An increasing number of polypeptide growth factors have been identified that regulate not only cell proliferation but an extraordinary range of cell activities, including matrix protein deposition and resolution, the maintenance of cell viability, cell differentiation, inflammation, and tissue repair. Normal cells appear to require growth factors for proliferation and for maintenance of viability. Cells that secrete a polypeptide growth factor have an advantage in growth. These factors can act either externally through cell surface receptors or perhaps internally during the transport of receptors and growth factors through the ER and Golgi, causing autocrine stimulation of cell growth. Depending on the cell type, growth factors can also be potent inhibitors of cell growth rather than stimulating growth, and the effects can depend on the presence or absence of other growth factors. Platelet-derived growth factor has been shown to be nearly identical to the product of the v-sis gene of the simian sarcoma virus, which appears to cause cell transformation through its interactions with the PDGF receptor activating the tyrosine kinase activity of the PDGF receptor. Similarly, two proto-oncogenes, c-erbB and c-fms, encode growth factor receptors. The EGF receptor activity of the v-erb oncogene product appears to be constitutively activated without the need for growth factor, perhaps because of the truncation at the amino terminus deleting the EGF binding domain. The induction of the myc and the fos proteins by growth factor stimulation of quiescent cells, as well as the potential for the p21 product of the ras oncogene to act as an intermediate in transducing adrenergic signals, provide direct evidence that these pathways are important for stimulation of cell growth. Cells transformed by the v-sis oncogene always appear to bear PDGF cell surface receptors, which suggests that this oncogene has a specific requirement of the PDGF receptor for transformation. In contrast, cells transformed by the v-erbB and v-fms oncogenes are not stimulated by EGF or by CSF-1. Thus it seems likely that the tyrosine kinase activity of the corresponding receptor is ubiquitously expressed in these cases. Major questions remain unanswered. In particular, what are the mechanisms by which growth factors initiate pathways leading to DNA synthesis? What are the physiological substrates of the growth factor receptor tyrosine kinase? Considerable effort also is needed to further define the cellular specificity of the different growth factors, particularly within intact tissues, and to determine how the various growth factors interact [1)]

1)
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