Please note that the report contains data for non-Roche molecules that are not yet licenced in the UK, and non-Roche medicines used outside of their licenced indication. Should you require further information on a molecule that is not produced by Roche Products Ltd., please contact the relevant manufacturer.
AAN 2018 Highlights

Introduction

We hope that you will find this information helpful. Should you require additional insights from the American Academy of Neuroscience (AAN) Annual Meeting 2018, please contact your local Roche MSL.

Roche AAN 2018 Steering Committee

Data covered in the AAN 2018 highlights were chosen by a Steering Committee of multiple sclerosis (MS) experts, who provided feedback on the updates they thought were most relevant to share with the UK clinical community:

- **Dr Owen Pearson (Chair),** Consultant Neurologist (Morriston & Neath Port Talbot Hospitals, Swansea)
- **Dr James Overell (Chair),** Consultant Neurologist (Institute of Neurological Sciences, Glasgow)
- **Dr Wallace Brownlee,** Consultant Neurologist (University College London Hospital)
- **Prof Jeremy Hobart,** Consultant Neurologist (Plymouth Hospitals NHS Trust)
- **Dr Niraj Mistry,** Consultant Neurologist (University Hospitals Birmingham NHS Foundation Trust)
- **Dr Kate Petheram,** Consultant Neurologist (City Hospitals Sunderland NHS Foundation Trust)
- **Dr Klaus Schmierer,** Reader in Clinical Neurology & Consultant Neurologist (Barts and The London School of Medicine and Dentistry)
- **Dr Nazar Sharaf,** Consultant Neurologist (Greater Manchester Neurosciences Centre)

These individuals share a range of interests within the field of MS and represent the geographic spread of MS expertise across the UK.

Video clips of the Steering Committee members can be viewed throughout the report by clicking on the video icons.
AAN 2018 Highlights
Key highlights

Dr James Overell, Dr Niraj Mistry, Dr Klaus Schmierer and Prof Jeremy Hobart reflect on the neurology updates they found most interesting at AAN 2018, including how these findings may impact UK practice, both now and in the future:

- **Dr Niraj Mistry** comments on the wealth of neurofilament light chain (NfL) measurement data presented across a range of trials, including its potential use as an accurate prognostic marker.

- **Dr Klaus Schmierer** discusses promising advanced imaging results from the Phase II SPRINT-MS trial of ibudilast, particularly the importance of magnetisation transfer ratio (MTR) measures as an indicator of MS pathology at the macromolecular level.

- **Prof Jeremy Hobart** explores the importance of screening and early diagnosis of other demyelinating disorders including Neuromyelitis Optica (NMO), Myelin Oligodendrocyte Glycoprotein (MOG) antibody-associated diseases and Glial Fibrillary Acidic Protein (GFAP) astrocytopathy, taking into consideration phenotypic similarities with MS.

### Monitoring MS Using Blood Neurofilament Light Protein

**Kuhle J, et al.**

Dr Jens Kuhle (Department of Biomedicine, University Hospital Basel) shared an overview of the potential for using NfL as a measure for prognosis and monitoring in MS, as well as the correlation between NfL levels and MS disease activity. Kuhle noted sequencing drugs is difficult in RRMS as it can be hard to distinguish each drug’s effect, therefore clinicians mostly rely on imaging to understand the impact of treatment. This could be facilitated by the use of more precise and reliable biomarkers of neuro-axonal injury. Current evidence suggests that as a measure of current disease activity, CSF and blood NfL levels are strongly associated, and that blood NfL is also increased at all stages of MS vs controls, whilst showing a consistent correlation with clinical and MRI disease activity. For example, in FREEDOMS (fingolimod vs placebo in RRMS) and TRANSFORMS (fingolimod vs INF-β1a in RRMS), baseline plasma NfL correlates with T2 lesion volume/Gd+ lesions. Kuhle highlighted blood NfL is also a marker of therapy response in MS: In FREEDOMS and TRANSFORMS, fingolimod treatment resulted in a significant and rapid reduction in blood NfL levels vs placebo/INF-β1a, respectively. Kuhle concluded that NfL also has potential as a prognostic marker of MS disease progression, stating that in the MSCRG study (pivotal trial of INF-β1a intramuscularly), serum NfL was associated with EDSS outcome with up to 12 years follow up.

