A new approach for nutrition communication within IDF?

This first issue of Nutrition Newsletter represents a new approach to opening up a channel for discussions on nutritional matters of relevance to the dairy industry within IDF. The present issue comprises the lectures presented during the Nutrition Week which was organized in Uppsala, Sweden, in May 1992. It is intended that the various groups involved in nutrition matters within IDF should be able to use the newsletter for publication of their reports.

According to the terms of reference of Group F38, "Education in Nutrition", one of the purposes of this group is to stimulate the exchange of information within the field of human nutrition. Up until now Nutrition Weeks have been organized in order to discuss relevant topics, but it has been suggested that there should also be some form of newsletter in order to spread the message as relatively few people may be able to attend the Nutrition Weeks.

At present there are a number of expert groups within IDF’s Commission F who deal with nutritional matters:

- F20 Role of cultured and culture-containing dairy foods in health (Chairperson: E. Renner);
- F22 Significance of milk in the diet from the angle of calcium metabolism (Rapporteur: G. Shaafsma);
- F24 Milk protein and nutrition (Chairperson C. Barth);
- F37 Milk lipids in the diet and health (Rapporteur: M. Gurr);
- F38 Education in nutrition (Chairperson: L. Hambraeus).

In order to further stimulate and intensify the discussion on nutritional matters of relevance for the dairy industry a special Nutrition Coordination Group has been established. Although there has been some debate regarding the terms of reference and members of the coordination group, it was intended to comprise nutrition experts within the IDF organization in order to discuss and give advice on nutritional matters to other groups and commissions. It was recommended that the nutrition expert within Commission F should act as chairperson of the Nutrition Coordination Group since it would belong to Commission F. However, for various reasons, I, who recently retired as nutrition expert and permanent member of Commission F, was asked to act as chairperson for the next 2-year period.

It is hoped that the establishment of a Nutrition Newsletter will help to enhance the exchange of information within the field of nutrition.

Leif Hambraeus, MD, Dr Med. Sci.
Professor of Human Nutrition, University of Uppsala, Sweden
Chairperson of Group F38 and Nutrition Coordination Group
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5 Calcium balance in adolescent girls, relative importance of genetic and nutritional factors (V. Minkovic, USA)  
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1. PRESENT SITUATION REGARDING LEGISLATION ON THE USE OF HEALTH PROMOTION IN MARKETING OF FOOD ITEMS

1. HEALTH CLAIMS IN THE MARKETING OF FOOD PRODUCTS - SWEDISH EXPERIENCES

Nils-Georg Asp
Ph. D. MD, Professor of Applied Nutrition, Lund University Managing Director, The Swedish Nutrition Foundation, Sweden

Nutrition claims represent an evaluation of a food from the health point of view based, for instance, on the nutritional content of the product. Drugs are products designed to prevent, diagnose, alleviate or cure diseases or their symptoms. Consequently, until recently foods marketed with health claims were classified as drugs, as is still the case in other countries.

In 1989, the pharmaceuticals department of the National Board of Health and Welfare (now reorganized as the Medical Products Agency) stopped applying the legislation on drugs to products that are basically foods. A condition for this is that marketing does not contain specific instructions for dosage or other information that may only be used with respect to pharmaceutical preparations.

In recognition of the need to avoid misleading or false information when using health claims for the purpose of marketing food products, the food industry has prepared a programme of action in consultation with the National Food Administration and the National Board for Consumer Policies.

The programme is in four parts:

(A) A summary of generally accepted principles, limiting the use of health claims to generally recognized and well-documented causal connections between food and health.

(B) An expert on nutritional matters at the Swedish Nutrition Foundation will be available for giving advice regarding claims planned to be used for marketing purposes.

(C) The trade organizations and companies involved and the Swedish Nutrition Foundation will arrange seminars and courses for marketers.

(D) An evaluation of the programme of action is planned.

Two levels of health claims can be differentiated:

(1) Information on, for example, the relationship between saturated fat intake, blood cholesterol and cardiovascular disease, in connection with information on the total and saturated fat content of the product.

(2) Claims stating that a certain product is effective, for instance, in reducing blood cholesterol.

The programme relates, in the first place, to the first kind of claims. The second type of claims must only be used if they can be supported by well-documented, published studies of the product, or another product of identical composition. Prior consultation with the Medical Products Agency is recommended.

The following connections between physiological conditions and diet can be considered well established and will therefore form a basis for decisions on the acceptability of health claims in the marketing of food products.

- Obesity - energy content.
- Cholesterol level in the blood - content of saturated fats.
- Blood pressure - content of salt (sodium chloride).
- Atherosclerosis - reduction of high blood cholesterol level and blood pressure.
- Constipation - dietary fibre content.
- Osteoporosis - calcium content.
- Caries - sucrose and other easily fermentable carbohydrates in products that are frequently eaten between meals.
- Iron deficiency - iron content.

A low cholesterol should not be used as a nutritional or health claim by itself.

General statements such as “good for the bones”, “good for the heart” and “good for the stomach” should be used with caution and only when accompanied by explanatory text.

No claims about the connection between diet and cancer should be made until further notice.

Generally, the programme states that claims should be presented in such a way as to enhance public confidence in food products and the food industry. Therefore, the use of health claims must not result in loss of confidence in food products, whether in individual cases or in general.

The programme has now been in action for one and a half years. Producers have successively adjusted to the programme, although clear cases of misuse have also occurred. Decision about marketing activities misusing health claims seem to have been taken by product managers without company management approval. There is a need for more information activities about the programme.

Producers of fish products have felt the programme too restrictive in not allowing specific claims about fish fat. Also regarding fermented dairy products there is a clear wish to use more extensive claims than allowed at present.

The programme “Health claims in the marketing of food products” can be obtained from SNF - The Swedish Nutrition Foundation, Ideon, S-223 70 Lund, Sweden, phone +46 46 18 2280, fax +46 46 18 2281.

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2. PRESENT SITUATION REGARDING LEGISLATION ON THE USE OF HEALTH PROMOTION IN MARKETING OF FOOD ITEMS - USA

Emerita Alcantara,
Ph. D., R.D. Dairy Council of Wisconsin, Westmont, IL, USA

The world of food is changing rapidly due to technological advances and changes in policy. International organizations are shifting their focus from worldwide food supply to an increasingly prevalent problem - "increases in the noncommunicable diseases related to the overconsumption of food". This emphasis has initiated policy changes worldwide in the use of nutrient and health claims. In addition, a number of changes have been made in the US regarding food and ingredient labelling as a result of the Nutrition Labelling and Education Act (NLEA).

The NLEA was a major form of legislation passed by the United States Congress in 1990 and was the spark for labelling reform in the US. The US Food and Drug Administration (FDA) is the government agency responsible for enforcing the NLEA provisions. To date, FDA has published 25 proposed regulations covering the different aspects of NLEA. A total of three proposals were published last summer and the other 22 this past November. Together, they total close to 600 pages in the Federal Register.

Since the publication of the NLEA proposals on 27 November 1991, a great deal has occurred. On 30 and 31 January 1992, a public hearing was held to present views regarding the FDA labelling proposals. A number of key issues were presented, including a draft regarding the "alternative approach" for comparative claims. In addition to the public hearing, approximately 40,000 written comments from interested parties were sent to the FDA by the 25 February 1992 deadline.

On 8 November 1992, FDA is scheduled to release the labelling regulations in their final form. If the FDA fails to issue final rules on or prior to this date, the proposed rules published in November of 1991 will be considered law. All food manufacturers must be in compliance with the new regulations by 8 May 1993. However, given the amount of concern and controversy regarding this date, and the fact that the US Department of Agriculture, who regulates labelling for meat and poultry products, has extended their labelling implementation date to May 1994, there is discussion as to whether FDA may extend the deadline as well.

The NLEA changes, in a major way, the manner in which food products will be labelled and marketed. Dairy manufacturers and marketers will have to conform to the required changes in the following areas: nutrition labelling, ingredient labelling, nutrient claims, and health claims. While these changes represent challenges, a number of marketing opportunities exist to promote the benefits of dairy products.

NUTRITION AND INGREDIENT LABELLING

Currently, a product must carry a nutrition label only if a nutrition claim has been made or a nutrient has been added. As a result of the NLEA, all FDA-regulated retail products must be nutrition labelled unless otherwise exempted (for example, foods served in restaurants, foods in small packages, etc.). All foods will also require full ingredient labelling as well. Originally, standardized products were exempt from this requirement and only optional ingredients needed to be listed.

The proposed nutrition label will differ due to some new terminology, added information, and a change in emphasis. Additional items including "calories from total fat", "saturated fat", "cholesterol", "sugars", "complex carbohydrate" and "dietary fibre" have been proposed as MANDATORY on the nutrition label. Nutrients that were once MANDATORY, like thiamin, riboflavin, and niacin, are being proposed as VOLUNTARY declarations. The "Percent US RDA" may also change to "Percent Daily Value". Another section called the "Nutrition Profile" has also been added.

NUTRIENT CONTENT CLAIMS

As a result of the NLEA, nutrient content claims such as "low fat", "fat free", "percent fat free", "reduced", "light", "high", and "source of" have to follow FDA definitions. In addition, FDA is proposing to require certain referral and/or disclosure statements to accompany nutrient content claims.

The FDA proposed an "alternative approach" to that presented in its original November 1991 proposal regarding the comparative terms, "reduced" and "less". The alternative approach defines "reduced", "less", "fewer" and "lower" as synonymous terms. In addition, no minimum percentage reduction would be required for a food to bear a comparative claim, but the amount of the nutrient must differ from the reference food by an amount exceeding "low" for that nutrient. For example, for a product to be labelled "reduced fat", it must differ from the reference food by an amount exceeding 3g per serving.

In using nutrient content claims in conjunction with a standardized term in naming a substitute food, the FDA has adopted in its proposal, general rules permitting the naming of a substitute food using a nutrient descriptor combined with a standard: (1) the nutrient term used must follow FDA definition, (2) the food must not be nutritionally inferior, (3) the food must also have similar performance characteristics - if not, a statement must be present stating the differences, and (4) optional ingredients could not be substituted unless allowed according to standard.

HEALTH CLAIMS

The one health claim most important to the dairy industry is the calcium and osteoporosis claim. To qualify for a claim: (1) the product must provide at least 20% of the Reference Daily Intake (RDI) for calcium - 180 mg, (2) the calcium must be bioavailable, (3) the phosphorus must not exceed the calcium by weight, and (4) it must not exceed the disqualifying levels of other nutrients that increase the
risk of other diet-related diseases. The proposed disqualifying levels are: 11.5 g fat, 4.0 g saturated fat, 45 mg cholesterol and 360 mg sodium per serving and per 100 g. These disqualifying levels have been questioned by some groups because they seem to foster a "good" food/ "bad" food concept, and are contrary to nutrition principles that individual food should be evaluated in the context of the total diet rather than in isolation.

If a product is able to make a claim it must bear a statement addressing: (1) that adequate calcium intake is not the only recognized risk in osteoporosis, (2) some populations are more at risk for developing osteoporosis, (3) that adequate calcium in life is linked to reduced risk of osteoporosis through optimizing peak bone mass during adolescence and early adulthood, (4) the degree of reduced risk should not be quantified, and (5) a total dietary intake greater than 200% of the RDI has no further benefit.

MARKETING OPPORTUNITIES
A number of claims can be made on dairy products. There is no other food group that supplies as much bioavailable calcium as the dairy group. A "High in calcium" claim can be made for products such as fluid milk, nonfat or lowfat yogurt, and cheeses such as Cheddar, Monterey, Provolone and Swiss. Other nutrients that can be highlighted depending on the product include vitamin A, vitamin D, protein, riboflavin, and phosphorus. A number of dairy products such as fluid milk, certain yogurts and ice cream can qualify for the "low sodium" claim. Others will be able to make a "cholesterol free" (for example, nonfat cottage cheese) and "fat free" (skim milk) claim. In addition to nutritional qualities, dairy products also are superior in taste and product quality.

In summary, the NLEA proposals will definitely influence how foods will be labelled. The product label of the future will have a considerably longer nutrition label. There will be more verbiage than before because of required disclosure and referral statements. All FDA-regulated products will require full nutrition and ingredient labelling and use nutrient and health claims according to FDA definition. However, given these restrictions, marketers, manufacturers, and advertisers should be able to work within the framework of these guidelines to produce and effectively market foods to the consumer.

