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An analysis of the potential role of functional foods in the primary prevention of coronary heart disease

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AN ANALYSIS OF THE POTENTIAL ROLE OF FUNCTIONAL FOODS IN THE PRIMARY PREVENTION OF CORONARY HEART DISEASE

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by

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DECLARATION

I hereby declare that this thesis, submitted in fulfilment of the requirements for the award of Doctor of Philosophy, in the Department of Biomedical Science, University of Wollongong, is my own work unless otherwise referenced or acknowledged. This document has not been submitted in whole, or in part, for qualifications at any other academic institution.

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Craig S Patch

26th May 2005

DEDICATION

To Eleanor, Kennedy, Lewis & Finlay

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“If I have seen farther than others, it is because I was standing on the shoulders of giants.”

Isaac Newton, c 1676

Newton acknowledges that he was able to make many varied scientific discoveries because he took advantage of all the knowledge and discoveries that had been made by others around him. I too acknowledge that I have benefited from the knowledge and wisdom of those whom I have worked with.

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LIST OF ABBREVIATIONS

%E	Percent energy
A	Attitude
AA	Arachidonic acid
ALA	alpha linolenic acid
ANOVA	Analysis of variance
APD	Accredited Practising Dietitian
ATP III	Adult treatment Panel 3
AusNut	Australia Nutrient tables
BMI	Body mass index
BMR	Basal metabolic rate
BP	Blood pressure
BS	Belief strength
CHD	Coronary heart disease
CHO	Carbohydrate
CI	Confidence interval
CVD	Cardiovascular disease
DH	Diet history
DHA	Docosahexaenoic acid
EDTA	Ethylenediaminetetraacetic acid
EE	Energy expenditure
EI_{rep}	Reported estimated intake
EPA	Eiocosahexaenoic acid
FR	Food record
FSANZ	Food safety Australia New Zealand
GM	Genetically modified
HDL-C	High density lipoprotein - cholesterol
HMGCoA	Hydroxymethylglutaryl coenzyme A
I	Intention

kcal	Kilocalorie
Kg	Kilogram
LDL-C	Low density lipoprotein - cholesterol
ALA	Linoleic acid
MC	Motivation to comply
MI	Myocardial infarction
Ml	Millilitres
mmol	Millimoles
MUFA	Monounsaturated fatty acid
n-3	Omega-3 Polyunsaturated fatty acid
n-6	Omega-6 Polyunsaturated fatty acid
NB	Normative belief
NCEP	National Cholesterol Education Program
NHMRC	National Health and Medical Research Council
NLEA	National Labelling and Education Act
NNT	Number needed to treat
OE	Outcome evaluation
P	P value
PAL	Physical activity level
PBC	Perceived behavioural control
PRO	Protein
PUFA	Polyunsaturated fatty acid
r	Correlation coefficient
R^2	Regression coefficient
s.d.	Standard deviation
SEM	Standard error of the mean
SFA	Saturated fatty acid
SN	Subjective norm
TC	Total cholesterol
TG	Triglycerides

TPB	Theory of planned behaviour
LCn-3	Long chain omega-3 fatty acids
WHO	World Health Organization

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Awards

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ABSTRACT

Coronary heart disease (CHD) is the single largest cause of death and disability in most modern western societies and poor diet is a significant risk factor in the pathogenesis of this disease. Despite significant advances in the treatment of CHD, effective primary prevention approaches remain elusive largely due to the difficulty of sustaining significant and long term dietary changes. In this doctoral program, the hypothesis that the use of functional foods is an effective strategy in the primary prevention of this lifestyle disease was examined. Methodological approaches grounded in the field of nutrition science and behavioural psychology, incorporating both quantitative and qualitative studies were used.

Results of a dietary trial in 25 patients, reflective of 'free-living' clinical practice conditions, found that a functional food-led intervention using plant sterol enriched margarine was an effective approach in the management of hypercholesterolemia. Sixty percent of subjects counselled on the inclusion of 25g per day of plant sterols achieved >15% reduction in total serum cholesterol, compared to none receiving standard dietary advice. In another dietary trial that was a randomised placebo controlled dietary intervention with parallel groups, 86 overweight, but otherwise healthy, subjects where asked to choose at least eight serves per day from a selection of either long chain omega-3 fatty acid (LCn-3) enriched foods (~125 mg LCn-3 per serve) or matched control foods. After six-months, participants were able to increase their dietary LCn-3 intake 4-fold (to ~1200 mg/day) and this was confirmed by a corresponding increase in the erythrocyte LCn-3 levels by 20%. This has implications for CHD reduction given the importance of dietary LCn-3.

Consumer and social research also suggest that functional foods can have an important role in public health. The Theory of Planned Behaviour (TPB) was used as a theoretical framework for a study examining prediction of intention to use n-3 enriched functional foods, using a purpose designed questionnaire and follow up of recorded intake. Regression analysis was able to show that the model was a significant predictor of intention ($R^2 = 0.725$, $P < 0.001$) and to a lesser extent behaviour ($R^2 = 0.298$, $P < 0.001$). Attitude was significantly positively associated with intention, whereas subjective norms and control beliefs were not. These results imply that the best prospects for modifying behaviour are likely to come through a change in attitude. Beliefs and attitudes of key influencers are a significant determinant of consumer attitude. Semi-structured interviews with stakeholders (e.g. nutrition professionals, scientists, regulators, media personnel and food industry representatives) revealed there are a number of areas of agreement in their attitudes towards functional foods, including the need for safety, taste, bioactivity and extensive evidence for claims. Divergent views were expressed in relation to the regulation of health claims and the levels of evidence required substantiating these claims. A strong feature of the areas of disagreement was lack of clear scientific evidence available to form opinions, and stakeholders often relied on pre-existing ideology. These differing views may impact negatively on consumer attitude to the use of functional foods.

In conclusion, analysis of clinical and consumer research suggests that use of functional foods can be an effective preventative health strategy for CHD as long as the maintenance of positive consumer confidence can be achieved.

CHAPTER 1

INTRODUCTION

Coronary heart diseases (CHD) collectively are the major cause of death and disability in most modern western societies. Primary prevention strategies are founded on the doctrine that prevention is better than cure. It has been demonstrated that population-based healthy eating primary prevention programs cost £14 - 560 (~AUD$ 34 -1373) per life year gained which is substantially cheaper than the cost of treatment (1). This would suggest that effective primary prevention strategies have the potential to reduce both the personal and financial burden of many lifestyle diseases. However, primary prevention programs are seen by many as achieving marginal gain at best and at worst are seen as ineffective (2).

To date, approaches to reduce CHD which have been largely based on educational and behavioural models of intervention, have met with limited success (2). They are underpinned by the rational premise that people, once given appropriate education and skills will make sound decisions regarding their long-term health status. Although there have been a number of programs which have successfully demonstrated improvements in knowledge, skill application and occasionally behaviour, they have seldom demonstrated improvements in risk factor reduction or improvements in morbidity and mortality outcomes (2). With tertiary treatment institutions under mounting resource and financial pressures,

there needs to be a redress of effective strategies that can potentially prevent these debilitating lifestyle diseases (3).

Recent advancements in food technology provide a new range of options for intervention programs which are only beginning to be explored. At the forefront of this technological boom are functional foods. These are foods which provide nutritional benefits beyond basic nutrition (4, 5). Currently there is no universally accepted definition. In its broadest definition functional foods can include not only newly formulated foods but also traditional whole foods (e.g. oats and walnuts) (5). Central to this research is the hypothesis that the use of functional foods is an effective primary prevention strategy as it applies to CHD.

With the unlikelihood that functional foods singularly will prevent CHD, it is important to place functional foods within an existing health promotion framework. Egger and Swinburn urge us to view lifestyle diseases as an epidemic, and state that epidemics historically have only been controlled after environmental factors have been modified and that existing intervention strategies for lifestyle diseases will only have limited impact if environmental factors are not addressed (6). There proposed model (epidemiological triad) supports a devotion of effort into three distinct, yet interrelating domains: host, environment and vector. For CHD, “host” refers to the biological and behavioural influences on diet and other lifestyle behaviours. “Environment” can be described in terms of the macro-environment and the micro-environment which are located in the physical, economic and socio-cultural environmental context (7). And finally the “vector” is represented by the food and its associated nutritional profile. It is suggested that effective interventions

need to address each of the components of the triad. By superimposing a functional food-led intervention onto components of the epidemiological triad model, effects of functional foods (vector) on biological and behavioural outcomes (host) as well as the effects of these on the interface with policy and legislation (environment) were investigated. In this thesis, evidence for the effectiveness of a functional food-led approach was gathered using dietary intervention studies, behavioural studies and a stakeholder analysis.

1.1 Methodological issues

Establishing evidence for effective CHD prevention using dietary intervention studies provides a number of unique challenges. Firstly, the pathophysiology of CHD develops over a long period of time and absolute rates of mortality in the populations are relatively small. In order to detect the effect of an intervention on mortality rates, trials require large sample sizes with long intervention and follow up periods. This is usually cost prohibitive, leaving the definitive studies unavailable. Secondly, there is no single cause for the development of this disease, and as such a number of attributable risk factors have been described. These include diet-related factors such as type 2 diabetes mellitus, obesity, elevated cholesterol, particularly LDL-cholesterol and high triglyceride levels, lifestyle-related risk factors such as physical inactivity and smoking and genetic factors such as gender and family history (8) and more recently a low level of erythrocyte eicosapentaenoic acid (EPA) + docosahexaenoic acid (DHA) (9). Often these intermediate markers of disease do not necessarily answer questions of mortality. Despite our best efforts to provide evidence of the effectiveness of primary prevention interventions,

there will always be uncertainty because of the lack of definitive evidence related to the disease endpoint itself. This should not lead to abandonment of research into primary prevention programs, but does serve as a cautionary tale which needs addressing through appropriate trial design and a reasoned approach to the application of newly acquired knowledge.

Another concern for research design is that the utility of findings from the research setting to a ‘real life – free living’ context is not always assured (10). Caution is required when interpreting results from controlled clinical contexts and transferring them into free-living settings. For this reason it is important to design intervention trials that are grounded in a free-living context ensuring that the study outcomes are not overly inflated due to the tightly controlled design where compliance is assured. Also, extrapolating scientific evidence from one paradigm to another may falsely raise expectations. For example, positive results obtained from a sample of hypercholesterolaemic patients cannot be extrapolated to the general population. Therefore, consideration of the use of functional foods for public health benefits or as part of individual therapeutic regimens requires certain distinctions to be made.

Dietary interventions designed to promote health may be categorised two ways: those that aim to modify dietary behaviour and reduce the level of risk factors in individuals, and those that aim to address the underlying determinants of health in populations as a whole (11). Each category of dietary intervention has specific considerations with respect to study design as well as the conclusions which can be drawn from the results. In relation to CHD, primary prevention interventions can be categorised as those targeted toward individuals that aim to reverse the

condition of diagnosed dyslipidaemia and those that aim to improve the dietary fat intake (reduce saturated fat and increase essential fatty acids) in the population.

Despite the favourable results that individual counselling has on patients with elevated cholesterol in controlled studies (where food is provided) the results in a clinical environment are far less impressive (12). This has led to the undermining of nutrition counselling as the front line treatment of dyslipidemia in favour of more expensive drug therapy (13). However, the recent release of plant sterol/stanol enriched food products has renewed interest in dietary therapy, culminating in the recommendation of plant sterol/stanol use as part of medical nutrition therapy by the authors of the US National Cholesterol Education Program (14). However, there are no studies in the context of clinical practice that support this statement.

1.2 The utility of functional foods in clinical practice

In this thesis the first study was designed to test the hypothesis that specific dietary prescription of margarine enriched with 2g/day of plant sterols/stanols is more effective than standard dietetic advice in achieving an improved lipid and nutrient profile in free-living subjects with hyperlipidaemia. A randomised parallel design trial of comparative 12-week interventions was conducted with patients referred by a general practitioner to a dietary outpatient clinic for the management of hyperlipidaemia. In total, twenty-five patients (15 women and 10 men) completed the study. The control group received counselling regarding diet for hyperlipidaemia was based on the National Cholesterol Education Program (NCEP) guidelines (14), whereas the intervention group was instructed to

incorporate ~25 g/day of margarine containing plant sterols/stanols, which delivered ~2 g of plant sterols/stanols. Changes in diet, body weight and serum total cholesterol, LDL cholesterol, HDL cholesterol and triglycerides were measured and reported at baseline and at three months. The number needed to treat (NNT), which is an index of effectiveness of the intervention, was calculated. This study gave an indication of the utility of functional foods in clinical practice.

1.3 The utility of functional foods in population interventions

Independent of the favourable effect that a reduction of hypercholesterolemia has on CHD risk, an adequate long chain omega-3 fatty acid (LCn-3) intake (EPA + DHA) has also been found to be beneficial for the prevention of CHD (15-17). Australians eat ~ 1/3 of the suggested intake of 650mg/day (18). Thus, from a population perspective there could be significant population health benefits from increasing the intake of this essential fatty acid in a large portion of the population. Therefore, in this thesis a second study was designed, this time with a population scope in mind, to test the hypothesis that a dietary strategy based on foods enriched with microencapsulated LCn-3 (fish oil) is more effective than a diet with non-enriched foods in achieving the target dietary recommendations of 1g/day of LCn-3 as well as providing cardioprotective benefits. An evaluation of reported dietary intakes in a 6-month randomised controlled parallel design supplemental trial was carried out. 86 overweight men and women with elevated triglycerides who reported low baseline fish intake were recruited and all subjects were supplied with a range of processed foods enriched with LCn-3 and instructed to consume 8 serves per day (equivalent to ~ 1 g/day LCn-3), or matched control foods.

Reported energy, macronutrient and fatty acid intakes were measured by diet history and 3-day food records in conjunction with red cell fatty acids and surrogate markers of CHD. Food preferences were also determined along with the impact of this style of eating on the whole diet. The effect that this functional food-led intervention had on the total diet and dietary biomarkers was assessed and the feasibility and practicalities of the widespread application of this approach discussed.

The ability to extrapolate results from these studies is somewhat limited by their design. In order to achieve the study design objective of placebo-controlled, randomised interventions subjects were provided with all test foods. The relatively high compliance in this study may not be achieved outside of the confines of such a controlled environment. Therefore, two more studies were designed for this thesis to investigate consumer beliefs, attitudes and ultimately predict the consumer behaviour in relation to functional foods.

1.4 Consumers beliefs and behaviours with respect to functional foods

There are a number of theories which aim to explain and predict human behaviour. These include, but are not limited to, the transtheoretical model (TTM), health belief model (HBM) and theory of planned behaviour (TPB). All have valid use in specific areas, with no one theory universally recognised. After a review of the literature the TPB was chosen as the model of choice to help understand consumer behaviour in relation to omega-3 (n-3) enriched functional foods. In brief TPB suggests that the best predictor of human behavior is a person's conscious

decision to perform the behavior. This behavioural intention is determined by several factors, including the attitude they hold towards the behavior, the degree of social pressure felt by the person to perform or not perform the behavior, and the degree of control that the person feels they have over performing the behaviour (19). These three factors in turn are determined by a number of beliefs and subsequently how each is evaluated. It has been demonstrated that the TPB, or modified versions of it, is applicable in explaining consumers' food choice (20-22), including supplement use (23) and genetically modified products (24), however this is the first time it has been applied to the area of functional foods.

