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INTRODUCTION

Forever and Everywhere

BRENDA HAMPTON THOUGHT SHE HAD finally gotten her blood pressure under control. She gratefully gulped down her new medication and kept a watchful eye on her vital signs, which had been volatile, to say the least.

Having faced years of renal failure with just one kidney, Brenda had been coping with “uncontrollable” highs and lows as the semi-functioning organ worked overtime. But with her blood pressure now stabilized, her doctors remained relatively unfazed about her laggard pulse of 40 beats per minute. Perhaps, they surmised, her body had just become accustomed to functioning at a heart rate that Brenda described as nearly “undetectable.”¹

But things changed when Brenda got home at the end of the day, in December 2021.

“My shoulders were bothering me, and so I administered a heating pad, went to sleep,” she said. “I woke up nauseated, sweat popping off of me—I felt like an elephant was sitting on my chest.”

Breathless after eventually falling, Brenda knocked on the floor to her

downstairs neighbor, who ultimately called her sister and an ambulance, which shuttled her to the hospital.

“I would never become dizzy or anything until I fell here—at the time when I had a heart attack,” she said. “It was an unusual heart attack. I had worked that Monday.”

Her medical team was puzzled by the circumstances of the heart attack but told Brenda that the event was likely connected to her kidney issues. And while there was no blood clot and Brenda didn’t require a stent, her bloodstream did contain the cardiac enzymes that marked a recent heart attack. The mysterious episode concluded with warnings from her doctors that she might be “leading up to a big one.”

Sitting next to her monitor a couple weeks after the attack, Brenda observed that her heart rate wasn’t even reaching 40 beats per minute—the lowest measure to register on her machine.

“I’m just sitting. I’m not doing anything stressful. I’m not worried about anything,” Hampton said. “I know it’s from getting in those chemicals.”



Experiences like Brenda’s—life-threatening illness following long-term exposure to toxic chemicals—have become disquietingly familiar in recent years. But what makes her story especially insidious is the nature of the chemicals that had infiltrated her drinking water. As it turns out, Brenda, and in fact, everyone in her small hometown of Courtland, Alabama, had been contaminated by compounds known as PFAS.

While just a brief acronym, PFAS stands for a much lengthier chemical name—the umbrella group called per- and polyfluoroalkyl substances, which include thousands of manmade chemicals. They have waterproof and stain-resistant properties and are used in all sorts of products—from Teflon pans to raincoats, mascara, and even certain types of firefighting foam. And unfortunately for the people of Courtland, PFAS also have a habit of leaching into nearby waterways.

Worse yet, these substances are known as “forever chemicals” for a reason: unlike many compounds, they don’t break down over time. Instead, they build up in the environment, and many also accumulate in animals and people, growing evermore concentrated and dangerous. Along with kidney and testicular cancer, studies have linked PFAS to a variety of other ills, from low birth weights and high cholesterol to thyroid dysfunction and liver damage.

PFAS chemicals are not only toxic and long-lasting; they are, as environmental activist Erin Brockovich points out, omnipresent. “All this time it’s been in the water. It’s gotten into the food chain. We’re eating it. It’s in plastics. We’re sleeping in it. It’s in everything we touch.”² Overall, the contamination is so widespread that the Centers for Disease Control and Prevention (CDC) estimated that PFAS are in the blood of at least 97 percent of Americans.³

Although most of us have PFAS coursing through our veins, these chemicals, like all pollutants, are not distributed evenly or equitably. Instead, communities like Brenda’s, downstream from production facilities, are particularly hard hit. So, too, are those who live near military bases, where soldiers used PFAS-laden firefighting foam in training drills; and farm towns, where chemical sludge was used to fertilize crops.

Throughout the following pages, we will meet a wide range of individuals who have become painfully familiar with the awkward acronym PFAS. The toxic compounds that trickled through Brenda’s tap have also infiltrated the infrastructure of Mark Favors’s faithful military family in Colorado, Lawrence and Penny Higgins’s agricultural oasis in Maine, Emily Donovan’s riverside community in North Carolina, and every American household.

In a note of hope, we will explore how these communities are pushing back against the chemicals and companies that have imperiled their health and happiness. As in so many cases of pollution, opportunism and sleight of hand by industry are central to this story. So, too, is

government neglect. On a federal level, it took decades for just six of what could be as many as 15,000 compounds to be regulated.

“We said ten years ago this is going to be the largest emerging contaminant in the history of this country, and it is,” added Brockovich. “It’s very scary, because it’s not going to go away.”

They’re forever, they’re everywhere, and they’re wreaking havoc across the nation and in every corner of the planet.

CHAPTER 1

A Glorious Future

TALL GRASS BORDERS EACH SIDE of northern Alabama’s County Road 150 as it passes by farm homes, groups of grain silos, and centuries-old plantations. The fields taper off toward a traffic intersection where the area’s lone gas station looms large: a Chevron-signed gateway to the Jesse Jackson Parkway and the town of North Courtland. By the time the road reaches a once predominantly Black high school, shuttered by the school board in 2022 following years of neglect, green has turned to gaunt.

Passing by the empty school, Brenda Hampton gestured through the sealed car window toward an adjacent house, announcing that the father and son who lived there are now deceased.

