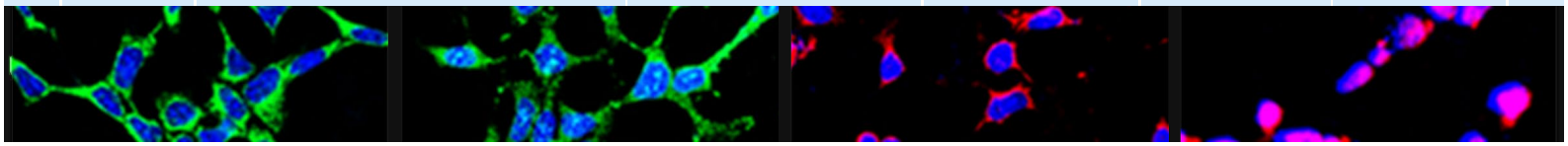
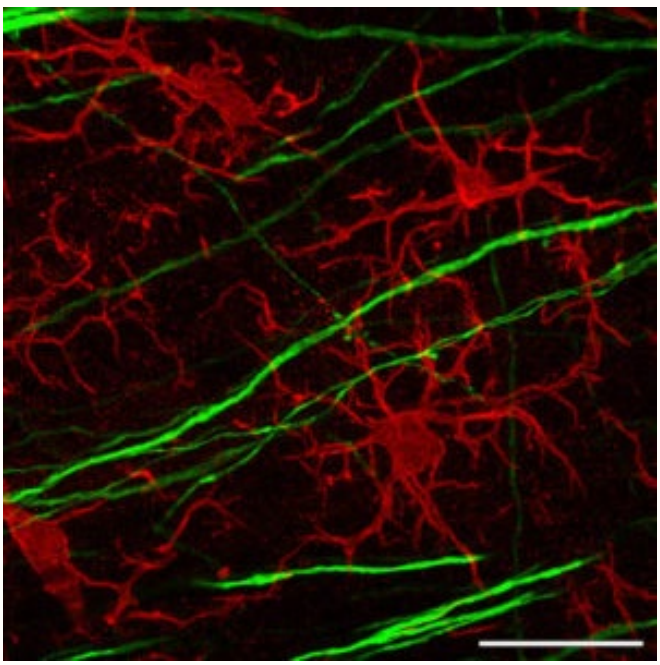


Early Inflammation May Play a Role in FTD

February 13, 2017 News in Brief Michelle Pflumm

Inflammation is a hallmark of neurodegenerative diseases including ALS and FTD. However, whether this process contributes to the onset of these diseases remains unclear. Now, University College London's Adrian Issacs and colleagues report that a mouse model of CHMP2B FTD exhibits early microglial activation in the brain more than 12 months *before* the first signs of the disease.

The study, published on January 16 in *Human Molecular Genetics*, found increased activated microglia in key FTD-affected regions of the brain. What's more, upon disease onset, these immune cells produced significant levels of the pro-inflammatory



Cause or consequence? *Immune system dysfunction is increasingly implicated in ALS and FTD due in part to the discovery of immune-related genes linked to these diseases. These genes include C9orf72 and TBK1 (see February 2015, March 2015 and March 2016 news). The defects include altered microglial function (O'Rourke et al., 2016). [Image: Activated microglia in the brain. Zeiss. CC BY-NC-ND 2.0].*

Search research news...
Enter search term, click "enter".
Sort by:
Date: ⊙ Relevance: ○

Filter articles published since 2015 by topic, disease, or article type.

Guidelines for filtering

Filter

Topic:
☐ Biomarkers
☐ Clinical Research
☐ Drug Development
☐ Genetics
☐ Laboratory Research
☐ New Methods
☐ Policy
☐ Research Models
☐ Risk Factors

Disease:
☐ Alzheimer's disease
☐ Amyotrophic lateral sclerosis
☐ Autism
☐ Cancer
☐ Crohn's disease
☐ FAP
☐ Frontotemporal dementia
☐ Hereditary neuropathies
☐ Huntington's disease

cytokines IL1β and TNFα. The symptoms, which occur late and include behavioral and motor deficits, resemble key aspects of the human disease. The CHMP2B form of FTD lacks TDP-43 pathology.

The results suggest that inflammation may occur early in the brain and drive the pathology of CHMP2B-linked disease. The study adds to growing evidence that immune system dysfunction may increase the susceptibility to neurodegenerative diseases (Miller et al., 2016; Miller et al., 2014).

Reference:

Clayton EL, Mancuso R, Nielsen TT, Mizielinska S, Holmes H, Powell N, Norona F, Overgaard Larsen J, Milioto C, Wilson KM, Lythgoe MF, Ourselin S, Nielsen JE, Johannsen P, Holm I, Collinge J, Frej A, Oliver PL, Gomez-Nicola D, Isaacs AM. Early microgliosis precedes neuronal loss and behavioural impairment in mice with a frontotemporal dementia-causing CHMP2B mutation. Hum Mol Genet. 2017 Jan 16. [PubMed].

Further Reading:

Miller ZA, Sturm VE, Camsari GB, Karydas A, Yokoyama JS, Grinberg LT, Boxer AL, Rosen HJ, Rankin KP, Gorno-Tempini ML, Coppola G, Geschwind DH, Rademakers R, Seeley WW, Graff-Radford NR, Miller BL. Increased prevalence of autoimmune disease within C9 and FTD/MND cohorts: Completing the picture. Neurol Neuroimmunol Neuroinflamm. 2016 Oct 28;3(6):e301. [PubMed].

Miller ZA, Rankin KP, Graff-Radford NR, Takada LT, Sturm VE, Cleveland CM, Criswell LA, Jaeger PA, Stan T, Heggeli KA, Hsu SC, Karydas A, Khan BK, Grinberg LT, Gorno-Tempini ML, Boxer AL, Rosen HJ, Kramer JH, Coppola G, Geschwind DH, Rademakers R, Seeley WW, Wyss-Coray T, Miller BL. TDP-43 frontotemporal lobar degeneration and autoimmune disease. J Neurol Neurosurg Psychiatry. 2013 Sep;84(9):956-62. [PubMed].

Share this: