British Journal of Nutrition (2010), **103**, 1223–1229
doi:10.1017/S0007114509992893

Diet quality and obesity in women: the Framingham Nutrition Studies

Dolores M. Wolongevicz[1], Lei Zhu[2], Michael J. Pencina[2], Ruth W. Kimokoti[1], P. K. Newby[3], Ralph B. D'Agostino[2] and Barbara E. Millen[1,4]*

[1]*Department of Family Medicine and the Graduate Medical Sciences Division, Boston University School of Medicine, Boston, MA, USA*
[2]*Department of Mathematics, Boston University, Boston, MA, USA*
[3]*Departments of Pediatrics and Epidemiology, Boston University Schools of Medicine and Public Health, Boston, MA, USA*
[4]*Boston Nutrition Foundation, Inc., Westwood, MA, USA*

(Received 10 June 2009 – Revised 5 October 2009 – Accepted 14 October 2009 – First published online 24 November 2009)

Obesity affects one in three American adult women and is associated with overall mortality and major morbidities. A composite diet index to evaluate total diet quality may better assess the complex relationship between diet and obesity, providing insights for nutrition interventions. The purpose of the present investigation was to determine whether diet quality, defined according to the previously validated Framingham nutritional risk score (FNRS), was associated with the development of overweight or obesity in women. Over 16 years, we followed 590 normal-weight women (BMI $< 25\,kg/m^2$), aged 25 to 71 years, of the Framingham Offspring and Spouse Study who presented without CVD, cancer or diabetes at baseline. The nineteen-nutrient FNRS derived from mean ranks of nutrient intakes from 3 d dietary records was used to assess nutritional risk. The outcome was development of overweight or obesity (BMI $\geq 25\,kg/m^2$) during follow-up. In a stepwise multiple logistic regression model adjusted for age, physical activity and smoking status, the FNRS was directly related to overweight or obesity (P for trend=0·009). Women with lower diet quality (i.e. higher nutritional risk scores) were significantly more likely to become overweight or obese (OR 1·76; 95 % CI 1·16, 2·69) compared with those with higher diet quality. Diet quality, assessed using a comprehensive composite nutritional risk score, predicted development of overweight or obesity. This finding suggests that overall diet quality be considered a key component in planning and implementing programmes for obesity risk reduction and treatment recommendations.

Diet quality: Nutritional risk score: Obesity: BMI: Dietary quality index

Obesity has reached epidemic proportions in the USA and affects one in three adult women aged 20 years or older[1]. Recent estimates indicate that 62 % of women are overweight or obese[1] (BMI $\geq 25\,kg/m^2$) and experts predict that obesity will soon surpass tobacco and become the leading cause of preventable death in the USA[2]. Women may be at greater risk of obesity than men. The sex difference is possibly related to differences in dietary patterns[3,4], as well as physiological (for example, decreased RMR) and behavioural (for example, reduced physical activity) changes associated with ageing[5], higher levels of body fat and the ability to store more fat[6], fluctuations in sex hormone concentrations[7], dysregulation of serotonin[8] and higher leptin levels[9]. More noteworthy is that women may also experience a larger burden of adverse health outcomes attributable to the disease, including CVD, hypertension, dyslipidaemia, respiratory and pulmonary disturbances, type 2 diabetes and osteoarthritis[10,11]. The direct and indirect costs of obesity in the USA are estimated to be more than $117 billion annually[12].

At present, guidelines for obesity prevention and treatment exist[13–17], but there is no consensus on an appropriate evidence-based preventive and treatment nutrition model or dietary interventions to sustain long-term weight loss (>1 year). It has been established that very-low-fat[18], low-fat[19] and low-carbohydrate[20] diets produce short-term weight loss, but evidence suggests that their long-term ineffectiveness may relate to their restrictive nature or deviation from the habitual, sustainable eating patterns of individuals[21]. Increasingly, experts are urging for the development of innovative nutrition intervention strategies that are guided by research which better characterises the links between the characteristics and quality of habitual long-term eating behaviours and obesity-related outcomes[12,22]. Epidemiological investigations that utilise comprehensive measures of diet exposures, such as dietary patterns and diet quality indices, may offer new insights into nutrition intervention planning for obesity risk reduction. Most research on diet quality indices and weight-related outcomes to date is cross-sectional in nature[23–32]. Of the two prospective studies that have been conducted, one did not assess obesity status[33] and the other did not stratify obesity results by sex[34].

The present study prospectively examined the relationship between diet quality and the development of overweight or

Abbreviations: Exam 3, third examination; FNRS, Framingham nutritional risk score; FOS, Framingham Offspring and Spouse Study.

* **Corresponding author:** Dr Barbara E. Millen, fax + 1 781 326 2217, email bmillen@bu.edu

obesity in women over a broad age range, a subject that is not adequately addressed in the literature. In the present investigation, we evaluated whether a previously validated, global diet index, the Framingham nutritional risk score (FNRS)[3], predicted the development of overweight or obesity over 16 years in healthy, normal-weight (BMI $< 25\,kg/m^2$) women.

