

Review

Dietary intake of people with severe mental illness: systematic review and meta-analysis

Scott B. Teasdale, Philip B. Ward, Katherine Samaras, Joseph Firth, Brendon Stubbs, Elise Tripodi and Tracy L. Burrows

Background

Severe mental illness (SMI) is thought to be associated with lower diet quality and adverse eating behaviours contributing towards physical health disparities. A rigorous review of the studies looking at dietary intake in psychotic disorders and bipolar disorder is lacking.

Aims

To conduct a systematic, comprehensive evaluation of the published research on dietary intake in psychotic disorders and bipolar disorder.

Method

Six electronic databases were searched for studies reporting on dietary intakes in psychotic disorders and bipolar disorder. Dietary-assessment methods, and dietary intakes, were systematically reviewed. Where possible, data was pooled for meta-analysis and compared with healthy controls.

Results

In total, 58 eligible studies were identified. People with SMI were found to have significantly higher dietary energy (mean

difference 1332 kJ, 95% CI 487–2178 kJ/day, $P = 0.002$, $g = 0.463$) and sodium (mean difference 322 mg, 95% CI 174–490 mg, $P < 0.001$, $g = 0.414$) intake compared with controls. Qualitative synthesis suggested that higher energy and sodium intakes were associated with poorer diet quality and eating patterns.

Conclusions

These dietary components should be key targets for preventative interventions to improve weight and other physical health outcomes in people with SMI.

Declaration of interest

S.B.T. and E.T. have clinical dietitian appointments within the South Eastern Sydney Local Health District and do not receive any further funding.

Keywords

Health behaviour; obesity; physical health; bipolar disorder; schizophrenia.

Copyright and usage

© The Royal College of Psychiatrists 2019.

People with severe mental illness (SMI) die approximately 15 years earlier than individuals in the general population,¹ identified as a significant human rights issue and major source of inequity.² The vast majority of these earlier deaths are attributable to physical health conditions, primarily cardiovascular disease.³ Understanding modifiable factors that may diminish or prevent the ‘scandal of premature mortality’ is essential.² In the general population, there is robust evidence that excessive energy intake and poor diet quality is associated with adverse physical health including cardiovascular disease and premature mortality.⁴ This evidence supports population-level state-sanctioned strategies that focus on nutrition as a cornerstone of health outcome determination.

For many people experiencing SMI, antipsychotic, antidepressant and mood-stabilising medications may be essential components of treatment.^{5,6} Many of these medications are associated with substantial weight gain, obesity and associated cardiometabolic abnormalities.^{7–9} It has been suggested that one of the key factors underlying these abnormalities are the effects of antipsychotic medication on dietary intake and eating behaviours.¹⁰ People receiving antipsychotic medication report increased appetite, decreased satiety and increased cravings for sweet foods and beverages.¹¹ A range of lifestyle interventions have attempted to mitigate the obesogenic effects of these medications; however, a clear understanding of the dietary intake in people experiencing psychotic illness is lacking. A key limitation in a previous systematic review of dietary patterns in schizophrenia¹² was lack of evaluation and critique of the quality of dietary intake assessment methods. Given the vast majority of dietary-assessment methods are subjective, the strength of methodology in collecting, interpreting and analysing dietary intakes is a crucial determinant for obtaining

meaningful conclusions. Thus, a comprehensive systematic review and meta-analysis of the dietary intake of people with SMI, taking into account dietary-assessment methodology, is warranted to identify possible dietary treatment targets for interventions to improve the physical health of this vulnerable population.

Method

Design

This study was pre-registered on the PROSPERO database (CRD42016048833) and conducted in accordance with the PRISMA¹³ and MOOSE statements¹⁴ (see supplementary Files 1 and 2 available at <https://doi.org/10.1192/bjp.2019.20> for PRISMA and MOOSE checklists).

Search strategy

An online search strategy was undertaken to identify studies published in the English language from 1975 to August 2017 through librarians within the University of Newcastle, Callaghan Campus. Cumulative Index to Nursing and Allied Health Literature (CINAHL), Cochrane, Embase, Medline, PsycINFO and Scopus databases were searched using common psychiatric and nutritional MeSH terms (feeding behavior OR eating OR food intake OR diet* OR nutrition* OR coffee OR caffeine OR beverages AND schizophrenia OR psychotic disorders OR bipolar disorder OR bipolar*; see supplementary File 3 for comprehensive search list). Electronic searches were supplemented with manual cross-checking

of the reference lists of relevant publications.¹² Cross-sectional and cohort studies in adults were included.

After the removal of duplicates, stage two involved the assessment of titles and abstracts of identified studies by two independent reviewers (S.B.T. and T.L.B.), with disagreements resolved by further discussion. *A priori* inclusion/exclusion criteria were applied to determine the eligibility of each publication for inclusion in the review, as per the following inclusion criteria.

Inclusion and exclusion criteria

Population

Adult populations (age ≥ 18 years or 'adults' depending on the database searched), DSM or ICD diagnosis of a severe mental illness (schizophrenia spectrum disorders, bipolar affective disorder, depression with psychosis, or other psychotic illness) or clinician-diagnosed first-episode of psychosis. There were no limitations employed for rates of psychotropic medication prescription, as reporting of this was infrequently included.

Intervention

Types of studies included cross-sectional, cohort and longitudinal designs.

Control

There were no limits on comparison groups, although only studies with matched controls were eligible for meta-analysis.

Outcome

One or more nutritional outcomes, including energy, macronutrients, micronutrients, fat subgroups, fibre, diet quality, food groups and caffeine. We excluded animal studies, studies of people with high-prevalence mental illness (depression and anxiety), or those with eating disorders (anorexia and bulimia nervosa), case studies, letters to the editor, intervention studies and studies with eating behaviour outcomes that lacked specific dietary intake outcomes. Alcohol was excluded as an outcome as we did not include substance use in our search terms. Excluded articles are summarised in Fig. 1.

Data extraction

Where necessary, corresponding authors of included studies were contacted for additional data for inclusion in meta-analysis. A follow-up email was sent 3 weeks later if corresponding authors did not reply to the initial request.

Data were extracted using standardised tables developed for this review and included study design, population demographics and dietary intake assessment methods. In cases of uncertainty regarding quality assessment, or data extraction, a third independent reviewer was consulted, until consensus was reached.

Study quality

Study quality was assessed twice for each individual study using a standardised tool from the American Academy of Nutrition and Dietetics.¹⁵ Two reviewers scored each study independently. The lead author resolved discrepancies between the different scorers. Scoring included ten quality criteria that were rated as being absent, present or unclear in each study. This included the assessment of population bias, study masking, a description of the assessment tool, statistical methods and study funding. An overall quality rating was assigned, with each study being rated as: (a) negative, if six or more answers to the validity questions are 'no', (b) neutral, if answers to validity criterion 2, 3, 6 and 7

do not indicate the study was strong, or (c) positive, if most of the answers to validity criteria were 'yes' (including criterion 2, 3, 6 and 7 plus one extra criterion). No studies were excluded based on quality ratings.

Data analysis

Studies were eligible for meta-analysis if they; (a) used a recognised dietary-assessment method (i.e. 24 h recall) or cited a validation study for the tool used, (b) had a matched control group who were assessed at the same time as the target group, and (c) reported outcomes in a compatible metric/measure. Comparisons against national health surveys and other population data sources were not eligible for meta-analysis.

All meta-analyses were performed using Comprehensive Meta-Analysis 2.0.¹⁶ To account for expected heterogeneity between studies, a random-effects model was used throughout.¹⁷ First, for studies that used validated, or recognised and acceptable, dietary-assessment tools, comparative meta-analyses were performed to calculate a pooled mean difference (and 95% confidence intervals) between participants with SMI and healthy controls in daily total energy intake, the primary outcome of interest, measured in kilojoules per day (kJ).

