

Role of Cellular Receptors in Cell Signaling

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EDITORIAL NOTE

Receptors are a type of proteins that function by interacting with a certain ligand molecule and causes immune responses. Immune cells such as monocytes, T cells, B cells, NK cells, and stem cells contain receptors. A ligand is a molecule that binds to a receptor and can be a peptide or other molecules such a neurotransmitter, pharmaceutical medicine, hormone, toxin, or parts of a virus or microbes exterior [1]. When a ligand binds to a receptor, it activates or inhibits the biochemical pathway associated with that receptor.

Receptors can control membrane channels, regulate cell binding, and stimulate cell growth, division, and cell death. Signal transduction, immunotherapy, and immunological responses all rely on receptors [2].

Intracellular receptors

Internal receptors, sometimes called intracellular or cytoplasmic receptors, are located in the cell cytoplasm and respond to hydrophobic ligand molecules which travel across the plasma membrane. Gene expression is the physiological process through which information in cell DNA is transformed into a sequence of amino acids, which leads to formation of proteins. A conformational change occurs when the ligand binds to the internal receptor, by viewing a DNA-binding site on the protein [3]. The ligand-receptor combination enters the nucleus, binds to certain regulatory regions of chromosomal DNA, and encourages transcription to begin. Internal receptors have the ability to directly alter gene expression.

Cell-surface receptors

Transmembrane receptors are also known as cell-surface receptors. They are the membrane-anchored or integral proteins on the cell surface that bind to external ligand molecules. This receptor crosses the plasma membrane and performs signal transduction, transforming extracellular signals into intracellular signals. Ligands interacting with cell-surface receptors will not be penetrated with the cell they are interacting with as they are particular to individual cell types. Cell-surface receptors are also known as cell-specific proteins or markers [4]. A cell-surface

receptor is composed of an extracellular ligand-binding domain (extracellular domain), a hydrophobic membrane-spanning area, and an intracellular domain. Depending on the type of receptor, the size and extent, these domains varies.

Mostly, in multicellular organisms, cell-surface receptors are involved in signaling. Ion channel-linked receptors, G-protein-linked receptors, and enzyme-linked receptors are the three types of cell-surface receptors [5].

Ion channel-linked receptors: Ion channel-linked receptors, also known as ionotropic receptors, regulate chemical signal transduction across the cell membrane in response to the chemical messenger (e.g., neurotransmitter) binding. When a ligand binds to ion-channel linked receptors, the extracellular domain of the receptor undergoes certain changes and allows a channel to open across the plasma membrane [6]. Specific ions (such as Na^+ , Ca^{2+} , H^+ and Mg^{2+}) and other essential molecules can pass through the open channel.

Neurotransmitters and peptide hormones are ligands that bind to ion channel-linked receptors, and the passing molecules are ions like sodium (Na^+) and potassium (K^+).

G-protein linked receptors: The largest cell surface receptors are G-protein-coupled receptors (GPCRs), which are made up of seven transmembrane proteins in the plasma membrane. GPCRs are responsible for activating membrane-bound trimeric G-proteins (GTP binding proteins), which then activate an ion channel (effector) or an enzyme in the cell membrane [7]. The acetylcholine (Ach) receptor, adrenergic receptor, metabotropic glutamate receptors, some olfactory receptors, peptide hormone receptors, and rhodopsin are all examples of G-protein-coupled receptors (a photosensitive receptor). G-protein-linked receptors contain seven transmembrane domains, but each has its own extracellular domain and G-protein-binding site.

When the ligand binds to the receptor, activation and release of Guanosine Di Phosphate (GDP) occurs. In the cell membrane, the activated G-protein activates either an ion channel (effector) or an enzyme. The G-protein subunits are then separated into α subunit and β subunit. As a result, one or both of these G-protein fragments might be able to activate additional proteins [8]. The GTP on the active G-protein α subunit is hydrolyzed to

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GDP and the β subunit is deactivated. The cycle begins again when the subunits re-associate to form the inactive G-protein.

Enzyme-linked receptors: An enzyme-linked receptor, also known as a catalytic receptor, is a transmembrane receptor that leads to the intracellular enzymatic activity when an external ligand binds to it. As a result, an enzyme-linked receptor is a membrane protein that contains enzymatic, catalytic, and receptor functions. They have a single transmembrane helix and two important domains, namely an extracellular ligand binding domain and an intracellular domain, which has catalytic function. The signaling molecule binds to the receptor on the outside of the cell, causing the change in the catalytic function on the receptor inside the cell [9].

The extracellular and intracellular domains of enzyme-linked receptors are generally extensive, but the membrane-spanning portion is only a single alpha-helical peptide strand. The tyrosine kinase receptor is an example of an enzyme-linked receptor [10].

REFERENCES

1. Jones B. The therapeutic potential of GLP-1 receptor biased agonism. *Br J Pharmacol.* 2021;4: 58-65.
2. Dragic T, Litwin V, Allaway GP, Martin SR, Huang Y, Nagashima KA, et al. HIV-1 entry into CD4+ cells is mediated by the chemokine receptor CC-CKR-5. *Nature.* 1996;381: 667-73.
3. Kim MB, Giesler KE, Tahirovic YA, Truax VM, Liotta DC, Wilson LJ. CCR5 receptor antagonists in preclinical to phase II clinical development for treatment of HIV. *Expert Opin Investig Drugs.* 2016;25: 1377-1392.
4. Stencel-Baerenwald JE, Reiss K, Reiter DM, Stehle T, Dermody TS. The sweet spot: defining virus-sialic acid interactions. *Nat Rev Microbiol.* 2014;12: 739-49.
5. Lee S, Mannstadt M, Guo J, Kim SM, Yi HS, Khatri A, et al. A Homozygous [Cys25]PTH(1-84) Mutation That Impairs PTH/PTHrP Receptor Activation Defines a Novel Form of Hypoparathyroidism. *J Bone Miner Res.* 2015;30: 1803-1813.
6. Weinstein LS, Shenker A, Gejman PV, Merino MJ, Friedman E, Spiegel AM. Activating mutations of the stimulatory G protein in the McCune-Albright syndrome. *N Engl J Med.* 1991;325: 1688-1695.
7. Huang K, Luo YB, Yang H. Autoimmune Channelopathies at Neuromuscular Junction. *Front Neurol.* 2019;10: 516-517.
8. McPhaul MJ, Marcelli M, Zoppi S, Griffin JE, Wilson JD. Genetic basis of endocrine disease. 4. The spectrum of mutations in the androgen receptor gene that causes androgen resistance. *J Clin Endocrinol Metab.* 1993;76: 17-23.
9. Shiang R, Thompson LM, Zhu YZ, Church DM, Fielder TJ, Bocian M, et al. Mutations in the transmembrane domain of FGFR3 cause the most common genetic form of dwarfism, achondroplasia. *Cell.* 1994;78: 335-342.
10. Sahni M, Ambrosetti DC, Mansukhani A, Gertner R, Levy D, Basilico C. FGF signaling inhibits chondrocyte proliferation and regulates bone development through the STAT-1 pathway. *Genes Dev.* 1999;13: 1361-1366.