Fat as a risk factor for overconsumption: Satiation, satiety, and patterns of eating

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ABSTRACT

Many people experience great difficulty in preventing energy intake from outstripping energy expenditure. Eating high-fat foods can facilitate the development of short-term positive energy balances by influencing satiation and satiety, the processes that control the size of eating episodes and the strength of postingestive appetite inhibition, respectively. An important feature of these processes is the relative potency of orosensory, postingestive (preabsorptive), and postabsorptive signals. Foods high in dietary fat have a weak effect on satiation, which leads to a form of passive overconsumption, and a disproportionately weak effect on satiety (joule-for-joule compared with protein and carbohydrate). This overconsumption (high-fat hyperphagia) is dependent upon both the high energy density and the potent sensory qualities (high palatability) of high-fat foods. A positive fat balance does not appear to generate a tendency for behavioral compensation, and there appears to be almost no autoregulatory link between fat oxidation and fat intake. The Leeds High Fat Study has found a higher frequency of obesity among high-fat than low-fat consumers, but the relationship between fat consumption and obesity is not a biologic imperative; analysis of the pathways between daily fat intake and patterns of eating has revealed high-risk eating episodes. The physiologic responses to fat ingestion appear to be weak compared with the potent orosensory properties of high-fat foods, and such responses cannot prevent overconsumption. A first stage in a health program should be to prevent passive overconsumption. J Am Diet Assoc. 1997;97(suppl):S63-S69.

What happens when people are enveloped in a food supply containing an abundance of high energy dense foods (usually high in fat) that are extremely palatable, actively promoted, and a major part of modern culture? Observation and evidence suggest that many persons cannot maintain an energy intake that does not exceed energy expenditure (which may be quite low). Can people eat freely, to derive pleasure and satisfaction from food, without fear of overconsumption? In addition, what happens when people try to reduce their fat intake? Can people adjust their pattern of eating without inducing compensatory responses? Considering the alarming and continuing increase in the frequency of obesity, it is clear that many people have great difficulty in preventing energy intakes from outstripping energy expenditures. Specific biological and psychological features appear to allow people to drift with apparent ease into a positive energy balance that results in an inevitable gain in weight.

FAT INTAKE, SATIATION, AND SATIETY

Although the body appears to contain potent physiologic responses that are triggered by fat ingestion, many studies have demonstrated that people who consume high-fat foods (either through personal choice or in experimental situations) tend to overconome energy (1,2). This easy overconsumption of energy does not occur readily with high-carbohydrate foods, although in some studies in which the proportions of fat and carbohydrate were varied, the two macronutrients were shown to have equivalent satiating power (3). Because of the potent action of protein on satiety, it is necessary to maintain protein as a constant when comparing fat and carbohydrate. In one study in which fat displayed a satiety action similar to carbohydrate, the fat foods contained much higher amounts of protein (4). Consequently, the apparently strong satiating action of fat could have been due to the presence of the protein. Therefore, the satiating power of dietary fat may be altered according to its association in a food with either protein or carbohydrate (5). Such an effect could explain instances where fat caused a potent inhibition of subsequent eating (6).

Satiation is the process that occurs while foods are being eaten; satiety is the state engendered as a consequence of consumption (7). Satiation controls meal size, while satiety measures the capacity of food to control subsequent hunger and eating and commonly is called satiating efficiency (8). In considering dietary fat as a risk factor in overconsumption, the effect on satiation is likely to be more important than that on postingestive satiety (9).

