Professor discovers genetic basis for hair loss

October 15 2010, by John H. Tucker

"Physician, heal thyself." That oft-quoted proverb describes the ground-breaking effort by Columbia professor Angela Christiano to discover the cause of the second most common form of hair loss after male-pattern baldness.

In 1996, Christiano's hair suddenly began falling out, leaving her scalp riddled with bald patches. The condition was diagnosed as alopecia areata, which affects about 2 percent of the population overall including more than 5.3 million people in the United States.

As she struggled with the disease, Christiano, who is affiliated with the Dermatology and the Genetics and Development departments at Columbia University Medical Center, decided to study hair loss.

"I looked at the literature and realized how little was known about genes that control hair growth," she says. "I couldn't believe that this disease I was living with was so much in the dark."

Earlier this year, an international team of researchers led by Christiano found that the immune genes carried by alopecia areata patients are nearly identical to those carried by patients suffering from rheumatoid arthritis, Type 1 diabetes and celiac disease. The study was published in the July 1 issue of *Nature*.

Christiano, the Richard and Mildred Rhodebeck Professor of Dermatology, is now hopeful that the drugs used to treat those

diseases—particularly rheumatoid arthritis—might also be used to treat alopecia areata. The team expects to begin clinical trials next year.

Unlike male-pattern baldness, alopecia areata has a sudden onset and is often marked by hair loss from the entire body, including the eyebrows, eyelashes and legs. (The word “alopecia ” comes from the Greek word for “fox,” an animal that frequently loses its hair.) The disease is classified as an autoimmune disorder, which means it’s caused when the body’s immune system attacks its own organs—in this case, the hair follicles.

The researchers discovered that a gene called ULBP3 acts as a homing beacon for killer immune cells. ULBP3 is turned off in normal hair follicles but turned on in alopecia areata follicles. In its activated state, ULBP3 attracts killer immune cells with a special receptor called NKG2D, which triggers an autoimmune attack.

Christiano and colleague Raphael Clynes, an assistant professor of medicine and microbiology at the medical center, are now trying to find a way to moderate the response.

“The easy thing to do would be to block the NKG2D receptor using certain antibody drugs that are currently being developed,” says Clynes. “The other tactic is to use a soluble receptor that ... blocks the interaction between the killer cells and the ULBP3’s ‘kill-me’ signal.”

In addition to hair loss, Christiano studies hypertrichosis, or excess hair growth. She earned her Ph.D. in genetics at Rutgers University and was a post-doctoral fellow in dermatology at Jefferson Medical Center in Philadelphia, where she performed research in epidermolysis bullosa, a potentially fatal blister disorder.

Hair loss is among the most emotionally devastating skin diseases, as

measured by impact on quality of life, says Christiano. For now, she says, alopecia patients should take comfort in knowing the disease has a genetic cause, which means that help may be on the way.

Today, Christiano has a full head of dark, wavy hair. After two years of treatment with steroids, the condition reversed itself. But she remains emotionally connected to the disease.

She recently spoke at a National Alopecia Areata Foundation patient conference in Indianapolis. After delivering a lecture to several hundred alopecia patients, many people burst into tears—and she did too.

“They finally had genes to talk about,” she says. “They felt empowered.”

Provided by Columbia University

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