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Journal of Pharmaceutical Care & Health Systems

Editorial

Development of Hybridism Monoclonal Antibody

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DESCRIPTION

Hybridized Monoclonal Antibody. In the past, it was very difficult to study individual antigenic determinants due to the inherent heterogeneity of the antibody population found in normal antisera. Monoclonal antibodies have solved the specificity and reproducibility issues associated with traditional antisera. The fusion of antibody-producing spleen cells and immortal myeloma cells is called a hybridoma. Hybridomas can adapt to growth in tissue culture and produce antibodies.

In addition, they secrete only one type of antibody, that is, an antibody against only one antigenic determinant. Hybridoma technology allows the production of monoclonal antibodies that react with only a single antigenic determinant. Monoclonal antibodies act on a single specific epitope and are uniform in terms of affinity and specificity.

This allows for customized monoclonal antibodies. Freezing hybridoma cells for later retrieval ensures a reproducible source of specific reagents. Therefore, monoclonal antibodies can be used in commercial and clinical diagnostic kits, imaging, histocompatibility testing, and in the diagnosis and treatment of a variety of diseases.

However, the antibody response to spontaneous infection or active immunization is polyclonal. In other words, many B cells are involved, each recognizing different antigenic determinants (epitope) of the immune antigen and secretion different immunoglobulins. Thus, immunized human or animal sera usually contain a mixture of antibodies, which can all be combined with the same antigen, but with different epitopes appearing on the surface of the antigen. Moreover, even antibodies that bind to the same epitope often have different abilities to bind to that epitope. This makes it very difficult to isolate significant amounts of a particular monoclonal antibody from a polyclonal mixture.

Although the production of monoclonal antibodies from rat or mouse cells has become routine, the construction of human hybridomas has not been so simple. This is because most human myeloma cells do not grow well in culture and do not produce stable hybridomas.

However, when human B cells isolated from the blood are infected with the Epstein-Barr virus, the causative agent of infectious mononucleosis, they proliferate in culture and continue to secrete immunoglobulins. Very few of them are likely to produce antibodies with the desired specificity, even from immunized subjects. However, in some cases, immunologists can identify and select cells that secrete the immunoglobulin of interest.

These cells can be grown in culture as a single clone that secretes a monoclonal antibody. Researchers used this procedure to obtain a human monoclonal antibody against Rh antigen. A simpler method of constructing human monoclonal antibodies can be achieved using recombinant DNA technology. When a mouse monoclonal antibody is constructed using the conventional method described above, the DNA encoding the antigen-binding portion of the antibody molecule can be isolated and fused with the human DNA encoding the antibody.

The hybrid DNA is then inserted into a bacterium that produces monoclonal antibodies in half the mouse and half the human. Antibodies produced in this way are less likely to elicit an anti-antibody response when administered to humans.

Further tweaks can be made to modify any part of the antibody that is not directly involved in binding to a particular antigen. This technique has been used to produce a number of different monoclonal antibodies for therapeutic use.

The human monoclonal antibody can also be produced using phage display technology. In this approach, human antibody proteins are constructed and expressed on the surface of phage (bacteriophage) vision particles. The fusion phage culture can then be purified to increase the amount of fusion protein available for further research. Adalimumab was the first fully human monoclonal antibody developed by phage display and approved for clinical use in human patients. It was approved in 2002 as a treatment for rheumatoid arthritis.

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Received date: September 6, 2021; **Accepted date:** September 20, 2021; **Published date:** September 28, 2021

Citation: Nanda R (2021) Development of Hybridism Monoclonal Antibody. J Pharma Care Health Sys.S8:e001.