Fat in the Diet and Obesity

Berit Lilienthal Heitmann¹ and Lauren Lissner²

¹Glostrup and Copenhagen University Hospital, Copenhagen, Denmark and ²Department of Community Medicine, Göteborg University Hospital, Göteborg, Sweden

**TRENDS IN FAT INTAKE**

During recent decades, trends of declining fat intake have been reported for most countries. However, both diet surveys and food disappearance data suggest that for most countries, fat intake is still above the recommended 30% of total energy. Countries like the USA and Norway have reported particular success in reduction of dietary fat intake (1,2). In the USA, for instance, a reduction in energy from fat from about 40% to 33% was achieved between 1960 and 1995 (1). In Norway, fat intake was reduced from 40% to 34% in only 15 years (2), making Norwegians a role model for the other Nordic countries, where a high fat consumption is the tradition. Finland (3) has followed this trend, and now reports patterns of decreasing fat intake similar to those in the USA and Norway. However, some countries, like Denmark and Sweden, still displayed intake figures for fat consumption as high as 39–43% until the late 1980s, and only recently have decreases been reported (4–6).

**INCONSISTENT TRENDS IN OBESITY AND FAT INTAKE**

Although trends in fat intake have been found to correlate closely with trends in cardiovascular disease, several studies have demonstrated that obesity is increasing at the same time that fat intake is decreasing (7). In fact, some of the countries that have experienced a substantial decrease in fat intake have noted the most dramatic increases in obesity prevalences. In Finland, for instance, prevalence of obesity rose from 10% to 14% in men and from 10% to 11% in women between the late 1970s and early 1990s (8) while at the same time fat intake decreased from approximately 38% to 34% (3). In the USA, data from the National Health and Nutrition Examination Survey (NHANES) show that while fat intake was decreasing, prevalences of severe obesity increased from 10% to 20% in men and from 15% to 25% in women (9). Although the possibility of ecological fallacy must be considered when interpreting such results, it cannot be excluded that the secular trends in obesity may have been even more dramatic if not for the decrease in fat intake.

Figure 10.1 shows trends in fat intake and obesity among adult Americans (9,10).

There are at least three interpretations for this paradox of opposite trends for fat intake and obesity. One is that people are decreasing their energy intake but also becoming less active. Another is that people are maintaining their energy intake despite the reduction in fat intake. A third explanation, is that fat intakes are not as low as reported. With regard to the first alternative, data from England have implicated sedentary activity as the most plausible explanation. For instance, trends in number of cars, or number of televisions per household, were more closely associated with the dramatic increase in severe obesity seen in England.
over the past two decades, than were trends in fat or energy intake (11). With regard to the second alternative, it has been suggested that total energy may well remain high, despite the proliferation of low fat products on the market. This may be due to the misperception that low fat products can be consumed without restraint (12). Finally, with regard to the third alternative, social desirability bias may cause people increasingly to report false low fat intakes, as official recommendations and public health campaigns to reduce fat intake are intensified and disseminated.

**BIAS IN DIET SURVEYS**

In dietary surveys, there is a great potential for bias deriving from a tendency for obese subjects to underreport their food intake to a greater extent than normal weight individuals (13,14). In addition, it was recently demonstrated that the obese underreport total energy more than energy from protein, suggesting that the other energy-yielding nutrients must have been underreported disproportionately (15) (Figure 10.2). This is in general agreement with the assumption that obese individuals underreport socially undesirable foods such as those rich in fat and/or simple carbohydrate. The consequence of both specific and non-specific underreporting by the obese is that the diet–obesity and diet–health associations may be distorted (16). In addition, diet–obesity associations found in dietary surveys may not reflect those found in the general population, since non-responders generally are more likely to be obese, more likely to be smokers, more likely to have a low educational level (17), and may have a different dietary consumption pattern than respondents. Finally, older food databases may not sufficiently capture nutrient composition of these new low fat products, and hence, add to the creation of biased diet–obesity relationships.

**EVIDENCE LINKING FAT TO OBESITY**

Cross-sectional evidence from studies linking intake data to degree of obesity clearly suggests that dietary fat is associated with overweight. In a recent review, Lissner and Heitmann (18) found that in more than 80% of the cross-sectional studies reviewed, dietary fat intake was associated with obesity. The diet of obese subjects has been found to contain 5–8% more fat than the diet of the normal weight control groups (19), and this is likely to be a conservative estimate if obesity-related underreporting of fat occurs. Furthermore, most experimental studies provide evidence that, compared to a covert low fat diet, spontaneous energy intake is
increased following ingestion of a high fat diet (20–22). In addition, a number of studies have suggested that appetite control is dissociated from fat since energy intake of a subsequent meal was not suppressed by adding up to 60 g fat (2.3 MJ) to the previous meal (for review see Astrup and Raben (19)). Likewise, one study demonstrated that 2-year maintenance of weight loss was more successful on an ad libitum low fat diet than on calorie counting (23). Finally, randomized dietary fat intervention studies consistently show that weight is lost on a calorically unrestricted low fat diet, although rates of weight loss per day are modest (ranges between 17 g and 37 g per day) (18).

