

compared to 1/14 LGI1-Ab+patients( $p=0.0024$ ) and 1/12 healthy controls.

**Conclusion** Neuropathic pain may be present in both LGI-Ab + and CASPR2-Ab+patients, and is immunotherapy responsive. Serum IgG from CASPR2-Ab+patients more frequently bound sensory neurons and dorsal root ganglia, suggesting pathophysiological differences which may underlie the more severe pain in these patients.

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#### DISEASE REACTIVATION AFTER CESSATION OF DISEASE-MODIFYING THERAPY IN RELAPSING-REMITTING MULTIPLE SCLEROSIS

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**Objectives** To evaluate the rate of return of disease activity after cessation of multiple sclerosis (MS) disease-modifying therapy.

**Methods** This was a retrospective cohort study from two large observational MS registries: MSBase and OFSEP. Patients with relapsing-remitting MS who had ceased a disease-modifying therapy and were followed up for the subsequent 12-months were included in the analysis. The primary study outcome was annualised relapse rate in the 12 months after disease-modifying therapy discontinuation stratified by patients who did, and did not, commence a subsequent therapy. The secondary endpoint was the predictors of first relapse and disability accumulation after treatment discontinuation.

**Results** 18 029 eligible treatment discontinuation epochs were identified for seven therapies. Rates of relapse started to increase 2-months after natalizumab cessation. Commencement of a subsequent therapy within 2-4 months reduced the magnitude of disease reactivation. After discontinuation of fingolimod, rates of relapse increased overall, and stabilised faster in patients who started a new therapy within 1-2 months. Magnitude of disease reactivation for other therapies was low, but reduced further by commencement of another treatment 1-10 months after treatment discontinuation. Predictors of relapse were higher relapse rate in the year before cessation, female sex, younger age and higher EDSS. Commencement of a subsequent therapy reduced both the risk of relapse (HR 0.76, CI 0.72-0.81) and disability accumulation (0.73, 0.65-0.80).

**Conclusion** Understanding the rate of disease reactivation after discontinuing different MS immunotherapies will help guide optimal wash-out times for therapeutic agents during treatment sequencing.

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#### PREDICTING INFECTION RISK IN MULTIPLE SCLEROSIS PATIENTS TREATED WITH OCRELIZUMAB: A RETROSPECTIVE COHORT STUDY

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**Objective** To examine factors determining risk of self-reported infections and antimicrobial use in patients receiving Ocrelizumab for MS.

**Methods** Retrospective, observational cohort study conducted in Ocrelizumab-treated patients at the Royal Melbourne Hospital. The association of clinical and laboratory factors with self-reported infection rate and antimicrobial use were estimated using univariate and multivariable logistic regression models.

**Results** 185 patients were included in the study, and 176 infections were reported in 89 patients (46.1%), and in 47 patients (25.3%) antimicrobial use was identified. In univariate analyses, a higher serum IgA was associated with reduced odds of infection (OR 0.44, 95% CI 0.25 - 0.76). In multivariable analyses, older age (OR 0.94, 95% CI 0.88 - 0.99), higher serum IgA (OR 0.37, 95% CI 0.17 - 0.80) and higher serum IgG (OR 0.81, 95% CI 0.67 - 0.99) were associated with reduced odds of infection. Older age (OR 0.85, 95% CI 0.75 - 0.96) and higher serum IgA (OR 0.23, 95% CI 0.07 - 0.79) were associated with reduced odds of antimicrobial use, whilst longer MS disease duration (OR 1.22, 95% CI 1.06 - 1.41) and higher EDSS (OR 1.99, 95% CI 1.02 - 3.86) were associated with increased odds of antimicrobial use.

**Conclusions** Higher serum IgA, IgG and older age were associated with reduced odds of infection. Our findings highlight non-uniformity of infection risk in Ocrelizumab-treated MS patients, and substantiate the need to monitor immunoglobulin levels pre-treatment and whilst on therapy.

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#### REAL-WORLD EXPERIENCE WITH OCRELIZUMAB IN THE MSBASE REGISTRY – AUSTRALIAN RRMS COHORT

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**Introduction** Ocrelizumab (OCR) is a humanised anti-CD20<sup>+</sup> monoclonal antibody for the treatment of Multiple Sclerosis.