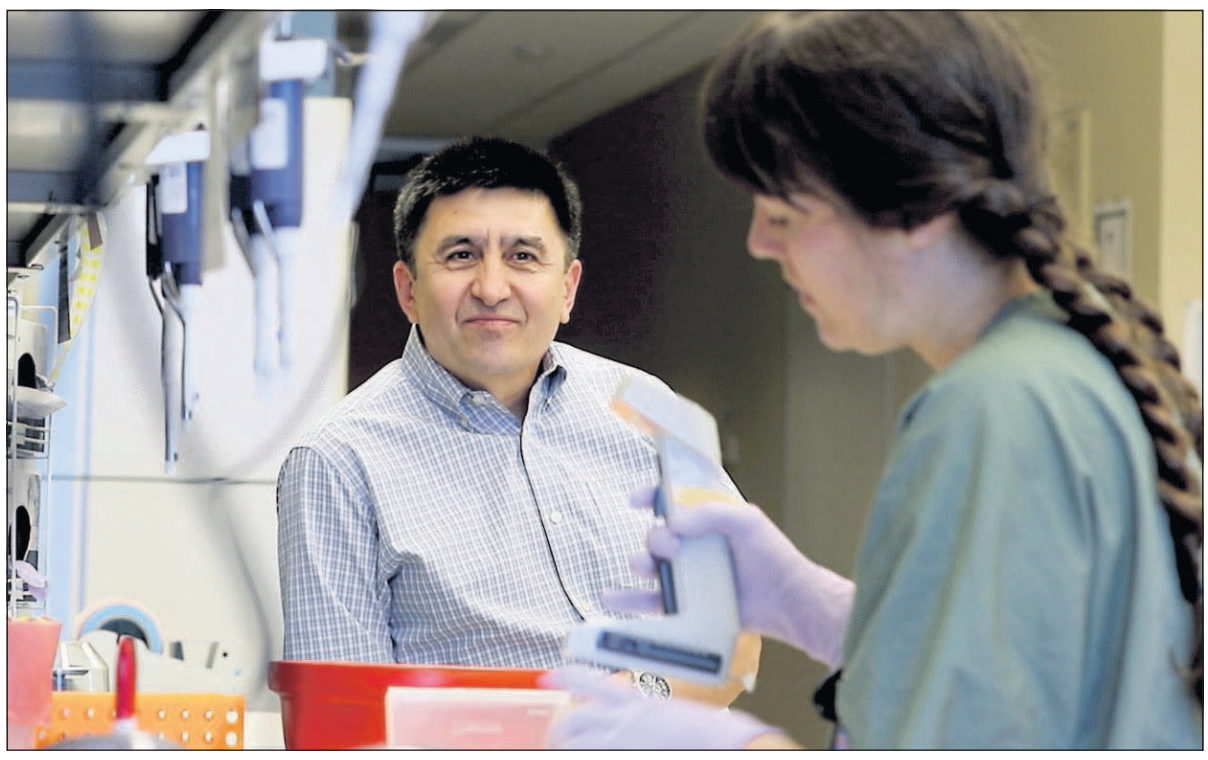
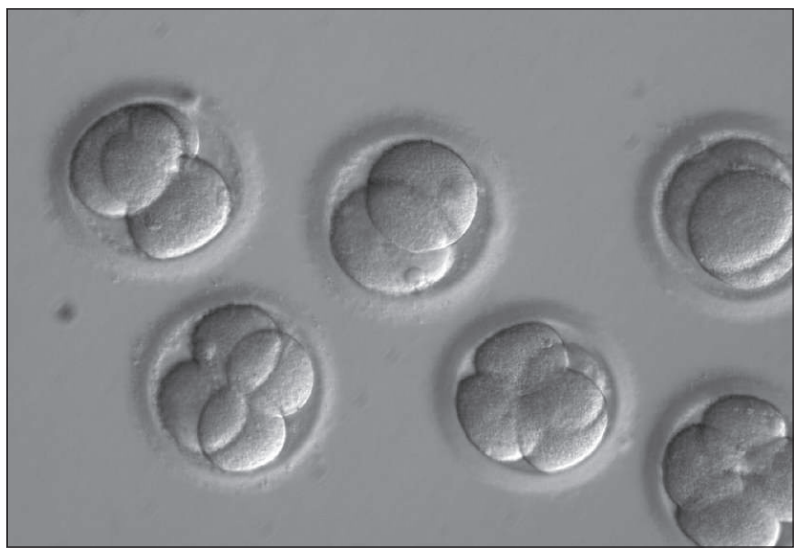
Embryo gene-repair holds promise for inherited disease

OHSU researchers first to repair gene that causes heart defects



Kristyna Wentz-Graff/Oregon Health & Science University via AP

In this July 31 photo provided by Oregon Health & Science University, Shoukhrat Mitalipov, left, talks with research assistant Hayley Darby in the Mitalipov Lab at OHSU in Portland. Mitalipov led a research team that, for the first time, used gene editing to repair a disease-causing mutation in human embryos, laboratory experiments that might one day help prevent inherited diseases from being passed to future generations.



