Relative bias in diet history measurements: a quality control technique for dietary intervention trials

Gina S Martin[1],*, Linda C Tapsell[1], Marijka J Batterham[1] and Kenneth G Russell[2]

[1]Department of Biomedical Science, University of Wollongong, Wollongong, NSW 2522, Australia:
[2]School of Mathematics and Applied Statistics, University of Wollongong, NSW 2522, Australia

Abstract

Objective: Investigation of relative bias in diet history measurement during dietary intervention trials.

Design: Retrospective analysis of human dietary data from two randomised controlled trials examining modified fat diets in the prevention and treatment of type II diabetes mellitus.

Setting: Wollongong, Australia.

Subjects: Thirty-five overweight, otherwise healthy subjects in trial 1 and 56 subjects with diabetes in trial 2.

Interventions: Diet history interviews and three-day weighed food records administered at one-month intervals in trial 1 and three-month intervals in trial 2.

Results: In a cross-sectional bias analysis, graphs of the association between bias and mean dietary intake showed that bias decreased in higher carbohydrate consumers in trial 1 ($r = -0.344$, $P < 0.05$). No other significant associations were found. In a longitudinal analysis, bias did not change over time in either trial. There were no significant differences in bias magnitudes between the trials, with the exception of monounsaturated fat measurement where bias was significantly greater and more positive in trial 2, indicating overestimation of monounsaturated fat intake with the diet history. Subjects in control and intervention groups underestimated energy, fat, saturated fat and alcohol intakes with the diet history in both trials. Overweight and obese individuals appeared to make the greatest contribution to the overall underestimation of saturated fat intake by the diet history regardless of whether they were in the control or intervention group and whether they were healthy or had diabetes.

Conclusion: Bias in diet history measurement appears to be macronutrient-specific, with energy, fat and saturated fat consistently underreported in the interview by subjects with and without diabetes and in both intervention and control groups in a dietary intervention trial. Relative bias analysis appears to be an informative tool in quality control for dietary intervention trials when biochemical markers are unavailable.

Quality control in the dietary components of intervention trials includes first identifying and then quantifying sources of measurement bias in dietary assessment methods. In this instance, bias is often associated with over- and underestimation of energy and macronutrient intakes by the chosen method. Biochemical markers of intake can identify potential sources of bias, but they are often expensive to obtain and carry another set of issues concerning the specificity of the markers themselves[1]. In addition, biochemical markers are limited in the information they can provide with respect to the measurement of 'whole' diet.

Where biochemical markers are unavailable, relative bias can be assessed retrospectively using statistical techniques. Cross-sectional bias analysis allows for the determination of association between bias and intake as well as the precision (variability in bias) at a particular data collection point in a trial[2], while longitudinal bias analysis provides information regarding changes in both bias and precision as the intervention progresses. Using relative comparisons to examine bias can expose the limitations of a chosen method, especially in intervention trials where sample sizes are small. Findings generated from these relative investigations may then provide the basis for research specifically designed to investigate error.

The limitations associated with current dietary assessment techniques have been well documented[3]. The diet history method (DH), an in-depth account of a person's

Corresponding author: Email gmartin@uow.edu.au

habitual dietary intake and the technique examined in this paper, is not without fault. It is susceptible to recall bias and, perhaps, encourages psychological tendencies to report what is socially acceptable[4]. It has also has been found to underestimate both energy[5] and fat intakes[6,7] in epidemiological studies. Research in this area needs to expand to incorporate different contexts, as the DH is used extensively in both dietary intervention trials and in the clinical setting[8,9].

In this study we report a retrospective analysis of relative bias in a DH by comparison with a three-day food record (FR) during the course of two dietary intervention trials using both cross-sectional and longitudinal approaches.

Methods

The data reported here were obtained from two dietary intervention trials conducted in the major coastal city of Wollongong, Australia. Both studies were randomised controlled trials examining the effect of a modified fat diet on metabolic variables in the insulin-resistant state. In both studies the dietetic approach involved manipulating current dietary patterns to meet the dietary targets. A profile of the dietary targets is given in Table 1.

Trial 1

Context
Trial 1 data were from a larger multi-centre study examining the effect of a diet high in monounsaturated fat (MUFA) on the risk factors for type II diabetes mellitus[10]. The Wollongong sample was recruited through local media advertisements and email in tertiary institutions in the area. The 35 participants were overweight, but otherwise healthy adults ranging in age from 29 to 42 years. Seventeen subjects were randomly assigned to the control group and 18 subjects were to follow the intervention diet. Subjects in the intervention group were required to increase their MUFA intake over the length of the trial (three months). The control group was to continue with their normal diet.