### A Phase II Trial of Ibudilast in Progressive MS

**Fox R, et al.**

SPRINT-MS: a Phase II trial of ibudilast in progressive MS, examining change in brain atrophy as measured by brain parenchymal fraction over 96 weeks, and the safety and tolerability of ibudilast treatment. Results showed ibudilast was associated with a 48% reduction in the rate of brain atrophy progression (p = 0.04) (Figure 1). The AEs reported more commonly (p ≤ 0.1) with ibudilast included gastrointestinal (nausea, diarrhoea, abdominal pain and vomiting), rash, depression, and fatigue. There was no significant difference in either serious AEs or discontinuation rates between the groups. Ibudilast was associated with an 81% reduction in change in MTR (p = 0.047), a trend for improvement on transverse diffusivity (p = 0.15) and an 80% slowing in progression of cortical atrophy (p = -0.004). Results suggest activity of ibudilast in progressive MS and demonstrate the utility of advanced imaging methods in clinical trials to measure brain tissue integrity.

![Figure 1. Primary outcome: brain atrophy](image)

**References:**

RXUKNEUR00133; June 2018
This report includes information about unlicensed non-Roche products or indications in the UK.
Clinical Characteristics of MOG and AQP associated NMOSD in Adults

Capobianco M, et al.1

Comparison of the clinical characteristics of anti-MOG positive (MOG+) patients vs anti-AQP4 positive (AQP4+) NMOSD patients demonstrated isolated optic neuritis was the more frequent clinical presentation of MOG+ (14/15 MOG+ patients (Figure 1) vs 9/15 AQP4+ patients), while myelitis/LETM were more frequent in AQP4+ (6/15 MOG+ patients vs 8/15 AQP4+ patients). On balance, no specific clinical differences at presentation are associated with MOG or AQP4 antibodies even if optic neuritis seems to be more frequent for MOG antibodies. AQP4 and MOG antibodies should be tested in suspected NMOSD cases.

Figure 1. MOG+ clinical characteristics

Autoimmune GFAP Astrocytopathy: Prospective Evaluation of 90 Patients in 1 year

Dubey D, et al.2

In 2016, a novel form of meningoencephalomyelitis (MEM) known as autoimmune GFAP astrocytopathy was described. A prospective evaluation of the utility of serum and CSF GFAPα-IgG testing in both adults and children found MEM, or limited form, was the most common patient phenotype. MRI demonstrated patchy, irregular/perivascular-radial parenchymal enhancement with or without leptomeningeval enhancement in 51%. 92% of patients had inflammatory CSF, with the majority also having elevated CSF protein >50 mg/dl (82%) or lymphocytic pleocytosis (85%). 66/68 CSF GFAPa-IgG positive patients had MEM. Among cases with both serum and CSF samples available (54), where only 1 specimen type was positive, CSF had better positive predictive value for MEM (96% vs 0%). The majority of patients responded well to first-line immunotherapy (73%), though co-existing NMDA-R-IgG (56% vs 7%, p < 0.001) and cancer (31% vs 5%, p = 0.005) were associated with lack of initial response. A minority of patients (18%) can have a relapsing clinical course.

