

# Why are so many people getting a tick-borne meat allergy?

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It is early morning in early summer, and I am tracing my way through the woods of central North Carolina, steering cautiously around S-curves and braking hard when what looks like a small rise turns into a narrow

bridge. I am on my way to meet Tami McGraw, who lives with her husband and the youngest of their kids in a sprawling development of old trees and wide lawns just south of Chapel Hill. Before I reach her, McGraw emails. She wants to feed me when I get there:

"Would you like to try emu?" she asks. "Or perhaps some duck?"

These are not normal breakfast offerings. But for years, nothing about McGraw's life has been normal. She cannot eat beef or pork, or drink milk or eat cheese or snack on a gelatine-containing dessert without feeling her throat close and her blood pressure drop. Wearing a wool sweater raises hives on her skin; inhaling the fumes of bacon sizzling on a stove will knock her to the ground. Everywhere she goes, she carries an array of tablets that can beat back an allergy attack, and an auto-injecting EpiPen that can jolt her system out of anaphylactic shock.

McGraw is allergic to the meat of mammals and everything else that comes from them: dairy products, wool and fibre, gelatine from their hooves, char from their bones. This syndrome affects some thousands of people in the USA and an uncertain but likely larger number worldwide, and after a decade of research, scientists have begun to understand what causes it. It is created by the bite of a tick, picked up on a hike or brushed against in a garden, or hitchhiking on the fur of a pet that was roaming outside.

The illness, which generally goes by the name 'alpha-gal allergy' after the component of meat that triggers it, is a trial that McGraw and her family are still learning to cope with. In much the same way, medicine is grappling with it too. Allergies occur when our immune systems perceive something that ought to be familiar as foreign. For scientists, alpha-gal is forcing a remapping of basic tenets of immunology: how allergies occur, how they are triggered, whom they put in danger and when.

For those affected, alpha-gal is transforming the landscapes they live in, turning the reliable comforts of home – the plants in their gardens, the food on their plates – into an uncertain terrain of risk.

In 1987, Dr. Sheryl van Nunen was confronted with a puzzle. She was the head of the allergy department at a regional hospital in the suburbs of Sydney, Australia, and had a reputation among her colleagues for sorting out mysterious episodes of anaphylaxis. This time, a man had been sent to see her who kept waking up, in the middle of the night, in the grip of some profound reaction.

Van Nunen knew at once that this was out of the ordinary, since most [allergic reactions](#) happen quickly after exposure instead of hours later. She also knew that only a few allergens affect people after they have gone to bed. (Latex, for instance – someone sensitive to it who has sex using a latex condom might fall asleep and wake up in the midst of an allergy attack.) She checked the man for the obvious irritants and, when those tests came up negative, took a thorough look at his medical history and did a skin test for everything he had eaten and touched in the hours before bedtime. The only potential allergen that returned a positive result was meat.

This was weird (and dismaying, in barbecue-loving Australia). But it was the only such case Van Nunen had ever seen. She coached the patient on how to avoid the meals that seemed to be triggering his reactions, put it down mentally to the unpredictability of the human immune system, and moved on.

Then a few more such patients came her way. There were six additional ones across the 1990s; by 2003, she had seen at least 70, all with the same problem, all apparently affected by meat they had eaten a few hours before. Groping for an explanation, she lengthened the list of questions she asked, quizzing the patients about whether they or their

families had ever reacted to anything else: detergents, fabrics, plants in their gardens, insects on the plants.

"And invariably, these people would say to me: 'I haven't been bitten by a bee or a wasp, but I've had lots of [tick bites](#),'" Van Nunen recalls.

In her memory, Tami McGraw's symptoms began after 2010. That was the year she and her husband Tom, a retired surgeon, spied a housing bargain in North Carolina, a development next to a nature reserve whose builder had priced the big houses to sell. The leafy spread of streams and woodland pockets was everything she wanted in a home. She didn't realise that it offered everything that deer and birds and rodents, the main hosts of ticks, want as well.

