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Tyrosine kinases in cancer

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Tyrosine kinases (TKs) play an important role in human neoplastic diseases. TKs are a class of receptors sharing protein structures akin to each other, comprising an extracellular domain (L25-S645), transmembrane helix (I646-M668) and a cytoplasmic domain (R669-A1210). There are 58 known TK receptors, out of which a subfamily of four receptors is the ErbB (erythroblastic leukaemia viral oncogene symbol) or HER (human epidermal growth factor receptor) group of receptors. Members of this subfamily include epidermal growth factor receptors (EGFRs), such as ErbB1/HER1, ErbB2/HER2, ErbB3/HER3 and ErbB4/HER4. Dimerisation of a receptor with the same type of ErbB receptor or a different type within the same family happens on the binding of ligand leading to activation of the receptor. The symbol ErbB is derived from the discovery of the first ErbB receptor, EGFR, when it was found that the avian erythroblastosis tumour virus has a mutant homologue of EGFR. This group of receptors determines key roles in tissue and organ development. But overexpression of each of these receptors is known to play a direct role in the pathogenesis of cancer. EGFR is one of the well-studied receptors among the ErbB family. Mutations and over-expression of this receptor lead to the development of a variety of solid tumours including non-small-cell lung carcinoma (NSCLC), head and neck squamous cell carcinoma (HNSCC), glioblastomas, etc. The over-expression of EGFR is the driver mechanism in lung cancer, breast cancer, pancreatic cancer, etc. It can thus be targeted in treating solid tumours with high throughput cancer genome-based molecular therapeutics.

Keywords: EGFR, Tumours, Cancer, Tyrosine kinases, Solid tumours, Human neoplastic diseases, ErbB, HER

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