YOUR TICKET TO
CLINICAL RESEARCH
Emory offers more than 1,300 research trials to patients 10

YOUR BRAIN
ON ECONOMICS
New neuro tools for policy-makers 14

VACCINES AND AUTISM
Is there a connection? 24

autism triggers
tracking genes, one family at a time 2
The best of times, the worst of times

No matter whom you talk to, the economy is the topic on everyone’s mind. With the stock market down and unemployment and home foreclosures up, the country is facing an economic downturn of historic proportions. The current state of national and international affairs has been characterized by many as a crisis, but it’s more appropriate to view it as the beginning of a major socioeconomic environmental change.

All of us have lived through major national and international crises associated with significant social and economic changes. The Great Depression, World War II, the Cold War, Vietnam, and 9/11 each affected society and our personal lives, but we survived them, often emerging stronger as a result. Unfortunately, the worst part of such periods of change is at the beginning, when we just don’t know how things will turn out. We are at such a point now—in the world, country, Atlanta, and certainly at Emory and the Woodruff Health Sciences Center (WHSC).

That’s why it is critical now more than ever that we steward our resources wisely and effectively. That means ensuring that our faculty and staff—the most valuable of all our resources—as well as our spaces, funds, and programs are employed in a way that meets the principles of prioritizing our goals, aligning sources with uses, and ensuring full transparency. In this issue of Emory Health, you’ll see highlights of the many ways in which WHSC is making the most of its resources to continue providing extraordinary advances in research, education, and patient care.

In so many ways, one could consider the current environment as the worst of times. But I hope as you read this issue, you’ll feel as I do—that this is also the best of times, a time when we’re fine-tuning our focus and strategically channeling our resources toward achieving our goals.

During these challenging times, the question each of us should really be asking is, “Where would I rather be in this environment?” For me, the answer is pretty simple: I’d want to be at an organization involved in health and education. I’d want to be at a place with a noble mission, inspirational vision, and high aspirations. I’d want to be at a place with unique assets and partners that provide great potential for success. That’s why there’s nowhere I’d rather be than here in Atlanta, at Emory, at the WHSC.

Thanks, as always, to you—our faculty, staff, students, alumni, and community supporters—for your continued leadership and friendship as we work toward transforming health and healing ... together.

Fred Sanfilippo, MD, PhD
Please share your feedback at evphafeedback@emory.edu.

The current state of national and international affairs has been characterized by many as a crisis, but it’s more appropriate to view it as the beginning of a major socioeconomic environmental change.
Emory geneticists and clinicians are working with families to unravel autism and its triggers one gene at a time.

At 18 months, Griffin Hatcher had missed some common developmental milestones. He wasn’t speaking by 2. At 3, he consistently ignored other children, preferring to lie on the ground and look at the spinning wheels of his toy car. His pediatrician said the boy was fine. So did another doctor. But Griffin’s parents, Molly and Brent Hatcher, insisted on a referral.

When the Hatchers arrived at developmental pediatrician Amy Pakula’s office, their journey—self-described as an “unpredictable, ever-changing, never-ending process of having a child with autism”—picked up speed. Pakula, on faculty at Emory and Marcus Autism Center, told them that while she could offer no cure for autism, they could work together to tackle the problems it produced.

Treatment with Prozac caused neighbors to ask why the boy suddenly seemed less anxious and more engaged. Griffin’s days filled with therapy—for speech, the activities of daily life, social interactions, and even occupational therapy on horseback. Following an individualized program prepared by Pakula, the 4-year-old entered a special preschool near his Flowery Branch home, where he could mix with typical kids.

“Amy never sugar-coated anything, and she went after every problem decisively and aggressively,” says Brent, a lawyer. “We felt a tremendous sense of relief and direction, like we now had a team.”

When it was time for public school, Pakula prepared another program, outlining in detail the support Griffin would need, including a paraprofessional to accompany him to some classes. It was up to the Hatchers to persuade the school system to accept—and pay for—this plan. They requested detailed reports from everyone who had worked with Griffin, and an outreach specialist from the Emory Autism Center reviewed their case. Griffin’s therapists attended the successful meeting with school officials.

Now almost through his first year of public school, Griffin can read, his handwriting is improving, and he performs better socially. On weekends, he asks to go to school. He enjoys his brother Davis, a
Two doctors told Molly Hatcher her son, Griffin, was fine, but she insisted on a referral. An Emory physician confirmed what she had suspected. Griffin had autism.

Joseph Cubells was one of the first scientists to point to a connection between a deletion syndrome known as 22q11 and autism.

Christa Lese Martin performs studies that identify specific chromosomal duplications in families who participate in an autism gene bank.

Stephen Warren discovered the fragile X gene can cause one form of autism and is now working to identify a drug treatment for the faulty gene.

David Ledbetter believes scientists could end up identifying more than 100 different kinds of autism, each with a different genetic basis.

Behind the science

When autism was first described in the 1940s, the blame often was assigned to emotionally distant "refrigerator" mothers who failed to properly bond with their babies. That long-disproved theory—and the pain and guilt it caused so many parents—was one of medicine’s most shameful mistakes, says Emory psychiatrist and geneticist Joseph Cubells.