Oregon Health & Science University via AP

In this microscope photo provided by Oregon Health & Science University, human embryos grow in a laboratory for a few days after researchers repair a heart disease-causing genetic mutation.

WASHINGTON (AP) — Altering human heredity? In a first, researchers safely repaired a disease-causing gene in human embryos, targeting a heart defect best known for killing young athletes — a big step toward one day preventing a list of inherited diseases.

In a surprising discovery, a research team led by Oregon Health and & Science University reported Wednesday that embryos can help fix themselves if scientists jump-start the process early enough.

It's laboratory research only, nowhere near ready to be tried in a pregnancy. But it suggests that scientists might alter DNA in a way that protects not just one baby from a disease that runs in the family, but his or her offspring as well. And that raises ethical questions.

"I for one believe, and this paper supports the view, that ultimately gene editing of human embryos can be made safe. Then the question truly becomes, if we can do it, should we do it?" said Dr. George Daley, a stem cell scientist and dean of Harvard Medical School. He wasn't involved in the new research and praised it as "quite remarkable."

"This is definitely a leap forward," agreed developmental geneticist Robin Lovell-Badge of Britain's Francis Crick Institute.

Today, couples seeking to avoid passing on a bad gene sometimes have embryos created in fertility clinics so they can discard those that inherit the disease and attempt pregnancy only with healthy ones, if there are any.

Gene editing in theory could rescue diseased embryos. But so-called "germline" changes — altering sperm, eggs or embryos — are controversial because they would be permanent, passed down to future generations. Critics worry about attempts at "designer babies" instead of just preventing disease, and a few previous attempts at learning to edit embryos, in China, didn't work well and, more importantly, raised safety concerns.

In a series of laboratory experiments reported in the journal Nature, the Oregon researchers tried a different approach.

They targeted a gene mutation that causes a heart-weakening disease, hypertrophic cardiomyopathy, that affects about 1 in 500 people. Inheriting just one copy of the bad gene can cause it.

The team programmed a gene-editing tool, named CRISPR-Cas9, that acts like a pair of molecular scissors to find that mutation — a missing piece of genetic material.

Then came the test. Researchers injected sperm from a patient with the heart condition along with those molecular scissors into healthy donated eggs at the same time. The scissors cut the defective DNA in the sperm.

Normally cells will repair a CRISPR-induced cut in DNA by essentially gluing the ends back together. Or scientists can try delivering the missing DNA in a repair package, like a computer's cut-and-paste program.

Instead, the newly forming embryos made their own perfect fix without that outside help, reported Oregon Health & Science University senior researcher Shoukhrat Mitalipov.

We all inherit two copies of each gene, one from dad and one from mom — and those embryos just copied the healthy one from the donated egg.

"The embryos are really looking for the blueprint," Mitalipov, who directs OHSU's Center for Embryonic Cell and Gene Therapy, said in an interview. "We're finding embryos will repair themselves if you have another healthy copy."

It worked 72 percent of the time, in 42 out of 58 embryos. Normally a sick parent has a 50-50 chance of passing on the mutation.

Previous embryo-editing attempts in China found not every cell was repaired, a safety concern called mosaicism. Beginning the process before fertilization avoided that problem: Until now, "everybody was injecting too late," Mitalipov said.

Nor did intense testing uncover any "off-target" errors, cuts to DNA in the wrong places, reported the team, which also included researchers from the Salk Institute for Biological Studies in California and South Korea's Institute for Basic Science. The embryos weren't allowed to develop beyond eight cells, a standard for laboratory research.

The experiments were privately funded; U.S. tax dollars aren't allowed for embryo research.

Genetics and ethics experts not involved in the work say it's a critical first step — but just one step — toward eventually testing the process in pregnancy, something currently prohibited by U.S. policy.

"This is very elegant lab work," but it's moving so fast that society needs to catch up and debate how far it should go, said Johns Hopkins University bioethicist Jeffrey Kahn.

And lots more research is needed to tell if it's really safe, added Britain's Lovell-Badge. He and Kahn were part of a National Academy of Sciences report earlier this year that said if germline editing ever were allowed, it should be only for serious diseases with no good alternatives and done with strict oversight.

