ECU, health department teaming up against HIV

By Brock Letchworth
The Daily Reflector

Pitt County’s public health department and East Carolina University’s Brody School of Medicine are working together to slow the spread of HIV and AIDS in the area.

With the help of a federal grant, the two entities are entering into a contractual agreement whereby the health department will acquire additional staff to locate individuals who are HIV positive and not receiving care.

HIV stands for human immunodeficiency virus and is the virus that causes AIDS. It is different from most other viruses because it attacks the immune system, which gives the body the ability to fight infections.

The health department will refer those who are HIV positive to the school of medicine for treatment.

Dr. John Morrow, county public health director, said the deal will help officials decrease HIV transmission in eastern North Carolina.

“We know that a lot of the folks who have HIV don’t get into care or they fall out of care, and we know that people who are HIV positive and receiving appropriate care or medical treatment are much less likely to transmit that infection to anyone else,” Morrow said. “We want to do whatever it takes to get them to appropriate care.”

Treatment slows the progression of HIV by controlling an individual’s viral load, Morrow said.

To help with locating infected individuals, the health department said it will hire ECU public health students. Those individuals will work with medical students to seek out people and expedite them to care.

A grant provided to the School of Medicine by the U.S. Department of Health Resources and Services Administration will fund the efforts, said Roy Carlton, administrator for the Brody department of infectious diseases. The university is using $20,000 of the grant to fund the new positions and pay for other expenses like travel for workers and patients.

“We just want to contact them, and see if there is anything we can do to make it easier for them to get into treatment,” Morrow said. “It doesn’t really matter whether they get that care from the school of medicine or not. We just want to make sure they get care somewhere.”

Morrow said officials have not decided how many students they will hire, and the positions are only funded through the end of July. Officials are not certain if they will seek additional funding when the grant expires.

There were 1,943 new individuals diagnosed with HIV statewide in 2007, Morrow said. The state average is about 1,700 per year.
Years of study unlock reason behind deaths of infant boys

By Mary Ann Roser
Cox News Service

As soon as the Messers saw their newborn son, they knew something was wrong. The baby, who had hardly kicked in the womb, emerged with a beautiful face but with a tiny cry and a body too weak to move.

The delivery room doctor at Scott & White Hospital in Temple, Texas, told Ann Messer and her husband, then-state Rep. Bill Messer of Belton, that Andrew would not live to be a year old. He was diagnosed with congenital muscular dystrophy.

Andrew was 5½ months old when he died in 1985.

The couple then started noticing that other women in the family were losing children. Two of Ann Messer’s cousins had sons die from what appeared to be the same disorder.

After 23 years, the Messers finally got some answers recently.

A team of scientists in the United States and Germany said it has discovered the gene that caused the rare disease — known as X-linked infant spinal muscular atrophy — that killed Andrew and three sons of Ann Messer’s cousins. It was announced in a paper published in The American Journal of Human Genetics.

The X-linked form is the most severe type of spinal muscular atrophy, a disease in which the big nerves in the spinal cord die and can’t send signals to the muscles, resulting in the “floppy” body the Messers saw in the delivery room. Without muscle tone, the condition worsens until the child can’t chew food or breathe, said Lisa Baumbach-Reardon, the paper’s senior author and an associate research professor in the department of pediatrics at the University of Miami’s Miller School of Medicine.

The symptoms of the disease, which is in the family of muscular dystrophy, are similar to those of amyotrophic lateral sclerosis — also known as Lou Gehrig’s disease — and in 75 percent of the cases, death occurs by age 2, Baumbach-Reardon said.

GENE DISCOVERY

THE CONDITION:
Researchers searching for the cause of X-linked infant spinal muscular atrophy found a defect in the UBE1 gene on the X chromosome.

THE EFFECT: The gene carries flawed instructions for destroying proteins that the body needs to eliminate, which can clog the body like a garbage disposal system that’s backed up. That means the body can’t function normally.

The flaw is similar to what happens with Parkinson’s disease, researchers said.