Methods

Participants

The cohort for this analysis is women of the Framingham Offspring and Spouse Study (FOS). Detailed methods have been described previously[35]. Briefly, in 1948 the Framingham Study was initiated to identify factors contributing to CVD development and to study the progression of CVD among residents from the town of Framingham (MA, USA). The original cohort consisted of 5209 men and women, aged 28–62 years, representing a two-thirds random sample of Framingham residents. The FOS began in 1971 and includes adult children and their spouses of the original Framingham Study cohort. This second-generation group of 5124 offspring and spouses are examined, on average, every 4 years. At each examination, following a standardised protocol, participants provide an updated, detailed medical history and undergo a complete physical examination with laboratory and non-invasive diagnostic testing. Of the 1956 women who attended the third examination (Exam 3; 1984–88), 1265 (65 %) had completed a 3 d dietary record[3]. Of those 1265 women, 590 were normal weight (BMI $< 25\,kg/m^2$), aged 25 to 71 years, without CVD (including CHD and stroke), cancer, or diabetes at baseline (Exam 3) and comprise the study sample for these analyses. Follow-up was assessed through to Exam 7 (1998–2001) for a total of 16 years.

The present study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the Human Subjects Institutional Review Board of the Boston University Medical Campus and Boston Medical Center. Written informed consent was obtained from all subjects.

Diet assessment and nutritional risk score

Diet was assessed from 3 d dietary records completed according to standardised research protocols[36,37]. At the Exam 3 clinic visit, participants were instructed by a registered dietitian to record all foods consumed over 2 weekdays and 1 weekend day with no deviation in their current eating habits. To quantify portion sizes, participants were trained using a validated two-dimensional pictorial food portion model[37]. The dietary records were reviewed and coded by trained coders following formal protocols. Nutrient intake calculations were performed using the Minnesota Nutrition Data System software (NDS version 2.6, Food Database 6A, Nutrient Database 23; Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN, USA). For each woman, 3 d mean nutrient intake estimates were determined. Diet exposure is considered Exam 3 for these analyses because this was the time point that the 3 d dietary records were collected.

The FNRS is a validated nutritional risk score to assess diet quality and is comprised of nineteen nutrients which include total energy, protein, total fat, monounsaturated, polyunsaturated and saturated fats, carbohydrate, fibre, alcohol, dietary cholesterol, Na, Ca, Se, vitamins C, B_6, B_{12} and E, folate and β-carotene[3]. Originally, these nutrients were selected for their relation to CVD risk following a review of the literature on diet and CVD. Macronutrients and alcohol are presented as a percentage of total energy, and fibre, dietary cholesterol and micronutrients are presented per 4184 kJ (1000 kcal). Although research has found monounsaturated fat to be beneficial[38], the rationale for ranking this fat subtype as a risk nutrient rather than a protective nutrient in the FNRS was done because the majority of monounsaturated fat consumed by FOS women in the mid-1980s was derived from animal rather than plant sources.

The FNRS is calculated using a ranking system of intakes of each nutrient. The ranking of individual nutrients in the score is based on the number of women in the sample (*n* 590) where each nutrient is ranked from 1 (low risk) to 590 (high risk) for each woman with completed 3 d dietary records. Ranks are assigned so that a woman with a desirable nutrient intake level (protective nutrients) receives a lower rank while a woman with an undesirable nutrient intake level (risk-promoting nutrients) receives a higher rank. Energy, protein, alcohol, total, saturated, and monounsaturated fats, dietary cholesterol, and Na intakes were ranked low to high, whereas polyunsaturated fat, carbohydrate, fibre, Ca, Se, vitamins C, B_6, B_{12}, and E, folate and β-carotene intakes were ranked high to low. The mean ranks of each individual nutrient are used to calculate the overall dietary risk score of each woman. These composite scores are then ranked and categorised into tertiles.

Outcome measure

Body weight was measured at clinic visits with a calibrated spring balance scale. Participants were weighed wearing lightweight hospital gowns. Standing height was measured using a stationary anthropometer with participants standing erect and head positioned in the Frankfurt plane. The main outcome was development of overweight or obesity at any time point during follow-up. To maintain statistical power, overweight and obesity were considered together. Overweight or obesity was defined according to the recommended BMI category (BMI $\geq 25\,kg/m^2$)[14].

Covariates

Anthropometric and metabolic measurements are routinely collected at clinic visits according to validated, published methods[39,40]: waist circumference at Exam 4, 1988–92, systolic and diastolic blood pressure (duplicate measurements), fasting lipids (total cholesterol, HDL, LDL, TAG) and fasting glucose.

Sociodemographic and behavioural characteristics are also assessed at clinic visits. Self-reported dietary behaviour was evaluated using the Framingham food habit questionnaire and included adherence to a modified diet (currently 'on a diet') and usual weight pattern described as stable (± 2·7 kg (5 pounds)) or fluctuating (± 4·5 kg (10 pounds)) body weight. Other self-reported characteristics included age, smoking status, parity, menopausal status, use of hormone replacement therapy, use of hypertension or lipid medications and physical activity.

Physical activity was assessed using a standardised questionnaire[41] at Exam 2 (1979–83) and not at Exam 3; these values were used in the analyses consistent with published Framingham protocols[33].

