The results have been disastrous for Brazil's science ministry and its main funding agency, the National Council for Scientific and Technological Development (CNPq), which receives from FNDCT a large share of the money it hands out as grants. This year, the council expected to receive 1.2 billion reais ($350 million) from the fund; it has gotten just 27% of that amount. As a result, CNPq has not announced some of its usual granting programs, including the Universal Call open to all research fields, and canceled others. Officials have also delayed announcing awards in a major competition to establish new virtual research institutes; the winners were supposed to be announced this past March. And CNPq is withholding payments on some 5500 existing grants it awarded in 2014's Universal Call. "The money just wasn't there," says Glaucius Oliva, a researcher at the Physics Institute of São Carlos and a former president of CNPq.

Herculano-Houzel is among the scientists waiting for payments. She holds a CNPq grant for 50,000 reais, but has gotten just 6500. That's better, however, than the zero payout she's received on another grant, awarded by a state funding agency. To keep her lab operating, she's loaning her own money to her project. "I owe myself some 15,000 reais already," she says.

Paulo Artaxo, a University of São Paulo physicist, worries the cuts will hamper one of Brazil's newest research efforts. Just last week, officials celebrated the opening of the 325-meter-high Amazon Tall Tower Observatory, which will collect atmospheric data deep in the Amazon (*Science*, 6 March, p. 1051). But the funding crisis is threatening "our ability to secure funds for its operation and long-term measurements," Artaxo says.

Scientists blame some of the cuts in FNDCT grants on the Rousseff administration's determination to protect one of its flagship programs, Science Without Borders, which is sending 100,000 students (mostly undergraduates) to study abroad. Last year, the program received 1 billion reais from the fund, and it may get the same sum this year. Science Without Borders "is important" but shouldn't come "at the expense of the entire scientific community," Nader says.

The Brazilian Academy of Sciences and SBPC are pleading with Rousseff to find new sources of funding—but so far to no avail. In the meantime, the science ministry officials have negotiated a $2.5 billion loan from the Inter-American Development Bank to help researchers through the crisis, but Brazilian officials and lawmakers have yet to approve the deal. ■

REPRODUCIBILITY

Many psychology papers fail replication test

An effort to repeat 100 studies yields sobering results, but many researchers are positive about the process

By **John Bohannon**

The largest effort yet to replicate psychology studies has yielded both good and bad news. On the down side, of the 100 prominent papers analyzed, only 39% could be replicated unambiguously, as a group of 270 researchers describes on page 943. On the up side, despite the sobering results, the effort seems to have drawn little of the animosity that greeted a similar replication effort last year (*Science*, 23 May 2014, p. 788). This time around, many of the original authors are praising the replications as a useful addition to their own research.

"This is how science works," says Joshua Correll, a psychologist at the University of Colorado, Boulder, and one of the authors whose results could not be replicated. "How else will we converge on the truth? Really, the surprising thing is that this kind of systematic attempt at replication is not more common."

That's encouraging news to Brian Nosek, a psychologist at the University of Virginia in Charlottesville who led the effort. "I don't know if replication is 'entirely ordinary' yet, but it is certainly more ordinary than it was [a few] years ago." he says. In that time, major psychology journals have started publishing replications alongside original research. "The change is pretty remarkable."

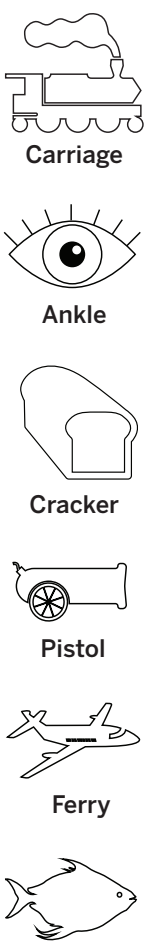
The mass replication effort began in 2011 with the goal of putting psychological science on more rigorous experimental footing. The strategy was to replicate a sample of published studies using an approach that Nosek has popularized through the Center for Open Science, a nonprofit he founded in 2013: publish your experimental design first, receive open peer review on it, and only then carry out the experiment and share the results, no matter the outcome. That should reduce the number of papers that report statistically significant results that are actually false positives.

