Symposium: Dietary Composition and Obesity: Do We Need to Look beyond Dietary Fat?

Dietary Fat Intake and Regulation of Energy Balance: Implications for Obesity[1,2]

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ABSTRACT Obesity represents a major threat to health and quality of life. Although obesity has strong genetic determinants, the increasing prevalence of obesity in populations around the world suggests that environmental factors are promoting or exacerbating the problem. Experts are calling for public health efforts to deal with the global epidemic of obesity. Such a campaign would require that we identify and modify environmental factors that promote obesity. Our current food supply is high in fat, and high fat diets have been suggested to promote obesity by increasing energy intake, thus increasing the probability of positive energy balance and weight gain. However, others argue that high fat diets are not promoting obesity. In this paper, we review evidence from animal studies, carefully controlled laboratory studies, cross-sectional studies, clinical trials and studies in individuals at high risk to develop obesity. Although there are many environmental factors promoting excess energy intake and discouraging energy expenditure, it is clear that consumption of a high fat diet increases the likelihood of obesity and that the risk of obesity is low in individuals consuming low fat diets. On the basis of the available data, the current public health recommendations to lower dietary fat intake appear to be appropriate. J. Nutr. 130: 284S–288S, 2000.

KEY WORDS: • *body weight regulation* • *energy balance*

Obesity has reached epidemic proportions in the United States (Flegal et al. 1998), and the prevalence is increasing in most countries around the world (WHO 1998). Although genetics appear to play a major role in the regulation of body weight (Bouchard and Tremblay 1997), the rapid increase in the prevalence of obesity in the U.S. over the past two decades suggests that environmental factors are promoting, or at least favoring obesity in susceptible individuals. These factors must be identified to initiate successful public health efforts to prevent obesity and its associated health risks.

The human body has some ability to regulate energy in that energy intake (EI)[4] can be adjusted to energy expenditure (EE) and vice versa. The fact that body weight is maintained at a relatively constant level in most people over many years suggests that this must be the case. Studies of over- and underfeeding show that EE is altered to compensate for and to oppose changes in energy balance (E_{BAL}) (Horton et al. 1995, Roberts et al. 1996). However, these changes are not sufficient to completely counter the effects on EI, and a change in body mass results. When the over- or underfeeding stops, subjects tend to return to their initial body weights, usually by adjusting EI, again suggesting that some regulation occurs. The rapid increase in the prevalence of obesity suggests that environmental factors are exerting constant pressure to increase EI and decrease EE, and that the strength of these factors is overriding the strength of our defense mechanisms (**Fig. 1**).

There is evidence that consumption of high fat (HF) diets increases total EI and that excess dietary fat is stored with a greater efficiency than similar excesses of dietary carbohydrate or protein. However, the lack of weight loss with a reduction in dietary fat in some interventions has been interpreted to suggest that dietary fat does not play a role in the development of obesity (Willett 1998). In this paper, we review evidence for the role of dietary fat in regulating energy balance and promoting the development of obesity.

The role of dietary fat in regulating energy balance

Dietary obesity in animals. Diets that contain 30% or more of energy from fat reliably produce obesity in rats, mice, dogs and primates as a result of increased EI and efficiency of energy storage (Hill et al. 1989, Sclafani 1989, West and York 1998). Alternatively, obesity is rare in animals fed a low fat (LF) diet (<20%), even when animals are maintained in small cages that limit physical activity. Although there are strains and some animals within strains that resist becoming obese when fed HF diets (Pagliassotti et al. 1997), the chances of

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[4] Abbreviations used: DIT, dietary-induced thermogenesis; E_{BAL}, energy balance; EE, energy expenditure; EI, energy intake; HF, high fat; LF, low fat; RCT, randomized control trial.