“The next house, the mother died,” she said, as the car crept down the quiet parkway on a humid day in March 2023. “The next house coming down the street, the husband and wife died. The next house was husband and wife. The next house was a female that died—a young girl died here, in this house, here.”¹

In two streets alone in this small section of North Courtland, the sixty-seven-year-old mother of three recounted, she has identified fifty-four cases of cancer and fourteen cases of kidney failure. This in a town

of fewer than 500 residents. “We’ve had a lot of people in this area to die from renal failure, a lot of them from rare cancers, very rare cancers,” Brenda said.

The death-rattled thoroughfare rolls right into adjacent Courtland, a stop sign serving as a de facto dividing line between the two towns. An abandoned Foodvalu supermarket sits vacant alongside the parkway: now the Dollar General is the sole place to buy groceries, and only canned goods and packaged food at that. Although more than 30 percent of the people in Courtland live below the poverty line, the town is dotted with relics of an affluent past—decaying but expansive plantation homes and a vacant, historic downtown—while its northern neighbor (one of the poorest communities in Alabama) hangs on by a vestigial limb.

“They don’t care for me over in North Courtland,” Brenda said. “They’re like a little nervous of me over there when I come in.”

Despite her neighbors’ suspicions, Brenda has long “serviced” both areas, offering families bottled water, adult diapers, and rides to medical appointments, free of charge. For nearly a decade, she has been supporting residents in need through her primarily self-funded nonprofit organization.

“My phone is constantly ringing from people in the community that need things,” she said.²

Like North Courtland, Courtland has experienced more than its fair share of death and disease. Across the town line, Brenda pointed out yet another home racked by illness. “I service this house—the husband died in there. He had renal failure,” she said. “The wife is still alive in there.”³

Within Brenda’s own circle, many of her nuclear family members, distant relatives, and friends have passed from unexplained illnesses. Her grandparents both died of kidney failure, as did her mother in 2001, just four years after Brenda had given her a kidney. In one single

week, her family experienced six deaths—related to causes including cancer, kidney failure, liver failure, and heart attacks.⁴

“We were at one funeral and then there was another. We buried my brother and my cousin on the same day,” she said, recalling the “double funerals” of her thirty-nine-year-old brother and her thirty-seven-year-old cousin. “It’s something to grieve one, but when you have double caskets, it just sort of messes with you.”

Brenda knew that her family had suffered tremendous loss, but it wasn’t until she experienced her own health scare that she started to suspect a link to her hometown. Brenda had grown up in Courtland before the single town split into central and northern townships. She attended the now-abandoned R. A. Hubbard High School, but a graduation gift from her aunt led her to Boston. For a while, she worked as a paralegal for her godmother and then started doing court-appointed investigative work for a variety of legal firms after she realized the importance of delving into the details of each defendant’s story.⁵ “That’s where I come in at—I knock on the door to actually see,” Brenda said.⁶

Working as a manager for McDonald’s in 2015, Brenda transferred from Boston to Tennessee to help her ailing father—and underwent a routine physical examination as part of that shift. When her tests came back, the results were shocking. “My kidneys and everything were off the charts,” she said. “They just said that I was going through renal failure then, that I had been exposed to something toxic, and I knew at the time that what I was working with, it was nothing toxic—hamburgers and fries.”⁷

After moving back to Courtland following the alarming physical exam, Brenda started to notice just how many people in town were sick. Her informal door-to-door survey revealed dozens of cancer cases, along with many instances of renal failure.

While she had lived in Boston since 1973, she made frequent trips home to Alabama, eating the same food and drinking the same water

as the residents of Courtland. “*Voilà*, I’m at death’s door,” she recalled. “And then they’re telling me that I’m affected by industrial toxins, and I’m like, what? How am I affected by industrial toxins?”⁸



Brenda’s story begins nearly a thousand miles away and almost ninety years ago, with an Ohio farm-boy-turned-chemist fresh out of graduate studies. The postdoctoral student, Roy Plunkett, had taken on his first job at a New Jersey laboratory of E. I. du Pont de Nemours and Company—which was also known as DuPont.⁹

In 1938, Roy was seeking out an alternative for the hazardous refrigerants that were used to keep food cold at the time. As part of this process, he stored a gas called tetrafluoroethylene (TFE) in cylinders at very low temperatures—assuming that he would still find a gas afterward. Yet when he sawed open the cylinder, he found a white powder instead.¹⁰ Roy’s initial reaction was one of disappointment; *Now we’ll have to start all over again!* he thought.¹¹

What had happened was that the substance had gone through a process called “polymerization,” in which a double bond between two carbon atoms in one TFE compound came apart in such a way that it could “attack” other TFE compounds.

“One of them hooked up with another one, with another one, with another one, until there were hundreds, thousands, millions of tetrafluoroethylenes,” said Kathy Davis, an associate professor of chemistry at Indiana’s Manchester University, Roy’s alma mater.¹²

Roy noticed that the powder was heat resistant, chemically inert, and so low in surface friction that most other materials would not stick to it.¹³ Yet the young scientist hadn’t yet realized what he had stumbled upon: an entirely new chemical that would eventually become a cash cow for DuPont—and one of the biggest environmental problems the world has ever seen.

The company ultimately trademarked Roy’s white powder, called

polytetrafluoroethylene (PTFE), as Teflon in 1944.¹⁴ Teflon had unique properties that made it particularly appealing to the US military: it was persistent, nonstick, nonreactive with other chemicals, and resistant to both high temperatures and corrosion. In fact, one of its first uses was to contain highly reactive materials, like uranium, in the making of the atomic bomb. Collectively, PTFE and chemicals like it came to be known as PFAS.

