Fats And Oils: The Facts

A review of the scientific literature regarding fats and oils and their importance in food and health.

MJ James, LG Cleland and Meadow Lea Foods Ltd.
Preface

Fats and Oils: The Facts is intended as a readily accessible resource to assist Dietitians, Nutritionists, Medical Practitioners, Students and all people interested in the relationships between diet, health and disease. This web version replaces the popular printed edition produced by the Meadow Lea Foods Advisory Centre in 1995. It addresses the place of fats and oils in food, both naturally and in manufacture, as well as the roles of dietary monounsaturates, omega-6 polyunsaturates, and omega-3 polyunsaturates in health and disease. It is not intended to provide exhaustive detail but it will provide the scope for further enquiry by readers who want greater detail in certain areas. Such areas include:

- Heart Disease
- Cancer
- Diabetes
- Obesity
- Infant Nutrition
- Inflammation
- Arthritis

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Since 1994, Meadow Lea Foods has supported independent, investigator-initiated research at the Royal Adelaide Hospital, Flinders Medical Centre Adelaide, Flinders University, Women’s and Children’s Hospital, CSIRO Human Sciences and Nutrition, Royal Melbourne Institute of Technology, University of Wollongong, University of Newcastle, Baker Medical Research Institute Victoria, Deakin University, Sydney University, University of Western Australia, Curtin University of Technology and the Royal Prince Alfred Hospital Sydney.

Meadow Lea Foods, the manufacturer of Gold’n Canola, is committed to the research and scientific understanding of healthy fats and to the promotion of their role in food and health. Gold’n Canola is pleased to support this valuable resource by including it on this website.

Meet the authors

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Fats and Oils: The Facts

By MJ James, LG Cleland, and Meadow Lea Foods Ltd

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Chapter 1. Introduction to Fats: their similarities and their differences

Where is the Fat?

Fat may:
- occur naturally (in red meat, white meat, fish, dairy foods, eggs, nuts, olives, seeds)
- be added as an ingredient in manufacture (in biscuits, cakes, ice cream)
- be absorbed during cooking (in frying).

Almost all foods contain some type of fat because all cells in all tissues, both plant and animal, contain some fat. In most cases, the fat is present only as a structural part of cell membranes and therefore, most foods contain very little fat (e.g. trimmed meat, most vegetables and fruits). The fat content of food is increased greatly when it contains some adipose tissue which is the fat storage tissue in the body (e.g. untrimmed meat) or when it is a fat storage vessel itself (e.g. seeds, nuts, eggs).

Mixtures of Fats

Tallow, butter, olive oil, canola oil, sunflower oil are each mixtures of fats.

Food ingredients such as tallow, butter, olive oil, canola oil, and sunflower oil are commonly referred to as fats. In fact, each of them is a mixture of individual fats which can differ in their chemistry. These chemical differences can determine the type of applications suited to the food ingredient and can determine the impact on human biology and health of the fat once it is eaten.

The chemistry of a fat or a mixture of fats is critically important, both for commercial and domestic food uses and for health considerations.

Chemical Similarities Amongst Fats

Most dietary fat is in the form of triglycerides. A triglyceride has a glycerol ‘backbone’ to which is attached three fatty acids. While this is the basic structure of all triglycerides, they can vary considerably in the type of fatty acids which are attached.
Chemical Variety Amongst Fats

Fatty acids can be saturates, monounsaturates, or polyunsaturates, depending on the number of ‘double-bonds’ in the molecule. Polyunsaturates are further divided into omega-6 polyunsaturates or omega-3 polyunsaturates and this terminology refers to the position of the double-bonds in the molecule. Omega-6 fatty acids have the first double bond after the sixth carbon (when counting from the methyl \([\text{CH}_3]\) end). Similarly, omega-3 fatty acids have the first double bond after the third carbon.

Figure 1.1 shows a schematic representation of examples of saturated, monounsaturated and polyunsaturated (omega-6 and omega-3) fatty acids.

Table 1.1 shows the common name and abbreviated form of the most common fatty acids in foods. Following this, Table 1.2 is a bar chart showing the fatty acid composition of some common oils and fats and Table 1.3 compares the amount of total fat, saturates, monounsaturates and polyunsaturates in a number of everyday foods. Table 1.4 further divides the polyunsaturates into the omega-6 and omega-3 content in a variety of foods that are primary dietary sources of these nutrients.
Figure 1.1  Schematic diagram of fatty acid structure

Stearic acid. A typical saturated fatty acid.

Oleic acid. A typical monounsaturated (omega-9) fatty acid.

Linoleic acid. An omega-6 polyunsaturated fatty acid.

Linolenic acid. An omega-3 polyunsaturated fatty acid.

Chemistry Determines Function

In the following chapters it will be made clear that the double-bond chemistry of fatty acids is a powerful and inescapable determinant of their function in food and their function in the body.
Table 1.1 The Predominant Fatty Acids Found In Edible Fats and Oils

<table>
<thead>
<tr>
<th>General class</th>
<th>Fatty acid common name</th>
<th>Chain length</th>
<th>Number of double bonds</th>
<th>Omega name</th>
<th>Abbreviated name</th>
<th>Found in</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturates</td>
<td>Butyric</td>
<td>4</td>
<td>0</td>
<td>na</td>
<td>C4:0</td>
<td>Butter/milk fat</td>
</tr>
<tr>
<td></td>
<td>Caproic</td>
<td>6</td>
<td>0</td>
<td>na</td>
<td>C6:0</td>
<td>Butter/milk fat</td>
</tr>
<tr>
<td></td>
<td>Caprylic</td>
<td>8</td>
<td>0</td>
<td>na</td>
<td>C8:0</td>
<td>Butter/milk fat</td>
</tr>
<tr>
<td></td>
<td>Capric</td>
<td>10</td>
<td>0</td>
<td>na</td>
<td>C10:0</td>
<td>Butter/milk fat</td>
</tr>
<tr>
<td></td>
<td>Lauric</td>
<td>12</td>
<td>0</td>
<td>na</td>
<td>C12:0</td>
<td>Butter/milk fat, coconut oil, palm kernel oil</td>
</tr>
<tr>
<td></td>
<td>Myristic</td>
<td>14</td>
<td>0</td>
<td>na</td>
<td>C14:0</td>
<td>Butter/milk fat, coconut oil, palm kernel oil</td>
</tr>
<tr>
<td></td>
<td>Palmitic</td>
<td>16</td>
<td>0</td>
<td>na</td>
<td>C16:0</td>
<td>All fats and oils</td>
</tr>
<tr>
<td></td>
<td>Stearic</td>
<td>18</td>
<td>0</td>
<td>na</td>
<td>C18:0</td>
<td>All fats and oils</td>
</tr>
<tr>
<td>Monounsaturates</td>
<td>Oleic</td>
<td>18</td>
<td>1</td>
<td>Omega-9</td>
<td>C18:1 Omega-9</td>
<td>All fats and oils</td>
</tr>
<tr>
<td>Polyunsaturates</td>
<td>Linoleic (LA)</td>
<td>18</td>
<td>2</td>
<td>Omega-6</td>
<td>C18:2 Omega-6</td>
<td>Most vegetable oils</td>
</tr>
<tr>
<td></td>
<td>Alpha-Linolenic (ALA)</td>
<td>18</td>
<td>3</td>
<td>Omega-3</td>
<td>C18:3 Omega-3</td>
<td>Canola oil, soyabean oil</td>
</tr>
<tr>
<td></td>
<td>Gamma-Linolenic</td>
<td>18</td>
<td>3</td>
<td>Omega-6</td>
<td>C18:3 Omega-6</td>
<td>Blackcurrant oil, evening primrose oil</td>
</tr>
<tr>
<td></td>
<td>Eicosapentaenoic (EPA)</td>
<td>20</td>
<td>5</td>
<td>Omega-3</td>
<td>C20:5 Omega-3</td>
<td>Fish oils</td>
</tr>
<tr>
<td></td>
<td>Docosahexaenoic (DHA)</td>
<td>22</td>
<td>6</td>
<td>Omega-3</td>
<td>C22:6 Omega-3</td>
<td>Fish oils</td>
</tr>
</tbody>
</table>
Table 1.2 The fatty acid composition of some common oils and fats

<table>
<thead>
<tr>
<th>Oil</th>
<th>Saturated fat</th>
<th>Polyunsaturates: Linoleic acid</th>
<th>Polyunsaturates: Alpha-linolenic acid</th>
<th>Monounsaturates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunola™ Oil</td>
<td>10</td>
<td>8</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td>Canola Oil</td>
<td>8</td>
<td>20</td>
<td>10</td>
<td>62</td>
</tr>
<tr>
<td>Safflower Oil</td>
<td>9</td>
<td>75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sunflower Oil</td>
<td>11</td>
<td>60</td>
<td></td>
<td>Trace 29</td>
</tr>
<tr>
<td>Olive Oil</td>
<td>14</td>
<td>10</td>
<td>76</td>
<td></td>
</tr>
<tr>
<td>Corn Oil</td>
<td>14</td>
<td>52</td>
<td></td>
<td>2 32</td>
</tr>
<tr>
<td>Soybean Oil</td>
<td>15</td>
<td>54</td>
<td></td>
<td>8 23</td>
</tr>
<tr>
<td>Peanut Oil</td>
<td>18</td>
<td>32</td>
<td></td>
<td>2 48</td>
</tr>
<tr>
<td>Cottonseed Oil</td>
<td>26</td>
<td>32</td>
<td>56</td>
<td></td>
</tr>
<tr>
<td>Palm Oil</td>
<td>51</td>
<td>10</td>
<td></td>
<td>Trace 39</td>
</tr>
<tr>
<td>Coconut Oil</td>
<td>91</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tallow</td>
<td>50</td>
<td>2</td>
<td>1</td>
<td>47*</td>
</tr>
<tr>
<td>Butterfat</td>
<td>64</td>
<td></td>
<td></td>
<td>2 1 33*</td>
</tr>
</tbody>
</table>

* Note: Tallow and butterfat contain approximately 5% trans fat which is declared as a monounsaturate in this table.