THE HOPE: Finding the gene means doctors can use a DNA test to diagnose a fetus or a boy who has the disease and eventually develop treatments.
The odds are 50-50 that a woman who is a carrier will produce a son with the disease. The Messers' first-born son, Will, 28, doesn't have it.

Finding the gene means that scientists now know how the disease is transmitted (from mother to son only) and that tests can be done to determine whether a woman is a carrier and whether an embryo or fetus has the mutation. Although therapies have not been developed for turning off the gene that causes the disease, technology exists for isolating embryos without the flawed gene so they can be implanted into the womb of a woman who is a carrier, ensuring a son free of the disease.

Finding the gene also means doctors can use a DNA test to diagnose a fetus or a boy who has the disease and perhaps one day develop treatments.

"We've been waiting so long for this breakthrough," said Ann Messer, 56, as she sat clutching a photograph of Andrew in her husband's Austin, Texas, office.

"It's a terrible tragedy," said Bill Messer, 56, now a Capitol lobbyist. "But a great good has come from it, not just for Ann's family but for all families."

Ann Messer and her cousins never believed the doctors who insisted that they had married men with the same flawed gene. The odds were simply too great. But that was the only explanation they had.

The gene discovery "confirmed what we thought 23 years ago," Messer said. "This is the proof."

Baumbach-Reardon said she doesn't know how many babies are born with the X-linked disease or how many women have miscarriages because of it, but she said she feels certain it is often undiagnosed or misdiagnosed.

The more common form of spinal muscular atrophy is believed to affect one in 6,000 births,

See GENE, D2
GENE
Continued from D1
she said. She identified 16 families worldwide with the
X-linked variety, including the Messers and Ann Messer's cousins, who joined
the study. She said it's possible that many of the 4 percent of
the babies born with spinal muscular atrophy might actu-
ally have the X-linked form.
Because the defective gene
is recessive and on the X
chromosome, the disease can be
passed on only to boys,
who inherit the X chromo-
some from their mothers and
a Y chromosome from their
fathers. Girls, who have two
X chromosomes, can carry
the defective gene but won't
develop the disease, Baumbach-Reardon said.

"It's fantastic to see this
work come out," said Sharon
Hesterlee, vice president
for translational research
at the Muscular Dystrophy
Association, which helped
finance the study. "It was a
big needle-in-the-haystack
problem for Lisa."

Baumbach-Reardon said
she spent 15 years seeking the
gene, following the work of
a Baylor College of Medicine
researcher, the late Dr. Frank
Greenberg, who suspected
that the gene was passed by
mothers to their sons but
couldn't prove it without the
gene.

Isolating the gene is a big
step toward understanding
how the disease process
works, but it's like the end
of a chapter, not the end of a
book, said Vishy Iyer, associ-
ate professor of molecular

LISA BAUMBACH-
REARDON, a University of
Miami geneticist who has
been tracking the UBE1 gene
that can cause infant spinal
muscular atrophy, says Ann
and Bill Messer's cooperation
with her work was invaluable.
genetics and microbiology
at the University of Texas at
Austin.

Years of work remain to
develop therapies, if there
are any, he said. It can take 10
years to develop a treatment
after a gene is discovered,
and finding money is always
a challenge, Baumbach-Reard-
on said.

Some experimental drugs
are in clinical trials for
spinal muscular atrophy,
Hesterlee said.

Having the Messers and
Ann Messer's cousins in
the study was a huge help,
Baumbach-Reardon said. The
families gave blood for study,
and tissue and blood samples
from some of their sons had
been preserved for future re-
search. All of that was needed
to unravel a family mystery.

Ann Messer said her
mother, Edna Whitworth of
Austin, had a miscarriage
and lost a 2-day-old son, prob-
ably because of the disease.
But her mother also had two
healthy boys, including Ann's
brother, Philip Whitworth, an
oil and gas lawyer in Austin.

Ann Messer's cousin Cath-
erine Blevins of Houston was
living in New Orleans in the
1980s when she gave birth to
a "floppy baby" who died at
18 months. Everyone thought
"it was one of those random
things that happened," Ann
Messer said.