Though these chemicals had early roots in World War II, their military application took off in the 1960s. At that time, the US Navy partnered with the Minnesota Mining and Manufacturing Company (3M) to develop a PFAS-based foam that could suppress jet-fuel-based fires by smothering the flammable liquids responsible for their ignition. The primary PFAS ingredient in the new “aqueous film-forming foam” (AFFF) was for many years perfluorooctanesulfonic acid (PFOS), one of the most notorious types of forever chemicals.

PFAS provided armor not only on the battlefield, but also in kitchenware and in other consumer products—with the surge in popularity of a Teflon-based nonstick coating. Before mass production of the material could occur, however, scientists had to overcome a tremendous hurdle: figuring out how to bond a nonstick powder to other solids. Techniques that ultimately proved effective included heating the PTFE to high temperatures, blending it with hydrocarbons, and mixing it with solvents that extracted some of the fluorine atoms. Early on, Teflon was used for insulating electrical equipment, coating industrial valve and pump components, and eventually, commercial food processing.¹⁵

Though Roy Plunkett may have been the brains behind the Teflon chemical, the scientist “had nothing to do with putting it on a frying pan,” his daughter-in-law, Susan Plunkett, wrote in a February 2022 text message. She did recall, however, that “a man approached Roy at a social event and told him that his discovery had saved his life because he had received an artificial heart valve made of Teflon.” And when Roy

received a medal for his invention in 1951, each guest went home with a nonstick muffin tin.¹⁶

The first person to commercialize nonstick pans coated with the material was actually across the Atlantic, in France. Engineer Marc Grégoire's invention was the result of a friendly marital challenge. An avid fisherman eager to avoid tangles,¹⁷ Grégoire decided to heat Teflon powder just below its melting point to coat his aluminum fishing gear.¹⁸ When his wife, Colette, got wind of his new hobby, she challenged him to coat her cookware with the material as well. He patented the technology in 1954 and the couple launched a company, Tefal, in 1956.

Soon after, Tefal opened a factory in Rumilly, a small town in the French Alps near the Swiss border. It attracted employees by offering relatively high salaries, even if the working environment was far from ideal. Bernard Truffet, the second-ever employee at the Rumilly factory, recounted “difficult conditions” from his first days on the job, lamenting how they “were building things cheaply.” He recalled one situation in which he was tasked with acquiring dozens of meters of bicycle chains for a conveyor belt and another in which a flood of Teflon erupted from a centrifuge. But the company's eventual transformation into an industrial titan brought significant change to the verdant Alps community, and many residents relished their boost in lifestyle. Henry Bouvier, an employee from 1967 to 2007, described the factory as “somewhat of an institution” in the region, noting that salaries were “obviously much higher” than those paid by small pharmacies or artisan shops in town.¹⁹

The forever-changed village still houses the Tefal global headquarters and continues to boast the coveted title of “the world frying pan capital.” In 2016, officials erected a monument of a giant metal frying pan at the town's entrance for the company's sixtieth anniversary, while celebrating with festivities along “Tefal Street”—the original home of a product that unexpectedly became a post–World War II phenomenon.²⁰

Nonstick pans became a major hit, both in Europe and across the pond. Tefal's products made it to the US market in December 1960, with stores ordering a million pans each month by mid-1961.²¹ That year, a photo of First Lady Jackie Kennedy holding a Tefal pan caused a significant boost in company sales.²² Historic promotions for the cookware introduced a so-called happy pan as an "amazing new concept in cooking" and the savior from getting "stuck in the kitchen."²³

A 1968 ad from Mirro Aluminum Company, a former cookware giant, promises "Hard-Bond Super-Tough Teflon" pots and pans, as a woman with a sixties-style flip-do glances flirtatiously from the stove at a man. A tagline trumpets the products as "for ladies who want more than just slickness from their Teflon."²⁴

Another ad, this time from DuPont, depicts a glowering woman, struggling to scrub off layers of grease from a steel pan.

"What a way to start married life," the ad declares in bold print. "There are better things in life than being married to a sink. Talking, walking, having a night out with your bread-winner. Anything, rather than scrubbing leftovers out of a pan."²⁵

The ads worked, and soon, nonstick pans became a cookware staple in households across America. Yet with heavy demand also came heavy manufacturing.

In 1951, DuPont opened a major plant called Washington Works, in Washington, West Virginia, along the Ohio River. At the plant, the Teflon manufacturing process also made use of a chemical called perfluorooctanoic acid (PFOA), which DuPont bought from 3M. The mining and manufacturing giant, meanwhile, opened several plants of its own, including a facility in Cottage Grove, Minnesota, in 1948.²⁶

Irene Dalbotten, who began her forty-year tenure at the plant shortly after it opened, told a local newspaper in 2008 that the factory started out small "but it grew fast."²⁷

One of the products the company manufactured there was

Scotchgard, a fabric protector that repels water and prevents stains.²⁸ Like Teflon, Scotchgard was also synthesized by accident—when a lab assistant spilled a liquid rubber concoction, which then splashed onto chemist Patsy Sherman’s shoes. As the scientists scrambled to clean her shoes with water and other substances, every attempt was repelled by the mixture. Sherman and fellow chemist Sam Smith transformed this inadvertent discovery into a product of its own, and sales of the Scotchgard stain repellent kicked off in 1956.²⁹ What they didn’t know, however, is that in addition to generating a lot of sales, this product would also turn out to have deadly consequences.