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3. USE OF HEALTH PROMOTION IN MARKETING FINLAND

Ulla-Marja Urho
The Dairy Council of Finland, Vuorikatu 22 A 11, SF-00100 Helsinki, Finland

Throughout history, health consciousness has guided our choice of what we eat. From a basically biological and organoleptic selection, cultural man has acquired knowledge and wisdom to help him choose his food. Today we are surrounded by experts. The complexity of health information today becomes evident in the contradictory attitudes consumers have towards food. What is most important - taste, a plentiful supply of food, or health? However, when the food is purchased its freshness, price and purity seem to mean more to the consumer than whether or not it is healthy.

According to a study by the International Food and Drink Monitor, Finns are more interested in their well-being and their diet than the average European. In the 1980s, Finnish eating habits shifted radically towards recommended nutritional principles; consumption of vegetables doubled and consumption of fat dropped by two-thirds.

All the same, when women do the shopping, as is still mostly the case even in Finland, it is the freshness and purity of a basic food that are even more important than its health aspect.

In order to follow a healthy diet, one must have information about food. Acquiring and applying information requires effort and knowledge of the basic principles of nutrition. In the consumer debate in Finland, the responsibility for raising the level of nutritional knowledge and food awareness has been increasingly left to the food industry. The authorities have expressed great concern, particularly over food labels.

The nutrition message can be transferred through different channels - packaging, advertising, school education and counselling and informative education of consumers. According to a recent study it is clear that in food advertisements consumers are interested in the prices of products and in recipes. Hints for preparing food are more sought after than further information on the nutritional content of food. An interview survey conducted by the Food Bureau in 1991 shows that

- 90% of consumers say they read food labels
- 75% of consumers read the labelling always or very frequently, and usually prior to purchase. The most commonly consulted entry is the sell-by date, which is checked by 80% of the consumers. Contents, ingredients, instructions for storage and use, and additives are examined by slightly less than half of all consumers. Every fourth consumer reads the nutritional information, more women than men do this. People paid less attention to food package labelling now than they did in a study conducted in 1985.

The nutritional information is the only entry that is considered more important now than it was 6 years ago. Over 80% of consumers consider nutritional information necessary, most of them because of the indication of fat content. Other reasons cited include monitoring vitamin and mineral content, avoiding sugar and comparing different products.

FOOD LABELLING
From the beginning of June 1991, new regulations concerning food labelling came into effect in Finland. When giving nutritional information the following points must be adhered to.

(1) The information must be based on laboratory studies. Values for energy-producing substances (protein, fat, carbohydrates and energy content) can be in table form.

(2) The amounts of protein, fat, carbohydrates and the energy content must always be stated.

(3) If a particular nutrient is emphasized on the package, the amount of this nutrient must also be shown, for instance:
- high in fibre - the amount of fibre
- low fat - the amount of fat
- high in vitamin C - the amount of vitamin C.

(4) The term "light diet" may be used if the energy content of a product is at least 30% less than that of a comparable normal food product.

(5) The amounts of sugar and fibre may be shown.

(6) The amounts of fatty acids and cholesterol may be shown as:
- saturated fats
- monounsaturated fats
- polyunsaturated fats
- or simply as saturated fats.

(7) If the cholesterol amount is shown, the above breakdown of fats and all other nutritional information should also be shown.

(8) Furthermore, the amounts of the following minerals and minerals may be shown: A, D, C, B1, B2, B6, B12, niacin, folic acid, potassium, magnesium, iron, zinc, iodine, copper, manganese and selenium.

(9) The amount of vitamins and minerals may be compared with the recommended daily dose - for instance, three glasses of milk (6 dl) provide about 70% of the recommended daily dose of vitamin B.

The following information is compulsory:

(1) Certain nutritional information must always be shown. For children's foods, the energy content and the amounts of protein, fat and carbohydrates must always be shown.

(2) The packages of milk, cream, sour milk products and yoghurt must show the fat content. The packages of cheeses, milk powder, margarines, butter and vegetable oil mixes and all sausages except salami-type sausages must show the fat content.

The following information is optional:

The declaration of nutritional content, that is, of energy content and the amounts of protein, fat, carbohydrates, vitamins and minerals. Nutritional information becomes compulsory if the package bears a nutrition-related claim such as "low-fat", "light-diet", high-fibre" etc.
NUTRITIONAL CLAIMS IN MILK MARKETING

The high nutritional value of milk has been recognized for a long time and used in milk marketing for some 60 years. In Finland, "THE MILK PROPAGANDA OFFICE" used health as a marketing device for milk way back in the 1930s. The themes then were snacks for sportsmen and school children.

I think the message has gone home; consumption of all liquid milk products is higher in Finland than in any other country, about 209 l per person in 1991, and milk itself accounted for 166 l of this total.

What about health claims and milk advertising today? Nutritional claims feature in the most recent advertising campaign too. It consists of:

- nutritional information and bulletins on milk cartons;
- commercial advertisements on TV and in magazines;
- information material distributed to nutrition specialists and through them to consumers;
- consumer publicity

What is the milk message?

The main target group for milk information are the adolescents. Advertising directed at them is based on the MILK ENERGY campaign. The health message is aimed mainly at adults. They are the gatekeepers.

Nutritional claims were strongly emphasized in milk marketing between 1962 and 1997. They are now making a comeback:

Milk works for grown-ups too - have you ever tried a glass of milk for stress, insomnia or a hangover? Many people say milk has a soothing effect.

Milk goes to work on bones, nails and teeth. It contains proteins, vitamins and minerals useful for the skin and the hair.

How about a glass of milk?

How has this come about?

The penetration, credibility and effect of the nutritional message on milk were maintained amongst both consumers and nutrition experts throughout the 1980s. A survey conducted from 1968 to 1988 showed that over two-thirds of all consumers agree that:

- milk is a natural vitamin and mineral drink;
- milk contains beneficial protein;
- milk reduces the risk of osteoporosis;
- milk is important for children’s teeth.

The points least agreed with were:

- the calcium in milk reduces high blood pressure;
- the recommended daily dose of milk is three glasses;
- milk is not fattening.

Consumers were also asked to estimate the effect of these claims on milk consumption. One-third of consumers thought that the claims of calcium strengthening children’s teeth and reducing the risk of osteoporosis had influenced their consumption of milk. The other claims were given less attention.

Nutrition experts have stronger views about milk and education. The recommended daily allowance of milk products (three glasses) has been proved, and the correlation between calcium in milk and a reduced risk of osteoporosis has been established. However, the experts consider milkfat to be a greater hazard than consumers do. The negative arguments concerning milkfat detract from the positive nutritional message of milk.

In Finland, as elsewhere, doctors and healthcare staff are much more likely to be believed in nutritional counseling than printed matter or advertisements, so healthcare staff are our main target group at the Dairy Council.

The following conclusions are my own and they can be argued about:

Nutritional information on food packages is demanded by the authorities and the consumers. However, little attention is paid to them in comparison to freshness, price and ingredients.

The detailed nutritional information is read by nutrition experts and by people following special diets (salt, sugar and lactose-free; high-fibre, cholesterol-free etc.).

Calcium is the best known and most positively perceived nutrition message in milk. It may not be worthwhile emphasizing the function of other nutrients in sales promotion.

In the wild chaos of advertising, complicated nutritional messages get lost. Even the most health-conscious consumer will not systematically change over to healthy alternatives.

Uniform nutritional information on food packages makes for easier comparison and less complicated foreign distribution.

Changes in nutritional information and health claims are to be expected in Finland at least in two respects:

- consumers would like to see packages furnished with symbols that would make selection easier (the authorities are awaiting results concerning key symbols already in use in Sweden);
- there are no directives on the use of health claims. Should it be acceptable for food to be marketed as promoting digestion, lowering blood pressure, reducing osteoporosis, preventing coronary disease or reducing cholesterol?

The last of these has been used liberally by the magazine industry on packaging, without the authorities considering the question.

- It is true that the health aspect is not in itself a selling point. Taste, convenience and the new organic foods are more effective pressures when consumers make their food choices.

WHAT IS IMPORTANT WHEN YOU BUY BASIC FRESH FOOD?

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Suomen Gallup 1989

WHAT DO CONSUMERS LOOK FOR ON FOOD ADVERTISEMENTS?

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Suomen Gallup 1990
THE NORDIC COUNTRIES, COMPARED TO EUROPE

WELL BEING
LONG TERM VISION OF THE IMPORTANCE OF FOOD AND BEVERAGES, COUPLED WITH AN ACTIVE SEARCH FOR PHYSICAL, EMOTIONAL AND INTELLECTUAL BALANCE.
• DIETETIC CONCERN
• ACTIVE COMPENSATION
• BALANCED DIET

WHY IS IT NECESSARY TO STATE THE NUTRIENT CONTENT?

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Food and Drink Monitor
RISK, 1991

WHAT DO YOU THINK ABOUT THESE STATEMENTS?

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<td>Milk has no additives</td>
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<td>Milk calcium lowers your blood cholesterol</td>
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<td>Milk calcium prevents osteoporosis</td>
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<td>Milk doesn't cause ear infection</td>
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<td>Grown ups need milk too</td>
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<tr>
<td>You should drink three glasses of milk a day</td>
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AGREE = completely or in principle
DISAGREE = totally or mainly

Consumer attitudes 1998
University of Helsinki,
Department of Nutrition

UNDERLYING SOCIAL FORCES IN SOCIETY

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International Food and Drink Monitor, RISK, 1991

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4. THE PRESENT SITUATION REGARDING LEGISLATION ON THE USE OF HEALTH PROMOTION IN THE MARKETING OF FOOD ITEMS IN AUSTRALIA

Lois Shuttleworth
Australian Dairy Corporation, Glen Iris, Victoria 3146, Australia

INTRODUCTION

Australia has had many changes to its food legislation that has affected the health promotion of food items in recent years. This paper introduces some of the most recent and proposed changes to Australia's food legislation affecting health promotion.

The most critical change has been the establishment of a new body responsible for food laws and regulations (known as standards) - the National Food Authority (NFA).

The NFA was introduced because the previous system for the introduction of food standards was criticized for being ineffective. Although it is too early to gauge the effectiveness of the new body, it is anticipated that the NFA will be more streamlined in its procedures with the result that changes to the Food Standards Code will be quicker.

Three areas of responsibility for the NFA outlined in the paper are health messages, product descriptions and food ingredients.

RESPONSIBLE BODY FOR ESTABLISHING FOOD STANDARDS - THE NATIONAL FOOD AUTHORITY (NFA)

Firstly to the new body responsible for establishing food standards, that is laws and regulations relating to food, the National Food Authority, or, as I will refer to throughout this paper, the NFA. The NFA commenced operations in August 1991 and assumed the role previously fulfilled by different government bodies and committees.

Matters of public health remain a state government responsibility while enforcement and administration are handled by local government in each city.

The NFA was formed in response to criticism of the previous procedures used to establish food standards. The previous system was criticized as it:

- was too slow and cumbersome and overly complex, with the average time to introduce a new standard or revise an old one being 5 years;
- reduced the efficiency and competitiveness of the Australian Food Industry;
- inhibited trade within Australia due to a lack of uniformity of food legislation between each of the 7 Australian states, and
- lacked a single body with responsibility for monitoring the system and developing standards.

The newly formed NFA was charged with the following objectives:

- to consider and make recommendations for the development and variation of food standards through the Food Standards Code;
- to provide uniformity in food standards throughout Australia as now any new food standard is automatically adopted by each of the 7 states;
- to promote consistency between domestic and international food requirements;
- to provide interested parties an opportunity to contribute to the development and review of food standards; including holding public hearings, and
- to operate within a minimum number of layers of responsibility.

The NFA involves the public and the food industry in decision making by way of:

- advertising proposed changes to food standards;
- seeking comments regarding proposals from organizations such as The Council of Australian Food Technology Associations and the Food Industry Council of Australia;
- seeking industry consultation, and
- allowing lobbying of NFA committees or council on specific issues.

The NFA is presently working on Revision of the Standards for Vitamins and Minerals, which will have implications for many food groups, including the dairy industry. Amazingly this Standard has been under review for 12 years without resolution.

Briefly, the proposed changes to the Standard will allow vitamins and minerals to be added to foods only on the basis of:

- restoring what has been lost during the processing of the food;
- fortifying foods which are substitutes for other commonly used food (for example, margarine for butter, soy drink for cows milk), and
- fortifying food when there is an identified and proven public health need to do so.

As a result of the proposed new Standard the addition of vitamins and minerals to food will become far more restricted than what exists now.

The proposed new Standard will be consistent with the Codex Alimentarius policy on Vitamins and Minerals.

Draft revised standards have been prepared and are available for public comment. Companies and individuals have until September 1992 to make comment on the draft standard before its review is finalized.