Table 1 Dietary targets for main variables in the two intervention trials

Dietary variable (% of energy)	Trial 1		Trial 2	
	Control	Intervention	Control	Intervention
Total carbohydrate	45	45	53	43
Total fat	37	37	27	37
Polyunsaturated fat	6	6	7	7
Monounsaturated fat	14	23	12	22
Saturated fat	17	8	8	8

Dietary data
Dietary intakes were assessed monthly by dietitians adopting a narrative DH interview (an open-ended approach)[8]. Subjects were asked to recall the dietary constituents of each meal in an average day over a one-month period. They were then questioned on dietary variation, portion sizes and frequencies of consumption. Three students were trained in DH administration and interviewers were different for each repeat interview to avoid potential interviewer effects. Nutrition assessment interviews were used to subjectively check compliance and recommendations were made to members of the intervention group who needed further dietary manipulation. Subjects were also required to provide a three-day weighed FR (two weekdays and one weekend day) during the period between dietary assessment interviews. Salter Slimmer scales, cups and spoons were provided. All subjects were instructed on household measures at their baseline interview and instructed to record all preparation techniques and recipes. Forms were provided for subjects to record weights and household measures. The dietitians checked the FRs for missing values and for clarification of portion sizes at each interview. Subjects were also provided with specific fats, oils and spreads along with recipes for preparing foods to be included in their diet throughout the three-month trial. Weights and heights were recorded at each interview using digital scales and a wall-mounted stadiometer, respectively. The dietary variables reported here were from data collected at baseline and monthly intervals until the end of the trial (three months). Dietary data were analysed with the Diet 1 nutrient analysis software package (Version 4, Xyris Software, Highgate Hill, Brisbane, Australia), which is based on the Australian Nutrient Database (NUTTAB 1995, Department of Human Services and Health, Canberra, 1995).

Trial 2

Context
The aim of trial 2 was to examine the effect of a high-MUFA diet on the metabolic indices of diabetes control. The intervention diet required a reduction in total carbohydrate in the intervention group and an increase in total fat to accommodate manipulations in dietary fatty acids (Table 1). Men and women between the ages of 45 and 65 years with type II diabetes mellitus, who had been referred to the Diabetes Education Service, were invited to participate. Subjects were recruited over a period of two years between the beginning of 1998 and the end of 1999. A total of 86 people participated in the trial and, out of those, 56 were chosen for the validity study of which 28 were in the control group and 28 were following the intervention diet (high-MUFA). Both groups were receiving dietary counselling at the Diabetes Service prior to the trial and were following low-fat diets.

Dietary data

All subjects were required to attend dietary interviews every three months until trial completion at one year. Four dietitians collected the dietary data using the narrative approach DH[8] with a recall time of three months. DHs were administered as in trial 1. Dietary interviews took 1–1.5 hours and were used for compliance assessment and further advice if necessary. The intervention group participants were provided with specific counselling on their MUFA intake, while the control groups were given general advice on low-fat diets. Participants were also required to complete a three-day weighed FR (two weekdays and one weekend day) during periods between dietary interviews as in trial 1. FRs were checked by the dietitians for missing data and portion size clarification at each interview. Subjects were supplied with oil and spreads to use in their food preparation. Weights and heights were recorded at all interviews using digital scales and a wall-mounted stadiometer, respectively. Dietary data were analysed with the FoodWorks nutrient analysis software package (Version 2.03, Xyris Software, Highgate Hill, Brisbane, Australia), which is based on the Australian Nutrient Database (NUTTAB 1995, Department of Human Services and Health, Canberra, 1995).

Dietary variables

MUFA was the main dietary variable in both trials and the ability of the DH to measure this variable was considered central to any evaluation of its performance. In accordance with the literature examining diet and the metabolic syndrome, investigators were concerned with the DH's measurement of energy and macronutrient consumption. For the purpose of this analysis, protein, carbohydrate, fat and alcohol have been expressed as percentages of energy intake (% protein, % CHO, % fat and % alcohol, respectively) and monounsaturated, polyunsaturated and saturated fat expressed as percentages of fat (% MUFA, % PUFA and % SFA, respectively).

Ethics

The University of Wollongong's Human Research Ethics Committee approved the data collection for this research.

