

A nutrition intervention is effective in improving dietary components linked to cardiometabolic risk in youth with first-episode psychosis

Scott B. Teasdale^{1,2*}, Philip B. Ward^{2,3}, Simon Rosenbaum^{1,2}, Andrew Watkins^{1,4}, Jackie Curtis^{1,2}, Megan Kalucy^{1,2} and Katherine Samaras^{5,6}

¹Early Psychosis Programme, South Eastern Sydney Local Health District, Bondi Community Centre, Bondi Junction, NSW 2022, Australia

²School of Psychiatry, University of New South Wales, NSW 2052, Australia

³Schizophrenia Research Unit, South Western Sydney Local Health District, Ingham Institute of Applied Medical Research, Liverpool Hospital, NSW 2170, Australia

⁴Faculty of Health, University of Technology Sydney, Ultimo, NSW 2007, Australia

⁵Department of Endocrinology, St Vincent's Hospital, Darlinghurst, NSW 2010, Australia

⁶Diabetes and Metabolism Division, Garvan Institute of Medical Research, Darlinghurst, NSW 2010, Australia

(Submitted 28 September 2015 – Final revision received 15 February 2016 – Accepted 17 February 2016 – First published online 4 April 2016)

Abstract

Severe mental illness is characterised by a 20-year mortality gap due to cardiometabolic disease. Poor diet in those with severe mental illness is an important and modifiable risk factor. The present study aimed to (i) examine baseline nutritional intake in youth with first-episode psychosis (FEP), (ii) evaluate the feasibility and acceptability of nutritional intervention early in FEP and (iii) to evaluate the effectiveness of early dietary intervention on key nutritional end points. Participants were recruited over a 12-month period from a community-based programme specifically targeting young people aged 15–25 years with newly diagnosed FEP. Individual dietetic consultations and practical group sessions were offered as part of a broader lifestyle programme. Dietary assessments were conducted before and at the end of the 12-week intervention. Participants exceeded recommended energy and Na intakes at baseline. Retention within the nutrition intervention was 67%, consistent with other interventions offered to FEP clients. There was a 47% reduction in discretionary food intake (–94 g/d, $P < 0.001$) and reductions in daily energy (–24%, $P < 0.001$) and Na (–26%, $P < 0.001$) intakes. Diet quality significantly improved, and the mean change was 3.6 (95% CI 0.2, 6.9, $P < 0.05$), although this finding was not significant after Bonferroni's correction. Increased vegetable intake was the main factor contributing to improved diet quality. Nutrition intervention delivered shortly after initiation of antipsychotic medication is feasible, acceptable and effective in youth with FEP. Strategies to prevent weight gain and metabolic decline will contribute to prevent premature cardiometabolic disease in this vulnerable population.

Key words: First-episode psychosis: Diet: Nutrition: Dietary and nutritional intervention: Schizophrenia

People with severe mental illness (SMI) such as schizophrenia, schizoaffective disorder, bipolar affective disorder and depression with psychotic features have a reduction in life expectancy of up to 20 years, which has been labelled a 'scandal of premature mortality'⁽¹⁾. Further, recent evidence shows that the mortality gap is widening⁽²⁾. Standard treatment for psychotic illness includes long-term antipsychotic medications (APM) combined with psychosocial interventions delivered by a multidisciplinary team^(3,4).

APM can induce rapid weight gain with associated metabolic abnormalities^(5,6). This rapid weight gain is most significant during the first 12 weeks after APM initiation, with a mean

increase of 8 kg during this period, progressing to 12 kg over the first 2 years and 18 kg over the first 4 years of treatment⁽⁵⁾. This weight gain contributes to the high rates of overweight and obesity in people with established SMI, with twice the rate of diabetes and rates of hypercholesterolaemia up to five times that of the general population^(7–9). Weight gain and cardiometabolic risk evolution associated with APM has been reviewed elsewhere⁽¹⁰⁾. In addition to APM, factors contributing to weight gain include energy-dense, highly processed, high-salt diets⁽¹¹⁾ and lower levels of physical activity⁽¹²⁾ compared with the general population, as well as higher rates of smoking and substance use^(13,14).

Abbreviations: APM, antipsychotic medication; EPP, Early Psychosis Program; FEP, first-episode psychosis; KBIM, Keeping the Body in Mind; SMI, severe mental illness.

* **Corresponding author:** S. B. Teasdale, fax +61 2 9387 1070, email Scott.Teasdale@sesiahs.health.nsw.gov.au

A high proportion of people receiving APM report considerably increased hunger and excessive night hunger, a decreased feeling of satiety and increased cravings for sweet foods and drinks^(15,16). Additional eating behaviours in this population include high levels of fast food consumption, low food literacy, fast-eating syndrome and habits such as only eating one main meal daily⁽¹⁶⁾.

Studies that have examined dietary intake of people with SMI have focused on people with chronic SMI, and are summarised in a systematic review that reported higher intakes of SFA and Na and lower intakes of fruit and fibre⁽¹¹⁾. Studies of energy intake found higher intakes in those with SMI compared with the general population^(17–19) and lower consumption of nutrient-dense foods such as vegetables, dairy products and legumes⁽²⁰⁾.

