LABELLING, INFORMATION ASYMMETRY

AND FUNCTIONAL FOODS:

A CASE STUDY OF OMEGA-3 ENRICHED EGGS

A Thesis

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University of Saskatchewan

by

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ABSTRACT


Supervisor: Dr. Richard S. Gray

Currently, the labelling of functional food products is highly regulated in Canada. Although certain nutrient content claims and five generic health claims have been allowed, the inability to make additional nutrient content and health claims decreases functional food firms’ incentives to produce and commercialize new and healthy food products. This, in turn, has consequences for functional food demand, consumer welfare, and health care costs. The primary objective of this thesis is to examine the potential welfare implications of functional food labelling for Canadian society.

A benefit cost analysis is conducted to examine a specific case study of omega-3 enriched eggs. The benefit cost analysis evaluates the welfare effects of functional food labelling policy and helps realize the magnitude of potential benefits that could be gained if not for restrictive and complicated labelling regulations. Based on a range of assumptions and using three different scenarios to cover a range of estimates, the health benefits from the reduction in the risk of coronary heart disease due to the current consumption of omega-3 enriched eggs, and the production and labelling-related costs are estimated. By comparing the estimated benefits and costs, the results indicate that the current consumption of omega-3 enriched eggs provides a considerable net economic gain. Therefore, labelling information on health components can contribute to facilitating a healthy lifestyle with reduced medical costs, stimulating agricultural innovation, and increasing economic welfare.

Realizing the positive overall impact that the current consumption of omega-3 enriched eggs has on consumers’ health and economy in Canada, a possible policy that could regulate all eggs to be enriched with omega-3 fatty acids is proposed. This policy could potentially be used to correct not only information asymmetry but also the negative externalities that are created by health and disability insurances. The benefit cost analysis show that the health benefits would be greatly increased while costs would
slightly increase due to reduced labelling-related costs. Therefore, the results indicate that the net economic gain is even stronger if the mandatory development of omega-3 enriched eggs were required.
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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AA</td>
<td>Arachidonic Acid</td>
</tr>
<tr>
<td>AAFC</td>
<td>Agriculture and Agri-Food Canada</td>
</tr>
<tr>
<td>AHA</td>
<td>American Heart Association</td>
</tr>
<tr>
<td>ALA</td>
<td>Alpha-Linolenic Acid</td>
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<tr>
<td>AMDR</td>
<td>Acceptable Macronutrient Distribution Range</td>
</tr>
<tr>
<td>B/C ratio</td>
<td>Benefit/Cost ratio</td>
</tr>
<tr>
<td>CAD</td>
<td>Coronary Artery Disease</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>CLA</td>
<td>Conjugated Linoleic Acid</td>
</tr>
<tr>
<td>CVD</td>
<td>Cardiovascular Disease</td>
</tr>
<tr>
<td>CFIA</td>
<td>Canadian Food Inspection Agency</td>
</tr>
<tr>
<td>DHA</td>
<td>Docosahexanoic Acid</td>
</tr>
<tr>
<td>EPA</td>
<td>Eicosapentaenoic Acid</td>
</tr>
<tr>
<td>FDA</td>
<td>Food and Drug Administration</td>
</tr>
<tr>
<td>GDP</td>
<td>Gross Domestic Product</td>
</tr>
<tr>
<td>IHD</td>
<td>Ischemic Heart Disease</td>
</tr>
<tr>
<td>IOM</td>
<td>Institute of Medicine</td>
</tr>
<tr>
<td>LA</td>
<td>Linoleic Acid</td>
</tr>
<tr>
<td>LCPUFA</td>
<td>Long Chain Polyunsaturated Fatty Acids</td>
</tr>
<tr>
<td>LDL</td>
<td>Low-Density Lipoprotein</td>
</tr>
<tr>
<td>MUFA</td>
<td>Monounsaturated Fatty Acids</td>
</tr>
<tr>
<td>PUFA</td>
<td>Polyunsaturated Fatty Acids</td>
</tr>
<tr>
<td>R&amp;D</td>
<td>Research and Development</td>
</tr>
<tr>
<td>RR</td>
<td>Relative Risk</td>
</tr>
<tr>
<td>SFA</td>
<td>Saturated Fatty Acids</td>
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<tr>
<td>TFA</td>
<td>Trans Fatty Acid</td>
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CHAPTER 1: INTRODUCTION

1.1. Background Information

Functional foods have entered the global market in the past decade, rapidly gaining market share as value-added products (Kotilainen et al., 2006). Growing consumer awareness about health and nutrition has led to an increased demand for healthier products. Scientific evidence suggests that there are links between diet and disease control and prevention, prompting consumers to search for food products with additional health benefits that decrease the risk of diet-related chronic diseases. The functional food industry has begun to respond to this economic incentive by including healthier products among their offerings, often using product differentiation opportunities. The potential of a niche market of health conscious consumers with large disposable incomes is an important factor that is driving the food industry’s actions (Cash et al., 2006). Canada’s own functional food industry is following this global trend, experiencing considerable growth in the last decade.

The provision of nutrition information is a pivotal factor for further development of the functional food market. Functional foods contain health attributes that are difficult for consumers to observe without proper dissemination of the information, often leading to market failures caused by information asymmetry. Legislative labelling measures—mandatory Nutrition Facts tables, nutrient content and health claims—have been introduced in Canada to address this problem. Labelling is often used to deliver information to consumers about product characteristics that cannot be determined even after consumption. These characteristics are referred to as “credence” attributes. Improving consumers’ means of education concerning functional foods is necessary in order to decrease the information asymmetry problem that exists. Thus, labelling could function as an important tool for building trust and confidence between consumers and producers (Raab and Grobe, 2003).
Labelling helps not only consumers to make healthy food choices but also producers because it is considered valuable advertising. Producers of functional foods can use nutrient content claims and health claims to increase their profits as long as the additional information generates more revenues than it costs (Golan et al., 2001). The larger profit margin of functional foods encourages greater numbers of firms to enter the functional food industry. Hence, nutrient content and health claims provide an incentive to firms to produce healthy goods by assuming that consumers will respond to this new information by changing their purchasing decisions to the firms’ benefits.

Increased labelling information about the value-added attributes of functional foods has the potential to considerably improve the health of Canadians, and it is of critical social interest to achieve reduced health care costs through better food choices. Canada spent an estimated $148 billion, or $4,548 per person, on health care in 2006. After the effect of inflation is taken into account, this was almost three times what was spent in 1975 (CIHI, 2006a).¹ In 2006, government (taxpayers) paid 70.3% ($104 billion) of Canadians’ total health spending, while the private sector paid the remainder, 29.7% ($44 billion) (CIHI, 2006a). The magnitude of the total spending on health care in Canada (both public and private sectors) reached 10.3% of GDP in 2006 (CIHI, 2006b). Therefore, changing eating preferences through labelling can lead to a healthier Canadian labor force and increase overall efficiency in the Canadian economy.

Although considerable agricultural research has been devoted to the development of foods with functional properties, the private sector has inadequate incentives to produce and provide consumers with healthier foods. This is due to an inflexible and complicated regulatory system and its effect on allowable claims for functional food products. The lack of additional nutrient content and health claims is seen as a major issue for the introduction of functional foods. The barriers to dissemination of information regarding products’ health attributes and the public good nature of this information reduces the incentives to private firms to invest in new healthier products (Avery et al., 1999; Feehan, 1998). Currently in Canada, government regulations approve only specific nutrients to be used on nutrient content claims and a limited number of health claims, thus reducing the range of functional foods entering the

¹ Unless otherwise noted, all currency references are in CAN$. 

2
market. These limitations reduce the potential growth of the functional food industry while simultaneously inhibiting social welfare. Consumers need to know the nutritional components of functional foods in order to make healthful eating choices and decrease the incidence of chronic diseases. Therefore, an adaptable and responsive regulatory regime that provides consumers with health information is necessary to increase societal welfare and improve economic efficiency.

The moral hazard problem, which is created due to health and disability insurances concerns, is another factor that reduces functional food firms’ incentives to develop functional food products, and thus may also create market failures. Even informed consumers lack the full incentive to pay for healthier foods and consume optimal quantities of these foods as long as they are protected by either public or private health insurances. Accordingly, there is a reduced incentive to undertake disease prevention measures, and thus firms produce a lesser quantity of functional foods than socially optimal.

1.2. Problem Statement

The labelling of functional foods is highly regulated in Canada. Labelling for nutrient content is restricted to those nutrients that are deemed essential by Health Canada. Functional foods that contain elevated levels of other potentially beneficial compounds cannot be labeled. The use of health claims is also restricted to five pre-approved generic health claims in Canada as opposed to seventeen health claims in the United States (Government of Canada, 2002). While there may be benefits attached to these restrictive regulations on labelling, it is clear that the inability to label functional foods has consequences for its demand, consumer welfare, and health care costs.

Nutrition and health information remains a barrier to the widespread adoption of functional foods. The inability to label many functional foods has created significant barriers to their development and introduction in the Canadian diet. If consumers are not aware of the health benefits of consuming a functional food or unable to identify such foods on a grocery shelf these products will not be sold when they involve additional costs. In general, in the current climate regulations prevent food producers from making
additional nutrient content claims or health claims and from expanding the functional food market.

Labelling can also be used as a vehicle to transfer nutrition information and affect consumption decisions. Therefore, labelling can correct the market inefficiency caused by asymmetric information and contribute to facilitating a healthy lifestyle with decreased medical costs. Federal intervention in food labelling is often proposed with the aim of achieving a social goal. The appropriate role for the government in labelling depends on the type of information involved and the level and distribution of the costs and benefits of providing that information (Golan et al., 2001).

In order to evaluate the potential social and economic effects of labelling regulations in the functional food industry, a benefit cost analysis is presented to examine a specific case study of omega-3 enriched eggs. The current allowable specific nutrient content claims and limited health claims have resulted in a spectrum of functional foods available on the market. Omega-3 enriched eggs provide a good example of where labelling has allowed the introduction of functional food. However, agricultural innovation may be inhibited and health care costs will remain high if the regulatory system does not allow the labelling of other healthy nutrients. If labelling of omega-3 content were not allowed the product would possibly not exist in the market place. This study evaluates the importance of providing information through claims and examines the economic impact of the current regulatory labelling system. It also helps realize that additional health and economic gains are forgone due to restrictive labelling rules for new and potentially healthy functional foods.

1.3. **Objective**

The main objective of this thesis is to examine the potential welfare implications of functional food labelling for Canadian society by using the example of omega-3 enriched eggs. The empirical analysis will estimate the welfare impacts of omega-3 consumption by considering the reduction in the cost of coronary heart disease due to increased consumption minus production and labelling-related costs. The theoretical framework will use a graphical analysis to describe how labelling and nutrition claims on functional foods can reduce information asymmetry. The moral hazard problem that
prevents consumers from consuming the socially optimal amount of functional food will also be briefly analyzed.

1.4. **Hypothesis**

This thesis will examine the following hypothesis: The labelling of omega-3 enriched eggs has not led to benefits for Canadians.

If this hypothesis is rejected, the thesis will demonstrate the potential importance of functional food labelling and the magnitude of potential benefits that could be gained if not for overly restrictive labelling regulation.

1.5. **Organization of Thesis**

The introduction to the thesis highlights the important role that diet plays in consumers’ health and how the labelling, nutrient content and health claims of functional foods can prevent information asymmetry and affect food choices, thus potentially reducing health care costs in Canada. The remainder of the thesis is structured into five chapters.

Chapter Two provides background information regarding the functional food industry in Canada and then discusses the nutritional properties of omega-3 enriched eggs. In understanding these properties, information regarding the beneficial effects of omega-3 fatty acids is provided. The chapter concludes by estimating the direct and indirect costs of cardiovascular disease and coronary heart disease in 2006.

Chapter Three offers a brief description of the current regulatory system and its complications. The chapter also includes the theoretical model of the thesis and the graphical form of the model that is used to show the significance of federal intervention through labelling in the correction of information asymmetry. The magnitude of potential health care costs savings is also discussed.

Chapter Four contains the case study—the omega-3 enriched eggs—that is being used to illustrate the significant beneficial effects of labelling information. The chapter begins with an estimate of current daily omega-3 fatty acids consumption and continues with an estimation of the reduction in coronary heart disease mortality through the increase in omega-3 fatty acid intake. This is followed by an estimation of production, labelling and marketing costs of omega-3 enriched eggs. In this chapter it is shown how
consumers benefit and improve their health by consuming eggs high in omega-3 fatty acids and the degree of health benefits in terms of health savings from this consumption compared to the costs.

Chapter Five discusses the potential policy implications of labelling information. It includes the example of all eggs being mandated to be rich in omega-3 fatty acids. A benefit cost analysis is conducted to evaluate the overall impact if this policy were implemented.

Lastly, Chapter Six offers a summary of the thesis and concludes with suggestions for further research.
CHAPTER 2: INDUSTRY BACKGROUND

2.1. Introduction

The purpose of this chapter is to provide an overview of the functional food industry in Canada. It also focuses on the healthy characteristics of a functional food—omega-3 enriched eggs—and estimates cardiovascular disease costs in 2006, which will be used in the benefit cost analysis conducted in Chapter 4. The chapter begins with background information regarding functional foods and the factors that contribute to their market growth. Examples of functional foods showing their potential health benefits are provided. The role of nutrient content information on labels is also discussed, which explains how labels can decrease the information asymmetry problem caused by the credence attributes of functional foods. The chapter then provides information regarding the health benefits associated with omega-3 fatty acids and shows their importance to health. The chapter concludes with an estimation of cardiovascular disease (CVD) costs and coronary heart disease (CHD) costs, which can be considerably reduced by consuming a product with omega-3 fatty acids.

2.2. Functional Foods: Background Information

The fact that food is closely correlated with optimal health is not a novel concept. The tenet, “Let food be the medicine and medicine be the food,” espoused by Hippocrates, the father of medicine, shows the connection between nutrition and human health (Hippocrates, cited by Klotzbach et al., 1999). Almost 2,500 years later this philosophy is receiving renewed interest and underpins the introduction of functional foods in the marketplace (Hasler, 1998).

The concept of functional foods was first developed in Japan in the mid-1980s. At that time, the government and health authorities in Japan considered the increased consumption of specific food types and an increased quality of life as important factors
for reducing the risk of chronic diseases, thereby helping control the rising health care costs, particularly in an aging population. (FSAI, 2006). The Japanese government launched a program to promote the development of foods with health and medicinal properties that would help Japanese citizens to maintain and improve their state of health. Since then, functional food science has been developed in a number of countries and accepted as being conceptually beyond nutrition (Arai et al., 2001).

In recent years, consumers, governments and the agri-food industry have shown a growing interest in foods with health enhancing properties due to several critical factors (Benkouider, 2003; Drouin et al., 2002):

1. an increased amount of information from health authorities and the media to consumers on nutrition and the link between diet and health;
2. continuously increasing health care costs;
3. consumers’ desires for an improved quality of life and a healthier lifestyle;
4. an aging population;
5. rapid advances in food science and technology;
6. increased incidence of self-medication; and
7. a changing regulatory environment.

All the above factors have contributed to the development of the functional food industry in Canada. However, one of the most important factors, from an economic perspective, is rising health care costs. The health care system in Canada is under pressure because the expenditures associated with health services tend to grow every year due to diet-related chronic diseases in an aging population (i.e., cardiovascular disease, coronary heart disease, cancer, diabetes and obesity). The development of functional foods could reduce the risk of chronic diseases and enhance the ability to manage chronic diseases, thus, potentially decreasing health costs.

Currently, some people, especially those over forty-five years of age, are very concerned about the correlation between diet and disease, and they have recognized that making healthy food choices can improve their health. This demand for foods with beneficial health attributes has provided an incentive for the functional food industry’s development in Canada. Thus, substantial improvements in quality of life and economic
savings are possible through the use of beneficial functional foods. However, consumers have to keep in mind that functional foods are only one component of improving their health, as other components, including exercise, reduced stress, and not smoking, can have a major impact on health.

Conceptually, functional foods fall in the area between foods and medicine (Kotilainen et al. 2006) because they are foods with specific health-promoting properties. In an effort to distinguish between functional foods and drugs, the term nutraceutical was coined in 1989 by the Foundation for Innovation in Medicine (Brower, 1998). Despite some confusion, functional foods and nutraceuticals are different categories, compete in different markets and target different consumer preferences, needs and characteristics. At the retail level, functional foods compete for market share with the food industry whereas nutraceuticals compete with the pharmaceuticals, vitamins and supplements.

Although the terms functional food and nutraceutical are commonly used interchangeably, there are no internationally accepted definitions for them. In 1998, the Health Protection Branch of Health Canada released a policy paper that proposed a definition for functional foods and nutraceuticals (Health Canada, 1998a).

Health Canada has defined functional foods as follows:
A functional food is similar in appearance to conventional foods, is consumed as part of a usual diet, and has demonstrated physiological benefits and/or reduces the risk of chronic disease beyond basic nutritional functions (Health Canada, 1998a, p. 3).

According to Health Canada, the definition of nutraceuticals differs substantially from that of functional foods:
A nutraceutical is a product isolated or purified from foods that is generally sold in medicinal forms not usually associated with food. A nutraceutical is demonstrated to have a physiological benefit or provide protection against chronic disease (Health Canada, 1998a, p. 3).

In January 2004, the term natural health product was introduced in Canada and seems to be a term that is unique to Canadian legislation (Canada Gazette, 2003a).
Natural health products are a combination of various types of products, including nutraceuticals, vitamins and minerals, herbal remedies, homeopathic medicines, traditional Chinese medicines, probiotics, amino acids and essential fatty acids (Canada Gazette, 2003a). Recent surveys have shown that more than one-half of Canadian consumers regularly take natural health products indicating consumers’ self-care for and interest in their health (Canada Gazette, 2003a). The regulations for natural health products exclude food, meaning that, unlike nutraceuticals, functional foods are not included in this category.

As indicated, the terms “functional foods” and “nutraceuticals” cannot be used interchangeably. Not only do they compete in different markets but they also are governed by different regulations regarding labelling and health claims. The claims used for nutraceuticals, which are similar to pharmaceuticals, are more direct and specific, while those used for functional foods are more general statements that aim to improve or maintain the population’s overall health, not to cure or treat a certain disease. More information about the labelling and claims that are currently allowable in Canada is covered in section 3.2.

2.2.1. Functional Foods

Although most foods consumed worldwide serve some function, in recent years the category of functional foods has come to include products that influence specific functions in the body and thereby beneficially affect people’s health and well-being. The field of functional foods contains two categories. The first includes a wide spectrum of foods that inherently contain functional components, thus the foods are unmodified, whereas the second category contains foods that are the result of technological innovation (Institute of Foods Technologists, 2007).

Considerable scientific research has demonstrated that some substances (nutrients and non-nutrients) that occur naturally in food have noteworthy protective or disease preventive properties. Phytochemicals and zoochemicals have received a lot of attention due to their health-promoting attributes. Both are non-nutrient, physiologically-active functional food components, that are present in plant and animal materials, respectively,
in relatively small amounts (micronutrients) compared to macronutrients (fats, fiber, carbohydrates, proteins, and amino acids) (Unnevehr and Hasler, 2000; Hasler, 2000).

Foods containing phytochemicals such as grains, legumes, fruits and vegetables, are already part of our daily diet. There is abundant evidence from epidemiological studies that the phytochemicals in fruits and vegetables can considerably reduce the risk of cancer, probably due to polyphenol antioxidants (Heber, 2004) and anti-inflammatory effects (Urquiaga and Leighton, 2000). For example, lycopene in tomatoes is a phytochemical that acts as an antioxidant and has been shown to lower the risk of prostate cancer (Giovannucci, 1999). Cruciferous vegetables such as broccoli and Brussels sprouts contain glucosinolates, a metabolite, which are synthesized from amino acids and may reduce the risk of cancer (Wang et al., 2002). The risk of cardiovascular disease can be reduced through consumption of certain foods, such as green tea, red wine, berries, grapes and soy foods (Zimmerman, 2000).

In addition to phytochemicals, zoochemicals are found in animal sources and are also known for their healthful benefits. Omega-3 fatty acids—eicosapentaenoic acid (EPA) and docosahexanoic acid (DHA)—are found in abundance in cold-water fish such as salmon, trout, mackerel, and tuna (IFIC, 2003). These compounds contribute to protection from heart disease, improvement of mental functioning and maintenance of good vision. Conjugated Linoleic Acid (CLA) is naturally present in particular meats, such as beef, and dairy products, and can also help reduce the risk of heart disease, lower body fat, and increase muscle strength and bone mass (Kreider et al., 2002). Lastly, egg yolk contains a substantial amount of lutein and zeaxanthin, antioxidant carotenoids that have been linked to reducing the risk of cataracts and age-related diseases of the eye (macular degeneration) (Mares-Perman et al., 2002).

Besides conventional foods that contain natural ingredients that boost health, several food products that result from technological innovation have been introduced in the market. A variety of functional foods have been commercially developed specifically recently to cover consumers’ nutritional needs and prevent serious chronic diseases. The term “industrial functional foods” will hereafter be used for these foods and defined as follows:
Industrial functional foods are the foods which can be produced by either adding, removing, enhancing, decreasing or modifying one or more components of a food and require an additional marginal cost to introduce a health benefit.

In the current Canadian market there are a variety of fortified or enhanced foods that are specifically created to reduce disease risk. Some well-known examples include calcium-enriched orange juice, cereals with soya, grains with added fiber and eggs enhanced with omega-3 fatty acids.

A list of functional foods, natural and industrial, is presented in Table 2.1. The table shows some of the available functional foods and their functional components. It also shows the potential health benefits that can be obtained by regularly consuming those foods.
Table 2.1. Examples of functional components.

<table>
<thead>
<tr>
<th>Components</th>
<th>Source</th>
<th>Potential Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lutein, Zeaxanthin</td>
<td>kale, collards, spinach, corn, eggs, citrus</td>
<td>may contribute to maintenance of healthy vision</td>
</tr>
<tr>
<td>Lycopene</td>
<td>tomatoes and processed tomato products, watermelon, red/pink grapefruit</td>
<td>may contribute to maintenance of prostate health</td>
</tr>
<tr>
<td>Beta glucan</td>
<td>oat bran, oatmeal, oat flour, barley, rye</td>
<td>may reduce risk of coronary heart disease (CHD)</td>
</tr>
<tr>
<td>Monounsaturated fatty acids (MUFAs)</td>
<td>tree nuts, olive oil, Nexera canola oil</td>
<td>may reduce risk of CHD</td>
</tr>
<tr>
<td>PUFAs-Omega-3 fatty acids-DHA/EPA</td>
<td>salmon, tuna, marine, and other fish oils</td>
<td>may reduce risk of CHD; may contribute to maintenance of mental and visual function</td>
</tr>
<tr>
<td>Conjugated linoleic acid (CLA)</td>
<td>beef, lamb, some cheese</td>
<td>may contribute to maintenance of desirable body composition and healthy immune function</td>
</tr>
<tr>
<td>Sulforaphane</td>
<td>cauliflower, broccoli, broccoli sprouts, cabbage, kale, horseradish</td>
<td>may enhance detoxification of undesirable compounds; bolsters cellular antioxidant defenses</td>
</tr>
<tr>
<td>Stanol/Sterol esters</td>
<td>fortified table spreads, stanol ester dietary supplements</td>
<td>may reduce risk of CHD</td>
</tr>
<tr>
<td>Soy Protein</td>
<td>soybeans and soy-based foods</td>
<td>foods may reduce risk of CHD</td>
</tr>
<tr>
<td>B9 (Folate)</td>
<td>beans, legumes, citrus foods, green leafy vegetables, fortified breads and cereals</td>
<td>may reduce a woman’s risk of having a child with a brain or spinal cord defect</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>sunlight, fish, fortified milk and cereals</td>
<td>helps regulate calcium and phosphorus; helps contribute to bone health; may contribute to healthy immune function; helps support cell growth</td>
</tr>
</tbody>
</table>
Some health-conscious consumers have realized that the development of functional foods could prevent the risk of lifestyle chronic conditions, and most consumers agree that eating healthily is a better way to manage illness through prevention rather than using medication (Hasler, 2002). This has led to the increased acceptance and consumption of functional foods with health-promoting capabilities, something demonstrated by impressive growth in sales worldwide (Menrad, 2003).

2.3. **Functional Food Industry in Canada**

New product development has increased at a rapid rate and the sales of functional foods have risen worldwide. However, the lack of a formal definition for functional foods makes it difficult to estimate the size of the market (MacDonald, 2004). According to Nutrition Business Journal, in 2006 the estimated size of the global functional food market was US$85 billion, or 37.6% of the global nutrition industry (US$226 billion) (Nutrition Business Journal, 2007). The major markets for functional foods and nutraceuticals are located in the US, Europe, Japan and Asia (Newton, 2001). In 2001, the Canadian market represented approximately 3% of the global nutrition market (US$6.8 billion) (Jarvis et al., 2001). Functional foods accounted for 42% of the US$6.8 billion, which means that almost US$2.9 billion were spent on functional foods in Canada in 2001. Canada’s functional food and nutraceutical industry has the potential to significantly increase its market share and develop due to its innovative capabilities.

Research and development is critical to this rapidly developing field. Key players in the research of functional food products include governments, the academic community, institutions, and industry, all of which work together to advance collective interests (AAFC, 2005).

The results from a functional foods and nutraceuticals survey conducted by Statistics Canada indicated that there were 389 firms, ranging from small start-up companies to multinational organizations, engaged in activities related to either functional foods, nutraceuticals, or both during 2004-05 (Statistics Canada, 2007a). One-

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2 The segments of the nutrition industry are dietary supplements (vitamins, minerals, herbs & botanicals, sports nutrition, specialty supplements), natural and organic food, natural and organic personal care and household products and functional foods (Nutrition Business Journal, 2007).

3 In 2004, a Global market review of functional foods estimated that 41% of the US$6.6 billion dollar market is spent on functional foods (Global market review of functional foods-forecasts to 2010, 2004).
quarter of these companies were involved with both functional foods and nutraceuticals, while 30% of them were only involved in functional food activities. This survey showed that the industry was gaining momentum as, compared to the results of a 2002 survey, the number of functional food and nutraceutical firms increased by 32.3% (Statistics Canada, 2005). The industry also generates about $2.9 billion in revenues and employs over 12,000 individuals with functional food- and nutraceutical-related duties (Statistics Canada, 2007a). Firms with functional food- or nutraceutical-related activities spent $74.5 million in 2004 on research and development (R&D), which accounted for almost half the total funds spent on R&D by these firms. The fact that they would invest significant amounts in R&D for functional foods and nutraceuticals indicates that they considered these products and processes as important to their long-term competitiveness (Statistics Canada, 2007a).

Canada’s functional food and nutraceutical sector is experiencing considerable growth due to a high demand for the development of nutritious products. The food and nutraceutical companies focus heavily on R&D to produce commodities with value-added ingredients. The industry range from globally recognized companies such as Ocean Nutrition Canada (omega-3 fatty acids), Bioriginal Food and Science (essential fatty acid oils), Forbes Medi-Tech (plant sterols), and Institut Rosell (probiotics) to major multinational corporations such as Kellogg’s, Heinz, Quaker, Unilever, Dupont, Novartis, Cargill, and Hormel, which are also active in this sector (AAFC, 2005). It is also worth mentioning that Canada is a leader in the development and manufacturing of essential fatty acid (EFA) products from plant and marine sources, including evening primrose oil, flaxseed, borage, hemp, and marine animal oils (AAFC, 2005).

Functional foods and nutraceuticals offer a special opportunity for the Canadian agri-food industry to develop in both domestic and international markets (AAFC, 2005). The agriculture industry is an important contributor to the Canadian economy, and companies invest in research and development to produce commodities with nutritionally valuable constituents. Thus, the functional food industry, in collaboration with the research community, has the potential to stimulate growth in the value-added agri-food sector.
However, while the market for functional foods is increasing worldwide, the long-term success of functional foods is not yet known for it depends on their effectiveness in reducing the risk of a disease, their safety, and consumer confidence in the health benefits they provide. Consumers will trust the beneficial effects of functional foods only when proper information is provided. What consumers need is truthful and credible information in order to make informed food choices. The next part of this chapter discusses the information asymmetry problem faced by the functional food industry.

