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Small protein plays big role in asthma severity

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A new culprit has been identified that likely plays a big role in the severity of asthma—a small protein chemokine called CCL26. These findings were published in the *Journal of Leukocyte Biology* and represent the first demonstration that CCL26 is a potent regulator of the migration of asthmatic eosinophils, commonly observed in asthmatic airways. Results from this discovery may lead to new drug targets for the treatment of asthma.

"We hope that these studies will help to develop a new treatment that would specifically abrogate bronchial inflammation and provide a specific, efficacious and well-tolerated alternative to the current therapy," said Michel Laviolette, M.D., a researcher involved in the work from the Centre de Recherche de l'Institut Universitaire de Cardiologie et de Pneumologie de Québec, Faculté de Médecine, Université Laval, Québec, Canada.

Specifically, data from the report suggest that the chemokine CCL26 plays a crucial role in asthma pathogenesis and its severity by supporting the recruitment of eosinophils early in the development of the disease, and possibly later in severe asthma associated with persisting lung eosinophilia. To make this discovery, scientists used blood from healthy and asthmatic subjects to isolate eosinophils and measure their migration response to CCL26 in vitro. Researchers also assessed their response to other chemokines, CCL11 and CCL24, and showed that only CCL26 induced an amplified eosinophil migration of asthmatic eosinophils compared to healthy cells. Interestingly, this additional migration

occurred after a 6-hour incubation and, in contrast to the migration induced by the other chemokines, was not eradicated by blocking the chemokine receptor CCR3 shared by these three chemokines.

"The control of eosinophils is central to asthmatic diseases and the underlying mechanisms are prime targets for treatments for the debilitating and sometimes life-threatening symptoms of asthma and allergy," said John Wherry, Ph.D., Deputy Editor of the *Journal of Leukocyte Biology*. "This report shows that CCL26 could be a novel drug target to regulate eosinophils in these diseases."

More information: Véronique Provost, Marie-Chantal Larose, Anick Langlois, Marek Rola-Pleszczynski, Nicolas Flamand, and Michel Laviolette. CCL26/eotaxin-3 is more effective to induce the migration of eosinophils of asthmatics than CCL11/eotaxin-1 and CCL24/eotaxin-2 . J Leukoc Biol August 2013 94:213-222; doi:10.1189/jlb.0212074