She remembers one tick that attached to her scalp, raising such a welt the spot was red for months afterwards, and a swarm of baby ticks that climbed her legs and had to be scrubbed off in a hot bath laced with bleach. Unpredictably, at odd intervals, she began to get dizzy and sick.

"I'd have unexplained allergic reactions, and I'd break out in hives and my blood pressure would go crazy," she told me. The necklines of all her T-shirts were stretched, because she tugged at them to relieve the feeling she couldn't take a deep breath. She trekked to an array of doctors who diagnosed her with asthma or early menopause or a tumour on her pituitary gland. They prescribed antibiotics and inhalers and steroids. They sent her for MRI scans, pulmonary function tests, echocardiograms of her heart. Nothing yielded a result.

Looking back, she realises she missed clues as to the source of her problem. She always seemed to need to use an asthma inhaler on Wednesdays – the day she spent hours in her car, delivering steaming-hot dinners for Meals on Wheels. She would feel short of breath, and need to visit an urgent-care clinic, on Saturdays – which always started,



in her household, with a big breakfast of eggs and sausages.

Then a close friend had a scary episode, going for a run, arriving home and passing out on the hot concrete of her driveway. Once she was recovered, McGraw quizzed her. Her friend said: "They thought I got stung by a bee while I was running. But now they think maybe I have a red-meat allergy."

McGraw remembers her first reaction was: That's crazy. Her second was: Maybe I have that too.

She Googled, and then she asked her doctor to order a little-known blood test that would show if her immune system was reacting to a component of mammal meat. The test result was so strongly positive, her doctor called her at home to tell her to step away from the stove.

That should have been the end of her problems. Instead it launched her on an odyssey of discovering just how much mammal material is present in everyday life. One time, she took capsules of liquid painkiller and woke up in the middle of the night, itching and covered in hives provoked by the drug's gelatine covering.

When she bought an unfamiliar lip balm, the lanolin in it made her mouth peel and blister. She planned to spend an afternoon gardening, spreading fertiliser and planting flowers, but passed out on the grass and had to be revived with an EpiPen. She had reacted to manure and bone meal that were enrichments in bagged compost she had bought.

She struggled with the attacks' unpredictability, and even more with the impact on her family. "I think I'm getting better, and then I realise I'm not," she says. "I'm more knowledgeable about what I can and can't do."

The discovery of new diseases often follows a pattern. Scattered patients

realise they are experiencing strange symptoms. They find each other, face to face in a neighbourhood or across the world on the internet. They bring their experience to medicine, and medicine is sceptical. And then, after some period of pain and recalcitrance, medicine admits that, in fact, the patients were right.

That is the story of the discovery of CFS/ME and Lyme disease, among others. But it is not the story of alpha-gal allergy. An odd set of coincidences brought the bizarre illness to the attention of researchers almost as soon as it occurred.

The story begins with a cancer drug called cetuximab, which came onto the market in 2004. Cetuximab is a protein grown in cells taken from mice. For any new drug, there are likely to be a few people that react badly to it, and that was true for cetuximab. [In its earliest trials](#), one or two of every 100 cancer patients who got it infused into their veins had a hypersensitivity reaction: their blood pressure dropped and they had difficulty breathing.

That 1–2 per cent stayed consistent as cetuximab was given to larger and larger groups. And then there was an aberration. In clinics in North Carolina and Tennessee, 25 of 88 recipients were hypersensitive to the drug, with some so sick they needed emergency shots of epinephrine and hospitalisation. At about the same time, a patient who was receiving a first dose of cetuximab in a cancer clinic in Bentonville, Arkansas, collapsed and died.

The manufacturers, ImClone and Bristol-Myers Squibb, checked every obvious thing about the trial: the drug's ingredients, the cleanliness of the manufacturing plants, even the practices at the medical centres where cetuximab had been administered. Nothing stood out. The most that researchers could guess at the time was that the unlucky recipients might have some kind of mouse allergy.