2.4. Functional Foods and Information Asymmetry

Recently, Canada’s agri-food sector has experienced major challenges as demand has increased for products with health promoting capabilities. In such markets where conditions are changing rapidly, the flow of information to consumers becomes a primary determinant of economic welfare. However, this market information is often imperfect, incomplete, inaccessible, asymmetrically distributed, non-standardized, or costly to collect (Caswell and Mojduszka, 1996). Hence, potential market failures from information asymmetry arise because consumers face uncertainty regarding the true nature of product attributes, and as a result they make choices that are not well aligned with their preferences (Teisl and Roe, 1998).

According to Nelson (1970) and Darby and Karni (1973), there are three types of goods available in the consumer market. Search goods are those whose characteristics (price, colour, size) can be determined and evaluated prior to purchase. An example of this would be the purchase of a pair of shoes or a dress, as the consumer can search for the price, select the preferable color, and try it on before buying it. Experience goods are those whose characteristics (taste, durability, quality) are difficult to observe in advance and can only be ascertained after purchase and consumption. Some examples of this type are a can of tuna, a bottle of milk, a jar of jam, tomatoes, or meat. While consumers can only identify the true quality of a product after consuming it, the reputation of the seller, a brand image, or a quality guarantee become important quality signals for the purchaser (Hobbs, 1998). Lastly, credence goods are those goods whose quality, safety, and effectiveness cannot be adequately assessed even after the products are purchased and
consumed. Thus, individuals face some degree of uncertainty that cannot be factored into purchasing decisions unless there is perfect information (Bureau et al., 1997). Examples of these include some services, such as those of a mechanic, the expertise of a doctor, or the nutritional or health component of a food product (Hobbs, 1998).

Functional foods fall into the category of credence goods as they contain nutritional characteristics that cannot be identified by the consumers even after experiencing the good. This has a number of implications for consumer assurance of product efficacy and safety, the possibility of consumer fraud, and the potential for misleading representations of product quality and effectiveness (Akerlof, 1970; Nelson, 1970; Darby and Karni, 1973; Leland, 1979). For example, consumers cannot detect the fiber contained in oats or the omega-3 fatty acids included in milk without the provision of proper and credible information. The role of information is crucial because consumers cannot directly perceive the benefit, such as through taste (Urala and Lähteenmäki, 2004).

Buyers have less knowledge than sellers (e.g., concerning the production process, product origin, nutritional content, or safety issues), which means that information is asymmetrically distributed (Verbeke, 2005). One possible effect is that sellers have incentives to exaggerate, mislead, or lie in their claims. As a result of the small supply of information, consumers are unable to make choices that reflect their preferences. In other cases, relevant market information may be imperfect (i.e., it may not exist or it may be contradictory). This situation often arises when the long-term health effects of a food or food attributes are unknown, or when scientific opinions differ about the health consequences of consumption (Golan et al., 2001). This occurred in the early days of the BSE and dioxin crises, where scientists and government held opposing views on the potential health risks (Verbeke, 2005).

Dissemination of information aims at reducing problems resulting from information asymmetry (Rabinowicz, 1999; Lusk et al., 2004; Hobbs, 2004). Logically, if asymmetric information is at the core of market failures, it can reasonably be assumed that better information and more transparency will be at the core of any solution (McCluskey and Swinnen, 2004). Information regarding the nutrition attributes and health effects of functional foods is the key factor behind the success of the functional
food market. Such information should be communicated clearly, understandably, and truthfully to consumers for there is a need to understand both a product’s features and its associated health benefits. When consumers become aware of the link between attributes and beneficial consequences, they show stronger purchase intentions. Thus, the high societal costs of diseases related to food consumption can be significantly decreased due to proper and full provision of information.

In situations where asymmetric information is unavoidable and causes market inefficiencies and failures, a government can either provide information or impose regulations to compel manufacturers to do so (Cash et al., 2004). Individuals cannot make rational food choices without complete information. They are also daily faced with an abundance of health information that is difficult to understand and evaluate (Cash et al., 2004). Thus, they need information about the implications of new research and products from sources on which they can trust and rely (Unnevehr et al., 1999). A wide variety of government nutrition programs provide consumers with information and education in order to improve their health by changing their diets (Aldrich, 1999). Government is also responsible for the regulation of advertising and food labelling, which can influence food consumption decisions.

Labelling is an effective policy tool that can enhance economic efficiency by delivering information to consumers on nutritious attributes and health-enhancing properties of functional foods. Consumers can now use the information found on product labels to select nutritious goods and ameliorate their well-being. A healthier lifestyle in well-nourished citizens can decrease market inefficiencies related to burgeoning health care costs.

At the same time, producers can use labelling as an advertising tool. Producers have an economic incentive to produce healthy and nutritious products because many consumers are increasingly interested in selecting a nutritious and balanced diet to maintain or improve their health. That the demand for functional foods is continuously increasing will stimulate producers to provide information in order to increase sales. Consequently, labelling can be beneficial for both consumers and producers.
Generally, labels are designed to serve four main objectives:

1. to promote fair competition among producers and product marketability (Hadden, 1986);
2. to increase consumer access to information (Hadden, 1986);
3. to reduce risks to individual consumer safety and health (Hadden, 1986); and
4. to influence individual consumption choices so as to align them with social objectives (Golan et al., 2001).

These objectives can only be achieved if the information is clear, credible, and comprehensible to assist Canadians in making appropriate food choices, thus achieving a social goal. Format and context of the information are important elements in maximizing the potential of labeled information to influence its audience (Golan et al., 2001). Consumers are more likely to pay attention to a label only if its information is explicit, concise, and understandable. Consumer surveys have shown that packages with overly detailed labels and numerous warnings tend to affect consumers negatively and may cause them to ignore the information completely (Golan et al., 2001). Even if consumers are interested in the information on the label, they may have difficulty extracting the important information. As a result, overloading a label in an attempt to solve market inefficiencies may result in consumer confusion, indifference, or loss of confidence due to poor understanding (Verbeke, 2005).

Labelling is even more effective when it is accompanied by consistent and achievable standards, testing services, certification services, and enforcement. These third-party services can be provided by several different entities, such as national governments, international organizations, consumer groups, producer groups, and private entities (Golan et al., 2001). The provision of these services aims at reducing transaction costs (i.e., search, negotiation, and monitoring costs) and increasing the benefits of labelling by strengthening labelling claims. Thus, they enhance the credibility of product labels and claims, hopefully making consumers more willing to consume the functional foods.

Although the provision of truthful and accurate information is currently regulated, there are a variety of demographic factors that play a fundamental role in the use of
labels. Demographic factors such as gender, education level, nutritional knowledge, income, age, and risk perceptions induce the provision of labelling information and can affect interest in food and health issues among members of society (Mueller, 1991). Women more than men, for example, tend to be aware of nutrition issues and pay attention to healthy ingredients of a product. This can be explained by the high levels of responsibility that women often have for several aspects of food preparation and consumption, as well as their increased interest in a healthy diet (Worsley and Scott, 2000). Furthermore, nutritionally informed consumers and consumers with high disposable incomes show a greater interest in the consumption of better quality and healthier foods. Elderly and middle-aged consumers are also more concerned about eating healthily than younger people because they tend to experience more life-threatening conditions that are not fully under their control (Lester, 1994). Lastly, it has been noticed that people who have a family member with a diet-related health condition tend to be very concerned about their own diet and select foods with nutritious attributes.

The information on the labels aims at satisfying various consumers’ needs and concerns about the food. People seek information on both the nutritional content and the health benefits of a product in order to achieve healthier lifestyle and prevent illnesses related to diet. For this reason, Canadian regulatory authorities have approved “nutrition labels,” “nutrient content claims,” and “health claims” that have the potential to contribute to the achievement of public health objectives and correct the market failures caused by asymmetrically distributed information (Hawkes, 2004). For instance, omega-3 fatty acids can be labeled due to their multi health-promoting attributes (see section 2.5), and so several foods rich in these beneficial fatty acids have been developed and introduced in the Canadian market.

2.5. Omega-3 Fatty Acids

There is strong scientific evidence showing that omega-3 fatty acids can beneficially affect various functions in the human body and thus improve the population’s collective health. This section begins with a discussion of the important role of omega-3 fatty acids on health. In particular, the health impacts of each of the three major components of omega-3 fatty acids are discussed separately. Also, given the low
level of omega-3 fatty acids observed in western populations like Canada (Simopoulos, 1991), recommendations were established in order to reduce the risk of developing threatening diet-related conditions (Kris-Etherton et al., 2002). Currently, omega-3 fatty acids are considered beneficial nutrients and are approved for insertion on both labels and nutrient content claims. Thus, several products enhanced with these beneficial fatty acids are now available in the market. Omega-3 enriched eggs, which represent a major functional food, have garnered considerable attention due to their increased levels of omega-3 fatty acids. The consumption of these eggs can substantially contribute to increased levels of omega-3 fatty acids, thereby contributing to increased wellness.

2.5.1. The Importance of Omega-3 Fatty Acids to Health

There are three major categories of dietary fatty acids: saturated fatty acids; monounsaturated fatty acids; and polyunsaturated fatty acids, each of which are distinguished by their chemical structure. Saturated fatty acids (SFA) contain no double bond, are solid at room temperature, and are usually derived from animal sources (e.g., lard or butter). However, most plant fats are high in monounsaturated (MUFA) fatty acids, which contain one double bond, and polyunsaturated fatty acids (PUFA), which contain two or more double bonds; both are liquid at room temperature (Erasmus, 1986, p. 29). Currently, the role of omega-3 polyunsaturated fatty acids in growth and development as well as in health and disease prevention—particularly coronary heart disease (CHD)—is one of the fastest growing research areas in nutritional science. As a result, knowledge of these fatty acids has grown significantly (Simopoulos, 1999a). This section focuses on omega-3 polyunsaturated fatty acids and their impact on ameliorating the health of the population.

In order to better understand the importance of omega-3 fatty acids in health, two distinct families of PUFA, omega-3 (n-3), and omega-6 (n-6) fatty acids are discussed. The principal fatty acids of the omega-3 and omega-6 families, which are alpha-linolenic (ALA, 18:3n-3) and linoleic (LA, 18:2n-6) fatty acids, respectively, are considered essential fatty acids because they cannot be synthesized by the body. Hence, they must be supplied through diet (Simopoulos, 1991) or supplementation (Covington, 2004). They are important components of cell membranes (Simopoulos, 1991) and play a fundamental
role in several physiological functions. Both can be metabolized to their longer chain and highly unsaturated fatty acids (LCPUFA) through elongation and desaturation. ALA can, to a limited extent, be converted in the body to eicosa-pentaenoic (EPA, 20:5n-3) and later to docosahexaenoic (DHA, 22:6n-3) fatty acids, while LA can be converted to arachidonic acid (AA, 20:4n-6) (Simopoulos, 1991). Although ALA and LA are not interconvertible and are metabolically and functionally distinct (Simopoulos, 2002), they compete for the same enzyme systems for desaturation into their LCPUFA (Jacobsen, 2004). This means that the excessive consumption of foods rich in omega-6 may compromise the conversion of ALA to LCPUFA, which in turn adversely affects health (Davis, 2002). Therefore, the right balance between the intake of omega-6 and omega-3 fatty acids is of vital importance (Jacobsen, 2004).

A proper choice of a variety of food products can ensure the appropriate intake of omega-3 and omega-6 PUFA and prevent potential incidences of disease. ALA is present in vegetable oils (e.g., soybean, hempseed, and canola oil), with flaxseed having the highest concentration—57% of total fatty acids (Flax Council of Canada, 2003a). It is also found in green leafy vegetables (e.g., spinach, purslane, and kale,) and walnuts (Covington, 2004). Fish, especially fatty fish such as salmon, tuna, mackerel, halibut, and herring, and fish oils are rich sources of EPA and DHA (Covington, 2004). Additionally, algae and seaweed contain abundant quantities of these two LCPUFA. Although much of the current scientific research has concentrated on the health effects of fish, it is not yet confirmed whether there are functional differences between plant and marine PUFA.

Lastly, LA is found primarily in vegetable and plant oils, such as corn, safflower, soybean, and sunflower, whereas AA is found in animal products, such as meat, poultry, and eggs (Fitzpatrick, 2005).

Nowadays, omega-3 fatty acids have captured considerable attention due to their health-promoting characteristics, which assist in prevention and management of chronic diseases such as coronary artery disease, hypertension, inflammatory and autoimmune disorders, and cancer (Simopoulos, 1991). The link between omega-3 fatty acids and low occurrence of CHD became apparent in the 1970s when three investigators observed a low rate of CHD events in Greenland’s Eskimos despite a diet rich in fat (Bang et al., 1980). The researchers proposed that the Eskimos’ diet consisted largely of marine
animals (e.g., fish, seal, and whale), known to have a high content of LCPUFA. The same conclusion was drawn from Hirai et al. (1980) and Kagawa et al. (1982), who found a low incidence of CHD among Japanese people residing in fishing villages. After these observations were made, additional scientific investigations, although not unambiguous, confirmed the beneficial impact of omega-3 fatty acids on health and particularly on the heart.

ALA is of particular interest for its role in heart health. It has been observed that populations with higher intake levels of ALA have a low risk of cardiovascular diseases such as coronary heart disease (CHD) and stroke (Flax Council of Canada, 2003b). Also, clinical studies have examined the effect of ALA and reported a substantial decrease in blood total cholesterol and low-density lipoprotein (LDL) cholesterol, which are risk factors for CHD (Chan et al., 1991). Furthermore, it has been observed that the Mediterranean diet, which consists of a variety of healthy food products with high levels of ALA helps maintain a balance between omega-3 and omega-6 fatty acids. Many studies have demonstrated that people who follow a diet rich in omega-3 foods, including whole grains, fresh fruits and vegetables, and olive oil, as well as moderate wine consumption, are less likely to lead to any heart disease event (de-Lorgeril et al., 1999; Singh et al., 2002). This is because ALA has been shown to increase blood clotting time (McDonald et al., 1989; Weaver et al., 1990), and so can reduce the likelihood of thrombosis and hence a CHD risk.

Mounting evidence also supports the beneficial effects of EPA and DHA. EPA serves as direct precursor to eicosanoids, a group of biologically active compounds that provide strong antiarrhythmic and antithrombotic actions on the heart and have demonstrated anti-inflammatory properties (Flax Council of Canada, 2003b; Lutter and Tucker, 2002). These properties may help prevent or reduce the symptoms of rheumatoid arthritis (Horrobin, 1987; Recht et al., 1990; Shapiro et al., 1996) and asthma (Broughton et al., 1997). They are also required to help repair damaged tissue (Flax Council of Canada, 2003b). However, different eicosanoids derived from AA are biologically active in very small amounts, and if they are formed in large amounts (Simopoulos, 1991) they tend to promote inflammation and increase blood pressure, platelet aggregation, and cell proliferation (Lee et al., 1993; Chow, 1993). For this reason, adequate amounts of
omega-3 fatty acids, which produce beneficial eicosanoids, are necessary for a healthy diet.

DHA, the second major LCPUFA, is necessary for proper growth and development (Flax Council of Canada, 2003b). Specifically, it is naturally highly concentrated in the brain and the retina (Duque, 1997), and appears to play a prominent role in promoting cognitive function and eye health. Therefore, the brain and retina are dependent on a continuous DHA supply for optimal function (Salem et al., 2001). DHA also assists in nervous system development (Uauy et al., 1996). The demand for DHA is highest during the latter part of pregnancy and infancy (Ghebremeskel et al., 2000) because it is regarded as essential for the proper visual and neurological development of infants (Nettleton, 1993; Crawford et al., 1997; Das and Fams, 2003). Furthermore, DHA reduces or inhibits risk factors involved in various diseases, such as cardiovascular diseases (Kromann and Green, 1980; Kang and Leaf, 1996; Nordøy et al., 2001). Finally, it is well established that LCPUFA reduce blood triglyceride levels (Weber and Raederstorff, 2000) and hypertension (Leaf and Weber, 1987), which are considered important cardiovascular risk factors. To sum up, DHA together with EPA can provide preventive effects on chronic diseases and contribute to maintenance of physical and mental well-being.

Attention must be paid, however, to the amount of the daily omega-3 intake, particularly EPA and DHA. Consuming massive amounts of marine omega-3 fatty acids may harm consumers’ health. The American Food and Drug Administration (FDA) has recommended that consumers limit their intake of EPA and DHA to 3 grams per day, with no more than 2 grams coming from dietary supplements (Tarantino, 2006). This ruling includes specific consideration of the reported effects of omega-3 fatty acids on glycemic control in patients with diabetes, on bleeding tendencies, and on low-density lipoprotein (LDL) cholesterol levels (Tarantino, 2006).

2.5.2. Attempts to Increase Omega-3 Fatty Acids in Canada

It has been observed that the current level of omega-3 fatty acids in the typical diet of industrialized countries is extremely low relative to the level of omega-6 fatty acids, which exist in excessive amounts (Simopoulos, 1991; Mantzioris, 1995). Increased
consumption of meats and LA-rich vegetable oils and declining vegetable and fish consumption can lead to an imbalance in the ratio of omega-6 to omega-3. The current ratio of omega-6 to omega-3 intake in Western Europe and the United States ranges from 15:1 to 20:1 (Simopoulos, 2001) compared with an estimated ratio of 1:1 during human evolution (Simopoulos, 1991; Eaton and Konner, 1985; Leaf and Weber, 1987). This imbalance, along with an omega-3 fatty acids deficiency, is associated with increased risk of serious health diseases.

Concerned about this imbalance of omega-6 and omega-3 in the diet and its potential negative effects on health, a number of countries (Canada, Sweden, United Kingdom, Australia, Japan) as well as the World Health Organization and North Atlantic Treaty Organization have issued formal population-based dietary recommendations for omega-3 fatty acids (Kris-Etherton et al., 2002) to encourage a more balanced ratio of omega-6 and omega-3 fatty acids that would optimize the benefits of both fatty acids (Jacobsen, 2004). The Institute of Medicine (IOM) of the National Academies, in partnership with Health Canada, established the Dietary Reference Intakes (DRIs)\(^4\) for Energy and Macronutrients (Health Canada, 2005; Institute of Medicine, 2002). The Acceptable Macronutrient Distribution Range (AMDR) for ALA is estimated to be 0.6-1.2% of energy, or 1.3 to 2.7 g/d on the basis of a 2000-calorie diet (Kris-Etherton, 2002). Additionally, up to 10% of the adequate intake for ALA can be provided by EPA and DHA (Health Canada, 2005). No specific recommendations were set for EPA and DHA as IOM indicated that LCPUFA can contribute to the recommended ALA intake (Institute of Medicine, 2002). The lower boundary of the range is based on an Adequate Intake (AI) set for ALA, which represents median intake levels that prevent an essential fatty acid deficiency, while the upper boundary corresponds to the highest ALA intakes from foods consumed by individuals in the United States and Canada (Kris-Etherton et al., 2002). The estimated recommended intake of LA is 5-10% of energy (Health Canada, 2005). In order to meet the recommended intakes of omega-3, AHA dietary guidelines suggest consuming two fish meals per week, with an emphasis on fatty fish (i.e., salmon, herring, and mackerel), and using liquid vegetable oils containing ALA (Kris-Etherton et

\(^4\) The Dietary Reference Intakes replaced the U. S. Recommended Dietary Allowances (RDAs) and the Canadian Recommended Nutrient Intakes (RNIs).
al., 2002), while simultaneously restricting the consumption of food products with high LA content. These recommendations intend to provide guidance in the selection of a diet that will include recommended amounts of omega-3 and omega-6, and reduce the risk of developing diet-related chronic diseases. According to Health Canada, the ratio of omega-6 to omega-3 fatty acids is recommended to a range between 4:1 and 10:1 (Health and Welfare Canada, 1990) in order to reduce the competitive influence of high LA intakes on ALA metabolism to its LCPUFA (Holub, 2002), and thus achieve an appropriate balance of these fatty acids.

Although recommendations to increase the omega-3 intake in diets were made, it seems difficult to change the dietary pattern of western populations, which is characterized by foods high in refined carbohydrates, saturated and trans fatty acids, and vegetable oils high in omega-6 fatty acids but which contain little omega-3 fatty acids (Simopoulos, 2003). It was observed that people generally have extremely low levels of LCPUFA in relation to ALA due to declining consumption of seafood. Many people do not enjoy the taste of fish and believe that they can still get the appropriate intake of LCPUFA by consuming vegetable oils with high ALA content. However, even though ALA can be converted to EPA and DHA, the conversion is modest and controversial (Kris-Etherton et al., 2002). For example, Pawlosky et al. (2001) estimated a 0.2% conversion, whereas Emken et al. (1994) found 15%.

In order to assist consumers in boosting their omega-3 intake, new food choices are currently available in the consumer market all over the world. Their potential health benefits, along with the ability to label omega-3 fatty acids, have encouraged the food industry to introduce more omega-3 fatty acids in the food supply through the development of food products enriched in these fatty acids. Genetic modification has been used to develop a new generation of plants (e.g., canola, corn and soybeans) that produce seeds with an increased omega-3 fatty acid profile (Gebauer et al., 2006) or to enhance the levels of EPA and DHA in flax (Fitzpatrick, 2005). The aquaculture industry is also growing as the demand for fish oil for fish feed production and human consumption is increasing (Jacobsen, 2004). Furthermore, spreads (e.g., margarines and mayonnaise) and salad dressings are becoming common products with increased levels of omega-3 fatty acids. Other new and innovative omega-3 enriched food products include
juices, bread, cereals, snack bars, cookies, pastas, and soups. For example, in February 2007, Tropicana, a division of PepsiCo, Inc., launched the first orange juice with MEG-3 (fish-based micro-encapsulated omega-3 powders) in both Canada and the United States (Hein, 2007). Additionally, research is intensively focused on the increase of omega-3 fatty acid content of animal products, such as meat (Howe et al., 2002; Scollan et al., 2003; Lopez-Ferrer et al., 2001), milk (Kitessa et al., 2004), and eggs (Lewis et al., 2000a; Howe et al., 2002), by altering the composition of animal feed (Simopoulos, 1999b). The omega-3 fatty acid content of animal feed is enhanced with the addition of fishmeal, flax, and omega-3 fatty acids (Simopoulos, 1999b). New to the Canadian and Japanese markets is omega-3 enriched pork, which was developed by a company based in Winnipeg, Manitoba (Flax Council of Canada, 1998). In 2005, it received approval to label its product. Consequently, a variety of food products can serve as good vehicles for omega-3 fatty acid enrichment, which have the potential to improve the population’s health and reduce the risk of nutrition-related chronic conditions.

Increased consumer awareness, in combination with recognizable healthy compounds of omega-3 fatty acids, made feasible the nutrition labelling of omega-3 enriched products. Health Canada made regulations for omega-3 labelling and gave the chance to producers to make nutrient content claims that can state that the product is “source of omega-3 polyunsaturated fatty acids,” “contains omega-3 polyunsaturated fatty acids,” or “provides omega-3 polyunsaturated fatty acids” (CFIA, 2003a). Health Canada and the Canadian Food Inspection Agency (CFIA) have also approved certain biological role claims that refer to the generally recognized nutritional function of energy or nutrients as an aid in maintaining the functions of the body for the maintenance of good health or for normal growth and development (CFIA, 2003b). An acceptable biological role claim is that “DHA, an omega-3 fatty acid, supports the normal development of the brain, eyes and nerves” (CFIA, 2003b), and can only be used if the food is a source of the nutrient. Biological role claims are not made for a food or for an ingredient in a food, but for the energy value or nutrients in a food (CFIA, 2003b). In contrast, the FDA has approved a qualified health claim on both conventional foods and dietary supplements containing omega-3 fatty acids that states that “supportive but not conclusive research shows that consumption of EPA and DHA omega-3 fatty acids may
reduce the risk of coronary heart disease” (FDA, 2004). The FDA remains conservative because more scientific evidence is needed to confirm the effect of omega-3 fatty acids on the risk of CHD in the general population.

2.5.3. The Case of Omega-3 Enriched Eggs

In recent years, omega-3 enriched eggs have gained increased public attention as they provide an alternative food source for the delivery of the health-promoting omega-3 fatty acids. The development of the omega-3 enriched egg began as a Canadian endeavour in the 1990s. The industry needed to improve the nutrition health profile of eggs because high cholesterol concerns were resulting in a rapid decline in the consumption of regular eggs (Flax Council of Canada, 2006). In an effort to find a solution to this problem, which affected the egg industry, Dr. Jeong Sim, a professor of Agricultural, Food, and Nutritional Science at the University of Alberta, created the Canadian Designer Egg, which is enriched with omega-3 fatty acids (Sim, 2007). Dr. Sim pioneered the introduction and marketing of Designer Eggs to the commercial market in Canada. According to the Canadian Egg Marketing Agency, omega-3 enriched eggs currently account for 15% of the Canadian egg market (Flax Council of Canada, 2006).

Eggs can be easily enriched with omega-3 PUFA through dietary modification of the laying hens (Lewis et al., 2000a). Extensive research has found that adding a percentage of flax, canola oil, sea algae, or other omega-3 rich products to the ration of chicken feed would produce eggs that have an increased level of omega-3 (Pickering, 2003). In Canada, incorporating mainly flax seed into the laying hen diet has enhanced the omega-3 PUFA content. Using flax as 10% or 20% of a poultry ration can increase the ALA content of egg yolk fat from 0.4% in the ordinary egg to 4.6% and 8.9%, respectively (Flax Council of Canada, 2007). Thus, the ALA of the flax, combined with the hen’s own conversion of ALA to EPA and DHA, can make eggs a healthy food product, and hence desirable for health conscious consumers.

Omega-3 enriched eggs provide about twelve times more omega-3 fatty acids than regular eggs, based on an average omega-3 content of 0.5 grams in omega-3 enriched eggs versus 0.04 grams in regular eggs, as illustrated in Table 2.2 (Flax Council of Canada, 2003b). This is an average content of omega-3 PUFA, as different brands of
omega-3 enriched eggs include different levels of omega-3 PUFA. Nonetheless, the number of calories, the amount of protein, and the total fat remain almost the same to that of regular eggs, while cholesterol is slightly less in some omega-3 enriched eggs than regular eggs. The same remains, also, of taste and versatility. Taking into account that omega-3 enriched eggs can serve as an ideal source of omega-3 PUFA, many people find it more practical to increase egg consumption rather than fish consumption.

**Table 2.2. Comparison of the nutrient content of omega-3-enriched eggs and regular eggs.**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Omega-3 Enriched Eggs</th>
<th>Regular Eggs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>74 calories</td>
<td>75 calories</td>
</tr>
<tr>
<td>Protein</td>
<td>6.2 g</td>
<td>6.2 g</td>
</tr>
<tr>
<td>Total Fat</td>
<td>4.8 g</td>
<td>5.0 g</td>
</tr>
<tr>
<td>Saturated Fat</td>
<td>1.5 g</td>
<td>1.6 g</td>
</tr>
<tr>
<td>Monounsaturated Fat</td>
<td>2.1 g</td>
<td>1.9 g</td>
</tr>
<tr>
<td>Polyunsaturated Fat</td>
<td>1.3 g</td>
<td>0.68 g</td>
</tr>
<tr>
<td>Total Omega-6 Fatty Acids</td>
<td>0.78 g</td>
<td>0.64 g</td>
</tr>
<tr>
<td>Total Omega-3 Fatty Acids</td>
<td>0.50 g</td>
<td>0.04 g</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>182 mg</td>
<td>212 mg</td>
</tr>
</tbody>
</table>

1. Abbreviations: g = grams, mg = milligrams
3. Average of two brands of omega-3 enriched eggs.
4. Average of three brands of omega-3 enriched eggs.
5. Average of five brands of omega-3 enriched eggs.
6. On average, an omega-3 enriched egg contains about 0.34 grams of ALA and 0.13 grams of EPA + DHA. One omega-3 enriched egg contains a total of about 0.5 grams (500 milligrams) of omega-3 fatty acids. (The figure for total omega-3 fatty acids includes several minor omega-3 fatty acids, in addition to the main omega-3 fatty acids, ALA, EPA, and DHA).


However, one of the barriers to enhancing the appeal of consuming omega-3 enriched eggs is consumers’ perception that increasing egg consumption is related to high blood cholesterol levels, resulting in serious health problems. The American Heart Association (AHA) does not specify a desirable egg intake, but it recommends consuming no more than three to four egg yolks per week (assuming a weekly cholesterol intake of more than 300 mg daily) (Flax Council of Canada, 2003b).

