



Receptor Tyrosine Kinases Mediated Cell Signalling Pathways

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A small number of signalling pathways, including receptor tyrosine kinases (RTKs) and G protein-coupled receptors (GPCRs), are activated by plasma membrane receptors in order for cells to respond to external cues. In addition to transmitting, these pathways also process, encode, and integrate both internal and external signals. In recent years, it has become clear that different spatio-temporal activation profiles of the same set of signalling proteins lead to different gene-expression patterns and various physiological responses. These findings suggest that the precise temporal control and relative spatial distribution of activated signal transducers are necessary for important cellular decisions such as cytoskeletal reorganisation, cell-cycle checkpoints, and cell death (apoptosis). Due to their crucial function in the control of embryogenesis, cell survival, motility, proliferation, differentiation, glucose metabolism, and apoptosis, RTK-mediated signalling pathways have drawn a lot of attention from scientists.

A significant number of serious human diseases, including diabetes, cancer, chronic inflammatory syndromes, and developmental defects, are caused by RTK signalling dysfunction. RTKs undergo allosteric transitions (the insulin receptor, for instance) or dimerization in response to stimulation, which activates the intrinsic tyrosine-kinase activity. Numerous cytoplasmic proteins receive a biochemical signal from subsequent phosphorylation of numerous tyrosine residues on the receptor, which causes them to move to the cell surface. Through intricate biochemical circuits of protein-protein interactions and covalent modification cascades, the resulting cellular reactions take place.

The earlier theories of discrete linear pathways, which connected extracellular signals to the expression of particular genes, have been replaced by an emerging picture of interconnected signalling networks, raising concerns about the specificity of signal-response events. In actuality, all RTK-mediated pathways share a common protein complement that mediates signal transduction downstream of RTKs. The expression of nuclear transcription factors is induced by the activation of kinase and phosphatase cascades, such as the mitogen-activated protein kinase (MAPK) cascades, which are both activated by GPCRs and RTKs. There is no single protein or gene that determines the specificity of a signal for any given receptor pathway.

Instead, the temporal and spatial dynamics of the components of downstream signalling control specificity. A classic example is the distinctive biological response to EGF and nerve growth factor stimulation of PC12 cell lines (NGF). Cell proliferation is temporarily induced by EGF, whereas cell differentiation is temporarily triggered by sustained MAPK activation by NGF. However, there are many variables that affect how MAPK cascades behave. Depending heavily on their subcellular localization and their recruitment to scaffold proteins, MAPK cascades can produce oscillations, abrupt switches, bistable dynamics.