

# UNIVERSITY OF BIRMINGHAM

## Research at Birmingham

### Response to Professor Kawada

Turner, Graham; Feltham, Maxwell; Ryan, Ronan; Marshall, Tom; Calvert, Melanie

DOI:

[10.1111/ene.13175](https://doi.org/10.1111/ene.13175)

License:

Other (please specify with Rights Statement)

*Document Version*

Peer reviewed version

*Citation for published version (Harvard):*

Turner, G, Feltham, M, Ryan, R, Marshall, T & Calvert, M 2017, 'Response to Professor Kawada', *European Journal of Neurology*, vol. 24, no. 1, e1. <https://doi.org/10.1111/ene.13175>

[Link to publication on Research at Birmingham portal](#)

#### **Publisher Rights Statement:**

This is the peer reviewed version of the following article: Turner, G., Calvert, M., Feltham, M., Ryan, R. and Marshall, T. (2017), Response to Professor Kawada. *Eur J Neurol*, 24: e1. doi:10.1111/ene.13175, which has been published in final form at 10.1111/ene.1317. This article may be used for non-commercial purposes in accordance with Wiley Terms and Conditions for Self-Archiving.

#### **General rights**

Unless a licence is specified above, all rights (including copyright and moral rights) in this document are retained by the authors and/or the copyright holders. The express permission of the copyright holder must be obtained for any use of this material other than for purposes permitted by law.

- Users may freely distribute the URL that is used to identify this publication.
- Users may download and/or print one copy of the publication from the University of Birmingham research portal for the purpose of private study or non-commercial research.
- User may use extracts from the document in line with the concept of 'fair dealing' under the Copyright, Designs and Patents Act 1988 (?)
- Users may not further distribute the material nor use it for the purposes of commercial gain.

Where a licence is displayed above, please note the terms and conditions of the licence govern your use of this document.

When citing, please reference the published version.

#### **Take down policy**

While the University of Birmingham exercises care and attention in making items available there are rare occasions when an item has been uploaded in error or has been deemed to be commercially or otherwise sensitive.

If you believe that this is the case for this document, please contact [UBIRA@lists.bham.ac.uk](mailto:UBIRA@lists.bham.ac.uk) providing details and we will remove access to the work immediately and investigate.

We thank Professor Kawada for his interest in our study regarding ongoing impairments following transient ischaemic attack (TIA).[1] We are particularly encouraged that he recognised the need to improve care for TIA patients presenting with signs of fatigue, psychological impairment or cognitive impairment.

The study of impairments post-TIA is currently under-researched. Indeed, the findings of our systematic review,[2] which aimed to establish prevalence of impairments post-TIA/minor stroke, were inconclusive because the quality of the included studies was not high. Our review identified the limitations of existing research and our current study was conducted in response to this gap in the literature. We addressed these limitations through subsequently conducting a retrospective cohort study which has a large sample size, included matched controls and adjusted for confounding variables and presence of impairments pre-TIA. We believe this robust study design provides reliable estimates to measure the association between TIA and subsequent impairments. However, future research is required to determine the incidence, prevalence and natural history of impairments post-TIA.

In response to Professor Kawada's second comment regarding the possibility of impaired cognitive function being present prior to TIA, for this reason we purposefully adjusted for presence of cognitive impairment prior to TIA in our main analysis. Furthermore, we conducted an exploratory analysis which excluded people with cognitive impairment recorded prior to TIA. In this exploratory analysis the association between TIA and cognitive impairment remained; indeed, the adjusted hazard ratio actually increased from 1.45 (95% CI, 1.28–1.65) to 1.54 (95% CI, 1.35–1.77). However, as we discussed in the limitations, it is possible that cognitive impairment prior to TIA may have been undetected or not recorded. We agree that caution also should be paid regarding inclusion of patients with dementia; however, only 4% and 3.4% of TIA patients and controls, respectively, had dementia recorded.

The mechanism of residual impairments post-TIA is unknown and in our discussion we hypothesised three potential explanations: (i) presence of cerebral infarction, i.e. minor stroke misdiagnosed; (ii) undetected microinfarcts; and (iii) psychological impact of the event. An alternative hypothesis could be that cognitive impairment is caused by vascular risk factors which predispose TIA. However, a systematic review by Blom et al. (2013) found inconsistent results for the association between vascular risk factors and cognitive decline in patients with dementia.[3] We agree that the mechanism of cognitive impairment necessitates further research and this information may facilitate the development of an intervention to improve cognition in TIA patients.

## References

[1] Turner GM, Calvert M, Feltham MG, Ryan R, Marshall T. Ongoing impairments following transient ischaemic attack: retrospective cohort study. *Eur J Neurol*. 2016; doi: 10.1111/ene.13088

[2] Moran GM, Fletcher B, Feltham MG, Calvert M, Sackley C, Marshall T. Fatigue, psychological and cognitive impairment following transient ischaemic attack and minor stroke: a systematic review. *Eur J Neurol* 2014;21: 1258–1267.

[3] Blom K, Emmelot-Vonk MH, Koek HL. The influence of vascular risk factors on cognitive decline in patients with dementia: a systematic review. *Maturitas*. 2013;76(2):113-7.