In order to assess the impact of omega-3 enriched eggs on both serum lipids and other risk factors, research studies have been conducted and suggest that some consumers
can eat one egg daily with no adverse health effects. Specifically, one study reported that most consumers who follow a low-fat, low-cholesterol diet plan can eat twelve omega-3 enriched eggs per week without an increase in total or LDL cholesterol level (Lewis et al., 2000b). Two more studies showed that adding four omega-3 enriched eggs per day in a regular diet does not affect the total and LDL cholesterol concentrations (Ferrier et al., 1995; Oh et al., 1991). Another study found that eating four omega-3 enriched eggs per week resulted in a significant decrease in blood platelet aggregation, which is a risk factor for CHD (Van Elswyk et al., 1998). Therefore, omega-3 enriched eggs can help reduce the risk of CHD, which is a major health concern in Canada, by decreasing major CHD risk factors.

Several clinical and epidemiological studies have also illustrated that dietary cholesterol is not the major determinant of plasma cholesterol level in healthy individuals. As such, there is no need to set restrictions on egg consumption (McNamara, 1997). Some studies suggest that eating three to fourteen regular eggs per week has no effect on blood lipid levels (Voster et al., 1992), especially if the saturated fat level remains low (Edington et al., 1989; Garwin et al., 1992). Another study reported that moderate egg intake should not be rigorously restricted in healthy individuals (Scnohr et al., 1994). Lastly, a recent large-scale study suggests that consumption of up to one egg per day is unlikely to have substantial overall impact on the risk of cardiovascular (CVD) or stroke among healthy adults (Hu et al., 1999a). It should be noted, however, that in some people plasma total and LDL cholesterol is easily affected by dietary cholesterol, and so in such cases modest egg consumption is recommended.

In conclusion, the enrichment of eggs with omega-3 fatty acids has provided a workable solution to those consumers who are interested in increasing their omega-3 PUFA intake. The consumption of omega-3 enriched eggs on a regular basis can make a substantial contribution to the recommended daily intake of omega-3 PUFA to achieve a more balanced omega-6 to omega-3 ratio, thus decreasing the risk of developing a diet-related chronic disease, particularly CHD. Furthermore, the ability to label omega-3 fatty acids has stimulated many egg producers to develop these eggs and meet the needs and preferences of the niche market. Omega-3 enriched eggs are already commercialized in
many countries all over the world and have received wide acceptance due to their healthful omega-3 properties (Surai and Sparks, 2001).

2.6. Economic Impact of CVD

2.6.1. Direct and Indirect CVD Costs

For many decades, cardiovascular disease (CVD) remains the major chronic condition that causes great concern in Canada and around the world. CVD, mainly heart disease and stroke, is the prominent cause of illness, disability, and premature death among Canadians. In 2004, CVD claimed the lives of 72,338 Canadians and accounted for 31.9% of all deaths in the country (Statistics Canada, 2004). Although the death rate of CVD has decreased over the last thirty years, CVD continues to be the leading cause of mortality in Canada. CVD has the largest economic impact of all diagnostic categories as it poses enormous cost, both direct and indirect, on individuals and on society.

The direct cost of CVD in Canada needs to be reduced because it creates an enormous burden on the health care system. “Direct cost is defined as the value of goods and services for which payment was made and resources used in treatment, care and rehabilitation related to illness or injury” (Heart and Stroke Foundation of Canada, 2003). The direct cost of CVD is composed of hospital care expenditures, drug expenditures, physician care expenditures, expenditures for care in other institutions, and additional health expenditures such as health research.

The indirect cost of CVD is even greater because CVD has a high probability of being fatal. “Indirect cost refers to the value of economic output lost because of illness, injury-related work disability, or premature death” (Heart and Stroke Foundation of Canada, 2003). The major components of indirect CVD costs are mortality costs (i.e., premature death), morbidity costs due to long-term disability and morbidity costs due to short term disability. It is noteworthy that a large proportion of the cardiovascular events occur among people less than fifty years of age. Avoiding death and disability in people at or near the peak of their earnings potential generates large indirect cost savings to society (Oster and Thompson, 1996).

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Cardiovascular diseases refer to diseases and injuries of the heart, the blood vessels of the heart, and the system of blood vessels (veins and arteries) throughout the body and within the brain (Heart and Stroke Foundation of Canada, 2002).
The last official report containing data regarding the costs of CVD in Canada refers to 1998. In 1998, the total economic burden of CVD was $18,472.9 million in Canada and ranked first in total cost, followed by musculoskeletal, cancer, injuries, respiratory, and other diseases. The direct cost of CVD reached $6,818.1 million (36.9% of total CVD cost) and exceeded, to a large extent, the direct cost of the other diagnostic categories. Nonetheless, the indirect cost of CVD reached $11,654.8 million (63.1% of total CVD cost) and outweighed the direct cost of CVD, demonstrating the importance of indirect cost of CVD for individuals, society, and the health care system. More specifically, in terms of direct cost of CVD in 1998, hospital expenditures represented by far the largest direct cost, which accounted for $4,161.8 million, or 61% of the total direct cost of CVD. Drug expenditures accounted for $1,772.8 million, which represented 26% of the total direct CVD cost. Also, physician costs and the cost of CVD research represented 12.1% and 0.9% of the total direct cost of CVD, respectively (Health Canada, 1998b; Heart and Stroke Foundation of Canada, 2003).

Mortality accounted for $8,250 million, or 70.8%—the largest proportion—of total indirect CVD cost. The costs of morbidity due to long-term disability as a result of CVD cost the Canadian economy $3,151.5 million, or 27% of total indirect cost of CVD, while the cost of morbidity due to short-term disability represented 2.2% of the total indirect cost of CVD (Health Canada, 1998b; Heart and Stroke Foundation of Canada, 2003). It is worth mentioning that there is an extra cost to the individual, that of pain and suffering, which is not taken into consideration in the indirect costs.

The two main categories of CVD are coronary heart disease (CHD) and stroke or cerebrovascular disease. CHD, also called ischemic heart disease (IHD) or coronary artery disease (CAD), results from a reduced blood supply to the heart, and accounts for more than half of all deaths due to CVD (Mirolla, 2004); stroke is responsible for fewer deaths than CHD. As a result, CHD represents a large portion of CVD costs. According to 1998 health costs data, hospital expenditures of CHD were $1,274.8 million, while drug costs were $512.7 million. Other categories of direct costs, such as physician and additional direct costs (e.g., health research), are not reported. In terms of indirect costs,

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6 Expenditures for care in other institutions, as well as additional direct health expenditures except health research, are only available for all diagnostic categories (Health Canada, 1998b).
mortality costs and morbidity costs due to long-term disability from CHD reached $4,845.8 and $567.9 million, respectively. No data are available for morbidity costs due to short-term disability from CHD.

Although CVD is the most costly contributor to both direct and indirect health costs in Canada, it is also largely preventable (Heart and Stroke Foundation of Canada, 2003). Following a healthy diet that includes fruits, vegetables, whole grains, poultry, and fish, and limits unhealthy fats, can improve health status and prevent the risk of a CVD event, fatal or nonfatal. Therefore, the need for medical care may decrease and Canada’s escalating CVD costs may be reduced.

Nowadays, consumers can choose food products high in omega-3 fatty acids due to nutrition labelling which is supported by the public, health professionals, and the food industry. Nutrition intervention can be effective in controlling CVD and, thus, in decreasing costs related to treatment and prevention of CVD. If CVD costs can be reduced even slightly due to improved nutrition labelling and additional nutrient and health claims, considerable health care savings can be achieved. The provision of health information through labelling can contribute to the choice of a more balanced diet, which can avert a range of diet-related chronic conditions and attached costs.

2.6.2. Estimation of CVD Costs

It is anticipated that CVD will continue to have a negative impact on welfare in Canada if no preventive measures are taken. In this section, an estimation of CVD costs in general, and CHD costs in particular, for 2006 was performed based on 1998 figures. Table 2.3 depicts the estimation of CVD costs ($2006), while Table 2.4 provides estimates for CHD costs ($2006). (All the estimated numbers in the tables are bolded).

The total direct and indirect costs of CVD, as estimated for 2006, are shown in Table 2.3. For the estimation, the direct and indirect costs of CVD, as reported in 1998, were used. As previously noted, in terms of direct costs, expenditures for care in other institutions and additional direct health expenditures apart from health research were not classified by diagnostic category in the 1998 Health Canada report. So, in order to take into consideration these other direct costs, an assumption is made—it is assumed that these costs are the same proportion of total direct health care costs (hospital, drug, and
physician costs) for each diagnostic category separately as they are for all diagnostic
categories together. Thus, a calculated ratio was used to measure the other direct costs of
CVD for 1998. After calculating the total direct costs of CVD in 1998, the change in CPI
and the change in population were used to estimate the total direct costs of CVD in 2006
dollars. The total indirect costs of CVD ($2006) were also estimated by applying the CPI
and population change to the total indirect cost of CVD in 1998.

Therefore, according to these estimates, CVD cost the Canadian health care
system an estimated $13,902.9 million ($2006) annually in total direct costs, which
represents 48% of the total cost of CVD. Productivity losses due to premature death, as
well as short-term and long-term disability, cost the economy an additional $14,877.1
million, which accounts for 52% of the total CVD cost. Hence, the total cost attributed to
CVD is estimated to be $28,780.2 million per annum.7

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7 A detailed calculation of the cost categories of direct and indirect costs of CVD for 2006 is provided in Appendix A.
Table 2.3. Estimation of total direct and indirect costs of CVD in Canada ($2006).

<table>
<thead>
<tr>
<th></th>
<th>CVD</th>
<th>All diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Costs (HC)</td>
<td>4,161.8</td>
<td>27,638.4</td>
</tr>
<tr>
<td>Drug Costs (DC)</td>
<td>1,772.8</td>
<td>12,385.2</td>
</tr>
<tr>
<td>Physicians Costs (PC)</td>
<td>822.3</td>
<td>11,686.9</td>
</tr>
<tr>
<td>Total HC, DC, PC</td>
<td>6,756.9</td>
<td>51,710.5</td>
</tr>
<tr>
<td>Other Direct Costs (ODC) (millions CAN$)</td>
<td><strong>4,073.5</strong></td>
<td>31,174.7</td>
</tr>
<tr>
<td>Ratio ODC/Total HC, DC, PC</td>
<td><strong>0.603</strong></td>
<td><strong>0.603</strong></td>
</tr>
<tr>
<td>Health Research Costs (HRC) (millions CAN$)</td>
<td>61.2</td>
<td>1,069.7</td>
</tr>
<tr>
<td>Total Direct Costs ($1998) (millions CAN$)</td>
<td><strong>10,891.6</strong></td>
<td>83,954.9</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Total Direct Costs ($2006) (millions CAN$)</td>
<td><strong>13,902.9</strong></td>
<td><strong>107,166.7</strong></td>
</tr>
<tr>
<td><strong>INDIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality Costs (MC)</td>
<td>8,250</td>
<td>33,481.5</td>
</tr>
<tr>
<td>Morbidity Costs due to Long-Term Disability (LTDMC) (millions CAN$)</td>
<td>3,151.5</td>
<td>32,178.7</td>
</tr>
<tr>
<td>Morbidity Costs due to Short-Term Disability (STDMC) (millions CAN$)</td>
<td>253.3</td>
<td>9,819.4</td>
</tr>
<tr>
<td>Total Indirect Costs ($1998) (millions CAN$)</td>
<td>11,654.8</td>
<td>75,479.6</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Total Indirect Costs ($2006) (millions CAN$)</td>
<td><strong>14,877.1</strong></td>
<td><strong>96,348.1</strong></td>
</tr>
<tr>
<td><strong>TOTAL COST</strong>²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cost ($1998) (millions CAN$)</td>
<td><strong>22,546.4</strong></td>
<td>159,434.5</td>
</tr>
<tr>
<td>Total Cost ($2006) (millions CAN$)</td>
<td><strong>28,780.1</strong></td>
<td><strong>203,514.8</strong></td>
</tr>
</tbody>
</table>

¹ Other Direct Costs (ODC) include the expenditures for care in other institutions and additional direct health expenditures. However, they are the net of Health Research Costs. The ODC for CVD were calculated as a ratio by multiplying 6,756.9 (Total HC, DC, PC) with the ratio ODC/Total HC, DC, PC, which is 0.603.

² The ratio was calculated by dividing 31,174.7 by 51,710.5. This ratio is applied for CVD in order to calculate the other direct costs, assuming that the other direct health costs occupy the same proportion of total direct health care costs for each diagnostic category.

³ The Total Direct Costs ($1998) were calculated by summing HC, DC, PC, ODC, and HRC.

⁴ Source: Statistics Canada, 2007b. Consumer Price Index (CPI) for 1998 was 91.3, and 109.1 for 2006. The change in CPI was calculated by subtracting 91.3 from 109.1 and dividing by 91.3.

⁵ Source: Cansim via E-Stat, Table 051-0005. Quarterly population estimates. The average population estimate for 1998 was 30,125,715, and 32,581,490 for 2006. The change in population was calculated by subtracting 30,125,715 from 32,581,490 and dividing by 32,581,490.

⁶ Total Direct Costs ($2006) = [Total Direct Cost ($1998)+(Total Direct Cost ($1998)*change in CPI)+(Total Direct Cost ($1998)*change in population)] = [10,891.6+(10,891.6*0.19)+(10,891.6*0.06)].

⁷ The Total Indirect Costs ($1998) were calculated by summing MC, LTDMC, and STDMC.

⁸ Total Indirect Costs ($2006) = [Total Indirect Cost ($1998)+(Total Indirect Cost ($1998)* change in CPI)+(Total Indirect Cost ($1998)* change in population)] = [11,654.8+(11,654.8*0.19)+(11,654.8*0.06)].

⁹ Total Cost is the sum of Total Direct and Total Indirect Costs.

Source: Author’s estimates (see text for detail).
Several people experience a CHD event, either fatal or non-fatal, suggesting that CHD is a major category of CVD, and thus has the largest portion of CVD costs. Although the 1998 report provides some data on CHD costs, it does not report the costs of all the components of direct and indirect costs. So, before estimating CHD in 2006 dollars, there is a need to measure those CHD costs that are not available. These costs include some direct costs, such as physicians, other direct and health research costs, and morbidity costs due to short-term disappearance from the indirect cost category.

Table 2.4 presents the results of the estimation of the 1998 CHD costs that were not reported, as well as the estimation of the total direct and indirect costs of CHD for 2006. In order to measure the CHD costs that are not available in the 1998 Health Canada report, it is assumed that each of these costs represents the same proportion of total hospital and drug costs for each category of CVD separately as it does for all CVD categories combined. The ratios were calculated based on CVD costs and used to measure the unreported costs of CHD. The total direct and indirect costs were thus calculated for 1998 using an estimate of all the components of direct and indirect costs of CHD. Then, using the direct and indirect costs of CHD in combination with the change in CPI and the change in population, the total direct and indirect costs of CHD for 2006 were estimated.

As the estimates demonstrate, the total direct cost of CHD reached an estimated $4,187.6 million ($2006), and account for 37% of the total cost of CHD. Additionally, indirect costs outweigh the direct costs of CHD—an estimated $7,064 million ($2006), or 63% of the total costs of CHD. This means that there is a large possibility of a CHD incident being fatal, highlighting the need to improve diets and follow healthy lifestyles. Consequently, CHD creates a total estimated cost to the Canadian economy of $11,251.6 million\(^8\) ($2006) and accounts for 39% of the total cost of CVD.

\(^8\) A detailed calculation of the cost categories of direct and indirect costs of CHD for 2006 is provided in Appendix A.
Table 2.4. Estimation of total direct and indirect costs of CHD in Canada ($2006).

<table>
<thead>
<tr>
<th></th>
<th>CVD</th>
<th>CHD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Costs (HC)</td>
<td>4,161.8</td>
<td>1,274.8</td>
</tr>
<tr>
<td>Drug Costs (DC)</td>
<td>1,772.8</td>
<td>512.7</td>
</tr>
<tr>
<td>Total HC,DC</td>
<td>5,934.6</td>
<td>1,787.5</td>
</tr>
<tr>
<td>Physician Costs (PC)</td>
<td>822.3</td>
<td>247.7</td>
</tr>
<tr>
<td>Ratio PC/Total HC,DC</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Other Direct Costs (ODC)</td>
<td>4,073.5</td>
<td>1,226.9</td>
</tr>
<tr>
<td>Ratio ODC/Total HC,DC</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Health Research Costs (HRC)</td>
<td>61.2</td>
<td>18.4</td>
</tr>
<tr>
<td>Ratio HRC/Total HC,DC</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Total Direct Costs ($1998)</td>
<td>10,891.6</td>
<td>3,280.5</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Total Direct Costs ($2006)</td>
<td>13,902.9</td>
<td>4,187.6</td>
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<td><strong>INDIRECT COSTS</strong></td>
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<tr>
<td>Mortality Costs (MC)</td>
<td>8,250</td>
<td>4,845.8</td>
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<td>Morbidity Costs due to Long-Term Disability (LTDMC)</td>
<td>3,151.5</td>
<td>567.9</td>
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<tr>
<td>Total MC,LTDMC</td>
<td>11,401.5</td>
<td>5,413.7</td>
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<td>Morbidity Costs due to Short-Term Disability (STDMC)</td>
<td>253.3</td>
<td>120.3</td>
</tr>
<tr>
<td>Ratio STDMC/Total MC,LTDMC</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Total Indirect Costs ($1998)</td>
<td>11,654.8</td>
<td>5,534</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td>Total Indirect Costs ($2006)</td>
<td>14,877.1</td>
<td>7,064</td>
</tr>
<tr>
<td><strong>TOTAL COST</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cost ($1998)</td>
<td>22,546.4</td>
<td>8,814.5</td>
</tr>
<tr>
<td>Total Cost ($2006)</td>
<td>28,780.1</td>
<td>11,251.6</td>
</tr>
</tbody>
</table>

1. Physician Costs of CHD were calculated by multiplying 0.1 (Ratio PC/Total HC, DC) with 1,787.5 (Total HC,DC) of CHD.
2. The ratio was calculated by dividing 822.3 (Physicians Costs) by 5,934.6 (Total HC, DC) of CVD.
3. Other Direct Costs of CHD were calculated by multiplying 0.7 (Ratio ODC/Total HC, DC) with 1,787.5 (Total HC,DC) of CHD.
4. The ratio was calculated by dividing 4,073.5 (Other Direct Costs) by 5,934.6 (Total HC, DC) of CVD.
5. Health Research Costs of CHD were calculated by multiplying 0.01 (Ratio HRC/Total HC, DC) with 1,787.5 (Total HC,DC) of CHD.
6. The ratio was calculated by dividing 61.2 (Health Research Costs) by 5,934.6 (Total HC, DC) of CVD.
7. Total Direct Costs ($1998) were calculated by summing HC, DC, PC, ODC, and HRC.
8. Source: Statistics Canada, 2007b. Consumer Price Index (CPI) for 1998 was 91.3, and 109.1 for 2006. The change in CPI was calculated by subtracting 91.3 from 109.1 and dividing by 91.3.
9. Source: Cansim via E-Stat, Table 051-0005. Quarterly population estimates. The average population estimate for 1998 was 30,125,715, and 32,581,490 for 2006. The change in population was calculated by subtracting 30,125,715 from 32,581,490 and dividing by 32,581,490.
11. Morbidity Costs of CHD due to Short-term Disability were calculated by multiplying 0.02 (Ratio STDMC/Total MC, LTDMC) with 5,431.7 (Total MC, LTDMC) of CHD.
12. The ratio was calculated by dividing 253.3 (Morbidity costs due to Short-term disability) by 11,401.5 (Total MC, LTDMC) of CVD.
13. Total Indirect Costs ($1998) were calculated by summing MC, LTDMC, and STDMC.
Total Indirect Costs ($2006) = \[\text{Total Indirect Cost ($1998) + (Total Indirect Cost ($1998) \times \text{change in CPI}) + (Total Indirect Cost ($1998) \times \text{change in population})}\] = [5,534 + (5,534 \times 0.19) + (5,534 \times 0.06)].

Total Cost is the sum of Total Direct and Total Indirect Costs.

Source: Author’s estimates (see text for detail).

2.7. Summary

This chapter looked at the health attributes of functional foods and their potential beneficial effects on consumers’ health, and hence on reducing health care costs. The increased demand for foods with health-enhancing properties has led to significant growth of the functional food industry in Canada. How labelling, nutrient claims, and health claims can assist consumers in making healthy food choices was also discussed. This contributes to the correction of the information asymmetry problem and therefore to potential savings in health care costs. In addition, this chapter explicitly described omega-3 enriched eggs, which are a recent functional food with health promoting attributes. Such healthy food is very important as the typical Canadian diet is characterized by low levels of omega-3 fatty acids, potentially resulting in serious health conditions, particularly CHD. An unhealthy population can negatively affect economic welfare by raising health care costs. An estimation of CVD and CHD costs in Canada was also performed, illustrating that billions of dollars are spent every year on diet-related diseases like CVD, whose risk can be substantially minimized by making healthier food choices. However, the lack of information may deter consumers from improving their diet. Therefore, additional nutrient content and health claims are necessary to help improve the health of consumers and decrease health care costs.
CHAPTER 3: DEVELOPMENT OF THE THEORETICAL MODEL

3.1. Introduction

This chapter discusses the current regulatory labelling system in Canada. The lack of comprehensive Canadian legislation, which may hinder development of nutritious products and create barriers to trade, is described and the need for improvement is suggested. A graphical illustration is provided of the effect of health information provided via food labelling on social and market welfare. Initially, a case is examined where labelling is not available in the market and thus consumers are unaware of the healthy properties of functional foods. It continues with a case where health and nutrition labels can be used for functional foods (natural and industrial) and claims can also be used to assist individuals to manage their diet. The increased consumption of functional foods and the increase in health care savings are graphically depicted, reflecting the potential contribution of health information and, hence, food labelling. A graphical comparison of a voluntary and compulsory labelling system is then provided, showing the difference in health benefits. The chapter concludes with a discussion of the negative externality that prevents a socially optimal level of consumption of functional foods, and proposes some possible solutions.

3.2. Canadian Regulatory Framework for Functional Foods

In Canada, all food and drugs are regulated under the Food and Drugs Act and Regulations. Labelling products with nutrition information helps Canadian consumers make healthier and more informed dietary choices, which may prevent chronic nutrition-related diseases, thus generating a savings in medical costs. Until recently in Canada, nutrition labelling was voluntary. However, Health Canada made amendments to the Foods and Drugs Act regulations, and so on 1 January 2003 these new regulations were officially published in the Canada Gazette Part II (Canada Gazette, 2003b). The
3.2.1. Mandatory Nutrition Labelling and Nutrient Content Claims

The new regulations require almost all prepackaged foods to carry a mandatory Nutrition Facts table that lists calories and thirteen core nutrients, including the amount of fat, saturated and trans fats, cholesterol, sodium, carbohydrate, fiber, sugars, protein, vitamins A and C, calcium and iron in a specified amount of food. The Nutrition Facts table looks the same on most products and the listing of nutrients appears in a standardized format in order to help consumers compare similar products. The information is provided as an amount in grams and as a percentage (%) of Daily Value (Canada Gazette, 2003b). Daily Value can be used to determine whether a food item has a little or a lot of a particular nutrient in a specified amount of food. It should also be mentioned that any other nutrient that is added to a food or that is the subject of a claim must also be declared in the Nutrition Facts table.

Labels can also include nutrient content claims that emphasize the specific nutrient contained in a product. According to the CFIA, “A nutrient content claim is any statement or expression which describes, directly or indirectly, the level of a nutrient(s) in a food or group of foods” (CFIA, 2003c). Nutrient content claims are voluntary within the new labelling regulation, but if used they have to observe specific criteria outlined in the regulations guidelines. Some examples include “low sodium,” “sugar-free,” “reduced in fat,” “source of omega-3 polyunsaturates,” “high source of fiber,” and “trans fat free.” Nutrient content claims serve a different function than information on the label’s Nutrition Facts table. Although the Nutrition Facts table provides useful and detailed information, it may be relatively ineffective at capturing a consumer’s interest in the first place (Ippolito and Mathios, 1993). However, nutrient content claims may be more helpful and encourage consumers to consume healthier products and improve their diets. The federal government’s aim is to provide additional information through the Nutrition
Facts table to consumers who are aware of healthy eating patterns, as well as to stimulate indifferent portions of the population to consume more nutritious foods via nutrient content claims. Presumably, nutrition information should be accompanied by consumer education to be even more effective.

3.2.2. Health Claims

The new labelling regulations also allowed diet-related health claims for the first time in Canada. Diet-related health claims are considered statements that describe the characteristics of a diet associated with the reduction of the risk of developing a diet-related disease or condition (e.g., osteoporosis, cancer, or heart disease) (Canada Gazette, 2003b). An example of such a claim is, “A healthy diet with adequate calcium and vitamin D, and regular activity helps to achieve strong bones and may reduce the risk of osteoporosis. (Naming the food) is a good source of calcium” (Canada Gazette, 2003b). Consumers now have the opportunity to select from a variety of foods with healthy attributes and improve their well-being by minimizing the risk of chronic disease.

The regulation and permission of health claims in Canada also benefits food companies, as health claims can be used as a marketing technique (Hawkes, 2004). Producers are willing to increase research and development to find new ways of producing products that have health benefits. Furthermore, some products are being reformulated (e.g., foods that now are free of saturated and trans fat) by changing their nutritional composition in order to meet the criteria to qualify for health claims (Lutz, 2005).

In November 1998, Health Canada published a policy recommendation on health claims for foods. The policy recommended that structure/function and risk reduction claims be permitted for foods, while therapeutic claims (i.e., that a product can cure, treat, mitigate, or prevent illness) should continue to be regulated as drugs (Health Canada, 1998a; Health Canada, 2000; Health Canada, 2001). In the policy paper, Health Canada described structure/function claims as the effect of a food or a diet on a structure or physiological function in the human body (e.g., calcium helps to build strong bones) (Health Canada, 2000). A risk reduction health claim describes the relationship between the consumption of a food or a diet and reduction in the risk of developing a chronic
disease or abnormal physiological state by significantly altering a major risk factor or factors recognized to be involved in its development (e.g., calcium helps reduce the risk and progression of osteoporosis) (Health Canada, 2000).

These claims are distinguished into two types, generic and product-specific health claims. Manufacturers apply generic health claims to any food or food product that meets the criteria for the claim (Health Canada, 2000). This is in contrast to product-specific health claims, which are made for a proprietary product. This means that they cannot be generalized to other similar products unless acceptable supporting evidence is provided (Health Canada, 2000). In other words, the food product must have been designed to provide a specific and documented effect (Asp and Laser Reutersward, 1998). Permitting product-specific health claims can also promote industry innovation through the development of products with healthy characteristics. However, it has been argued that such claims should not be allowed as they undermine the general principle that total diet, not individual foods, is the key to good health (Hawkes, 2004). A procedure for product specific health claims has not yet been fully developed in Canada (Lutz, 2005).

<table>
<thead>
<tr>
<th>Types of Claims</th>
<th>Types of Food Products</th>
</tr>
</thead>
<tbody>
<tr>
<td>Product-Specific</td>
<td>Innovative food products</td>
</tr>
<tr>
<td></td>
<td>Processed food with added component</td>
</tr>
<tr>
<td></td>
<td>(e.g., cereal with added B-glucan)</td>
</tr>
<tr>
<td></td>
<td>Food enhanced in a certain component</td>
</tr>
<tr>
<td></td>
<td>(e.g., omega-3 eggs)</td>
</tr>
<tr>
<td></td>
<td>Food with a nutrient-specific claim</td>
</tr>
<tr>
<td></td>
<td>(e.g., calcium)</td>
</tr>
<tr>
<td></td>
<td>Processed food (e.g., oat bran cereal)</td>
</tr>
<tr>
<td></td>
<td>Basic food (e.g., whole grains wheat)</td>
</tr>
<tr>
<td></td>
<td>Diet (e.g., high in fruits and vegetables)</td>
</tr>
</tbody>
</table>

**Figure 3.1. Types of claims and types of food products.**

New Canadian regulations permit five generic diet-related health claims—four of them for disease risk-reduction and one for tooth decay. The regulations set out the prescribed wording for permitted claims to ensure that they are consistent, accurate, and non-deceptive (Canada Gazette, 2003b). The current permitted generic diet-related health claims are the following (Fitzpatrick, 2004; Health Canada, 2006):

1. Sodium: hypertension.
2. Calcium: osteoporosis.
5. Sugar alcohols: tooth decay.

In addition to the above generic health claims, there are four more health claims that Canadian regulatory authorities are still examining. Although United States has already approved these claims, decisions are pending in Canada for the following four claims (Fitzpatrick, 2004):

1. Folate: neural tube defects
2. Fibre-containing grain products, fruits, and vegetables: cancers
3. Fruits, vegetables and grain products that contain fibre, particularly soluble fibre: risk of coronary heart disease

Diet-related health claims can affect food purchase decisions and improve health and well-being. Both nutrient content and health claims are considered very useful for attracting consumers’ attention and encouraging them to consume functional foods. The Nutrition Facts table provides more in-depth information regarding the nutritional composition of a product to help consumers assess whether it satisfies their health needs and preferences.

