

Response to letter by Prof Christian Nolte and colleagues

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European Stroke Journal
2022, Vol. 7(3) 341–342
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DOI: 10.1177/23969873221110378
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We thank the authors for their letter. This is a crucial point and one we are keen to address.

We interpret that the key concern is whether every person with ischaemic stroke or TIA should aim for an LDL cholesterol level of <1.8 mmol/l. The guideline on pharmacological interventions for long term secondary prevention after ischaemic stroke or TIA is not intended to cover in detail specific types of stroke, such as cervical artery dissection or specific types of cardioembolic stroke. These are covered in already published guidelines or in guidelines that are under development (see <https://eso-stroke.org/guidelines/eso-guideline-directory/>). The balance between making recommendations in a guideline, which is a general set of rules or piece of advice, and the required detail and nuance to manage individual patients is challenging to find. There is no better example of this than the issue they raise.

We agree there is a need for greater clarity. The authors correctly highlight that the main evidence supporting a recommendation for a lower LDL cholesterol target comes from the Treat Stroke to Target trial. To be included in this trial, participants had to have evidence of extracranial or intracranial stenosis, aortic arch plaque or a known history of coronary artery disease. We also agree that the benefit of high intensity statin therapy, or a lower LDL target, will be greatest in people with large artery disease, at least in absolute terms. In the SPARCL trial, while an eligible TIA was required to be assumed to have an atherosclerotic cause, this was not the case for stroke. Thus, people without proven atherosclerosis could have been included, provided they did not have atrial fibrillation. We believe the totality of evidence for intensive lipid lowering goes beyond those with a confirmed large artery stroke but agree it does not apply to every person.

We agree that there will be people for whom an LDL target of <1.8 mmol/l is not appropriate and where the benefit of this is not yet quantified. We could have raised this more specifically in the discussion where we discussed these principles in the last paragraph. We hope that the guideline consolidates the need to consider more

aggressive management of blood pressure, lipids and blood glucose (in people with diabetes). Thus, we agree with the authors that clinicians should be familiar with whom these targets apply, but that overall the evidence suggests the approach to secondary prevention should be more intensive. We believe this is an important step away from one size fits all, or ‘fire and forget’ approaches to management of cardiovascular risk factors.

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Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical approval

Not Applicable.

Informed consent

Not Applicable.

Guarantor


JD.

Contributorship

JD wrote the response and all authors reviewed and agreed with the submission.


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