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Cell signaling Molecules: An approach towards Drug delivery to several diseases

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ABSTRACT:

Review of the paper suggest that Many diseases result from aberration in signaling pathways. These disease include cancer, atherosclerosis ,some inflammation and tissue rejection. This problem focused on designing of essential drugs which intercepts cell signaling like Tyrosine kinase blockers, signal interceptors like protein kinase c blockers, Ras blockers, ca2 signaling inhibitors which inhibits growth of cancers cell in vivo and in vitro.

The signaling systems made up of receptor sites to which one or more ligand binds to trigger a cellular signal. We are trying to manipulate cellular process by designing ligands mimicking molecules such as therapeutic drug molecules to trigger or suppress specific cells activities as desired.

Introduction:

Cancer is driven by genetic and epigenetic alterations that allow cells to overproliferate and escape mechanisms that normally control their survival and migration. Many of these alterations map to signaling pathways that control cell growth and division, cell death, cell fate, and cell motility, and can be placed in the context of distortions of wider signaling networks that fuel cancer progression, such as changes in the tumor microenvironment, angiogenesis, and inflammation. Mutations that convert cellular proto-oncogenes to oncogenes can cause hyperactivation of these signaling pathways, whereas inactivation of tumor suppressors eliminates critical negative regulators of signaling. An examination of the PI3K-Akt and Ras-ERK pathways illustrates how such alterations dysregulate signaling in cancer and produce many of the characteristic features of tumor cells.

Oncogenic mutations can cause the affected genes to be overexpressed (e.g., gene amplification) or produce mutated proteins whose activity is dysregulated (e.g., point mutations, truncations, and fusions). Examples include proteins involved in signaling pathways that are commonly activated in many physiological responses, such as growth factor receptor tyrosine kinases (RTKs; e.g., the epidermal growth factor receptor, EGFR), small GTPases (e.g., Ras), serine/threonine kinases (e.g., Raf and Akt), cytoplasmic tyrosine kinases (e.g., Src and Abl), lipid kinases (e.g., phosphoinositide 3-kinases, PI3Ks), as well as nuclear receptors (e.g., theestrogen receptor, ER). Components of developmental signaling pathways, such as Wnt, Hedgehog (Hh), Hippo, and Notch can also be affected, as can downstream nuclear targets of signaling pathways—for example, transcription factors (e.g., Myc and NF-κB), chromatin remodelers (e.g., EZH2), and cell cycle effectors (e.g., cyclins)



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METHODOLOGY:

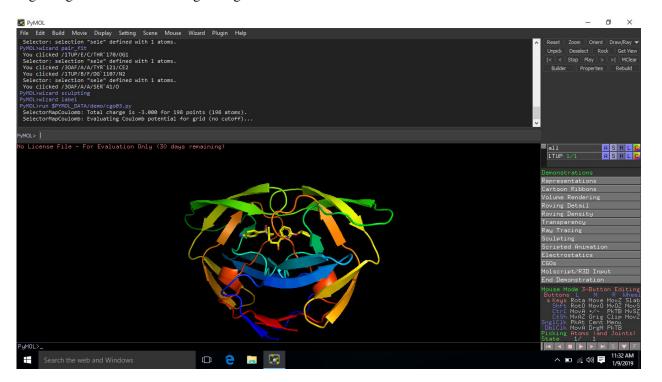
- 1) First analyze a proteome of a human cell- that complete set of proteins produced by one cells about 20000 proteins.
- 2)Picked up peptides which looked likely to be ligands and using computational method recognize potential peptide ligands by their characteristics N terminal signal peptide.
- 3)Also looked on specific cleavage site of Molecules.

Procedure:

We look after several pathways and found some signaling molecules which is responsible for cell proliferation and cancer cell progression and we targeted several molecules which will bind to that molecules hence we can stop the signaling pathway.

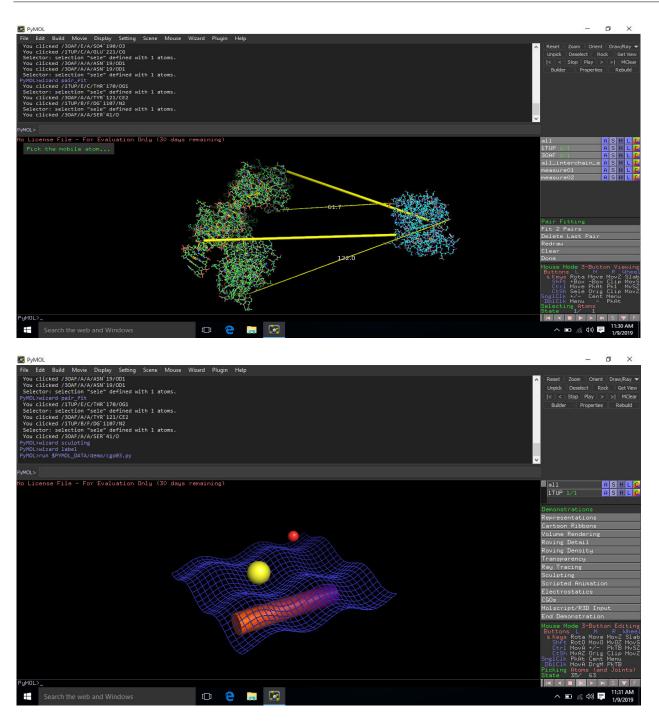
If we can design such molecule which will bind to that molecules active site..then the signaling pathway will stop.

Signaling molecules with designed ligand:



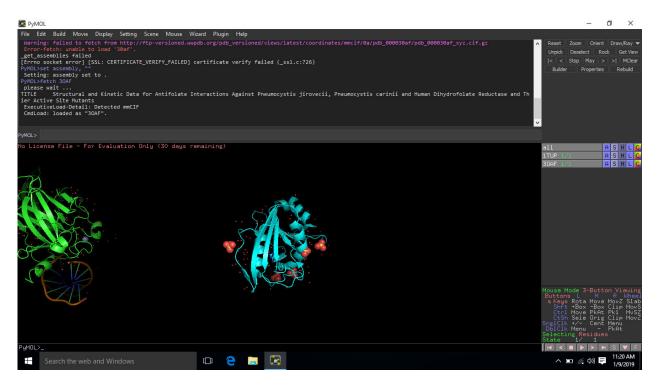


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These are the certain insillico approach towards the cell signaling molecules. Which will bind with particular sites to that signaling molecules.

Concluson:

These are some approaches towards the cell signaling molecules. First analyze a proteome of a human cell- that complete set of proteins produced by one cells about 20000 proteins.

2)Picked up peptides which looked likely to be ligands and using computational method recognize potential peptide ligands by their characteristics N terminal signal peptide.

3)Also looked on specific cleavage site of Molecules.