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Short Communication

Inhibition of RTKs Pathway in Cancer Cell Treatment

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DESCRIPTION

The phosphorylation of kinases and other signaling cascades is caused by the interaction between the receptor and ligand. Tyrosine Kinase Inhibitors (TKIs) either block the kinase domain of RTKs or the interaction between the receptor and the ligand, as in the case of trastuzumab in breast cancer. Tyrosine kinase domain inhibitors prevent the Receptor Tyrosine Kinases (RTKs) from being activated by competitively inhibiting the ATP binding site. The domain of Tyrosine Kinases Inhibitors (TKIs) maybe natural or synthetic compounds [1].

Inhibition of vascular endothelial growth factor

Vascular Endothelial Growth Factor (VEGF) is recognized to mediate angiogenesis, a crucial element in the development, progression, and multiplication of neoplastic cells. Therefore, blocking the growth factor has significant benefits for the treatment of cancer [2]. Angiogenesis, a tightly controlled process for neoplastic cells to generate blood vessels, promotes the survival of cancer cells. The development of monoclonal antibodies and/or small compounds that can inhibit the phosphorylation of the tyrosine kinase domain are two ways that the angiogenic growth factor can be inhibited.

Inhibition of epidermal growth factor

Epidermal Growth Factor (EGF) is necessary for the growth, development, angiogenesis, and metastasis of tumors. As a result, it is a promising target for the therapy of cancer because its overexpression is linked to the emergence of cancer [3]. Erlotinib and gefitinib are two examples of small-molecule EGF inhibitors that have shown to be very effective in the treatment of cancer. The development of monoclonal antibodies, such as Cetuximab, which block the extracellular receptor domain of RTKs, has also been crucial in the fight against cancer.

Inhibition of insulin growth factor

The Insulin Growth Factor (IGF) receptor plays a significant role in the development of tumors. In order to reduce the threat of cancer, it is necessary to limit the activation of this growth factor. Small-molecule inhibitors of the ATP binding domain or blocking substrate binding to the insulin growth factor receptor have been used to inhibit IGF, hence preventing the activation of tyrosine kinases important in the development of cancer [4].

Inhibition of fibroblast growth factor

Fibroblast Growth Factor (FGF) has crucial functions in migration, apoptosis, and proliferation. FGF changes have been linked to cancer. Since FGF is linked to carcinogenesis and the formation of cancer, inhibiting this growth factor has shown to stop the spread of cancer. Inhibitors of small-molecule kinases can stop RTK signaling. According to studies, they exhibit broad specificity for VEGF, FGF, and other RTKs like AZD4547. Monoclonal antibodies have also been used to suppress the Fibroblast Growth Factor Receptor (FGFR).

CONCLUSION

RTKs are essential for the growth and development of neoplasms. An in-depth understanding of the conformation and mechanisms of RTK inhibition is necessary for the formulation of novel and more effective TKIs. It is important that various treatment regimens that can reduce toxicity, eliminate recurrence caused by cancer cells that are in a dormant state, and improve a cancer-free state.

When the RTK is in the resting state, or when ligands are not interacting with it, basic enzymatic processes are thought to be ongoing. Even in the absence of ligand interaction, it is thought that the resting state oscillates between dynamic modes. A soluble tyrosine kinase still phosphorylates the Tyrosine Kinase Domain (TKD) in the resting state of RTK, such as c-Src, which activates signaling. The juxtamembrane domain and the C-terminal tail engage with the TKD in a way that prevents activation, but residual kinase activity may still occur.

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