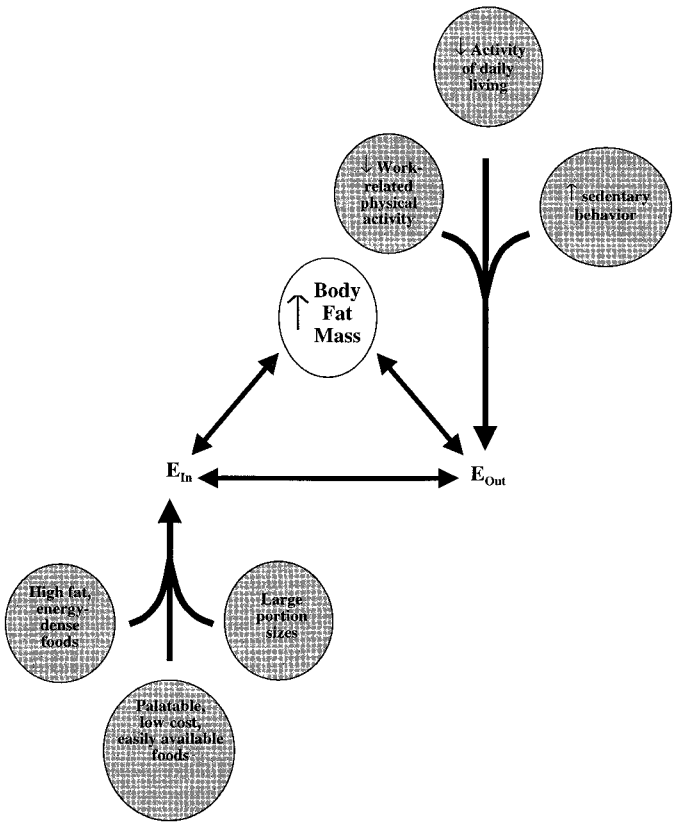

FIGURE 1 The effect of environmental factors on energy balance. When energy intake (E_{in}) equals energy expenditure (E_{out}), the system is in energy balance and body fat mass is stable. In the current environment, factors (in circles) on the left are driving E_{in} up, whereas factors on the right are driving E_{out} down, creating constant pressure toward positive energy balance, leading to an increase in the body fat mass.

becoming obese increase with increasing dietary fat (Salmon and Flatt 1985). Dietary obesity can be reversed by switching rodents from a HF to a LF diet, but the extent to which this occurs depends on the extent and duration of the dietary obesity (Hill et al. 1989).

Laboratory studies in human subjects. Several groups have studied the effect of diet composition on short-term (≤2 wk) energy regulation in humans in which the outcome was a surrogate for development of obesity, such as change in EI or in energy or fat balance (Lissner et al. 1987, Stubbs et al. 1995, Thomas et al. 1992). The advantage of these studies is the precision with which the composition of the diet can be manipulated, the self-selection of foods, and the use of measured rather than self-reported intake. Limitations include the need for subjects to consume required foods and differences in palatability and energy density between diets. Nonetheless, results are remarkably consistent with almost every study, showing higher total EI (~200 to 700–800 kcal/d) when consuming a HF diet (>40% of total energy). Subjects may consume more with consumption of HF diets because of the higher energy density (Stubbs et al. 1995), palatability (Schiffman et al. 1998) or other behavioral or metabolic differences (Reed et al. 1992). However, most of these studies compared diets containing 40% or more energy from fat to those containing 20% or less. In human subjects, we have very little information about the effects of dietary fat intake between 20 and 40% on energy intake.

The mechanisms by which excess dietary fat leads to positive E_{BAL} have been investigated in a number of paradigms. Dietary-induced thermogenesis (DIT) is lower when HF diets are consumed, compared with LF diets (Flatt et al. 1985). However, at an intake of 2000 kcal/d, the difference in DIT between a 20 and a 40% fat diet (assuming protein is constant) would be very small (~24 kcal/d). A more important factor is the efficiency with which excess dietary fat is stored. A greater proportion of excess energy is stored during fat overfeeding (~90–95%) compared with an equivalent amount of carbohydrate overfeeding (~75–85%) (Horton et al. 1995). Carbohydrate overfeeding produced progressive increases in carbohydrate oxidation and total EE, whereas fat overfeeding had minimal effects of fat oxidation and total EE. Although these differences may not persist during extended overfeeding, repeated bouts of HF overfeeding could lead to an accumulation of more excess energy than would repeated bouts of carbohydrate overfeeding. This scenario probably occurs in most individuals on a day-to-day basis.