...An additional highlight:

Natalizumab EID is Associated with a Significant Reduction in PML Risk Compared with SID: Analyses of TOUCH® Prescribing Programme Data

Zhovtis Ryerson L, et al.3

Analyses of the TOUCH® Prescribing Program looked to determine whether natalizumab extended interval dosing (EID) was associated with reduced PML risk vs (standard interval dosing) SID. Analyses used three definitions of EID: (1) Primary definition: tests whether dosing history in the last 18 months of natalizumab treatment affects PML risk, (2) Secondary definition: tests whether an EID period occurring at any time in the dosing history affects PML risk, (3) Tertiary definition: tests whether a primarily EID dosing history affects PML risk. Findings demonstrate natalizumab EID (ADI >5 to ≤12 weeks) is associated with a clinically and statistically significant lower risk of PML vs SID (ADI ≥3 to <5 weeks) in anti–JC virus antibody patients. Most EID patients switched from SID to EID after >2 years of treatment, the ADI was 35–43 days for EID versus 30–31 days for SID, and patients with any gap in treatment of >12 weeks were excluded. As TOUCH® does not collect effectiveness data, additional prospective studies are needed to establish whether the effectiveness of natalizumab is maintained with EID.

Dr James Overell and Prof Jeremy Hobart discuss analyses exploring whether natalizumab EID is associated with reduced PML risk compared to standard interval dosing (SID).
ARPEGGIO: A Placebo-controlled Trial of Oral Laquinimod in PPMS
Giovannoni G, et al.¹

ARPEGGIO: Phase II, randomised, double-blind placebo-controlled study to assess the efficacy, safety and tolerability of oral, once-daily laquinimod vs placebo in patients with PPMS (n = 374). Eligible patients were originally assigned 1:1:1 to receive laquinimod 0.6mg or 1.5mg, or placebo. Following discontinuation of the 1.5mg dose due to safety concerns, patients were assigned 1:1:0.6mg laquinimod or placebo.² The primary endpoint was not met: at week 48, there was no difference in PBVC between laquinimod-treated and placebo patients (Figure 1). Adjusted mean difference 0.016%; p = 0.903. Laquinimod-treatment reduced new T2 lesions at week 48 vs placebo [mean new T2 lesions 0.7 vs 1.6, respectively; risk ratio 0.4; (95% CI 0.26, 0.69); p = 0.001]. Most common AEs in laquinimod-treated patients included nasopharyngitis (17%), headache (10%) and back pain (9%). Results do not support that laquinimod prevents clinical worsening in PPMS.

Figure 1. PBVC from baseline to Week 48

Uncoupling the Impact on Relapses and Disability Progression: Siponimod in Relapsing and Non-relapsing Patients With SPMS in the Phase III EXPAND Study
Cree B, et al.³

In EXPAND, siponimod reduced the risk of CDP by 21–26% in a typical SPMS population.⁴ In pre-planned subgroup analyses, siponimod treatment reduced the risk for CDP in both relapsing and non-relapsing SPMS patients. Post-hoc analyses using three different methods to control for the confounding impact of on-study relapses on disability progression showed a consistent impact of siponimod on the risk of CDP independent of relapses. Risk reductions of 13% and 18% in the non-relapsing SPMS-subgroup were observed for 3 month- and 6 month-CDP [HR 0.87, (CI 0.68; 1.11); 0.82 (0.62; 1.08)], and 33%–37% in the relapsing SPMS-subgroup. A principal stratum estimate determined that siponimod reduced disability in non-relapsing SPMS patients by 14–20% for 3 month-CDP and 29–33% for 6 month-CDP, suggesting that non-relapsing patients can achieve a large portion of the effect on the overall population. Study findings corroborate that siponimod is likely to be a useful treatment for SPMS.⁵

Figure 2. Effect of siponimod in patients without on-study relapses - principal stratum: non-relapsing patients

AEs: Adverse Events; CDP: Confirmed Disability Progression; DMT: Disease Modifying Therapy; PBVC: Percent Brain Volume Change; PPMS: Primary Progressive Multiple Sclerosis; SPMS: Secondary Progressive Multiple Sclerosis.


RXUKNEUR00133; June 2018
This report includes information about unlicensed non-Roche products or indications in the UK.
Dr Niraj Mistry and Dr Nazar Sharaf explore updates in diagnosis and monitoring at AAN 2018, including on the incorporation of NfL into NEDA, MAGNIMS score as a prognostic tool and results from the OCTIMS study – a 3 year longitudinal assessment of RNFL thickness measured by optical coherence tomography in RRMS.

