

Even before a child takes its first breath, prenatal exposure to urban pollution ups the odds of developing asthma, cognitive problems, and obesity

by TIM PAUL

photographs by **ROJ RODRIGUEZ**

A FILTER FROM RAMONA TAVERAS' AIR CONDITIONER IS CLOGGED WITH THE RESIDUE OF AIR POLLUTION.



AMONA TAVERAS KNEW

next to nothing about asthma until one evening last year when her 4-year-old son, Justin, started gasping for air. A vein on the

right side of his neck bulged as he wheezed and struggled to breathe. There was only one thing to do. She put aside the dinner she was cooking and rushed him in a cab to the emergency room. It wouldn't be the last such trip. In the past year, Justin has been in and out of the ER three times, even after a specialist prescribed an inhaler. Taveras' younger son, Danny, was also diagnosed with asthma, though, thankfully, his attacks have been less severe than his brother's. Taveras, a native of the Dominican Republic, wonders if where she lives could have something to do with her sons' illness. Her apartment is in the Bridge Towers, a housing complex built astride Interstate 95 near the George Washington Bridge. In a single day, close to 300,000 cars and trucks pass under her building. According to the Port Authority of New York and New Jersey, the bridge is the busiest crossing in world.

No matter how often she cleans, Taveras says, her apartment is never free of dirt. "Right after I'm done," she says, "I go back to my dresser and there is a layer of soot."

As it turns out, Northern Manhattan has one of the highest rates of asthma and asthma hospitalization in the nation. Other health problems also abound in this low-income, largely Hispanic neighborhood. Onequarter of the area's children are obese, and close to a third have a developmental delay affecting their cognitive or motor skills well above the national average. Deaths from cancer among both children and adults are also elevated.

Could there be a link between the soot in the air and Taveras' asthmatic children, not to mention the other health problems plaguing their neighborhood?

This is the kind of question that research-



HOME ABOVE A HIGHWAY

INTERSTATE 95 PASSES BENEATH THE HIGH-RISE WHERE THE TAVERAS FAMILY LIVES. LOOKING EAST FROM THEIR 28TH-FLOOR WINDOW, THEY CAN SEE A BUSY INTERCHANGE CONNECTING NORTHERN MANHATTAN AND THE BRONX.

ers at the Columbia Center for Children's Environmental Health at the Mailman School have sought to answer since the Center was founded 14 years ago. Researchers there, led by Environmental Health Sciences Professor Frederica Perera, DrPH, have uncovered a wealth of disturbing data linking the urban pollution to childhood diseases and developmental problems. They have illuminated a virtually invisible process that begins even before birth and harms multiple tissues and organs right down to a child's DNA.

Simply put, they've confirmed that there's something in the air.

A SHOCKING DISCOVERY

There are, of course, many noxious substances pervading urban air. Among the most intriguing to researchers at the Center is a category of chemicals released when organic material—such as tobacco, oil, coal, wood, and gasoline—is burned. These chemicals, called polycyclic aromatic hydrocarbons, or PAHs, are ubiquitous in New York City, with the highest concentrations in heavily trafficked areas. The dark soot particles that Taveras keeps wiping from her dresser? That's full of PAHs, which, despite their name, are odorless.

Perera's interest in PAHs grew out of discoveries she made as a Columbia graduate student. In laboratory experiments, her mentor, the late Bernard Weinstein, MD, and colleagues had shown that PAHs attach themselves to DNA, leaving behind molecular fingerprints, known as adducts, that could be detected in the lab. For her dissertation on molecular epidemiology, Perera showed for the first time that it was possible to measure these DNA adducts in human blood samples and lung tissue. It was a breakthrough that helped open the door to a new scientific field—molecular epidemiology—that would, as Perera explains it, "pry open the black box between exposure and disease."

Another critical discovery lay around the corner. To establish a baseline of zero exposure to PAHs in the environment, Perera collected umbilical cord blood samples. A mother's placenta was believed to be a barrier against these pollutants, so the young researcher expected cord blood to be pristine. To her amazement, many samples had high levels of adducts—even in cases where the mother wasn't a smoker.

The finding was alarming. Studies had determined that adducts were not merely signs of PAH exposure, they were a kind of genetic damage that could ultimately lead to cancer. Seeing adducts in cord blood meant that damage from cigarette smoke or other forms of pollution could begin in the womb. "This made a serious impression that opened a whole new realm of investigation into prenatal exposures," says Perera. "I knew then we had to find out how these babies were being harmed and how to prevent it from happening."

