Kinase C receptors perform key function in immune system, aims to develop anti-disease treatments

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By its very nature, the immune system has a somewhat unwieldy, flexible repertoire of â€œto dosâ€ and â€œdonâ€™tsâ€ that depends on everything from the structure of the molecule to its local location. For this reason, a stable immune cell, in addition to being able to clear out a specific pathogen, also has a broad scope in terms of doing so. Now, researchers at Kyoto Universityâ€™s Osaka Wing Chinoi Center for Integrated Fluid Dynamics have discovered a mechanism that controls the expansion of one type of immune cell and could open the door to new anti-disease approaches.

For the study, scientists artificially introduced multiple kinase C, a protein specific to the cells we recognize as white blood cells, in cells of the peripheral blood system. The cells were then exposed to several unexpected substances, including antigens in quantities previously unseen in the cell. Kinase C controlled whether the cell divided rapidly or not. This experimental finding has implications for developing immunotherapies for inflammatory diseases in which fast cell division is usually a contributory factor. In the future, therapeutic drugs should contain both a rapid-dividing kinase C product as well as factors that inhibit its folding. Kinase C-subunit assembling kinase C-specific protein I temphysiology/Kinase C II receptor I>ÃœÃŸ

Ava Kojima, lead author of the study and first author of the paper, has been awarded a 2009 Danish Cancer Society Helen Raneri Fellowship.

TOKYO â€“ When scientists looked at small molecules, such as sugars, in the blood of people with acute myeloid leukemia (AML), we saw something unexpected: cells started to divide at a rate of 67 fold per millimeter. One reason the cells began to proliferate so quickly is because of kinase C receptors, which play a big role in initiating immune response, and are located on the cell surface. Having identified the the gene responsible for these receptors, we set out to understand their behavior.

The most common theory about these kinase C receptors was that they acted as tags on amino acids. Kinase C removes empty amino acids from the RNA, causing the molecule to have a new set of labels.

The strange thing about the sugar family was that the breakdown product was not converted into fuel or biologically important molecules. They werenâ€™t typically sugar-rich and were primarily toxic. Therefore, we wondered if these sugar molecules were actually structural components of the kinase C receptor.

To find out, we created a replica of the in vivo kinase C receptor. This replicant could be a key component in understanding the interactions between our bodyâ€™s immune system and the environment. If our inside is kept clean, the outside will remain clean, which is the reason it takes longer for foreign substances to penetrate and cause symptoms.

In the test, we inactivated the kinase C receptor by also interfering with its transporter. The replicant accurately predicted changes in expression and unfolded marker receptors.

The replicant also underwent three chemical reactions. At the first, the receptor exhibited a different expression pattern than normal. When subsequent chemical reactions took place, the receptor remained oriented in the upright and upright position compared to normal.

So we can say that the replicant in vivo is a machine that can recognize the proteins, polymer derivatives, and the residues that are included in the kinase C receptor. The replicant not only determines whether the receptor will roll over or roll over, but also controls whether a signaling molecule will be transformed into a sugar or has other characteristics. Our findings could be considered as a technology for disease prevention and treatment of inflammatory diseases. A testing system for the similar model of the immune system could improve the identification of new anti-disease drugs.

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