PASSIVE OVERCONSUMPTION

Interestingly, most experiments on fat and appetite have used some variation of the preload or fixed meal presentation in

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which individuals are required to consume an obligatory amount of fat (or fat mixture). The consequences of this mandatory consumption (determined by the researcher) then are measured. This procedure offers a measure of satiety (not satiation). This approach probably provides only limited information about the effect of fat on appetite and may preclude complete understanding of the effect that dietary fat has on energy intake. An alternative procedure, sometimes called concurrent evaluation (10), can be used to assess the effect that fat content of foods has on the amount willingly consumed (satiation) when individuals are provided with a range of foods and allowed to eat freely to comfortable fullness. Use of this procedure has demonstrated that people consume much greater quantities of energy from a range of high-fat foods than from foods high in general carbohydrates (11) or sucrose (12). This effect, which is particularly strong in obese people (13), has been termed high-fat hyperphagia (14) or passive overconsumption (15). Because the effect almost certainly is due, in large part, to the high energy density of high-fat foods, it can be regarded as a passive form of high consumption rather than as actively driven eating. Use of the term passive means only that there is no deliberate intention on the part of the eater to overconsume. People normally are not endeavoring to eat as much as they possibly can; rather, a number of mechanisms contribute to passive overconsumption. For example, ingested fat can stimulate food intake, and the orosensory effects of fat appear to stimulate intake in rats (16). Additionally, the combination of fat and alcohol can stimulate overeating independently of energy density (17).

Although high-fat foods induce remarkably high levels of energy intake (primarily fat energy) in meals or snacks, the experimental design still permits a measure of postigestive effects on satiety. Interestingly, these very high levels of consumed fat energy do not induce any noticeable intensification of satiety when compared with lower energy intakes of high-carbohydrate foods. Therefore, high-fat foods can stimulate high intakes of fat energy without a proportionate increase in satiating power. Consequently, a single meal (or snack) of high-fat foods normally leads to a significant increase in that day's food consumption. These effects may explain the relationship of the eating pattern to the long-term overconsumption of energy on a high-fat (low-food quotient) diet (18).

The methodology previously outlined demonstrates overconsumption in connection with the intake of high-fat foods, as seems to happen in real life, indicating a genuine point of contact between "real world" events and laboratory research. The methodology emphasizes that it is necessary to measure the effects of fatty foods while they are being consumed (satiation) as well as after consumption (satiety). Indeed, it appears that the most important action of dietary fat on appetite occurs while it is being consumed rather than after ingestion.

THE FAT PARADOX
The relationship between fat and satiety presents a paradox. Although fat in the intestine appears to generate potent satiety signals (19), exposure to high-fat foods leads to a form of passive overconsumption that suggests a weak effect for fat on satiety (12). The paradox can be expressed as the puzzle of fat-induced satiety and high-fat hyperphagia.

Although fat induces physiologic responses that should inhibit food intake, rats placed on high-fat diets or given fat supplements take in excessive amounts of energy and gain weight rapidly. Humans given a range of high-fat foods also increase their energy intake and gain weight compared with those eating a medium- or low-fat diet. In addition, high-fat foods markedly increase meal size (measured in terms of energy), an effect particularly evident in obese individuals (13).

Although emulsified fat delivered to the intestine (duodenum or jejunum) produces prompt satiety signals, fat consumed orally takes some time to reach the intestine in similar form, and its action is likely to be diluted by other nutrients. Hence, consumed fat may engender more slowly arising satiety signals. Two features of fat favor the rapid consumption of energy: Fat produces potent oral stimulation, which facilitates intake, and high-fat foods normally have a high energy density, which means a large amount of fat energy can be consumed before fat-induced satiety signals become operative. These satiety signals apparently are too delayed to prevent the intake of large amounts of this food. One of the consequences of a food supply containing readily available, very palatable, high-fat foods is that the natural fat-induced satiety mechanism becomes overwhelmed.

Indeed, in a series of experiments using a research design that measures both satiation and satiety, we have observed striking differences with participants allowed to consume from ranges of high-carbohydrate or high-fat foods on separate occasions (20). With the high-fat foods (high energy density), participants invariably consume a significantly greater amount of energy (sometimes double) when eating to a point of comfortable fullness. However, the actual weight of food consumed (total mass) is significantly less with the high-fat foods. The primary function of a satiety signal should be to limit the amount of food being put into the mouth (an inhibition of eating behavior). Therefore, the fat paradox can be resolved.

