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B-vitamin status in relation to cognitive health over 4 years in healthy older adults

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Advancing age is associated with a decline in cognitive function which can range from mild cognitive impairment to dementia. Epidemiological evidence suggests that sub-optimal B-vitamin status may be associated with greater cognitive impairment in both healthy older adults and in patients with dementia[(1)]. In addition, evidence from randomised control trials has demonstrated a beneficial effect of B-vitamin supplementation on cognitive function[(2,3)] but the evidence is not entirely consistent[(4)]. The aim was to investigate B-vitamin status in relation to cognitive decline over a 4 year follow-up period in healthy older adults. We hypothesised that poorer B-vitamin status at baseline would lead to a greater than expected rate of cognitive decline.

In a retrospective study, healthy older adults ($n = 154$; aged 60–88years) who had been previously screened for cognitive function were reassessed four years after initial assessment. Cognitive function was assessed at both timepoints by the Mini-Mental State Examination (MMSE), the most widely used cognitive screening tools in a clinical setting. Participants were initially recruited as being cognitively healthy at baseline, i.e. having an MMSE score between 25–30.

At the 4 year follow-up assessment, when participants were aged 73·4 ± 7·1 years, mean cognitive MMSE scores had declined from 29·1 ± 1·3 to 27·5 ± 2·3 ($P < 0·001$). Although most participants showed a typical rate of cognitive decline expected for healthy older adults (i.e. a decrease of 0·2–0·6MMSE points per year)[(5)], cognitive decline occurred at an accelerated rate in a sub-set of participants (i.e. greater than 1 MMSE point per year; $n = 38$). Baseline predictors of accelerated cognitive decline were investigated (Table).

	Beta (β)	Odds Ratio	95% CI	*P*
Baseline Factors				
Age	0·12	1·12	(1·06–1·18)	<0·001
Gender	−0·21	0·81	(0·38–1·71)	0·580
BMI	0·01	1·00	(0·92–1·09)	0·976
Education	−0·49	0·61	(0·26–1·42)	0·254
Smoking	1·17	3·23	(0·62–16·72)	0·162
B-vitamin Biomarker Status				
Plasma Hcy (μmol/l; 12·8–25·4 *vs* 6·1–12·5)	−0·43	0·65	(0·27–1·57)	0·335
Red cell folate (nmol/l; 191–719 *vs* 726–2206)	0·67	1·95	(0·87–4·34)	0·104
Serum B12 (pmol/l; 118–231 *vs* 233–672)	0·02	1·02	(0·45–2·32)	0·971
Plasma PLP (nmol/l; 15·4–43·3 *vs* 43·9–198·4)	1·40	4·06	(1·80–9·23)	0·001

For each B-vitamin biomarker, lowest tertile (or highest tertileHcy) was compared with other two tertiles. Values were considered significant if $P \leq 0·05$.Abbreviations; CI: confidence interval; Hcy: homocysteine.

After adjustment for age, a low baseline concentration of vitamin B6, as measured using pyridoxal-5-phosphate (PLP;<43·3 nmol/l) was associated with a 4-fold higher risk of having accelerated cognitive decline. Neither folate nor vitamin B12 concentrations were significantly associated with cognitive decline. In conclusion, lower vitamin B6 status at baseline was strongly associated with an accelerated rate of cognitive decline over the 4 year period. Vitamin B6 may be an important (often overlooked) protective factor in maintaining cognitive function in ageing.

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