

# Surprising find helps explain why women get chronic chlamydia infections

June 23 2011

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(Medical Xpress) -- Researchers at Duke University Medical Center used mice to learn why genital Chlamydia infection remains chronic in women. The findings have important implications for developing strategies to treat Chlamydia and perhaps other chronic infections in the future.

If you expose mice to the human form of the [bacterial pathogen Chlamydia](#), the mice don't develop a chronic infection – they don't develop the disease that humans get.

The researchers removed the robust inborn response mice have against the pathogen and thought they might get the chronic infection, but the mice still were able to clear the infection, unlike humans.

“Our new data, combined with the published literature suggests, that Chlamydia trachomatis co-opts an inborn immune mechanism in humans to cause a self-limiting but sustained infection,” said lead author Jörn Coers, Ph.D.

The study was published in *PLoS Pathogens* on June 23.

“Using a mouse model, we show that in the absence of a robust inborn [immune response](#), Chlamydia is cleared by an unusually pronounced adaptive immune response, that is created in the presence of this pathogen,” said Jörn Coers, Ph.D., Assistant Professor in the Duke Department of Molecular Genetics and Microbiology.

The finding is important as *Chlamydia trachomatis* is the most common sexually transmitted bacterium, and it causes chronic genital infections in women, a common cause of pelvic inflammatory disease and infertility in women.

Coers and his team found that if they removed the innate immune response artificially, this situation worked in favor of the host. “Ultimately the mouse is in an even better position to clear the infection than a normal mouse would be,” Coers said. “That is the major finding of the paper – that the pathogen requires an early immune response to limit a later immune response.”

“*Chlamydia* are okay with getting a beating early on, as long as they can stick it out in the long run,” Coers said. “We think other bacteria, like *Mycobacterium tuberculosis* or *Salmonella typhi*, might also be present in this chronic pattern, surviving a first punch, and then emerging again at intervals to grow throughout the host’s lifetime.”

However, the early immune responses of mice and humans are mechanistically quite distinct. While the mouse immune response kills *Chlamydia*, the human response only puts it to sleep. “While dormant, *Chlamydia* doesn’t trigger a strong secondary immune response and it is probably for that reason that *Chlamydia* can establish chronic infections in humans,” Coers said

Coers said that *Chlamydia trachomatis* co-evolved with humans to be susceptible to an early innate immune response found in humans but not in mice. “For us to understand how exactly *Chlamydia* takes advantage of the human immune response, how *Chlamydia* enters dormancy and causes [chronic infections](#), we need to study mice that artificially launch the early immune response of humans instead of their own. This is something we’re working on right now.”

Because dormant bacteria are effectively hidden from an antibiotic treatment, a solution might be to drive them out of dormancy and simultaneously treat them with antibiotics, curing the chronic infection, Coers said.

Provided by Duke University

Citation: Surprising find helps explain why women get chronic chlamydia infections (2011, June 23) retrieved 25 April 2024 from <https://medicalxpress.com/news/2011-06-women-chronic-chlamydia-infections.html>

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