Figures are percentages.
### Table 1.3 The fat content of some everyday foods

<table>
<thead>
<tr>
<th>FOOD</th>
<th>SERVING SIZE (grams)</th>
<th>TYPE OF FAT PER SERVE (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Total</td>
</tr>
<tr>
<td><strong>Breads and Cereals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cornflakes</td>
<td>45</td>
<td>0.5</td>
</tr>
<tr>
<td>Bread</td>
<td>30</td>
<td>0.8</td>
</tr>
<tr>
<td>White rice</td>
<td>100</td>
<td>0.2</td>
</tr>
<tr>
<td>Pasta</td>
<td>100</td>
<td>0.3</td>
</tr>
<tr>
<td><strong>Fruit and Vegetables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avocado</td>
<td>75</td>
<td>16.9</td>
</tr>
<tr>
<td>Olives</td>
<td>20</td>
<td>0.7</td>
</tr>
<tr>
<td>Other fruits and vegetables</td>
<td>100</td>
<td>0.1</td>
</tr>
<tr>
<td><strong>Meat</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rump steak (grilled, trimmed)</td>
<td>100</td>
<td>9.5</td>
</tr>
<tr>
<td>Fish (bream, steamed)</td>
<td>100</td>
<td>5.4</td>
</tr>
<tr>
<td>Chicken (breast, baked, lean, skin off)</td>
<td>100</td>
<td>4.8</td>
</tr>
<tr>
<td>Poached egg</td>
<td>50</td>
<td>5.6</td>
</tr>
<tr>
<td><strong>Dairy Products</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk</td>
<td>200</td>
<td>7.6</td>
</tr>
<tr>
<td>Reduced fat milk</td>
<td>200</td>
<td>3.6</td>
</tr>
<tr>
<td>Yoghurt</td>
<td>200</td>
<td>6.8</td>
</tr>
<tr>
<td>Cheddar cheese</td>
<td>20</td>
<td>6.8</td>
</tr>
<tr>
<td><strong>Fats and Oils, Spreads</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polyunsaturated spread (eg. Meadow Lea)</td>
<td>5</td>
<td>3.75</td>
</tr>
<tr>
<td>Monounsaturated spread (eg. Gold’n Canola)</td>
<td>5</td>
<td>3.5</td>
</tr>
<tr>
<td>Spread containing plant sterols for lowering cholesterol absorption. (eg. Logicol™)*</td>
<td>5</td>
<td>3.35</td>
</tr>
<tr>
<td><strong>Butter</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canola oil</td>
<td>15</td>
<td>15.0</td>
</tr>
<tr>
<td>Olive oil</td>
<td>15</td>
<td>15.0</td>
</tr>
<tr>
<td>Sunola® oil</td>
<td>15</td>
<td>15.0</td>
</tr>
<tr>
<td>Sunflower oil</td>
<td>15</td>
<td>15.0</td>
</tr>
</tbody>
</table>

**Source:** Nutritional Values of Australian Foods, English & Lewis 1991, and Meadow Lea Foods.

*Logicol is a registered trademark of Meadow Lea Foods Ltd. * Product contains 0.4g sterols per 5g serve. ** Sunola is the registered name for Meadow Lea Foods’ high oleic acid sunflower oil.
### Table 1.4: Common dietary sources of Omega-6 and Omega-3 Fatty Acids

<table>
<thead>
<tr>
<th>FOOD</th>
<th>TOTAL FAT (g/100g)</th>
<th>OMEGA-6 (mg/100g)*</th>
<th>OMEGA-3 (mg/100g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>18:3 (ALA)**</td>
<td>20:5 (EPA), 22:5 (DPA), 22:6 (DHA)**</td>
</tr>
<tr>
<td><strong>Nuts and Seeds</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Almond (blanched)</td>
<td>55.8</td>
<td>13500</td>
<td>0</td>
</tr>
<tr>
<td>Hazelnut</td>
<td>61.4</td>
<td>7000</td>
<td>100</td>
</tr>
<tr>
<td>Peanut (roasted, skin, salted)</td>
<td>51.7</td>
<td>16300</td>
<td>0</td>
</tr>
<tr>
<td>Pecan</td>
<td>71.9</td>
<td>24200</td>
<td>600</td>
</tr>
<tr>
<td>Pine nut</td>
<td>70.9</td>
<td>39800</td>
<td>0</td>
</tr>
<tr>
<td>Sesame seed</td>
<td>55.6</td>
<td>24400</td>
<td>0</td>
</tr>
<tr>
<td>Walnut</td>
<td>69.2</td>
<td>43200</td>
<td>6300</td>
</tr>
<tr>
<td><strong>Spreads and oils</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polyunsaturated spread</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(eg. Meadow Lea)</td>
<td>75</td>
<td>30000</td>
<td>2000</td>
</tr>
<tr>
<td>Canola spread</td>
<td>70</td>
<td>11000</td>
<td>5800</td>
</tr>
<tr>
<td>(eg. Gold’n Canola)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Olive oil spread</td>
<td>75</td>
<td>17500</td>
<td>1900</td>
</tr>
<tr>
<td>(eg. Olive Grove)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Butter (regular)</td>
<td>82</td>
<td>1400</td>
<td>700</td>
</tr>
<tr>
<td>Canola oil</td>
<td>100</td>
<td>20000</td>
<td>10000</td>
</tr>
<tr>
<td>Flaxseed/linseed oil</td>
<td>100</td>
<td>16000</td>
<td>57000</td>
</tr>
<tr>
<td>Soybean oil</td>
<td>100</td>
<td>54000</td>
<td>8000</td>
</tr>
<tr>
<td>Sunflower oil</td>
<td>100</td>
<td>60000</td>
<td>tr</td>
</tr>
<tr>
<td>Peanut oil</td>
<td>100</td>
<td>32000</td>
<td>2000</td>
</tr>
<tr>
<td><strong>Cheese, Eggs, Meat</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cheddar (reduced fat)</td>
<td>23.8</td>
<td>400</td>
<td>200</td>
</tr>
<tr>
<td>Cream cheese</td>
<td>33.1</td>
<td>600</td>
<td>300</td>
</tr>
<tr>
<td>Regular egg (chicken)</td>
<td>10.1</td>
<td>900</td>
<td>0</td>
</tr>
<tr>
<td>Omega-3 enriched egg</td>
<td>11.8</td>
<td>793</td>
<td>297</td>
</tr>
<tr>
<td>Beef (rump steak, lean)</td>
<td>2.7</td>
<td>140</td>
<td>29</td>
</tr>
<tr>
<td>Chicken breast (no skin)</td>
<td>1.3</td>
<td>180</td>
<td>7</td>
</tr>
<tr>
<td><strong>Fresh Fish</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barramundi</td>
<td>2.1</td>
<td>82</td>
<td>35</td>
</tr>
<tr>
<td>Bream</td>
<td>4</td>
<td>75</td>
<td>26</td>
</tr>
<tr>
<td>Cod, Antarctic</td>
<td>4.1</td>
<td>46</td>
<td>0</td>
</tr>
<tr>
<td>Flathead</td>
<td>1.6</td>
<td>22</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 1.4 continues on the following page.
### Table 1.4 continued…

<table>
<thead>
<tr>
<th>FOOD</th>
<th>TOTAL FAT (g/100g)</th>
<th>OMEGA-6 (mg/100g)*</th>
<th>OMEGA-3 (mg/100g)</th>
<th>TOTAL n-3 #</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>18:3 (ALA)**</td>
<td>20:5 (EPA), 22:5 (DPA), 22:6 (DHA)***</td>
<td></td>
</tr>
<tr>
<td>Gemfish</td>
<td>6.4</td>
<td>248</td>
<td>103</td>
<td>721</td>
</tr>
<tr>
<td>Mackerel, blue, with skin</td>
<td>3.3</td>
<td>204</td>
<td>34</td>
<td>1108</td>
</tr>
<tr>
<td>Mullet</td>
<td>3.0</td>
<td>359</td>
<td>14</td>
<td>595</td>
</tr>
<tr>
<td>Oyster, Sydney rock</td>
<td>4.0</td>
<td>184</td>
<td>109</td>
<td>1024</td>
</tr>
<tr>
<td>Perch, golden</td>
<td>2.3</td>
<td>265</td>
<td>79</td>
<td>460</td>
</tr>
<tr>
<td>Salmon, Atlantic</td>
<td>7.1</td>
<td>592</td>
<td>108</td>
<td>1836</td>
</tr>
<tr>
<td>Salmon, Australian</td>
<td>1.5</td>
<td>48</td>
<td>5</td>
<td>615</td>
</tr>
<tr>
<td>Trevally</td>
<td>1.8</td>
<td>294</td>
<td>5</td>
<td>629</td>
</tr>
<tr>
<td>Whiting</td>
<td>0.5</td>
<td>45</td>
<td>3</td>
<td>132</td>
</tr>
<tr>
<td><strong>Canned Fish</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sardine (canned in oil, drained)</td>
<td>15.7</td>
<td>1839</td>
<td>329</td>
<td>2615</td>
</tr>
<tr>
<td>Australian Salmon (Safcol)</td>
<td>3.4</td>
<td>86</td>
<td>36</td>
<td>981</td>
</tr>
<tr>
<td>Pink Salmon (John West)</td>
<td>6.8</td>
<td>116</td>
<td>69</td>
<td>1454</td>
</tr>
<tr>
<td>Red Salmon (Paramount)</td>
<td>10.4</td>
<td>178</td>
<td>89</td>
<td>1740</td>
</tr>
<tr>
<td>Tuna, canned in oil</td>
<td>23.2</td>
<td>10700</td>
<td>930</td>
<td>487</td>
</tr>
</tbody>
</table>

Source: Taken from Meyer, Tsivis, Howe, Tapsell & Calvert 1999. Polyunsaturated fatty acid content of foods: differentiating between long and short chain omega-3 fatty acids. Food Australia 51(3):82-95. All data were adapted from a variety of sources. Some data is sourced directly from Meadow Lea Foods.

* Omega-6 is the linoleic acid (18:2n-6) content
** ALA is α-linolenic acid (18:3n-3).
*** These are long-chain omega-3 polyunsaturates; 20:5 is eicosapentaenoic acid (EPA), 22:5 is docosapentaenoic acid (DPA), and 22:6 is docosahexaenoic acid (DHA).
# The total omega-3 content is the combined total of ALA and the long-chain omega-3 polyunsaturates.
*du; data unavailable.

Due to the biological nature of food, all values are representative only. Foods selected are those that contribute a significant proportion of dietary fat.
Chapter 2. Role of Fats in Foods

Fats have Important Roles in Natural and in Manufactured Foods

Fats contribute to our enjoyment of foods in the following ways:

- **Flavour.** In some foods, fats and oils are used to add flavour. For example, extra virgin olive oil used on salads, or milk fat in ice cream. Fats and oils may also accentuate other flavours in foods by acting as carriers for the flavour compounds.

- **Appearance.** In baked foods, fats help ‘shorten’ the product to give a soft, crumbly texture. They can also provide a sheen to foods and help baked goods hold air during mixing and baking. Fats are important structurally in ice-cream and whipped creams because they help to produce a light and airy texture.

- **Mouthfeel.** In many foods, fats and oils affect the way the food feels in the mouth. In the food industry, this is called *mouthfeel*. For example, there is a considerable difference in mouthfeel between skim milk and regular milk.

- **Moisture retention.** Fats and oils help some food stay moist. For example, breads and cakes made without fat tend to lose moisture and become dry and stale rapidly.

- **Effective Cooking.** Fats and oils can help cook food rapidly. In frying, for example, there is a rapid and uniform transfer of heat to the food being cooked. Frying is common in most cultures.

Thus, fats and oils are integral to food manufacture and domestic cooking because they can add to palatability, taste, texture, and overall enjoyment of food.

The Chemistry of Fats Determines Their Application in Food

The extent of saturation (or unsaturation) has practical importance for the use of fats in food applications

A shortening agent used for baking must be solid at room temperature. An oil for salad dressing must be liquid at room temperature. A fat used for repeated frying must be stable to heat. These characteristics of fats are self-evident to anyone who cooks. Also, they are characteristics which are determined in large part by the double-bond chemistry, or extent of saturation. As discussed in the previous chapter, the description of fats as saturated or unsaturated refers to the number of double-bonds in the fatty acids. Now, it will be seen that this classification of fats has practical importance for their applications in food manufacture and preparation.

The more saturated a fat mixture is, the more likely it is to be solid at room temperature. For example, lard, tallow, butter, and palm oil contain mainly saturated fats. However, butter and palm oil are softer at room temperature compared with lard and tallow. This is because butter and palm oil contain a proportion of unsaturated fats mixed with the saturates.
As the proportion of saturated fats decrease and the proportion of unsaturated fats increase, the melting point of a fat is lowered. Thus, polyunsaturated fats are liquid at room temperature and generally, they remain liquid in the refrigerator. Monounsaturated fats also are liquid at room temperature, but they begin to solidify in the refrigerator. Saturated fats are solid at room temperature and in the refrigerator.