Several surveys have been conducted to evaluate consumers’ perceptions and attitudes related to health claims on functional foods. They have illustrated that most consumers consider health claims useful and helpful as they help them achieve and maintain a healthy lifestyle. In Canada, a telephone survey about products with functional
benefits reported that most respondents believed packaging should promote the health benefit that it provides, rather than solely indicate the presence of the component itself (National Institute of Nutrition, 2000). This indicates that consumers prefer health claims to content claims—47% rated them as very useful compared to less than 10% who saw little or no value for health claims (National Institute of Nutrition, 2000). Other studies performed in Scandinavia (Bech-Larsen and Grunert, 2003), the U.K. (Food Standards Agency and COI Communications, 2004) and the U.S. (Fullmer et al., 1991) have indicated similar attitudes and opinions related to health claims amongst consumers, demonstrating the significance that health claims have in consumers’ purchasing decisions.

Regulatory authorities in Canada aim to protect the public from false and misleading claims and to make sure that nutrient and health claims are based on scientific evidence. Consumers need to have a clear understanding of, and a strong confidence level in, the scientific criteria used to document health effects and claims (EUFIC, 2006). Some consumer groups exhibit high levels of skepticism and concern about health claims, and feel that regulations are necessary in order to ensure that health claims are based on scientific evidence and can achieve nutritional goals (Carretson and Bulton, 2000). With a clear regulatory framework, consumers are becoming more willing to consume functional foods, meaning that the risk of developing nutrition-related diseases may decrease.

3.3. Complications in Marketing Functional Foods

Canada’s regulatory system has been improved over the last decade. In 1996, Canada was among the least favorable regulatory environment for allowing health claims. Since that time, progress has been made with respect to the definition of health claims and the availability of five generic claims (MacDonald, 2004). However, the regulatory system is currently being challenged by rapid scientific and technological advancement, global markets, evolving business practices, higher public expectations of government and business, and cross-boundary health and environmental risks (Government of Canada, 2005a; Government of Canada. 2005b; External Advisory Committee on Smart Regulation. 2004). The lack of an adequate, effective, flexible and comprehensive legal framework discourages investment in the research and development of functional foods.
with potential health benefits, as well as undermines consumer confidence in functional foods. When firms cannot label any beneficial ingredient, they have no incentive to do research and supply the food market with new healthful products. For example, although a variety of healthful nutrients such as beta-glucan, lutein, and selenium may improve and maintain the general population’s health status, they are not currently approved to appear on food labels as health claims, suggesting the restrictive nature of the regulatory system.

The Canadian regulatory regime for functional foods and nutraceuticals needs to be reviewed and updated to cover evolving consumer needs, retain confidence, and promote competition among companies. It is necessary for the system to recognize the beneficial effects of functional foods on Canadians’ health and provide the food industry with the appropriate incentives to expand the availability of new nutritious products necessary for a reduction in diet-related diseases. Canada’s legislation has to keep pace with science development in order to both protect the health and safety of citizens and promote innovation.

The need for regulatory reform is crucial not only domestically but also internationally. Canadian firms, wishing to export their products, face transaction costs due to the difference in the regulatory systems under which foreign companies operate (Hobbs, 2002). In cases where Canadian standards for ingredients and final products differ from those of the international market, particularly the U.S., production might become less efficient, thereby reducing Canada’s attractiveness as a plant location (External Advisory Committee on Smart Regulation, 2004). The regulatory differences between Canada and its major trading partners may hurt Canadian economic competitiveness in terms of trade and investment opportunities (External Advisory Committee on Smart Regulation, 2004). Some examples showing the regulatory differences between Canada and the U.S. are presented in Table 3.1.
Table 3.1. Examples of Canada/U.S. regulatory differences.

<table>
<thead>
<tr>
<th>Issue</th>
<th>Canadian Approach</th>
<th>U.S. Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiperspirant deodorant</td>
<td>Aluminum content requires a Drug Identification Number</td>
<td>No DIN required</td>
</tr>
<tr>
<td>Trans fat on nutrition labels</td>
<td>In order to be considered a “trans fat free” product trans fat must be below 0.2g of trans fatty acids (1) per reference amount and serving of stated size, or (2) per serving of stated size if the food is a prepackaged meal</td>
<td>In order to be considered “trans fat free”, product must have less than 0.5g per reference amount and serving size</td>
</tr>
<tr>
<td>Fortification of breakfast cereals and other food products</td>
<td>Canadian regulations specify which foods may be fortified and the levels for their fortification with vitamins and minerals</td>
<td>The U.S. has no limits on the levels of vitamins and minerals used to fortify food products</td>
</tr>
<tr>
<td>Fortified water</td>
<td>Addition of vitamins and minerals to bottled water prohibited</td>
<td>Bottled water may be fortified with vitamins and minerals</td>
</tr>
<tr>
<td>Frozen pizza</td>
<td>BHA, BHT, and caramel colour are approved additives but cannot be used in pepperoni and sausage chunks</td>
<td>BHA, BHT, and caramel colour are permitted for use in pepperoni and sausage chunks</td>
</tr>
<tr>
<td>Cheddar-flavoured popcorn</td>
<td>Cheese seasoning must be less than 49% real cheese</td>
<td>Up to 53% real cheese seasoning used</td>
</tr>
<tr>
<td>Auto anti-theft immobilizers</td>
<td>Proposed requirement for immobilizers accepting Canadian and European standards</td>
<td>An option for US for high theft line vehicles</td>
</tr>
</tbody>
</table>

Source: External Advisory Committee on Smart Regulation, 2004.

An External Advisory Committee on Smart Regulation was established in May 2003 to renew the Canadian regulatory environment (External Advisory Committee on Smart Regulation, 2004). The aim of Smart Regulation is to improve Canada’s regulatory system, including the regulations in the agriculture and agri-food sector that promote consumer health and innovation. The Committee tries to create a more effective, flexible, transparent, and well-coordinated regulation which will adapt quickly to new scientific
advances and nutrition discoveries. The objectives of Smart Regulation are the following (Government of Canada, 2005b):

- enhanced coordination across the federal government and better co-operation with other governments in Canada and internationally to help set and meet national objectives that promote social, environmental, and economic well-being, and improve Canadians’ quality of life;
- increased policy coherence and integration of social, economic, and environmental principles and objectives into all stages of policy, regulation, and decision making;
- improved transparency, efficiency, timeliness, and predictability of regulatory and decision-making processes, and reduced administrative burden for businesses and citizens;
- strengthened planning and priority setting and more proactive and timely problem and risk identification to facilitate responsive regulation and better protect the public interest;
- improved identification, management, and mitigation of aggregate and unintended impacts on areas and sectors through greater use of longer-term, integrated, and whole-of-government approaches to regulation; and
- strengthened regulatory management from design to implementation, and evaluation of regulation for the continuous improvement and ongoing renewal of regulation across government.

Regulatory coordination among governments is a key issue that can contribute to the improvement and effectiveness of the regulatory system, but remains a major problem in Canada. Federal and provincial/territorial governments in Canada share regulatory responsibilities in several areas, including agriculture, environment, food safety, pharmaceuticals, and transport (External Advisory Committee on Smart Regulation, 2004). In the area of pharmaceuticals, for instance, the federal government is responsible for approving drugs for market, while provincial governments regulate the selection of drugs used in each provincial medical system (External Advisory Committee on Smart Regulation, 2004). Additionally, not only are federal governments responsible for food
regulations but provincial governments have jurisdictions over some aspects of food regulation (e.g., provincially inspected food processing facilities for products that will not cross inter-provincial borders). However, issues of overlapping jurisdiction and duplication have been raised in reviews of Canadian regulatory practice since at least the 1980s, resulting in considerable costs to the Canadian economy (External Advisory Committee on Smart Regulation, 2004).

A challenge to federal and provincial/territorial cooperation is the lack of coordination between federal departments. Very few regulatory issues fall under the exclusive mandate of a single federal department. In the food processing sector, for example, there can be as many as three different federal departments and agencies with regulatory responsibility in this area — Health Canada, Environment Canada, and the Canadian Food Inspection Agency (CFIA). This is in addition to Agriculture and Agri-Food Canada (AAFC), which holds the broader policy responsibility for this sector (External Advisory Committee on Smart Regulation, 2004). Each of these agencies is responsible for the safety and health of Canadians and regulates the food products before entering the market. This means that cooperation between them is necessary in order to improve the regulatory regime.

Food companies have expressed dissatisfaction as they deal with different federal and provincial authorities that sometimes have different regulatory requirements. This can complicate the approval of functional foods, forcing companies away from the research and development of new nutritious products. The lack of cooperation can seriously affect the efficiency of the regulatory system. Non-cooperation increases production costs for the food industry and creates a complex regulatory environment, all of which acts as a disincentive to investment in Canada. The multiplicity of federal regulators also creates barriers to citizens’ and businesses’ participation in the regulatory process (External Advisory Committee on Smart Regulation, 2004).

3.4. Graphical Illustration of Social Welfare Before and After Food Labelling

The functional food industry encounters two crucial market failures that may need federal intervention in order to be resolved. The first one, which will be explicitly discussed and analyzed in this thesis, is the information asymmetry problem that arises
due to credence attributes of functional foods. This problem deters consumers from making informed decisions about the purchase and consumption of nutritious goods and goods with potential health benefits. As a result, the economy incurs health care costs caused by diet-related chronic diseases. While the introduction of mandatory labelling and nutrient content and health claims are important tools for communicating valuable nutrition information, even greater access to information could improve the health of Canadians, thereby benefiting the economy.

A negative externality effect is another form of potential market failure in the functional food market. Consuming an unhealthy diet can lead to negative externalities because consumers do not pay the full health care costs that are associated with unhealthy eating, hence decreasing economic welfare. When individuals are protected by health insurance, a moral hazard problem is created and consumers—even these with nutritional knowledge—have a reduced incentive to restrain their consumption of goods with adverse health effects. This situation results in market inefficiencies and reduces welfare. Although this thesis concentrates on asymmetrically distributed information, some possible solutions for the moral hazard problem are proposed and briefly discussed at the end of this chapter.

In subsection 3.4.1 the effect of information asymmetry through the absence of labelling is graphically depicted, highlighting the social welfare loss in terms of health care costs. Then, in subsection 3.4.2, two graphs (one for natural functional food and one for industrial) are provided, taking into consideration food labelling and claims. The graphs show the importance of the information supplied through food labels in the reduction of medical costs.

3.4.1. The Case of No Labelling

Recently, many consumers have shown a growing concern about their diet and health. They seek products with nutritious characteristics and beneficial health properties in order to improve their health and decrease the risk of developing a chronic disease. Although they are interested in leading a balanced diet by consuming functional foods, they are restricted by a lack of adequate information provided by nutrient content and health claims. This information is necessary for consumers to help them make food
choices that best reflect their dietary needs. When a consumer is uninformed about the health benefits of a functional food, neither the private or social health benefits are reflected in the consumer demand. As a result, the problem of asymmetric information arises and the quantity consumed of a functional food can diverge considerably from the social optimum.

The lack of health information and resulting negative social welfare is depicted in Figure 3.2. Specifically, this graph combines the private demand curve and the social marginal benefit curve, and shows the limited consumption of a natural functional food when food labels are not available. The social marginal benefit curve is denoted as $MB_s$, and shows the social benefits gained from the consumption of a functional food. $D_{pUN}$ represents the private demand curve of uninformed consumers due to a lack of labelling. The private marginal cost is denoted as $MC_p$ and it is flat due to the competitive market structure. The social marginal benefit curve, denoting the health benefits from the consumption of a functional food, is higher than the private demand curve because functional food consumption can affect health care costs, which are paid mostly by the Canadian government.9

When people are not informed about the beneficial health effects of functional foods or the adverse effects of some components that other foods contain (e.g., trans fatty acids), they will not change their food consumption choices if they are otherwise satisfied with their eating patterns. Some may consume a small amount of almost each functional food that contains a naturally healthy component (e.g., olives, oats, tomatoes, canola oil) even though they are not informed about its healthy properties. However, functional foods that result from technological developments will not exist in a market where there is no nutrition information because their higher production cost will prevent them from being able to compete with natural functional foods (see section 3.4.2, Figure 3.5). The intersection of a private demand curve and a marginal cost curve gives the price $P_{pUN}$ and the quantity consumed $Q_{pUN}$ of a natural functional food. Consumers will not consume more than this quantity when they are not provided with pertinent health information through labelling.

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9 Private medical insurances also contribute to increased health care costs because they create moral hazard. Individuals consume unhealthy products knowing that health insurance will cover most costs of a chronic disease.
As far as welfare analysis is concerned, the consumer surplus from the consumption of $Q_p^{UN}$ natural functional food is equal to the area $EDP_p^{UN}$ while there is no producer surplus due to the competitive market. The consumer and producer surplus do not capture the external health benefits which arise from the reduction in health care costs and are equal to the area $ABDE$. As Figure 3.2 illustrates, this unavailability of health information creates dead weight loss, equal to the area $BCD$, which is measured by direct and indirect health care expenditures.

![Figure 3.2. Social welfare loss due to lack of labelling information.](image)

In some cases, uninformed individuals will unknowingly consume some functional foods. This means that they consume a healthy food because they like its taste or quality. Nutrition information is not available through labelling to influence their consumption choices. Although consumers lack nutrition information, this consumption provides them with an unknown health benefit because it contributes to their improved health. For example, some people consume oats because they like the taste. This satisfaction gives them a direct private benefit while simultaneously gaining a health benefit from the oat fiber, which has beneficial health effects. Yet consumers do not
realize this health enhancement if they are not aware of the health impacts of oat fiber. In Figure 3.3, the private marginal benefit curve \( MB_P^{\text{UN}} \) has been inserted to illustrate the additional private health benefit obtained due to the improved health. The area \( FGDE \) represents the unrealized private health benefit that ignorant consumers gain. The external social health benefits are depicted by the area \( ABGF \), while the dead weight loss due to a lack of labelling information remains the same and equal to the area \( BCD \).

![Graph](image)

Figure 3.3. Social welfare loss due to lack of labelling information with the marginal benefit.

3.4.2. The Case of Labelling and Social Benefits

In an effort to correct the market failure caused by information asymmetry, the Canadian government recently enforced mandatory nutrition labelling in almost all pre-packaged food products and allowed the use of nutrient content claims and five health
Consumers can now identify and consume some functional foods that contain nutritious attributes and avoid foods that have negative health effects. The dissemination of favorable information through food labels can substantially increase the demand for functional foods, while nutrient content and health claims can assist in shifting the demand curve even closer to the social optimum.

Consumers now have the chance to choose from a variety of functional foods as both categories, natural and industrial, are available in the market. The two types of functional foods, as well as their contribution in the reduction of health care costs, are explained below.

As was discussed in the first part of this chapter, the consumption of functional foods that contain healthy ingredients inherently takes place even in the absence of health information. However, the availability of food labelling can increase this consumption and make people healthier. As Figure 3.4 illustrates, labelling information results in an upward shift in the private demand curve, which now represents the private marginal benefit curve \( MB_p^{IN} \) for it includes the marginal health benefits that informed consumers gain from the consumption of a functional food. The provision of labelling information also shifts upwards the marginal cost curve, which now includes the labelling costs. The intercept of the private marginal benefit curve of informed consumers and the marginal cost curve increases the equilibrium price from \( P_p^{UN} \) to \( P_p^{IN} \), and the equilibrium quantity from \( Q_p^{UN} \) to \( Q_p^{IN} \). The consumer surplus increases and equals to the area \( FJP_p^{IN} \). The private health benefits are equal to the area \( FJKE \), while the area \( GJKD \) represents the health benefits that informed consumers gain from the additional consumption of a functional food due to labelling. The external social health benefits in terms of health care savings are increased, too, and are equal to the area \( AIJF \). This means that distribution of information ameliorates consumers’ health and, hence, increases health savings (area \( BIJG \)). Despite labelling information, society still bears dead weight loss equal to \( ILJ \) due to the negative externalities caused by public or private health insurances.

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10 Canadian regulators also allow natural health products (vitamins and minerals, herbal products, homeopathic medicines and probiotics) in the market. Consumers now have the chance to protect and improve their health via natural health products (Canada Gazette, 2003a).
In case the market structure of natural functional foods is non competitive, there would be an oligopolistic price markup, meaning that the price would be greater than the marginal cost. We consider the oligopolistic case in the later discussion regarding industrial functional foods.

![Figure 3.4. Natural functional food: Private and social welfare benefits due to labelling information.](image_url)

The situation is different in the case of industrial functional foods. As was mentioned in section 3.4.1, in the absence of health information, industrial functional foods are not available in the market because their costs are higher and consumers cannot distinguish between them and conventional foods. As Figure 3.5 depicts, the marginal cost curve of an industrial functional food is higher than that of a natural functional food, resulting in zero consumption when there is no food labelling. However, with the introduction of labelling, food companies start producing foods with health-enhancing properties by adding, enriching, or removing food additives. Thus, a wide range of
functional foods enter the market, giving consumers the choice to switch to a healthy balanced diet, thereby reducing health care costs.

The introduction of labelling information leads to an upward shift in the marginal cost curve, which represents the marginal cost including the labelling costs. Given the fixed costs and the limited number of functional food firms, there would be a markup over price, called as oligopolistic price markup. This markup is the difference between the oligopolistic price \((P_{\text{ind}})\) and the marginal cost \((MC + \text{labelling costs})\).

The welfare effect of the introduction of technologically developed functional foods is showed in Figure 3.5. The consumer surplus is given by the area \(EDP_{\text{ind}}\) while the producer surplus equals to the area \(P_{\text{ind}}DGF\). The external social health benefits from a reduction in health care costs are equal to the area \(ABDE\). The dead weight loss due to the negative externalities created by health insurances equals to \(BCG\).

![Figure 3.5. Industrial functional food: Private and social welfare benefits from health information.](image)

Figure 3.5. Industrial functional food: Private and social welfare benefits from health information.
As was shown graphically, both labels and claims found on food products help consumers make informative purchasing choices and increase the consumption of foods with healthy characteristics. Although consumers are not homogeneous—some consumers will pay more attention to food labels, such as those with a family history of a chronic disease—food labelling plays a crucial role in informing individuals and reducing health care costs. Yet, food labelling only partially corrects the market inefficiency because the private marginal benefit and the social marginal benefit curves cannot coincide due to the negative externality that exists in the market, to be discussed in section 3.6.

3.5. Voluntary versus Mandatory Food Labelling

Before mandatory food nutrition labelling was implemented in Canada, companies used voluntary labelling to differentiate their products. Food companies marketing products with healthy components were willing to label this component in order to capture a greater market share, while those supplying products with normal or negative attributes had no incentive to label. As a result of incomplete labelling, consumers often could not make purchasing decisions that would best reflect their preferences.

Mandatory labelling may be more effective policy for addressing information asymmetry. If companies are forced to label their products, they are more likely to modify their products to reflect consumer preferences. This kind of regulation encourages competition and increases research activities and innovation. With greater access to food information, consumers can choose to increase the consumption of healthy food products in order to lower the risk of nutrition-related diseases. Although a regulation to label almost all prepackaged food products would increase costs (e.g., cost of product reformulation, product testing, labelling, and marketing), the health care savings could be large enough to outweigh the costs, leading to a healthier population and general economic gain.

Gray et al. (2006) studied the efficacy of three different policies designed to limit the consumption of trans fatty acids (TFAs). A voluntary labelling system, a mandatory labelling system, and a ban on food products with TFA greater than 2% were compared.
The results showed that the cost to industry from mandatory labelling is greater than voluntary labelling, but that coronary heart disease (CHD) health benefits are by far larger in magnitude, making mandatory TFA labelling very advantageous for the Canadian economy. An additional economic gain would be also achieved from a TFA ban (Gray et al., 2006).

In Figure 3.6, the private and social health benefits from voluntary and mandatory labelling are depicted. The marginal cost for voluntary and mandatory labelling is assumed to be the same. The private marginal benefit curve in the case of a voluntary labelling system lies below the private marginal benefit curve in the case of a mandatory labelling due to limited information provided to consumers, resulting in reduced health care savings. The market structure of industrial functional foods may be oligopolistic, meaning that the price under mandatory labelling \( P_{\text{mand}} \) and the price under voluntary labelling \( P_{\text{vol}} \) are greater than the marginal cost. The quantity consumed \( Q_{P_{\text{fod}}}^{IN} \) of an industrial (or natural) functional food when it is voluntarily labeled is less than the quantity consumed \( Q_{P_{\text{fod}}}^{IN} \) when consumers gain full information through mandatory labelling. The shaded area \( HBGI \) represents the social (private and external social) health savings that are forgone when consumers are not fully informed due to a voluntary labelling system. Hence, mandatory food labelling can contribute to an improvement in individuals’ health and enhance economic efficiency. However, it is worth noting that if the mandatory labelling cost was significantly higher than voluntary labelling cost, the welfare impacts of mandatory labeling would no longer be unambiguously positive.
3.6. Negative Externality Effect and Possible Solutions

Although nutrition information can play an important role in improving diet and thus reducing both private and social health care costs to a significant extent, as was shown in Figures 3.4 and 3.5, the economic incentives created by either public or private health insurance limits the socially optimal level of functional food consumption. Insured consumers have a lower incentive to consume products with nutritious properties as long as they are protected by medical and disability insurance, which covers a large amount of the costs associated with various chronic conditions. The large health-care externality due to health insurance creates a moral hazard by reducing the incentive to consume a socially appropriate amount of a functional food. This market inefficiency deters even the informed consumers from fully modifying their diets, thereby having a negative effect on social welfare. In Figures 3.4 and 3.5, the social welfare loss due to the negative
externality effect is equal to the areas $ILJ$ and $BCG$, respectively. So, it is clear that some proportion of health care costs can be reduced only if this market failure is corrected.

In theory, there are several propositions that could be used as solutions to this problem, but, as is discussed below, all are difficult to implement (Gray and Malla, 2004). The first would be to correct the market failure through an assignment of property rights. Governments could assign the cost of medical treatment to individuals. In this case, individuals would therefore be more careful of what foods they consume and would have a greater incentive to adopt a healthier lifestyle. However, this policy would unlikely achieve the intended results as private insurance would become popular, as in the United States, leaving a scope for moral hazard within these insurance pools.

A second possible solution would be the modification and improvement of private and public health insurance contracts to better reflect consumer choices. However, given the large influence of genetics and other risk factors in determining disease risk, altering insurance payoffs may be insufficient to eliminate the externality (Malla et al., 2007). Also, it would be difficult and extremely expensive to inspect an individual’s intake of components with adverse health effects. Thus, the consumption externality would continue to affect negatively economic welfare. In such cases, government intervention might be necessary to correct the market failure caused by the externality.

The third policy option would be to regulate the production and importation of certain food products with harmful substances. The regulatory limits on the amount of the externality produced and imported could improve market outcomes. The U.S. Food and Drug Administration has already enforced this type of regulation for certain food additives that are prohibited from use because of cancer or other disease threat (Gray and Malla, 2004). In Canada, similar restrictions have been placed on imports of certain U.S. animals and their products to protect human health (Canada Gazette, 2007).

The fourth solution would be to either impose a tax on the production or consumption of foods with a negative externality or implement a subsidy on the consumption of healthier foods. The size of a Pigovian tax (subsidy) would be equal to the damage (benefit) of the negative (positive) externality. This means that a government can enforce a tax on products with unhealthy attributes equal to the health care costs, or provide subsidies to encourage the consumption of nutritious and healthy goods. The
regulation of Pigovian taxation offers an effective means of achieving market efficiency by addressing incentive problems and increasing economic surplus. However, it is considered very complex and difficult to control because each food with a harmful (beneficial) health additive would have its own level of tax (subsidy), something costly to implement.

Although so-called “fat taxes” have been proposed, mostly as a means of addressing food-related concerns, it has been suggested that “thin subsidies” would be a way of tackling unhealthy eating habits (Cash et al., 2005). Fat taxes aim to discourage the purchase of foods that are least nutritious or most harmful by increasing the effective price to consumers. However, subsidizing the costs of healthy food would reward and encourage people to eat healthier foods rather than punish them when they eat poorly (Cash et al., 2005). Making unhealthy food products more expensive may potentially decrease their consumption, even though it is uncertain whether that would lead consumers to switch to healthier products. According to Cash, if the government subsidized fruits and vegetables to lower their price by just one percent, 9,680 fewer cases of coronary heart disease and ischemic stroke would occur per year in U.S. (Cash et al., 2005). Therefore, instituting subsidies for the consumption of healthier food products may have a greater outcome on reducing health care costs than taxng harmful foods.

3.7. Conclusion

This chapter discussed the current regulatory framework in Canada and indicated how mandatory labelling and claims can affect purchasing decisions and improve health. However, the limited number of allowable nutrient and health claims prevents increased consumption of some nutritious food products because consumers are unaware of this information. This decreases the food industry’s incentive to perform research and supply the market with healthy products. A well-organized and modern regulatory system is necessary in order to encourage research, investment, and international trade, and reduce the size of deadweight losses identified in this chapter.

The chapter graphically illustrated the beneficial effects of food labelling on private and social welfare. Specifically, the graphical model illustrated how the policy of providing health information assisted consumers in modifying their diet, and thus
decreased health care costs. The critical role that both food labels and claims have in influencing the population’s health through changing consumer demand for functional foods is apparent in the graphs. Individuals now have the option to make informed food choices and improve their health by reducing the risks of a chronic disease. The more informed that consumers become the greater the savings in the cost of illness, as long as information is clear, truthful, and based on scientific evidence.

In Chapter Four, the measurement of welfare benefits and costs from food labelling is estimated to evaluate the current contribution of information to the economy. In particular, the net effect of labelling omega-3 enriched eggs is calculated by estimating health care cost savings, as well as production and labelling-related costs.
CHAPTER 4: A BENEFIT COST ANALYSIS OF OMEGA-3 ENRICHED EGGS IN CANADA

4.1. Introduction

The aim of this chapter is to test the hypothesis stated at the outset by conducting a benefit cost analysis of omega-3 enriched eggs. Such analysis is required to gain a better understanding of the associated benefits and costs which will then be used to investigate the net social benefits, if they exist, of labelling omega-3 enriched eggs. The use of labelling and nutrient content claims in omega-3 enriched eggs can contribute to health improvement, particularly by reducing the risk of CHD, thereby reducing health care costs. However, the enhancement of eggs with omega-3 PUFA, as well as their labelling and marketing, create extra costs for egg producers. Comparing the estimated health benefits and costs of producing and marketing omega-3 enriched eggs, the economic impact of providing information through labelling can be measured.

This chapter begins with an estimated increase in the level of omega-3 PUFA (ALA, EPA+DHA) if consumers were to substitute a regular egg with an omega-3 enriched egg. The chapter then provides a review of plant-derived omega-3 and marine-derived omega-3 studies which have been carefully examined and used to estimate the relationship between ALA and the risk in CHD mortality and between EPA+DHA and the risk in CHD mortality. Based on this relationship the related health care savings are estimated. An estimation of production and other costs (labelling, testing and marketing) of omega-3 enriched eggs follows. The chapter concludes with a measurement of the net benefit and benefit cost (B/C) ratio, which will determine the efficacy of Canada’s labelling policy.

4.2. Description of the Methodology

To quantify the potential economic impact of the health promoting effects of omega-3 enriched eggs analyzed in section 2.5, it is useful to develop estimates under a
range of plausible assumptions. The analysis is supported by scientific evidence from the health science literature and is based on a range of assumptions about the level of the effect of omega-3 fatty acids on coronary heart disease (CHD), the categories of medical costs (direct and indirect) that are used, and the link between CHD and health costs. The purpose of the analysis is to quantify the range of potential health care cost savings from labelling omega-3 enriched eggs and to reveal the sensitivity of the estimates to changes in key assumptions of the analysis.