Higher energy intake contributes to weight gain and diabetes and CVD risk⁽²¹⁾; this is compounded by high levels of sedentariness and lower BMR in people with SMI^(22,23). Given the dietary behaviours described above, interventions to reduce energy intake and improve diet quality, by increasing core foods such as fruits, vegetables, whole-grains, lean meats and dairy foods and by reducing discretionary foods such as processed foods high in kilojoules, SFA, added sugars, added salts and/or alcohol, could be key interventions to improve the physical health of people with SMI. Further, reducing Na intake is a valid method of reducing the risk of CVD⁽²⁴⁾, and lowering the glycaemic load may reduce the risk of type 2 diabetes and CVD^(25,26).

Despite evidence that lifestyle intervention improves the physical and biochemical measures in both early psychosis^(27,28) and enduring SMI^(29–31), few studies have evaluated the impact of lifestyle interventions on nutritional intake. To date, no studies have assessed the impact of preventative nutrition interventions delivered to youth with first-episode psychosis (FEP) and the feasibility and acceptability of such interventions in this vulnerable population.

The aims of this study were as follows: (i) to report the dietary intake of youth with a FEP, (ii) to evaluate the feasibility and acceptability of a 12-week intensive dietitian-delivered nutrition intervention soon after initiation of APM and (iii) to evaluate its effectiveness in reducing energy intake and improving diet quality.

Methods

Design

Dietetic consultations were one component of the multidisciplinary Keeping the Body in Mind (KBIM) lifestyle and life skills intervention that was evaluated in a cluster-controlled study described elsewhere⁽²⁸⁾. The intervention included an exercise physiologist-led physical activity programme and behavioural support, along with the dietetic intervention described below. All clients from one clinical service (Bondi Early Psychosis Program (EPP)) were offered the KBIM package of care. The comparison site (Liverpool Early Psychosis Intervention Program (EPIP)) received treatment as usual involving best-practice early intervention for FEP, without the KBIM lifestyle and life skills intervention. The study received ethics approval from the South Eastern

Sydney Local Health District Human Research Ethics Committee (ref no: 13/040; LNR/13/POWH/85).

Participants/setting

All clients referred to the Bondi EPP, an early intervention service within a community healthcare centre, over a 12-month period, between February 2013 and February 2014, were invited to participate in the on-site nutrition intervention as part of the KBIM programme. The EPP clinical programme includes a multidisciplinary team focused on the recovery of normal life trajectory, using second-generation APM, other psychotropic medications as indicated (e.g. mood stabilisers and antidepressants), combined with community-based case-management and targeted behaviour and group programmes. Clients referred to the Liverpool EPIP served as a control group for the broader lifestyle intervention study; however, baseline and post-intervention nutritional intakes were not assessed at this site. Referral pathways for both EPP and EPIP included (i) acute inpatient units, (ii) general practitioner referral or (iii) transfer from another health service. Inclusion criteria were as follows: (i) first-episode psychosis, (ii) aged 15–25 years old and (iii) less than 1 month since commencement of APM. All participants met criteria for FEP before commencing APM. Those with more than 1 month of treatment with APM were excluded, as such treatment may already have resulted in significant weight gain and/or cardiometabolic disturbances. No eligible participants were prescribed medications to treat cardiometabolic complications including hypercholesterolaemia, type 2 diabetes and hypertension at commencement of the study. Psychiatric diagnoses were confirmed by treating psychiatrists during the course of treatment according to Diagnostic and Statistical Manual of Mental Disorder (DSM-5) criteria⁽³²⁾.

Intervention

The dietitian offered weekly structured individual dietetic consultations for 12 weeks and focused on specific educational modules, utilising motivational interviewing and SMART (Specific, Measurable, Achievable, Relevant, Time Specific) goals⁽³³⁾. A full assessment including medical and physical health history, together with a comprehensive diet history, was completed at the initial appointment. Subsequent consultations ranged from 30 to 60 min in duration and included a 24-h recall, diet history or food diary review plus education module(s) and goal setting. Educational modules delivered to all participants within the individual dietetic consultations targeted weight management, core foods and nutritional adequacy, and recommended serves as per the *Australian Dietary Guidelines*⁽³⁴⁾. Additional topics included meal structure, portion sizes, mindful eating, healthier eating and takeaway choices, label reading and budgeting. These subsequent topics were prioritised by the dietitian based on individual needs.

The dietitian facilitated a weekly group shopping tour and cooking session with assistance from a mental health nurse specialist. The dietitian and group members walked to the local supermarket to purchase ingredients, and then returned to the community centre to prepare lunch. During the weekly

shopping tour and cooking session, education was provided and reinforced regarding nutritional requirements, healthy food choices, fresh food selection, food energy density, salt content, food safety as well as a variety of cooking skills. Both components of the nutrition intervention were offered weekly as part of standard care for people attending the Bondi EPP.

Participants also had the opportunity to take part in a weekly sports group and had access to an on-site gym supervised by an exercise physiologist.

Outcome measures

Energy intake, macronutrient and micronutrient intakes and glycaemic load were derived from a validated semi-quantitative/FFQ, the Dietary Questionnaire for Epidemiological Studies (DQES), developed by the Cancer Council of Victoria (Australia)⁽³⁵⁾. Following an explanation from the dietitian, participants self-completed the DQES based on intake over the last 3 months. Further assistance from the dietitian was provided if requested. Completed DQES forms were returned to the Cancer Council of Victoria for analysis, providing data on outcomes listed above. Feasibility and acceptability of the dietary consultations and group sessions were evaluated by a consumer survey (described elsewhere⁽³⁶⁾), through examination of attrition during the course of the intervention and through the number of consultations and weekly group sessions attended. The Australian Recommended Food Score (ARFS) was used to determine the proximity of core foods intake compared with the *Australian Dietary Guidelines*⁽³⁴⁾. The ARFS is a validated tool for use in the Australian population, producing an overall score, out of a possible 74 points, with reference to core food groups^(37,38). Discretionary food intake was measured separately by daily intake (g).