Ann got pregnant, and two
months after Andrew was
born, Indiana cousin Connie
Alexander, who now lives in
Fort Worth, Texas, gave birth
to a son who had the same
"floppy" look as Andrew. He
lived 11 months. The puzzle
was starting to come to-
together.

By then, Blevins had had
another son, who looked just
like the other sick babies. He
lived to age 18 with the help
of a feeding tube, a ventila-
tor to help him breathe and
constant care.

None of the boys had
mental impairments, and
Andrew, despite his health
problems, was a sweet, happy
child, Ann Messer said.

"He was very cherished for
the 5½ months we had him," she
said.

Just knowing what caused
the illness is important to
families, and those with
a family history can seek
testing and get answers, the
Messers said.

"There are a lot of bless-
ings in all of this," Bill Mess-
er said, "and it's a great one
(we're getting) this week."
Study ties too much, too little sleep to ill health

By Mike Stobbe
The Associated Press

People who sleep fewer than six hours a night — or more than nine — are more likely to be obese, according to a new government study that is one of the largest to show a link between irregular sleep and big bellies.

The study also linked light sleepers to higher smoking rates, less physical activity and more alcohol use.

The research adds weight to a stream of studies that have found obesity and other health problems in those who don't get proper shut-eye, said Dr. Ron Kramer, a Colorado physician and a spokesman for the American Academy of Sleep Medicine.

"The data is all coming together that short sleepers and long sleepers don't do so well," Kramer said.

The study released Wednesday is based on door-to-door surveys of 87,000 U.S. adults from 2004 through 2006 conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention.

Such surveys can't prove cause-effect relationships, so — for example — it's not clear if smoking causes sleeplessness or if sleeplessness prompts smoking, said Charlotte Schoenborn, the study's lead author.

It also did not account for the influence of other factors, such as depression, which can contribute to heavy eating, smoking, sleeplessness and other problems.

Smoking was highest for people who got under six hours of sleep, with 31 percent saying they were current smokers. Those who got nine or more hours also were big puffers, with 26 percent smoking.

The overall U.S. smoking rate is about 21 percent. For those in the study who sleep seven to eight hours, the rate was lower, at 18 percent.

Results were similar, though a bit less dramatic, for obesity: About 33 percent of those who slept less than six hours were obese, and 26 percent for those who got nine or more. Normal sleepers were the thinnest group, with obesity at 22 percent.

For alcohol use, those who slept the least were the biggest drinkers. However, alcohol use for those who slept seven to eight hours and those who slept nine hours or more was similar.

In another measure, nearly half of those who slept nine hours or more each night were physically inactive in their leisure time, which was worse even than the lightest sleepers and the proper sleepers. Many of those who sleep nine hours or more may have serious health problems that make exercise difficult.

Many elderly people are in the group who get the least sleep, which would help explain why physical activity rates are low. Those skimpy sleepers who are younger may still feel too tired to exercise.

ON THE NET

- National Center for Health Statistics: www.cdc.gov/nchs
- American Academy of Sleep Medicine: www.aasmnet.org/
AG: Close colleges to illegal aliens

The advice derails a movement to grant in-state tuition to graduates of N.C. high schools.

BY KRISTEN COLLINS
STAFF WRITER

Public colleges in North Carolina should not admit illegal immigrants as students, the state Attorney General's Office advised in a letter released Wednesday.

If followed, the advice would reverse policies at the state's 58 community colleges and at the 16 four-year institutions in the University of North Carolina system, which allow illegal immigrants to attend. It also damages a movement to grant in-state tuition to illegal immigrant teenagers who have attended North Carolina high schools.

"It really closes the door of opportunity for a lot of kids," said Andrea Bazin, president of the Triangle Community Foundation, a philanthropic organization, and a longtime Hispanic advocate.

The letter to the general counsel for the Community College System, which was dated Tuesday but released to The News & Observer on Wednesday, said that higher education is a public benefit that illegal immigrants are not entitled to under federal law.