Statistical analysis

All data were analysed using the SPSS statistical package (Version 10, SPSS Inc., Chicago, IL, USA). The significance level was set at $\alpha = 0.05$ for all analyses. Population characteristics at baseline were examined for differences between the trials using analysis of variance (ANOVA) with sex and trial as factors.

Cross-sectional bias analysis

Paired t-tests were used to identify significant differences between mean energy and macronutrient intakes derived from the DH and the FR within both trials. Correlation coefficients were used to assess the presence of a linear association between the results of the two methods. Bias in DH measurement was defined as the difference between the DH and the FR (DH − FR) and could be positive or negative. The association between bias and mean dietary intake ((DH + FR)/2) at baseline was assessed using a technique described by Bland and Altman[2]. For each dietary variable, the limits of agreement were set at two standard deviations (2SD) from the mean bias (95% confidence intervals of the bias). The DH and FR measurements were considered to be in agreement if bias calculations for an individual fell between the limits of agreement. In addition, the greater the degree of separation of the confidence intervals, the greater the variability in bias (intra-individual variation in measurement) and hence the lower the relative precision of the DH. The statistical significance of the regression line expressing bias in terms of dietary intake was used to determine bias movement over the range of dietary intakes[11].

Longitudinal bias analysis

The mean bias (DH − FR) in DH measurement was calculated for the intervention and control groups as well as for the total sample of each trial at each of the data collection points. The extent of intra-individual variation in bias was examined using the SD of the bias (SD_{diff}) at each time point. A three-way repeated measures analysis of covariance (ANCOVA), with age, sex, body mass index (BMI), group (intervention or control) and trial (trial 1 or trial 2) as covariates, was used to assess changes in bias over time. Individual *post hoc* analyses were then performed on all significant interaction terms.

Table 2 Characteristics (mean (SD)) of subjects providing data for both intervention trials

Variable	Trial 1			Trial 2		
	Male ($n = 12$)	Female ($n = 23$)	All ($n = 35$)	Male ($n = 25$)	Female ($n = 31$)	All ($n = 56$)
Age (years)	46.7 (6.3)	45.9 (7.4)	46.2 (7.0)	53.8 (7.9)	52.2 (7.1)	53.1 (7.5)*
Weight (kg)**	86.1 (13.6)	68.8 (13.8)	75.2 (15.8)	93.6 (12.7)	83.5 (13.1)	89.1 (13.7)*
Height (cm)**	178.9 (6.6)	162.1 (5.4)	166.7 (8.5)	174.3 (7.4)	160.5 (7.9)	168.1 (10.2)
BMI (kg m^{-2})	27.4 (3.7)	26.2 (4.5)	26.6 (4.2)	30.5 (4.1)	32.3 (4.1)	31.3 (5.0)*

*, Mean significantly different from trial 1 at $P < 0.01$; **, means for males and females significantly different in both trials at $P < 0.01$.

Table 3 Comparison between the DH and the FR measurements at baseline in both intervention trials

	Trial 1					Trial 2				
Variable	DH ($n = 35$) Mean (SD)	FR ($n = 35$) Mean (SD)	Paired t-test	Correlation	Mean bias (SD$_{diff}$)	DH ($n = 54$) Mean (SD)	FR ($n = 54$) Mean (SD)	Paired t-test	Correlation	Mean bias (SD$_{diff}$)
Energy (kJ)§	8466 (1974)[a]	9401 (2191)[b]	$P < 0.01$	0.70**	−935 (1868)	7641 (1880)[a]	7803 (1922)[b]	NS†	0.47**‡	−244 (1959)
% Protein	18.77 (3.03)[c]	18.20 (2.61)[d]	NS	0.54**	0.57 (2.74)	21.72 (3.58)[c]	21.08 (3.54)[d]	NS	0.42*	0.55 (3.86)
% CHO	46.31 (5.94)	44.49 (7.58)[e]	$P < 0.05$	0.74**	1.83 (5.09)	44.31 (7.69)	43.68 (6.26)[e]	NS	0.57**	0.86 (6.51)
% Fat	32.03 (5.90)	33.68 (7.12)[f]	NS	0.62**	−1.63 (5.79)	29.07 (7.37)	29.73 (6.29)[f]	NS	0.57**	−0.88 (6.40)
% Alcohol§	2.94 (3.33)	3.83 (4.23)	NS†	0.51**‡	−0.89 (4.09)	2.57 (5.30)	2.86 (5.89)	NS†	0.80**‡	−0.20 (3.62)
% MUFA§	38.17 (3.83)	38.80 (4.16)	NS†	0.34**‡	−0.49 (6.23)	42.34 (5.36)	41.47 (5.24)	NS	0.24 (0.35**)	0.93 (6.51)
% PUFA§	17.77 (5.78)[g]	16.49 (4.82)[h]	NS	0.61**	3.63 (10.10)	18.59 (5.79)[g]	17.49 (4.72)[h]	NS†	0.16 (0.30*)‡	0.75 (6.57)
% SFA	43.97 (6.04)[i]	44.71 (6.75)[j]	NS	0.64**	−0.67 (7.36)	39.07 (6.57)[i]	41.03 (6.98)[j]	NS	0.17 (0.31*)	−1.47 (8.63)

