

The inflammatory response



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The body is designed to defend itself against invading bacteria, and infection. The skin and mucous membranes are the first line of defence, the invasion of foreign bacteria can pass this first line of defence and immediately triggers the second line of defence. The second line of defence is the inflammatory response (McCance & Huether, 2009). The mechanism of the inflammatory response is to protect the injured site by killing the agent responsible, limiting its effects on the rest of the body and initiating the healing process (Porth, 2007).

According to Botwinski (2001), during infection bacteria grow and divide, and release potent toxins that cause damage to the body's cells. These toxins trigger the initiation of the inflammatory response. The changes that occur are initiated by the interactions between bacterial products and inflammatory mediators. Inflammatory mediators are chemicals that are released by protective cells or plasma when harmful agents invade the body. Inflammatory mediators include histamine, prostaglandins, and leukotrienes (Kumar, Abbas, Fausto, Robbins, & Cotran, 2005).

The main cells involved are the mast cells and are located in connective tissue in close contact with blood vessels. Mast cells play a key role in the inflammatory response, when stimulated by infection they release a potent substance called histamine. When histamine leaks into the tissues it causes changes in the surrounding blood vessels. The two changes that occur in the blood vessels is blood vessel dilation and increased capillary permeability. The changes are designed to maximise the movement of plasma proteins and circulating cells out of the blood flow and into the site of infection (McCance & Huether, 2009).

At the onset of injury the histamine that is released causes the blood vessels at the site to constrict for a short time then dilate (Nair, 2009). This widening of the blood vessels increases blood flow, and therefore increases the amount of oxygen, nutrients, and white blood cells being delivered to the site of injury (Botwinski, 2001). New blood vessels can also open up at the site and the area becomes flooded with blood. This increase in blood flow produces the characteristics of redness and warmth and are the earliest symptoms seen in the inflammatory response (Porth, 2007).

The blood vessel wall confines blood products and cells in the circulation and prevents it from leaking out into the surrounding tissues of the body. This is done by cells called endothelial cells that tightly line the walls (Braun & Anderson, 2006). The chemicals that are released at the site of injury bind with receptors on the endothelial cells and cause's them to retract producing gaps in their walls. This structural change allows healing fluid and cells to escape out into the affected area (Porth, 2007). Braun & Anderson (2006) describe that the harmful bacteria are diluted by this increased amount of fluid.

The increased fluid and pressure produces the swelling seen at the site of infection. Nerve endings can also be stimulated as a result from the increasing pressure and this is what can cause the pain associated with infection (Nair, 2009). An important part of the inflammatory response is to send circulating white blood cells to the infected area. They are attracted in large numbers to the scene of injury as a result from blood vessel dilation and the release of bacteria substances and inflammatory mediators (Kumer et al. 2005). The white blood cells job is to provide a defence by killing

invading bacteria, and getting rid of dead tissue. The main white blood cell in the inflammatory response is the neutrophil and arrives first at the site of inflammation approximately six to twelve hours after the initial injury (McCance & Huether, 2007). Macrophages are cells that live in various tissue locations and like the neutrophils they are released in the blood stream and attracted to the substances released at the site of infection.

Macrophages and neutrophils are called phagocytes, and share the same job in the inflammatory response, which is to clean up the damage by ingesting and killing the invading bacteria (McCance & Huether, 2005). According to Sherwood (2009), the phagocytes floating in the circulation stick to the inside of the blood vessel wall, this process is called mirgination. Diapedesis then occurs, in this process the phagocytes squeeze through the gaps of capillary walls that were formed earlier on in the inflammatory response.

Chemical mediators called chemotaxins accumulate at the site of infection and attract the phagocytes. Once inside the surrounding tissue the phagocytes make their way through the tissue to areas where there are higher concentrations of chemotaxins, this process is called chemotaxis (Roitt & Delves, 2001). Once at the affected area the neutrophils and macrophages eliminate the invading bacteria from the inflammation site by a process called phagocytosis. Phagocytosis is “ cell-eating” and is a three stage process composed of recognition and attachment, engulfment, and killing and degradation (Kumar et al. , 2005).

Phagocytes have receptors on their surface that enable them to recognise and attach to the receptors on the surface of the microorganism, this

attachment prevents the bacteria from “ getting-away”. In the process of engulfment the phagocytes stretch two surface like projections called pseudopods around the microorganism until it completely surrounds it, the microorganism is then trapped inside. Potent chemicals and enzymes are released inside the phagocyte and these break down and kill the invading microorganism. The pus that forms at the infected site is the accumulation of these phagocytic cells both living and dead (Sherwood, 2009).

McCance & Huether (2009) describe that there are three protein systems that are also initiated during the inflammatory response. They are the complement, clotting and kenin systems, and consist of protein enzymes. These systems work along side the protecting cells and help them carry out their roles. The complement system consists of potent defensive proteins that help in the destruction of invading bacteria. The clotting system produces fibrinous tissue that acts as a boundary by trapping the bacteria and maximising the activity of the phagocytic cells.

The clot that is formed also minimizes blood loss and prevents spread of infection (Botwinski, 2001). The kenin system consists of mainly bradykinin a protein that cause’s dilation of vessels, vascular permeability, and pain (McCance & Huether, 2009). The protective mechanisms of the inflammatory response prepare the site for healing and regeneration of the destructed tissue. Depending on the severity of infection and damage this can be a long process and is finished when structure and function is returned to normal (McCance & Huether, 2009).