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Epilepsia®

GRAY MATTERS

Letters

The risk of Stevens-Johnson syndrome and toxic epidermal necrolysis in new users of antiepileptic drugs: Comment on data sparsity

Dear Editor-in-Chief:

We, enthusiastically, read the article authored by Frey and colleagues that was published in *Epilepsia* in December 2017.[1] The study was conducted to examine the risks of Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN) in association with use of all antiepileptic drugs (AEDs) in the United Kingdom. The authors reported that there is a strong association between SJS/TEN and new use of carbamazepine (odds ratio [OR] 92.57, 95% confidence interval [CI] 19.89-∞], phenytoin (OR 49.96, 95% CI 10.13-∞), and lamotrigine (OR 26.90, 95% CI 4.88-∞), which is questionable. It has been stated that huge effect-size estimates with remarkably wide CIs are yielded in the studies with insufficient observations in the *exposure-outcome* combinations, which is known as *data sparsity*.[2,3] As a matter of fact, these inflated effect-size estimates are biased due to *sparse data bias*.[2] We assessed the data on the association between SJS/TEN and new use of carbamazepine, phenytoin, and lamotrigine, and serious data sparsity is expected (see our Table 1).

The *Penalization through Data Augmentation* is one of the new and efficient statistical methods that can be used to reduce the *sparse data bias*.[2] We reanalyzed the authors' reported associations using the *penalization* methods and it was found that shrunk ORs with shorter CIs were obtained, which means that the less-biased ORs has been yielded (see our Table 1).

The take-home message for the readers is that the *sparse data bias* is common in medical research,[4,5] and that needs to be reduced by using the advanced statistical methods.

TABLE 1 The crude association between Stevens-Johnson syndrome and toxic epidermal necrolysis (SJS/TEN) and new use of carbamazepine, phenytoin, and lamotrigine using ordinary and *penalized* logistic regression

	Cases (n = 480)	Controls (n = 1920)
Carbamazepine		
≤84 days prior to the index date	16	0
Reference group	464	1920
Estimated OR (95% CI)		
Ordinary logistic regression	92.57 (19.89-∞)	
Penalized logistic regression	21.12 (6.07-73.41)	
Phenytoin		
≤84 days prior to the index date	5	0
Reference group	475	1920
Estimated OR (95% CI)		
Ordinary logistic regression	26.90 (4.88-∞)	
Penalized logistic regression	6.05 (1.46-25.01)	
Lamotrigine		
≤84 days prior to the index date	9	0
Reference group	471	1920
Estimated OR (95% CI)		
Ordinary logistic regression	49.96 (10.13-∞)	
Penalized logistic regression	11.34 (3.04-42.28)	

AUTHOR CONTRIBUTIONS

All authors were involved in the manuscript conception, design, drafting and revising, and final approval of the submitted version.

DISCLOSURE

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Response: The risk of Stevens-Johnson syndrome and toxic epidermal necrolysis in new users of antiepileptic drugs: Comment on data sparsity

Dear Editor-in-Chief

We have read the comment on our article with great interest, and we would like to thank Dr Safiri and Dr Ashrafi-Asgarabad for their interest in our study. However, we do not think that the suggested penalized odds ratios (ORs), which were quantified using data augmentation, accurately reflect the association between Stevens-Johnson syndrome (SJS) and aromatic antiepileptics.

Drs Safiri and Ahran-Asgarabad deemed our presented ORs for the association between SJS and carbamazepine (OR 92.57, 95% confidence interval [CI] 19.89-∞), phenytoin (OR 49.96, 95% CI 10.13-∞), and lamotrigine (OR 26.90, 95% CI 4.88-∞) as too extreme and biased by sparse data, and instead presented penalized ORs (eg, OR 6.05, 95% CI 1.46-25.01 for phenytoin), which they quantified using the data augmentation method. Although Greenland et al emphasized that the method of data augmentation strongly relies on preexisting evidence, the authors do not report on the basis of which existing evidence their penalization priors were chosen or which 95% prior intervals were applied, which prevents us from accurately interpreting the suggested results.[1,2] SJS is a rare disease that is caused mainly by new use of a few specific drugs, of which aromatic antiepileptics by far bear the highest risk of triggering SJS. We previously calculated absolute risks of SJS/toxic epidermal necrolysis in new users of trigger drugs other than antiepileptics of 1-6 cases/100 000 new users (one study not published yet),[3] whereas the absolute risk among new users of aromatic antiepileptics was 20-45 cases/100 000 new users.[4] Thus, the expected relative risk estimates for SJS in association with new antiepileptic use can be expected to be very high, which was also suggested in the comprehensive hospital-based EuroSCAR case-control study, which reported ORs of 72 (95% CI 26-225) for carbamazepine and 26 (95% CI 7.8-90) for phenytoin.[5] We agree that extremely high relative risks from observational studies need to be interpreted carefully. However, in the absence of any prior evidence suggesting lower ORs, using a penalization prior which artificially corrects ORs toward the null might provide a false sense of certainty. We therefore think that the more conservative approach we chose is the method of choice here, whereby the wide confidence intervals indicate the level of uncertainty due to small sample size.