Today, the only known causes of autism are genetic. Increasingly precise genetic assessments, like those being developed at Emory, are correcting another earlier misconception: autism is not a single disorder, any more than cancer or mental illness is. Rather it is a neurobiologically diverse group of autism spectrum disorders (ASDs). How many are there? Emory geneticist David Ledbetter says that scientists could end up identifying 100 or more different kinds, each with a different genetic basis and each accounting for just a small percentage of total cases. His research, and that of colleague Christa Lese Martin, focuses on genes or chromosomal events that by themselves produce a specific ASD. So far scientists have found at least 15 of these, including the fragile X gene discovered by Emory genetics chair Stephen Warren. Most geneticists now believe that many ASDs result from the interaction of multiple faulty genes, many not yet discovered. To complicate the impact even further, some of these genes are likely to be “susceptibility” genes that not only must interact with each other but also with one or more environmental triggers.

Complicated, says Ledbetter, but encouraging. “We are just beginning to understand how specific genotypes can result in different autistic phenotypes [similar patterns of cognitive, linguistic, and behavioral problems]. That has immense implications for diagnosis and personalized intervention—both behavioral, and eventually, pharmaceutical.”
The Emory Autism Center (EAC), with the largest staff of specialized autism providers in Georgia, offers diagnosis, family support, and treatment as a vital source of professional training. Last year, the center provided clinical care to more than 840 children and adults and strategies for families to reinforce learning at home and in the community. The center’s specialists also consult with community physicians and community and state entities.

The center’s Walden Lab School frequently is cited as one of the nation’s top five models of early autism intervention, based on integrating children with autism into classes with typically developing toddlers, preschoolers, and pre-kindergarteners. The EAC’s Monarch Program provides training and consultation services to schools K–12 that want to improve their autism services for adolescents and adults, including Asperger’s support groups.

Research at the EAC focuses on early intervention, including teaching and social conditioning, work that has changed how autism is treated across the country. EAC researchers also collaborate with the CDC, investigating reports of increased autism prevalence. psychiatry.emory.edu/clinical_sites/autism_center.cfm, 404-727-8350.

The family array
The idea of using genetics to define a disorder is not new. In the 1960s, after scientists found the extra chromosome that causes Down syndrome, they began to discover chromosomal imbalances characterizing specific syndromes almost monthly. Today, thanks to the vast amount of genetic information available from the Human Genome Project and to new high-throughput cytogenetic microarray technology, a similar renaissance is occurring for autism. For example, Emory scientists developed one such technology, EmArray, now used in a clinical laboratory consortium of more than a dozen U.S. laboratories and 40 additional labs in Canada, Europe, and South America.

Microarrays are glass slides or silicon chips to which nucleic acid probes are chemically attached. With these arrays, scientists can examine thousands of genes simultaneously, detecting both chromosomal additions and deletions and subtle differences in genes and gene expression. The Emory Genetics Laboratory each year provides microarray genetic testing for more than 2,000 children with developmental delays referred by physicians from across the country. Of those, approximately 400 are related to autism. Emory lab was one of the first to use cytogenetic microarray testing for autism. Using this technique and the current state of knowledge about genetic bases, a specific genetic diagnosis for autism can be identified in 5% to 8% of cases. For others, such as Griffin Hatcher, a specific genetic diagnosis remains elusive.

Knowing where to look
But maybe not for long. Families who are willing to share medical information and genes are invaluable to progress in understanding autism, says Ledbetter. The Autism Genetics Resource Exchange (AGRE) was the first collaborative gene bank for autism. More than 1,000 families—a majority of “multiplex” families with two or more children diagnosed with ASD—have contributed clinical and genetic information, including DNA samples, from both affected and unaffected family members to the gene bank. Warren serves on the Autism Speaks board, while Ledbetter serves on AGRE’s scientific board. Ledbetter, Christa Martin, and a colleague from UCLA perform cytogenetic and molecular studies that identify specific chromosome duplications among families who participate in the gene bank.

A second repository—the Simons Foundation Autism Research Initiative (SFARI)—focuses on “simplex” families, who have one child with ASD. The initiative collects clinic-based assessments and genotyping on the child with ASD as well as the parents and unaffected siblings. Among other research, the initiative sponsors a family collection study led by Emory’s genetics department, the Emory Autism Center, and Marcus Autism Center (an affiliate of Children’s Healthcare of Atlanta that works with children with developmental disabilities and collaborates with Emory, Georgia Tech, and others). As the only SFARI program in the Southeast, Emory is recruiting 150 families from those seen in Atlanta facilities or found through local autism support groups, school psychologists, and other community activities. In all, SFARI hopes to recruit 2,000 simplex families. As an example of their power and cooperation, the databases are allowing Warren and his colleagues to conduct a study to determine whether genetic variations on the X chromosome play a larger-than-suspected role in causing autism. One reason to think they might be is the fourfold excess of males with autism, a pattern that could arise because males, with only one copy of the X chromosome, would be more likely than females to be affected by X-linked mutations. Another reason is that the X chromosome is home to a large number of genes (including fragile X), whose mutations are known to cause problems with brain development.

The study, funded by the Simons Foundation, looks at the entire X chromosome of 300 autistic males, using microarray-based genomic selection technology developed by Emory geneticist Michael Zwick. The ambitious three-step process begins with identification of all deletions, duplications, and gene variations on each boy’s X chromosome. Next comes a resequencing (or comparison to the human genome) of the coding portion of every gene, thus detecting any variations in how each gene “codes” or instructs for the production of the protein that carries out the function of the gene. Finally, the research focuses on epigenetic
That's why she recommends that families undergo genetic testing for known causes and why she encourages families to participate in family databases.