The state could pass a law allowing students access regardless of their immigration status, the letter says. Otherwise, a policy prohibiting illegal immigrants "would more likely withstand judicial scrutiny," wrote J.B. Kelly, general counsel in the office of Attorney General Roy Cooper.

Kelly recommended that the colleges revert to a policy that would allow illegal immigrants to take only non-college level courses, such as adult high school and English as a second language.

The community colleges asked for the attorney general's opinion in December, after then-President Martin Lancaster directed all 58 campuses to admit illegal immigrants. Until then, the campuses set their own policies, and about a third barred illegal immigrants. Though the letter is addressed only to the community colleges, the advice applies to all state-funded higher education institutions.

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COLLEGES
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Officials in the Attorney General’s Office could not be reached for comment on the letter, which Kelly specified was advisory and not a formal opinion. Both are nonbinding recommendations, but an opinion goes through a more formal review process.

Lancaster, reached just hours before his retirement party Wednesday, said only that he was disappointed.

“Every lawyer is entitled to their opinion,” he said, “but the Attorney General’s Office carries more weight than mine.”

Gov. Mike Easley also supported Lancaster’s decision. Efforts to reach Easley on Wednesday were unsuccessful.

Leaders at the University of North Carolina System and the Community College System also declined to comment. Audrey Bailey, a spokeswoman for the community colleges, said President Scott Ralls was out of town at a funeral and had not yet reviewed the letter. “We’re taking it under advisement,” Bailey said.

Joni Worthington, a spokeswoman for the UNC system, said UNC had not received the letter.

Currently, only a handful of illegal immigrants are students at North Carolina’s universities and community colleges. The UNC system says 27 of its more than 200,000 students are illegal immigrants. The community colleges have reported that 340 of their 271,000 degree-seeking students are here illegally.

All pay out-of-state tuition, which is greater than the cost of their instruction — meaning the schools make a profit from these students.

However, many say that North Carolina should not provide higher education to illegal immigrants, regardless of the cost. And some argue that the costs are higher than the schools’ calculations show, when taking overhead and infrastructure into account.

“The key word is ‘illegal.’”

Federal law requires that elementary and secondary education be provided to all children, regardless of their immigration status. State Sen. Richard Stevens, of Cary, said the state should provide only what is required.

“The key word is ‘illegal,’ ” Stevens said. “They got here illegally and now they want to claim all the benefits.”

Stevens said he asked the Attorney General’s Office to consider the federal statute that became the basis of its advice. If the office hadn’t issued the letter, Stevens said he would have supported legislation in the upcoming session to expressly bar illegal immigrants from colleges and universities.

Now, he said, that bill may not be necessary.

Advocates, including Bazán, called the advice “extreme.” Several states provide in-state tuition to illegal immigrants, and she said that in years of fighting for a similar policy in North Carolina, she has never heard this federal law invoked.

The letter says that the “scope and applicability” of the federal law barring higher education is “unsettled” and cites conflicting legal rulings on the issue. It also says that enforcement of the federal law is the responsibility of the Department of Homeland Security, but that the department has not provided any guidance on how to interpret the law regarding higher education.

“Permanent underclass”

Bazán said that, by denying illegal immigrant students a chance at college, “we are creating this permanent underclass.”

“We’re not talking about thousands of kids,” Bazán said. “We’re talking about just a handful of kids who have made it to the top of their class and want to contribute.”

Peter Kaufman, a University of North Carolina history professor, works with some of those students. He runs the Scholars’ Latino Initiative, which mentors Hispanic high school students who want to go to college.

Kaufman said he has four illegal immigrant students in the program this year, two of whom plan to attend public colleges in North Carolina. He said all are teens who had no choice about moving to the United States, and that all have succeeded despite poverty and limited English skills.

“These are kids who are extraordinarily disciplined and gifted,” Kaufman said. “But we can’t tell them that achievement is going to mean clear sailing.”

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Bypassing Medicine to Treat Diabetes

By altering the gut's production of hormones, gastric bypass surgery may be able to eliminate type 2 diabetes. But scientists worry that this radical operation can also cause dangerously low blood sugar.

