Nutrient intakes and the common mental disorders in women

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ABSTRACT

Background: There is an increasing recognition of the role of nutrition in depression and anxiety. Magnesium, folate and zinc have all been implicated in depressive illness, however there are few data on these nutrients in anxiety disorders and the data from population-studies are limited.

Aims: In a large, randomly-selected, population-based sample of women, this study aimed to examine the relationship between the dietary intakes of these three micronutrients and clinically determined depressive and anxiety disorders and symptoms.

Methods: Nutrient intakes were determined using a validated food frequency questionnaire. The General Health Questionnaire-12 measured psychological symptoms, and a clinical interview (Structured Clinical Interview for DSM-IV-TR, non-patient edition) assessed current depressive and anxiety disorders.

Results: After adjustments for energy intake, each standard deviation increase in the intake of zinc, magnesium and folate was associated with reduced odds ratio (OR) for major depression/dysthymia (zinc: OR=0.52, 95% confidence interval (CI) 0.31 to 0.88; magnesium: OR=0.60, 95% CI 0.37 to 0.96; folate: OR=0.66, 95% CI 0.45 to 0.97). There was also an inverse association between the intake of magnesium and zinc and GHQ-12 scores (zinc: zβ=-0.16, 95% CI −0.29 to −0.04; magnesium: zβ=-0.14, 95% CI −0.26 to −0.03). These relationships were not confounded by age, socioeconomic status, education or other health behaviours. There was no relationship observed between any nutrient and anxiety disorders.

Conclusion: These results demonstrate an association between the dietary intakes of magnesium, folate and zinc and depressive illnesses, although reverse causality and/or confounding cannot be ruled out as explanations.

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1. Introduction

In recent years, there has been an increasing interest in the role of nutrition in depression. Apart from the extensive investigations focused on long-chain omega-3 fatty acids (for review see Parker et al., 2006), three micronutrients in particular have been examined in depression: zinc, magnesium and folate. Zinc is a trace mineral involved in modulating the function of NMDA receptors (Takeda, 2000), which in turn play a critical role in synaptic plasticity in areas of the brain salient to depressive illness, such as the hippocampus and amygdala (Nestler et al., 2002). In animal models of depression, zinc treatment produces antidepressant-like effects (Krocza et al., 2000, 2001; Nowak et al., 2003b; Rosa et al., 2003) and enhances the efficacy of common antidepressant medications (Krocza et al., 2001). In humans, decreased serum zinc is described in individuals with MDD
while serum zinc levels are negatively correlated with illness severity (Maes et al., 1994) and treatment resistance (Maes et al., 1997b). Moreover, zinc supplementation has been shown to enhance the efficacy of antidepressant therapy (Nowak et al., 2003a), while two very recent studies have shown inverse relationships between the dietary intake of zinc and self-reported depression in pregnant women (Roy et al., 2011) and female students (Amani et al., 2011).

Magnesium is another micronutrient thought to play role in depression. A magnesium-deficient diet has been shown to increase depression- and anxiety-related behaviour in mice (Singewald et al., 2004), while magnesium treatment appears to improve such behaviours in animal models (Poleszak et al., 2004, 2005). In humans, Jacka et al. (2009) have reported an inverse relationship between dietary magnesium intake and self-reported depression, but not anxiety, in a large sample of community-dwelling men and women in Norway. However, to date there have been no comprehensive studies investigating the link between the dietary intake of either zinc or magnesium and clinically-determined depressive and anxiety disorders in humans.

Finally, folate deficiencies have been associated with depression in a range of clinical and epidemiological studies. Symptoms of depression such as apathy, fatigue, insomnia, irritability and impaired concentration, are commonly seen in various states associated with deficiencies of folate and the results of more than 20 clinical studies have indicated that serum folate deficiency affects as many as one-third of psychiatric patients (Alpert et al., 2000). Serum folate levels are related to treatment response in major depression (Fava et al., 1997; Alpert et al., 2000). Serum folate levels are related to treatment response in major depression (Fava et al., 1997; Alpert et al., 2000). Serum folate levels are related to treatment response in major depression (Fava et al., 1997; Alpert et al., 2000). Serum folate levels are related to treatment response in major depression (Fava et al., 1997; Alpert et al., 2000). Serum folate levels are related to treatment response in major depression (Fava et al., 1997; Alpert et al., 2000).

