy in 10 fractions) was delivered with complete clinical regression of the tumour in the treated area. Interestingly, the mass anterior to the left pinna which was present prior to commencing chemotherapy has not recurred.

Chemotherapy has been employed only occasionally in this malignancy. Many of the cases were treated adjuvantly after surgical resection of measurable and evaluable disease; the effectiveness of therapy in these cases cannot be evaluated [2, 3]. In cases of advanced disease, little evidence of response has been found despite a variety of chemotherapy agents being employed [1, 4–6]; only one convincing case of eccrine sweat gland carcinoma responding to chemotherapy has been described [7]. The current case differs from those reported previously in that the chemotherapy regimen included cisplatin and, as did the other responding case, doxorubicin. In contrast to other reports [1, 4], our patient achieved substantial symptomatic improvement as well as clinical control of the tumour in the treatment field with local irradiation. This suggests that patients with advanced eccrine carcinomas may achieve some palliative benefit from both irradiation and chemotherapy, despite the poor results reported in the past. It further suggests that, in patients suffering from this malignancy for whom palliative chemotherapy appears to be appropriate, CAP, or other regimens employing doxorubicin and/or cisplatin should be considered.

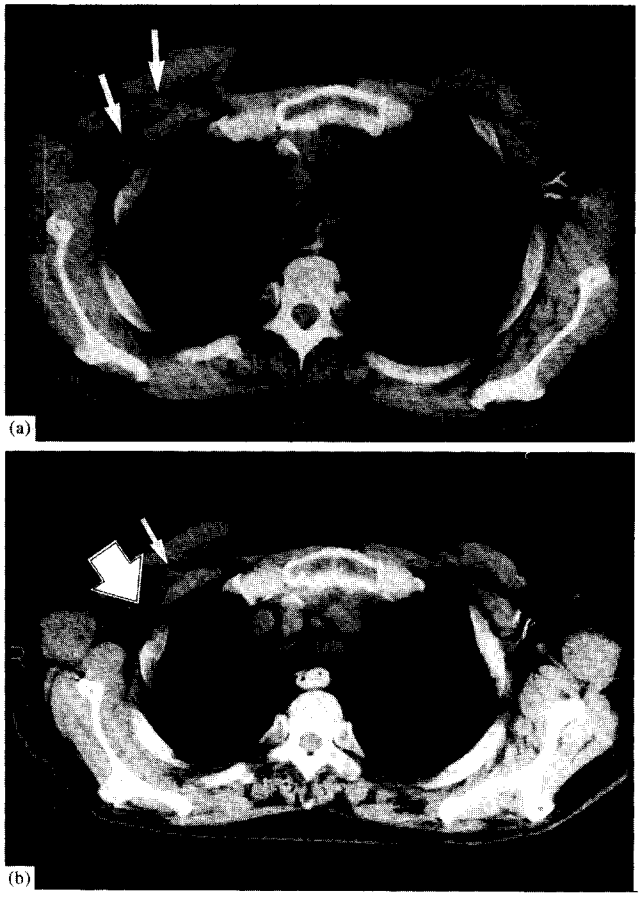

Figure 1. (a) Computerised tomography of the thorax demonstrating enlarged axillary lymphadenopathy (arrows). (b) Computerised tomography of the thorax demonstrating regression of axillary lymphadenopathy (arrows) after one course of repeat chemotherapy.

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