Furthermore, given the low numbers of exposed patients in our study population, which is an inherent problem when studying rare diseases like SJS, we took several precautions to avoid sparse data bias. First, we refrained from conducting multivariable adjustment of our ORs (confounding is not a major issue when studying SJS), but instead matched cases and controls on age, sex, and index date as suggested by

Greenland et al and quantified the proportion of patients who were concomitantly exposed to other high-risk drugs. Furthermore, we conducted exact logistic regression whenever a zero cell was observed to avoid sparse data bias.[2]

In conclusion, we agree that data augmentation is a valid new method to avoid sparse data bias, but we do not necessarily agree that this method should have been used in our study.

DISCLOSURE

None of the authors have any conflict of interest to declare. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Sparse data and use of logistic regression

The performance of logistic regression, commonly used to analyze a binary response variable, is questionable in the presence of sparse data. When the logistic regression yields very high odds ratios (ORs) with very wide confidence intervals (CIs), this alerts us to the possibility that we are dealing with sparse data. The reason for this "blowing up" of parameter estimates is that the function one is trying to maximize does not have a clear maximum. Thus, even when the model converges, the regression coefficients and standard errors that are obtained are very large, signaling estimation difficulties. The degree of this "small-sample bias" (a misleading term, because this problem can happen with very large datasets) is mainly dependent on the number of cases in the less frequent of the 2 categories. For example, there may be substantial bias, even with a sample size of 100 000, if there are only 20 events in the sample. The recommended method to analyze sparse data is to use a penalized maximum likelihood estimation method.[1,2] The idea behind penalized maximum likelihood estimate is that the method penalizes the likelihood by subtracting a "penalty" from the log-likelihood such that it will shrink the final estimates.[3] The choice of penalty is guided by background information—for example, that large values for the parameter are usually implausible.

An alternative approach to analyzing sparse data is to use an exact logistic regression method. The idea behind this method is the same as the exact inference (Fisher's exact test) for a 2×2 table. For large datasets and for models requiring many covariates (especially if they are continuous and not discrete), this method is computationally very intensive and usually not used in regular practice. When not adjusting for many covariates, the use of exact methods can mitigate the sparse data problem. However, it

has been shown that even exact methods give biased estimates,[4] and penalization methods are therefore seen as preferable for sparse data analysis. If sufficient prior information is not available, or for variables whose effects are thought to be very large, a weaker penalization (equivalent to using weakly informative Bayesian priors[5]) may be used.

In their study of Stevens-Johnson syndrome and toxic epidermal necrolysis and its association with antiepileptic drug use, Frey et al[6] have used exact methods to estimate the ORs and CIs "where there were no exposed cases/controls." Even so, the resulting ORs are large and the CIs are very wide (including infinite values). Therefore, it is possible that in this case, penalization would have been the preferred method to stabilize the estimates, as suggested by Safiri and Ahran-Asgarabad.[7] The response by Frey et al.[8] notes correctly that Safiri and Ahran-Asgarabad do not include detailed information on the penalization priors. This lack of information does not allow the reader to evaluate the validity of the resulting estimates.

In summary, in the analysis of rare events, especially when dealing with large samples, researchers need to be vigilant. If possible, they should examine frequency tables of the outcome and the exposure variables. As data become more complex, and in the presence of many covariates, this is not feasible. In such situations, sensitivity analyses are valuable for detecting sparse data problems, done by augmenting the observed data with a small amount of additional data. If the model estimates are unchanged with the additional information, then the data are likely not sparse; conversely, if the parameter estimates are sensitive to the additional information, then the data being analyzed are likely sparse. The researchers then should take appropriate steps to deal with sparse data, such as making use of penalized likelihood ratio methods.

