Abstract. This paper provides a description of dietary fats, including saturated, monounsaturated and polyunsaturated fats, as well as rans fatty acids and dietary cholesterol. The main concerns about dietary fats for human health are discussed, with particular reference to obesity, Coronary Heart Disease and cancer. The paper then looks at red meat as a source of dietary fat, concentrating on Australian beef and the types of fat found in the beef. The impact of marbling on the level of fat found in Australian beef is discussed.

Introduction

Meat has always been central to the Australian diet. From the earliest days of colonisation, meat for consumption was in abundance as a by-product of the highly successful wool trade. The flocks and herds that developed at that time and in the following decades provided vast quantities of relatively cheap meat for the Australian population and still later, when refrigerated ships were available, Australia made significant contributions to the meat supply of populations in other countries.

By the 1890s, medical experts were warning about potential problems with the high meat intakes in the colonies although at that time their concern was largely ignored. Concern centred on the “heat-producing” aspects of meat that were felt to be unsuitable to hot climates; potential problems with contamination and deterioration were identified and concern was expressed that excessive consumption of meat could lead to piles, or eruptions of the skin. Others believed it to weaken the stomach and alimentary functions and thus cause heart affections, general debility and nervous disorders. If these symptoms were indeed evident in the population, or related to the dietary patterns of the time, it is likely that the low intake of vegetables and fruits may have played as important a part as the high meat intake mirroring current day concerns about dietary balance.

This heavy reliance on meat lasted well into the 1900s and began to change in the years between the First and Second World Wars with more marked changes after the Second World War. From the late 1960s, concern started to appear in the scientific literature about the rising incidence of chronic diseases in western societies like Australia and diet was identified as one key influence on this trend. Concern centred on the high overall intake of food relative to changing activity patterns and to the fat content of diets, particularly the saturated fat. As a corollary to this, advice started to appear in professional literature and in the media to reduce dietary fat intake, particularly saturated fat. One of the consumer messages that gained currency at that time was the need to reduce the intake of animal foods such as meats and dairy products. In recent years has been modified to encourage consumption of lean or low fat versions of these foods rather than reduction per se or elimination, however there is much confusion and ambivalence about red meat and its role as a contributor to dietary fat intake and through this, energy intake.

What are dietary fats?

Chemically, most of the fats in foods are triglycerides, made up of a unit of glycerol (glycerine) combined with three fatty acids (which may be the same or different). The differences between one fat and another are largely due to the fatty acids they contain (which together make up 90% of the weight of the molecule).

Fats are the most concentrated form of energy (38 kilojoules or 9 calories per gram). They are the chemical form in which most of the energy reserve of animals and some seeds is stored. Cholesterol, a lipid has important functions in the body. It is part of the cell membrane of all cells, part of the myelin in the brain and nervous system and the starting material for synthesis in the body of bile acids, adrenocortical and sex hormones.

There are various types of fat which can have different effects in the body. The biologically important fats are the:

- Neutral fats - largely storage fats which are triglycerides made up of 3 fatty acids bound to glycerol. There are many fatty acids with different properties but they fall into 3 general classes which differ in their structure by the number of reaction sites (double bonds) present and where these reaction sites occur in the molecule. These classes are saturated fatty acids (no double bonds), monounsaturated...
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- Fatty acids (one double bond) or polyunsaturated fatty acids (more than one double bond).
- Phospholipids, which are structural fats such as those found in membranes and in other cell structures, made up of 2 fatty acids, glycerol, phosphate and choline (for lecithin) or ethanolamine (for cephalin).
- Sterols which include the various steroid hormones as well as cholesterol their precursor.

**Saturated Fats**

In these fats, the majority of the fatty acids, contain no double bond, ie are fully saturated with hydrogen. Saturated fats are usually solid at room temperature. They are predominantly found in milk, cream, butter and cheese and in some meats (most of the land animal fats) and in palm oil and coconut oil. Most predominantly saturated fats contain one or more of the fatty acids palmitic (16:0), myristic (14:0) and lauric (12:0). When they predominate in the dietary fat they tend to raise plasma cholesterol.