As secondary outcomes, we also compared SMI and control groups on daily mean intake of each macronutrient and micronutrient examined in a sufficient number of studies to justify meta-analysis (>2), using the standard units of measurements for these nutrients. If non-standard means were used, we calculated means and standard deviations from the data of the studies where possible. Along with examining differences using standard nutrient measurement values, the overall difference between SMI and control groups for daily intake of each macro/micronutrient was computed as Hedge's *g*, with resultant effect sizes classified as small (<0.2), moderate (>0.2 to <0.8) or large (>0.8). Statistical significance was set at $P < 0.05$. For all analyses, the variance between studies was assessed using Cochran's *Q* and reported as I^2 , which quantifies the degree of variance resulting from between-study heterogeneity, rather than by chance.

For the primary outcome, we also applied several tests to measure and adjust for publication bias: (a) Egger's regression test was used to quantify the risk of publication bias influencing findings, (b) the 'fail-safe N '¹⁸ was calculated to determine the number of unpublished null studies that would invalidate the findings, and (c) Duval & Tweedie's trim-and-fill analysis was used to re-calculate the pooled difference after adjusting for any studies potentially reflecting publication bias.

Results

Identification and selection of studies

Electronic database searches identified 5538 unique titles after accounting for duplicates, as summarised in Fig. 1. Five additional titles were sourced by screening a relevant references.¹² A review of titles and abstracts led to the exclusion of 5394 titles. Full-texts were assessed for the remaining 149 titles, of which 91 were excluded for reasons detailed in Fig. 1. In total, 58 studies were identified for critical appraisal and included in this review.^{19–76}

The studies were conducted in 17 countries, with the majority conducted in the USA (14 studies, $n = 4885$ participants), UK (10 studies, $n = 575$), Spain (6 studies, $n = 2637$) and Australia (6 studies, $n = 1899$). The majority of studies were cross-sectional in design (48 studies, $n = 33\,915$), and smaller numbers of cohort (6 studies, $n = 924$) and case-control (2 studies, $n = 275$) studies

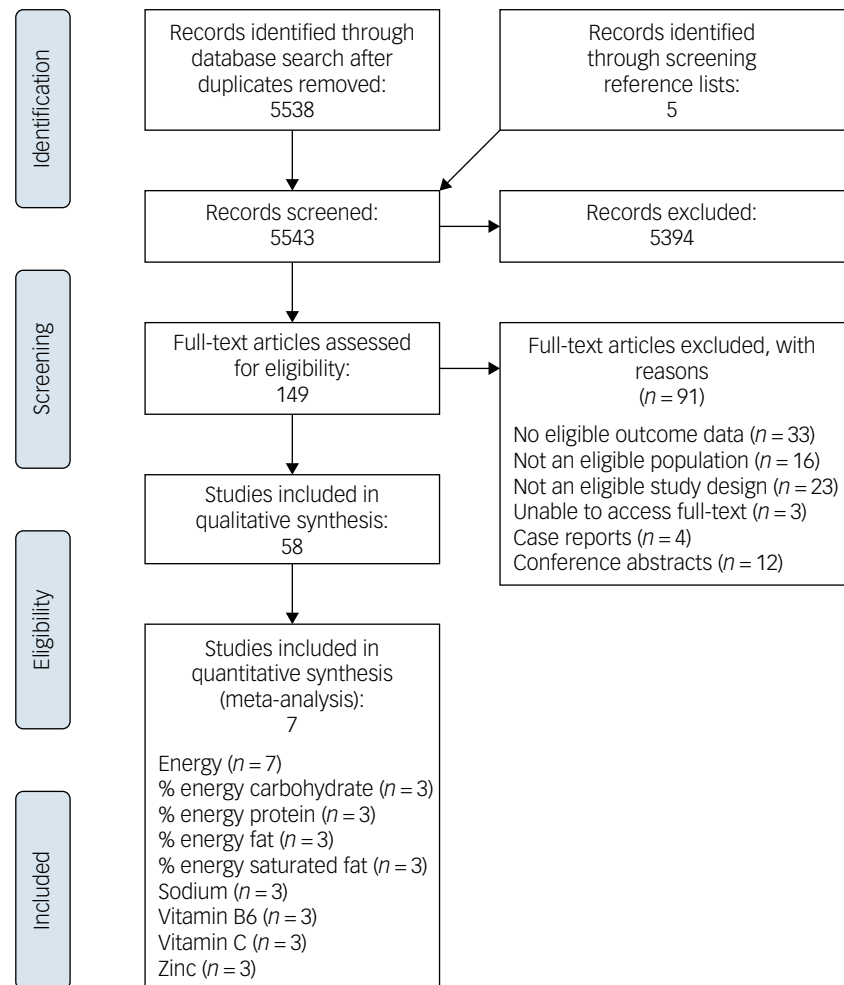


Fig. 1 PRISMA flow chart.

were included, and 1 study each for longitudinal ($n = 352$) and cross-cue reactivity ($n = 15$).

Characteristics of included studies

Participants

The 58 studies included a total of 35 481 people with SMI and 5465 non-psychiatric controls. Diagnoses within the studies were: (a) limited to schizophrenia spectrum disorders (27 studies, 47%, $n = 26\ 230$), (b) mixed SMI diagnoses (20 studies, 34%, $n = 8301$), (c) limited to bipolar affective disorder (7 studies, 12%, $n = 673$), and (d) limited to first-episode psychosis (4 studies, 7%, $n = 277$).

The majority of studies described participants as out-patients or community-dwelling (40 studies, $n = 6944$), followed by in-patients (8 studies, $n = 886$) and mixed settings (2 studies, $n = 22\ 072$). Eight studies did not report participant setting. A total of 19 studies (33%) used controls ($n = 5465$) and 20 studies (34%) used population data as a comparator group.

Participants were receiving a range of psychotropic medications including antipsychotics, mood stabilisers, antidepressants and benzodiazepine medications. Medications were reported in a range of formats including (a) chlorpromazine equivalents, (b) per cent on psychotropic medications as a whole, (c) per cent prescribed an antipsychotic medication, (d) per cent receiving antipsychotic polypharmacy, (e) per cent prescribed a mood stabiliser medication, and (f) detailed description of the specific antipsychotic and mood stabiliser medications prescribed. There were 17 studies

(29%) that did not describe medication prescription. See supplementary File 4 for complete details.

Dietary intake assessment methods

Seventeen different types of dietary-assessment methods were identified. Only 12 studies (21%) cited validation studies for the nutrition assessment method used. No study cited validation of the dietary-assessment tool used in a SMI population. One study reported piloting their food frequency questionnaire in 15 people with SMI, however, these data were unpublished. A further 22 studies (38%) used recognised acceptable dietary-intake assessment measures such as 24 h recall; however, only 11 studies (50%) reported that dietitians or other trained interviewers completed dietary intake assessment and analysed it using nutrition data-analysis software, and only four studies used multiple, non-consecutive recalls. Weighed-food records, a more objective measure of dietary intake, was used for two of these studies. In these two studies, one study assessed 7-day dietary intake, and the other 2-day dietary intake, both assessed by a dietitian/nutritionist. One study used a 3-day photographic food record assessed by a dietitian, also considered a more objective measure of dietary intake. For the remaining studies for which validity of the assessment tool was unclear, 14 studies (24%) reported the assessment tool very broadly, such as 'standardised questionnaire' or 'verbal questions' and 9 studies (16%) report using food frequency questionnaires without citing validity, or questions taken from national health surveys.