BACKPACKS AND BABIES

In 1997, Perera and her colleagues began an ambitious, longterm project designed to examine how prenatal exposures to air pollution might impact health throughout childhood. Over the next nine years, the researchers recruited more than 700 pregnant women from Northern Manhattan and the Bronx, most of them black or Hispanic and a third of them living below the poverty line. With continuous funding by the National Institute of Environmental Health Sciences (NIEHS) and the Environmental Protection Agency, the Mothers and Newborns Study has helped establish the fetal origins of disease. To solve the problem of measuring the women's exposure to air pollution, the researchers came up with a brilliant strategy. The mothers wore a lightweight backpack filled with air-sampling equipment for 48 hours during their third trimester of pregnancy. The pack went everywhere they went, indoors and out; it rested at their bedside while they slept. A batterypowered pump continuously sucked in air through a filter that collected gas and particles—including PAHs.

"Previous studies collected air samples from set locations like the top of a building. These backpacks did something much more powerful by giving us a snapshot of exposure for each pregnant woman as she lived her life," says Mailman Environmental Health Sciences Professor Patrick Kinney, ScD, who helped design the devices.

When the backpack filters were analyzed, the research team was shocked to find that PAHs were present in 100 percent of the air samples. Exposure happened everywhere. In fact, indoor air was often worse than outdoor samples, especially in the winter, when

some apartment buildings use what is known as residual heating oil. Because of New York's legacy as a shipping and oilrefining center, the city is the only place in the country where residual heating oil is still legal. Literally the bottom of the barrel, these oils burn thick and dirty.

Perera and her team paired the prenatal air monitoring with measurement of the PAH-DNA adducts in cord blood. Given the high level of PAH exposure, researchers expected to see plenty of PAH-DNA adducts in the children's cord blood. In fact, 42 percent of the cord blood samples had this telltale sign of exposure.

Over the years, the Mothers and Newborns Study followed the children born to the 700 mothers. Asthma was a major concern. Because of their minuscule size and volatility, PAHs are easily inhaled into the airway. Research by Associate Professor Rachel Miller, MD, with Perera and other colleagues showed that children exposed prenatally to PAHs combined with exposure to secondhand smoke had greater odds of asthma at ages 5 and 6. The psychological state of the mother was another potentially aggravating factor, albeit one that acted independently of PAHs. "Reports of difficulty coping with stressful situations during pregnancy increased the odds that a child had



wheeze," says Miller. "Whether it is fear of crime or worries about having enough food on the table, low-income families have many stressors."

Asthma made sense on an intuitive level: Bad air goes directly into the airways. But the research team discovered a range of less obvious health risks.

A series of studies by Perera with Clinical Population and Family Health Professor Virginia Rauh, ScD, MSW, and others found early signs of impaired development in the form of reduced birth weight and head circumference. Later research confirmed that nearly a third of the children showed deficits in at least one neurodevelopmental domain. Children exposed to the highest levels of prenatal PAHs scored well below average on tests for cognitive development at age 3 and showed reductions in IQ at age 5. A more recent study found signs of anxiety, depression, or attention problems in this group at ages 6 and 7. "We were able to show this link using both the air monitoring data and the cord adducts as measures of prenatal exposure," says Perera. Adds Rauh: "This is concerning because these symptoms could adversely affect social relationships and academic performance in the early school years and potentially lead to further behavior prob-

lems as the children mature." Exactly how these changes happen is still being worked

happen is still being worked out. One clue came from a study of rat brains at Meharry Medical College in Nashville, Tenn., that found PAH exposure interfered with the ability to regulate neuronal cell differentiation. As a result, the animals exhibited depressionlike behaviors and memory problems.

In fact, there's evidence for several possible mechanisms. PAHs bind to placental growth factors, restricting the exchange of oxygen and nutrients. They impact DNA through the formation of adducts and alter gene expression. They are toxic to the

immune system and inhibit the brain's ability to clean up antioxidants. And they mimic estrogen, disrupting the body's endocrine system, which regulates growth and development, tissue function, metabolism, and even mood.

The endocrine system is also involved in the creation of fat tissue. Researchers at Ohio State University showed that mice exposed to PAHs gain in fat mass, and studies of cell cultures found that exposures to PAHs interfere with lipolysis, the process by which fat cells shed lipids and shrink in size. Were PAHs doing the same thing in children?

