from depression and psychosis to obsessive–compulsive disorder has been linked to the abrupt changes in biology and physiology that occur when the body responds to infection, especially in childhood. And some researchers have traced the possible chain of events back a generation. Studies have highlighted that pregnant women could react to infection in a way that influences their baby's developing brain, which could lead to cognitive and neurodevelopmental problems in the child.

One consequence of this 'maternal immune activation' (MIA) in some women could be to increase the risk of autism in their children. And two papers published online this week in *Nature* (S. Kim *et al. Nature* http://dx.doi.org/10.1038/nature23910; 2017 and Y. S. Yim *et al. Nature* http://dx.doi.org/10.1038/nature23909; 2017) use animal models to examine how this might happen, as well as suggest some possible strategies to reduce the risk.

Kim *et al.* looked at the impact of MIA on the brains and behaviour of mice. They found that pregnant female animals exposed to circumstances similar to a viral infection have offspring that are more likely to show atypical behaviour, and they unpick some of the cellular and molecular mechanisms responsible. Some of their results confirm what scientists already suspected: pregnancy changes the female mouse's immune response, specifically, by turning on the production of a protein called interleukin-17a. But the authors also conducted further experiments that give clues about the mechanisms at work.

The types of bacteria in the mouse's gut seem to be important. When the scientists used antibiotics to wipe out common gut microorganisms called segmented filamentous bacteria in female mice, this seemed to protect the animals' babies from the impact of the simulated infection. The offspring of mice given the antibiotic treatment did not show the unusual behaviours, such as reduced sociability and repetitive actions. Segmented filamentous bacteria are known to encourage cells to produce more interleukin-17a, and an accompanying News & Views article (C. M. Powell *Nature* http://dx.doi.org/10.1038/nature24139; 2017) discusses one obvious implication: some pregnant women could use diet or drugs to manipulate their gut microbiome to reduce the risk of harm to their baby if an infection triggers their immune response. Much science still needs to be done before such a course could be recommended — not least further research to confirm and build on these results.

Yim *et al.* analysed the developing brain of mice born to mothers who showed MIA. They traced the abnormalities to a region called the dysgranular zone of the primary somatosensory cortex (S1DZ). The authors genetically engineered the mice so that neurons in this region could be activated by light, and they showed that activation of S1DZ induced the same telltale atypical behaviours, even in mice that were born to mothers with no MIA.

"It's tempting to draw parallels with mechanisms that might increase the risk of autism in some people."

It's unusual to be able to demonstrate such a direct link between the activities of brain regions and specific behaviours — although plenty of work on mental disorders makes a strong theoretical case for linking particular conditions to over- and underactive brain zones and circuitry.

Encephalitis lethargica, for example, has been linked to changes in the deep regions of the basal ganglia, and the disease produces symptoms that are similar to those often seen in autism, including stereotyped and repetitive behaviours. Yim *et al.*'s study shows that the S1DZ region projects to one of those deep brain regions — the striatum — and that this connection helps to trigger repetitive actions in the animals. But S1DZ also connects to a separate, distinct, region in the cortex, and this is what seems to drive the changes in sociability.

Taking the two studies together, it's tempting to draw parallels with mechanisms that might increase the risk of autism in some people and explain some of its symptoms. Scientists and others should be cautious about doing so — much can change when results from animal models are applied to human biology. But the studies do offer some intriguing leads. ■

Face the heat

The giraffe's long neck could have evolved to help the animals keep their cool.

How did the giraffe get its long neck? The obvious answer — and some of you are probably shouting it at the page or screen right now — is that it evolved as a benefit that allowed the animals to reach and eat higher leaves. Perhaps. Probably, even. That was certainly Charles Darwin's explanation. But it's not certain, and other possible origins for one of the animal kingdom's most distinctive features are still a topic of debate among zoologists and evolutionary biologists alike.

One such idea is reported in the *Journal of Arid Environments* (G. Mitchell *et al. J. Arid Environ.* **145,** 35–42; 2017). Long-necked giraffes, scientists argue, can point their heads and necks towards the Sun, exposing less of their skin and making it easier for them to keep cool and survive the hot, dry conditions they often endure.

Improved thermoregulation is one of the later evolutionary explanations offered for the giraffe's long neck — a debate that goes back to before the time of Darwin. The French naturalist Jean-Baptiste Lamarck suggested that giraffes' necks became stretched as they constantly reached for foliage (an idea very much ahead of its time but for which he is sometimes unfairly ridiculed). Darwin and his contemporary Alfred Russel Wallace then famously turned this Lamarckism on its head, pointing out that the long neck would have come first, and this would have handed the taller individuals a significant advantage over shorter giraffes.

That idea stood largely unchallenged until, in a letter to this journal in 1949, Chapman Pincher took issue and pointed out that the legs of a giraffe are also unusually long (all the better for a swift escape from predators) (C. Pincher *Nature* **164,** 29–30; 1949). The long neck, he said, must therefore have evolved as a way for the animal to be able to reach past its own legs when it leans to reach the ground to take a drink of water. (Never very popular, Pincher's suggestion lasted only as long as it took scientists to find and examine fossil ancestors of the giraffe, and point out that those animals had managed perfectly well with long legs and short necks for millions of years.)

Other, more credible, alternatives to the dominant 'competing browsers' idea have emerged. One of the most popular is that long necks help male giraffes use their heads to bash rivals, or that females prefer them. Both would suggest that long-necked males are sexually selected.

And then there is thermoregulation. Originally, the suggestion was that long necks (and legs) significantly tilted the balance between volume and surface area that determines how quickly animals (and other bodies) gain and lose heat. Giraffes might look as if they have a larger than usual surface area compared with barrel shaped rhinos, elephants and others — but do they? It turns out that few people have tried to measure the surface area of enough giraffes to be sure. That's what the scientists do in the latest study.

They looked at measurements made for dozens of giraffes culled in Zimbabwe. They found that, pound for pound, the surface area of a giraffe is actually no larger than would be expected for any other animal of the same mass. And the creatures are no better at keeping cool, until, the scientists go on to suggest, they turn to face the Sun — as many giraffes are seen to do on hot days. ■