Ethics, evidence, and the environment in dementia risk reduction

Authors' reply

Timothy Daly's response to our Comment[1] introduces three important considerations that we welcome.

Firstly, there is an ethical obligation to adopt population-based risk reduction policies over individual-based policies to counter the so-called moralisation of health behaviours.

We agree that this ethical imperative exists. There is a clear socioeconomic gradient in almost all known chronic diseases and their risk factors, including in dementia.[2] Exclusively placing the onus for addressing this problem on individuals, rather than on society, is akin to saying that poor individuals are feckless.[3]

Arguably, however, the ethical imperative for population-based approaches does not simply involve avoiding the moralisation of health behaviours, but extends to asking what works and for whom? A strong body of evidence points to the fact that simply educating people about their increased risk of ill-health and encouraging them to do something about it is not sufficient to induce sustained behavioural changes, and might disproportionately benefit individuals with the greatest resources (eg, material, social, cognitive).[4] Instead, the greatest health benefit for the individuals who need it the most might come from altering the social, cultural, and economic context in which we all live.

Furthermore, although we agree that an individualised focus on risk can, and has, contributed to the moralisation of ill-health, the extent to which the modifiable risk factors for dementia identified by the *Lancet* Commission[5] are subject to such moralisation could be questioned. Although some risk factors are subject to moralisation, others, such as education and social isolation, might be less so. Nonetheless, the broader ethical imperative for population-based measures to reduce health inequalities surely holds, as it is a widely held belief that society must play a role in educating and connecting its citizens. This role includes being transparent about the logic that underpins efforts to change the wider societal context.

Secondly, the randomised control trial design is not well suited to evaluating population-based approaches to dementia risk reduction.

Consistent evidence from high-quality observational studies, such as the Cognitive Function and Aging studies,[6] show that age-specific incidence of dementia has been reduced by improvements in population health, and could, therefore, be further reduced in the future. In an ideal world, studies quantifying precise effect sizes, attributable to specific public health interventions (eg, every cycle lane built prevents *x* in 1000 cases of dementia, reducing dementia-related health-care and social-care costs by *y*), would convince policy makers to take further action on risk factors that are modifiable across an individual's life course. However, timeframes and the interconnectedness of risks and protective factors arguably make this type of exercise naive.

As Daly points out, these kinds of interventions are not amenable to a randomised control design, widely known as the gold-standard study design for suggesting an intervention is efficacious. As a result, the interventional evidence that has been attempted for dementia risk reduction, such as the FINGER study[7] referenced by Daly, has focused on individual-based interventions. As outlined in our Comment,[1] it is not feasible to scale the intensive support offered to individuals within these studies to the level of population need; and in any case the results are rather unconvincing even in those volunteering to participate.

An alternative study design could be the cluster randomised trial, often used by public health academics. However, these trials are limited by two key challenges: (1) dementia generally results from cumulative exposure to multiple risk factors over an individual's life course, requiring unfeasibly long study duration, and (2) there is a relatively low incidence of dementia before very old age, which reduces the statistical power of results.

We must, therefore, look to alternatives to provide robust, but practical, evaluations of population-based approaches to dementia risk reduction, such as modelling studies[8] and natural experiment studies.[9] As for all study designs, there are limitations and challenges to these designs. Modelling studies rely on the robustness and precision of the estimates that inform their assumptions, whereas natural experiment studies are complex to design, execute, and interpret,[10] and residual confounding can never be completely removed. Clearly, further careful, high-quality work in this area could provide opportunities to build the evidence base for population-based dementia risk reduction.

Thirdly, population-based approaches must aim to narrow the health inequalities that are exacerbated by individual-based approaches.

Daly makes an important point: it is not sufficient to say that individualistic interventions reliant on conscious behaviour will increase health inequalities. We must also set out how population-based interventions can reduce them, and how these interventions can contribute to an equitable approach to public health. Here, we point again to Marmot's work on proportionate universalism,[3] and its emphasis on the delivery of services at a scale and intensity that is proportionate to the level of need and disadvantage of a given population subgroup.

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1 Walsh S, Govia I, Wallace L, et al. A whole-population approach is required for dementia risk reduction. *Lancet Healthy Longev* 2022; **3:** e6–8.

2 Kivimäki M, Batty GD, Pentti J, et al. Association between socioeconomic status and the development of mental and physical health conditions in adulthood: a multi-cohort study. *Lancet Public Health* 2020; **5:** e140–9.

3 Marmot M, Bell R. Fair society, healthy lives. *Public Health* 2012; **126:** S4–10.

4 Milne R, Altomare D, Ribaldi F, Molinuevo JL, Frisoni GB, Brayne C. Societal and equity challenges for Brain Health Services. A user manual for Brain Health Services—part 6 of 6. *Alzheimers Res Ther* 2021; **13:** 173.

5 Livingston G, Huntley J, Sommerlad A, et al. Dementia prevention, intervention, and care: 2020 report of the *Lancet* Commission. *Lancet* 2020; **396:** 413–46.

6 Matthews FE, Arthur A, Barnes LE, et al. A two-decade comparison of prevalence of dementia in individuals aged 65 years and older from three geographical areas of England: results of the Cognitive Function and Ageing Study I and II. *Lancet* 2013; **382:** 1405–12.

7 Ngandu T, Lehtisalo J, Solomon A, et al. A 2 year multidomain intervention of diet, exercise, cognitive training, and vascular risk monitoring versus control to prevent cognitive decline in at-risk elderly people (FINGER): a randomised controlled trial. *Lancet* 2015; **385:** 2255–63.

8 McRae I, Zheng L, Bourke S, Cherbuin N, Anstey KJ. Cost-effectiveness of dementia prevention interventions. *J Prev Alzheimers Dis* 2021; **8:** 210–17.

9 Seblova D, Fischer M, Fors S, et al. Does prolonged education causally affect dementia risk when adult socioeconomic status is not altered? A Swedish natural experiment in 1.3 million individuals. *Am J Epidemiol* 2021; **190:** 817–26.

10 Ogilvie D, Adams J, Bauman A, et al. Using natural experimental studies to guide public health action: turning the evidence-based medicine paradigm on its head. *J Epidemiol Community Health* 2020; **74:** 203.