**Monounsaturated Fats**

In these, the main fatty acid is oleic acid (18:1), which has one (mono) double (unsaturated) bond. Olive oil, canola and peanut oils are rich in oleic, and it is the most abundant fatty acid type in most muscle meats.

**Polyunsaturated Fats**

In these, the main fatty acid contains two or more double bonds. There effects of these fatty acids differs according to where the double bond occurs in the chain They are liquid at room temperature. The most common polyunsaturated fatty acid is linoleic (18:2); its double bonds are in the omega (w)–6 position. It occurs predominantly in seed oils, eg sunflower, safflower and corn oils but there is a small amount in meats. Smaller amounts of polyunsaturated fatty acids with double bonds in the omega-3 position also occur in the diet. Best known are those found predominantly in fatty fish, their names abbreviated to EPA (20:5) and DHA (22:6). Another omega-3 polyunsaturated fatty acid, ALNA (18:3) occurs in small amounts in leafy vegetables. There is more of this in canola oil and most in flaxseed oil. Omega-3 fats are also found in meat to varying degrees depending on feeding regimes.

**Trans Fatty Acid**

These are a form of unsaturated fatty acid that is straight at a double bond (rather than bent as in the usual cis form); not common in nature but formed during some manufacturing process, eg hydrogenation of edible oils to make hard margarines.

**Dietary Cholesterol**

Cholesterol occurs in all the cell membranes of land animals. Oils and fats from plants never contain cholesterol. Eating cholesterol doesn’t necessarily increase cholesterol in human blood plasma because when it is absorbed the liver tends to reduce its own endogenous cholesterol synthesis (about half the body’s cholesterol is made in the body from acetate).

**What are the main concerns about dietary fats in terms of human health?**

There are three major areas of concern that have been expressed in relation to inappropriate intake of dietary fats:

- obesity (and its correlates such as high blood pressure and Type 2 diabetes) where concern is related to the total fat (and thus potentially excess energy) in the diet not to specific types of fat; fat has a high energy value per unit weight compared to protein and carbohydrate (over twice the energy value);
- heart disease, related in part to total fat because of the link to obesity, but also to individual types of fat consumed; and
- cancer, either related to total fat intake or polyunsaturated fat intake.

**Obesity**

Obesity is increasing in many countries throughout the world including Australia. While there is a substantial genetic contribution to such weight gain, underlying the increase in adiposity is an alteration in the usual energy balance. Individuals may be consuming more than they require and/or be less active.

Dietary fat intake can be major factor in the development of obesity. Fat is the macronutrient with the highest energy value per unit weight, 2.25 times that of carbohydrates and proteins. There is good epidemiological evidence linking higher fat intakes with increased prevalence of obesity. The WHO (World Health Organization 2000), the British Obesity Task Force (Department of Health 1994), the Scottish Intercollegiate Guidelines Network (Scottish Intercollegiate Guidelines Network 1991) and a major academic review (Bray and Popkin 1998), all emphasise the major role of fat consumption in the development of obesity and of cutting fat intake in its dietary management. Fat intake alone, is not the sole cause of obesity - genetic predisposition, total energy intake and reduced physical activity are also important. However, in dietary terms, reduction of fat may help prevent and certainly helps treat obesity and its metabolic consequences.

There is also evidence that a high fat intake, particularly of saturated fats (Borkman et al. 1991), can lead to insulin resistance and increased likelihood of Type 2 diabetes and lipid disorders that can be detrimental to heart health.

The National Health and Medical Research Council has developed a strategic plan for the prevention of overweight and obesity (NHMRC 1997) that recognises the role of a high fat diet in the development of obesity and the need for healthier diets and increased physical activity to help reduce the problem.

None of this should be taken to mean that reducing fat intake is all that is needed to deal with the contemporary epidemic of overweight and obesity. Many Australians have become
obese in the last 20 years while fat intakes do not appear to have increased (either as g/d or as % energy) and may have declined slightly. Physical activity remains one of the keys to control of obesity but limiting fat intake will help in making this an effective strategy to combat obesity.