The dietary override of physiologic satiety signals has a number of implications. First, it is clear that the effect of fat per se should be separated from the effects of high-fat foods. References to the effect of dietary fat and satiety should recognize that the effects of fat delivered in controlled amounts to the intestine may demonstrate effects that still are present but that are eclipsed when people eat large quantities of high-fat foods. Second, the recognition of this paradox suggests nutritional and pharmaceutical strategies for reducing fat consumption. Any technique that would advance or intensify fat-induced satiety signals or that would prevent the effects of high-fat foods on satiation would prevent the passive overconsumption of fat energy that heralds good control of appetite.

One further implication of the uncoupling of eating behavior and energy intake is that, through passive overconsumption, fatty foods have a strong tendency to generate an immediate positive energy balance. This is apparent in periods of rest, normal activity, and even high levels of exercise (cycling or running) (21). Because this positive energy (and fat) balance does not generate any strong compensatory responses, the short-term positive energy balance probably is incorporated into body energy stores.

FAT INTAKE AND FAT CONSUMERS
One useful procedure to investigate the effect of fat on short-term energy balance is to use laboratory experiments to model natural eating patterns. In this way, the effect of foods of varying types upon satiation and satiety (precisely monitored) during free consumption can be measured. Another procedure is to characterize natural high- and low-fat consumers identified through large scale surveys or epidemiologic studies.

The Leeds High Fat Study (22) identified such consumers in Leeds, United Kingdom. In addition, the database from the National Diet and Nutrition Survey of British Adults (DNSBA) (23) was reanalyzed to define the relationship between dietary fat, food consumption, and body mass index (BMI).

High- and low-fat consumers were defined as consuming >46% or ≤36% of their food energy as fat, respectively. When
nutrient intake is defined as percentage of total food energy consumed, carbohydrate intake falls as fat intake increases (Figure 1).

DNSBA data indicate that the greatest contributor to fat in the diet is meat and meat products, which provide 25% of the total intake of fat. Both men and women in the high-fat groups derived the greatest percentage of their fat intake from meat and meat products, whereas cakes, pastries, and biscuits (cookies) contributed the most fat to the diet in the low-fat groups. Male high-fat consumers ate significantly more high-fat meat products (bacon, lamb, meat pies, sausages, burgers, fried white fish), butter, whole milk, cheese, eggs, savoury snacks (i.e., crisps), desserts, and alcohol. They also ingested less breakfast cereal (including high-fiber cereals), skimmed milk, low-fat spreads, chicken, yogurt, potatoes (not fried), fruit, and table sugar. The women's dietary patterns were similar to the men's, but there were fewer differences between high- and low-fat groups in terms of consumption of high-fat meat products. Bacon, meat pies, and sausages were the only meat products that differentiated the female high- and low-fat groups. These differences were consistent with variations in food intake found between high- and low-fat consumers in the Leeds study.

THE PUZZLE OF SUGAR AND FAT IN THE DIET

A growing confusion has emerged over the role of sugar and fat in energy balance and their influence on body weight. It long has been contested that foods high in sugar and fat are specifically targeted by food cravers (24) and, therefore, represent a basis for overeating. This hypothesis is supported partially by the observation that obese women rank sweet fat foods highly in their listing of preferences (25). More recently, the combination of sweet high-fat foods in the diet has been regarded as a potential source of weight gain (26), and this has been accompanied by a recommendation to reduce both fat and sugar in the diet as a means of curtailing the rise in obesity.

However, much publicity has been given to the concept of the "sugar-fat seesaw" (27), which is based on the observation that when the energy consumed as sugar or fat is expressed as the percentage of total energy consumed, a reciprocal relationship becomes apparent. Because of this inverse association, it is argued by some that it is impossible to reduce intakes of both fat and sugar because a reduction in one automatically will cause a rise in the other (28).