Note: Data with the same alphabetical superscript are significantly different. Correlation coefficients in parentheses indicate coefficients adjusted by the removal of outliers for bias.
*, Significant at $P < 0.05$; **, significant at $P < 0.01$; [a–d, g, i], significantly different at $P < 0.01$; [e,f, h, j], significantly different at $P < 0.05$; NS, not significant.
† Wilcoxon signed rank test.
‡ Spearman's rank order correlation.
§ Mann–Whitney tests for differences between trials.

Results

Demographics

Table 2 illustrates the demographic characteristics of subjects participating in both trials. The participants in trial 2 had significantly greater mean age ($P < 0.01$) and mean weight ($P < 0.01$) and, consequently, had greater mean BMI ($P < 0.01$) than their trial 1 counterparts.

Cross-sectional bias analysis

Means and SDs for consumption of energy and macronutrients measured by the DH and the FR at baseline are given in Table 3. In trial 1, mean energy intake was greater with the DH ($P < 0.01$) and mean CHO intakes were greater with the FR ($P < 0.05$). All other differences in mean macronutrient measurement were not significant. In trial 2, measurements from the DH were not significantly different to those from the FR. Correlation coefficients between values measured by the DH and the FR were significant, except for the fatty acids in trial 2. However, when outliers for bias were removed correlation coefficients improved and were significant. Mean bias was low for energy and macronutrients in both trials with the exception of PUFA, which showed large intra-individual variation in measurement (low precision).

Bias that moved from positive to negative with increasing % CHO intake in the diet ($r = -0.34$, $P < 0.05$) was evident in trial 1 (Fig. 1). It appears that higher CHO consumers tended to report more accurately than those who consumed smaller amounts of CHO relative to energy intake. High CHO consumers underestimated their intake with the DH in trial 1. No other significant trends in bias with intake were observed and therefore bias plots for energy and macronutrients in both trials are not shown. These plots also showed good agreement between the DH and the FR for the measurement of energy and all macronutrients, with almost all cases falling between the limits of agreement. Low precision was shown for the measurement of energy, fat and SFA measurement in trial 1. Similar results were found for energy and SFA in trial 2.

Longitudinal bias analysis

Table 4 illustrates the mean bias and SD$_{diff}$ for macronutrient and energy intakes at all data collection points in both trials. Data for MUFA, PUFA and SFA obtained in the first month in trial 1 were not available. Similarly, data from the second and third months for protein, CHO and alcohol were also unavailable. The residuals from the repeated measures ANCOVA were found not to differ significantly from normality. The assumption of homogeneity of variance appeared valid. Therefore, no transformation of the response variable was required for this analysis.