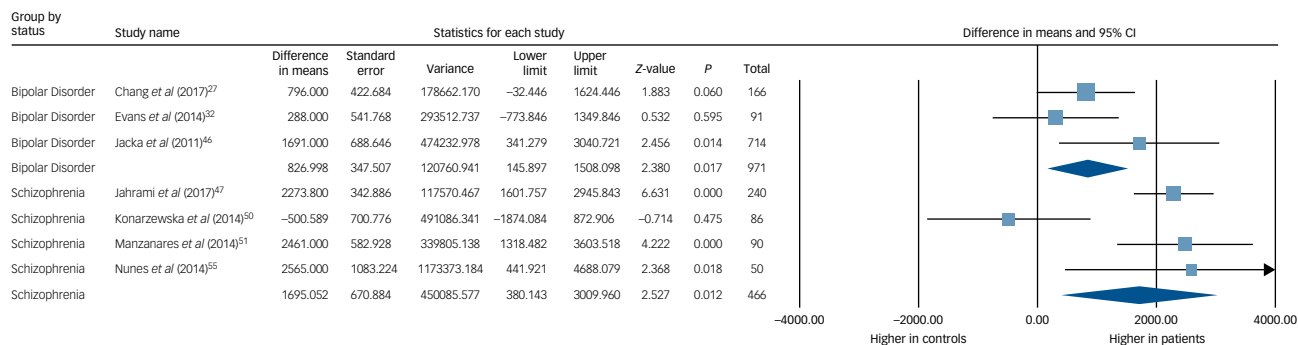


Fig. 2 Meta-analysis of energy intake in bipolar disorder and schizophrenia.

All comparisons: energy in kilojoules.

Dietary outcomes

A wide variety of dietary outcomes were assessed. The most common dietary outcome measures were energy intake (22 studies), macronutrients (carbohydrate, protein, fat) (20 studies), individual fatty acids or fat subgroups (20 studies), fibre (16 studies), food groups/categories (14 studies), caffeine or coffee intake (12 studies), overall dietary patterns (9 studies) and micronutrients (9 studies). One study also reported health (dietary) knowledge as a secondary outcome. Dietary outcomes were reported in a range of different metrics. For these reasons it was difficult to conduct direct comparisons.

Quality of studies

In total, 39 studies (67%) received a neutral score, 13 studies (22%) received a negative score and 6 studies (10%) received a positive score. Key areas of weakness included: (a) lack of concurrent controls, and comparability of groups on important confounding factors, (b) lack of use of independent assessors and masking for data collectors (when concurrent comparator groups were used), (c) measurements not being based on standard, valid and reliable methods and procedures, (d) measurements implemented at unclear level of precision, and (e) inconsistent measures used across groups. Individual study quality data are outlined in supplementary File 5.

Quantitative analysis

In two studies that only reported median averages,^{46,50} medians were used as an imputed mean and standard deviations were estimated from the pooled standard deviations across all other studies. For the one study that did not report standard deviations for controls,⁵⁴ standard deviations was imputed from a study of similar sample size.⁴²

Energy

Seven studies ($n = 1448$) reported energy intakes for both people with SMI and controls.^{27,32,46,47,50,51,55} Mean energy intake among individuals with SMI was 1332 kJ/day higher than among the controls (95% CI 487–2178 kJ/day, $P = 0.002$), with a moderate effect size for the difference between groups (7 studies, $g = 0.463$ (95% CI 0.159–0.767), $P = 0.003$). Although there was heterogeneity across the study data ($Q = 25.7$, $P < 0.001$, $I^2 = 76.7\%$), there was no evidence of publication bias ($P = 0.328$ for Egger's regression test), and the fail-safe N was 67 (estimating that 67 unpublished 'null' studies would need to exist for the actual P -value to exceed 0.05). A trim-and-fill analysis did not identify any outliers, and the random-effects point estimate remained at $g = 0.463$. These findings were in line with the two studies that used weighed-food records that found: (a) energy intake was significantly higher in

the schizophrenia group compared with the general population,⁶⁴ and (b) energy intake increased with the commencement of olanzapine, in line with the weight gain observed.³⁵

Subgroup analysis found the mean difference in energy intake for the schizophrenia spectrum cohorts compared with controls was +1695 kJ/day (95% CI 380–3010 kJ/day), $P = 0.012$, and the mean difference between energy intake in bipolar disorder-only cohorts compared with controls was +827 kJ/day (95% CI 146–1508 kJ/day), $P = 0.017$. The between-group difference in energy intake for bipolar disorder-only and schizophrenia spectrum-only cohorts was not statistically significant ($Q = 1.32$, $P = 0.251$). These are illustrated in Fig. 2.

Additional nutrients

Three studies ($n = 387$) reported data able to be pooled for meta-analysis for sodium, vitamin B6, vitamin C and zinc.^{47,50,55} Sodium intake was significantly higher in the SMI group (mean difference 332 mg, 95% CI 174–490 mg, $Z = 4.121$, $P < 0.001$, $I^2 = 12\%$, $g = 0.414$). There was no significant difference in pooled intakes of vitamin B6, vitamin C and zinc between people with SMI and controls (all $P > 0.2$). See Table 1 for meta-analysis results for energy and nutrient intakes.

Qualitative synthesis

Dietary patterns and diet quality scores were assessed in eight studies and two studies, respectively. The SMI group was found to have less healthy dietary patterns in eight studies^{36,46–49,58,76} and women had lower diet quality in one study that assessed diet quality,²⁰ and a mean diet score within the 'unhealthy' category for the other study assessing diet quality.⁶³ No included study found healthier dietary patterns for the SMI group when compared with the control or population data.

Four studies reported a relationship between dietary patterns and SMI. A higher 'Western' and 'modern' dietary pattern was positively associated, and 'traditional' dietary pattern negatively associated with bipolar disorder.⁴⁶ A 'cereal' dietary pattern (bread, rice, confectionary etc.) was positively associated with schizophrenia whereas a 'vegetable' dietary pattern was not.⁷² Higher energy intake, and lower protein intake, were positively associated with general symptom severity in early psychosis in one study,⁵¹ whereas life stress was positively associated with increased refined sugar intake in people experiencing psychosis but negatively associated with refined sugar intake in both high-risk for psychosis and healthy participants, in the same study. A fourth study in Japan found men who had infrequent intakes of vegetables, mayonnaise, potatoes, soy products, seaweed

Table 1 Meta-analysis of dietary energy and nutrient intakes

Comparison	Studies, <i>n</i>	Hedges <i>g</i>	95% CI	Z-value	<i>P</i>	<i>I</i> ²	Mean difference	95% CI	Z-value	<i>P</i>	<i>I</i> ²
Energy (kJ)	7	0.463	0.159 to 0.767	2.986	0.003	77	1332	487 to 2178	3.089	0.002	76
Sodium (mg)	3	0.414	0.181 to 0.646	3.488	<0.001	12	322	174 to 490	4.121	<0.001	0
Vitamin B6	3	0.484	-0.532 to 1.499	0.933	0.351	95	0.4	-0.4 to 1.2	0.999	0.318	96
Vitamin C (mg)	3	0.132	-0.530 to 0.794	0.391	0.696	88	8.7	-47.0 to 64.4	0.305	0.760	86
Zinc (mg)	3	0.369	-0.233 to 0.971	1.202	0.229	85	1.6	-1.4 to 4.5	1.038	0.299	85

and fish products had more pronounced psychiatric symptoms, although this correlation was not found in women.⁷⁶

Fruit and vegetable intake was found to be lower in the SMI group compared with controls/population data in three studies^{48,52,62} and less than country/region-specific recommendations in nine studies^{26,34,39,41,43,52-54,63} and higher compared with the general population in one study.⁷⁷ Low intakes, or lower intakes than comparison groups, were found for fish^{54,63} and nuts and vegetable oils.²⁰ Large intakes, or higher intakes than comparison groups, were found for carbonated beverages,²⁰ sweetened beverages,³⁰ soft drinks,⁶⁹ cakes and other sweets,⁶⁹ white bread,⁴³ hydrogenated oils²⁰ and fast food/takeaway foods.^{43,62} In addition, one study found poor diet literacy⁴¹ and one study found difficulties obtaining and/or cooking food⁴⁸ for people with SMI.

