13th Post-ECTRIMS Meeting: review of the new developments presented at the 2020 ECTRIMS Congress (II)

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Introduction. For more than a decade, after the ECTRIMS Congress, Spain has hosted the Post-ECTRIMS meeting, where neurologists with expertise in multiple sclerosis (MS) meet to review the new developments presented at the ECTRIMS.

Aim. This article, published in two parts, summarises the presentations of the post-ECTRIMS meeting, held online on 16 and 17 October 2020.

Development. This second part highlights the importance of gender and age in understanding the pathology of the disease and optimising its management. The advances made in paediatric MS, from a neuropsychological and neuroimaging point of view, are presented. In turn, special attention is paid to the findings that contribute to a more personalised approach to therapy and to choosing the best treatment strategy (pharmacological and non-pharmacological) for each patient. Similarly, results related to possible strategies to promote remyelination are addressed. Although there are no major advances in the treatment of progressive forms, some quantitative methods for the classification of these patients are highlighted. In addition, the study also includes results on potential tools for the assessment and treatment of cognitive deficits, and some relevant aspects observed in neuromyelitis optica spectrum disorders. Finally, the results of the papers considered late breaking news at the ECTRIMS-ACTRIMS are detailed.

Conclusions. Most of the advances presented were related to the knowledge of paediatric MS, remyelination strategies and cognitive assessment in MS.

Key words. ACTRIMS. Congress. ECTRIMS. MS. Multiple sclerosis. Post-ECTRIMS.

Introduction

This article is the second part of the summary of the papers presented at the 13th post-ECTRIMS Meeting, held online on 16-17 October 2020.

Gender-associated factors in pathogenesis and management

Although differences in drug metabolism and adverse events (AEs) have been observed between men and women, gender is often not taken into account in the design of clinical trials, nor in the reporting of AEs in studies and on drug product data sheets [1].

Among the factors that may influence susceptibility to the disease due to gender are the hormonal changes that occur in women throughout their lives [2]. The strongest evidence comes from data collected during pregnancy, where increased sex hormones are accompanied by a decreased risk of relapse, while the decrease in hormones in the postpartum period leads to a rebound in activity, although these attacks have been reduced with the use of disease-modifying therapies (DMT) [3]. In a study using data from 1,640 pregnancies from the MSBase registry, it was concluded that the use of highly effective DMT before conception can prevent relapses during pregnancy [4]. Menarche, pregnancy and lactation do not appear to influence long-term disability [5]. After menopause, however, a more progressive phase does appear, although it may be associated with ageing *per se* [6].

Multiple sclerosis (MS) is two to three times more common in women [7] perhaps because the X chromosome expresses multiple genes with an immune function, with the brain being the organ where they are expressed the most [8]. Also, differences in X chromosome DNA methylation between the parents may lead to differences in gene expression in the two sexes during the immune response [9]. Transcriptional regulation may differ depending on the sex. In experimental autoimmune en-

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cephalomyelitis (EAE) p38 signalling in microglia appears to be protective only in males [10].

Advances in paediatric multiple sclerosis

Results from the Swedish multiple sclerosis register

The prevalence of paediatric MS is 5-10% [11]. The prognosis of this disease is not as favourable as previously believed. They take around 10 years longer to reach progressive stages, but they do so at an earlier age. The course of the disease is different, with a higher number of relapses in the first five years, and a faster and more complete recovery from them [12].

Using the Swedish MS register (SMSreg), a slowing of information processing and impairment of executive functions was observed as of early stages of the disease. This cognitive impairment is early and independent of age and disease duration [13].

The impact of early-onset MS also has community health implications. These people are less likely to pursue higher education studies than the general population, and require more disability pensions at a younger age [11]. This spills over to the next generation, where children of people with MS have fewer higher qualifications and lower purchasing power [11].

Neuroimaging

Paediatric patients have a high inflammatory activity. Already at a very early age, black holes and atrophy can be observed, even despite a short disease duration and the absence of progression [13]. One of the great challenges is to find correlations between the changes observed with neuroimaging techniques in the white matter and in the grey matter, as well as, in turn, between each of these changes and cognitive function or fatigue [14].