Statistical analysis

Age-adjusted mean levels of baseline characteristics and nutrient intakes were computed for each nutritional risk score tertile. The general linear model (GLM) procedure in SAS (analysis of covariance; SAS Institute, Cary, NC, USA) was used to compute the age-adjusted means for continuous variables. Logistic regression (LOGISTIC procedure in SAS; SAS Institute, Inc.) was used to compute age-adjusted proportions for dichotomous variables. Bonferroni's correction was used to adjust for multiple comparisons in analyses of baseline characteristics and nutrient intakes. The primary research goal was to examine the association between diet quality, assessed by the FNRS, and the development of overweight or obesity. Stepwise multiple logistic regression was used to evaluate variables that were related to overweight or obesity. Covariates that were considered include age, physical activity, menopausal status, smoking status and parity. Metabolic, anthropometric and demographic variables that did not differ at baseline according to nutritional risk score tertile were not identified to be confounders and were thus not added to the model. The final model adjusted for age (continuous), physical activity (continuous) and smoking status (never, former or current smokers). The main analyses are not adjusted for total energy because it is a component of the nutritional risk score and all nutrients are energy-adjusted in the index using the nutrient-density method. OR were calculated for each nutritional risk score tertile of the FNRS, with the lowest tertile as the referent group. The *P* value for trend was determined using the tertile groups of the FNRS in a continuous form. For main outcome statistical testing, α was set at 0·05.

Secondary analyses consistent with our primary diet quality model which adjusted for age, physical activity and smoking status were conducted to determine the relationship of individual risk score components to overweight or obesity. Intakes of each index component were ranked low to high and categorised into tertiles and OR were calculated for each intake tertile, with the lowest tertile as the referent group. The *P* value for trend was determined using the tertile groups of intake for each individual nutrient in a continuous form. For these *post hoc* analyses, α was set at 0·05.

All analyses were performed using SAS software (version 9.1, 2003; SAS Institute, Inc.).

Table 1. Baseline characteristics of 590 healthy, normal-weight (BMI $< 25\,kg/m^2$) women in the Framingham Offspring and Spouse Study according to diet quality*

(Least squares (LS) means and 95 % confidence intervals or percentages)

	Framingham nutritional risk score†					
	Higher diet quality Tertile 1 (*n* 197)		Tertile 2 (*n* 194)		Lower diet quality Tertile 3 (*n* 199)	
Characteristic	LSmean	95 % CI	LSmean	95 % CI	LSmean	95 % CI
Age (years)	48·2[a]	46·8, 49·5	46·3[a,b]	45·0, 47·6	45·7[b]	44·4, 47·1
Weight (kg)‡	58·9	58·0, 59·7	58·2	57·4, 59·1	58·8	57·9, 59·6
BMI (kg/m^2)	22·0	21·7, 22·2	21·9	21·7, 22·2	22·3	22·0, 22·5
Waist circumference (cm)	73·3	72·3, 74·3	73·5	72·4, 74·5	75·0	74·0, 76·0
Physical activity index§	34·1	33·5, 34·8	33·4	32·8, 34·1	34·1	33·4, 34·7
Current smoker (%)		14·3[a]		19·8[a]		41·1[b]
Smoking (pack years)	6·3[a]	4·0, 8·5	9·8[a]	7·5, 12·0	15·2[b]	12·9, 17·4
Current dieter (%)		90·2		94·3		94·8
Fluctuating weight (%)		7·9		7·5		13·4
Postmenopausal (%)		47·9		41·0		38·5
On hormone replacement therapy (%)		3·6		6·1		4·6
Parity (no. of births)	2·3	2·1, 2·5	2·4	2·2, 2·7	2·5	2·3, 2·7
Systolic blood pressure (mmHg)	116·5	114·6, 118·4	117·1	115·2, 119·0	116·6	114·7, 118·5
Diastolic blood pressure (mmHg)	74·2	73·0, 75·4	74·8	73·6, 75·9	74·4	73·3, 75·6
Hypertension treatment (%)		7·7		9·1		8·1
Total cholesterol (mmol/l)‖	5·19	5·07, 5·32	5·24	5·12, 5·36	5·29	5·17, 5·41
HDL-cholesterol (mmol/l)	1·54	1·49, 1·59	1·63	1·57, 1·68	1·59	1·54, 1·64
LDL-cholesterol (mmol/l)	3·24	3·12, 3·35	3·23	3·11, 3·34	3·26	3·15, 3·38
TAG (mmol/l)¶	0·92	0·85, 0·98	0·86	0·79, 0·92	0·95	0·89, 1·01
Lipid treatment (%)		0·5		0·5		0·5
Glucose (mmol/l)**	4·84	4·78, 4·91	4·82	4·76, 4·88	4·85	4·79, 4·91

[a,b] Values within a row with unlike superscript letters were significantly different ($P<0·05$).

* The general linear model (GLM) procedure in SAS (analysis of covariance) was used to obtain age-adjusted means for continuous variables and to identify subgroups that differed significantly. Logistic regression (SAS procedure LOGISTIC) was used to obtain age-adjusted proportions for dichotomous variables and to identify subgroups that differed significantly. Both sets of analyses used Bonferroni's correction for each variable.