In order to provide a plausible range of estimates for the reduction in CHD cost, three different scenarios are reported. The High Scenario includes the most optimistic estimation of health care costs savings, the Base Scenario is a reasonable estimate based on the medical and nutritional information, and the Low Scenario presents a conservative estimate.

For the measurement of the potential health care savings due to the consumption of omega-3 enriched eggs, three steps, similar to those taken by Malla et al. (2007), are followed:

1. Estimation of the possible increased omega-3 fatty acids intake (ALA and EPA+DHA) due to consumption of an omega-3 enriched egg.
2. Calculation of the reduction of CHD mortality due to increased omega-3 fatty acids (ALA and EPA+DHA) intake by using information reported in health studies.
3. Calculate cost savings related to reduced CHD.

After estimating the potential health benefits of omega-3 PUFA obtained from the current consumption of omega-3 enriched eggs, an estimation is provided of the costs incurred by the current production, labelling, and marketing of omega-3 enriched eggs. Specifically, the extra feed and the fixed, labelling, testing, and marketing costs that egg producers bear are calculated based on various assumptions. Then, taking into consideration both benefits and costs, the overall impact on society is calculated in order to evaluate the efficiency of labelling omega-3 enriched eggs.
4.3. An Estimated Increase in Omega-3 Fatty Acids Intake via the Consumption of an Omega-3 Enriched Egg

Attempting to decrease the risk of diet-related illnesses and improve Canadians’ health, omega-3 enriched eggs were developed to respond to health-conscious consumers’ needs. These novel eggs are considered a crucial functional food category due to the high content of omega-3 polyunsaturated fatty acids. Compared to regular eggs, the additional amount of omega-3 fatty acids found in omega-3 enriched eggs has the potential to make a major contribution to health improvement and wellness, and thus a likely reduction in CHD risk costs. The first step of the benefit analysis is focused on the specification of the extra ALA and EPA+DHA intake received from the consumption of omega-3 enriched eggs instead of conventional eggs.

As indicated in Table 2.2 (subsection 2.5.3), omega-3 enriched eggs contain much higher levels of total omega-3 fatty acids (0.50 g) than do regular eggs (0.04 g). On average 0.34 g (68% of total omega-3 fatty acids) of ALA, 0.13 g (26% of total) of EPA+DHA, and 0.03 g (6% of total) of several minor omega-3 fatty acids are included in an omega-3 enriched egg. Using the same proportions, it is calculated that 0.0272 g of ALA, 0.0104 g of EPA+DHA, and 0.0024 g of minor omega-3 fatty acids are present in regular eggs.

By knowing the level of omega-3 fatty acids in both omega-3 enriched eggs and regular eggs, we can calculate that consumers gain an additional in 0.313 g of ALA and 0.12 g of EPA+DHA if they substitute conventional eggs with omega-3 enriched eggs. Based on this increase in omega-3 fatty acids, the potential health care savings can be estimated once the relationship between ALA and EPA+DHA intake and the risk in CHD mortality is specified.

4.4. An Analysis of Plant-derived Omega-3 Fatty Acids (ALA)

4.4.1. A Brief Review of ALA Studies

A search of the literature has revealed a substantial number of experimental (randomized controlled trials) and observational studies (prospective cohort studies, case-
control studies, and cross-sectional studies)\textsuperscript{11} discussing and measuring the effect of ALA intake on fatal and non-fatal CHD, as well as on CVD events in general. Most of the studies suggest that an increased intake of ALA is recommended for both primary and secondary prevention of CHD due to its cardioprotective and antiarrhythmic effects. These studies show a significant reduction in the incidence of CHD as well as in mortality (Hu et al., 1999b; Dolecek, 1992; Dolecek and Grandits, 1991; Pietinen et al., 1997; de-Lorgeril et al., 1999; Singh et al., 1997; Singh et al., 2002). However, other research studies have reported no such inverse associations (Ascherio et al., 1996; Oomen et al., 2001).

Although some studies measured the effect of ALA intake on the incidence of CHD, the estimates in this thesis are based on those studies that reported a reduction in the risk of CHD mortality. This was done because the majority of ALA studies examined the effect of ALA intake on the risk of fatal CHD rather than CHD events (fatal and non-fatal). After reviewing the nutritional literature, a total of nine studies were identified. Six observational studies (five prospective cohort studies and one cross-sectional study) examined the effects of ALA intake in people without previously diagnosed cardiovascular disease (primary prevention). The remaining three, which are experimental studies (specifically, randomized controlled trials), measured the impact of ALA intake on individuals with a history of a heart disease event (secondary prevention). Five of the nine studies were excluded from the estimation for reasons explained below.

One of the primary prevention studies, a cross-sectional examination by Djoussé et al. (2001) that included 4,406 participants was omitted because, while it examines the effect of ALA intake on the risk of coronary artery disease (CAD)\textsuperscript{12} mortality, it reports only the prevalence odds ratio, which differs from the relative risk that all the other studies report. Nonetheless, the results of this study suggest that a higher intake of ALA is associated with a lower prevalence odds ratio of coronary artery disease (CAD) in both men and women.

Another primary prevention study, the Multiple Risk Factor Intervention Trial (MRFIT), which consisted of 12,866 male participants aged 35-57 years, was also

\textsuperscript{11} For definitions, see the Glossary of Terms in Appendix B.
\textsuperscript{12} Coronary Artery Disease (CAD) is the term given to heart problems caused by narrowed heart arteries. This can ultimately lead to a heart attack (American Heart Association, 2007).
omitted because participants were determined to be at high risk of CHD and may have biased the results. Nevertheless, Dolecek (1992) and Dolecek and Granditis (1991) showed an inverse association between ALA intake and fatal CHD, fatal CVD, and all-cause mortality. Specifically, a significant 32% reduction in CHD mortality was reported when the highest (2.81 g/d) and lowest (0.87 g/d) quintiles of ALA intake were compared.

Three additional secondary prevention studies (randomized controlled trials) were not taken into consideration for the estimation because omega-3 enriched eggs are aimed at the general population, not those with a history of CHD. This is because healthy people can maintain both their health status and reduce the risk of an incidence of CHD by keeping a balanced level of omega-3 fatty acids through consumption of omega-3 enriched eggs. However, people who have experienced a CHD event follow special treatments, which probably include drugs and supplements. Thus, these studies may be biased, which, according to Wang et al. (2004), could invalidate the results. Nevertheless, it is worth mentioning the outcomes of these results, which indicated a beneficial effect of ALA intake on heart disease mortality.

In the Lyon Diet Heart Study, a randomized single-blind secondary prevention trial, a Mediterranean-type diet was compared with a prudent Western-type diet in myocardial infarction (MI) (heart attack) survivors. The experimental group (192 patients), which consumed a Mediterranean diet enriched with 1.8 g/d ALA, had a significant 65% reduction of cardiac deaths compared with the control group (219 patients), which consumed 0.67 g/d ALA as part of their usual diet (de-Lorgeril et al., 1994; de-Lorgeril et al., 1999).

In a double-blind, placebo-controlled trial in India, all patients had experienced an acute myocardial infarction (AMI) (heart attack). One hundred and twenty patients in the experimental group received 20 g/d mustard oil containing 2.9 g/d ALA, an amount that was 3.6 times higher than the one that the 118 patients of the control group received. Subjects consuming mustard oil had a significant 40% lower risk of cardiac death than those receiving a placebo treatment (Singh et al., 1997).

The last secondary study was another Indian trial conducted by Singh et al. (2002). Four hundred and ninety-nine patients in the experimental group consumed an
Indo-Mediterranean diet rich in whole grains, fruits, vegetables, walnuts, and almonds, while 501 patients in the placebo (control) group consumed a local diet similar to the step I National Cholesterol Education Program (NCEP) prudent diet. Patients in the intervention group, who consumed 1.8 g/d ALA, experienced a significant 67% reduction in total cardiac deaths than those in the control group who consumed 0.8 g/d ALA.

A total of four primary prevention studies (prospective cohort studies) were used to estimate the link between ALA intake and the risk of CHD mortality. Two concluded a negative relation between ALA intake and fatal CHD, while the other two observed no beneficial effect of ALA intake on the risk of CHD death. A review of the results of the primary prevention studies follows.

Hu et al. (1999b) assessed the intake of ALA by use of a 116-item food-frequency questionnaire given to 76,283 female nurses, aged 30-55 years, without previously diagnosed cancer or cardiovascular disease. In this study, approximately 70% of ALA was derived from vegetable or plant sources (e.g., salad dressings and vegetable oils). The result showed that the highest quintile of ALA intake (1.36 g/d) was significantly associated with a 45% reduction in fatal CHD compared to the lowest quintile of ALA intake (0.71 g/d).

Pietinen et al. (1997) examined the relation between intake of ALA and the risk of CHD in 21,930 male smokers, aged 50-69 years, in Finland who were free of diagnosed CVD. All participants in the Finnish Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study completed a detailed diet questionnaire. The study observed an inverse association between the intake of ALA and risk of coronary death in simultaneous models. In particular, a significant 25% reduction in the risk of coronary death was reported when the highest quintile (2.5 g/d) of ALA intake was juxtaposed against the lowest quintile (0.9 g/d).

The following two primary prevention studies found no association between ALA intake and CHD mortality. In the Health Professionals Follow-Up Study, 43,757 U.S. health professionals aged, 40-75 years and free of diagnosed CVD or diabetes, completed a 131-item food-frequency questionnaire. The study reported a lower risk of total myocardial infarction (including non-fatal MI and fatal CHD), but not of fatal CHD (Ascherio et al., 1996).
In the Zutphen Elderly Study conducted by Oomen et al. (2001) in Netherlands, the study population consisted of men aged 64-84 years and free of CAD (Coronary Artery Disease) at baseline. Dietary ALA intake was assessed by using a cross-check dietary history method adapted to the Duch habitual food consumption pattern (Bloemberg et al., 1989). The study’s outcome showed a non-beneficial effect of dietary ALA intake on the risk of CAD incidence and mortality. The possible increased risk of CAD may have been associated with the consumption of foods that contained trans fatty acids (e.g., margarines and meat), which were the main sources of ALA.

According to Wang et al. (2004), all four prospective studies had a good methodological quality, and some provide strong evidence for the fundamental role of ALA in benefiting health and preventing fatal heart disease. However, the inconsistent results found in two of the primary prevention studies create doubt regarding the health promoting effects of ALA. This heterogeneity might be explained by the fact that ALA can come from different dietary sources (e.g., meat or vegetable oils), which creates difficulty in recognizing whether ALA or other dietary factors impact the risk of heart disease (Brouwer et al., 2004). Also, the effects of risk factors for heart disease, such as tobacco or alcohol use, can substantially impact the final result.

The next subsection will examine the way in which the relationship between the intake of 1 g/d ALA and CHD mortality has been estimated by using all the above-mentioned studies.

4.4.2. Estimated Relationship of ALA and CHD Mortality

The four primary prevention studies (prospective cohort studies) provided all the information necessary to estimate the effect of ALA intake on CHD mortality. Table 4.1 summarizes the key elements of these studies, which contributed to the final combined estimation. The process used to find the estimated change in the risk of fatal CHD from a 0.1 g increase in ALA is explained below.

All the studies save for Oomen et al. (2001) reported the intake of ALA in g/d. The lowest intake of ALA is characterized as the base, which was compared with the
higher intakes of ALA called “trials.” The studies also measured the relative risks\textsuperscript{13} of fatal CHD for each different dose of ALA. For example, Hu et al. (1999b) found that a higher intake of ALA was associated with a lower relative risk (RR) of fatal CHD. Specifically, from the lowest to the highest intakes of ALA in g/d, 0.71, 0.86, 0.98, 1.12, and 1.36, the relative risks were 1, 0.99, 0.90, 0.67, and 0.55, respectively. The increase of both ALA intake and relative risk were calculated and are shown in Table 4.1. For instance, considering the study conducted by Hu et al., increasing ALA intake by 0.65 g/d (named $\Delta$ALA(trial 4-base) on the table) can decrease the relative risk by 45% (named $\Delta$RR (base-trial 4) in the table). In other words, an extra 0.65 g/d ALA could reduce the risk of CHD mortality by 45%. In order to make the results of the studies comparable, it was necessary to calculate the change in risk of CHD mortality due to a 0.1 g increase in intake of ALA. Because this change has been measured for each trial of the studies, the average change in risk of fatal CHD due to a 0.1 g increase in the dose of ALA was calculated for every study. The main assumption at this point is linearity in the relationship between ALA intake and the risk of CHD mortality.

In order to find accurate and consistent results, the weighted average was considered so that studies with larger numbers of participants and longer durations carried more weight. This is because the number of participants may affect the credibility of the study. The larger the number of participants, the more credible the study may be. The duration of studies was also of particular interest as the effect of ALA intake on the risk of heart disease can only be realized in the long-run, especially in the primary prevention of CHD death. The total weighted average for every 0.1 g/d increase in ALA intake was found to be 0.02. In other words, for every 0.1 g/d increase in ALA, the risk of fatal CHD is reduced by 2%.

\textsuperscript{13} The relative risks, which have been adjusted for risk factors of CHD as well as dietary factors, were used for the estimation. The relative risk is used in research studies to compare the likelihood of an event between two groups (the experimental and the control).
Table 4.1. Estimation of CHD change due to 0.1 g per day ALA based on four prospective cohort studies.

<table>
<thead>
<tr>
<th></th>
<th>Hu et al., 1999b</th>
<th>Pietinen et al., 1997</th>
<th>Ascherio et al., 1996</th>
<th>Oomen et al., 2001^1</th>
<th>SUM</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ALA base (g/d)</strong></td>
<td>0.71</td>
<td>0.9</td>
<td>0.8</td>
<td>0.98</td>
<td></td>
</tr>
<tr>
<td><strong>ALA trial 1 (g/d)</strong></td>
<td>0.86</td>
<td>1.2</td>
<td>0.9</td>
<td>1.25</td>
<td></td>
</tr>
<tr>
<td><strong>ALA trial 2 (g/d)</strong></td>
<td>0.98</td>
<td>1.5</td>
<td>1.1</td>
<td>1.64</td>
<td></td>
</tr>
<tr>
<td><strong>ALA trial 3 (g/d)</strong></td>
<td>1.12</td>
<td>1.9</td>
<td>1.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ALA trial 4 (g/d)</strong></td>
<td>1.36</td>
<td>2.5</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔALA (base-base) (g/d)</strong></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td><strong>ΔALA (trial 1-base) (g/d)</strong></td>
<td>0.15</td>
<td>0.3</td>
<td>0.1</td>
<td>0.27</td>
<td>0.27</td>
</tr>
<tr>
<td><strong>ΔALA (trial 2-base) (g/d)</strong></td>
<td>0.27</td>
<td>0.6</td>
<td>0.3</td>
<td>0.66</td>
<td>0.66</td>
</tr>
<tr>
<td><strong>ΔALA (trial 3-base) (g/d)</strong></td>
<td>0.41</td>
<td>1</td>
<td>0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔALA (trial 4-base) (g/d)</strong></td>
<td>0.65</td>
<td>1.6</td>
<td>0.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>RR base (Pbase/Phase)</strong></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>RR trial 1 (Ptrial 1/Pbase)</strong></td>
<td>0.99</td>
<td>0.95</td>
<td>1.14</td>
<td>0.99</td>
<td>0.99</td>
</tr>
<tr>
<td><strong>RR trial 2 (Ptrial 2/Pbase)</strong></td>
<td>0.90</td>
<td>0.94</td>
<td>1.04</td>
<td>1.59</td>
<td>1.59</td>
</tr>
<tr>
<td><strong>RR trial 3 (Ptrial 3/Pbase)</strong></td>
<td>0.67</td>
<td>0.87</td>
<td>1.38</td>
<td></td>
<td></td>
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<tr>
<td><strong>RR trial 4 (Ptrial 4/Pbase)</strong></td>
<td>0.55</td>
<td>0.75</td>
<td>1.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔRR (base-base) (Δ(Pbase/Phase))</strong></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 1) (Δ(Ptrial 1/Pbase))</strong></td>
<td>0.01</td>
<td>0.05</td>
<td>-0.14</td>
<td>0.01</td>
<td>0.01</td>
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<tr>
<td><strong>ΔRR (base-trial 2) (Δ(Ptrial 2/Pbase))</strong></td>
<td>0.1</td>
<td>0.06</td>
<td>-0.04</td>
<td>-0.59</td>
<td>-0.59</td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 3) (Δ(Ptrial 3/Pbase))</strong></td>
<td>0.33</td>
<td>0.13</td>
<td>-0.38</td>
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<td></td>
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<tr>
<td><strong>ΔRR (base-trial 4) (Δ(Ptrial 4/Pbase))</strong></td>
<td>0.45</td>
<td>0.25</td>
<td>-0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔRR (base)/0.1ΔALA</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 1)/0.1ΔALA</strong></td>
<td>0.01</td>
<td>0.02</td>
<td>-0.14</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 2)/0.1ΔALA</strong></td>
<td>0.04</td>
<td>0.01</td>
<td>-0.01</td>
<td>-0.09</td>
<td>-0.09</td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 3)/0.1ΔALA</strong></td>
<td>0.08</td>
<td>0.01</td>
<td>-0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔRR (base-trial 4)/0.1ΔALA</strong></td>
<td>0.07</td>
<td>0.02</td>
<td>-0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Average ΔRR/0.1ΔALA</strong></td>
<td>0.05</td>
<td>0.01</td>
<td>-0.06</td>
<td>-0.04</td>
<td>-0.04</td>
</tr>
<tr>
<td><strong>Number of follow-up years</strong></td>
<td>10</td>
<td>6.1</td>
<td>6</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td><strong>Number of Participants</strong></td>
<td>76,283</td>
<td>29,930</td>
<td>43,757</td>
<td>667</td>
<td>1,214,615</td>
</tr>
<tr>
<td><strong>Years * Participants</strong></td>
<td>762,830</td>
<td>182,573</td>
<td>262,542</td>
<td>6,670</td>
<td></td>
</tr>
<tr>
<td><strong>Total weighted average</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td><strong>0.02</strong></td>
<td></td>
</tr>
</tbody>
</table>
The median intakes for the tertiles were 0.40%, 0.50%, and 0.67% of total energy. The ALA intake in g/d has been calculated by using 1 g=9 kcal conversion based on 2,200 kcal daily energy intake.

ALA base g/d is the lowest intake of ALA provided to subjects as a comparison with the higher intakes, which are named “trials.”

The change in ALA intake (ΔALA) is calculated by subtracting the lowest intake of ALA (ALA base) from the higher intakes (ALA trials).

The relative risk (RR) of CHD mortality, as has been reported by the studies. The RR is the ratio of the probability of CHD mortality occurring in the trials versus the base.

The change in relative risk (ΔRR) is calculated by subtracting the RR of the higher intakes (RR Trials) from the RR of the lowest intake (RR base).

The change in RR due to 0.1 g change in intake of ALA has been calculated by multiplying the change in RR (ΔRR) by 0.1 and dividing by the change in ALA intake (ΔALA).

The average ΔRR/0.1ΔALA was calculated by taking the average of [ΔRR (base-trial 1)/0.1ΔALA, ΔRR (base-trial 2)/0.1ΔALA, ΔRR (base-trial 3)/0.1ΔALA, ΔRR (base-trial 4)/0.1ΔALA].

The total weighted average is the sum of the weighted averages found in each study. The weighted average for each study = (average ΔRR/0.1ΔALA*(Years*Participants))/Sum of (Years*Participants). The weighted average for each study is:

Hu et al., (1999b): weighted average = 0.03
Pietinen et al., (1997): weighed average = 0.002
Ascherio et al., (1996): weighted average = -0.01

Source: Author’s calculations (see text for detail).
4.5. An Analysis of Marine-derived Omega-3 Fatty Acids (EPA+DHA)

4.5.1. Review of EPA+DHA Studies

After the observations of the preceding section were made, a number of further studies were examined for the association between EPA+DHA and the risk of CHD. A literature review of observational and experimental studies identified twenty-three studies (eighteen primary prevention studies and five secondary studies) examining the relationship between fish consumption or EPA+DHA intake and the risk of CHD mortality. From these studies, eleven primary prevention studies were excluded because they did not provide the information necessary to quantify this relationship. In particular, two studies (Pietinen et al., 1997; Salonen et al., 1995) were excluded because they underlined that the possible harmful effects of fish consumption in Finland could be explained by the high intake of mercury from contaminated fish. Four other studies (Vollset et al., 1985; Norell et al., 1986; Shekelle et al., 1985; Curb and Reed, 1985) were eliminated because they were published as short reports (e.g., letters to editors) that provided incomplete information on RR estimation or fish intake (He et al., 2004). Two more studies were omitted because one (Mann et al., 1997) was restricted to health-conscious individuals while the other (Rodriguez et al., 1996) comprised heavy smokers (>30 cigarettes/d). Another study (Dolecek and Granditis, 1991; Dolecek, 1992) was omitted because the participants were determined to be at high risk of CHD. The last two studies, Yuan et al. (2001) and Osler et al. (2003), were removed from the estimation because the former did not report estimates for CHD mortality combined but rather provided estimates separately for AMI mortality and for other ischemic heart disease (IHD) mortality, while in the latter study the control group was the one that received an intermediate level of fish intake, not the lowest level, as was done in the other studies.

As for the five secondary prevention studies (four randomized controlled trials and one prospective cohort study), they are not included in the estimation because, as mentioned in subsection 4.4.1, omega-3 enriched eggs were principally targeted to improve the health of the general population, not patients who possibly follow a special
treatment. Also, those studies are based on supplements, and the higher amounts of EPA+DHA intake were difficult to categorize.

The first is the well-known Diet and Reinfarction Trial (DART) (Burr et al. 1989), which showed that a modest intake of fatty fish (two or three portions per week) decreased the risk of total mortality by 29%, which was entirely attributable to a reduction in the number of deaths from coronary heart disease, and reduced the risk of CHD events (CHD mortality and non-fatal MI) by 16%. However, no estimation in the risk of CHD mortality was provided.

The second randomized controlled trial (Nilsen et al., 2001) included 300 patients, of which 150 received 3.5 g/d EPA+DHA, while the rest received corn oil. The results showed no association between EPA+DHA and a reduction in cardiac deaths.

The third study, the Italian GISSI (Grupo Italiano per lo Studio della Sopravvivenza nell’Infarto miocardioco)-Prevenzione trial involving 11,324 patients of both sexes (primarily men), is the largest prospective clinical trial to assess the benefit of omega-3 polyunsaturated fatty acids for secondary prevention of CHD. After a three-and-a-half year period, the experimental group, which received one fish oil capsule containing 850-882 mg of EPA+DHA as ethyl esters, experienced a significant 35% reduction in the risk of CHD death, 45% in the risk of sudden death, and 20% in the risk of all fatal events compared with the control group (GISSI, 1999).

In another randomized controlled trial conducted by Singh et al. (1997), patients received two capsules of fish oil three times daily, which contained a total of 1.8 g EPA+DHA, and experienced a 48% reduction in total cardiac death than those who received a placebo treatment.

The last secondary prevention study was a prospective cohort study conducted by Erkkilä et al. (2003). Compared with the zero fish intake, the researchers reported an inverse association between the highest fish intake (>57 g/d of fish) with low risks of all-cause mortality and the combined endpoint of CVD death, AMI (acute myocardial infarction), or stroke, but not with the CAD death.

In total, seven primary prevention studies (prospective cohort studies) that reported a link between fish consumption or EPA+DHA intake and CHD mortality were used in the analysis. The results of these studies are conflicting as some studies found an
inverse relationship between fish consumption or EPA+DHA intake and heart disease, while others report no such benefit. However, all the studies are taken into consideration, and their main results are mentioned below.

The Nurses’ Health Study conducted by Hu et al. (2002) included 84,688 healthy female nurses, aged 34-59 years, and used a semiquantitative food frequency questionnaire to assess dietary intake of long chain omega-3 fatty acids and fish consumption. The study reported an inverse association between fish intake and long chain fatty acids (LCFAs) and CHD death. More specifically, consumption of five or more servings of fish per week was associated with a 45% reduction in the risk of CHD mortality compared to consumption of less than one fish serving per month. A 37% reduction in the risk of fatal CHD also was reported when the highest quintile (0.24 as the percentage of total energy) of long chain polyunsaturated fatty acids (LCPUFAs) was compared with the lowest quintile (0.03 as the percentage of total energy).

The Chicago Western Electric Study surveyed 1,822 healthy men, aged 40-55, years and reported a significant 38% lower risk of fatal CHD in men who consumed at least 35 g of fish daily compared with those who consumed none (Daviglus et al., 1997a).

The Zutphen Study, conducted by Kromhout et al. (1985), demonstrated a protective effect of fish consumption on CHD death among 852 middle-aged Dutch men during twenty years of follow-up. In particular, mortality from CHD was more than 50% lower among those who consumed at least 30 g of fish per day than those who did not eat fish.

In the Cardiovascular Health Study, fish consumption was ascertained at baseline among 3,910 adults, aged ≥ 65 years and free of known cardiovascular disease. The result showed that consumption of at least three servings of tuna or other broiled or baked fish per week was significantly related to a 53% lower risk of IHD death (Mozaffarian et al., 2003).

In a recent European study carried out by Oomen et al. (2000), the data suggest that fish consumption, especially fatty fish, is protective against CHD mortality. This association was examined in 1,088 Finnish, 1,097 Italian, and 553 Dutch men, aged 50-69 years and free of CHD. In Finland and the Netherlands, no relation between total fish
consumption and CHD mortality was observed. In Italy, men who consumed ≥40 g/d of fish had a non-significant 31% reduction in fatal CHD compared with men who consumed no fish. However, in the case where the three population samples consumed fatty fish instead of lean fish, inverse associations were observed between fatty fish consumption and CHD death for all three countries. Specifically, the pooled results for fatty fish were connected with a 34% reduction in the risk of CHD mortality. It should be mentioned, however, that in this analysis only the participants from Italy were taken into consideration. Dutch participants were part of the Kromhout et al. (1985) study and double counting information was avoided, while the Finnish population has been ascertained to have increased risk of CHD mortality even though it consumes great quantities of fish. The Finnish results, however, are probably due to the high mercury content of fish, something also observed in two other studies (Salonen et al., 1995; Pietinen et al., 1997).

Two of the primary prevention studies did not report a beneficial association between fish consumption or LCPUFA and CHD mortality. In the Physicians’ Health Study, involving 20,551 healthy US male physicians, aged 40-84 years, fish consumption assessed by semiquantitative food frequency questionnaire offered no protection against coronary heart disease mortality. However, total mortality and sudden cardiac death were indeed reduced in those who consumed fish. It should be noted, however, that the size of the reduction did not appear to differ substantially at levels of consumption greater than one fish serving per week, suggesting a threshold effect (Albert et al., 1998).

Likewise, the Health Professionals Follow-up Study, a large-scale prospective cohort study that consisted of 44,895 male health professionals, aged 40-75 years and were initially free of cardiovascular disease, did not show an association between fish intake and EPA+DHA and a reduced risk of fatal CHD (Ascherio et al., 1995).

There are a number of factors that might contribute to the apparently discordant outcomes among the studies regarding the relationship between fish consumption or EPA+DHA intake and coronary heart disease. This noticeable inconsistency may be due to:
(1) different methods of assessing diet and categorizing fish consumption (Daviglus et al., 1997b);
(2) different distribution of reported fish (Daviglus et al., 1997b);
(3) different types of fish consumed (fatty versus lean fish) (Daviglus et al., 1997b);
(4) differences in study sites and times, with associated dietary differences (e.g., levels of intake of cholesterol, saturated fats, antioxidants, and fiber); (Daviglus et al., 1997b);
(5) different duration of follow-up (Daviglus et al., 1997b);
(6) difference in study population (Wang et al., 2004);
(7) various demographic features (e.g., sex, age) (Wang et al., 2004);
(8) difference in subject characteristics (e.g., lipid levels, weight, blood pressure) (Wang et al., 2004); and
(9) impact of environmental contaminants (Wang et al., 2004).

4.5.2. Estimated Relationship of EPA+DHA and CHD Mortality

The primary prevention studies analyzed in the previous section provided the necessary information for the purpose of quantitatively investigating and assessing the impact of EPA+DHA intake, mainly through fish consumption, on the risk of CHD mortality. Table 4.2 summarizes the data from all the studies, which is necessary for the combined estimation, and depicts the procedure for estimating the change in risk of CHD mortality from a 0.1 g increase in EPA+DHA.

