recently, 5 cases of paclitaxel-associated radiation-recall reactions [5–7] and a single case of radiation-recall mucositis associated with docetaxel have been reported [8]. The precise mechanism is unknown. One hypothesis suggests that cytotoxic treatment after radiotherapy causes a 'remembered' reaction in the remaining surviving cells within the previously irradiated field. An alternative proposition suggests that radiation induces heritable mutations within surviving cells which then produce a subgroup of defective stem cells that are unable to tolerate the second insult of chemotherapy [9]. Paclitaxel [10] has been found to be a radiosensitiser, but it is unclear how this might correlate with an ability to reactivate latent radiation effects in normal tissues.

It is interesting to note that the skin reaction occurred in only two of the four regions of irradiated skin, i.e. anteriorly where the skin doses were 18.7 Gy and 21.5 Gy over the spine and pelvic areas, respectively, but not posteriorly where the corresponding skin doses were 8.7 Gy and 16.8 Gy, respectively. The existence of four levels of skin doses suggests a 'threshold' radiation dose for the recall reaction of between 16.8 and 18.7 Gy in this case. It would be interesting to look for a similar 'threshold' dose from the data in other cases, but unfortunately, in most cases, only the tumour dose, rather than the skin dose, are reported.

To our knowledge, our patient represents the first case of radiation-recall dermatitis with docetaxel. In the few reported cases of recall reactions associated with taxanes, one case of radiation-recall dermatitis has been reported to be recurring with repeated administration of paclitaxel [6]; however, our patient treated with docetaxel and 2 patients reported by Shenkier and associates [7] who were treated with paclitaxel did not have signs of recurrent reactions with repeated therapy using the same drugs. This suggests that there may be a specific time period during which patients are susceptible to recall dermatitis. Alternatively, in our case, there may be a drug dose threshold since the two cycles in which the dermatitis did not occur were associated with a dose reduction.

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Acknowledgements—Docetaxel was provided by Rhône-Poulenc Rorer Limited as part of "A Phase II Study of Docetaxel in the Treatment of Patients with Advanced or Metastatic Breast Cancer". We thank Mr Peter Yu for his help in the analysis of the radiation dosages.

European Journal of Cancer Vol. 33, No. 4, pp. 699–700, 1997

Printed in Great Britain
0959-8049/97 $17.00 + 0.00

PII: S0959-8049(96)00490-X

Cathepsin D Content in Malignant Tumours of Corpus Uteri

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DUE TO degradation of the basement membrane and digestion of the extracellular matrix, lysosomal proteases may participate in invasion and metastasis of cancer. Of particular interest is cathepsin D, elevated levels of which have been correlated with poor prognosis of breast cancer [1, 2]. The enhanced expression of cathepsin D has also been reported for tumours of different origin [1]. The aim of the present study was to determine the concentration of cathepsin D in normal and malignant tissues of corpus uteri (30 matched pairs) and to assess the relationship between cathepsin D content and some clinical and pathohistological parameters.

Characteristics of the tumours and patients studied are given in Table 1. The classification of tumours and histo-

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Received 10 Sep. 1996; revised 31 Oct. 1996; accepted 22 Nov. 1996.

Table 1. Pathohistological and clinical characteristics of the malignant tumours of corpus uteri and patients

	Number of pairs
Total	30
Clinical stage	
I	25
II	3
III	2
Myometrial invasion	
<50%	15
>50%	14
Unknown	1
Tumour grade	
GI	17
GII	7
GIII	5
Unknown	1
Hystological type	
Carcinomas	
Type I (endometrioid, mucinous mixed endometrioid-mucinous)	24
Type II (serous, mixed endometrioid-serous, anaplastic)	5
Malignant muller mixed tumour	1
Lymphovascular invasion	
No	21
Yes	9
Age of patients (mean, range years)	60 (41–84)
<50	5
>50	25
Group	
Good (Type I, myometrial invasion <50%, grade I)	11
Poor (Type II, myometrial invasion >50%, grades II and III)	19

pathological grade was defined according to FIGO (1988). The samples representing matched pairs were obtained from the tumour and adjacent normal tissue. The cathepsin D content was measured in cytosol of homogenated samples [3], using a commercially available solid-phase two-site immunoradiometric assay (CIS Bio International, Gif-sur-Yvette, France). The total protein in cytosol was determined by Bradford's method [4]. The statistical significance of differences between concentrations of cathepsin D in matched pairs of normal and tumour tissues were calculated by Wilcoxon signed rank test. The differences were considered significant at $P < 0.05$.

We found that the concentrations of cathepsin D were significantly higher in tumours (35.0 ± 19.02 pmol/mg protein) than in normal tissue (11.87 ± 5.21 pmol/mg protein; $P < 0.001$) with a tumour to normal tissue ratio of 2.95. The concentrations of cathepsin D did not seem to relate to clinical stage (stage I versus stages II and III together), myometrial invasion, tumour grade, histological type of tumour, lymphovascular invasion, age of patients and the type of tumour according to its prognosis, although the sample sizes were too small for formal statistical analysis.

The literature concerning the role of cathepsin D in tumours of corpus uteri are scarce [5–8]. The significantly higher cathepsin D content determined in malignant endometrial tissues in our study confirm the previously published suggestions of its being of potential value as an endometrial tumour marker [5–8] as is with breast cancer [1, 2], head and neck cancer [9], ovarian [10] and cervical cancers [7]. Scambia and associates [7] reported an inverse correlation between cathepsin D levels and the stage and myometrial invasion in endometrial tumours, whereas Maudelonde and associates [5] and Nazeer and associates [6] found a positive correlation between high cytosolic levels of cathepsin D and depth of myometrial invasion. These conflicting results (like those obtained with breast cancer [2]) suggest that further studies are required to establish definitively the prognostic value of cathepsin D in endometrial tumours.

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