In this study, we aimed to priori to examine the dietary intake of those three micronutrients that are supported by extant evidence and the common mental disorders and symptoms in a large, randomly-selected, population-based sample of adult women. Our hypothesis was that women with lower dietary intakes of each of these micronutrients would be more likely to have clinical depressive and anxiety disorders and higher levels of psychiatric symptomatology.

2. Methods

2.1. Participants

The Geelong Osteoporosis Study (GOS) is an ongoing epidemiological study based in south-eastern Australia. An age-stratified, randomly-selected, population-based sample of 1494 women aged 20–94 years was recruited between 1994 and 1997, with a participation of 77.1% (Pasco et al., in press). These women have continued to return for biennial follow-up assessments that include comprehensive measures of lifestyle, medical and family history, medication use, as well as anthropometric, body composition and bone fragility measures. At the ten-year GOS assessments, a psychiatric component was introduced to the study. Of the 1127 women who participated in the GOS 10-year follow-up (Pasco et al., in press), participants for whom psychiatric or dietary data were not available at the time of the study (n = 81) were excluded from the analyses, resulting in a sample of 1046 women aged 20–93 years available for analysis. The Barwon Health Human Research Ethics Committee approved the study, and written informed consent was obtained from all participants.

2.2. Dietary assessments

Habitual diet was assessed using the Cancer Council Victoria dietary questionnaire (Giles and Ireland, 1996). This is a comprehensive, computer-read, food frequency questionnaire (FFQ), validated for assessing habitual dietary intake in the Australian population (Hodge et al., 2000). This questionnaire reports participant’s usual consumption of 74 foods and six alcoholic beverages over the preceding 12 months using a 10-point frequency scale. Nutrient intakes, including energy, were computed from the dietary data using NUTTAB95 nutrient composition data.

2.3. Psychiatric assessments

The Structured Clinical Interview for DSM-IV-TR Research Version, Non-patient edition (SCID-I/NP) (First et al., 2002) was the primary diagnostic instrument for the common mental disorders. This is the gold-standard assessment tool comprising a validated, semi-structured clinical interview for the major axis one psychiatric disorders in DSM-IV-TR. Diagnoses of current major depressive disorder, dysthymia and anxiety disorders were our outcomes of interest, as they comprise the high prevalence mental disorders. Researchers trained in psychology and administration of the SCID-I/NP conducted these interviews. Psychiatric symptoms were measured with the self-reported General Health Questionnaire (GHQ-12) (Goldberg and Hillier, 1979).

2.4. Covariates

Socioeconomic status was determined using Socioeconomic Index For Areas (SEIFA) index scores based on the 2006 Australian Bureau of Statistics census data. It was decided a priori to use the Index of Relative Socioeconomic Advantage and Disadvantage, which accounts for high and low income and the type of occupation from unskilled employment to professional positions. A low score on this index identifies the most disadvantaged (quintile 1), and a high score identifies the most advantaged (quintile 5). Information regarding the highest educational level attained was derived from baseline assessments, and comprised four categories, from primary schooling only (1), to (4) university or other tertiary qualification. Physical activity was assessed by self-report questionnaire and ranged from (1) chair or bedridden, or limited activity throughout the home, through to (4) very active. Alcohol consumption in grams per day was ascertained from the dietary questionnaire and was subsequently categorised according to 2001 recommendations for women ((NHMRC), 2001) as the following: zero consumption (1), 1–14 standard drinks per week (2), 15–29 standard drinks per week (3), and ≥30 standard drinks per week (4).
and 15 or more standard drinks per week (3). Current cigarette smoking, yes/no, was self-reported. Height was measured to the nearest 0.1 cm and body weight was measured to the nearest 0.1 kg. Body mass index (BMI) was calculated from these measurements as weight/height$^2$ (kg/m$^2$). Although nutritional supplement use was self-reported by participants, the data were of insufficient detail or quality to adequately determine intakes of the investigated nutrients. However, supplement use (yes/no) was also tested as a potential confounder, as those taking nutrient supplements were more likely to have a common mental disorder ($p = 0.02$).

2.5. Statistical analyses

In calculating the intakes of individual nutrients such as zinc, nutrients in alcohol were added to those calculated from food intake in the FFQ to calculate a total intake amount for each nutrient per day. All nutrient and GHQ-12 scores were positively skewed and were normalised using a natural log transformation. All exposure variables and GHQ-12 scores were subsequently standardised using z-scores and expressed as standard deviation units.

