# Course Papers

# Molecular Biology for Clinical Oncologists

## A postgraduate teaching course organised by John Yarnold and Simon Powell

## Academic Radiotherapy Unit, Royal Marsden Hospital, in association with the Institute of Cancer Research, London, U.K., 26–29 June 1991

The Academic Unit of Radiotherapy at the Royal Marsden Hospital in association with the Institute of Cancer Research organised a 4 day postgraduate teaching course entitled 'Molecular Biology for Clinical Oncologists' in London, 26–29 June 1991. The course concentrated on explaining recent advances in cancer research achieved through the applications of molecular techniques, particularly as they relate to the pathogenesis of human cancer and therapeutic responses to radiation and drugs. No prior knowledge of the field was assumed. Invited speakers were clinical scientists or scientists directly involved in laboratory programmes related to the topics they presented. The following articles are based on the course book that was provided.

The first two days explained how cancer genes are recognised and how they disrupt cell behaviour. Specific examples were chosen to illustrate multistage carcinogenesis in adult and paediatric cancers. The principles and applications of the polymerase chain reaction (PCR) to applied research were also discussed. The third day included presentations on the future of gene therapy. The last day focussed on how radiation and drugs induce damage in DNA, how damage is modified by repair mechanisms, and how residual DNA damage determines cell fate. A second teaching course on Molecular Biology for Clinical Oncologists is planned for 1–3 July, 1992. Further information can be obtained from J. R. Yarnold at the Royal Marsden Hospital.

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# Understanding DNA Damage and Repair: How Useful will it be Clinically?

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UNDERSTANDING OF DNA damage and repair is potentially useful clinically in two major ways; the prediction of response in the individual patient and the modification of response to cytotoxic therapy. We are currently unable to predict reliably which patients will respond to therapy. Tumour resistance remains a problem for both local and systemic therapies. Approaches to overcoming this include the development of new drugs within recognised groups with different binding specificities and the modification of repair by enzyme inhibition. The more effective use of radiation alone or in combination may be helped by the appreciation that certain types of damage are less repairable than others (as in high linear energy transfer radiations by external beam or incorporated radioisotopes), or how molecules such as thiols modify damage. Predictive assays of sensitivity to both radiation and cytotoxic therapy are currently being assessed based primarily on biological measures such as cell proliferation. Should these prove useful, direct measures of DNA damage may provide the same information more rapidly and reliably. The transition from the laboratory, to clinical practice, however, presents several problems particularly in the testing of new treatment strategies based on damage modification. The current philosophy of testing is based on randomised trials; large groups of patients in broad prognostic categories such that the small improvements currently considered realistic can be detected. It seems unlikely that any one of the modifications we have been considering will produce sufficiently large improvements across even a single tumour category to be detected in this way. Realistic goals for studies, therefore, must be identified, with appropriate patient selection for each

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