Recent analyses of some large databases appear to support the sugar-fat seesaw. For example, using data from the MONICA study and the Scottish Heart Study, the dietary components of fat and sugar were calculated as quintiles and then related to BMI (29). Ascending quintiles of fat consumption were related inversely to the quintiles of sugar intake, and a high BMI was associated with the highest quintile of fat intake (and, therefore, the lowest quintile of sugar). Accordingly, BMI was positively related to the fat:sugar ratio. This pattern also is reflected in the analysis of the United Kingdom database forming the DNSBA (30) in which a high intake of sugar (as a percentage of total energy) was associated with the lowest BMI. This collection of studies appears to confirm that the intake of fat clearly is related to obesity, while the intake of sugar is associated with leanness. It appears to follow that sugar and fat are related inversely in the diet, thereby making consumption of less of one lead inevitably to consumption of more of the other.

However, the issue is not completely resolved by these analyses of large databases. It has been noted previously, in the Leeds High Fat Study, that fat and carbohydrate intakes are related inversely when these nutrients are expressed as percentages of energy in the total diet. When a similar analysis for fat and sugar is conducted on a large database such as the DNSBA (OPCS) survey, these two dietary components are related inversely when expressed as percentages (the sugar-fat seesaw), but positively related when expressed as grams (31). Consequently, different outcomes are obtained when exactly the same data are represented in two different ways (Figure 2).

At present, it is not clear whether the observed relationships between fat and sugar reflect processes in physiology, nutri-
tion, or food choice (eating behavior). When people are allowed to eat from ranges of high-fat or high-sucrose foods, passive overconsumption only occurs with fat (32). It follows that fat promotes overconsumption while sucrose probably prevents it. As long as foods containing either fat or sucrose are separately clearly, then processes of food choice probably will ensure that high intakes of sucrose (in people with a sweet tooth) will be related inversely to fat intake (people who prefer fat). Of course, some people have a preference for both sweet items and fatty foods. There also is a question of whether single foods that combine fat and sugar (e.g., cakes, chocolates, pastries) are eaten by the sweet-tooth, fat-tooth, or both groups. Does the presence of sweetness (sucrose) in these products increase their palatability, making them more likely to be consumed and thereby inevitably raising fat intake? The expression of nutrient intakes in absolute weights demonstrates that large amounts of sucrose and fat can coexist in the diets of particular persons.

One interpretation is that low-fat diets are not likely to stimulate passive overconsumption or to result in a short-term positive energy balance. Low-fat consumers will eat large amounts of carbohydrate, some of which will be sugars, but this sugar does not appear to constitute a threat of obesity. In contrast, high-fat foods have the potential to cause overconsumption and sweet fat foods are particularly potent under some conditions (33). In some persons the sweet characteristics of fatty foods will facilitate consumption (through processes influencing food choice); sweetness in this case will be associated with a high fat intake and contribute to a positive energy balance and weight gain. Thus, sweetness, in the form of sucrose or high-intensity sweeteners, can contribute to a positive energy balance in high-fat consumers who also have a sweet tooth.

Some evidence for this hypothesis can be seen from an examination of the data from one national database in the United Kingdom. In Figure 3, absolute intakes of sugar and fat are displayed for four groups of persons varying in BMI. The levels of total sugar intake are approximately equivalent across the BMI categories, but there is a clear and progressive increase of fat intake from the lowest to the highest BMI group. The lowest BMI group has a substantially greater ratio of sugar to fat than the obese group, but the consumption of sugar is not displacing fat from the dietary intake. The feature that distinguishes the highest BMI group from the lowest is not a decline in total sugar intake but an increase in fat consumption. Consequently, a high intake of fat is compatible with a high intake of total sugars, which appears to occur in persons who are attracted by both sweetness and fat in foods. Of course, the cross-sectional data displayed in Figure 3 do not allow any statement about causality. It cannot be claimed (on the basis of these data) that high sugar and high fat intakes actually cause obesity, although it can be hypothesized that this dietary scenario would encourage a positive energy balance. The data do suggest that the physiologic status of high BMI persons does not impede the consumption of high levels of sugar and fat.

