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Changes in ganglioside antibody positivity rates during the COVID-19 pandemic

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ABSTRACT

Reports suggested an association between SARS-CoV-2 infection and GBS, but subsequent studies produced conflicting results regarding the incidence of GBS during the pandemic. This study assessed positivity rates for GQ1b, GM-1, GD1a, and GD1b for tests performed January 2016, through March 2021, at a national laboratory. Relative to pre-pandemic levels, positivity rates during the pandemic declined by 61% for GQ1b and 24% for GM-1, while unchanged for GD1a and GD1b. These findings suggest heterogeneity with positivity rates of GBS-associated ganglioside antibodies during the COVID-19 pandemic. Mitigation strategies during the pandemic may have reduced the frequency of certain forms of GBS.

1. Introduction

Many neuroimmunologic disorders are thought to result after a prior viral or bacterial infection. Various mechanisms have been proposed to trigger these presumed autoimmune disorders from molecular mimicry to bystander activation (Wanleenuwat et al., 2019). With the occurrence of the COVID-19 pandemic, case reports have suggested a temporal association between COVID-19 and some neuroimmunologic disorders, including disorders such as inflammatory antiganglioside neuropathies.

One such neuroimmunologic disorder is Guillain-Barré syndrome (GBS), which includes acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). There are clear infectious triggers for GBS, including viral infections such as Epstein Barr virus, Zika virus, Haemophilus influenzae and bacterial infections such as Campylobacter jejuni (Koike and Katsuno, 2021). Several case reports have suggested a temporal association with SARS-CoV-2 infection and the subsequent occurrence of GBS (Table 1). This led to several epidemiologic studies to examine if the incidence of GBS increased during the COVID-19 pandemic. While several studies suggested an increase in GBS during the pandemic (Fragiel et al., 2021; Abu-Rumeileh et al., 2021; Filosto et al., 2021), a study from the United Kingdom found a reduction in the number of cases despite using a number of techniques to determine whether there was an association between COVID-19 and GBS (Keddie et al., 2021). In particular, cases of COVID-19 and GBS in various regions did not appear to correlate and the authors concluded there was no epidemiologic evidence that SARS-CoV-2 was causative of GBS (Keddie et al., 2021). However, in these studies GBS was viewed as a homogenous disorder. Interestingly, over 50% of GBS cases have identifiable antibodies present to various gangliosides (Cutillo et al., 2020), making it possible to examine whether the COVID-19 pandemic affected the different forms of GBS in a heterogeneous fashion.

In this study, we examined the occurrence of four ganglioside antibodies associated with GBS based on testing at a US reference clinical laboratory. Our goal was to assess changes in the incidence of these GBS-associated antibodies during the COVID-19 pandemic compared to pre-pandemic testing.

2. Materials and methods

All testing was performed by Quest Diagnostics. Detection of anti-GQ1B, anti-GM1, anti-GD1a, and anti-GD1b antibodies was performed using covalent ELISA technology tests developed and validated by Quest Diagnostics.

2.1. Study population

All GQ1b, GM1, GD1a, and GD1b, results from tests performed

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Abbreviations: GBS, Guillain-Barré Syndrome.

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January 1, 2016 through March 31, 2021 that included a company-wide unique identifier were selected for potential inclusion in this study. The study population for each test result was limited to one result per patient; if any test detected antibodies, that patient was classified as positive. If antibodies were detected multiple times for the same patient, the earliest detection date was used to assess cohort trends in positivity over time. If the same patient was negative multiple times, the first negative date was used to assess cohort trends in positivity over time. Patients with indeterminate results were excluded as their status could not be classified.

2.2. Definitions

Patients were assessed geographically by United States Health and Human Services (HHS) Region. When patient state data were not available, the ordering clinician's account state was used. The “pandemic” period was defined as the period from April 1, 2020, through March 31, 2021 to align with quarterly analysis.

