

Featured Research Studies

Pediatr Neurol. 2008 Mar;38(3):163-76. L

Fetal mechanisms in neurodevelopmental disorders.

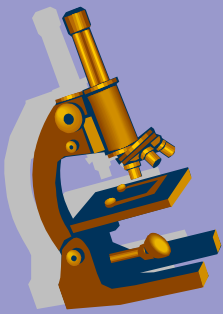
Connors SL, Levitt P, Matthews SG, Slotkin TA, Johnston MV, Kinney HC, Johnson WG, Dailey RM, Zimmerman AW.

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Normal development of the central nervous system depends on complex, dynamic mechanisms with multiple spatial and temporal components during gestation. Neurodevelopmental disorders may originate during fetal life from genetic as well as intrauterine and extrauterine factors that affect the fetal-maternal environment. Fetal neurodevelopment depends on cell programs, developmental trajectories, synaptic plasticity, and oligodendrocyte maturation, which are variously modifiable by factors such as stress and endocrine disruption, exposure to pesticides such as chlorpyrifos and to drugs such as terbutaline, maternal teratogenic alleles, and premature birth. Current research illustrates how altered fetal mechanisms may affect long-term physiological and behavioral functions of the central nervous system more significantly than they affect its form, and these effects may be transgenerational. This research emphasizes the diversity of such prenatal mechanisms and the need to expand our understanding of how, when altered, they may lead to disordered development, the signs of which may not appear until long after birth.

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<http://www.ncbi.nlm.nih.gov/pubmed/18279750?dopt=AbstractPlus>



Rheumatology (Oxford). 2008 Feb;47(2):208-11.

A subset of fibromyalgia patients have findings suggestive of chronic inflammatory demyelinating polyneuropathy and appear to respond to IVIg.

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OBJECTIVES: The aetiopathogenesis of the fibromyalgia syndrome (FMS) remains unknown. Recent reports, however, suggest that a subgroup of FMS subjects has an immune-mediated disease. Therefore, our primary objective was to study FMS subjects for evidence of an immune-mediated demyelinating polyneuropathy. Our secondary objective was to determine the effects of treating these FMS subjects with the immune modulator, intravenous immunoglobulin (IVIg).

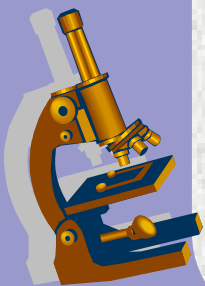
METHODS: Fifty-eight FMS subjects, 26 rheumatic non-FMS subjects and 52 non-rheumatic non-FMS subjects were studied. Subjective measures of paraesthesias, weakness, stocking hypaesthesia, pain, fatigue and stiffness were made. Objective measures of tenderness, proximal muscle strength and electrodiagnostic (EDX) evidence of polyneuropathy and demyelination were also made. Eleven other FMS subjects underwent sural nerve biopsy.

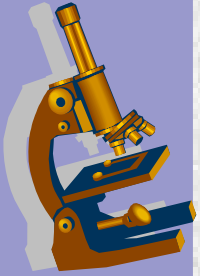
RESULTS: Paraesthesias, subjective weakness and stocking hypaesthesia were more common in FMS than in rheumatic non-FMS ($P < \text{or} = 0.0001$). Proximal muscle strength was less in FMS than in rheumatic non-FMS ($P < \text{or} = 0.0001$). EDX demonstrated a distal demyelinating polyneuropathy, suggestive of chronic inflammatory demyelinating polyneuropathy (CIDP), in 33% of FMS subjects. No rheumatic non-FMS subject had polyneuropathy ($P = 0.005$), or demyelination ($P = 0.05$). Fifteen FMS/CIDP subjects were subsequently treated with IVIg (400 mg/kg each day for 5 days). Pain ($P = 0.01$), tenderness ($P = 0.001$) and strength ($P = 0.04$) improved significantly. Fatigue and stiffness trended towards improvement.

CONCLUSIONS: A significant subset of FMS subjects have clinical and EDX findings suggestive of CIDP. IVIg treatment shows promise in treating this subset. These observations have implications for better understanding and treating some FMS patients.

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The hygiene hypothesis and psychiatric disorders

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The hygiene hypothesis proposes that several chronic inflammatory disorders (allergies, autoimmunity, inflammatory bowel disease) are increasing in prevalence in developed countries because a changing microbial environment has perturbed immunoregulatory circuits which normally terminate inflammatory responses. Some stress-related psychiatric disorders, particularly depression and anxiety, are associated with markers of ongoing inflammation, even without any accompanying inflammatory disorder. Moreover, pro-inflammatory cytokines can induce depression, which is commonly seen in patients treated with interleukin-2 or interferon- α . Therefore, some psychiatric disorders in developed countries might be attributable to failure of immunoregulatory circuits to terminate ongoing inflammatory responses. This is discussed in relation to the effects of the immune system on a specific group of brain serotonergic neurons involved in the pathophysiology of mood disorders.

Trends in Immunology
Article in Press, Corrected Proof

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