MedicalXpress

Novel coronavirus well-adapted to humans, susceptible to immunotherapy

February 19 2013

The new coronavirus that has emerged in the Middle East is well-adapted to infecting humans but could potentially be treated with immunotherapy, according to a study to be published on February 19 in *mBio*, the online open-access journal of the American Society for Microbiology. The study indicates that the virus HCoV-EMC can penetrate the lining of the passageways in the lung and evade the innate immune system as easily as a cold virus can, signs that HCoV-EMC is well-equipped for infecting human cells. The study also reveals that the virus is susceptible to treatment with interferons, components of the immune system that have been used successfully to treat other viral diseases, opening a possible mode of treatment in the event of a large-scale outbreak.

"Surprisingly, this coronavirus grows very efficiently on human epithelial cells," says co-author Volker Thiel of The Institute of Immunobiology at Kantonal Hospital in St. Gallen, Switzerland. Thiel says these new data indicate that although HCoV-EMC may have jumped from animals to humans very recently, it is just as well adapted to infecting the human respiratory tract as other, more familiar human coronaviruses, including the SARS virus and the common cold virus, HCoV-229E.

HCoV-EMC first came to light in June when it was isolated from a man in Saudi Arabia who died from a severe respiratory infection and kidney failure. Since that time, public health officials have identified an additional 10 infected persons, nine of whom had traveled in the Middle

East and one who had recent contact with an infected person. The emergence of HCoV-EMC, which is related to the SARS virus, has raised concern that it may eventually lead to a pandemic much like the SARS pandemic of 2002-2003, which is estimated to have sickened over 8,000 people and killed 774 worldwide.

For the *mBio* study, Thiel and his colleagues tested how well HCoV-EMC could infect and multiply in the entryways to the human lung using cultured bronchial cells manipulated to mimic the airway lining. The lining of the lung, or epithelium, represents an important first barrier against respiratory viruses, but they apparently don't put up a big fight against HCoV-EMC, says Thiel. He and his colleagues found that human airway epithelial cells are highly susceptible to HCoV-EMC infection and that the virus is able to multiply at a faster initial rate than the SARS virus.

"The other thing we found is that the viruses [HCoV-EMC, SARS, and the common cold virus] are all similar in terms of host responses: they don't provoke a huge innate immune response," Thiel says. This is an indication that HCoV-EMC is already well adapted to the human host and that the virus uses that same strategy other coronaviruses use for evading the host's non-specific immune mechanisms.

The authors asked themselves whether boosting this weak immune response might diminish the virus' ability to infect airway epithelial cells. They found that pre-treating the cells with lambda-type interferons, proteins that are released by host cells in response to infection and enable communication between cells to mount an immune response, significantly reduced the number of infected cells. This is encouraging from a treatment standpoint, note the authors, since interferons have also shown a good deal of promise for treating SARS and another viral illness, Hepatitis C.

Thiel and co-author Ronald Dijkman emphasize that their work with HCoV-EMC would not have been possible without the efforts of many different research groups from Switzerland, Germany, The Netherlands, and Denmark.

Ongoing collaboration is crucial, they say. Future research to head off outbreaks of HCoV-EMC and other emerging diseases requires cooperation and trust among scientists and health agencies, a goal that is not always achieved. The future of this virus is uncertain, Thiel points out, but access to samples from a wider range of patients and epidemiological work could answer some fundamental questions, including where the virus is coming from and what the true prevalence of the virus is.

"We don't know whether the cases we observe are the tip of the iceberg," says Thiel. "Or whether many more people are infected without showing severe symptoms."

Provided by American Society for Microbiology