DELETION MUTANTS IN THE TRANSFORMING GENE OF THE HARVEY SARCOMA VIRUS.
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In order to study the functional domains of the Harvey Sarcoma Virus transforming protein, deletion mutants were constructed. The transforming gene of Harvey Sarcoma virus was subcloned from a molecular clone in a form which was active in the NIH-cell-transforming assay. Deletions extending into the coding portion of the gene from the 3' end were constructed with exonuclease Bal31 and an oligonucleotide linker containing the recognition sequence for the restriction enzyme BclI was added. This sequence contains the stop coden TGA.

The deletions were sequenced and those where the TGA of the BclI-linker was in frame with the transforming protein were selected. A clone containing the non-coding 3' portion of the gene was constructed, and the deletions reconstituted. Preliminary results suggest that deletion of as little as eight amino-acids into the carboxy end of the protein results in a non-transforming protein.

LOCALIZATION OF THE H-2 COMPLEX IN THE DISTAL PART OF CHROMOSOME 17 IN THE T19O TRANSLOCATION IN MICE. Iwona Włodarska, Alina Czarnomska and Kazimierz Dux, Department of Tumour Biology, Institute of Oncology, Warsaw, Poland.

It is not clear so far, which of two marker chromosomes in the reciprocal translocation T/1;17/19OCa carries the H-2 complex located on chromosome 17.

In our experimental system five single cell clones have been isolated from the hyperdiploid ascites leukaemia induced by X-irradiation in heterozygous T19O/tf mice. In one of these clones the long marker chromosome was absent. The cells did not express the H-2^{tW1} specificities characteristic for T19O and have been not rejected after intraperitoneal inoculation into tf/tf mice preimmunized by skin T19O/tf allografts. In four other clones with both marker chromosomes, the H-2^{tW1} specificities were expressed.

We conclude that the H-2 complex responsible for the expression of tw1 specificities is located on the long marker chromosome containing the distal part of chromosome 17.

THE USE OF SISTER CHROMATID EXCHANGE (SCE) TO DETECT EXPOSURE TO GENOTOXIC EXPOSURE.
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SCE (sister chromatid exchange) may be examined in lymphocytes taken from persons exposed to chemicals in vivo. In this case the cells have not only been exposed to the chemical, but also to the degradation products.

Children have lower SCE than adults but the SCE count seems rather stable after 12 to 15 years of age.

SCE has been found to be higher in smokers than in non-smokers. This has been shown both in cigarette, pipe and cheroot smokers.

Anticontraceptive drugs do not enhance SCE.

The effect of industrialized food is under investigation, the preliminary findings giving no suspicion of SCE inducing properties in this kind of food. Neither anaesthetic personnel nor patients subjected to anaesthesia with halothane, fluroxene, enflurane and isoflurane have shown enhanced SCE in their lymphocytes.

Workers exposed to inorganic lead showed a SCE level which increased with increased zinc protoporphyrin in the blood.