Table 1
Antibody positive cases of Guillain-Barre syndrome or Miller-Fisher syndrome.

| Author            | N | Time course                  | Neurological symptoms                                                                 | GBS specific antibody          | Comments                                                                 |
|-------------------|---|------------------------------|---------------------------------------------------------------------------------------|--------------------------------|--------------------------------------------------------------------------|
| Guillot et al., 2021 | 3 | Neurological symptoms developed 5–21 days after COVID-19 symptoms | One patient each developed cranial neuropathy and meningo-polyradiculitis, brainstem encephalitis, and delirium with associated involuntary movements and ataxia Quadruparalysis, decreased tactile and pain sensations in lower extremities, urinary retention, perinatal areflexia, DTR increased in all limbs. Electrophysiology was indicative of acute motor axonal neuropathy and MRI showed hyperintense lesions in the spinal cord at T2 | Anti-GD1b IgG | Questionable pathogenicity of anti-GD1b due to highly variable clinical presentations. Also identified a case of anti-Caspr2 encephalitis. |
| Manaccio et al., 2021 | 1 | Neurological symptoms developed 15 days after COVID-19 symptoms | Patient one had anoma, aguesia, right intermural ophthalmonemoparesis, right fascicular oculomotor palsy, ataxia. Patient two had areflexia, aguesia, bilateral abducens palsy. Ascending areflexic paralysis of lower extremities, absent DTR, aguesia, anosmia, MRI negative for demyelination. | Anti-GD1b IgM | Rare case of both myelitis and GBS with antibody positivity. COVID-19 nasal swab was negative, but COVID-19 antibodies were found in the blood. |
| Gutiérrez-Oritz et al., 2020 | 2 | Neurological symptoms developed 3–5 days after COVID symptoms | Patient one was positive for Anti-GD1b IgG, patient two negative Positive for Anti-GM1, anti-GD1a, anti-GD1b, anti-GQ1b | Anti-GM1 IgG | Miller-Fisher syndrome was probable in one patient and polyneuritis cranialis was likely in the other. |
| Dufour et al., 2021 | 1 | Neurological symptoms developed 21 days after positive COVID test | Ascending areflexic paralysis of lower extremities, absent DTR, aguesia, anosmia, MRI negative for demyelination. | Anti-GQ1b IgG positive | Resolution of symptoms was achieved with IVIG, but no neurophysiological studies were performed. |
| Kopscik et al., 2020 | 1 | Neurological symptoms started 2 months before positive COVID test | Progressive weakness, numbness, difficulty walking, cranial nerve abnormalities, dysmetria, ataxia, and absent lower extremity reflexes. | Anti-GM1 IgG was in the equivocal range, all others negative | Patient did not have typical COVID-19 symptoms such as fever or respiratory involvement. Neurological symptoms developed before positive test for COVID-19. |
| Lantos et al., 2020 | 1 | Neurological symptoms started 2 days after COVID-19 symptoms developed | Reduced sensation and paresthesia in lower limbs, left eye drooping, blurry vision, enlargement of left cranial nerve 3 on MRI | Anti-GM1 IgG | MFS was diagnosed, despite negative autoantibodies, due to consistent symptomatology with MFS. |
| Gigli et al., 2021 | 8 | Unclear time course | Paresthesias, tetraparesis in multiple patients | 1 patient positive for anti-GD1a IgG and anti-GT1b IgG, 5 negative, 2 not tested | While these patients may have GBS, the association seems questionable based on the unclear time course and lack of positive COVID-19 tests. |
| Chan et al., 2021 | 2 | Neurological symptoms developed 16–23 days after onset of COVID-19 symptoms | Patient one had paresthesias, gait disturbance, facial weakness, dysarthria, dysphagia, CSF results consistent with GBS. Patient two had paresthesias, gait disturbance, absent reflexes in the legs, facial weakness, autonomic dysfunction, respiratory failure. | Patient one was not tested, patient two was positive for anti-GM2 IgG/IgM | Electrophysiology was deferred in both patients due to infection control measures. |
| Lowery et al., 2020 | 1 | Neurological symptoms developed 14 days after onset of COVID-19 symptoms | Gait ataxia, left facial and bilateral lower extremity weakness, dysphagia, quadriparesis, global areflexia, cranial nerve 3, 4, and 6 weakness. | Positive for Anti-GQ1b IgG | MFS with GBS overlap was diagnosed. |
| Petrelli et al., 2020 | 1 | Neurological symptoms developed 17 days after onset of COVID-19 symptoms | Hypoesthesia, loss of mobility, upper limb flaccid paralysis, DTR absent on right side, electromyography had absence of a demyelinating pattern, but showed axonal-only motor neuropathy | Positive for anti-GM1 IgG and anti-GD1a IgG | GBS diagnosed based on presence of autoantibodies and symptomatology. |
| Civardi et al., 2020 | 2 | Neurological symptoms developed 10 days after onset of COVID-19 symptoms | Lower limb weakness, paresthesia, generalized areflexia, nerve conduction studies showed demyelinating pattern. Eventually developed quadruplegia and neuromuscular respiratory failure | Positive for anti-GM1 IgG, anti-GD1b IgG, anti-GD1b IgM | GBS diagnosed based on presence of autoantibodies and symptomatology. |