Neuropsychological profile

Cognitive impairment in paediatric patients is not limited to a slowing of processing speed. Other cognitive domains such as episodic memory, visuospatial and visuomotor function and language are also impaired, regardless of the time course of MS [15].

Ageing

Ageing and multiple sclerosis phenotypes

Age modifies the clinical phenotype [16]. The older the age, the longer the time to recovery from the relapse, the greater the disability if initiation of DMT is delayed, and the lesser the effect of the DMT will be [17]. Additionally, the pre-progression stage depends only on age, and not on the previous clinical stage [18]. The changes in biological mechanisms that take place during ageing play against MS. Senescence of genetic-epigenetic mechanisms would modulate senescence of the immune system, microglia, the neuron-astrocyte complex and oligodendrocyte functions. The future development of treatments should take into account these mechanisms affected by ageing [17].

Neuroimaging

Age also modifies the radiological phenotype [19]. Age is one of the endogenous factors that increases the likelihood of conversion [20], and is associated with a lower likelihood of gadolinium (Gd^+)-enhancing T_1 lesions and a greater likelihood of atrophy.

An age-brain paradigm has been established with which the age of a patient's brain can be predicted on the basis of its degree of cerebral atrophy. The difference between the calculated age and the actual age has been termed brain-PAD (brain-predicted age difference) and the greater the difference, the older the brain is. A high brain-PAD occurs in patients with MS and is associated with a faster disability progression [21].

Immunological perspective and intervention

The concept of 'inflammaging' describes chronic inflammation that increases with age and, together with immunosenescence, contributes to neurodegenerative diseases [22]. Premature immunosenescence plays an important role in the progression of MS. Levels of nerve growth factor β differ depending on age, and pro-inflammatory interleukins (IL-18) are increased in older patients, especially in untreated males [23]. Possible interventions suggested to decrease 'inflammaging' include calorie restriction and reduced methionine intake [24].

Personalised treatment approaches

Identification of the optimal treatment for each patient

Personalised medicine aims to tailor treatment to individual patient characteristics, taking into ac-

count various factors (Fig. 1). As the therapeutic arsenal grows, and it is unknown whether a patient will respond favourably to a given treatment [25], choosing which one to use becomes quite a challenge [26]. For this reason, several prediction models have been developed, including the two-stage model, which takes the risk variables of each patient into account. This model was applied to 3,590 patients receiving natalizumab, dimethyl fumarate, glatiramer acetate or placebo in several clinical trials and results showed that the baseline risk of relapses modifies the relative and absolute effects of the drugs [27].

Specific biomarkers of treatment response

The implementation of certain biomarkers will allow for an increasingly personalised form of medicine. A prospective follow-up study in relapsing remitting MS (RRMS) has shown that, in patients with similar demographic and clinical characteristics, low percentages of $CD19^+$ B cells in blood or plasmablasts before starting alemtuzumab treatment predicted a lower risk of autoimmune AEs [28].

The predictive value of serum neurofilament light chains (NfL) for disease activity has been studied in patients with clinically isolated syndrome (CIS) and RRMS with DMT. The results showed that NfL levels are predictive of activity and could therefore be used to monitor the patient [29].

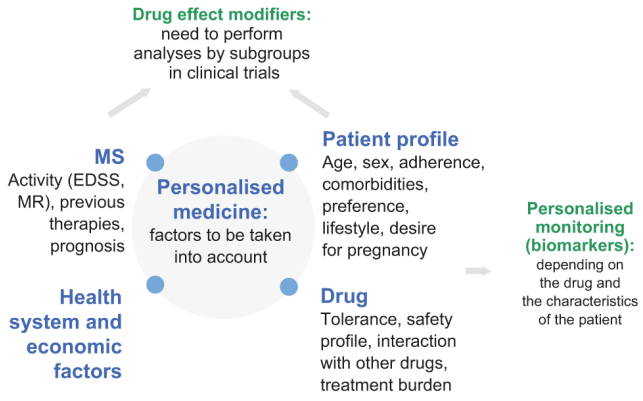
Disease-modifying strategies

Escalation vs. induction

The choice between moderately effective and safe drugs with subsequent escalation or more effective drugs from the outset is a key issue in clinical practice [30]. No information from clinical trials on the efficacy of different treatment strategies is available, although randomised clinical trials (TREAT-MS and DELIVER-MS) are under way to determine whether a given treatment strategy best prevents long-term disability and atrophy [31].