"I cannot do my job in the clinic without the genetics, and they cannot gather the data they need without the help of the families," Pakula says.

Not just for kids

Most autism research and organizations focus on children. But these kids grow up. Every week, psychiatric geneticist Joseph Cubells sees a handful of autistic adults, and he suspects some people with the most severe cases went undiagnosed in earlier eras and today are either confined to mental institutions or among the homeless.

Although Cubells sees many patients with Asperger's (a form of autism with normal intelligence), sometimes above normal, language and intellectual development), most of his ASD patients come from physician referrals for co-existing psychiatric problems, from depression to psychosis. The challenge of getting feedback from often linguistically impaired patients, coupled with a paucity of research on psychotropic medications in adults with ASD, makes his job a big one. Many patients arrive already taking a potentially toxic mix of four or five prescriptions accumulated over the years by a series of clinicians desperate to do something.

"One of the rewards of working with adults with autism," says Cubells, "is that it almost invariably involves working with families. Patients whose families stayed involved generally do better."

Families also are helping researchers learn what to expect from patients with similar genotypes, says Cubells. Take, for example, the deletion syndrome known as 22q11, for which Cubells and colleagues at Emory and Children's Healthcare of Atlanta have developed a new clinical. Affecting one in 4,000, the chromosomal anomaly is second only to Down syndrome in frequency of occurrence but far more variable. It's one of the 15- or so known causes of autism, and more than a third of patients with the deletion have an autism diagnosis. An even higher number exhibit autistic-related problems of communication, social interaction, and stereotype behavior.

An unknown number of people with the 22q11 deletion are "clinically silent," at least in early years, but they may develop serious heart, renal, immunologic, and endocrine disorders. Schizophrenia also may emerge in up to 30% of these adults, something not seen in other forms of ASD.

A study by Cubells, co-investigator Opal Ouelay, and other Emory and Children's researchers was one of the first to point to an overlap between the 22q11 deletion abnormality and autism. Currently, the researchers are comparing autistic features in patients known to have 22q11 deletion syndrome with autistic features in patients with other chromosomal disorders that show a high rate of autism, such as fragile X. It is already known that the deletion is often characterized by poor early language development.

"If these studies show other systematic character-istics, we'll be closer to understanding the relationship between specific genes and certain behaviors," says Cubells. That could lead to better diagnoses. For example, Ouelay already is working on possible biomarkers for this form of autism and others as well as personalized behavioral and educational interventions. Eventually, scientists believe this genetic understanding could identify drug targets to counteract the actions of a specific genetic change.

The fragile X hope

There's a long way to go before that happens, but Stephen Warren sees hope. He may be on the verge of identifying a drug treatment to compensate for a faulty fragile X gene, known to cause one form of autism. Five percent of people with autism carry this gene. Furthermore, 30% of people with fragile X meet the criteria for autism in the Diagnostic and Statistical Manual, the bible for practitioners diagnosing psychiatric disorders, and 80% of those with fragile X show autistic traits, such as repetitive behaviors.

Soon after discovering the fragile X gene, Warren discovered the protein it produced. That enabled him to develop the first biologic diagnostic test and better explain why fragile X could have such a wide range of effects, depending on how much protein is produced by the damaged gene.

It also allowed him to identify a compound that causes fruit flies with a fragile X-like disorder to give up their fragile X-type behaviors and develop more normal biochemistry and brain wiring (Nature Chemical Biology, April 2008). Getting a drug from the lab to patients can take as long as 15 years, so Warren deliberately tested only compounds that already had survived FDA safety testing. With that hurdle eliminated, he anticipates clinical trials could begin in humans within the next two years. If the compound works as well in humans with fragile X as it has in the insect model, this treatment could open up for many other forms of ASDs.

When autism was first recognized, moms got the blame. Today, say Emory researchers, mothers and fathers deserve much of the credit for advances in under-standing autism and treating this disorder. As advocates, lobbyists, spokespeople, and almost universal participants in the family-initiated AGRE and SEARR programs, families touched by autism are researchers' biggest supporters.

The unusually strong alliance of scientists, clinicians, and families comes at a time of incredible new research tools, says Warren. For understanding autism, it's the perfect storm. The Human Genome Project and technology that can spot differences in individual genomes, such as EmArray and Zwick's genomic selection microarray, enable scientists to take data provided by families and chip away at autism, one ASD subset, one chromosomal unreliability, one gene, at a time. What they are finding may help personalize treatment and, eventually, as Warren's fruit flies suggest, mitigate, even prevent its problems.

more autism resources

An affiliate of Children's Healthcare of Atlanta, Marcus Autism Center works with children with autism and related disorders. The center diagnoses and treats children with a wide range of neurologic problems, including autism spectrum disorders. They work with parents to find ways to help children cope with their disability, including carefully managed therapy to teach children to communicate barrier posed by the disability.

The Early Intervention Program at Marcus Autism Center is an evidence-based program for children, age 18 months to 8 years, with autism spectrum disorders, providing intervention services for families who share a goal of transitioning children into public or private school systems. The Pediatric Neurodevelopmental Center provides evaluation and treatment for children by developmental pediatricians, child and adolescent psychiatrists, social workers, and others.

