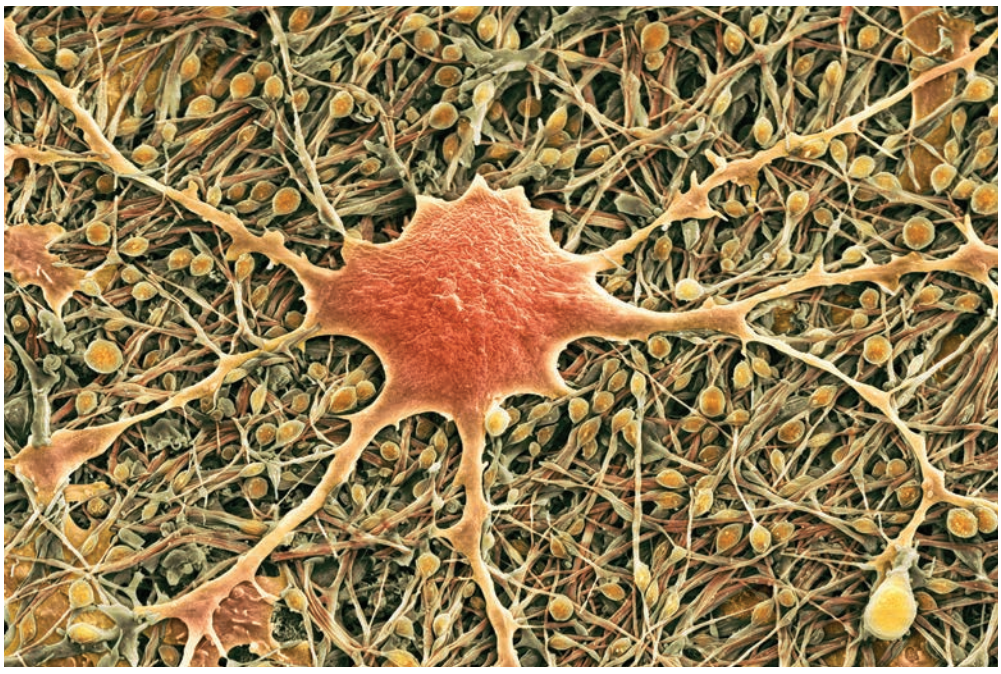


GLIAL GLITCHES: Childhood-onset schizophrenia may begin in faulty glial cells—brain cells that protect neurons and help them build communication networks.

Where Does Schizophrenia Begin? New Evidence Points to Glial Cells

Researchers at the Medical Center have identified a potential culprit behind the wiring problems in the brains of people with schizophrenia.

When the researchers transplanted human brain cells generated from individuals diagnosed with childhood-onset schizophrenia into mice, the animal's nerve cell networks did not mature properly, and the mice exhibited behaviors similar to those seen in people with the disease.

The findings, published in the journal *Cell*, suggest that childhood-onset schizophrenia may be due to glial cell dysfunction, according to Steve Goldman, the Dean Zutes Chair in Biology of the Aging Brain and codirector of the Center for Translational Neuromedicine, who was the lead author of the study.

"The inability of these cells to do their job, which is to help nerve cells build and maintain healthy and effective communication networks, appears to be a primary contributor to the disease," he says.

The research provides scientists with a foundation to explore new treatments for the disease. Because schizophrenia is unique to humans, scientists have been limited in their ability to study it. The researchers developed a new animal model that can be used to accelerate testing drugs and other therapies in schizophrenia.

—Mark Michaud

An Eye Test That Could Help Diagnose Autism

New research on rapid eye movements could herald a new tool to help physicians identify a sub-group of people with autism.

The rapid eye movements that humans make when shifting attention from one object to another, known as saccades, are essential to understanding and interacting with the world.

Saccades are controlled by the cerebellum, a densely packed structure of neurons that plays a role in motor control as well as emotion and cognition, due to its connections to the rest of the brain. There is growing evidence that the structure of the cerebellum is altered in some people with autism.

A series of experiments led by John Foxe, the Kilian J. and Caroline F. Schmitt Professor in Neuroscience and director of the Del Monte Institute for Neuroscience, and Edward Freedman, an associate professor in the Department of Neuroscience, suggested that the sensory motor controls in the cerebellum responsible for eye movement were impaired in subjects with autism.

Thus, saccade adaptation measures may prove useful in early detection of the disorder.

The research appears in the *European Journal of Neuroscience*.

—Mark Michaud

Bacterial or Viral? Genes May Hold the Key

Antibiotics are lifesaving drugs, but overuse is leading to one of the world's most pressing health threats: antibiotic resistance.

Antibiotics help fight bacterial infections, but are not effective against viruses. But because physicians have lacked a foolproof means to confirm bacterial infections, they often prescribe antibiotics, even though an infection may be viral.

A team led by Ann Falsey, professor and interim chief of the Infectious Diseases Division at the Medical Center, is developing a tool to help physicians identify bacterial infections.

Falsey's team conducted a battery of microbiologic tests on blood samples from participants who had been hospitalized with lower respiratory tract infections. Thomas Mariani, a professor of pediatrics and biomedical genetics, used genetic and statistical analysis to pinpoint 11 genetic markers in the blood that correctly classified the patients with bacterial infections 80 to 90 percent of the time.