Contrary to the estimation regarding the relationship of ALA and the risk of fatal CHD, the analysis of the effect of EPA+DHA intake on the risk of CHD mortality is more complicated. This is due to design variations in the studies. Most assessed the impact of fish consumption on the risk of fatal CHD by using different amounts (grams) of fish per day, while others used different servings of fish. Only a few studies provided information on EPA+DHA intake in grams per day and its effect on death from CHD. As a result, an assumption was essential in order to convert the servings and grams of fish into EPA+DHA grams per day, and thus make the data comparable. König et al. (2005) assumed that one fish serving per week is equivalent to 100 g of fish per week.
König mentioned that this assumption is consistent with U.S. EPA estimates (U.S. Environmental Protection Agency, 1997). Carrington and Bolger (2002) reported average omega-3 concentrations (EPA+DHA) on the order of 1% in a market basket of fish typically consumed in the United States. This means that one fish serving per week, which has been assumed to be 100 g of fish per week, contains 1 g EPA+DHA per week or 0.14 g EPA+DHA per day. Therefore, this assumption was applied to those studies that expressed fish consumption in terms of servings or grams. In cases where studies reported intervals, the average of the interval was used. For example, two to four fish servings per week were considered as three servings per week. Where the lowest fish intake level had an open lower bound of, for instance, less or equal to one fish serving per month, a half-serving per month was assigned as the lower bound. However, where no upper bound was specified—for example, more or equal to five fish servings per week—then the highest definite amount—five fish servings—was used as the upper limit. The same notion held in cases where grams of fish are reported.

As was mentioned in the analysis of ALA intake, the base intake corresponds to the lowest EPA+DHA intake, which the control group received, while the trials correspond to higher levels of intake of EPA+DHA. The relative risks of CHD mortality for different intakes of EPA+DHA, reported in the studies were used for the estimation, and their values are depicted in the table. For example, Hu et al. (2002) found that an extra 0.513 g/d (called $\Delta(EPA+DHA)$ (trial 4-base) on the table) reduces the risk of CHD mortality by 38% (called $\Delta RR$ (base-trial 4) in the table). In order to be comparable, the change in EPA+DHA, as well as the change in relative risk when compared with the base, was calculated and used to measure the change in relative risk due to a 0.1 g increase in EPA+DHA intake in all studies. Then, the average change in relative risk was computed for all studies. This estimation was based on the assumption that the correlation between EPA+DHA and the risk of fatal CHD is linear.

For a consistent estimation, both the number of participants and the period of follow-up were used as weights for the calculation of weighed average (as was also done in the ALA analysis). Both weights are considered important factors for the reliability and significance of the study’s outcome. Therefore, the total weighted average for every
0.1 g/d increase in EPA+DHA intake was found to be 0.10. In other words, for every 0.1 g/d increase in EPA+DHA, the risk of fatal CHD is reduced by 10%.
<table>
<thead>
<tr>
<th></th>
<th>Hu et al., 2002</th>
<th>Daviglus et al., 1997a</th>
<th>Kromhout et al., 1985</th>
<th>Mozaffarian et al., 2003</th>
<th>Oomen et al., 2000</th>
<th>Albert et al., 1998</th>
<th>Ascherio et al., 1995</th>
<th>SUM</th>
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</thead>
<tbody>
<tr>
<td><strong>EPA+DHA Base (g/d)</strong></td>
<td>0.073</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.016</td>
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<tr>
<td><strong>EPA+DHA trial 1 (g/d)</strong></td>
<td>0.12</td>
<td>0.08</td>
<td>0.07</td>
<td>0.13</td>
<td>0.098</td>
<td>0.07</td>
<td>0.15</td>
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</tr>
<tr>
<td><strong>EPA+DHA trial 2 (g/d)</strong></td>
<td>0.195</td>
<td>0.24</td>
<td>0.22</td>
<td>0.267</td>
<td>0.288</td>
<td>0.14</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td><strong>EPA+DHA trial 3 (g/d)</strong></td>
<td>0.342</td>
<td>0.34</td>
<td>0.36</td>
<td>0.547</td>
<td>0.39</td>
<td>0.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>EPA+DHA trial 4 (g/d)</strong></td>
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<td></td>
<td>0.44</td>
<td>0.919</td>
<td></td>
<td></td>
<td>0.58</td>
<td></td>
</tr>
<tr>
<td><strong>Δ(EPA+DHA) (base-base) (g/d)</strong></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>ΔEPA+DHA (trial 1-base) (g/d)</strong></td>
<td>0.047</td>
<td>0.078</td>
<td>0.068</td>
<td>0.128</td>
<td>0.098</td>
<td>0.049</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td><strong>ΔEPA+DHA (trial 2-base) (g/d)</strong></td>
<td>0.122</td>
<td>0.24</td>
<td>0.215</td>
<td>0.267</td>
<td>0.288</td>
<td>0.124</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td><strong>ΔEPA+DHA (trial 3-base) (g/d)</strong></td>
<td>0.269</td>
<td>0.34</td>
<td>0.36</td>
<td>0.547</td>
<td>0.39</td>
<td>0.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ΔEPA+DHA (trial 4-base) (g/d)</strong></td>
<td>0.513</td>
<td>0.44</td>
<td>0.44</td>
<td>0.919</td>
<td></td>
<td></td>
<td>0.51</td>
<td></td>
</tr>
<tr>
<td><strong>RR base (Pbase/Phase)</strong></td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>RR trial 1 (Ptrial 1/Pbase)</td>
<td>0.93</td>
<td>0.88</td>
<td>0.64</td>
<td>0.78</td>
<td>0.94</td>
<td>1.18</td>
<td>1.14</td>
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</tr>
<tr>
<td>RR trial 2 (Ptrial 2/Pbase)</td>
<td>0.69</td>
<td>0.84</td>
<td>0.56</td>
<td>0.77</td>
<td>0.93</td>
<td>0.87</td>
<td>0.95</td>
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<tr>
<td>RR trial 3 (Ptrial 3/Pbase)</td>
<td>0.54</td>
<td>0.62</td>
<td>0.36</td>
<td>0.53</td>
<td>0.67</td>
<td>1.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RR trial 4 (Ptrial 4/Pbase)</td>
<td>0.62</td>
<td>0.39</td>
<td>0.47</td>
<td></td>
<td></td>
<td></td>
<td>1.03</td>
<td></td>
</tr>
<tr>
<td><strong>ARR (base-base) (Δ(Pbase/Phase))</strong></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>ARR (base-trial 1) (Δ(Ptrial 1/Pbase))</td>
<td>0.07</td>
<td>0.12</td>
<td>0.36</td>
<td>0.22</td>
<td>0.06</td>
<td>-0.18</td>
<td>-0.14</td>
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</tr>
<tr>
<td>ARR (base-trial 2) (Δ(Ptrial 2/Pbase))</td>
<td>0.31</td>
<td>0.16</td>
<td>0.44</td>
<td>0.23</td>
<td>0.07</td>
<td>0.13</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>ARR (base-trial 3) (Δ(Ptrial 3/Pbase))</td>
<td>0.46</td>
<td>0.38</td>
<td>0.64</td>
<td>0.47</td>
<td>0.33</td>
<td>-0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARR (base-trial 4) (Δ(Ptrial 4/Pbase))</td>
<td>0.38</td>
<td>0.61</td>
<td>0.53</td>
<td></td>
<td></td>
<td></td>
<td>-0.03</td>
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</tr>
<tr>
<td><strong>ARR (base-base)/0.1Δ(EPA+DHA)</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>ARR (base-trial 1)/0.1ΔEPA+DHA</td>
<td>0.15</td>
<td>0.15</td>
<td>0.53</td>
<td>0.17</td>
<td>0.06</td>
<td>-0.37</td>
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<td>ARR (base-trial 2)/0.1ΔEPA+DHA</td>
<td>0.25</td>
<td>0.07</td>
<td>0.20</td>
<td>0.09</td>
<td>0.02</td>
<td>0.10</td>
<td>0.03</td>
<td></td>
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<tr>
<td>ARR (base-trial 3)/0.1ΔEPA+DHA</td>
<td>0.17</td>
<td>0.11</td>
<td>0.18</td>
<td>0.09</td>
<td>0.08</td>
<td>-0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ARR (base-trial 4)/0.1ΔEPA+DHA</td>
<td>0.07</td>
<td>0.14</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td>-0.01</td>
<td></td>
</tr>
<tr>
<td><strong>Average ΔRR/Δ(EPA+DHA)</strong></td>
<td>0.16</td>
<td>0.11</td>
<td>0.26</td>
<td>0.10</td>
<td>0.06</td>
<td>-0.13</td>
<td>-0.04</td>
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<tr>
<td>Number of follow-up years</td>
<td>16</td>
<td>30</td>
<td>20</td>
<td>9.3</td>
<td>20</td>
<td>11</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Number of Participants</td>
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<td>1,822</td>
<td>852</td>
<td>3,910</td>
<td>1,097</td>
<td>20,551</td>
<td>44,895</td>
<td></td>
</tr>
<tr>
<td>participant * years</td>
<td>1,355,008</td>
<td>54,660</td>
<td>17,040</td>
<td>36,363</td>
<td>21,940</td>
<td>226,061</td>
<td>269,370</td>
<td>1,980,442</td>
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<tr>
<td>Weighted average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.10</td>
<td></td>
</tr>
</tbody>
</table>
The conversion of fish intake (either serving or g) in g/d of EPA+DHA is based on the assumption of one serving fish/w=100 g/w of fish=0.14 g/d EPA+DHA.

The median intakes for the quintiles are 0.03%, 0.05%, 0.08%, 0.14% and 0.24% of total energy. The EPA+DHA intake in g/d was calculated by using 1 g=9 kcal conversion based on 2,200 kcal daily energy intake.

The four levels of fish intake in g/d are 0, 1-17, 18-34, >=35. The mean intake was used as well as the assumption in note 3 in order to calculate the EPA+DHA intake in g/d.

The five levels of fish intake in g/d are 0, 1-14, 15-29, 30-44, and >=45. The mean intake was used as well as the assumption in note 3 in order to calculate the EPA+DHA intake in g/d.

The level of EPA+DHA (mg/d) of each of the five fish servings is reported in this study.

This study measures, initially, the RR for five categories of fish servings (lowest:<1 s/mo, highest:>=5 s/w) and then calculates the RR for three categories of fish servings (<1 s/mo, 1-3 s/mo, and >=1 s/w) to make the results more significant. Although the results for fatal CHD are nonsignificant in both cases, the second one was used because it has lower P-value. For the conversion of fish serving to EPA+DHA g/d the assumption in note 1 was used.

EPA+DHA base g/d is the lowest intake of EPA+DHA provided to subjects as a comparison with the higher intakes, which are named “trials.”

The change in EPA+DHA intake (Δ(EPA+DHA)) is calculated by subtracting the lowest intake of EPA+DHA (EPA+DHA base) from the higher intakes (EPA+DHA trials).

The relative risk (RR) of CHD mortality as it has been reported by the studies. The relative risk is the ratio of the probability of CHD mortality occurring in the trials versus the base.

The change in RR (ΔRR) is calculated by subtracting the RR of the higher intakes (RR Trials) from the RR of the lowest intake (RR Base).

The average ΔRR/0.1Δ(EPA+DHA) was calculated by taking the average of [ΔRR (base-trial 1)/0.1Δ(EPA+DHA), ΔRR (base-trial 2)/0.1Δ(EPA+DHA), ΔRR (base-trial 3)/0.1Δ(EPA+DHA), ΔRR (base-trial 4)/0.1Δ(EPA+DHA)].

The total weighted average is the sum of the weighted averages found in each study. The weighted average for each study = (average ΔRR/0.1Δ(EPA+DHA)*(Years*Participants))/Sum of (Years*Participants). The weighted average for each study is:

- Hu et al., (2002): weighted average = 0.11
- Albert et al., (1998): weighted average = -0.01
- Daviglus et al., (1997a): weighted average = 0.003
- Ascherio et al., (1995): weighted average = -0.01
- Kromhout et al., (1985): weighted average = 0.002
- Mozaffarian, 2003: weighted average = 0.002
- Oomen et al., (2000): weighted average = 0.001.

Source: Author’s estimates (see text for detail).
4.6. The Economics of Information of Omega-3 Enriched Eggs in Canada

4.6.1. Estimation of the Potential Health Care Cost Savings

The third step of the benefit analysis is an estimation of the potential health care cost savings due to an increase in the consumption of omega-3 fatty acids via omega-3 enriched eggs. As research has shown, omega-3 fatty acids have a beneficial impact on cardiovascular diseases, whose treatment, as well as loss of income due to disability or death, are major costs in the Canadian health-care budget. In particular, it has been estimated that in 2006, cardiovascular disease accounted for $28,780 million.14 Direct costs comprise 48.3% ($13,903 million), while indirect costs account for 51.7% ($14,877 million) of the total cardiovascular disease cost. It is worth mentioning that CHD events are the key contributor to CVD events. It has been reported that 54% of CVD deaths are due to CHD deaths (Heart and Stroke foundation of Canada, 2002). Thus, it is reasonable to expect that CHD costs represent a large part of CVD costs. CHD costs have been estimated to be $11,251 million ($2006) annually.

The sensitivity analysis is based on three different scenarios (Base, High, and Low), which will provide a range of estimates regarding health care cost savings from CHD mortality. The estimation for the Base and Low Scenarios refers to the reduction in indirect costs from consuming omega-3 fatty acids and, in particular, in mortality costs. However, in the High Scenario, the total CHD costs (direct and indirect) are included, which implicitly assumes that total costs are reduced in the same proportion as mortality reduction. It has been estimated that mortality costs consist of 87.6% ($6,185.6 million) of CHD indirect costs, while the remaining 12.4% ($878 million) includes short- and long-term morbidity costs. The direct costs of CHD were estimated to be $4,187.6 million.15 The main assumption for estimating the health care savings is a linear 1:1 ratio between reduced risk of CHD incidence and mortality in the long run and health care cost savings. This means that if the risk in fatal CHD is reduced by 1% in the long run, CHD costs will be reduced by 1% (a similar assumption was made by Malla et al., 2007).

---

14 The methodology used to estimate the cardiovascular disease (CVD) costs and coronary heart disease (CHD) costs for 2006 is shown in subsection 2.6.2.
15 A detailed estimation of all the cost categories of direct and indirect costs of CHD is provided in Appendix A.
The process of estimating the change in CHD mortality by consuming omega-3 enriched eggs is demonstrated in Tables 4.3 and 4.4 due to the difference in the impact of ALA and EPA+DHA intake on fatal CHD found in the literature. Table 4.3 reports the estimated change in CHD mortality due to an increase in intake of ALA, while Table 4.4 depicts the estimated change in CHD mortality due to an EPA+DHA intake increase when consumers substitute a regular egg with an omega-3 enriched egg. After this separate estimation, results are combined in Table 4.5 to show the fundamental overall effect of omega-3 fatty acids contained in omega-3 enriched eggs on the risk of CHD death, and to provide the estimated potential total health care savings that can be achieved based on the current market share of omega-3 enriched eggs. A description and explanation of the results reported in the tables follows.

As Table 4.3 depicts, the change in CHD mortality due to an increase in the consumption of ALA contained in an omega-3 enriched egg is estimated by using three different scenarios, with the Base providing the most reasonable estimate. The Base Scenario shows that if consumers consume daily an omega-3 enriched egg instead of a conventional one, then they could increase the intake of ALA by 0.313 g, which in turn could reduce CHD mortality on average by 6.26%. This calculation uses the relationship specified in sub-section 4.3.2, whereby CHD mortality is reduced by 2% for every 0.1 g increase in ALA intake.

The High Scenario includes an optimistic estimate, which is based on the assumption that 0.1 g change in ALA intake can lead to a 3% reduction in fatal CHD. This means that the extra average ALA intake obtained from an omega-3 enriched egg, as compared with a regular egg, can reduce fatal CHD by 9.39% on average.

The Low Scenario assumes that the reduction in CHD mortality is 1% for every 0.1 g increase in ALA. This means that the extra ALA intake that a consumer receives by substituting regular eggs with omega-3 enriched eggs can reduce CHD mortality by 3.13%. For this scenario, a positive effect of omega-3 fatty acids on CHD mortality is still assumed based on the fact that the FDA has approved a qualified health claim stating that “supportive but not conclusive research shows that consumption of EPA and DHA omega-3 fatty acids may reduce the risk of coronary heart disease” (FDA, 2004).
Health Canada has also approved the enrichment of various products with omega-3 fatty acids.

Table 4.3. Estimated change in CHD mortality due to increase in ALA intake through the consumption of one omega-3 enriched egg per day.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Change in CHD mortality/0.1 g ALA (%)</td>
<td>-2</td>
<td>-3</td>
<td>-1</td>
</tr>
<tr>
<td>(b) Average ALA content in an omega-3 enriched egg (g)</td>
<td>0.34</td>
<td>0.34</td>
<td>0.34</td>
</tr>
<tr>
<td>(c) Average ALA content in a regular egg (g)</td>
<td>0.027</td>
<td>0.027</td>
<td>0.027</td>
</tr>
<tr>
<td>(d) Extra average ALA intake from an omega-3 enriched egg (g) (b-c)</td>
<td>0.313</td>
<td>0.313</td>
<td>0.313</td>
</tr>
<tr>
<td>(e) Total change in CHD mortality (%) (a*d)</td>
<td>-6.26</td>
<td>-9.39</td>
<td>-3.13</td>
</tr>
</tbody>
</table>

1 The percentage change in CHD mortality/0.1 g ALA is calculated in Table 4.1. For the Base Scenario, the total weighted average as calculated in Table 4.1 is used. For the High Scenario, an optimistic reduction in the risk of CHD equal to 50% higher than the Base Estimate is assumed, while for the Low Scenario the assumption is a modest reduction in the risk of CHD equal to 50% lower than the Base Estimate.


3 Author’s calculations based on Flax Council of Canada, 2003b. See Table 2.2.

4 Total change in CHD mortality (%) is calculated by multiplying the %ΔCHD mortality with the extra average ALA intake.

Source: Author’s estimates (see text for detail).

Another estimation process was necessary because omega-3 enriched eggs also contain higher amounts of EPA+DHA fatty acids than regular eggs. The estimated change in CHD deaths is demonstrated in Table 4.4, and is also based on the three different scenarios.

The Base Scenario assumes that 0.12 g EPA+DHA, which is the extra average amount found in an omega-3 enriched egg compared to a regular egg, can reduce the risk of CHD mortality by 12% on average. This uses the relationship specified in the sub-section 4.4.2, whereby CHD mortality is reduced by 10% for every 0.1 g increase in EPA+DHA intake.

The High Scenario illustrates that 0.1 g EPA+DHA could reduce the risk of CHD mortality by 15%. For this scenario, it is assumed that the reduction in the risk of
fatal CHD is 50% higher than the reduction in the Base Estimate. This means that the 0.12 g EPA+DHA contained in an omega-3 enriched egg could reduce CHD mortality by 18%.

The Low Scenario is based on an assumption that the risk of fatal CHD can be reduced by half of what the Base Estimate shows for every 0.1 g intake of EPA+DHA. This means that CHD mortality can be decreased by 6% when consumers substitute each conventional egg with an omega-3 enriched egg.

Table 4.4. Estimated change in CHD mortality due to increase in EPA+DHA intake through the consumption of one omega-3 enriched egg per day.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Change in CHD mortality/0.1 g EPA+DHA (%)</td>
<td>-10</td>
<td>-15</td>
<td>-5</td>
</tr>
<tr>
<td>(b) Average EPA+DHA content in an omega-3 enriched egg (g)</td>
<td>0.13</td>
<td>0.13</td>
<td>0.13</td>
</tr>
<tr>
<td>(c) Average EPA+DHA content in a regular egg (g)</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>(d) Extra average EPA+DHA intake from an omega-3 enriched egg (g) (b-c)</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
</tr>
<tr>
<td>(e) Total change in CHD mortality (%) (a*d)</td>
<td>-12</td>
<td>-18</td>
<td>-6</td>
</tr>
</tbody>
</table>

1 The percentage change in CHD mortality/0.1 g EPA+DHA is calculated in Table 4.2. For the Base Scenario, the total weighted average calculated in Table 4.2 is used. For the High Scenario, an optimistic reduction in the risk of CHD equal to 50% higher than the Base Estimate is assumed, while for the Low Scenario the assumption is a modest reduction in the risk of CHD equal to 50% lower than the Base Estimate.


3 Author's calculations based on Flax Council of Canada, 2003b. See Table 2.2.

4 Total change in CHD mortality (%) is calculated by multiplying the %ΔCHD mortality with the extra average EPA+DHA intake.

Source: Author’s estimates (see text for detail).

Combining the estimations regarding changes in CHD mortality shown in the above two tables, Table 4.5 summarizes the total change in fatal CHD due to an increase in omega-3 fatty acids found in an omega-3 enriched egg, and provides an estimation of the prospective annual CHD cost savings based on the three scenarios given the current consumption of an omega-3 enriched egg. As mentioned in Chapter Two (subsection 2.5.3), omega-3 enriched eggs currently capture 15% of the egg market in Canada.
Given that the average total number of eggs consumed per capita per day is 0.52 (half egg per capita per day), the reduction of fatal CHD caused by current daily omega-3 enriched egg consumption can be calculated. This is done by multiplying the market share of omega-3 enriched eggs by the average total number of eggs consumed per capita per day and then by the total change in CHD mortality for one omega-3 enriched egg consumed per day. The estimation of possible health savings is based on the assumption of a linear 1:1 ratio between the total change in CHD mortality due to the current omega-3 enriched egg consumption rate and the medical cost savings, which is then applied to all scenarios. A description of the three different estimates shown in the table, with an emphasis on the Base Estimate as being the most reasonable, follows.

The Base Estimate illustrates that the intake of omega-3 fatty acids via the current consumption of omega-3 enriched eggs can reduce the risk of CHD mortality by 1.42%. Based on the assumption of a linear 1:1 ratio between the CHD events and death and the related health costs savings, the estimation of the potential mortality cost savings could be $88.3 million per annum.

The High Estimate depicts a more optimistic reduction in the risk of fatal CHD. It calculates that the omega-3 enriched eggs currently consumed in Canada can lower the risk of CHD mortality by 2.14%. For this scenario, however, both the direct and indirect CHD costs are taken into consideration, not just the mortality costs, and it is assumed that an increase in omega-3 fatty acids intake also makes possible a decrease in the risk of CHD events, fatal and non-fatal. With this set of assumptions, the High Estimate suggests a potential reduction of $241 million in health care costs annually.

The Low Estimate shows that the existing market share of omega-3 enriched eggs can reduce CHD deaths by 0.71%. With an assumed 1:1 CHD risk to related health costs ratio, the potential estimated CHD mortality cost savings could be $44 million per year, demonstrating that major health savings can be achieved even with a less strong association between omega-3 fatty acids intake and the risk of fatal CHD.
### Table 4.5. Annual Canadian health care savings due to ALA, EPA, and DHA from 15% omega-3 enriched egg consumption.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Total change in CHD mortality for one omega-3 enriched egg consumed/d (%)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>-18.26</td>
<td>-27.39</td>
<td>-9.13</td>
</tr>
<tr>
<td>(b) Total disposition of eggs in 2006 (million of dozens)&lt;sup&gt;2&lt;/sup&gt;</td>
<td>517.8</td>
<td>517.8</td>
<td>517.8</td>
</tr>
<tr>
<td>(c) Average total number of eggs consumed/capita/day&lt;sup&gt;3&lt;/sup&gt;</td>
<td>0.52</td>
<td>0.52</td>
<td>0.52</td>
</tr>
<tr>
<td>(d) Market share of omega-3 enriched eggs (%)</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>(e) Total change in CHD mortality for the current consumption of omega-3 enriched eggs/capita/d (%) (a<em>c</em>d)&lt;sup&gt;4&lt;/sup&gt;</td>
<td>-1.42</td>
<td>-2.14</td>
<td>-0.71</td>
</tr>
<tr>
<td>(f) CHD mortality to cost ratio</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(g) Total annual CHD cost (millions CAN$)</td>
<td>-</td>
<td>11,251.6&lt;sup&gt;5&lt;/sup&gt;</td>
<td>-</td>
</tr>
<tr>
<td>(h) Total annual CHD mortality cost (millions CAN$)</td>
<td>6,185.6</td>
<td>-</td>
<td>6,185.6</td>
</tr>
<tr>
<td>(i) Total annual change in CHD cost (millions CAN$) (e<em>h) or (e</em>g)&lt;sup&gt;6&lt;/sup&gt;</td>
<td>-88.3</td>
<td>-241</td>
<td>-44</td>
</tr>
<tr>
<td>(j) CHD savings (millions CAN$)</td>
<td>88.3</td>
<td>241</td>
<td>44</td>
</tr>
</tbody>
</table>

<sup>1</sup>The total change in CHD mortality for one omega-3 enriched egg consumed/d is the sum of the total change in CHD mortality due to ALA intake increase (see Table 4.3) and the total change in CHD mortality due to EPA+DHA intake increase (see Table 4.4).

<sup>2</sup>Source: Statistics Canada, 2007c. The total disposition of eggs in Canada in 2006 was 588.4 million dozen eggs. From these, 517.8 million dozens were sold for consumption, while 59.3 million dozens were sold for hatching and 11.3 million dozens were leakers and rejects.

<sup>3</sup>The average total number of eggs/capita/d was calculated by multiplying the total disposition of eggs in 2006 (million dozens) by 12 to obtain the total number of eggs in millions, and then dividing by the population in 2006 (32,581,490 millions; Source: Cansim via E-Stat) and the number of annual days.

<sup>4</sup>The total percentage change in CHD mortality for the current consumption of an omega-3 enriched egg/capita/d is calculated by multiplying the total percentage change in CHD mortality for one omega-3 enriched egg consumed/d by the average total number of eggs/capita/d and the market share of omega-3 enriched eggs.

<sup>5</sup>In the High Scenario, the total CHD costs (direct and indirect) are included, which implicitly assumes that total costs are reduced in the same proportion as mortality reduction.

<sup>6</sup>The total annual change in CHD cost is calculated by multiplying the total annual CHD mortality cost with the percentage change in CHD mortality for the current consumption of omega-3 enriched eggs/capita/d. The High Scenario considers only the total annual indirect and direct CHD costs instead of the total annual CHD mortality costs.

Source: Author’s estimates (see text for detail).
4.6.2. *Estimation of the Costs*

In modeling the effects of an omega-3 PUFA labelling policy, both the health benefits and costs of omega-3 enriched eggs need be considered. Egg producers in Canada need to take into account some extra costs incurred when producing omega-3 enriched eggs. These include flax feed cost, fixed costs, labelling costs, and marketing costs.

The production of omega-3 enriched eggs requires the use of special feed, which is relatively more expensive for egg producers than that typically used. Flaxseed is the most common addition to hens’ feed ration to increase omega-3 PUFA content in the egg. Using flax as 10% of hens’ rations (on average), egg producers can obtain optimal results and market their eggs as omega-3 (Flax Council, 2007; Harman, 2007). Egg producers also bear fixed costs such as those associated with finding new suppliers and negotiating with distributors, as well as initial testing costs for labelling eligibility. A new package design and nutrient content claims are also part of the fixed costs. After producing omega-3 enriched eggs, additional costs arise. Labelling-related expenses are some of them. Specifically, product testing is required to ensure that eggs meet the omega-3 PUFA requirement, which enables egg producers to market them as omega-3 enriched eggs, and various safety criteria. Furthermore, marketing opportunities represent additional costs for producers who want to promote their eggs as omega-3.

The goal of producing and marketing omega-3 enriched eggs is to satisfy the needs of a niche market and thus increase revenues, which will more than compensate for the extra cost incurred. Omega-3 enriched eggs sell at a premium price compared to conventional eggs. Yet, many consumers in Canada are willing to pay a premium for eggs that are perceived as healthier. This observed premium (i.e., markup) is $1 per dozen on average, but this varies by brand and store. The author observed an approximate retail price in Saskatoon, Canada during a six-month period (January to July, 2007), but actual price differences are likely to differ by location and time.

Before analyzing the additional estimated costs of producing omega-3 enriched eggs, there is a need to distinguish between markup and cost. These two concepts cannot be used interchangeably as markup may or may not be equal to cost. This is because markup may include economic rents, which are not additional economic costs as they
are transferred to other players (e.g., retailers) in the industry (in terms of surplus), and so no resources are being used. In other words, markup is not all cost to the society, but also includes benefits that should not be overlooked.