The Marcus School offers educational programs for children who exhibit aggressive and disocial behavior and helping them learn to modify behaviors so they can participate in neighborhood schools and enjoy a better quality of life at home. Marcus and Children's collaborate closely with Emory, Georgia Tech, and others to provide autism resources for the community. marcus.org, 404-785-9400.

web connection:

To view a series of videos on autism and genes, see whsc.emory.edu/autism_vide.html.

Also visit the Emory Autism Center, psychiatry.emory.edu/clinical_sites_autism_center.cfm (404-727-8350) and the Emory Genetics Laboratory, genetics.emory.edu/ege/index.php (404-778-8509).
Early in his career, neurobiologist Don Stein noticed something unusual in studying the behavior of brain-injured rats. After a brain injury, some females seemed to recover promptly while the male rats did not. Some 40 years later, after much trial and effort, Stein was able to prove that progesterone, a reproductive hormone, explained why. The hormone seemed to slow or block damaging chemicals released after brain injury, protecting brain cells from destruction. Both male and female rats with brain injury developed less brain swelling and recovered better when they were treated with progesterone soon after the injury.

With the help of hundreds of volunteers, Emory is on the road to finding new medical treatments, clinical trial by clinical trial.

Miles

Miracle

By Quinn Eastman  •  Photography by Jack Kearse
“The major reason why studies fail is that not enough people are enrolled. We’re losing potentially valuable treatments because they can’t be adequately tested.”

—Carlston Dampier, medical director, Emory’s Office for Clinical Research.

As long as a decade ago, a federal review concluded that the consent process had become “a disclaimer for institutions rather than for information for the participant” and “may be inappropriate individually deterring participants from participating.”

Retired engineer McCamie Davis and his wife, Starla, are taking part in a clinical trial to see if a vaccine can slow progression of his Alzheimer’s disease. [1129x504 to 1313x720]
He has built a career researching what goes on in the brain when people formulate decisions. Now he's started the Center for Neuropolicy with the goal of giving policy-makers a step further in order to boost the effectiveness of policy-makers. If policy-makers understood how they make decisions, they could stop wasting time on ineffective legislation. (For example, cigarette taxes have been less successful in convincing people to stop smoking than social pressure, Berns says.)

Policy-makers tend to act when consequences come into the present. Berns points to global warming as an example. Many see global warming as a long-term problem, but so far governments haven't done much to stem it.

"Humans are myopic," he says. "We have a hard time conceiving of ourselves one year from now, much less 20 or 30 years from now. There's no incentive to make long-term decisions. That's a problem in this country."

Blame our myopic nature on dopamine, a chemical in the brain that either increases or reduces the activity of nerve cells. Dopamine is called the "pleasure chemical," but Berns tags it the "chemical of anticipations." Dopamine affects brain processes that control emotional response and ability to experience pleasure and pain. "Dopamine is like crack," he says. "It's here, and then it's gone."

Myopia, though, can be overridden by focused attention, Berns says. "We can make projections, but the question is how to bring them into the consciousness of the people you are trying to affect," he says. "I don't have the answer for that. Collective decision-making is political, but politics are biologic.

The human brain evolved to function in social groups. By discovering how our brains are wired to behave in group settings, we can begin figuring out solutions to problems of global impact.

Berns started making the rounds in intelligence and military communities in December. His center and the U.S. Air Force hosted a conference to spur interest in research collaboration. One topic of discussion was how abstract social values, such as religious and political ideologies, become distorted in the brain and subvert basic survival value judgments, which occur in war and terrorism.

Ideological values are "sacred," have no monetary value, and therefore are powerful in their emotional pull. Are there circuits in the brain that predispose people to fall into certain groups? Do political ideologies tap into those circuits?

The answers to those questions, Berns hopes, will lead to more effective government. We have the technology to solve our most daunting problems, but "the real problem is human nature," he says.

By Kay Torrance • Illustration by Stanisława Kódmán

**FeAture**

A FULL COURSE PLAN

By Kay Torrance

Illustration by Stanisława Kódmán

**The myth of rational thinking or HOW ONE EMMRY NEUROSCIENTIST WANTS TO HELP POLICY-MAKERS BY PEERING INTO THEIR BRAINS**

Greg Berns doesn't want you to make a decision by yourself. He doesn't trust you. People don't make rational decisions, he contends, and you are likely to muck it up. Don't be offended by his reasoning, though. He says that there are biological reasons why we all get it wrong.

Just by way of his resume, Berns would seem likely to make better decisions than a lot of us. He's a poster boy for overachievement, with degrees in physics, biomedical engineering, and psychiatry. He's written two books on decision-making. Yet he too knows that he makes decisions like everyone else. He's vulnerable to the influence of irrational thinking, the emotional sway that invades his—and everyone else's—reasoning.

all combined to develop findings around how the brain makes financial decisions.

Early on, neuroscientific researchers began looking at why people made financial decisions they did, mostly because economists had gotten it wrong.

Economists had long assumed that with proper information or instruction, people would make good financial decisions, systematically and without emotion.

"We know from studies that people don't make rational decisions," Berns says. "The problem with economic models is that they assume a certain level of rationality by people, that people will maximize their benefits."