To find out, Environmental Health Sciences Professor Robin Whyatt, DrPH, and Andrew Rundle, DrPH, associate professor of Epidemiology, examined the evidence for any link between children's weight and BMI measures and their prenatal exposure to PAHs. They found that children exposed at high levels were more than twice as likely to be obese by age 7 as those exposed to lower levels. "Not only was their body mass higher, but it was higher due to body fat rather than bone or muscle mass," says Rundle. The study, he notes, is the first to link air pollution to obesity.

Of course, PAHs weren't the only reason these kids put on the pounds. "Obesity is a complex disease with multiple risk factors," says Rundle. "What this study tells us is that the epidemic of obesity isn't just the result of individual choices like diet and exercise. For many people who don't have the resources to buy healthy food or don't have the time to exercise, prenatal exposure to air pollution may tip the scales, making them even more susceptible to obesity."

BROADER RESEARCH, BROADER IMPACT

As the research continues to unfold, surprises lurk around the corner. Some of the children born into the study are now 14 years old. The teenage years, says Perera, are another period of heightened vulnerability. Like the fetal period, there is rapid growth, and hormonal factors are at play. Could air pollution be a factor in early-onset puberty, which is commonly seen in this community? Will DNA damage from adducts or epigenetic changes raise the risk of chronic diseases in later years? Some research suggests that prenatal damage-in the form of epigenetic changes-might even be passed along to succeeding generations.

Since the start of the Mothers and Newborns Study, Center scientists have collected a mountain of evidence on health problems linked not only to early exposure to PAHs, but also to chemicals such as phthalates and bisphenol A (BPA)—both found in plastics—and the pesticide chlorpyrifos. These findings have been reinforced by Perera and her colleagues in parallel studies in Poland, China, and a group living near the World Trade Center on September 11, 2001. All told, upwards of 2,000 mothers and children are being followed. The crucial lesson is that the nine months of fetal development are, as Perera puts it, a period of "exquisite susceptibility."

As the fetus grows, Perera adds, there is an "elaborate choreography" of rapid cellular differentiation and proliferation that raises the possibility of problems occurring when the fetal environment is changed even slightly. Meanwhile, the fetus is without an adult's immune defenses and DNA repair systems. While the placenta protects against 90 percent of toxins the mother is exposed to, the remainder has the potential to inflict much worse damage in the fetus than it would in the mother. All this points to a need for prevention, says Perera. "See the red flags and do something in time."

Doing something in time is precisely what concerned citizens, advocacy groups, and policymakers are focused on. In New York and elsewhere, they are grabbing hold of research on PAHs and successfully pushing for policies that will lead to cleaner air. In New York, these efforts have already led to new regulations and a cleaner bus fleet. (See next page.)

And when policies change, the Columbia Center for Children's Environmental Health is there to assess the impact. Its research has found that between 1998 and 2006, levels of PAHs recorded by the backpacks were down significantly. Asthma hospitalization rates were also down. The trend is likely to continue, particularly with the advent of a city initiative to eliminate the use of residual heating oil, although Perera cautions that even very low exposures can impact fetal development. Another complicating factor is climate change, which scientists predict will exacerbate the effects of air pollution.

And there are pockets of the city where dirty air remains a constant danger. Just ask Ramona Taveras. Her sons by her side, she gazes from her 28th-story window and watches as five lanes of truck-heavy traffic inch east toward a tangle of highways. "This is my view," she says. "This is the air we breathe."



HOW THEY DO THEIR DIRTY WORK

Prenatal exposure to polycyclic aromatic hydrocarbons (PAHs) from exhaust fumes, tobacco smoke, and other sources can have lasting effects on a child, according to a series of landmark studies by the Columbia Center for Children's Environmental Health. A variety of mechanisms may be at work: PAHs are endocrine disrupters that can impact hormones controlling growth, metabolism, and other functions. They affect genes by directly damaging DNA and may alter their expression through epigenetic changes. Animal studies show they can also interfere with how fat cells break down lipids. A look at some of the impacts:

BRAIN

- Lower IQ scores by age 5 in children with the highest exposures.
- Anxiety, depression, or attention problems at ages 6 and 7.

AIRWAY

 Increased odds of asthma at ages 5 and 6 when exposure is combined with secondhand smoke

BODY FAT

 Two times the risk of obesity by age 7 for those with greatest prenatal exposure. Excess weight due to fat not muscle mass.

CANCER RISK

DNA damage and structural changes in babies' chromosomes. Both are known to raise the risk of cancer later in life.