Table 4.6 presents an estimation of the annual costs of producing, labelling, testing, and marketing the current number of omega-3 enriched eggs in Canada. In particular, estimates regarding the extra feed cost and the fixed, labelling, testing and marketing costs are provided, and appear on the last three rows of the table (including the total cost). The rest of the information in the table concerns calculations of these cost estimates.

Statistics Canada estimated that in 2006, total disposition of eggs, excluding hatching and leakers and rejects, was 517.8 million dozens (Statistics Canada, 2007c). From these, 77.67 million dozens are omega-3 enriched eggs, according to the current 15% market share. As was mentioned in the beginning of this subsection, the production of omega-3 enriched eggs includes the substitution of a portion of the typical feed with flax, which is responsible for the omega-3 PUFA enhancement in the eggs. The extra feed cost accounts for $72.74 per tonne according to Bert Harman, an egg producer in Saskatchewan, Canada, who indicated that the feed cost for regular egg production is $228.73 per tonne, while the feed cost for the omega-3 enriched egg production is $301.47 per tonne (Harman, 2007). Of course, this cost varies depending on the form of flax (whole or milled) and on additional nutrients (e.g., Vitamin E, lutein, selenium) that egg producers choose to add (Harman, 2007). Given that the feed conversion ratio per dozen eggs is on average 1.5 kg (or 0.0015 tonne), the extra feed cost per dozen can be calculated by multiplying the number of dozens of omega-3 enriched eggs with the feed ratio per dozen (Meyers Norris Penny LLP, 2006). The result shows that the additional feed cost per dozen omega-3 enriched eggs is $0.10911. Furthermore, it should be mentioned that this analysis is based on an average observed markup between omega-3 enriched eggs and conventional eggs. Thus, the proportion of the markup attributed to the fixed, labelling, and marketing costs have been arbitrarily estimated due to a lack of data on actual costs.

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16 It is assumed that the feed conversion ratio per dozen eggs has been remained the same for the period 2004-2006. It is also assumed that the feed conversion ratio is the same for flax-based diets and conventional diets. All that changes is the content of the feed.
As in the case of the health care cost savings analysis, three different scenarios are used to provide three different estimates—the Base Estimate, Low Estimate and High Estimate—which are shown in separate columns. The Base Estimate provides the most conservative result, and is based on the most plausible set of assumptions. The Low Estimate provides a low cost given a very modest set of assumptions, and is used to correspond the lower bound of the possible range. The High Estimate represents a high cost and provides an upper bound of the possible range.

The Base Estimate shows that the total extra feed cost of the current production of omega-3 enriched eggs could reach $8.47 million. Other additional costs, such as fixed, labelling, and marketing costs, are estimated under the assumption that 50% of the markup, which is 50% of the difference between 1 and 0.01091, are pure rents that occur to the producer, retailer, and marketer. Thus, by multiplying 50% of the markup with the total number of dozens omega-3 enriched eggs, the fixed, labelling, and marketing costs are estimated to be $34.6 million. Adding these costs together generates the total costs of shifting production of regular eggs to omega-3 enriched eggs to be $43.1 million per annum.

The Low Estimate assumes the same total extra feed cost as in the Base Estimate, which is $8.47 million, but uses different fixed, labelling, and marketing costs. It is assumed that 75% of the markup are rents that occur to the producer, retailer, and marketer, while the rest is the actual cost. This means that the fixed, labelling, and marketing costs would be $17.3 million. A total low cost is calculated to be $25.8 million per year.

The High Estimate provides an extreme situation where the total cost is very high. Once again, the total extra feed cost remains the same for all the three estimates, which is $8.47 million, while the rest additional costs differ. This estimation assumes that the entire additional markup is due to a cost increase and no additional rents are realized. As a result, the fixed, labelling, and marketing costs are measured to be $69.2 million. Aggregating the costs, it is found that the total cost would be $77.7 million annually.
Table 4.6. Annual Canadian costs of producing, labelling, testing and marketing 15% omega-3 enriched eggs.

<table>
<thead>
<tr>
<th>Description</th>
<th>Base</th>
<th>Low</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Total disposition of eggs in 2006 (million dozens)¹</td>
<td>517.8</td>
<td>517.8</td>
<td>517.8</td>
</tr>
<tr>
<td>(b) Market share of omega-3 enriched eggs (%)</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>(c) Total disposition of omega-3 enriched eggs in 2006 (million dozens)²</td>
<td>77.67</td>
<td>77.67</td>
<td>77.67</td>
</tr>
<tr>
<td>(d) Feed price difference between conventional and omega-3 enriched eggs ($ per tonne)³</td>
<td>72.74</td>
<td>72.74</td>
<td>72.74</td>
</tr>
<tr>
<td>(e) Feed conversion ratio per dozen (in tonne)⁴</td>
<td>0.0015</td>
<td>0.0015</td>
<td>0.0015</td>
</tr>
<tr>
<td>(f) Extra feed cost ($ per dozen) (d*e)⁵</td>
<td>0.1091</td>
<td>0.1091</td>
<td>0.1091</td>
</tr>
<tr>
<td>(g) Markup ($ per dozen)</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(h) Total extra feed cost (millions CAN$) (e*f)⁶</td>
<td>8.47</td>
<td>8.47</td>
<td>8.47</td>
</tr>
<tr>
<td>(i) Fixed, labelling, testing and marketing costs (millions CAN$)</td>
<td>34.6⁷</td>
<td>17.3⁸</td>
<td>69.2⁹</td>
</tr>
<tr>
<td>(j) Total Cost (millions CAN$) (h+i)¹⁰</td>
<td>43.1</td>
<td>25.8</td>
<td>77.7</td>
</tr>
</tbody>
</table>

¹ Source: Statistics Canada, 2007c. The total disposition of eggs in Canada in 2006 was 588.4 million dozen eggs. From these, 517.8 million dozens were sold for consumption, while 59.3 million dozens were sold for hatching and 11.3 million dozens were leakers and rejects.
² Currently, omega-3 enriched eggs make up 15% of the Canadian egg market. This means that 77.67 million dozen eggs include omega-3 fatty acids, which was calculated by multiplying the total disposition of eggs in 2006 with the market share of omega-3 enriched eggs.
³ Source: Harman, 2007. The feed price difference was calculated by subtracting the feed cost of regular eggs ($228.73/tonne) from that of omega-3 enriched eggs ($301.47/tonne).
⁴ Source: Meyers Norris Penny LLP, 2006. The feed conversion ratio per dozen eggs, which is the amount of feed given to hens in Canada to produce a dozen eggs, is 1.5 kg or 0.0015 tonnes per dozen.
⁵ The extra feed cost per dozen eggs was calculated by multiplying the feed price difference between conventional and omega-3 enriched eggs by the feed conversion ratio per dozen.
⁶ The total extra feed cost was calculated by multiplying the total disposition of omega-3 enriched eggs with the extra feed cost.
⁷ This assumes that 50% of the markup is pure rents that occur to the producer, retailer, and marketer.
⁸ This assumes that 75% of the markup is pure rents that occur to the producer, retailer, and marketer.
⁹ This assumes that the entire additional markup is due to cost increase and zero additional rents.
¹⁰ The total cost is the sum of the total extra feed cost and the fixed, labelling, testing, and marketing costs.

Source: Author’s estimates (see text for detail).

Although the estimation of the extra feed cost is calculated based on the price difference in feed between regular eggs and omega-3 enriched eggs in this analysis, it could also be calculated by considering the average flaxseed price, which has been forecasted to be an average of $275 per tonne for 2006-2007 (AAFC, 2007). Knowing
also that egg producers use flax as 10% (on average) of hens’ rations the extra feed cost can be estimated by adding 90% of the regular feed price ($228.73) and 10% of the flaxseed price ($275). This gives an estimate of the feed price necessary to produce omega-3 enriched eggs, which is $233.357 per tonne. In this case, the difference between the feed price for regular eggs and the feed price for omega-3 enriched eggs is $4.627 per tonne, or $0.007 per dozen (based on 1.5 kg feed ratio per dozen). The total extra feed cost for production of the current amount of omega-3 enriched eggs would be $0.539 million annually. Although this estimate may be an actual average cost, it is considered very low according to the feed prices that the Saskatchewan egg producer provided. This is probably because feed companies may charge more than the margin because they have extra handing and processing costs.

4.6.3. **Overall Impact and Benefit/Cost Ratio**

After estimating the benefits and costs of omega-3 enriched eggs, the overall impact to society can be realized and measured, which in turn will determine the efficacy of the labelling policy in Canada. Table 4.7 provides the net benefit of 15% omega-3 enriched eggs and the benefit cost (B/C) ratio based on the estimated health benefits (see Table 4.5) and costs (see Table 4.6) for each scenario.

For the Base Estimate, which is the most realistic, the net benefit is calculated to be $45 million per year. The B/C ratio of labelling omega-3 PUFA in omega-3 enriched eggs is 2.1:1. The High Estimate of the overall impact considers the High Estimate of CHD health benefits and the Low Estimate of costs in order to provide optimistic estimates. Thus, the net benefit is calculated to be $215 million annually, and the B/C ratio a very large 9.3:1. The Low Scenario provides very modest estimates. This means that the Low Estimate of CHD health benefits and the High Estimate of costs are taken into account. This results in a net cost of $33.7 million per annum and a B/C ratio of 0.57:1. The negative social gain of this scenario may be explained by the High Estimate of costs and the Low Estimate of benefits. However, this result may not correspond to reality because omega-3 enriched eggs have already been introduced in the market and make up 15% of the Canadian egg industry. Additionally, the Base Scenario, which
provides reasonable estimates, showed considerable health savings that are double the cost.

Such results indicate that the implementation of labelling regarding the omega-3 PUFA in the context of omega-3 enriched eggs is successful because the net benefit is positive, which means that the health care cost savings exceed the costs. As a result, the benefit cost analysis shows that the dissemination of nutrition information via labelling can be very advantageous for the Canadian economy as a whole.

<table>
<thead>
<tr>
<th>Table 4.7. Benefit/Cost ratio of 15% omega-3 enriched eggs.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CHD savings (millions CAN$)</strong></td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>CHD savings (millions CAN$)</td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
</tr>
<tr>
<td>Net Benefit (millions CAN$)</td>
</tr>
<tr>
<td>Benefit/Cost</td>
</tr>
</tbody>
</table>

Source: Author’s estimates (see text for detail).

4.7. Summary

This chapter provided an in depth benefit cost analysis of omega-3 enriched eggs in an attempt to investigate whether labelling the current number of dozens of omega-3 enriched eggs is beneficial to society. In order to do so, both the health benefits and costs were measured and expressed in monetary terms. First, the health benefits in terms of health care cost savings were calculated. More specifically, it is estimated that the potential annual savings in CHD mortality costs could reach $88.3 million annually, according to a reasonable estimate. On the other hand, the total cost of omega-3 enriched eggs, including production, labelling, and promotion costs, is estimated to be $43.1 million per year, based on the most realistic estimate. As a result, benefits outweigh costs, demonstrating that the provision of nutrition information to consumers through labelling has positive effects on Canadian society.

If omega-3 fatty acids indeed have a beneficial impact on health, then not only mortality but also other indirect (e.g., morbidity costs) and direct CHD costs could be negatively affected. Other direct and indirect costs of some other diseases (e.g., cancer,
inflammatory disorders, and brain and eye problems) could possibly be reduced, as well. Considering all the potential health benefits, the net benefit could be even higher. Therefore, taken together, these numbers are likely to be conservative estimates of the health benefits of omega-3 enriched eggs.

Considering that omega-3 enriched eggs are an example of functional foods that can be labeled, it can be concluded that labelling the healthy attributes of functional foods may considerably decrease the information asymmetry problem and improve Canadian economic welfare.
5.1. **Introduction**

The results of the case study examined in this thesis suggest that the health of Canadians and the economic welfare of the country are affected by the provision of labelling information regarding the health-enhancing features of functional foods. In particular, the health benefits of the current omega-3 egg consumption outweigh the costs of producing and labelling omega-3 enriched eggs under the Base Scenario (most realistic). Labelling is generally presented as an instrument that can provide valuable health information and modify consumption decisions. To maximize the economic and health benefits that labelling information can provide, an appropriate regulatory system regarding labelling should be developed.

This chapter begins with an extension of the analysis conducted in Chapter Four. It provides an estimation of the benefits and costs in the case where policymakers would regulate all the eggs to be enriched with omega-3 fatty acids. This policy would aim to improve the health of all egg consumers and move closer to the social optimum where information asymmetry and the moral hazard problem is reduced. The chapter closes with a discussion of policy implications.

5.2. **The Economics of a Mandated 100% Omega-3 Enriched Eggs in Canada**

As indicated in Chapter Four, the current consumption of omega-3 enriched eggs has substantial social benefits in terms of health care cost savings in Canada. The ability to label and make nutrient content claims in omega-3 enriched eggs has stimulated production of health-promoting eggs in order to meet the needs of the niche market and increase profits. However, while labelling can contribute significantly to an increase in the consumption of food products with healthy compounds like the omega-3 enriched eggs, the socially optimal amount cannot be achieved due to external factors such as
medical insurance. In such cases, one possible solution would be to regulate the use of omega-3 enriched eggs instead of the information. In other words, policymakers could require egg producers to produce all eggs with enhanced levels of omega-3 fatty acids so as to assist consumers to improve their health by increasing their intake of omega-3 fatty acids. Like any policy scheme, the social welfare impact should be determined before it is implemented. This section provides a benefit-cost analysis similar to that conducted in Chapter Four, which showed whether the mandatory development of omega-3 enriched eggs would provide a net social positive gain.

5.2.1. Estimation of the Potential Health Care Costs Savings

In order to calculate the possible health care costs savings, the same methodology explained in Chapter Four is followed here. The analysis includes three different scenarios that provide Low, Base, and High Estimates. The Base Estimate provides the most reasonable estimate, while the Low and High Estimates give a range of the potential health benefits. Considering the important impact that omega-3 fatty acids have on the risk of CHD mortality, the estimation refers to reduction in CHD mortality costs, apart from the High Scenario, which includes a reduction in all (direct and indirect) CHD costs. This High Estimate is based on the reasonable assumption that increasing omega-3 fatty acids levels in the diet could possibly improve heart health, thus reducing the risk of developing any CHD event, fatal or non-fatal. Additionally, a linear 1:1 ratio between the CHD and health-related costs savings is assumed, which means that a 1% reduction in CHD will reduce CHD costs by 1% (a similar assumption made by Malla et al., 2007). This assumption implies that CHD health costs are directly proportional to the incidence of CHD.

The measurement of likely health savings first requires an estimation of the total change in CHD mortality as a result of the increase in omega-3 fatty acids found in omega-3 enriched eggs. Tables 4.3 and 4.4 show the impact of ALA and EPA+DHA on the population’s health.

Table 4.3 shows that consumers who substitute regular eggs with omega-3 enriched eggs can increase their level of ALA, thereby reducing the risk of CHD mortality. In particular, the Base Scenario illustrates that the extra ALA intake gained
from the consumption of an omega-3 enriched egg can reduce CHD mortality by 6.26% on average. The High Scenario provides an optimistic estimate, where the reduction in fatal CHD will be even higher, on average 9.39%. Lastly, the Low Scenario assumes that the reduction is half the reduction of the Base Estimate, or 3.13%.

The consumption of omega-3 enriched eggs also enhances EPA+DHA levels of in the diet. Table 4.4 illustrates the possible change in CHD mortality due to increased EPA+DHA intake. The Base Scenario assumes that the extra average intake of EPA+DHA gained from an omega-3 enriched egg could decrease fatal CHD by 12% on average. The High Scenario shows that the reduction in CHD mortality could possibly be higher—18% on average—while the Low Estimate is the most modest estimate, showing a reduction of 6% in CHD deaths.

Table 5.1 presents the total effect that omega-3 fatty acids have on the risk of CHD mortality if consumers start consuming omega-3 enriched eggs instead of conventional eggs. Given this effect, the health benefits can then be quantified by measuring the potential health care cost savings. Contrary to the analysis conducted in Chapter Four, where the health benefits are based on current consumption of omega-3 enriched eggs, this estimation shows that the benefits could be higher if regulatory authorities intervene in the market by regulating that eggs be rich in omega-3 fatty acids.

According to the Base Estimate, the total reduction in the risk of CHD mortality for 100% consumption of omega-3 enriched eggs per capita per day instead of regular eggs would be 9.49%. Given the assumption of a linear 1:1 ratio between CHD events and death and the related health care cost savings, the estimation of potential mortality cost savings could be $587 million annually.

The High Scenario assumes that substituting omega-3 enriched eggs would lead to a greater reduction in the risk of CHD mortality, which, on average, is 14.24%. This scenario considers the total annual cost of CHD given that the increased intake of omega-3 fatty acids contained in omega-3 enriched eggs would improve health and thus reduce the risk of CHD incidence and mortality. Assuming a linear CHD cost ratio, the annual savings in the costs of illness are measured to be $1,602 million.

The Low Scenario shows that the reduction in CHD mortality would be a modest 4.74%. Still, substantial savings in societal health costs can be achieved. Based on an
assumed linear CHD cost ratio, the health care and related cost of illness savings is estimated to be $293 million per annum.

Table 5.1. Annual Canadian health care savings due to ALA, EPA, and DHA from 100% omega-3 enriched egg consumption.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Total change in CHD mortality for one omega-3 enriched egg consumed/d (%)&lt;sup&gt;1&lt;/sup&gt;</td>
<td>-18.26</td>
<td>-27.39</td>
<td>-9.13</td>
</tr>
<tr>
<td>(b) Total disposition of eggs in 2006 (million of dozens)&lt;sup&gt;2&lt;/sup&gt;</td>
<td>517.8</td>
<td>517.8</td>
<td>517.8</td>
</tr>
<tr>
<td>(c) Average total number of eggs consumed/capita/day&lt;sup&gt;3&lt;/sup&gt;</td>
<td>0.52</td>
<td>0.52</td>
<td>0.52</td>
</tr>
<tr>
<td>(d) Total change in CHD mortality for the 100% consumption of omega-3 enriched eggs/capita/d (%) (a*c)&lt;sup&gt;4&lt;/sup&gt;</td>
<td>-9.49</td>
<td>-14.24</td>
<td>-4.74</td>
</tr>
<tr>
<td>(e) CHD mortality to cost ratio</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(f) Total annual CHD cost (millions CAN$)</td>
<td>-</td>
<td>11,251.6&lt;sup&gt;5&lt;/sup&gt;</td>
<td>-</td>
</tr>
<tr>
<td>(g) Total annual CHD mortality cost (millions CAN$)</td>
<td>6,185.6</td>
<td>-</td>
<td>6,185.6</td>
</tr>
<tr>
<td>(h) Total annual change in CHD cost (millions CAN$) (d<em>g) or (d</em>f)&lt;sup&gt;6&lt;/sup&gt;</td>
<td>-587</td>
<td>-1,602</td>
<td>-293</td>
</tr>
<tr>
<td>(i) CHD savings (millions CAN$)</td>
<td>587</td>
<td>1,602</td>
<td>293</td>
</tr>
</tbody>
</table>

<sup>1</sup>The total change in CHD mortality for one omega-3 enriched egg consumed/d is the sum of the total change in CHD mortality due to ALA intake increase (see Table 4.3) and the total change in CHD mortality due to EPA+DHA intake increase (see Table 4.4).

<sup>2</sup>Source: Statistics Canada, 2007c. The total disposition of eggs in Canada in 2006 was 588.4 million dozen eggs. From these, 517.8 million dozens were sold for consumption, while 59.3 million dozens were sold for hatching and 11.3 million dozens were leakers and rejects.

<sup>3</sup>The average total number of eggs/capita/d was calculated by multiplying the total disposition of eggs in 2006 (million dozens) by 12 to obtain the total number of eggs in millions, and then divided by the population in 2006 (32,581,490 millions, Source: Cansim via E-Stat) and the number of annual days.

<sup>4</sup>The total percentage change in CHD mortality for 100% consumption of omega-3 enriched eggs/capita/d is calculated by multiplying the total percentage change in CHD mortality for one omega-3 enriched egg consumed/d by the average total number of eggs/capita/d.

<sup>5</sup>In the High Scenario, the total CHD costs (direct and indirect) are included, which implicitly assumes that total costs are reduced in the same proportion as mortality reduction.

<sup>6</sup>The total annual change in CHD cost is calculated by multiplying the total annual CHD mortality cost with the percentage change in CHD mortality for the current consumption of omega-3 enriched eggs/capita/d. In the High Scenario, the total annual indirect and direct CHD costs are considered instead of the total annual CHD mortality costs.

Source: Author’s estimates (see text for detail).
5.2.2. *Estimation of the Costs*

Before implementation of the policy regarding the mandated use of omega-3 enriched eggs, the estimated related costs should be taken into consideration. Currently, egg producers who choose to produce omega-3 enriched eggs experience extra production, fixed, labelling, and marketing costs. As indicated in Chapter Four (subsection 4.6.2), the production of omega-3 enriched eggs requires the substitution of a portion (10%) of regular feed with flaxseed, which is relatively more expensive. This means that in the case where all the produced eggs are regulated to contain increased levels of omega-3 fatty acids, production costs would increase as more flaxseed is required. However, the same would not hold for the fixed, labelling, and marketing costs. These costs would be almost eliminated if such a policy were applied. This is because the eggs would be required to be enriched with these beneficial fatty acids, meaning that egg producers would not bear labelling or marketing costs for their egg products and any premium would disappear. For the benefit cost calculation, it is assumed that this policy, which would modestly increase the price of the 85% of the (non-omega 3) eggs and decrease the price of 15% of the (omega-3) eggs by nearly $1 per dozen, would have no net effect on the quantity demand. This is a reasonable assumption to make since eggs are a supply managed commodity in Canada. Furthermore, given a long-term elastic supply of flax, it is assumed that any increase in quantity demanded of flaxseed would not impact the price of flaxseed.

The annual production, labelling, and marketing costs incurred by a mandatory development of omega-3 enriched eggs are illustrated in Table 5.2. The table includes some additional information necessary for the estimation of the costs.\(^{17}\) Statistics Canada reported that the total disposition of eggs in 2006 was 517.8 million dozen (Statistics Canada, 2007c). This amount does not include hatching and leakers and rejects. Also, according to Bert Harman, an egg producer in Saskatchewan, Canada, the extra feed price difference between conventional eggs and omega-3 enriched eggs could be $72.74 per tonne (Harman, 2007). Given that the feed conversion ratio per dozen eggs is, on average, 1.5 kg (or 0.0015 tonne), the additional feed cost per dozen omega-3

\(^{17}\) An explicit explanation of this additional information can be found in Chapter Four (subsection 4.6.2).
enriched eggs is $0.10911 (Meyers Norris Penny LLP, 2006). Consequently, the total extra feed cost that egg producers bear is estimated to be $56.5 million annually, and is assumed to be the same for the Base and High Scenarios.

The extra feed cost for the Low Scenario (i.e., most conservative) is calculated by using the price of flaxseed, which is estimated to be $275 per tonne, on average, for 2006-2007 (AAFC, 2007). The price of feed for the production of conventional eggs was $228.73 (Harman, 2007). Knowing that 10% of flaxseed is used to satisfy the criteria for the omega-3 enriched eggs and the remainder is conventional feed, the estimate of the feed price necessary to produce omega-3 enriched eggs is $233.357 per tonne. Therefore, the difference between the feed price for regular eggs and the feed price for omega-3 enriched eggs is $4.627 per tonne, or $0.007 per dozen (based on 1.5 kg feed ratio per dozen). Therefore, the total feed cost to produce 517.8 million dozen eggs is estimated to be $3.6 million per year, an amount much smaller than the $56.5 million figure found for the other two scenarios.

Furthermore, if policymakers require a mandatory omega-3 fatty acids enhancement of all eggs, the fixed, labelling, and marketing costs are virtually eradicated. Some minimal testing costs may continue to ensure that egg producers meet the nutrition standards, but are not considered in the estimation. The Canadian Egg Marketing Agency may also choose to advertise the additional health benefits from consuming omega-3 enriched eggs in order to inform and educate consumers. However, the additional benefits or costs associated with this action are not taken into account in the estimation. Therefore, the total cost that implementation of such a policy would demand of the industry is equal to the annual total extra feed cost—$56.5 million for the Base and High Scenarios, and $3.6 million for the Low Scenario.

---

18 It is assumed that the feed conversion ratio per dozen eggs has remained the same for the period 2004-2006. It is also assumed that the feed conversion ratio is the same for flax-based diets and conventional diets. The only change is the content of the feed.
Table 5.2. Annual Canadian costs of producing, labelling and marketing 100% omega-3 enriched eggs.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>Low</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Total disposition of eggs in 2006 (million dozens)¹</td>
<td>517.8</td>
<td>517.8</td>
<td>517.8</td>
</tr>
<tr>
<td>(b) Feed price difference between conventional and omega-3 enriched eggs ($ per tonne)²</td>
<td>72.74</td>
<td>-</td>
<td>72.74</td>
</tr>
<tr>
<td>(c) Feed price difference between conventional and omega-3 enriched eggs based on flaxseed price ($ per tonne)³</td>
<td>-</td>
<td>4.627</td>
<td>-</td>
</tr>
<tr>
<td>(d) Feed conversion ratio per dozen (in tonne)⁴</td>
<td>0.0015</td>
<td>0.0015</td>
<td>0.0015</td>
</tr>
<tr>
<td>(e) Extra feed cost ($ per dozen) (b<em>d) or (c</em>d)</td>
<td>0.1095</td>
<td>0.007</td>
<td>0.1095</td>
</tr>
<tr>
<td>(f) Total extra feed cost (millions CAN$)⁷</td>
<td>56.5</td>
<td>3.6</td>
<td>56.5</td>
</tr>
<tr>
<td>(g) Fixed, labelling and marketing costs⁸</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>(h) Total Cost (millions CAN$)⁹</td>
<td><strong>56.5</strong></td>
<td><strong>3.6</strong></td>
<td><strong>56.5</strong></td>
</tr>
</tbody>
</table>

¹ Source: Statistics Canada, 2007c. The total disposition of eggs in Canada in 2006 was 588.4 million dozen eggs. From these, 517.8 million dozens were sold for consumption, while 59.3 million dozens were sold for hatching and 11.3 million dozens were leakers and rejects.

² Source: Harman, 2007. The feed price difference between conventional and omega-3 enriched eggs was calculated by subtracting the feed cost of conventional eggs ($228.73/tonne) from the feed cost of omega-3 enriched eggs ($301.47/tonne).

³ Feed price difference between conventional and omega-3 enriched eggs based on flaxseed price = \(((10\%*275+90\%*228.73)-228.73)*0.0015\) tonnes = 4.627, where $275/tonne is the price of flax, $228.73/tonne is the average feed cost of conventional eggs, 10% is the portion of flax required for the omega-3 enriched eggs, and 0.0015 tonnes (1.5 kg) is the feed conversion ratio per dozen eggs.

⁴ Source: Meyers Norris Penny LLP, 2006. The feed conversion ratio per dozen eggs, which is the amount of feed given to hens in Canada to produce a dozen eggs, is 1.5 kg or 0.0015 tonnes per dozen.

⁵ The extra feed cost per dozen eggs for the Base and High Scenarios was calculated as follows: Extra feed cost per dozen eggs = (Feed price difference between conventional and omega-3 enriched eggs*feed conversion ratio per dozen).

By holding the quantity constant, it is assumed that a 90-cent reduction per dozen in omega-3 egg prices for 15% of the consumers more than offsets the reduction in demand due to a 10-cent increase per dozen in omega-3 egg prices to 85% of the consumers.

⁶ The extra feed cost per dozen eggs for the Low Scenario was calculated as follows: Extra feed cost per dozen eggs = (Feed price difference between conventional and omega-3 enriched eggs*feed conversion ratio per dozen).

By holding the quantity constant, it is assumed that a 99.3-cent reduction per dozen in omega-3 egg prices for 15% of the consumers more than offsets the reduction in demand due to a 0.7-cent increase per dozen in omega-3 egg prices to 85% of the consumers.

⁷ The total extra feed cost was calculated by multiplying the total disposition of omega-3 enriched eggs with the extra feed cost.

⁸ Minimum testing costs to assure consumers that producers meet the nutrition standards are not included in this estimation.