Statistical analysis methods

Baseline evaluation compared general dietary patterns including energy, fibre, Na, alcohol and discretionary food intakes with estimated energy requirements and national standards for recommended intakes⁽³⁹⁾. BMR and estimated energy requirements were calculated using the Schofield equation⁽⁴⁰⁾. Energy under-reporting was classified as an energy intake (EI):BMR ratio <1.2. The pre-post evaluation assessed change in a range of nutritional factors that can impact on cardiometabolic health, including core food groups, discretionary foods, energy intake (kJ), Na intake (mg) and glycaemic load. Outcome measures were assessed using paired-sample *t* tests. Non-parametric χ^2 analysis was used for categorical variables. Partial η^2 (η_p^2) effect sizes were calculated for mean change scores. Partial η^2 (η_p^2) effect sizes were considered small at 0.10, medium at 0.25 and large at 0.40. Pearson's correlations were calculated between mean change scores and mean number of contacts with clinicians. Repeated-measures ANOVA were calculated for mean change scores in anthropometric measures. Analyses were conducted using SPSS, version 22 package. A Bonferroni's correction was applied to mean change scores assessed before and after intervention.

Results

During the recruitment period, twenty-seven participants completed baseline measures for the 12-week programme (twelve females and fifteen males, mean age = 20.5 (SD 2.4) years). Clients were predominantly Caucasian (*n* 14, 52%), and in decreasing frequency Asian (*n* 5, 19%), Middle Eastern (*n* 3, 11%), Indigenous Australians (*n* 2, 7%), Maori/Pacific Islanders (*n* 2, 7%) and African-Americans (*n* 1, 4%). Diagnoses were as follows: schizophreniform disorder (*n* 12, 45%), bipolar affective disorder (*n* 8, 30%) schizoaffective disorder (*n* 2, 7%), schizophrenia (*n* 2, 7%), major depression with psychotic features (*n* 2, 7%) and brief psychotic disorder (*n* 1, 4%). A range of mood-stabiliser, antipsychotic and antidepressant medications was prescribed. In total, 50% of the sample had unchanged medication for the duration of the intervention. Some changes in antipsychotic dosage occurred for the remaining participants (28% decreased, 17% increased). For one patient, APM was switched during the course of the intervention. Demographic, diagnostic and medication data are provided in Table 1.

Table 1. Characteristics of clients referred to the Bondi Early Psychosis Program and those who completed the intervention (Numbers and percentages; mean values and standard deviations)

| | Referrals (<i>n</i> 27) | | Completers (<i>n</i> 18) | | Statistical test | <i>P</i> |
|---------------------------------|-----------------------------|------|------------------------------|------|----------------------|----------|
| | <i>n</i> | % | <i>n</i> | % | | |
| Demographic | | | | | | |
| Age | | | | | | |
| Mean | | 20.5 | | 20.0 | <i>t</i> (25) = 1.53 | 0.50 |
| SD | | 2.4 | | 2.3 | | |
| Female | 12 | 44 | 9 | 50 | $\chi^2 = 0.68$ | 0.68 |
| Ethnicity | | | | | | |
| Caucasian | 14 | 52 | 9 | 50 | | |
| Indigenous | 2 | 7 | 1 | 6 | | |
| Australians | | | | | | |
| Asian | 5 | 19 | 4 | 22 | $\chi^2 = 4.44$ | 0.49 |
| African-American | 1 | 4 | 0 | 0 | | |
| Maori/Pacific Islander | 2 | 7 | 1 | 6 | | |
| Middle Eastern | 3 | 11 | 3 | 16 | | |
| DSM-IV diagnoses | | | | | | |
| Brief psychotic disorder | 1 | 4 | 0 | 0 | | |
| Schizophreniform | 12 | 45 | 9 | 50 | | |
| Schizophrenia | 2 | 7 | 1 | 6 | $\chi^2 = 7.88$ | 0.16 |
| Schizoaffective disorder | 2 | 7 | 0 | 0 | | |
| Bipolar affective disorder | 8 | 30 | 6 | 33 | | |
| Major depression with psychosis | 2 | 7 | 2 | 11 | | |
| Psychotropic medications | | | | | | |
| Mood-stabiliser | | | | | | |
| Lithium | 4 | 15 | 3 | 17 | $\chi^2 = 0.15$ | 0.59 |
| Sodium valproate | 2 | 7 | 1 | 6 | $\chi^2 = 0.27$ | 1.00 |
| Antipsychotic | | | | | | |
| Risperidone | 6 | 22 | 5 | 28 | $\chi^2 = 0.96$ | 0.63 |
| Quetiapine | 9 | 33 | 5 | 28 | $\chi^2 = 0.32$ | 0.68 |
| Olanzapine | 5 | 19 | 4 | 21 | $\chi^2 = 0.96$ | 0.63 |
| Aripiprazole | 4 | 15 | 1 | 6 | $\chi^2 = 3.67$ | 0.09 |
| Paliperidone | 2 | 7 | 2 | 11 | $\chi^2 = 1.08$ | 0.54 |
| Venlafaxine | 2 | 7 | 0 | 0 | $\chi^2 = 0.27$ | 1.00 |
| Antipsychotic polypharmacy | 2 | 7 | 1 | 6 | $\chi^2 = 0.27$ | 1.00 |
| Antidepressant | | | | | | |
| Citalopram | 2 | 7 | 2 | 11 | $\chi^2 = 1.08$ | 0.54 |
| Escitalopram | 2 | 7 | 2 | 11 | $\chi^2 = 1.08$ | 0.54 |
| Sertraline | 1 | 4 | 0 | 0 | $\chi^2 = 2.07$ | 0.33 |