In the Open Science Collaboration, 270 psychologists from around the world signed up to replicate studies; they did not receive any funding. The group selected the studies to be replicated based on the feasibility of the experiment, choosing from those published in 2008 in three journals: *Psychological Science*, the *Journal of Personality and Social Psychology* (*JPSP*), and the *Journal of Experimental Psychology: Learning, Memory, and Cognition*. Not only were all 100 replications preregistered, but the authors of the original studies were invited to collaborate in the design of the replication.

The results lend support to the idea that scientists and journal editors are biased—consciously or not—in what they publish. For example, even in studies that could be replicated, the size of the effect—a measure of how much of a difference there was between the experiment groups—was on average only half as big as the original studies. The bias could be due to scientists throwing out negative

Real effect?

A 2008 study found that people were slower to correctly name an object when shown the names of similar objects. A replication of this study failed to find that effect.



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results, for example, or journal editors preferentially accepting papers with bigger effects. Some of the replications even found the opposite effect compared with the original paper. "This very well done study shows that psychology has nothing to be proud of when it comes to replication," says Charles Gallistel, president of the Association for Psychological Science.

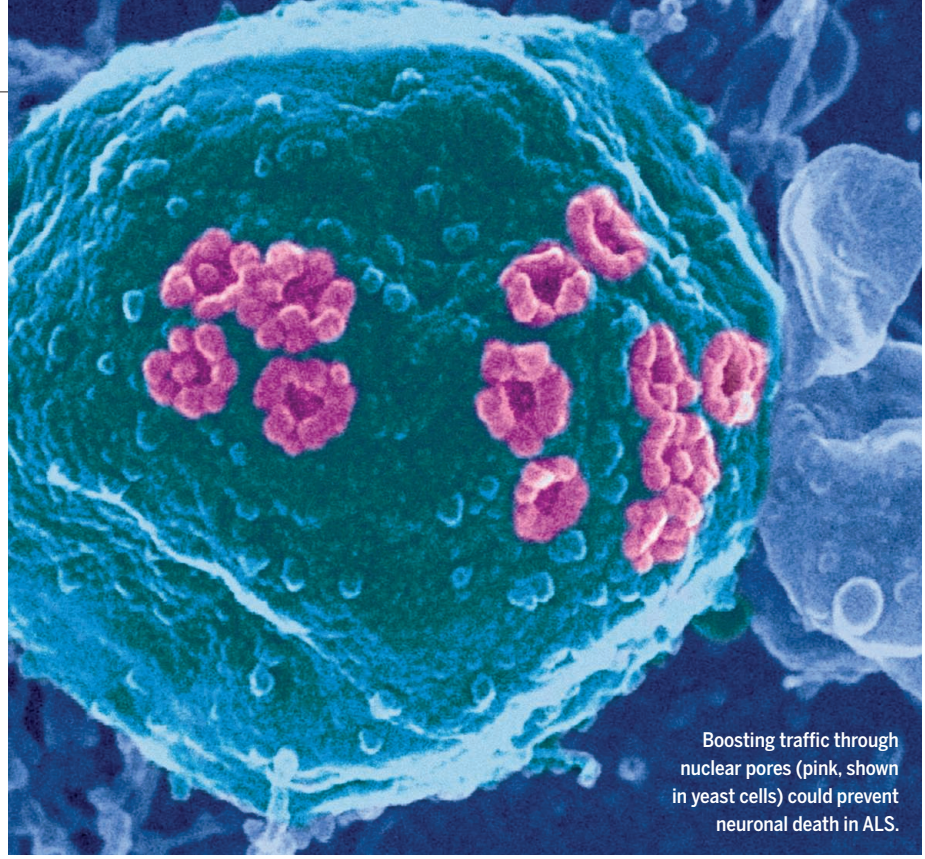
"Their data are sobering and present a clear challenge to the field," says Lynne Cooper, a psychologist at the University of Missouri, Columbia, who became one of the editors of *JPSP* in January. Already, she says, the journal is instituting reforms. In order to encourage researchers to test published results, *JPSP* will publish more replications, Cooper says. The journal is also launching new policies that will encourage "authors, editors, and reviewers ... to reexamine and recalibrate basic notions about what constitutes good scholarship," she says. The details have not yet been announced.