Fats which are liquid at room temperature are called ‘oils’

Because the monounsaturated and polyunsaturated fats are generally liquids at room temperature, they are called ‘oils’. In general, the oils are vegetable in origin. Thus, the fats derived from olive, canola, sunflower, soybean, cottonseed, and peanuts are all oils. However, some vegetable derived fats are semi-solids at room temperature because they have a significant proportion of saturated fats, even thought they are called ‘oils’. Palm oil is in this category.

The extent of saturation is important for stability as well as melting point.

In addition to being a determinant of melting point, the extent of unsaturation is also a determinant of the ‘stability’ of a fatty acid. Instability used in this sense refers to the chemical degradation of fatty acids by oxidative breakdown. If all fats were kept in the refrigerator in the absence of air, they would all be stable to a similar extent. However, in the real world of domestic and commercial food preparation and storage, they are exposed to oxygen and to heat.

Fatty Acid Stability
- oxygen from the air and heat combine to degrade fatty acids
- saturates and monounsaturates are more stable than polyunsaturates
- the degradation can produce destructive free radicals and off-flavours
- the end-point is rancidity

The oxygen in air can react with the double bonds in a fatty acid to initiate its destruction. As often occurs in chemical reactions, this process in increased by heat. As well as losing the fatty acid in this process, the by-products of the reaction are free radical compounds which can initiate chain reactions leading to destruction of other molecules. This leads to ‘off-flavours’ and unsuitability for consumption. The end-stage is commonly known as ‘rancidity’.
In most cases, the problem is not as dramatic as it appears. This is due to the presence of natural anti-oxidants such as vitamin E in the monounsaturated and polyunsaturated vegetable oils. As the name implies, anti-oxidants protect against oxidative reactions which in this case, are destructive. In normal applications, these agents keep the oils stable and sometimes, extra anti-oxidants are added. However, in the high temperatures achieved during frying, particularly with repeated use of an oil for frying, the natural anti-oxidants are overwhelmed and breakdown occurs.

Therefore, in commercial applications where repeated use of a frying fat is necessary (in fish and chip shops for example), saturated fats are used in high proportions due to their stability relative to polyunsaturates. Unfortunately, saturated fats can have adverse health effects and this will be discussed in later chapters. A solution for the future is to increase the proportion of monounsaturated oils for these applications as in general, they are very stable compared with polyunsaturates.

Chemistry of Fats: Importance beyond food

Clearly, the classification of fats by their level of saturation has substantial relevance for commercial and domestic use of fats in food preparation. This classification is relevant also when considering the effects on human biology of the different fats once they are ingested. The following chapters explore the relationships between dietary fats, health and disease.
Dietary fat is necessary for energy and all fats, regardless of their chemical structure, contain the same amount of energy (37 kilojoules/gram). But fats also have biological functions which vary with their chemical structures and this variety in their functions is essential for maintenance of good health. Therefore, when considering the relationship between dietary fat and health, it is most important to distinguish between the different types of dietary fat and their balance in the diet.

When fats are eaten as triglycerides, the following pathways occur to deliver individual fatty acids to tissues. After that, there are a number of possible fates.

Although the possible fates are shown in separate boxes, some fatty acids will undergo several of the events shown. These are now considered in more detail.
### Conversion to Other Fatty Acids

**Dietary Fatty Acids:**
- are mainly 18-carbon in length
- can be lengthened by the addition of more carbons
- the 20- and 22-carbon fatty acids have important biological properties

Besides the double-bond chemistry, there is another aspect of fatty acids which is an important determinant of their function, particularly in the body. This is the ‘chain length’ which is the number of carbon atoms in a single molecule (as shown in Chapter 1). Most of the dietary fatty acids are 18-carbon in length. Once ingested and incorporated into cells, they can be lengthened to 20- and 22-carbons. Once lengthened, additional double bonds can be added. The significance of these processes is that 20-carbon fatty acids are the precursors for the formation of hormone-like agents called prostaglandins, thromboxane, and leukotrienes.

The scheme below shows the common sources of fatty acids in the diet and their potential conversions once incorporated into cells.

<table>
<thead>
<tr>
<th>Fatty Acids</th>
<th>18-Carbon Fatty Acids</th>
<th>20-Carbon Fatty Acids</th>
<th>22-Carbon Fatty Acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sources</td>
<td>Monounsaturates</td>
<td>Polyunsaturates</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(omega-9)</td>
<td>omega-6</td>
<td>omega-3</td>
</tr>
<tr>
<td></td>
<td>oleic acid</td>
<td>linoleic acid</td>
<td>α-linolenic acid</td>
</tr>
<tr>
<td></td>
<td>(these are dietary essential fatty acids)</td>
<td>(these are dietary essential fatty acids)</td>
<td>(these are dietary essential fatty acids)</td>
</tr>
<tr>
<td>Sources</td>
<td>Diet and synthesised in the body</td>
<td>Diet only</td>
<td>Diet only</td>
</tr>
<tr>
<td></td>
<td>olive, sunola® oil, canola oil, meat</td>
<td>sunflower, corn, soybean, safflower oil</td>
<td>flaxseed, canola, soybean oil</td>
</tr>
<tr>
<td>Dietary Intake</td>
<td>Large intake (8-15% dietary energy)</td>
<td>Large intake (7-8% dietary energy)</td>
<td>Minor intake (0.3-0.4% dietary energy)</td>
</tr>
<tr>
<td>Lengthened and more double bonds added</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sources</td>
<td>arachidonic acid</td>
<td>eicosapentaenoic acid</td>
<td>docosahexaenoic acid</td>
</tr>
<tr>
<td></td>
<td>(AA)</td>
<td>(EPA)</td>
<td>(DHA)</td>
</tr>
</tbody>
</table>

The dietary intake levels shown above are based on approximate current levels in Western diets. The excess of omega-6 fat intake compared with omega-3 fat intake has practical consequences because the omega-3 and omega-6 fatty acids are homologues of each other, i.e. very similar except for the position of one double-bond. Therefore, they compete for the same enzymes which metabolise them. However, the excess of omega-6 over omega-3 fatty acids leads to poor metabolism of ingested omega-3 fatty acids to the longer chain omega-3 fatty acids, EPA and DHA. This is an issue of simple competition.
Incorporation into Cell Membranes

**In cell membranes, fatty acids:**
- are not ‘free’ but are incorporated into phospholipids such as lecithin
- can affect membrane fluidity and the function of membrane-bound enzymes

Fatty acids do not stay as ‘free’ fatty acids in cells. They are rapidly incorporated into the phospholipids of the cell membrane. While there are a number of different types of phospholipid, all of them contain two fatty acids linked to a backbone molecule. Lecithin is one of the common phospholipids.

The unique chemistries of the different types of fatty acids have unique physical effects on cell membranes. While the outer membrane of the cell is like a wall in some respects, and like a selective sieve in other respects, it is not rigid like a wall or a sieve. Rather, it is dynamic and fluid. The more unsaturated and the longer are the fatty acids, the more fluid is the membrane which in turn, affects the function of membrane enzymes and other membrane proteins. Therefore, it is significant that the long-chain omega-3 fatty acids in membranes are generally more unsaturated and longer than the omega-6 fatty acids.

Conversion to Biologically Active Hormone-Like Agents

**Arachidonic acid is:**
- at high levels in cells due to the current diet
- crucially important for the production of eicosanoids which have widespread influence on health and disease

In the scheme above which outline fatty acid conversions, the 20-carbon fatty omega-6 acid, arachidonic acid can be seen. It is mainly synthesized from linoleic acid and a small amount is ingested preformed in meat and fish. Arachidonic acid is present in large amounts in the tissues of people on a typical Australian or American diet. This is because these diets contain large amounts of linoleic acid from sunflower oil and related oils. It is also because the diets contain only small amounts of competitor omega-3 fats which can decrease the levels of arachidonic acid.

Arachidonic acid is the precursor of biologically active substances which collectively are known as eicosanoids. They include prostaglandins, thromboxane, and leukotrienes. Eicosanoids can be made by all tissues in the body and they are important for an astonishing array of normal body functions.
While eicosanoids are necessary for maintenance of good health, if there is an imbalance in their production, there can be an alteration from health to disease as a result.

### Balance of Omega-3 / Omega-6 Fats - Health or Disorder?

It is not suggested that all of the disorders shown above are caused by an imbalance in eicosanoid production. The imbalance may be primary or secondary to development of the disorder. However, this does not diminish the importance of eicosanoid control whether it be for prevention or therapy.

### Omega-3 and omega-6 fats can be antagonists of each other

Omega-3 fats can decrease prostaglandin production from omega-6 arachidonic acid. Therefore, a balance which involves increased omega-3 and decreased omega-6 dietary fats may be beneficial when excessive eicosanoid production is clearly a problem, such as:

- excessive thromboxane production in thrombosis
- excessive prostaglandin and leukotriene production in inflammatory disorders

In addition to affecting eicosanoid production, omega-3 fats can decrease production of other compounds such as cytokines, adhesion molecules, and metalloproteinases. These compounds are involved in:

- inflammation such as occurs in arthritis
- atherosclerotic plaque formation
- cartilage degradation such as occurs in arthritis

### Summary and Conclusion

Dietary omega-6 and omega-3 fats can have profound effects in the body via a number of possible pathways. The following chapters examine the evidence for involvement of dietary fats in a number of health problems.

### References

Chapter 4. Dietary Fat and Health – The Issues

What are the Issues?

- the amount of dietary fat
- the type of dietary fat

Dietary Fats and Health – What are the Issues?

Along with protein and carbohydrate, fat is one of the three principal macronutrients. It is needed for many normal cellular structures and processes. If insufficient fat is present in the diet, saturated and monounsaturated fats are synthesized within the body. However, the omega-3 and omega-6 fats cannot be synthesized in the body and, because they are necessary for normal physiological processes, they are dietary essential fats.

Thus, there is no argument that fat is necessary for maintaining good health. For many nutritionists, the principal issue is ‘How much fat should there be in the diet?’. This question has dominated the debate for so long that it has overshadowed a much more important question; namely, ‘What kind of fat should there be in the diet?’

The Amount of Dietary Fat and the Type of Dietary Fat are Separate Issues

It is important to recognise that these are separate questions and, when one examines the linkages between dietary fat and health, it becomes apparent that the amount of dietary fat is probably less important than the type of fat.

These issues are discussed in the following chapters on obesity, heart disease, arthritis, diabetes, infant nutrition, colon and breast cancer, inflammatory bowel diseases, and psoriasis.
Chapter 5. Obesity

The Amount of Dietary Fat and Obesity

Dietary Fat and Obesity - 1
- studies between countries and cross-sectional studies suggest higher fat intake is associated with obesity
- perhaps the availability of high-fat foods in the community leads to excess energy intake by some people,
  BUT
- this conclusion is not supported by analysis of trends

In comparisons between countries, those with a higher average fat intake have a higher prevalence of obesity. Cross-sectional studies within countries also have shown higher fat intake to be associated with obesity. These results suggest that dietary fat content is an important determinant of weight gain. It could be concluded that the availability of high-fat foods in the community may lead some people to eat in excess of energy requirements.

Dietary Fat and Obesity - 2
- in the US, the prevalence of obesity has increased while the community fat intake has decreased, AND
- in the UK, the prevalence of obesity has greatly increased while community energy intake has decreased

However, the strength of this statement is challenged by the data showing that the prevalence of obesity in the US increased by 33% in the decade from 1976 during a period when the average fat intake decreased. Also, the prevalence of obesity in the UK has greatly increased during a period when total energy intake has decreased.