**Coronary Heart Disease**

Obesity is a risk for coronary heart disease and as such, high total fat intake which might lead to excess energy intake is an indirect risk factor. However, in relation to heart disease, type of fat consumed is probably more important in most cases.

**The evidence against saturated and trans fatty acids**

Much of the early evidence linking saturated fats to increased coronary heart disease risk can from animal studies and human experimental studies looking at risk factors such as raised plasma cholesterol. For human population studies, the prospective cohort study where large numbers of people are assessed at one or more a points in time (for example diet, exercise, family background etc) and followed for many years to look at disease outcome (i.e. actual heart attacks/deaths), is considered the most reliable. The first of these studies to report a positive association of saturated fat intake with subsequent CHD mortality was the classic Seven Countries Study (Keys et al. 1957; Bronte-Stewart 1958; Hegsted et al. 1965; Beynen and Katan 1989; Mensink and Katan 1992; Hergsted et al. 1993; Clarke et al. 1997).

As far as it has been possible to separate the effects of individual fatty acids, it is only lauric (12:0), myristic (14:0) and palmitic (16:0) that have the LDL and total cholesterol-raising effect. Stearic (18:0) does not appear to have this effect (Hegsted et al. 1965; Clarke et al. 1997; McGandy et al. 1970) and lauric appears less active than palmitic and myristic (McGandy et al. 1970).

The cholesterol-raising effect of trans unsaturated fatty acids (Anderson et al. 1961) was rediscovered in the early 1990s (Mensink and Katan 1990) and confirmed in different laboratories (Nestel et al. 1992; Judd et al. 1994). Unlike C12 to 16 saturated fatty acids, trans fatty acids are reported to lower HDL-cholesterol and to increase Lp(a). Trans-fatty acids were reported to be associated with CHD in the Nurses Health Study (Willet et al. 1993), although the reliability of the food composition data was queried (Applewhite 1993). Re-analysis of dietary data and longer follow up of the Nurses Cohort by Willett’s group supported their earlier finding on trans fatty acids (Hu et al. 1997). However, adipose tissue percentages of trans fatty acids have not been associated with CHD cases in Europe (Roberts et al. 1995; Aro et al. 1995).

The UK Committee on the Medical Aspects of Food Policy recommended as early as 1984 that trans-fatty acids should be regarded as equivalent to saturated fatty acids for the purposes of preventing CHD (Department of Health and Social Security 1984). The NHMRC Working Party on the Role of Polyunsaturated Fats in the Australian Diet (NHMRC 1992) came to the same conclusion; so did the Heart Foundation’s 1994 review of dietary fats and blood cholesterol (Sharpe et al. 1994).

The Heart Foundation’s 1999 position statement on dietary

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Table 1. Overweight and Obesity in Australia.

<table>
<thead>
<tr>
<th></th>
<th>Men %</th>
<th>Women %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Overweight</td>
<td>Obese</td>
</tr>
<tr>
<td>NHF 1980 (Hu et al. 1999)</td>
<td>34.1</td>
<td>7.2</td>
</tr>
<tr>
<td>NHF 1983 (Ascherio et al. 1994)</td>
<td>34.2</td>
<td>6.4</td>
</tr>
<tr>
<td>NHF 1989 (Sacks et al. 1994)</td>
<td>38.6</td>
<td>9.3</td>
</tr>
<tr>
<td>NHF 1993 (NHMRC 1997)</td>
<td>43.0</td>
<td>18.3</td>
</tr>
</tbody>
</table>

NHF = National Heart Foundation (surveys in capital cities)

NNS = National Nutrition Survey (randomly sampled the whole nation from voters rolls).
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fats (National Heart Foundation of Australia 1999), based on a thorough review of the literature with ranking of the strength of evidence recommended that saturated fatty acids and trans fatty acids together contribute no more than 8% of total energy intake. The 2000 Dietary Guidelines for Americans recommends a saturated fat intake of 10 percent of kilojoules (US Department of Agriculture 2001). This was recommended by the NHMRC in 1992 (NHMRC 1992) and is a feasible target for the Australian average to reach from the present level of 12.5 percent of energy.