A study with more than five years' follow-up showed that the induction strategy was more effective than the escalation strategy in controlling disability progression. This effect increased in the long term (up to 10 years), even though patients in the escalation group had been switched to more effective drugs [32].

Figure 1. Factors to be considered in a personalised approach to medicine. EDSS: Expanded Disability Status Scale; MR: magnetic resonance; MS: multiple sclerosis.



Discontinuation of treatment

It appears that discontinuation of DMT in older patients may be safe [33]. The combination of an age ≥ 45 years with no relapses or radiological activity for ≥ 4 years has been associated with a high probability of continuing without relapses after discontinuation of DMT; while ≥ 45 years with long disease duration and high EDSS predicted risk of progression [34]. It should also be noted that almost half of elder people are very satisfied with their DMT and do not consider discontinuing it, even if their disease is inactive [35].

Radiologically isolated syndrome

Radiologically isolated syndrome continues to give rise to a large amount of controversy and there is insufficient information on its evolution, risk of progression and long-term prognosis [36]. The current recommendation is not to treat these patients, and to consider this period as a window of opportunity in which changes in lifestyle can be implemented [36]. There are two ongoing phase III clinical trials that aim to unify criteria in the management of these patients.

Treatment dosage

A study conducted at two centres in Catalonia with patients treated with rituximab has shown that low doses of rituximab had the same effectiveness and a

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better safety profile than higher doses, with both groups being demographically and clinically homogeneous [37]. The RIDOSE-MS phase III clinical trial, which is evaluating two rituximab dosing regimens (500 mg every 6 months vs. every 12 months), is currently under way.

Strategies to promote remyelination

Role of astrocytes in remyelination

Oligodendrocytes are in close contact with astrocytes from the beginning of brain development, resulting in an intertwining of the endings of both cells. Through this junction, astrocytes provide oligodendrocytes with nutritional support and cholesterol molecules that will be used by oligodendrocytes for the synthesis of myelin [38]. Once MS appears, astrocyte reactivity increases and astrocyte gene expression changes during the remyelination process. In the early stages, genes of the Nrf2 pathway are over-expressed. Later, this pathway is mitigated and at the end of the process another type of genes associated with the biosynthesis of cholesterol are over-expressed. These two pathways compete; when the Nrf2 pathway is up-regulated, the cholesterol pathway is down-regulated. Based on these observations, it is suggested that the study of the neuroprotective and pro-myelinating functions of the Nrf2 pathway should be complemented as a therapeutic target [39].

Remyelination mechanisms

In EAE, older mice have a higher number of myelin debris-laden lysosomes within the phagocytes, which cannot be expelled to the outside. Liver X receptors (LXRs) are involved in this process of removing myelin debris-laden lysosomes. In older mice, this clearance mechanism is impaired because of an accumulation of lysosomes within the cell that crystallise and perpetuate a pro-inflammatory environment around the cell. When older mice are supplemented with an LXR agonist, the cells release lysosomes, and the clearance pathway and capacity for remyelination is restored [40]. Resolution of the inflammatory environment is necessary for oligodendrocytes to remyelinate [41].

Role of amino acids

Chronic active lesions have latent inflammatory demyelination at their edge, remyelination failure and axonal degeneration. These lesions occur even in patients treated with DMT [42]. In EAE, an increase in essential amino acids has been detected around the lesion, especially early on in the disease. The protein Slc7a5 (large neutral amino acid transporter; LAT1), which regulates the flow of amino acids from the inside to the outside of the cell, is over-expressed within the microglia of the lesion, suggesting that excess amino acids are introduced into the cell. Blocking LAT1 regulates the amount of amino acids within the microglia, which changes its phenotype from amoeboid-like to branched-like. Following this intervention, there is an increase in oligodendrocyte differentiation and remyelination [43].

Pharmacological management of progressive multiple sclerosis

Treatments available

The number of drugs approved to treat the progressive forms is still limited. For secondary progressive MS (SPMS) with active disease, siponimod has been approved. In addition, interferons, ocrelizumab, cladribine and ozanimod can be used in these patients [44]. Other drugs such as simvastatin and ibudilast are currently being studied in advanced clinical stages.

