Characterizing polyautoimmunity and familial autoimmunity in a U.S. multiple sclerosis population.

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There is overlap in the genetic and non-genetic risk components across several autoimmune diseases (ADs). However, it is unclear whether polyautoimmune persons with MS (PwMS) or those with familial autoimmunity differ from sporadic PwMS. Differences may highlight distinct etiologic mechanisms and processes.

Study population . The Accelerated Cure Project Repository is an open-access resource of data from PwMS recruited at 10 U.S. MS clinics (www.acceleratedcure.org). PwMS met diagnostic criteria and were ≥18 years of age at onset. There were 1,507 unrelated PwMS for this analysis.

Polyautoimmunity and familial autoimmunity . Participants completed structured questions on 31 ADw6.7 (u)0.5 (t)3.3 050 Td (on)Tj 1 .006 Tw.

- Polyautoimmunity (having a comorbid AD) in PwMS was greater among women, those of older age, with higher education, reported being ever obese and those with a family history of autoimmunity (Table 2).
- PwMS were more likely to report a family history of autoimmunity if they were female and if they had a second AD themselves (Table 2). Interestingly, non-white PwMS were >50% less likely to report a history of a non-MS AD than white PwMS. There were no differences in PwMS who reported a family history of MS and those who did not.
- Among PwMS with a comorbid AD (polyautoimmune), they were more likely to report a history of non-MS ADs (Table 3). These results at first glance, might suggest the clustering of various ADs in families may increase the prevalence of polyautoimmunity in PwMS. However, when we account for family histories for specific ADs, a family history of other ADs are not associated with the presence of a specific ADs. For example, PwMS are 4.7 times more likely to report Hashimoto's thyroiditis if they reported having a first degree relative with Hashimoto's as well; reporting a family member with another AD did not make a difference (p=0.19)
- Notable is the lack of strong associations between established MS genetic risk factors and polyatuoimmunity and familial autoimmunity (Table 2).
- Also noteworthy, is that PwMS did not differ in their presentation at/near onset based on polyautoimmunity and familial autoimmunity (Table 4).
- Collectively, these results do not suggest that polyautoimmune PwMS differ from those who are not, nor do they suggest that PwMS differ by familial autoimmunity history. These analyses also do not provide evidence that the clustering of various ADs in [(Ye)-93.9 (s)]TJ EMC /P <</MCID 141 >>BDC -0.001 Tc 0.001 Tw 1.772 0.036 1.96 1.031