A comparison of the nutrient intake of a community-dwelling first episode psychosis cohort, aged 19-64, with data from the United Kingdom population

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A comparison of the nutrient intake of a community-dwelling first-episode psychosis cohort, aged 19–64 years, with data from the UK population

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Abstract

Psychosis increases the risk of CVD, obesity and type 2 diabetes and reduces life expectancy. There are limited data comparing the dietary habits of community-dwelling first-episode psychosis sufferers — with autonomy over diet — and the general population. The data represent the retrospective evaluation of nutritional data collected between 2007 and 2013 from 143 individuals from the UK population receiving treatment for first-episode psychosis. Differences in mean nutrient intakes between the study cohort and the national sample were tested for statistical significance using independent t tests, incorporating Satterthwaite’s correction where required. Mean total energy intake was lower for males (P = 0.049) and higher for females (P = 0.016) in the cohort than in the corresponding subgroups of the national sample. Females in the study cohort consumed 12.9 (95% CI 4.3, 21.5) g more total fat per d, whilst males consumed 7.7 (95% CI 0.5, 14.9) g less protein per d than the national sample. Males in the study also showed significantly lower mean intakes than nationally of folate, Fe, Se, vitamin D and Zn, but not vitamin C. The proportion of individuals not meeting the lower reference nutrient intakes, particularly for Se (males 54.0% and females 57.1%) and for Fe amongst females (29.6%), is cause for concern regarding potentially severe deficiencies. Further exploration of dietary habits within first-episode psychosis is warranted to assess whether individuals make beneficial dietary changes for their physical and mental health and wellbeing following dietary change intervention. It would also be pertinent to assess any correlation between diet and mental health symptomology.

Key words: First-episode psychosis: Dietary assessment: Micronutrient deficiencies: Metabolic syndrome

Severe mental illness, such as schizophrenia, increases the risk of CVD, obesity and type 2 diabetes and reduces life expectancy[1–7]. Schizophrenia is a condition characterised by episodes of psychosis, hallmarked by an alteration of perception, thoughts, mood or behaviour[8]. There are several key factors potentially affecting the food consumption patterns in those affected, including socio-economic status[9,10], an illness-induced lack of motivation and a sedentary lifestyle compared with the general population[8,11–13].

A recent systematic review reporting thirty-one published studies on dietary patterns in schizophrenia cited only four which related to first-episode psychosis[14]. Whilst these[15–18] – all case–control studies – provided useful information, it is unclear whether the study groups reported had control over their own dietary choices. Autonomy over dietary choice is important because acutely psychotic individuals in the community may fail to eat meals as a consequence of the chaos of psychotic symptoms[19], and could help determine which aspects of the diet would benefit from dietary change intervention. There are limited data comparing the dietary habits of community-dwelling first-episode psychosis sufferers with the general population or with an optimum
nutrient intake. One study[^18^], for example, assessed dietary intake through recording food frequency over the previous 1 year as part of the Health and Lifestyle 2 (HAL2) questionnaire. This assessed habitual frequency, may inaccurately reflect current intake, in light of recent mental health problems, particularly if the individual has a short duration of psychosis.

Antipsychotic medication, which whilst improving sympto-
mology, is known to cause carbohydrate craving[^20^,^21^] and hyperphagia in individuals[^22^–^27^]. Animal models demonstrate that abdominal fat deposition can occur whilst undergoing treatment with antipsychotic medication, irrespective of a corres-
ponding change in energy intake[^28^]. This effect, found to be reversible following the cessation of the pharmacological agent[^29^], emphasises the need for robust nutritional assessment and intervention in this population.