Over time, 3M grew, eventually launching plants all around the world that made not only PFAS but a wide range of products—from office supplies to electrical equipment to masks used during the COVID-19 pandemic. And one lucky spot to gain a massive manufacturing branch was an industrial complex just outside of Decatur, Alabama, right in Brenda Hampton’s neck of the woods.



Continuing her tour of Courtland, Brenda approached the silent downtown square, proclaiming there’s “nothing happening”—and not just because it was a Sunday. Dusty windows betrayed dark, vacant spaces, shadows of the lives no longer there.³⁰

“The lady that lived in here—she’s dead,” Brenda said, gesturing to a historic old house. “This used to be Hughes Drug Store. It is gone. This was the movie theater.”

An ice cream parlor next door hinted at some signs of life, but blank windows quickly revealed that there was no one working and certainly no ice cream inside. Brenda was quick to confirm that “nothing happens in there” and that “they just decorate it.” This was, however, the space where she had launched her community service effort.

“I gave out water, food, clothing, and everything from this ice cream shop,” Brenda said. An adjacent antique store—which now sells JAX

Wholesale, Amazon, and Target returns—became a regular gathering site for her meetings with other volunteers.

Red brick building facades—deemed historic by the National Parks Service³¹—stood largely intact, but no guests frequented the long-shuttered Old Sherrod Hotel, a 1930s hub for the construction crews who built the nearby Wheeler Dam. Nor were there any patrons in the locked and dark Ms. Jane’s Antiques, whose wares lined the windows of the historic A. F. Rebman and Company’s 1890 building.

Courtland on occasion gets families from “up north” seeking out an idyllic Southern spot to settle down. “A lot of them have been requesting, like, a little Mayberry town, laid back, cost of living pretty cheap,” Brenda said. “I’ve had several families that have come in this town, and then, they’d be asking me, ‘Well, where’s downtown Courtland?’ And I go, ‘You’re *in* downtown Courtland.’”

“This used to be a thriving town. There used to be a clothing shop right there,” she recalled. “My mother had a restaurant over here where they got the City Hall at. That’s where all the Black businesses were at.”

A wrought-iron street clock, two stories high, stands in front of the A. F. Rebman building, ticking toward a future that has left Courtland behind. Still stuck in place beneath the clock is a mounting block (also called a horse block or carriage block), its three stone steps used for a far more sinister purpose than simply climbing onto a saddle.

“These used to be where they sold a lot of slaves,” Brenda said. “This would be for the expensive one, this is one that’s not so good,” she continued, pointing to the top and middle steps. Then gesturing toward the third, she added, “That’s the bottom of the totem pole.”

Courtland, like so many other former hubs of the Deep South, still bears emblems of its pre-Civil War history. While plantations tended to have fewer enslaved people in this region, which was far less fertile than many other parts of the South, the town remained largely segregated for

more than a century after emancipation, and the legacy of slavery is still woven into the town fabric.

“People did grow cotton around here, but it was mostly smaller farms,” said John Allison, archivist for Morgan County, home to the city of Decatur and adjacent to Lawrence County, where Courtland is located.³²

Brenda described a slow-paced rural culture that defines Alabama, which she views as less developed than other Southern states. “That’s why they are still taking advantage of Alabama,” she said. “Nobody goes by any rules, nobody questions anything.”³³

Brenda holds that attitude—and the region’s limited agricultural potential—responsible for the twentieth-century drive that lured industrial giants like 3M to erect their plants along the banks of the Tennessee River.



About twenty miles upstream from Courtland, Decatur was once a sparkling Southern city replete with dress shops, cab service, and culture, as Brenda recalled from her grandparents’ lore. They, and their parents before them, worked as sharecroppers in the surrounding farm fields and came into Decatur to enjoy the vibrancy of urban life.

“They would hang their head in shame—they couldn’t believe it,” she said, of the current landscape.³⁴

In its early days in the 1800s, Decatur’s location along the Tennessee River at the intersection of two railway lines made it a hub of trade and transport, while the creation of the Tennessee Valley Authority in 1933 and subsequent construction of the Wheeler Dam brought further development to the region. In 1953, the *Alabama Municipal Journal* deemed Decatur “Alabama’s fastest growing industrial empire,” and a Chamber of Commerce pamphlet from 1961 proclaimed, “Nowhere in America can be found a better balance between agriculture and industry,” noting that the city’s seven-mile waterfront

included thirty-three industries representing a total investment of over 100 million dollars.”³⁵

The City of Decatur and the larger Morgan County made things easy for companies and manufacturers, offering an industry-friendly environment to those interested in entertaining a move to this corner of the Deep South, particularly in unincorporated rural areas.

“That’s how these plants come in here—they sit on the outskirts of the city,” Brenda said.³⁶

It was during this period that residents of Decatur first became acquainted with 3M. Phil Rath, who would end up managing the Decatur plant until his retirement in 1984, began eyeing the spot in 1959, and the company opened its factory within two years. Among the lures of the city was the presence of manufacturing giants—legacy chemical company Chemstrand, as well as Wolverine, Goodyear, and a flour mill—employers that had made the region ripe for industrial expansion while promoting river development and job growth.