N = number of patients in study.

2.3. Statistical analyses

Differences in proportions between groups were analyzed using the chi-square test. Trends in positivity rates among age groups were analyzed using the Cochran-Armitage test for trend. HHS region 9 (California, Arizona, Hawaii, Nevada) was used as the reference group in statistical analysis because it had the most patients. Multivariable logistic regression models were performed to assess the impact on positivity of potential changes in the demographic factors of patients tested for each antibody during the pandemic. The model used a stepwise entry criterion of p < 0.05 and excluded patients with missing values for any included factor. Data analyses were performed using SAS® Studio 3.6 on SAS® 9.4 (SAS Institute Inc., Cary, NC, USA). This Quest Diagnostics Health Trends® study was deemed exempt by the WCG Institutional Review Board (Puyallup, Washington).
45,051 patients with GM1 results, 19,711 patients with GD1a results, and 18,962 patients with GD1b results. A small number of patients were excluded due to having only indeterminate/inconclusive results (4 for GQ1b, 3 for GM1, 1 for GD1a, and 3 for GD1b), leaving a final analytic cohort representing over 99.9% of potential patients for each outcome (Table 2).

Patient demographics and their respective associations with positive outcomes are shown in Table 2. Notable findings included a significantly higher proportion of males compared to females testing positive for GQ1b (p < 0.001), GD1a (p < 0.001), and GD1b (p = 0.010). GQ1b positivity rate decreased with increasing age groups (p < 0.001 for trend). Conversely, there was a statistically significant increase in the GM1 and GD1a positivity rates with increasing age groups (p < 0.001 for trend).

Reflect a continuation of a declining trend that was demonstrated prior to the COVID-19 pandemic. This area is controversial in the literature. Some studies suggest an association between COVID-19 infection and GBS, while a larger study published by Keddie et al. utilizing a number of methodologies did not show an association and actually reported a decrease in cases of GBS in the United Kingdom. Studies on GBS are fraught with issues regarding case definition and ascertainment bias (Sevjar et al., 2011). We examined positivity rates for antibodies associated with various forms of GBS and found that GQ1b positivity rates declined dramatically after the onset of the pandemic. GM1 positivity rates also declined significantly during the pandemic, but this may reflect a continuation of a declining trend that was demonstrated prior to the pandemic.

4. Discussion

In this study, we examined the positivity rates for several ganglioside antibodies that had been shown to be temporally associated with SARS-CoV-2 infection in case reports (see Table 1). Specifically, we examined whether positivity for antibodies associated with GBS changed during the COVID-19 pandemic. A logistic model adjusting for demographic factors confirmed this association (AOR 0.78, 95% CI 0.72–0.85) GM1 positivity rates also declined significantly in each of the prior two years; however, it is notable that the positivity rate in Q1 2021 (11.4%, 95% CI 10.3–12.6%) was the lowest rate during the study period.

Although it did drop substantially in the most recent quarter where data was available, the GD1a positivity rate was not significantly lower during the pandemic period compared to the entire pre-pandemic period (6.5%, 95% CI 5.8–7.2%; versus 7.2%, 95% CI 6.8–7.6%, p = 0.109; Fig. 2A). The GD1b positivity rate was not lower during the pandemic compared to the prior year (1.8%, 95% CI 1.4–2.2%; versus 1.9%, 95% CI 1.5–2.4%; p = 0.559; Fig. 2B). Logistic regression models adjusting for demographic factors confirmed the lack of association.