At baseline, there was no difference between groups in mean weight (KBIM = 67.6 (sd 13.2) kg, control = 75.8 (sd 18.8) kg), BMI (KBIM = 23.2 (sd 3.1) kg/m², control = 24.8 (sd 3.3) kg/m²) or waist circumference (KBIM = 83.4 (sd 10.5) cm, control = 83.0 (sd 9.1) cm) (all *P*-values > 0.39). The multidisciplinary KBIM lifestyle and life skills intervention restricted antipsychotic-induced weight gain to a mean 1.1 kg (95% CI -0.3, 2.5) during the 12-week intervention, whereas the control group had a mean weight gain of 7.8 kg (95% CI 4.8, 10.7), $F_{1,28} = 24.4$ ($P < 0.001$). There was no change in waist circumference in the KBIM group, -0.3 (95% CI -2.3, 1.6) cm, whereas the control group gained a mean 7.1 cm (95% CI 4.8, 9.4), $F_{1,28} = 27.1$ ($P < 0.001$).

All participants completed the DQES at baseline. Baseline energy, discretionary food, Na, glycaemic load and diet quality intakes are described in Table 2. The mean reported energy intake was not significantly different from the estimated energy requirement ($t(26) = -0.4$, $P = 0.67$). Energy was under-reported in 41% ($n = 11$, EI:BMR range 0.63–1.10). After accounting for under-reporting, reported energy intake, 11 385 (sd 3601) kJ, was significantly higher than the estimated energy requirement, 9550 (sd 1574) kJ, 1833 (sd 3067) kJ, $t(15) = 2.39$, $P = 0.03$.

Two-thirds of the participants (nine females and nine males, mean age = 20.0 (sd 2.1) years; range 17–24 years) remained engaged at the completion of the 12-week nutrition intervention. Reasons for disengagement included the following: five were unable to be contacted, three discontinued treatment with the mental healthcare service, one was re-admitted to inpatient care during intervention and one was incarcerated during the

intervention period. There were no significant differences in age ($P = 0.5$), sex ($P = 0.7$), ethnicity ($P = 0.5$) or diagnoses ($P = 0.2$) between those who remained engaged in the programme and those who disengaged. There was no difference in rates of mood-stabiliser, antidepressant, antipsychotic prescription or antipsychotic polypharmacy between those who remained engaged in the programme and those who disengaged ($P \geq 0.09$).

The mean number of contacts with the dietitian was 8.2 (median = 7.5, range 5–12) (individual consultations = 6.1 (median = 6.5, range 2–12) and shopping/cooking group = 2.1 (median = 2, range 0–5)). The mean number of contacts with the exercise physiologist was 10.9 (median = 11, range 0–25). There was a trend to significance between the number of contacts with the dietitian and the number of contacts with the exercise physiologist ($r = 0.46$, $P = 0.05$).

At completion of the 12-week programme, significant quantitative and qualitative changes were evident. Daily energy intake was reduced by 1956 kJ (95% CI -2897, -1020, $\eta_p^2 = 0.51$, $P < 0.001$). This was reflected in reductions in carbohydrate (-54 g/d, $P < 0.001$), sugar (-15 g/d, $P = 0.08$), protein (-13 g/d, $P = 0.04$) and fat (-22 g/d, $P = 0.001$) intakes. Fibre intake was not significantly different before and after evaluations. Energy was under-reported in 50% of the participants who remained engaged at the 12-week point ($n = 9$; EI:BMR range 0.82–1.10). There was no relationship between energy underestimation at baseline and change in weight over the 12-week intervention ($R = 0.23$, $P = 0.37$) (Table 3).

After the 12-week programme, discretionary food intake reduced by 47% (-94.0 g, 95% CI -138.9, -48.3 g, $\eta_p^2 = 0.53$,

Table 2. Baseline nutritional intake for twenty-seven participants (all participants), compared with sixteen participants (accounting for under-reporting), and recommended intakes

| Unit of measure | Baseline results | | | | Baseline results (accounting for under-reporting) | | | | Estimated energy requirement recommended daily intake ⁽⁴¹⁾ | |
|---------------------|------------------|----|---------------|----|---|----|--------------|----|---|--------------------|
| | Male (n 15) | | Female (n 12) | | Male (n 9) | | Female (n 7) | | Male | Female |
| | n | % | n | % | n | % | n | % | | |
| Energy | 11 102 | | 7260 | | 13 624 | | 8503 | | 10 989 | 8202 |
| Discretionary foods | 4218 | 38 | 2613 | 36 | 4866 | 36 | 3107 | 37 | Limit | Limit |
| Fibre | 25 | | 19 | | 26.1 | | 23.0 | | 30 | 25 |
| Na | 3162 | | 2253 | | 4322 | | 2598 | | 2300 ^{UL} | 2300 ^{UL} |
| Alcohol | 10 | | 8 | | | | | | 20 | 20 |

^{UL}, Upper limit.