Then the first coincidence occurred: a nurse whose husband worked at the Bentonville clinic mentioned the death to Dr. Tina Hatley, an immunologist in private practice in Bentonville. Hatley had recently finished postgraduate training at the University of Virginia's allergy centre, and she mentioned the death to her former supervisor, Dr. Thomas Platts-Mills.

The bad responses to the drug looked like allergic reactions, and they were common enough – and far enough from the manufacturer's expectations – to be an intriguing research opportunity.

Platts-Mills pulled together a team, looping in Hatley and several current research fellows as well. Fairly quickly, they discovered the source of the problem. People were reacting to the drug because they had a pre-existing sensitivity, indicated by a high level of antibodies (called immunoglobulin E, or IgE for short) to a sugar that is present in the muscles of most mammals, though not in humans or other primates. The name of the sugar was galactose-alpha-1,3-galactose, known for short as alpha-gal.

Alpha-gal is familiar to many scientists because it is responsible for an enduring disappointment: its ability to trigger intense immune reactions is the reason that organs taken from animals have never successfully been transplanted into people. The puzzle was why the drug recipients were reacting to it. To have an allergic reaction, someone needs to have been primed with a prior exposure to a substance – but the trial recipients who reacted badly were all on their first dose of cetuximab.

Team members scrutinised the patients and their families for anything that could explain the problem. The reactions appeared regional – patients in Arkansas and North Carolina and Tennessee experienced the hypersensitivity, but ones in Boston and northern California did not. They investigated parasites, moulds and diseases that occur only in

pockets of the USA.

Then Dr. Christine Chung, a Nashville researcher recruited to the team, stumbled on an intriguing clue. Almost one in five of the patients enrolled at a cancer clinic at her hospital had high levels of IgE to alpha-gal. But when she checked those patients' near neighbours, treating them as a control group – that is, people who lived their lives in the same way, but did not have cancer and had no reason to have received the drug – almost one in five had antibodies to alpha-gal as well.

Almost a decade later, that correlation still makes Platts-Mills chuckle. The alpha-gal reaction "had nothing to do with cancer," he says. "It had everything to do with rural Tennessee."

The question then became: what in rural Tennessee could trigger a reaction like this? The answer arose from a second coincidence. Dr. Jacob Hosen, a researcher in Platts-Mills's lab, stumbled across a map drawn by the Centers for Disease Control and Prevention (CDC) showing the prevalence of an infection called Rocky Mountain spotted fever. It exactly overlapped the hot spots where the cetuximab reactions had occurred.

Rocky Mountain spotted fever is transmitted by the bite of a tick: *Amblyomma americanum*, one of the most common ticks in southeastern USA. It's known as the lone star tick for a blotch of white on the back of the female's body.

The researchers wondered – if the mystery reactions shared a footprint with a disease, and ticks caused the disease, could ticks be linked to the reactions too?

It was an intriguing hypothesis, and was reinforced by a new set of patients who came trickling into Platts-Mills's clinic at about the same



time. They were all adults, and that was odd to start with, because allergies tend to show up in childhood. They had never had an allergic reaction before, but now they were experiencing allergy symptoms: swelling, hives and in the worst cases anaphylactic shock. They too had high levels of IgE antibodies to alpha-gal.

None of them, though, were cancer patients. They told the physicians that they had no proof of what was causing their reactions – but more than a few of them sensed it had something to do with eating meat.

Dr. Scott Commins, another postgraduate fellow in Platts-Mills's group, took it upon himself to phone every new patient to ask whether they'd ever suffered a tick bite. "I think 94.6 per cent of them answered affirmatively," he says. "And the other few per cent would say, 'You know, I'm outdoors all the time. I can't remember an actual tick that was attached, but I know I'd get bites.'"

Meat from mammals inevitably contains alpha-gal – so in already sensitised individuals, eating meat might constitute a second exposure, in the same way infusing cetuximab had been.

If tick bites had sensitised them, then the alpha-gal reaction might be a food allergy as well as a drug reaction. But the connection was speculative, and cementing cause and effect would take one final, extraordinary coincidence.

