Immune system kick-started in moist nasal lining in sinusitis, asthma and colds

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Scientists at Johns Hopkins have outlined a new path for potential therapies to combat inflammation associated with sinusitis and asthma based on a new understanding of the body's earliest immune response in the nose and sinus cavities.

Researchers say their findings, to be published in the May edition of the *Journal of Allergy and Clinical Immunology*, are the first evidence describing how viral agents, such as the rhinovirus responsible for the common cold, can kick start the body's mobilization of immune white blood cells in the moist, mucous membrane lining of the nasal passages.

While such responses are key to maintain health in the face of pathogens, they can also become a source of illness due to resulting inflammation. This can lead to potentially life-long problems, including tissue swelling, nasal polyp formation, sneezing, stuffy and runny nose, sore throat, cough, headache, chills, fever and difficulty breathing.

Thus, blocking these reactions, the researchers point out, could interrupt the cascade of feel-awful symptoms that ensue.

The focus of the study is B7-related proteins, called B7 homologs, which trip white blood cell response in a pathogen attack.

Using purified cold virus and its genetic material as bait, the scientists found that production of two B7 homologs spiked in response: Levels of B7-H1 jumped almost ninefold and levels of B7-DC tripled.

Until now, says senior study investigator Jean Kim, M.D., Ph.D., viruses were known to reside in and infect the physical epithelium, invading surface membrane cells and revving up the immune system's main blood cell defenses, "but no one knew the major steps involved in or precisely how this immune response was triggered."

"The inside surface of our nose and sinuses is much more than a protective cover, and we have good scientific evidence to show that epithelial cells on these mucosal membranes are very powerful mediators - middlemen - in diseases that result in inflammation," adds Kim. An assistant professor at the Johns Hopkins University School of Medicine and an expert in the molecular origins of inflammation, Kim is also an authority on nasal and sinus infections.

Moreover, Kim notes, study results demonstrate how the body's immune system is interconnected, where one key part, the physical lining that filters out and captures invading viruses and environmental allergens, can trigger the other key part, which leads to targeted white blood cell action.

"Now that we have a better understanding of the immune pathway, we can start to develop therapies that could potentially block the triggering reactions for sinusitis and asthma, which are both made worse when people are infected with the common cold virus," she says.

Sinusitis is the most common respiratory complaint in the United States. The condition is often linked with asthma, which affects more than 30 million, including 9 million children. Each year, 62 million Americans catch a cold.

The study also explains a common failure in current therapy.

According to Kim, nasal and oral steroids are frequently prescribed for many of the 15 percent of the American adult population who suffer

from sinusitis, nasal polyps or asthma. Steroids complement drugs taken for symptomatic relief, such as decongestants and pain relievers.

But corticosteroid drugs, she says, do not work for everyone and their effectiveness often wanes over time.

This may be related to the B7 homolog triggers in the mucous membranes, Kim says, as study results showed that corticosteroid therapy does not fully shut down or prevent their overproduction.

In the study's first set of experiments, researchers found that levels of two of five key proteins tested, B7-H1 and B7-DC, rose sharply after samples of nasal cell concentrate were exposed to genetic material from cold viruses. Spiked production was detected using antibodies chemically tagged to glow when bonded to a specific B7 homolog.

However, when researchers pretreated the cell scrapings with a well-known anti-inflammatory corticosteroid, called flucticasone propionate, the drug failed to stop overproduction of either B7-H1 or B7-DC.

In the final set of study experiments, six adult volunteers were infected with the cold virus and monitored for variations in their immune response during infection, which typically lasts a week to 10 days.

Analysis of daily scrapings of surface cells lining the nose showed that production of B7-H1 and B7-DC peaked on the second and third days, when cold symptoms were also at their worst. These protein levels, as a measure of severity of the immune response, dropped quickly afterwards, and at the same time as scores of symptom severity went down. It was this evidence that verified the triggering connection between the cold virus and the immune white cell response inside the nose and sinuses, says Kim.

Kim says that researchers' next steps are to analyze the biological control mechanisms for producing the B7 homologs in the nasal lining, and to map out any chemical interactions that result, to look for ways of breaking the cycle of inflammation involved in sinusitis, asthma and colds.

Source: Johns Hopkins Medical Institutions

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