IN 1980, BARIATRIC SURGEON WALTER Pories of East Carolina University School of Medicine in Greenville, North Carolina, performed his first gastric bypass surgery on an obese patient with type 2 diabetes, then a second, then a third. He noticed right away that the patient no longer needed insulin. Family doctors confirmed that what Pories had considered a transient phenomenon seemed like something more: Each person's diabetes had disappeared, even before they'd lost much weight. Pories was convinced that the doctors had erred. "I said, 'You guys don't know how to work up diabetes. Diabetes is an incurable disease.'" A few years later, in 1984, Pories and an endocrinologist took matters into their own hands. "We marched right down to the lab, very self-righteous," and accused the lab employees of incorrectly measuring blood sugar levels. "'If you're a doctor, you like to blame other people,'" Pories explains.

As the number of patients with vanishing diabetes mounted, Pories recognized that the effect was real. Still, the concept that diabetes could be reversed surgically was so outlandish, he says, that "we didn't dare publish" the results. Instead, Pories began tracking his patients. In 1995, he reported in the Annals of Surgery that among 146 people with diabetes who had had the surgery in the past 14 years, 121, or 83%, had quickly become diabetes-free. The result was far superior to that achieved by any other treatment at the time—or now.

"The surgical world noted that paper," says endocrinologist David Cummings of the University of Washington, Seattle. But it took "another 10 years for the rest of us" to catch up, he says. Now, endocrinologists are beginning to pay close attention to the effects of gastric bypass surgery, which had long been a backwater of medicine, in part because obesity was not considered a genuine disease.

As America and other countries confront rising rates of obesity, with few treatments that shrink the widest waistlines, the surgery's popularity is soaring. The most common form in the United States, Roux-en-Y gastric bypass, was performed on more than 120,000 people in 2007, according to estimates. That's almost double the number 5 years ago. Doctors often learn from their patients, and the hundreds of thousands of people who have had gastric bypass surgery are now prompting an overhaul in our understanding of metabolism and diabetes. Scientists are also going back to animals to figure out the impacts of the procedure. They are finding that the surgery's rerouting of the intestines and closing off of much of the stomach appears to have drastic effects on gut hormones and disease, independent of the weight loss that accompanies it.

These effects can also have dire consequences. Beginning in 2000, F. John Studio, an endocrinologist at the Mayo Clinic in Rochester, Minnesota, began seeing patients with some alarming symptoms: confusion, abnormal behavior, seizures, and unconsciousness. In each case, the culprit was a low level of blood sugar that struck after eating, which is when it rises in healthy people. Every patient, it turned out, had undergone gastric bypass surgery months or years earlier. The Mayo Clinic now sees at least two new patients a month with this unusual hypoglycemia disorder, which was the topic of a meeting at the Joslin Diabetes Center in Boston earlier this month. "A lot of surgeons have removed part or even all of the pancreas, which churns out insulin, from many of these patients."

How to decipher and harness the surgery's metabolic effects is prompting much debate. On the one hand, some surgeons are already operating on less obese people with diabetes as a treatment for that disease. But others would prefer to wait until the science catches up, especially because the surgery isn't harmless, with a death rate ranging from 0.1% to 2%, depending on where it's performed. "Surgeons have for too long acted in a vacuum ... Most of them aren't thinking about the mechanisms of what they're doing," says John Dixon, an obesity researcher at Monash University in Melbourne, Australia. "But we need to dissect out what's happening in these patients."

Early clues

Gastric bypass was inspired by similar intestinal operations employed for ulcers and gastric cancer that induced dramatic and enduring weight loss and were reported to reverse diabetes as far back as the 1950s. "As soon as we started doing the operation, we were aware of the fact that before the patients got out of the hospital, they no longer needed insulin," says Edward Mason, a retired surgeon from the University of Iowa in Iowa City who developed the procedure for weight loss. Most current forms of gastric bypass, hyperinsulinemic hypoglycemia following gastric bypass: pathogenesis and treatment symposium, Boston, Massachusetts, 7 April.