"Some will be tempted to conclude that psychology is a bad apple," says Charles Carver, a psychologist at the University of Miami who was one of the editors of *JPSP* in 2008. He insists this is not the case. "This is a problem of science, medical science no less than behavioral science." Replication efforts in other fields are equally low, says John Ioannidis, a biologist at Stanford University in Palo Alto, California, who suspects the true proportion of psychology papers that are not false positive is "something like 25% ... [which] seems to be in the same ballpark as what we see in many biomedical disciplines."

Like most researchers contacted by *Science*, Correll says the exercise was "worth the effort," no matter the outcome. That's not to say Correll is disavowing his earlier results. In his 2008 *JPSP* study, his team asked subjects to identify images of weapons while simultaneously showing them images of people of different races. The goal was to test the idea that differences in the reaction times indicate a person's implicit racial bias. They found that the variation in people's response times had a nonrandom signature known as 1/f noise that predicted their bias.

But when Etienne LeBel's lab at the University of Western Ontario in Canada repeated the experiment, they found 1/f noise, but the prediction did not hold. Correll says the failure "does not convince me that my original effects were [a] fluke. I know that other researchers have found similar patterns ... and my lab has additional data that supports our basic claim." He is already working on the follow-up study. ■

IMAGE: DR ELENA KISELEVA/SCIENCE PHOTO LIBRARY



Boosting traffic through nuclear pores (pink, shown in yeast cells) could prevent neuronal death in ALS.

NEUROSCIENCE

Plugged pores may underlie some ALS, dementia cases

Multiple groups reveal that "stutter" mutation kills nerve cells by clogging channels into the nucleus

By **Emily Underwood**

It is famous for robbing Lou Gehrig of his life and Stephen Hawking of his mobility and voice, but just how amyotrophic lateral sclerosis (ALS) destroys motor neurons in the brain and spinal cord remains a mystery. Now, scientists are converging on an explanation, at least for a fraction of the ALS cases caused by a specific mutation. In cells with the mutation, the new work shows, pores in the membrane separating the nucleus and cytoplasm become clogged, preventing vital molecules from passing through and creating a fatal cellular traffic jam.

For now, the work applies only to the mutation dubbed C9orf72—a DNA stutter in which a short nucleotide sequence, GGGGCC, is repeated hundreds to thousands of times in a gene on chromosome 9. Nor do the multiple labs reporting results this week agree on exactly what plugs those nuclear pores and how the cells die. Still, the work is "a major breakthrough" in ALS research, says Amelie Gubitz, program director of the neurodegeneration division at the National Institute of Neurological Disorders in Bethesda, Maryland. The groups worked independently, starting with different hypotheses and experimental designs, yet reached similar conclusions, making the finding more convincing. And it suggests that boosting traffic through nuclear pores could be a new strategy for treating some cases of ALS and frontotemporal dementia (FTD), another neurodegenerative condition C9orf72 can cause.

Based on past work by their own and other groups, neuroscientists Jeff Rothstein and Tom Lloyd at Johns Hopkins University in Baltimore, Maryland, suspected that the long strands of excess RNA produced by C9orf72 cause neurodegeneration by binding to, and thus sequestering, key cellular proteins. The team tested the idea in fruit flies with the mutation, which display damage in the nerve cells of their eyes and in motor neurons.

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