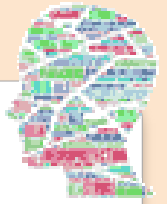


# MOLECULAR CHARACTERIZATION OF NMO AND MS PATIENTS



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## Introduction

Neuromyelitis optica (NMO) and Multiple Sclerosis (MS) are central nervous system demyelinating diseases. NMO has been recognized and differentiated from typical MS as a distinct disease on the basis of epidemiologic, genetic, pathogenic and clinical features. Clinical experience suggests that therapeutics used in MS have not been shown to be effective in NMO. Although the cause of both disorders is not known, abundant data advocate that are autoimmune diseases. In NMO the fundamental immunological process is driven by humoral mechanisms suggesting a Th2 immunopathological pattern, differing from the Th1 seen in MS.

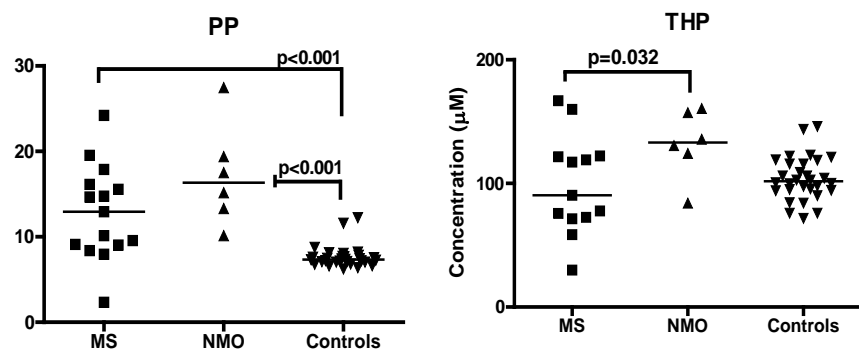
## Methods

Sera from clinically definite MS and NMO patients and apparently healthy subjects were screened in this study.

**Cytokines:** commercially available quantitative sandwich ELISA kits were used according to the manufacturers' instructions to quantify serum levels of proinflammatory TNF- $\alpha$  (Quantikine R&D) and IFN- $\gamma$  (Human ELISA Biotrak System, Amersham Biosciences) and regulatory cytokines IL-10 (Quantikine, R&D)

**REDOX Balance:** Serum levels of oxidative stress markers Malondialdehyde (MDA), advanced oxidation protein products (AOPP), peroxidation potential (PP), total hydroperoxides (THP) were evaluated using colorimetric techniques previously described

## Results



## Conclusions

Almost undetectable levels of TNF- $\alpha$  in R-NMO patients and a decreased production of IL-10 in both demyelinating diseases indicate a relevant pathogenic breakdown in T cell mediated immunoregulation.

A marked pro-oxidant environment is also developed probably as a consequence of the chronic inflammatory process contributing to the pathogenesis as well.

EDSS is as close to TIMP-1 and MMP-9 as is to the MMP-9/TIMP-1 ratio with a good degree of correspondence between the distances among points implied by the MDS map and the matrix input, by a stress of 0.0086.