In primary progressive MS (PPMS), the only approved DMT is ocrelizumab. Study results were not positive for interferons, glatiramer acetate and rituximab. However, long-term efficacy was seen in a single-centre study in patients treated with interferons, and in younger patients with ≥ 1 Gd^+ lesion treated with rituximab.

Time in treatment and progression

One retrospective study [45] found that, in PPMS, longer exposure to DMT could delay the time to reach an EDSS of 7 (note that the patients treated in this case were younger, with a lower EDSS and shorter disease duration). Treating younger patients and initiating treatment earlier could improve long-term disability outcomes.

Quantitative methods of classifying RRMS and SPMS

The diagnosis of SPMS is challenging, as there are no clear and agreed diagnostic criteria. Methods for classifying RRMS and SPMS, such as EXPAND

[46], Melbourne [47] or decision tree [48] differ in several of their criteria, including the number of EDSS assessments required, demonstration of progression by EDSS or taking age into consideration. Table I shows the results of a study aimed at validating these classification methods [49], and of another study that determined the characteristics of patients clinically assigned to an RRMS course who were reclassified as SPMS when the decision tree was applied [50].

Table I. Studies on quantitative methods of classifying RRMS and SPMS.

	Method	Results
Forsberg et al [49]	Data from registries in the Czech Republic (11,336 patients), Denmark (10,255), Germany (23,185), Sweden (11,247) and the United Kingdom (5,086) were used. Three classification methods were applied – method 1: EXPAND criteria; method 2: University of Melbourne definition; method 3: Karolinska Institute decision tree. The classifications were compared with clinical assignment, and the sensitivity (SPMS as true positive), specificity (RRMS as true negative) and accuracy were calculated	The overall classification performance (sensitivity, specificity, accuracy) among classifiable patients was – method 1: (0.47; 0.85; 0.79); method 2: (0.77; 0.87; 0.85); method 3: (0.84; 0.83; 0.84). The proportions of patients not classifiable with each method were – method 1: 20.0%; method 2: 32.2%; method 3: 0%
Hillert et al [50]	The same registry data were used as in Forsberg et al. The Karolinska Institute decision tree method was applied	Between 5% and 25% of patients were misclassified: 8,372 RRMS patients were reassigned as SPMS, the proportion of SPMS increasing from 17% to 31%. Reassigned patients were younger, of greater age at onset and had undergone a more rapid progression to SPMS. The proportion of patients with DMT was 36% in those clinically assigned to SPMS, while it was 69% in those reassigned as SPMS

EDSS: Expanded Disability Status Scale; RRMS: relapsing-remitting multiple sclerosis; SPMS: secondary progressive multiple sclerosis.

Cognitive dysfunction

Evaluation and monitoring

In recent years, tests have been developed in electronic format for neuropsychological evaluation. Advantages include their ease of use and reduced learning effect, although technical supervision is important during their administration [51].

Remote administration (by phone) of neuropsychological testing appears to be on a par with face-to-face administration, providing a valid and reliable measure of cognitive function. This has been demonstrated with the California Verbal Learning Test (CVLT) [52,53] and the Symbol Digit Modalities Test (SDMT) [53], both of which are useful tests for identifying cognitive dysfunction [54].

Radiological predictors of cognitive impairment

Cognitive impairment in MS is determined by lesions, structural damage and the degree of efficiency of the neural network [55-59]. Even if structural damage is significant, the efficiency of the network prevents this from translating into major cognitive symptoms. In patients with CIS, an association has been found between involvement of the hippocampus and verbal episodic memory [56], and between the posterior lobes of the cerebellum and processing speed [58]. From a functional point of view, cognitive impairment could be due to a failure of compensatory mechanisms consisting in the recruitment of additional brain areas to perform a task. This failure in these mechanisms may be due to compensation not occurring or its occurring in a maladaptive manner [60].

Treatment

Several phase III clinical trials with DMT have included cognitive impairment among the study variables although, in most cases, it was not defined as a primary variable. Thus, while positive effects have been obtained, the nature of the evidence is not sufficiently robust to make concrete recommendations on the use of specific DMT in cognitive impairment. This would require randomised clinical trials with cognition as the primary outcome variable [61].

