B cells scaffold protein gene could be a drug target for lupus patients

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In a healthy human body, the immune system carries the responsibility to defend the body against all harmful agents that cause diseases, like some bacteria, viruses and other disease agents. The process that follows the entry of the agents to the body and how the body reacts to this situation is called immune response. The immune response involves many proteins and cells, starting from the antigen recognition, passing through special signal transduction by the immune system to activate the cells responsible for the antigen clearance. B lymphocytes (B-cells) are a type of blood cells produced in the bone marrow and play an important role in the immune response. Once B cells are activated due to entrance of harmful agents in the body, these cells pass through different phases starting from immature B cells to antibody producing cells. In the last phase these cells gain the ability to produce proteins called antibodies that trace and eliminate these antigens. The immune system produces sometimes wrong signals leading to the production of abnormal antibodies in the absence of the antigen, these antibodies sometimes attack the cells and the tissues of the human body causing a group of diseases called autoimmune diseases.

Systemic lupus erythematosus (SLE) is an autoimmune disease mainly affecting women in the child bearing age. Common symptoms of this disease are arthritis (joint pain and swelling), fever, fatigue, skin rashes, weight loss, anaemia, sun sensitivity, central nervous system inflammation, muscle pain and weakness.

Since B-cells are responsible for the antibody production, the genes and the proteins involved in B-cells activation and conversion to antibodies producing cells were an intense research area when studying autoimmune diseases. Recently, B-cells scaffold protein gene (BANK1) was discovered to play an important role in B-cells activation and in this lab this gene was found to be associated with SLE, that means that this gene in some way has a role either in causing the disease or affecting the severity of the disease.

In previous studies, the idea was that the BANK1 protein has only one form that represents the normal protein. In this study, it was discovered that BANK1 protein has another form. This new discovered protein form is smaller (truncated) missing some parts of the normal protein. The expression levels of the two transcripts that produce the two protein forms were measured in SLE patients and in healthy people.

The results showed that one of theseforms (the short form) is expressed more in healthy human than the full length (normal) form. Ongoing research in this lab is taking place to measure the expression levels of both isoforms in patient's samples.
So if the full-length form of the gene is expressed more in patient samples, more studies have to be done to determine the role of the full length form in causing the disease or in the disease severity, that could be a good starting point to produce a drug target to cure or to reduce the severity of lupus disease.

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