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DNA, increased cytokine production and constitutive activation of microglia. Rats lacking ATM had significant loss of motor neurons and microgliosis in the spinal cord, consistent with onset of paralysis. Betamethasone treatment extended *Atm* knockout rats' lifespan, prevented microglial activation and significantly decreased neuroinflammation and motor neuron loss. These findings provide a basis for neurodegeneration in A-T patients and rationale for anti-inflammatory drugs as treatment options for A-T patients and more generally in neurodegenerative diseases.

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Protection against development of a mouse model of multiple sclerosis by a parasite-derived 68-mer peptide

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Helminths (parasitic worms) can exert protective effects on autoimmune diseases by modulating the type of immune response, and deliberate infection with helminths is being explored as a potential therapeutic strategy for autoimmunity. However, the use of live helminths as therapeutic agents for autoimmune disease has a number of drawbacks, and it would be preferable to instead identify and use purified immunomodulatory components of the helminths. Previously it has been shown that the immunomodulatory activity of the liver fluke *Fasciola hepatica* resides in its excretory-secretory products (FhES), and further analysis of FhES has identified 3 major components: a 68 amino acid alpha helical cathelicidin-like peptide (FhHDM1), a cathepsin L-cysteine protease (FhCL1), and peroxiredoxin (FhPrx). In the current study, the ability of these three components to modify the course of a relapsing-remitting experimental autoimmune encephalomyelitis (EAE) model of multiple sclerosis (MS) was tested. FhHDM1 was the most effective, significantly ($p < 0.0001$) reducing the overall severity of the disease and the number of EAE relapses compared to mice treated with vehicle alone or mice treated with FhPrx. The effects were long-lasting, with mice continuing to show benefits for up to 70 days following a single course of FhHDM1 treatment. Preliminary investigation of the mechanism of action of FhHDM1 suggests that it does not affect the adaptive arm of the immune response, but instead exerts its effects by modulation of innate pro-inflammatory responses. The data suggest that this parasite-derived peptide has potential as a novel therapeutic agent for treatment of MS.

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Protective roles for microglia in neuroinflammation

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Microglia are central nervous system (CNS)-resident immune cells. They are often referred to as brain macrophages and indeed share many phenotypic features with them. However, it is now recognized that they are of a separate lineage and are not replaced from blood-derived precursors, at least under normal circumstances. Microglia are implicated in neuroinflammatory and neurodegenerative diseases including multiple sclerosis (MS). We have shown that in a mouse model for MS- experimental autoimmune encephalomyelitis (EAE) numbers of microglia expressing CD11c significantly increase. These $CD11c^+$ microglia are effective antigen presenting cells, but poor inducers of Th1 or Th17 responses. Interestingly, $CD11c^+$ microglia express neuroprotective insulin-like growth factor 1 which suggests a neuroprotective rather than proinflammatory role. Here we show that $CD11c^+$ microglia predominated in the neonatal brain and expressed genes governing neuronal and glial survival, migration and differentiation. These cells were localized in sites of primary myelination such as cerebellum and corpus callosum. They expanded rapidly after birth and then contracted to become