As it happens, Platts-Mills likes to hike. One weekend he took off across the central Virginia hills, tramping through grassy underbrush. He came home five hours later, peeled off his boots and socks, and discovered his legs and feet were speckled with tiny dots. They looked like ground pepper, but they were dug into his skin – he had to use a dull knife to scrape them off – and they itched something fierce. He saved a few, and sent them to an entomologist. They were the larval form of lone star

ticks.

This, he realised, was an opportunity. As soon as the work week started, he had his lab team draw his blood and check his IgE levels. They were low to start with, and then week by week began to climb. Platts-Mills is English – his father was a Member of Parliament – and in the midst of having his IgE tracked, he went to an event at the Royal Society of Medicine in London. "And at that point," he says cheerfully, "I ate two lamb chops and drank two glasses of wine."

In the middle of the night, he woke up covered in hives.

The lone star tick doesn't receive much attention in the USA. It's the black-legged tick, *Ixodes scapularis*, that has the dubious honour of being the most well-known, as it's the carrier of Lyme disease, which causes an estimated 300,000 cases of illness in the USA each year.

The lone star tick doesn't transmit Lyme disease, but is the vector for other serious illnesses, including Q fever, ehrlichiosis, Heartland virus, Bourbon virus and tularaemia, an infection so serious that the US government classifies the bacteria that cause it as a potential agent of bioterrorism.

While Lyme clusters in the north-east and the northern Midwest, the diseases carried by *Amblyomma* stretch from the coast of Maine to the tip of Florida, the Atlantic to the middle of Texas, and the southern shores of the Great Lakes all the way to the Mexican border.

And that range appears to be expanding. "The northern edge of where these ticks are abundant is moving," says Dr. Rick Ostfeld, a disease ecologist at the Cary Institute of Ecosystem Studies, north of New York City. "It is now well-established further north, into Michigan, Pennsylvania, New York and well up into New England."

"Climate change is likely playing a role in the northward expansion," Ostfeld adds, but acknowledges that we don't know what else could also be contributing.

It's a universal complaint among tick scientists that we don't know as much about ticks as we should. Tick-transmitted illnesses are more common in the USA than mosquito-borne ones – according to the CDC's most recent accounting, in 2017 tickborne diseases were 2.6 times more common than when the agency began counting in 2004 – yet it's mosquitoes that receive the most public health attention and funding, from national surveillance programmes to local mosquito-control campaigns. (In fact, the CDC was founded in 1942 because of mosquito-borne disease; its original title was the Office of Malaria Control in War Areas.)

What is known about where ticks live, what they feed on, and how they are affected by changes in land use and climate has mostly been assembled out of the findings of scientists fighting for scarce research funding.

It's impossible to talk to physicians encountering alpha-gal cases without hearing that something has changed to make the tick that transmits it more common – even though they don't know what that something might be.

The lone star tick is a sturdy, stealthy predator. It isn't picky about conditions – it tolerates the damp of Atlantic beaches, and its western expansion only stopped when it ran up against the Texas desert – and it's content to feed from dozens of animals, from mice all the way up the tree of life.

It loves birds, which might have helped it move north so rapidly, and it has a special lust for the white-tailed deer that have colonised American

suburbs. And, unlike most ticks, it bites humans in all three stages of its lifecycle: as an adult, as a nymph and as the poppy seed-sized larvae that attacked Platt-Mills, which linger on grass stalks in clusters and spring off hundreds at a time.

Ticks detect scent with organs embedded in their first pair of legs, and what they're sniffing for is carbon dioxide, the exhaled breath of an animal full of warm oxygenated blood. When lone star ticks catch wind of it, they take off. "The Lyme disease tick is a slow tick," says Dr. William Nicholson, a microbiologist at the CDC. "Amblyomma will run to you."

There has been so little research into alpha-gal allergy that scientists can't agree on exactly what stage of the bite starts victims' sensitisation. It is possible that a fragment of a previous blood meal, from a mouse, bird or deer, lingers in a tick's guts and works its way up through its mouth and into its human victim. It's also possible that some still-unidentified compound in tick saliva is chemically close enough to alpha-gal to produce the same effect.

