Fat in food and the obesity epidemic

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Publication Details
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Abstract
Dietary fat is strongly implicated in the development of insulin resistance and obesity, both major public health problems today. While the amount of dietary fat is relevant, the type of fat is important in fuel utilisation, storage and appetite regulatory mechanisms. Human calorimetry research confirms the importance of dietary fat in energy balance, but more work needs to be done to uncover the impact of type of dietary fat in weight control. Population and intervention research confirm the importance of fat in dietary interventions, bearing in mind the contribution of physical activity to energy balance. The food industry has an important role to play in strategic food innovation which addresses this expanding knowledge base.

Keywords
epidemic, food, fat, obesity

Disciplines
Arts and Humanities | Life Sciences | Medicine and Health Sciences | Social and Behavioral Sciences

Publication Details

This journal article is available at Research Online: http://ro.uow.edu.au/hbspapers/1295
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Dietary fat is strongly implicated in the development of insulin resistance and obesity, both major public health problems. While the amount of dietary fat is important in fat utilisation, storage and appetite regulatory mechanisms. Human calorimetry research confirms the importance of dietary fat in energy balance, but more work needs to be done to uncover the impact of type of dietary fat in weight control. Population and intervention research activity confirm the importance of fat in dietary fat intake and in limiting the contribution of physical activity to energy balance. The food industry has an important role to play in strategic food innovation which addresses this expanding knowledge base.

Obesity and dietary fat

Obesity and Type 2 diabetes mellitus (T2DM) are major public health concerns in Australia today. National surveys show that 38% of men and 28% of women are overweight and a further 18% are obese, with the prevalence in children and adolescents steadily increasing (1997). The direct costs and social burden to society have been estimated at $646 million with an additional $272 million in indirect costs (AIHW 1992). An estimated 7% of Australians have T2DM, with an associated healthcare cost likely to exceed $1 billion annually (McCorry 1996). Obese children and adolescents are particularly at risk of developing T2DM as they present with a high prevalence of impaired glucose tolerance (Sinha & others 2002).

Obesity and T2DM are linked under the rubric of ‘metabolic syndrome’ that is used to describe the co-occurrence of multiple risk factors for diabetes and heart disease, such as abdominal blood lipids, glucose intolerance, insulin resistance, high blood pressure and disorders of clotting regulation (Meigs 2000). Obesity carries a paradigmatic risk for T2DM: insulin resistance, hyperinsulinemia and increased circulating free fatty acids (Boromport 1999), but whether obesity causes insulin resistance or vice versa is not known. Dietary fat has been strongly implicated in the development of insulin resistance (Stolten & others 2001), and in obesity (Astrup 1998), but the story is not simple and approaches to the problem need to be carefully thought through. Because fat lies at the heart of the problem, focusing on dietary fat will illuminate preventive and treatment strategies. As suppliers of food, the food industry needs to be involved in this process, and can help by developing beneficial new products, bearing in mind the challenge of accommodating economic imperatives with a focus on health and nutritional improvement. Promotion of reduced energy dense diets (Seidell 1999) and fat modified foods (Zock & Katan 1999) would be most appropriate and made possible through strategic food innovation and nutritionally sound marketing campaigns.

Where is the evidence?
Mechanistic and whole body research

The significance of dietary fat in the energy balance equation can be explained on a number of levels. At the molecular level, dietary fat is less well utilised (oxidised) as fuel than protein and carbohydrate, so when overconsumption occurs fat is preferentially stored and the energy efficiency of this storage is high. In this sense energy efficiency is not fat’s friend (Plant 1995). The problem is that people do not eat only fat; they eat foods and meals which contain different combinations of energy sources (fat, protein and carbohydrate) and of fatty acids (polysaturated, saturated and monounsaturated). Polysaturated fatty acids (PUFA) have been shown to be more readily utilised for energy than longer chain saturated fatty acids (SFA) in feeding studies involving animals (Leyton & others 1987) and humans (Simmons & Schoeller 1988, De Lany & others 2000). It has been proposed that obesity results from a defect in the oxidation of dietary long chain triglycerides, which are readily taken up by adipose tissue (Bannert & others 1998). At the same time research in molecular biology and genetics has shown that, in animal models, high fat (SFA) can promote fat cell proliferation (Okuno & others 1997) and lipogenesis, whereas PUFA promote fat oxidation (Chazbe 2001). Animal model studies examining the expression of appetite control mechanisms have also shown that dietary SFA are obesogenic, whereas dietary PUFA (and especially n-3 PUFA) may even protect against obesity (Wang & others 2002). Thus, while the amount of dietary fat is relevant, it would be foolish to ignore the impact of the type of fat on fuel utilisation, storage, and appetite regulatory mechanisms – and then in the context of whole foods and whole diets.

