

Understanding the roots of cancer

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Cancer is a life-changing disease that affects millions of people worldwide. Scientists all over the world are daily trying to win the fight against cancer, find novel therapies and give cancer sufferers hope for cure. But why is it so difficult to defeat cancer? Who are we fighting against? Who is the enemy? Cancer is sneaky; it is our own cells that become transformed and start this war and finding a selective drug targeting only cancer cells is the main obstacle in treating this disease. One way to think of it is that there are good and bad cells. The good ones that we have had peacefully with us all our life and the bad ones that suddenly decided to change, grow faster and even move abnormally around our body. In order to win the fight against cancer, it is important to understand how cells function in our body, how they communicate with each other as well as the reason why they change and grow uncontrollably.

Cell processes including division and motility are regulated by signals the cell receives when a ligand binds to various receptors at the cell surface. This ligand binding results in transmission of signals inside the cell. In other words, cells communicate with each other and in order to regulate how fast they will grow or divide, they have various receptors “antennas” at the cell membrane. These receptors receive the signal, become activated and through proteins transfer the information to the nucleus where the genetic material is located. How this information spreads inside the cell until it reaches the nucleus resembles how news spread in a society. For instance, a person becomes aware of information that is heard on the news, feels the need to spread the information to others and this becomes a chain reaction in which news is usually spreading fast and lead to a specific behavior in the society. Now imagine proteins acting in this way and regulating the information flow inside the cell.

There is a group of receptors called Platelet-derived growth factor receptors (PDGFR), whose activation is essential for regulation of cell division, migration and survival. Among the proteins activated by this receptor, there is one called Extracellular regulated kinase (Erk) 5. Several studies have shown that Erk5 is important for cell division and migration but it has also been involved in various aggressive cancer types and resistance to therapy.

In this degree project, our aim was to understand the mechanisms by which PDGFR activates Erk5 in cells. Our findings that according to cell type, there are different ways of activating Erk5 in response to PDGF indicate the complexity of signaling events concerning Erk5. The central idea is that by finding components in the pathway leading from the receptor to Erk5 we may discover targets for therapeutic intervention.

Degree project in biology, Master of Science (2 years), 2014

Examensarbete i biologi 45 hp till masterexamen, 2014

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