Epidemiologic studies. Most, but not all cross-sectional studies show a modest positive relationship between dietary fat intake and one or more indices of obesity (Lissner and Heitmmann 1995). There are major limitations to these studies, including reliance on self-reported EI, adjustments for potential confounders, the inability of diet surveys to capture variability in dietary fat intake and the fact that group comparisons do not always characterize obesity in a population with accuracy. In the Diet and Nutrition Survey of British Adults, for example, the mean body mass index was similar in habitual LF (<35%) and HF (>45%) consumers, although the distribution was skewed to the right in the latter group (Blundell and Macdiarmid 1997).

Clinical trials. We identified 12 prospective, randomized-control trials (RCT) in 13 cohorts that compared a LF diet with a usual or control diet, and EI was measured (**Table 1**). Eight of these studies were ≥1 y in duration. These studies produced reductions in self-reported dietary fat intake of 2.8–18% with concomitant decreases in EI of 40–570 kcal/d (Bloemberg et al. 1991, Boyar et al. 1988, Boyd et al. 1990, Buzzard et al. 1990, Gatenby et al. 1997, Kasim et al. 1993, Lee-Han et al. 1988, Pritchard et al. 1996, Sheppard et al. 1991, Simon et al. 1997, Stefanick et al. 1998, Weststrate et al. 1998). Reducing intake of dietary fat also appears to be effective in reducing body weight. A recent review (Bray and Popkin 1998) found 28 intervention studies where subjects were asked to reduce dietary fat without energy restriction. There was an average weight loss of 1.6 g/day for each 1% reduction in dietary fat. A recent meta-analysis indicated that in 15 of 16 identified studies, reducing dietary fat led to a greater, yet modest decrease in body weight (2.5 kg, 95% confidence interval = 1.5–3.5 kg, $P < 0.0001$) compared with the control groups (Astrup et al., unpublished data). There were significant positive correlations between the reduction in dietary fat and amount of weight loss ($r = 0.37$) and between initial body weight and weight loss ($r = 0.52$). This latter result suggests that the greatest effect of a LF diet on weight loss might be seen in overweight subjects. However, none of these studies were specifically designed to assess the effects of a reduction in dietary fat on either body weight or EI, and few studies examined the effects in overweight or obese individuals.

A non-RCT by Knopp et al. (1997) has been used as evidence that LF diets are not effective in producing weight loss (Willett 1998). Hypercholsteremic, moderately overweight men were assigned to one of four dietary conditions (22, 25, 26, and 27% fat). Because all groups lost an equivalent amount of body weight (2–3 kg), Willett (1998) has argued that decreasing dietary fat does not contribute to weight loss.

TABLE 1

Randomized control trials of low fat diets fed ad libitum. Data taken data are not