† The risk score was calculated from the consumption of nineteen nutrients (protein, carbohydrate, fibre, Ca, alcohol, total fat, polyunsaturated fat, monounsaturated fat, saturated fat, cholesterol, Na, Se, vitamin C, vitamin B_6, vitamin B_{12}, folate, vitamin E, β-carotene, total energy), which were ranked for each woman in the sample.

‡ To convert kg to pounds, divide by 0·454.

§ Physical activity index scores range from 24 (total bed rest) up to 120.

‖ To convert mmol/l cholesterol to mg/l, divide by 0·00259.

¶ To convert mmol/l TAG to mg/l, divide by 0·00113.

** To convert mmol/l glucose to mg/l, divide by 0·00555.

British Journal of Nutrition

Results

Women did not differ across tertiles in most baseline characteristics (Table 1). Women with higher diet quality (i.e. lowest nutritional risk tertile) were significantly older and smoked less currently and during their lifetimes. Those with the poorest diet quality scores (i.e. highest nutritional risk tertile) had lower intakes of energy, carbohydrate, fibre and all micronutrients (except vitamin B_{12}) and higher intakes from alcohol and total, saturated and monounsaturated fats (Table 2).

The overall incidence of overweight or obesity over 16 years was 44 % (*n* 258; Table 3). In multiple logistic regression analyses adjusted for age, physical activity and smoking status, women in the highest nutritional risk tertile were 1·76 (95 % CI 1·16, 2·69) times more likely to become overweight or obese compared with those in the lowest (referent) nutritional risk tertile (*P* for trend = 0·009). We also explored associations with quartiles and results were similar (data not shown). In additional analyses, including total energy intake in the regression model attenuated the findings (data not shown).

In *post hoc* analyses, energy, fibre, alcohol and vitamin E were inversely associated, while protein was positively associated, with development of overweight or obesity (*P* for trend < 0·05). In multiple logistic regression analyses adjusted for age, smoking status and physical activity, compared with the lowest tertile of intake, the odds of becoming overweight or obese were lower in the highest tertile of energy intake (OR 0·56; 95 % CI 0·37, 0·84), fibre intake (OR 0·51; 95 % CI 0·34, 0·78), alcohol intake (OR 0·67; 95 % CI 0·46, 0·99) and vitamin E intake (OR 0·64; 95 % CI 0·42, 0·95) and higher in the highest tertile of protein intake (OR 1·89; 95 % CI 1·25, 2·86).

Discussion

Diet quality, assessed using the validated nineteen-nutrient Framingham composite nutritional risk score, predicted the development of overweight or obesity over 16 years in disease-free FOS women, aged 25–71 years, at baseline. Women with the poorest diet quality (i.e. highest nutritional risk) consumed diets that were lower in energy, carbohydrate and micronutrients and higher in total fat, particularly saturated fat, and alcohol. These findings suggest that overall diet quality appears to be an important component of the diet–obesity relationship and provide potential new insights for use in future translational research on developing preventive nutrition strategies.

We performed independent single-nutrient analyses of FNRS index components which, for comparison purposes, controlled for covariates consistent with our primary model. All results were found to be in agreement with those of the overall composite FNRS score. Higher energy, fibre and vitamin E intakes were associated with a lower risk of developing overweight or obesity. Higher protein consumption was associated with a higher risk of becoming overweight or obese in this sample of women who also consumed a higher-carbohydrate, higher-fat diet. While it is difficult to draw conclusions regarding alcohol, as consumption levels

Table 2. Baseline daily nutrient intake profiles of 590 healthy, normal-weight (BMI < 25 kg/m^2) women in the Framingham Offspring and Spouse Study according to diet quality*

(Least squares (LS) means and 95 % confidence intervals)

	Framingham nutritional risk score†					
	Higher diet quality Tertile 1 (*n* 197)		Tertile 2 (*n* 194)		Lower diet quality Tertile 3 (*n* 199)	
Nutrient	LSmean	95 % CI	LSmean	95 % CI	LSmean	95 % CI
Energy (kJ)‡	7297[a]	7032, 7561	6966[a]	6702, 7223	6026[b]	5762, 6289
Protein (% energy)	16·7[a,b]	16·1, 17·2	16·3[a]	15·8, 16·9	17·3[b]	16·8, 17·8
Total fat (% energy)	33·3[a]	32·5, 34·1	37·8[b]	37·0, 38·6	40·7[c]	39·9, 41·5
Saturated fat (% energy)	11·1[a]	10·8, 11·5	13·1[b]	12·7, 13·4	14·9[c]	14·6, 15·3
Monounsaturated fat (% energy)	11·7[a]	11·4, 12·0	13·8[b]	13·5, 14·1	14·9[c]	14·6, 15·3
Polyunsaturated fat (% energy)	7·9	7·5, 8·3	8·1	7·7, 8·5	7·6	7·2, 8·0
Carbohydrate (% energy)	50·3[a]	49·3, 51·3	43·9[b]	42·9, 44·9	38·3[c]	37·3, 39·3
Fibre (g/4184 kJ)	17·1[a]	16·5, 17·7	12·6[b]	12·0, 13·1	9·0[c]	8·4, 9·5
Alcohol (% energy)	2·0[a]	1·3, 2·7	3·5[b]	2·8, 4·2	4·7[b]	4·0, 5·4
Cholesterol (mg/4184 kJ)	209·7[a]	194·2, 225·2	243·0[b]	227·6, 258·4	240·0[b]	224·6, 255·5
Na (mg/4184 kJ)	2549[a]	2435, 2664	2553[a]	2439, 2667	2344[b]	2230, 2458
Ca (mg/4184 kJ)	762[a]	727, 797	627[b]	592, 662	505[c]	470, 540
Se (μg/4184 kJ)	107[a]	103, 112	100[a]	96, 105	87[b]	82, 91
Vitamin C (mg/4184 kJ)	137·0[a]	130·2, 143·8	84·6[b]	77·9, 91·4	50·1[c]	43·3, 56·9
Vitamin B_6 (mg/4184 kJ)	1·8[a]	1·7, 1·9	1·3[b]	1·3, 1·4	1·1[c]	1·0, 1·1
Vitamin B_{12} (μg/4184 kJ)	5·9	4·5, 7·2	6·7	5·4, 8·0	4·6	3·3, 5·9
Folate (μg/4184 kJ)	313[a]	301, 326	215[b]	202, 228	144[c]	131, 157
Vitamin E (mg/4184 kJ)	9·7[a]	9·2, 10·3	8·1[b]	7·6, 8·7	6·2[c]	5·7, 6·7
β-Carotene (μg/4184 kJ)	4735[a]	4246, 5224	3359[b]	2873, 3846	2057[c]	1570, 2544

