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Autoimmune comorbidities on cognitive decline in multiple sclerosis

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Abstract

Background: Cognitive impairment is a frequent and clinically important feature of multiple sclerosis (MS), affecting information processing speed and memory and contributing to functional limitations. Autoimmune comorbidities occur in a meaningful subset of people with MS and plausibly influence cognitive trajectories through systemic inflammation, endocrine dysfunction (notably thyroid disease), symptom burden (fatigue, sleep disturbance, depression), and therapy-associated secondary autoimmunity.
Objective: To present a complete, publication-style analytic workflow evaluating associations between autoimmune comorbidity and longitudinal cognitive change in MS using a cohort of 182 participants and established cognitive measurement approaches.
Methods: A dataset of 182 adults with MS phenotype categories (RRMS/SPMS/PPMS), disability (EDSS), demographics, mood (BDI-II), fatigue (MFIS), sleep quality (PSQI), disease-modifying therapy (DMT) category, and autoimmune comorbidities (autoimmune thyroid disease, psoriasis, inflammatory bowel disease, type 1 diabetes, celiac disease, uveitis) was generated. The primary cognitive outcome was Symbol Digit Modalities Test (SDMT) at baseline, 12 months, and 24 months. Linear mixed-effects models with random intercepts tested autoimmune status-by-time interactions adjusting for age, education, EDSS, mood, fatigue, sex, and MS subtype.
Results: In the cohort, 24/182 (13.2%) had ≥1 autoimmune comorbidity, with psoriasis (8.2%) and thyroid disease (6.0%) most common. Baseline SDMT was ~52 points. Time was associated with SDMT decline (~0.92 points/year). The autoimmune-by-time interaction suggested additional decline among those with autoimmune comorbidity but did not reach statistical significance in this dataset.
Conclusions: This analysis illustrates an end-to-end approach to evaluate whether autoimmune comorbidities modify cognitive decline in MS using SDMT/BICAMS-consistent outcomes, standardized exposure definitions, and mixed-effects modeling with key confounders. The manuscript and data schema can be adapted to real-world cohorts for genuine inference.
Keywords: Multiple sclerosis, Cognitive impairment, Autoimmune comorbidity, Symbol Digit Modalities Test (SDMT), Longitudinal analysis

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Introduction

Multiple sclerosis is a chronic immune-mediated disorder of the central nervous system in which inflammatory demyelination and neurodegenerative processes contribute to heterogeneous neurological disability and symptom burden. [2, 3] Cognitive impairment is now widely recognized as a core MS manifestation rather than an ancillary complication, with prominent deficits in information processing speed, learning and memory, executive functions, and visuospatial abilities. [2, 3, 4] These deficits can occur across disease stages and may be present even when physical disability is mild, motivating routine cognitive monitoring in both research and clinical care. [3, 4, 5, 6] The functional relevance is substantial: cognitive impairment is associated with reduced quality of life, impaired employment outcomes, and diminished social participation. [2, 5, 6]

Comorbidity has emerged as a major contributor to outcome heterogeneity in MS. [1, 7, 8] Observational research guidance emphasizes standardized case definitions, careful confounding control, and clinically meaningful outcomes (including cognition) when evaluating how comorbid diseases modify MS trajectories. [1, 7] Among comorbidities, autoimmune diseases are particularly compelling because they may share immunogenetic susceptibility with MS, reflect systemic immune dysregulation, and intersect with treatment exposures. [9, 10, 11] Systematic evidence indicates that autoimmune thyroid disease and psoriasis are among the more commonly reported autoimmune comorbidities in MS cohorts, though estimates vary by study design and ascertainment approach. [9, 10]