Reference	Primary outcomes	Duration	Study subjects	Dietary goal	Group	F/M	BMI
Lee-Han et al. 1988	Breast dysplasia	1 y	Women with mammographic dysplasia	Reduce fat intake to 15%	I	29/0	23.3
					C	28/0	22.3
Boyd et al. 1991	Blood lipids	1 y	Women with mammographic dysplasia	Reduce fat intake to 15%	I	100/0	22.4
					C	106/0	22.8
Buzzard et al. 1990		3 mo	Women with stage II breast cancer	Reduce fat intake to 15%	I	17/0	28.6
					C	11/0	28.6
Insull et al. 1990	Blood lipids, body weight	2 y	Women at risk for breast cancer	Reduce fat intake to 20%	I	184/0	
					C	119/0	
Sheppard et al. 1991	Body weight	2 y	Women at risk for breast cancer	Reduce fat to 20%	I	158/0	26
					C	94/0	25
Bloemberg et al. 1991	Blood lipids	26 wk	Hypercholestermic men	Personalized diets to produce improvement in cholesterol profile	I	0/39	26.0
					C	0/41	26.3
Kasim et al. 1993	Blood lipids	1 y	Women at risk for breast cancer	Reduce fat intake to 15%	I	34/0	25.2
					C	38/0	28.0
Pritchard et al. 1996	Bone mineral density	1 y	Overweight men	Reduce fat intake to 20–22%	I	0/18	28.9
					C	0/19	28.7
Simon et al. 1997	Breast cancer risk	1 y	Women at risk for breast cancer	Reduce fat intake to 15%	I	65/0	24.8
					C	68/0	25.0
Stefanick et al. 1998	Blood lipids	1 y	Adults with moderate dyslipidemia	NCEP Step 2 diet (<30% fat)	I	45/0	26.3
					C	46/0	
					I	0/46	27.0
					C	0/49	
Westrate et al. 1998	Energy intake, body weight, blood lipids, antioxidant status	6 mo	Healthy, nonobese adults	Replacement of full-fat with reduced fat items	I	58/59	24.8
					C	51/52	25.0
Gatenby et al. 1997	Energy intake	10 wk	Healthy, nonobese females	Replacement of full-fat with reduced fat items	I	17/0	22.7
					C	13/0	24.2

[1] Data are means (SD).
[2] Abbreviations used: BMI, body mass index; I, intervention group; C, control group; HF, high fat diet; LF, low fat diet; NR, not reported; NC, no change; NCEP, National Cholesterol Education Program

This is an unconvincing argument. First, the range of differences in dietary fat across groups was small. Second, it may be that the reduction to 27% accounted for the majority of the weight loss effects and that further reductions provided minimal additional benefit. There may exist a threshold of dietary fat below which the changes of overeating are small and above which they are high. At present, it is not known if such a threshold exists or whether it is the same in all populations.

Low fat diets and the prevention of weight regain. Additional support for the role of LF diets in regulating body weight comes from studies of formerly obese individuals, a group considered to be at high risk for weight regain. After weight loss induced by an energy-restricted diet, weight regain at 2 y was less in subjects assigned to an ad libitum LF diet (5.4 kg) than those consuming a fixed energy intake diet (11.3 kg) (Toubro and Astrup 1997). Subjects in the National Weight Control Registry, a database of over 2000 individuals successfully maintaining a weight loss of at least 30 pounds for at least 1 y, report eating a diet low in fat (24%) and participating in very high levels of physical activity (Klem et al. 1997).

SUMMARY

The debate surrounding the role of HF diets in producing obesity has gained much attention in the scientific literature. This is surprising to us given the available data. Although we cannot point to a specific definitive study showing that HF diets cause or LF diets prevent obesity, the available data are consistent in suggesting that this is the case. We could not find a single study showing an advantage of HF diets over LF diets in reducing EI and preventing obesity. It is clear that the chances of becoming obese increase when consuming HF diets and decrease when consuming LF diets. Reductions in dietary fat consistently lead to proportional reductions in EI and modest, but potentially important reductions in body weight. The greatest potential effect of a low fat diet may not be in producing weight loss but in preventing weight gain.

Exactly why HF diets promote excess EI is not clearly understood, although energy density appears to be an important factor (Poppitt and Prentice 1996, Roberts et al. 1998). Nonetheless, reducing dietary fat is still a desired outcome, although a better understanding of energy density may lead to additional ways to modify the diet to prevent obesity. Whether manipulations in energy density, palatability and variety of snack foods play a role in reducing ad libitum intake of dietary fat requires more investigation.

