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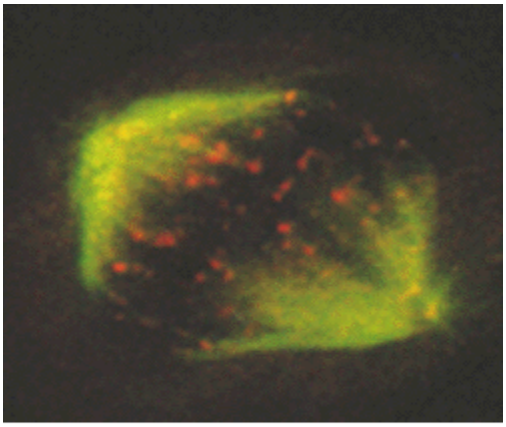
Cancers



An immortal HeLa cell, originally derived from a human tumor, undergoing cell division. It is stained with anti-tubulin (green) and anti-CENP-E (red) antibodies. CENP-E, a kinesin, is associated with the centromeres of paired sister chromatids during metaphase. [Photograph by Tim Yen and colleagues, Fox Chase Cancer Center, PA, USA. Reproduced from Endow, SE (1993) Trends Genet. 9, 52-55, with permission.]

Cancer occurs when cell division gets out of control. Usually, the timing of cell division is under strict constraint, involving a network of signals that work together to say when a cell can divide, how often it should happen and how errors can be fixed. Mutations in one or more of the nodes in this network can trigger cancer, be it through exposure to some environmental factor (e.g. tobacco smoke) or because of a genetic predisposition, or both. Usually, several cancer-promoting factors have to add up before a person will develop a malignant growth: with some exceptions, no one risk alone is sufficient.

The predominant mechanisms for the cancers featured here are (i) impairment of a DNA repair pathway (ii) the transformation of a normal gene into an oncogene and (iii) the malfunction of a tumor supressor gene.

Diseases

Lung carcinoma, small cell

Malignant melanoma

Multiple endocrine neoplasia

Neurofibromatosis

The p53 tumor suppressor protein

Pancreatic cancer

Polycystic kidney disease

Prostate cancer

Harvey Ras oncogene

Retinoblastoma

Tuberous sclerosis

Von Hippel-Lindau syndrome