**HIGH-FAT CONSUMPTION AND BMI**

One consequence of a positive energy balance induced by exposure to high-fat foods should be an increase in body weight. Evidence linking dietary fat and BMI already exists. It follows that natural high- and low-fat consumers should show differences in average BMI and the frequency of obesity. Computations have been carried out with DNSBA. The sample was defined according to the percentage and the absolute amount of fat consumed. To ensure the inclusion of valid data, persons who reported being ill or who were dieting during the study period were excluded from analysis. In addition, it is argued
that a ratio of energy intake (EI) to basal metabolic rate (BMR) of less than 1.2 is not compatible with habitual intake and normally signifies underreporting of energy intake (34). Therefore, those who had an EI:BMR of less than 1.2 were excluded.

The most meaningful relationship is the distribution of BMIs for the high-fat and low-fat groups, classified by fat grams consumed per day (Figure 4). There is a greater positive skew in the distribution of BMIs of high-fat consumers. Defining obesity as a BMI of more than 30, there were 19 times more obese subjects in the high-fat group than there were in the low-fat group. Indeed, among the sample of low-fat consumers, only one person was obese. Thus, a low-fat diet appears to offer substantial protection against the development of obesity.

However, not everyone in the sample identified as eating a high-fat diet was obese. Indeed, there were many normal weight and even some underweight people among the high-fat consumers. This suggests that some people can resist the weight-increasing properties of high-fat diets. This resistance could be behavioral or physiological. For example, some persons may expend extremely high levels of energy in physical activity or they may have high BMIs or high rates of fat oxidation. Although a high-fat diet facilitates weight gain and promotes obesity, it does not appear to constitute a biologic imperative that inevitably leads to obesity. Of course, in cross-sectional data such as these it is not possible to know the length of time for which people have been consuming a particular type of diet, which obviously would affect body weight.

**FAT INTAKE AND EATING PATTERNS — LEEDS HIGH FAT STUDY**

If the passive overconsumption that can occur when people are exposed to high-fat foods can, in turn, generate a positive energy balance that is not offset by later compensation, then fluctuations in fat intake become particularly important. This issue could affect the relationship between high-fat and low-fat consumers and BMI. For example, persons with a low mean intake of dietary fat (<35% food energy) occasionally could consume a surfeit of fat at a particular meal or during a particular day. This high consumption probably would not endure long enough to induce a rise in fat oxidation and, therefore, the positive energy balance would be incorporated into stores. It has been demonstrated that an increase in fat oxidation only occurs after 3 to 7 days of consistent high-fat feeding (35,36). With shorter periods of high-fat eating, the body would deal with accumulating positive fat balances. Therefore, it is important to examine daily fluctuations in fat intake.

In the Leeds High Fat Study, a small cohort of high- and low-fat consumers taken from the larger sample have been investigated in detail using food frequency questionnaires, 7-day weighed food diaries, and intensive interviewing. Daily profiles of nutrient and energy intake have been plotted from the diary records. Examples of a low-fat and a high-fat consumer are shown in Figure 5.

The profiles indicate considerable variation in mean daily fat intake for each person, which was true for most of the people in the cohort. Although the daily mean fat intake for the high-fat consumer is 63.7 g (the United Kingdom average for women is 78.0 g), on 2 of the 7 days of the food record, fat intake markedly exceeded this value, reaching almost 140 g on 1 day. For the high-fat consumer, the mean daily intake of 154 g failed to reveal daily fluctuations varying between 80 and 210 g.

