reported unwelcome, persistent advances from men at the conference. The analysis does not reveal what percentages of respondents reported these experiences, but does say that 15% of respondents were women.

Terrence Sejnowski, president of the foundation that oversees the conference, told *Nature* that the foundation's board, and others, had read the report with great interest, and thanked the authors for the analysis. "It provides us with valuable information for understanding our community," he said.

DIVERSITY MEASURES

The survey was carried out by Katherine Heller, a machine-learning researcher at Duke University in Durham, North Carolina, and Hal Daumé, a machine-learning researcher at the University of Maryland in College Park, who are the diversity and inclusion chairs at this year's event.

In December 2017, Sejnowski and the chairs of the boards of the 2017 and 2018 conferences acknowledged that several events held at or in conjunction with the 2017 conference had fallen short of the standards required to "provide an inclusive and welcoming environment for everyone". They said that they would take immediate action, including recruiting the diversity and inclusion chairs, formalizing the process for reporting concerns and strengthening an existing code of conduct, by which all attendees and sponsors will have to abide in future.

Their statement came shortly after several female machine-learning researchers spoke out about their experiences at last year's event in Long Beach, California, and other AI conferences, including a joke about sexual assault, allegedly made by a member of a band composed of leading researchers at a party coinciding with the 2017 event.

Other measures to improve inclusion include subsidized childcare and a diversity meeting. There are also now several ways for conference-goers with concerns to notify organizers.

And on 16 November, the board abandoned the commonly used acronym, NIPS, and renamed the event NeurIPS. A March 2018 letter to the board, signed by 122 academics at Johns Hopkins University in Baltimore, Maryland, said the NIPS acronym was "prone to unwelcome puns" and revealed further goings-on at the conference, including an unofficial sister event named "TITS" and T-shirts spotted bearing the slogan "my NIPS are NP-hard".

Researchers have mixed views about whether the board's efforts will bring meaningful change. Raia Hadsell, a machine-learning researcher at DeepMind in London who has been attending the conference for more than a decade has not witnessed a "rampant culture of discrimination, bias or harassment" at the event but has seen and experienced problematic behaviour. "I find it infuriating to be asked whether I am a recruiter, or a 'plus one', or whether I 'did the work myself' — do men ever, ever get asked questions like that?" she says.

She thinks that the machine-learning community wants to address the problems, but that their complexity makes it difficult. "I think that there will still be a problem come December in Montreal."

Elana Fertig, a computational biologist at Johns Hopkins University who signed the March letter to the board, says that altering the name is a powerful first step that has heightened awareness of the issues and shows that change is possible. But two of Fertig's students decided earlier this year not to attend the event because of the reported culture. And she worries about a backlash against the name change, noting that there were negative, sometimes threatening, comments that accompanied the debate over the change. ■

NEUROSCIENCE

Alzheimer's researchers seek better mice

Several teams are developing animal models that more closely mimic the disease in people.

BY SARA REARDON

Drug companies have spent billions of dollars searching for therapies to reverse or significantly slow Alzheimer's disease, to no avail. Some researchers argue that the best way to make progress is to create better animal models for research, and several teams are now developing mice that more closely simulate how the disease devastates people's brains.

The US National Institutes of Health (NIH), the UK Dementia Research Institute and the Jackson Laboratory — one of the world's biggest suppliers of laboratory mice — are among the groups trying to genetically engineer more-sophisticated rodents. Scientists are also probing the complex web of mutations that influence neurological decline in mice and people.

"We appreciate that the models we had were insufficient," says Bruce Lamb, a neuroscientist at Indiana University in Indianapolis who directs the NIH-funded programme. "I think it's sort of at a critical juncture right now."

Alzheimer's is marked by cognitive decline and the build-up of amyloid-protein plaques in the brains of people, but the disease does not occur naturally in mice. Scientists get around this by studying mice that have been genetically modified to produce high levels of human amyloid protein. These mice develop brain plaques, but no memory problems.

"I think it's sort of at a critical juncture right now."

Many experimental drugs that have successfully removed plaques from mouse brains have not lessened the symptoms of Alzheimer's disease in people. One high-profile stumble came last month, when three companies reported that their Alzheimer's drugs — from a class called BACE inhibitors — had failed in late-stage clinical trials. Although the drugs successfully blocked the accumulation of amyloid protein in mice, they seemed to worsen cognitive decline and brain shrinkage in people.

The drive for better mice comes as genomics studies are linking the most common form of Alzheimer's — late onset — to dozens of different genes. This diversity suggests that each case of the disease is caused by a different mix of genetic and environmental factors. "There is no single Alzheimer's disease," says Gareth Howell, a neuroscientist at the Jackson Laboratory in Bar Harbor, Maine.

Howell argues that scientists' reliance on inbred lab mice with only a few engineered mutations might have limited research. His own work suggests that, in mice, just as in people, genetic diversity plays a part in determining how neurodegeneration progresses.

When Howell's team modified two genes associated with early-onset Alzheimer's in both lab mice and their wild cousins, all of ▶

the animals developed amyloid plaques. But although the more-inbred lab mice did not display any outward signs of Alzheimer's, a portion of the genetically diverse wild mice experienced memory problems. The researchers think that a combination of plaques and unknown genetic factors caused these symptoms. They presented the results this month at a meeting of the Society for Neuroscience in San Diego, California.

Another study, by neuroscientist Catherine Kaczorowski at the Jackson Laboratory, suggests that animals' genetic make-up affects how they respond to environmental triggers. Her group bred genetically diverse wild mice with lab mice that had mutations that cause amyloid plaques to form. Some of the resulting offspring were more likely to develop cognitive problems if they ate a high-fat diet, but other mice on the diet had a lower risk of these symptoms, Kaczorowski reported at the San Diego meeting.

JACKSON LABORATORY

An Alzheimer's model mouse.

Understanding how this expanded universe of genetic factors affects Alzheimer's risk will require a host of new animal models with different combinations of mutations. Several efforts to engineer these next-generation mice are already under way.

In 2016, the NIH started the MODEL-AD consortium to develop more Alzheimer's mice and make them available to researchers. Project scientists engineer mice with different genetic mutations associated with early- or late-onset Alzheimer's, and test the animals to see whether they display signs of the disease. They then post descriptions of each mouse type in an online database. Lamb says that the team has released about 30 mouse varieties, and received more than 500 orders for the animals from academic scientists and biotechnology firms.

And in January, the UK Dementia Research Institute in London launched a similar programme. Scientists there are developing model mice whose brains show the amyloid plaques and tangles of another protein, called tau, that occur in people with Alzheimer's. To mimic the brain inflammation that the disease causes, the group is implanting neural immune cells grown from human stem cells into the brains of mice.

Ultimately, researchers hope that the models will reveal ways to predict whether a person will respond to a particular Alzheimer's therapy. And having a better understanding of how inflammation and genes drive the disease could help to identify it in people before plaques and tangles have formed, says Rudolph Tanzi, a neurologist at Harvard University in Cambridge, Massachusetts. "That's why it's so important to have those animal models available and really start working on all these genes."

But Bart de Strooper, a molecular biologist at the Catholic University of Leuven in Belgium, urges caution. De Strooper, who directs the UK programme, says that none of the next-generation animals is likely to be a perfect analogue for people. "The biggest mistake you can make," he says, "is to think you can ever have a mouse with Alzheimer's disease." ■

CORRECTION

The News Feature 'Why extreme rains are getting worse' (*Nature* **563**, 458–460; 2018) erroneously located Elizabeth Kendon in Reading. She is, in fact, at the Met Office in Exeter.