Bias magnitudes did not change with time in either trial (Table 4). The values for SD$_{diff}$ were large for all
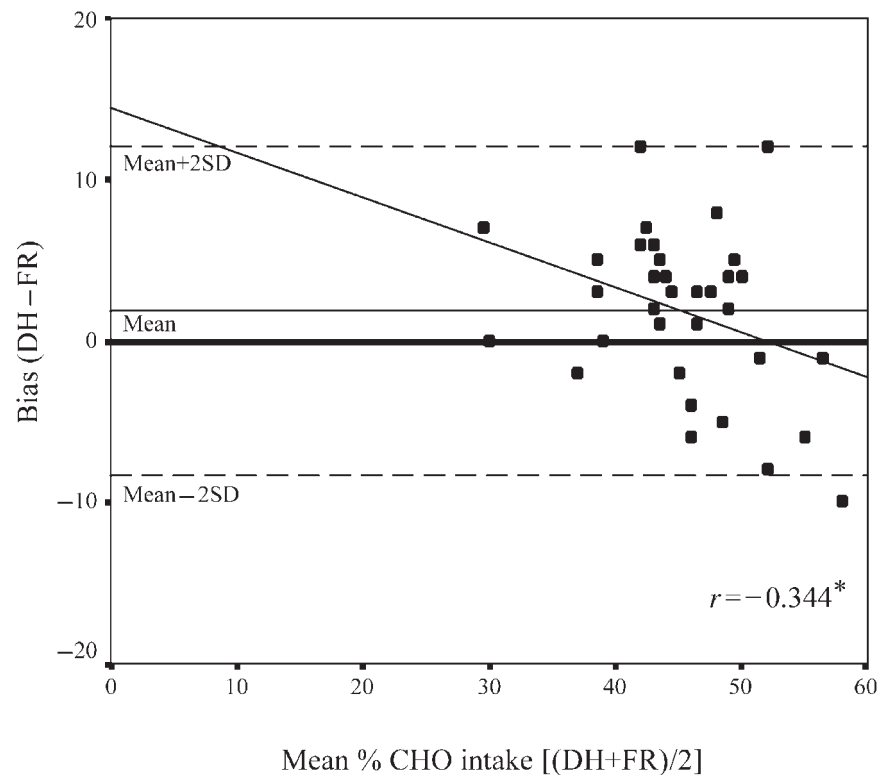
Fig. 1 Bias plot showing the association between bias in CHO measurement and the mean intake of CHO as a percentage of energy at baseline in trial 1 ($n = 35$) (*, significant at $P < 0.05$)

macronutrient and energy intakes, indicating considerable intra-individual variation in measurements made with the DH and the FR. Large variability in bias for alcohol measurement was evident at all data collection points and for PUFA measurement at baseline in both trials. Bias, averaged over all time points and in all subjects, for energy, fat, alcohol, MUFA and SFA intake measurement indicated an underestimation by the DH relative to the FR in trial 1 in both intervention and control groups. Bias was similar in direction in trial 2 for all dietary variables apart from that of MUFA, which was overestimated by the DH in trial 2 when averaged over all time points in both intervention and control groups.

There was no trend in bias for fat measurement over time. Mean bias was then averaged over all time points. Overall bias in the two trials indicated an underestimation of fat intake by the DH. Bias in measuring fat intake was also found to be significantly greater in males ($P < 0.01$) and in the control groups ($P < 0.05$) when the subjects from the two trials were combined (Table 4). Bias was negative in both males and females for fat intake, indicating an overall underestimation of fat intake by the DH regardless of sex. Despite a significant interaction between trial and group ($P < 0.05$), fat was underestimated by all subjects regardless of group in both trials. Bias was also found to be significantly different between trial 1 and trial 2 for measuring % MUFA intake ($P < 0.05$). The bias was significantly greater in trial 2 and indicated an

overestimation by the DH relative to the FR, while the bias in trial 1 was significantly smaller and negative (underestimation by the DH).

In the combined sample, bias in % SFA intake decreased significantly with increasing BMI and reached zero at a BMI of 24–25 kg m^{-2} (Fig. 2). Bias then increased in the negative direction. Those within the healthy weight range, between 20 and 25 kg m^{-2}, overestimated intakes of SFA with the DH relative to the FR. Overweight and obese subjects (BMI > 25 kg m^{-2} and BMI > 30 kg m^{-2}, respectively) underestimated saturated fat intake with the DH. Overall, the correlation between the DH and the FR was $r = -0.38$ ($P < 0.01$). When outliers were removed, the linear association between the DH and the FR improved ($r = -0.47$, $P < 0.01$).

Discussion

This research aimed to investigate relative bias in DH measurements in two intervention trials using a simple calculation of the difference between the DH and a three-day FR in both cross-sectional and longitudinal analyses. Despite the limitations of this analysis, we found no significant changes in bias with time. The bias in all variables, except MUFA, was unaffected by differences between the two trials. There were notable similarities between the two trials with respect to the direction of bias for reporting energy and specific macronutrients. It is

Table 4 Mean (SD$_{diff}$) of the bias (DH − R) for energy and nutrient intakes for all data collection points in both intervention trials[a]