The dairy industry will include in its response to the proposed changes that:

- soy drinks should not be allowed to be fortified with calcium or other essential nutrients so as to imitate the nutritional composition of milk;
- the general fortification of foods has the risk of misrepresenting the value of some foods; for example, sugared children's cereals fortified with vitamins and minerals.

FOOD STANDARDS CODE - SECTION A1(19)

Next I will turn to a section in the Food Standards Code that presently restricts health messages in advertising or messages on food labels. The section requires that food manufacturers cannot promote a product that:
claims or infers a therapeutic effect or the prevention of a disease;
- uses the words "health" or words of similar meaning as a part of the name of the food;
- by word, design or implication could be interpreted as advice of a medical nature;
- names or gives reference to any disease or psychological condition or;
- misrepresents the property or quality of a food.

Enforcement of this section has not been strictly adhered to, with the consequence that some companies have chosen their own interpretation of the section. Part of the reason for this is that local government in the city responsible for prosecution of alleged breaches of the Food Standards Code is known to be resistant to taking legal action unless they are certain of success, as they cannot afford to pay the legal costs if they should lose.

In October 1991, the section was well and truly breached by a milk manufacturer of a reduced fat, high calcium, milk product. The manufacturer launched a television commercial informing the consumer of the risks of osteoporosis, and how their calcium-enriched product could help prevent this disease. Using the Grim Reaper (previously used in Australia for the introduction of the AIDS campaign), the advertisement was violent and designed to shock, and it received strong criticism from the community and the media.

The manufacturer was called to the Media Council, a self-regulating body, to be given an opportunity to defend the television commercial against the claim that it was unduly violent. The advertisement was found to be inappropriate for general viewing and was voluntarily withdrawn by the manufacturer.

An alternative television commercial has since been launched which, although it still mentions the benefits of calcium-enriched milk for strong bones, is more in line with the requirements of the section.

THE PRESENT POSITION ON THE USE OF HEALTH MESSAGES IN THE PROMOTION OF FOOD

Section A1(19) of the Food Standards Code has been criticized for being too restrictive. As a result, a proposal is being considered by the NFA which would allow specific health messages to be used in the promotion of certain foods.

The NFA has prepared the following guidelines in developing the new direction for these health messages:
- the health message must be stated in terms of the nutrient's function and place in the total diet;
- consideration must be given to the potential for conflicting benefits and disadvantages of a nutrient, and
- reference to a disease or physiological condition is acceptable only if the evidence for such a link is proven. The example used is the link between vitamin C and scurvy.

We believe that the connection between calcium and osteoporosis will be interpreted in the same way.

The health messages have been prepared for specific nutrients being calcium, fats, fibre and sodium. The final health messages which are proposed will be trialled for correct interpretation by consumers. These messages would then be incorporated into the Food Standards Code. Such messages would be the only ones permitted for use.

(i) Calcium messages
The proposed health message for calcium will be used only with high calcium content foods where one serving provides at least 300 mg of calcium. Parts of the message will be mandatory while others will be optional.

The parts of the proposed calcium message which must be mentioned are:
- calcium is essential in a balanced diet;
- calcium is vital throughout life for the development and maintenance of strong, healthy bones and teeth, and to keep nerves and muscles functioning, and
- one serving of x g or ml of this product provides y mg of calcium.

The optional message proposed is:
- milk and dairy foods are the major sources of calcium in the Australian diet.

Problems exist with the calcium claims. For example, the average consumer is unlikely to be able to interpret what a "serve" is, let alone what a mg of calcium represents as part of the total daily requirement.

Further to this, the claims are cumbersome and verbose and the uses of the mandatory and optional messages are unclear. For example, do all three mandatory messages have to be used at the one time?

(ii) Fat messages
The fat messages can be used only where not more than 30% of energy is from fat. The proposed health message for fat includes two mandatory claims:
- some fat is essential in every balanced diet, although a diet high in saturated fats has been associated with ill health, whereas a diet low in total fat, especially saturated fat, contributes to good health in adults;
- this food is a low/reduced fat food and contains x g of total fat and y g of saturated fat.

Once again the statements are cumbersome and verbose. The fat claims are not supported by the dairy industry because the first statement focuses inequitably on saturated fats. This statement will be strongly resisted.

These health claims are at least 1-2 years from adoption as they are still under review by the NFA and will be subject to further public scrutiny.

THE PRESENT POSITION ON NUTRIENT CLAIMS IN PRODUCT DESCRIPTIONS OR NAMES

Product descriptions relating to a food being "low in fat" or being "cholesterol free" have been commonplace on labels and in advertising in Australia. Although these are acceptable product statements, there has been growing criticism of such product descriptions as being often inappropriate and confusing to the consumer.

The NFA is now reviewing product claims of "reduced fat", "low fat", "low saturated fat", "saturated fat free", "low in cholesterol" and "cholesterol free".

Draft guidelines have been prepared which set out the conditions each product claim must meet, and are given below.

CLAIMS
Reduced fat
Low fat or Low in fat

CONDITIONS
Minimum reduction in fat from the regular product of 25%
- Less than 3 g of fat per 100 g
- In the case of total meal foods, less than 20% energy from fat.
- Where foods are naturally low in fat, "low fat" is not to be included as part of the product name.
Reduced saturated fat  Minimum reduction in fat from the 
the regular product of 25%.

Low saturated fat  To be less than 1 g of fat per 100 g 
or less than 7% of energy from 
saturated fat.

Low in cholesterol  To be no more than 20 mg/100 g 
and low in saturated fat.

Cholesterol free  To be no more than 3 mg/100 g 
and low in saturated fat.

Agreement on these product claims is still some time 
away as they are at a review stage and will require further 
public scrutiny. Previous public comment on the claims 
resulted in considerable criticism as to how the conditions 
could apply to all foods. The dairy industry is once again not 
happy with the isolation of saturated fats and has argued 
against these conditions on this basis.

I have not mentioned the Government's draft National 
Food and Nutrition Policy that has until this month been 
available for public submission, and will do so now briefly.

The National Food and Nutrition Policy aims to encour-
age all sectors of the community, including manufacturers, 
to work towards a common goal in the interests of the health 
of all Australians. This will encompass food production 
and promotion which meets the dietary guidelines. This 
includes foods which are low in fat, salt and sugar. It is 
anticipated that introduction of the National Food and 
Nutrition Policy is still some years away.

RECENT CHANGES TO PRODUCT INGREDIENTS
I will now turn to two recent changes to the Food 
Standards Code affecting product ingredients. The first is 
the introduction of reduced and low fat ice cream.

(i) Reduced and low fat ice cream
In November 1991 legislation was passed for the use of 
the terms “low fat” and “reduced fat” ice cream on 
labels. Previously these products had to be called ice 
confection. Such terminology had a negative connota-
tion to the consumer in regards to product quality. To 
be an ice cream, a product required over 10% milkfat. 
The Code now defines ice cream as follows:
- regular ice cream with a fat content of not less than 
  10%,
- reduced fat ice cream with a fat content not more 
  than 6.5%, and
- low fat ice cream not more than 4% milkfat.

To date, a number of reduced and low fat ice creams 
have been launched on to the market very success-
fully. The second change is regarding table spreads.

(ii) Table spreads
In August 1991 another change to product ingredients, 
which also affects the dairy industry, was made to the 
Standard for Table Spreads. Briefly the changes are:
- all spreads other than margarine and butter (which 
  are 80% fat) can be labelled “Table Spreads”;
- the term “reduced fat” applies to spreads with a fat 
  content of over 30% but not more than 60%. 
  Previously the level was 30-50%;
- the term “low fat” applies to spreads with a fat 
  content not more than 30%. Previously there was no 
  provision for “low fat” spreads;
- the words “butter” and “margarine” can now be used 
in conjunction with terms such as “reduced fat” and 

“low fat”, but only as a description of a “spread”. For 
example, a product previously called a “reduced fat 
spread” can now be called a “reduced fat butter 
spread”;
- the words “dairy blend” can be used in conjunction 
with “reduced” and “low fat”, and are no longer 
exclusively for the description of a full fat (80%) butter 
or margarine;
- where, previously, a dairy blend had to contain milk-
  fat as a majority component of the total fat, that is 
55% milkfat and 25% vegetable oil, the standard 
now permits the milkfat level to be as low as 50% of 
the total fat content, with vegetable oil as high as 
50% of the total fat content.

The new standard has resulted in a range of new pro-
ducts for the dairy industry. These new products are 
meeting consumer demand for a low fat table spread 
which has the flavour of butter and the spreadability of 
margarine.

CONCLUSION
In conclusion, a lot has and is presently happening with 
food legislation in Australia. The health messages and pro-
duct descriptions presently under review will have far-reac-
ching consequences on health promotion of food products 
and it is for this reason that finalization of them is still some 
years a way.

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II. THE FAT DILEMMA

1. THE RELATIONSHIP BETWEEN DAIRY PRODUCTS AND CARDIOVASCULAR DISEASE

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INTRODUCTION

Diseases of the heart and blood vessels represent the leading cause of death in the United States and most industrialized countries in the world. Epidemiologic studies have identified a number of risk factors for this multifactorial disease and numerous health agencies have initiated intervention programs in an attempt to reduce risk factors in the population. Of major importance in considering cardiovascular disease risk are hypercholesterolemia, hypertension, cigarette smoking, glucose intolerance and obesity. Each makes independent contributions to heart disease risk and are known to interact to compound the overall risk profile. Obviously with a disease of such magnitude having defined risk factors, recommendations of interventions to reduce the lifestyle factors involved in increased cardiovascular disease risk becomes a rational and desirable approach to reducing incidence.

HYPERCHOLESTEROLEMIA, CARDIOVASCULAR DISEASE AND DIETARY RECOMMENDATIONS

Hypercholesterolemia constitutes a significant risk factor for heart disease and there is little debate that elevated plasma cholesterol levels, primarily low density lipoprotein (LDL) cholesterol, account for a significant portion of cardiovascular disease incidence in the population. Intervention studies suggest that for every 1% reduction in plasma cholesterol there is a corresponding 2% reduction in heart disease risk. Based on the evidence from epidemiological and intervention studies, the National Cholesterol Education Program (NCEP) of the National Heart, Lung and Blood Institute, National Institutes of Health has recommended that the American public modify their diet to lower plasma cholesterol levels, and that for those individuals with high-risk plasma cholesterol levels after dietary intervention, hypercholesterolemic drug therapies be initiated [1]. Ideally, the goal is to have a plasma total cholesterol of less than 200 mg/dl (5.2 mmol/l) to attain a low-risk classification. Even though 50% of the population has a cholesterol level below 5.2 mmol/l, dietary intervention is recommended for every man, woman, and child over the age of 2 years. Similar dietary recommendations have been made by other health agencies [2, 3].

Based on the current understanding of dietary effects on cholesterol and lipoprotein metabolism, predictions can be made regarding the effects of the recommended dietary changes on plasma lipid levels and heart disease risk [4]. In the United States reduction of saturated fat calories to 10% and of cholesterol to less than 300 mg/day would be predicted to reduce the average plasma cholesterol level by 0.36 mmol/l, approximately 6.5%. If the proposed 2% reduction in heart disease risk is achieved for every 1% reduction in plasma cholesterol is true, then these changes would achieve a 12-14% reduction. Based on these considerations it would appear that dietary intervention on a population basis will have significant effects on heart disease incidence and mortality [5, 6].

While the evidence for a beneficial population response to dietary intervention is fairly strong, the major benefits attained are in that 50% of the population classified as moderate to high risk. Due to the relatively lower incidence rate in the average-risk population and the small increments in risk relative to the plasma cholesterol level in this group, the overall benefits are small in this group [5]. Clearly, population based interventions treat everyone in an attempt to benefit those at increased risk for cardiovascular disease.

DAIRY PRODUCTS AND CARDIOVASCULAR DISEASE RISK

The major recommended dietary change resulting in a lower plasma cholesterol level is the decrease in saturated fat calories, with any decrease in dietary cholesterol intake resulting in a much smaller plasma cholesterol lowering effect. The available evidence indicates that the decrease in plasma cholesterol resulting from a reduction in saturated fat intake is due primarily to the reduction in two saturated fatty acids - myristic acid (C14:0) and palmitic acid (C16:0). Studies have shown that intake of stearic acid (C18:0) has a hypocholesterolemic effect and should not be considered as a plasma cholesterol raising saturated fat. One source of myristic and palmitic acids in the diet is dairy fat (Figure 1.1) and accordingly it has been recommended that intake of full-fat dairy products be reduced.

