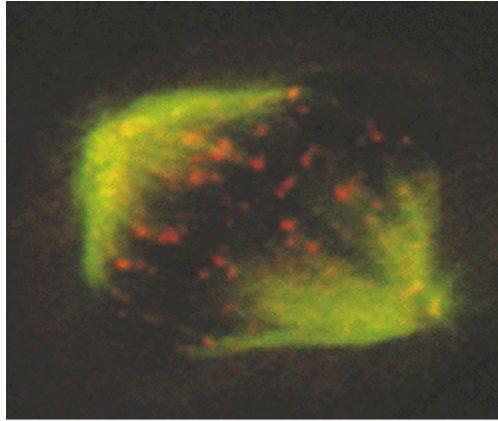


Cancers



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 suppressor gene.

Diseases

Breast and ovarian cancer

Burkitt lymphoma

Colon cancer
Leukemia, chronic myeloid
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

Did you know ...?

Is **cancer** the leading cause of death in Americans? Or is it heart disease, stroke, or accidental injuries?