Unintended effects. Roux-en-Y gastric bypass surgery reduces the stomach to a fraction of its original size and skips past part of the small intestine, which causes profound metabolic changes in the gut.
Beyond fat. From the early days, doctors recognized that for many patients, diabetes vanished after gastric bypass.

and Mason's original operation, have one element in common: A newly created exit from the stomach is connected to a piece of small intestine a few feet lower down, "bypassing" the upper portion of the small intestine. In addition, the stomach is drastically restricted, by about 95%. (Another weight-loss surgery, gastric banding, seals off most of the stomach but leaves the intestines intact and is not considered gastric bypass.) Today, most gastric-bypass patients shed 50% of their body weight and keep it off.

Mason, now 87 years old, recalls that he and others explained away the reversals of type 2 diabetes because their patients weren't eating right after surgery, which would lower blood glucose levels and, in turn, their need for insulin. (The surgery does not have the same effect on type 1 diabetes, in which afflicted individuals cannot produce insulin.) But Pories' study years later slowly began to convince people that something more fundamental was occurring.

Almost a decade later, a second report strengthened the case. In 2003, Philip Schauer, a bariatric surgeon now at the Cleveland Clinic in Ohio, published follow-up data from 1160 obese people who in the preceding 5 years had undergone Roux-en-Y gastric bypass, which gets its name from a French surgeon who developed the technique. Of the 191 people with diabetes or impaired glucose metabolism who could be tracked down, 83%, precisely the figure reported by Pories, no longer had the problem.

Although impressive, it's not yet clear if these success rates will hold up in clinical trials. These are "typically the observations of a single surgeon or group of surgeons" and "very anecdotal," says David D'Alessio, an endocrinologist at the University of Cincinnati in Ohio.

Getting at biology

After years of absence, science is slowly making inroads into gastric bypass surgery. "The development of the field was not based on real research," says Francesco Rubino, a bariatric surgeon at Weill Cornell Medical College in New York City. "That has tarnished the field somewhat."

Recently, however, a growing number of studies are suggesting that the surgery has a profound effect on gut hormones, which could explain its impact on appetite, diabetes, and the low blood sugar that's turning up. One of the first clues emerged in 2002, when Cummings looked into a well-recognized oddity. Gastric bypass restricts the stomach, forcing people to eat smaller meals. One might then expect "that people would be compelled to sip milkshakes all day long," says Cummings. That's not what happens. Many move away from calorie-dense foods altogether.

Curious, Cummings began examining levels of ghrelin, a hormone produced mainly by the stomach that stimulates appetite. Most people have peaks and valleys in ghrelin levels throughout the day as they consume meals and then become hungry again. In those who've had gastric bypass, Cummings found, ghrelin levels in blood were low and changed little all day, suggesting that something about the surgery dampens ghrelin production and hence appetite.

The role this plays in diabetes resolution has not been firmly established, and researchers are now more closely examining how gastric bypass affects other hormones. Rubino's work, for example, has focused on the intestines, which produce a different suite of chemicals and hormones from those the stomach churns out. In 1999, Rubino turned to rats to examine whether the surgery's effects on diabetes were due to calorie restriction and weight loss alone. He tried to tease apart distinct features of his "patients"—the rats, in this case—and different features of surgery. When performed on lean animals with type 2 diabetes, gastric bypass had the same positive effects on the diabetes as in obese ones, suggesting that weight loss was largely irrelevant. Furthermore, Rubino performed the
intestinal bypass portion of the operation, skipping past the duodenum and the jejunum that link up to the stomach, but leaving the stomach intact. There was a "direct antidiabetic effect," he says.

Rubino's rat work dovetails with a popular theory: that a hormone produced by the intestines called glucagon-like peptide 1 (GLP-1) lies behind the vanishing diabetes in many gastric bypass patients—and may be linked to the hypoglycemia that later strikes others, most of whom did not have diabetes before the surgery. The GLP-1 theory is that the small intestine goes into overdrive making hormones in gastric bypass patients. Because of the surgical rerouting, food "empties directly into this part of the intestine that it normally wouldn't see at that stage" of digestion, says Mary-Elizabeth Patti, an endocrinologist at the Joslin Diabetes Center.