"Our genes react differently to a virus than they do to bacteria," says Mariani. "Rather than trying to detect the specific organism that's making an individual sick, we're using genetic data to help us determine what's affecting the patient."

Falsey and Mariani plan to continue their research, noting that the genetic classifiers selected from the study population may not prove to be universal to all patients.

The study appears in *Scientific Reports*.

—Emily Boynton

THOMAS DEERINCK/SCIENCE PHOTO LIBRARY

Sharpen Your Image—With Freeform Optics

Researchers at the Institute of Optics have described an optical device with potential applications ranging from improved satellite and diagnostic imagery to more precisely matching the paint color on a living room wall.

The device is a type of spectrometer—an optical instrument that takes light and breaks it down into components to reveal a catalog of information about an object.

Unlike traditional spectrometers, this one is designed using freeform optics. A relatively new type of optical design, it replaces rotationally symmetrical, and often perfectly spherical, optical surfaces with "freeform" ones that rely on a more complicated geometry.

Freeform design enables a device to efficiently correct aberrations with fewer, smaller lenses and mirrors.

Described in *Light: Science & Applications*, the device—designed by Jacob Reimers, a PhD candidate in the lab of Jannick Roland, the Brian J. Thompson Professor of Optical Engineering—is five times more compact than similar spectrometer designs using more conventional mirrors.

DECENTERED: Designed with freeform optics, Reimers's prototype spectrometer is lighter, more compact, and more efficient than traditional spectrometers.

It also allows a three-fold increase in the bandwidths analyzed and is 65 times more effective at correcting aberrations that affect field of view and resolution.

"Spectrometers monitor the environment, help examine patients, and are broadly used for many other applications. What we found here can be applied to spectrometers used in all of these other applications," says Rolland.

—Bob Marcotte

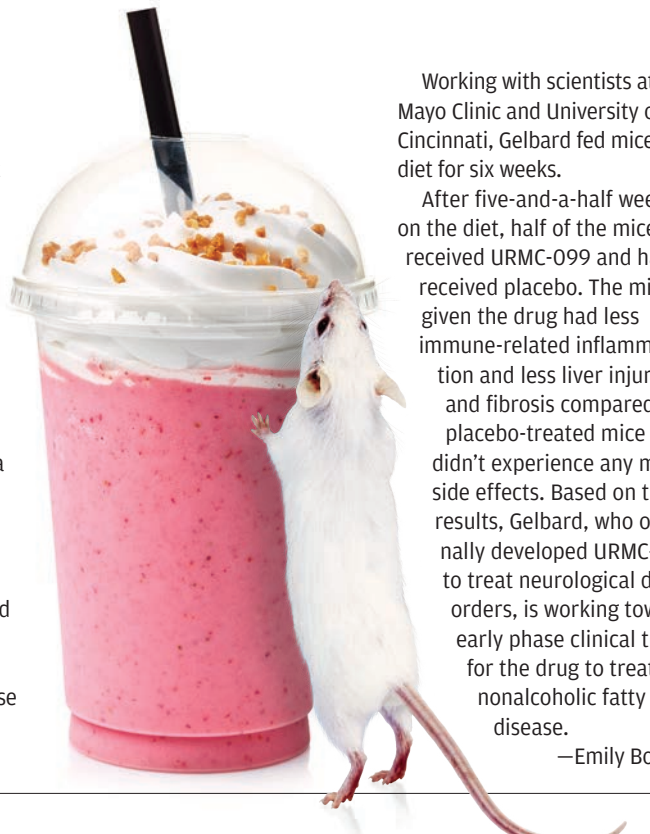
Of Mice and Milkshakes

Not surprisingly, a fast-food diet is no better for mice than for humans. But a drug developed at the Medical Center protected mice that were fed a fast-food diet from one of the diet's many potential ills: nonalcoholic fatty liver disease.

In a study published in the journal *JCI Insights*, scientists reported that the drug, dubbed "URMC-099," reversed liver inflammation, injury, and scarring in mice. The mice had developed the symptoms of fatty liver disease after consuming a diet high in fat, sugar, and cholesterol that had been designed to replicate a fast-food diet and recreate the features of the illness found in people.

Eating high volumes of fatty and sugary foods triggers inflammation in the liver, and the body responds by sending immune cells to neutralize the threat. Unfortunately, the immune response can rage out of control, creating even more inflammation and further damaging the liver. URMC-099, which was developed in the laboratory of Harris (Handy) Gelbard, a professor of neurology and the director of the Center for Neurotherapeutics Discovery, dials back the immune response to a normal level.

"URMC-099 seems to break this vicious cycle of persistent inflammation by restoring balance between immune cells and liver cells," says Gelbard. "The drug's ability to turn down the volume on the immune response allows the liver to regain its normal functions."



Working with scientists at the Mayo Clinic and University of Cincinnati, Gelbard fed mice the diet for six weeks.

After five-and-a-half weeks on the diet, half of the mice received URMC-099 and half received placebo. The mice given the drug had less immune-related inflammation and less liver injury and fibrosis compared to placebo-treated mice and didn't experience any major side effects. Based on the results, Gelbard, who originally developed URMC-099 to treat neurological disorders, is working toward early phase clinical trials for the drug to treat nonalcoholic fatty liver disease.

—Emily Boynton

J. ADAM FENSTER (OPTICS); STEVE BOERNER (PHOTO ILLUSTRATION)/ADOBE STOCK (ORIGINAL IMAGES)