The specific mechanism for dietary fat in obesity development is generally believed to depend on passive over-consumption on the high fat diet, due mainly to its high energy density and also palatability related factors. However, the effect is likely to vary under different circumstances. For instance, fat intake has been reported to predict weight gain in sedentary, but not inactive women, suggesting that certain subgroups of the population may be particularly sensitive to over-consumption of a high fat diet (24). The hypothesis that genetic factors determine the identity of fat-sensitive individuals will be discussed in more detail later.

A possible energy independent effect of the fat related to a lower satiating power has also been suggested (25,26). Oscai et al. (27) demonstrated that rats developed obesity when fed fat-rich, but not low fat equi-caloric diets. Other mechanisms include a particular storage preference (28) and/or a lower thermic effect of fat compared to carbohydrate (29) or protein. Proposed mechanisms linking fat to obesity development are given in Table 10.1 (29).

### EVIDENCE UN-LINKING FAT FROM OBESITY

A number of observational studies have suggested that the role of fat in obesity development may be exaggerated, and suggest that the population differences in weight do not appear to be due primarily to the fat intake (30). For instance, in a recent literature review, it was found that short-term hypophagia on low fat diets is compensated in the longer term (18). A report from the National Centre for Health Statistics has shown that Americans today eat less fat but more calories than earlier, a finding that may explain the rise in obesity, but acquits the role of fat (31). In addition, results from
prospective observational studies give inconsistent results, and do not support the relation between a high fat intake and subsequent weight gain. Klesges et al. (32), for instance, found a clear positive association between dietary fat and subsequent weight changes, while Colditz et al. (33), using the same dietary instrument, and Kant et al. (34), using a different instrument, could not find such an association. Other studies have also shown inconsistent results (35–37). Indeed, Katan (38) recently reviewed the literature on long-term fat trials, and found that low fat diets in these trials had resulted in decreases in body weight of only 0.4–2.6 kg relative to control diets. Based on this, he concluded that the evidence from the long-term trials could not support the idea that a high proportion of fat in the diet could be responsible for the 10–15 kg weight gain that people in affluent societies experienced between adolescence and middle age.

Results from feeding studies in humans, with covert manipulation of energy density in the diet, suggest that increase in the fat content does not result in an increase in energy intake, when density is constant (39). Also Stubbs et al. (40,41) manipulated macronutrient intake in iso-caloric diets and found that the energy density of the diet was dissociated from the fat content, since spontaneous energy intake remained unchanged as the fat increased from 20% to 60%.

In experimental animals, Ramirez and Friedman (42) demonstrated that increasing the energy density of the chow food led to an increase in energy intake, but did not affect the weight of the food eaten. Results like these suggest that fat is only less satiating than carbohydrate if the energy density is tied to the fat content, and hence, challenge the specific role that has been attributed to fat in obesity development. Rather, results like these imply that it is the bulk, or the weight, of food that controls satiety. Indeed, Westerterp et al. (43) recently demonstrated that change in the fat content of the diet resulted in change in body composition only when energy intake was simultaneously changed, and several studies have found the weight of food eaten to be more constant than daily energy intake (20,22,44). Hence, an increasing energy density may result in passive over-consumption only because individuals habitually eat a constant weight of food per day (40,41).

Finally, it has been suggested that the benefits of decreasing the density of one meal, by removing selected high fat items from the diet, may be compensated by the inclusion of high fat items in a later meal (45). On the other hand, this leaves a greater potential for diluting diets, for instance with fibre. In fact, it may be argued that the benefits of a low fat diet with regard to obesity may depend not on the low fat per se, but on the accompanying high fibre content (Table 10.1).

A SPECIFIC ROLE FOR GENES?

Genetic susceptibility for weight gain may be influenced by dietary factors, such as fat intake (46). Indeed, a few studies have indicated that development of obesity is, in part, due to differential effects of fat in the diet for those who are genetically predisposed, compared to those who are not (47–49). In this context, studies in both animals and humans have demonstrated that food intake seems to play a specific role for obesity development in association with a predisposition to obesity (37,50,51). For instance, Selefani and Assimon (52) found that obesity prone mice ate more high fat, but less sugar-rich foods than leanness prone mice. In addition, obesity prone mice have been found to gain weight at a much faster rate than wild-type mice fed the same high fat diets (50), suggesting a gene–environment interaction between the high fat diet and the subsequent weight gain. Furthermore, compared to non-obese controls, impaired ability to increase the fat/carbohydrate oxidation ratio in response to a high fat diet has been suggested in post-obese women (51), implying that this obesity prone group is particularly susceptible to weight gain on such a diet. Although not all studies have been able to document gene–environment interactions relating a high fat intake to weight gain (53,54), a study using identical twins found that weight gain in response to controlled overfeeding was more similar within identical twin pairs than between pairs of twins, suggesting a specific genetic influence (47). Finally, the specific role of fat in obesity development may be restricted to those who are predisposed, only. This was the case in one study where women with a familial history of obesity had a stronger risk of major weight gain compared to women without such a predisposition, when consuming a high fat diet (37).