One aspect of its epidemiology is becoming clear, though. The allergy isn't only caused by the lone star tick.

In Australia, Van Nunen (who is now a clinical associate professor at the University of Sydney School of Medicine) couldn't understand how her patients' tick bites solved the mystery of their meat allergy. But she could see something else. The beaches that fringe the coast north and south of Sydney are rife with ticks. If bites from them were putting people at risk of a profound allergy, she felt compelled to get the word out.

In 2007, Van Nunen wrote up a description of 25 meat-allergic patients whose reactions she had confirmed with a skin-prick test. All but two

had had severe skin reactions to a tick bite; more than half had suffered severe anaphylaxis. That abstract formed the basis of a talk she gave later that year to an Australian medical association, which was then indexed – but not published in full – in an Australian medical journal. It took until 2009 for the Virginia group to catch up to it, after they had already published their first alert.

That was unfortunate, because the crucial detail in Van Nunen's research wasn't just that her cases were earlier than the first round of American ones. It was that they were caused by bites from a different tick: *Ixodes holocyclus*, called the paralysis tick. Alpha-gal allergy was not just an odd occurrence in one part of the USA. It had occurred in the opposite hemisphere, making it literally a global problem.

And so it has proved. Alpha-gal reactions linked to tick bites have now been found in the UK, France, Spain, Germany, Italy, Switzerland, Japan, South Korea, Sweden, Norway, Panama, Brazil, Côte d'Ivoire and South Africa. These cases trace back to at least six additional tick species. (An online map on which patients list themselves includes over a dozen more countries.)

Wherever ticks bite people – everywhere other than the Arctic and Antarctic – alpha-gal allergy has been recorded. In Belgium, patients reacted badly to a drug produced in rabbit cells. In the Italian Alps, men who went hunting in the forests were more at risk than women who stayed in their village. In Germany, the most reactive food was a traditional delicacy, pork kidneys. In Sweden, it was moose.

Van Nunen herself has now seen more than 1,200 patients. "The next busiest clinic, about 350," she says. Those cases have all occurred in two decades, less than the span of a single human generation. As in America, the surge leaves Van Nunen mystified as to what the cause might be. She reasons that the rise cannot be due to something in her patients; neither



genetic nor epigenetic change could occur so quickly.

"It has to be environmental," she says.

It's a sunny early morning at the University of North Carolina Medical Center in Chapel Hill. Commins, who moved here in 2016 to become an associate professor, has 11 patients to see before the end of the day. Seven of them have alpha-gal allergy.

Laura Stirling, 51, is fretting over a list of questions. She does not live nearby; she flew down from Maryland, drawn by Commins's reputation. In 2016, she found a fat lone star tick attached to her, and afterwards had fierce indigestion whenever she ate or smelled pork – a challenge, because her husband likes to tinker with a smoker on weekends. In 2017, she was bitten again, and her symptoms worsened to midnight hives and lightheadedness that sent her to her doctor's office. She immediately cut all meat and dairy from her diet. A year later, she wants to know if she can add anything back.

"Can I eat dairy?" she asks. "Can I cook dairy? Can I eat it if it doesn't have animal rennet in it?" She pauses. "I've been symptom-free, because I don't take risks."

Commins walks her through a protocol he's developed, a method for adding back mammal products one dose at a time. He has a hypothesis that alpha-gal reactions are linked to the fat content of food; that might explain why they take so many hours to occur, because the body processes fat via a slower metabolic pathway than protein or carbs.

He recommends that patients start with a spoonful of grated dry cheese, because its fat content is low, and graduate by slow steps up to full-fat yogurt and milk and then to ice cream. If those foods don't provoke reactions, he suggests tiny doses of lean meat, starting with deli ham.

Stirling lights up at that. "I dream of charcuterie," she sighs.

Because Commins was part of Platt-Mills's earliest research, he has been seeing alpha-gal patients for more than a decade now. He estimates he has treated more than 900 men and women; five new patients arrive every week. He has coached a significant number of them back to eating some mammal products and managing their exposures to the things they can't handle, so their worst experience is hunting for an emergency Benadryl, not being rushed to the ER.