### Table 2

Demographics of patient testing and positivity for GBS-associated ganglioside antibodies.

|         | GQ1b Total | GQ1b Positive | GM1 Total | GM1 Positive | GD1a Total | GD1a Positive | GD1b Total | GD1b Positive |
|---------|------------|---------------|-----------|--------------|------------|---------------|------------|---------------|
| Sex     |            |               |           |              |            |               |            |               |
| Female  | 25,006     | 660 (2.6)     | 45,048    | 7734 (17.2)  | 19,711     | 1390 (7.1)    | 18,959     | 556 (2.9)     |
| Male    | 12,434     | 394 (3.1)     | 23,824    | 4050 (17.0)  | 9779       | 804 (8.2)     | 9197       | 305 (3.3)     |
| Age Group (years) | | | | | | | | |
| <18     | 532        | 23 (4.3)      | 418       | 60 (14.4)    | 218        | 8 (3.7)       | 183        | 3 (1.6)       |
| 18–29   | 1422       | 67 (4.7)      | 1868      | 235 (12.6)   | 910        | 48 (5.3)      | 831        | 24 (2.9)      |
| 30–49   | 5008       | 172 (3.4)     | 7893      | 1337 (16.9)  | 3717       | 237 (6.4)     | 3513       | 78 (2.2)      |
| 50–69   | 10,498     | 271 (2.6)     | 19,581    | 3401 (17.4)  | 8391       | 589 (7.0)     | 8205       | 276 (3.4)     |
| ≥70     | 7538       | 127 (1.7)     | 15,273    | 2700 (17.7)  | 6469       | 508 (7.9)     | 6220       | 175 (2.8)     |
| Physician setting/specialty | | | | | | | | |
| Neurology | 9427     | 103 (1.1)     | 19,214    | 3287 (17.1)  | 8673       | 609 (7.0)     | 8957       | 241 (2.7)     |
| Hospital | 6565       | 305 (4.7)     | 11,220    | 1925 (17.2)  | 4404       | 368 (8.4)     | 3967       | 152 (3.8)     |
| General Practice | 2151 | 39 (1.8) | 1868 | 235 (12.6) | 910 | 48 (5.3) | 831 | 24 (2.9) |
| Internal Medicine | 952 | 12 (1.3) | 2958 | 495 (16.7) | 1092 | 71 (6.5) | 950 | 29 (3.1) |
| All Others (ref) | 5864 | 200 (3.4) | 8119 | 1414 (17.4) | 3857 | 235 (6.1) | 3497 | 87 (2.5) |
| Health and Human Services Region | | | | | | | | |
| 1: CT, MA, ME, NH, RI, VT | 1609 | 55 (3.4) | 2370 | 594 (2.2) | 1601 | 115 (7.2) | 1070 | 37 (3.5) |
| 2: NJ, NY | 3060 | 60 (2.0) | 6878 | 1117 (16.2) | 2833 | 213 (7.5) | 2206 | 54 (2.5) |
| 3: DE, DC, MD, PA, VA, WV | 2247 | 90 (4.0) | 3879 | 606 (15.6) | 1381 | 95 (6.9) | 1415 | 40 (2.8) |
| 4: AL, FL, GA, KY, MS, NC, SC, TN | 5857 | 122 (2.2) | 9324 | 1576 (16.9) | 3978 | 293 (7.4) | 4615 | 132 (2.9) |
| 5: IL, IN, MI, MN, OH, WI | 2597 | 88 (3.4) | 2917 | 585 (20.1) | 1306 | 92 (7.0) | 1664 | 61 (3.7) |
| 6: AR, LA, NM, OK, TX | 1785 | 55 (3.1) | 3789 | 630 (16.6) | 1584 | 122 (7.7) | 1144 | 36 (3.2) |
| 7: IA, KS, MO, NE | 726 | 9 (1.2) | 1042 | 269 (25.8) | 802 | 53 (6.6) | 599 | 19 (3.2) |
| 8: CO, MT, ND, SD, UT, WY | 261 | 14 (5.4) | 342 | 39 (11.4) | 100 | 4 (4.0) | 84 | 1 (1.2) |
| 9: AZ, CA, HI, NV (ref) | 6067 | 135 (2.2) | 12,301 | 2059 (16.7) | 5276 | 338 (6.4) | 5761 | 163 (2.8) |
| 10: AK, OR, ID, WA | 581 | 13 (2.2) | 1215 | 249 (20.5) | 825 | 62 (7.5) | 388 | 13 (3.4) |

Chi-square test p < 0.05**; p < 0.01.
to the pandemic. These findings may suggest, as others have concluded, that while COVID-19 may be able to trigger neuroimmunologic conditions such as GBS, that mitigation strategies such as social distancing, mask wearing, and hand hygiene could reduce exposure to infectious agents that might otherwise trigger some forms of GBS, particularly that associated with GQ1b.

