

HIV Disrupts Lung Clock, Raising COPD and Emphysema Risk

The HIV Tat protein disrupts the lungs' molecular clock, the timekeeping system that regulates daily lung function and immune response.

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People living with HIV face a greater risk of developing lung diseases at a much younger age, even if they have never smoked.

Florida International University researchers have now uncovered a previously unknown mechanism that helps explain how HIV causes emphysema and chronic obstructive pulmonary disease (COPD).

In a study published in [Communications Biology](#), the team found that an HIV protein, known as Tat, essentially disrupts the lungs' internal molecular "clock" — the timekeeping system that regulates daily lung function and, importantly, plays a key role in immune response. This unleashes a cascade of chronic inflammation that damages airway tissue, setting the stage for disease.

"This is the first study to show HIV is interfering with this important system," said Hoshang Unwalla, lead study author and professor at the [FIU Herbert Wertheim College of Medicine](#). "It opens the door for new ways to potentially slow or prevent disease development."

Unwalla has spent decades studying HIV's effects on the lungs. His lab was among the first to show that the virus can infect airway cells in the lungs and form reservoirs — pockets of HIV-infected cells that hide out and are not eradicated by antiretroviral therapies.

"When there are HIV reservoirs, the HIV-infected cells are still making these harmful proteins," Unwalla explained. "Tat, in particular, is the most damaging."

In this study, Unwalla and his team led a series of experiments using lung samples from HIV patients, as well as lab-grown lung cells and a mouse model engineered to produce Tat exclusively in the lungs.

They discovered Tat increased levels of a small regulatory molecule. This had a sort of domino effect: The molecule shut down production of a protein, SIRT1, essential for keeping the lungs'

clock running. The result: More inflammatory molecules.

In the mouse model, early signs of emphysema started to appear at around four months of age, the equivalent to early adulthood (age 25) in humans.

Unwalla's research team also focused on ways to reset the clock.

"When we treated the cells with the SIRT1 activator, we were able to reduce inflammatory molecules," said Kingshuk Panda, a PhD candidate in Unwalla's lab. "This suggests that if we can target this pathway, we may be able to improve outcomes."

These findings could have broader implications beyond the lungs. Because trillions of clock genes are present throughout the body, Unwalla says Tat could be dysregulating clocks in other organs as well.

Today, Unwalla's lab is focused on stopping HIV at its source. They developed a patented CRISPR-based gene-editing approach that works like molecular scissors to silence HIV reservoirs.

"Using this approach, our goal is to stop infected cells from producing these damaging proteins," Unwalla said. "That way, we may be able to reduce complications like COPD and improve the quality of life for people living with HIV."

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