



**For Rachel Altmann '88 and Tyler Morrison '90, life changed in waves of sound and light.**

The couple's first child, Benjamin, was born big and healthy in 1996. So Altmann and her doctor were both surprised in 2003 when, after seven months of pregnancy with her second child, Nina, Altmann's belly quit growing. The obstetrician had just bought a new ultrasound machine and decided to test it out.

"It was a very scary picture," says Morrison. "You could see the skull, and the whole center was filled with black fluid. I thought, 'My baby has no brain.'"

Until a neurosurgeon operated right after birth to relieve excess spinal fluid, no one could know how much Nina's brain would bounce back.

"That whole first 24 hours was awful," Altmann remembers. But it was just the introduction to a tangle of crisis, confusion, and hope that will stretch through Nina's life.

Nina has Fanconi anemia, a rare, recessive genetic disease that strikes about one in 360,000 newborns in the United States. The illness cripples an important DNA-repair system, leaving patients at high risk for cancers and aplastic anemia, a failure of the bone marrow to make new blood cells. While a few Fanconi anemia patients reach middle age, others die in childhood. The average life expectancy is 22. Treatments are risky and there is no cure, not even a good theory how to fix a problem built into every cell of the body.

The illness can wrap families like Nina's in confusion, isolation, and loss, says Nancy Cincotta, a social worker at Mt. Sinai School of Medicine in New York. "It hits you so hard it kind of challenges your

whole world. A lot of people define their lives as life before your child was diagnosed and life after."

Altmann and Morrison now live across that line. Nina's diagnosis means new routines: sleepless nights, endless blood draws and MRIs, and the knowledge a crisis will someday strike. It spurs them to support research into Fanconi anemia and to preserve Nina's quality of life. It has led Morrison to change jobs and Altmann to put off a return to the workforce. And it has reshaped the way they think of friends, family, and life.

"It has really opened my heart," Altmann says. "Nina walks into a room and everything changes. She just has something that sort of gets people's attention."

At two-and-a-half, Nina does look different. She's slight and knee-high, with glasses and a shuffle to her walk, all features of her illness. But her personality is what steals the show. She is cheerful, outgoing, and direct, bustling around the family's cozy wooden home telling mom when to make toast, asking dad to hold her upside down or take her upstairs to play a little foosball.

The family lives on a broad, windy hilltop above the Columbia Gorge in Corbett, Oregon. Toys flank the piano and books line shelves built into the ogive roof. Morrison, a software engineer, installed a solar-electric system to



ONE IN THREE HUNDRED SIXTY THOUSAND

BY ANDY DWORKIN  
PHOTOS BY LISA BAUSO

AS RESEARCHERS SEARCH FOR CAUSES OF A RARE GENETIC DISORDER, ONE FAMILY COPES WITH ITS EFFECTS ON THEIR GROWING DAUGHTER.



*Fanconi anemia has affected Nina Morrison's eyesight and physical development. She learned to walk late last year, after her second birthday.*

power his computers. He began installing a wind generator as well, but the turbine has spent two years in a box, a project delayed—like the half-built front porch railing—by the demands of Nina's illness.

"You don't have the energy and emotional resources for a lot of things you did," Morrison says.

Coming out of Reed, the duo took a typically atypical path to this home.

Morrison grew up in Hawaii, where his mother worked for social service agencies. His father was a musician, artist, and architect who used traditional tools to restore an old hotel on the slopes of Kilauea into the Volcano Art Center. Morrison worked alongside. At Reed he took a passel of math and physics courses before majoring in psychology. For his thesis, he programmed a Space Invaders-type game to test players' reactions to varying levels of rewards, a humanized version of running rats through mazes.

Altmann split her childhood between Chicago and Kenya, where her field-biologist parents studied baboons. She majored in music at Reed, writing a thesis on African American gospel music and Mahalia Jackson. She met Morrison through the Reed College Sound Collective.

After college, Altmann worked at a Portland radio station and got her master's in teaching. Then she

and Morrison moved to Hawaii, where he ran his own software company and she worked her "dream job" as an environmental educator at the Hawai'i Nature Center. In October 1997, they brought baby Benjamin back to the mainland and bought a used 40-foot city bus. Morrison ripped out the seats so the family could live up front, and wired in solar panels to turn the back into a rolling software lab. For months, they wandered the country, stopping for Benjamin to play by a lake, or for Morrison to send code via the internet. In 2000, they parked the bus in Corbett.