Results for macronutrients and micronutrients were mixed when compared with reference groups and recommended intakes, with no clear findings emerging. There were trends for studies to find lower mono- and polyunsaturated fats,^{24,28,29,55} and higher intakes of total and saturated fat^{51,54,57,64,65,72} and trans-fats⁵⁵ in the SMI groups when compared with a comparator (control or population data). In addition, higher sugar intake was found in two studies.^{57,72}

Results for fibre intakes were mixed when compared with controls or population data, although the SMI group consumed less than the national/region recommendations in nine studies^{24,29,42,44,47,50,54,65,68} and adequate intake in one study.⁵⁵ For caffeine intake, five studies found higher caffeine intakes, or high caffeine intakes (≥ 200 mg/day), to be more frequent in the SMI group compared with the reference group^{19,37,44,65,66} and one study found no difference.²² One study found people with psychosis to have a higher frequency of 'high' coffee consumption (≥ 5 cups/day) compared with other mental illnesses including depression and alcohol use disorder.⁷⁵ Seven studies found smokers in the SMI groups to have the highest caffeine intakes.^{19,22,23,25,37,65,66} Differences in reporting of caffeine and caffeinated drinks as outcomes meant that data could not be pooled for meta-analysis.

Discussion

Main findings

This is the first meta-analysis of dietary intake in people with either psychosis or bipolar disorder and showed significantly higher total energy and sodium intakes compared with controls. This study also found consistent reports of less healthy dietary patterns including low intakes of fruit and vegetables, and high intakes of takeaway and other convenience foods, and sugar-sweetened beverages. A previous qualitative synthesis in schizophrenia and schizoaffective disorder¹² suggested poorer dietary patterns in this population. Poorer dietary patterns are not limited to schizophrenia and schizoaffective disorder and included all those with a SMI diagnosis, although energy intake appears to be highest for those with psychotic disorders. This was also the first study to systematically review dietary intake assessment methods and strength of reporting, highlighting a need to improve scientific rigour in this area.

Interpretation of our findings and comparison with findings from other studies

The strongest evidence was found for higher energy and sodium intakes in SMI populations, with statistically significant differences compared with controls (+1332 kJ/day and +322 mg/day higher in those with SMI, respectively) when pooled intakes obtained from validated assessment tools were compared with matched controls. These findings are consistent with the two studies using weighed-food records (considered the most accurate method) that found: (a) a significant increase in caloric intake coinciding with weight gain,³⁵ and (b) a schizophrenia group that consumed more energy, sugar and fat compared with the general population.⁶⁴ Both meta-analyses revealed moderate effect sizes ($g = 0.46$ and $g = 0.41$, respectively), and are of considerable clinical relevance, particularly an increased energy intake of 1332 kJ per day, given that: (a) the general population is already overconsuming energy⁷⁸ and salt,⁷⁹ and (b) this is compounded with high levels of sedentary behaviour⁸⁰ and low cardiorespiratory fitness,⁸¹ which helps to explain the alarming rates of obesity, metabolic syndrome, diabetes, cardiovascular disease and premature mortality in people with SMI.³

The weight change dynamics paradigm of Hall and coworkers⁸² predicts that every 100 kJ intake excess will have an eventual body weight change of 1 kg. Applied to the findings from this study, someone with SMI would weigh on average 13 kg more than the general population (mean 8 kg and 17 kg in the bipolar disorder and schizophrenia groups, respectively) from dietary factors alone.

Increased energy and sodium intakes are likely explained by increased hunger and preference for 'discretionary foods' such as sweetened beverages and convenience foods, which are high in sugar, salt and fat (and therefore energy) and low in beneficial nutrients such as fibre, vitamins and minerals. Reasons for increases in appetite remain to be clarified. A wide variety of neuroreceptor and neuroendocrine factors regulate eating behaviour and appetite in SMI.⁸³ Dopamine, serotonin, muscarinic and histamine receptors have all been implicated in antipsychotic-induced increases in hunger, with drugs with high affinity for 5HT_{2c} and muscarinic receptors associated with the greatest risk of weight gain.^{84,85} Compounded with the lower levels of physical activity among people with SMI,⁸⁰ this helps to explain the stark difference in weight and BMI status between people with SMI and the general population.⁸⁶

Intakes of the micronutrients vitamin B6, vitamin C and zinc were not significantly different from controls in our analyses, although these were limited to the few studies that included such data. Given a previous analysis found blood levels of micronutrients were significantly lower in SMI compared with healthy controls,⁸⁷ more well-designed dietary intake studies should investigate micronutrients, particularly those with a close relationship to mental health, such as folate.

Strengths and limitations

Search strategy

The search strategy was limited to articles written in English and therefore articles written in another language were not reviewed.

In addition, grey literature was not searched for this review. The study aimed to focus on naturalistic cohort data of real-world patients' dietary consumption such that the results would reflect clinical reality, hence the inclusion of cohort/cross-sectional studies, and the exclusion of randomised controlled trial data.

Medication

Attempts were made to disentangle the effects of antipsychotic and mood-stabilising medication; however, because of insufficient reporting, limited conclusions could be made from this review. Given the differing effects on metabolic health of antipsychotics and mood-stabilising medications, more research is needed to explore the specific effects of individual medications on dietary intake and eating behaviours.

Dietary-assessment methods and tools

Qualitative synthesis found a large range of dietary-assessment and analysis methods and outcomes were employed, a clear challenge for interpreting dietary intake. A large proportion (37%) either used an unvalidated tool or did not report whether the tool had undergone validation, which limits generalisability of any findings. Those studies that used validated assessment or recognised, acceptable methods were often not specific to mental health populations; that is, it is unknown if the tools perform accurately or individuals with mental health diagnoses can accurately report on dietary intake. However, the two studies using weighed-food records and one study using a photographic food diary (i.e. more objective measures of dietary intake), found results in line with the results obtained in the current review. The overall strength of reporting in many studies was also limited, with the majority considered to be neutral (i.e. neither strong nor weak, $n = 40$).

The dietary intake methods used in studies included in this meta-analysis were based on self-report, so may reflect subjective bias. This review comprehensively and systematically reviewed all published studies, providing a best-guess insight into dietary intake in people with SMI. Misreporting is a common issue in the general population, with an average energy underreporting of approximately 20%, and higher in people who are obese (~30%). Given that people with SMI commonly experience additional barriers, including cognitive impairment, lack of motivation and poor memory, misreporting could be expected to be more common, and to have a larger impact, in this population suggesting the findings on energy intake may be an underestimation. Comparisons against population data can be misleading as the population data can be captured years or even decades earlier, are generally unmatched to the target group and may have used a different nutrition assessment method to the target group. These provide potential explanations for conflicting results, which have been reported.^{47,74}

Results from this review suggest that no dietary-assessment method or tool has been thoroughly validated in SMI. There is a clear need for subjective measures of dietary assessment to be compared with objective measures, such as biomarkers, to assess the accuracy of self-report measures in people with SMI. Biomarkers, such as carotenoids which are reflective of fruit and vegetable intake,⁸⁸ or doubly labelled water which is reflective of total energy intake,⁸⁹ are validated specific measures of dietary intake in the general population. Although direct relationships between diet and objective biomarkers likely also exist in mental illness, consideration of certain factors is needed when interpreting results. For example, inflammation associated with mental illness may reduce the levels of these biomarkers. Given cognitive impairment, poor memory, motivation difficulties and potential recall bias, those assessing dietary intake need expertise or training in dietary-

assessment method selection and implementation, to ensure appropriate use of particular methods. Given short-term, 'snapshot' assessments of dietary intake were commonly used (such as the use of 24 h recalls), long-term dietary intake in SMI may require further investigation. Meta-analyses in this review were also limited because of the range of metrics used to report outcomes, placing greater importance on complementary qualitative synthesis.