Not only are macronutrients of significance in severe men-
tal illness, micronutrients are also essential cofactors in men-
tal function, via a potential range of mechanisms, including: deficiency methylation, impaired mitochondrial function, alteration of gene expression and the impaired growth and development of neurons[^30^]. Folate is an integral component of methylation processes, notably the formation of neurotrans-
mitters controlling mood[^31^,^32^]. Logically, therefore an adequate folate intake must be advocated in individuals with severe mental illness. Likewise, there is emerging evidence for vitamin D as a functioning neurosteroid[^32^], implicating it in the development and general function of brain homeostasis. Evidence indicates that vitamin D helps reduce Ca[^2] levels in the brain, inhibits glutathione metabolism and protects against reactive oxygen and nitrogen species[^33^]. Additionally, vitamin D has been specifically associated with psychotic disorders. Individuals with schizophrenia were found to have significantly lower plasma levels of vitamin D[^34^,^35^]. Whilst some consider that this could be linked to dietary habits[^34^], others, however, found that the diets were not lacking in Ca-rich dairy foods[^35^]. In addition to these studies conducted in adult cohorts, there is evidence of vitamin D deficiency associated with the prevalence of psychotic features in adolescents[^36^,^37^]. A large proportion of vitamin D is sourced from sunlight and this could well be lacking in those with psychosis through limited outdoor exposure brought about from the social isolation and seden-
tary lifestyle often associated with psychosis[^38^]. It is important thus to ensure that a baseline dietary intake is established and then education around optimal dietary intake be provided, where warranted.

Antioxidant systems are especially important for those with psychosis. There is evidence that oxidative damage to neurons has been associated with, and may therefore contribute to, the pathophysiology of schizophrenia[^39^–^41^]. Micronutrients are involved in the production of several of the body’s antioxidant systems. The importance of Fe, Cu and Mn has been cited as transition metals that can cause free radical damage[^42^]. Se is an essential component of the glutathione peroxidase group[^43^]. An alteration from physiologically optimal Mn, Cu, Zn, Se and Fe levels may play a role in the development of schizophrenia and that dietary supplementation or dietary improvement may be effective through the corresponding increase in antioxidant activity[^44^].

The aim of the present paper was to begin to address the paucity of nutritional information and present population comparative data for key macro- and micronutrients from community-dwelling individuals with first-episode psychosis from the UK population who are responsible for their own diets.

**Materials and methods**

**Study design and population**

The primary data described represent the retrospective evalua-
tion of nutritional data collected from individuals receiving treatment for first-episode psychosis. These subjects are part of the UK population, have a diagnosis of first-episode psych-
osis and are undergoing treatment from the National Health Service (NHS). No individuals displaying acute symptoms of psychosis, as determined by a health professional, were offered a nutritional assessment as this was not a health care priority for them at that time. Individuals in the psychosis study series, to be comparable with the national sample, were all community-dwelling (not hospital residents) and were either directly in control of their food purchases, or household foods were purchased via a partner or relative, at the time of the survey.

The cohort under study (n=143) comprises eighty-seven males and fifty-six females, who were asked by a health professional (not the nutritionist) from the Early Intervention in Psychosis service if they would like a nutritional assessment. All cases thus consented to, and completed, the nutritional assessment voluntarily as part of their treatment within the Early Intervention Service. The subjects ranged in age from 19 to 64 years. The comparative data described herein are published data collected from a sample of the UK general popu-
lation by the Department of Health’s Food Standards Agency (The National Diet and Nutrition Survey 2008–2011). Subject details of this comparative study series have been described elsewhere[^45^], though in summary: full datasets were compiled on nutrient intake from 519 male and 667 female participants aged 19–64 years, that consensually opted to take part in the National Study.

**Ethics**

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving patients were approved by the NHS host institution’s Research Governance Department. (This study did not require ethical approval.) Verbal informed consent was obtained from all patients relating to their participation in a nutritional assess-
ment for the purposes of their healthcare needs. Verbal con-
sent was witnessed and formally recorded in their patient notes. The dataset was fully anonymised before statistical ana-
lysis was conducted. Individuals in the psychosis study series were informed throughout the process that they could stop participation in the nutritional assessment at any time, without
Results

