Here are some important genes (c stands for c ellular [as opposed to v iral genes]): (I present these because the names, more often than not, are **opaque**. I mean to shed a bit of light but not to have you memorize the names. If this list is useful, great; if not, skip on past! The complaint I have is that this is a story with characters but no character development; [thus, the story fails].)

c-*fos*
c-*myc* Transcription factors;
c-*jun* products of cellular genes;
c-*myb* oncogenes...
c-*ets* All have viral counterparts...
c-*rel*

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src
ras

MAPK Mitogen activated protein kinase
MEK Map kinase kinase
JNK Jun kinase
JAK Janus kinase (two kinases at either end of molecule) (aka just another kinase)
ERK Extracellular regulated kinase
ZAP-70 Zeta-activating protein
Lck Lymphocyte kinase
Fyn Fybroblast endothelial kinase
(ELK) (Don t be mislead by the K. Here the initials abbreviate a transcription factor.)

And here are some prominent and well-studied oncogenes:

src An ONCOGENE originally identified as the transforming determinant of ROUS SARCOMA VIRUS. The *v-src* product ($pp60^{v\text{-}src}$) has tyrosine-specific kinase activity. The *c-src* product also has tyrosine kinase activity, but *c-src* is expressed at very low levels in most normal cells; cells transformed by RSV usually contain relatively high levels of $pp60^{v\text{-}src}$. *The* c-src *and v-src* products are similar but not identical, differing in their C-terminal amino acid sequences; this difference in structure may be at least partly responsible for the tranforming capacity of $pp60^{v\text{-}src}$.

ras Designation for a family of ONCOGENES first discovered in HARVEY MURINE SARCOMA VIRUS (H-*ras* or Ha-*ras*) and KIRSTEN MURINE SARCOMA VIRUS (K-*ras* or Ki-*ras*); cellular *ras* genes have been highly conserved during evolution and occur, *e. g.,* in humans, rodents and *Saccharomyces cerevisiae*. The *ras* gene codes for highly related proteins (generic designation: p21) containing 189 amino acid residues; p21 proteins have GTP-binding, GTP hydrolysing, and autophosphorylating activities. A *ras* oncogene can be activated, *e. g.*, by

chemical carcinogens; transforming *ras* genes can differ from normal cellular *ras* genes by a single point mutation which results in a single amino acid substitution involving the 12^{th} or 61^{st} amino acids in p21.

fos An ONCOGENE from FBJ (FINKEL-BISKIS-JINKINS murine sarcoma virus: a retrovirus complex consisting of a replication-competent murine leukemia virus [FBJ-MuLV] and a replication defective transforming murine sarcoma virus [FBJ-MSV].) FBJ-MSV carries the oncogene v-*fos* and induces osteosarcomas in mice after a latent period of ca. 3 weeks. The v-*fos* product does not have tyrosine case activity. Homologues of c-fos have been identified in the genomes of various vertebrates as well as in *Drosophila*.

jun A proto ONCOGENE...that encodes part of the AP-1 transcription factor...

myc An ONCOGENE originally identified as the transforming determinant of avian myelocytomatosis virus (MC29). The MC29 v-*myc* product is a *gag-myc* fusion protein ($P110^{gag\text{-}myc}$) which has no protein kinase activity; it binds to dsDNA and occurs -- possibly as a chromatin component -- in the nucleus. In humans, c-*myc* is located on chromosome 8 and is involved in the pathogenesis of Burkitt's lymphoma. In chickens, c-*myc* activation by avian leukosis virus appears to result in the development of lymphoid leukosis.

myb An ONCOGENE originally identified as the transforming determinant in avian myeloblastosis virus v-*myb* is an altered form of cellular sequence *amv* differing from *amb* in gene structure, transcript structure, gene product structure, and in the intracellular location (nucleus) of its product. V-myb^{+} avian acute leukemia virus (AMV) can transform chicken hematopoietic cells in culture, but differs from other acutely transforming retroviruses in that it does not transform fibroblasts in culture; it causes a rapidly fatal leukemia only in chickens.