The case for monounsaturated fatty acids (MUFA)

Oleic acid (18:1,cis) predominates among the mono-unsaturated fatty acids. There are however small amounts in the diet of palmitoleic (16:1), 17:1, eicosenoic (20:1) and erucic acid (22:1). When dietary fats are changed from mostly saturated to mostly monounsaturated, total and LDL-cholesterol fall. But polyunsaturated fatty acids are more cholesterol-lowering (Keys et al. 1957; Hegsted et al. 1965; Clarke et al. 1997; McGandy et al. 1970). Some oils rich in oleic acid have more effect on plasma cholesterol than others. This seems to depend on their content of phytosterols (cholesterol-lowering) and squalene (cholesterol-raising) (Truswell and Choudhury 1998). In prospective cohort studies monounsaturated fatty acid intake has not usually been associated with significant increase or decrease in coronary events. In the Seven Country Study “monoenes” were negatively correlated but not significantly so (Keys 1970). In the younger cohort in Framingham, MUFA intake was significantly positively associated with CHD incidence (Posner et al. 1991). In the US Nurses Study a negative association of MUFA with CHD was marginally significant after several adjustments (Willett et al. 1993). There has been no preventive trial with monounsaturated fats.

The FAO/WHO Consultation made no specific recommendations about monounsaturated fat intake (FAO 1994) and the National Heart Foundation of Australia (1999) notes there is little evidence that mono-unsaturated fatty acids have an independent effect on coronary end points. The position statement recommends that “a proportion of dietary saturated fatty acids should be replaced by monounsaturated fatty acids as a strategy for reducing the intake of saturated fatty acids”.

The role of w-6 polyunsaturated fatty acids (those found predominantly in plants)

In recent decades, there have been marked decreases in coronary heart disease in countries like Australia and the US and the changing balance of unsaturated to saturated fatty acids over time in those countries correlates better with the fall in CHD than do changes in total or saturated fat intake.

The National Heart Foundation of Australia concludes that there is good evidence that replacing saturated fatty acids with w-6 polyunsaturated fatty acids reduces the risk of coronary events and deaths. It is recommended that w-6 polyunsaturated fatty acids contribute 8 to 10% of total energy intake (National Heart Foundation of Australia 1999).

In human nutrition, linoleic acid (18:2) is almost synonymous with “w-6 PUFA”. In experimental animals, rats and marmosets, long-term feeding of diets rich in w-6 PUFA also reduces the frequency of dangerous arrhythmias when a coronary artery is ligated (McLenann et al. 1985; McLennan 1993; McLennan et al. 1992). In experimental studies with humans, oils rich in linoleic acid have also consistently lower plasma total and LDL-cholesterol (Keys et al. 1957; Bronte-Stewart 1958; Hegsted et al. 1965; Beynen and Katan 1989; Mensink and Katan 1992; Hergsted et al. 1993; Clarke et al. 1997), even when the oil increases the overall fat intake (Bronte-Stewart et al. 1956; Rassias 1991).

Three prospective cohort studies in humans have estimated linoleic intake from blood samples. In all three studies there was a negative association with subsequent coronary heart disease (Miettinen et al. 1982; Kingsbury et al. 1969; Simon et al. 1995). Another nine prospective cohort study publications estimated intake of fatty acid types and followed subjects to see who developed CHD. In 3 of these, significant inverse association of PUFA or 18:2 with CHD was found in the US Nurses Study (Hu et al. 1997; Hu et al 1999) and the Western Electric Study (Chicago) (Shekelle et al. 1981). In the other 6 studies no significant association was found. In some of these the dietary method may not have had enough detail of fat composition (eg 24 hour recall) or the range of PUFA intakes was too small.

Eight intervention trials (Rose et al. 1965; Medical Research Council 1968; Dayton et al. 1969; Leren 1970; Turpeinen et al. 1979; Moiittinen et al. 1983; Burr et al. 1989; Franz et al. 1989) prescribed substitution of polyunsaturated fats (ie omega-6) for saturated fat in the experimental group (Table 3). Diet was the only lifestyle factor changed. These eight trials involved a total of 17,529 subjects in four countries, half in the increased polyunsaturated to saturated group and half in the usual diet group. Those on the higher polyunsaturated diets had only 81% of the deaths of the control group.