Variable	Trial 1				Trial 2			
	Baseline	Time 1	Time 2	Time 3	Baseline	Time 1	Time 2	Time 3
Energy (kJ)								
Intervention	−969.67 (1560.03)	−753.61 (1431.34)	−818.28 (1505.15)	−745.39 (2272.00)	−477.08 (2192.99)	67.04 (1999.31)	−1354.58 (2059.30)	−382.49 (1325.06)
Control	−898.24 (2197.24)	−519.24 (1184.54)	−517.29 (1485.93)	−896.24 (1642.12)	−27.61 (1727.23)	73.97 (1857.04)	46.60 (4120.23)	−232.21 (908.35)
All	−934.97 (1868.18)	−639.77 (1303.38)	−672.09 (1481.58)	−818.66 (1963.62)	−244.02 (1959.51)	70.14 (1914.96)	−794.10 (3084.41)	−317.50 (1151.06)
% Protein								
Intervention	0.56 (3.07)	−0.17 (2.62)	–	–	1.70 (4.11)	0.24 (4.22)	1.01 (4.14)	0.33 (5.25)
Control	0.59 (2.42)	0.00 (2.21)	–	–	−0.52 (3.33)	−0.21 (3.65)	0.37 (3.97)	−5.67 (2.86)
All	0.57 (2.74)	−0.09 (2.40)	–	–	0.55 (3.86)	0.03 (3.93)	0.74 (4.03)	0.06 (4.35)
% CHO								
Intervention	2.67 (4.82)	2.89 (4.95)	–	–	−0.42 (6.13)	1.23 (7.50)	1.60 (5.11)	1.09 (5.10)
Control	0.94 (5.36)	1.06 (2.86)	–	–	2.03 (6.74)	−1.50 (8.44)	−0.37 (5.72)	−0.29 (5.83)
All	1.83 (5.09)	2.00 (4.12)	–	–	0.86 (6.51)	0.02 (7.97)	0.75 (5.40)	0.49 (5.39)
% Fat[b]								
Intervention	−2.39 (6.12)	−2.67 (4.91)	0.39 (6.11)	−0.61 (4.19)	−1.49 (6.42)	−3.01 (6.48)	−1.25 (6.65)	−2.39 (5.15)
Control	−0.82 (5.49)	−0.71 (3.24)	−0.82 (4.71)	−1.12 (4.06)	−0.32 (6.45)	2.33 (8.10)	−0.49 (7.49)	1.14 (6.04)
All	−1.63 (5.79)	1.71 (4.25)	−0.20 (5.43)	−0.86 (4.07)	−0.88 (6.40)	−0.56 (7.67)	−0.92 (6.94)	−0.86 (5.75)
% Alcohol								
Intervention	−1.11 (4.90)	−0.28 (4.61)	–	–	0.13 (3.99)	1.49 (4.93)	−1.38 (5.55)	0.89 (4.54)
Control	−0.65 (3.16)	−0.41 (1.70)	–	–	−0.52 (3.28)	−0.42 (1.84)	0.51 (3.08)	−0.20 (1.30)
All	−0.89 (4.09)	−0.34 (3.46)	–	–	−0.20 (3.62)	0.61 (3.92)	−0.62 (4.77)	0.42 (3.53)
% MUFA[c]								
Intervention	−0.94 (4.90)	–	−1.67 (5.05)	−0.78 (2.71)	0.69 (4.81)	0.96 (4.41)	2.22 (4.23)	3.12 (3.32)
Control	−0.02 (7.51)	–	0.59 (3.24)	−2.71 (12.24)	1.14 (7.86)	3.23 (4.31)	3.78 (4.74)	3.34 (4.75)
All	−0.49 (6.23)	–	−0.57 (4.36)	−1.71 (8.67)	0.93 (6.51)	1.99 (4.47)	2.89 (4.47)	3.22 (3.95)
% PUFA								
Intervention	2.06 (5.96)	–	−0.11 (3.80)	0.78 (2.86)	0.13 (6.20)	2.16 (5.48)	1.13 (4.57)	1.34 (4.66)
Control	5.31 (13.17)	–	0.18 (2.90)	−0.76 (2.93)	1.33 (6.96)	1.60 (5.32)	2.53 (3.53)	0.68 (3.45)
All	3.63 (10.10)	–	0.03 (3.34)	0.03 (2.96)	0.75 (6.57)	1.90 (5.36)	1.73 (4.17)	1.06 (4.14)
% SFA[d]								
Intervention	−2.33 (5.41)	–	0.50 (5.37)	−0.11 (3.82)	−0.91 (7.33)	−2.60 (6.09)	−3.35 5.82	−4.47 (5.77)
Control	1.63 (9.16)	–	−1.00 (2.24)	1.41 (7.07)	−1.99 (9.79)	−5.06 (7.92)	−6.34 5.76	−4.09 (5.30)
All	−0.67 (7.36)	–	−0.23 (4.17)	0.63 (5.61)	−1.47 (8.63)	−3.94 (7.01)	−4.59 5.92	−4.31 (5.51)

