# MOG-IgG-ASSOCIATED DISORDER AND SYSTEMIC LUPUS ERYTHEMATOSUS DISEASE: SYSTEMATIC REVIEW.

Leticia Caroline Breis; Marco Antônio Machado Schlindwein; Isabelle Pastor Bandeira; Laura Fiuza Parolin; Pedro Weingrill, Rodrigo Kleinpaul, Marcus Vinícius Magno Gonçalves

Universidade da Região de Joinville – UNIVILLE, Joinville, SC - Brazil
Instituto de Neurologia – NEUROVIE, Joinville, SC - Brazil
Universidade Federal de Minas Gerais – UFMG, Belo Horizonte, SC - Brazil

## INTRODUCTION

Myelin oligodendrocyte glycoprotein (MOG) is expressed in oligodendrocyte and outer surface of myelin sheath[1]. MOG associated disorders (MOGAD) is a neurological entity which embraces acute disseminated encephalomyelitis (ADEM) and Neuromyelitis optica spectrum disorder (NMOSD), especially manifesting as transverse myelitis and optic neuritis[3]. The possible association of MOGAD and Systemic Lupus Erythematosus (SLE) is a relatively recent issue which is still not clearly understood. Therefore, we carried out a Systematic Review of the existing literature, which consists only in observational studies so far.

## METHODS

**The report of this systematic review was made according to the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement[6].** There was no previous protocol registered. We have systematically screened MEDLINE/ PUBMED (April 21st, 2020 - April 21nd, 2020), ScienceDirect (May 4, 2020), Google Scholar SciELO, LILACS, and Cochrane (May 5, 2020) in order to detect eligible papers.

## RESULTS

| Author | Year | Main Findings |
|---|---|---|
| Lalive et al.[5] | 2011 | Immunization of mice with MOG leaded to Auto-reactive B cell activation, MOG IgG antibodies elevation and anti-RNA and anti-ssDNA antibodies production |
| Mader et al.[9] | 2011 | MOG-IgG1 was found in Neuromyelitis optica (NMO) or High-Risk NMO patients without AQP4-IgG. (II) 27 SLE patients joined the control group, and 2 of them (2/27) had positive MOG-IgG. |
| Kovacs et al.[8] | 2016 | 5/6 patients had SLE before the NMO/NMOSD onset. 79% of the samples were AQP4-IgG positive and none had anti-MOG antibodies. |
| Asgari et al.[7] | 2018 | 2/30 (6,7%) of the NPSLE patients had AQP4-IgG and fulfilled the criteria of NMOSD. None of the AQP4-IgG seronegative patients had IgG antibodies against MOG. |
| Mader et al.[4] | 2018 | 3/6 of the NMOSD patients without AQP4-IgG had high titers MOG-IgG. |
| Soelberg et al.[11] | 2018 | Two patients (4%) had MOG IgG antibodies. One patient had MOG-IgG Optic Neuritis and weakly positive anti-Sm, without SLE symptoms or dsDNA antibodies |
| Bilodeau et al.[10] | 2019 | Case report: A 32-years old Native American woman presented with SLE onset and MOG-IgG associated LETM on post-partum period. |
| Probstel et al.[12] | 2019 | Among the 174 SLE patients, 14 (8%) had antibodies against MOG, and 6 of those had NPSLE (42,9%) |

## DISCUSSION

**Patients with NMOSD can develop SLE and vice versa, and NPSLE and NMOSD produce a similar pattern of demyelination[4].** When testing for MOG-IgG, it is important to keep in mind that this must be preferentially done during acute relapses and that immunosuppressive treatments for SLE can attenuate antibody levels[2,7].

## CONCLUSIONS

Not much is known about the association of MOGAD and SLE. **The overlap between these diseases (mainly NPSLE and NMOSD, due to the similar pattern of demyelination) is defended by some authors[4]; however, the existence of a separate disease is also possible due autoimmune predisposition[12].**
It is important to highlight that we have only observational studies in humans so far. This is a problem since many factors can contribute to the divergent data we found, such as different drugs, numbers of patients and immunoassays.

## REFERENCES

**Contact: Letícia Breis**
**breisleticia@gmail.com**
**+55 47 991760101**