The Effect of Diet, Lifestyle and/or Cognitive Interventions in Mild Cognitive Impairment: a Systematic Review

Background

As our population grows older, the risk of developing cognitive disorders increases and is a major public health challenge. Mild Cognitive Impairment (MCI) is described as a transitional stage between the expected cognitive decline of normal ageing and that of dementia2 and is suggested to be the optimum time point for preventative intervention.3,4. Nutrition and cognitive decline has been examined in terms of a range of nutrients/dietary patterns, investigating the role that single nutrients, such as n-3 PUFA5 as well as whole diet interventions, such as the DASH diet6, a ketogenic diet7 or the Mediterranean diet8 may have. Therefore, a rationale exists to systematically review the dietary intervention studies in the literature among this patient group.

Methods

Inclusion Criteria

- Diagnosis of Mild Cognitive Impairment
- Community dwelling participants
- Randomised Controlled Trial

Exclusion Criteria

- Diagnosis of dementia or any other form of cognitive impairment other than MCI
- Participants with psychiatric problems e.g. depression or any significant medical comorbidity
- Individuals who are hospitalised, in a rehabilitation or long term care facility
- Pilot studies

Primary Outcome

- Validated cognitive measure (neuropsychological and/or cognitive test)
- Data on quality of life, adverse events and biomarker analysis (e.g. structural MRI or amyloid imaging) if available

Secondary Outcome

- Incident Dementia/Alzheimer’s Disease (AD)

Database search: Ovid MEDLINE, EMBASE, PsycINFO, Web of science and Scopus

Records identified = 2130

Records after duplicates removed = 1480

Titles and abstracts screened = 1480

Excluded = 1447

Full text articles screened = 33

Studies included in analysis = 12

Results

<table>
<thead>
<tr>
<th>Reference</th>
<th>Intervention</th>
<th>Cognitive outcome measured</th>
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</thead>
<tbody>
<tr>
<td>Ma (2016)</td>
<td>Folic Acid (400 μg) (n=180, 6 months)</td>
<td>Memory*, Visuospatial skills*</td>
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<tr>
<td>de Jager (2012)</td>
<td>0.8mg folate acid, 0.5mg vitamin B12, 20mg vitamin B6 (n=266, 2 years)</td>
<td>Memory*; Executive function*; Global cognition</td>
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<tr>
<td>Dekosky (2008)</td>
<td>Gingko Biloba (120-mg (n=482, 6.1 years)</td>
<td>Diagnosis of Dementia</td>
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<td>Lee (2013)</td>
<td>n-3 fatty acids (430 mg of DHA and 150 mg of EPA) (n=36, 12 months)</td>
<td>Memory*; Executive Function; Attention; Visuospatial Skills; Psychomotor speed; Global cognitive function</td>
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<tr>
<td>Petersen (2005)</td>
<td>Vitamin E (2000 IU) (n=769, 3 years)</td>
<td>Development Alzheimer’s disease; Memory; Executive Function; Language; Visuospatial skills; Overall Cognitive Function</td>
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<tr>
<td>Krikorian (2010)</td>
<td>Chromium Picolinate (1000 mcg) (n=26, 12 weeks)</td>
<td>Memory; IMR*</td>
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<tr>
<td>Desideri (2012)</td>
<td>Cocoa Flavanols (990mg/520mg/45mg) (n=90, 8 weeks)</td>
<td>MMSE; Trail making test, Part A and B*; Verbal fluency test*</td>
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<tr>
<td>Krikorian (2010)</td>
<td>Concord grape juice (n=12, 12 weeks)</td>
<td>Memory* (verbal learning only)</td>
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<tr>
<td>Krikorian (2010)</td>
<td>Wild blueberry juice (n=9, 12 weeks)</td>
<td>Memory* (V-PAL test only)</td>
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<tr>
<td>Horie (2016)</td>
<td>Nutritional counselling (healthy eating/calorie restriction) (n=80, 12 months)</td>
<td>Memory; Executive Function; Language; Psychomotor Speed</td>
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<tr>
<td>Bayer-Carter (2011)</td>
<td>High–saturated fat/low–GI Vs low–saturated fat/low–GI diet (n=49, 4 weeks)</td>
<td>Memory; Executive Function; Motor Speed; AD Biomarkers* (CSF Aβ42 only)</td>
</tr>
<tr>
<td>Krikorian (2012)</td>
<td>High carbohydrate Vs very low carbohydrate diet (n=23, 6 weeks)</td>
<td>Memory</td>
</tr>
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</table>

Discussion and Conclusion

Diet supplementation, with either Vitamin E10 or Ginkgo Biloba12, had no statistically significant effect on progression from MCI to dementia and/or AD. There was heterogeneity in the results for cognitive function. Some studies showed improvements in a few of the cognitive tests used but not all, with some of the improvements observed not being maintained until intervention completion. The studies which investigated B vitamins and folic acid10,11 and cocoa flavanols16 showed the most consistent results in terms of cognition.

The mixed evidence may be explained by the heterogeneity of studies included, on the basis of:

- the variation in cognitive outcome measures used
- differences in the diet intervention type (supplements vs single food products vs dietary patterns)
- variations in sample size and duration of intervention
- the small number of dietary intervention studies conducted

These factors make it difficult to provide conclusive evidence to support the effect of diet on cognitive outcomes. Nonetheless, the review highlights the need for well-designed, robust RCTs to further explore the role of diet in cognitive decline.