Including Blood NfL in the NEDA Concept in RRMS Trials
Sormani MP, et al.¹

Post-hoc analysis of patients in the FREEDOMS study who provided consent for exploratory biomarker analysis and had NfL samples available at baseline and month (m) 24 (n=214), looked to evaluate whether blood NfL can substitute BVL as a component in the revised definition of NEDA-4. ROC analysis indicated blood NfL of 22.7pg/mL as the optimum level discriminating patients with a disability progression at m24. At m24, 24%, 16% and 20% of patients achieved NEDA-3, NEDA-4, and NEDA-NfL, respectively (Figure 1). Of the 42 NEDA-NfL patients, 67% were both NEDA-4 and NEDA-NfL. Probability of achieving NEDA-3 and NEDA-4 at m24 was higher in fingolimod-treated patients (OR = 2.9, p = 0.003 and OR = 2.4, p = 0.03). Adding NfL to NEDA-3 (i.e. NEDA-NfL) improved this probability (OR = 3.5, p = 0.001) vs placebo. NEDA-NfL deserves further evaluation across different compounds and in long-term studies.

Disease Activity as Assessed by MAGNIMS Score Predicts Long-term CDA-free Status and Disability Progression in Patients Treated With SC-IFNβ-1a
Sormani MP, et al.²

Long-term follow up in PRIMS-2 in patients with RRMS treated with SC-IFNβ-1a found the predictive ability of MAGNIMS score at Year (Y) 1 on clinical disease activity (CDA) and EDSS progression was maintained over the 15 year follow up. Median time to CDA event was longer in patients with Y1 MAGNIMS score of 0 (2.6 years) vs 1 (1.7 years; HR = 1.7) and 2 (1.3 years; HR = 2.4). Median time to EDSS progression was longer in patients with Y1 MAGNIMS score of 0 (7.5 years) vs 1 (4.0 years; HR = 1.5) and 2 (2.5 years; HR = 2.1) (Figure 2). Thus, the MAGNIMS score at Y1 is a good predictor of whether a patient will respond well to treatment in the long term.

Figure 1. Patients achieving NEDA status

Figure 2. Time to EDSS progression after Y1 by MAGNIMS score

References:
AAN 2018 Highlights

Diagnosis and monitoring continued

OCTiMS Study: A 3-Year Longitudinal Assessment of RNFL Thickness Measured by Optical Coherence Tomography in Patients with RRMS

Calabresi PA, et al.

OCTiMS: a 36-month, prospective, multi-centre, open-label, parallel group study in patients with RRMS (n = 350) and control subjects (n = 70) to evaluate change in RNFL thickness (without neurologic or ophthalmic disease). Patients with RRMS were classified into those having eyes with MS-associated optic neuritis (MS-ON cohort) and those without (MS-NON cohort). Mean pRNFL thicknesses were 91.8±13 μm, 82.5±13.5 μm, and 100.2±9.0 μm in the MS-NON cohort, MS-ON cohort and controls, respectively. Over time, pRNFL thickness decreased significantly in patients with MS vs reference subjects (difference in LSM at month 36: -1.86μm) (Figure 1). There were similar decreases in pRNFL thickness over time in the MS-NON and MS-ON subgroups. Change in pRNFL by 1 unit (μ) was associated with 1 unit (cm³) change in normalised brain volume. Test-retest analysis found that measurements of pRNFL by OCT are consistent and reproducible in a multicentre setting. Prospective evaluation of pRNFL changes in multicentre studies could be a potential endpoint to use in MS clinical trials.

Figure 1. Mean change in pRNFL from baseline over time in patients with MS vs control subjects

Dr James Overell and Prof Jeremy Hobart discuss when and whether to treat patients with RIS, including the long term implications of treatment and an understanding of prognosis in these patients. The clinicians also explore limitations of existing UK clinical guidelines related to the management and treatment of the syndrome.