Add into the mix that most of us don't know that we are irrational, says Emory economist Mónica Capra, who is a member of the center. "People believe they themselves are rational, even if everyone else isn't," she says.

"We want to minimize energy needed to make a decision so we use rules of thumb. We also tend to over-weigh low probability and under weigh high probability."

One case in point. Why do we still save so little and spend too much even when we know the consequences? Are we just not listening? To get some answers, economists began reading what the psychologists and neuroscientists were finding.

Since 2002, when psychologist Daniel Kahneman of Princeton and economist Vernon Smith of George Mason University were awarded the Nobel Prize in economics, neuroeconomics has been gaining traction. Centers for neuroeconomics have opened in universities in the United States, Europe, Israel, and Japan. The first scholarly neuroeconomic journal appeared in 2008, and another is planned. The first edited textbook on the subject appeared last fall, and the Wall Street Journal debuted its first neuro-economic columnist last July.

Neuroeconomists have essentially rede-fined some economic models, says Elliot Bendoly, an information systems and operations management specialist at Emory and a member of the Center for Neuropolicy.

"Economic models affect the debate in Congress, how we live," he says. "It's not that people are deviating from the ideal economic model, it's that the ideal is wrong. The hope is that as economic models develop, they incorporate human behavior."

How the brain affects policy

Borns would like to take neuroeconomics a step further in order to boost the effectiveness of policy-makers. If policy-makers understood how they make decisions, they could stop wasting time on ineffective legislation. (For example, cigarette taxes have been less successful in convincing people to stop smoking than social pressure, Berns says.)

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Home away from home

In the living room, heart transplant recipient Chester Howard listens to his son, Brandon, strum a guitar. Across the hall in one of three kitchens, Inga Boyce stirs a pot of goulash on the stove. On the upper floors, others relax, read books, or send emails to friends.

It is a typical night in the Mason Guest House, a private retreat that offers affordable lodging for transplant patients and their families. The house accepts guests dealing with any phase of transplant, whether they are getting an initial evaluation, undergoing a transplant, or waiting to transition to home after surgery.

Located on Emory’s Clairmont campus, the house welcomes transplant families from Emory and other centers in Atlanta. “We really wanted our house to have the intimacy of a home,” says Jennie Perryman, director of policy and outcomes management at the Emory Transplant Center. “We wanted our guests far enough away from the hospital to be comfortable but close enough to feel secure.”

The Mason Guest House opened in October 1995 with support from the Carlos and Marguerite Mason Trust. Emory donated the land, and the Masons donated $1.625 million in an initial gift and a $600,000 endowment for maintenance.

The Tudor-style Mason Guest House echoes what they learned. There are three floors, with 15 bedrooms with private bath, computer and exercise rooms, and a laundry. Guests are served a continental breakfast, and they can bring in their own food and cook. There are rooms for families to gather and socialize around a grand piano or television, or, as Perryman says, “places to just be.” Rooms come with twin or queen beds in décors from Country French to sleek and modern. The cost? A night’s stay is $35 for standard rooms and $80 for a two-bedroom suite.

The majority of guests, 60%, are Georgians, says Willie Skipper, who manages the day-to-day operations of the house. He can quote other statistics too. Many stay only one or two nights, with the average length-of-stay in 2008 being 11 days. Wednesday and Thursday are the busiest days, and the hours after 6 p.m. when families are returning from the hospital are the busiest times.

Backed by a team of guest services coordinators, Skipper makes sure that guests have what they need when they need it. He’s there to offer not only a comfortable, inviting house but also a welcoming smile and an ear to listen. Last year, the staff got a 99% approval rating.

“We all believe in the mission of the Mason Guest House,” Perryman says. “If there is a one-liner for what we want to create, it is a home away from home.”—Rhonda Mullen

Birdsong

On the ground floor of Emory University Hospital Midtown, 100 joyful voices greet patients and visitors at the north entrance. The little voices, music to human ears, belong to a group of exotic birds that reside in the hospital’s two aviaries.

“A visit to the aviaries is the same sort of ritual you make when visiting a favorite park or a favorite city,” says patient Wendy Darling. “Whatever else is going on, you make sure you visit your favorite place.”

Darling goes to the hospital once a week for allergy shots. “At times, it’s unpleasant, but in the end, I know that the birds will be there. Whether I get to watch them for a minute or half an hour, I always enjoy my time with them,” Darling says.

More than two dozen types of birds, from blue capped cordon bleu finches to frosted peach canaries, are housed in well-appointed, climate-controlled habitats. Their homes are complete with scenic murals, seasonal decor, toys, and a variety of fruits and vegetables straight from the hospital’s pantry. All the birds are exotic, and although they are indigenous to specific regions, such as the African grasslands, all are captive born and captive bred by a licensed breeder.

Donated by the Emory Crawford Long Hospital Auxiliary, the aviaries and their feathered residents are overseen by Shari Creech, manager of the hospital’s building support services. “After our former COO’s wife, Barbara, retired from the auxiliary in 2003, I was asked to step in and supervise,” says Creech. “I was thrilled, but I really didn’t know much about birds.”