Dietary Fat and Obesity - 3
- tightly controlled dietary studies indicate no difference between low-fat and high-fat diets when energy intake is equivalent
- long-term studies of weight loss have not shown substantial advantages of low-fat diets

Thus, results of population studies with free-living subjects do not support the contention that the availability of dietary fat is the sole or principal determinant of excess weight gain. In addition, tightly controlled dietary studies in metabolic units show that the percentage of fat in the diet has no special effect on body weight when the caloric intake is controlled. For weight gain, low-fat diets were equivalent to high fat diets when the energy content of both was similar. Also, in long term studies on weight loss, there were modest short term advantages with low fat diets but there was little or no difference in the longer term (e.g. after 1 year).
Summary and Conclusions

Dietary Fat and Obesity - Conclusion

- unlikely that community wide recommendations to reduce fat intake will reduce the prevalence of obesity
- obesity is the result of many ‘causes’ coming together in some individuals
- physical exercise will be as important as diet
- it is a complex problem demanding individualised solutions

Overall, one cannot conclude that dietary fat must be reduced to low levels to control obesity in the community. It has been stated that:

“obesity...will not be solved solely by reducing the percentage of fat in the diet” \(^3\).

Undoubtedly, there are many ‘causes’ which coalesce in various ways in different people to produce obesity. For example, there is evidence that the decreased level of physical activity which accompanies a modern lifestyle is a substantial contributor\(^2\). This must also be considered at the community level and in the clinical management of individuals with obesity.

Once present, obesity must be treated on an individual basis with multi-faceted solutions tailored to individual needs. Whether low-fat diets have a place in individual treatment of obesity is a matter of discretion. While reduction in dietary fat will be important for some individuals, advice to follow diets low in fat is not likely to be succesful at the community level\(^7\).

References

Chapter 6. Heart Disease

Coronary Heart Disease.

Primary Events Resulting from Atheromatous Coronary Arteries:
- ischaemia, which is insufficient blood flow to heart muscle, causes angina
- formation of thrombus; in a coronary artery this can cause myocardial infarction
- arrythmia, which is faulty heart muscle contraction, can cause cardiac arrest and sudden death

While there can be many causes of heart failure, coronary heart disease (CHD) is the most common. CHD is usually the result of atherosclerosis of one or more coronary arteries. The narrowing of vessels by atheromatous plaque can lead to insufficient oxygen supply to the heart muscle and result in chest pain (angina). Plaques may crack, allowing the entry of blood into them and leading to formation of a clot (thrombus). The thrombus may occlude the artery which can result in death of some heart muscle (an infarction). In addition, decreased blood flow to the heart muscle (ischaemia) may cause an arrythmia and cardiac arrest.

Secondary Events are Due to:
- progressive "pump" failure which causes heart failure
- arrythmia, which can cause aggravation of heart failure, cardiac arrest, and sudden death

Following survival from a heart attack, the area of infarcted heart muscle can become an area of poorly contracting tissue. If this area is large, the heart will no longer work properly as a pump. This 'pump' failure can lead to progressive heart failure, or to arrythmias. Some arrythmias are called ‘malignant’ because they can lead to sudden death.

Risk Factors
- smoking, overweight, lack of exercise, family history
- elevated LDL and lowered HDL cholesterol, elevated blood triglycerides
- diabetes, hypertension

While the initiating and promoting events in atheroma formation are poorly understood, there are many known risk factors for CHD. These include cigarette smoking, overweight, physical inactivity, elevated LDL cholesterol, lowered HDL cholesterol, elevated blood triglycerides, and diabetes. Hypertension is not a risk factor for CHD, but prolonged hypertension leads to ventricular hypertrophy which can interact with CHD to precipitate heart failure.

Treatment

Anti-thrombotic Treatment
- low-dose aspirin
- trials with new anti-platelet agents

In "at risk" subjects, preventive treatments aim to have an anti-thrombotic action, to lower cardiac oxygen demand (by slowing heart rate), and to decrease the resistance to cardiac output (by lowering blood pressure). Low-dose aspirin is used to suppress platelet activity and thereby reduce thrombotic episodes. Beta-blockers, calcium channel antagonists, angiotensin-converting enzyme inhibitors, and angiotensin receptor antagonists are used to achieve the latter effects. For angina, nitrates are used to effect rapid vasodilation of coronary arteries and provide symptomatic relief.
In addition, the risk factor of elevated plasma LDL cholesterol is addressed through use of inhibitors of cholesterol synthesis. Current dietary advice is aimed also at reducing plasma cholesterol and this can be achieved via reduced intake of saturated fats and use of margarines containing phytosterols. Attention should also be given to life-style factors such as regular exercise, smoking, etc.

Role of Dietary Fat in Heart Disease

**Amount of Dietary fat and Heart Disease**
- both low-fat and high-fat diets have been associated with low rates of heart disease
- the association of high rates of heart disease with fat intake is due to the high saturated fat content of the diet

The traditional Japanese diet is low in fat (≈ 20% of dietary energy) and is associated with a low rate of heart disease. By contrast, the Greenland Eskimo diet and the Mediterranean diets have relatively high fat content (≈ 40% of dietary energy) and are associated also with low rates of heart disease. In further contrast, some northern European diets which had high fat content were associated with high rates of heart disease.

**Type of Dietary Fat and Heart Disease: Between Country Comparisons**
- direct association with saturated fat intake
- inverse association with monounsaturated, omega-3 and omega-6 polyunsaturated fat intake

The apparent discrepancies between total fat intake and prevalence of heart disease are most likely explained by the type of fats in the various diets. The northern European diets were high in saturates whereas the Mediterranean and Greenland diets were high in monounsaturates and omega-3 polyunsaturates.

There is evidence from dietary intervention studies and prospective cohort studies that there is a relationship between increased dietary saturated fat intake and increased incidence of heart disease or increased incidence of secondary events after established heart disease.
In Western diets, omega-6 polyunsaturates have increased in the last 40 years. In general, these have been used as substitutes for saturated fats—margarine for butter, vegetable oil for tallow. This change has been coincident with a decline in the rate of heart disease and thus, omega-6 fats also appear to be beneficial when eaten instead of saturated fats. Because in many studies the increased intake of omega-6 polyunsaturates was directly related to the decreased intake of saturated fats, it has been difficult to discern the ‘stand-alone’ effects of omega-6 polyunsaturates. In fact, several studies found an association between death from heart attack and the Keys and Hegsted scores. The Keys and Hegsted scores take account of dietary saturated fats, omega-6 polyunsaturates, and cholesterol. Many of these early studies did not initially consider omega-3 fat intake.

Overall, these results indicate that the type of fat is more important than the amount of fat which is eaten.

A considerable amount has been published regarding saturated fats and omega-6 polyunsaturated fats, blood cholesterol levels, and heart disease. There is relatively less documentation on the effects of omega-3 polyunsaturated fats which appear to have protective effects for heart disease which are unrelated to effects on blood cholesterol levels. These are considered in detail here.

### Evidence for Preventive Effects of Omega-3 Fats

Dietary omega-3 fats are beneficial for heart disease. One can have confidence in this statement due to the weight of consistent evidence from studies of different kinds. The studies fall into four categories: comparisons between countries (ecological), comparisons of the diet of people who have, or do not have heart disease (case-control), observations of the health and diet of groups as they change over time (cohort studies), and observations of the health of groups after the diet is deliberately changed (dietary intervention).
Ecological Observations relating to Omega-3 Fats

**Ecological Studies**
- Communities with higher omega-3 fat intake have lower rates of death from heart disease

On their traditional diets, Greenland inuits and Japanese have higher intakes of omega-3 fats than that in the US and Australia due to their higher intake of fish and marine mammals. Higher intakes are reflected in the blood levels of omega-3 fats, particularly EPA, and are coincident with reduced risk of cardiovascular mortality (see Table).

<table>
<thead>
<tr>
<th></th>
<th>US</th>
<th>Japan</th>
<th>Greenland</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary intake of fish derived omega-3 fats(^a)</td>
<td>&lt;0.1%</td>
<td>1%</td>
<td>4%</td>
</tr>
<tr>
<td>Cardiovascular mortality(^b) (% of all deaths)</td>
<td>45</td>
<td>12</td>
<td>7</td>
</tr>
</tbody>
</table>

\(^a\) % of dietary energy\(^10\); \(^b\) from ref\(^11\)

While there are many other differences in lifestyle between countries, the results support a preventive effect of dietary omega-3 fats.

**Case-Control Study relating to Omega-3 Fats**

**Case-Control Study**
- Much lower risk of having a heart attack when omega-3 intake was higher and blood levels of omega-3 fats were higher

In a study from the Seattle area, the diet of people who had experienced a heart attack was compared with that of people who had not had a heart attack, but who were otherwise matched for age and sex. The data were used to calculate the odds of having a heart attack given a certain level of intake of omega-3 fats\(^12\). The results indicated that a weekly intake of omega-3 fats of about 3.4 g resulted in a 60% reduction in the odds of having a heart attack. Similar results were obtained from analysis of blood cell levels of omega-3 fats.

<table>
<thead>
<tr>
<th>Comparing omega-3 intakes between study groups</th>
<th>Reduction in risk of heart attack</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.4 g / week intake versus none</td>
<td>60% reduction(^12)</td>
</tr>
</tbody>
</table>
Cohort Studies related to Omega-3 Fats - Prevention of Primary Events in Heart Disease

The results from six large cohort studies involving both men and women indicate beneficial effects of omega-3 fats derived from fish or from vegetable oils. The table sets out summaries of the findings.

<table>
<thead>
<tr>
<th>Study</th>
<th>Comparisons</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zutphen study13</td>
<td>≥ 30g fish daily (approx 2 meals / week) vs. no fish</td>
<td>64% risk reduction for cardiovascular death</td>
</tr>
<tr>
<td>Chicago Western Electric Study14</td>
<td>≥ 35g fish daily (approx 2 meals / week) vs. no fish</td>
<td>67% risk reduction for non-sudden cardiac death. Statistically significant trend for level of fish intake and risk reduction.</td>
</tr>
<tr>
<td>US Physician's Health Study15</td>
<td>One fish meal / week vs. less than one / month</td>
<td>52% risk reduction for sudden cardiac death.</td>
</tr>
<tr>
<td>Health Professionals Study16</td>
<td>1% dietary energy increase in ( \alpha )-linolenic acid</td>
<td>59% risk reduction for total MI (fatal + non-fatal)</td>
</tr>
<tr>
<td>MRFIT (Multiple Risk Factor Intervention Study)17</td>
<td>Regression analyses for CHD death and omega-3 fat intake, both fish fatty acids and ( \alpha )-linolenic acid</td>
<td>Statistically significant regressions for fish fatty acids and ( \alpha )-linolenic acid. For highest vs lowest quintiles, risk reductions = 50% and 42% for fish fatty acids and ( \alpha )-linolenic acid, respectively.</td>
</tr>
<tr>
<td>Nurses Health Study18</td>
<td>1.4 vs 0.7 g / day ( \alpha )-linolenic acid</td>
<td>45% risk reduction for fatal CHD</td>
</tr>
</tbody>
</table>

Consider the Zutphen study. The results indicate that men who had approximately two fish meals per week had a 64% reduction in risk for cardiovascular death. Consider the Health Professionals Study. The results indicated that an increase in the dietary intake of the plant omega-3 fat, \( \alpha \)-linolenic acid, was associated with a 59% reduction in the risk of a myocardial infarction, either fatal or non-fatal.

