David Paul hansemann and the role of chromosomes in cancer

Chromosomes were described, named and identified as the basis of the cellular hereditary material in the 1870s-80s. Abnormal – including asymmetric – mitoses were discovered in tumour cells at almost the same time. In the same period Weismann’s theories of ‘plasmas’ and ‘differentiation by chromosomal loss’ were widely believed. Also, early studies of chromosomes had identified loss of chromosomes (as ‘polar bodies’) in oogenesis. In 1890, David Paul Hansemann (1858-1920), was an Assistant to Rudolf Virchow (1821-1902) in Berlin and noted asymmetric mitoses in cancer cells. Hansemann recognised the paramount features of tumours as loss of tissue differentiationi and increased capacity for independent existence (i.e. “autonomy” – ability to grow in remote tissues and form metastases). Probably because Virchow insisted that tumour formation must involve only an abnormality of a “physiological” tissue process (not a new process), Hansemann looked for a cell process which might be a counterpart of this particular combination of changes – i.e. a normal cell process in which changes in chromosomal content, reduction of differentiation and greater autonomy all occurred. Hansemann proposed that oogenesis was the “prototype process” because (i) the egg comes about by reduction divisions (ii) it is less differentiated than ovarian epithelial cells (in fact, it is ‘de-differentiated’) and (iii) the egg can survive for days free in the endometrial cavity. Hansemann called the normal process “anaplasia”, and proposed that it could occur to variable degrees in different cases of tumour according to the degrees of chromosome imbalance in the tumour cells.

Unfortunately for Hansemann’s theory, within ten years, Weismann’s ‘chromosome loss theory’ was found to be incorrect, and ejection of polar bodies was found to be a very specific phenomenon associated with the formation of (hapoid) ova. However, for practising pathologists, Hansemann’s ideas provided useful terminology. This was because previous classifications had divided tumours into only two types, for example “homologous vs heterologous” or “homeoplastic vs heteroplastic” without offering words to describe variations of abnormalities. Hansemann’s terminology provided a way of describing the continuous variation of abnormalities which tumours exhibit. Thus “anaplasia” and “(de)differentiation” became popular and have been used to describe tumours ever since, even though the original cell biological ideas on which they were founded are no longer believed.

In the early twentieth century, the directions of cancer research moved towards investigating Mendelian genetics in relation to tumours, and the mechanisms of action of viral, physical and chemical carcinogens. Only in the last 30 or so years, have the roles of chromosomal abnormalities in tumour formation – which were first considered in detail by Hansemann-again received significant attention.

Biography

Leon Bignold graduated in medicine from the University of Western Australia in 1971, completed a research doctorate 1978, qualified as a histopathologist in 1980 and has been working in academic and diagnostic pathology ever since. He has published over 70 scientific papers and edited vol 96 of EXS (Cancer: Cell Structures, Carcinogenesis and Genomic Instability, Birkhäuser, 2006). With colleagues, he has published a volume (Birkhäuser, 2007) on David Paul Hansemann (1858-1920), who was the first to suggest a chromosomal theory of cancer, and another on Rudolf Virchow (Birkhäuser, 2008). Dr Bignold is currently completing a volume on genomic models for complex clinical, pathological and therapeutic aspects of tumors.

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