![Figure 1.1: Fatty acid composition of dairy fat. Data presented as percentage of individual fatty acids.](image-url)
Epidemiologic studies have shown a positive association between intake of both dairy products and butter with coronary heart disease mortality rates (Figure 1.2); however, the relationship is weak and exhibits a high degree of variability. As shown in Figure 1.2, at the average levels of intakes of dairy products and butter, rates of coronary heart disease mortality range from a low of 150 to a high of 550 deaths per 100,000. Obviously there must be other factors involved in the wide range of coronary heart disease mortality rates in these various populations. In a similar manner, studies within populations have usually not found a significant relationship between milk and cheese consumption and total mortality. These data suggest that intake of dairy products does not have a strong relationship to coronary heart disease or total mortality rates across or within populations.

Clinical studies of the effects of dairy products on plasma cholesterol levels are inconsistent with some studies suggesting a plasma cholesterol raising response to intake of full-fat dairy products while others do not find a significant effect [8-11]. Some studies have even suggested that dairy products contain a "hypercholesterolemic milk factor" which negates the plasma cholesterol raising effects of its saturated fat content (reviewed in [8]). While the existence of a putative "hypercholesterolemic milk factor" remains in doubt, the studies do appear to indicate that the plasma cholesterol raising effects of full-fat dairy products is less than would be predicted based on its fatty acid composition and that some aspect of the physicochemical structure of the fat, the mineral content, or the protein composition alters the plasma cholesterol response to the intake of dairy products. Animal studies have in many cases been difficult to interpret; however, in many of these studies the data suggest that intake of whole milk has a less than predicted plasma cholesterol raising effect. Why the saturated fatty acids in dairy products do not have the predicted effect on plasma cholesterol levels remains unclear. Another question has been the potential differences between fermented and unfermented dairy products with regard to effects on plasma cholesterol levels.

**DIETARY INTERVENTIONS, PLASMA CHOLESTEROL LOWERING, AND INDIVIDUAL RISK**

When the efficacy of dietary interventions in lowering plasma cholesterol levels in individuals, as compared to populations, is considered the relative merits become complicated since individuals exhibit heterogeneity of response to dietary changes which precludes simple predictions based on formulas [12,13]. Numerous studies have demonstrated that the response to changes in dietary cholesterol and saturated fat are highly variable between individuals and that subjects do not have the same degree of plasma cholesterol lowering when consuming a low-fat, low-cholesterol diet. Approximately one-third of the population is sensitive to dietary cholesterol which, even on a population basis, makes little if any contribution to elevated plasma cholesterol levels. In a similar manner, changes in dietary fat saturation result in highly variable responses with some individuals exhibiting significant plasma cholesterol lowering while others have little if any response [12,13].

**DIETARY INTERVENTIONS AND GOOD CHOLESTEROL, BAD CHOLESTEROL**

While it is clear that an elevated plasma LDL cholesterol is a risk factor for heart disease, studies have also indicated that low levels of plasma high density lipoprotein (HDL) also constitute a risk. Dietary interventions to lower plasma LDL cholesterol levels also lower plasma HDL and the current debate regarding low-fat versus moderate-fat diets represents this concern [14]. The concern deals with the fact that shifting from a typical diet to the recommended diet often lowers not only LDL but also HDL [13, 14] and that the heterogeneity of responses is such that some individuals lower HDL levels more than LDL. Such findings have led to the suggestion that a moderate-fat diet, similar

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*Figure 1.2: Relationship between dairy (■) and butter (□) intake and coronary heart disease (CHD) mortality rates. Data from a study of 20 countries [6]. Solid lines represent correlation, dotted lines represent mean intakes.*
to the Mediterranean diet with higher levels of monounsaturated fatty acids, may be a more effective dietary intervention to lower LDL without affecting plasma HDL cholesterol [14]. There are also concerns raised regarding low-fat diets in that the relative benefits for males and females might differ [15] in that the protection afforded women by high plasma HDL cholesterol levels might be compromised by the HDL lowering effect of dietary interventions.

**DIETARY INTERVENTIONS AND GENETICS**

Numerous studies have indicated that heart disease risk has a large genetic component [16] and that even in the absence of hypercholesterolemia risk can be elevated due to genetic variations in lipoprotein structure and metabolism [17, 18]. Recent studies have demonstrated that the plasma cholesterol response to dietary interventions is also affected by genetic factors and play a major role in the sensitivity of individuals to dietary modifications [19-21]. Certain genetic patterns are associated with increased sensitivity to diet whereas other patterns indicate a resistance to changes in plasma cholesterol by reductions in dietary saturated fat and cholesterol. The documented heterogeneity of plasma cholesterol responses to dietary interventions has a significant genetic component which is only now being recognized.

The existence of genetic factors determining heart disease risk and the efficacy of dietary interventions in risk reduction raises the question of whether a population-based approach to risk reduction will be of benefit to those with genetic predispositions to heart disease or to resistance to diet-mediated plasma cholesterol lowering. Emphasis on a population based approach minimizes the opportunity to devise patient-specific dietary interventions which may be necessary to attain effective risk reduction in individuals with lipoprotein-related increased risk for heart disease in the absence of clear hypercholesterolemia.

**DIETARY INTERVENTION AND LIFE EXPECTANCY**

The data indicate that a population based dietary intervention approach to hypercholesterolemia and heart disease risk can be effective. Unfortunately, the effect, in terms of increased life expectancy, is relatively small. Modeling studies indicate that for the average individual life-long adherence to a fat modified diet will result in a 3 - 4 month increase in life expectancy [5, 22, 23]. While on a population basis this can be viewed as a saving of 62 million life years, on an individual basis the effect is rather modest [5]. The calculated effects on life expectancy do not address benefit in terms of the quality of life and this remains an area of uncertainty. What is clear from the modeling studies is that the largest benefit will be gained by those with the highest risk.

**SPECIAL GROUPS WITH SPECIAL NEEDS**

Dietary recommendations have a tendency to instill a "good food, bad food" attitude in the mind of the public. Elderly, afraid of high quality nutritious foods because of saturated fat and cholesterol, parents with overzealous approaches to restricting the diets of children, and misleading advertising claims as to the "healthiness" of some products are all responses to dietary guidelines. For many groups adequate nutrition is a more immediate concern than fat calories and cholesterol content. For many the advice to lower total and saturated fat and cholesterol in the diet equals to a zero tolerance approach to nutrition because few individuals understand how many calories come from fat or how much cholesterol an item might contain. In many cases the concept becomes "if too much is bad, then none at all must be better." Such attempts at healthy eating are anything but healthy. One must also be concerned about the current attempts to convince the public that even 30% of calories as fat is too high and that we should consume less than 20%. The potential risks in terms of HDL lowering and mild glucose intolerance must be given serious consideration when such dietary extremes are proposed.

Animal products in the diet provide many essential nutrients involved in growth and development and in health maintenance, and reduction or exclusion of these products from the diets of children, pregnant women, and the elderly is not a trivial matter. Population-based recommendations for dietary changes must be accompanied by some aspects of nutrition education if the interventions are to be both effective and safe. The argument that mass dietary intervention will do no harm is not absolutely true. From a nutritional perspective this may not be the case since many in the population may exclude high quality, nutritionally dense foods out of fear due to saturated fat and cholesterol. Every meal should not be a life or death decision for the public. As sources of essential, high quality nutrients dietary products in the diet represent an important source of essential nutrients. In dealing with a public having little knowledge of the scientific aspects of nutrition it may be that a "good food - bad food" mentality results in many individuals losing valuable sources of nutrients from the diet. In many cases the major health concern is not so much what one eats but rather how much one eats and whether or not the foods have nutritional density or simply represent high calorie, fun foods.

**SUMMARY**

Epidemiologic surveys, animal studies, and clinical investigations have shown a positive association between saturated fat calories and plasma cholesterol levels and elevated plasma cholesterol levels are clearly associated with cardiovascular disease risk. Based on this evidence, the recommendation to reduce saturated fat intake to lower plasma cholesterol levels and cardiovascular disease risk has been widely disseminated to the public. One source of saturated fat in the diet are dairy products, and the intake of full-fat dairy products has been discouraged. Dairy products play a significant role in maintaining a balanced, healthy diet and supply a number of essential vitamins and nutrients; however, the fat content of many dairy products is a concern to health professionals and consumers. It is important to balance the contributions of dairy products to nutrition and health with considerations of the saturated fat content of these products. There are reports in the literature suggesting that intake of dairy products does not result in the predicted plasma cholesterol raising effect expected from their fatty acid composition. Whether this lack of a major cholesterol raising effect of dairy products is due to the physicochemical properties of the fat or to a putative "hypercholesterolemic milk factor" remains unknown. No matter the mechanism, the evidence fails to document a relationship between intake of dairy products, elevated plasma cholesterol levels, and cardiovascular disease incidence either across or within populations.

From a nutritional perspective, dairy products are an important part of a balanced diet. Efforts to reduce saturated fat intake must be balanced with appropriate nutrition education to maintain adequate intakes of vitamins, minerals and other essential nutrients supplied by animal pro-
products in the diet. Dairy products are an important source of calcium essential for growth and development and for minimizing the risk of osteoporosis. Infants and children benefit from dairy products in the diet important for normal growth and caloric expenditure. Limitations placed on quality, nutritiously sound foods such as dairy products should be viewed with due skepticism and must be based on solid scientific evidence of benefit in cardiovascular disease risk reduction.

LITERATURE


2. ANIMAL FAT AND HEALTH, SOME RECENT NORWEGIAN EXPERIENCES

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An excessive intake of dietary fat is related to some of our most prevalent chronic diseases, in particular coronary heart disease (CHD) and possibly certain forms of cancer [1]. The unfavourable effects on health of a high intake of animal fats have primarily been ascribed to their content of certain saturated fatty acids and cholesterol, components found to be of particular importance in the pathogenesis of atherosclerosis [2]. The most potent atherogenic fatty acids are those with 12, 14, and 16 carbon atoms, that is, lauric, myristic and palmitic acid. These are abundant in milkfat, meat and meat products. For this reason these products have been the focus of discussions regarding diet and cardiovascular diseases.

It should be stressed, however, that classification of fat into vegetable and animal is only a practical classification. The nutritional properties of fats are not so much determined by their source as by their fatty acid composition. Previously, in Norway for example, margarine was a more important source of saturated - (and trans) fatty acids than animal fat. Also, fish fat has properties very distinct from land animal body fat and should therefore, from a nutritional point of view, not be classified as animal fat. What is important from a nutritional point of view is that about half of our dietary fatty acids are saturated and that 2/3 of these are derived from dairy products and meat. For this reason animal fat deserves special attention.

The questions I want to focus on in this discussion are the following:

1. What is the relationship between animal fat, or more precisely saturated fat, and disease?
2. Is it possible to alter the dietary habits of the population, both at the individual and at the national level, to reduce the fat, in particular the saturated fat content of the diet?
3. Is such a reduction associated with reduced risk, and reduced mortality from diseases claimed to be related to intake of total fat and certain fatty acids?

To illustrate my points I will to a large extent use experiences from Norway as examples.

ATHEROSCLEROSIS AND DIETARY LIPIDS

The underlying cause of coronary heart disease is atherosclerosis. The dietary fat hypothesis for the development of atherosclerosis goes back to the first part of this century when it was discovered that dietary fat could cause atherosclerosis in experimental animals, that elevated serum lipids were associated with atherosclerosis and that dietary fat influenced serum lipids. During the last 75 years, in spite of numerous attempts, it has not been possible to disprove the hypothesis and the essential elements of the hypothesis are still the same. In recent years considerable support for the hypothesis has come from sophisticated cell biological work but for our discussion the epidemiological evidence may be the most relevant.

During the early fifties Ancel Keys pointed to the association between total fat intake and total mortality from CHD. In the classic Seven Countries Study Keys and co-workers subsequently showed the significant correlation between CHD mortality and serum cholesterol, between CHD mortality and saturated fat, and further a strong correlation between fat intake, in particular saturated fat intake and serum cholesterol [3].

Metabolic studies, primarily by Keys and Hegsted and their co-workers, demonstrated that different types of fat affected serum cholesterol in different ways [2]. In short, these studies showed that when exchanged for carbohydrates, saturated fatty acids with 12-16 carbon atoms, that is, lauric acid, myristic acid and palmitic acid, raised serum cholesterol, while oleic acid decreased it. Oleic acid and stearic acid were neutral. Dietary cholesterol affected serum cholesterol only to a moderate degree. From their results Keys and Hegsted derived their well-known equations linking changes in serum cholesterol to changes in intake of fatty acids and of cholesterol. A large number of subsequent studies have demonstrated the high degree of predictability of these equations. Recent studies, like those by Mensink and Katan [4] and Ginsberg et al. [5] have shown that also under more normal experimental conditions with ordinary foods and young adults will changes in dietary lipids be followed by changes in serum cholesterol close to predicted from the equations. What could not be predicted from the equations of Keys and Hegsted were individual changes in LDL-cholesterol and HDL-cholesterol which may be different to total serum cholesterol. Since total cholesterol is mainly determined by LDL-cholesterol, this does not in any way alter the main conclusions about the effect of dietary fat on serum cholesterol.