DISCLOSURE

The author has no conflict of interest to disclose. I confirm that I have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Photosensitivity as an early marker of epileptic and developmental encephalopathies

To the Editors:
The well-performed and informative retrospective study by Specchio et al.[1] showed the importance of intermittent photic stimulation (IPS) in diagnosing progressive neuronal ceroid lipofuscinosis (NCL2) at an early stage of the disease (<2 years of age). We wish to highlight that photoparoxysmal electroencephalographic (EEG) responses (PPRs) appeared to be the only evidence of this neurode-

generative disease before the onset of psychomotor regression in 14 patients. Other seizure or EEG variables were not predictive.

Recently, EEG responses to IPS were studied in 53 patients with *SCN1A*-related Dravet syndrome (DS) in a similar way and with comparable results.[2] PPRs were most frequently induced in the second year of life soon after the first seizure(s) and before occurrence of DS type signs or symptoms.[2] Furthermore, the 42% PPR-positive patients developed more severe disabilities than those without a PPR, regardless of *SCN1A* mutation type or sex. With other genes involved in progressive myoclonic syndromes starting at the infant age (potassium channel–related gene *KCTD7*) or later (*PRICKLE1*, *CSTB*, *NHLRC1*), PPRs can also be recorded frequently at an early stage of the disease.[3–6] When the encephalopathic process advances, the strong reaction to IPS can even disappear, as has been shown in Unverricht-Lundborg disease by Ferlazzo et al.[6]

There is a clear relationship between spontaneous discharges and PPRs at the early stages of disease, although the dominant localization of these could differ among the various syndromes; in NCL2 they are predominantly temporal, whereas occipital regions are mainly involved in DS patients and in *KCTD7*-related progressive myoclonus epilepsy.[1–6] PPRs are most typically for epileptic encephalopathies elucidated time-locked at low flash frequencies, but can occur at higher flash frequencies (only) as well.[1–6]

Thus, we want to stress that IPS is an important method for early diagnosis of a variety of progressive encephalopathic diseases. Delay in the performance of an EEG with photic stimulation in early onset epileptic and developmental encephalopathies could result in a dramatic delay in the etiologic diagnosis based on further genetic testing.[1–6] It is advised to use a standardized IPS methodology in all infants and children soon after their first seizures have occurred.[7]

DISCLOSURE

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Announcements

Epilepsia – May 2018 – Announcements

Young Epilepsy Section: YES Kick-Off Workshop

12–13 May 2018
London, UK
Information: https://www.ilae.org/congresses/young-epilepsy-section-yes-kick-off-workshop

EILAT Conference on New Antiepileptic Drugs and Devices (EILAT XIV)

13–16 May 2018
Madrid, Spain
Website: https://www.eilatxiv.com/

8th Congress of the Polish Society of Epileptology

17–19 May 2018
Warsaw, Poland

Infantile Epilepsy in Light of New ILAE Classification – New Terminology, Etiology and Treatment Perspectives

28 May 2018
Tbilisi, Georgia
Information: https://www.ilae.org/congresses/infantile-epilepsy-in-light-of-new-ilae-classification-new-terminology-etiology-and-treatment-perspectives

30th Annual Meeting of the European Academy of Childhood Disability (EACD)

28–31 May 2018
Tbilisi, Georgia
Information: https://www.ilae.org/congresses/30th-annual-meeting-of-the-european-academy-of-childhood-disability-eacd

Joint Annual Meeting of the Swiss League Against Epilepsy and the Swiss Society of Clinical Neurophysiology

30–31 May 2018
Aarau, Switzerland
Website: http://www.sgkn-congress.ch/

9th Simposio Internacional de Epilepsias

31 May–1 June 2018
Santiago, Chile
More information: https://www.ilae.org/congresses/9th-simposio-internacional-de-epilepsias

Norwegian League Against Epilepsy 2018 Chapter Congress

1–2 June 2018
Trondheim, Norway

37th Congresso da Liga Brasiliera de Epilepsia

6–9 June 2018
São Paulo, SP, Brazil
Website: http://congresso.epilepsia.org.br/2018/

4th East European Course on Epilepsy

13 June 2018
Shishkinn, Chernihiv Region, Ukraine
Website: http://ulae.org.ua/eece/2018/

54th Annual Meeting of the German Society of Epileptology (DGfE) e. V.