One limitation of this study is that we could not determine the specific temporal association of COVID-19 exposure with positivity for ganglioside antibodies. This was partly because SARS-CoV-2 seropositivity is several fold higher than molecular testing positivity, mostly because of the large number of asymptomatic infections occurring in the general population (Rogawski et al., 2021; Stefanelli et al., 2021). Thus, we only compared the positivity rate of antiganglioside antibodies during the pandemic to pre-pandemic levels. In addition, some of the variation in test positivity may represent seasonal variation known to occur with GBS (Webb et al., 2015).

As the exact reason for testing was unknown, there is potential selection bias. However, we were able to view the top 30 ICD-10 codes associated with the ordering of anti-ganglioside testing and this information for the top 10 codes is provided in Supplementary Table 1. Because Quest Diagnostics does not perform all GBS-associated antibody testing in the country, these data should be interpreted as a large, but not exhaustive sample of national data. In fact, this study is one of the largest to date assessing neuroimmunological complications during the COVID-19 pandemic. However, we were not able to review patient charts for specific symptoms of Guillain-Barré to determine reliability of ICD-10 diagnosis codes. In addition, our estimates may be conservative; not every case of GBS demonstrates antibody positivity. Moreover, some clinicians do not order antibody testing even if they suspect GBS. However, the frequency of vitamin D deficiency may reflect the known association of low vitamin D levels observed in GBS and CIDP (Elf et al., 2014). Notably, as vaccines became available for SARS-CoV-2, reports began to surface of GBS in association with vaccination (Allen et al., 2021; Maramattom et al., 2021). One study of 702 patients known to have GBS then vaccinated for SARS-CoV-2 found only one patient required short-term medical care for recurring symptoms (Shapiro Ben...
David et al., 2021). For this reason, we specifically examined the positivity rates for ganglioside antibodies prior to the time when vaccines became available to the general public.

5. Conclusions

These data suggest that heterogeneity in terms of the effect that the COVID-19 pandemic has had on rates of GBS associated with ganglioside antibodies. In particular, while positivity rates for GD1a and GD1b remained largely unchanged during the pandemic, rates of positivity for GQ1b and GM1 were significantly reduced during the pandemic. While these findings do not exclude the possibility that immune responses to SARS-CoV-2 may trigger autoimmune responses to gangliosides as suggested in several case reports (see Table 1), they do suggest that mitigation strategies taken during the pandemic could possibly have reduced the frequency of certain forms of GBS, such as those mediated by GQ1b and GM1.

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References

Abu-Rumeileh, S., Abdelbaky, A., Foschi, M., Tumani, H., Otto, M., 2021. Guillain-Barre syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. J. Neurol. 268, 1133–1170.

Allen, C.M., Ransamy, S., Tarr, A.W., et al., 2021. Guillain-Barre variant occurring after SARS-CoV-2 vaccination. Ann. Neurol. 90, 315–318.

Chan, M., Han, S.C., Kelly, S., Tamimi, M., Giglio, B., Lewis, A., 2021. A case series of Guillain-Barré Syndrome After COVID-19 infection in New York. Neurol. Clin. Pract. 11, e576-e578.

Civardi, C., Collini, A., Geda, D.J., et al., 2020. Antiganglioside antibodies in Guillain-Barré syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. J. Neurol. Neurosurg. Psychiatry 91, 1361–1362.

Cutillo, G., Saariaho, A.-H., Meri, S., 2020. Physiology of gangliosides and the role of antiganglioside antibodies in human diseases. Cell. Mol. Immunol. 17, 313–322.