“Those were all significant workplaces for Decatur,” Rath told *The Decatur Daily*. “Before that, people had to leave Decatur to make a living.”

The local news story attributed much of the plant’s early successes to Rath, surmising that “perhaps his years as a plant manager at 3M made him a problem-solver, or maybe it was growing up on a ranch in Roundup, Montana, or rearing ten children.” After his retirement from the company, Rath also “put his business and family skills to work to improve Decatur in many ways: as a city councilman.”³⁷

Historical literature commissioned by the mayor and city council of Decatur in 1968 and published in 1970 hailed 3M as “a great and nationally famous corporation.” Noting that 3M specialized in a broad range of industrial chemicals, the literature explained that the plant hosted a division, managed at the time by Rath, that produced materials like plastics and textile-treating compounds. Another part of the

company with a major Decatur presence was a separate division responsible for manufacturing film.³⁸

The number of workers at the Decatur 3M plant continued to grow over time. Beginning with 100 employees in 1961, the factory rapidly expanded its workforce—employing more than 1,000 people for a collective \$11.3 million in wages and salaries in 1975.³⁹

But back in 1961, when this tenfold rise in employment was just a dream, the Alabama Chamber of Commerce declared in its pamphlet that this “Fresh Water Fishing Capital of the South” had “every reason to look forward to a glorious future,” thanks to the combination of an “enlightened citizenry” and “devoted community leaders.”

“Decatur’s future growth and success can most certainly be assured,” the pamphlet boasted.⁴⁰

For a while, during Brenda’s childhood, those golden promises seemed to materialize. “People were able to afford better homes and better transportation,” Brenda said. “And in the area where 3M is, once that comes in, CEOs always talk to another plant to find out how the area is, so it brought in other plants.”

The lure of jobs kept 3M popular in the community. Yet as the chemical giant grew and grew, decades-old mom-and-pop businesses, along with legacy industries, eventually began to shut their doors. When, for example, the International Paper mill closed down in 2014, it was Lawrence County’s largest employer, taking with it 1,096 jobs and \$771,000 annual tax revenue from Courtland.⁴¹

As of mid-2022, manufacturing still made up a significant share of both Courtland and North Courtland’s economies, responsible for about 35 percent and 39 percent of jobs, respectively, while Decatur clocked in at 20 percent.⁴² Yet the dream of middle-class livelihoods appears to have gone up in smoke alongside the plumes from the manufacturing plants. Courtland’s median household income was almost \$40,000, while North Courtland’s was nearly \$31,000 in a 2022 federal

survey.⁴³ Decatur, less reliant on manufacturing jobs, was higher at more than \$55,000—but still a good \$20,000 below the national average.⁴⁴

The economic push and pull of the chemical industry was already a significant burden for Brenda and her neighbors to shoulder. But whether 3M and its compatriots were creating jobs or hurting the community's other financial prospects, they were certainly producing vast amounts of waste—byproducts that ended up in landfills that were unequipped to handle them and in waterways that carried the pollution to nearby towns. And although that waste was there to stay, the same could not be said for all the companies and the jobs they provided. That eventual emptiness and contamination, Brenda said, caught up with the area and “everything just died out.”⁴⁵

“It changed the landscape here,” she recalled. “Then the stores and things closed—no grocery store, things like that. That’s what happened in this area. . . . It was devastated.” And in the case of PFAS, the threat wasn’t just to neighborhood stores and historic town centers but to the residents themselves.

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CHAPTER 2

A Very Toxic Compound

AS THE DECATUR 3M PLANT was ramping up production of PFAS and the popularity of products like Teflon surged in the middle of the twentieth century, evidence was emerging that the substances also had a dark side.

In the 1950s, researchers inside and outside DuPont started to see an odd phenomenon: people exposed to heated Teflon developed flu-like symptoms, feeling chest discomfort, followed by a dry cough, increased heart and breathing rates, fever, shivering, and sweats. A 1951 paper in *The Lancet* documented four such cases—including two that involved DuPont employees.¹ The accounts continued to trickle in, with an internal memo from 1959 discussing the experience of employee John Kropenski, who came down with a case of the “shakes.” Earlier in the day that he developed these symptoms, Kropenski had cut a Teflon pipe while he had an open pack of cigarettes in his pocket. The tremors began after he smoked the cigarettes later that evening.²

About a year later, a New York-based doctor documented a similar phenomenon in one of his patients, a middle-aged man who worked with Teflon. The doctor, Henry Wharton, said in a letter to the FDA

that the man was experiencing “angina-like” chest pain as well as dizziness and shortness of breath. The patient noticed that his co-workers had similar symptoms, and his foreman told him that these symptoms were “caused by Teflon and that they all know about it,” the doctor wrote. Wharton took a chest X-ray of the patient, observing abnormalities and questioning whether “Teflon might be carcinogenic or whether it might produce” a lung disease. In response, an FDA official said the agency had “relatively little occasion to consider” Teflon’s health effects—a response that foreshadowed years of regulatory neglect to come.