First-episode psychosis

As the pooled prevalence of recovery in first-episode of psychosis is 38%,⁹⁰ and weight gain and metabolic decline are most rapid in first-episode psychosis in the earlier stages of psychotropic medication treatment,⁷ it was deemed imperative to review studies that included clinician-diagnosed first-episode psychosis. The four identified studies in first-episode psychosis found unfavourable dietary intakes,^{59,60,62,74} in line with the results for established, enduring SMI. This is particularly important, since increased food intake and the majority of associated weight gain is believed to begin following initiation of antipsychotic treatment.^{11,91} Furthermore, first-episode psychosis has been identified as a 'critical period' for targeting lifestyle behaviours in order to prevent obesity and metabolic dysfunction from arising later in life.⁹²

Impact of diet on brain health

There was limited information obtained from the systematic review for the impact of diet on brain health and mental illness symptomatology; however, evidence in this area is growing. There appears to be a bidirectional relationship between diet quality and depression,⁹³ with emerging randomised controlled trial evidence finding that improvements in diet quality correlate with improvements in depressive symptoms.^{94,95} Additionally, dietary intake appears to be a factor in brain health, which may be of particular relevance given the neurodegeneration involved with SMI. High blood sugar and a Western diet, which is high in processed, non-nutritious foods, and low in commonly recommended foods of a healthy diet, are associated with smaller hippocampal volume.^{96,97} Further, a meta-analysis has also demonstrated the potential preventative action of diet on the development of a series of brain ailments including cognitive impairment (eight studies, relative risk (RR) = 0.60, 95% CI 0.43–0.83) and depression (nine studies, RR = 0.68, 95% CI 0.54–0.86).⁹⁸

Future recommendations

Future recommendations include:

- use of technology to assist people with mental illness to record/remember foods consumed;
- use of food models/images and a food checklist to assist people with remembering food and beverage items and to estimate portion sizes;
- using a trained interviewer or diet expert such as dietitian;
- using an online dietary assessment primer or published review⁹⁹ to choose the most appropriate assessment method; and
- using relevant guidelines such as Strengthening the Reporting of Observational Studies in Epidemiology – nutritional epidemiology (STROBE-nut)¹⁰⁰ to enhance reporting of dietary intake studies.


Additionally, further research should be dedicated to the following areas:

- comprehensive evaluation of dietary intake relative to psychotropic medications;

- (b) dietary intake in the early stages of illness as targets for preventative intervention;
- (c) dietary intake pre-illness onset to observe if unhealthy dietary intake precedes illness onset (i.e. at-risk mental state), and whether any dietary factors may indicate the onset of illness;
- (d) the effect of dietary patterns on psychiatric symptoms, and characteristic of illness such as cognitive impairment, in people with SMI; and
- (e) evaluating the validity and reliability of dietary-assessment methodologies in people with SMI to determine appropriate methods for future use, or facilitate the development of new methods. Validation should be completed using energy equations to determine accuracy and where possible use objective biomarkers to validate.

Implications

Overall, the findings of this study are clinically important, as poor dietary intake, particularly low intakes of fruit and vegetables, and high intakes of fast food and other convenience foods, and sweetened beverages, may lead to greater risk for future cardiometabolic illness. Dietary interventions should be a standard part of care for people with SMI, to help mitigate the physical health disparities in this population compared with the wider population.

Scott B. Teasdale, BNutrDiet, BAppSci(Nutr&Food) , Senior Mental Health Dietitian, Keeping the Body in Mind Program, South Eastern Sydney Local Health District; and School of Psychiatry, University of New South Wales, Australia; **Philip B. Ward**, BMedSci, PhD, Professor of Psychiatry, School of Psychiatry, University of New South Wales; Schizophrenia Research Unit, South Western Sydney Local Health District; and Ingham Institute for Applied Medical Research, Australia; **Katherine Samaras**, MBBS, PhD, FRACP, Senior Staff Specialist (Endocrinology), Department of Endocrinology, St Vincent's Hospital; Diabetes and Metabolism Division, Garvan Institute of Medical Research; and St Vincent's Clinical School, University of New South Wales, Australia; **Joseph Firth**, PhD, Senior Research Fellow, NICM Health Research Institute, Western Sydney University, Australia; and Division of Psychology and Mental Health, Faculty of Biology, Medicine and Health, University of Manchester, UK; **Brendon Stubbs**, PhD, Head of Physiotherapy, Physiotherapy Department, South London and Maudsley NHS Foundation Trust; and Health Service and Population Research Department and Department of Psychological Medicine, Institute of Psychiatry, Psychology and Neuroscience, King's College London, UK; **Elise Tripodi**, MNutrDiet, Mental Health Dietitian, Keeping the Body in Mind Program, South Eastern Sydney Local Health District, Australia; **Tracy L. Burrows**, BHSc(N&D), PhD, FDAA, Associate Professor in Nutrition and Dietetics, School of Health Sciences and Priority Research Centre for Physical Activity and Nutrition, University of Newcastle, Australia

Correspondence: Scott Teasdale, Keeping the Body in Mind Program, Bondi Community Centre, 26 Llandaff St, Bondi Junction, NSW 2022, Australia.
Email: scott.teasdale@health.nsw.gov.au

First received 17 Feb 2018, final revision 21 Jun 2018, accepted 11 Jan 2019

Funding

J.F. is supported by a Blackmores Institute Fellowship and an MRC Doctoral Training Grant (P117413F07). B.S. holds a Clinical Lectureship supported by Health Education England and the NIHR Integrated Clinical Academic (ICA) Programme (ICA-CL-2017-03-001). B.S. is also partly supported by the Maudsley Charity and the National Institute for Health Research (NIHR) Collaboration for Leadership in Applied Health Research and Care South London at King's College Hospital NHS Foundation Trust. The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR or the Department of Health and Social Care. T.B. is supported by a Brawn Research Fellowship, University of Newcastle.

Acknowledgements

The authors thank Janelle Skinner and Rebecca McKenna for assisting with data extraction, and Michelle Hsu, Tegan Hayes and Cin Yee Hsia for assisting with study quality checks.

Supplementary material

Supplementary material is available online at <https://doi.org/10.1192/bjp.2019.20>.