The case for omega-3 (w-3) polyunsaturated fatty acids (n-3 PUFA)

Unlike omega-6 PUFA with only one fatty acid usually considered, there are three important fatty acids in the omega-3 series:

- α-linolenic (ALNA) (18:3) occurs in leafy plants, canola, flaxseed oils
- eicosapentaenoic (EPA) (20:5) richest source is oily fish
- docosahexaenoic (DHA) (22:6) richest source is oily fish

All three occur in low concentrations in human and other animal tissues and in human milk. EPA is the precursor of the 3 series of prostaglandins and the 5 series of leukotrienes.
DHA is found in very high concentration in the photoreceptors of the retina and the membranes of the brain. Amongst other benefits, omega-3 fats have been associated with reduced coronary heart disease risk.

The FAO/WHO Consultation on Fats and Oils (FAO 1994) recommended that the ratio of linoleic to α-linolenic acid in the diet should be between 5:1 and 10:1. Individuals with a ratio in excess of 10:1 should be encouraged to consume more w-3 rich foods such as green leafy vegetables, legumes, fish and other seafood. Another expert workshop in the Netherlands (de Deckere et al. 1998) concluded that there should be separate recommendations for plant (α-linolenic 18:3) and marine (EPA 20:5; DHA 22:6) w-3 PUFAs and that the w-3/w-6 ratio will not be helpful.

The National Heart Foundation of Australia (1999) recommends at least two fish meals per week (preferably oily fish); consumption of both plant and marine w-3 PUFAs since it is possible that they protect against CHD by different mechanisms with plant w-3 PUFA intakes at least 2 g/day.

**The role of dietary cholesterol**

The cholesterol-elevating effect of dietary cholesterol is less consistent than that of saturated fats (Beheyen and Katan 1989). Dietary cholesterol only occurs in animal fats, which are also the major source of saturated fatty acids in the diet (Baghurst et al. 2000). The position of the NHMRC on dietary cholesterol up to now has been that at the public health level, advice to reduce saturated fat will bring with it lower cholesterol intakes.

The National Heart Foundation (1999) recommends that individuals with plasma cholesterol greater than 5 mmol/L or with other risk factors should restrict their intake of cholesterol-rich foods. This is clinical, rather than public health advice.

**Dietary Fats and Cancer**

Higher intakes of dietary fat have been linked in the past to an increased risk number of cancers including hormone-related cancers and cancers of the gut, notably the colon. However, evidence from animal studies and early attempts to study this in humans do not appear to be holding up to scrutiny with the availability of more sophisticated human population studies.

In relation to breast cancer, early animal experiments suggested a role for fat and, in particular, for polyunsaturated fats in promoting breast cancer. In experiments with rats given chemical carcinogens, more mammary tumour development was found with diets containing moderate amounts of w-6 PUFA than with saturated fats. A systematic review of all the human case-control and prospective cohort studies reporting linoleic acid or PUFA and cancer incidence (Zock and Katan 1998) in humans showed that whilst the case-control studies showed, if anything, a somewhat reduced risk with PUFA, better quality prospective studies showed no link between PUFA and breast cancer risk. In the conclusions of a study looking at the vast body of evidence from human trials of total fat intake and breast cancer, the authors also concluded that it was unlikely that a reduction in total fat consumption by middle-aged and older women would substantially reduce their risk of breast cancer.

A similar trend in the evidence appears to hold for colon cancer. Although some early human case-control studies claimed a link between total fat and colon cancer, a recent review showed no association between total fat and colon cancer in a total of 8 reported prospective human studies (HMSO 1998).

Thus, it would seem now that there is little or no increased risk from cancers in humans from total or type of fat intake.

**Summary of the effects of fats on health**

Total fat is providing just over one-third of dietary energy in Australia. It appears to have declined a little but it is still relatively high from a world perspective. For anyone overweight a reduction of total fat to 20 to 25% energy should be a major part of dietary management, together with more physical exercise. This is a public health matter because about half the Australian adult population were overweight in the 1995 National Nutrition Survey (Australian Bureau of Statistics 1997). The biological effects and health risks of dietary fats and oils are determined in large part by their predominant fatty acids.