Table 1. Summary statistics for daily energy and macronutrient intakes

<table>
<thead>
<tr>
<th></th>
<th>Sample Mean Energy (kJ/d)</th>
<th>Sample Median Energy (kJ/d)</th>
<th>Sample SD Energy (kJ/d)</th>
<th>National Mean Energy (kJ/d)</th>
<th>National Median Energy (kJ/d)</th>
<th>National SD Energy (kJ/d)</th>
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<tbody>
<tr>
<td>All</td>
<td>7962.2</td>
<td>7690.2</td>
<td>224</td>
<td>7421.8</td>
<td>7112.9</td>
<td>199</td>
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<tr>
<td>Males</td>
<td>8359.6</td>
<td>8270.5</td>
<td>208</td>
<td>7843.4</td>
<td>7494.9</td>
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<tr>
<td>Females</td>
<td>7342.9</td>
<td>7222.5</td>
<td>191</td>
<td>6952.1</td>
<td>6642.3</td>
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<table>
<thead>
<tr>
<th></th>
<th>Sample Mean Total fat (g/d)</th>
<th>Sample Median Total fat (g/d)</th>
<th>Sample SD Total fat (g/d)</th>
<th>National Mean Total fat (g/d)</th>
<th>National Median Total fat (g/d)</th>
<th>National SD Total fat (g/d)</th>
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<tr>
<td>All</td>
<td>27.0</td>
<td>25.1</td>
<td>68.3</td>
<td>74.8</td>
<td>74.2</td>
<td>68.3</td>
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<tr>
<td>Males</td>
<td>28.2</td>
<td>26.8</td>
<td>78.9</td>
<td>82.3</td>
<td>82.3</td>
<td>78.9</td>
</tr>
<tr>
<td>Females</td>
<td>25.2</td>
<td>23.1</td>
<td>59.9</td>
<td>56.9</td>
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<td>59.9</td>
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<table>
<thead>
<tr>
<th></th>
<th>Sample Mean Saturated fat (g/d)</th>
<th>Sample Median Saturated fat (g/d)</th>
<th>Sample SD Saturated fat (g/d)</th>
<th>National Mean Saturated fat (g/d)</th>
<th>National Median Saturated fat (g/d)</th>
<th>National SD Saturated fat (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>25.0</td>
<td>23.8</td>
<td>65.8</td>
<td>27.6</td>
<td>27.3</td>
<td>65.8</td>
</tr>
<tr>
<td>Males</td>
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<td>23.8</td>
<td>75.9</td>
<td>27.3</td>
<td>27.3</td>
<td>75.9</td>
</tr>
<tr>
<td>Females</td>
<td>25.1</td>
<td>23.8</td>
<td>59.9</td>
<td>59.9</td>
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</table>

<table>
<thead>
<tr>
<th></th>
<th>Sample Mean Protein (g/d)</th>
<th>Sample Median Protein (g/d)</th>
<th>Sample SD Protein (g/d)</th>
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<th>National Median Protein (g/d)</th>
<th>National SD Protein (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>74.8</td>
<td>71.8</td>
<td>68.3</td>
<td>71.8</td>
<td>68.3</td>
<td>68.3</td>
</tr>
<tr>
<td>Males</td>
<td>78.3</td>
<td>74.9</td>
<td>78.9</td>
<td>74.9</td>
<td>78.9</td>
<td>78.9</td>
</tr>
<tr>
<td>Females</td>
<td>73.1</td>
<td>69.4</td>
<td>65.0</td>
<td>65.0</td>
<td>65.0</td>
<td>65.0</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Sample Mean Carbohydrate (g/d)</th>
<th>Sample Median Carbohydrate (g/d)</th>
<th>Sample SD Carbohydrate (g/d)</th>
<th>National Mean Carbohydrate (g/d)</th>
<th>National Median Carbohydrate (g/d)</th>
<th>National SD Carbohydrate (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>230.6</td>
<td>219.0</td>
<td>66.3</td>
<td>57.2</td>
<td>57.2</td>
<td>57.2</td>
</tr>
<tr>
<td>Males</td>
<td>240.9</td>
<td>250.0</td>
<td>75.5</td>
<td>68.9</td>
<td>68.9</td>
<td>68.9</td>
</tr>
<tr>
<td>Females</td>
<td>204.7</td>
<td>207.4</td>
<td>44.3</td>
<td>35.7</td>
<td>35.7</td>
<td>35.7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Sample Mean NMES (g/d)</th>
<th>Sample Median NMES (g/d)</th>
<th>Sample SD NMES (g/d)</th>
<th>National Mean NMES (g/d)</th>
<th>National Median NMES (g/d)</th>
<th>National SD NMES (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>66.3</td>
<td>57.2</td>
<td>57.2</td>
<td>57.2</td>
<td>57.2</td>
<td>57.2</td>
</tr>
<tr>
<td>Males</td>
<td>75.5</td>
<td>68.9</td>
<td>68.9</td>
<td>68.9</td>
<td>68.9</td>
<td>68.9</td>
</tr>
<tr>
<td>Females</td>
<td>44.3</td>
<td>35.7</td>
<td>35.7</td>
<td>35.7</td>
<td>35.7</td>
<td>35.7</td>
</tr>
</tbody>
</table>