The first step in testing the hypothesis that the TPB predicts intention and use of functional foods enriched with n-3 was the implementation of exploratory focus group research. As suggested by the authors who described the TPB, the first step in exploring behaviour is to unveil the salient beliefs toward the behaviour of interest, which in this case is toward functional foods enriched with n-3 fatty acids (19). Focus group interviews were conducted with adult community based residents living in the Illawarra region of New South Wales, Australia. Forty-two participants (29 female; 13 male) aged 30-80y were recruited by advertisement and attended one of six focus groups which were recorded and transcribed *verbatim*. Content analysis was carried out and sub-categories were developed to capture the emerging themes according to the TPB model. Each aspect of the model was explored and salient behavioural, normative and control beliefs were discussed and reported. The consumer attitudes and purchase intentions identified in this study will be helpful to educators as they plan messages and strategies to

guide dietary choices related to products with n-3 fatty acids. In addition the beliefs identified and categorised from this study were then used to formulate a 54 item questionnaire which was used in the subsequent study. The questionnaire, in conjunction with information on dietary intake, was used to quantify responses regarding intention to use, beliefs and attitude toward n-3 enriched functional foods. A cross-sectional self-administered questionnaire was completed by community based residents living in the Illawarra region of New South Wales, Australia. Two sub-samples were surveyed via questionnaire: community members who responded to a local media advertisement (n = 79), and subjects in a dietary intervention trial for type 2 diabetes mellitus (n = 50). Using dietary intake data in conjunction with the TPB variables, questionnaire items were constructed to measure behaviour as well as intention to consume n-3 enriched functional foods. Results from sub-samples did not differ and were combined for analysis. Using regression analysis it was demonstrated that the model was a significant predictor of intention as well as being externally valid when intake data was considered. This work will help identify cognitive targets for the promotion of n-3 enriched foods, which are important to identify if we are to increase the population intake of n-3 through the addition of this active ingredient through a range of processed foods. However, primary prevention strategies based on education and promotion alone will be ineffective if the environmental factors highlighted in the epidemiological triad are not addressed (6).

1.5 Environmental influences on the utility of functional foods

In order for a functional food-led intervention to have a significant impact on CHD a supportive environment through effective policy and legislation is required. McConnon *et al* (2002) argue that the success of functional foods in both commercial and health promoting terms is reliant on the resolution of incongruence between stakeholder beliefs and attitudes towards these foods as this will have an effect of consumer confidence (25).

The final study of this thesis tested the hypothesis that beliefs about functional foods are related to underlying ideology. Using semi-structured interviews the beliefs and attitudes of stakeholders towards the development and promotion of functional foods were unveiled. A sampling frame was drawn up of potential stakeholders in Australia using the categories as outlined by McConnon *et al* (25). A purposive sample of individuals was used representing general practitioners (GP's), nutritionists, industry, media, regulatory bodies and authoritative organisations from various States. Interviews were audio taped and later transcribed to allow systematic analysis of the discussion. Using the theoretical framework proposed by McConnon *et al* (2002)(25) as well as reviewing the literature pertinent to this area (26, 27), ten open ended questions were devised to determine stakeholder awareness, beliefs, attitude issues regarding the development and promotion of functional foods. The results of the stakeholder interviews are discussed in relation to current national and international trends in food policy and regulation. Outcomes from this study provide a better

understanding of the areas of agreement and disagreement between the various stakeholders, and ways to reconcile incongruence are suggested.

1.6 Summary

The primary prevention of CHD presents us with two challenges: to treat those with overt symptomatic risk factors and to prevent CHD in people with no identifiable risk factors. Neither of these challenges is currently being met with any widespread effect (28). The studies reported here present an alternative primary prevention paradigm using innovations in food technology as an underlying driver. To answer the question as to the effectiveness of a functional food-led intervention this work has gone beyond the traditional approach of controlled clinical studies to incorporate dietary and behavioural psychology methodologies. In addition, the interface between functional foods, consumer behaviour and policy has been investigated and some of the implications of this intervention approach for food regulation have been discussed.

1.6.1 Hypotheses

The advent of functional foods is an important technological innovation which has the potential to help overcome the natural aversions humans have to dietary change. However, evidence of the effectiveness of a functional foods-led intervention is required. In addition, the use of functional foods by consumers must be congruent with the socio-cultural beliefs of the target population and supported by appropriate public policy. To confirm the central hypothesis that the use of

functional foods is an effective primary prevention strategy, the following hypotheses were tested:

1. Specific dietary prescription of margarine enriched with 2g/day of plant sterols/stanols is more effective than standard dietetic advice in achieving an improved lipid and nutrient profile in free-living subjects with hyperlipidaemia.

2. A dietary strategy based on foods enriched with microencapsulated fish oil is more effective than a diet with non-enriched foods in achieving the target dietary recommendations of 1g/day of LCn-3 and providing the cardioprotective benefits of fish oils.

3. The behaviour model, Theory of Planned Behaviour, can be used to predict consumer's intention and use of functional foods enriched with LCn-3.

4. Stakeholder beliefs about functional foods will influence the consumer environment.

1.6.2 Aims

More specifically the aims of this thesis are to:

i. Determine the effectiveness of prescribing 25g of margarine per day (containing 2 g plant sterols/stanols) to hypercholesterolaemic patients referred to an outpatient clinic on biochemical and dietary outcomes.

ii. Describe the feasibility of incorporating LCn-3 enriched foods in the diets of people with low fish intakes and the subsequent impact on the total diet, making clear reference to the quality and validity of the dietary data through the use of biomarkers.

iii. Gain an understanding of the salient beliefs underlying Australian consumer attitudes and purchase intentions with regard to n-3 enriched functional foods using focus group discussions.

iv. Identify the nature, strength and relative importance of influences on intentions to consume foods that are good sources of n-3 fatty acids using the Theory of Planned Behaviour (TPB).

v. Ascertain the beliefs and attitudes of key leaders (stakeholders) towards the development and communication of functional foods.

CHAPTER 2

LITERATURE REVIEW[1]

2.1 Coronary Heart Diseases

Coronary heart diseases (CHD), also known as coronary occlusion, ischemic heart disease (IHD), atherosclerotic heart disease or coronary thrombosis, are the leading cause of death in most Western societies including Australia (29). This has wide public health implications. In 2002, CHD was the single largest cause of death and the most common cause of sudden death, and accounted for 26,063 deaths which represent 19.5% of the total number of deaths and accounted for 51.8% of cardiovascular deaths in Australia (29). Over half of the CHD deaths were from sudden heart attacks (acute myocardial infarction) (29). Overall, males are twice as likely as females to die from CHD (29). From the results of the Australian 2001 National Health survey, 1.9% (355,600) of those surveyed individuals reported having manifestations of CHD (29), whereas in 2001-02 it was estimated that there were 48,700 fatal and non-fatal CHD events (29). Cardiovascular diseases are the

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most costly disease for the health system and in 1993-94 it accounted for $3,719 million or 12% of total direct health system costs in that year (29). Coronary heart diseases were the major contributor to this cost, accounting for 25% ($894 million) of the total (29). Furthermore cardiovascular drugs accounted for 16.8% of all costs of prescription drugs listed on the Pharmaceutical Benefits Scheme in 1994 ($27.3 million per year) (29). From 1992-1994, there was an average increase of 4.0% per year in these prescription drugs (29).

The clinical manifestations of CHD are chronic arterial obstructions or acute arterial occlusions in various territories, which ultimately lead to the condition where there is an inadequate supply of blood to the heart muscle leading to ischemia (30). In some cases ischemia may become so severe that it causes a myocardial infarction (coronary infarction, coronary thrombosis, or heart attack). This may result from the progression of atherosclerosis within the coronary arteries, or result from the formation of a blood clot within the arteries (thrombosis), or most likely a combination of both that ultimately leads to a necrosis of the cardiac muscle cells (30). Atherosclerosis is a disease of the tunica intima (arterial inner lining of the lumen) of the large and medium sized arteries, characterised by the development of fibrous, fatty deposits called plaques or atheromas (31). These atheromas eventually become calcified, rendering them rigid and narrow.

The pathogenesis is only understood in part as yet. The development of an atheroma (or plaque) in the intima of the major arteries marks the development of the pathogenesis of atherosclerosis. Arterial wall abnormalities, blood composition abnormalities and haemodynamic alterations are generally accepted to be

causative (Virchow's triad) (8). There is strong support for a causative relationship between hypercholesterolaemia and atherosclerosis originally evidenced by the premature development of CHD in younger individuals with familial hypercholesterolaemia (32). There is also direct evidence from animal models of the effect of elevated LDL-cholesterol on atherogenesis (32). Research suggests this forms a cytotoxic compound and initiates the cascade of events eventually leading to atherosclerosis (33). Shear-stress induced micro injuries of the endothelium in hemodynamically compromised regions together with local coagulation activation associated with microinflammation of the plaque are currently thought to cause plaque rupture (8). The resultant local clot formation is the ultimate reason for organ failure.

2.2 Dietary risk factors

No authentic cause of atherosclerosis has been isolated, but is believed to be multifactorial (34). To date, aetiologic research has only established risk factors which provide the basis for some efficient but incomplete means for prevention. Most of this evidence relating to risk factors is epidemiologic and therefore consists of statistical evidence for probability of evidence (34). The three factors most consistently associated with an increase in the incidence of CHD include hypertension, hypercholesterolemia and cigarette smoking. Other modifiable risk factors include obesity, lack of exercise, hypertriglycerideaemia and low HDL cholesterol levels. There are also a number of non-modifiable risk factors such as male sex, diabetes mellitus, and family history of premature CHD and the presence of definitive atherosclerosis.

There is considerable evidence that dietary fats lead to increases in blood levels of cholesterol, and ultimately to atherosclerosis, and in the past this has been the theoretical underpinning to many health promoting and dietary prevention strategies (35). Initially, expert committees from major scientific associations such as the National Heart, Lung and Blood Institute and the American Heart Association initiated clinical trials with the aim of decreasing plasma cholesterol levels in an attempt to decrease the risk of atherosclerotic disease (the diet-heart hypothesis) (36). However it is now becoming apparent that the role of diet in the prevention of CHD is more complicated than this simple cause and effect relationship would suggest.

2.3 The diet-heart disease hypothesis

Keys *et al* first suggested the link between serum cholesterol, dietary fat intake and cardiovascular disease in the landmark Seven Countries study (37). Populations living in Japan and Crete were shown to have the lowest rates of CHD and tended to have a lower serum cholesterol and consume a diet low in saturated fat, while being high in polyunsaturated fatty acids (PUFA) (38). However, it is apparent that although there was a positive relationship between serum cholesterol and rates of CHD, there was wide inter-population variation in CHD rates at all levels of serum cholesterol. For example, at a cholesterol level of 6.00mmol/l, the US and Finland had an incidence of cardiovascular disease (CVD) between 30-45%, whereas Japan and Crete had an incidence of <10% (39).

In addition to elevated levels of plasma cholesterol (TC), increased levels of triglycerides (TG), as well as a reduced level of high-density lipoprotein cholesterol (HDL) have been linked to the development of premature CHD (40-42). In a prospective observational study, the Framingham Study, it was concluded that coronary risk starts to increase with total cholesterol values over about 5.00 mmol/L (41). In addition, for patients with established CHD, elevated lipids and lipoprotein remain predictive of future CHD events and even death (43). Elevated triglycerides have also been suggested as having a predictive role in CHD (44). The authors concluded that high plasma cholesterol; high blood pressure and cigarette smoking are predictors of coronary risk. In addition, the Strong Heart Study (2003) found the ratio of total and HDL cholesterol to be a good predictor of CVD in both male and female type 2 diabetics, although a stronger predictor in men (45). However the investigators acknowledged that they could only demonstrate 'guilt by association' and could not prove that they cause CHD (46).

Hu *et al* followed 85,941 women from 1980 to 1994 from the Nurses' Health study and investigated the incidence of CHD (15). The age specific incidence rates of coronary disease showed a decline in over time. The relative risk of all coronary disease decreased by 31% from 1980 – 1992. This model suggested that an improvement in diet explained a 16% decrease while the increase in body-mass index explained an 8% increase in the incidence of CHD. Researchers noted that the dietary changes were from a decrease in the intake of saturated and trans fatty acids, whereas the intake of polyunsaturated fatty acids (PUFA), cereal fibre, marine n-3 polyunsaturated fatty acids (LCn-3) and folate increased (15). In

summary they concluded that a 10% decrease in serum cholesterol levels is associated with a 30% reduction in CHD after adjusting for confounders and measurement errors (47) which is consistent with earlier risk modelling which reported a 2-3% relative risk reduction with each 1% reduction in serum cholesterol (37). In addition, there is a wealth of observational, experimental and genetic evidence to suggest that increasing HDL cholesterol will also lower the risk of CHD (48-51). The ratio of total to HDL cholesterol is now considered more important than these individual factors alone in estimating the risk of CHD (52-54). Therefore it is important to assess the effects of dietary interventions on both of these biomarkers.

Using these epidemiological associations as a theoretical base, drug intervention trials have been carried out since the 1970's. In 1994, the Scandinavian Simvastatin Survival Study conducted a double-blind trial using 4444 patients with existing CHD (55). Those patients receiving 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (HMGCoA reductase inhibitor (statin)) showed total cholesterol, LDL-cholesterol, HDL- cholesterol changes of –25%, –35% and +8% respectively. Using the combined end-points of coronary deaths and nonfatal myocardial infarct (MI), a reduction in incidence of 37% was achieved over the entire study (5.4 years) (55). Comparable results were reported by using Pravastatin (56). Similarly, dietary studies into the secondary prevention of heart disease have shown a reduction in serum cholesterol by 8 -12% through dietary manipulation – aiming for low saturated fats, high n-3, high fruit and vegetables – has resulted in a 25 - 45% reduction in cardiovascular mortality (57-59).

Can the same benefits be achieved with dietary interventions aimed at reducing total cholesterol in primary prevention? Epidemiological and experimental evidence indicate that a diet high in SFA is associated with high levels of serum cholesterol which are in turn related to CHD, although there are some significant anomalies in the evidence where some of the earlier intervention studies failed to improve CHD prognosis (60). However, it can be noted that the trials were conducted on low risk patients and used high fat diets (~ 40% of energy as lipids), comprising of low saturated fat and cholesterol levels, but very high level of PUFA (15 to 20% of energy), particularly n-6 (60). An epidemiological investigation into the relationship between dietary intake and CHD mortality could not find any significant association between any of the dietary components and mortality (61). It was concluded that a better understanding of the pathway between dietary intake and coronary disease is needed, as the current diet-lipid-heart hypothesis may be overly simplistic (61). Furthermore a number of primary prevention trials failed to determine this association. In the Multiple Risk Factor Intervention Trial, 12,866 high risk males between the ages of 35 and 57years were recruited to a randomised primary prevention trial (62). Men randomly assigned to the intervention group were counselled on lowering cholesterol, received dietary advice for reducing hypertension, and instructed to cease smoking, whereas those in the usual care group were referred to usual community follow up. After an average of 7 years, and despite significant reductions in risk factors in the intervention group, both CHD mortality and total mortality did not differ between groups (62).

In summary, the diet – heart disease hypothesis is characterised by the following syllogism: that an elevated intake of saturated fat, and total fat, and dietary cholesterol will raise serum cholesterol, which in turn leads to atherosclerosis and ultimately results in CHD morbidity and mortality (63). As this hypothesis would suggest, a reduction in dietary saturated and total fat should result in a reduction in morbidity and mortality from CHD. It is the lack of direct evidence that is missing in order to prove the hypothesis that has prompted others to question its validity (64).