Cohort Studies

- 42% to 67% reduction in risk of death from heart attack arising from consumption of omega-3 fats
- benefits for omega-3 fats from fish and from vegetable oils

Overall, the risk reductions arising from consumption of omega-3 fats were large, ranging from 42% to 67%. 
Dietary Intervention Studies with Omega-3 Fats - Prevention of Secondary Events in Heart Disease

**Dietary Intervention Studies**
- conducted in people who had already had a heart attack
- large decrease in risk of second heart attack when diet was changed to increase omega-3 fat intake
- benefits with omega-3 fats from fish, fish oil, and vegetable oil (canola)

<table>
<thead>
<tr>
<th>Diet and Reinfarction Trial (DART)19</th>
<th>Comparisons</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPA intake in the two groups was 2.3 vs 0.7 g / week</td>
<td>32% decrease in CVD death in fish advice group (no change in non-fatal MI)</td>
<td></td>
</tr>
</tbody>
</table>

| Lyon Diet-Heart Study20 | Mediterranean-type diet advice or usual hospital advice | For cardiac death + non-fatal MI: 73% decrease at 27 months 72% decrease at 46 months Plasma α-linolenic acid inversely correlated to CVD outcomes |

| GISSI Prevenzione Study21 | Allocated to ± fish oil (0.85 g/day n-3 ethyl esters), 3.5 year follow-up | Fish oil use associated with: 45% decrease in sudden deaths |

The result from three large studies with men and women who had already had a heart attack, indicate that an increase in the intake of omega-3 fats from either fish, fish oil or vegetable oil, will decrease the risk of further heart attack and death (see Table). For example, in the Lyon Diet-Heart Study, patients in the intervention group changed their diet to include more fruit, vegetables, and beans and to replace saturated fats with canola or olive oil and canola margarine. At 27 months of follow-up, there was a 73% decrease in risk of myocardial infarction, both fatal and non-fatal, and the decreased risk correlated with blood levels of α-linolenic acid.

**What are the mechanisms?**

**Potential Mechanisms for the Beneficial Effects of Omega-3 Fats in Heart Disease**
- reduction of risk factors
- reduction of thrombosis
- reduction of atheromatous plaque formation
- reduction of irregular heart rhythms

There are several possibilities. Omega-3 fats can decrease certain risk factors for heart disease, they can decrease platelet aggregation and blood clots (thrombus), they can decrease white blood cell activity (atherogenesis), and they can decrease irregular heart rhythms (arrhythmias).
Reducing the Recognised Risk Factors with Dietary Fats

**Omega-3 Fats and the Recognised Risk Factors for Heart Disease**

- for cholesterol, reducing saturates is the important issue – replacement with monounsaturates or omega-6 or omega-3 polyunsaturates is similar
- cholesterol can be reduced also with margarines to which phytosterols have been added
- omega-3 fats will reduce triglycerides
- omega-3 fats can reduce hypertension
- obesity is a complex issue which requires advice tailored to individual needs and preferences

1. **Blood Cholesterol**: Reduction of blood cholesterol levels is accomplished by drugs and by reducing saturated fat intake. The latter is accomplished by trimming the fat from meat, using low-fat dairy products, replacing butter with margarine, and by using vegetable oils for cooking in place of solid fats. The margarine and vegetable oils can be monounsaturated, omega-6 or omega-3 polyunsaturated because the effects on cholesterol lowering are similar.

In addition, the ingestion of phytosterols, which have been included in some margarines, can lower blood cholesterol.

Note that, although trans fatty acids act like saturated fats with regard to cholesterol elevation, Australian margarines have little trans fatty acid content (by comparison with US margarines).

2. **Blood Triglycerides**: Elevated triglycerides are a risk factor for CHD. Of the dietary fats, only omega-3 fats lower plasma triglycerides.

3. **Hypertension**: Omega-3 fats from fish oil can modestly decrease blood pressure.

4. **Overweight and Obesity**: The Body Mass Index (BMI) is a parameter which takes account of height and weight. A BMI > 25 kg/m² is considered to be in the overweight range. The role of total fat content in the diet of an individual and their BMI is a subject of some controversy. This topic has been discussed in Chapter 4. Briefly, it has been pointed out that populations such as the US have experienced an increase in obesity while the average fat intake has fallen. Also, controlled intervention studies with fat-reduced diets generally have been unsuccessful. It was concluded that “obesity … will not be solved solely by reducing the percentage of fat in the diet.”

Clearly, overweight and obesity is a complex issue which needs to be addressed on an individual basis and attention to physical activity will be an important component.
Thrombosis and Dietary Fats

**Omega-3 Fats and Thrombosis**
- will reduce thrombus formation and therefore, thrombotic complications

By suppressing production of the pro-aggregatory platelet product, thromboxane, omega-3 fats suppress platelet aggregation. Since platelet aggregation is a major event initiating thrombosis, omega-3 fats may decrease the thrombotic complications of CHD.

Atherogenesis, White Blood Cell (Leukocyte) Activity and Dietary Fats

**Omega-3 Fats and Atherogenesis (plaque formation)**
- they decrease the atherogenic potential of leukocytes by decreasing production of inflammatory agents and growth factors
- they decrease blood vessel changes that favour plaque formation
- they decrease atheromatous plaque formation in an animal model

Leukocytes are the blood cells that respond to injury or infection with a protective inflammatory response or an immune response. However, leukocytes are prominent cells in the atheromatous plaque which suggests that early plaque formation has an inflammatory component.

Omega-3 fats can decrease inflammatory prostaglandin, leukotriene and cytokine production by leukocytes. They can also decrease leukocyte production of growth factors which are involved in atheromatous plaque formation. In addition, they can decrease blood vessel changes (expression of adhesion molecules) which attract the leukocytes. All of these biochemical actions can decrease the atherogenic potential of leukocytes. Therefore, it is not surprising that dietary omega-3 fats have been shown to prevent atheroma formation in an animal model.

Arrhythmias and Dietary Fats

**Omega-3 Fats and Arrhythmias**
- both omega-6 and omega-3 fats have anti-arythmic actions
- omega-3 fats are superior to omega-6 fats
- canola oil was superior to sunflower or soybean oil

Once heart muscle is damaged by an infarct, the normal co-ordinated electrical conductivity is interrupted. This can lead to an arrhythmia, particularly during periods of reduced blood flow to the heart muscle (ischaemia). When arrhythmias involve the main pumping chamber of the heart, the left ventricle, they can be fatal. Arrhythmias are thought to be the cause of ‘sudden death’ in heart disease.

It has been shown in animals and in heart cell cultures that omega-3 fats derived from fish oil or vegetable oil, as well as omega-6 fats have anti-arythmic effects. In the cell cultures omega-6 fats had variable effects, sometimes being pro-arythmic due to their conversion to eicosanoids (see Chapter 3). In animal studies, omega-3 fats were superior to omega-6 fats, which showed up in the stronger anti-arythmic effects of canola oil compared with sunflower or soybean oils.
Summary and Conclusions

There is no argument that reducing saturated fat intake is important. These can be replaced with monounsaturates or omega-6 polyunsaturates. However, the greatest benefits will be seen when omega-3 polyunsaturates are used also. Considering this last point and considering that omega-6 fats are antagonists to omega-3 fats at many levels (absorption, metabolism, biological activity), monounsaturates can provide a background more favorable to the beneficial effects of omega-3 fats.

Translating this to practical advice means:

- increasing the intake of fish and omega-3 containing vegetable oil products such as canola and flaxseed, but
- using monounsaturated oil products (olive oil, sunola oil) where necessary, e.g. in frying

### Dietary Fat and Heart Disease
- the type of fat is more important than the amount of fat
- reduce saturated fat intake
- omega-3 fats are beneficial
- monounsaturates provide the best accompaniment to omega-3 fats

### In practical terms, consider that:
- fish contains omega-3 fats
- canola oil and flaxseed oil contain omega-3 fats
- olive oil and sunola oil contain monounsaturates

### Is this mixture of dietary fats experimental?
- hardly, it is that which has been used in the southern Mediterranean for thousands of years
Rheumatoid arthritis is an inflammatory disorder which affects many systems, but the most obvious problem is joint inflammation. In early disease, the pain, swelling, and tenderness result from inflammation of the joint lining (the synovium). In the healthy state this lining is only a single cell thick. With persistent inflammation, it becomes thickened and there is an outgrowth onto the cartilage surface. This abnormality is associated with progressive degradation of the articular cartilage that covers the ends of the bones. In advanced disease, this damage leads to joint deformities and joint failure. For example, deformities in the joints of the fingers and wrist can lead to an inability to perform tasks such as turning a tap or opening a jar or packet.

Rheumatoid arthritis is 3-fold more likely in women than men and there are known genetic predispositions. However, unlike normal inflammation which is a response to trauma or infection, there is no apparent reason for initiation of the joint inflammation, and unlike the normal situation, the inflammation persists instead of naturally resolving.

**Treatment**

**Treating the Symptoms**
- non-steroidal anti-inflammatory drugs (NSAIDs), selective COX-2 inhibitors
- steroids

**Treating the Underlying Disease**
- hydroxychloroquine, sulphasalazine
- gold salts
- leflunomide
- methotrexate, cyclosporin

The dominant approach to therapy for rheumatoid arthritis involves use of pharmaceuticals. Aspirin-like agents known as non-steroidal anti-inflammatory drugs (NSAIDs) are used for relief from pain, swelling and stiffness. Amongst these drugs are indomethacin, ibuprofen, diclofenac, naproxen and many others. While they are effective, gastric toxicity is a major problem with chronic use of these agents. The latter problem has been addressed by the introduction of new subclass of NSAIDs known as COX-2 inhibitors which have similar anti-inflammatory effectiveness to other NSAIDs, but with greatly reduced gastric toxicity.

In addition to NSAIDs, a group of drugs known as slow-acting anti-rheumatic drugs are used also, often in combinations, in an attempt to slow the progression of the underlying destructive disease. Their effectiveness in slowing cartilage degradation is questionable and a major problem also with many of these drugs is toxicity (eyes, blood, kidney, liver, skin). Toxicity, as well as lack of efficacy, are major reasons for patients discontinuing their use of drugs in this class.
Evidence for Preventive Effects of Omega-3 Fats

The Japanese, who have a diet rich in omega-3 fats derived from fish, have a lower rate of rheumatoid arthritis than Western communities\(^1\). This observation is made more significant by the fact that a gene which confers susceptibility to rheumatoid arthritis is more prevalent in Japan. Thus, based on the genetic makeup, one would predict an increased prevalence of rheumatoid arthritis when in fact, the opposite is seen.

Further evidence for preventive effects of omega-3 fats is found in the results of a case-control study from the Seattle area. It was reported that fish consumption was higher in healthy controls than in the women who had rheumatoid arthritis. In fact, being in the top 10\% of omega-3 fat intake (>1.6g/day) was associated with an approximate 70\% decreased probability of having (seropositive) rheumatoid arthritis\(^2\).

Evidence for Therapeutic Effects of Omega-3 Fats

Placebo-controls are important in most clinical trials and this is true especially in arthritis studies where the ‘placebo’ effect can be as high as 40\%. This consideration is important when evaluating the evidence for efficacy of diets, dietary supplements, and ‘neutraceuticals’.

At least 12 double-blind placebo-controlled trials of fish oil use in rheumatoid arthritis have been published and all studies indicated a beneficial effect of the increased omega-3 intake. This conclusion is supported by review of each study and by meta-analysis\(^3,4\). The number of tender joints was the measure which improved in most studies, but other measures to improve were the duration of morning stiffness, grip strength, and time to fatigue. There was also an indication of an NSAID sparing effect. It was apparent that 12 weeks was the minimum time at which effects were seen\(^5\).