By the early 1960s, stories about what came to be called “polymer fume fever,” along with other ill-effects of inhaling Teflon, were circulating beyond company walls. One account, published in a letter to the *Canadian Medical Association Journal*, alleged that a person who smoked a Teflon-contaminated cigarette had died—though the writer later retracted the story.³

Nevertheless, in response to these stories, DuPont published a pamphlet called “The Anatomy of a Rumor,” decrying the accusations. The pamphlet’s author, DuPont toxicologist John A. Zapp Jr., acknowledged the existence of polymer fume fever—likening it to influenza—but stressed that there are “no lasting physiological effects” and linked the symptoms to soot pollution, as opposed to gases related to Teflon. In addition to redirecting blame for polymer fume fever, Zapp also emphasized the safety of Teflon pans.⁴

A manager at a company that used Teflon wrote to the *Journal of Teflon* in June 1962 to praise the missive as “extremely informative”—and he requested thirty copies to distribute to his employees, who had regular contact with the material. “We have all been exposed to these rumors in various forms, and possibly unknowingly some of our people have assisted in passing some of them as impressions along to our customers,” he wrote.

Despite these assurances, even prior to the 1960s scientists were

beginning to demonstrate that the impacts of PFAS exposure might extend beyond a temporary “flu.” In a 1956 paper, researchers with Stanford University found that PFOA could bind to human blood.*⁵ It was also during this era that evidence began to emerge about the potential of these substances to harm animal organs. For example, a 1955 study published in the journal *Cancer Research* found that embedding Teflon under rats’ skin could trigger the growth of cancerous tumors.⁶

As alarming findings began to accumulate, DuPont’s own personnel also started to discover adverse health effects in animals. Toxicologist Dorothy Hood wrote in a 1961 company memo that several PFAS, including PFOA, were toxic and could cause the enlargement of rat livers in low doses. She described another PFAS, known by the initialism AHT as “a very toxic compound.” “It is recommended that all three materials . . . be handled with extreme care,” Hood wrote. “Contact with the skin should be strictly avoided.”⁷

By the following February, and at the same time in 1962 that Dupont was downplaying health hazards associated with inhaling Teflon, company scientists documented additional health issues stemming from PFOA. DuPont pathologist G.W.H. Schepers wrote that month in an internal company memo that rats fed PFOA had moderately enlarged livers, shrunken pancreases, and slightly enlarged kidneys, adrenal glands, and testes.⁸ A few days later, Schepers followed up to explain that in three experiments, rats fed PFOA were killed by damage to their stomachs, intestines, brains, lungs, and pancreases. Meanwhile, another company document suggested that the problem might apply to an entire group of PFAS compounds known as surfactants—substances such as soaps, detergents, and lubricants that decrease a liquid’s surface tension and, in the case of PFAS, enable water and oil to mix into a foaming agent.⁹

* References to PFOA in this book also include variations of the substance known as APFO, an ammonium salt of PFOA.

Following these initial findings, DuPont researchers extended their experiments to also include dogs, noting that the canine research “might be useful in detecting liver injury in personnel.”¹⁰ Sure enough, the studies showed liver enlargement.¹¹ By 1966, DuPont was trying to figure out how to get rid of waste from its Teflon plant. While acknowledging that some of the solid wastes contained “toxic” PFAS, the company claimed to have found a suitable method of disposal—as long as the toxic parts were “reduced to an acceptable level.” All one needed to do was bake the waste for five hours. Without taking this action, it warned, some of the toxic PFAS “would be leached into the groundwater.”



DuPont may have been grappling with its PFAS waste problem in the mid-1960s, but federal regulators had already begun to take notice of the potential issues posed by the substances in 1959. In February of that year, a man named Henry McNulty wrote to the Food and Drug Administration (FDA) asking for its views on the use of “Teflon-Tefal” in cooking utensils, as he had hoped to import them to the United States from France. However, his business aspirations would hit a speed bump after he received negative responses from the FDA’s A. J. Lehman.¹² Replying that he could not “comment favorably” on the use of the product in cooking utensils, Lehman explained that the material “appears to be unstable under cooking temperatures” and that “some of its decomposition products are quite toxic.”¹³

McNulty tried again, following up with additional information, but Lehman’s opinion remained unchanged. “I have reviewed the data and have again discussed the problems with others of the staff,” the FDA official stated. “I am sorry to say that I cannot offer any other opinion than that expressed in my letter of Feb. 10.”¹⁴

But later that same year, in September of 1959, Lehman appeared to change his tune. He, alongside other FDA officials, met with DuPont representatives to discuss the company’s own foray into PFAS-coated

cookware. At the meeting, the employees discussed two problems—“the inhalation hazard and the ingestion hazard”—hazards that Zapp, the DuPont toxicologist who also wrote the “Anatomy of a Rumor” pamphlet, roundly refuted. Some back-and-forth followed, according to a DuPont memo, but ultimately an FDA toxicologist agreed that no hazard existed. DuPont employees, meanwhile, presented data showing that “only minute bits” of the coating transferred to food when the Teflon pans were used.

“It’s like bits of glass or porcelain coming off,” Lehman understood.