There is an immediate need for research aimed at achieving population reductions in dietary fat intake. However, a food supply that is high in fat is not the only factor promoting

at the last measurement are reported, and thus intermediate presented[1,2]

Fat intake at start of intervention	Fat intake at end of intervention	Mean change in fat intake	Energy intake at start of intervention	Energy intake at end of intervention	Mean change in energy intake	Greatest change in body weight	Change in body weight at end of study
%	%	%	*kcal*	*kcal*	*kcal*	*kg*	*kg*
36.4 (7.2)	25.8 (8.8)	NR	1872 (330)	1655 (396)	NR	−1.1	−0.9
35.7 (6.1)	35.9 (7.3)	NR	1828 (405)	1881 (503)	NR	+0.6	+0.6
37.0 (7.0)	21.0 (8.0)	NR	1753 (382)	1543 (361)	NR	−2.0	−1.0
37.0 (6.0)	35.0 (6.0)	NR	1742 (406)	1742 (439)	NR	0.0	0.0
38.4 (4.3)	22.8 (7.8)	NR	1840 (419)	1365 (291)	NR	−2.8	−2.8
39.4 (3.7)	35.4 (6.3)	NR	NR	NR	NR	−1.3	−1.3
39.1 (6.1)	22.6 (7.0)	−18.2 (8.4)	1735 (396)	1321 (317)	−413 (335)	−3.1	−1.7
39.0 (6.8)	36.8 (7.9)	−0.9 (10.1)	1708 (438)	1555 (407)	−154 (449)	−0.4	−0.1
39.2 (6.3)	NR	−18.3 (7.9)	1743 (396)	NR	−395 (399)	−3.2	−1.9
38.9 (6.4)	NR	−1.0 (6.7)	1720 (446)	NR	−107 (466)	−0.4	−0.1
38.5 (7.1)	33.5	−5.0 (6.5)	2637 (676)	NR	NR	−0.9	−0.9
38.3 (9.0)	36.8	−1.5 (5.9)	2591 (695)	NR	NR	0.0	NC
36.3 (6.4)	17.6 (5.8)	NR	1927 (562)	1572 (404)	NR	−3.4	−3.4
35.6 (6.2)	33.8 (7.4)	NR	1697 (476)	1499 (464)	NR	−0.8	−0.8
38.2 (5.9)	25.4 (6.4)	NR	2605 (608)	1792 (406)	NR	−6.3	−6.3
38.5 (6.1)	39.2 (5.7)	NR	2223 (417)	2318 (417)	NR	+1.2	+1.2
36.0 (6.8)	18.0 (5.6)	NR	1876 (642)	1570 (379)	NR	−3.9	−1.5
35.9 (6.8)	33.8 (7.4)	NR	1676 (473)	1594 (513)	NR	−2.4	−2.4
NR	NR	−5.7 (7.4)	NR	NR	−220 (356)	−2.7	−2.7
NR	NR	−0.2 (6.7)	NR	NR	−19 (367)	+0.8	+0.8
NR	NR	−8.0 (8.1)	NR	NR	−285 (541)	−2.8	−2.8
NR	NR	−0.7 (5.9)	NR	NR	−25 (482)	−0.7	−0.7
39.1 (3.4)	33.9 (3.8)	NR	2510 (789)	2438 (669)	NR	NR	NR
39.9 (4.6)	42.2 (4.6)	NR	2510 (717)	2724 (645)	NR	NR	NR
37.0 (20.6)	33	−3.5 (4.0)	1986	1711	−275	NC	NC
34.0 (14.4)	NR	+1.0 (3.6)	2225	1933	−292	NC	NC

obesity in the U.S. Although not the focus of this review, it is clear from the literature that declining levels of physical activity are also contributing to the obesity epidemic. In a physically active society, our food supply might not produce high levels of obesity. We should expand our public health efforts to reduce dietary fat intake and increase physical activity.

We must move beyond the debate about the role of high fat diets in producing obesity. The message to the public should be to advocate consumption of a low fat diet, with <30% of calories coming from fat. Although we lack definitive data to suggest that this level is appropriate for the entire population, the available data suggest that this level of fat intake should greatly reduce the likelihood of overeating in most individuals.

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