Anti receptor kinase (suffix ~nib)

“At present, 58 receptor tyrosine kinases (RTKs) are known, grouped into 20 subfamilies.

They play pivotal roles in diverse cellular activities including growth, differentiation, metabolism, adhesion, motility and death.

RTKs are composed of an extracellular domain, which is able to bind a specific ligand, a transmembrane domain, and an intracellular catalytic domain, which is able to bind and phosphorylate selected substrates.

Binding of a ligand to the extracellular region causes a series of structural rearrangements in the RTK that lead to its enzymatic activation.

In particular, movement of some parts of the kinase domain gives free access to adenosine triphosphate (ATP) and the substrate to the active site.

This triggers a cascade of events through phosphorylation of intracellular proteins that ultimately transmit (“transduce”) the extracellular signal to the nucleus, causing changes in gene expression.

Many RTKs are involved in oncogenesis, either by gene mutation, or chromosome translocation, or simply by over-expression.

In every case, the result is a hyper-active kinase, that confers an aberrant, ligand-independent, non-regulated growth stimulus to the cancer cells.”(16)

From these 20 subfamilies of Receptor Tyrosine Kinases (RTK), seven families are promising field of investigation and only two families of RTK represent now the most promised field of drug development in AMD; fibroblast growth factor receptor (FGFR) family and vascular endothelial growth factor receptor (VEGFR) family.

RTK Class I Epidermal growth factor receptor family, RTK Class II Insulin receptor family RTK Class III Platelet-derived growth factor receptor RTK Class IV Fibroblast growth factor receptor (FGFR) family, RTK Class V Vascular endothelial growth factor receptor (VEGFR) family, RTK Class XII RET receptor family (RET proto-oncogene) RTK Class VIII Eph receptor family.

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