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Letter to the Editor

David Paul Hansemann: Chromosomes and the origin of the cancerous features of tumor cells

Chromosomes were described, named and identified as the basis of the cellular hereditary material in the 1870–1880s. Abnormal – including asymmetric – mitoses were discovered in tumor cells at almost the same time. These various authors up to 1890, including Arnold (1879), Martin (1881), Pfitzner (1886), Cornil (1886), Klebs (1889) and Hauser (1890) (references with English summaries in [1]) did not consider that abnormal chromosomes or mitoses cause tumours, but are simply manifestations of the neoplastic state.

In 1890, David Paul Hansemann (1858–1920), was an *Assistent* to Virchow in Berlin and noted asymmetric mitoses in cancer cells. In relation to tumors in general, he recognised the paramount features of malignancies as loss of tissue specialisation and increased capacity for independent existence (i.e. 'autonomy' – ability to grow in remote tissues and form metastases). Probably because Virchow insisted that tumor formation must involve only an abnormality of a 'physiological' tissue process (i.e. 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 specialisation 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 specialised than ovarian epithelial cells, and (iii) the egg can survive for days free in the endometrial cavity. Hansemann called the process 'anaplasia' and in later works, he suggested that the basis of the cancer cell is loss of the ability to maintain symmetric mitoses, or at least loss of ability to preserve chromosomal integrity. He considered that populations of chromosomally unbalanced cells would arise, some of which have the (ovum-like) anaplastic features, but still have features of the cell type from which they arose. In response to reports that not all tumours exhibit asymmetrical mitoses or chromosomal abnormalities, Hansemann suggested that the chromosomal lesions might simply be too small to be visible.

Hansemann's ideas were criticized by many authors (summarized in [1]) but several authors (Hauser, 1903; Moore and Farmer, 1904; and Boveri, 1914; see [1]) produced theories of tumors involving alterations of cellular hereditary material. None of these was as comprehensive as Hansemann's, and Boveri's in particular was largely based on only one mitotic abnormality (quadripolarity) and one new cell biological observation (wandering of cells of the blastomere in doubly-fertilized – 'dispermic' – eggs) [1–3]. Also 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 studied by Hansemann – again received significant attention.

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