[a,b,c] Mean values within a row with unlike superscript letters were significantly different (*P* < 0·05).

* The general linear model (GLM) procedure in SAS (analysis of covariance) was used to obtain age-adjusted means for continuous variables and to identify subgroups that differed significantly. This set of analyses used Bonferroni's correction for each variable.

† The risk score was calculated from the consumption of nineteen nutrients (protein, carbohydrate, fibre, Ca, alcohol, total fat, polyunsaturated fat, monounsaturated fat, saturated fat, cholesterol, Na, Se, vitamin C, vitamin B_6, vitamin B_{12}, folate, vitamin E, β-carotene, total energy), which were ranked for each woman in the sample.

‡ To convert kJ to kcal, divide by 4·184.

Table 3. Development of overweight or obesity over 16 years in 590 healthy women in the Framingham Offspring and Spouse Study according to diet quality

		Framingham nutritional risk score*			
	Overall incidence	Higher diet quality Tertile 1 (*n* 197)	Tertile 2 (*n* 194)	Lower diet quality Tertile 3 (*n* 199)	*P* for trend†
Overweight or obese‡					
Subjects (*n*)	258	73	79	106	
Subjects (%)	43·7	37·1	40·7	53·3	
OR					
Age-adjusted		1·00	1·16	1·92	0·001
95 % CI		Reference	0·77, 1·75	1·29, 2·88	
Multivariate-adjusted§		1·00	1·07	1·76	0·009
95 % CI		Reference	0·71, 1·62	1·16, 2·69	

* The risk score was calculated from the consumption of nineteen nutrients (protein, carbohydrate, fibre, Ca, alcohol, total fat, polyunsaturated fat, monounsaturated fat, saturated fat, cholesterol, Na, Se, vitamin C, vitamin B_6, vitamin B_{12}, folate, vitamin E, β carotene, total energy), which were ranked for each woman in the sample.
† The *P* value for trend was determined using the tertile groups of the Framingham nutritional risk score in a continuous form. Significance testing was at $P<0{\cdot}05$.
‡ Overweight defined as BMI 25–29·9 kg/m^2; obesity defined as BMI ≥ 30 kg/m^2.
§ Stepwise multiple logistic regression model adjusted for age, smoking and physical activity.

were low (<5 % of energy intake in all tertiles), it is noteworthy that alcohol appeared to have a protective effect in the single-nutrient analysis; yet when combined with the set of nutrients in the composite FNRS, women with lower overall diet quality consumed more alcohol and were at greater risk of becoming overweight or obese. Thus, it is important to emphasise that single-nutrient comparisons may not reflect the true magnitude or direction of risk due to food and nutrient collinearity, nutrient synergy and other complex metabolic and biochemical interactions.

To our knowledge, this is one of the first long-term prospective investigations of overweight or obesity status specifically in women in relation to diet quality. As the FNRS contains both macro- and micronutrients, it may better represent total diet quality and its reflection of the nutrient density of a diet may explain its ability to predict development of overweight or obesity. This finding is consistent with an earlier prospective investigation that demonstrated a direct relationship with a five-point diet quality index and weight gain[33]. This index included five nutrients (total and saturated fat, dietary cholesterol, Na and carbohydrate) and a score of 1 was given for each nutrient if mean intake levels met the 2000 *Dietary Guidelines for Americans*[42]. Adults with the highest scores gained less weight over 8 years than those with lower scores. Average weight gain was about 1·4 kg (3 pounds) for those with higher-quality diets (i.e. higher scores) compared with 2·3–3·6 kg (5–8 pounds) for those with poorer-quality diets (i.e. lower scores) ($P<0{\cdot}05$). A recent longitudinal investigation[34] conducted over 18 months demonstrated that for every 1-unit increase on the original 1990 healthy eating index[43] or a modified healthy eating index, based upon the 2005 *Dietary Guidelines for Americans*[13], the odds of obesity decreased by 3 % in a combined sample of white men and women in multinomial logistic regression analysis.