Saturated fatty acids raise plasma LDL-cholesterol, a major risk factor for coronary heart disease (CHD). In three large prospective epidemiological studies, saturated fatty acid intake was directly associated with subsequent CHD. Trans unsaturated fatty acids appear to behave similarly, though their consumption is now small in Australia. Saturated plus trans fatty acid intakes averaged over 12.5% energy in 1995. A population average of 10% energy is recommended.

Intake of omega-6 polyunsaturated fatty acids (essentially linoleic) should be in the range 6 to 8% of energy because there is strong evidence that they protect against CHD by lowering plasma LDL-cholesterol and probably by reducing the risk of dangerous cardiac arrhythmias.

Omega-3 polyunsaturated fatty acids occur predominantly in fish and a few vegetable oils ut are present in other foods including red meats. Present intake of these fatty acids is low (approx 200 mg). It would appear desirable to double this intake as a measure aimed to reduce the risk of CHD. Because of the relative amounts of red meat compared to fatty fish consumed in Australia, there is evidence that red meats are providing up to 30% of the intake of omega-3 fats in Australia at the current time.

Monounsaturated fatty acids do not raise plasma cholesterol and do not have the action of polyunsaturated fatty acids on arrhythmia. Present intake levels appear to be satisfactory except in individuals who need to reduce total fat as part of body weight management.

Dietary cholesterol intake will decline if people eat smaller amounts of saturated fat, since these two lipid classes usually...
occur in the same foods.

**Red Meat as a source of dietary fat**

**How much fat does red meat contribute to the Australian diet?**

In Australia, red meat consumption has fallen markedly in the past few decades in part because of consumer concerns about dietary fat, particularly saturated fats. In contrast, in some countries, particularly Asia, red meat consumption is increasing from a low base and a high degree of marbling is preferred because of its cooking qualities and relative tenderness. In recent years concerns over increasing rates of heart disease and obesity are beginning to have an effect in raising concerns about dietary change in those countries and it will be interesting to see if this has any effect on marbling preferences or amounts of red meat consumed in Asian markets in the coming decades.

In Australia, today’s consumers also commonly remove much of the visible fat on meats before cooking. In the last Australian National Nutrition Survey (1995/6), around two-thirds of red meat cuts were fat-trimmed before consumption (57% for men aged 25-44 years and 73% for women in the same age group, with further proportions – about 17% - (not specified whether they trimmed). This same survey showed that red meat at that time contributed only 9% of total dietary fat for men and 7% for women together with 10% of the saturated fat for men and 8% for women. Indeed, red meats contribute more unsaturated fats (14% men, 10% women) than saturated fats. However, red meat also contributed 19% and 15% of the dietary cholesterol for males and females respectively.

**Where is the fat in meat?**

Fat in meat can be present in a number of forms as subcutaneous (selvedge) fat, as intermuscular fat (seam fat); as intramuscular fat (the fat within the muscle fibres or cells which is mostly structural phospholipids but with some triglycerides) and finally as marbling fat (triglyceride or storage fat) found in the areas between muscle fibres or cells. In the literature the term “intramuscular” is sometimes used to encompass both cellular lipids as well as marbling fat or used synonymously with “marbling fat”. This has led to some confusion in the literature. In cases where the meat is very lean, the cellular phospholipids can account for up to a third of the fat content but in highly marbled meats the triglyceride dominates. This has led to some confusion in interpretation of fatty acid data.

**How fatty is Australian beef?**

Compared to most of red meat from countries like the US and to meats available in Australia for most of the last century, most selvedge or seam-trimmed meats available at this point in time for consumption in Australia are quite lean. In Australia, the dominant source of fat in meat, as purchased, is the subcutaneous and/or seam fat which can makes a big difference to the total fat content of some cuts of red meat. This is not always the case however in countries where highly marbled beef is preferred where the marbling fat makes a major contribution to total fat.

