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Intestinal worm infections increases severity of STIs in females — research finds

New research has found that intestinal worm infections may put sub-Saharan Africa women at increased risk of sexually transmitted viral infections (STIs). The findings were published this week in the *Cell Host and Microbe* journal.

Rates and severity of sexually transmitted viral infections in sub-Saharan Africa are very high and are one of the world's leading causes of pathological disease. Worm infection rates are also very high in this region; but do not colonise the female reproductive tract. Worm infections in the intestine can change immunity in other parts of the body.

This research was led by researchers from the University of Cape Town (UCT) in collaboration with the University of Bonn, Norwegian University of Science and Technology, University of Liege, University of Birmingham, and French National Centre for Scientific Research.

Lead author, Dr William Horsnell from UCT's Institute of Infectious Disease and Molecular Medicine, said: "We have found that intestinal worm infection can change female reproductive tract (FRT) immunity by causing a worm associated immune response in the FRT, even though the worms never reside there. In particular we found that worms induced eosinophils in the vagina. These are immune cells associated with anti-worm immunity and can cause allergic disease. Their role in the FRT is not known."

Horsnell said intestinal worm infections strikingly increase vaginal HSV2 infection induced pathology. "We identified that worm infections cause a very large increase in tissue necrosis following vaginal HSV2 infection. We could prevent this worm induced increase in HSV2 pathology by specifically inhibiting worm induced changes in the FRT, especially by impairing the eosinophil response."

According to the researchers, these findings were very unexpected. The findings show that worm infections that never colonise the FRT still cause a strong change in FRT immunity. The consequence of this change is that following a viral sexually transmitted infection the pathology caused by the virus is hugely increased.

Horsnell commented: "To date research into STIs has largely neglected the role of worm infections in influencing the severity of these important diseases. We identified that eosinophils cause this effect. Very little has ever been studied addressing the role of eosinophils in the FRT."

He said the research is important for health workers as it may help them to explain why STIs can be more virulent in areas where worm infections are common.

"We identified that this effect is driven by a particular type of immune cell: eosinophils. When we target these cells with molecules that can deplete them, we can prevent the increase in pathology. This suggests that this pathology can be targeted and maybe prevented or reduced by using existing drugs. The research also shows that eosinophils can have a very important role to play in vaginal immunity. This has never been so strongly demonstrated before."

The researchers hope that this work will lead to more support of research to understand how parasitic worm infections indirectly influence control of sexually transmitted infections.

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