2.4 The diet – heart disease hypothesis revised

Although research has focused on the role of SFA, serum lipids and atherosclerosis for the last 3 decades, more recent attention has turned to the role of specific dietary fats, and in particular n-3 on specific manifestations on CHD, namely sudden death (65-67). N-3 fatty acids are a family of naturally occurring PUFA. It is in the position of the double bonds within their hydrocarbon chain that gives n-3 fatty acids their name and also their physical and physiological properties. For example, in n-3 the terminal double bond (i.e. closest to the methyl end of the hydrocarbon chain) is on C3. In n-6 the terminal double bond is on C6. The n-6 and n-3 families of PUFA have a range of members, ranging from linoleic acid (LA; 18:2n-6) and α-linolenic acid (ALA; 18:3n-3), both of which are plant-derived oils. In fact, n-6 and n-3 double bonds can not be inserted into fatty acids by animal enzymes, only by plants through the action of the Δ12- and Δ15-desaturase respectively (68). Although animals cannot derive these fatty acids, mammals have a requirement for these, and thus they are regarded as essential fatty acids. Although mammalian cells cannot synthesis these fats, they can

metabolise them by further saturation and elongation. For example linoleic acid can be converted to γ-linolenic acid (18:3n-6) by Δ6-desaturase and then γ-linolenic acid can be elongated to dihomo- γ-linolenic (68). This can be further desaturated by Δ5-desaturase to yield arachidonic acid (20:4n-6). Using the same series of enzymes α-linolenic acid is converted to EPA (20:5n-3) (68). Because the n-6 and n-3 series utilise the same enzymatic pathways (Δ6-desaturase reaction) which is a rate limiting step, there is competition between these fatty acid families.

Further metabolism of EPA to DHA (22:6n-3) is mediated by the formation of docosapentaenoic acid (DPA; 22:5n-3), then the addition of two additional carbons (24:5n-3) and desaturation at the Δ6 position to form 24:6n-3 and finally β-oxidation to yield DHA (69). It is important to note that recent studies have revealed that the conversion of ALA into its longer chain derivatives is not efficient in humans, and some suggest that conversion of ALA to DHA is around 15% (70-72). Similarly arachidonic acid can be metabolized by the same enzymatic series to yield, in turn, 22:4n-6, 24:4n-6, 24:5n-6 and 22:5n-6 (73).

The long-chain derivatives of LA and ALA acid (AA and EPA respectively) are important biologically as they exert potent physiological effects (68). EPA and AA are competitively metabolized via the cyclooxygenase, lipoxygenase and cytochrome P450 pathways to eicosanoids such as prostaglandins (PG), thromboxanes (TX) and leukotrienes (LT) (74). Arachidonic acid is the substrate for the synthesis of 2-series PG, 2-series TX and 4-series LT, whereas EPA is the substrate for the 3-series PG, 3-series TX and 5-series LT. The competitive metabolism of these substrates has important implications in the regulation of

smooth muscle contraction, platelet aggregation, inflammation, immune function and cell proliferation (74). For example, EPA-derived TXA 3 shows reduced proaggregatory and vasoconstricitive properties compared to the AA-derived TXA 2, while the antiaggregatory and vasodilative efficacy of PGI 3 equals that of PGI 2 (74). Because ALA and LA compete for the Δ-6-desaturase enzyme in the desaturation chain elongation pathway, both incorporation of ALA into the tissues and plasma as well as the conversion of EPA and DHA, are influenced by LA levels. The net result of a higher ALA levels and subsequent EPA metabolite is the suppression of TXA 2 (and promotion of TXA 3) which has a proaggregatory vasoconstrictor, as LA conversion to arachidonic acid is decreased (64). In addition, PGI 3, the antiaggregatory prostaglandin derived from EPA, has effects comparable to those AA-derived PGI 2 (75). Subsequent research revealed that the other 3-series eicosanoids derived from EPA (e.g. PGE 3, LTB 5) also have reduced physiological activity compared to their 2-series counterparts (75).

Thus the net results in prostanoid synthesis from n-3 results in reduced proaggregatory and vasodilatory effects (74). The differences between the EPA and AA metabolism are likely to be the basis for the observed reduction in thrombosis and anti-inflammatory properties of dietary n-3 fats (75). Thus it is thought that a lower n-6:n-3 ratio has a beneficial effect on primary and secondary prevention of atherosclerosis, thrombosis and embolic phenomena, hypertriglyceridaemia and hypertension (74).

Presently most Western countries consume diets with relatively low levels of this LCn-3. A good dietary source of LCn-3 is fish. Fish can be classified as either lean

fish, where fat is stored in the liver (e.g. whiting, cod) or fatty fish, where the oil is contained in the flesh (e.g. salmon, tuna, herring, mackerel). Essentially all fish oil is high in LCn-3, although the proportions of EPA, DPA and DHA can vary. The major dietary source of AA is from meat and meat products, but most AA comes from the conversion of LA rather than from the diet (76). In contrast, major sources of LA come in the form of cooking oils (e.g. corn oil, sunflower oil and rapeseed oil) and margarines made from these oils, which make a significant contribution to the intakes of these fatty acids (68). This results in a higher intake of n-6 in modern Western societies over the last 35 years (68). For example in the UK the average intake of LA among adults increased from ≈ 11g in 1970 to ≈ 14g in 1990 (68). However, more recently margarines derived from canola and flax have been introduced into the food supply and contain more ALA. ALA can be metabolized to EPA and DHA through the desaturation chain elongation pathway (64). The physiological action of the n-6 series is interwoven with the actions of the n-3 series, and it is an imbalance between the two that has a detrimental effect on vascular health. The correction of this n-3 imbalance and LCn-3 deficiency in the modern diet is a key step to improving the cardiovascular risk in our population (77).

Interest in n-3 and their health benefits were stimulated by earlier research into the dietary habits of Greenland Eskimos. It was suggested that the reasons for the low cardiovascular disease rate, which was <10% of that predicted given their high intake of fat, was due to a diet which was high in seal meat and whale blubber (17, 78). The suggested cardioprotective component was LCn-3, with the intakes being

estimated to average as much as 5-15g/day (78). In comparison to Danes, Eskimos ate a diet that was lower in linoleic acid (LA) and higher in n-3; had platelet phospholipids that contains proportionately less LA, less AA, and more EPA; and had a lower mean platelet count and longer mean bleeding (79). The Japanese also exhibit a low mortality from CHD, with fatty fish also contributing significantly to the traditional Japanese diet (80). These results have been replicated in population studies that have found a positive association between communities with a high fish intake and lower rates of CHD (17, 81). Data from a number of epidemiological and case-controlled studies dating from the 1960's have strongly supported the inverse relationship between high LCn-3 blood concentration and intake on CHD mortality rates in Western countries (65-67, 79, 82-100). For example, the Zutphen study provided early epidemiological evidence of the importance of fish oil in the prevention of CVD (100). This study, conducted in 1960, observed a cohort of 852 middle aged men who had no history or symptoms of heart disease. After 20 years the incidence of heart disease was inversely related to fish intake. The authors concluded that 35 g fish/day equated to a 50% reduction in mortality from coronary artery disease (100). However, this area is not without controversy as others have failed to show significant benefits of modest intakes of fish oil on CHD rates (96, 101, 102).

Further support for an increase in LCn-3 comes from research into Paleolithic nutrition. N-3 fats were abundant in the diet of our Paleolithic ancestors (103). The original sources of n-3 in the food supply were from ubiquitous algae in the sea and of grasses and leaves on the land (104). This small amount of n-3 in the algae and

grasses became more concentrated as it was consumed up the food chain, particularly in fish and larger grazing animals (104). With modern domestic animal generally being fed on cereal based products, meat is generally low in these essential fatty acids (105). Although interesting, evidence from controlled intervention trial is needed to substantiate the hypothesis.

A secondary prevention study, the Diet and Reinfarction Trial (DART) randomised 2033 survivors of myocardial infarction who were at very high risk of dying from a heart attack with the next few years into a low fat diet or high fibre diet. The intervention group consumed ~2.3g EPA/day, whereas the control group consumed ~0.7g EPA/day. After 2 years of intervention the low dose fish oil group had a reduced mortality (29%), with no effect on blood pressure, serum lipids and no difference in ischaemic events, and the authors concluded that this suggests an antiarrhythmic effect (58, 106). Similar results have been reported by Singh *et al* (1992) (107). The clinical effectiveness of fish oil was most strikingly demonstrated in the GISSI Prevenzione study (108). This was also a secondary prevention study, but this time using a fish oil supplement, Marchioli *et al* (2002) randomised 11,323 survivors of myocardial infarction to either n-3 supplements (885mg of EPA + DHA/day), vitamin E (300mg/day), both or neither (control) (108, 109). After 12 months, compared to the control group, they demonstrated a significant reduction of cardiovascular deaths by 30% and a significant reduction of sudden death by 45% in the fish oil group, but not the others (108). There were no effects on cholesterol, blood pressure and a small reduction in triglyceride. Survival curves for the n-3 treatment group diverged early after randomisation and mortality was

significantly lowered after 3 months of treatment. The authors concluded that the early effect of low-dose (~1g/day) n-3 on total mortality and sudden death supports the hypothesis of an antiarrhythmic effect of this therapy (108). Equally impressive results have been reported with ALA. The Lyon heart study was a prospective, randomised single-blinded secondary prevention trial and compared the effect of a Mediterranean diet rich in ALA (precursor of long chain n-3 fatty acids) rich diet to the usual post-infarct prudent diet. Overall mortality was reduced and an adjusted risk ratio of 0.30 achieved (38, 110). An interesting addendum in these studies was the finding that significant reductions in CHD death were observed with little or no reduction in serum cholesterol (111).

When comparing these results to those obtained through drug interventions, the clinical validity of fish oils therapy in the secondary prevention of CHD becomes apparent. A recent meta-analysis of patients with CHD to determine the effectiveness of HMGCoA inhibitor (Statin) therapy concluded that there was a reduction in CHD mortality or non-fatal myocardial infarction of 25% (RR, 0.75; 95% CI 0.71-0.79), all cause mortality 16% (RR, 0.84; 95% CI, 0.79-0.89), and CHD mortality 23% (RR, 0.77; 95% CI, 0.71-0.83) (112). These values are no better that the results achieved in the fish oil supplementation trials.

There are a number of mechanisms via which LCn-3 may reduce the risk of heart disease. It may protect against both the pathological processes leading to the disease (i.e. atherosclerosis) and the process leading to death (i.e. stroke and myocardial infarction). Dietary n-6 and n-3 reduce triglyceride accumulation in the skeletal muscle and potentially in the cardiomyocytes and β cells (75, 113-116).

Elevated triglycerides are regarded as an independent risk factor of CVD and has been outlined in a number of reviews (117-119). The resultant lower tissue lipid levels are associated with an increase in insulin sensitivity (120). PUFA concomitantly suppress lipid synthesis in the liver, up regulating fatty acid oxidation in liver and skeletal muscle and increasing body glycogen (120). This repartitioning activity of PUFA has been observed in both animal and human models (120-122). LCn-3 are also anti-inflammatory (123), which is recognized to be a contributing factor to atherosclerosis, starting off the cascade of vessel wall intima damage culminating in leukocyte and smooth muscle migration (124). Further down regulatory pathways, protective actions attributed to LCn-3 include decreasing growth factor (125) and adhesion molecules (126-128). Long chain n-3 have also been found to reduce blood pressure (129-131) and as revealed in a recent meta-analysis, an intake of a median dose of 3.7g/day of LCn-3 can reduce systolic and diastolic blood pressure of 1.7 mmHg (95% CI: 0.3, 3.1) and 1.5 mmHg (95% CI: 0.6, 2.3), respectively (132). LCn-3 have also been shown to promote arterial compliance which is also an athero-protective benefit (133). Finally, as demonstrated in a recent study by Erkkila *et al* (2004) fish intake is associated with a reduced progression of coronary artery atherosclerosis (134).

In addition to the beneficial effects of PUFA which are exerted through changes in vascular membrane fatty acid composition and subsequent alterations in hormone signaling, fatty acids exert a direct effect within the heart. For example diets rich in PUFA from both plant (n-6) or marine (n-3) origin reduce the incidence of fatal cardiac arrhythmias after induced simulated myocardial ischemia in rat models

(135-138) and in the marmoset monkey (139). Also, a comparison of the effect of the different PUFA indicates an enhanced antiarrhythmic effect of dietary fish oils (140). Fish oils typically contain 30-40% PUFA, mainly as EPA or DHA, and provide the greatest antiarrhythmic protection (67, 141, 142). Furthermore, due the preferential accumulation of DHA in myocardial cell membranes, its association with arrhythmia prevention and metabolic efficiency and the selective ability of pure DHA to prevent ventricular fibrillation, suggest that DHA is the bioactive component of fish oil in the heart (143). There is also strong evidence to suggest that these effects rely on the incorporation of DHA into the myocardial cell membranes and are dependent upon habitual intake (143). Given that sudden ventricular fibrillation accounts for up to 50% of cardiovascular deaths in developed nations (29) coupled with the fact that prior symptoms often do not occur, prevention may best be approached through community wide nutritional changes (141).

For a putative risk factor to be clinically useful several requirements need to be met (144) and LCn-3 fulfils many of these (9). There is substantial epidemiological data (145). A relationship between membrane EPA + DHA and cardiac death is also biologically plausible which is characterised by the following hypothesised relationship: 1. dietary n-3 intake increases cell membrane and free fatty acid n-3 levels, 2. higher n-3 levels favourably alter cardiac ion channel function, 3. altered ion channel function modifies the cardiac action potential, and 4. alteration in the action potential reduces myocardial vulnerability to ventricular fibrillation, which is the major life threatening arrhythmia that results in sudden cardiac death (67). In addition, LCn-3 may enhance plaque stability as well as being anti-atherosclerotic

via other mechanisms (146, 147). Most importantly secondary prevention trials have shown that by changing a risk factor a disease outcome is altered (109). These results culminate in a strong association between this biomarker and disease which is independent of other risk factors (87) and systematic review evidence concludes that dietary advice to those with CHD can reduce morbidity and mortality (148).

Although these results provide substantial evidence for the LCn-3 in secondary prevention of CHD, the prevention of sudden cardiac death in the community in patients without clinically recognised CHD remains a challenge. Currently there is no primary prevention intervention trial linking fish intake or fish oil and the reduced incidence of CHD and this area is not without conflict. For example, the Cochrane Collaboration published a review on the role of n-3 for the prevention of CVD and concluded that it was not clear that n-3 alter cardiovascular mortality in high risk populations (149). Nevertheless there is enough evidence to support population-wide guidelines about fish consumption (150).

2.5 Summary of dietary risk factors for CHD

What is emerging from the literature is that there are a number of dietary factors which have been implicated in either the promotion or prevention of CHD via two potential mechanisms – atherogenicity and thrombogenicity. Saturated fatty acids (SFA) have been shown to be cholesterol raising (and by inference atherogenic) as well as thrombogenic (30, 151). PUFA of the n-6 series, and PUFA of the n-3 series (152), monounsaturated fatty acids (MUFA), dietary fibre (153, 154) and

antioxidants (155) have demonstrated protective effects. In addition, both clinical and animal-experimental evidence suggests that the effect of n-3 on the risk of sudden cardiac death relates primarily to reduced vulnerability to ventricular fibrillation, rather than to a reduction in atherosclerosis or nonfatal myocardial infarction (63). Doses of 3-5g/d of EPA + DHA have been shown in experimental studies to decrease triglycerides (75, 113-116), decrease inflammation (123) and blood pressure (132), whilst increasing vessel relaxation and increasing erythrocyte deformability (133). However, excessive n-6 intake, through direct competition, has been shown to increase thrombogenicity via the promotion of platelet aggregation (126-128) and decreasing levels of growth factor (125). It is worthy of noting that all of the doses in these studies that have been shown to be effective are significantly higher than what is usually associated with prevention of heart disease, suggesting that it may be the combined, or even synergistic effects of all of these marginal effects that is important, although this is yet to be fully understood. With this basis of scientific evidence a number of authoritative organisations, using expert panels, have published dietary recommendations to guide dietary therapy.

2.6 Dietary recommendations for the primary prevention of CHD

In a recently published review of some 150 studies on the link between diet and cardiovascular health, the authors concluded by suggesting three major approaches as being the most effective in preventing cardiovascular health: 1. replacing SFA and *trans*-fats (solid fats produced artificially by heating liquid vegetable oils and has similar effects to SFA) with MUFA and PUFA; 2. Increasing

consumption of n-3 from either fish or plant sources such as nuts; and 3. eating a diet high in a variety of fruits, vegetables, wholegrain, nuts and avoiding foods with a high glycaemic load (156).