**What type of fats are found in meats like beef?**

Contrary to common belief, the amount of saturated fat in beef is actually less than the total amount of unsaturated fat on a per edible portion basis (60). Most of the saturated fat is palmitic acid (C16:0) and stearic acid (C18:0) – about 40% of the saturated fat in beef is stearic acid, a saturated fatty acid that does not raise plasma cholesterol. The average fatty acid profile of raw lean meats as available in Australia is shown below in Table 2 but the fatty acid profile can vary quite markedly across individual animals, across breeds, across feeding regimes and to a limited extent across type of fat i.e. whether we are considering that from within muscle fibres, between muscle fibres (marbling), intermuscular fat or seam fat or subcutaneous fat. However, these cross-depot variations are quite small within a given animal.

**Table 2. Fatty Acid Content of Different Raw Lean Meats (g/100g edible portion).**

<table>
<thead>
<tr>
<th>Fatty Acids</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>C14:0</td>
<td>0.067</td>
</tr>
<tr>
<td>C16:0</td>
<td>0.334</td>
</tr>
<tr>
<td>C18:1</td>
<td>0.337</td>
</tr>
<tr>
<td>C18:2W6</td>
<td>0.089</td>
</tr>
<tr>
<td>C18:3W5</td>
<td>0.029</td>
</tr>
<tr>
<td>C20:1</td>
<td>0.013</td>
</tr>
<tr>
<td>C20:4W6</td>
<td>0.033</td>
</tr>
<tr>
<td>C20:5W6</td>
<td>0.019</td>
</tr>
<tr>
<td>C22:5W5</td>
<td>0.022</td>
</tr>
<tr>
<td>C22:6W6</td>
<td>0.003</td>
</tr>
<tr>
<td>n-6 polyunsaturated</td>
<td>0.873</td>
</tr>
<tr>
<td>n-3 polyunsaturated</td>
<td>0.321</td>
</tr>
<tr>
<td>Total W6</td>
<td>0.103</td>
</tr>
<tr>
<td>Total W9</td>
<td>0.073</td>
</tr>
<tr>
<td>Total W12</td>
<td>0.135</td>
</tr>
<tr>
<td>W8:W6</td>
<td>0.33</td>
</tr>
</tbody>
</table>

There has been a suggestion in the literature that marbling fat may be compositionally different to other meat fats and therefore be more “healthy”. This originated because of confusion over the term “intramuscular fat” which is often is used synonymously with marbling fat. True intramuscular fat is highly unsaturated, with significant percentages of polyunsaturates, whereas marbling fat is an adipose tissue with a composition similar to those of other sites such as seam fat. In other cases, the term “intramuscular” is used to include both marbling fat and the true intramuscular (within muscle-cell) fat and this again can lead to some confusion about
purported benefits and relative “healthiness” of the various fat components. There also seems to be some confusion over the effects of grain-feeding versus pasture feeding on fatty acid profiles and the effects of increased marbling per se. Although grain feeding or lot feeding can lead to increased marbling, this is not always the case and the two issues need to be considered separately.

How does marbling fat fit in to the picture?

What evidence is there for this assertion that marbling meat may have specific health attributes?

One aspect to consider is the cholesterol content. Sweeten et al. (1990) in the US showed that the contribution of marbling fat serve of raw lean beef was some 2mg/100g of meat. This constituted a very small proportion of the total cholesterol in the average US meat serve (60-70mg) so in terms of cholesterol, marbling fat will have little effect on the overall contribution of red meat to cholesterol intake. The story is not as positive, however, when considering the issue of contribution to total fat or fatty acid type.

Firstly, no matter what the composition of the fatty acids in marbled versus other meat fats, all fats provide a concentrated energy source and marbled meat is substantially more energy dense than lean meat. The greater the marbling the greater the disparity. In this context, consumption of marbled meats if eaten in significant amounts, could be detrimental to health depending on the dietary context.