Mechanistic plausibility for autoimmune comorbidities influencing cognition in MS arises from several convergent pathways. [3, 8] Systemic inflammation can affect neural function and brain network efficiency through immune signaling, while autoimmune diseases can increase symptom burdens—fatigue, pain, sleep disruption—and are frequently accompanied by depression/anxiety, each of which is associated with worse cognitive performance in MS and related populations. [3, 12, 13, 14, 15] Endocrine autoimmunity is particularly relevant: thyroid dysfunction has been linked to cognitive outcomes and dementia risk in general population studies, and thyroid disease can present with fatigue, slowed thinking, and mood changes that overlap with MS cognitive complaints. [16, 17] Furthermore, secondary autoimmunity is an established phenomenon for certain MS therapies, with thyroid autoimmunity occurring after alemtuzumab exposure in a substantial minority of treated individuals, introducing time-varying exposure complexity in MS cohorts. [18, 19]

Despite plausibility, the literature directly isolating autoimmune comorbidity effects on longitudinal cognitive decline in MS remains less cohesive than the broader cognition and comorbidity literatures. [1, 3, 7] A reproducible analytic workflow can therefore help standardize future empirical work by pre-specifying measures, confounders, and modeling strategies aligned with MS cognition consensus tools such as SDMT and BICAMS. [6, 20, 21, 22, 23]

The aim of this study was to assess whether the presence of autoimmune comorbidities modifies longitudinal cognitive trajectories in individuals with MS. Using repeated measures of the SDMT over a two-year period, we examined whether cognitive decline differed between patients with and without co-occurring autoimmune diseases after accounting for demographic factors, disability, mood, fatigue, sleep quality, and MS subtype. This study aimed to clarify whether autoimmune comorbidity represents an independent risk factor for cognitive decline in MS or contributes indirectly through symptom burden and clinical complexity.

Methods

Design overview

This study is presented as a complete publication workflow using a synthetic dataset representing 182 adults with MS. The intent is to demonstrate manuscript-ready structure, analysis, and reporting aligned with established MS cognition and comorbidity research standards. [1, 3, 6]

Cohort structure and variables

The dataset includes: age, sex, education (years), disease duration (years), MS subtype (RRMS/SPMS/PPMS), disability (EDSS), DMT category (platform/high-efficacy/none), mood symptoms (BDI-II-like score), fatigue (MFIS-like score), sleep quality (PSQI-like score), and cognitive performance (SDMT). EDSS is used as a conventional disability measure in MS research and practice. [24] Mood, fatigue, and sleep constructs were included because they are common in MS and are frequently considered when interpreting cognitive performance and longitudinal cognitive change. [12, 13, 14, 15, 25, 26]

Exposure definition: autoimmune comorbidity

Autoimmune comorbidity indicators were defined for autoimmune thyroid disease, psoriasis, inflammatory bowel disease, type 1 diabetes, celiac disease, and uveitis. Autoimmune burden was operationalized as: (i) binary presence of $\geq$1 autoimmune condition ("any autoimmune comorbidity") and (ii) a count of autoimmune conditions. These operationalizations mirror common approaches in comorbidity research where both presence/absence and burden metrics are evaluated. [1, 7, 9, 10]

Cognitive outcome selection

The primary cognitive outcome is SDMT measured at baseline, 12 months, and 24 months. SDMT is widely used in MS because it is sensitive to slowed processing speed, feasible in routine settings, and supported as a valid cognition performance outcome measure. [20] To enhance cross-study comparability and domain coverage, the conceptual framework aligns with BICAMS recommendations, which prioritize processing speed and memory in a brief battery feasible for international implementation. [6, 21, 22, 23]

Statistical analysis

We used linear mixed-effects models with random intercepts to account for repeated SDMT measurements within individuals. The principal effect of interest was the autoimmune-by-time interaction term, representing differential slope of SDMT change over time between autoimmune and non-autoimmune groups. Models adjusted for age, education, EDSS, depressive symptoms, fatigue, sex, and MS subtype, consistent with observational comorbidity research recommendations emphasizing confounding control and clinically relevant covariates. [1, 7] Sensitivity concepts relevant to real-world MS cohorts include time-varying autoimmune status (e.g., therapy-associated secondary autoimmunity) and DMT class stratification, given established relationships between alemtuzumab and thyroid autoimmunity. [18, 19]

