

How the 'police' of the cell world deal with 'intruders' and the 'injured'

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Dr Piccinini in the School of Pharmacy at The University of Nottingham. Credit: The University of Nottingham

The job of policing the microenvironment around our cells is carried out by macrophages. Macrophages are the 'guards' that patrol most tissues of



the body - poised to engulf infections or destroy and repair damaged tissue.

Over the last decade it has been established that macrophages are capable of detecting changes in the microenvironment of human tissues. They can spot pathogen invasion and tissue damage, and mediate inflammatory processes in response, to destroy microbial interlopers and remove and repair <u>damaged tissue</u>. But how do these sentinels of the cell world deal with infection and <u>tissue injury</u>?

Dr Anna Piccinini, an expert in inflammatory signalling pathways in the School of Pharmacy at The University of Nottingham, has discovered that the macrophage's 'destroy and repair service' is capable of discriminating between the two distinct threats even deploying a single sensor. As a result, they can orchestrate specific immune responses - passing on information in the form of inflammatory molecules and degrading tissue when they encounter an infection and making and modifying molecular components of the tissue when they detect tissue damage.

Dr Piccinini's research is published today, Tuesday 30 August 2016, in the academic journal *Science Signaling*. Her findings could provide future targets for the treatment of diseases with extensive tissue damage such as arthritis or cancer where inflammation plays an increasingly recognised role.

Dr Piccinini said: "Although we know <u>macrophages</u> are capable of detecting different threats using a single sensor we have never really understood whether they use the same or distinct defence strategies depending on the kind of threat they are confronted with. And, what is interesting is that we are also understanding better how macrophage behaviour affects the microenvironment around them."



This research was funded by the Medical Research Council, Arthritis Research UK and the Kennedy Trust for Rheumatology Research. The work was carried out at the University of Oxford in Professor Kim Midwood's laboratory.

Dr Piccinini said: "This research paves the way to identify specific molecular events that arise from the cell's specific response to infection and sterile <u>tissue damage</u> and in future may allow us to find specific targets to manipulate inflammation."

Provided by University of Nottingham

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