References

- 1 Walker ER, McGee RE, Druss BG. Mortality in mental disorders and global disease burden implications: a systematic review and meta-analysis. *JAMA Psychiatry* 2015; **72**: 334–41.
- 2 Thornicroft G. Physical health disparities and mental illness: the scandal of premature mortality. *Br J Psychiatry* 2011; **199**: 441–2.
- 3 Correll CU, Solmi M, Veronese N, Bortolato B, Rosson S, Santonastaso P, et al. Prevalence, incidence and mortality from cardiovascular disease in patients with pooled and specific severe mental illness: a large-scale meta-analysis of 3,211,768 patients and 113,383,368 controls. *World Psychiatry* 2017; **16**: 163–80.
- 4 Gakidou E, Afshin A, Abajobir AA, Abate KH, Abbafati C, Abbas KM, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016. *Lancet* 2017; **390**: 1345–422.
- 5 Lieberman JA, Stroup TS, McEvoy JP, Swartz MS, Rosenheck RA, Perkins DO, et al. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *N Engl J Med* 2005; **353**: 1209–23.
- 6 Galletly CA, Castle D, Dark F, Humberstone V, Jablensky A, Killackey E, et al. Australian and New Zealand clinical practice guidelines for the treatment of bipolar disorder. *Aust N Z J Psychiatry* 2016; **50**: 1–117.
- 7 Álvarez-Jiménez M, González-Blanch C, Crespo-Facorro B, Hetrick S, Rodríguez-Sánchez JM, Pérez-Iglesias R, et al. Antipsychotic-induced weight gain in chronic and first-episode psychotic disorders. *CNS Drugs* 2008; **22**: 547–62.
- 8 Correll CU, Manu P, Olshansky V, Napolitano B, Kane JM, Malhotra AK. Cardiometabolic risk of second-generation antipsychotic medications during first-time use in children and adolescents. *JAMA* 2009; **302**: 1765–73.
- 9 Vancampfort D, Stubbs B, Mitchell AJ, De Hert M, Wampers M, Ward PB, et al. Risk of metabolic syndrome and its components in people with schizophrenia and related psychotic disorders, bipolar disorder and major depressive disorder: a systematic review and meta-analysis. *World Psychiatry* 2015; **14**: 339–47.
- 10 Elman I, Borsook D, Lukas SE. Food intake and reward mechanisms in patients with schizophrenia: implications for metabolic disturbances and treatment with second-generation antipsychotic agents. *Neuropsychopharmacology* 2006; **31**: 2091–120.
- 11 Blouin M, Tremblay A, Jalbert ME, Venables H, Bouchard RH, Roy MA, et al. Adiposity and eating behaviors in patients under second generation antipsychotics. *Obesity* 2008; **16**: 1780–7.
- 12 Dipasquale S, Pariante CM, Dazzan P, Aguglia E, McGuire P, Mondelli V. The dietary pattern of patients with schizophrenia: a systematic review. *J Psychiatry Res* 2013; **47**: 197–207.
- 13 Stroup DF, Berlin JA, Morton SC, Olkin I, Williamson GD, Rennie D, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. *JAMA* 2000; **283**: 2008–12.
- 14 Moher D, Liberati A, Tetzlaff J, Altman TG, Altman D, Antes G, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *Ann Intern Med* 2009; **151**: 264–9.
- 15 Academy of Nutrition and Dietetics. *Evidence Analysis Manual: Steps in the Academy Evidence Analysis Process*. Evidence Analysis Library, 2016.
- 16 Borenstein M, Hedges LV, Higgins JPT, Rothstein HR. *Comprehensive Meta-Analysis Version 2*: 104. Biostat, 2005.
- 17 DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials* 1986; **7**: 177–88.
- 18 Orwin RG. A fail-safe N for effect size in meta-analysis. *J Educ Stat* 1983; **8**: 157–9.
- 19 Adolfo AB, AhnAllen CG, Tidey JW. Effects of smoking cues on caffeine urges in heavy smokers and caffeine consumers with and without schizophrenia. *Schizophr Res* 2009; **107**: 192–7.
- 20 Amani R. Is dietary pattern of schizophrenia patients different from healthy subjects? *BMC Psychiatry* 2007; **7**: 15.
- 21 Archie SM, Goldberg JO, Akhtar-Danesh N, Landeen J, McColl L, McNiven J. Psychotic disorders, eating habits, and physical activity: who is ready for life-style changes? *Psychiatr Serv* 2007; **58**: 233–9.
- 22 Arrojo-Romero M, Armas Barbazán C, López-Moriñigo JD, Ramos-Rios R, Gurpegui M, Martínez-Ortega JM, et al. Caffeine consumption in a long-term psychiatric hospital: tobacco smoking may explain in large part the apparent association between schizophrenia and caffeine use. *Schizophr Res* 2015; **164**: 234–41.
- 23 Baethge C, Tondo L, Lepri B, Baldessarini RJ. Coffee and cigarette use: association with suicidal acts in 352 Sardinian bipolar disorder patients. *Bipolar Disord* 2009; **11**: 494–503.
- 24 Bly MJ, Taylor SF, Dalack G, Pop-Busui R, Burghardt KJ, Evans SJ, et al. Metabolic syndrome in bipolar disorder and schizophrenia: dietary and lifestyle factors compared to the general population. *Bipolar Disord* 2014; **16**: 277–88.