A most interesting study to illustrate the predictability of the Keys' and Hegsted's equations is the recently published study by Sandström et al. [6]. For 8 months they maintained a group of young students on a diet containing 30% of energy of fat according to the Nordic Nutrition Recommendations, down from 37% in their usual diet. Saturated fat was reduced from 14 to 8% of energy. A significant reduction in serum cholesterol and in blood pressure was observed and the difference was maintained for the whole experimental period of 8 months. From the data given in the paper it is possible to calculate the expected change in serum cholesterol according to Keys and Hegsted. The observed value (0.49 mmol/l) was remarkably close to the expected values (0.58 and 0.54 mmol/l, respectively).

Results from a Norwegian study, where the amounts and types of fatty acids in the diet were determined by the double portion technique in a randomly selected group of 40-year-old men, showed that the values for saturated fat intake and serum cholesterol fell exactly on the correlation line as calculated by Keys et al. from the Seven Countries Study [7]. The Norwegian study showed that palmitic acid, myristic and lauric acid accounted for 44% of the total fatty acid intake (total saturated fat was 58%), higher than in any of the groups of the Seven Countries study.
It can be concluded that the main factor determining the mean serum cholesterol of a population is the intake of saturated fat. Because of the importance of saturated fatty acids as determinants of serum cholesterol of a population the question about the relation of dairy fat and meat to CHD has been raised. Turpeinen [8] pointed to the fact that in international comparisons a higher correlation is found between CHD mortality and dairy fat than between CHD mortality and total fat. Similar findings have been reported from Italy where a high correlation between CHD mortality of different regions and the per capita consumption of milk and cheese was found [9].

THE CHD EPIDEMIC IN NORWAY

Mortality from cardiovascular diseases (CVD) started to increase in Norway in the 1920s. This increase went unnoticed until after the second world war when the mortality data were analysed. It was then soon realized that we were facing a new epidemic and that the disease in question primarily had to do with exogenous factors.

In 1951 Strøm and Jensen [10] published their classic data on the decline in mortality from CVD diseases in Norway during the war, mainly due to reduced mortality from ischemic heart disease. The rapid change indicated that it was primarily the thrombotic and not the atherosclerotic component that had been most affected by the factors in question. This view was supported by the findings that thromboembolic complications after major surgery at the Oslo City Hospital fell from 32 to 6 per 1000 operations during the war and increased again to 20 in 1947 [11].

What were the explanations for these dramatic changes? Storm considers that a change in nutrition was the most important factor. In Norway during the war the energy supply fell by 10-20%, resulting in a slight degree of undernutrition and weight loss in the population as a whole. Dietary studies among working class people in the cities showed a very low fat content of the diet, 22-24% of energy, due to reduced intake of margarine, cream, whole milk, cheese and meat. This was compensated by an increased intake of cereals, potatoes, root vegetables and fish. Total reduction in animal and other saturated fat was thus considerable.

Reduction in smoking and increased physical activity may also have contributed. During the post-war years there was a rapid increase in the incidence of myocardial infarction and within a few years Norway ranked among the top countries with regard to CHD mortality.

The mortality peak was reached in the early 1970s. Since then we have, like many other countries, experienced a decline for both sexes and for all age groups [12]. A closer look at the different age groups discloses that in males the youngest age groups have experienced the most marked decline, for the age group below 50 the decline has been about 60%. In females, also the oldest groups have benefited from the decline.

What are the factors most likely to be responsible for the recent decline in CHD mortality?

Smoking has probably played a minor role. Since the early 1970s the percentage of daily smokers has decreased somewhat in males but increased in females. Total sale of tobacco has decreased by approximately 15% from 1975 to 1985.

The total burden of hypertension has certainly played a role. This is reflected in the continuous decrease in cerebrovascular mortality. After a transient increase around 1960 (concomitant with the start of treatment for hyperten-

NATIONAL TRENDS IN FOOD PATTERN

Changes in dietary habits may have contributed significantly to the decline in CHD mortality.

The long-term trends in carbohydrate and fat consumption in Norway show that during the last century there has been a downward trend in carbohydrate and an upward trend in fat consumption until around 1970. At its peak, fat made up 41% of total energy, about half of it saturated. During the last 15-20 years there has been a remarkable increase in total fat consumption and actually fat contributes with 34% of the total energy, about 40% of which is saturated [13].

The decrease in margarine consumption may explain a large part of this decline. A small decline in butter consumption is also discernible while the consumption of cheese has increased.

In very recent years there has in addition been a major change in the consumers' choice of milk varieties. The consumption of whole milk (3.9% fat content) has been reduced while that of skimmed milk and, since 1984, that of low fat milk (1.5% fat) has increased. The sales of low fat milk now exceed that of whole milk and if the trend continues, whole milk may possibly disappear from the marked within a few years.

The decreased margarine consumption since the early 1970s has reduced total fat consumption by 5.8 kg while the change in total milkfat consumption amounts to about 4 kg of fat per capita per year.

Consumption of meat has not changed to any large extent while that of fish may have increased somewhat in recent years. Dietary cholesterol was reduced from 450 mg/d in 1975 to 358 mg/d in 1990.

The P/S ratio has increased from 0.36 to 0.43 due to a relative increase in the use of soft margarines.

CORONARY HEART DISEASE PREVENTION PROGRAMMES

What are the explanations for these changing trends in food consumption pattern? Probably they are the results of preventive measures directed both at high risk groups and at the population as a whole.

Encouraged by the results of the Oslo diet and smoking trial [14], large-scale screening and intervention studies have been conducted in Norway during the last 15-20 years [15-18]. These studies have focused mainly on high risk groups. Parallel to these studies, the population strategy has been applied in a national nutrition policy programme that has been in action since 1979 [19, 20].

THE OSLO DIET AND SMOKING TRIAL [14]

The results of this study have to a large extent been used as a justification for large-scale prevention programmes in Norway.

This trial demonstrated that it is possible to alter the dietary habits of a large group of high risk individuals, that this change reduces serum cholesterol, incidence of CHD and also reduces total mortality. It was a randomized and
controlled trial. The participants, selected from 16,202 men aged 40-49, were 1232 normotensive men free of disease but at high risk based on serum cholesterol (range 7.9 mmol/L, mean 7.5) and smoking habits (80% smokers). The participants in the intervention group were advised to reduce the amount of saturated fat, increase polyunsaturated fat and to reduce total energy intake if body weight was high, and to stop smoking. The dietary advice was given by a dietitian in the course of a 30 min talk and with follow-up every 6 months. In addition the wives of the subjects were invited in groups of 30-40 together with their husbands for diet and smoking information. In a group of good diet responders 28% of energy was derived from fat at 4 years as compared to 44% in the control group [21]. The saturated fat intake was reduced by 58% and cholesterol intake by one half.

Serum cholesterol was reduced 13% in the intervention group against 3% in the control group. Only 25% stopped smoking against 17% in the control group.

At the end of the study after 5 years the incidence of fatal and non-fatal myocardial infarction and sudden coronary death was 47% lower in the intervention group than in the control group (p = 0.028 by 2-sided test). The difference in total mortality was not statistically significant after 5 years. At a second follow-up after 13 more years there was a borderline significant difference also in total mortality [22] that has become highly significant after 15 years of follow-up (I. Hjermann, personal communication). This finding is of importance when compared to the large drug trials where no difference in total mortality has been observed in the intervention groups compared to the control groups. Statistical analysis of the results by Cox's model showed that at least 2/3 of the reduction in incidence was explained by reduction in serum cholesterol and less than 1/3 explained by reduced smoking. A remarkable finding was that in the intervention group only one individual underwent a coronary bypass operation against 8 in the control group.

LARGE-SCALE INTERVENTION PROGRAMMES

Encouraged by these results large-scale intervention studies have subsequently been conducted by the Community Health Service in Oslo and by the National Health Screening Service in different Norwegian counties [15-18]. Originally three counties were selected as a pilot study. Since 1986 the programme has gradually been extended and the total country has now been included and more than 200,000 people at about 40 years of age have been screened. In certain counties up to three follow-up screenings have been performed. In size, this programme can be compared to the MRFIT study in the US. The aim of these studies has been to apply both the high risk and the population strategy by giving special advice to high risk individuals (10-12% of total) and to give general advice to the population.

A mortality follow-up of the programme [23] has confirmed, as expected, cholesterol as a main risk factor for CHD. The same correlation between serum cholesterol and mortality is seen in groups screened at different time periods and also the risk curve is almost identical to that from the MRFIT study in the US [24]. This consistency in risk relation, over time and in different populations, between serum cholesterol and mortality from CHD indicates that serum cholesterol is biologically related to the development of CHD.

The association between serum cholesterol and CHD mortality is shifted upwards by increasing blood pressure and by smoking, but does not appear to be altered by body mass index.

An association between serum cholesterol and total mortality has also been confirmed [23].

The difference in mortality from CHD between the counties can be explained entirely by the differences in total risk score. In Finnmark with the highest CHD mortality there are more smokers and the mean serum cholesterol is higher than in Sogn og Fjordane, the county with the lowest CHD mortality. From the first to the last screening there was a marked reduction in risk factors and total risk score. In particular mean cholesterol fell in all counties.

When we look at the serum cholesterol values of the first screenings only, there is a clear tendency towards a reduction of mean serum cholesterol of about 10% during a 15 year period. This is about what would be expected from the changes in fat intake of the population as a whole and it can probably explain a large extent the recent decline in CHD mortality.

From the mortality data it is difficult to evaluate the relative importance of the high risk versus the population strategy in the observed overall decline in CHD mortality. There might have been some effect on mortality in those confined to the highest risk group. Their mortality from myocardial infarction and sudden death was somewhat lower than expected from the linear relation between risk and mortality. When we look at the total population of the different counties screened, however, the mortality data do not invariably follow the national downward trends. It is therefore difficult to judge the effect of the high risk strategy compared to the more general population strategy.

One explanation for the adverse mortality trend in some of the screened counties may be that detection and drug treatment of hypertension under the age of 50 does more harm than good. Another, that persons who are not recalled after the first screening tend to relax any strictures they may previously have placed on their lifestyle. Presumably a much larger fraction of the population has to be defined as being at high risk for the high risk strategy to be successful. It then becomes a matter of definition if the strategy is to be considered high risk or population strategy. What we have learned from the Norwegian experience is that population morbidity and mortality are governed by powerful forces in society which are difficult to influence or to predict. This does not mean that we should not try to change the situation. On the contrary, research on how to develop and successfully apply the population strategy should be given the highest priority in the future. Such work requires an interdisciplinary approach and should not be left to the medical profession alone.

NATIONAL NUTRITION POLICY - POPULATION STRATEGY

Personally I tend to believe that most of the changes seen in dietary habits have come about as a result of measures directed primarily towards the population as a whole and not as a result of high risk strategy measures. The Norwegian nutrition policy adopted by the Parliament in 1975 [19, 20] and the existence of a National Nutrition Council have been instrumental in this regard. The main nutrient goal was to reduce total fat content of the diet from 41% to 35% of total energy by 1990. This was seen as a reasonable compromise between agricultural and nutritional interests. Already in 1982 a scientific committee recommended the lowering of total fat content of the diet to 30% of the energy [24] but this recommendation was not incorporated into the official health policy. In a forthcoming report to the Parliament on health prevention and nutrition it will be proposed that the new goal be set at 30% of the
energy from fat with emphasis on a further reduction in saturated fat.

As we have seen, the main goal of the 1975 report, reduction of total fat to 35% of energy by 1990, was reached in 1988. From this point of view the Norwegian nutrition policy has been a success. Unfortunately it has not been possible to evaluate the effect of the different components of this policy. My personal view is that measures directed towards the demand side (the consumers) have been more important than those directed towards the supply side. Education of the public by all means and use of the media have certainly been of major importance. Only slowly has the supply side accepted the nutritional arguments.