Statistical analyses

Statistical analyses of the comparison between both datasets were carried out using SPS (for Windows v20) (SRS Inc.). For selected nutrients, descriptive statistics (mean, median, standard deviation, 95th and 2nd percentiles) were calculated in line with those published for data from the National Diet and Nutrition Survey. Differences in mean total energy intake were tested for statistical significance using independent t-tests incorporating Satterthwaite’s correction where required. Analyses of variance were also performed to examine the effects of sex and study series on energy and nutrient intakes. Differences in mean total energy intake were tested for statistical significance using independent t-tests incorporating Satterthwaite’s correction where required. Analyses for selected nutrients, descriptive statistics (mean, median, standard deviation, 95th and 2nd percentiles) were calculated in line with those published for data from the National Diet and Nutrition Survey. Differences in mean total energy intake were tested for statistical significance using independent t-tests incorporating Satterthwaite’s correction where required. Analyses of variance were also performed to examine the effects of sex and study series on energy and nutrient intakes.

Data from the two study series were compared using independent t-tests. Differences in mean total energy intake were tested for statistical significance using independent t-tests incorporating Satterthwaite’s correction where required. Analyses of variance were also performed to examine the effects of sex and study series on energy and nutrient intakes.
The data represent the nutritional habits of those with a first episode of psychosis. Whilst the duration of untreated psychosis may be variable, the dietary intake of these individuals well represents a first episode of psychosis study series at the base line of admission to services due to the validated methods used. It is interesting to note that the total daily energy intake in males is within recommended guidelines, yet is lower than in the general population. This could be due to a lower proportion of micronutrients consumed in the study group as nutritional status and micronutrient intake amongst females is lower than in the national sample. Amongst males, a lower mean intake of all the micronutrients considered except vitamin D (vis. JPN, respectively, and there is mounting evidence that the general population does not consume sufficient vitamin D(6)).

Table 3 shows the numbers and percentages of participants in the study whose dietary intakes meet or exceed the recommended daily intakes for each nutrient. The results indicate that the majority of participants meet or exceed the recommended daily intake for vitamin C, vitamin E, and folate, but that lower intakes are prevalent for vitamin A, vitamin D, calcium, and iron. The proportion of participants meeting the recommended daily intake for vitamin B12 is also lower than the general population.

Table 2. Summary statistics for daily micronutrient intakes

<table>
<thead>
<tr>
<th>Vitamin C (mg/d)</th>
<th>Vitamin D (μg/d)</th>
<th>Folate (μg/d)</th>
<th>Fe (mg/d)</th>
<th>Se (μg/d)</th>
<th>Zn (mg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample</td>
<td>National</td>
<td>Sample</td>
<td>National</td>
<td>Sample</td>
<td>National</td>
</tr>
<tr>
<td>Mean</td>
<td>77.2</td>
<td>85.7</td>
<td>2.1</td>
<td>220.9</td>
<td>260.0</td>
</tr>
<tr>
<td>Median</td>
<td>56.0</td>
<td>69.8</td>
<td>1.5</td>
<td>199.0</td>
<td>244.0</td>
</tr>
<tr>
<td>Male</td>
<td>77.9</td>
<td>88.4</td>
<td>2.3</td>
<td>230.0</td>
<td>298.0</td>
</tr>
<tr>
<td>Female</td>
<td>53.0</td>
<td>67.6</td>
<td>2.6</td>
<td>125.0</td>
<td>172.0</td>
</tr>
</tbody>
</table>

* Independent t-test comparing means of study cohort and national sample.

