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Study indicates different treatment may be needed for infection-related breathing problems

DALLAS – Jan. 31, 2007 – New research suggests that different treatments may be needed for chronic asthma, depending on whether it results from allergies or lung infections.

Previous studies have shown that certain lung infections such as *Mycoplasma pneumoniae* can linger on and contribute to a person later experiencing symptoms of asthma.

Researchers have now identified a particular gene that influences how severe a *M. pneumoniae* infection may be, which in turn suggests that a different strategy might be needed for treating asthma resulting from this and similar lung infections rather than allergies.

“What this shows is that infectious asthma might have a different mechanism than allergic asthma. Most people think asthma is asthma, but it may be multifaceted,” said Dr. Robert Hardy, an infectious disease specialist at UT Southwestern.

That’s an important implication because the latest statistics show that asthma is on the rise. According to the U.S. Centers for Disease Control, more than 20 million Americans currently have asthma and another 10 million have been diagnosed with asthma at some point in their life. Roughly 6.5 million American children, or nearly 9 percent of the nation’s pre-adult population, have asthma, figures released in December show.

Dr. Hardy, an assistant professor of internal medicine and pediatrics, has been using mice to study how certain pneumonia bacteria contribute to chronic asthma and, in this latest study, identified how a particular gene may contribute to more severe lung infection. The research appears in the January edition of *Infection and Immunity*.

Pneumonia is a lung infection typically characterized by breathing difficulties and spread by coughing and sneezing. Symptoms often include headache, fever, chills, coughs, chest pains, sore throat and nausea. Dr. Hardy’s research involves pneumonia caused by the bacterium *M. pneumoniae*, commonly called walking pneumonia, a typically less severe form of the disease that accounts for 20 percent to 30 percent of community-acquired pneumonia.

To investigate the mechanism by which *M. pneumoniae* causes lung disease and respiratory difficulties, the UT Southwestern researchers inoculated two different types of mice with this

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bacterium. The study contrasted the reaction of one normal group of mice with another group lacking a particular gene called *IL-12*, which is involved in immune response. The mice engineered without the gene showed significantly less lung inflammation than the mice that naturally had the gene, with some indicators showing seven times less inflammation.

“*M. pneumoniae* might be more of a cofactor in developing chronic asthma than a direct cause, similar to how high cholesterol or diabetes makes people more vulnerable to heart attacks,” Dr. Hardy said, pointing to a number of previous studies. “It’s probably not the only thing, but it’s one of them. In some people it might incite asthma or it might exacerbate it.”

Because the *M. pneumoniae* bacterium is difficult to kill and often remains in the lungs even after antibiotic treatment and the symptoms fade, Dr. Hardy said, it is important to find better treatments to prevent it from lingering.

Other UT Southwestern researchers involved in the study were Dr. George McCracken, professor and chief of pediatric infectious diseases; Dr. Ana Gomez, assistant professor of pathology; Drs. Christine Salvatore, Asuncion Mejias and Cynthia Somers, pediatrics postdoctoral trainees; Kathy Katz-Gaynor, pediatrics research associate; and Monica Fonseca-Aten and Susanna Chavez-Bueno, former pediatric postdoctoral trainees.

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