The cross-sectional literature using *a priori* diet quality indices or scores as correlates of obesity are also consistent with our findings[31,32]. For example, in multivariate-adjusted analyses, Guo *et al.*[31] found that American women with the lowest diet quality measured using the healthy eating index were 1·7 (95 % CI 1·2, 2·6) times more likely to be obese than women with higher-quality diets. In Spanish adults, Schroder *et al.*[32] demonstrated a significant decrease in obesity risk for those in the top quartile of healthy eating index adherence (OR 0·68; 95 % CI 0·52, 0·89) compared with those in the first quartile in multivariate-adjusted analyses. In this same investigation[32], men and women in the top Mediterranean diet score adherence quartile were 0·68 (95 % CI 0·52, 0·89) times less likely to be obese compared with those in the first quartile. The FNRS also may have been predictive of overweight or obesity, as it may better capture dietary variety as reflected in a broad range of macro- and micronutrients. This is in agreement with cross-sectional data from Kennedy *et al.*[44] who found that adults consuming a diet with more variety had significantly lower BMI and were less likely to have BMI > 25 kg/m^2.

Our research is a step towards moving diet quality index research into the translational phase of designing dietary interventions and informing current guidelines for obesity prevention and risk reduction. This is essential, as the prevalence of obesity in adult women increases substantially each decade beginning in their twenties and does not decline until they reach their seventies[45]; also, long-term weight loss in women is disappointing, with estimates that only 5 % of initially overweight or obese women successfully maintain at least 5 % of their lost weight at 9 years[46]. The FNRS captures nutrient intake levels in an overall pattern that poses the greatest risk, as well as the greatest benefit[13]. As demonstrated here, a higher-quality diet appears to be less obesity promoting and provides support for national nutrition guidelines, which emphasise a balanced eating plan. While the FNRS cannot be applied directly 'as is' to design targeted nutrition interventions, we have previously linked the FNRS with food-related dietary patterns, derived using cluster analysis, in FOS women[3]. Since the clusters are based on foods and food groups, this previous research provided increased interpretability, as well as a framework for application of this score in conjunction with food-based approaches to dietary assessment and intervention. Translational research has not identified an optimal composite diet quality index method for obesity risk assessment. Therefore, as translational research moves forward we would advocate that preventive nutrition interventions be guided by the

combination of composite nutrient index (diet quality) and dietary pattern approaches.

The major strengths of the present investigation are the longitudinal design with a long duration of follow-up, inclusion of a relatively large sample of disease-free normal-weight women over a broad age range who had otherwise varying risk factor status and direct measurement of anthropometric outcome variables (as opposed to self-report). Additionally, the FNRS is nutrient-based, which is consistent with the foundation of the *Dietary Guidelines for Americans* [13] for determining adequacy of intake and overall diet quality. Other strengths include consideration of potential confounders (age, physical activity, smoking, parity, menopausal status) and calculation of nutritional risk (diet quality) using energy-adjusted index components. In contrast, our reliance on 3 d dietary records may introduce error into estimates of nutrient intake and result in a lower estimate of diet–obesity relationships, as 3 d dietary records may not fully capture usual diet, which would be more strongly connected with weight. While it is also true that we cannot assess nutritional risk over time, intakes in the FOS cohort have been shown to be stable[47]. Lastly, 98 % of FOS women are white, which would limit the generalisability of our findings, since food and nutrient intake patterns have been shown to differ by ethnicity[48].

In conclusion, we identified a link between diet quality, assessed using the FNRS, and risk of becoming overweight or obese in women. This finding has significant implications for obesity prevention and treatment, as it suggests the importance of comprehensively assessing nutritional risk, particularly at the time of initiating weight-gain prevention recommendations and weight-loss interventions. Fundamental to obesity management is identification of the behaviour to target in interventions. With respect to diet, focusing on food intake behaviour is essential. A nutritional risk score that is comprehensive and nutrient-based, like the FNRS, allows assessment of overall nutrient intake patterns of individuals to identify areas for translation to dietary changes in food patterns, rather than using a more uniform set of intervention guidelines that are potentially irrelevant or more restrictive than appropriate to the individual. These findings suggest that obesity risk reduction may be possible through strategies which optimise the nutrient quality of the individual's habitual eating behaviour. They also suggest that diverse dietary patterns in a population may be possible to maintain, as long as their nutrient composition and quality are enhanced by the use of nutrient-dense foods and suitable recipe modifications. Using this approach in future research would require the assessment of the individual's (or population's) habitual dietary patterns and their nutritional risk profiles and the development of options for dietary intervention that achieve an optimal balance between the individual's (or population's) food preferences, dietary patterns and nutrient-dense foods.

Acknowledgements

The present study was supported by grants R01-HL-60700 and R01-HL-54776 from the National Heart, Lung and Blood Institute (NHLBI) and funding from the Department of Family Medicine and the Division of Graduate Medical Sciences, Boston University School of Medicine. The Framingham Study is supported by NIH/NHLBI N01-HC-25195.