Mechanistic studies investigate the specific effects of dietary fat on fuel utilisation, body fat distribution, satiety, appetite and energy balance, with special emphasis on the class of fatty acids and their composition. Such studies have provided considerable insights into the mechanisms by which fat is metabolised and stored in the body, but there is still much more to know. Precise data are available from studies using whole-body indirect calorimetry where people spend time in a respiration chamber (a closed room), where by measuring oxygen consumed and carbon dioxide produced, energy expenditure and fat and carbohydrate utilisation are estimated. By manipulating diet, within-person and group comparisons (eg obese vs lean) can be conducted (Murgatroyd 1994). To date, this research has...
demonstrated, for example, that only a proportion of dietary fat is oxidised after a meal and that the amount oxidised is far less than the amount consumed (Thomas & others 1992, Sonko & others 2001). Further, the adaptation to high fat intakes is dependent on the type of fat being consumed. Subjects can adapt to high fat intakes within seven days (Schrauwen & others 1997) and are able to burn increased amounts of fat (Hill & others 2000, Sonko & others 1992), but the latter can adapt better when glycogen stores are depleted through previous exercise (Sonko & others 1992). This indicates the ability to predict fat balance following a shift to higher fat intakes has been linked to the energy cost of concomitant physical activity (Smith & others 2000). Thus, while fat balance lies at the core of energy balance, this relationship appears to be strongly mediated by physical activity and yet go to see whether the type of fat also influences this.

If the fat component of the diet is reduced, the carbohydrate component is increased, and more specifically, the polyunsaturates. Where these fractions have been manipulated, differences in diet-induced thermogenesis and satiety have been demonstrated in controlled studies (Westerterp & others 1999). While this appears to offer support for a lower fat intake, the levels of fat studied have been extreme (10 vs 60% energy), and do not reflect typical eating patterns with the current food supply. In a clinical setting, increasing the protein fraction can reduce hunger (50% energy) does not seem to increase weight loss or reduce the concomitant fall in resting energy expenditure in people consuming 60% energy for 12 weeks (2002). Likewise, while an upper level of fat tolerance seems reasonable, ad libitum consumption of carbohydrate may be impractical due to the methodological constraints in observing the true effects.

Using the precision of continuous indirect calorimetry combined with computer simulations it has been shown that carbohydrate is converted to fat with 50% overfeeding, regardless of the type of carbohydrate (McLoughlin 2005). Thus, from a thermodynamic level, simply reducing fat and not maintaining energy balance with the remaining dietary components is still a challenge.

Clinical and population studies

In an attempt to convert knowledge of fat balance to eating behaviours, many studies have focused on interventions to reduce intakes of foods. Although these studies have been able to show reduced energy intake with low fat diets, like the calorimetry study conducted by Hill & others 2000), some have failed to develop the effect of lower dietary PUFA:SFA ratios. These studies are consistent with an earlier Dutch study that demonstrated a reduced range of dietary fat and SFA with an indirect measure of insulin resistance, but dietary PUFA appeared to be utilised in the body, composition, energy balance and insulin resistance but still very incomplete understanding. When we consider fat as not just energy but a food component, the development of molecules, the notion of "you are what you eat" has particular meaning.

The reasons to she are already in nature, but in controlling so many aspects of our environment, we must now take responsibility for the consequences. Food industry and society are now tied up with economic imperatives. Our current climate comprising an energy dense, abundantly available food supply combined with technology that reduces our physical activity suggests we need to make changes to re-balance the energy equation. On the one side, increasing physical activity is vital. On the other, we need demonstrably to reduce intake of the type of fat, are less energy dense, and at the same time fit within an exciting modern cuisine which blends all benefits. This will require attacking the future. A systematic approach to establishing the evidence of benefits in health outcomes will require strong evidence base, including robust, public health and government funding bodies. For fat, in the first instance this will require making changes in substance to focus on the evidence base and assessments of efficacy under clinical trials. This is the New Nutrition, but it looks like it is here to stay.

References

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Summary

We are clearly moving into a new era in food and nutrition where nutrition problems, which require a sophisticated and integrated understanding of energy balance, are emerging on a large scale. It will be a challenge to see if new knowledge and new technologies can match the pace. Obesity and T2DM are approaches are closely linked to lifestyle, physical activity, the environment, and the food industry. The current challenge is to understand the environment, but still very incomplete understanding. When we consider fat as not just energy but a food component, the development of molecules, the notion of 'you are what you eat' has particular meaning.

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