Table 3. Mean change of eighteen participants from baseline to after the 12-week intervention (Mean values, standard deviations and 95% confidence intervals)

| | Mean at baseline | sd | Mean at 12 weeks | sd | Mean change | 95% CI | Paired-sample <i>t</i> test | Effect size |
|---------------------------|------------------|------|------------------|------|-------------|---------------|-------------------------------|-------------------|
| Energy (kJ/d) | 8263 | 2205 | 6306 | 1858 | -1958 | -2900, -1021 | | |
| Energy (kcal/d) | 1975 | 527 | 1507 | 444 | -468 | -693, -244 | $t(17) = 4.4$, $P < 0.001^*$ | $\eta_p^2 = 0.51$ |
| Discretionary foods (g) | 200.3 | 77.6 | 106.3 | 69.5 | -94.0 | -139.8, -48.3 | $t(17) = 4.3$, $P < 0.001^*$ | $\eta_p^2 = 0.53$ |
| Na (mg) | 2708 | 791 | 1995 | 648 | -713 | -1063, -364 | $t(17) = 4.3$, $P < 0.001^*$ | $\eta_p^2 = 0.52$ |
| Glycaemic load | 113.5 | 37.1 | 80.0 | 25.7 | -33.5 | -47.5, -19.5 | $t(17) = 5.0$, $P < 0.001^*$ | $\eta_p^2 = 0.60$ |
| Diet quality score (ARFS) | 28.1 | 11.4 | 31.6 | 11.8 | 3.6 | 0.2, 6.9 | $t(17) = 2.3$, $P = 0.038$ | $\eta_p^2 = 0.23$ |

ARFS, Australian Recommended Food Score.

* Statistically significant after applying Bonferroni's correction.

Table 4. Mean change in diet quality score (Australian Recommended Food Score) by subgroup (Mean values, standard deviations and 95% confidence intervals)

| | Maximum score | Mean at baseline | sd | Mean at 12 weeks | sd | Mean change | 95% CI | Paired-sample <i>t</i> test |
|----------------|---------------|------------------|------|------------------|------|-------------|-------------|-----------------------------|
| Vegetable | 22 | 8.44 | 5.40 | 10.94 | 6.15 | 2.50 | 0.26, 4.74 | $t(17) = 2.36, P = 0.03$ |
| Fruit | 14 | 5.44 | 3.33 | 5.33 | 3.31 | -0.11 | -1.36, 1.14 | $t(17) = 0.19, P = 0.85$ |
| Grain | 14 | 4.89 | 1.38 | 5.17 | 1.25 | 0.28 | -0.52, 1.07 | $t(17) = 0.74, P = 0.47$ |
| Protein | 14 | 5.28 | 3.03 | 5.72 | 2.22 | 0.44 | -0.54, 1.43 | $t(17) = 0.95, P = 0.35$ |
| Dairy products | 7 | 2.28 | 1.07 | 2.50 | 1.15 | 0.22 | -0.25, 0.69 | $t(17) = 1.00, P = 0.33$ |
| Fat | 1 | 0.50 | 0.51 | 0.39 | 0.50 | -0.11 | -0.35, 0.12 | $t(17) = 1.00, P = 0.33$ |
| Alcohol | 2 | 1.22 | 0.73 | 1.56 | 0.62 | 0.33 | -0.01, 0.67 | $t(17) = 2.06, P = 0.06$ |

$P < 0.001$), with associated reductions in Na intake, -713 mg (95% CI $-1063, -364, \eta_p^2 = 0.52, P < 0.001$), and glycaemic load, -33.5 (95% CI $-47.5, -19.5, \eta_p^2 = 0.60, P < 0.001$). Reductions in discretionary food intake correlated with reductions in overall energy intake ($r = 0.57, P = 0.01$). There was an increase in overall diet quality score (3.6 (95% CI 0.3, 6.8, $\eta_p^2 = 0.23, P = 0.03$)), not statistically significant after applying Bonferroni's correction for multiple comparisons.

To further examine the components of diet quality, an analysis of diet quality subgroups was performed, with results shown in Table 4. The trend in improved diet quality score was driven by increased vegetable variety and frequency (2.4 (95% CI 0.3, 1.0, $P = 0.031$)).

The correlation between greater contact with the dietitian and lower energy intake approached significance ($r = 0.45, P = 0.06$), and there was also a trend for the relationship between lower Na intake and number of contacts with the exercise physiologist ($r = 0.46, P = 0.05$). The magnitude of change in weight, waist circumference, discretionary food, glycaemic load and diet quality was not significantly correlated with contact with either the dietitian or the exercise physiologist (all r -values < 0.45 , all P -values > 0.06).

Discussion

The present study demonstrated that (i) the dietary intake in youth early in their first episode of psychosis appears to be higher than required, driving the rapid weight gain seen with the commencement of APM, (ii) a 12-week dietary intervention was feasible and accepted by the majority of clients and (iii) the intervention was effective in improving dietary intake.