To supplement her knowledge, Creech sought out the advice of local pet store owner and exotic bird expert Teri Chacon. Chacon continues to consult with Creech and Mattie Williams, who is charged with the day-to-day care of the birds. “Every three months, we do an entire inventory,” says Chacon. “Sometimes someone has a sore toe, or is molting and looking a little rough, they get put in a special cage in a special area and Mattie cares for them until they recover,” says Chacon.

Williams says she loves her job. “I make sure these little babies are clean, get the best food, and stay out of trouble,” she says. “They’re just like children. You turn your back on them, and they’re into something. If they fall out of a nest, I have to put them back.”

Williams has noticed that visitors love the birds, too. “People say the birds are calming and relaxing. It really does something for the visitors, their spirit and their minds.”

As for Darling, she’s particularly fond of the Rosey Bourkes parakeets. “Most of the birds are finches, small and flighty, whereas the parakeets are much larger, even bigger than lovebirds,” Darling says. “They have lovely gray and pink coloring and a sedate nature. Often, I’ll watch them sitting lined up in a row, observing all the other birds. I think I can relate to them.”—Robin Tricoles

 Alumni House

Located in the Mason Guest House with his son, Brandon, Below, Inga Boyce (right), whose son had a double lung transplant, and DeLoris Moore, who cared for her daughter after a heart transplant, were recent guests.

look for these birds...

Type: Finch

Genus: Erythrina

Size: 4 in. to 5 in. length
Average: 6 in. wingspan
Greatest Span: 6 in. wingspan
Living Span: 10 to 12 years
Field: Anywhere

Look for: Fancy fancy canary

Type: Canary

Genus: Serinus

Size: 2 in. length
Average: 4 in. wingspan
Greatest Span: 5 in. wingspan
Living Span: 10 to 15 years
Field: Anywhere

Look for: Gloster fancy canary

Type: Serinus

Size: 2 1/2 in. length
Average: 5 in. wingspan
Greatest Span: 6 in. wingspan
Living Span: 5 to 7 years
Field: Anywhere

Look for: Gloster gloster fancy canary

Type: Serinus

Size: 2 1/2 in. length
Average: 5 in. wingspan
Greatest Span: 6 in. wingspan
Living Span: 5 to 7 years
Field: Anywhere

Look for: Gloster gloster fancy canary

Type: Serinus

Size: 2 in. length
Average: 4 in. wingspan
Greatest Span: 5 in. wingspan
Living Span: 10 to 15 years
Field: Anywhere

Look for: Gloster fancy canary

Type: Serinus

Size: 2 in. length
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Greatest Span: 5 in. wingspan
Living Span: 10 to 15 years
Field: Anywhere

Look for: Gloster fancy canary
New application for an old technique

Many women who have uterine fibroids go through their days with no noticeable symptoms. They may even be unaware they have fibroids at all. However, for a small percentage who have symptoms, daily life can be interrupted continually by pain.

Uterine fibroids can cause a host of disruptive symptoms: unusually heavy or long menstrual periods, pain during sexual intercourse, pressure on the bladder leading to frequent trips to the bathroom, bloating, and pain in the pelvis, legs, or lower back. They affect 20% to 40% of women 20 years or older and occur in half of African American women. So far, doctors are unable to pinpoint why fibroids are more common in African Americans or why women develop them at all. But they do know that heredity and obesity are factors.

Women with problematic uterine fibroids traditionally have had only two options—a hysterectomy or a myomectomy (surgical removal of the fibroids). In fact, unwanted fibroid symptoms trigger approximately 150,000 hysterectomies each year.

Recently, an old technique is providing women who suffer with uterine fibroids with a nonsurgical alternative. Physicians have used embolization for more than two decades to treat pelvic bleeding of recovery that accompany hysterectomies.

An embolization is performed through a small puncture in a groin artery. Dye is injected into the artery to identify which blood vessels supply the uterus and fibroids. The radiologist then guides a wire and catheter into the identified vessels and injects small particles that block the blood supply to the fibroids. The fibroids and the uterus shrink approximately 60% in the first year. Heavy periods usually take a few cycles to lessen. The procedure takes approximately an hour followed by a day’s stay in the hospital for intravenous medication. Patients usually can resume normal activity after a week.

“Most of the women I’ve treated report a significant improvement in their symptoms at their first-month check-up,” Peters says.

—Kay Torrance

WEB CONNECTION To schedule a consultation for uterine fibroid embolization, contact 404-778-7777 or visit www.emory-healthcare.org.
An ounce of prevention

In the flu pandemic of 1918, Georgia’s first recorded case was reported on October 19. Soon after, the Atlanta City Council temporarily closed all public gathering places, including schools, libraries, theaters, and churches. By the end of the year, thousands of additional cases were reported in Georgia, as were hundreds of deaths. Millions died worldwide.

Today scientists have a far better understanding of the flu virus and are working to prevent another pandemic. At Emory, two groups of researchers and their collaborators recently developed two approaches to lessen morbidity and mortality associated with flu during a normal season. Both strategies show promise in mitigating a worldwide outbreak.

The impact of vaccines

A new predictive epidemiologic model shows that giving infants the currently recommended 7 valent pneumococcal conjugate vaccine (PCV7) saves lives and money during a normal flu season by preventing related bacterial infections. According to the model, the vaccine would prevent more than 100,000 deaths during a flu pandemic, while saving $7 billion in costs.