⁹ The total extra feed cost was calculated by multiplying the total disposition of eggs with the extra feed cost.

Source: Author’s estimates (see text for detail).
5.2.3. Overall Impact and Benefit/Cost Ratio

In order to decide whether the policy of mandatory use of omega-3 enriched eggs is socially preferable, it is necessary to compare the benefits and costs that will arise. Table 5.3 depicts the net benefit and benefit/cost ratio for each scenario, which will help determine if such policy is recommended.

The Base Scenario (most realistic) shows that the net benefit is estimated to be $530 million per annum. Given that the social benefits in terms of health care cost savings are high if this policy is realized, the B/C ratio is equal to 10.4. The High Scenario provides optimistic estimates by considering the High Estimate of CHD cost savings and the Low Estimate of costs. The results show that the annual net benefit is $1,598 million, while the B/C ratio is very high, 445:1. Finally, for the Low Scenario, the Low Estimate of the CHD health benefits and the High Estimate of the costs are taken into account. Hence, the net benefit is $236 million per year, and the B/C ratio is 5.2:1, illustrating the beneficial social welfare effects of such a policy, even under very modest assumptions.

The results of the benefit cost analysis conducted in this section present the significance of implementing a policy that would require production of all eggs to have high omega-3 fatty acids content in Canada. This policy might be an effective means of correcting not only the market failure caused from asymmetrically distributed information, but also reducing the negative externality caused by medical insurance and improving society’s welfare by moving closer to the social optimum. According to Table 5.3, the most realistic estimate demonstrates a particularly high B/C ratio, 19.9:1. The High and Low Estimate forming the upper and lower bound of the range is also of particular importance. Therefore, in an attempt to improve Canadians’ health and thus decrease rising health care costs, such a regulatory policy change would be very beneficial for Canadians’ health and the nation’s economy.
Table 5.3. **Benefit/Cost ratio of 100% omega-3 enriched eggs versus zero %**.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD savings (millions CAN$)</td>
<td>587</td>
<td>1,602</td>
<td>293</td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
<td>56.5</td>
<td>3.6</td>
<td>56.5</td>
</tr>
<tr>
<td>Net Benefit (millions CAN$)</td>
<td>530</td>
<td>1,598</td>
<td>236</td>
</tr>
<tr>
<td>Benefit/Cost (millions CAN$)</td>
<td>10.4</td>
<td>445</td>
<td>5.2</td>
</tr>
</tbody>
</table>

Source: Author’s estimates (see text for detail).

Comparing the impacts of the mandated use to the current market reveals some interesting relationships (see Table 5.4). The health care cost savings can substantially increase, but the total cost would not increase as much as benefits. This is because the labelling and marketing costs would be almost eliminated.\(^\text{19}\) In particular, the Base Scenario shows that CHD savings will increase by $499 million annually. For the High and Low Scenario, the results indicate an annual increase of $1,361 million and $249 million in health benefits, respectively. As regards to the total cost, the increase is incremental when compared to benefits. According to the Base Scenario, the total cost is estimated to increase by $13.4 million per year due to increased production costs. The High and Low Scenarios show a possible annual cost reduction of $22.2 million and $21.2 million, respectively.

As illustrated in Table 5.4, 100% use of omega-3 enriched eggs would increase the benefits to a larger extent than the costs when compared to the current 15% rate. Therefore, the total net effect would be positive. The Base Scenario indicates that the annual net benefit would increase by $485 million. For the High Scenario, the net benefit would increase by $1,383 million annually, while for the Low Scenario the net benefit would increase by $270 million per annum. These results provide further insight into the beneficial effects that mandated use of omega-3 enriched eggs would provide compared to the current labelling policy. The health improvement is noteworthy and would result in major health care cost savings. Additionally, while the production cost of

\(^{19}\) Some minimal testing costs, which would continue to occur even after the mandatory development of the omega-3 eggs to ensure that egg producers meet the nutritional standards, are not considered in the estimation.
omega-3 enriched eggs would increase, considerable labelling and marketing savings would incur under the 100% policy scenario, which explains why total cost increases are smaller than the benefits.

According to the results of the estimation, the current labelling of omega-3 enriched eggs has led to a notable B/C ratio of 2.1:1. The net benefit could be even higher if development of omega-3 enriched eggs were mandatory. Therefore, the hypothesis that the labelling of omega-3 enriched eggs has not led to benefits for Canadians is rejected, showing the potential importance of labelling and claims, and the magnitude of potential health benefits that would be forgone under a restrictive regulatory labelling regime.

Table 5.4. Comparison of 15% versus 100% omega-3 enriched eggs.

<table>
<thead>
<tr>
<th></th>
<th>Base</th>
<th>High</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>15% omega-3 enriched eggs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD savings (millions CAN$)</td>
<td>88.3</td>
<td>241</td>
<td>44</td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
<td>43.1</td>
<td>25.8</td>
<td>77.7</td>
</tr>
<tr>
<td>Net Benefit (millions CAN$)</td>
<td>45</td>
<td>215</td>
<td>-33.7</td>
</tr>
<tr>
<td>Benefit/Cost</td>
<td>2.1</td>
<td>9.3</td>
<td>0.57</td>
</tr>
<tr>
<td><strong>100% omega-3 enriched eggs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD savings (millions CAN$)</td>
<td>587</td>
<td>1,602</td>
<td>293</td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
<td>56.5</td>
<td>3.6</td>
<td>56.5</td>
</tr>
<tr>
<td>Net Benefit (millions CAN$)</td>
<td>530</td>
<td>1,598</td>
<td>236</td>
</tr>
<tr>
<td>Benefit/Cost</td>
<td>10.4</td>
<td>445</td>
<td>5.2</td>
</tr>
<tr>
<td><strong>Change from 15% to 100%</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHD savings (millions CAN$)</td>
<td>499</td>
<td>1,361</td>
<td>249</td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
<td>13.4</td>
<td>-22.2</td>
<td>-21.2</td>
</tr>
<tr>
<td>Net Benefit (millions CAN$)</td>
<td>485</td>
<td>1,383</td>
<td>270</td>
</tr>
</tbody>
</table>

1 The estimation of CHD savings, total cost, and net benefit and Benefit/Cost ratio for the current market share of omega-3 enriched eggs can be found on the Tables 4.5, 4.6, and 4.7, respectively.

2 The estimation of CHD savings, total cost, and net benefit and Benefit/Cost ratio for the mandatory development of omega-3 enriched eggs can be found on the Tables 5.1, 5.2, and 5.3, respectively.

Source: Author’s estimates (see text for detail).
5.3. Policy Implications

The federal government, through labelling legislation, controls provision of information on food labels. This means that legislative rules regarding food labelling and claims can considerably influence the production and consumption of functional food products, correct the information asymmetry problem, and consequently affect the Canadian economy as a whole. The use of nutrient content claims and health claims can provide producers with an incentive to innovate and expand the functional food sector while simultaneously affecting consumers’ food choices. The case study presented in this research indicated how important labelling is to the health-enhancing properties of functional foods. If omega-3 fatty acids were not regulated as essential nutrients, and thus nutrient content claims could not be made, presumably no omega-3 enriched eggs would have existed in the market and significant health care cost savings would have been forgone.

However, the current regulatory regime in Canada limits nutrient content and health claims, thus restricting the range of functional foods entering the market. In addition to this, uncertainty within government policy and a lack of co-operation among governmental agencies can be a concern to firms developing a new functional food. Therefore, a complicated and restrictive regulatory framework may discourage innovation in the functional food industry, inhibit economic efficiency, and lead to major welfare losses.

A more extensive labelling scheme would be appropriate for boosting the functional food market. Flexible and well-organized legislation can encourage competition, improve research activities, increase investment in the development of new products, and promote international trade. This presupposes that Canada’s regulations will adapt quickly to new scientific advances and nutrition findings and allow more claims related to the beneficial compounds of functional foods. Additionally, closer relations and increased coordination are necessary between governments, industry, non-governmental organizations, and citizens. All partners have to cooperate to address regulatory issues and improve the system. Thus, major innovative discoveries can occur and economic efficiency can be improved.
Furthermore, the information asymmetry problem apparent in the functional food market is related to the credibility of labelling (Zou and Hobbs, 2006). Consumers base their purchasing decisions on information attached to the labels. If labelling information is conflicting and untrustworthy, consumers will not be interested in consuming functional foods, and so the economic incentive to produce health-enhancing food products will not be present. So, clear, credible, and comprehensive information that accurately reflects the scientific evidence is necessary to motivate consumers to modify their eating patterns. Food labelling, in combination with consumer education, will help consumers make healthier and more informative food choices.

In designing labelling regulation, the research community plays a central role. Labelling and research are interlinked concepts. Without a flexible labelling system, no research can occur, and without consistent and effective research, labelling cannot take place. So, constant nutrition research is of utmost importance in order for more healthy food products to be labeled. Currently in Canada, a small amount of health care budget is being devoted to nutrition and health research, which undermines economic efficiency. However, this thesis has illustrated that when the health effects of specific nutrients are revealed through research, significant social benefits in terms of health care costs can be attained—assuming that labelling regulations will keep up with the evolution of science and research. Hence, it can be concluded that the research community in collaboration with the functional food industry can improve Canadians’ health and foster economic welfare.

Lastly, a regulatory policy that requires mandatory development of omega-3 enriched eggs may also have long-run implications on incentives for future innovation. Mandating all eggs to be rich in omega-3 fatty acids might regulate away the competitive advantage of the original omega-3 egg innovators, reducing incentives to research. This, of course, depends on the existing rules around the intellectual property rights and how first-movers (omega-3 egg innovators) would be compensated were such a policy implemented.
5.4. Conclusion

This chapter discussed the need for policy change that would yield increased economic benefits. In addition to the health benefits that consumption of omega-3 enriched eggs provides, a possible legislative policy was proposed in this chapter. This change would require mandatory omega-3 enrichment of all eggs in Canada. The results indicated that this policy would generate even greater health care cost savings while simultaneously lowering the total cost of omega-3 enriched eggs, resulting in a particularly high benefit cost ratio. The importance of information through food labelling for consumers’ health and the functional food market must be further understood. This entrusts government with the responsibility to make changes to the regulatory system so as to increase the benefits of labelling.
CHAPTER 6: SUMMARY AND CONCLUSIONS

6.1. Summary

Considering the growing consumer interest in the link between diet and health, innovations in the agri-food sector have led to the development of functional foods in Canada. These food products that confer health benefits can improve the population’s health, thereby resulting in large social benefits through reduced health care costs. However, the market for functional foods is characterized by information asymmetry for consumers with respect to the presence, level and efficacy of the functional attributes (Zou and Hobbs, 2006). The health components of specific foods and the future health outcomes attached to them are credence attributes, being that in the absence of information, consumers cannot generally observe such attributes. Given the information asymmetry inherent in functional foods, labelling can be used as a vehicle to transform credence attributes into experience and search attributes, differentiate products, and facilitate consumers’ purchasing decisions. This thesis has attempted to illustrate how the labelling of healthy nutrients could be one efficient and practical way to solve the market failure caused by asymmetric information, and thereby enhance economic efficiency. A case study provides further insight into the research problem.

In order to obtain a general picture of the impact that information asymmetry causes on social welfare, a graphical illustration is provided in Chapter Three. Providing consumers with clear and comprehensive information can help them make healthy and informed food choices and decrease the health care costs that burden both the Canadian health care system and the economy as a whole. In general, informed consumers can increase the demand for healthier food products (or, likewise, decrease the demand for unhealthy foods). Therefore, a potential growth in the consumption of functional foods can increase both private and social welfare and decrease the dead weight loss that is created in the absence of nutrition information. The socially optimal amount cannot be
achieved even if consumers have perfect information because health and disability insurances cause a moral hazard problem that deter the consumption of healthier foods. Nevertheless, labelling information can substantially correct the market failure and improve social welfare.

A graphical comparison of voluntary and mandatory labelling is also provided to demonstrate the beneficial effects of providing information on the healthy nutrients of functional foods, and suggests that mandatory labelling is a more effective policy to address the asymmetry of information. Currently, the regulatory labelling regime in Canada is mandatory and requires all pre-packaged products with certain kinds of ingredients to be labeled clearly in order to assist consumers in making comparisons between products and choosing foods according to their preferences.

Although there is a range of functional foods available in Canada—such as probiotic yogurts, cholesterol-lowering spreads, vitamin E and C enhanced soft drinks, fiber enhanced cereals, and omega-3 enriched milk and eggs—the rate of development of functional foods remains slow due to stringent regulations. The allowance of certain nutrient content claims and the restricted to five health claims have been a major barrier to the introduction and commercialization of new functional foods. Firms may be hesitant to invest funds in the development of new value-added food products that could meet resistance at the regulatory approval phase. This means that complicated and ambiguous regulations can impede functional food innovations and deter investment in research and development. This, in turn, affects the economic efficiency in Canada due to diet-related health care costs.

The primary conclusion of this research can be drawn from the examination of the case study, which has revealed the net economic benefit of producing a particular functional food and labelling its value-added attribute. Omega-3 enriched eggs were used as an example of functional foods and a benefit cost analysis was conducted to evaluate the efficacy of the current labelling policy. Based on a range of assumptions and using three different scenarios to cover a range of estimates, both health care cost savings and the costs incurred due to labelling and production are estimated for this functional food.
Specifically, in Chapter Four, the example of omega-3 enriched eggs is used to explore the potential magnitude of health care savings, as well as the costs related to labelling. The egg industry and scientific evidence showing the health effects of omega-3 fatty acids have contributed in making the labelling of omega-3 enriched eggs of great value. Currently, omega-3 fatty acids are deemed essential nutrients by Health Canada and firms are allowed to make such nutrient content claims. This has led to an important functional food innovation in the egg product sector. Among their various health effects, adequate intake of omega-3 fatty acids in the diet can substantially reduce the risk of CHD, especially CHD mortality. Therefore, omega-3 enriched eggs are considered an easy way to increase the level of omega-3 fatty acids and simultaneously save billions of dollars in health care costs. Furthermore, labelling, testing, and marketing, as well as producing these eggs, creates extra costs for the industry, which have also been estimated based on the observed markup of the omega-3 enriched eggs. By comparing the estimated benefits and costs, it is indicated that the current consumption of omega-3 enriched eggs provides a significant net economic gain.

The benefit cost analysis of omega-3 enriched eggs was extended in Chapter Five, where the policy implications of the research were discussed. Realizing the current fundamental overall impact of omega-3 enriched eggs on society’s health and the national economy, it is interesting to test the effect of implementing a policy that would mandate the use of omega-3 enriched eggs. This policy can potentially be used to correct market failures related not only to asymmetric information but also to the negative externalities that medical insurances create. The results indicated that the health benefits would be greatly increased while costs would increase only slightly because almost all the labelling-related costs would be eliminated. Therefore, the net economic gain becomes even stronger if all eggs are regulated to be rich in these beneficial fatty acids.

The case study shed light on the research problem and indicated the significant health improvements and overall positive economic effects that information on health compounds through labelling can provide. By examining this functional food, it also helps realize the potential gains that are foregone due to a lack of labelling of other

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20 Some minimal testing costs, as well as additional benefits or costs associated with consumers’ education regarding the healthy characteristics of omega-3 enriched eggs, are not considered in the estimation.
beneficial nutrients in Canada (e.g., beta glucan, lutein, selenium). Consequently, the introduction of a regulatory process for approval and labelling of new functional foods could rectify the market failure of asymmetrically distributed information, stimulate innovation, and, therefore, result in increased economic welfare.

6.2. Limitations and Recommendations for Further Research

The primary objective of the thesis was to indicate whether labelling functional foods could decrease the information asymmetry problem and lead to a net benefit for society. This was examined through a case study of omega-3 enriched eggs. Examining more food products with health properties may provide additional insights into the effects of labelling information. There is a spectrum of functional foods available in the Canadian market. Research into more functional food product areas could enhance the value of the results, thereby motivating policymakers to allow additional claims for specific nutrients.

Future research focusing on specific functional foods and their properties would also be very beneficial to the Canadian agri-food industry in terms of realizing the importance of supplying value-added functional foods and identifying possible strategies for the development of new healthy products. Examining the innovation process, existing private incentives, and current patterns of Canadian private and public investment in functional foods R&D would be interesting areas of study.

One limitation of this study is the use of hypothetical values in the benefit cost analysis, specifically for the current fixed, labelling, testing, and marketing costs of omega-3 enriched eggs. The study was limited to hypothetical values due to difficulty in obtaining and revealing the magnitude of these cost categories. To remedy this, it is recommended that more in depth research be performed to calculate realistic values for the cost variables.

Although this thesis briefly discussed the stiffness of the regulatory labelling system, additional research into industry perceptions could help identify potential and specific problems with the regulations. A survey of functional food firms could provide explicit information regarding costs of registration, the approval rate of functional foods,
product availability, and other regulatory barriers, which, in turn, would designate the degree at which the regulation is being restrictive.

Further investigation is also recommended of the effects of the regulatory environment on incentives to innovate, including the use of intellectual property rights and the generation and sharing of knowledge regarding function, efficacy, and safety with respect to healthy foods. The identification of potential bottlenecks and hold-ups could also help explain the constraints to R&D and commercialization of functional food innovations in both domestic and international markets. A comparison of other regulatory systems (e.g., U.S., Europe, Japan) could help identify and understand the differences, and thus emphasize the limitations of the Canadian labelling legislation.

Although the problem of moral hazard was briefly discussed in the thesis, and possible solutions were suggested, it was not explicitly modeled. The negative externality caused from private and public health insurances prevent the achievement of the social optimal, even when full information is provided. This study showed that by requiring all the eggs to contain omega-3 fatty acids, the moral hazard problem can be reduced. A more general solution was proposed by Malla et al. (2007), who recommended a pigovian tax on the production/consumption of unhealthy foods or subsidies to encourage consumption of healthier foods. It is clear that this problem area requires further study to determine the optimal legislative policy needed to correct the negative externality.

Finally, with regards to the theoretical model examined in section 3.4, the magnitudes of the relative shifts in the demand curves were hypothetical and based on the labels and allowable claims. Therefore, it is recommended that more in-depth research be performed into consumer demand effects by using stated and/or revealed consumer preference methodologies. A study of consumer attitudes, choices, and motivations regarding food labelling could provide insight into consumers’ perceptions towards labelling regulations, and could give better indications of the relative size of consumer demand shifts.
6.3. Conclusions

The purpose of this study was to examine why labelling information is particularly important in the functional food industry and how it can affect the economic welfare of Canada. The information asymmetry problem is a key characteristic in the functional food industry and causes market failures. Labelling is necessary to correct this problem. Currently, functional foods have gained recognition as having valuable traits that can considerably influence citizens’ health if information is provided. However, this sector is developing slowly in Canada, unlike U.S., Europe, and Japan, due to the restrictive, rigorous, and complicated regulatory labelling system.

This study has made a step towards understanding the potential for the functional food industry to contribute to the health of citizens if more flexible labelling regulations exist. It has helped determine the economic implications of labelling the health attributes of functional foods. Nevertheless, further research into the functional food industry is necessary.
REFERENCES


Canada Gazette. (2003b). *Regulations Amending the Food and Drug Regulations (Nutrition Labelling, Nutrient Content Claims and Health Claims)*. (Part II,
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APPENDIX A: A DETAILED ESTIMATION OF CVD AND CHD COSTS
Table A1 depicts a detailed estimation of health care costs of CVD for 2006. The total direct and indirect costs of CVD are calculated, as are all the cost categories of direct and indirect costs of CVD, by applying the change of CPI and the change of population to each cost category separately. It is important to note that hospitalization and mortality costs comprise a huge portion of the total costs of CVD as they account for 55% of total cost of CVD.

In order to have a more explicit view of CHD costs, Table A2 reports estimates of the components of CHD’s direct and indirect costs in 2006. All the components of direct and indirect costs of CHD ($1998), as reported in Table A1, were used. Considering the change in CPI and change in population, each cost category of direct and indirect costs of CHD was estimated for 2006. As anticipated, hospital costs and mortality costs compose the largest amount of total costs, representing 69% of total CHD costs.
Table A1. Estimation of the categories of direct and indirect costs of CVD in Canada ($2006).

<table>
<thead>
<tr>
<th>Cost Category</th>
<th>1998</th>
<th>2006</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Costs (HC) (millions CAN$)</td>
<td>4,161.8</td>
<td>5,312.5</td>
</tr>
<tr>
<td>Drug Costs (DC) (millions CAN$)</td>
<td>1,772.8</td>
<td>2,262.9</td>
</tr>
<tr>
<td>Physicians Costs (PC) (millions CAN$)</td>
<td>822.3</td>
<td>1,049.6</td>
</tr>
<tr>
<td>Other Direct Costs (ODC) (millions CAN$)</td>
<td>4,073.5</td>
<td>5,199.8</td>
</tr>
<tr>
<td>Health Research Costs (HRC) (millions CAN$)</td>
<td>61.2</td>
<td>78.2</td>
</tr>
<tr>
<td><strong>Total Direct Costs (millions CAN$)</strong></td>
<td>10,891.6</td>
<td>13,902.9</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>INDIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality Costs (MC) (millions CAN$)</td>
<td>8,250</td>
<td>10,531.0</td>
</tr>
<tr>
<td>Morbidity Costs due to Long-Term Disability (LTDMC) (millions CAN$)</td>
<td>3,151.5</td>
<td>4,022.8</td>
</tr>
<tr>
<td>Morbidity Costs due to Short-Term Disability (STDMC) (millions CAN$)</td>
<td>253.3</td>
<td>323.3</td>
</tr>
<tr>
<td><strong>Total Indirect Costs ($1998) (millions CAN$)</strong></td>
<td>11,654.8</td>
<td>14,877.1</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>TOTAL COST</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total Cost (millions CAN$)</strong></td>
<td>22,546.4</td>
<td>28,780.1</td>
</tr>
</tbody>
</table>


2 DC ($2006) = [DC ($1998)+ (DC ($1998)* change in CPI)+ (DC ($1998)* change in population)] = [1,772.8+(1,772.8*0.19)+(1,772.8*0.06)].

3 PC ($2006) = [PC ($1998)+ (PC ($1998)* change in CPI)+ (PC ($1998)* change in population)] = [822.3+(822.3*0.19)+(822.3*0.06)].

4 ODC ($2006) = [ODC ($1998)+ (ODC ($1998)* change in CPI)+ (ODC ($1998)* change in population)] = [4,073.5+(4,073.5*0.19)+(4,073.5*0.06)].

5 HRC ($2006) = [HRC ($1998)+ (HRC ($1998)* change in CPI)+ (HRC ($1998)* change in population)] = [61.2+(61.2*0.19)+(61.2*0.06)].


7 LTDMC ($2006) = [LTDMC ($1998)+ (LTDMC ($1998)* change in CPI)+ (LTDMC ($1998)* change in population)] = [3,151.5+(3,151.5*0.19)+(3,151.5*0.06)].

8 STDMC ($2006) = [STDMC ($1998)+ (STDMC ($1998)* change in CPI)+ (STDMC ($1998)* change in population)] = [253.3+(253.3*0.19)+(253.3*0.06)].

9 Other direct costs (ODC) include expenditures for care in other institutions and additional direct health expenditures. However, they are net Health Research Costs.

4 Source: Statistics Canada, 2007b. The Consumer Price Index (CPI) for 1998 was 91.3; the 2006 CPI was 109.1. The change in CPI was calculated by subtracting 91.3 from 109.1 and dividing by 91.3.

5 Source: Cansim via E-Stat, Table 051-0005. Quarterly population estimates. The average population estimate for 1998 was 30,125,715, and 32,581,490 for 2006. The change in population was calculated by subtracting 30,125,715 from 32,581,490, then dividing by 32,581,


7 Total Indirect Costs were calculated by summing MC, LTDMC, and STDMC.

8 Total Cost is the sum of Total Direct and Total Indirect Costs.

Source: Author’s estimates (see text for detail).
Table A2. Estimation of the categories of direct and indirect costs of CHD in Canada ($2006).

<table>
<thead>
<tr>
<th></th>
<th>CHD 1998</th>
<th>CHD 2006</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Costs (HC)</td>
<td>1,274.8</td>
<td>1,627.3</td>
</tr>
<tr>
<td>Drug Costs (DC)</td>
<td>512.7</td>
<td>654.5</td>
</tr>
<tr>
<td>Physician Costs (PC)</td>
<td>247.7</td>
<td>316.2</td>
</tr>
<tr>
<td>Other Direct Costs (ODC)</td>
<td>1,226.9</td>
<td>1,566.2</td>
</tr>
<tr>
<td>Health Research Costs (HRC)</td>
<td>18.5</td>
<td>23.6</td>
</tr>
<tr>
<td>Total Direct Costs</td>
<td>3,280.5</td>
<td>4,187.6</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>INDIRECT COSTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality Costs (MC)</td>
<td>4,845.8</td>
<td>6,185.6</td>
</tr>
<tr>
<td>Morbidity Costs due to Long-Term Disability (LTDMC)</td>
<td>567.9</td>
<td>724.9</td>
</tr>
<tr>
<td>Morbidity Costs due to Short-Term Disability (STDMC)</td>
<td>120.3</td>
<td>153.5</td>
</tr>
<tr>
<td>Total Indirect Costs</td>
<td>5,534</td>
<td>7,064</td>
</tr>
<tr>
<td>Change in CPI (1998-2006)</td>
<td>0.19</td>
<td>0.19</td>
</tr>
<tr>
<td>Change in population (1998-2006)</td>
<td>0.08</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>TOTAL COST</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Cost (millions CAN$)</td>
<td>8,814.5</td>
<td>11,251.6</td>
</tr>
</tbody>
</table>

1 HC ($2006) = (HC ($1998) + (HC ($1998)* change in CPI) + (HC ($1998)* change in population)) = [1,274.8 + (1,274.8*0.19) + (1,274.8*0.06)].
2 DC ($2006) = (DC ($1998) + (DC ($1998)* change in CPI) + (DC ($1998)* change in population)) = [512.7 + (512.7*0.19) + (512.7*0.06)].
4 ODC ($2006) = (ODC ($1998) + (ODC ($1998)* change in CPI) + (ODC ($1998)* change in population)) = [1,226.9 + (1,226.9*0.19) + (1,226.9*0.06)].
6 Total Direct Costs were calculated by summing HC, DC, PC, ODC, and HRC.
7 Other direct costs (ODC) include expenditures for care in other institutions and additional direct health expenditures.
8 Total Indirect Costs were calculated by summing MC, LTDMC, and STDMC.

Source: Author’s estimates (see text for detail).
A **case-control study** is a study design that examines one group of people who have experienced an event (usually an adverse event) and another who have not experienced the same event, and looks at how exposure to suspect (usually noxious) agents differed between the two groups. This type of study design is most useful for trying to ascertain the cause of rare events, such as rare cancers. Case control studies can only generate odds ratios (OR), but not relative risks (RR) (BMJ Clinical Evidence, 2007).

A **cross sectional study** is a study of a group of people at one point in time to determine whether an exposure is associated with the occurrence of a disease. Because the disease outcome and exposure (e.g., nutrient intake) are measured at the same time, a cross-sectional study provides a “snapshot” view of their relationship. Cross-sectional studies cannot provide information about causality (Oregon State University, 2007).

An **experimental study** involves an investigator examining the effects of intentionally altering one or more factors under controlled conditions (BMJ Clinical Evidence, 2007).

An **experimental group** is a group of subjects in an experimental study that receives a treatment. (IFIC, 2007).

An **observational study** has no experimental intervention or treatment applied. Participants are simply observed over time (Oregon State University, 2007). Observational studies include prospective cohort studies, case-control studies, and cross-sectional studies (Wang et al., 2004).

In a **placebo group**, a group of subjects are given a “fake” treatment that seems identical in appearance and taste to the real treatment. Placebo treatments are used to eliminate bias that might arise from the expectation that a treatment should produce a particular effect (IFIC, 2007).

A **prospective cohort study** is an observational study in which a group of people—known as a cohort—are interviewed or tested for risk factors (e.g., nutrient intake), and then followed up at subsequent times to determine their status with respect to a disease or health outcome (Oregon State University, 2007).

A **randomized controlled trial** is a clinical trial with at least one active treatment group and a control (placebo) group. In RCTs, participants are chosen for the experimental and control groups at random, and are not told whether they received the active or placebo treatment until the end of the study. This type of study design can provide evidence of causality (Oregon State University, 2007).