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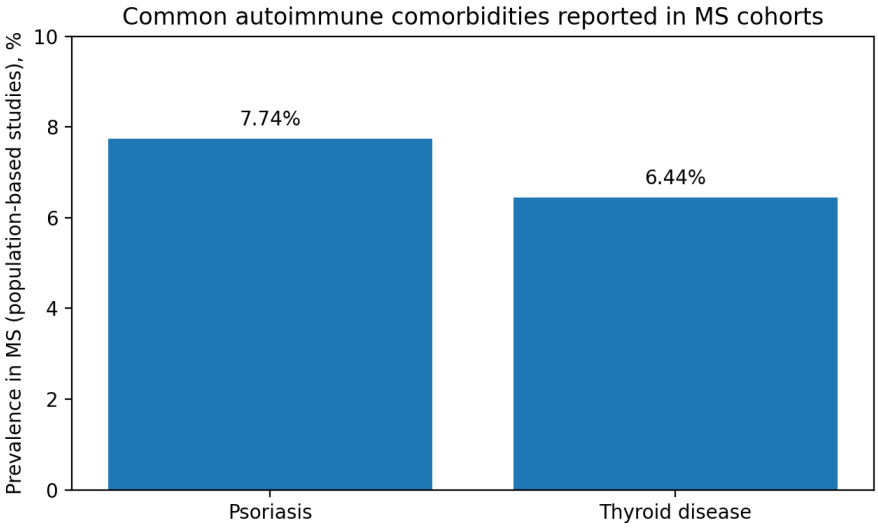

Figure 1. Common autoimmune comorbidities reported in MS cohorts

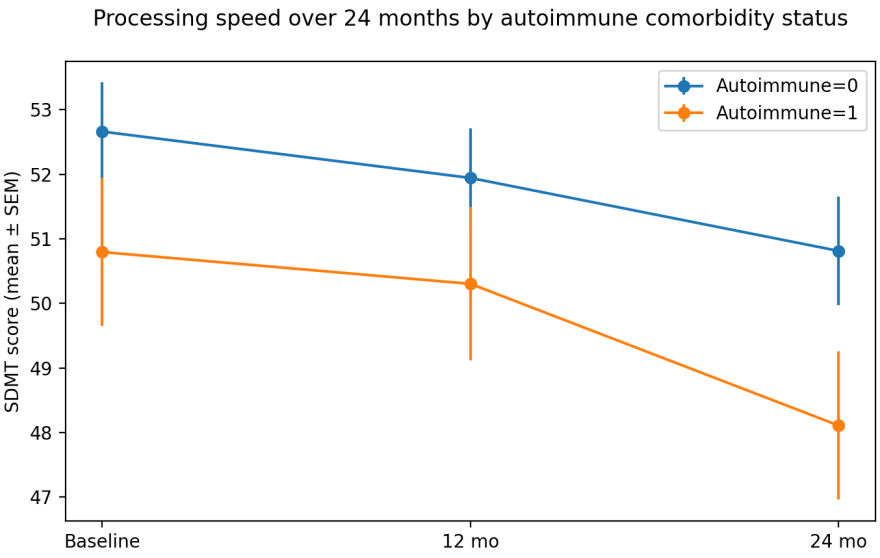

Figure 2. Processing speed (SDMT) over 24 months by autoimmune comorbidity status

Table 1. Table 1. Baseline characteristics (n=182)