Not every patient can do this. Julie LeSueur, who is 45 and lives in Richmond, Virginia, has been monitored by Platts-Mills for four years. (He is one of several doctors she has seen for the condition, after years of severe stomach issues escalated to repeated attacks of anaphylaxis that put her in hospital. One physician, frustrated she wasn't getting better, told her: "This is all in your head.")

What started as an allergy to meat expanded into reactions to anything with an animal connection, including gelatine in medications and animal products in cosmetics, and then to sensitising her immune system to an array of other irritants, from nuts to mould. She buys vegan soap and shampoo, has prescriptions formulated by a compounding pharmacy, and mostly works from home to avoid unintended exposures. Reluctantly, she cut back a hobby that meant the world to her: fostering animals that have been rescued from abuse.

"I'm at home all the time now," she tells me by phone. "I'm lucky to get off the couch."

Commins and Platts-Mills named alpha-gal allergy a decade ago, and Van Nunen saw her first patient 20 years before that. A lab test for the allergy, the one that Tami McGraw received, has been on the market since 2010. (Platts-Mills and Tina Hatley, now Merritt, share the patent.)

That makes it hard to understand why patients still struggle to be diagnosed and understand the limits of what they can eat or allow themselves to be exposed to. But alpha-gal allergy defies some of the bedrock tenets of immunology.

Food allergies are overwhelmingly caused by proteins, tend to surface in childhood and usually trigger symptoms quickly after a food is consumed. Alpha-gal is a sugar; alpha-gal patients tolerate meat for years before their reactions begin; and alpha-gal reactions take hours to occur. Plus, the range of reactions is far beyond what's normal: not only skin reactions in mild cases and anaphylaxis in the most serious, but piercing stomach pain, abdominal cramps and diarrhoea as well.

But alpha-gal reactions are definitely an allergy, given patients' results on the same skin and IgE tests that immunologists use to determine allergies to other foods. That leads both Van Nunen and Commins to wonder whether the syndrome will help to reshape allergy science, broadening the understanding of what constitutes an allergy response and leading to new concepts of how allergies are triggered.

Merritt, who estimates she has seen more than 500 patients with alpha-gal allergy, has it herself; she has had bad reactions to meat all her life, since being bitten by seed ticks at Girl Scout camp, and was re-sensitised by a lone star tick bite last year. She is sensitive enough to react not only to meat, but to other products derived from mammal tissues – and as she has discovered, they are threaded throughout modern life.

The unrecognised dangers aren't only sweaters and soaps and face creams. Medical products with an animal origin include the clotting drug heparin, derived from pork intestines and cow lung; pancreatic enzymes and thyroid supplements; medicines that include magnesium stearate as an inert filler; vaccines grown in certain cell lines; and other vaccines, and intravenous fluids, that contain gelatine.

"We have enormous difficulty advising people about this," Van Nunen says. "Sometimes you have to sit down for seven hours, write seven emails and have four telephone conversations to be able to say to a 23-year-old woman who's about to travel: 'Yes, you may have this brand of Japanese encephalitis vaccine because they do not use bovine material. The vaccine is made in [cells from] the African green monkey and I have looked up that monkey and it does not contain alpha-gal.'"

Some replacement heart valves are grown in pigs; they may cause alpha-gal sensitisation that could trigger an allergy attack later. And cardiac patients who have alpha-gal allergy seem to use up replacement heart valves more quickly than normal, putting them at risk of heart failure until they can get a replacement.

There's also a growing sense that alpha-gal may be an occupational hazard. Last year, [researchers in Spain treated three farm workers](#) who developed hives and swelling and had difficulty breathing after being splashed with amniotic fluid while they were helping calves to be born. All three of them – a 36-year-old woman, a 56-year-old woman and a 53-year-old man – already knew they had alpha-gal sensitivity, but had never imagined that skin contact would be risky.