"The thing we realized, going to all these places, was Portland was as good a place as any," Morrison says.

Portland is better than most places. The Fanconi Anemia Research Fund ([www.fanconi.org](http://www.fanconi.org)) is based in Eugene (University of Oregon President Dave Frohnmayer and his wife, Lynn, started the group in 1989, after their daughters were diagnosed with the disease). Researchers at Oregon Health & Science University in Portland have helped track down genes behind the condition and are studying possible treatments.

Surprisingly, with so much local expertise, it took doctors almost two years to diagnose Nina. She was delivered in April 2003—at 37 weeks and just over



4-1/2 pounds—with a constellation of problems that implied some genetic disease. But “her pediatric geneticist said he didn’t know what she had, and we’d probably never know,” Altmann says.

Nina was born with crossed eyes and corneas clouded an opaque white. “I spent a long time thinking she was going to be blind her whole life,” Morrison says. There were neurological and digestive tract abnormalities. And her thumbs were small and slightly out of place, a detail lost in the medical shuffle. “We kept asking people to look at the thumbs,” Morrison recalls. “They never did, or did and said not to worry about it.”

Over the next year, surgeons operated four times. A scan revealed normal brain anatomy. Morrison put the image on the family’s website with the caption, “My baby has a brain.”

“I had started to think: If she’s going to make it, which it looks like she is, she’ll be okay,” Morrison says. “Maybe it’s just been a crappy two years.”

The relief was short-lived. Nina showed little interest in food, so her parents began dishing up homemade avocado ice cream and substituting heavy cream for milk to boost her caloric intake. In January 2005, Nina was admitted to Doernbecher Children’s Hospital to be fed through a tube.

In the hospital, Altmann asked for a genetics consultation. Dr. Markus Grompe, one of the world’s top Fanconi anemia experts, took the call. He read Nina’s chart, then asked to see her. Nina was asleep, and Altmann didn’t want to wake her. “Well,” Grompe said, “just tell me about her thumbs.” Altmann’s jaw dropped.

Some Fanconi patients show few symptoms. But many, like Nina, have an array of defects including kidney and digestive problems, small stature, and malformed thumbs. “I was kind of blown away that she hadn’t been diagnosed previously,” Grompe says.

It is unclear why Fanconi anemia causes such diverse problems. But in the past decade, scientists have uncovered some of the disease’s secrets.

They now know there are roughly a dozen disease subtypes, each pegged to a different mutated gene. Those genes normally help find and fix broken DNA strands. Since DNA frequently breaks during replication and protein production, such genes are crucial for stopping biological chaos, especially cancers.

Nina has one of the rarer forms of Fanconi anemia, recorded in only a couple dozen people. “All indications from those two dozen patients are that this is by far the worst kind of Fanconi anemia you can have,” Altmann says. Nina’s hematologist, Dr. Peter Kurre, calls her prognosis “really fuzzy.”

What is clear is that Nina will eventually face a crisis—either anemia, or a blood, brain, or kidney



Amy Frohnmayer, who has Fanconi anemia, headed off to Stanford University last fall. Two of her sisters have died from the disease.

## RESEARCH

### on the cutting edge

**W**hen Amy Frohnmayer started kindergarten in the early 1990s, no one knew which genes caused Fanconi anemia, the disease she shares with roughly 3,000 people worldwide, including Nina Morrison.

By the time Amy started college last year, scientists had identified 11 genes, and had begun to understand how these genes normally defend healthy bodies against disease. That research—supported by the Oregon-based Fanconi Anemia Research Fund founded by Amy’s parents, Dave and Lynn—has inspired some optimism for families facing a disease that kills most patients by their twenties.

The condition was first described in 1927 by Swiss pediatrician Guido Fanconi. Important scientific breakthroughs came when researchers realized that all patients were sensitive to a type of genetic damage called crosslinking, which occurs when the bases that form the DNA molecule stick together during replication. That leads to constant breaking of DNA strands, something most people fix well, but not Fanconi patients.