At baseline, reported energy intake did not differ significantly from estimated energy requirement. However, once accounted for under-reporting, reported energy intake was significantly higher than estimated energy, coinciding with frequent reporting of increased hunger in those commencing antipsychotic treatment, leading to rapid weight gain, similar to that seen in the control group. Future studies utilising weighed food records in the early stages of FEP treatment are required to confirm these findings.

A mean contact with the dietitian of 8.2, range 5–12, suggests that participants accepted the intervention. The individual consultations had a higher attendance rate compared with the cooking group. The low attendance rate in the cooking group may be explained in part by the session being offered at a set

time each week, in contrast to the individual consultations, which were tailored to the participants' availability.

The results of the intensive 12-week intervention suggest that overall intake of food in this target group can be improved, with poor-quality, energy-dense, discretionary foods replaced by core foods such as vegetables. The reduction in energy intake provides a means to attenuate antipsychotic-induced weight gain. Indeed, the multifaceted KBIM program demonstrated that weight gain could be prevented in the majority of youth commencing APM⁽²⁸⁾.

The 12-week intervention also resulted in a reduction in Na intake, a proven risk factor for hypertension and heart disease. Na intake was reduced to within the Australian recommended upper limit of intake (< 2300 mg/d)⁽²⁴⁾.

Although intake of discretionary foods, which are characterised by high-sugar, high-fat, non-nutritious foods, decreased significantly, it was not possible to assess the change in added sugar consumption. Future studies should assess the impact of lifestyle intervention on added sugar with recent studies suggesting a link to CVD⁽⁴¹⁾ and recent changes in World Health Organization⁽⁴²⁾ recommendations.

A recent study in enduring mental illness demonstrated that a telephone-delivered nutrition intervention reduced inactivity and improved fruit and vegetable intake, as well as overall diet quality in those with established illness⁽⁴³⁾. Importantly, the study also found significant increases in quality of life and global functioning. Although there were increases in participant vegetable intake in our study, fruit intake remained relatively unchanged. This may have been due to participants already meeting the recommendations of 2 servings of fruit/d at baseline assessment. In addition to monetary reimbursement and free fruit and vegetables boxes provided in Baker *et al.*'s⁽⁴⁵⁾ study, inclusion criteria specified only those consuming < 7 fruit and vegetable serves/d. This may account, in part, for the large effect sizes seen in the study. Future studies assessing the cost-benefits of differing delivery methods would assist the design and delivery of nutrition interventions into standard practice.

At present, there are no recommended ranges for diet quality score when using the Australian Recommended Food Score. Mean diet quality scores in FEP clients were found to be lower than mean scores in established illness⁽⁴³⁾ and in Australian adult women of the general population⁽⁴⁴⁾.

This study has a number of limitations, most notably the lack of age-, sex- and socio-demographic characteristic-matched controls. This was due to the pragmatic nature of this study, which aimed to evaluate the effectiveness of a real-world

intervention offered to all clients of a service, such that randomisation to a comparison condition could not be undertaken. Although there is considerable evidence of decline in physical health during in the first 12 weeks of antipsychotic therapy^(5,45), it is likely that nutritional status declines in parallel as weight gain and metabolic deterioration develop.

Although there was potential for a Hawthorne effect⁽⁴⁶⁾, there were no significant relationships between contact with the dietitian or exercise physiologist and dietary improvement. Similar to the majority of nutrition assessment tools, the responses to the dietary questionnaire were self-reported, potentially reducing accuracy and increasing risk of bias. Although the study had a relatively small sample size, the data showed clear improvements across a number of nutritional parameters.

In total, 67% successfully completed 12 weeks of intensive intervention, demonstrating the feasibility and acceptability of the intervention. Youth with FEP can be difficult to engage, with 30% attrition rates for a range of interventions offered in FEP services⁽⁴⁷⁾, and these drop-out rates are similar to other lifestyle programmes conducted in people without SMI^(48,49).

Clinicians delivering the intervention also noted a number of potential benefits as a result of the nutrition intervention that warrant further investigation. Having a nutrition programme embedded within the EPP was associated with increased attendance by clients at the community centre, allowing additional opportunities for client contact by mental health clinicians. Peer contact within the cooking group may have contributed to improved social skills and self esteem. Previous studies^(50,51) have demonstrated that improved diet quality can increase psychosocial function in those with depression. Correlations between diet quality and psychosocial function scores in psychotic illness have not been evaluated, and future studies should assess the relationship between diet quality and symptomatology.

Conclusion

The integration of dietetic services into community early psychosis programmes is feasible and acceptable to youth with FEP with high attendance rates over a short-term period of 12 weeks. If dietary changes are sustained, intensive and early nutrition intervention may reduce future risk of cardiometabolic disease in SMI, helping meet the targets identified in the Healthy Active Lives international declaration for youth with psychosis (www.iphys.org.au). These targets aim to create a world where 'young people experiencing psychosis have the same life expectancy and expectations of life as their peers who have not experienced psychosis'. Early intervention in the first episode of psychosis may have a greater long-term impact on the health of people with SMI and represents a greater return on clinician effort than our current practice of late addressing of poor metabolic health in established chronic mental illness.

Acknowledgements

The authors acknowledge staff and clients of the Bondi EPP for their assistance with this study.

The Mental Health and Drug & Alcohol Office (MHDAO), Ministry of Health, NSW, North Sydney, Australia provided funding. The MHDAO had no role in the design, analysis or writing of this article.