- 25 Bobes J, Arango C, Garcia-Garcia M, Rojas J. Healthy lifestyle habits and 10-year cardiovascular risk in schizophrenia spectrum disorders: an analysis of the impact of smoking tobacco in the CLAMORS schizophrenia cohort. *Schizophr Res* 2010; **119**: 101–9.
- 26 Brown S, Birtwistle J, Roe L, Thompson C. The unhealthy lifestyle of people with schizophrenia. *Psychol Med* 1999; **29**: 697–701.
- 27 Chang YW, Assari S, Prossin AR, Stertz L, McInnis MG, Evans SG. Bipolar disorder moderates associations between linoleic acid and markers of inflammation. *J Psychiatr Res* 2017; **85**: 29–36.
- 28 Clayton EH, Hanstock TL, Hirneth SJ, Kable CJ, Garg ML, Hazell PL. Long-chain omega-3 polyunsaturated fatty acids in the blood of children and adolescents with juvenile bipolar disorder. *Lipids* 2008; **43**: 1031–8.
- 29 Ellingrod VL, Taylor SF, Brook RD, Evans SJ, Zöllner SK, Grove TB, et al. Dietary, lifestyle and pharmacogenetic factors associated with arteriole endothelial-dependent vasodilatation in schizophrenia patients treated with atypical antipsychotics (AAPs). *Schizophr Res* 2011; **130**: 20–6.
- 30 Elmslie JL, Mann JJ, Silverstone JT, Williams SM, Romans SE. Determinants of overweight and obesity in patients with bipolar disorder. *J Clin Psychiatry* 2001; **62**: 486–91.
- 31 Evans SJ, Assari S, Harrington GJ, Chang YW, Burant CF, McInnis MG. Plasma linoleic acid partially mediates the association of bipolar disorder on self-reported mental health scales. *J Psychiatr Res* 2015; **68**: 61–7.
- 32 Evans SJ, Ringrose RN, Harrington GJ, Mancuso P, Burant CF, McInnis MG. Dietary intake and plasma metabolomic analysis of polyunsaturated fatty acids in bipolar subjects reveal dysregulation of linoleic acid metabolism. *J Psychiatr Res* 2014; **57**: 58–64.
- 33 Fawzi MH, Fawzi MM, Said NS, Fawzi MM, Fouad AA, Abdel-Moety H. Effect of Ramadan fasting on anthropometric, metabolic, inflammatory and psychopathology status of Egyptian male patients with schizophrenia. *Psychiatry Res* 2015; **225**: 501–8.
- 34 Fusar-Poli P, De Marco L, Cavallin F, Bertorello A, Nicolasi M, Politi P. Lifestyles and cardiovascular risk in individuals with functional psychoses. *Perspect Psychiatr Care* 2009; **45**: 87–99.
- 35 Gothelf D, Falk B, Singer P, Kairi M, Philiip M, Zigel L, et al. Weight gain associated with increased food intake and low habitual activity levels in male adolescent schizophrenic inpatients treated with olanzapine. *Am J Psychiatry* 2002; **159**: 1055–7.
- 36 Gupta A, Craig TK. Diet, smoking and cardiovascular risk in schizophrenia in high and low care supported housing. *Epidemiol Psychiatr Soc* 2009; **18**: 200–7.
- 37 Gurpegui M, Aguilar MC, Martinez-Ortega JM, Diaz FJ, de Leon J. Caffeine intake in outpatients with schizophrenia. *Schizophr Bull* 2004; **30**: 935–45.
- 38 Gurpegui M, Aguilar MC, Martinez-Ortega JM, Jurado D, Diaz FJ, Quintana HM, et al. Fewer but heavier caffeine consumers in schizophrenia: a case-control study. *Schizophr Res* 2006; **86**: 276–83.
- 39 Hahn LA, Galletly CA, Foley DL, Mackinnon A, Watts GF, Castle DJ, et al. Inadequate fruit and vegetable intake in people with psychosis. *Aust N Z J Psychiatry* 2014; **48**: 1025–35.
- 40 Hamera E, Schneider JK, Deviney S. Alcohol, cannabis, nicotine, and caffeine use and symptom distress in schizophrenia. *J Nerv Ment Dis* 1995; **183**: 559–65.
- 41 Hardy S, Gray R. The secret food diary of a person diagnosed with schizophrenia. *J Psychiatr Ment Health Nurs* 2012; **19**: 603–9.
- 42 Haruyuki ITO, Kumagai T, Kimura M, Kolke S, Shimizu T. Dietary intake in body mass index differences in community-based Japanese patients with schizophrenia. *Iranian J Public Health* 2015; **44**: 639–45.
- 43 Heald A, Pendlebury J, Anderson S, Narayan V, Guy M, Gibson M, et al. Lifestyle factors and the metabolic syndrome in schizophrenia: a cross-sectional study. *Ann Gen Psychiatry* 2017; **16**: 12.
- 44 Henderson DC, Borba CP, Daley TB, Boxill R, Nguyen DD, Culhane MA, et al. Dietary intake profile of patients with schizophrenia. *Ann Clin Psychiatry* 2006; **18**: 99–105.
- 45 Henderson DC, Cagliero E, Copeland PM, Borba CP, Evins E, Hayden D, et al. Glucose metabolism in patients with schizophrenia treated with atypical antipsychotic agents: a frequently sampled intravenous glucose tolerance test and minimal model analysis. *Arch Gen Psychiatry* 2005; **62**: 19–28.
- 46 Jacka FN, Pasco JA, Mykletun A, Williams LJ, Nicholson GC, Kotowicz MA, et al. Diet quality in bipolar disorder in a population-based sample of women. *J Affect Disord* 2011; **129**: 332–7.
- 47 Jahrami HA, Faris MAE, Saif ZQ, Hammad LH. Assessing dietary and lifestyle risk factors and their associations with disease comorbidities among patients with schizophrenia: a case-control study from Bahrain. *Asian J Psychiatr* 2017; **28**: 115–23.
- 48 Kilbourne AM, Rofey DL, McCarthy JF, Post EP, Welsh D, Blow FC. Nutrition and exercise behavior among patients with bipolar disorder. *Bipolar Disord* 2007; **9**: 443–52.
- 49 Kilian R, Becker T, Krüger L, Schmid S, Frasch K. Health behavior in psychiatric in-patients compared with a German general population sample. *Acta Psychiatr Scand* 2006; **114**: 242–8.
- 50 Konarzewska B, Stefańska E, Wendolowicz A, Cwalina U, Golonko A, Malus A, et al. Visceral obesity in normal-weight patients suffering from chronic schizophrenia. *BMC Psychiatry* 2014; **14**: 35.
- 51 Manzanares N, Monseny R, Ortega L, Montalvo I, Franch J, Gutiérrez-Zotes A, et al. Unhealthy lifestyle in early psychoses: the role of life stress and the hypothalamic-pituitary-adrenal axis. *Psychoneuroendocrinology* 2014; **39**: 1–10.
- 52 McCreadie RG, Scottish Schizophrenia Lifestyle Group. Diet, smoking and cardiovascular risk in people with schizophrenia: descriptive study. *Br J Psychiatry* 2003; **183**: 534–9.
- 53 Mucheru D, Hanlon MC, Campbell LE, McEvoy M, MacDonald-Wicks L. Social dysfunction and diet outcomes in people with psychosis. *Nutrients* 2017; **9**: 80.
- 54 Nenke MA, Hahn LA, Thompson CH, Liu D, Galletly CA. Psychosis and cardiovascular disease: is diet the missing link? *Schizophr Res* 2015; **161**: 465–70.
- 55 Nunes D, Eskinazi B, Cambolm Rockett F, Delgado VB, Schwelger Perry ID. Nutritional status, food intake and cardiovascular disease risk in individuals with schizophrenia in southern Brazil: a case-control study. *Rev Psiqiatr Salud Ment* 2014; **7**: 72–9.
- 56 Osborn DP, Nazareth I, King MB. Physical activity, dietary habits and coronary heart disease risk factor knowledge amongst people with severe mental illness. *Soc Psychiatry Psychiatr Epidemiol* 2007; **42**: 787–93.
- 57 Ratliff JC, Palmese LB, Reutenauer EL, Liskov E, Grilo CM, Tek C. The effect of dietary and physical activity pattern on metabolic profile in individuals with schizophrenia: a cross-sectional study. *Compr Psychiatry* 2012; **53**: 1028–33.
- 58 Roick C, FritzWieacker A, Matschinger H, Heider D, Schindler J, Riedel-Heller S, et al. Health habits of patients with schizophrenia. *Soc Psychiatry Psychiatr Epidemiol* 2007; **42**: 268–76.
- 59 Ryan MC, Flanagan S, Kinsella U, Keeling F, Thakore JH. The effects of atypical antipsychotics on visceral fat distribution in first episode, drug-naive patients with schizophrenia. *Life Sci* 2004; **74**: 1999–2008.
- 60 Ryan MCM, Collins P, Thakore JH. Impaired fasting glucose tolerance in first-episode, drug-naive patients with schizophrenia. *Am J Psychiatry* 2003; **160**: 284–9.
- 61 Saarni S, Saarni SI, Fogelholm M, Fogelholm M, Heliövaara M, Perälä J, Suvisaari J, et al. Body composition in psychotic disorders: a general population survey. *Psychol Med* 2009; **39**: 801–10.
- 62 Samele C, Patel M, Boydell J, Leese M, Wessely S, Murray R. Physical illness and lifestyle risk factors in people with their first presentation of psychosis. *Soc Psychiatry Psychiatr Epidemiol* 2007; **42**: 117–24.
- 63 Simonelli-Muñoz AJ, Fortea MI, Salorio P, Gallego-Gomez JJ, Sánchez-Bautista S, Balanza S. Dietary habits of patients with schizophrenia: a self-reported questionnaire survey. *Int J Ment Health Nurs* 2012; **21**: 220–8.
- 64 Stokes C, Peet M. Dietary sugar and polyunsaturated fatty acid consumption as predictors of severity of schizophrenia symptoms. *Nutr Neurosci* 2004; **7**: 247–9.
- 65 Strassnig M, Singh Brar J, Ganguli R. Nutritional assessment of patients with schizophrenia: a preliminary study. *Schizophr Bull* 2003; **29**: 393–7.
- 66 Strassnig M, Singh Brar J, Ganguli R. Increased caffeine and nicotine consumption in community-dwelling patients with schizophrenia. *Schizophr Res* 2006; **86**: 269–75.
- 67 Strassnig M, Singh Brar J, Ganguli R. Dietary fatty acid and antioxidant intake in community-dwelling patients suffering from schizophrenia. *Schizophr Res* 2005; **76**: 343–51.
- 68 Sugawara N, Yasui-Furukori N, Sato Y, Saito M, Furukori H, Nakagami T, et al. Dietary patterns are associated with obesity in Japanese patients with schizophrenia. *BMC Psychiatry* 2014; **14**: 184.
- 69 Sugawara N, Yasui-Furukori N, Yamazaki M, Shimoda K, Mori T, Sugai T, et al. Attitudes toward metabolic adverse events among patients with schizophrenia in Japan. *Neuropsychiatr Dis Treat* 2016; **12**: 427–36.
- 70 Suvisaari JM, Saarni SI, Perälä J, Suvisaari JV, Härkänen T, Lönnqvist J, et al. Metabolic syndrome among persons with schizophrenia and other psychotic disorders in a general population survey. *J Clin Psychiatry* 2007; **68**: 1045–55.
- 71 Treuer T, Hoffman VP, Chen AK, Irimia V, Ocampo M, Wang G, et al. Factors associated with weight gain during olanzapine treatment in patients with schizophrenia or bipolar disorder: results from a six-month prospective, multinational, observational study. *World J Biol Psychiatry* 2009; **10**: 729–40.
- 72 Tsuruga K, Sugawara N, Sato Y, Saito M, Furukori H, Nakagami T, et al. Dietary patterns and schizophrenia: a comparison with healthy controls. *Neuropsychiatr Dis Treat* 2015; **11**: 1115–20.
- 73 Wallace B, Tennant C. Nutrition and obesity in the chronic mentally ill. *Aust N Z J Psychiatry* 1998; **32**: 82–5.