From Science to Policy: SEEKING ENVIRONMENTAL JUSTICE

IN THE WINDOW ON THE SECOND floor of a former police precinct building on 152nd Street and Amsterdam Avenue, a tapered metal cylinder hangs over the sidewalk. It's not a wind chime or a weather station, but the intake for an Aethalometer, a sophisticated device used to measure PAH-laden airborne black carbon. The device, installed by Mailman School researchers, is stationed in the corner of the conference room of the community environmental group We Act for Environmental Justice. Its measurements are no secret—a Twitter account (@aethan) blasts out timely readings in idiosyncratic style: "G-Willikers, Batman! Look, the We Act Aethalometer reading is at 532-nanograms per cubic meter."

The dynamic duo of Mailman and We Act had its origins in the mid-1990s, when public health graduate students teamed with the We Act Earth Crew youth interns in a street-level study of air pollution in Northern Manhattan. They found a high degree of variation, with hot spots on busy thoroughfares, such as 125th Street, and near bus depots. "Until then, regulators assumed that you could get a picture of air pollution in the city using a couple of rooftop monitors. Our work dispelled that notion," says Environmental Health Sciences Professor Pat Kinney, who led the study. "Turns out, it really matters what's happening on the ground."

The research gave credence to what many, including We Act Executive Director Peggy M. Shepard, knew firsthand: Northern Manhattan neighborhoods and low-income people of color were disproportionately burdened with air pollution. Shepard co-founded We Act in 1988 to lead a community struggle for environmental justice pitted against such glaring disparities as the fact that the neighborhoods north of 99th Street were home to six of the city's seven diesel bus depots. "Northern Manhattan communities were being treated as New York City's dumping ground," says Shepard. "Communities of color were being excluded from democratic decisionmaking about issues affecting their health."

We Act's work with Mailman became a blueprint for new kind of community-academic partnership. It is a cornerstone of the mission of the Columbia Center for Children's Environmental Health and written into grants from the EPA and NIEHS that fund 14 children's environ-

mental health centers across the country. These partnerships call for scientists and community leaders to join forces in translating the research and getting the word out—often in bilingual brochures and information sessions—on how to protect children from air pollution and other risks.

But to really protect kids takes more than education; it takes new urban policies. That's where the rubber met the road in pairing Center science with We Act advocacy. The most visible source of pollution was the city's diesel buses. One-third of the fleet-the largest in the nationdrove in and out of depots in Northern Manhattan, their diesel engines emitting 50 times the pollution of a gas-powered bus. Armed with Mailman School scientists' data, We Act launched a campaign against "uptown dirty diesel" that, between 2000 and 2006, encouraged the city to convert its fleet to engines powered by compressed natural gas, diesel-electric, or ultra-low-sulfur diesel. Today, not a single city bus belches black diesel smoke-a change that helps the entire city.

"Scientific research gave us the muscle to make that happen," says Shepard. "The evidence was incontrovertible: Diesel exhaust increased the threats of a whole range of health problems from asthma to developmental delays, threatening the health of over a half million pre-



NYC CLEAN-AIR BUSES

MAILMAN SCHOOL RESEARCH HELPED PERSUADE THE CITY TO CONVERT ITS BUS FLEET—THE LARGEST IN THE NATION— FROM DIRTY DIESEL TO A MIX OF CLEANER FUELS.

> dominately African-American and Latino New Yorkers in Northern Manhattan neighborhoods."

> Greening the urban environment has been a priority for Mayor Michael Bloomberg, whose administration has acknowledged the contributions of the Columbia Center for Children's Environmental Health. "They created a scientific basis for policy development, " says Daniel Kass, deputy commissioner at the city's Department of Health. "The research on PAHs, the vulnerability of children, the association of exposures with birth outcomes, and later even causal relationships—all these things drive policymakers to recognize the value of trying to intervene where it's possible to prevent exposures."

> In 2011, the city announced Clean Heat, an initiative to finally end the use of dirty, low-grade residual heating oil. More than 10,000 residential buildings and 200 public schools will make the switch to cleaner fuels. This summer, City Hall announced \$100 million in financing to help older apartment buildings make the transition. To hasten the change in Northern Manhattan, We Act has hired an organizer to reach out to tenants, building owners, and superintendents. For Mayor Bloomberg, Clean Heat is a point of pride. Other than the city's anti-smoking legislation, he calls it "the single biggest step that we've taken to save lives."