years. Six month, 12 month and 24 month adherence rates (MPR ≥80%) were as follows: interferon (n=149) 94.0%, 87.2% and 67.1%; glatiramer (n=55) 78.2%, 70.9% and 56.4%; natalizumab (n=18) 94.4%, 83.3% and 66.7%; and fingolimod (n=15) 73.3%, 80.0% and 66.7%. Overall adherence with injectable drugs seemed higher at any time point than oral drugs: injectable drugs 93.6% (6 months), 86.7% (12 months) and 70.0% (24 months) compared with 73.5%, 70.6% and 55.9%, respectively, for oral drugs.

Conclusion and relevance This retrospective analysis showed high 6 month to 24 month adherence rates for injectable DMD in multiple sclerosis. Both interferon and natalizumab had higher adherence rates than reported elsewhere in the literature. Oral DMD had lower adherence rates than injectable DMD but more consistent rates with other studies in the literature.[1]

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No conflict of interest.

6ER-012 PERSISTENCE FOR DISEASE MODIFYING DRUGS FOR MULTIPLE SCLEROSIS

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Background and importance Persistence of therapy is fundamental to achieve disease management goals. Due to the chronic nature of multiple sclerosis treatment, interventions by hospital pharmacists is fundamental to patient persistence with disease modifying drugs (DMD), reducing relapses and slowing disease progression.

Aim and objectives The study aimed to assess persistence for multiple sclerosis therapy.

Material and methods This was a retrospective cohort study based on hospital drug claims for multiple sclerosis. Patients with at least one claim for interferon, fingolimod, dimethyl fumarate, glatiramer acetate, natalizumab or teriflunomide were eligible if naïve or had switched between 2012 and 2017. Naïve were defined as patients without any claim in the previous 365 days. Switchers were defined as patients who changed to other drugs anytime during the study period (2012–2017). The main outcome was persistence, defined as time from initiation to discontinuation of a given DMD, which was considered as a gap in therapy when a subsequent claim for the same drug occurred >90 days after the end of the previous claim. The proportion of persistent patients was reported for 6 and 12 months. Time to event analysis was performed with the Kaplan–Meier estimator and semi-parametric Cox proportional hazard regression model.

Results A total of 87 patients were included with a mean age of 43.0 (SD 10.9), of whom 44.8% (n=39) were naïve and 55.2% (n=48) were switchers. Overall, 55.2% of patients were receiving treatment with injectable drugs (glatiramer acetate 24.1%; interferon 23.0%; natalizumab 8.1%) and 44.8% with oral drugs (fingolimod 19.5%; teriflunomide 18.4%; dimethyl fumarate 6.9%). For the overall sample, median time to discontinuation was 4.5 years. Median time to discontinuation for injectable DMD was significantly lower (median 1.2 years) than for oral DMD (median not reached) (log rank <0.001). The risk of discontinuing treatment was 10.0 times higher for patients receiving treatment with injectable DMD compared with oral DMD (HR=10.0, 95% CI 3.0 to 33.5). The probability of persistence for injectable DMD decreased substantially from 6 months (70.2%, 95% CI 57.2% to 86.1%) to 12 months (52.5%, 95% CI 37.9% to 72.9%).

Conclusion and relevance Treatment with oral drugs was associated with higher persistence in patients with multiple sclerosis.

REFERENCES AND/OR ACKNOWLEDGEMENTS

No conflict of interest.

6ER-013 ENRICHED DEVELOPMENTAL BIOLOGY MOLECULAR PATHWAYS: IMPACT ON ANTIPSYCHOTIC INDUCED WEIGHT GAIN

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Background and importance Psychotropic induced weight gain (PIWG) may lead to an increased risk of cardiovascular diseases, metabolic disorders and, ultimately, treatment discontinuation. Identification of the genetic makeup at risk for PIWG could characterise subjects at risk for this possible severe side effect and help move a step forward in the direction of personalised treatment in psychiatry.

Aim and objectives The hypothesis tested in the study was that PIWG might be genetically driven. Analysis of the complete molecular pathways may grant sufficient power to tackle the biologic variance of PIWG.

Material and methods A genetic sample from the CATIE trial (n=765; 556 men, mean age 40.93±11.03 years) treated with diverse antipsychotic drugs was investigated. A molecular pathway analysis was conducted in an R environment for the identification of the molecular pathways enriched in variations associated with PIWG.

Results The developmental biology molecular pathway was found to be significantly (p adj=0.018) enriched in genetic variations significantly (p<0.01) associated with PIWG. A total of 18 genes were identified and discussed. The developmental biology molecular pathway was involved in the regulation of β cell development, and the transcriptional regulation of white adipocyte differentiation. Interestingly, this finding was a result of a hypothesis free approach.

Conclusion and relevance The results correlate with previous evidence and are consistent with our earlier results in the STAR*D sample. Furthermore, the involvement of β cell development and transcriptional regulation of white adipocyte differentiation pathways stress the relevance of peripheral tissue rearrangement, rather than increased food intake, in the biologic modifications that follow psychotropic treatment and may lead to PIWG. Further research is warranted.

REFERENCES AND/OR ACKNOWLEDGEMENTS

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