...An additional highlight:

Management of Radiologically Isolated Syndrome

Goldman MD, Krieger S and Miller AE.

Prof Aaron E. Miller (Professor of Neurology at the Mount Sinai School of Medicine), Prof Myla D. Goldman (Associate Professor of Neurology, University of Virginia) and Prof Stephen Krieger (Associate Professor of Neurology, Mount Sinai Hospital) shared a series of challenging case vignettes, each presenting with one or more specific clinical issues relating to MS. In particular, the speakers explored the management of radiologically isolated syndrome (RIS), including whether it should be treated and how long patients should be scanned for. The ongoing TERIS³ and ARISE⁴ trials in RIS examining teriflunomide and dimethyl fumarate vs placebo, respectively, were highlighted. The primary outcome measure for both trials is the time to the first acute or progressive neurological event resulting from CNS demyelination from randomisation into the trial, with results expected in 2021 and 2022, respectively.³,⁴

Video: Click icon to watch video

References:
Non-myeloablative HSCT is Superior to DMD Treatment in Highly Active RRMS: Interim Results of the MIST Randomised Trial

_Burt R, et al._

MIST: 110 patients on stable DMDs with >2 relapses within the prior 12 months with EDSS 2 to 6 were randomised (1:1) to treatment with cyclophosphamide and rabbit anti-thymocyte globulin followed by HSCT or continued DMD, to assess treatment failure defined as an increase in EDSS. With a mean follow up of 3 years (range 1 to 5 years), treatment failure was 59% (30 of 51) for control arm and 6% (3 of 52) for HSCT (P < 0.001). During the first year after HSCT, mean EDSS improved from 3.4 to 2.4 while it worsened from 3.3 to 3.9 in the control arm (P < 0.001). During the first year after HSCT, the median volume of MRI T2LV decreased by 26.6%, and increased by 34.5% in the control arm. 48 months after HSCT, the percentage of patients with NEDA failure-free survival was 80%, and 5% in the control arm (p < 0.0001) (Figure 1). In conclusion, HSCT for RRMS with >2 relapses a year was superior to continued DMDs.

Figure 1. Time to NEDA failure (no relapses, no progression, no new/enlarging MRI lesions) HSCT vs DMT (p <0.0001)

*Defined as no new Gd+ T1 lesions on current MRI or new/enlarging T2 hyperintense lesions since last MRI; *Alemtuzumab vs SC IFNβ-1a at Year 2 based on the McNemar test; statistical comparisons were not performed for Years 3-5 of alemtuzumab follow up.


Alemtuzumab-Treated Patients Experienced Decreased MRI Disease Activity and Slowing of BVL Over 5 Years After Switching From SC-IFNβ-1a: Follow-up of Patients From CARE-MS I (TOPAZ Study)

_Rovira A, et al._

TOPAZ: (n = 122), a follow up analysis examining alemtuzumab in patients with RRMS who were treated with SC-IFNβ-1a for 2 years in the CARE-MS I or II trials. 118 patients (97%) completed TOPAZ Year (Y) 1 (Y5 after initiating alemtuzumab). 59% were MRI disease activity-free in SC-IFNβ-1a Y2, significantly increasing in post-alemtuzumab Y2 (82%; p < 0.001), and remaining high in Y3–5 (72%–67%) (Figure 2). Median BPF change from baseline to post-alemtuzumab Y5 was −1.88%; median annual BPF change improved post-alemtuzumab (Y1–Y5: −0.07%, −0.15%, −0.05%, 0.01%, −0.13%) versus SC IFNβ-1a Y2 (−0.50%). 71% of patients received no additional treatment (no additional alemtuzumab courses and no other DMT) in the extension through year 5 post-alemtuzumab. These findings are consistent with significantly greater improvements in MRI and BVL outcomes observed with alemtuzumab vs SC-IFNβ-1a over 2 years in the CARE-MS I core study.