The KBIM programme was designed by J. C., P. B. W. and K. S., who also obtained funding. A. W., J. C. and K. S. provided clinical supervision. S. B. T. delivered the nutrition intervention and collected data. S. B. T. analysed the data with input from S. R., K. S. and P. B. W. S. B. T., S. R., A. W., K. S. and P. B. W. interpreted the data. S. B. T. led the manuscript preparation with input from corresponding authors. All authors contributed to the intellect of the manuscript.

The authors declare that there are no conflicts of interest.

References

1. Thornicroft G (2011) Physical health disparities and mental illness: the scandal of premature mortality. *Br J Psychiatry* **199**, 441–442.
2. Lawrence D, Hancock KJ & Kisely S (2013) The gap in life expectancy from preventable physical illness in psychiatric patients in Western Australia: retrospective analysis of population based registers. *BMJ* **346**, f2539.
3. McGorry P, Killackey E, Lambert T, *et al.* (2005) Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of schizophrenia and related disorders. *Aust N Z J Psychiatry* **39**, 1–30.
4. McGorry P, Killackey E & Yung A (2007) Early intervention in psychotic disorders: detection and treatment of the first episode and the critical early stages. *Med J Aust* **187**, S8–S10.
5. Álvarez-Jiménez M, González-Blanch C, Crespo-Facorro B, *et al.* (2008) Antipsychotic-induced weight gain in chronic and first-episode psychotic disorders. *CNS Drugs* **22**, 547–562.
6. Correll CU, Manu P, Olshansky V, *et al.* (2009) Cardiometabolic risk of second-generation antipsychotic medications during first-time use in children and adolescents. *JAMA* **302**, 1765–1773.
7. Miller B, Paschall CB & Svendsen D (2006) Mortality and medical comorbidity among patients with serious mental illness. *Psychiatr Serv* **57**, 1482–1487.
8. Newcomer JW (2006) Antipsychotic medications: metabolic and cardiovascular risk. *J Clin Psychiatry* **68**, 8–13.
9. Hennekens CH, Hennekens AR, Hollar D, *et al.* (2005) Schizophrenia and increased risks of cardiovascular disease. *Am Heart J* **150**, 1115–1121.
10. Blanchard E & Samaras K (2014) Double jeopardy: diabetes and severe mental illness. Addressing the special needs of this vulnerable group. *Diabetes Manag* **4**, 339–353.
11. Dipasquale S, Pariante CM & Dazzan P (2013) The dietary pattern of patients with schizophrenia: a systematic review. *J Psychiatr Res* **47**, 197–207.
12. Jerome GJ, Rohm Young D, Dalcin A, *et al.* (2009) Physical activity levels of persons with mental illness attending psychiatric rehabilitation programs. *Schizophr Res* **108**, 252–257.
13. Cooper J, Mancuso SG & Borland R (2012) Tobacco smoking among people living with a psychotic illness: the second Australian Survey of Psychosis. *Aust N Z J Psychiatry* **46**, 851–863.
14. Bahorik AL, Newhill C & Queen C (2014) Under-reporting of drug use among individuals with schizophrenia: prevalence and predictors. *Psychol Med* **44**, 61–69.
15. Treuer T, Hoffmann VP, Chen AK-P, *et al.* (2009) 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* **10**, 729–740.
16. Blouin M, Tremblay A, Jalbert ME, *et al.* (2008) Adiposity and eating behaviors in patients under second generation antipsychotics. *Obesity (Silver Spring)* **16**, 1780–1787.
 17. DeMyer MK, Ward SD & Lintzenich J (1968) Comparison of macronutrients in the diets of psychotic and normal children. *Arch Gen Psychiatry* **18**, 584–590.
 18. Gothelf D, Falk B, Singer P, *et al.* (2002) Weight gain associated with increased food intake and low habitual activity levels in male adolescent schizophrenic inpatients treated with olanzapine. *Am J Psychiatry* **159**, 1055–1057.
 19. Strassnig M, Brar JS & Ganguli R (2003) Nutritional assessment of patients with schizophrenia: a preliminary study. *Schizophr Bull* **29**, 393.
 20. McCreadie RG (2003) Diet, smoking and cardiovascular risk in people with schizophrenia descriptive study. *Br J Psychiatry* **183**, 534–539.
 21. Swinburn BA, Caterson I, Seidell JC, *et al.* (2004) Diet, nutrition and the prevention of excess weight gain and obesity. *Public Health Nutr* **7**, 123–146.
 22. Cuerda C, Velasco C, Merchán-Naranjo J, *et al.* (2013) The effects of second-generation antipsychotics on food intake, resting energy expenditure and physical activity. *Eur J Clin Nutr* **68**, 146–152.
 23. Sharpe J-K, Byrne NM, Stedman TJ, *et al.* (2005) Resting energy expenditure is lower than predicted in people taking atypical antipsychotic medication. *J Am Diet Assoc* **105**, 612–615.
 24. National Heart Foundation of Australia (2006) Position statement: the relationship between dietary electrolytes and cardiovascular disease. <http://www.heartfoundation.org.au/CollectionDocuments/Dietary-Electrolytes-CVD-Position-Statement.pdf> (accessed February 2015).