The Adult Treatment Panel (ATPIII) of the National Cholesterol Education Program (NCEP) recommend as part of their Therapeutic Lifestyle Changes for the reduction of CVD risk, a diet which contains 25 - 30% of total calories from fat (< 7% SFA, up to 10% PUFA, and up to 20% MUFA, 50 - 60% of total calories from carbohydrates (CHO), ~ 15% of total energy from protein (PRO), < 200mg cholesterol/day, dietary fibre 20 –30g/day and total energy to be balanced with energy expenditure to maintain desirable body weight or prevent weight gain (14). There is a recommendation to include 2g per day of plant sterols into the diet for those with elevated serum cholesterol (14).

In addition, the International Society for the Study of Fatty Acids and Lipids (ISSFAL) makes the official statement that the consumption of LCn-3 may reduce the risk of CHD (157). More recently the Food and Drug Administration in the US (FDA) has agreed to a qualified health claim for n-3, stating *"supportive but not conclusive research shows that consumption of EPA and DHA omega-3 fatty acids may reduce the risk of coronary heart disease"* (158). Although to date there are no human studies that support the direct effect of LCn-3 and the primary prevention of CHD, these uncertainties have been addressed by a number of secondary prevention randomised controlled trials (RCT). Therefore, based on the balance of evidence ISSFAL supports the use of LCn-3 in the prevention of CHD, and intakes of 800-1000 mg per day appear to be a prudent approach (75). Safety of intakes

up to 3000 mg per day of DHA/EPA has been approved by the FDA and this level in generally regarded as safe (GRAS) in the American food supply (75). Recommendations from the UK and Europe after an experts meeting suggested values of up to 650 mg per day for the general population (75). In fact, recently the American Heart Association recommended that n-3 be consumed as a supplement if the diet contained an insufficient amount of this fat (159). In summary, for a 8360kJ (2000kcal) diet, ISSFAL recommends a daily intake of n-3 fatty acids of 0.65g EPA + DHA and 2.22g ALA, with the n-6:n-3 ratio reduced to substantially less than 10 (157, 160).

2.7 Behaviour change theory in dietary interventions to prevent CHD

The goal of primary prevention is defined as the treatment of risk factors when no clinical manifestations of atherosclerosis are apparent yet (8). Primary prevention of CHD can either be individually oriented involving screening for and treating risk factors (high-risk approach) or it can be directed to the whole population (161). Although the priorities of primary prevention programs are to: 1. educate people about risk factors and lifestyle changes to reduce risk; 2. identify and alter risk factors to prevent the onset of CHD leading to heart attack or stroke, primary prevention strategies in the past have taken on many forms which have resulted in mixed results, therefore undermining the perceived benefits of this approach.

Much progress has been made in recent years in improving the cardiovascular health of Australians. Death rates from CHD have fallen substantially, by around 70% since the late 1960's (29). However, despite this CHD remains Australia's

greatest health concern. As most of the improvements in this rate have been on the impact of cardiac survival (advances in treatment and care) there are huge gains to be obtained through primary health care strategies (28).

Epidemiological observational studies, both prospective and case control, show repeatedly that dietary factors are associated with CHD (79, 162-164), and therefore contribute substantially to the preventable burden of this lifestyle disease. Clinical trials have confirmed many of these associations leading to the notion that people's dietary intake can affect health (68). The scientific consensus is supportive of an approach to improving dietary intake, and as dietary change remains an important modifiable behaviour, it follows that primary prevention intervention is advisable (165). However, non-pharmacological treatments such as smoking cessation, exercise and improved nutritional habits are generally poorly followed and their effectiveness is widely questioned (166, 167).

Nutrition intervention programs focus on foods people eat (168) as well as employing behavioural science theory and methods to understand dietary behaviour and design and evaluate dietary change programs (169). Early behavioural nutrition research used basic learning theory to address issues of how much and when a person chose to eat (169). These interventions included nutritional counselling and adult learning theory in program design.

More recently, nutritionists and nutrition researchers have been exploring behaviour change models and intervention strategies to find ways of having a greater impact on behaviour change (170). The theories most commonly employed

in primary prevention nutrition programs include Social Cognitive Theory (SCT) also sometimes called Self Efficacy Theory and previously called Social Learning Theory; Theory of Planned Behaviour; Health Belief Model (HBM); the Transtheoretical Model (TTM), also called the stages of change; Health Belief Model; and Self Control Theory (170). Population approaches have also been employed using a number of theories from Community Education Theory to Health Education and more recently Social Marketing (170). Each of these will be reviewed briefly.

2.7.1 Nutritional counselling

Nutrition counselling is the process of guiding a client toward a healthy nutrition lifestyle by meeting normal nutritional needs and solving problems that are barriers to change (170). To alter food habit successfully, counsellors work with clients to change behaviour based on a range of psychological, socio-cultural and physiological factors. During the 1980's, the focus was on behaviour modification, giving way to goal setting and client centered counselling in the 1990's (170). There is an array of counselling philosophies, theories and models that are available to deal with the complex field of behaviour change. The range includes Person-centered therapy which involves clients actively participating in clarifying needs and exploring potential solutions (171); Behaviours therapy which is based on the premise that behaviours are learned and therefore new behaviours can be re-conditioned (170); Gestalt therapy which operates in the present and aims for clients to take responsibility for their own actions (172); Cognitive therapy which is based on the premise that negative self-talk and irrational ideas are self-deflating

learned behaviours and most frequently the source of their problems (173); and Family therapy which aims to assist the individuals within the context of the family to change themselves (174).

2.7.2 Adult Learning Theory

Historically, adult learning theory was regarded as an extension of childhood learning theory. Approximately 25 years ago the concept of andragogy was introduced into the field of education (175). Andragogy, “the art and science of helping adults learn”, was contrasted with pedagogy, the art and science of helping children learn (175). Adults can learn from a continuum of approaches from teacher-directed learning to student-directed learning depending on the situation (175).

2.7.3 Theory of Planned Behaviour

The Theory of Planned Behaviour (TPB) provides a model that can help explain behaviour and intention to perform a given behaviour. In turn, a person’s behavioural intention is determined by the attitude they hold towards the behaviour, the degree of social pressure felt by the person to perform or not perform the behaviour as well as the degree of control felt by a person over performing the behaviour (19). These three factors are determined by a number of beliefs and subsequently how each is evaluated. The TPB, or modified versions of it, has been applied to a range of health related behaviours (20-22).

2.7.4 Health Belief Model

This is probably one of the most influential models governing health related interventions. It postulates that cognitive factors influence an individual's decision to make and maintain a specific health behaviour change. The individual's behaviour will change if they believe that they are susceptible to a health concern which will impact on their life, and they are capable of making the necessary behavioural changes to reduce the risk (176). Traditional approaches to dietary management of cholesterol lowering are largely based on this premise.

2.7.5 Transtheoretical Model (TTM)

The TTM (also known as the stages of change model) states that people progress through five distinct motivational stages in attempting to change their behaviour: pre-contemplation, contemplation, preparation, action and maintenance (177). In addition to helping understand behaviour, this model can be used as a counselling guide, as strategies will have varying effectiveness depending on the stage of change the individual is at. In addition this model can be used as a measure of outcome effectiveness. For example, an intervention may be considered effective if a client moved from "I do not need to make a change" to "maybe I should give some thought to change".

2.7.6 Self-Efficacy Theory

Self-efficacy has been used as a stand alone model, as well as incorporated into other behavioural change models. Self-efficacy has been defined as "our personal belief of how capable we are of exercising control over events in our life" (178). As

strong self-efficacy has been found to correlate with the attainment of health behaviour change, it is believed that a person's confidence in ability to accomplish behavioural change is more important than the skill *per se* (179).

2.7.7 Social Cognitive/Learning Theory

Social cognitive theory states that people are neither driven by inner forces nor automatically shaped and controlled by external stimuli. Rather, human functioning is explained in terms of a model of triadic reciprocally in which behaviour, cognitive and other personal factors, and environmental events all operate as interacting determinants of each other (180). Here, it is said, that behaviour change occurs through taking action, observations of others taking action, and evaluation of the results of those actions.

2.7.8 Social Marketing Theory

Kotler *et al* (2002) define social marketing as "the use of marketing principles and techniques to influence a target audience to voluntarily accept, reject, modify or abandon a behaviour for the benefit of individuals, groups or society as a whole" (181). This relies on the voluntary compliance of consumers and often cannot promise a direct benefit for this change. Despite distinctions in scope, social marketing shares many similar techniques with commercial sector marketing.

A review of some of the individual randomised trials and other individual-level intervention studies that have examined the effectiveness of various nutrition interventions will be presented, including those conducted with healthy individuals as well as at risk individuals. Also, a review of interventions focusing on both

community groups (e.g. family, providers, worksites, point-of-purchase) and entire communities will be presented. These studies will be presented with reference to the theoretical model underpinning the intervention and discussed in relation to the outcome achieved.

2.8 Individual-level dietary intervention studies

A large number of individual-randomised studies have been conducted to test the efficacy on outcome measures such as food based outcomes (e.g. fish intake or fruit and vegetable intake), dietary goal based outcomes (e.g. percent fat intake) and CHD biomarker outcomes (e.g. serum cholesterol levels).

The 1990's saw a large number of individual interventions aiming to increase fruits and vegetables reporting largely positive results. Significant increases in fruit and vegetable (F/V) intake were reported in an individual case study using behavioural therapy over a relatively short time frame of 5 months (182) as well as in a 6 month intervention with 33 women with low F/V intake (183). Lutz *et al* (1999) conducted a 6 month intervention using 710 males and females using interventions based on social cognitive theory and transtheoretical model with the aim of increasing F/V intake (184). Subjects were randomised to receive regular newsletters, tailored messages or goal setting advice at monthly intervals. Significant increases in F/V intake in all intervention groups was achieved (184). Similar results have been reported using telephone calls (185), nutritional counselling (186), coupons along with educational programs (187), messages about change (188), and multiple mailed contacts which resulted in significant decreases in fat scores (an indirect

measure of fat intake) (189). Another personalised health promotion program using targeted counselling demonstrated improvements in diet scores over a 2 year period, but not 5 years (190). In the Netherlands, significant increases in fish consumption were achieved in both group sessions and tailored letters after a 16 week intervention (191).

Similarly there have been a number of studies which have reported similar successful outcomes in reducing both total and saturated fat intake. The MRFIT trial was a randomised intervention of 12,866 males at high risk of CHD who received nutrition counselling to reduce total fat, saturated fat and low fat foods or control and were followed up for an average of 6 years. The intervention group achieved significantly greater decreases in all outcome measures (192). Large reductions in fat consumption in relation to controls have been reported by Boyd *et al* (1997) after a two year period of nutrition counselling of 220 premenopausal women (193), by Coates *et al* (1999) after 18 months provision of group education to 2208 women (194) and in trials on men undergoing nutritional counselling (195) and a prescription of dietary plans (196). Another trial found that dietitian-led interventions produced significant reductions in dietary fat and cholesterol intake compared with material and videotape intervention groups (197). While these studies predominately used nutrition counselling as a theoretical underpinning, White *et al* (1994) investigated the effect of using a program based in cognitive theory (198). Using group sessions with self monitoring tools in conjunction with nutritional and behavioural strategies they were able to demonstrate significant,

large reductions in fat intake, which was maintained for at least one year after intervention in 1050 women aged between 45-69 years (198).

Several studies have targeted individuals with risk factors for chronic disease. The Oslo Study found that regular dietary counselling produced significant long-term declines in serum cholesterol compared to control participants (199). However, a community-wide screening and follow-up project in a low-socioeconomic area produced significant changes in diet following group education compared with pamphlet intervention, but no difference in serum lipids or CHD risk (200).

Metabolic ward studies have shown that dietary changes can reduce blood total cholesterol concentration by 10-15% in settings with very strict dietary control (201-204). However these results should be used with caution when extrapolating to free-living populations. For example, Denke *et al* (1995) showed that dietary counselling could achieve cholesterol reductions of up to 10%, and therefore would prove value as a clinical treatment option (205). However, a study published in 1991 suggested that dietary advice to individuals produced small reductions in total cholesterol concentration (less than 4%) rendering it of little value in clinical management (206). Tang *et al* (1998) published a systematic review of 19 randomised controlled dietary intervention trials under free-living conditions aimed at lowering total cholesterol (12). The overall weighted mean reduction in blood cholesterol across all dietary comparisons was only 5.3%, using trials of at least six months duration (12).

A meta-analysis of randomised controlled trials of dietary interventions of at least 3-months concluded that dietary interventions in primary prevention can achieve only modest improvements in diet and cardiovascular disease risk status that are maintained for 9 – 18 months (207). For example, after 3 – 6 months dietary fat as a percentage of energy reduced by -2.5% (CI - -3.9%, -1.1%) in the intervention groups and serum cholesterol, -0.22 (CI = -0.39,-0.05) mmol/L in the intervention groups (207). Dietitians are specifically trained and motivated to provide high-quality dietary advice. Having a battery of approaches available for individual clients, dietitians appear to achieve better outcomes than doctors at lowering cholesterol in the short to medium term, though clinically the difference is small, and there is no evidence to suggest that this strategy is better than self-help resources or nurses (208).

2.9 Community-level dietary intervention studies

Community level interventions using a range of theoretical models such as SLT (209), TTM (210-212), participatory strategies model (213), community organisation (214, 215), socio-ecological (216), social marketing theory (217) and a multitude of models (218) appear successful in improving fruit and vegetable intakes as well as the selection of low fat products. Some studies, although not stating the theoretical underpinning of the study, but using similar techniques such as counselling, print material, advertising and food policy changes, have reported similar positive effects of interventions (219-224), although others using similar approaches and theoretical behaviour change models have reported little or no effect (225-232). Also, a large number of interventions designed to reduce total fat

and saturated fat intake as a percent of energy have all achieved positive outcomes using TTM and Educational theory (233-235) and without an underlying intervention strategy specified (236-239).

Now turning attention to population interventions targeting a reduction of serum cholesterol, of the 6 published studies, half showed a positive effect whereas the other half showed no significant impact of the intervention. In 1994, Luepker *et al* used an intervention based on SLT employing mass media, health professional training, screening and education across three separate communities and found no sustained differential change in total cholesterol (240). Two subsequent interventions using nutrition education and training in counselling found a similar result (241, 242). However, positive effects have been reported in interventions using mass media campaigns in 5 cities (243), community counselling and screening (244) and food policy changes along with educational programs in a contained community of a Kibbutz in Israel (245).

Several community interventions aimed at reducing CHD have not achieved desired outcomes that would be predicted from the individual dietary behaviour change interventions. In the Malmö preventive project (2000) between 1974 and 1992, a total of 22 444 men and 10 902 women attended screening. Around 25% of the screened subjects underwent various interventions (lifestyle modification and drug therapy) aimed at treatment of hypertension, hyperlipidaemia, IGT etc (161). The behavioural approach was an individually oriented, high-risk approach with resources for both screening and intervention. Although positive effects on risk factors such as hyperlipidaemia were found, no effect of intervention on mortality

and cardiovascular morbidity in the intervention group as a whole was reported (246, 247). The authors concluded that risk factor screening and intervention in an urban setting such as the one in Malmö, and the use of contemporary methods of the trial, are not enough to reduce all cause mortality more than implied by the population trends. They did note that it is important to consider the heterogeneity in the determinants of disease, as people living in more socially deprived areas have more risk factors and a higher incidence of CHD than found in the less deprived areas (161). In a similar trial in another part of Sweden, middle aged men were screened and treated for cardiovascular risk factors (248). However, no overall beneficial effect could be detected in total or CHD mortality in comparison with a predetermined control group. The MARGARIN study (2000) investigated the impact of an intensive group education on the Mediterranean diet and after one-year of intervention a posted leaflet with dietary guidelines was provided (249). Beneficial changes in dietary habits were recorded in the intervention group, however, saturated fat and total fat was still too high. Of the 262 hypercholesterolaemic persons with at least 2 other cardiovascular risk factors, the intensive program of dietary education did not significantly lower cholesterol (-3%) more that the control (-2%) and after 1 year both groups gained ~1% body weight.

