

Example of medicinal chemistry essay

[Science](#), [Genetics](#)



Mechanism and Progress of Anti-Angiogenic Drugs

Cancers are one of the common and devastating conditions affecting human being for a long time. The landmarks and progress made in achieving consummate and effective drugs for the management of this conditions are worth mentioning. Researchers have embarked on finding solutions to the nagging issue of cancer among human beings, which are commonly based on the behavior of the mutant carcinogenic cells in the body. Understanding the environment that favor mutation and proliferation of mutate genes is a remarkable approach towards getting a long life solution. In this respect, it has been identified that targeting blood vessels development in a tumor tissue will limit the growth. Ideally, blood supply is vital for cell proliferation and growth since it helps in supplying nutrients and oxygen, much needed for metabolism. Therefore, limiting supply of oxygen and nutrients to the mutant cells cut down tumor cells proliferation and growth.

Some of the antiangiogenic drugs that have been approved by the Food and Drug Administration body include Avastin (bevacizumab) and Zactima (vandetanib) (Pecorino, 2009). Avastin was established from a hypothesis of Judah Folkman that limiting nutrients and oxygen supplied to cancer cell can help in treating cancer. The drug acts through inhibiting the initiation of new blood vessels development. Avastin binds to all strains of vascular endothelial growth factor at the receptor binding domain. In this manner, the drug inhibits successful interaction between VEGF-A and the target receptors (Arora & Scholar, 2005). It should be noted that this interaction, when uninterrupted, leads to endothelial cell proliferation and growth causing genesis of blood vessels. Zactima was fully approved for the treatment of

medullary thyroid cancer by Food and Drugs Administration on 2011. The drug is an active inhibitor of vascular endothelial growth factor receptor 2 tyrosine kinase. Inhibiting VEGF –receptor 2 inhibit proactive angiogenesis. Therefore, the drug reduces the development of cancerous cell in the thyroid gland and through involvement of the tyrosine kinase mechanism.

Tyrosine kinases are a group of enzymes that are involved in the transfer of gamma phosphate group to the targeted protein. The phosphate group transferred from adenosine triphosphate. These enzymes are involved in mediating and traducing cellular signals on initiation, limitation or cessation of a number of processes in the body. These processes may be involved in proliferation, apoptosis, growth of the cells, and metabolic activities in cells. The role of these factors in a cell can offer a consummate target for combating cancer development. This is because there are ample tyrosine kinase mediated processes surrounding the development of tumor cells. Therefore, drugs such as Avastin and Zactima inhibit tyrosine kinase responsible for growth and angiogenesis. (Arora & Scholar, 2005).

Role of Sodium Channels in Epilepsy

Epilepsy is a condition that is distinguished by recurrent and unprovoked seizures. Epilepsy results from abnormal neural activities, or accelerated synchronous neural activities in the brain. The bottom line of epileptic episodes is related to the mode of impulse transmission from one neural end to the other. A number of mutations along or in the protein components of voltage-gated mechanism and also the ligand gate ion channels are believed to have imminent contribution to the development of epilepsy. These changes in the channel structure disrupt the transmission of an impulse

leading to seizures, hence epilepsy. One of these channels that are believed to have influence on development of seizures is the sodium channel. Researchers have established that the gating mechanism of the sodium ion pump has some effects and contribution to the development of epilepsy. According to Stafstrom, 2005, the potential of sodium ion causing epilepsy is benched on issues of closing, opening and inactivated of the impulse transfer path. It is believed that the sodium ion factor has profound effects on the amplitude of the impulse dome. Despite that fact the mechanism is also supported by other ions in the body such as calcium, chloride and potassium, the sodium ions have imminent alterations on firing mechanism in the impulse transfer channel, hence a high possibility of causing epilepsy. In some cases of epilepsy, there is profound evidence of mutation on the sodium channels genes resulting to affected impulse transmission that can translate to epilepsy (Stafstrom, 2007).

Sodium channel mutation or defects can lead to repetitive firing of the impulse from the sources, causing confusion. The sodium ions are also associated with maintenance of prolonged depolarization plateau potentials. Some of the epileptic disorders that are highly associated with sodium channel defect include generalize epilepsy with febrile seizures plus disorder and severe myoclonic epilepsy of infancy. Among the genes that may be affected in these conditions at the sodium channel genes in the chromosome 20 include SCN1A and SCN2A. These mutations may lead to slowed inactivation, accelerated activation, accelerated rate of inactivation recovery, and abnormal voltage-dependent activation with hyperpolarizing shift, which are all linked to epilepsy. Sodium ion effect has attracted the

interest of treating epilepsy and lots of drugs have their effect on sodium ion gating mechanism. Phenytoin, Propofol, Lamotrigine and Valproic acid are ideal drugs that reduce the effect of sodium ion potential among the patients. The effectiveness of these drugs supports the role of sodium channels in epileptic conditions (Graves, 2006).

References

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