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Child conduct problems and social skills in a middle-income country

We commend Baker-Henningham *et al*[1] for carrying out a relevant and important intervention study on pre-school children with conduct problems and poor social skills in a middle-income country. Classroom and school intervention studies are sparse from low- and middle-income countries and this work is a step in the right direction. However, we would like to highlight certain issues. First, the authors chose pre-school children (age 3–6 years) as the target population for their intervention, whereas the typical age at onset of conduct disorder is 11.6 years.[2] They also did not mention explicitly whether the children had a syndromal diagnosis of conduct disorder. Assessment of attention-deficit hyperactivity disorder, visual and hearing deficits, intellectual disability and pervasive developmental disorder would have led to better interpretation of the results, as these conditions may have an impact on the outcome of conduct problems.[3] In addition, children with low attendance were excluded from the study, even though it is known that children with severe conduct problems are less likely to attend school. This might have led to an inadvertent selection of children with less severe conduct problems in the study. Further, statistically significant improvements were not found in the parent reports of conduct problems. This suggests that the improvements were limited to the school setting and did not generalise to the home environment. Interventions such as the Incredible Years Teacher Training programme help teachers to manage difficult pupils better in school and to promote friendships, and deserves a place in the teachers' training curricula. Baker-Henningham *et al* included only children with severe problems for assessment and significant results were seen in those with low-to-moderate levels of conduct problems. Evidence for other psychiatric disorders suggests that improvement is more apparent in those with a more severe form of the disorder and the effects are less when the symptoms are subthreshold and approach normalcy.[4] The result is that severely disordered children are expected to benefit more. This in turn may have a domino effect on the behaviour of other children. The developmental complexities of child behaviour are immense. Interventions that help both children and the community are likely to pay dividends as these children mature.

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Assessing the role of cerebrovascular disease in the incidence of geriatric depression

The association between vascular disease and late-life depression is an example of how a common 'medical' disorder could have clinically significant neuropsychiatric sequelae. With cerebrovascular disease, a prominent cause of mortality and disability in the aged population, this is a major public health issue. In their longitudinal study of white matter changes and depression incidence, Firbank *et al*[1] note that the cross-sectional nature of existing work prohibits conclusions about the direction of causality, with their prospective investigation a welcome contribution to this exciting field. A temporal sequence of white matter disease before depression supports the use of neuroimaging in screening 'at risk' individuals and implicates cardiovascular risk factors in the pathogenesis of geriatric mood disorder. However, other recent studies have suggested that this sequence could be bidirectional.[2] As I argue elsewhere,[3] the relationship between physical disease and mood disorder in the elderly is likely to be aetiologically complex and characterised by reciprocity.

Firbank *et al* present results from the LADIS study and conclude that in their patients, progression of white matter disease was associated with depression incidence. However, I believe the analytical methods used by the authors affect the significance of this finding and warrant discussion.

A cohort study of harm typically compares individuals exposed to a risk factor (white matter changes) with those unexposed. The two groups are followed to monitor the incidence of the adverse effect (depression incidence), which allows for the calculation of a relative risk (the hazard ratio). However, in this study the authors used the equivalent of a *t*-test for non-parametric data to compare the level of white matter changes between groups of patients according to their depression status. When the results are presented in this manner, depression status effectively becomes the exposure. Therefore, although it is possible for the authors to conclude that exposure to depression at baseline did not lead to white matter changes, their claim that white matter changes predict depression incidence seems less certain. The presence of overlapping 95% confidence intervals between cohorts also introduces doubt about whether the true value of white matter changes between populations is significantly different, although a wide confidence interval in those patients with depression onset in year 3 of the study is likely related to the small number of patients in this group.

Firbank *et al* then make a careful attempt to identify and control for potential confounders in their regression analysis. Here, however, the 95% confidence interval for the relationship between white matter changes and depression includes 1 (unity). With such marginal significance, the fate of those patients who were lost to follow-up (over 30%) seems increasingly relevant. Moreover, I wonder why the authors chose to use the Folstein Mini-Mental State Examination as a correlate of cognitive impairment, when executive dysfunction is often most problematic in these patients.[4]

Future studies might dichotomise patients into 'high white matter changes' and 'low white matter changes' exposure cohorts to more accurately quantify risk and demonstrate a biological gradient for the effects of vascular disease on mood disorder.

1 Firbank MJ, Teodorczuk A, van der Flier WM, Gouw AA, Wallin A, Erkinjuntti T, et al. Relationship between progression of brain white matter changes and late-life depression: 3-year results from the LADIS study. *Br J Psychiatry* 2012; **201**: 40–5.

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Authors' reply: We thank Dr Mosley for his interest in our paper. As noted in the discussion to the paper, we agree with him that the relationship between physical disease and mood disorder is complex. However, we do not agree regarding the other points raised.

Our two-group statistic presented in the paper is quite appropriate. We compared progression of white matter changes between those with and without depression at the 3-year point, and demonstrated with a high significance that those with depression had a greater progression of white matter changes prior to depression. If, as suggested by Mosley we dichotomise the white matter change progression, then high white matter change progression is still significantly associated ($P<0.01$) with both depression at 3 years, and any depression over the 3 years.

Patient drop-out is a problem in any longitudinal study, and our drop-out rate is fairly typical for the study population. However, participants who drop out are typically less well than those who do not, and it is likely that those developing depression are more likely to drop out. The effect of drop-out is thus more likely to have weakened the association between white matter changes and depression than otherwise.

We agree that executive dysfunction is associated with depression. However, it is also associated with white matter changes,[1] and the purpose of the regression analysis was to investigate whether depression could be accounted for by factors other than white matter changes, rather than attempting to identify the best risk factors, and hence we used the Mini-Mental State Examination as a well-recognised measure of general cognitive ability.

When combined with the previous findings in our cohort[2] demonstrating that baseline white matter changes predict incident depression, we feel confident that, in this cohort at least, our findings robustly demonstrate that vascular disease as measured by white matter changes is a risk factor for depression.

Declaration of interest

J.T.O. is an editorial board member for *Psychological Medicine*, is Deputy Editor of *International Psychogeriatrics*. He has been a consultant for GE Healthcare, Servier and Bayer Healthcare, and has received honoraria for talks from Pfizer, GE Healthcare, Eisai, Shire, Lundbeck, Lilly and Novartis.

1 Debette S, Markus HS. The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and meta-analysis. *BMJ* 2010; **341**: c3666.

2 Teodorczuk A, Firbank MJ, Pantoni L, Poggesi A, Erkinjuntti T, Wallin A, et al. Relationship between baseline white matter changes and development of late life depressive symptoms: 3 year results from the LADIS study. *Psychol Med* 2010; **40**: 603–10.

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Corrections

Where are the hypotheses when you need them? *BJP*, **201**, 178–179. Reference 4 should read:
Wood S, Stride C, Threapleton K, Wearn E, Nolan F, Osborn D, et al. Demands, control, supportive relationships and well-being amongst British mental health workers. *Soc Psychiatry Psychiatr Epidemiol* 2011; **46**: 1055–68.

Association between maladaptive parenting and child self-control over time: cross-lagged study using a monozygotic twin difference design. *BJP*, **201**, 291–297. Figures 1 (p. 293) and 2 (p. 294): the outcome, top right of each figure, should read 'Emotional difficulties'.

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