2.10 Limitations of primary prevention strategies

With this background in mind it reinforces the fact that changing life-long behaviour is a complex process, and the results are often disappointing. Despite the increasing knowledge about the pathophysiological processes and resultant risk factors of CHD and subsequent awareness and education about nutrition and

exercise, CHD remains our number one lifestyle disease. Evidence from lifestyle trials, in which both individual and population based preventative strategies have been successful is limited (161). With the absence of disease symptoms it may be difficult for individuals to change and adhere to a prescribed treatment. A large gap exists between research and practice in the effective implementation of validated lifestyle primary health initiatives to reduce the incidence of CHD.

Prevention of CHD represents a great challenge with the potential to reduce a considerable portion of the global burden of this disease (250). Results obtained from a number of trials (248, 251) lead us to believe that a hospital-based prevention program alone is not effective in reducing the incidence of CHD, nor is individual screening due to the vast numbers of at-risk individuals and lack of long term resources (251). Therefore an individual approach is encouraged to be combined with population-based approaches (161). These programs are required to be based on evidence, noting that evaluation of larger scale community projects will always be much more difficult than evaluation of individually targeted projects.

This raises the question about why primary prevention strategies fail. Possibly there is a lack of compliance thus rendering interventions ineffective. In part this can be attributed to the lack of knowledge of the psychological and social mechanisms involved in changing attitudes and behaviours, not only of individuals but of the entire population (161). Similar concerns have been raised by others, and in part, this was the reason why a long term primary prevention trial was abandoned because of the poor ability of free-living subjects to adhere to long-term dietary recommendations to reduce saturated fat, highlighting the perceived

difficulty of achieving dietary change (161). In addition the resource burden of primary prevention strategies may be too great for patients, clinicians and the health care system (252). Finally, lifestyle trials have traditionally been narrow in their scope. They rely on singular contexts (individual or community) are based on singular theoretical models (TTM, SCM, social marketing) and rarely consider the wider political or environmental factors. To this end, the World Health Organisation (WHO), the National Institute for Heart , Lung and Blood Disease (NHBLI), as well as other organisations have all recommended radical changes in diet and lifestyle interventions (161). The authors recognise that population-based interventions need the development of a new methodology, not least within the social and behavioural processes involved in preventive efforts in the population. Thus a new intervention paradigm is required that helps individuals overcome the natural aversion to dietary change and one that is going to be supported by a large portion of the population.

2.11 A new paradigm for the primary prevention of CHD

Mortality from several causes has been reduced through prevention programs that changed population behaviours (7). Examples in Australia and New Zealand are smoking related diseases (253), road deaths (254), lung cancer (255), cervix cancer (256) and sudden infant death syndrome (SIDS) (257). Mortality rates from CVD provide a mixed picture. Mortality rates peaked in 1968 and have since fallen by over 60%, and this is consistent in industrialised countries (29). However, it has been reported that more than 70% of this overall decline in mortality has occurred amongst patients with existing coronary heart disease (258). Evidence suggests

that emergency care, medical (259) and surgical treatment and follow-up care (58, 260, 261) have influenced this rate due to the survival of cardiac events which underscores the importance of secondary prevention (15). However, it is much more difficult to detect a positive preventative effect of interventions directed toward relatively healthy populations (192, 240, 248, 262).

Of the range of theoretical frameworks in common usage in health promotion for influencing population behaviours and disease end-points the ‘epidemiological triad’ has been widely used in addressing both communicable and non-communicable epidemics and only recently has it been applied to lifestyle diseases (7). Figure 2-1 shows how it relates to CHD specifically (7). The agent (the final common pathway) for CHD is an imbalance of dietary fatty acids. Host factors are a combination of non-modifiable, such as, genetic factors, age, gender, whereas others such as beliefs, attitudes and behaviours are modifiable.

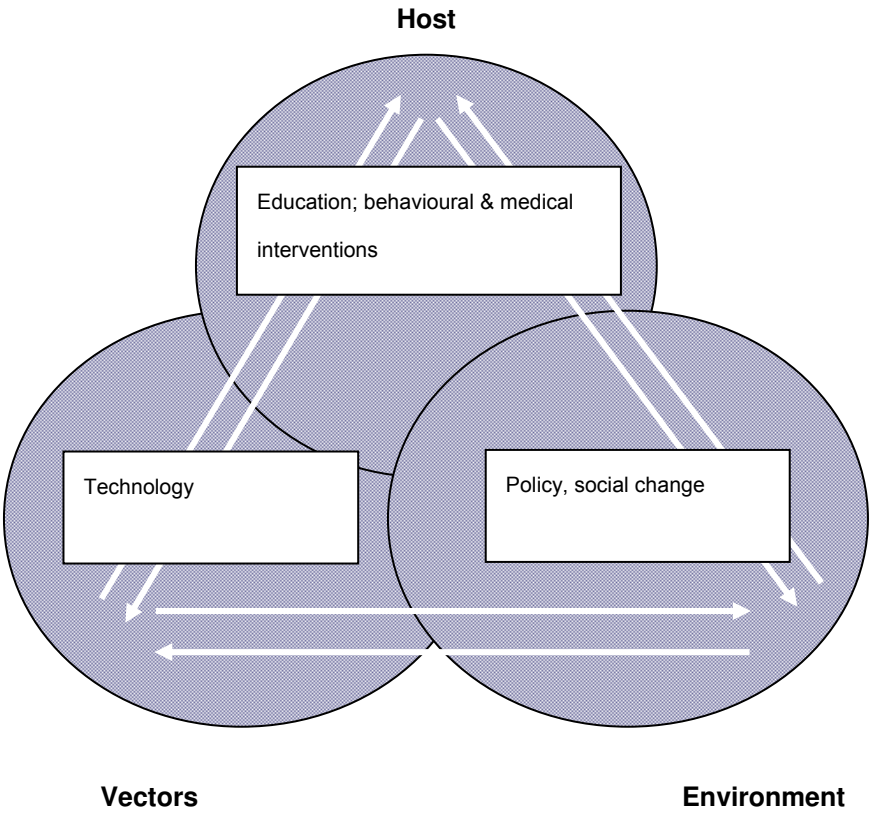

Figure 2-1 The epidemiological triad as it applies to cardiovascular disease (the 'agent' is dietary fatty acid imbalance) (6)

Behaviours are a complex interaction of psychological factors, including habits, emotions, beliefs and attitudes developed through a background of learning history (263). The vectors for a poor dietary profile are high saturated fat foods (264) and foods low in n-3 (265). The environment is external to the individuals and provide a structure within which people live and is considered a powerful determinant of behaviours (7).

Host-related activities to date have been the focus of primary prevention strategies, and tend to be education based (individuals or group education) or medical

(pharmaceutical). Technological innovations such as novel food enriched with bioactive ingredients or modified in fat content provide vector-related solutions. These novel foods are commonly referred to as functional foods. A functional food is defined as *"… any modified food or food ingredient that may provide a health benefit beyond that conferred by traditional nutrients the food contains"* (266) or *"foods with health benefits beyond basic nutrition"* (267). In this instance, vector-related solutions could be physical (making functional foods widely available), economic (making the costs of functional foods affordable), policy (altering rules related to the promotion of these foods i.e. health claims) and sociocultural (influencing attitudes, beliefs and perceptions). Whilst there is overlap between the different sectors of the triad, interventions historically focus on one corner.

What has been learned from ameliorating other epidemics is that population behaviour change can be achieved knowing that: knowledge itself is a weak determinant of behaviour but well communicated, action specific messages influence behaviours; television-based communications are effective but expensive; and environmental strategies are powerful. All elements of the triad must be addressed simultaneously. In the case of CHD, vector innovations such as low fat food were the first phase; now food innovations which include bioactive ingredients provide new opportunities. Social attitudes are influenced and shaped by policy changes (again nutrition claims and fat claims provided first phase opportunities, now health claims legislation changes offer new opportunities); here advocacy from powerful commercial groups often influence policy. Finally, the

normalisation of consumers' behaviours (e.g. acceptance of eating novel foods enriched with fish oil) is powerful in sustaining long term behaviour changes.

2.11.1 Vector-based innovations

There has been an incremental increase in research into the bioavailability and application of functional foods and food ingredients (268). Interest in functional foods is increasing because of higher health care cost and recent scientific discoveries linking dietary habits with the development of many diseases (269). Functional foods have been designed with health improvement in mind.

Dietary guidelines are pointing nutrition educators to two priorities in the prevention of CHD, namely promoting dietary habits that reduce serum cholesterol as well as increasing their dietary LCn-3 intake. However, it has been widely acknowledged that individuals find it difficult to adhere to stringent dietary goals for elevated cholesterol (12) and the there is an inability of the population at large to consume enough LCn-3 from fish alone (18). Thus the combined efforts of food technologists, scientists, nutritionists and food industry have led to the development of a number of functional foods, of particular note products enriched with plant sterol/stanol and LCn-3.

Since the 1950's plant sterols were found to have cholesterol-lowering properties. The major plant sterols in the diet are β-sitosterol, campesterol and stigmasterol. Research over the past decade has focused on the esterified form of plant sterols which is added to food products - for reasons of effectiveness and safety. Esterification renders plant sterols soluble in dietary fat which is the most effective

vehicle to deliver sterols to the small intestine where cholesterol absorption occurs (270). Plant sterols reduce the absorption of both dietary and biliary cholesterol from the intestinal tract by 30-50% (271). The exact mechanism is yet to be elucidated, however it is generally assumed that the presence of increased quantities of plant sterols in the gut lowers the micellar solubility of cholesterol thereby, lowering the amount of cholesterol available for absorption. Recent advancements in food technology have seen the emergence of food products such as margarine, milk, yoghurt and cereal products being enriched with plant sterols/stanols and promoted as a food which can help lower serum cholesterol.

Another food innovation has been the development of LCn-3 enriched foods such as margarine, bread, eggs and yoghurt. This has been made possible by the advent of microencapsulation which allows for LCn-3 to be added to a range of food stuffs (272). Microencapsulation is the process whereby small particles of oil are encased in a secondary material (encapsulent) which is usually a complex carbohydrate compound (272). This increases the shelf life of the oil as it reduces exposure to the environment which would oxidise the n-3 and eliminates the ‘fishy’ taste that is accompanied by LCn-3 (272, 273). This has the net effect of protecting the organic structure throughout all phases of the processing and in addition to ensuring that taste and texture is not affected (272, 273). Both the bioavailability and taste is not affected by oil that is microencapsulated (272-274).

There is little doubt that these two food types are a triumph of food technology, however their effectiveness in promoting health is yet to be confirmed. There is therefore an opportunity to use these recent advances in food technology and the

development of functional foods (Vector) and test them against the elements of the theoretical model. To demonstrate the effectiveness of these products in a functional food-led intervention, an examination of both the biological and psychological effects of these foods (Host) is required along with the consideration of the wider social context (Environment).

2.11.2 Host-based considerations

Clinical studies on the effects of spreads enriched with plant-sterol-esters or plant-stanol-esters (hydrogenated form of sterols) have consistently shown reductions in serum total and LDL-cholesterol, with little or no change in serum HDL-cholesterol or triglyceride. In controlled situations, plant sterol intake of up to 2g per day - added to products such as margarine, oils, spreads and milk - have been shown to reduce serum cholesterol by up to 15% (270, 275-278). The cholesterol lowering effects of plant sterols is independent of, and additive to, the effects of dietary manipulation and cholesterol lowering medication. A recent study showed that the reduction in LDL-cholesterol by 10% achieved by a low-fat diet was enhanced by a further 12% when plant sterols were added to the diet through salad dressing (279). Since the hypercholesterolaemic action of sterols is mechanistically different from the statin class of drugs, it might be expected that the effects are also additive to those of drug therapy, however one study comparing the efficacy of sterol-enriched spreads between subjects with and without cholesterol lowering medication revealed that the relative fall in LDL-cholesterol was the same in both groups (280). Despite the favourable results reported so far, all studies to date have been in controlled settings where subjects have been supplied with sterol

containing products. Translation of these efficacy trials into the clinical setting has been slow. One reason is that evidence is still required from studies investigating subjects who assemble the diets for themselves on a routine basis, thus determining the true applicability of this strategy in the clinical setting (281).

Despite the body of evidence suggesting the importance of LCn-3, dietary goals are difficult to achieve through the consumption from fish alone (18). Although there have been a wide range of foods enriched with this essential fatty acid, only a few studies have investigated their efficacy. A study by Mantzioris *et al* (2000) investigated the effectiveness of a diet that incorporated foods rich in n-3 fatty acids (282). Healthy male volunteers were supplied with foods enriched with α–linolenic acid (cooking oil, margarine, salad dressing, and mayonnaise) and EPA and DHA (sausages and savory dip) as well as foods naturally high in n-3 (flaxseed and fish). Intake averaged 9.0 g/day of ALA and 1.8 g/day of EPA and DHA which was confirmed by an increase in plasma, platelet and monocellular phospholipids (282). However, this was a pre test – post test study, with 15 subjects over 4 weeks (282). Metcalf *et al* (2003) took this concept one step further and tested the effects of providing a wide range of foodstuffs containing n-3 from fortification (283). Foods enriched with LCn-3 included margarine spread, milk, sausages, luncheon meat and French onion dip was provided to 16 subjects. Again this was a before-after study for duration of 4-weeks. The consumption of ALA increased from 1.4 to 4.1 g/day and EPA and DHA increased from 1.2 to 1.52 g/day which was confirmed by corresponding increases in serum and platelets (283). There has been a suggestion by these authors and others that a range of novel foods

provides the opportunity for wider public consumption of LCn-3 (282, 283), but this may be a little premature. Although Mantzioris *et al* reported positive effects on monocyte-derived eicosanoids and cytokine production, the design of these studies require caution in interpretation of these results. The short time frame limits the assertion that these foods could *"support large-scale programs on the basis of the therapeutic and disease-preventive effects of n-3 fatty acids"*(282). Finally, the effects of these foods on the composition of the total diet as well as other biomarkers of CHD warrant attention.

A general principle in relation to vectors is that small changes in high volume vectors can have significant population impacts (7). However at the core of this principle is the assumption that a modified vector will be widely accepted by consumers as well as being accessible in terms of location and cost. In this case there is an assumption that these foods will be incorporated into the diet such that the other dietary considerations are not compromised. Therefore for this class of functional food to support large scale programs for the primary prevention of CHD an understanding of consumer acceptance is fundamentally important. Factors that promote or constrain the use of these foods need to be understood. The unveiling of psychosocial variables can be used to maximise interventions, predict the potential impact of an intervention and help guide future interventions. Whilst there have been a few studies that have investigated consumer views towards functional foods in general, (284-288) there are no studies that have investigated the factors that prevent or promote the use of LCn-3 functional foods in the wider community.

2.11.3 Environment – based considerations

To date the food industry has responded to public health guidelines and has contributed to influencing the food supply (Vectors) in several ways. For example, in response to a rapid rise in CHD there was a national call in Finland for the Government to intervene (289). The results were a removal of saturated fat from the food supply or its replacement with unsaturated fats. The results were exciting; with mean cholesterol levels in Finland dropping by 15% along with rates of CHD by about 75% of their peak levels (289). In many westernised countries farmers and processors have responded more recently by producing leaner meat and dairy whilst manufacturers have provided a number of foods substantially reduced in harmful components. At the heart of this issue is the ability of companies to gain a marketing advantage from nutritionally enhanced foods and nutrition and health claims are one way to achieve this (11, 26). Countries such as the US, Canada, UK, Europe, Japan have established processes for the management of health claims. In contrast Australia has been slower in adopting this policy, with the Health ministers of the Australian New Zealand Food Standards Council (ANZFSC) agreeing in May 2002 and again in December 2003 to set policy principles for a new regulatory system that will allow the use of health claims within a risk management framework (290).