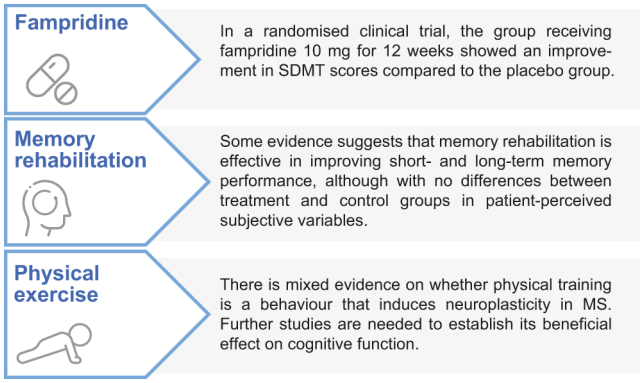
For the time being (Fig. 2), there is evidence of a beneficial effect on certain cognitive processes of interventions such as dalfampridine treatment [62], cognitive rehabilitation [63] and physical exercise [64]. Further studies are needed to confirm this evidence.

Innovations in symptomatic therapy and rehabilitation

Although the impact of fatigue on quality of life and social burden is high, it remains one of the most difficult symptoms to treat, despite the fact that some pharmacological and non-pharmacological approaches have been proposed.

Non-pharmacological approaches include psychological therapies such as cognitive training, which appears to offer an improvement in 50% of patients, and energy-saving strategies, which have shown a

Figure 2. Results from studies on pharmacological and non-pharmacological interventions for cognitive impairment in multiple sclerosis. SDMT: Symbol Digit Modalities Test.



moderate favourable impact [65]. Neurofeedback may also improve cognition. A pilot study with 14 patients showed that patients who learned to regulate the sensory motor EEG rhythm after receiving visual feedback showed an improvement in MRI parameters, which in turn correlated with an improvement in cognition [66].

As for pharmacological therapies, the TRIUMPHANT-MS study on the use of amantadine, modafinil and methylphenidate for five weeks showed no change in the Modified Fatigue Impact Scale (MFIS). In addition to not improving fatigue, these treatments gave rise a higher number of AEs compared to the placebo group [67]. The COMBO-MS study was designed to analyse the effectiveness of cognitive behavioural therapy, treatment with modafinil or a combination of the two. In addition, it aims to identify whether treatment response occurs as a function of MS subtypes or other covariates, such as sleep and depression.

Global perspectives on neuromyelitis optica spectrum disorders

Neuromyelitis optica spectrum disorders (NMOSD) is still considered a rare disease [68] that is characterised by more severe relapses with worse recovery than MS and a negative impact on quality of life [69].

The discovery of anti-aquaporin-4 antibodies (AQP4-IgG) has been one of the major milestones in the diagnosis of NMOSD. However, not all the techniques used for its detection have the same sensitivity and specificity. The cell culture technique appears to be the most accurate. AQP4-IgG-positive paediatric cases respond well to treatment, especially first-line rituximab [70].

Naive B-cell behaviour has been studied in anti-MOG and anti-AQP4 positive patients in different scenarios [71]. In the former, prior to treatment, anti-AQP4 patients showed an increase in transitional B cells ($CD36^+$ and $CD27^-$) that was not observed in anti-MOG patients. After treatment with steroids, in both anti-MOG and anti-AQP4 patients, there was a decrease in transitional B cells. This suppression was correlated with the time of treatment administration.

Late breaking news

As usual in recent editions, the event ended with some late breaking news. Table II describes these studies, framed within various themes such as remyelination processes and their possible therapeutic management [72,73] biomarkers in cerebrospinal fluid (CSF) [74], microstructural and metabolic correlates of the first demyelinating event [75], association between gut bacteria and relapses in paediatric MS [76] and the role of NfLs as a biomarker in pregnancy [77].

Conclusions

The importance that demographic factors, particularly age, may have in MS was discussed at the last post-ECTRIMS. Proactive management in paediatric patients was proposed and advances in the study of the accuracy of serum prognostic biomarkers, such as NfL, were highlighted. Quantitative classification methods were discussed that could help to identify RRMS patients who have started a progressive course. Regarding remyelination processes, inflammation has been shown to be relevant, so that control of latent inflammation could favour remyelination.