Elf, K., Askmark, H., Nygren, I., Punga, A.R., 2014. Vitamin D deficiency in patients with Guillain Barre syndrome - a case report, systemic review and implication for vaccine development. Brain Behav. Immun. Health. 12, 100203.

Filosto, M., Cotti Piccinelli, S., Gazzina, S., et al., 2021. Guillain-Barre syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. J. Neurol. Neurosurg. Psychiatry 92, 1361–1362.

Fragiel, M., Miro, O., Llorens, P., et al., 2021. Incidence, clinical, risk factors and outcomes of Guillain-Barre syndrome associated with SARS-CoV-2 infection. J. Neurol. Neurosurg. Psychiatry 91, 1361–1362.

Gigli, A., Slootjes, S.M., Sellimi, A., et al., 2021. Immune-mediated neurological syndromes in SARS-CoV-2-infected patients. J. Neurol. 2021 (268), 751–757.

Keddie, S., Fakpoor, J., Mouzele, C., et al., 2021. Epidemiological and cohort study finds no association between COVID-19 and Guillain-Barre syndrome. Brain. 144, 682–693.

Koike, H., Katsuno, M., 2021. Emerging infectious diseases, vaccines and Guillain-Barré syndrome. Clin. Exp. Neuroimmunol. https://doi.org/10.1111/cen.12644.

Kopczik, M.R., Gourigan, B.K., Presley, B.C., 2020. A case report of acute motor and sensory polyneuropathy as the presenting symptom of SARS-CoV-2. Clin. Pract. Cases Emerg. Med. 4, 352–354.

Lantos, J.E., Strauss, S.B., Lin, E., 2020. COVID-19-associated miller fisher syndrome: MRI findings. Am. J. Neuroradiol. 41, 1184–1186.

Lowery, M.M., Taimur Malik, M., Seemiller, J., Tsai, C.S., 2020. Atypical variant of Guillain Barre syndrome in a patient with COVID-19. J. Crit. Care Med. 6, 231–236.

Maramattom, B.V., Krishnan, P., Paul, R., et al., 2021. Guillain-Barre syndrome following ChAdOx1 S-nCoV-19 vaccine. Ann. Neurol. 90, 312–314.

Masuccio, F.G., Barra, M., Claudio, G., Claudio, S., 2021. A rare case of acute motor axonal neuropathy and myelitis related to SARS-CoV-2 infection. J. Neurol. 268, 2327–2330.

Petrocelli, C., Scandurra, R., Paglieriti, M., Logullo, F.O., 2020. Acute motor axonal neuropathy related to COVID-19 infection: a new diagnostic overview. J. Clin. Neurophysiolog. Dis. 22, 120–121.

Rogowski, E.T., Guettin, K.A., Becker, L., et al., 2021. Assessment of seroprevalence of SARS-CoV-2 and risk factors associated with COVID-19 infection among outpatients in Virginia. JAMA Netw. Open 4, e2035234.

Sevjar, J.J., Baughman, A.L., Wise, M., Morgan, O.W., 2011. Population incidence of Guillain-Barre syndrome: a systematic review and meta-analysis. Neuroepidemiology. 36, 123–133.

Shapiro Ben David, S., Potsman, L., Rahamim-Cohen, D., 2021. Rate of recurrent Guillain-Barré syndrome after mRNA COVID-19 vaccine BNT162b2. JAMA Neurol. 78, 1409–1411. https://doi.org/10.1001/jamaneurol.2021.3287.

Stefanelli, P., Bellis, A., Pedele, G., et al., 2021. Prevalence of SARS-CoV-2 IgG antibodies in an area of northeastern Italy with high incidence of COVID-19 cases: a population-based study. Clin. Microbiol. Infect. 27, 633.e1–633.e7.

Wanleenawat, P., Iwanowski, P., Koabchi, W., 2019. Antiganglioside antibodies in neurological diseases. J. Neurol. Sci. 408, 116576.

Webb, A.J.S., Brain, S.A.E., Wood, R., Rinaldi, S., Turner, M.R., 2015. Seasonal variation in Guillain-Barre syndrome: a systematic review, meta-analysis, and Oxfordshire cohort study. J. Neurol. Neurosurg. Psychiatry 86 (11), 1196–1201.