Multivariate linear regression analyses were used to assess the relationship of nutrient intakes as exposures and GHQ-12 scores as outcomes. Logistic regression models were also developed to estimate odds ratios with 95% confidence intervals using current major depressive disorder/dysthymia and anxiety disorders as the outcomes of interest. For each analysis, the following covariates were entered sequentially in order to test their contribution to the mental health outcomes: age, socioeconomic factors (SEIFA scores and education), health behaviours (physical activity, alcohol consumption and smoking); and energy intake. BMI and supplement use were also tested in the models as explanatory variables. These models took into account potential non-linear associations and interactions.

There were no missing data on dietary variables. There were missing data for GHQ-12 scores ($n = 23$) and listwise exclusions were used when GHQ-12 was the outcome variable, resulting in a sample of 1000 women for these analyses. There were 24 participants with missing data on BMI. There were no missing data on any other variables.

3. Results

Table 1 presents characteristics of the study participants with and without depressive and anxiety disorders. Comparisons revealed no evident differences between those with and without these disorders on any of the included variables.

Median intakes and interquartile ranges of nutrients were as follows: zinc (9.6 mg/day, IQR 7.5 to 12.2); magnesium (240.9 mg/day, IQR 189.5 to 304.4); and folate (261.6 μg/day, IQR 322.8 to 210.7). While 70% of women in the sample met or exceeded the recommended daily intake (RDI) for zinc (8 mg/day), only 26% of women met or exceeded the RDI of magnesium (320 mg/day), and 8% met or exceeded the RDI for folate (400 μg/day). The correlations between nutrients were high (all $r > 0.7$, $p < 0.001$), precluding the inclusion of all nutrients in the same statistical models.

Table 2 presents the results of linear regression analyses with nutrient intakes as exposures and GHQ-12 scores as the outcome variable of interest. After adjustment for all covariates, the intake of both zinc and magnesium was inversely related to GHQ-12 scores. Each standard deviation increase in zinc and magnesium was associated with an approximate 0.15 standard deviation decrease in GHQ-12 scores. There tended to be an inverse relationship between folate intake and GHQ-12 scores.

Table 3 presents the results of logistic regression analyses. Age, socioeconomic status, health behaviours and BMI did not confound the relationships examined. The only identified confounder was energy intake, and all figures cited are concordantly adjusted. For each standard deviation increase in nutrient intakes, the likelihood of current depressive disorders was reduced by between a third and a half. The relationship between nutrient intakes and anxiety disorders was not statistically significant (all $p > 0.05$).

4. Discussion

Hypotheses regarding the association between the nutrients magnesium, folate and zinc and mental health were largely supported by the data, with the intake of each of these nutrients from food associated with reduced psychiatric symptomatology and the likelihood of depressive disorders. However, the relationship between nutrient intakes and anxiety disorders, while in the same direction, was not significant.

4.1. Magnesium

The inverse relationship between magnesium intake and GHQ-12 scores was in the same direction, and of almost identical magnitude to that previously demonstrated in a large community-based study undertaken in Norway (Jacka et al., 2009). In that study of nearly 6000 adults, after adjustments for a wide range of lifestyle, socioeconomic and medical variables, each one standard deviation decrease in magnesium intake was associated with a 0.11 standard deviation increase in depression score on the Hospital Anxiety and Depression Scale. In that study of nearly 6000 adults, after adjustments for a wide range of lifestyle, socioeconomic and medical variables, each one standard deviation decrease in magnesium intake was associated with a 0.11 standard deviation increase in depression score on the Hospital Anxiety and Depression Scale. Also in common with the Norwegian study (Jacka et al., 2009) was the relationship of nutrient intakes to anxiety disorders, wherein the associations were in the same direction as to depression but failed to reach statistical significance. It is unclear as to why the relationship between nutrient intakes and anxiety disorders may be less apparent. One explanation is that biological pathways that underpin depression and anxiety differ, with differential mechanisms influenced by the presence or absence of nutrients. Another explanation is that of reverse causality, wherein the influence of depression on dietary habits is different to that of anxiety.