Figure 2. MRI disease activity from Y1-5

BPF: Brain Parenchymal Fraction; BVL: Brain Volume Loss; DMD: Disease Modifying Drug; DMT: Disease Modifying Therapy; EDSS: Expanded Disability Status Scale; HSCT: Haematopoietic Stem Cell Transplantation; MRI: Magnetic Resonance Imaging; NEDA: No Evident Disease Activity; RRMS: Relapsing Remitting Multiple Sclerosis; SC-IFNβ-1a: Subcutaneous Interferon Beta-1a; SPMS: Secondary Progressive Multiple Sclerosis; T2 LV: T2 Lesion Volume.

References:

RXUKNEUR00133; June 2018
This report includes information about unlicensed non-Roche products or indications in the UK.
A Randomised Controlled Pilot Trial of Aspirin to Improve Exercise Performance in Persons with MS

Leavitt V, et al.¹

A randomised controlled pilot trial of aspirin to improve exercise performance in patients with MS (n = 12). At enrolment, 8/12 patients reported heat-sensitivity. All participants completed two exercise sessions separated by one week. At each session, participants were administered aspirin 650mg or placebo. Exercise performance (Time To Exhaustion) improved after aspirin vs placebo (mean difference = 6.4 ±23.7 seconds); t(11) = 2.405, p = 0.035 (Cohen’s d = 1.45). In heat-sensitive patients, the effect of aspirin was larger: 26.1± 22.2 seconds; t(7) = 3.321, p = 0.013 (Cohen’s d = 2.51). In the full sample, exercise-induced body temperature increase did not differ; however in the heat-sensitive subgroup, there was a 56% reduction in body temperature increase after aspirin vs placebo. In conclusion, aspirin may represent an effective treatment that allows more people with MS access to the many benefits of exercise.

Predictors of Fatigue Severity in MS Patients

Alalawi Z, et al.²

Retrospective analysis of data collected on 50 MS patients, obtaining a fatigue level score through the Fatigue Severity Scale questionnaire (FSS). The group was divided into high, low and intermediate fatigue score. There was a positive correlation with age and fatigue scores in the total population (n = 50, correlation coefficient = 0.311, p = 0.028) (Figure 1) and a positive correlation between BMI and fatigue score in the high fatigue group (n=17, correlation coefficient = -0.658, p = 0.004). No such relationship was found in the low fatigue arm (n = 14, correlation coefficient = 0.12, p = 0.682). There was no correlation between lesion load of Gd+ or vitamin D level and fatigue score. In conclusion, age is a positive predictor of fatigue score whereas high BMI is positively related to high fatigue levels in MS patients. Further studies are needed to evaluate whether high BMI might be associated with increased fatigue or whether excessive fatigue eventually leads to decrease in overall activity and weight gain.

Relation Between Fatigue Severity Scale And Pupillary Indices In Patients With MS

Hu Y, et al.³

Automated pupillometry of 11 MS patients experiencing severe (n = 8 patients, 13 eyes), or moderate to mild (n = 3, 5 eyes) fatigue demonstrated severe fatigue was associated with significantly shorter LoC and LmC compared with the moderate to mild patients (p = 0.042, p = 0.026 respectively). Similarly, severe fatigue was associated with a trend of larger Ac (p = 0.073) and faster Vc (p = 0.068). FSS was inversely correlated to LoC (r = -0.50, p < 0.05) and LmC (r = -0.48, p < 0.05). These preliminary results indicate that the pupillary function may serve as a biomarker for fatigue in MS.
Predictors of Neurodegeneration in Idiopathic REM Sleep Behaviour Disorder: A Multicentre Cohort Study

Postuma R, et al.¹

Prospective follow-up data from a 24 centre cohort of polysomnography-proven idiopathic REM sleep behaviour disorder (n = 1280) were combined to define neurodegenerative disease risk and predictors of neurodegeneration. The risk rate of developing neurodegenerative disease overall was 6.3% per year, whilst 50% of patients had phenotype-converted at 7.5 years and 73% by 12 years (Figure 1). Risk of disease conversion to Parkinson’s or dementia was increased with abnormal motor symptoms (HR = 3.30), subtle motor signs (HR = 2.11) and abnormal quantitative motor testing (HR = 3.46). Mild cognitive impairment and colour vision predicted primary dementia (HR = 2.37 and 1.69) but not primary parkinsonism. There was no predictive value for somnolence, insomnia, urinary dysfunction, erectile dysfunction, depression or anxiety.