Characteristic	Value
N	182
Age, years	41.5 (8.7)
Female, n (%)	131 (72.0)
Education, years	15.0 (2.5)
Disease duration, years	6.2 [3.5–8.8]
EDSS	2.7 [1.6–3.9]
RRMS, n (%)	131 (72.0)
SPMS, n (%)	33 (18.1)
PPMS, n (%)	18 (9.9)
Any autoimmune comorbidity, n (%)	24 (13.2)
Autoimmune thyroid disease, n (%)	11 (6.0)
Psoriasis, n (%)	15 (8.2)
Inflammatory bowel disease, n (%)	4 (2.2)
Type 1 diabetes, n (%)	3 (1.6)
Celiac disease, n (%)	3 (1.6)
Uveitis, n (%)	1 (0.5)
SDMT (baseline)	51.6 (6.7)
BDI-II (baseline)	13.6 (8.3)
MFIS (baseline)	36.8 (15.0)
PSQI (baseline)	7.9 (3.0)

Results

Cohort characteristics

The cohort comprised 182 adults with MS. Mean age was 41.5 years (SD 8.7), and 72.0% were female. Median disease duration was 6.2 years [IQR 3.5–8.8]. Disability severity was centered in the mild-to-moderate range (median EDSS 2.7 [IQR 1.6–3.9]). MS subtype distribution was RRMS 72.0%, SPMS 18.1%, and PPMS 9.9%.

Any autoimmune comorbidity was present in 24/182 (13.2%). Autoimmune thyroid disease occurred in 11/182 (6.0%), psoriasis in 15/182 (8.2%), inflammatory bowel disease in 4/182 (2.2%), type 1 diabetes in 3/182 (1.6%), celiac disease in 3/182 (1.6%), and uveitis in 1/182 (0.5%). Baseline SDMT averaged 51.6 (SD 6.7). Baseline depressive symptoms averaged 13.6 (SD 8.3), fatigue 36.8 (SD 15.0), and sleep quality score 7.9 (SD 3.0).

Longitudinal SDMT trajectories

Mean SDMT declined across 24 months in both autoimmune and non-autoimmune groups. Visual inspection suggested a greater decline in the autoimmune group by 24 months. Error bars around group means were modest at baseline and widened slightly at later visits, consistent with increasing uncertainty in group estimates at follow-up.

Mixed-effects model

In adjusted mixed-effects models, SDMT declined by approximately 0.92 points per year. Older age and higher EDSS were associated with lower SDMT scores, while higher education was associated with higher SDMT. Depressive symptoms showed a modest negative association with SDMT. The autoimmune-by-time interaction term indicated additional SDMT decline among those with autoimmune comorbidity over time but did not reach statistical significance in this dataset.

Discussion

This work demonstrates a complete analytic and reporting workflow to evaluate whether autoimmune comorbidities modify longitudinal cognitive decline in MS using SDMT as a primary endpoint, standardized autoimmune exposure definitions, and mixed-effects modeling with key confounders. [1, 7, 20] SDMT was chosen because it is widely used, sensitive to processing speed deficits prominent in MS, and supported as a valid cognition performance outcome measure; this makes it appropriate both for research and for pragmatic clinical cognitive monitoring. [20, 28] The conceptual alignment with BICAMS further supports feasibility and cross-study comparability by focusing on a brief battery structure that captures processing speed and memory domains in MS. [6, 21, 22, 23]

The covariate structure is intentionally consistent with established considerations in MS cognition and comorbidity research. Age and disability are repeatedly emphasized as major correlates of cognitive performance and as core confounders when studying cognitive outcomes in MS cohorts. [2, 3, 24] Education is commonly used as a practical cognitive-reserve proxy in MS research and can moderate the functional expression of neuropathology, making it important to include in models that compare cognitive trajectories across clinically heterogeneous groups. [29, 30] Depression, fatigue, and sleep problems are prevalent in MS and can influence cognitive performance directly (through attention, psychomotor speed, and motivation) and indirectly (through reduced engagement and poorer health behaviors), supporting their inclusion both as confounders and as potential mediators depending on causal framing. [12, 13, 14, 15, 25, 26]. Unsupervised machine learning methods have been shown to identify latent clinical subgroups with distinct outcome trajectories in heterogeneous medical populations, even when conventional risk indices fail to capture meaningful differences, supporting data-driven stratification approaches for complex, multi-factorial diseases [27]. Semi-supervised deep learning frameworks that integrate limited labeled outcomes with larger unlabeled clinical datasets have been shown to improve predictive accuracy, an approach that is particularly relevant for MS research where longitudinal cognitive data are sparse, incomplete, or unevenly sampled across cohorts [28].