In the near future, the development of electronic devices and scales is expected to change the approach to cognitive screening in MS. Assessment of focal damage to structures involved in cognitive impairment by means of MRI could become a surrogate marker of the disease.

The data presented provide information that will allow for an increasingly personalised approach to patients, taking into account both their personal

Table II. Late breaking news.

	Aim	Methodology	Results	Conclusions
Lohrberg et al [72]	To investigate the effects of primary astrocytic loss on the regeneration and remyelination of lesions	They studied autopsy material from patients with CPM; in EAE, they characterised the activation and differentiation of OPCs	There was rapid activation of the parenchymal NG2+ OPC reservoir in the astrocyte-free lesion, leading to extensive OPC proliferation. One week after the onset of the lesion, the majority of parenchymal OPC-derived cells expressed BCAS1, indicating the transition to a pre-myelinating state	Astrocyte-oligodendrocyte interactions are important for remyelination. The impact of astrocyte dysfunction on remyelination efficiency needs to be further determined
Brown et al [73]	To evaluate the safety and efficacy of bexarotene (an RXR agonist used in cutaneous T-cell lymphoma) as a remyelinating therapy	Double-blind, phase 2a trial of RRMS patients aged 18-50 years treated with dimethyl fumarate, randomly assigned to bexarotene or placebo for 6 months	The outcome of the main variable was negative: there was no difference between the groups in the change in the mean magnetisation transfer rate of the submedian lesions; there were, however, significant changes in the supermedian lesions and in the latency of the visual evoked potentials. In the bexarotene group, 20% discontinued and 48% required dose reduction	Although the result of the primary variable was negative, the results of secondary/exploratory variables show that bexarotene exerts a small biological effect. Tolerance of bexarotene was poor
Watanabe et al [74]	To examine the capacity to differentiate NMOSD, MOG and RRMS by 4 neurotrophic granulocyte biomarkers in CSF: Ela, MPO, MMP-8 and NGAL	CSF from patients with NMOSD, MOG and RRMS was evaluated for Ela, MPO, MMP-8, NGAL and compared with markers of neuronal (NfL) and astrocyte (GFAP, S100B) damage with conventional ELISA or SIMOA	All the patients had high levels of NfL, although GFAP levels only elevated in NMOSD. In MOG, Ela, MPO and MMP-8 they were increased compared to controls and acute RRMS. In acute NMOSD, the S100B and GFAP levels were increased in 89% and 83% of patients, respectively, compared to mean MOG values. In acute NMOSD, EDSS correlated with all 4 biomarkers and GFAP, but not with NfL and S100B	Since all 4 neutrophil granulocyte biomarkers can be measured within a few hours, compared to a response time of up to 2 weeks for cell-based assays for AQP4 and MOG, they could support individual decision-making for acute therapeutic intervention
Collorone et al [75]	To use quantitative multi-parametric MRI techniques to detect clinically relevant alterations not captured by conventional MRI	In a 3T scan, structural scans of the brain and spinal cord, and NODDI of the brain were obtained in CIS or MS patients (n=46), and controls (n=13). NDI and ODI were measured with NODDI, and TSC was measured with 23Na MRI	Patients showed higher ODI in the normal-appearing white matter, including the corpus callosum, where they also showed lower NDI and higher TSC vs. controls. In grey matter, controls vs. patients had lower ODI in the frontal, parietal and temporal cortex; lower NDI in the parietal, temporal and occipital cortex; and higher TSC in the limbic and frontal cortex. There was no difference in brain volumes between patients and controls. In patients, higher ODI in the corpus callosum was associated with worse performance in the gait test	Increased axonal dispersion in the normal-appearing white matter and reduced axonal density in the corpus callosum suggests that this structure may be affected early on. NODDI alterations were observed in grey matter in specific areas. The 23Na MRI technique could detect clinically relevant pathology in very early MS
Horton et al [76]	To estimate the association between gut microbiota and subsequent disease activity in paediatric-onset MS	Stool samples were collected and analysed by 16S rRNA sequencing of the V4 region. ASVs were identified using DADA2	Among 270 ASVs included in the analyses, 20 were significant ($p<0.05$), e.g. the presence of Blautia stercoris was associated with an increased risk of relapses. Six ASV modules were identified. Higher module-specific gene values were associated with a higher risk of relapse. Four ASVs associated with an increased risk of relapses were included in this module: Blautia massiliensis, Dorea longicatena, Coprococcus comes and an unknown species of the genus Subdoligranulum	There are no major differences in the composition of the gut microbiota associated with relapses, but there are some bacterial species of the Blautia genus that seem to be associated with relapses
Yaldizli et al [77]	To assess whether the interruption of DMT due to pregnancy leads to an increase in NfL levels	Pregnancies were documented prospectively from the Swiss MS cohort study. Serum samples were collected every 6 to 12 months and analysed using SIMOA	DMT was discontinued in 13 out of 72 pregnancies. NfL levels were on average 22% higher during vs. outside the postpartum period. There were 29 relapses during the postpartum periods, which were associated with 98% more NfL. The effect of the postpartum period on NfL disappeared after including exposure to DMT in the model. DMT patients had 12% lower NfL levels vs. non-DMT patients	NfL could be a sensitive and minimally invasive measure of disease activity in pregnancy. Strategies that allow DMT to be continued during pregnancy may be justified