FAT INTAKE AND CANCER

While the situation appears encouraging concerning CHD, the situation is far less clear when it comes to forms of cancer that have been claimed to be related to fat intake. Also the evidence that relates diet to cancer is far less clear than in the case of CHD. The evidence comes mainly from international comparisons like when mortality from breast cancer was related to fat intake in different countries. Such associations have not been consistently confirmed in cohort studies. Over the 15-20 years when fat intake has decreased in Norway there has been a continuous rise in the incidence of breast cancer, colorectal cancer and prostate cancer, all proposed to be related to fat intake. It is also noteworthy that the parts of the country with the highest CHD mortality, as in Finnmark county, also have the lowest incidence of these cancer forms. One explanation for these diverging time trends between cancer and CHD may be that cancer has a much longer latency period and that any decline can only be expected in the future. Only time will show if this is so.

CONCLUSIONS

The Norwegian experience shows that it is possible to reduce total and saturated fat intake of the population and that this reduction is accompanied by a lower serum cholesterol level in the population. Concomitant with the changing trends in dietary habits cardiovascular disease mortality has declined. A further reduction of fat intake from the actual 34% of energy towards 30% means further reduction of visible and invisible fat, exchange of red meat and meat products by leaner meat, more fish and more vegetable foods in our daily diet.

LITERATURE

16 The Cardiovascular Disease Study in Norwegian Counties. Results from the second screening. National Health Screening Service, Oslo (1989).  
3. ASSESSMENT OF TRANS FATTY ACID INTAKE IN EUROPEAN COUNTRIES: IS IT REQUIRED?

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INTRODUCTION
Dietary trans fatty acids originate from both natural and industrial (food processing) sources. It is believed that trans fatty acids do not exert harmful effects, provided the diet contains sufficient linoleic acid [1, 2]. However, recent research [3] indicates that high dietary levels of trans as compared to cis monounsaturated fatty acids lower plasma HDL cholesterol and slightly raise plasma LDL cholesterol. Thus trans fatty acids in the diet seem comparable to dietary saturated fatty acids with respect to cardiovascular risk. Therefore, it is timely to assess trans fatty acid intake in European countries and to identify the contribution made by dairy products.

TRANS FATTY ACID INTAKE
Table 3.1 shows data on estimations of average trans fatty acid intake in seven western countries. It should be stressed that these averages are based on estimations of food intake from dietary surveys and food disappearance data, combined with data on the fatty acid composition of foods. Both sources are liable to major inaccuracies and do not allow comparisons across countries. Therefore true trans fatty acid intake remains uncertain. Moreover, and this is even more important, knowledge about the distribution of trans fatty acid intake within groups of the populations is scarce. It seems possible that individual intake figures could largely exceed average intake values.

Table 3.1: Estimated intake of trans fatty acids in some western countries (g/day)

<table>
<thead>
<tr>
<th>Country</th>
<th>Trans Fatty Acids (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA</td>
<td>7.6</td>
</tr>
<tr>
<td></td>
<td>8.1</td>
</tr>
<tr>
<td></td>
<td>13.3</td>
</tr>
<tr>
<td></td>
<td>8.3</td>
</tr>
<tr>
<td>Canada</td>
<td>9.6</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>12.0</td>
</tr>
<tr>
<td>Netherlands</td>
<td>10.9*</td>
</tr>
<tr>
<td>Finland</td>
<td>5.6</td>
</tr>
<tr>
<td></td>
<td>3.0*</td>
</tr>
<tr>
<td></td>
<td>1.5-1.9*</td>
</tr>
<tr>
<td>Sweden</td>
<td>7</td>
</tr>
</tbody>
</table>

* Analysed by IR spectroscopy or GLC in composite total diets.

Most estimations come to an average intake of 8-12 g/day corresponding to 10-12% of total fat intake. In some countries, like Sweden and Finland, trans fatty acid intake seems relatively low and shows a tendency to decrease [13, 14].

CONTRIBUTION BY DAIRY PRODUCTS
Milkfat contains about 3-6% of total fatty acids as trans fatty acids, mainly monoenes with the trans unsaturation in positions 6-16 of 18:1, with elaidic acid (11-trans) being the most predominant. In spreads and shortenings the trans fatty acid content is highly variable and depends on the degree of hydrogenation. Most spreads fall within the range of 10-40% trans fatty acids. The contribution to trans fatty acid intake by dairy products for some countries is shown in Table 3.2.

Table 3.2: Contribution of dairy products to trans fatty acid intake (%)

<table>
<thead>
<tr>
<th>Country</th>
<th>Trans Fatty Acid Intake (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Netherlands</td>
<td>13.8</td>
</tr>
<tr>
<td>UK</td>
<td>14.2</td>
</tr>
<tr>
<td>USA</td>
<td>6.0</td>
</tr>
<tr>
<td>Finland</td>
<td>25</td>
</tr>
</tbody>
</table>

Table 3.2 indicates that dairy products contribute to a limited extent to trans fatty acid intake. The main contribution to trans fatty acid intake is made by hydrogenated vegetable oils and fish oils, beef only contributing to a very small extent (less than 5%).

CONCLUSION
Trans fatty acid intake in western countries is not very well defined, for example, distribution of intake is unknown and comparability across countries is not possible. The contribution of dairy products to trans fatty acid intake seems modest. An international study using a protocol that allows comparisons across countries is required to assess true trans fatty acid intake in Europe.

LITERATURE
III. THE CALCIUM AND OSTEOPOROSIS PROBLEM

1. THE IMPORTANCE OF PEAK BONE MASS FOR SKELETAL STATUS IN OLD AGE AND CALCIUM REQUIREMENT DURING GROWTH

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INTRODUCTION

Prevention of osteoporosis is of major concern to health authorities. In many western countries its incidence increases due to aging of the population and factors of which the relative importance has not yet completely been identified. Among these factors are decreased physical activity, low calcium intake, high sodium and protein intake and suboptimal provision of vitamin D [1, 2]. A low bone mass appears to be the main risk factor for sustaining a bone fracture (Colles' fracture, vertebral compression fracture or hip fracture) as a result of minor or even no trauma. It has now been well established in several studies [3-5], including the 10-year study undertaken by NIZO [5], that bone mass at a younger age is a major determinant of bone mass in later life. Therefore measures to prevent osteoporosis should aim at promotion of peak bone mass development in young people in addition to inhibition of bone loss. Thus according to Hansen et al. [3] bone mass in women in later life, when fractures occurs, can be predicted rather adequately by a combination of a base line bone mass measurement at menopause and biochemical estimation of the future bone loss. Here we would like to concentrate on calcium intake at a young age as an important factor for peak bone mass development.

CALCIUM REQUIREMENT DURING GROWTH

The calcium requirement during growth can be considered as the amount of calcium needed for bone development plus the amount needed to cover the endogenous calcium losses via skin, urine and faeces.

| Table 1.1: Estimated calcium retention calculated from figures on body weight gain and body calcium content in different age and sex groups |
|---|---|---|---|
| Sex, age | Body weight (kg) | Body calcium content (g/kg) | Calcium retention (mg/day) |
| Boys | 0-1 | 3.5-9.5 | 9.0-10.0 | 164 |
| | 1-10 | 9.5-13.5 | 10.0-12.7 | 117 |
| | 10-19 | 34.5-68.5 | 12.7-15.6 | 215 |
| | 19-25 | 68.5-72.0 | 15.6-17.0 | 85 |
| Girls | 0-1 | 3.5-9.5 | 9.0-10.0 | 164 |
| | 1-10 | 9.5-13.5 | 10.0-12.2 | 112 |
| | 10-19 | 34.5-60.0 | 12.2-14.6 | 156 |
| | 19-25 | 60.0-60.0 | 14.6-15.0 | 13 |

Calcium retention

Although the age at which the bone mass reaches its peak is not precisely known, it seems safe to assume that after the age of about 25 years only a small and insignificant increase of cortical bone may occur [6]. On the basis of figures on weight gain and data on forearm bone density of Caucasians at different ages the average calcium retention values in different age and sex groups can be computed [7] (Table 1.1).

It should be stressed that calcium retention varies with the rate of skeletal growth and that retention may be as high as 400 mg/day at puberty [8].

Losses through the skin

In most calcium balance studies calcium losses through the skin have been neglected. A reasonable average value seems to be 25 mg/day for adults [7] and this value can be used to calculate losses in other age groups, using the metabolic body weight for extrapolation (Table 1.2).

| Table 1.2: Obligatory calcium losses (mg/day) in different age and sex groups |
|---|---|---|---|---|
| Sex, age | Urine | Skin losses | Faeces | Total |
| Boys | | | | |
| 0-1 | 24 | 4 | 14 | 42 |
| 1-10 | 61 | 10 | 35 | 106 |
| 10-19 | 115 | 19 | 66 | 200 |
| 19-25 | 148 | 25 | 85 | 258 |
| Girls | | | | |
| 0-1 | 24 | 4 | 14 | 42 |
| 1-10 | 61 | 10 | 35 | 106 |
| 10-19 | 108 | 18 | 62 | 188 |
| 19-25 | 131 | 22 | 75 | 228 |

Urinary excretion

In adults the mean urinary calcium excretion at low calcium intakes will be close to 150 mg/day [9]. This value corresponds to 6 mg/kg metabolic weight, a value that can be used to estimate the obligatory calcium excretion in different age and sex groups (Table 1.2).

Endogenous faecal excretion

In adults the daily secretion of calcium into the intestinal tract is about 150 mg [10]. Part of this quantity is reabsorbed. So at low calcium intake endogenous faecal calcium excretion is about 85 mg/day [10, 11] and this value is used to quantitate the endogenous faecal excretion in the
different age and sex groups, using the metabolic body weight as a basis for extrapolation (Table 1.2).

**Intestinal absorption**

Table 1.3 shows the sum of retention and obligatory losses of calcium for the different age and sex groups. These sums vary between 206 g/day in infants to 415 mg/day in adolescent boys and represent the amounts of calcium to be absorbed from dietary sources. To estimate the minimum calcium requirements, the key parameter is true intestinal absorption.

**Table 1.3: Amount of calcium to be absorbed, estimated minimum requirement and RDA in different age and sex groups**

<table>
<thead>
<tr>
<th>Sex, age (mg/day)</th>
<th>To be absorbed¹ (mg/day)</th>
<th>Minimum requirement² (mg/day)</th>
<th>RDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1</td>
<td>206</td>
<td>350-500</td>
<td>400-600</td>
</tr>
<tr>
<td>1-10</td>
<td>223</td>
<td>350-500</td>
<td>800</td>
</tr>
<tr>
<td>10-19</td>
<td>415</td>
<td>700-900</td>
<td>1200</td>
</tr>
<tr>
<td>19-25</td>
<td>343</td>
<td>600-800</td>
<td>1200</td>
</tr>
<tr>
<td>Girls</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1</td>
<td>206</td>
<td>350-500</td>
<td>400-600</td>
</tr>
<tr>
<td>1-10</td>
<td>218</td>
<td>350-500</td>
<td>800</td>
</tr>
<tr>
<td>10-19</td>
<td>344</td>
<td>600-800</td>
<td>1200</td>
</tr>
<tr>
<td>19-25</td>
<td>241</td>
<td>400-600</td>
<td>1200</td>
</tr>
</tbody>
</table>

¹ Sum of calcium retention (Table 1.1) and total obligatory losses (Table 1.2).

² Estimated on a basis of 40-60% true calcium absorption.

It is widely recognized that the efficiency of calcium absorption is increased during periods of increased physiological needs as at low calcium intakes. Absorption values as high as 75% in young children and as low as 15% in adults have been reported [12-14]. On the basis of current knowledge, it seems reasonable to accept a true calcium absorption of 40-60% in young growing persons for the purpose of estimating the minimum calcium requirements. This range corresponds to a net calcium absorption of about 30-40% (intake minus excretion, expressed as a percentage of intake).

**Minimum requirements and RDAs**

Application of true calcium absorption of 40-60% to the amounts that have to be absorbed from the diet results in an estimation of the average minimum requirements. Comparison of these values with the current USA RDAs [12] (Table 1.3) leads to the conclusion that for almost all age and sex groups the RDA provides a reasonable margin of safety, allowing for interindividual variability. However, the RDA seems unnecessarily high for girls aged 19-25 years.

**DISCUSSION**

It appears that a reasonable estimation can be made of the minimum calcium requirements in young people. The values obtained are well below the current USA RDAs for this nutrient. Therefore it does not seem likely that levels of intake in excess of these RDAs will lead to peak bone mass values that are superior to those that will be attained with the RDAs. Appropriate levels of physical activity are probably much more powerful in promotion of bone development.

