Study gives new insight on inflammation

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Scientists' discovery of an important step in the body's process for healing wounds may lead to a new way of treating inflammation.

A study published today in Current Biology details how an international team of researchers led by Monash University's Australian Regenerative Medicine Institute (ARMI) discovered the mechanism, which shuts down the signal triggering the body's initial inflammatory response to injury.

When the body suffers a wound or abrasion, white blood cells, or leukocytes, travel to the site of the injury to protect the tissue from infection and start repairing the damage. However, this period of inflammation need only be temporary. If the body allows the inflammatory stage to continue for too long, the next phase of healing is compromised.

Previous research identified the initial signal that calls the leukocytes to the site of the injury, but how this early signal was switched off, letting the leukocytes know that they were no longer urgently needed, was unknown. The latest findings show that an enzyme called myeloperoxidase is the key to this process.

The team studied zebra fish with modified leukocytes and tissues that fluoresced different colours, enabling leukocyte movement and the concentration of chemical signals to be monitored simultaneously. By observing the tiny, transparent fish under a microscope, the researchers were able to observe individual white blood cells and how they are

regulated in the inflammatory phase of the healing process.

Lead researcher Professor Graham Lieschke of ARMI said the findings suggested new possibilities for treating inflammation.

"White blood cell activity is important for determining the balance between repair, scarring and healing. Understanding what regulates leukocyte activity during inflammation should ultimately allow us to manipulate this system and maximise healing and repair," Professor Lieschke said.

"Our research has identified a new pathway to target with anti-inflammatory drugs. There is a significant need for new treatment options as current drugs are not effective in all circumstances."

Professor Lieschke said the findings were especially relevant to understanding and treating the hereditary disease myeloperoxidase deficiency, which affects leukocyte function in approximately one in every 2000 people.

Provided by Monash University