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The Role of Antibodies in the Light of the Theory of Evolution

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Abstract

The phenomenon of facilitation of viral infections by antibodies (ADE antibody dependent enhancement) as well as the resistance of agammaglobulinemia patients to certain viruses are in contradiction with the protective role of antibodies affirmed by classical immunology. This must be compared to the opsonizing antibodies that promote the specific phagocytosis of extra-cellular bacteria. However, questions about the role of antibodies have been raised since the beginning of the history of immunology.

More recently, Pierre Sonigo has shed light on the contradictions between the finalist interpretation of the role of lymphocytes and the theory of evolution: how can it be explained that cells are selected to protect the organism they constitute? The role of anti-viral and antiintracellular bacteria antibodies could be to allow phagocytosis by the cells: either directly by the Fc fragment of immunoglobulins, or via the complement for many cell types. This makes it easy to understand the selection of antibodysecreting cells. Natural selection favors the cells that produce the most affine Ig and thus guides the maturation of the proB cell to the plasma cell.

A review of recent publications in theoretical immunology is consistent with this hypothesis. The theory of evolution should be integrated at every level of research and teaching in immunology, as it is for biology as a whole.

Introduction

The phenomenon of antibody facilitation of viral infections has recently been re-discussed in relation to the clinical aspect of Covid-19 (Yushun et al., 2020 and Banoun, 2020) and vaccines against this infection (Roper and Rehm, 2009). Antibody dependent enhancement is the accepted mechanism to explain severe reinfections due to dengue virus—among others—(Taylor et al., 2015) as well as the higher occurrence of severe dengue in vaccinated (compared to unvaccinated, Feinberg and Ahmed, 2017).

This effect of antibodies appears to contradict immunological theory, which states that the “role” of antibodies is to protect organisms against pathogens, including viruses.

However, contradictory observations have long been noted.

Already in 1956, a review was published (Good and Zak, 1956) which referred to “the clinical paradox posed by the apparently satisfactory resistance of patients with agammaglobulinemia to certain viral infections and the failure of their response to the virus antigen…. ”. As noted by Burnet (1968), measles immunity is independent of antibodies, but depends solely on cellular immunity. The same demonstration has recently been made for immunity to VSV (vesicular stomatitis virus, Moseman et al., 2012).

These observations have been reviewed by Sanna and Burton (2000). They are criticized because all patients diagnosed wuth aγglobulinemia would have received IM immunoglobulin as early as the early 1950s, and thus the complete null phenotype has been little studied. The authors suggest that the treatment given to these patients indicates that antibodies may play a role in viral infections. Some viral infections that these patients developed before treatment virtually disappeared after the initiation of treatment. However, it cannot be denied that the aγglobulinemia patients were discovered by the bacterial infections they developed and not by the viral infections.

It appears that humoral immunity plays a role in HSV (Herpes simplex virus, neurologically disseminated enterovirus) infections for viruses with nervous tropism and for persistent viral infections.

Moreover, it is difficult to attribute the discovery of an infection in an antibody deficient patient to a particular virus: the serological diagnosis is inoperative; it was necessary to wait for the culture of the viruses and especially the PCR to affirm a viral infection.

Therefore, this last publication does not call into question the first observations on aγglobulinemia patients: patients are very sensitive to bacterial infections and for the majority of viral infections, their sensitivity is comparable to that of the general population.

Much more recently, the pandemic at Covid-19 has mobilized thousands of researchers and allowed significant advances in immunology and virology. One study compared serologies and cell type immunity in Covid-19 index patients and their contacts: only index patients became seropositive, but both groups showed robust and specific cell type reactivity to SARS-CoV-2 (the virus responsible for Covid-19) (Gallais et al., 2020).

Similarly Sekine et al. (2020) showed that most individuals with asymptomatic or moderate Covid-19 generated highly functional durable memory T-cell responses in the absence of a corresponding humoral response.

The role of antibodies in bacterial infections is well established in the defense against extracellular bacterial infections (as opposed to intracellular infections) (Berche, 1988). The frequency of infections in patients with genetic abnormalities of phagocytes is indicative of the importance of phagocytosis.

Bacteria, once phagocytized, are degraded and then exocytosed, making the antigens accessible to the cells of adaptive immunity. The antibodies then act by promoting phagocytosis, which is the natural process of fighting pathogenic bacteria: they are

opsonizing antibodies, another term for facilitators. Could the neutralization and agglutination of bacteria observed in vitro not occur in vivo?

Complement (a group of serum proteins) plays an important role in phagocytosis. These proteins act on the one hand by binding to specific antibodies, but complement is also activated by the alternating pathway induced directly by surface bacterial antigens: lipopolysaccarides, capsule polyosides, lipotechoic acids, and this in the absence of antibodies.

However, as with viral infections, the first line of defense is innate immunity: during a primary infection it takes 7 to 10 days to mount a specific humoral reaction, and it is non-specific phagocytosis that operates first. A good example is pneumococcal pneumonia. Pneumococcus is a commensal upper airway bacterium that becomes pathogenic when it acquires a phagocytosis-resistant capsule. In young adults the evolution is typical: after incubation of 1 to 3 days, a sudden onset, high fever, cough, the evolution is favorable in 8-10 days with a sudden improvement. This improvement corresponds to the appearance of specific antibodies. The inflammatory (polynuclear) reaction aided by the opsonizing specific antibodies will destroy the pneumococci and lead to healing.

How can this opsonizing role of antibacterial antibodies be related to the phenomenon of the facilitation of viral infections by the antibodies?

Pierre Sonigo, one of the discoverers of the AIDS virus in the 1980s, reflected on the theory of immunology and the problems it poses in relation to the theory of evolution (Kupiec and Sonigo, 2003).

Before giving a brief summary of his theses, a historical overview of this science can account for the theoretical gaps that accompanied its birth.

Read the full peer-reviewed article here.

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