In healthy people, GLP-1 has a variety of effects, including increasing insulin secretion, and a diabetes drug on the market, called Byetta, mimics the effects of GLP-1. Physiologist April Strader of Southern Illinois University in Carbondale is now performing an intestinal surgery in rats that leaves the stomach intact and promotes the animals to secrete more GLP-1. Strader is examining whether that in turn causes proliferation of insulin-producing cells in the pancreas.

**Linking the good and bad**

GLP-1's impact on the pancreas may also explain the hypoglycemia originally seen by the Mayo Clinic. One sharp contrast between the disappearance of diabetes and the hypoglycemia stemming from the surgery is that the former occurs immediately or within weeks, whereas the latter takes several years to show up. At the Boston meeting, the 40 or so surgeons, endocrinologists, pathologists, and others gathered there admitted that they couldn't explain this but wondered whether changes to the pancreas over time generated the low-blood-sugar problems, whereas diabetes improvement from obese individuals. He attributes this difference of opinion to his more extensive analysis, which did not identify an upsurge in insulin-producing cells.

Butler did make one intriguing find, however. Obese people tend to produce more insulin over time to accommodate the growing amount of tissue that requires the hormone. Based on the appearance of the islet cells, Butler deduced that the gastric bypass pancreases hadn't made the adjustment to their host's new weight: They were still producing just as much insulin as before the surgery, effectively increasing the insulin available. That this occurs after meals would make sense, because this is when the pancreas normally releases extra insulin. In these patients, the insulin they secrete would far exceed what's needed.

Stephen Bloom, an obesity researcher at Imperial College London, notes that it's far from clear whether GLP-1 has the same effect on human pancreases as it does on those of rodents. Furthermore, the small intestine secretes dozens of hormones, many of them poorly understood. "It's still too soon to rule out everything else," says Strader. Rubino agrees that researchers need to think expansively. Instead of largely undigested food stimulating hormone secretion from the intestines it's dumped into, it's possible that when food does not touch the walls of the duodenum, as happens in gastric bypass, that has hormonal effects of its own. Rubino says his recent findings in animals suggest such antidiabetic effects.

**Surging popularity**

As research picks up pace, gastric bypass surgeries continue unabated, and some surgeons, particularly outside the United States and Europe, are beginning to operate on less obese patients with diabetes. Bariatric surgery is "kind of the Wild West," says D'Alessio. There's "huge demand, no regulation, everybody's got their own operation, and patients are willing to do whatever it takes to get it."

Currently, U.S. National Institutes of Health guidelines recommend that gastric bypass surgery be considered only for people who have a body mass index (BMI) of at least 35. (A BMI of 18.5 to 25 is considered normal.) At a meeting in Rome last year, 78% of attendees supported lowering the limit to a BMI of 30 for those with diabetes. Should the number be even less? "We need more data to know if a lower bar is okay or if there should be any bar at all" when the goal is diabetes treatment, says Cummings.

But many still view gastric bypass as extreme therapy for diabetes. Some who undergo the operation have serious problems, such as infections, gallstones, and hernias, that can require additional surgery. And given the time lag between gastric bypass and the severe hypoglycemia that Service, Patti, and others are just now documenting, no one knows how prevalent the side effect will be nor how much such patients will affect the cost-benefit analysis. The death rate from gastric bypass surgery also scares many diabetes researchers. "We had a death in a 28-year-old recently; she had a complication but didn't want to come to the hospital," says Bloom. "When you see that and have to go to the funeral, you don't think it's such a harmless procedure."

Yet type 2 diabetes isn't harmless, either, contributing to more than 1 million deaths worldwide each year. "There is a barrier we need to get over" in considering gastric bypass as a diabetes treatment, says Rubino. He points to a paper published last summer, concluding that the surgery reduces diabetes deaths by 92%. "It's the most profound effect in terms of mortality from diabetes ever reported," Rubino says. "What is the price of that?"