We have previously proposed that one pathway by which magnesium may be linked to depression is via its contribution to immune system functioning (Jacka et al., 2009). Systemic inflammation and cell-mediated immune activation are prominent features of major depression (Maes, 2011). Magnesium has strong anti-inflammatory effects (Almoznino-Sarafian et al., 2007) and studies in animal models have shown that induced magnesium deficiency in rats results in a systemic pro-inflammatory/pro-oxidant state (Weglicki et al., 1992). In humans, large community studies such as the National Health and Nutrition Survey (NHANES) in the US have reported an inverse relationship between magnesium intake and systemic
inflammation (King et al., 2005; Song et al., 2005). Pro-inflammatory states are also implicated in a range of health conditions comorbid with depression, such as cardiovascular disease, osteoporosis, obesity and the metabolic syndrome (Shoelson et al., 2007; Sutherland et al., 2004), and the low intakes of magnesium reported in our study may have implications for these diseases in addition to depression.

4.2. Folate

The relationship between folate intake and the mental health measures was less prominent than that of magnesium and zinc, with only a trend towards an association between dietary folate and reduced GHQ-12 scores. It is may be that this is a result of the consistently very low levels of intake in the sample obscuring this relationship. However, the relationship between higher folate intake and a lower likelihood for major depression/dysthymia is in accordance with previous community-based studies showing a relationship between folate intake and the likelihood of depression in cross-sectional studies (Murakami et al., 2008; Tolmunen et al., 2003), and over time (Tolmunen et al., 2004a). For example, in a study of 2313 middle-aged Finnish men, intakes of folate were measured at baseline and discharge diagnoses of clinical depression over approximately 10 years were ascertained from national registries. After controlling for numerous covariates, including depressive symptoms at baseline, the dietary

Table 1
Characteristics of study sample: comparisons between those with and without depressive/anxiety disorders.

<table>
<thead>
<tr>
<th></th>
<th>No (n=905)</th>
<th>IQR</th>
<th>Yes (n=118)</th>
<th>IQR</th>
<th>p-Value</th>
</tr>
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<td>34–67</td>
<td>49</td>
<td>34–60</td>
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<td>BMI (kg/m²)</td>
<td>26</td>
<td>23–31</td>
<td>27</td>
<td>24–31</td>
<td>0.64</td>
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<tr>
<td>Energy (kJ/day)</td>
<td>6561</td>
<td>5208–8055</td>
<td>6880</td>
<td>5562–8489</td>
<td>0.13</td>
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<tr>
<td>Socioeconomic status</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (low)</td>
<td></td>
<td>%</td>
<td></td>
<td>%</td>
<td>0.38</td>
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<tr>
<td>2</td>
<td></td>
<td>140</td>
<td>16</td>
<td>20</td>
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<td>3</td>
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<td>197</td>
<td>22</td>
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<td>Education</td>
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<td>4</td>
<td>7</td>
<td>6</td>
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<tr>
<td>Some secondary</td>
<td>380</td>
<td>42</td>
<td>48</td>
<td>41</td>
<td></td>
</tr>
<tr>
<td>Completed secondary</td>
<td></td>
<td>224</td>
<td>25</td>
<td>27</td>
<td></td>
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<tr>
<td>Post-secondary</td>
<td>265</td>
<td>29</td>
<td>36</td>
<td>30</td>
<td></td>
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<tr>
<td>Physical activity</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chair or bedridden/limited activity</td>
<td>46</td>
<td>5</td>
<td>7</td>
<td>6</td>
<td>0.48</td>
</tr>
<tr>
<td>Sedentary</td>
<td>144</td>
<td>16</td>
<td>24</td>
<td>20</td>
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<td>Active</td>
<td>477</td>
<td>53</td>
<td>62</td>
<td>53</td>
<td></td>
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<tr>
<td>Very active</td>
<td>238</td>
<td>26</td>
<td>25</td>
<td>21</td>
<td></td>
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<tr>
<td>Current smoker</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>116</td>
<td>13</td>
<td>23</td>
<td>20</td>
<td>0.05</td>
</tr>
<tr>
<td>No</td>
<td>789</td>
<td>87</td>
<td>95</td>
<td>80</td>
<td></td>
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<tr>
<td>Alcohol consumption</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zero consumption</td>
<td>173</td>
<td>19</td>
<td>24</td>
<td>20</td>
<td>0.89</td>
</tr>
<tr>
<td>1–14 standard drinks per week</td>
<td>601</td>
<td>66</td>
<td>79</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td>15 or more standard drinks per week</td>
<td>131</td>
<td>15</td>
<td>15</td>
<td>13</td>
<td></td>
</tr>
</tbody>
</table>

Table 2
Linear regression analyses examining food-derived nutrient intakes and GHQ-12 scores (all transformed and z-score standardised, to be interpreted as standard deviations)a.