The logic of the Leeds High Fat Study has carried the investigation one stage further to identify the particular pattern of eating that occurred on the days of the highest fat consumption for both high-fat and low-fat consumers. These patterns, shown in Tables 1 and 2, indicate how fat consumption can be traced from daily averages to specific eating episodes involving particular types of foods. This type of analysis has revealed different pathways to a high fat intake and demonstrated the particular vulnerabilities in eating patterns that constitute risk factors for a high fat intake.

The effect of daily fluctuations such as these on biologic end points such as obesity (or other disease states) is not known, but the effects may be different from those expected from laboratory studies in which constant high-fat or low-fat manipulations are administered. For a low-fat consumer, it can be surmised that occasional days of high fat (and high energy) intake would lead to a positive fat (and energy) balance; if this were not compensated by later behavioral or metabolic adjustment, then a small increase in body energy stores would occur. Repeated occurrences would be expected to have an effect on BMI or distribution of body fat (reflected in the waist-hip ratio).

**FAT SUBSTITUTION — PREVENTION OF WEIGHT GAIN**

Present epidemiologic data indicate that average BMI is continuing to increase in most countries, which implies that many individuals are in a state of positive energy balance that normally means a positive fat balance (37). Consequently, a first stage in dealing with the problem of obesity must be to arrest the present rate of weight accumulation by preventing the occurrence of a positive energy (fat) balance.
Table 1
Pattern of eating leading to a high intake of fat on one day in a low-fat consumer (see Figure 5, ID 3063, Wednesday)*

<table>
<thead>
<tr>
<th>Meals</th>
<th>Sources of fat in meals</th>
<th>Fat content (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cereal (bran hearts)</td>
<td>Bran hearts</td>
<td>5.9</td>
</tr>
<tr>
<td>with semiskim milk</td>
<td>Semiskim milk</td>
<td>1.4</td>
</tr>
<tr>
<td>Coffee (milk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midmorning snack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coffee (with milk)</td>
<td>Whole milk</td>
<td>1.0</td>
</tr>
<tr>
<td>Lunch (buffet)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small ham sandwich</td>
<td>Butter</td>
<td>40.8</td>
</tr>
<tr>
<td>Small chicken sandwich</td>
<td>Mayonnaise</td>
<td>35.7</td>
</tr>
<tr>
<td>(with mayonnaise)</td>
<td>Cheddar cheese</td>
<td>13.8</td>
</tr>
<tr>
<td>Small egg mayonnaise</td>
<td>White rolls/bread</td>
<td>8.9</td>
</tr>
<tr>
<td>finger roll</td>
<td>Onion bhajia</td>
<td>7.5</td>
</tr>
<tr>
<td>Small cheese finger roll</td>
<td>Tuna (in oil)</td>
<td>4.0</td>
</tr>
<tr>
<td>Small tuna fish finger roll</td>
<td>Egg</td>
<td>3.2</td>
</tr>
<tr>
<td>Onion bhajia</td>
<td>Ham</td>
<td>1.2</td>
</tr>
<tr>
<td>Glass of diet cola</td>
<td>Chicken</td>
<td>0.5</td>
</tr>
<tr>
<td>Mid-afternoon snack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coffee (black)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuna chili</td>
<td>Tuna</td>
<td>5.6</td>
</tr>
<tr>
<td>Brown rice (boiled)</td>
<td>Brown rice</td>
<td>0.8</td>
</tr>
<tr>
<td>Salad (lettuce, cucumber, tomato)</td>
<td>Banana</td>
<td>0.3</td>
</tr>
<tr>
<td>Banana</td>
<td>Lettuce</td>
<td>0.1</td>
</tr>
<tr>
<td>Evening snack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crispbread with low-fat spread</td>
<td>Low-fat spread</td>
<td>5.6</td>
</tr>
<tr>
<td>Coffee (milk)</td>
<td>Semiskim milk</td>
<td>1.0</td>
</tr>
<tr>
<td>Total fat intake:</td>
<td></td>
<td>138.0</td>
</tr>
</tbody>
</table>

*ID: 3063 (Wednesday) female low-fat consumer, BMI = 24.3, average daily fat intake = 64 kg.