D. M. W. was responsible for the concept and design of the experiment and prepared the manuscript. B. E. M. provided overall direction to the research and overall critical review of reported intellectual content, and contributed to writing of the manuscript. L. Z. carried out the statistical analyses and critically reviewed the statistical content reported. M. J. P. and R. B. D. oversaw the statistical analyses and contributed to the interpretation of the data and critical review of the statistical content reported. P. K. N. contributed to the statistical design and writing of the manuscript. R. W. K. contributed to interpretation of the data and writing of the manuscript.

None of the authors had personal or financial conflicts of interest.

References

1. Ogden CL, Carroll MD, Curtin LR, *et al.* (2006) Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* **295**, 1549–1555.
2. Mokdad AH, Marks JS, Stroup DF, *et al.* (2004) Actual causes of death in the United States, 2000. *JAMA* **291**, 1238–1245.
3. Millen BE, Quatromoni PA, Copenhafer DL, *et al.* (2001) Validation of a dietary pattern approach for evaluating nutritional risk: the Framingham Nutrition Studies. *J Am Diet Assoc* **101**, 187–194.
4. Millen BE, Quatromoni PA, Pencina M, *et al.* (2005) Unique dietary patterns and chronic disease risk profiles of adult men: the Framingham nutrition studies. *J Am Diet Assoc* **105**, 1723–1734.
5. Simkin-Silverman LR & Wing RR (2000) Weight gain during menopause. Is it inevitable or can it be prevented? *Postgrad Med* **108**, 47–50.
6. Blaak E (2001) Gender differences in fat metabolism. *Curr Opin Clin Nutr Metab Care* **4**, 499–502.
7. Lovejoy JC (1998) The influence of sex hormones on obesity across the female life span. *J Womens Health* **7**, 1247–1256.
8. Roca P, Proenza AM & Palou A (1999) Sex differences in the effect of obesity on human plasma tryptophan/large neutral amino acid ratio. *Ann Nutr Metab* **43**, 145–151.
9. Hellstrom L, Wahrenberg H, Hruska K, *et al.* (2000) Mechanisms behind gender differences in circulating leptin levels. *J Intern Med* **247**, 457–462.
10. Patterson RE, Frank LL, Kristal AR, *et al.* (2004) A comprehensive examination of health conditions associated with obesity in older adults. *Am J Prev Med* **27**, 385–390.
11. Muennig P, Lubetkin E, Jia H, *et al.* (2006) Gender and the burden of disease attributable to obesity. *Am J Public Health* **96**, 1662–1668.
12. United States Department of Health and Human Services (2001) *The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity*. Rockville, MD: US Department of Health and Human Services, Public Health Service and Office of the Surgeon General.
13. United States Department of Health and Human Services & United States Department of Agriculture (2005) *Dietary Guidelines for Americans, 2005*. Washington, DC: US Government Printing Office. http://www.health.gov/dietaryguidelines/dga2005/document/default.htm (accessed 21 May 2009).
14. National Institutes of Health (1998) *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Evidence Report*. National Institutes of Health publication no. 98-4093. Bethesda, MD: National Heart