<table>
<thead>
<tr>
<th>GHQ-12 scores</th>
<th>β</th>
<th>95% CI</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc</td>
<td>−0.16</td>
<td>−0.29 to −0.04</td>
<td>0.01</td>
</tr>
<tr>
<td>Magnesium</td>
<td>−0.14</td>
<td>−0.26 to −0.03</td>
<td>0.02</td>
</tr>
<tr>
<td>Folate</td>
<td>−0.09</td>
<td>−0.18 to 0.01</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Table 3
Logistic regression analyses examining food-derived nutrient intakes and categorical depressive and anxiety disorders (odds ratios and 95% confidence intervals)a.

<table>
<thead>
<tr>
<th></th>
<th>MDD/Dysthymia (n=60)</th>
<th>Anxiety Disorders (n=80)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR 95% CI p-value</td>
<td>OR 95% CI p-value</td>
</tr>
<tr>
<td>Zinc</td>
<td>0.52 0.31 to 0.88 0.01</td>
<td>0.76 0.48 to 1.19 0.23</td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.60 0.37 to 0.96 0.03</td>
<td>0.81 0.54 to 1.23 0.32</td>
</tr>
<tr>
<td>Folate</td>
<td>0.66 0.45 to 0.97 0.03</td>
<td>0.81 0.58 to 1.31 0.21</td>
</tr>
</tbody>
</table>

a Adjusted for age, SES, education, PA, alcohol consumption, smoking and energy intake.
consumption of folate below the median was associated with a 2.5-fold increased risk of MDD after all adjustments (Tolmunen et al., 2004a). In the Finnish sample, the average intakes of folate were below the recommended daily intake and this is concordant with our study, wherein the large majority of women consumed less than the RDI of folate.

It is thought that disturbed one-carbon metabolism, which is a consequence of folate deficiency, may be an important factor in depression (Coppen and Bolander-Gouaille, 2005). As methylfolate is required for the synthesis of methionine from homocysteine, folate deficiencies result in increased plasma levels of homocysteine (Tolmunen et al., 2003). Increased total plasma homocysteine is a marker of allostatic load, and high levels have been repeatedly shown in individuals with depression (Bjelland et al., 2003; Bottiglieri et al., 2000; Tolmunen et al., 2004b). Moreover, methionine is a precursor of S-adenosylmethionine (SAM), which is essential for numerous methylation processes in the brain involving monoamines, neurotransmitters and membrane phospholipids (Bottiglieri, 2005). Folate also interacts with inflammatory processes, with folate supplementation demonstrating anti-inflammatory effects in humans (Solini et al., 2006).

4.3. Zinc

Of the nutrients studied, it was zinc intake that demonstrated the strongest relationship to both psychological symptoms and depressive disorders. These results complement previous studies showing a significant reduction in serum zinc levels in patients with MDD compared to controls. For example, Maes et al. (1994) examined levels of zinc in the serum of 48 patients with differing degrees of depression: minor depression, MDD without melancholia and MDD with melancholia, compared to 32 healthy controls. The authors reported that serum zinc was decreased in these patients in a dose–response manner, wherein those with MDD had the lowest levels of zinc (approximately 12% lower than controls) and those with minor depression had intermediate values compared to controls. There was also an inverse relationship between depression severity and zinc levels. Importantly, these decreases were not attributable to either depression-associated anorexia, or to HPA axis hyperactivity, as measured using the dexamethasone test. This finding was replicated in two other studies of patients with MDD, wherein those with MDD had lower levels of serum zinc compared to healthy controls (Maes et al., 1997b, 1999b). In these studies, no relationship between zinc status and either severity or duration of illness, depression-related anorexia, or cortisol levels after dexamethasone challenge (Maes et al., 1997b, 1999b) was observed. However, there were significant associations between serum zinc and markers of systemic immune system activation, including inverse correlations with plasma interleukin-6, serum neopterin and the CD4/CD8 T cell ratio (Maes et al., 1994, 1997a, b) and positive correlations with serum albumin and transferrin, two negative acute phase proteins (Maes et al., 1997b). Therefore, the authors proposed that the observed decreases in serum zinc were secondary to the inflammatory response in depressive illness. In these studies, habitual diet was not measured, and it is not possible to rule out differences in dietary intake as a factor in these findings.