Benefits Seen even though Omega-3 Fats were:
- taken on top of existing medication
- taken with high levels of antagonistic omega-6 polyunsaturates
- all patients had long-standing disease

Therefore, it is possible that:
- the strength of the benefits for some patients will be greater than that seen in the studies so far

Note that there were potentially moderating factors in the reported studies. Firstly, fish oil was given as an addition to continued medication. Thus, the beneficial effects were additional to the drug effects. Secondly, fish oil was given as a supplement to typical Western diets. These are generally high in omega-6

\(^{1}\) James, M., Cleland, L. G., & Meadow Lea Foods Ltd (2000).
\(^{2}\) Case-control study from Seattle.
\(^{3}\) At least 12 double-blind placebo-controlled studies using fish oil capsules with rheumatoid arthritis patients.
\(^{4}\) Beneficial effects seen for swollen joints, tender joints, morning stiffness.
\(^{5}\) Suggestion of drug-sparing effects.
polyunsaturates which decrease tissue levels of omega-3 fats. Thirdly, in all studies patients had long-standing disease (median duration was 10 years) where there is frequently irreversible joint damage which defies the best available treatments. Overall, it is probable that the magnitude of the beneficial effect of omega-3 fats is greater than that which was observed in the trials.

What are the Mechanisms?

Eicosanoids have been mentioned in the chapter ‘Fats in the diet’. The eicosanoids, prostaglandin E_2 (PGE_2) and leukotriene B_4 (LTB_4), are produced within inflamed joints. Together, they cause pain and swelling. The cytokines, tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β), also appear to have a role in the symptomology as shown by the dramatic disease suppressive effects of anti-TNF-α therapy with monoclonal antibodies or soluble receptor antagonists. The cytokines are probably responsible also for triggering cartilage degradation. Suppression of PGE_2 synthesis is an action of all non-steroidal anti-inflammatory drugs (NSAIDs) and suppression of cytokine synthesis or action is a target of newly developed anti-inflammatory agents.

Because omega-3 fats can suppress production of these inflammatory agents (eicosanoids and cytokines), it is most likely that these actions are responsible, at least in part, for their beneficial effects in rheumatoid arthritis. However, because the biochemical suppressive effects of omega-3 fats on these inflammatory messengers are generally less than those of drugs, one cannot be confident that these actions of omega-3 fats explain all of their beneficial effects.
Summary and Conclusion

There is consistent evidence for a beneficial effect of omega-3 fats in rheumatoid arthritis, suggesting that diet has a role in therapy for this inflammatory disorder. In most patients, it is likely that diet will have a shared role with current drug treatments. Such an approach would cater well for the majority of patients who seek a more natural approach to disease control. The promotion of a combined dietary / drug approach centred on omega-3 fats also allows for collateral health benefits, e.g. reduced cardiovascular disease for which rheumatoid arthritis patients are especially at risk (as discussed in the chapter on heart disease). Furthermore, the use of omega-3 fats in the family setting can be expected to provide some protection from rheumatoid disease to susceptible first-degree relatives.

References

Chapter 8. Diabetes

What is it?

In both Type I or Type II diabetes mellitus, symptoms include tiredness, thirst, and frequent urination. Elevated fasting blood glucose (hyperglycaemia) is a defining feature. Type I, or insulin-dependent, diabetes mellitus results from diminished insulin secretion due to auto-immune damage to the insulin-secreting cells of the pancreas. Type I disease is treated by insulin replacement and will not be discussed here.

Type II, or non-insulin-dependent, diabetes (NIDDM) results from diminished insulin action, also described as insulin resistance. Frequently, the diabetes symptoms occur in conjunction with obesity, altered plasma lipids (increased triglycerides and decreased HDL), elevated or reduced blood insulin, and hypertension. This cluster is known also as metabolic syndrome or syndrome X. Importantly, these alterations in blood lipids, body weight, and blood pressure are indicators of increased risk for cardiovascular disease, which is one of the main long-term problems associated with NIDDM.

The cause and effect relationships between these derangements are uncertain, but there is general agreement that insulin resistance is an early event in the disease and it could give rise to the metabolic changes seen in syndrome X. There appears to be a genetic component because there is a high concordance between identical twins compared with non-identical twins and other first-degree relatives. However, there is also an environmental component because obesity alone can cause insulin resistance.

A central role for insulin resistance.

The major function of insulin is to allow transport of glucose from the blood into tissues (muscle etc). In addition, insulin is important for fat metabolism. Therefore, elevated plasma triglycerides as well as elevated plasma free fatty acids are found in NIDDM. Correction of the blood lipid levels with drugs does not improve the insulin resistance.

The association between insulin resistance and hypertension is confounded by obesity in many studies, but there is sufficient evidence from human and animal studies to indicate a causal effect of insulin resistance.

Obesity appears to be one of the precipitating causes, rather than the result of insulin resistance, since weight loss can improve insulin sensitivity.
Current Treatment

Diet, lifestyle change, and drugs are used in combinations according to individual needs and preferences.

**Weight Loss if Necessary**
- Diet
- Exercise

If overweight, weight loss through moderately reduced energy intake and exercise will improve blood lipids and insulin resistance. It is not clear whether exercise alone improves insulin sensitivity or whether the effects are secondary to loss of adipose tissue\(^6\). Thus, increased exercise may be beneficial also in patients with normal bodyweight.

While low-fat (<30% energy), high complex carbohydrate diets are commonly prescribed, it is not clear that these are preferable over higher fat diets. For example, a high carbohydrate diet increases blood insulin and triglyceride levels, changes which are undesirable. Certainly, increased dietary fibre ‘neutralises’ the potentially adverse effects of increased dietary carbohydrate\(^7,8\). Thus, plentiful fruit and vegetables, cereals and grains are important components of this type of diet.

**High Carbohydrate / Low Fat Diet**
- commonly recommended
- but can elevate blood triglycerides
- dietary fibre particularly important

There is not universal acceptance that a high carbohydrate / low fat diet is the only option in Type II diabetes. It has been shown that a high monounsaturated fat (40% energy), low carbohydrate diet (40% energy) was more effective in reducing insulin resistance and blood levels of glucose, insulin, and triglycerides than a low fat (20% energy), high carbohydrate (60% energy) diet\(^9\). Thus, if excess weight is not a problem, the amount of fat may not need to be as low as previously thought. Of course, this does not apply to saturated fats which should be limited to less than 10% of dietary energy.

**High’ Fat / Low Carbohydrate Diet**
- increased insulin sensitivity
- good glycaemic control
- lower blood triglycerides
- saturated fat intake must be minimised

**Drugs**
- if diet is not sufficient, drugs can be used to lower blood sugar

The oral hypoglycaemic drugs which are used have the actions of increasing insulin secretion by the pancreas (e.g., tolbutamide, glipizide) or decreasing insulin resistance (metformin), or decreasing starch breakdown (acarbose).

Type of Dietary Fat

There is no argument that saturated fat intake should be limited. This is because it elevates blood cholesterol (LDL) levels and it is associated with insulin resistance, independent of body mass\(^10\).

**Type of Fat**
- low saturated fat
- monounsaturated fat, omega-6 and omega-3 polyunsaturates are all good alternatives to saturated fat in respect to insulin sensitivity and lowering blood cholesterol
- omega-3 fats have the additional benefit of lowering blood triglycerides

The effects of omega-6 and omega-3 polyunsaturates are beneficial, with some suggestion for more widespread benefits from the latter. In studies with NIDDM patients and safflower oil (omega-6) or fish oil (omega-3) supplementation, there are beneficial effects from both for lowering of LDL cholesterol, but only fish oil lowers triglycerides, with greater effects seen in the patients with elevated triglycerides at baseline\(^11,12\).
some studies, blood glucose control deteriorated with both omega-6 and omega-3 supplementation, suggesting these polyunsaturates may have deleterious effects. However, the effect appears to be due to the increased energy intake, i.e. the fact that they are supplements and not part of the usual diet11.

Prevention as well as Treatment
• epidemiological evidence for protective effects of fish in the diet

In rat studies, it was shown that omega-3 fats taken as fish oils had a profound protective effect in preventing the development of insulin resistance13. This has not been observed in human studies12,14. However, there is epidemiological evidence from Greenland, Finland and the Netherlands that omega-3 fats taken as fish may protect against the development of insulin resistance and diabetes15,16,17.

Summary and Conclusions.

Whether low fat (<30% energy) or moderate fat (35% energy) diets are used, the type of fat is important. Saturated fat must be limited (<10% energy). The remaining fat could be monounsaturated (olive or high oleic sunflower oil) or omega-6 polyunsaturated (sunflower oil) or omega-3 polyunsaturated (canola oil, fish and fish oil). There may be additional benefits from omega-3 fats in terms of triglyceride lowering. Additionally, there may even be protective effects from omega-3 fats for the development of NIDDM in healthy individuals although this latter point remains conjecture.

Summary
• diet, exercise, drugs used in combinations as necessary
• diet must limit saturated fat intake
• some problems with high carbohydrate / low fat diet
• may be benefits from lower carbohydrate and moderate fat diet
• fats can be monounsaturated, omega-6 or omega-3 polyunsaturated
• omega-3 fats may be preventive for emergence of NIDDM

References
Neural tissue and omega-3 fats

In adult humans, the omega-3 fatty acid, docosahexaenoic acid (DHA), is present in brain grey matter and retina in very high amounts compared to any other tissue (except sperm)\(^1\). This is true also in infants where the brain DHA content increases through pregnancy, particularly in the third trimester. Brain DHA increases also in the first few months after birth, although only in breast-fed infants.

The high DHA content in retina and brain suggests a particular role for this omega-3 fat in vision. Thus, it is not surprising that animal studies (with rhesus monkeys) have shown that severe omega-3 deficiency during pregnancy and lactation results in abnormal electroretinograms and visual impairment\(^3\).

Fatty Acid Composition of Breast Milk and Infant Formula

Whereas breast milk is a good source of omega-3 and omega-6 fats, conventional infant formulae were quite deficient in long-chain omega-3 fats, e.g. DHA, as well as in long-chain omega-6 fats.

Formula does contain the short chain omega-3 fats which are precursors for DHA formation. However, this may not be sufficient because there is poor conversion to DHA by the infant. In part, this is due to metabolic competition from the excess of short chain omega-6 fatty acids. Clearly, this has implications for the DHA content of brain and retina in newborn infants.
Implications for Newborn Infants

**Compared with breast-fed infants, formula-fed infants had:**
- lower DHA levels
- poorer visual acuity

**Compared with breast-fed infants, infants using fish oil supplemented formula had:**
- similar DHA levels
- similar visual acuity

In formula fed infants, blood cell DHA decreases rapidly after birth and by 4 – 6 months of age, DHA levels can decrease by more than 50%. This contrasts markedly with the situation in breast-fed infants where DHA levels are maintained.

Furthermore, the low DHA levels in formula fed infants have correlated with substantially lower visual acuity scores at 16 and 30 weeks of age. Several studies have shown this effect, suggesting that the lowered DHA content is functionally significant for the retinal / visual cortex axis. The proof for this came from a study where formula was supplemented with DHA (fish oil). The infants who received this formula had DHA levels and visual acuity scores similar to those of breastfed infants.

Such studies have led to the supplementation of commercial formula with DHA. Arachidonic acid is included also as there was initial indication that slight growth retardation occurred in the absence of arachidonate.

Looking beyond fatty acids, breast milk contains growth factors and antibodies which are not present in formula and thus, breast milk remains the best food for healthy infants.

**Implications for Pregnancy and Lactation**

- **Lactating Mothers**
  - breast milk DHA increases with dietary DHA, but
  - no evidence currently that DHA supplementation of the maternal diet confers extra benefits

The DHA content of breast milk varies with the diet of the mother. However, there is no current evidence for benefits to the infant of maternal dietary supplementation with fish oils. Certainly, the short-chain omega-3 fat in the diet (ω-3-linolenic acid) appears to be adequately converted by the mother to support DHA breast-milk levels. Nevertheless, it would be prudent to choose omega-3 containing foods where possible.