On the basis of the evidence discussed here, fat substitutes can help to prevent passive overconsumption, allowing people to experience the sensory properties of fat without substantial weakening of appetite control. It follows that if fat exerts a disproportionately weak effect on satiety, then removing some of this fat (by substitution) will not decrease unduly the satiating power of the food. More importantly, because of the marked effect of high energy dense (high-fat) foods on satiation (meal termination), fat substitutes should reduce meal size by allowing a feeling of comfortable fullness to be reached after intake of fewer fat calories. Fat-substituted foods may have the capacity to reduce passive overconsumption markedly.

There already is good evidence that substitution of natural fat with olestra (up to 50 g/d) does not generate any strong compensatory appetite response (to make up for the fat energy displaced) when the substitutions are made at single lunch or dinner meals (38). Moreover, when fat substitution occurred gradually across the day in three normal meals or in six snack-size meals, study participants did not experience an increase in hunger, and there was no tendency to “catch-up” the energy on the following day (39). However, it should be noted that a larger compensatory response (equal to 74% of the fat energy substituted) was observed when fat was reduced (by substitution) from 30% to 20% of total energy (40). Longer term exposure to fat-substituted foods also has indicated that reasonable fat reduction can occur on a day-to-day basis without inducing a compensatory increase in appetite (41).

Consequently, on the basis of current knowledge, it can be concluded that foods low in fat (by omission or substitution) have the potential to prevent unnecessary (passive) overconsumption and to prevent people from unwittingly accumulating positive fat balances, thereby arresting the current upward trend in weight gain. Whether unrestricted consumption of such foods actually can induce a negative energy (fat) balance and bring about a weight loss awaits investigation.

A SCENARIO FOR A HUMAN PREDICAMENT

It has been estimated that the average daily fat intake for men in the United Kingdom is 108 g (42). However, experimental studies and data collected from free-living persons indicate that people can consume much more than 130 g of fat in a single meal and nearly 200 g in a full day. It appears to be relatively easy to consume large amounts of fat. Because people do not consciously plan to eat as much fat as possible, we have called this passive overconsumption. The mechanisms that cause this passive overconsumption include the high energy density of fat, the very high palatability of high-fat foods, culturally approved high-fat food habits, aggressive marketing by segments of the food industry, and human preferences for foods with a fatty texture and the flavors associated with fat. These contributory factors overwhelm the first line of inhibitory processes, namely, the preabsorptive fat-induced physiologic satiety signals. The primary effect of high-fat foods on the short-term positive energy balance occurs during consumption while the food is in contact with the orosensory receptors. This means that the processes controlling satiation are too weak or too slow to prevent the intake of a huge amount of energy. The subsequent disproportionately weak effect of fat on postconsumptive satiety works against the development of energy compensation (a reduction of later intake to compensate for the high fat load).

Postabsorptive metabolic adjustments that follow food consumption (fuel utilization) do not appear to provide a strong feedback for the control of food intake. At least these processes do not appear to be strong enough to modulate habitual patterns of food intake held in place by long-term condition-
ing, physiologic rhythms, and environmental contingencies. In addition, the induction of fat oxidation to a level commensurate with fat intake does not appear to occur until after several days of uniform high-fat feeding. Any satiety signal associated with this fat oxidation is too late to prevent the consumption of the (already) eaten fat and does not appear strong enough to disrupt habitual eating patterns.

Consequently, although the physiologic system does contain inhibitory processes that influence the termination of eating episodes and the duration of intervals between episodes, these processes can be overwhelmed by the super potent fat-containing food products that can generate a positive energy balance (particularly when energy expenditure is low). The human psychobiologic predilection to derive pleasure from eating allows the environment to overcome physiology.

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