Health claims bring with them the concerns about the level of honesty and accuracy of claims and label information generally (291). Examples show us that the regulators are often behind the marketers in this regard (292, 293). In Australia, where health claims are currently prohibited with one exception, a study found that

7.4% of food and drink advertisements contained illegal health claims (26). In addition, a major threat to the effective use of functional foods is the exaggerated and often contradictory health claims observed in Europe, Asia and the US. For example, only 1% of Japan's functional foods are directed at the significant health problems such as high blood pressure, with adequate scientific justification (294). In part the problems relate to the strength of the evidence base for these claimed benefits which range from very strong, based on multiple clinical trials, to the merely suggestive, relying on epidemiological or *in vitro* studies (295). A strong scientific base is a necessary requirement for any health claims about functional foods (295). Furthermore consideration needs to be given to the ethical and social consequences of their introduction into the food supply (296). For example, who has access to these foods and what will be the cost to the consumer? Thus, careful consideration is required about our future regulatory frameworks to ensure that consumers are not misled about the potential benefits of functional foods. McConnon *et al* suggests that in order for functional foods to reach their maximum potential the food industry, government and health professionals must work together to improve communication between themselves and consumers (25). An initial step in this process is the clear articulation of the beliefs and attitudes of these stakeholders and the identification of incongruence which may ultimately affect this communication process.

In summary, in this thesis the effectiveness of a functional food-led intervention will be measured against each component of the epidemiological triad model. Two dietary interventions, behavioural studies using both qualitative and quantitative

methods and a stakeholder analysis were conducted and the methodological considerations will now be discussed.

CHAPTER 3

METHODOLOGY

3.1 The concept of 'evidence' in relation to primary prevention interventions

Despite the postulation that functional foods can be used to improve health, it is highly unlikely that these novel foods alone will be the solution. For functional foods to be effective they must be placed within the context of current primary health intervention frameworks. Thus an important conceptual issue concerns the type of scientific evidence required to substantiate the effects of primary prevention strategies (297).

Traditionally, in evidenced based medicine there has been an emphasis on a hierarchy of evidence to rank recommendations according to the available type and amount of evidence, of which the randomised controlled trial carries the greatest weight (298). There is little doubt that the push by medical societies and federal agencies to ensure practice was governed by this type of evidence in clinical and pharmaceutical research has improved the quality of clinical practice. Unfortunately this has motivated medical and nutrition societies to try to apply this scheme to all research questions in the medical field, including public health.

A critical evaluation of these hierarchies of evidence and their application is required. Because they were originally developed out of a specific type of research question – mainly the evaluation of therapeutic treatments – their utility in the evaluation of primary prevention strategies is questioned (297). The hierarchies are

based on the ranking of studies according to their susceptibility to bias (297). This means that hierarchy refers to the internal validity of the study designs. Although internal validity is important in terms of experimental design, its external validity is the relevance to the research question (297). For example, a randomised controlled trial can be designed to test the effects of eating 10 tubs of yoghurt a day on health, but obviously very few people would actually eat this. There is thus a trade off between vulnerability to bias and external validity (297). The question here is whether existing schemes for levels of evidence and grading recommendations are appropriate for recommendations /guidelines dealing with behavioural lifestyle modifications. To answer this question we firstly have to differentiate between studies on therapeutic effectiveness or clinical preventive measures on the one hand and observational studies on the relationship between lifestyle and disease on the other (297).

A clinical preventive measure such as a vaccine or a clinical treatment or the prescription of a pharmaceutical has to be applied before the treatment can be evaluated. Therefore, assessment via an experimental study is appropriate. In contrast, primary behaviour change interventions usually advise people how to modify their behaviour (such as eating low fat foods or more fish or abstaining from alcohol) within a common range of behaviours. For example, the recommendation to stop smoking for lung cancer prevention was implemented without the support of a RCT. Because lifestyle behaviours are observable in populations it suggests that the assessment of the overall effect of specific patterns can often be obtained from observational studies (297). In addition, there are significant barriers if an

experimental design were required to prove insights from observation. For example, apart from ethical barriers of exposing some individuals to an adverse behaviour (e.g. smoking) some studies take years if not decades to complete because of the latency periods of many of these lifestyle diseases. There is also an inherent impossibility of blinding of many nutrition or other lifestyle interventions. Finally, dietary interventions that may involve the increase of one food group (e.g. fish) will inevitably result in the decrease of another, for example red meat. Thus the effects of the whole diet are important and often it is difficult to disentangle whether benefits are due to an increase in one food or the corresponding decrease in another.

Reliance solely on RCTs is both not feasible and not able to provide adequate answers for the effectiveness of dietary primary prevention interventions. Recently levels of evidence have been published which take into account that different medical areas require a different set of levels of evidence (299). However this approach relies heavily on Cohort design studies. Another approach to cope with the uncertainty in evaluating observational study results on disease relationships is the application of causal criteria that includes aspects such as consistency, temporality, biological gradient, biological plausibility and coherence (300). It has been suggested that contemporary approaches to evaluation of evidence are based on systematic summaries of all available data on a given topic rather than on single studies or results from single study types (297). In addition innovative designs, models and methodologies are needed for each research setting including consumer research (301).

In summary, RCTs are not available to establish the effectiveness of most primary prevention interventions, but still an answer is needed and recommendations are required. In order to assess the effectiveness of a functional foods-led intervention as a primary prevention strategy a different set of research questions need to be answered and therefore different types of study design are required. A paradigm shift from the traditional hierarchy of evidence to an application of causal criteria is required for guiding public health interventions.

This research drew from medical, dietetic, psychological and sociological methodologies to answer the antecedent hypotheses. Firstly, in order to evaluate the effectiveness of functional foods, trial design resembled ’free-living’ conditions as this is the context where these products will exert their potential health benefits. It was therefore important in evaluating effectiveness to design trials as close to free-living conditions as practical. Secondly, dietary assessment approaches needed to quantify not only the functional nutrient of interest, but also the total diet, and here attention to dietary methodology was given. Also, in order to ascertain consumer beliefs and attitudes toward functional foods as well as determinants of behaviour, both a qualitative and quantitative approach were applied to fully understand the reaction of consumers. Finally, to understand the wider political environment, qualitative methodology provided useful tools in investigating stakeholder views. Each of these approaches will be considered in turn.

3.2 Dietary interventions

Generally, evidence-based dietary interventions are hampered by 3 factors: a lack of nutritional intervention studies; the difficulty of translating nutritional interventions into practice; and the difficulty of translating interventions into a primary prevention setting (302). Compared with research into the efficacy and safety of pharmaceuticals, which is based on hard clinical endpoints, the study of food and mixed diets is more complex. In fact, due to the heterogeneity of the population and the different amounts and compositions of foods used by different populations, objective definitive trials are almost unfeasible (303). Providing proof of effectiveness is difficult due to the delay between the application of an intervention and the observation of its effects, which may be many years (303).Therefore upstream pathophysiological (304) and nutritional indicators (73, 305) may be used as surrogate endpoints.

While evidence of the efficacy of dietary components are required from clinical trials, it is equally important that evidence of the effectiveness from research in practice environments is obtained, since the applicability of controlled studies to general community settings needs to be confirmed (10). It is often believed that the positive results obtained in controlled trials can be extrapolated into a free-living context, as it is assumed that efficacy equates to effectiveness. However, efficacy aims to establish a relationship between a bioactive component and therapeutic benefit, whereas effectiveness also establishes this relationship whilst considering the question of compliance. In order to determine the effectiveness of functional foods, behavioural issues relating to the belief in the benefit of use, taste,

preference and price need to be investigated as they can ultimately affect compliance (20, 22). In addition, the net effect on the whole diet needs to be considered given the importance of other dietary aspects involved in reducing CHD risk (306).

3.2.1 Trial design

Historically dietary interventions have been evaluated on the basis of a hierarchy of evidence, such as that of the National Health & Medical Research Council (NH&MRC) (298). Because of the unique ability of randomised controlled trials to control for confounders, which are either known or unknown, they provide the best evidence of efficacy for interventions. Traditionally clinical nutrition practice has used results to from RCT research to develop health promotion strategies. However, this has not always led to the outcomes that have been promised. In part this is due to the assumption that efficacy equates to effectiveness. This is an important distinction if we are to determine the effectiveness of interventions for diseases with a multitude of aetiologies. For example, confounders such as compliance and the total diet profile can markedly reduce the predicted viability of an intervention under investigation.

It is for these reasons that the dietary intervention studies that were used in this research were designed to resemble as close to a free-living context as possible. The first dietary trial investigated the effectiveness of plant sterol prescription within a clinical context (Chapter 4). As previously summarised, although there were numerous publications of the efficacy of plant sterols/stanols in reducing serum

cholesterol and LDL, no studies had investigated effectiveness in a free-living context where subjects had to purchase and consume products containing this functional ingredient. The design used an existing outpatient clinic to recruit, manage and follow up subjects. All outcome measures were those obtained through routine practice (i.e. blood chemistry was collected by accredited laboratories at the direction of the medical practitioner) which would be used as markers for clinical decision making. The second dietary trial (Chapter 5) investigated the effectiveness of LCn-3 enriched foods on biomarkers of cardiovascular health. Within the study design considerations ensured that subjects were free to incorporate these foods into their diet at will. There was no specific counselling other than a brief instruction on how much of the products they should consume. Also, to increase the power of this study, a variety of prototype n-3 enriched food products were provided free to the subjects as additional foods not currently available on the market.

Despite the very best of intentions to design trials that mimic real life situations there will always be limitations. For example, recruitment bias, where certain individuals are more likely to participate in trials because of a personality trait can lead to an overestimation of compliance and ultimate effectiveness of a treatment. In the fish oil trial, food products were provided and as a result real life barriers such as cost, access and availability of these foods were removed.

3.2.2 Dietary recording methods

In order to address secondary questions about the adherence to intervention goals, and about the differences in observed outcomes between groups in trials, the accurate assessment of diet is important (301). To do this there is a reliance on dietary assessment methods such as the DH and FR. The DH is considered to be the benchmark method used to assess dietary intakes (307). The DH is designed to estimate subjects' usual intake over a specified period of time, usually 3-months (308). It has an established place as a dietary assessment method and lends itself well to clinical practice (309). The DH method is also valuable in intervention trials where dietary manipulation is required at the time of assessment (310). Furthermore, the DH method has demonstrated its ability to produce reliable group means for energy and macronutrients (309, 311). The FR on the other hand has a greater precision in determining food amounts, is cheaper to administer and has less reliance on memory (312).

It is common practice to validate methods for measuring intakes of a particular nutrient on the basis of a positive correlation between individuals using different dietary assessment techniques (313-315). However, measurement errors associated with multiple assessments are most likely to also be correlated, making an accurate assessment of nutrient exposure problematic (301). For example, limitations in the DH method include interviewer bias, inaccuracy of portion size reporting, errors in reporting of frequency, and a need for subjects to follow regular eating habits (316). In contrast, FRs tend to underreport dietary intake, are a poor representation of usual diet due to the limited number of days recorded, and distort

food habits due to the recording process (317). Despite these obvious limitations, the use of multiple dietary assessment measures, combined with the use of biomarkers allow for comprehensive assessment of dietary intake that is less likely to be skewed by common biases or errors.

With error in self-report of dietary intake being the single greatest impediment to understanding the effect of diet on disease risk, the reliance on biomarkers has received recent attention (301). The Women's' Health Initiative program in the US was designed to demonstrate the feasibility of collecting a comprehensive panel of biological markers of dietary intake (318). That study reviewed biomarkers for energy (doubly labelled water), protein (24-hour urine collections) and carbohydrate (plasma vaccenic acid). The authors propose that fat (and alcohol) intake should be determined by subtracting protein and carbohydrate (as measured by biomarkers) from the estimation of energy (318). Our primary dietary outcome of interest was n-3 intake, and as such a biomarker for this fatty acid class was required.

Biochemical validations make it possible to compare assessed dietary intake of some nutrients against biochemical markers (319). In the case of fatty acid compounds, demonstrated correlations between dietary intake and erythrocyte cell membrane concentrations have been found, and provide a useful biomarker which was used in the fish oil study (Chapter 5) (319-321). However, It is important to be aware that the biochemical marker method itself is prone to error with results often confounded by the fact that they reflect relative percentage and not absolute

amounts as well as different methods of collection, sampling site and analytical technique used (322).

In the sterol/stanol study no biomarkers were used to validate dietary data or determine plant sterol/stanol intake. This obvious limitation was a trade off with the experimental design which was to replicate real-life clinical conditions.

3.2.3 Sample

Two trials were used to look at the important primary prevention strategies aimed at reducing the prevalence of CHD, namely cholesterol reduction in those with hypercholesterolemia and increasing LCn-3 in low fish eaters. For the plant sterol study, those identified as 'at risk' groups but with no overt cardiovascular comorbidities provided the basis for investigation. Samples have been drawn from patients identified with hypercholesterolaemia (TC ≥ 5.50 mmol/L) by General Practitioners. Those who were of non-English speaking background, taking lipid lowering medication for less than 3-months prior to referral or had changed dosage within the last 3-month period were excluded, along with subjects following dietary regimens for allergy or intolerance, renal disease and diabetes mellitus.

For the fish oil study, low fish eaters who had a BMI ≥ 25 kg/m^2 and plasma triglycerides > 1.6 mmol/L were included. Although healthy subjects were selected, a criterion of elevated triglycerides was included as a triglyceride lowering effect of LCn-3 is considered as a potentially important cardiovascular biomarker. Potential subjects with a diagnosis of diabetes, recent symptomatic heart disease; angina pectoris; history of myocardial infarction or stroke; peripheral vascular disease;

major surgery within the last 3-months; BP >170/100; liver or renal disease (plasma creatinine >120 mmol/L); regular non-steroidal anti-inflammatory drug therapy; antihypertensive drugs, lipid lowering or other drugs affecting lipid metabolism; those eating more than one fish meal per week or regularly taking fish oil supplements; or an inability to consume the test foods were excluded. Both of these samples represent sub-sets of the population which have well established risk factors for CHD for whom targeted primary prevention strategies are advocated.

3.3 Consumer research

In this thesis the primary interest in consumer research was to predict behaviour in relation to the use of functional foods. Qualitative research methods have long been used in the social sciences and are the principle methods used by researchers interested in studying behaviours as well as customs. They are particularly useful in answering complex questions confronting health researchers and health care practitioners. The term “qualitative research” refers to specific research techniques used to gather data about the social world (such as questionnaires in survey research or focus groups) (323). Although qualitative research may appear neither unified nor well defined, it is often characterised by three distinguishing features. Firstly, qualitative research generally deals with talk text or words rather than numbers, which does not preclude measurement, or that it cannot be explanatory. A second feature is that it studies people in their natural setting, rather than an experimental situation. This has particular relevance to this research given the importance of establishing evidence in a ‘real life’ context. And

finally, the use of a “multi-method” approach where questions are asked of social phenomena from a range of perspectives is also a distinguishing feature. In a health behaviour context, such as the use of functional foods, we have therefore added a range of qualitative research methods to address the important questions about this social phenomenon.

A frequent criticism is that qualitative methods are necessarily subjective (and, therefore biased) and that such research is difficult to replicate and amounts to little more than anecdote, personal impression or conjecture (323). This concern can be allayed by ensuring that these techniques are applied appropriately and interpreted cautiously (324).This research uses qualitative techniques to play a role in both validating quantitative research findings as well as providing a alternate perspective on the same social phenomena. So instead of viewing quantitative and qualitative approaches as methodological opposites, each can be used to complement the other.

An important feature of qualitative research is the link between theory and method. The choice of research methods is inextricably linked to a particular theoretical perspective or set of explanatory concepts that provide a framework for thinking about the issue of consumer research.

