

Researchers uncover origin of virus-fighting plasma B cells

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Plasma B cells, which move through the body releasing antibodies, are an important component of the body's fight against viral infections and other invaders. They are relatively rare, however, and increasing their ranks is a major goal of vaccination. However, scientists do not yet fully understand how precursor cells are selected either to enter the body's fluids as plasma cells or remain in the places, known as germinal centers, where they undergo reprogramming to become more specific attackers of an infection.

Now, in work published in *Immunity*, a group of researchers led by RIKEN and Osaka University have discovered an important mechanism that governs how B [cells](#) are chosen to become plasma cells. They found that the key to the commitment to a new state involves the strength of the interaction between the B cells and another type of immune cell, called T follicular helper cells.

It is known that [plasma cells](#) express a transcription factor called IRF4, and conversely do not express a transcription factor called Bcl6. The group wondered whether the fate of the cells was already being determined while they were in the [germinal centers](#)—groups of cells located in lymph nodes and the spleen where B cells mature. They examined the expression of these two factors in B cells still located in germinal centers, and found that in a subset of cells, Bcl6 was expressed at a low level and IRF4 at a high level. They also found that these cells express a cellular marker called CD69.

To determine whether these cells they had identified were indeed precursors to plasma B cells, they compared the cells they had identified with germinal center-derived plasma B cells, and found that the gene sequences of their B cell receptors, which govern the antibodies they produce, were very similar, indicating that they were from the same cell group. It was also clear that these cells shared the same developmental features with germinal center-derived plasma B cells, suggesting that they were indeed precursors.

Lastly, they decided to examine the relationship between these cells and T follicular helper cells, which are important for the maturation of B cells. Since the T follicular helper cells stimulate B cells with a surface protein called CD40, the team created B cells that express CD40 at low levels, and found that the number of plasma B cells dropped dramatically. Further experiments showed that the strength of the interaction between the plasma B cell precursors and the T follicular helper cells determines whether the B cell precursors are chosen to be plasma B cells or to remain in the germinal center to undergo further mutation before either being selected to perish or to become a high-affinity plasma B cell.

Tomohiro Kurosaki of the RIKEN Center for Integrative Medical Science, who led the study, says, "Understanding how the body generates high-affinity [plasma](#) B cells, which are important in fighting [viral infections](#) such as influenza, which kills around 1,000 people per year in Japan, is important for creating more powerful vaccinations. Our work has given us important insights into how these cells are produced, and we hope it will be useful in this effort."

More information: Wataru Ise et al, T Follicular Helper Cell-Germinal Center B Cell Interaction Strength Regulates Entry into Plasma Cell or Recycling Germinal Center Cell Fate, *Immunity* (2018). [DOI: 10.1016/j.immuni.2018.03.027](https://doi.org/10.1016/j.immuni.2018.03.027)

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