 25. Barclay AW, Petocz P, McMillan-Price J, *et al.* (2008) Glycemic index, glycemic load, and chronic disease risk – a meta-analysis of observational studies. *Am J Clin Nutr* **87**, 627–637.
 26. Mirrahimi A, de Souza RJ, Chiavaroli L, *et al.* (2012) Associations of glycemic index and load with coronary heart disease events: a systematic review and meta-analysis of prospective cohorts. *J Am Heart Assoc* **1**, e000752.
 27. Alvarez-Jimenez M, Gonzalez-Blanch C, Vazquez-Barquero JL, *et al.* (2006) Attenuation of antipsychotic-induced weight gain with early behavioral intervention in drug-naive first-episode psychosis patients: a randomized controlled trial. *J Clin Psychiatry* **67**, 1253–1260.
 28. Curtis J, Watkins A, Rosenbaum S, *et al.* (2015) Evaluating an individualized lifestyle and lifeskills intervention to prevent antipsychotic-induced weight gain in first-episode psychosis. *Early Interv Psychiatry* (epublication ahead of print version 26 February 2015).
 29. Skouroliakou M, Giannopoulou I, Kostara C, *et al.* (2009) Effects of nutritional intervention on body weight and body composition of obese psychiatric patients taking olanzapine. *Nutrition* **25**, 729–735.
 30. Álvarez-Jiménez M, Hetrick SE & González-Blanch C (2008) Non-pharmacological management of antipsychotic-induced weight gain: systematic review and meta-analysis of randomised controlled trials. *Br J Psychiatry* **193**, 101–107.
 31. Bruins J, Jörg F, Brüggeman R, *et al.* (2014) The effects of lifestyle interventions on (long-term) weight management, cardiometabolic risk and depressive symptoms in people with psychotic disorders: a meta-analysis. *PLOS ONE* **9**, e112276.
 32. American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders (DSM-5[®])*. Washington, DC: American Psychiatric Publishing.
 33. Doran GT (1981) There's a SMART way to write management's goals and objectives. *Manag Rev* **70**, 35–36.
 34. National Health and Medical Research Council (2013) *Australian Dietary Guidelines*. Canberra: NHMRC.
 35. Giles G & Ireland P (1996) *Dietary Questionnaire for Epidemiological Studies (Version 2)*. Melbourne: Cancer Council Victoria.
 36. Teasdale SB, Rosenbaum S, Watkins A, *et al.* (2015) Preventing antipsychotic-induced weight gain in first-episode psychosis: transitioning dietitians into routine care. *Nutr Diet* (epublication 30 October 2015).
 37. Collins C, Watson J, Burrows T, *et al.* (2011) *Validation of an Adult Food Frequency Questionnaire and Development of a Diet Quality Food Score for Children and Adults*. Newcastle: University of Newcastle.
 38. Wirt A & Collins CE (2009) Diet quality – what is it and does it matter? *Public Health Nutr* **12**, 2473–2492.
 39. National Health and Medical Research Council (2006) *Nutrient Reference Values for Australia and New Zealand*. Canberra: NHMRC.
 40. Schofield WN (1984) Predicting basal metabolic rate, new standards and review of previous work. *Hum Nutr Clin Nutr* **39**, 5–41.
 41. Yang Q, Zhang Z, Gregg EW, *et al.* (2014) Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* **174**, 516–524.
 42. World Health Organisation (2015) *Sugars Intake for Adult and Children: Guideline*. Geneva: WHO.
 43. Baker AL, Turner A, Kelly PJ, *et al.* (2014) 'Better Health Choices' by telephone: a feasibility trial of improving diet and physical activity in people diagnosed with psychotic disorders. *Psychiatr Res* **220**, 63–70.
 44. Collins CE, Young AF & Hodge A (2008) Diet quality is associated with higher nutrient intake and self-rated health in mid-aged women. *J Am Coll Nutr* **27**, 146–157.
 45. Correll CU, Robinson DG, Schooler NR, *et al.* (2014) Cardio-metabolic risk in patients with first-episode schizophrenia spectrum disorders: baseline results from the RAISE-ETP study. *JAMA Psychiatry* **71**, 1350–1363.
 46. Roethlisberger F, Dickson W, Wright HA, *et al.* (1939) *Management and the Worker: An Account of a Research Program Conducted by the Western Electric Company, Hawthorne Works, Chicago*. Cambridge, MA: Harvard University Press.
 47. Doyle R, Turner N, Fanning F, *et al.* (2014) First-episode psychosis and disengagement from treatment: a systematic review. *Psychiatr Serv* **65**, 603–611.
 48. Evans S, Newton R & Higgins S (2005) Nutritional intervention to prevent weight gain in patients commenced on olanzapine: a randomized controlled trial. *Aust N Z J Psychiatry* **39**, 479–486.
 49. Kwon JS, Choi JS, Bahk WM, *et al.* (2006) Weight management program for treatment-emergent weight gain in olanzapine-treated patients with schizophrenia or schizoaffective disorder: a 12-week randomized controlled clinical trial. *J Clin Psychiatry* **67**, 547–553.
 50. Jacka FN, Kremer PJ, Berk M, *et al.* (2011) A prospective study of diet quality and mental health in adolescents. *PLoS ONE* **6**, e24805.
 51. Kulkarni A, Swinburn B & Utter J (2015) Associations between diet quality and mental health in socially disadvantaged New Zealand adolescents. *Eur J Clin Nutr* **69**, 79–83.