Watching assassin cells at work

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L'Oréal Fellow Misty Jenkins and her colleagues at the Peter Mac have a paper published on Sunday that reveals how our immune cells kill damaged damaged, virus-infected or cancerous cells taking between one and two minutes to make the hit. Then these serial killers are free to strike again. And although the killer cells themselves are vulnerable to

the poisons they use, they are somehow able to protect themselves from damage.

That's the chilling picture painted in the paper in the 1 September edition of the *The Journal of Immunology* of which Peter MacCallum Cancer Institute researchers Dr Jamie Lopez and Dr Misty Jenkins are joint first authors. Jenkins was last week was awarded a prestigious L'Oréal Australian and New Zealand For Women in Science Fellowship for her work on killer T cells.

The findings should open the way to better understanding and more efficient cooperation with the immune system in treating disease.

The Peter MacCallum research used a new live-cell microscope technique developed by Lopez and Dr Ilia Voskoboinik to log exactly what goes down during a hit. The live imaging technique allows immunologists to determine the exact time at which the hit is initiated. It can be used to unravel other fundamental details of how the cells of the immune system operate, the researchers the write.

When killer cells lock onto a victim they rearrange themselves internally to direct a series of granules—poison-filled grenades—onto the outer membrane of their target. The granules contain a compound called perforin which punches a hole in the target cell membrane to allow the poisons to enter. The cell promptly commits suicide.

The Peter MacCallum group found that in both humans and mice, this process typically took no longer than 100 seconds. The researchers also showed that if immune cells themselves were the target, they were just as susceptible as any other cell. But the poisoning process never seems to harm the killer cell. So somehow it manages to protect its membrane from perforin damage. The researchers are already investigating how this happens.

More information: www.jimmunol.org/content/191/5/2328