Asymptomatic, the most notable difference is in the total fat intake amongst females (p = 0.004), females in the sample was on average 12.9% (95% CI, 4.3, 18.4) more than in the national sample. Indeed, at per cent in the distribution than those in the national sample, 7.7% (95% CI, 0.03%) makes the study consume an average 7.7% of total protein consumed. Amongst males, a lower intake of protein was also observed in the study group, on average 7.7% (95% CI, 0.03%).
Table 3. Comparison of study subjects' micronutrient intakes with reference nutrient intake (RNI) and lower RNI (LRNI)*

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Males</th>
<th>Females</th>
<th>Males</th>
<th>Females</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>At or above RNI</td>
<td>At or above LRNI but below RNI</td>
<td>Below LRNI</td>
<td>Total</td>
<td>At or above RNI</td>
<td>At or above LRNI but below RNI</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>56%</td>
<td>64%</td>
<td>39%</td>
<td>56%</td>
<td>62%</td>
<td>67%</td>
</tr>
<tr>
<td>Folate</td>
<td>46%</td>
<td>49%</td>
<td>41%</td>
<td>46%</td>
<td>42%</td>
<td>42%</td>
</tr>
<tr>
<td>Fe</td>
<td>18%</td>
<td>15%</td>
<td>22%</td>
<td>18%</td>
<td>22%</td>
<td>22%</td>
</tr>
<tr>
<td>Se</td>
<td>54%</td>
<td>42%</td>
<td>52%</td>
<td>42%</td>
<td>58%</td>
<td>52%</td>
</tr>
<tr>
<td>Zn</td>
<td>10%</td>
<td>6%</td>
<td>6%</td>
<td>6%</td>
<td>6%</td>
<td>6%</td>
</tr>
</tbody>
</table>

* There are currently no recommended intakes for vitamin D.
† Aged 50 years or under only. Two females aged over 50 years had levels above the RNI for their age group.
‡ There is currently no LRNI for Cu.

Evidence of greater stress reported throughout the early and pre-illness phases for individuals.(19)

This finding of increased total energy and carbohydrate intake, particularly in females, within first-episode psychosis concurs with a previous study of those with schizophrenia.(51)

When nutrient intakes of community-dwelling schizophrenic individuals in the USA were compared with those of the National Health and Nutrition Examination Study (NHANES) dataset,(54), total carbohydrate intake was significantly higher than that of the general population. The total energy intake reported was again significantly higher than that of the general population, with the largest difference reported in females.(3) A higher fat intake and lower fibre intake relative to the general population have also been reported from a cohort of 102 middle-aged, community-dwelling individuals with schizophrenia(51). One study reported a lower fat intake in the schizophrenia group, compared with the controls.(52)

Of thirty-one published studies from schizophrenic cohorts, 44% cited an increased intake of fat; however, the differences observed in those studies could be due to the enduring psychosis and potential hyperphagic effect of antipsychotic medication.(20,21) Published case–control studies of first-episode psychosis service users have found only one macronutrient – a higher intake of saturated fat – to be higher than that of the general population(19), which was found to worsen post-antipsychotic treatment for 6 months(16), whilst others found no difference.(17,18). The proportions of individuals not meeting the lower RNI, particularly for Se (males 54.0% and females 57.1%) and for Fe amongst females (29.6%), is cause for concern regarding potentially severe deficiencies.

In this study the suboptimal micronutrient deficiencies, particularly in males, are concerning because this may indicate that whilst the diet is broadly supplying the energy needs of the individual, the quality of food supplying that energy is substandard. This could have other implications for health, particularly in light of the evidence that micronutrients may be specifically linked to mental health.

Carbohydrate intake is higher in females, although there is no difference between NME: sugar intakes, whereas another study has found that those with schizophrenia (not limited to first-episode psychosis) have a diet that is significantly higher in sugar(33). If this trend toward increased sugar consumption being correlated to increased duration of psychosis was to be repeated in further studies it could have implications,
first, for healthcare provision, and moreover the recommendations for sugar consumption in this population subgroup. It is also noteworthy that the study cohort’s NME sugar intake was not significantly higher in both sexes than the population, but higher than the current recommendation for both males (75·5 v. 55 g/d) and females (52·1 v. 45 g/d) \(^{46}\). NME sugar consumption has decreased in the general population in the UK in the last decade; the intake of the population, based upon the 2008/2009 rolling assessment programme data, indicates that it is still higher than recommended by the Food Standards Agency \(^{45}\). Despite this, intakes are higher than recommended and this reinforces the suggestion that dietary education is warranted, to help reduce sugar intake – or at least prevent an increase – in light of links between sugar and markers of the metabolic syndrome, and the higher prevalence of physical co-morbidities in those with schizophrenia \(^{1–7}\). A high NME sugar intake was found to be associated with type 2 diabetes in one meta-analysis \(^{34}\) and again more recently \(^{55}\). When the effect of sugar consumption is measured in terms of the glycaemic load, it has reportedly led to an increased CHD risk \(^{56}\), which was not mirrored by the total carbohydrate intake. In a review published over a decade ago, high-sucrose diets, when consumed excessively, increased the incidence of hypertriglyceridaemia in obese individuals \(^{57}\). Further convincing evidence shows that NME sugar in sugar-sweetened beverages contributes to weight gain and obesity \(^{54}\). It is also possible that individuals are aware of key public health advice relating to nutrition and the association with metabolic co-morbidities, such as type 2 diabetes and obesity. Further work in this area should consider the measurement of the nutritional knowledge of community-dwelling individuals with first-episode psychosis.