“We know, for years that bacterial infections can develop after influenza,” says global health professor Keith Klugman at Emory’s Vaccine Center and a Georgia Research Alliance Eminent Scholar. “We involved sifting through human blood to find those that made the right antibodies or vaccinating mice to ‘humanize’ the mouse antibody genes (altering them so they resemble human antibodies).”

Previously, methods for making human monoclonal antibodies had been laborious, says Rafi Ahmed, director of the Emory Vaccine Center and a Georgia Research Alliance Eminent Scholar. They involved sifting through human blood to find those that made the right antibodies or vaccinating mice to “humanize” the mouse antibody genes (altering them so they resemble human antibodies).

“Before just on more than one year, Olsen has been tending the field (and steering the ship) of the Emory Eye Center as director. It’s a challenging job. Besides administration and fund-raising, he does everything his faculty members do: patient care, teaching, and research.

Olsen’s resume shows a progression of honors, from his MD at the University of Minnesota to award-winning research on proteins of age-related macular degeneration (AMD), the development of new surgical instruments and methods, and six awards for distinguished teaching—at the universities of Kansas, Wisconsin, and Minnesota, as well as Emory.

An active physician-scientist, Olsen has an NIH grant to study the way proteins function in damaged retina cells. Additionally, he’s researching ways to replace those damaged cells with healthy ones from elsewhere in the retina, and in collaboration with biomedical engineers at Georgia Tech, he’s developing instruments especially for such surgery. Another ongoing project is his collaboration with Eye Center colleagues on the use of synthetic bear bile acids to treat diseases that can cause loss of vision, such as AMD and retinitis pigmentosa.

In the mid-1990s, Olsen spent two years on Emory’s campus as a retina fellow, specializing in diseases of the retina. The experience made him eager to return.

“Training at Emory gives you a deep appreciation for the culture of this place,” Olsen says. “Emory offers one of the most challenging ophthalmology programs in the country, but the people here work well together and share their knowledge generously. It’s not surprising that many of our trainees, like me, want to come back and join our faculty.”

On his 2008 return to Emory, Olsen inherited a program that ranks in the top 10 in the country. His vision for the program of its future, like his job itself, is wide-ranging.

“While training the future, we need to keep pace with a large and growing number of patients, Olsen plans to move forward on other endeavors close to his heart, including institutional partnerships with the CDC and Georgia Tech. Research initiatives already under way may include the Ocular Oncology Group, which combines the expertise of ophthalmology with research at Emory Winship Cancer Institute, and the Predictive Health Group, a team of ophthalmology faculty helping develop genetics profiling. Ultimately, all these efforts benefit patients, Olsen says.

Olsen’s lifelong, farm-influenced habit of rising early brings him to the office around 4:45 a.m. “Since age 7, when I came out of the doctor’s office with my first pair of glasses and could finally see clearly, I’ve wanted to help people see better,” Olsen says.—Ginger Pyron
The Fonda Center is approaching the challenge on multiple fronts, from sex education to violence prevention. It has developed a curriculum to help young people make smooth transitions to adulthood that youth grow up in social environments marked by violence, in which they learn to respect their bodies as a health problem nationally and in Georgia. Intimate partner violence, a serious public health problem, affects nearly 1 in 9 women and 1 in 11 men. According to the 2007 report from the Georgia Department of Human Resources, one out of every six Georgia high school students was hit, slapped, or physically hurt on purpose by a boyfriend or girlfriend during the prior 12 months. The Fonda Center will offer the program statewide.

Another recent grant, from the Robert Wood Johnson Foundation (RWJF), allows the Fonda Center to teach youth how to develop healthy relationships and prevent intimate partner violence, a serious public health problem nationally and in Georgia. According to a 2007 report from the Georgia Department of Human Resources, one out of every six Georgia high school students was hit, slapped, or physically hurt on purpose by a boyfriend or girlfriend during the prior 12 months. The Fonda Center will offer the dating violence prevention program to 7th graders in the Atlanta Public Schools. The effort will be supported by a national social marketing campaign from the RWJF and multiple local agencies who are banding together to reduce teen dating violence.

As an ongoing effort, each summer the Fonda Center trains 60 Atlanta teen leaders from local high schools to deliver an abstinence curriculum for 8th grade classrooms, under the guidance of staff from Grady Hospital's teen services.

“In high school, Lehr was happy to serve as her assistant, helping others become connected to health when she met a faculty member, Frances Nagata, who also worked as a sex therapist. “She was the first person in my life who ever talked openly about it,” Lehr says. When Nagata started the sexuality class in the nursing school, Lehr was happy to serve as her assistant, helping others become more comfortable with the topic. When her mentor left in 1984, it was a natural fit for Lehr to take on full responsibility for the class. Since then, the course has evolved to address current issues and new information. Transgendered sexuality and homosexuality have become major topics in class, along with a range of other sex-related topics that often connect to patients and nurses: sexuality and disability, rape, and sexuality and spirituality.

