NEWS

Molecular mechanisms: Immune molecule boosts brain size

BY **JESSICA WRIGHT**

23 OCTOBER 2012

Capacious cavity: Brain structures that contain cerebrospinal fluid (blue) are larger in mice with activated immune systems (top) than in controls (bottom).

Mice with elevated levels of the immune molecule interleukin-6 have abnormally large brains, according to a study published 23 August in the *International Journal of Neuroscience*[1].

Epidemiological studies suggest that infection during pregnancy can **affect the fetal brain**. The **placenta** filters out infectious particles, making the mother's immune molecules the most likely cause of the brain and behavior changes seen in her child. In particular, cytokines — molecules that send activating signals to immune cells — have been linked to **brain abnormalities in the child**.

The cytokine interleukin-6 (IL-6) is **elevated in the brains and blood** of people with autism. In mice, boosting IL-6 levels leads to anxiety and cognitive and social deficits, and injecting it into a pregnant mouse leads to **social deficits in her pups**. Conversely, an antibody that inactivates IL-6 improves autism-like behaviors in these pups[2].

A study published in June showed that boosting the levels of IL-6 in mice changes the shape and length of **dendritic spines**, the signal-receiving branches of neurons[3]. In the new study, the same team looked at brain volume in these mice using magnetic resonance imaging.

Mice expressing excess IL-6 have brains that are 18 percent larger than those of controls, the

study found. This is likely to be the result of enlarged lateral ventricles, brain structures that contain cerebrospinal fluid. The ventricles are about 15 times bigger than those in controls, deforming nearby brain regions such as the hippocampus and the corpus callosum.

Children with autism have atypically large heads, or **macrocephaly**, up to **4 years of age**. The new study suggests that this could be the result of elevated cytokines, the researchers say.

References:

1: Wei H. *et al. Int. J. Dev. Neurosci.* **30**, 554-559 (2012) **PubMed**

2: Smith S.E. *et al. J. Neurosci.* **27,**10695-10702 (2007) **PubMed**

3: Wei H. *et al. Biochim. Biophys. Acta* **1822**, 831-842 (2012) **PubMed**