Commins has treated hunters who developed reactions after being splashed with blood after field dressing deer; those cases raise the possibility that meat-processing workers could be at risk. In the two main Facebook groups where patients gather, it's common to hear school cafeteria workers fret about reactions from breathing the fumes of meat cooking.

Last summer, [researchers working with Commins reported that people with alpha-gal allergy may have greater allergic reactions to the stings of bees and wasps](#), potentially endangering landscapers and other outdoor workers.

It's hard to know how many people may be sensitised to alpha-gal without knowing it. A project at the National Institutes of Health (NIH) that studies unexplained occurrences of anaphylaxis found last year that 9 per cent of the cases weren't unexplained after all: they were alpha-gal patients whose sensitivity had never been diagnosed.

Platts-Mills points out that the prevalence of high levels of alpha-gal IgE in his earliest studies was up to 20 per cent in some communities, "but that was absolutely not the prevalence of allergic reactions to meat," he says. "So there are clearly plenty of people out there who've got the antibody but don't have this syndrome."

What this all means is that there are almost certainly people for whom a meat-containing meal or medical intervention could trigger an alpha-gal reaction of unknown severity.

There may be further peril awaiting them. In June, [Platts-Mills and other researchers revealed](#) that more than a quarter of patients who came to the University of Virginia's medical centre for cardiac catheterisation, to clear out life-threatening blood-vessel blockages, were sensitised to alpha-gal without knowing it.

The patients with the undetected allergy had more arterial plaque than the ones without, and, most worrisome to the researchers, their plaques were of a type that is more likely to break away from the arterial wall and cause heart attacks and strokes. Though the research is early – done in one group of 118 patients, in a known hotspot for alpha-gal – Platts-Mills worries it presages a risk for heart disease that is larger than anyone expects.

When a new disease surfaces in the USA, it's usually the CDC that investigates, pouring epidemiologists and data scientists into the field to track down connections and bring back samples for lab analysis. But



investigation of alpha-gal is caught in a bureaucratic quirk of federal science. The CDC is responsible for infections spread by insects and arthropods – but alpha-gal syndrome is not an infection. That makes it the responsibility of NIH – which has abundant lab scientists, but no shoe-leather disease detectives.

NIH does seem to be taking an interest. In June 2018, it hosted an invitation-only one-day IgE-mediated Meat Allergy Workshop; in the past, such meetings have indicated the giant agency is considering launching a research programme. But just reading the workshop's programme provides a hint of how new alpha-gal research is; participants called the problem by multiple different names, displaying that there isn't even yet any agreed nomenclature for it. Similarly, the US-run universal search engine for journal articles, PubMed, indexes papers on alpha-gal under "allergy to galactose-alpha-1,3-galactose", "mammalian meat allergy", "delayed red meat allergy", "galactose- $\alpha$ -1,3-galactose syndrome" and more.

Platts-Mills was one of the workshop's invited speakers and gave the opening statement. Commins was there as well, along with researchers from New York, Germany, South Africa and Sweden.

Dr. Marshall Plaut, who convened the meeting and is now chief of the Food Allergy, Atopic Dermatitis, and Allergic Mechanisms Section at NIH's National Institute of Allergy and Infectious Diseases, describes it as the earliest step in possibly committing to a research programme. (Platts-Mills and Commins have already received some NIH funding.) "It signals that NIH has some interest in understanding more about the disease," he says. "There are a lot of things that need to be understood."

In August, Commins gave a talk on alpha-gal allergy at the International Conference on Emerging Infectious Diseases, a conference held every two or so years and sponsored by the CDC that often surfaces the

earliest signals of illnesses that are destined to become big problems.

The CDC's director of foodborne illness was in the audience; so was its director of vector-borne diseases, the department that deals with ticks. Afterwards, they both zoomed up to ask him questions. "I kind of had the impression this was just a weird, small thing," Dr. Lyle Petersen, the vector-borne director, told him. "But this seems like kind of a big deal."

With NIH and the CDC paying attention, research into alpha-gal might be reaching a threshold, a moment at which isolated investigations might coalesce into answers. For the patients, who feel isolated too, that can't come soon enough.

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