4.8 Immune response, antigens and antibodies

Name: _____ Class: _____ Date: _____ **Total: 19 marks**

KEY WORDS humoral ( ) • plasma cell • variable region • monoclonal antibody • secondary response

Objective

Practise statements 6.8-6.12: non-specific responses, antigens, the humoral and cellular immune responses, antibody structure, the four kinds of immunity and vaccination.

1 Key knowledge and worked examples

■ The two arms of the specific response

Humoral : antigen binds a B cell's surface antibody → clonal selection → B effector cells divide into plasma cells secreting antibody; B memory cells remain. **Cellular** : T helper cells activate other cells (interleukins); T killer cells destroy infected cells by perforin; T memory cells persist.

■ The antibody molecule

Four chains (two heavy, two light), disulfide bridges, a **variable region** that differs per clone – the binding site – and a constant region. The **antigen-antibody complex** then triggers phagocytosis or lysis.

■ Four kinds of immunity

Natural active (infection), artificial active (vaccine), natural passive (mother's antibodies across placenta and milk), artificial passive (antiserum injection – immediate, short-lived, no memory).

■ The secondary response

Second encounter: memory cells divide at once – antibody rises in days, to a far higher titre, with IgG predominating. Vaccination schedules exploit this.

■ Worked example: reading an antibody-titre graph

Day 0 injection: titre rises by day 7, peaks day 21, falls. Booster at day 60: titre peaks in 5 days at 10× the first peak – clonal expansion from memory B cells. Passive antibody given instead would show an immediate high titre falling without any rise – no memory.

2 Practice

2.1 Name the cell that secretes antibodies. [1]

2.2 Describe the difference between active and passive immunity. [2]

2.3 State two roles of T helper cells. [2]

3 Exam-style questions

3.1 Describe the structure of an antibody molecule and explain how its shape gives each antibody specificity for one antigen. [5]

3.2 Explain why a mother's vaccination against a pathogen protects her newborn, naming the type of immunity the baby acquires and its limitation. [4]

3.3 Monoclonal antibodies are made by fusing a B cell with a tumour cell. Explain why the tumour partner is needed, why many different B cells must first be screened, and one use of the final monoclonal antibody in diagnosis. [5]

Solutions

Original-task marking: M1 method, A1 accuracy, B1 reasoning.

2.1 Plasma cell.

2.2 Active – the body makes its own antibodies (and memory); passive – antibodies made elsewhere are given to the patient.

2.3 Release interleukins that stimulate B cell division and macrophage activation; help T killer cells develop.

3.1 Four polypeptide chains – two heavy, two light – joined by disulfide bonds; each chain's end carries a variable region whose amino-acid sequence is unique to the clone; the two variable regions form a single binding site complementary in shape and charge to one antigen's epitope; the constant region handles effector functions like phagocyte binding.

3.2 Vaccination gives the mother high antibody titres; IgG crosses the placenta and passes into breast milk. The baby acquires natural passive immunity – immediate protection, but the antibodies are broken down within months and no memory cells are made, so protection fades.

3.3 Normal B cells divide only a few times; the tumour cell's endless-division property makes the hybridoma immortal, producing antibody indefinitely. Each B cell makes antibody to ONE antigen; the mixture from immunisation must be screened to find the clone binding the wanted target. Use: a monoclonal that binds a hormone or tumour marker in a pregnancy or diagnostic test.