**LITERATURE**

Bone mass increases rapidly during childhood and adolescence, reaching a peak in the third decade of life. Thereafter, bone loss takes place in both sexes [1]. In women, a superimposed acceleration of bone loss is seen in relation to the menopause. The menopause-related bone loss of trabecular bone starts early in the perimenopausal period and follows an exponential pattern, with substantial bone loss in the first 3 postmenopausal years and a rapid decrease thereafter [2]. The menopause-related cortical bone loss shows an exponential pattern as well, but the bone loss occurs less rapidly in the first postmenopausal years and slows down over a more protracted period of time [3].

It has been well established that the cause of postmenopausal bone loss is oestrogen deficiency. The mode of action of oestrogen on the maintenance of the skeleton is, however, not precisely known. Recently, oestrogen receptors have been located in osteoblast-like cells, suggesting that there might be a direct effect. Studies also suggest that oestrogen decreases the sensitivity of the skeleton to parathyroid hormone-induced bone resorption. Other possible pathways of the effect of oestrogen are through calcitonin secretion and through the calcium balance.

Heaney et al. [4] studied calcium balance in premenopausal and postmenopausal women who maintained their habitual calcium intake. It was observed that all women had a negative calcium balance, with the calcium balance being more negative in the oestrogen-deficient postmenopausal subjects than in the premenopausal subjects and the postmenopausal women who received oestrogen replacement therapy. The calcium balance correlated with the dietary calcium in both the oestrogen-deficient and oestrogen-replete women. With linear regression analysis it was calculated that the mean dietary calcium intake necessary to achieve a zero calcium balance lies in the order of 1000 mg/day for premenopausal women and around 1500 mg/day for postmenopausal women. Based on this study and additional circumstantial evidence, it was postulated that menopause-related bone loss can be prevented, at least in part, by increasing the calcium intake [5]. However, it has not yet been established whether the more negative calcium balance in oestrogen-deficient women is a cause or a consequence of the menopause-related bone loss.

In two European longitudinal studies of, respectively, 3 and 12 years duration, no significant relationship was observed between habitual dietary calcium intake and early postmenopausal bone loss [6, 7]. In several studies, calcium supplementation in early postmenopausal women was shown to reduce the cortical bone loss to some extent, but no significant effect on the rate of trabecular bone loss was observed. However, the statistical power of these studies was not sufficient to exclude an effect. In all studies in which calcium supplementation was compared to postmenopausal oestrogen replacement therapy, calcium supplementation was observed to be less effective in decreasing bone loss.

We studied the effect of calcium supplementation in 248 perimenopausal women during a 3-year trial [8, 9]. The subjects were selected from a random sample of the municipal register. Exclusion criteria were: hysterectomy or oophorectomy, use of gonadal hormones, renal failure, urolithiasis and metabolic bone disease. The subjects were randomized in three groups: 1/3 received no calcium supplementation, 1/3 received 1000 mg elemental calcium/day, and 1/3 received 2000 mg calcium/day. The main study parameters were: menstrual history, dietary calcium intake, bone mineral density of the lumbar spine (BMD) measured with Dual Photon Absorptiometry, metacarpal cortical thickness (MCT) and biochemical parameters of bone and calcium metabolism.

In the women who had been menstruating up to the last year of the trial, calcium supplementation significantly reduced lumbar bone loss throughout the study (mean change of lumbar BMD after 3 years: -3.2% of the initial value in the controls versus +1.6% in the supplemented groups, P < 0.01). In the women who stopped menstruating before or during the study, the lumbar bone in the supplemented subjects was significantly reduced in the first supplementation year, but not in the last 2 years of the study. The total bone loss over the entire study period in the supplemented subjects was equal to that in the controls. It could be concluded with 95% confidence that the difference in lumbar bone loss between the supplemented and the controls was greater than 0.5% of the initial value.

The metacarpal cortical bone loss (-3.0% of the initial value in the controls versus -2.0% in the treated subjects), serum alkaline phosphatase (AP), osteocalcin and urinary hydroxyproline excretion decreased significantly in the supplemented subjects in all menopausal groups throughout the study, but a menopause-related increase remained apparent. Apart from a more pronounced decrease of AP in the 2000 mg group, no significant difference was observed in the effect of calcium supplementation between the two treatment groups in any of the parameters.

The median of the average estimated dietary calcium intake of the four examinations was 1109 mg/day. One-third of the subjects had an intake between 310 and 948 mg/day, 1/3 between 950 and 1254 mg/day. The daily dietary calcium intake of the highest tertile ranged between 1255 mg and 2480 mg. Neither in the control group nor in the supplementation groups was a significant difference in the rate of bone loss between the tertiles of calcium intake observed. The maximal difference (95% confidence limits) in the yearly rate of bone loss expressed as percentage of the initial value between the lowest and highest tertile of dietary calcium intake that could be excluded was 1.0%
and 0.9% for lumbar BMD and MCT, respectively, in the controls and 0.7% and 0.6%, respectively, in the treated subjects. Even if the lowest tertile of dietary calcium supplementation was excluded from the analysis, the effects of calcium supplementation remained apparent.

These results indicate that calcium supplementation substantially reduced cortical and trabecular bone loss in the years immediately preceding menopause. Although it reduced postmenopausal cortical bone loss to some extent, it had no relevant clinical effect on the menopause-related accelerated bone loss. This suggests calcium intake does not play an important role in the modulation of menopause-related bone loss. Since the effect of calcium supplementation did not depend on the dietary calcium intake of the subjects, it is likely that the effect of calcium supplementation is other than can be achieved with an ordinary diet.

LITERATURE

3. CALCIUM INTAKE AT A YOUNG AGE AND INCIDENCE OF OSTEOPOROSIS

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INTRODUCTION

The peak adult bone mass at the end of the skeletal growth period at 25-30 years of age has been suggested as the decisive criterion regarding the prevention of osteoporosis [1]. Sowers et al. [2] as well as Shah and Belonje [3] observed that the peak adult bone mass of young women who consumed more than 800 mg of calcium per day was greater than that of women whose calcium intake was lower. However, a sufficient calcium intake is important also during adulthood in order to prevent bone loss beyond the biologically caused extent.

Without doubt, milk and milk products are essential to ensure an adequate calcium intake. For this reason the relationship between the calcium intake through milk and milk products and the bone density as well as the development of osteoporosis was studied in young adults and elderly people. The amounts of calcium taken up with milk and milk products were evaluated by means of a retrospective questionnaire. As in such a study nutritional and medical aspects are involved, a cooperation between nutrition scientists and medical people doing research in the area of osteoporosis was established.

METHODS

A group of 65 elderly individuals (48 females, 17 males; age 50 years and more) was studied. Osteoporosis had been diagnosed clinically and radiologically. Disorders which might have changed the resorption or metabolism of calcium (for example previous gastric or intestinal resection, malabsorption syndromes) were not present in any of them. These patients were compared with an age-matched group of 76 controls (50 females and 26 males). This control group was free of bone disease. A group of young adults (40 females and 40 males, 25-30 years) was also included in the study.

Bone density was measured at the proximal radius of the nondominant forearm (in most cases the left one) by single photon absorptiometry (SPA absorptiometer, Norland). The in-house normal laboratory range of the method was established by respective measurements in a group of 180 healthy subjects. The bone mineral content determined in this manner is directly proportional to bone strength [4].

The following bone-specific laboratory parameters were determined in all persons: serum calcium, serum phosphat, alkaline phosphatase, sex hormones (testosterone and estrogens), gonadotropins (FSH = follicle-stimulating hormone and LH = luteotropic hormone), parathyroid hormone (intact and midregional), 25-hydroxy-vitamin D₃ and osteocalcin as specific parameters of bone turnover.

The dietary intake of calcium via milk and milk products was assessed by means of a food-frequency questionnaire which included two and three different periods of life for young adults and elderly people, respectively: at the time of the study, 20-30 years prior to it (only for elderly people), and during childhood and adolescence. The questionnaire inquired about the consumption of 18 food items or food groups during the respective periods. For evaluating the intake of calcium, however, only the data applying to milk and milk products were used because these items are the dominant calcium suppliers in the human diet. The inquiries in the questionnaire about nondairy foods and eating habits at meals were included to avoid the answers being influenced by a one-sided emphasis on milk and milk products by which the interviewees might have been led to attach special importance to that product group.

In the present study the criterion for the amount of calcium intake via milk and milk products is a relative parameter called the calcium intake index [5]. Two auxiliary parameters are used to calculate this index:

![Figure 3.1: Influence of calcium intake (CII = calcium intake index) via milk and milk products in childhood and adolescence (C/A) on the bone mineral content in male (m) and female (f) young adults (y) and elderly (e) subjects.](image-url)
the consumption frequency score, where four given consumption frequencies are coded by using a score of points, and
- the calcium weighing factor which evaluates the food items with regard to their differing contribution to the intake of calcium.

CALCIUM INTAKE AND BONE DENSITY
Calcium intake through milk and milk products, as expressed by the calcium intake index, has a direct and distinct effect on the bone parameters of young adults. Figure 3.1 shows that the bone mineral content rises in both groups with increasing calcium intake through milk and milk products, but that the same amounts of calcium lead to higher bone mineral values in young males than in young females.

In the case of the elderly subjects it was also studied whether calcium intake via milk and milk products during different periods of life affects the bone parameters in advanced age. The results gave clear-cut evidence that the index determined for calcium intake via milk and milk products in childhood and adolescence is essentially responsible for the bone mineral content in the elderly (Figure 3.1). As in young adults, the same calcium intake index leads to sex-dependent differences in the bone parameter values of the elderly - a finding that supports the recommendation of a higher calcium intake for women.

CALCIUM INTAKE AND PREVENTION OF OSTEOPOOROSIS
In the elderly people, the intake of calcium via milk and milk products during the three periods of life was determined in the age-matched controls as well as in the osteoporotic patients (Figure 3.2). The following results can be derived from this survey:
- The calcium intake via milk and dairy products of the male and female osteoporotic had been markedly lower during childhood and adolescence (55% and 46%, respectively) than that of their age-matched controls. This finding confirms that inadequate calcium intake during these early periods of life is a decisive risk factor for the development of osteoporosis.
- 20-30 years prior to the present study the osteoporotic patients increased their calcium intake through milk and milk products considerably, so that for this period no significant differences exist between them and the control subjects. At that time of life, however, bone formation has already been completed, so that increased calcium intake cannot offset the calcium deficiency that existed during childhood and adolescence. The risk of developing osteoporosis can no longer be diminished.
- At the time of the present study the calcium intake of the osteoporotic patients and controls was nearly identical. The patients probably knew that an adequate calcium intake was necessary to prevent further aggravation of their illness.
- In the case of the controls it was found that their calcium intake via milk and dairy products had been at a uniformly high level throughout their lives. This life-long adequate calcium supply can therefore be regarded as an effective means of preventing osteoporosis.

FACTORS INFLUENCING THE BONE-SPECIFIC LABORATORY PARAMETERS
Whether calcium intake can influence the bone-specific laboratory parameters is a question of special interest. The results obtained indicate that calcium influences the levels of osteocalcin, a parameter of bone turnover. Increased calcium intake via milk and milk products is associated with decreased osteocalcin values. This association was particularly demonstrable in the young females and in the male and female osteoporotics (Figure 3.3). Decreasing osteocalcin levels resulting from a higher intake of calcium thus indicates a reduced rate of bone turnover. The results also demonstrate that an adequate supply of calcium suppresses the production of parathyroid hormone, thus reducing the release of calcium from bone.

These findings lead to the conclusion that an adequate calcium intake - which can best be ensured through milk and milk products - is not only a prerequisite for optimal bone formation but also a means of preventing enhanced bone resorption. This association was already evident in the young women and was of special relevance in the osteoporotic patients. An adequate intake of calcium via milk and milk products was an important protective factor against bone resorption in the male as well as in the female osteoporotics, as expressed by the 40-50% reduction of the serum osteocalcin levels.

SUMMARY
The bone mineral content of young adults as well as of osteoporotic patients and age-matched controls without bone disease was measured by single-photon absorptiometry. A retrospective dietary survey was additionally made to study the relationship between calcium intake and bone mineral content in different periods of life.

The bone mineral content and bone mineral density of young adults is directly related to the calcium intake through milk and dairy products.
The osteoporotics had a significantly lower bone mineral content than the controls. Calcium intake through milk and milk products in childhood and adolescence had been significantly lower in the patients than in the controls whereas in the later periods of life (20-30 years prior to the study and at the time of the study) there were no significant differences in the calcium intake of the two groups. It was also found that an adequate intake of calcium protected against increased bone resorption, as evidenced in particular by the reduced levels of serum osteocalcin, a parameter of bone turnover.