[a] Repeated measures ANCOVA.
[b] Significant interaction between trial and group at $P < 0.05$. Bias is significantly greater in males than in females in combined trial populations at $P < 0.01$. Bias is significantly greater in the control group in combined trial populations at $P < 0.05$.
[c] Bias is significantly different between trial 1 and trial 2 at $P < 0.05$.
[d] Bias is affected by BMI in combined trial populations at $P < 0.01$. See Fig. 2.
Note: – indicates data not available and therefore not presented.
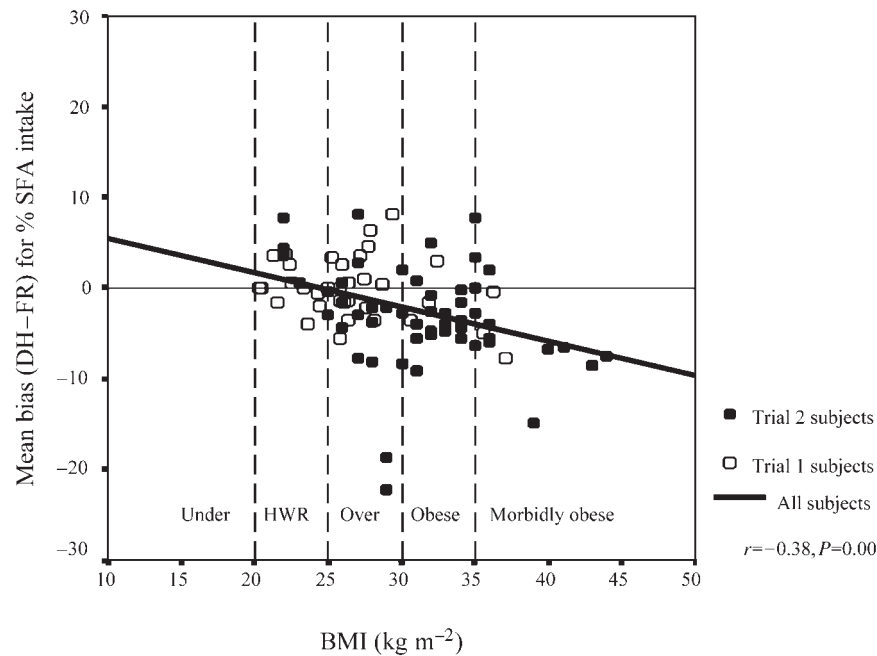
Fig. 2 Plot showing the effect of BMI on mean bias for % SFA intake as averaged over all available data collection points in subjects from both trials (Under – underweight; HWR – healthy weight range; Over – overweight)

important to note, however, that the FR comes with its own set of issues regarding accuracy of measurement and, therefore, macronutrient-specific biases in the DH may also be due to inherent biases within the reference method. Apparent biases in the DH method may then be a result of over- or underestimation by the reference method.

Cross-sectional bias analysis

In trial 1, underestimation of energy intake by the DH is consistent with findings from doubly labelled water studies[5] and criterion validity investigations[12,13]. However, despite the significant differences in mean energy intakes measured with the DH and the FR in trial 1, there was good agreement at an individual level between the two methods for energy intake. A possible interpretation is that, while the DH and the FR were essentially measuring similar quantities, their relative difference was sufficiently consistent at the group level to yield significantly different means. This could also be indicative of low precision in the measurement of energy intake, as indicated by the large SD_{diff} (separation of limits of agreement) in trial 1.

In both trials the data from the DH and FR appeared to be linearly associated. However, the failure to show a linear relationship for MUFA, PUFA and SFA intakes in trial 2 was not necessarily problematic as cases were found to be within the 95% confidence intervals in their respective bias plots and correlation coefficients improved upon removal of outliers. No significant trends in bias were evident in the bias plots[2] apart from CHO intake in trial 1. High CHO consumers tended to underestimate their intake in the DH interview, which may be due to difficulty

in remembering all foods containing carbohydrate or, perhaps, underestimation of the amounts actually consumed at such high CHO intakes.

Longitudinal bias analysis

Greater recall times and lengthier periods between intake assessments in trial 2 did not result in larger biases or larger increases in bias with time than those observed in trial 1. In fact, there were no evident changes in bias magnitudes with time in either trial. Overall bias in the DH measurement of energy, protein, CHO, fat, alcohol, PUFA and SFA was also unaffected by contextual differences between the trials.