"What we do not want is for rogue clinicians to start offering treatments" that are unproven, as has happened with some other experimental technologies, he stressed.

Among key questions: Would the technique work if mom, not dad, harbored the mutation? Is repair even possible if both parents pass on a bad gene?

Mitalipov is "pushing a frontier," but it's responsible basic research that's critical for understanding embryos and disease inheritance, noted University of Pittsburgh professor Kyle Orwig.

In fact, Mitalipov said the research should offer critics some reassurance: If embryos prefer self-repair, it would be extremely hard to add traits for "designer babies" rather than just eliminate disease.

"All we did is un-modify the already mutated gene."

Unwanted record: Biggest ever dead zone in Gulf of Mexico

NEW ORLEANS (AP) — There's an unwanted record in the Gulf of Mexico: This year's "dead zone ," a largely human-caused phenomenon where there's too little oxygen to support marine life, is the biggest ever measured.

The low-oxygen, or hypoxic , zone covers 8,776 square miles — about the size of New Jersey, the National Oceanic and Atmospheric Administration said Wednesday. The area is more than 3 percent larger than the 2002 dead zone.

"We predicted it would be large, and it is large," said scientist Nancy Rabalais, who has been measuring the dead zone since 1985.

She said the area was actually larger, but the July mapping cruise had to stop before reaching the western edge.

"The structure of the water column was changing, so I'm not sure how much larger it would have been," said Rabalais, of the Louisiana Universities Marine Consortium .

Rabalais said winds from the west and southwest apparently had also compressed the eastern half of the low-oxygen area closer to shore than she'd ever seen it. Without those winds, it probably would have covered a broader area, she said.

Studies in the spring had predicted the third-largest dead zone ever — nearly 8,200 square miles.

Those studies are based on examining nitrogen and phosphorus in the Mississippi River. The nutrients, which get carried down from the river, feed plankton blooms that die and sink to the bottom, where their decay uses oxygen.

The smallest measurements were during or after droughts: 1,696 square miles in 2000 and 15 square miles in 1988.

"This large dead zone size shows that nutrient pollution, primarily from agriculture and developed land runoff in the Mississippi River watershed is continuing to affect the nation's coastal resources and habitats in the Gulf," NOAA said in a news release.

- EMPLOYMENT OPPORTUNITY -

Part Time – Inside Salesperson

Great work environment. Super awesome team. Good base pay PLUS commissions. Retirement plan. Weekends off. Interested?

We are looking for a motivated, self-confident individual to join our inside sales team at East Oregonian in Pendleton.

We have an opening for a part time inside salesperson position.

No newspaper experience? No problem, as long as you understand the importance of great customer service, working hard and a desire to enjoy your job.

Could this be you?

We are seeking a dynamic salesperson to make prospecting phone calls to potential customers and businesses of all types, service existing accounts, be organized and have excellent computer and communication skills. If you've been looking for the opportunity to excel in a sales environment, this is it! The right candidate will be organized with a high attention to detail, have a desire to learn and grow their skills and work well in a team environment. Must be very accurate and detail oriented plus have excellent customer service and communication skills. Job qualifications include accuracy and speed when typing and spelling, excellent organizational, phone and communication skills. No sales experience required. Part-time, wage plus commission. Benefits include Paid Time Off (PTO) and 401(k)/Roth 401(k) retirement plan.

Send resume and letter of interest to **EO Media Group**,
PO Box 2048 • Salem, OR 97308-2048,
by fax to **503-371-2935** or e-mail **hr@eomediagroup.com**

EAST OREGONIAN

Come work with us! We are an awesome team.

The solar eclipse is coming August 21

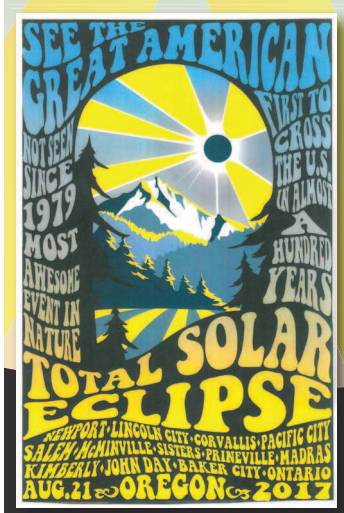
Come get the coolest eclipse glasses in town at your [illegible] East Oregonian office, [illegible] our [illegible] County Fair booth, Aug. 8-12

$1 EACH

Best [illegible] in town

SOLD OUT

Pick [illegible] for your friends and family while supplies last.



East Oregonian
1-800-522-0255

211 SE Byers Ave. Pendleton 333 East Main St. Hermiston