The agency ultimately did require some further clarification from DuPont, but the company received “no major objections” from the FDA officials, who foresaw “no major obstacles” in getting federal approval.¹⁵ A few months earlier, the FDA also authorized Teflon’s use in handling milk. Similar issues later cropped up for another DuPont product: Zonyl, which used PFAS to make food wrappers resistant to grease. The FDA raised objections in 1966 about the substance leaching into food and causing liver problems. However, DuPont was able to stave off the regulators once again, even though, after the product got FDA approval, it would again find that dogs who ate it experienced liver issues.¹⁶



While the FDA gave PFAS the green light, pesky questions about the health effects of the chemicals would continue to plague 3M and Dupont in the years that followed. After university researchers found organic fluorine—a possible indicator for PFAS—in 104 out of 106 blood samples from five different US cities in the 1970s, they alerted 3M that they were suspicious of its own Scotchgard and DuPont’s Teflon.¹⁷ In 1975, 3M sought to tamp down any qualms, telling University of Florida scientist Warren Guy—who had expressed concerns about the role of Teflon or Scotchgard in fluorine’s pervasiveness—that “this was no time for speculation.” In an internal memo, 3M

employee G. H. Crawford described these concerns as “far-fetched” and “unlikely,” but said that of the products Guy had flagged as possibly responsible, Teflon was “least unlikely.” However, Crawford downplayed the matter, writing, “This was not (I hasten to say) suggested to Dr. Guy.”¹⁸

Despite brushing off the researchers, 3M started to look for the substance in the blood of its employees—and found it. In fact, in 1978, the company described fluorine concentrations in their blood to be “proportional to the length of time” that they worked there, persisting in people who had previously been exposed to PFAS but hadn’t been around the compounds for fifteen to twenty years.¹⁹ There was also some early evidence that the substances’ reach was global. A 1979 DuPont memo cited 3M’s detections of organic fluorine in the blood of peasants in a Chinese village, though at levels “significantly lower . . . than values found in developed countries.”²⁰ DuPont also found fluorine in the blood of its workers, but the company decided against informing the EPA.

Worse yet, there were signs that these chemicals were taking a significant health toll—not just in animal studies but in people. A 1978 internal review of the medical records of those workers who had long-term exposure to PFOA revealed “borderline elevation of liver function tests,” wrote Sidney Pell, a manager of DuPont’s epidemiology section.²¹

The finding may have presented a legal quandary for DuPont—not only putting their own employees at risk, but also causing potential harm to contractors who worked at the plant. In fact, there was some internal debate at DuPont over whether to inform West Virginia janitorial company Winans about the risks to their staff. Ultimately, Dupont decided—in the interest of preempting any legal or media scrutiny—to come clean and recommend taking employee blood samples.

“This sampling is required to place DuPont in the best legal and ethical position should something happen with C-8 [i.e., PFOA] in the

future or if we're investigated by either the media and/or government," Dupont's A. R. Behnke wrote in a memo.²²

He then laid out a playbook as to how such investigations have occurred at other plants, which involved a beneficial opportunity for DuPont: an offer to conduct the tests for Winans, for a price. "The contractor pays DuPont to test his people w/ the testing cost added to the price of the contract," Behnke explained in the handwritten note. "This is the route I'd advise taking."²³

As the 1980s rolled around, 3M identified something it considered so problematic about PFOA that it took the rare step of informing the EPA: evidence of a link to birth defects. In a study from 1981, 3M researchers found that the fetuses of pregnant rats that had consumed the substance developed eye deformities, confirming two previous studies. Yet in its alert to the EPA, 3M stressed that "no human health problems have been observed" that were related to PFAS exposure.²⁴

In addition to notifying the EPA about the issue, 3M also alerted its customer, DuPont, in March 1981. Within days of hearing from 3M, DuPont acted—moving women out of jobs that could potentially expose them to the substance.²⁵ Bruce Karrh, DuPont's medical director, wrote that there had been one employee with "heavy" PFOA exposure who had experienced a miscarriage.²⁶ 3M took similar action, moving twenty-five Decatur women "of childbearing potential" out of jobs to prevent their exposure.²⁷ By mid-May, DuPont had compiled a list of employee pregnancies and recent births. Three were listed as having had a "normal child" (though PFOA was found in one of these women's umbilical cord blood), while two other women gave birth to babies with eye abnormalities. One also had a nose abnormality. Yet within a few months, DuPont appeared to reverse course. A November 1981 staff meeting memo from DuPont said that it seems "very unlikely" that PFAS exposure causes birth defects.²⁸

Yet other, even deadlier illnesses were also cropping up, with DuPont

finding an excess of certain cancers in its employees. In April 1989, the company said that its scientists had identified more cases of leukemia than expected, while that December, they detected high levels of mouth, throat, and kidney or other urinary cancers among male employees. The company ultimately determined that there was no statistically significant excess in cancers overall.²⁹

Nevertheless, 3M was beginning to worry not only about its employees' unusual illnesses, but also about what they could mean for its bottom line. An internal memo noted that since 1984, blood levels had started increasing. The document listed two primary concerns: "employee health" and "corporate liability."³⁰

In 2000, the health effects—and their potential liability—had become so stark that, after pressure from the EPA, the company agreed to begin phasing out PFOS and PFOA, though it would continue to produce many of their chemical cousins.

When asked about this long and sordid history, 3M did not respond directly to any particular findings, but the company commented on the toxicity studies broadly. Carolyn LaViolette, a spokesperson for 3M, said in a May 2024 statement that such studies "are typically designed to produce adverse effects and identify the doses at which toxicity occurs." These doses are "generally many orders of magnitude higher" than what humans are exposed to through the environment, she added.

"Subsequent animal studies performed by 3M at lower exposure levels did not show adverse effects at exposure concentrations still well above environmental exposure levels in humans," LaViolette said.

