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CONFERENCE REPORT

The XIX Congress of the International Society for Analytical Cytology Colorado Springs, 27 February to 5 March 1998

David A. Rew

University of Leicester, U.K.

Introduction

The XIX Congress of the International Society for Analytical Cytology was held in Colorado Springs between 27 February and 5 March 1998. The Congress attracted some 1000 international delegates from many scientific and clinical disciplines. The Congress provided an opportunity to review advances in cell, molecular and tumour biology, and the frontier technologies of the analytical cell sciences relevant to cancer research. The structure of the Congress, with tutorials, frontier lectures, technical workshops and plenary sessions, has a strong educational theme.

Cell production and cell death

The control of tissue and tumour proliferation is now known to be effected primarily through the cell cycle phase related expression of the cyclin and cyclin-dependent kinase series of proteins, as shown cytometrically by F. Traganos and Z. Darzynkiewicz (New York Medical College). The controls of apoptosis, or programmed cell death, are also now well defined, with key roles for the proteins c-myc, bcl2 and p53 and for signalling mechanisms such as insulin-like growth factor (IGF-1) and the CD95 surface receptor (G. Evan, Imperial Cancer Research Fund (ICRF), London). Investigation of such key regulatory processes is revealing new modulatory targets for therapeutic research.

The CD95 (APO-1/Fas) receptor is now the likely common receptor for cell death-inducing signals. The caspases are another recently recognized group of proteolytic enzymes which mediate apoptotic cell death in response to these signals, as shown by a group from the Cancer Research Centre in Heidelberg. The molecular signals and determinants of apoptosis rather than the process itself are now the focus of research. J. Hickman (Cancer Research Centre, Manchester, U.K.) showed how changes in the Bak protein in response to drug treatment may be one of these early determinants of apoptosis.

The problem of control of cell numbers and cell form in normal tissues was addressed by Martin Raff (University College, London). Control of tissue and organ development and final size is a function of control of cell numbers. Normal cells which do not receive signals (growth factors) from adjacent cells undergo apoptosis in isolation in very large numbers during development, while specific genetic modifications of key proteins such as bcl2 and p27 produce abnormalities of size in otherwise normal mice. Thyroid hormone has a key regulatory role in this process.

Genes and cancer

Fluorescence *in situ* hybridization (FISH) is a well-established tool for measuring gene copies in cell and tissue samples using specific chromatic probes. M. Press (University of Southern California) showed in a series of 324 archival breast tumours how increased expression of the HER-2/neu gene which codes for an epidermal growth factor-like receptor might be an independent predictor of outcome. Genetic techniques are now being used to trace developmental or evolutionary pathways for individual tumours which manifest as p53 protein abnormalities and aneuploidy. F. Waldman (University College, San Francisco) and C. Cornilesse (University of Leiden, Holland) have found that the genotype of metastases often resembles that of early rather than late-stage invasive primary cancers.

Deliveries of gene therapy may best be achieved using tissue specific viral vectors. Key regulatory genes such as p53 suggest new targets for gene-directed therapy. F. McCormick (University College, San Francisco) reported success in a mouse model with an adenovirus in killing by lytic infection tumour cells which lack normal p53, while preserving normal cells with normal p53. This study has now moved to Phase I and II clinical trials.

Conventional cancer therapy

The complexity and interactivity of regulatory pathways and mechanisms provides considerable redundancy against

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