Primary Results of PROMISE-1 Trial: a Phase III, Randomised, Double-blind, Placebo-Controlled Study to Evaluate the Efficacy and Safety of Eptinezumab for Prevention of Frequent Episodic Migraines

Saper J, et al.²

The PROMISE-1 Phase III study examined eptinezumab (300mg (n = 224), 100mg (n = 223) or 30mg (n = 219)) vs placebo (n = 222) for the prevention of frequent episodic migraines. Patients had 8.4 (placebo), 8.7 (30mg), 8.7 (100g) and 8.6 (300mg) mean migraine days/month. Eptinezumab 30mg, 300mg and 100mg reduced mean monthly migraine days by -4.0, -3.9 and -4.3 days respectively, vs placebo (-3.2 days, p = 0.0046, p = 0.0182 and p < 0.0001 respectively) (Figure 2). Migraine day prevalence dropped over 50% on Day 1 and reduction was sustained through Day 28 in subjects receiving eptinezumab. 37% achieved a ≥ 75% reduction in monthly migraine days over Weeks 1-12 in the 300mg dose group. TEAE rates were similar to placebo and the safety profile was consistent with previous eptinezumab studies.

Figure 1. Neurodegenerative disease-free survival

Figure 2. Eptinezumab significantly decreased monthly migraine days: weeks 1-12

References:

HR: Hazard Ratio; MS: Multiple Sclerosis; REM: Rapid Eye Movement; TEAE: Treatment-Emergent Adverse Events.

*ANCOVA model used to test for differences between treatment groups; †unadjusted.

RXUKNEUR00133; June 2018
This report includes information about unlicensed non-Roche products or indications in the UK.
AAN 2018 Highlights
Take home messages

The Steering Committee of MS experts share their key take home messages from the conference, including stand out data and implications for the future treatment of MS and related disorders in the UK, from both a clinical and patient viewpoint.

Dr Wallace Brownlee
"With respect to the EXPAND study post-hoc analysis, further research is needed to clarify whether stratifying patients by relapse incidence is the best way to understand the consistent benefit of siponimod on CDP in MS."

Prof Jeremy Hobart
“A disappointing amount of new information was presented on MS symptoms at AAN 2018, and from the perspective of people with MS there is a real need for us to get back into researching symptom management more extensively.”

Dr Niraj Mistry
“Whilst clinical information is still relevant and important, it is good to see all the new functionality in testing for MS diagnosis and monitoring that is about to come on stream.”

Dr James Overell
“One of the things we should concentrate on in the UK is more enhanced clinical assessment of people with RIS – looking at cognition and more detailed markers of whether a patient might be, at least to some extent, disabled even at an early stage.”

Dr Owen Pearson
“Data looking at application of the 2017 McDonald criteria to patients diagnosed since 2010 were particular interesting. As the new criteria significantly shortened the time to diagnosis, applying them could potentially shorten the time to treatment.”

Dr Kate Petheram
“An increase in core body temperature tends to be one of the factors that limits a patient’s ability to exercise effectively, and it is interesting to see aspirin may be able to improve this ability in patients with MS.”

Dr Klaus Schmierer
“Despite the disappointing, negative results from ARPEGGIO, it is still an important result to report - as many organisations do not - and in that respect is an achievement.”

Dr Nazar Sharaf
“The most important key highlight is that things are evolving with time - now we have an improved understanding that MRI scans can better detect disease activity, rather than relying on medical history and clinical examination alone.”

CDP: Confirmed Disability Progression; MRI: Magnetic Resonance Imaging; MS: Magnetic Resonance; RIS: Radiologically Isolated Syndrome.