Referring to the studies in general, she added that 3M has "shared significant information about PFAS over the decades," while also publishing many of its related findings "in publicly available scientific journals dating back to the early 1980s."³¹

As for DuPont, the company spun off its PFAS business in 2015—and then later underwent a merger and a separation. A spokesperson for

the corporation argued that as a result, DuPont is no longer the same company that carried out these studies, and therefore did not provide a response. The company to which it spun off its PFAS business also declined to comment, declaring that it did not exist until 2015.



As executives at DuPont and 3M were discovering the lethal effects of PFAS, the consequences were becoming painfully obvious at the plant outside Decatur. Even in the glory days, when 3M seemed like an economic savior for communities along the Tennessee River, red flags were popping up along its banks. Fifteen years after it opened the Decatur plant, 3M found PFOS in a Decatur plant worker's blood sample—a finding that repeated itself in several employees in 1979 and became even more striking in the decades that followed.³²

3M was finding not only a wide presence of the chemicals in Decatur workers' blood, but also that the exposures were making people sick. One study based on data collected in the 1990s found high risk levels for prostate and colorectal cancers, while another noted that workers had an increased risk of death from bladder cancer. The first study revealed that around the time that 3M was downplaying any potential health harms of PFOS to the EPA,³³ the company was internally identifying conditions to watch among its own personnel. 3M scientists listed liver and bladder cancers, as well as endocrine and reproductive disorders, as being of interest based on “a substantial body of literature,” including PFOA and PFOS research.³⁴

There was also evidence that the exposures were not limited to within the walls of the facility; in 1979, PFAS compounds were discovered in fish caught in the Tennessee River around Wheeler Dam, about twenty-six nautical miles from the company's Decatur plant.³⁵

While 3M's facilities have long been landmarks on the Tennessee River shores, they are by no means the only such plants in the region—or the only ones polluting local waterways. Steps away from 3M Decatur

sits another industrial giant: Daikin, an appliance manufacturer headquartered in Osaka, Japan. Driving along the highway adjacent to the area, passersby can see the company's cream-colored buildings flanked by intersecting gray structures. On a warm June 2023 day, as Brenda's car careened past the compound, at least one chimney was emitting a white substance into the atmosphere.

In addition to making air-conditioning equipment, Daikin also deals in PFAS. The company began making fluoropolymers—coatings that resist heat, oil, stains, grease, and water and can be made from PFAS—at the Decatur site in 1994. But the chemicals Daikin used didn't stay on Daikin's property—they went into the nearby air, water, and of course, the Tennessee River. A 2004 presentation stated as much; while stressing that soil monitoring showed “no clear pattern,” it lists the application of sludge to the land and residue from air emissions as “potential sources of contamination.”³⁶

While for years—and indeed decades—troubling findings were being discussed behind closed doors in corporate offices and government agencies, by the 1990s the local media was starting to take notice. A November 1999 article in *The Decatur Daily* sounded the alarm, citing the area industry's own descriptions of “their ultimate potential disaster.”

“At the Daikin America Inc. and 3M chemical plants, the worst-case scenario for a chemical accident would be the total release of a railcar filled with up to 180,000 pounds of hydrogen fluoride,³⁷ a dangerous toxic chemical,” the article stated. Any potential accidents involving the substance—which is required in the synthesis of PFAS, among other compounds—“could also span 25 miles and affect an estimated population of more than 75,000 people,” the article added. However, to mollify readers, it noted that “the worst-case scenario is extremely unlikely to happen.”³⁸

Likely or not, the threats posed by PFAS were becoming more widely recognized, with tangible consequences for its manufacturers and their

factories. It was around this time that 3M agreed to stop producing PFOS and PFOA, a move that affected the products churned out on the banks of the Tennessee River, and by extension, about a hundred of 3M's 975 Decatur employees.

"It has already been a year of change for 3M," a December 2000 *Decatur Daily* article stated, citing the May 2000 corporate decision to phase out certain product lines that contained "persistent chemicals that do not easily break down in the environment."³⁹

Among the items they shelved were certain Scotchgard products and firefighting foams, as well as grease-proof paper, pet food packaging, and some surfactants that were used in paint to smooth its spread. While Jim King, 3M's Decatur plant manager, stressed that the chemicals had not been proven dangerous, he acknowledged that their persistence was unsettling nonetheless.

"It's not a good public image if you're dealing with something that lingers in the environment, even if there's nothing wrong with it," he said.

The plant at that point started refocusing its efforts on a light adhesive used on the back of 3M Post-it notes and an adhesive for reflective strips on firefighter's outer clothing. Despite the news, King said at the time that "Scotchgard is alive and well," noting that affected products would be reformulated.⁴⁰ King went on to retire just a year later, in 2001, and he died in 2019.

But things at 3M were perhaps not as "alive and well" as they seemed. A series of tests from the early 2000s detected PFAS in the Tennessee River,⁴¹ showing that the pollution was flowing downstream: the water and fish collected there had higher concentrations than those upstream.^{42, 43} The levels identified in these fish were "very high," according to Rutgers environmental and occupational health professor Robert Laumbach.⁴⁴ PFOS was also found in the wastewater leaching out of Decatur's landfill, in local sediment, and in sludge coming out of the city's wastewater treatment plant.⁴⁵

(continued...)

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