3.3.1 Theoretical model of behaviour

The Theory of Planned Behaviour (TPB) (325) was used in this thesis and provides a model that can help explain consumers’ purchase behaviours in relation to functional food products. It has been demonstrated that the TPB, or modified

versions of it, has been applied to areas of consumers' food choice (20-22), supplement use (23) and genetically modified products (24). In addition, Baranowski *et al* (1999) published a review article on the psychosocial correlates of dietary intake and concluded that models with psychosocial variables predicting dietary fat and fruit and vegetable intake had a generally low predictive power (R^2 < 0.30), with no single model out predicting others (168). However, they went on to note that when models predicted narrower categories of behaviour such as the TPB (e.g. milk consumption), predictive power tended to be higher (168). Thus it could be predicted that this model will perform well against single action behaviour such as consumption of n-3 enriched functional foods. It was also suggested by the authors that in order to increase the predictive power of models, future research should combine variables from separate theories and incorporate variables that moderate the relationship of psychosocial to dietary behaviour (e.g. genetics of taste, stage in the life course).

3.3.2 Qualitative methods

As the TPB model had not been applied to consumer behaviour related to functional food use, beliefs and attitudes need to be determined. It has been suggest that for each behaviour under investigation it is important to determine the salient beliefs related to the behaviour in question (19). Focus group methodology is recommended and others have reported using this technique to assist with questionnaire design when using the TPB (23, 326, 327). Therefore, results obtained from a set of focus group interviews served to define the parameters of the TPB model and to test the language for questionnaire design.

Focus groups are a form of group interview that capitalises on communication between research participants in order to generate data. Here people are encouraged to talk to one another as well as the researcher, exchanging questions concerns and anecdotes to express points of view (328). Group work also assists in understanding an issue through the different ways people communicate with one another through the use of jokes, arguments and teasing which is useful as people's attitudes are not necessarily encapsulated in reasoned, logical responses to direct questions (328).

In this research, focus group procedures adhered to the guidelines as suggested by Krueger (1994) (329). Firstly, an interview guide was developed using the TPB theoretical framework. Questions were devised to determine consumer awareness, beliefs, attitude and barriers to using n-3 fatty acid functional foods. All focus groups interviews were conducted in a neutral setting (a community hall). Each group was heterogeneous for age, sex, socioeconomic status and occupation. All focus groups were recorded and transcribed *verbatim* and transcripts were analysed using a computer software package (Chapter 6).

3.3.3 Focus group sample and group composition

Focus groups studies consist of anything from half a dozen to over 50 groups, depending on the aims of the study and the resources available (328). It was reasoned that a sample of 12 groups would generate enough data to suit our needs. The aim of the study was to work with a representative sample of consumers with each group composition being heterogeneous in order to explore

the different perspectives within each group setting. It is inappropriate to assume that these group data are representative of the population, in the sense that these views would be expressed in the community independent of the group structure. In this sense the data obtained from the groups can be viewed metaphorically as a "guidepost" rather than a "landmark".

Using the TPB model, transcribed focus group conversation was categorised into one of the three areas of belief related to behaviour: *behavioural beliefs*, which are assumed to influence attitudes toward the behaviour; *normative beliefs*, which constitute the underlying determinants of subjective norms; and *control beliefs,* which provide the basis for perceptions of behavioural control. Content analysis was carried out and sub-categories were developed to capture the emerging themes. These themes provided the basis for questionnaire design.

3.3.4 Quantitative methods

To be able to test the ability of the model to predict behaviour a questionnaire needed to be developed. Questionnaire development followed the process outlined by Ajzen & Fishbein (1980). As recommended by the authors, clarity of the single action behaviour is critically important (325). The behavioural criteria are comprised of four elements: the action, the target at which the action is directed, the context in which it occurs, and the time at which is performed. In this study the single target behaviour was defined as *"eating foods enriched with omega-3 fats in the proceeding 2 weeks"*. Even though clinical research points to LCn-3 (from fish) as being more bioactive than ALA (from plants), this distinction was not made in

the questionnaire as it was apparent from focus group research that consumers did not distinguish the different classes of n-3. In order to measure behaviour we surveyed the subjects 2 weeks after completion of the survey to ascertain the behaviour under question, namely the consumption of n-3 enriched foods.

One of the advantages of this model is the ability to predict behaviour from intention. Although intentions are immediate antecedents of actions, the observed relationship between intention and behaviour depends on two factors: first, the measure of intention has to correspond to the behavioural criterion in action, target, context, and time; second, a measure of intention will predict behaviour only if the intention does not change before the behaviour is observed. Here the careful selection of intention is required. For this research intention was ascertained using the following measure of intention, *"I intend to eat one or more foods with added omega-3 oils over the next two weeks"*.

As outlined by the theory, a person's intention to perform a given behaviour is the immediate determinant of that behaviour. According to the TPB model, a personal or attitudinal component, a social or normative component and a control component determine behavioural intention (325). To assess a person's attitude toward behaviour a standard 7-point Likert scaling system was used. This alone does little to explain the complexity of behaviour, therefore the factors that determine these intentions are of interest. Beliefs can be viewed as underlying a person's attitudes, subjective norms and control that may ultimately determine intention and behaviour (325). A number of possible salient beliefs obtained from focus group interviews were used and converted into statements, anchored on a 7-

point bi-polar Likert scale. The implementation of the questionnaire was via postal survey and is also reported in detail in Chapter 7.

Whilst the reduction of behaviour to a set of statistical relationships is tempting, caution in interpreting these results is required. At best statistical models have been shown to explain about 30% of behaviour (168, 330). With the area of functional foods being a contemporary issue, very little research has been published on the psychosocial correlates which determine behaviours about their use. For these reasons this research is as much exploratory as it is explanatory.

3.3.5 Questionnaire sample

As with the dietary intervention studies this research drew samples from an adult population that would benefit most from the introduction of n-3 enriched functional foods in reducing CHD risk. In addition, recruitment targeted a mixture of both consumers without existing disease as well as with morbidities that may increase the likelihood of developing CHD (e.g. diabetes mellitus), varieties of socioeconomic backgrounds, age brackets and income ranges. The literature has suggested that these factors may be independent determinants of healthy foods choice (331).

Samples meeting these criteria were drawn from the general community of interested consumer's via media (print and radio) as well as subjects with type 2 diabetes mellitus (otherwise healthy) participating in an unrelated dietary intervention trial. Stratifying the sample based on co morbidities as well as recruiting from a general sample facilitated the investigation of socioeconomic and

health factors, in addition to beliefs and attitudinal factors about functional food use.

3.4 Stakeholder study

Although the collaboration of the food industry, government and health professionals is regarded as being important for functional foods to reach their maximum potential, no studies have investigated the attitudes of these stakeholder groups in the Australian context. Theoretically stakeholders are of interest because their needs, wants, desires, perceptions and conceptualisations differ (324). We can view stakeholders as networks of human beings, actors or agents with distinct roles to play based on distinct ideology which is defined as *"scheme of ideas at basis of political etc. theory or system"* (332). It is the interest in the underlying assumptions of these ideologies which is fundamental to this research. A qualitative methodology was used to expose and challenge underlying assumptions and making ideologies explicit.

3.4.1 Analytical strategy

From the literature five key domains which were derived which are regarded as being key issues of likely controversy between stakeholders. These were related to issues surrounding processing and production, the ability of functional foods to achieve tangible health benefits, evidence required for health claims, the regulatory environment (including substantiation authority, health claim wording authority and public disclosure of evidence) and intellectual property and propriety issues (25, 333). This framework provided a foundation for analysis.

Although focus group methodology could be used in this study, semi-structured interviews have been suggested as a more viable option for eliciting views from professional groups (324). Reasons cited are that professionals tend to be less likely to express true beliefs in an open forum, and there is a strong probability that discussion will end in verbal point scoring between opposing views, as opposed to an exploration of these issues (324). An interview guide was developed using the theoretical groupings of McConnon *et al* (2002) and Kwak *et al* (2001) to develop the categorisation of key issues to be investigated with stakeholders (25, 333). All interviews (except for two face-to-face interviews) were conducted over the phone, recorded and transcribed *verbatim* by an independent third person. Transcripts were analysed using a computer software package for qualitative research analysis, NVivo 2.0 (2002, QSR International Pty Ltd), which was used for data management and coding (334).

Stakeholders may present themselves as people, as role occupants, as groupings, as occupational groupings and pressure groups and many more categories. Identifying stakeholders for this research presented a number of hurdles. The general guideline is that a stakeholder is any interest group affected by or affecting the phenomena, which in this case is the development and communication of functional foods. In addition, the stakeholder grouping is usually constructed based on those having a relatively consistent experience and point of view. Practically, where an imagined stakeholder interest group (e.g. dietitians/nutritionists) has significant internal differences of opinion (e.g. between dietitians employed in

clinical, public health and food industry settings) the grouping should be further broken down.

The process of conducting qualitative, stakeholder analysis, involves making continuous skilled researcher craft judgments – whom to talk to and what to ask (324). When researching complex social areas orientating advice can be significantly shaped by these fine judgments. Furthermore, this research, as in any kind of applied research, is sponsored by, and intended to serve the interests of, one or more of the stakeholders. Whilst this does not automatically invalidate the process it needs to be stated and considered when drawing conclusions from the research. Perhaps stakeholder analysis is a clearer method than many other forms of research about these difficult but crucial underlying choices.

3.4.2 Sample

Stakeholders were first identified, using the literature as a guide. McConnon *et al* (2002) identified key stakeholders when considering the development of functional foods (25). These were defined as the food industry, consumers, health sector and government. These categories were further subdivided into eleven categories regarded as representing the key stakeholders in relation to functional food development in Australia. Key leaders in the field of Nutrition and Dietetics were consulted to structure a sampling frame based on these key groupings. The objective was twofold; firstly to sample a wide range of views from a variety of states and territories within Australia, and secondly to recruit equal numbers of

subjects presumed to have positive and negative views about functional food development.

One approach to identifying stakeholders identified in the literature is to follow a process of sequential sampling. This process involves collecting information from one interested party who will identify other stakeholders who can be followed up. This process is followed until nothing new turns up (324). This process was rejected in favor of a more structured form of sampling described above for two reasons. Firstly, the sequential sampling approach can be regarded as an optimal but requiring considerable effort and resources that were not available within the time frame (324). Secondly, with the recently established National Centre of Excellence in Functional Foods, there was the unique opportunity to draw on the expertise of Centre members in identifying key stakeholders within the Australian context. Specific details of the stratification are outlined in Chapter 8.

3.4.3 Limitations and quality issues in qualitative research

Qualitative research suffers from the “stigma of the small *n*” (335) because of the small samples under investigation and due to not seeking statistical representiveness. However this feature is irrelevant to the strength of this approach (335). Nonetheless, as with all forms of research attention to the quality of research is important.

The first issue of quality is with validation. There are no easy methods of validating qualitative research. There are ways to improve the validity which requires the exercise of judgment on the researcher. Firstly, since methods used in research

influence the objects of enquiry, it is important to provide a clear account of the process of data collection and analysis. This helps the reader judge the conclusions made from the methods used and the way the data was collected. These details have been included in the methods section of the qualitative studies presented in this thesis.

Closely related to this concept is the issue of reflexivity. This means the ways in which the researcher and the research process have shaped the data collection, including the role of prior assumptions and experience, which may influence the interpretation of the results (335). In view of this, personal and intellectual biases are declared at the outset of any research reports, which enhance the credibility of the findings. The final technique that has been employed to reduce bias is termed “fair dealing”. By this the researcher ensures that the research design explicitly incorporates a wide range of different perspectives so that the viewpoint of one group or person is not presented as if it represents the sole truth of the situation (336).

An additional dimension of relevance is the extent to which findings can be generalised beyond the research setting. In this sense forms of stratified sampling were used in order to ensure that the range of settings chosen was representative as well as the reporting of results with sufficient descriptive detail for the reader to be able to judge whether or not the findings apply to other situations.

CHAPTER 4

FUNCTIONAL FOODS ARE RELEVANT FOR INDIVIDUALS IN MANAGING CHD RISK[2]

4.1 Introduction

Despite extensive evidence for the efficacy of plant sterols/stanols in reducing serum cholesterol, evidence of effectiveness in a practice setting is absent. Forty-one clinical trials have studied the efficacy of plant sterol esters and plant stanol esters in reducing mean total cholesterol (TC) and low-density lipoprotein cholesterol (LDL) (337). When plant sterols/stanols are added to foods such as margarine, up to a 15% reduction in serum TC and LDL has been reported and the effect is additive with diet and drug interventions (337). To this end the Adult Treatment Panel (ATP III) of the National Cholesterol Education Program (NCEP) recommend the addition of plant sterols (2g/day) to the diet (14). However, all

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[2] *A significant portion of this chapter has been accepted for publication in the following peer reviewed journal:*

Patch CS, Tapsell LC, Williams PG. Plant sterol/stanol prescription is an effective treatment strategy for managing hypercholesterolemia in outpatient clinical practice. Journal of the American Dietetic Association. 2005;105:46-52.

CP was responsible for the design of the study, organisation and leading the study, data collection and analysis, critical discussion of the analysis and preparation of the manuscript. LT and PW were responsible for critical discussions of the study design, critical discussions of the analysis and preparation of the manuscript.

studies to date have been in controlled settings using willing volunteers and providing subjects with sterol containing products. These results are yet to be replicated in subjects who assemble the diets for themselves on a routine basis, thus determining the true effectiveness of this strategy in the clinical setting (281).

It is commonplace when determining evidence for functional foods to equate efficacy with effectiveness. Whereas efficacy aims to establish a relationship between a bioactive component and a therapeutic benefit, effectiveness goes one step further in answering the question of compliance i.e. do subjects comply with the therapy and does the therapeutic relationship hold. Whilst it may be correct to assume that efficacy often equates to effectiveness with therapeutic drugs, with functional foods, issues relating to the belief in the benefit of use, taste, preference and price may affect intake and ultimately compliance (20, 22). In addition, the net effect on the whole diet needs to be considered in those who select functional foods, given the importance of other dietary aspects such as restricting saturated fat and cholesterol and increasing n-3 fatty acids, in reducing CHD risk (306). It is therefore important in evaluating effectiveness to design trials in free-living contexts.

A clinical setting with standard practices is a useful setting for this research. However, the problem associated with recruitment in clinical trials has been acknowledged in the literature and obtaining the numbers needed to achieve statistically significant results can be problematic (338). The Number Needed to Treat (NNT) is becoming increasingly popular as an index for reporting results of randomised controlled trials and other clinical trials (339). The NNT represents the

clinical effort that is required by treatment in order to achieve a positive outcome, compared to the expected response in a control group. This provides a useful measure of effectiveness and overcomes some of the issues of recruitment of large numbers of subjects.

The aim of this study was to test the effectiveness of plant sterol/stanol prescription in consenting patients referred to an outpatient clinic that were randomised to either standard practice (a low fat, low saturated fat, high fibre diet) or standard practice with the additional prescription of 25g of a plant sterol or plant stanol containing margarine. This was a free-living trial. No food products were supplied to subjects, and the margarines prescribed were available commercially.

4.2 Methods

4.2.1 Participants and Study Design

Subjects were recruited through an established dietary outpatient clinic over an 18 - month period (Appendix A & B), from February 2001 to August 2002. Subjects were included if they were aged between 30 - 75 years and had a fasting serum total cholesterol concentration > 5.50 mmol/L. Those who were of non-English speaking background, taking lipid lowering medication for less than 3 months prior to referral or had changed dosage within the last 3-month period were excluded, along with subjects following dietary regimens for allergy or intolerance, renal disease and diabetes mellitus. Details of the study were explained to all subjects and written informed consent provided before participation in the study. The goal was to recruit 80 participants (allowing for 10 drop outs); on a sequential basis

using a blocking protocol for total serum cholesterol to ensure the cholesterol levels at baseline did not differ between the groups. Within each block, the allocation to treatment or control group was random. The study was conducted on an outpatient basis over 12 weeks and no foods or supplements were provided to subjects. At baseline (week 0) and week 12 subjects filled out a 3-day food record (two week days and 1 weekend day). One week later (week 1 and 13) a detailed dietary history was conducted along with weight, waist circumference measurement and an activity questionnaire. Fasting blood samples were collected prior to baseline ~2-3 weeks and at 12 weeks. The human research ethics committee of the University of Wollongong and the Illawarra Area Health Service approved the study (Appendix C).

