Parallel Symposium No. 3

Cytogenetics of Solid Tumours

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PS 3.1

CHROMOSOME CHANGES IN PEDIATRIC TUMORS

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There is evidence for a genetic component in the pathogenesis of some tumors in children, especially from studies on retinoblastoma and Wilms'tumor. Most, if not all, sporadic pediatric tumors have chromosome abnormalities, and these are specific for a particular type of tumor. Moreover, cytogenetic analysis helps in establishing a correct diagnosis as in the case of a group of tumors generically referred to as undifferentiated, small, round cell or small bluecell tumors in children.

PS 3.2

CYTOGENETICS IN THE DIAGNOSIS OF SOFT TUMORS

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The number of soft tissues characterized by a consistent specific chromosome abnormality is still small as is the number of cases chromosomally investigated of each subtype. However few examples have already proven the diagnostic value of cytogenetics in this type of tumors.
Moreover, it has been shown that the presence of a chromosomal abnormality is not an indicator of malignancy.

PS 3.3

Oncogenesis and tumor progression of testicular germ cell tumors. A cytogenetic model. - Sérgio CASTEDO - Dept. of Medical Genetics, Medical Faculty of Porto, Hospital S. João, 4200 PORTO, PORTUGAL.

Testicular germ cell tumors (TGCT) of adults can be divided clinically and morphologically in two main entities: seminomas and nonseminomatous germ cell tumors (nonseminomas). Essentially there are two therories about the pathogenetic relationship between seminomas and nonseminomas: one favoring independent origins of these two entities via carcinoma in situ (CIS); the other suggesting a single origin for these tumors with seminoma as a stage after CIS through which all TGCT progress.
The cytogenetic studies of TGCT describe generally hyperdiploid to hypertriploid chromosome complements with higher modal chromosome numbers in seminomas as compared to nonseminomas.
Regarding the origination of aneuploidy in TGCT, the cytogenetic reports published so far suggest that polyploidization or cell fusion are possible mechanisms. The demonstration of an isochromosome for the short arm of chromosome 12 - i(12p) - in nearly 80% of all TGCT suggests that the i(12p) marker plays an important role in the oncogenesis of this group of tumors, irrespective of the histological subtype. However, molecular studies carried out on fresh tumors and on TGCT-derived cell lines have recently indicated that the formation of an i(12p) probably occurs after aneuploidization.
The comparison of the chromosomal patterns of CIS, seminomas, and non-seminomas suggests that they share a common origin and that chromosomal loss is probably a crucial event during tumor progression of TGCT.

PS 3.4

The Promise of Molecular Studies in Human Cancer

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The recent successes in elucidating the molecular bases of some human pathologies have demonstrated the power of molecular genetics integrating both cytogenetics and molecular biology. The developments of molecular oncology have led to the recognition of oncogene activation as a possible factor in human oncogenesis. Consistent with this idea is the fact that several proto-oncogenes are located near chromosomal breakpoints involved in translocations specific of certain malignancies. One possibility is that translocations and other chromosome abnormalities affect the expression of oncogenes contributing either in the initiation or in the progression of the tumoral process.

The most common cytogenetic and molecular markers present in several solid tumors correspond to losses of genetic information (deletions, isochromosomes, losses of heterozygosity). Therefore, it is conceivable that anti-oncogenes (or tumor suppressor genes) represent primary targets of carcinogenesis. The search for chromosomes losses has already led to the mapping of loci corresponding to predisposition for inherited cancer syndromes.

Molecular genetics has already been implicated in clinical oncology by helping to recognize tumors subtypes. One of the challenges for the future is its involvement in prognosis.