that, as it was believed, could direct the transformation of a normal cell into a tumor cell. The favorite theory of the time was that virus-mediated cell-to-cell transmittance of oncogenes was the origin of all forms of cancer. This view was later proven to be incorrect because, from the standpoint of cancer development, the crucial issue is the comparison between oncogenes in normal cells (proto-oncogenes) and in tumor cells. Oncogenes are identified by the use of three-letter abbreviations; in addition, cellular and viral oncogenes are sometimes distinguished by *c-* and *v-* prefixes, respectively (e.g., *c-src* and *v-src*).

The first oncogene to be identified was *v-src*, discovered in 1970 as a component of a cancer-causing virus in chickens known as the Rous sarcoma virus. This is a member of the retroviruses family, characterized because their RNA genetic material is transcribed into DNA by the enzyme reverse transcriptase. This reverse transcription permits the integration of the genetic material of retroviruses into the chromosomal DNA in the cells. However, retroviruses play a relatively limited role in the development of cancer under natural conditions, with human T-lymphotropic virus type 1 (HTLV-1) the only known example in humans in which a retrovirus infection contributes to the origin of a cancer. Other kinds of viruses with DNA as their genetic material can also contribute to the development of tumors in humans, although other factors in addition to the virus infection are required for the cancer to develop. Certain types of papillomaviruses play a role in the development of cervical cancer in the genital tract, whereas Epstein–Barr virus is an important factor in the development of Burkitt's lymphoma in Africa and nasopharyngeal cancer in Asia.

In 1975, Bishop and Varmus demonstrated the true origin of the *v-src* oncogene by its detection in different species throughout the animal kingdom. It controls cell growth and division, and in humans it is involved in a variety of cancers, such as colon, liver, lung, breast, and pancreatic cancer.[8] Accordingly, this oncogene is not a true viral gene but, rather, a cellular gene picked up by the virus during its replication in cells.

In 1982, the first human oncogene, currently known as *H-RAS*, was cloned and characterized from the T24 bladder carcinoma.[9] Approaches to the true genetic complexity of cancer evolved as a result of the Human Genome Project (1988–2003), which led to the knowledge that among a total of approximately 25,000 human genes, mutations of approximately 200 are able to promote abnormal growth and cell division as well as evasion of programmed death, leading to cancer. Nevertheless, the regulation of growth and division of cells is much more complex than originally believed. Cellular oncogene products with different properties act in different positions in elaborate signal systems to transmit signals from one cell to another or within a single cell.

Several oncogene products function as receptors in the cytoplasmic membrane of the cells and catalyze the phosphorylation of the amino acid tyrosine. There are two groups of oncogene products with phosphokinase activity: tyrosine/phosphokinases, which lack receptor function and are located on the inside of the cytoplasmic membrane, and serine/threonine phosphokinases, which are found in the cytoplasm. Thus, oncogene products function as links in signal chains stretching from the surface of the cell to the genetic material in the nucleus. In the cytoplasm, there is one more group of oncogene products, such as Ras, that are related to the important cellular signal factors known as G proteins. Finally, several oncogene products, such as Myc, Myb, Fos, and ErbA, are located in the nucleus of the cell and direct the transcription of DNA into RNA, playing a critical role in the selection of proteins to be synthesized by the cell.

In the development of a tumor, a normal cellular oncogene may be hyperactive or an oncogene product may be altered so that it can no longer be regulated in a normal way. Oncogenes with point