—JENNIFER COUZIN
Today’s debate: Scholarships

Focus college aid on need

Our view:
In tight times, merit-based grants divert public money from the needy.

The University of Colorado at Boulder is a prestigious research university located in a picturesque setting. Its graduate schools consistently rank in the nation's top 10.

Considering all that, the university gets less respect from state politicians than it deserves. On lists ranking public universities by funding they receive from their state, Colorado rests near the bottom.

On the surface, then, it would be easy to take the university's side in its money struggles with the governor and Legislature. Except for one thing: If the school is so pinched for dollars, why does it hand out a quarter of its aid to students who don't need it?

Boulder is one of scores of public universities that dole out taxpayer-financed “merit aid” to students whose families can afford the tuition there. A 2006 College Board study concluded that the practice is both widespread and significant. Admissions directors use those dollars to attract exceptionally bright students or unusually talented athletes who make the university look more prestigious or attractive.

In flush economic times, such expenditures might be excusable. But today's tough times raise questions about giving away so much public money in the pursuit of higher rankings.

In competing with private colleges, public universities already have a substantial built-in cost advantage from state subsidies — an advantage that averages $7,072 a year for out-of-state students and $17,527 for in-state students.

To be sure, higher education costs have gotten onerous for all but the very wealthy, and even state-school tuitions are a strain on middle-class budgets. But boosting merit aid to non-needy students hurts poor students the most by siphoning away money that would otherwise be available to them.

As it is, middle- and upper-income undergraduates receive larger grants from institutions than do students from low-income families, according to the National Center for Public Policy and Higher Education, a nonprofit organization that examines college costs and access. The average institutional award is $4,700 for students with parental income below $20,000 a year and $6,200 for those with parental income above $100,000.

Some states lacking prestigious public universities hand out merit assistance to retain in-state students with good grades, the very students most at risk of leaving the state — the “brain drain” dilemma. But states such as California that built high-quality public schools, UCLA or Berkeley, don't have to offer merit aid to attract students. Unfortunately, many states see doling out merit money as cheaper than building first-rate universities.

The best way to improve quality is to raise the standards for admission, and then direct financial aid to those who meet those standards but couldn't otherwise afford to attend.
We need to compete

Opposing view:
Merit aid helps public universities attract and reward top students.

By Bruce D. Benson

It would be easy to conclude that in difficult economic times, top public research universities should devote all their financial aid to need-based rather than merit-based programs. Easy, but shortsighted.

Merit-based aid bolsters crucial research endeavors, raises the academic bar across the board, rewards student achievement and helps us compete with private universities for the best students.

At the University of Colorado, about three-quarters of the financial aid we disburse is need-based. A significant number of students who get merit-based aid also draw from the need-based pool. While providing needy students access to a top-flight education is part of our mission, so is ensuring the quality of a university education and meeting the compelling needs of our state and nation.

As one of the country’s top public research university systems, it is critical that we attract the best and brightest students to our classroom and research activities.

Discovery and innovation require a community of scholars, and we create it in part by enticing students with merit aid. While research is most common at the graduate level, providing undergraduates such opportunities fosters a mindset and a pipeline that sustain the entire endeavor.

Research is important, but top students also raise the bar across disciplines. Their presence helps inspire not only their classmates but also the faculty.

Public universities compete against private ones for top students. While private universities’ tuition is considerably higher, the perception that their sticker price is offset by substantial merit-based aid lures a significant number of high-achieving students.

Additionally, squeezing merit-based aid hurts an already pinched middle class, where families generally earn too much to qualify for need-based aid but not enough to comfortably pay tuition. Not all those students are meritorious, but shouldn’t those who have worked hard and achieved all their lives be rewarded?

Good work might be its own reward, but in America we have a long history of encouraging and rewarding excellence. Devoting a quarter of our financial aid money to those rewards, and the subsequent benefits a university receives, is a worthy investment.

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