Several genes have been proposed, for instance
Table 10.2  Evidence for and against a genetic component

- Obesity prone mice have been found to prefer high fat diets.
- Weight gain in obesity prone mice is higher than in wild type mice fed the same high fat diets.
- Subjects with a familial predisposition to obesity seem to have an impaired ability to increase the fat/carbohydrate oxidation ratio in response to a high fat diet.
- One study found that women with a familial history of obesity had a stronger risk of dietary fat-related weight gain compared to women without such a predisposition.
- Two studies found no evidence of a genetic predisposition for dietary fat-related weight gain.

Long and obese/obesity prone individuals seem to vary in their response to fat manipulation, since obese individuals have been found to compensate less well than lean unrestrained individuals for energy intake in response to preloads of varying energy densities (56). Furthermore, not only do obese subjects generally report liking high fat foods more so than lean subjects (57,58) they also may not be as sensitive to the satiety value of fat as lean subjects (20,59). Indeed, the literature suggests that the selection of macronutrients is, in part, heritable (60). For instance, obese rodents have been found to avoid sweets compared to their lean littermates, and genus that mediate the consumption of sugar have been mapped, and are being isolated and characterized. For sweet taste preference, major gene effects have been described, mapping studies of genetic loci have been published, and single-gene mutants have been discovered. However, only a few studies have assessed the role of genetic variability in dietary fat preference, but so far no genes have been characterized (60,61). In addition, it has been proposed that the development of obesity may be viewed as a regulatory mechanism by which the impaired lipid oxidation rate in the body is raised to match the dietary fat intake (27). This capacity to increase lipid oxidation after consumption of a high fat diet may vary greatly among individuals, and may depend on both physiological and genetic factors.

Finally, the genetically determined ability to taste bitter substances relates to obesity and may also be associated to fat preferences. Obese subjects are less sensitive to the bitterness of phenylthiocarbamide, and hence, the gene that determines this bitter taste polymorphism may either have effects on both dietary fat perception and body weight, or be linked to genes contributing to these phenotypes (60) (Table 10.2).

CONCLUSION

In summary, the epidemiological evidence linking fat intake to obesity must be considered together with evidence that obese individuals underreport food intake in general, and may underreport fat intake in particular. Such reporting biases would imply that studies finding that obese individuals consume more dietary fat than non-obese are likely to have underestimated the difference in intake. Furthermore, these reporting biases may distort the fat–obesity associations in unpredictable ways, creating both spurious and conservative associations (16,62). However, obesity related social desirability bias may not explain the opposing secular trends between fat intake and obesity prevalence at the population level, since these recent dietary trends are often based on aggregate-level food disappearance data, rather than reported diets.

Although the available epidemiological studies indicate that dietary fat does not play the leading aetiological role in the epidemic of obesity, the likelihood that the data are systematically biased makes it impossible to speculate on the true magnitude of any such effect. Thus, methodological constraints necessitate cautious interpretations of epidemiological data. However, the evidence from experimental research, linking fat intake to obesity, seems to suggest that energy density, and not fat intake, is the most likely mechanism here. However, the distinction between fat and energy density is somewhat artificial, since these dietary properties tend to be parallel.

Although the role of restricting dietary fat has been advocated in both prevention and treatment for obesity for a number of years, the data available
today suggest that dietary fat is probably one small part of a multifactorial aetiology. In this regard, there is some evidence that a low fat diet may be protective to specific subgroups of the population, and hence, be more relevant to obesity in a preventive rather than a treatment perspective. One such group may be subjects with a propensity to weight gain, who may be identified by their familial clustering of obesity. Another group may include the physically inactive, who could benefit from an extra margin of safety provided by a low fat diet in protection against positive energy balance. Other subgroups who might benefit are ex-smokers, during the initial quitting period, and consumers who are able to use fat-reduced products in a rational way. However, it must be kept in mind that low fat diets offer no panacea, since individuals with a predisposition for overeating appear to learn how to do so, even on fat-reduced diets.

In conclusion, focusing on total fat intake may be beneficial to some individuals, but should not remove the focus from total energy balance. The vast amount of studies on dietary fat intake and obesity may have created a misperception that as long as a food is low in fat, there is no limit to the amount that can be consumed without gaining weight.

REFERENCES

28. Flatt JP. The difference in the storage capacities for carbohy-