In conclusion, it can be stated that the data presented in this paper support the hypothesis that adequate calcium intake through milk and milk products in childhood and adolescence is a decisive marker for obtaining a maximum bone mass (peak adult bone mass) and for the prevention of osteoporosis. Furthermore it can be stated that increased calcium intake in later years may not reduce the risk of osteoporosis resulting from inadequate calcium intake during childhood and adolescence, but may prevent bone resorption.

LITERATURE


ACKNOWLEDGEMENTS

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4. THE IMPORTANCE OF CALCIUM INTAKE AND BIOAVAILABILITY FOR BONE DEVELOPMENT AND BONE STRUCTURE IN RATS

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The role of dietary calcium in the etiology and prevention of osteoporosis is discussed controversially. Some authors regard a lack of calcium as an initiating factor, most authors consider dietary calcium as an accelerating factor with respect to osteoporosis. Several investigations have shown that the intake of calcium, either habitual or supplemented, has a slowing-down effect on bone demineralization in postmenopausal women. Almost the same number of investigations have reported no effect. This contrast may be due to methodological differences. The main problem encountered with long-term studies in humans is the control of other confounding factors, such as physical activity, vitamin D status, and dietary components. Questionnaires or 3-day protocols used to assess the calcium intake bear a high risk of making false assumptions. This problem has recently been discussed in detail [1-4].

A strictly controlled long-term experiment with ovariectomized rats was carried out. This animal model has been established (proven ?, used ?) to simulate the hormonal situation of postmenopausal women. 140 rats were kept on semisynthetic diets and were allocated to one of three groups, differing only in their intake of calcium: low, medium, and high calcium groups equivalent to 0.1, 0.5 and 1.0% Ca/kg diet, respectively. After 7 months of rearing, the animals were ovariectomized and each group was divided into two subgroups, receiving 0.1 or 1.0% dietary calcium. Two balance periods were performed, the first at 4 months and the second at 9 months of the experiment. Four months after ovariectomy the animals were sacrificed and representative bone samples were taken for analyses. Parameters were bone mass, bone density and bone structure.

The aim of the experiment was to investigate the effect of calcium intake (1) during growth on bone mineralization at "adulthood", (2) during growth on bone demineralization after "menopause", (3) after menopause on bone demineralization after "menopause".

The results of the balance studies can be summarized as follows. A high calcium intake during growth increased fecal and urinary calcium excretion significantly but not calcium retention at "adulthood". A low calcium intake during growth decreased fecal excretion significantly and increased significantly calcium retention after "menopause" as a consequence of compensatory regulation. A high calcium intake after "menopause" increased fecal and urinary excretion significantly and tended to increase calcium retention after "menopause".

The results of the bone development parameters were as follows. A high calcium intake during growth increased bone mass and bone density significantly, but not bone structure at "adulthood". A high calcium intake during growth decreased the loss of bone density significantly, but not of bone mass or bone structure after "menopause". A low calcium intake after "menopause" increased significantly the loss of bone mass, bone density and bone structure after "menopause".

CONCLUSION
In a controlled long-term experiment with rats it could be demonstrated that calcium intake - during growth and after ovariectomy - had a significant effect on mineralization and demineralization of bone tissue. If calcium intake during growth was low, a compensation in calcium balance was seen at the time after ovariectomy, which could compensate for bone mass but not enough for bone density. A calcium balance per se does not necessarily reflect the degree of mineralization and demineralization of bone tissue.

LITERATURE
5. CALCIUM BALANCE IN ADOLESCENT GIRLS, RELATIVE IMPORTANCE OF GENETIC AND NUTRITIONAL FACTORS

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INTRODUCTION
Skeletal tissue provides the best example of growth process from infancy to maturity. The rate of this process and thereby the time required to achieve mature adult skeleton is variable. Most organ tissues including skeleton go through the developmental stages from fetal life to young adulthood. The most critical period in skeletal development is during the time of the most rapid bone modeling and turnover of adolescence. Nutritional requirements of all nutrients closely follow the developmental process and this particularly applies to calcium. Calcium deficiency during this critical phase of skeletal growth could negatively influence skeletal development and increase the risk of osteoporosis later in life. Attainment of peak bone mass is also under genetic influence.

SKELETAL GROWTH AND BONE MASS
The human skeleton develops through infancy, childhood and adolescence to a peak bone mass at maturity level. Most of the skeletal mass will be accumulated by the average age of 18 years, as recently established [1]. Thereafter, there is a minimal change in bone mass and density with age up to the time of menopause. Some skeletal sites continue to lose bone immediately after the age of 18 (proximal femur, and trabecular bone in the vertebrae) and the other sites show continuous apposition of bone up to the time of menopause (forearm, total spine, head) [1]. At menopause bone loss gradually occurs at all sites resulting in the bone mass which is decreased to the extent that fractures occur with minimal or moderate trauma. This time interval between attainment of bone mass in adolescence and the menopause is called bone maturity period. Since bone mass is one of the main determinants of fracture, high bone mass at skeletal maturity is considered the best protection against age-related bone loss [2]. Small differences in bone mass and density at a maturity level of 5-10% could contribute to a substantial reduction in the incidence of osteoporotic fractures.

The rate of bone modeling varies greatly with different biological stages and this is being reflected in a concomitant rise in serum osteocalcin [3], alkaline phosphatase, and hydroxyproline excretion in the urine [4]. Infants will almost triple their weight during the first year of life and adolescents will accumulate 57% of the total skeletal mass between Tanner's sexual developmental stage 2 and 5 (4-year interval) [1]. This correlates with the variation in osteoid pattern with age in human vertebrae [5]. The percent of osteoid covering trabecular bone surface is highest during infancy and adolescence, and decreases to a minimal value in early adulthood [5]. The amount of osteoid covering bone surface indicates the stage of relative immaturity of the skeleton primarily dictated by bone modeling/turover. To what extent mineral deficiency during periods of rapid skeletal modeling contributes to this phenomenon remains to be established.

CALCIUM REQUIREMENT DURING ADOLESCENCE
Over 99% of body calcium is in the skeleton. It contributes not only to the structural support but also serves as a large reservoir for calcium homeostasis. Low calcium intake does not have any deleterious effect on bone health of young individuals. It has been suggested that calcium deficiency during adolescence could influence adult height and/or peak bone mass and density and therefore reduce the fracture resistance among the elderly [6].

To achieve maximal peak bone mass, dietary calcium and its absorption need to be adequate to satisfy skeletal modeling and consolidation and also obligatory losses in urine, feces, and sweat. Growing individuals, contrary to adults, therefore need to be in positive calcium balance to meet those “extra” needs of skeletal growth and consolidation. Bone modeling period with the net positive bone tissue balance contributes to the constant demand for calcium throughout the developmental process. Thus, the question is how much dietary calcium is needed to assure optimal calcium balance during the various stages of sexual development of adolescence. As recently shown there is a significant positive relationship between calcium intake and body retention (calcium balance) of this element for adolescents [6, 7]. Lower calcium intakes, in spite of the relatively higher absorption efficiencies during puberty, were associated with lower calcium retentions in the body, and higher intakes with higher calcium retentions. Calcium retention in this age group represents skeletal retention since young individuals are free of the soft tissue calcification found in the elderly. The importance of a positive calcium balance in teenagers is further emphasized by the need to meet not only skeletal growth but losses of calcium through the skin which may amount to as much as 60 mg/day [7, 8]. Interestingly, during the period of rapid growth calcium excretion in the urine is practically not related to calcium intake [6, 7]. A weak relationship between urinary calcium and calcium intake exists during childhood when the rate of growth declines [7]. Such a relationship between urinary and dietary calcium definitely exists in adults [9]. The explanation for this is that rapidly growing individuals are retaining calcium they absorb rather than excreting it in the urine.

While the degree of positive calcium balance necessary to achieve any given bone mass can be calculated, the genetically possible value for optimal peak bone mass is unknown. We assume that it is close to the threshold balances, as was found recently. The existence of an intake threshold for calcium in humans has been established [10]. This is the level of calcium below which skeletal accumulation was a function of intake, and above which skele-
Table 5.1: Partition parameters for the split-regression model, by age group

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Partitiona</th>
<th>Rb</th>
<th>Threshold intake mg/day</th>
<th>Threshold balance mg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>88, 36</td>
<td>+0.825</td>
<td>1090</td>
<td>+603 ± 91c</td>
</tr>
<tr>
<td>2-8</td>
<td>75, 24</td>
<td>+0.555</td>
<td>1390</td>
<td>+246 ± 126</td>
</tr>
<tr>
<td>9-17</td>
<td>67, 66</td>
<td>+0.556</td>
<td>1480</td>
<td>+936 ± 184</td>
</tr>
<tr>
<td>18-30</td>
<td>122, 41</td>
<td>+0.601</td>
<td>957</td>
<td>+114 ± 133</td>
</tr>
</tbody>
</table>

a Number of subjects below and above the threshold, respectively.

b Composite R.

c x ± s.d. From Matkovic and Heaney [10].

total accumulation was constant, irrespective of further increases in calcium intake. Identifying and locating such a threshold was helpful for determining nutritional requirements for calcium [10]. Threshold intakes and balances were estimated by fitting 517 individual calcium balances to a two-component, split, linear regression model. The threshold values at which balances no longer rose with intake were higher than the current values for the recommended dietary allowance (RDA) for calcium in the US for all age groups concerned from infancy to young adulthood and specially for adolescents (Table 5.1) [10]. This threshold intake should provide sufficient calcium to insure maximal skeletal retention of calcium which will lead to peak bone mass formation.

The above findings should provide solid guidelines for the establishment of the RDA for calcium during adolescence.

ATTAINMENT OF PEAK BONE MASS AND HEREDITY

Genetic factors have been implicated in the pathogenesis of osteoporosis: a relationship between heredity and bone mass has been established in horizontal comparison models using twins [11]. In such studies the relative weight of genetic and environmental influences on bone can be assessed. Family resemblance studies have also noted a significant relationship between bone mass in mothers and daughters. All previously published studies have been done in adults and not during the acquisition of bone mass [11]. The role of fathers has, however, been established, perhaps due to the assumption that osteoporosis is a disease of women, that transmission of the pertinent genetic information is probably through the female sex, or that it is partially sex-linked disease.

To evaluate the effect of heredity on the acquisition of bone mass, we selected 24 parent-daughter pairs [6]. The age range of mothers was 35-56 years and 38-53 years for fathers. The highest correlations in bone mass were found between the mean parents’ values (mother + father) and the daughters’ values. These ranged from 0.53 and 0.46 for height and metacarpal length to 0.72 for distal forearm density and 0.73 for cross-sectional area of the metacarpal. In general, bone size and mass variables correlated more strongly than density variables. There was no correlation between random selection of mother’s, father’s, or mean parent’s variables with that of teenage girls (no biological connection) in this study, which again suggested a hereditary contribution. None of the univariate correlations between daughters and parents accounted for more than about 50% of the variance of the daughters’ variables. This was further, moderately increased by a stepwise regression procedure and canonical analysis. The data from this study supported the hypothesis that peak bone size, mass, and density in young women are influenced by genetic information not only from the mothers but from both fathers as well [8]. It should, however, be mentioned that family resemblance studies cannot completely control for environmental factors (to the degree possible in twin studies), although there was no correlation in bone mass and density between mothers and fathers.

LITERATURE


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6. GENERAL PRACTITIONERS AND PREVENTION OF OSTEOPOROSIS: ATTITUDES, KNOWLEDGE AND BEHAVIOUR (THE DUTCH SITUATION)

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To get a profound understanding of the professional behaviour of GPs in nutritional matters, one has to study not only the content of general practice, the decision-making process of GPs, factors influencing professional behaviour of medical doctors, and the curative nature of GPs, one has also to take into account recent developments regarding the desired shift from curative to more preventive work and what is really happening in this field: The scientific discussions on criteria for preventive action in the Netherlands, the report “Setting priorities in prevention” (1990), the development of standards by the Netherlands Association of GPs and the concept proposal of GPs organizations to the Department of Health regarding a programmatic building of preventive behaviour of GPs.

The attitudes, knowledge and behaviour of GPs regarding prevention of osteoporosis - as far as are known - are summarized, with special attention to the Health Council Report “Prevention of osteoporosis” (1991). The theory of diffusion of innovations (Rogers, 1983) is used to try to predict some future developments.

It is concluded that our knowledge about attitudes, knowledge does not exist an appropriate test or method (simple and cheap) to determine osteoporosis. Both increase of knowledge and translation of knowledge and skills to the practice of GPs is needed. Questions from patients/clients are influential, as are both scientific and other media in their agenda-setting function.

The volume and type of coverage is influencing the discussion. Even when all factors are optimal - which they are not in this case - diffusion of innovations takes time. We need to be realistic about the rate of progress.

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