13–16 June 2018
Stadthalle Fürth
Rosenstraße 50 • 90762 Fürth, Germany
Website: http://www.epilepsie-tagung.de/

2018 PAME Conference (Partners Against Mortality in Epilepsy)

14–16 June 2018
Alexandria, Virginia, USA
Website: http://pame.aesnet.org/16-19June 2018

4th Congress of the European Academy of Neurology

16–19 June 2018
Lisbon, Portugal
Website: https://www.ean.org/lisbon2018/

12th Baltic Sea Summer School on Epilepsy (BSSSE 12)

24–29 June 2018
Vilnius, Lithuania
Website: www.epilepsiestiftung-wolf.de

Epileptic Channelopathies—Clinical Spectrum and Treatment Perspectives

28–29 June 2018
3rd Dianalund International Conference on Epilepsies
Danish Epileptic Center, Sørup Herregård, Ringsted, Denmark
Program and registration: https://www.ilae.org/congresses/epileptic-channelopathies-3rd-dinalund-international-conference-on-epilepsies

12th Asian and Oceanian Epilepsy Congress

28 June–1 July 2018
Bali, Indonesia
Website: www.epilepsybali2018.org

4th Dianalund Summer School on EEG and Epilepsy

15–21 July 2018
Dianalund, Denmark
Application and Announcement: https://www.ilae.org/congresses/4th-dianalund-summer-school-on-eeg-and-epilepsy

16th Advanced San Servolo Epilepsy Course

16–27 July 2018
San Servolo (Venice), Italy
Application and Announcement: https://www.ilae.org/congresses/16th-advanced-san-servolo-epilepsy-course

8th International Summer School for Neuropathology and Epilepsy Surgery (INES 2018)

26–29 July 2018
Erlangen, Germany
Information: https://www.ilae.org/congresses/8th-international-summer-school-for-neuropathology-and-epilepsy-surgery-ines-2018

4th Summer School on Imaging in Epilepsy: SuSIE 2018

12–15 August 2018
Marburg, Germany
Website: http://www.imaging-in-epilepsy.org/

13th European Congress on Epileptology

26–30 August 2018
Vienna, Austria
Website: www.epilepsyvienna2018.org

ESTM 2018 Vienna: Epilepsy Surgery Techniques

31 August–1 September 2018
Vienna, Austria
Satellite symposium for the European Congress on Epilepsy
Website: http://www.estm2018.at/

Congreso de Epilepsia: 2018. Liga Agentina—LACE

13–14 September 2018
Chapter website: http://www.lace.org.ar/

9th International Summer School for Neuropathology and Epilepsy Surgery (INES 2018)

17–20 September 2018
Beijing, China
Information: https://www.ilae.org/congresses/9th-international-summer-school-for-neuropathology-and-epilepsy-surgery-ines-2018

International Symposium on Severe Infantile Epilepsies: Old and New Treatments (ISSET 2018)

20–22 September 2018
Vatican City, Rome, Italy
Website: http://www.ptsroma.it/isset2018/

Cleveland Clinic Epilepsy Update & Review Course

22–24 September 2018
Cleveland, Ohio, USA
CME Credits available
Website: http://www.clevelandclinicmeded.com/live/courses/epilepsy-update/

10th Latin American Congress on Epilepsy

29 September–2 October 2018
San José, Costa Rica
Website: http://epilepsysanjose2018.org/

CLAE/LCCE 2018 Scientific Meeting

21–23 September 2018
St. John's, Newfoundland
Website: https://canadianleagueagainstepilepsy.wildapricot.org/page-1816302

LAE British Chapter Annual Scientific Meeting

26–28 September 2018
Birmingham, UK
Website: http://www.ilaebritishconference.org.uk/

6th Global Symposium on Ketogenic Therapies for Neurological Disorders: Embracing Diversity, Global Implementation and Individualized Care

5–9 October 2018
Jeju, Korea
Website: www.ketoconnect.org

46th Annual Meeting of the International Society for Pediatric Neurosurgery (ISPN 2018)

7–11 October 2018
Tel Aviv, Israel
Website: http://www.ispnmeeting.org/2018

Hungarian Chapter of the ILAE

12 October 2018
Chapter website: http://www.epilepszia.hu/

Journées Françaises de l'Epilepsie

16–19 October 2018
Centre de Congres, Lyon, France
Website: https://www.jfe-congres.fr/

32nd Epilepsy Society of Australia Annual Scientific Meeting

31 October–2 November 2018
Brisbane, Australia
Website: https://www.epilepsy-society.org.au/conferences/esa-asm.asp

Swedish Chapter National Meeting

15 November 2018
Lund, Sweden

Annual Meeting of the Austrian and German Societies for Epileptology and the Swiss Epilepsy League ("Dreilaendertagung")

8–11 May 2019
Basel, Switzerland
Website: www.epi.ch/fach

33rd International Epilepsy Congress

22–26 June 2019
Bangkok, Thailand
Website: http://internationalepilepsycongress.org/