From recognition to activation

The primary immune response is the adaptive response to a first encounter with a particular antigen. Different immune cells cooperate to build a specific defence.

1. MACROPHAGES PRESENT ANTIGENS

A macrophage engulfs a pathogen by phagocytosis and digests it. It can display pathogen-derived antigen fragments on its surface, attached to MHC proteins. This links pathogen capture with recognition by T-helper cells.

2. MATCHING T-HELPER CELLS RESPOND

A T-helper cell with a complementary receptor recognises the presented antigen. With the necessary activation signals, it becomes activated and multiplies. T-helper cells release cytokines that help coordinate other immune cells.

3. ANTIGEN RECOGNITION IS SPECIFIC

Antigen presentation

An antigen fragment is displayed on a cell surface.

Clonal selection

The antigen selects lymphocytes with matching receptors.

Clonal expansion then increases the selected cell population by mitosis. Selection identifies the match; expansion makes more matching cells.

4. TWO COORDINATED ROUTES

B-lymphocytes can form plasma cells that secrete antibodies into body fluids. T-killer cells act on infected body cells. These routes can overlap in time; they are not two separate infections or a rigid single-file sequence.

Antigen presentation and T-cell roles: OpenStax, Microbiology 18.3.
B-cell activation and antibody response: OpenStax, Microbiology 18.4.

Scope: Cambridge Biology 9700 syllabus, section 11.1, outcome 3, p. 31.

Two routes to protection

Antibodies target antigens outside cells; T-killer cells eliminate infected host cells. Keep the cells, their products and their targets distinct.

1. B-CELL ROUTE: ANTIBODY PRODUCTION

Matching B-lymphocyte

A B cell binds a specific antigen, takes it in and presents fragments. For the T-dependent response shown here, a matching activated T-helper cell supplies contact signals and cytokines.

Clonal expansion

Activated B cells divide by mitosis.

Plasma cells

Secrete specific antibodies. These can neutralise toxins or viruses and help mark targets for removal by phagocytes.

Memory B cells

Persist after the response. They are living cells, not stored antibodies, and can respond on a later encounter.

2. T-KILLER ROUTE: INFECTED-CELL DESTRUCTION

Activated T-killer cells recognise matching antigen fragments presented by infected body cells. They trigger the target cell's death, helping stop intracellular pathogens from multiplying.

Do not swap the jobs: plasma cells secrete antibodies; T-helper cells coordinate; T-killer cells destroy infected host cells.

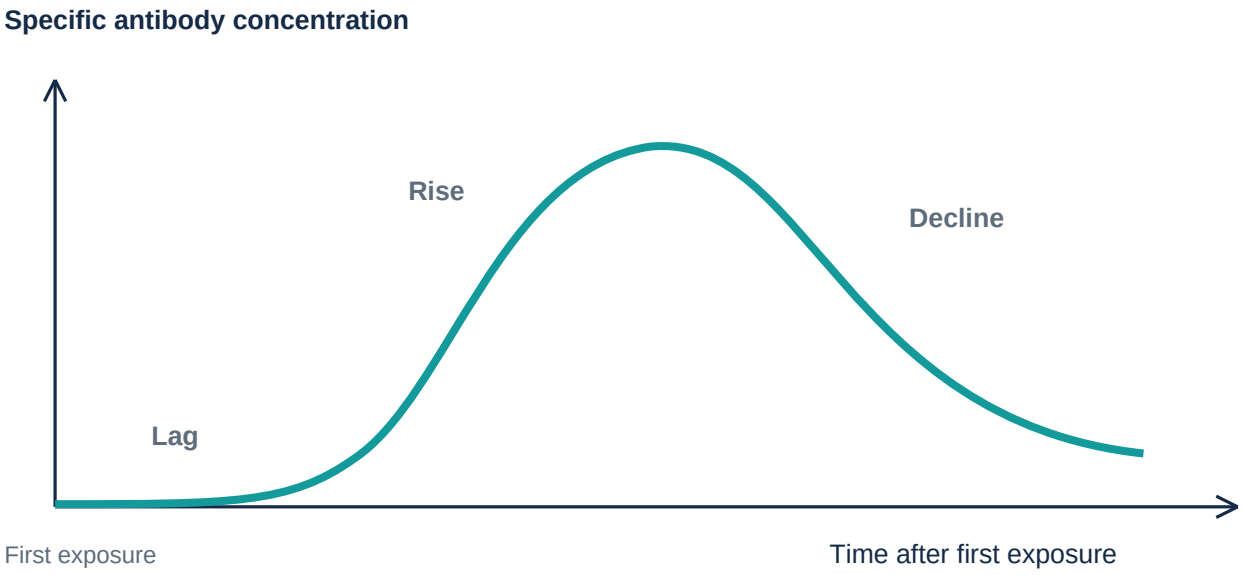
B-cell differentiation: British Society for Immunology, B Cells.
T-helper and cytotoxic T cells: OpenStax, Microbiology 18.3.

Scope: Cambridge Biology 9700 syllabus, section 11.1, outcome 3, p. 31.

Why the first response takes time

Recognition, activation, clonal expansion and differentiation must occur before large amounts of specific antibody can be produced.

1. A PRIMARY ANTIBODY RESPONSE



Schematic only: the time scale and peak vary with the antigen and individual.

2. READ THE CURVE AS A CELLULAR PROCESS

During the lag, matching lymphocytes are activated and clones expand. Antibody concentration rises as plasma cells secrete antibodies. It later falls as production declines and antibodies are broken down. A lag in this graph does not mean innate defences are inactive.

3. WHAT REMAINS AFTER THE RESPONSE?

Some activated lymphocytes form memory cells. Some plasma cells can also be long-lived, so a falling antibody level does not mean all protection has vanished. Memory is the bridge to the secondary response, explored in the next lesson.

Antibody-response timing: OpenStax, Anatomy and Physiology 2e, 21.4.
Primary response and memory: OpenStax, Microbiology 18.4.

Scope: Cambridge Biology 9700 syllabus, section 11.1, outcome 3, p. 31.