**References**

Chapter 10. Breast Cancer

Animal Studies

In rat models, breast cancer is favoured by high fat diets containing omega-6 polyunsaturates. However, the apparent promoting effect of omega-6 fats is seen only up to a ceiling, above which further increases in omega-6 fat intake have no further promoting effect\(^1\)\(^,\)\(^2\). By contrast, omega-3 polyunsaturates have a protective effect on cancer development with stronger inhibition depending on an intermediate, rather than a higher omega-3 / omega-6 ratio\(^1\)\(^,\)\(^3\).

Conjugated linoleic acid (CLA) is a variant of linoleic acid which is present in dairy products, beef and lamb, but not vegetable oils. Even at low levels in the diet (maximum effect at <1% of dietary energy), CLA markedly inhibited breast cancer development in rats, with the effect being much greater than that of omega-3 fats and independent of the background dietary fat\(^3\)\(^,\)\(^4\).

Monounsaturates and trans fatty acids had little or no effect in animal models of breast cancer\(^7\).

Human Studies – Total Fat

Initial indications that high dietary fat levels may increase breast cancer came from comparisons between countries with low fat and high fat intakes. However, these are comparisons primarily between poor and wealthy nations where there are many differences in addition to levels of fat intake\(^5\)\(^,\)\(^6\). There is a relationship between breast cancer and fat intake between levels of 10 to 30% of dietary energy. However, amongst the developed countries in the fat intake range 30 to 45%, there is little or no apparent relationship\(^6\).

Cohort studies can provide more uniform groups than ecologic studies. A pooled analysis of seven prospective cohort studies (337,000 women) showed no significant change in risk for breast cancer over a range of fat intakes from approximately 15 to 45 % energy\(^7\).

Human Studies – Type of Fat

Data from ten case-control studies and a pooled analysis of seven large cohort studies do not provide any support for an association between intake of omega-6 or omega-3 polyunsaturates and risk for breast cancer\(^8\). In the pooled analysis, when the risk of having breast cancer for those in the top 20% of omega-6 intake versus those in the bottom 20% of intake were compared, there was no significant difference in the breast cancer risk\(^7\). In other words, there was no discernible effect of omega-6 fats on breast cancer risk.
It is expected that human studies into the effects of CLA will soon appear. Results from the first study have indicated a strong negative association between CLA levels in breast tissue and the risk of breast cancer. There was an average 85% reduction in risk for women who had the highest levels of conjugated linoleic acid\(^9\). Further studies will be necessary to assess the value of these initial findings.

Summary

Whereas animal studies have shown a tumour enhancing effect of total fat intake and a protective effect of omega-3 fats, results from human studies have not supported either of these findings. The apparent preventive effect of conjugated linoleic acid is expected to lead to re-analysis of existing human data as well as prospective studies.

References

Chapter 11. Colon Cancer

Animal Studies

**Animal Studies and Colon Cancer**
- no evidence for tumour promoting effect of dietary total fat or omega-6 polyunsaturates
- protective effect of omega-3 fats from fish oil or vegetable oil

Diets high in saturated fat or omega-6 polyunsaturates have appeared to favour tumour formation in chemically-induced colon cancer in rats. However, high energy intake and weight gain also favoured tumour formation, and when these have been controlled, it is not clear that there is any independent effect of these dietary fats. The apparent effect of omega-6 fats may reflect only a minimum requirement for these fats, rather than a true promoting effect.

A clearer result is seen in the tumour suppressive effects of omega-3 fats from either fish oil or vegetable oil in the chemically-induced colon cancer models. Importantly, the results extend to a mouse model of the human condition, adenomatous polyposis coli (APC) which is a genetically inherited disposition for colon cancer. In these mice, which have naturally occurring intestinal polyps and tumours, the omega-3 fat, DHA, suppressed tumour development, although the effect was largely limited to females.

Human Studies

**Human Studies and Colon Cancer**
- associated with increased total energy intake
- no independent association with total fat intake
- protective effect of fish consumption

There are divergent conclusions arising from studies on total fat intake and colon cancer. Most case-control studies (but not all) have found that colon cancer risk increases with total fat intake. But there was also an association with total energy intake. A meta-analysis which examined thirteen case-control studies and made an allowance for total energy intake concluded that there was no specific association with fat intake. This conclusion is supported also by the majority of cohort studies. In other words, replacing dietary fat with carbohydrate or protein would not alter risk for colon cancer.

With regard to the type of fat, the main consistent finding is that fish consumption is associated with decreased risk for colon cancer. This is the conclusion from a review of individual case-control and cohort studies, as well as a combined ecologic study across several European countries. It is also the conclusion of a very large integrated series of case-control studies across Northern Italy where the total number of colorectal cancer cases accumulated was 926. In this study, the consumption of ≥ two fish meals per week approximately halved the risk of colon and rectal cancer.

The concordance between animal and human studies suggest a genuine protective effect of omega-3 fats for colon (and rectal) cancer.
Don’t Forget the Fibre

**Overall Agreement between Animal and Human Studies that:**
- dietary fat alone does not promote the occurrence of colon cancer
- omega-3 fats are protective
- dietary fibre or fruit and vegetables are protective

It is difficult to reduce the effects of diets to a single component such as fat. There is an interaction with dietary fibre such that a high fat, low fibre diet is the most conducive for colon cancer occurrence in animal models, and vice versa. Systematic reviews of more than one hundred human studies are consistent for the protective effects of fruit and vegetable consumption. Whether it is fibre or micronutrients is not clear from these studies. However, there are known mechanisms for the beneficial effects of fibre and these include: fermentation in the gut to short chain fatty acids such as butyrate which have tumour suppressing properties, binding bile acids which have tumour promoting effects, diluting potential carcinogens, and increasing transit rate through the gut.

**References**

Chapter 12. Inflammatory Bowel Diseases

Ulcerative Colitis

**Ulcerative Colitis and Fish Oil**
- clinical benefits were modest, but they were additional to those from drug use
- in some patients, dietary fish oil reduced steroid requirements
- once in remission, fish oil can slow the rate of relapse

Studies with fish oil use in ulcerative colitis have been conducted in two ways. In three double-blind placebo controlled studies, patients entered the study during a relapse, i.e. they had active disease\(^1\). On average, there was a modest beneficial effect for weight gain and other clinical measures. Also, there was a reduction in steroid use in the fish oil group, but not the control groups and although it was not consistent, some patients greatly reduced steroids and showed large improvements\(^2\). The investigators from two studies acknowledged the potential for benefits through decreasing the side effects of long-term steroid use\(^2\). In another study where patients entered the trial during remission, i.e. the disease was not active, fish oil use delayed the time to relapse\(^4\).

**Crohn’s Disease**

**Crohn’s Disease and Fish Oil**
- no effect in active disease
- for disease in remission, the results are equivocal

In two double-blind, placebo-controlled studies with fish oil in Crohn’s disease, fish oil had no effect on clinically active disease\(^3\) and did not prolong remission when disease was inactive\(^5\). However, in a third study, where patients entered the trial during remission, there was a striking beneficial effect of fish oil in decreasing the relapse rate\(^6\). The latter study used an enteric-coated fish oil preparation which achieves higher tissue omega-3 levels than other fish oils. Also, patients had low-grade disease activity by biochemical measures even though they were in clinical remission and this may have been a favourable starting point for the fish oil effects to be manifest\(^7\).

**Conclusion**
- some patients with ulcerative colitis may benefit from dietary omega-3 fats, but the effects overall will be modest
- omega-3 fats may prolong remissions in Crohn’s disease
- because diarrhoea is a problem for all of these patients, common food items containing omega-3 fats will be more practical than use of fish oil capsules

References
Chapter 13. Psoriasis

What is it?

Psoriasis is a disease characterised by patches of red, scaly skin. Against this common background, psoriasis may take a number of forms, e.g. plaque, guttate (drop-like), and pustular. It can be mild, when treatment with topical agents may be sufficient. In more severe disease, treatment involves oral drugs such as the retinoids and cyclosporin A. These agents have potentially serious side effects. About 15% of psoriasis sufferers have an arthritis that is distinctive to psoriasis.

Evidence for Effects of Omega-3 Fats

Greenland eskimos (Inuits) who consume their traditional diet, which is very high in omega-3 fats, have a greatly reduced incidence of psoriasis\(^1\). While this is consistent with a preventive effect of omega-3 fats, genetic factors may be involved also because genetics is a determinant for the occurrence of psoriasis.

In patients who already have psoriasis, fish oil supplementation has shown a beneficial effect in two out of five double-blind placebo controlled trials\(^2,3\). There were no benefits demonstrated in the other three trials\(^3-5\). The reasons for this discrepancy probably relate to the different trial designs. In one of the two studies which showed a benefit, fish oil was used in conjunction with ultra-violet radiation therapy\(^3\). In all other studies, no other therapy apart from topical creams, was used\(^2,4-6\). This may be significant because in an open-label study, benefits were only seen when fish oil was used with ultra-violet radiation\(^7\). Another reason for discrepant findings may be that different types of psoriasis respond differently. For example, no effect of fish oil was seen in patients with plaque-type psoriasis, but a dramatic improvement was seen with pustular psoriasis\(^7\).

Severe Psoriasis and Omega-3 Fats

Two studies with patients hospitalised for severe psoriasis merit attention. In a randomised, double-blind, placebo-controlled design, omega-3 or omega-6 fats were infused intravenously. This generates high blood levels of these fats very quickly. The omega-3 fats provided large and rapid benefits compared with omega-6 fats, and the effects were larger with guttate-type psoriasis compared with plaque-type psoriasis\(^8,9\).
Omega-3 Fats Limit Drug Side Effects

Fish oil has been useful in reducing the elevated blood triglyceride levels which arise from use of oral retinoids\textsuperscript{[1,11]}. Also, fish oil can reduce the adverse effects on kidney function which arise from use of cyclosporin A\textsuperscript{[12]}.

Summary and Conclusions

Omega-3 fats appear to be useful, particularly in certain types of psoriasis. More research is needed in order to predict who might benefit the most. Once the psoriasis is severe enough to need drugs, such as the retinoids and cyclosporin A, omega-3 rich diets may be useful in limiting the potentially serious side effects of these drugs.

References

There is no argument that recommendations should be based on evidence. How does this work at a practical level in nutritional research?

Drugs versus Food and the ‘Gold-standard’

Once ingested, some fats can have biological effects similar to drugs, i.e. they can have pharmacological activity. For example, omega-3 fats can inhibit production of the inflammatory mediator, prostaglandin $E_2$. This is an activity the same as that of all non-steroidal anti-inflammatory drugs. However, when one comes to examine the evidence for health benefits of fats, the type of evidence is usually quite different from that in the drug regulatory environment.

In Australia, health claims can be made for new drugs after they have been accepted for registration with the Therapeutic Goods Administration (TGA). The TGA allows such claims after examining the evidence which, invariably, comes from double-blind comparator-controlled clinical trials. This type of evidence is considered the ‘gold-standard’ because it can show that a drug causes an effect.

For dietary fats and other nutrients, this type of trial is the exception rather than the rule. There are several reasons for this.

Firstly, large scale double-blind comparator-controlled clinical trials are expensive. With the benefits deriving from patent protection as occurs with drugs, the pharmaceutical industry can conduct such trials. However, foods in general are not patentable and without patent protection, it is not practical for the food industry to conduct these trials.

Secondly, dietary interventions are complex. Unlike the simple provision of a drug to be taken in fixed amounts daily, it is difficult to change one aspect of a diet without changing others and often, a large change in daily habits is necessary.