Variability in bias was evident in all measures of intake, particularly with PUFA measurement. The wide range of PUFA-containing foods in the Australian food supply may have caused the discrepancy between the measurement of actual intake by the FR and usual intake by the DH, given the difficulty associated with recalling a nutrient that has large variability in the diet. The magnitude of the variability in measurement of alcohol in both trials was expected and has been reported in the literature in the past[14,15]. Alcohol is often omitted from reports of dietary intake, causing lack of agreement between FR and DH[8]. Variability in alcohol measurement also results in poor intra-class correlation coefficients for DH reproducibility[16].

Underreporting of fat intake was evident in both trials and in both intervention and control groups, and may reflect a social desirability to report less fat within both populations[8,17]. Social desirability and social approval are response variables that produce biases in a number of research contexts[18] and this has been largely supported by

dietary measurement investigations[4,19−22]. Subjects may be reluctant to report fat-containing foods during the interview for fear of social non-acceptance. The pronounced bias in fat reports from the control group in people with diabetes may have resulted from the low-fat diets they were prescribed during dietary counselling prior to the trial. Interestingly, it has been found that people with diabetes (trial 2) tend to report intakes that are in line with their prescription diets following dietary intervention[23,24]. This may have been the case in trial 2, where people in the control group reported intakes in their interviews that were in line with their dietary prescription (i.e. a low-fat diet). Prescription-related reporting could also explain the positive bias for MUFA intake with the DH in trial 2. People with diabetes in trial 2 may have reported intakes of MUFA which were in line with the intervention goals, but not reflected in their FRs. In contrast, the negative bias in MUFA measurement in trial 1 could have resulted from changes in food intake during the recording period to resemble those of the programme goals (increase in MUFA), something that has been seen with food records in the past[25].

In the case of SFA, differences in bias were dependent on the individual's body fatness or BMI. Reporting of SFA intake improved in subjects who were within the healthy weight range and then declined as subjects became fatter. SFA intake was underestimated by the DH in overweight and obese individuals. Again, a social desirability to report less fat may be reflected in a reduction in SFA reporting in overweight and obese individuals, i.e. a failure to accurately report SFA-containing foods like cakes, biscuits, pies and takeaway foods for fear of social non-acceptance. Outliers contributed to the SD_{diff}; however, their removal from the analysis only strengthened the negative relationship between bias and BMI in SFA reports. Because bias is given as a mean difference, often the full magnitude of bias at the group level is underestimated. Measurement of variability in bias (for example, by SD_{diff}) may be more useful to researchers in terms of bias at the individual level. In addition, the large intra-individual variation in measurement reinforces examining SD_{diff} in bias investigations as group means can dilute inter-subject differences in bias. Large variability in bias can be indicative of the need to consider the study context in adopting dietary assessment methods, which minimise bias.

It must be noted that bias in this study is relatively small in terms of macronutrients. Calculations of the difference in measurement between the two methods in terms of amounts of each macronutrient would be the equivalent of 3 g protein, 9 g CHO, 4 g fat, 3 g alcohol, 0.4 g MUFA, 3 g PUFA and 0.5 g SFA for all subjects at baseline in trial 1. In trial 2 the differences at baseline equate to 3 g protein, 4 g CHO, 2 g fat, 0.5 g alcohol, 0.5 g MUFA, 0.4 g PUFA and 0.9 g SFA. When these differences are translated into actual foods on an individual basis they are almost negligible;

however, a food-level analysis was beyond the scope of this paper.

Conclusion

Bias occurs with all measurement methods used in dietary assessment; however, the ability of investigators to simply identify or even quantify the sources of bias promotes better methodology for the future. We have examined bias in a DH method by cross-sectional and longitudinal means and found there to be no evidence of changes in bias over time during an intervention trial, regardless of trial length and the frequency of dietary monitoring. Subjects in both trials from both the intervention and control groups underreported energy, fat, alcohol and SFA intakes with the DH. Bias in both trials and in both groups also pointed towards an underestimation in reported SFA intake by the DH in overweight and obese individuals. It must be noted that our inability to influence the subject selection process meant that we performed this analysis knowing that subjects were probably interested in nutrition, which may have improved their reporting and recording capabilities in addition to enhancing their motivation to meet the dietary targets. Even though neither trial was designed to answer our research question, this simple retrospective method for determining relative bias can provide insight into sources of bias in dietary data from intervention research, which can then be investigated further using specific biochemical markers of intake.

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