Almost all beef available on the Australian market is pasture-fed (grass-fed) although often “finished” in feedlots for up to 70 days. The predominant type of feeding can affect both the content and composition of the resulting meat. Because of the type of and more extensive practice of grain-feeding cattle in the US, the average US beef cut is more marbled, contains more fat and has less omega-3 fatty acids than the average Australian beef cut (Meat and Livestock Australia 2001). In the 1980s, the minimum total intramuscular/marbling lipid content in US beef was stated to be 9% (Greenburg 1986). The current US food data base shows figures ranging from 3.7% fat upwards with most of the leaner, trimmed cuts being at least 6-7% fat. Sweeten et al. from the US (1990) claimed that about 6-7% intramuscular fat (true IM plus marbling) is about the upper limit for compliance with current dietary guidelines. This level of fat conformed at the time of the study, with the US modest/moderate marbling or average to high US choice grade. Savell and Cross (1988) in the US also developed a “window of acceptability” for fat content of meat based on health and palatability considerations. Their window ranged from 3 - 7.3% fat equivalent to beef longissimus dorsi from carcasses that graded in the lower range of US Select at that time (3 - 4.7% fat) to carcasses that graded in the higher range of US Choice (4.28% to 8.7% fat).

A study by Sinclair and O’Dea (1987) in the late 1980’s in Australia gave figures ranging from 1.6 - 6.5 % for a variety of samples of Australian pasture-fed beef (Gold-branded for domestic markets) and 1.5 - 5.4 % for various cuts of Australian grain-fed beef (from a Victorian feed-lot company). Both sets of Australian cattle used in the latter study were less than 2 yrs of age but no details were given about length of grain-feeding. Thus, in the Australian context, for animals under 2 years, there was little difference between these pasture and grain-fed animals in total intramuscular fat. However, for the grain-fed Australian beef there was a substantial increase in the ratio of 18:2n-6 to 18:3n-3 compared with the grass fed beef probably due to differences in fatty acid composition of feed. The linoleic acid to linolenic acid ratio in grain-fed animals was 15 compared to just under 4 for pasture fed samples.

This study, however, also showed that there was no potential “health” benefit to be gained by an increased ratio of marbling fat in terms of the fatty acid ratios or type of fat produced. Across the cuts of meats of varying intramuscular fat, the level of phospholipids (structural cell lipids) was relatively constant and independent of fat content but the level of triglycerides (in the marbling fat) was proportional to total fat. For meats with total intramuscular fat in the range of 2%, the phospholipid content contributed just over one third of the fatty acids whereas at 5% intramuscular fat, they contributed less than 15%. The triglycerides were rich in saturated and monounsaturated fats but the phospholipids were rich in polyunsaturates. The meat samples with lower total fat

![Figure 1. Distribution of fatty acids in subcutaneous and intramuscular fat from US beef (adapted from Sinclair and O’Dea 1987).](image-url)
(less marbling) also had lower proportions of saturated and monounsaturated fats and higher relative polyunsaturates. The P:S ratio showed an inverse relationship with intramuscular fat levels or marbling. In other words, the greater the marbling the higher not only the total fat but the higher the proportion of saturates and monounsaturates and the lower the proportion of polyunsaturates.

Generally, the fatty acid composition of marbling fat is similar to subcutaneous fat from the same animal. Tune (2001), in an accompanying seminar paper reports a slightly higher saturated fatty acid level, notably of stearic acid and a lower monounsaturated level with polyunsaturates being similar and Sweeten et al. (1990) in the US found little difference in % composition of subcutaneous and marbling fat in terms of P:M:S ratios although marbling fat in this case appeared to have slightly less PUFA (Fig. 1). As stearic acid is not cholesterogenic, it is unlikely that the level of changeover in stearic and monounsaturates would be of major concern but it this differential between marbling and subcutaneous fat. The predominant fatty acids (Sweeten et al. 1990) in marbling fat appear to be oleic acid (about 38%), followed by palmitic (about 25%) and the stearic (about 14%) but these can vary depending on the overall level of saturation in the individual animal.

It may be possible to improve the “healthiness” of the fatty acid profile of beef manipulate fatty acid composition by selective breeding, by genetic engineering and/or by altering feeding regimes, but there appears to be no unique role for marbling fat per se and, with increasing concern about obesity and its sequelae, increases in total fat (and this energy density) in meats brought about by increased marbling may offset gains obtained by improvement in fatty acid profile.

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