Commentary: Activity each day keeps dementia away—does social interaction really preserve cognitive function?

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Glei et al.1 observe that reported social activities are inversely related to decline in Mental Status Questionnaire scores in a cohort of 2387 elderly Taiwanese followed-up for 11 years. This is consistent with other studies and extends the observational evidence to an East Asian, albeit industrialized, population. These observations are usually taken as support for the ‘use it or lose it’ hypothesis but could it really be that the social or intellectual function of playing ‘mahjong’ or ‘bingo’ is to defer cognitive decline?

This study illustrates well the difficulties of undertaking observational epidemiology in this area. First, there is marked attrition in any study of elderly people mainly owing to death. This may distort any reported association because of selective censoring. The authors note that the true association may have been stronger if censored subjects had been excluded. Second, those subjects with more social engagement are self-selecting, which allows the possibilities that residual confounding (the effect of an unmeasured factor) and/or reverse causality (incipient decline affecting the seeking of cognitive stimulation) may explain this association. In this study, the authors have tried to account for reverse causality by using a ‘lagged model’ thereby adjusting for performance at earlier rounds.

However, confounding remains a serious concern. A life course approach to cognitive function would postulate that both biological and psychosocial factors operating in early life may be important.2 Such factors may establish maximal cognitive function or cognitive reserve;3 this is analogous to the role of exercise on bone mass and subsequent osteoporosis. Individuals with greater cognitive capacity would not only be more likely to engage in social activities, but would be better able to compensate once pathology had begun, and hence sustain such activities as well as perform better on mental testing. While social activities remained an ‘independent’ predictor after adjustment for education and income in this study, the latter appear to be of greater predictive value and it is possible that the social activities variable is merely part of a latent pre-morbid cognitive capacity variable that includes educational attainment. As birth cohorts mature, we will be in a better position to design interventions that will be sufficient to unravel the mechanisms underlying this association. If one could identify what characteristics of engagement appeared important, we would be in a better position to design motivational interventions have used group therapy.6,7 There have been several studies of memory and learning-based interventions although the effects have been of short duration and have not transferred between cognitive domains.6 Neurobiological evidence is indirect, with transgenic mouse models of Alzheimer’s disease housed in more stimulating environments than standard laboratory cages showing reduced deposition of pathological beta-amyloid in their brains8 and a small number of imaging studies showing the effect of interventions on neural substrates in healthy human volunteers.9 Extrapolating these findings to elderly persons with pathology requires care. In contrast, genetic studies provide strong evidence that cognitive decline, at least in terms of non-familial Alzheimer’s disease, is strongly inherited using twin studies suggesting a heritability of between 60 and 80%.10 However, the possibility that the expression of genotype may be strongly related to environmental factors cannot be discounted. In conclusion, although the idea of ‘use it or lose it’ is appealing in its simplicity, the evidence for it affecting pathology remains weak and indirect.

The attractiveness of the ‘use it or lose it’ hypothesis, however, is that it ‘covers a multitude of sins’. Are we talking about preventing the onset of cognitive decline or retarding the progression of pathology after it has begun? Alternatively, is it a matter of optimizing biological potential through improving cognitive strategies? The little evidence that is available suggests a limited role for this model.6 Another possibility is that we are helping people to enjoy life’s evening through enhancing motivation and the improved cognitive performance this brings.

The study by Glei et al. is further evidence that a fatalistic approach to cognitive decline with age is unnecessarily pessimistic. However, future studies should try to unpack the social engagement ‘black box’ as generic measures of exposure are insufficient to unravel the mechanisms underlying this association. If one could identify what characteristics of engagement appeared important, we would be in a better position to design interventions for trial evaluation.

References


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