While students become more comfortable talking about sexuality, they also gain empathy and information, as when they had an opportunity to talk with a guest speaker in a wheelchair about his sex life. “I think that's really important for the students to hear,” says Lehr. “Then when they see other people in wheelchairs, they know there may be some very similar concerns and issues for them.” —Dana Goldman

WEB CONNECTION
For more information on the NCI designation, see who.cancer.gov/about-nci/slides.html. A related video is available at who.cancer.gov/about-nci/videos.html. An audio slide show is available at who.cancer.gov/about-nci_videos.html.
When a child is diagnosed with a neurodevelopmental disorder, it’s understandable that parents sometimes ask what they could have done or not done during pregnancy, childbirth, or the baby’s infancy. I spend a lot of time talking with families about the many possible causes about which they worry, many gleaned from the Internet. It is rewarding to be able to assure them that they are not at fault. The causes of these disorders are largely unknown, and therefore without currently recognized preventive measures.

Do vaccines cause autism?  
An Emory pediatrician follows the evidence

By Amy Pakula, MD

Because of the extensive public awareness that surrounds autism, parents are less likely to look internally for a cause and more likely to look externally—to diet, the environment, and especially the vaccines that children receive. Concern over a possible role for vaccines in causing autism has immediate and potentially life-changing implications for parents, and describe the science in causing autism has immediate and potentially life-changing implications for parents, and take the time to explain the science.

I explain to families that we are only at the beginning of understanding the basis of this puzzling disorder. Most scientists believe that many, if not most, forms of autism will turn out to be caused by a mixture of errant genes, a few that have a specific effect by themselves, some that must be present in a specific combination of mutations, and some that are “susceptibility” genes that require a specific combination of mutations, and some that are “susceptibility” genes that require environmental triggers to activate changes that in turn produce autism.

Could that have implications for vaccinations and autism? Could some component of the vaccines, the immune system’s reaction, or something not yet known play a role in the development of autism for at least a subset of children? Well, science is very conservative when it comes to saying that some factor could never play a role in some problem, especially with a growing understanding of individual variation and the clinical diversity of autism. Some do theorize that yet-to-be-determined environmental factors present in modern society may uniquely impact genetically susceptible individuals. It is quite clear that the current prevalence of autism spectrum disorders is high, and only a part of the explanation for this lies in the broader awareness of symptoms by both professionals and the public, or in the expansion of diagnostic criteria to include a broader variety of developmental, communication, and behavioral criteria.

That is why I support continuing research on many possible causes of autism, including vaccines. However, as a pediatrician, I don’t have the luxury of waiting to see if future studies continue to reinforce what previous studies already have found. Instead, like virtually all physicians, I must practice “evidence-based medicine.” That means I recommend that babies receive vaccines as recommended by the American Association of Pediatrics (AAP), NIH, and the CDC.

Evidence is strong that vaccines have and continue to have an enormous, truly miraculous impact on the health and survival of infants and children, including the prevention of diseases that can result in brain and other neurodevelopmental damage. And so far, according to a large and growing number of studies, no evidence exists for a causal relation between vaccines and autism. In 2004, the Institute of Medicine (IOM) released a report by a committee of independent experts that had, at the request of the CDC and NIE, evaluated evidence on potential links between childhood vaccines and health problems. An earlier IOM report had found no association between vaccines and autism, but institute members believed more evidence was needed regarding thimerosal, a mercury-based compound previously used as a vaccine preservative. In larger dosages, mercury itself is known to cause neurologic damage. After reviewing five large studies in the United States, United Kingdom, Denmark, and Sweden along with 14 additional studies and information from parents and clinicians, the new IOM committee concluded that available evidence argues against the existence of a causal relationship between thimerosal-containing vaccines and autism. It was an interesting time to look at these studies because of the changes in vaccines in recent years. Currently, to be as cautious as possible, all routinely recommended vaccines manufactured for U.S. infants are either thimerosal-free or contain only trace amounts, and the quantity of completely thimerosal-free vaccines increases yearly as manufacturing capacities expand. In some countries, such as Denmark, vaccines have had no thimerosal for years. But none of these changes have produced a decline in autism.

These findings, however, did not cause our nation’s leading research institutions to close the door on further investigation of a possible role for vaccines and autism. The CDC is conducting or supporting a number of studies conducted with the NIH, AAP, and academic and research institutions.

Where research can help

The need for more research into the epidemiology of autism is why I encourage families to participate in autism family databanks and research studies. They do so gladly. The results of past and current research studies are why I continue to recommend that families get all recommended immunizations for their children. In our vaccine-rich society, we too often take for granted that our children will not get serious communicable diseases, but the recent outbreak of Hemophilus influenza type B (HIB), with its risk of meningitis leading to deafness and brain damage, shows how wrong this idea is. As a medical resident, before the HIB vaccine was developed in the 1990s, I saw the neurodevelopmental havoc, sometimes death, which this still-present bacterium could cause in infants and children.

Finally, the significant advances now being made in research, especially related to the genetic basis of some forms of autism, are why I am optimistic that eventually, sooner rather than later, we will better understand what really causes autism and consequently what we can do to both treat and prevent it.
EDYE BRADFORD fell in love with the people at Emory Winship Cancer Institute during her treatment for colon cancer in 2006. The surgeon gave her hugs. The nurses made her laugh. Her care team fought so fervently to restore her health, she called them “the three musketeers.”

Emory is well-known for its expertise, and Bradford is now cancer-free. Deeply moved by the experience, she has included Emory in her will. “In my small way, I am providing a means to knowledge,” she explains, “and that is my real gift.”

Learn how you can support Emory in your estate plans. Call 404.727.8875 or visit www.emory.edu/giftplanning.