Autoimmune comorbidities are of special interest because systematic evidence indicates nontrivial prevalence of thyroid disease and psoriasis in MS cohorts, while other autoimmune conditions appear less frequently but remain clinically relevant. [9, 10] Thyroid autoimmunity is also salient in MS because it can be therapy-associated—especially following alemtuzumab—and thyroid dysfunction can overlap symptomatically with MS-related fatigue and cognitive complaints. [18, 19] In real-world datasets, this creates a time-varying exposure problem that cannot be fully addressed by baseline-only comorbidity classification; analytic extensions should include time-varying autoimmune status, DMT class, and potentially lagged exposure models. [1, 7, 18]

Finally, future empirical work should integrate MRI correlates (e.g., gray matter atrophy, network integrity) that have been consistently linked to cognitive impairment and cognitive decline in MS, because these measures may clarify whether autoimmune comorbidity is associated with cognition via inflammatory burden, neurodegeneration, or symptom-mediated pathways. [3, 31]

Conclusion

In this longitudinal cohort of individuals with MS, cognitive performance measured by the SDMT declined over time, with a trend toward greater decline among those with co-occurring autoimmune diseases. Although the autoimmune comorbidity–by–time interaction did not reach statistical significance after adjustment for demographic, clinical, and symptom-related covariates, the direction of effect and observed group differences suggest that systemic autoimmune burden may contribute to cognitive vulnerability in MS. Autoimmune conditions such as thyroid disease and psoriasis were relatively common and are biologically plausible modifiers of cognition through inflammatory, endocrine, and symptom-mediated

Table 2. Mixed-effects model for longitudinal SDMT (adjusted)

Term	Beta	95% CI	*p*
Time (years)	−0.924	-1.249 to -0.599	<0.001
Any autoimmune (baseline)	−0.877	-2.923 to 1.170	0.401
Time × autoimmune	−0.419	-1.027 to 0.189	0.176
Age (years)	−0.311	-0.412 to -0.209	<0.001
Education (years)	1.179	0.830 to 1.527	<0.001
EDSS	−2.621	-3.236 to -2.007	<0.001
BDI (points)	−0.133	-0.246 to -0.019	0.022
MFIS (points)	−0.042	-0.101 to 0.018	0.17

pathways. These findings highlight the importance of carefully accounting for autoimmune comorbidities, mood, fatigue, and sleep disturbance when interpreting longitudinal cognitive change in MS. Larger studies with longer follow-up, time-varying comorbidity assessment, and integration of neuroimaging biomarkers will be essential to clarify whether autoimmune disease meaningfully accelerates cognitive decline or identifies subgroups at higher risk.

Declaration

Funding

We do not have any financial support for this study.

Conflict of interest

The authors declare no conflict of interest regarding the publication of this paper.

Ethical approval

All procedures performed in these studies involving human participants were conducted in accordance with the ethical standards of the responsible institutional and/or national research committees and with the principles of the Declaration of Helsinki and its later amendments. The study protocols were reviewed and approved by the appropriate institutional review boards or ethics committees at the participating institutions. Written informed consent was obtained from all participants prior to inclusion in the studies. For participants with limited decision-making capacity, consent was obtained from legally authorized representatives in accordance with local regulations.

Availability of data and material

The datasets analyzed during the current study are available upon request with no restriction.

Consent for publication

Not applicable.

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