- 74 Williamson K, Kilner K, Clibbens N. A comparison of the nutrient intake of a community-dwelling first-episode psychosis cohort, aged 19-64 years, with data from the UK population. *J Nutr Sci* 2015; **4**: e28.
- 75 Winstead DK. Coffee consumption among psychiatric inpatients. *Am J Psychiatry* 1976; **133**: 1447-50.
- 76 Noguchi R, Hiraoka M, Watanabe Y, Kagawa Y. Relationship between dietary patterns and depressive symptoms: difference by gender, and unipolar and bipolar depression. *J Nutr Sci Vitaminol* 2013; **59**: 115-22.
- 77 Archie SM, Goldberg JO, Akhtar-Danesh N, Landeen J, McColl L, McNiven J. Psychotic disorders, eating habits, and physical activity: who is ready for life-style changes? *Psychiatr Serv* 2007; **58**: 233-9.
- 78 Duffey KJ, Popkin BM. Energy density, portion size, and eating occasions: contributions to increased energy intake in the United States, 1977-2006. *PLoS Med* 2011; **8**: e1001050.
- 79 Appel LJ, Frohlich ED, Hall JE, Pearson TA, Sacco RL, Seals DR, et al. The importance of population-wide sodium reduction as a means to prevent cardiovascular disease and stroke: a call to action from the American Heart Association. *Circulation* 2011; **123**: 1138-43.
- 80 Vancampfort D, Firth J, Schuch FB, Rosenbaum S, Mugisha J, Hallgren M, et al. Sedentary behavior and physical activity levels in people with schizophrenia, bipolar disorder and major depressive disorder: a global systematic review and meta-analysis. *World Psychiatry* 2017; **16**: 308-15.
- 81 Vancampfort D, Rosenbaum S, Schuch FB, et al. Cardiorespiratory fitness in severe mental illness: a systematic review and meta-analysis. *Sports Med* 2017; **47**: 343-52.
- 82 Hall KD, Heymsfield SB, Kermitt JW, Klein S, Schoeller DA, Speakman JR. Energy balance and its components: implications for body weight regulation. *Am J Clin Nutr* 2012; **95**: 989-94.
- 83 Werneke U, Taylor D, Sanders TAB. Behavioral interventions for antipsychotic induced appetite changes. *Curr Psychiatry Rep* 2013; **15**: 347.
- 84 Lett TA, Wallace TJ, Chowdhury NI, Tiwari AK, Kennedy JL, Müller DJ. Pharmacogenetics of antipsychotic-induced weight gain: review and clinical implications. *Mol Psychiatry* 2012; **17**: 242-66.
- 85 Reynolds GP, Kirk SL. Metabolic side effects of antipsychotic drug treatment—pharmacological mechanisms. *Pharmacol Ther* 2010; **125**: 169-79.
- 86 Mitchell AJ, Vancampfort D, Sweers K, van Winkel R, Yu K, De Hert M. Prevalence of metabolic syndrome and metabolic abnormalities in schizophrenia and related disorders—a systematic review and meta-analysis. *Schizophr Bull* 2011; **39**: 306-18.
- 87 Firth J, Carney R, Stubbs B, Teasdale SB, Vancampfort D, Ward PB, et al. Nutritional deficiencies and clinical correlates in first-episode psychosis: a systematic review and meta-analysis. *Schizophr Bull* 2018; **44**: 1275-92.
- 88 Burrows TL, Williams R, Rollo M, Wood L, Garg ML, Jensen M, et al. Plasma carotenoid levels as biomarkers of dietary carotenoid consumption: a systematic review of the validation studies. *J Nutr Intermed Metab*, 2015; **2**: 15-64.
- 89 Hill RJ, Davies PSW. The validity of self-reported energy intake as determined using the doubly labelled water technique. *Br J Nutr* 2001; **85**: 415-30.
- 90 Lally J, Ajnakina O, Stubbs B, Cullinane M, Murphy KC, Gaughran F, et al. Remission and recovery from first-episode psychosis in adults: systematic review and meta-analysis of long-term outcome studies. *Br J Psychiatry*, 2017; **211**: 350-8.
- 91 Fountaine RJ, Taylor AE, Mancuso JP, Greenway FL, Byerley LO, Smith SR, et al. Increased food intake and energy expenditure following administration of olanzapine to healthy men. *Obesity* 2010; **18**: 1646-51.
- 92 Teasdale SB, Ward PB, Rosenbaum S, Samaras K, Stubbs B. Solving a weighty problem: systematic review and meta-analysis of nutrition interventions in severe mental illness. *Br J Psychiatry* 2017; **210**: 110-8.
- 93 Quirk SE, Williams LJ, O'Neil A, Pasco JA, Jacka FN, Housden S, et al. The association between diet quality, dietary patterns and depression in adults: a systematic review. *BMC Psychiatry* 2013; **13**: 175.
- 94 Jacka FN, O'Neil A, Opie R, Itsiopoulos C, Cotton S, Mohebbi M, et al. A randomised controlled trial of dietary improvement for adults with major depression (the 'SMILES' trial). *BMC Med* 2017; **15**: 23.
- 95 Opie RS, O'Neil A, Jacka FN, Pizzinga J, Itsiopoulos C. A modified Mediterranean dietary intervention for adults with major depression: dietary protocol and feasibility data from the SMILES trial. *Nutr Neurosci* 2018; **21**: 487-501.
- 96 Cherbuin N, Sachdev P, Anstey KJ. Higher normal fasting plasma glucose is associated with hippocampal atrophy The PATH Study. *Neurology* 2012; **79**: 1019-26.
- 97 Jacka FN, Cherbuin N, Anstey KJ, Sachdev P, Butterworth P. Western diet is associated with a smaller hippocampus: a longitudinal investigation. *BMC Med* 2015; **13**: 215.
- 98 Psaltopoulou T, Sergentanis TN, Panagiotakos DB, Sergentanis IN, Kosti R, Scarmeas N. Mediterranean diet, stroke, cognitive impairment, and depression: a meta-analysis. *Ann Neurol* 2013; **74**: 580-91.
- 99 Shim JS, Oh K, Kim HC. Dietary assessment methods in epidemiologic studies. *Epidemiol Health* 2014; **36**: e2014009.
- 100 Lachat C, Hawwash D, Ockè MC, Berg C, Forsum E, Hörnell A, et al. Strengthening the Reporting of Observational Studies in Epidemiology—nutritional epidemiology (STROBE-nut): an extension of the STROBE statement. *Nutr Bull* 2016; **41**: 240-51.

