

## Abstracts

with those who had high serum glucose in all measurements. Two and 3 normal blood glucose measurements conferred a 61% and 67% reduced risk of developing diabetes, respectively.

This finding was irrespective of group assignment in the three-arm trial, which lasted 3 years and tested an intensive lifestyle intervention, metformin, and placebo in the prevention of diabetes in people with high risk at baseline. Only those participants who did not develop diabetes during the trial (72% (1 990/2 761)) were included in the observational study. Serum glucose was measured twice a year.

Unexpectedly, among people without normal glucose measurements, those randomised to a lifestyle intervention had a 30% higher risk of developing diabetes than those who received placebo. It is not clear how to interpret this finding; it could be that people who responded poorly to a lifestyle intervention might have somehow been more susceptible to developing diabetes, or those who responded well to placebo might have been less susceptible.

In any case, the study seems to support early and aggressive lowering of blood glucose in people at high risk of diabetes. The linked comment suggests that future research should explore whether a similar strategy might reduce the risk of complications, such as blindness or vascular disease, in full-blown diabetes.

Diabetes Prevention Program Research Group. *Lancet* 2012;379(9833):2243-2251; doi:10.1016/S0140-6736(12)60525-X.

### One in 10 tuberculosis cases in China is multidrug resistant

About a million new cases of tuberculosis are detected in China each year. A nationally representative survey of about 4 000 people with tuberculosis showed that, in 2007, one-third of new cases as well as more than a half of previously treated cases had tuberculosis resistant to at least one of the first-line drugs – isoniazid, rifampicin, ethambutol, and streptomycin. One in 10 people had multidrug-resistant tuberculosis – where the organism is resistant to at least isoniazid and rifampicin – and 1 in 120 was also resistant to second-line drugs ofloxacin and kanamycin. In most cases, the organism was already resistant at the point of infection.

Drug-resistant tuberculosis is on the rise worldwide, says a linked editorial. If the epidemic is to be contained, diagnosis and treatment need to be improved. Global policies already call for testing microbial resistance in all patients, but most programmes still focus only on patients considered to be at high risk.

New drugs, such as delamanid and bedaquiline, may soon be approved for second-line treatment on the basis of placebo-controlled trials. However, we don't know whether they can be given concurrently or how best to incorporate them into existing regimens.

Zhao Y, et al. *N Engl J Med* 2012;366(23):2161-2170.

## SINGLE SUTURE

### Immune cells gobble up healthy but idle brain cells

Use it or lose it: a class of immune cell demolishes idle circuits and connections in the brain, even a healthy one. Understanding more about the process could help prevent the onset of degenerative brain diseases.

Until now, microglia have been dismissed as simple immune cells that do little more than protect brain cells from damage and tidy up in the aftermath of disease.

'The idea they can clean up brain debris has been well established in studies of brain disease,' says Beth Stevens of Boston Children's Hospital. 'But now, even without damage, we've found them to respond to subtle changes in synaptic function.' Stevens and her colleagues manipulated mice to make one eye more active than the other, creating a disparity in activity between the two neural circuits linking the eyes to the brain. With the help of dyes to distinguish the signals from the left and right eyes, they saw in postmortems that microglia had preferentially pruned the connections, or synapses, from circuits serving the underactive eye. Synapses were marked out for destruction through labelling with an immune chemical called C3.

'We think C3 is an "eat me" signal,' says Stevens.

Finding out why some synapses but not others are earmarked for elimination could point to ways of preventing degenerative brain diseases.

'Early synapse loss is a feature of many neurodegenerative diseases,' says Stevens. 'So there's lots of excitement about finding out what the trigger is.'

'Microglia were considered to be just garbage collectors,' says Francesca Peri of the European Molecular Biology Laboratory in Heidelberg, Germany. 'They are turning out to be far more interesting – but before we can understand their role in disease, we need to understand what these cells do normally.'

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New Scientist, 1 June 2012.