Despite all of these impediments, there have been dietary intervention studies in which different groups of subjects deliberately received an intervention to change their dietary fat intake. In the heart disease area, the DART study and the Lyon Diet-Heart Study are both examples where dietary fat was altered through foods. In these cases, omega-3 fat intake was increased using fish or using canola oil. Also in heart disease, the GISSI

* The cost for bringing a new drug from discovery to successful registration and marketing is in the range of 300 million to 600 million USD.
study is an example where dietary fat intake was altered by providing a dietary omega-3 supplement in capsules. In rheumatoid arthritis, much of the evidence for benefits of omega-3 fats comes from double-blind placebo-controlled studies where the omega-3 fats were provided in capsules as supplements to the usual diet. Dietary intervention studies such as these are the types of studies that provide high standards of evidence that a clinical effect is the result of the dietary intervention or change.

Standards of Evidence – Epidemiological

This type of evidence derives from ecological (between countries), case-control, cross-sectional and cohort studies, as described in the chapter ‘Heart Disease’. While associations between fat intakes and states of health may be revealed, these remain associations only. Unlike dietary intervention studies which are capable of showing that ingestion of a certain dietary fat leads to some clinical effect, epidemiological studies can only suggest causality. Nevertheless, there are systematised criteria which do allow causality to be inferred based on results from epidemiological research.

Nutrition Recommendations are Based on the Congruence of different Types of Evidence

Due to the relative scarcity of controlled intervention studies, dietary recommendations are usually based on informed judgements that the cumulative evidence arising from various types of studies is sufficiently congruent to be conclusive. While judgements can be debated, this is the system most used in nutritional research.

Although this system may appear less robust than total reliance on controlled clinical trials, it should be remembered that epidemiological evidence alone has provided the link between smoking and lung cancer, between folate and neural tube defects, and between asbestos mining and mesothelioma. It should also be noted that double-blind placebo-controlled drug studies can be poor predictors of the effectiveness of the drug in everyday clinical practice. This is true particularly with anti-rheumatic drugs, all of which have emerged through the standard clinical trial route.

Keeping all of this in mind, what picture emerges from the fore-going review of dietary fats, health and disease? How does one put it all together?

References

Chapter 15. Dietary Fats and Health – the Conclusions

Total Fat Intake

With regard to total fat intake, the level in Australia has fallen from 37% of dietary energy in the mid-1980’s to 33% of dietary energy in the late 1990’s1,2. This is the midpoint of the target range nominated by the Nutrition Taskforce of the Better Health Commission and the American Heart Association3,4. There is no reason apparent for recommending that it be lowered further. Further decreases are unlikely to suppress the incidence of new cases of obesity or heart disease. Also, future recommendations for treatment of diabetes Type II may include fat intakes around these levels.

There may even be a negative aspect to recommending lower total fat intakes for control of blood cholesterol levels, for example. This is because the decreased palatability and acceptability of very low fat diets can lead frequently to long-term failure4. The same ends can be achieved with regard to cholesterol lowering by substituting monounsaturated or omega-3 or omega-6 polyunsaturated fats, for saturated fats. To recommend diets which are unnecessarily difficult may discourage patients and reduce their commitment to other important lifestyle changes such as smoking cessation and increased exercise.

Omega-3 Fat Intake

The evidence strongly supports the health benefits of omega-3 fats as preventives and ‘treatments’ for heart disease and rheumatoid arthritis, and there is some evidence pointing to benefits in prevention and ‘treatment’ of psoriasis, inflammatory bowel disease and diabetes Type II. While the word ‘treatment’ suggests a prescriptive and singular course of therapeutic action, the role of omega-3 fats will frequently be as adjuncts to drug treatments, e.g. alongside aspirin and cholesterol-lowering agents in heart disease, alongside anti-inflammatory drugs in arthritis, alleviating hypertriglyceridaemia in diabetes which is not addressed by hypoglycaemic agents, and alleviating the renal side effects of drug treatments in psoriasis.

The vexed question is ‘How much is necessary for benefits?’ It is problematic due to lack of data in some areas and because vegetable oils and fish oils contain different omega-3 fats. The omega-3 fat from vegetable oil is α-LNA and the omega-3 fats from fish oil are EPA and DHA. α-LNA may confer benefits in its own right or it may confer benefits only after being converted to EPA. This is unknown.
With regard to preventive effects, the finding of protection from heart disease with 1 to 2 fish meals / week has probably guided the various recommendations on long chain omega-3 fats (EPA + DHA). Amongst the international recommendations are:

- at least 200 mg / day EPA+DHA\(^5\)
- population average should be 0.5% of dietary energy (1 to 1.2g) and safe up to 2% of dietary energy (4 to 5g / day)\(^6\)
- at least 220mg EPA and 220mg DHA / day\(^7\)

**Fish Oil-Derived Omega-3 Fats**
- for disease prevention, international recommendations range from 0.2 to 1.2 g / day
- in treatment of heart disease and rheumatoid arthritis, benefits were seen with 0.3 to 2 g / day (and higher)

With regard to treatment effects, there is less information as there is a paucity of dose response studies. In the rheumatoid arthritis studies with fish oil, some benefits were seen with doses of 1-2 g / day and there were dose responses evident with higher doses\(^8\). It must be remembered that these studies were conducted under unfavourable circumstances and thus benefits may be seen at lower doses (see Arthritis chapter). In the GISSI study for heart disease, benefits were seen with 1g / day of long chain omega-3 fats and in the DART study, benefits were seen with 300 mg / day of EPA (see Heart Disease chapter).

For the vegetable derived omega-3 fat, \(\alpha\)-LNA, it has been recommended generally that intakes should be at least 1% of dietary energy. Among the international recommendations are:

- 2 g/day or 1% of dietary energy\(^5\)
- population average should be 1% (2 to 2.4 g) and safe up to 2.5% of dietary energy (5 to 6 g/day)\(^6\)
- 2.2 g/day or 1% of dietary energy\(^7\)

**Vegetable Oil-Derived Omega-3 Fats**
- international recommendations are for intakes around 2g/day with safe levels to 6g/day
- intakes of 2g/day will mean an approximate 4-fold increase in intake for many Australians

An amount of 1% of dietary energy (around 2g / day) could represent an approximate 4-fold increase for many Australians. This recommendation is well justified since these levels were associated with substantial benefits for heart disease in the Lyon Diet-Heart Study, the Nurses Health Study and the Health Professionals Study\(^9-11\) (see Heart Disease Chapter).

**Omega-3 Fats and Health – Summary**
- intake of vegetable oil omega-3 fats to be at least 2 g/day
- intake of fish oil omega-3 fats to be at least 0.2 g/day and, depending on whether preventive or treatment effects are sought, can be 1-2 g/day

Overall, there are benefits to be derived from an increase in omega-3 fat intake from vegetable oil and from fish or fish oil. \(\alpha\)-LNA intake at 2 – 3g / day and fish oil–derived omega 3 fats at a minimum of 200 mg / day are reasonable targets. At these levels, there are no known safety issues.
Higher levels may be needed for benefits in specific conditions such as rheumatoid arthritis.

At the very high levels of omega-3 intake seen in the traditional Greenland eskimo (Inuit) diet (approximately 7 g / day EPA + DHA)\(^{12}\), there was a bleeding tendency and an increased incidence of haemorrhagic strokes\(^ {13}\). Therefore, it is advisable to avoid particularly high levels of omega-3 intake and to take special caution if untreated hypertension or bleeding disorders are present. Fortunately, it is extremely difficult to even approach the levels of intake seen in the traditional Inuit diet, and for practical purposes, omega-3 fats can be considered to be quite safe.*

**Omega-6 Fat Intake**

With the increased use of polyunsaturated oils and margarines from around 1970 onwards, there has been an unprecedented increase in the intake of omega-6 fats, principally linoleic acid, to levels of around 7% of dietary energy. As stated by the NHMRC Working Party into Polyunsaturated Fats in the Australian Diet:

> “There is no experience of any population that has consumed large amounts of linoleic acid over long periods”.\(^ 1\)

However, linoleic acid has now been consumed at unprecedented high levels for at least 30 years by large numbers of people with no apparent ill effects. Despite this, there is no reason apparent for recommending an increase in linoleic acid intake.

**Omega-6 Fats and Health Benefits**

- a substantial decrease in heart disease has occurred during the period of increased omega-6 fat intake
- intake of saturated fats has also declined during this period as they were replaced by omega-6 fats

Animal studies suggested linoleic acid promoted cancer formation (see Breast and Colon Cancer chapters), but this has not been borne out in human experience. During the last few decades, mortality from heart disease has decreased during the period when linoleic acid intake has increased, suggesting health benefits for heart disease. In general, linoleic acid has replaced saturated fats as polyunsaturated margarine was substituted for butter and vegetable oil was substituted for tallow\(^ 1\). Thus, the benefits for heart disease derive from a decline in saturated fat intake as much as they do from an increase in omega-6 polyunsaturated fat intake.
From this viewpoint, there appears to be no reason for recommending any change in linoleic acid intake. But, when it is considered that linoleic acid can decrease the metabolism and the tissue levels of omega-3 fats, a decrease in linoleic acid will allow more efficient use of short-chain and long-chain dietary omega-3 fats. Australian dietary surveys have not published omega-3 and omega-6 fat intake data. However, the ratio of dietary omega-3 / omega-6 was estimated in 1992 to be as low as 1:30. In dietary studies with free-living subjects using sunflower oil products, the omega-3 / omega-6 ratio was 1:20.

The ratio derived from UK Dept of Health recommendations is 1:6 with the recommendation for linoleic acid intake at 6% of dietary energy. A Working Party in the US has recommended an upper limit of linoleic acid intake of 3% of dietary energy. This would represent an approximate halving of current intake levels. While one can debate what that final intake level should be, it is clear that the current dietary omega-3 / omega-6 ratio is low and that it should be increased. The most efficient way to increase this ratio is to increase omega-3 intake and decrease omega-6 intake.

Monounsaturated Fats

- probably have no ‘special benefits’
- as a replacement for saturates, they are equivalent to omega-6 fats with regard to cholesterol-lowering
- they are not antagonistic to omega-3 fats
- therefore, they are preferred as a replacement for saturated fats

It is well recognised that Mediterranean diets rich in monounsaturated fats are associated with low levels of heart disease, these diets are also low in saturated fats and have some omega-3 fat content. With regard to biological plausibility of a ‘special effect’ for monounsaturated fats, none have emerged. However, they have a very useful role in a healthy diet. With regard to cholesterol lowering, they are equivalent to the other unsaturated fats as a replacement for saturates (see Heart Disease chapter). In addition, they are not antagonists of omega-3 tissue levels or metabolism. Therefore, they are excellent replacements for dietary omega-6 fats as well as saturated fats.

Saturated Fats

There is little argument here. Saturated fat should be reduced to levels less than 10% of dietary energy.
1. Omega-3 fat intake should be increased. Vegetable oil omega-3 fats should be at least 2 –3 g/day and fish oil omega-3 fats should be at least 200 mg/day.

2. Omega-6 fat intake should be decreased in order to more efficiently use the dietary omega-3 fats.

3. Monounsaturated fats are good replacements for saturated fats and omega-6 fats.

4. Saturated fats should be decreased to < 10% of dietary energy.

Summary

For practical on dietary ingredients which can be used to achieve these changes, including menu plans and recipes, please refer to other sections within the Gold’n Canola website, particularly the ‘Health, Diet and You’ book.

References


“Menhaden oil has been granted GRAS (Generally Regarded As Safe) status by the US Food and Drug Administration for usage up to 3 g/day which would supply approximately 1 g/day of EPA+DHA.”