Lung and Blood Institute. http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.htm (accessed 21 May 2009).
15. National Institutes of Health (2002) *Third Report of the National Cholesterol Education Program (NCEP) on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) – Final Report*. National Institutes of Health publication no. 02-5215. Bethesda, MD: National Heart, Lung and Blood Institute. http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3_rpt.htm (accessed 21 May 2009).
16. National Institutes of Health (2004) *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) – Final Report*. National Institutes of Health publication no. 04-5230. Bethesda, MD: National Heart, Lung and Blood Institute. http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.htm (accessed 21 May 2009).
17. Bantle JP, Wylie-Rosett J, Albright AL, *et al.* (2008) Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care* **31**, Suppl. 1, S61–S78.
18. Ornish D, Scherwitz LW, Billings JH, *et al.* (1998) Intensive lifestyle changes for reversal of coronary heart disease. *JAMA* **280**, 2001–2007.
19. Astrup A, Grunwald GK, Melanson EL, *et al.* (2000) The role of low-fat diets in body weight control: a meta-analysis of *ad libitum* dietary intervention studies. *Int J Obes Relat Metab Disord* **24**, 1545–1552.
20. Nordmann AJ, Nordmann A, Briel M, *et al.* (2006) Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. *Arch Intern Med* **166**, 285–293.
21. Wierenga ME & Oldham KK (2002) Weight control: a lifestyle-modification model for improving health. *Nurs Clin North Am* **37**, 303–313.
22. Katz DL (2005) Competing dietary claims for weight loss: finding the forest through truculent trees. *Annu Rev Public Health* **26**, 61–88.
23. Fitzgerald AL, Dewar RA & Veugelers PJ (2002) Diet quality and cancer incidence in Nova Scotia, Canada. *Nutr Cancer* **43**, 127–132.
24. Massari M, Freeman KM, Seccareccia F, *et al.* (2004) Research Group of the RIFLE Project. An index to measure the association between dietary patterns and coronary heart disease risk factors: findings from two Italian studies. *Prev Med* **39**, 841–847.
25. Schroder H, Marrugat J, Vila J, *et al.* (2004) Adherence to the traditional Mediterranean diet is inversely associated with body mass index and obesity in a Spanish population. *J Nutr* **134**, 3355–3361.
26. Fogli-Cawley JJ, Dwyer JT, Saltzman E, *et al.* (2006) The 2005 Dietary Guidelines for Americans adherence index: development and application. *J Nutr* **136**, 2908–2915.
27. McNaughton SA, Ball K, Crawford D, *et al.* (2008) An index of diet and eating patterns is a valid measure of diet quality in an Australian population. *J Nutr* **138**, 86–93.
28. Trichopoulou A, Orfanos P, Norat T, *et al.* (2005) Modified Mediterranean diet and survival: EPIC-elderly prospective cohort study. *BMJ* **330**, 991–997.
29. McCullough ML, Feskanich D, Rimm EB, *et al.* (2000) Adherence to the Dietary Guidelines for Americans and risk of major chronic disease in men. *Am J Clin Nutr* **72**, 1223–1231.
30. Ledikwe JH, Smiciklas-Wright H, Mitchell DC, *et al.* (2003) Nutritional risk assessment and obesity in rural older adults: a sex difference. *Am J Clin Nutr* **77**, 551–558.
31. Guo X, Warden BA, Paeratakul S, *et al.* (2004) Healthy eating index and obesity. *Eur J Clin Nutr* **58**, 1580–1586.
32. Schroder H, Marrugat J & Covas MI (2006) High monetary costs of dietary patterns associated with lower body mass index: a population-based study. *Int J Obes* **30**, 1574–1579.
33. Quatromoni PA, Pencina M, Cobain MR, *et al.* (2006) Dietary quality predicts adult weight gain: findings from the Framingham Offspring Study. *Obesity* **14**, 1383–1391.
34. Gao SK, Beresford SA, Frank LL, *et al.* (2008) Modifications to the Healthy Eating Index and its ability to predict obesity: the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr* **88**, 64–69.
35. Feinleib M, Kannel WB, Garrison RJ, *et al.* (1975) The Framingham Offspring Study. Design and preliminary data. *Prev Med* **4**, 518–525.
36. Posner BM, Martin-Munley SS, Smigelski C, *et al.* (1992) Comparison of techniques for estimating nutrient intake: the Framingham Study. *Epidemiology* **3**, 171–177.
37. Posner BM, Smigelski C, Duggal A, *et al.* (1992) Validation of two-dimensional models for estimation of portion size in nutrition research. *J Am Diet Assoc* **92**, 738–741.
38. Martinez-González MA & Bes-Rastrollo M (2006) The cardioprotective benefits of monounsaturated fatty acid. *Altern Ther Health Med* **12**, 24–30.
39. Cupples LA & D'Agostino RB (1987) Some risk factors related to the annual incidence of cardiovascular disease and death by using pooled repeated biennial measurements: Framingham Heart Study, 30-year follow-up. In *The Framingham Study: an Epidemiological Investigation of Cardiovascular Disease* [WB Kannel, PA Wolf and RJ Garrison, editors]. National Institutes of Health publication no. 87-2703, Section 34 (NTIS PB87-177499). Washington, DC: Department of Health and Human Services.
40. Meigs JB, D'Agostino RB Sr, Wilson PW, *et al.* (1997) Risk variable clustering in the insulin resistance syndrome. The Framingham Offspring Study. *Diabetes* **46**, 1594–1600.
41. Kannel WB & Sorlie P (1979) Some health benefits of physical activity. *Arch Intern Med* **139**, 857–861.
42. US Department of Agriculture & US Department of Health and Human Services (2000) *Nutrition and Your Health: Dietary Guidelines for Americans*. Washington, DC: US Government Printing Office.
43. Kennedy E, Ohls J, Carlson S, *et al.* (1994) *The Healthy Eating Index Final Report*. Alexandria, VA: US Department of Agriculture, Food and Nutrition Service.
44. Kennedy ET, Bowman SA, Spence JT, *et al.* (2001) Popular diets: correlation to health, nutrition, and obesity. *J Am Diet Assoc* **101**, 411–420.
45. Flegal KM, Carroll MD, Ogden CL, *et al.* (2002) Prevalence and trends in obesity among US adults, 1999–2000. *JAMA* **288**, 1723–1727.
46. Sarlio-Lahteenkorva S, Rissanen A & Kaprio J (2000) A descriptive study of weight loss maintenance: 6 and 15 year follow-up of initially overweight adults. *Int J Obes Relat Metab Disord* **24**, 116–125.
47. Millen BE, Pencina M, Kimokoti R, *et al.* (2004) *The Framingham Nutrition Studies: Insights into Weight History, Dietary Patterns, Obesity Prevention, and Risk Reduction. Predictors of Obesity, Weight Gain, Diet, and Physical Activity Workshop*. Bethesda, MD: Department of Health and Human Services and National Institutes of Health National Heart, Lung and Blood Institute. http://www.nhlbi.nih.gov/meetings/workshops/predictors/abstracts/millen.htm (accessed 19 May 2009).
48. Wirfalt AK & Jeffery RW (1997) Using cluster analysis to examine dietary patterns: nutrient intakes, gender, and weight status differ across food pattern clusters. *J Am Diet Assoc* **97**, 272–279.

British Journal of Nutrition