

DIE

RADIOIMMUNODETECTION (RID) OF HUMAN TUMOURS GROWING IN NUDE MICE  
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Nude mice (BALB/c, nu/nu) xenotransplanted with various human tumours (two types of adenocarcinoma of the rectum, the bladder carcinoma T24 cell line and the mammary carcinoma MDA-MB-231 cell line) were employed for RID studies. Mice were injected with the following  $^{131}\text{I}$  labelled antibodies and/or their F(ab')<sub>2</sub> fragments: rabbit Ab against human placental ferritin, monoclonal Ab 7E9 directed against the T24 cell line and monoclonal Ab HBCa 12 raised against the MDA-MB-231 cell line. Positive tumour images without blood background subtraction were gained within a 4-7 day interval after specific antibody administration. Some tumour, as well as antibody characteristics influence the success of tumour visualization.

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DIN

ULTRASTRUCTURE OF INVASION OF LUNG PARENCHYME BY SQUAMOUS CELL CARCINOMA

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We collected multiple samples from 50 resected bronchogenic squamous cell carcinomas to study ultrastructurally the invasion of lung parenchyme.

In the tumour periphery, where the initial contact between tumour and surrounding lung parenchyme takes place, the tumour cells were found to insert themselves into the epithelial compartment of the parenchyme without destroying the alveolar epithelial cells and without migrating through the alveolar basal lamina to reach the interstitial tissue compartment.

In the deeper tumour areas, an extensive stroma had developed, which fully deteriorated the original tissue architecture. However, an essentially intact basal lamina still surrounded most tumour cell groups, even when these were irregular and thin. Loss of basal lamina, which was found in part of the tumours studied, appeared to be a gradual process, and did not seem to greatly influence the behaviour of the tumour cells. Also in these deeper tumour areas, fully intact alveolar epithelial cells were present next to the tumour cells.

These results demonstrate that tumour invasion is a much more organized process than often assumed.

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DRO

ANTITUMOUR ACTIVITY OF MALONATO PLATINUM (II) COMPLEXES: MECHANISM OF ACTION

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Platinum complexes show antitumour activity provided they induce a specific lesion in the DNA which triggers a physiological, genetically determined response equivalent to SOS-functions in bacteria. Malonato platinum complexes (Pt-mal) react extremely slowly with DNA. In bacteria they fail to induce SOS-functions within a two hr experiment. However, Pt-mal complexes are active against tumours in a manner similar to cisplatin which reacts readily with DNA and induces SOS-functions. Therefore, we propose that Pt-mal complexes are changed into more reactive species in the biological milieu. The changes in the MEM cell culture medium indicate that no enzymes are involved.

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