The findings presented here indicate that even within 12 weeks of diagnosis with first-episode psychosis, diets are nutritionally poorer than the population. There are thus pertinent public health issues due, first, to the literature that links nutrients to health in the general population and then moreover to schizophrenia’s association with an increased risk of developing physical co-morbidities \(^{15,56–61}\). This cannot be dissociated from the continued disparity that exists in the life expectancy of those with severe mental illness \(^{2,7}\).

There are several potential consequences of a low micronutrient intake on first-episode psychosis individuals. First, does a potential link exists between their lack of micronutrients and the precipitation of their mental illness? Second, could this micronutrient-deficient diet be hampering their recovery, specifically through a neurotransmission-mediated response to prescribed medications? These processes require key nutrients, including folate, Se and Fe, to effect these changes. Furthermore, as the physiological requirement for vitamin D is met through diet and the duration and intensity of the exposure to sunlight, a chronic deficiency is plausible amongst those with psychosis, due to a possible co-morbid social phobia \(^{62}\) and consequent lack of outdoor exposure.

As part of the nutritional education of those with first-episode psychosis, it is prudent to consider the nutritional quality of daily energy sources. A plan of regular nutritional assessments is advisable to ensure that this situation is monitored, in light of the evidence that those with chronic or relapsing schizophrenia have significantly higher micronutrient intake compared with controls and are more likely to develop the metabolic syndrome than the general population.

**Limitations**

This paper compares the mean intake of a self-selected study series with first-episode psychosis with the general population. Whilst the study series may be comparable with the sample in the National Diet and Nutrition Survey in as far as these individuals were also self-selecting, this may not be fully representative of the population’s dietary intake.

Questions may also arise over the validity of self-reported dietary intakes in general; however, in some cases food diaries were backed up by members of the individuals’ care networks (partners or family members) and the concordance of total energy intake with the general population shows that drawing comparisons from the two datasets is valid. The generalisability of these results is limited though because no physiological measures, e.g. haematological or fingernail samples, were taken from the psychosis cohort to improve the validity of the participant reporting in the food diaries and any incomplete or inaccurate micronutrient data from the food composition tables. This would be advantageous in any future study of this type.

It is limiting that no information was available relating to the socio-economic status or food purchasing patterns of the psychosis cohort. Socio-economic status has long been linked to food purchasing patterns, nutrient consumption \(^{63}\) and thus possibly health outcomes, yet whilst that data would have been useful, it is important to note that psychosis can affect any individual, irrespective of socio-economic or lifestyle factors. Likewise, a measure of other variables that affect dietary intake behaviour, such as cooking ability, influence of family and peers and knowledge of the links between nutrition and health \(^{64,65}\) was not recorded for the psychosis cohort; however, this would warrant further investigation. Prescribed antipsychotic medication has also been linked to an alteration of food intake and metabolism \(^{20–29}\); thus a limitation of this study, and one that would warrant further consideration, is the lack of data pertaining to both the initiation and dosage of treatment with antipsychotic medication and any effect that may have on the dietary habits of those with psychosis.

**Conclusion**

Further exploration of dietary habits with first-episode psychosis would be warranted to assess whether individuals make dietary changes that would benefit their physical and mental health and wellbeing following dietary change intervention. It would also be pertinent to assess any relationship between diet and mental health symptomology.

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References