ASV: amplicon sequence variants; BCAS1: breast carcinoma amplified sequence 1; CIS: clinically isolated syndrome; CPM: central pontine myelinolysis; CSF: cerebrospinal fluid; DADA2: Divisive Amplicon Denoising Algorithm-2; DMT: disease-modifying therapies; EAE: experimental autoimmune encephalomyelitis; Ela: elastase; MMP-8: matrix metalloproteinase 8; MOG: anti-MOG antibody-associated diseases; MPO: myeloperoxidase; NDI: neurite density index; NGAL: neutrophil gelatinase-associated lipocalin; NMOSD: neuromyelitis optica spectrum disorders; NODDI: neurite orientation dispersion and density imaging; ODI: orientation dispersion index; OPC: oligodendrocyte precursor cells; RM: magnetic resonance; RXR: retinoid X receptor; SIMOA: single molecule array assay; TSC: total sodium concentration.

characteristics and those of their disease, while also detecting, as accurately as possible through biomarkers, their diagnosis, evolution and potential response to the treatments available.

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XIII Reunión Post-ECTRIMS: revisión de las novedades presentadas en el Congreso ECTRIMS 2020 (II)

Introducción. Desde hace más de una década, tras el Congreso ECTRIMS, se celebra en España la reunión post-ECTRIMS, donde neurólogos expertos en esclerosis múltiple (EM) se reúnen para revisar las novedades presentadas en el ECTRIMS.

Objetivo. En el presente artículo, publicado en dos partes, se resumen las ponencias de la reunión post-ECTRIMS, celebrada los días 16 y 17 de octubre de 2020 virtualmente.

Desarrollo. En esta segunda parte se destaca la importancia del género y la edad en la compresión de la patología de la enfermedad y la optimización de su manejo. Se exponen los avances realizados en la EM pediátrica desde un punto de vista neuropsicológico y de neuroimagen. Por su parte, cobran especial protagonismo los hallazgos que contribuyen a realizar un enfoque del tratamiento más personalizado y a elegir la mejor estrategia de tratamiento (farmacológica y no farmacológica) para cada paciente. De igual forma, se abordan los resultados relacionados con las estrategias posibles que promuevan la remielinización. Aunque no hay grandes avances en el tratamiento de formas progresivas, se destacan algunos métodos cuantitativos para la clasificación de estos pacientes. Además, se incluyen los resultados sobre herramientas potenciales de evaluación y tratamiento de los déficits cognitivos, y algunos aspectos relevantes observados en el espectro de los trastornos de la neuromielitis óptica. Por último, se detallan los resultados de las ponencias consideradas como noticias de última hora en el ECTRIMS-ACTRIMS.

Conclusiones. Se presentaron avances principalmente sobre el conocimiento de la EM pediátrica, las estrategias de remielinización y la evaluación cognitiva en la EM.

Palabras clave. ACTRIMS. Congreso. ECTRIMS. EM. Esclerosis múltiple. Post-ECTRIMS.