Normal Fanconi genes help find and fix crosslinked DNA. Intriguingly, these genes somehow interact with BRCA1, a gene that, when mutated, predisposes carriers to breast cancer. BRCA2, a related gene, adds breast cancer risk with one mutant copy, and can cause Fanconi anemia in people with two mutant forms.

In the long run, people without Fanconi may benefit more from the research than the sick children involved. The links to adult cancers make Fanconi a kind of lab for studying the processes behind those conditions. Other cutting-edge research “may make Fanconi Anemia a poster child for where to take gene therapy next,” says Oregon Health & Science University’s Dr. Peter Kurre, who treats Nina Morrison.

The Fanconi Anemia Research Fund (541/687-4658, [www.fanconi.org](http://www.fanconi.org)) provides seed money (\$1.3 million in 2004) to scientists testing ideas too new to draw major government grants, says Lynn Frohnmayer. The group hosts a summer camp, and publishes handbooks for families and doctors. The Frohnmayers founded the group after their daughters were diagnosed; complications killed Katie Frohnmayer in 1991, at age 12, and Kirsten Frohnmayer in 1997, at 24. Amy, 18, is doing well, and is in her freshman year at Stanford University.



*Benjamin, age 9, Rachel, Nina and Tyler live on a former farm near the Columbia Gorge in Corbett, Oregon.*

cancer. And then, Nina's illness will make treatment risky. The Fanconi defect makes cells highly sensitive to damage from radiation and many cancer-fighting drugs. Marrow transplants can fix aplastic anemia and prevent leukemia, but they hand families a dilemma. The transplants work best when patients are relatively healthy, but the immune system has to be suppressed to reduce the risk of rejection. Fanconi anemia patients face a 40 percent risk of death within a year of transplant.

"It's an exceedingly difficult decision," Kurre says. "You look at your healthy child and say, 'Is it time? Or do I wait a year?'"

Such uncertainty gnaws at Morrison and Altmann. "I'm a data-driven person," he says. "Give me all the data, I'll analyze it and make a decision." But with Fanconi anemia, "there's not enough data . . . It kind of leaves you feeling utterly helpless."

That feeling of helplessness hits other areas of life, too. The couple are concerned that the family could be subject to genetic discrimination. Paying for Nina's care has also brought struggles. While the family had a major medical policy during her birth, the insurer tried to make them pay \$40,000 of the nearly \$100,000 hospital bill. Morrison spent several months fighting that charge, then tried to find full medical coverage. But no insurer would take on a low-profit patient like Nina. Finally, Morrison suspended his software business to work for Extensis, a Portland software company whose group health policy will enroll a child with a major illness.

The reaction to Nina's illness among some longtime acquaintances also surprised the couple. When Altmann first emailed a dozen mothers in a playgroup about Nina, only four responded. "I have

a friend I have known since I was five," Altmann says. "She's never acknowledged anything to me."

The barrage of problems would make it easy to give in to fatalism, and Altmann and Morrison admit they ride an endless emotional roller coaster. But even at low times, Nina herself inspires love and hope. "The biggest positive we focus on is Nina," Altmann says. "She is so cheerful, even when she's sick—joyful, energetic, funny, and phenomenally cute."

That spirit has inspired friends and strangers to offer everything from kind words to donations. Last summer, the playgroup came through, organizing a "Tiny Tot Trot" walk and lunch to raise funds for research. Altmann and Morrison have met others facing the same illness through an Internet list-serve and a camp in Maine for Fanconi families.

"You form a different family, a family you didn't want to belong to," says Lynn Frohnmayer. She adds that, in her view, Fanconi patients see their world more with hope than despair. At a recent camp, five teens spoke about their experience. Four of them, she recalls, said they "felt lucky" to have this disease. "They felt they had been forced to view every day as a precious gift."

Morrison and Altmann may not feel lucky. But Nina's illness has made them think about life in new ways.

"I have a hard time with an omnipotent deity that makes innocent children suffer," Morrison says. But, he adds, Nina "touches people spiritually, emotionally. Maybe people who aren't going to live as long as most of us pack the same amount of spiritual connectedness into a shorter time, so it's more intense."

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