Communication and Marketing Department
Isebe loThungelwano neNtengiso
Kommunikasie en Bemarkingsdepartement

Private Bag X3, Rondebosch 7701, South Africa
Welgelegen House, Chapel Road Extension, Rosebank, Cape Town
Tel: +27 (0) 21 650 5427/5428/5674 Fax: +27 (0) 21 650 5628

www.uct.ac.za

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HIV and TB co-infection skews SARS-CoV-2 T cell response, study finds

A study led by the University of Cape Town (UCT) has provided new information on the relationship between SARS-CoV-2–specific CD4 response to COVID-19 severity and impact of HIV and tuberculosis (TB) co-infection.

COVID-19 is caused by SARS coronavirus 2 (SARS-CoV-2).

Published in the *Journal of Clinical Investigation*, the researchers assessed the magnitude, function and phenotype of SARS-CoV-2-specific CD4 T cells in 95 hospitalised COVID-19 patients (38 of them being HIV and/or TB co-infected) and 38 non-COVID-19 patients, using flow cytometry.

"We found that SARS-CoV-2-specific CD4 T cell attributes, rather than magnitude, associates with disease severity, with severe disease being characterised by poor polyfunctional potential and reduced proliferation capacity," said Dr Catherine Riou, lead researcher and senior research officer at UCT's Wellcome Centre for Infectious Diseases Research in Africa (CIDRI-Africa).

"HIV and TB co-infection skewed the SARS-CoV-2 immune response. HIV mediated CD4 T cell depletion associated with suboptimal T cell and antibody responses to SARS-CoV-2 and a decrease in the polyfunctional capacity of SARS-CoV-2-specific CD4 T cells was observed in COVID-19 patients with active TB," said Riou. "These results corroborate the important role of SARS-CoV-2-specific T cells in COVID-19 pathogenesis and support the concept of altered T cell functions in patients with severe disease."

The results also revealed that COVID-19 patients displayed reduced frequency of *Mycobacterium tuberculosis*–specific CD4 T cells.

Professor Robert J Wilkinson, co-author and director of CIDRI-Africa, said many viruses, including SARS-CoV-2, cause a temporary immunosuppressive effect, which could lead to the reactivation of subclinical bacterial infection. Thus, in a TB-endemic country such as South Africa, many concerns have been raised about the possibility that COVID-19 could reactivate latent TB.

Wilkinson suggested that HIV infected patients with SARS-CoV-2 need to be started on antiretroviral therapy (ART) if not already established.

"HIV infected persons should be stabilised on ART and then prioritised for vaccination. The co-occurrence of TB reduces the effectiveness of the anti- SARS-CoV-2 immune response and TB should be suspected as a complication of SARS-CoV2," he said.

Associate Professor Katalin Wilkinson, principal laboratory research scientist at the Francis Crick Institute in London, commented: "The clinical and epidemiological interactions of COVID-19 with TB and/or HIV pose an additional health threat. Although comorbidities associated with HIV and TB may primarily drive COVID-19 severity in these populations, it is also plausible that HIV and/or TB-associated immune dysregulation may contribute to heightened risk."

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Issued by: UCT Communication and Marketing Department

Ridovhona Mbulaheni
Media Liaison Assistant
Communication and Marketing Department
University of Cape Town
Rondebosch
Tel: (021) 650 2333
Cell: (064) 905 3807
Email: ridovhona.mbulaheni@uct.ac.za
Website: www.uct.ac.za