This study is the first to examine the dietary consumption of zinc and its relationship to clinically determined depressive and anxiety disorders and the results support a role for dietary zinc in depressive illnesses, although not in anxiety disorders. There are many reasons to consider zinc as an important component of habitual diet in regards to depressive illness. As well as playing a role in modulating the function of NMDA receptors (Takeda, 2000), and in the stabilisation of cellular membranes (Maes et al., 1997b), adequate zinc is required for the functioning of cells involved in mediating immunity, such as neutrophils and Natural Killer (NK) cells, while zinc deficiency affects cytokine production and the functioning of macrophages and T and B cells (Prasad, 2008). Chronic zinc deficiency also activates the HPA axis, leading to the chronic overproduction of cortisol (Fraker and King, 2004). Moreover, zinc is known to possess anti-inflammatory properties, down-regulating the gene expression of pro-inflammatory cytokines such as TNF-α and IL-1β (Prasad et al., 2004). Zinc is also an essential factor in polyunsaturated fatty acid (PUFA) metabolism, e.g. zinc is required for the activity of desaturase enzymes, which are responsible for the conversion of essential fatty acids to PUFAs (Russo et al., 1997). In depression, lower serum zinc is associated with reduced levels of EPA and DHA, suggesting that reduced zinc may contribute to the depletion of n-3 PUFAs commonly observed in depression (Maes et al., 1999a). Another important aspect of zinc is the role it plays as a neuro-protector. Zinc is a potent antioxidant as it protects cellular membranes from lipid peroxidation (Maes et al., 1997b); induces the production of metallothionein, which has the ability to scavenge hydroxide — a component of reactive oxygen species (ROS) (Prasad et al., 2004); and protects against DNA damage in humans (Prasad et al., 2004). Zinc also upregulates the expression of the brain derived neurotrophic factor (BDNF) gene in the cortex and hippocampus (Nowak et al., 2005). BDNF is a neuroprotective agent — protecting neurons from oxidative stress and promoting neurogenesis (Duman et al., 1997). In acting as an anti-inflammatory and neuroprotective agent, zinc may thus play a role in depressive illness. In this study, 30% of women consumed less than the RDI of zinc, which may have implications for a range of disease outcomes in addition to that of mental health.

4.4. Limitations

This study is limited by a cross-sectional design, which does not afford a determination of the direction of the relationship between dietary nutrient intakes and mental health. Reverse causality, wherein dietary intakes are a consequence of mental ill health, is another potential explanation for our findings, and there may also be differences in the reporting of diet between those with and without depressive and anxiety disorders. Reverse causality has been examined in studies examining the link between mental health and diet, and the available, although limited, data do not support this explanation (Akbaraly et al., 2009; Jacka et al., 2011; Sanchez-Villegas et al., 2009). Although we adjusted statistically for other potential confounding factors, such as health behaviours and socioeconomic status, we may not have fully captured relevant aspects of those factors. Furthermore, there may be other, unidentified confounding variables that we have not accounted.

for. We could not determine the intake of nutrients from supplements, although these may not be nutritionally equivalent. Finally, it may be the case that these nutrients are simply markers of dietary quality. Our previous study identified a dietary pattern ("Traditional"), comprising vegetables, fruits, lean red meats, fish and wholegrains, as being inversely associated with these mental health outcomes (Jacka et al., 2010), and each of these nutrients were moderately to highly correlated with this dietary pattern and each other, precluding the inclusion of each in the same statistical models (McGee et al., 1984) (data not shown). Strengths of the study include the use of gold-standard assessment tools for both nutritional intakes and psychiatric outcomes, the large sample size, the inclusion of a range of potential confounding variables, and a randomly-selected sample of women across the adult age range.

5. Conclusion

The results of this study support the contention that the consumption of nutrient-dense foods plays a role in depression, but not anxiety. Magnesium and folate are prominent components of foods recommended as part of the Australian National Healthy Eating guidelines (NHMRC, 2003), such as leafy green vegetables, nuts, legumes and wholegrains, while zinc is found in lean meats and seafoods, which are also important components of a healthy diet. Of concern are the very low intakes of magnesium and folate recorded in women in our study. This is in line with findings from the 2008 Victorian Population Health Survey, wherein only 10.5% of women met the recommended five or more serves of vegetables per day (Department of Human Services, 2008). Promoting an increased consumption of nutrient-dense foods may be a useful strategy in the prevention and treatment of depression in the community.

Conflict of interest

All authors have no conflict to declare.

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