4.2.2 Treatment Conditions

Control group

Subjects were individually instructed by the same Accredited Practising Dietitian to consume a diet low in saturated fat, consistent with the Therapeutic Lifestyle Changes dietary approach as outlined by NCEP (14). In summary, the diet aims for 25 - 30% of total calories from fat (<7% saturated fatty acids, up to 10% polyunsaturated fatty acids, and up to 20% monounsaturated fatty acids), 50 - 60% of total calories from carbohydrates, ~15% of total energy from protein, <200mg cholesterol/day, fibre 20 – 30g/day and total energy balanced with energy expenditure to maintain desirable body weight or prevent weight gain (14). Sterol/stanol use was not included as part of dietary instruction, however if subjects

were currently using plant sterol/stanol containing margarine they were encouraged to continue. This was regarded as standard practice and there would be ethical implications to actively discouraging the use of these products. Subjects were provided with information sheets offering practical suggestions and an individual diet plan. Furthermore, all subjects were encouraged to exercise (e.g. 30-minute brisk walking) at least 3 sessions per week. Counselling sessions consisted of a 1-hour session at week 1 and two 30-minute sessions at week 3 and 6.

Intervention group

The intervention diet was identical to the control diet in all but one aspect. All subjects on the intervention diet were instructed to consumer 25g (5 teaspoons) of either Flora's *Pro-active*® (Unilever Australasia, Sydney, Australia) or Meadow Lea's *Logicol*™ (Goodman Fielder, Sydney, Australia) margarine (these were the only plant sterol and plant stanol containing products respectively on the Australian market at the time of the study). The information sheets and diet plans explicitly stated these products and the amount to be consumed each day (Appendix D). Subjects were instructed to modify their fat intake to accommodate the sterol/stanol dosing and encouraged to consume additional carotenoid containing vegetables to prevent a possible decrease in plasma carotenoids as a result of the sterol/stanol intake (340). This message was the focus of each follow-up clinic visit.

4.2.3 Dietary Measurement

A dietary history (DH) (Appendix E) interview was conducted by the same dietitian (CP) at baseline and at 12 weeks. The DH method lends itself well to clinical practice where it provides clinicians with an opportunity to subjectively examine meal patterns and provide dietary advice while the patient is still present (309). Furthermore the utility of this method has been demonstrated in a number of sample groups, including short-term intervention studies (311, 341, 342). The approach taken was a narrative style DH interview (open-ended) described in detail elsewhere (341). In summary, the meal based diet history interview noted the types, amounts and frequency of consumption of all foods consumed routinely within a 3-month reference time. The interview was completed with a food frequency checklist of major food categories, snack and drink items (including alcohol), and sources of n-3 fatty acids (fish, nuts, soy foods), as well as questions on food preparation practices. The same assessor was the interventionist in this study; therefore the DH was not blinded. Subjects were also required to keep an estimated 3-day food record (FR) (Appendix F) (two weekdays and one weekend day) after the collection of the DH. Forms were provided along with instructions on how to estimate food portion sizes using standard kitchen measuring equipment. Food records were checked for missing values and clarification.

4.2.4 Anthropometric Measurement

Weight and height was recorded at each interview using calibrated balance scales and a wall mounted stadiometer. All subjects were clothed (light shirt). Height was

measured to the nearest 0.5cm and weight to the nearest 0.1kg. Waist and hip circumferences were measured at each time point. Waist circumference was measured using a standard cloth measuring tape (units in centimeters and inches); at natural waist when clearly identifiable or midway between the lower rib and iliac crest) and hip circumference measured at the greater trochanter. For each circumference, measurements were recorded to the nearest 0.5 cm. Activity level was obtained from a self-reported description of usual activity, including details of number of sessions a week of activity, the duration of each session and the intensity of each activity.

4.2.5 Biochemical Measurement

In keeping with standard clinical practice, a trained phlebotomist at accredited pathology laboratories within the Illawarra region (Southern IML Pathology) drew blood samples from subjects after an overnight fast. Subsequently, samples were analysed for concentrations of total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride. The pathology laboratories were fully accredited with the National Association of Testing Authorities (NATA) and the Royal College of Pathologists of Australasia (RCPA), which is a mandatory requirement for pathology laboratories in Australia.

4.2.6 Analysis and Statistics

Dietary data were analysed with Foodworks nutrient analysis software package (Professional Version 3.1, Xyris Software, Highgate Hill, Brisbane Australia), which

is based on the Australian Nutrient Database (AUSNUT 2000, Department of Human Services and Health, Canberra).

The required sample calculation of n = 80 was based on detecting a difference in TC between the groups of 10% at 80% power. A Bland Altman plot (343) was prepared to determine possible bias between actual and recalled total fat (%kJ) with the two dietary assessment methods. As described by Bland and Altman (343) the average of the reported fat intake in the DH and the FR (*x* axis) are plotted against the difference between the DH and FR recorded fat intake (*y* axis). The limits of agreement were set at 2 s.d. of the difference above and below the mean. Comparing the reported intake using the DH and FR using Pearson's correlation coefficient assessed the validity of sterol/stanol intake. Changes between week 0 and week 12 were studied for all variables. Dietary data and baseline measurements were assessed using a two-factor analysis of variance with diet and sex as fixed factors. The effect of dietary intervention was assessed using paired Student's *t*-tests when comparing the change in variables (dietary, anthropometric and biochemical data) between groups. For non-normally distributed data, Wilcoxon signed ranks test was used for within group comparisons, and Mann-Whitney *U* test were conducted to determine the proportion of subjects reaching dietary and biochemical goals between the groups. Changes in clinical outcomes were analysed with an intention to treat model-using analysis of variance i.e. subjects were included in the analysis whether or not they consumed the prescribed dose of plant sterol/stanol. The relationship between plant sterol intake and serum cholesterol was determined using Pearson's correlation coefficient. The

number needed to treat (NNT) index as described elsewhere was used to report effectiveness of the intervention (339). The NNT was calculated using the proportions of subjects on the control or experimental treatment who achieve a total cholesterol reduction of ≥ 15%. NNT is defined as (339):

$$NNT = \frac{1}{\pi_1 - \pi_2}$$

Here π_1 and π_2 are defined as the proportions of subjects on the control or experimental treatment (respectively) that experience the defined outcome. A P - value less than 0.05 were considered to be statistically significant. SPSS for Windows (version 10.0, 1999, Chicago, IL) was used for all statistical analyses.

4.3 Results

4.3.1 Subject demographics

During the 18-month time course of the study forty-two participants met the eligibility criteria, 32 agreed to participate producing a group randomisation of 15 control and 17 intervention subjects. Twenty-five subjects (15 women, 10 men) completed the study producing a group randomisation of 11 control and 14 intervention subjects. One subject withdrew after a myocardial infarct, another subject was hospitalised for psychiatric illness and was unable to complete the final assessment phase, the clinic lost contact with another subject, and four withdrew after the initial assessment. Baseline characteristics of all subjects admitted to the

study are shown in Table 4-1. The groups did not differ in terms of age, sex, body mass index, level activity, and waist: hip ratio, medication use, or serum lipid levels.

Table 4-1 Participant characteristics at baseline

	Control Group (n = 15)	**Intervention Group (n = 17)**
Age (y) (mean ± s.d.)	59.2 (±13.2)	55.2 (±13.4)
Female/Male	11/4	8/9
BMI (kg/m^2) (mean ± s.d.)	29.8 (±3.3)	31.4 (±5.9)
Waist/Hip ratio (mean ± s.d.)	0.92 (±0.1)	0.94 (±0.12)
Cholesterol lowering medication (n)[†]	2	3
Frequency of activity		
Nil	5	6
1-2 times per week	3	3
3-4 times per week	2	2
5-6 times per week	1	2
>6/7 times per week	0	1
Total cholesterol (mmol/L) (mean ± s.d.)	6.64 (±0.8)	6.89 (±1.1)
[*]LDL cholesterol (mmol/L) (mean ± s.d.)	4.25 (±0.6)	4.35 (±0.8)
[**]Triglyceride (mmol/L) (mean ± s.d.)	2.40 (±1.4)	2.28 (±1.1)
[***]HDL cholesterol (mmol/L) (mean ± s.d.)	1.27 (±0.3)	1.26 (±0.3)

[*]n = 23, [**]n = 30, [***]n = 27

There were no significant differences between any of the groups at baseline using Student's t-test for normally distributed data and Wilcoxon sign ranked test for categorical data.

[†]Refers to number of patients taking cholesterol lowering medication who had not reported a change in dose for >3 months.

4.3.2 Dietary data

Using the Bland-Altman plot there was no relationship between the average of the reported fat intake using DH and FR with the difference between the two methods (Figure 4-1). All but one value was within the 2 s.d. confidence limits as

recommended by Bland and Altman (343) indicating that there was no systematic bias. Strong correlations were found between the DH and FR method ($r = 0.782$, $P < 0.001$) for plant sterol/stanol consumption. Results of the dietary analyses using DH data are reported in Table 4-2. Overall, nutrient intakes were not significantly different between the two groups at baseline and at the end of the study (12 weeks). However, the change in protein was higher in the intervention group (22%) compared to the control group (5%) as well as the change in alcohol intake (-7g/day, 2g/day in the intervention and control group respectively). The average intake of plant sterols/stanols increased by 1.3g/day in the intervention group and reduced by 0.3g/day in the control group ($P < 0.05$), and 20% achieved the goal intake of ≥ 2g/day compared to none in the control ($P < 0.05$). After the intervention period, 43% ($P < 0.05$) of the intervention subjects achieved a polyunsaturated fat intake within the NCEP Step 1 diet recommendations (≥ 7%kJ) whereas none achieved this goal in the control. There were no other differences between the groups in achieving the NCEP recommendations (Table 4-3).

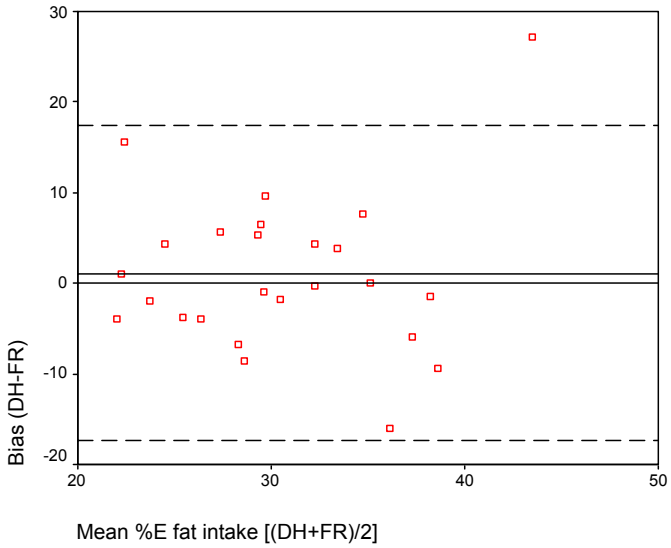

Figure 4-1 Bland-Altman plot of the mean difference between the DH and FR for fat (%kJ) versus the mean of the DH and FR, with limits of agreement of 2 s.d. from the mean difference

Table 4-2 Mean (± s.d.) of dietary composition at week 0 and 12 as determined by DH

Lipid	Control group (n = 11)	Intervention group (n = 14)
Energy		
Week 0 (kJ)	8059 (±2658)	8899 (±3298)
Week 12 (kJ)	7700 (±2395)	7219 (±1718)
^Change %	-4.5 (±21)	-18.9 (±30)
Protein		
Week 0 (%kJ)	19.7 (±2.3)	17.2 (±3.2)
Week 12 (%kJ)	20.7 (±2.0)	21.0 (±3.0)
^Change %	5.2 (±13.8)*	21.9 (±21.4)*
Fat		
Week 0 (%kJ)	29.7 (±5.3)	31.9 (±8.9)
Week 12 (%kJ)	27.4 (±5.4)	29.9 (±6.4)
^Change %	-8.0 (±14.8)	-6.4 (±34.3)
CHO		
Week 0 (%kJ)	47.4 (±4.8)	44.4 (±6.8)
Week 12 (%kJ)	48.0 (±4.7)	44.1 (±7.0)
^Change %	1.2 (±8.7)	-0.7 (±31.2)
SFA		
Week 0 (%kJ)	10.1 (±2.9)	10.9 (±6.9)
Week 12 (%kJ)	8.0 (±2.3)	7.3 (±2.1)
^Change %	-20.8 (±27.5)	-32.1 (±37.5)
MUFA		
Week 0 (%kJ)	12.4 (±2.6)	12.9 (±3.3)
Week 12 (%kJ)	11.7 (±2.6)	13.3 (±3.6)
^Change %	-5.6 (±17.8)	2.9 (±37.9)

PUFA		
Week 0 (%kJ)	4.4 (±1.1)	5.3 (±1.5)
Week 12 (%kJ)	4.0 (±1.2)	6.3 (±2.4)
^Change %	10.1 (±32.4)	18.6 (±56.2)
Cholesterol		
Week 0 (mg)	201.3 (±106.4)	236.7 (±166.1)
Week 12 (mg)	170.8 (±58.4)	153.1 (±64.9)
^Change %	-15.1 (±34.6)	-35.3 (±41.4)
Dietary fibre		
Week 0 (g/day)	28.5 (±10.1)	24.2 (±8.5)
Week 12 (g/day)	35.3 (±17.3)	26.5 (±8.9)
^Change %	24.1 (±69.4)	9.5 (±40.9)
Alcohol		
Week 0 (g/day)	3.4 (±6.1)	15.2 (±24.1)
Week 12 (g/day)	5.6 (±8.8)	8.4 (±16.7)
^^Change (g/day)	2.2 (±3.9) *	-6.8 (±10.7) *
Sterol/stanol intake		
Week 0 (g/day)	0.8 (±1.0)	0.2 (±0.5)
Week 12 (g/day)	0.5 (±0.8)	1.5 (±0.8)
^^Change (g/day)	-0.32 (±0.7) *	1.26 (±0.9) *

*P < 0.05, **P < 0.01, significantly different between the groups

$$\text{^Change \%} = \frac{\text{week 12 – week 0}}{\text{week 0}} \text{ X 100\%.}$$

$$\text{^^Change} = \text{week 12 – week 0}$$

Table 4-3 Number of subjects meeting selected fatty acid targets at baseline and 12 weeks (n = 25)

Time	Group	Plant sterol/stanol		Saturated fat intake		Polyunsaturated fat intake	
		≥ 2g/day	< 2g/day	≥ 10%kJ	< 10%kJ	≥ 7%kJ	< 7%kJ
Baseline	Control	0	0	4	7	0	11
	Intervention	0	0	7	7	2	12
12 weeks	Control	0*	11	2	9	0*	11
	Intervention	3*	11	2	12	6*	8

*P < 0.05, **P < 0.01, significantly different between the groups, Mann-Whitney *U* Test.

4.3.3 Serum lipids in response to treatment

Total cholesterol, plasma triglyceride, HDL cholesterol and LDL cholesterol concentrations did not differ significantly over time or between groups. Concentrations of plasma lipids at the beginning and end of the trial are shown in Table 4-4 and Figure 4-2. After the 12-week intervention the reductions in total cholesterol and LDL cholesterol were more than double in the intervention group compared to the control group, but these differences did not reach statistical significance. There was a strong inverse relationship between 12-week dietary sterol/stanol intake and change in serum cholesterol (r = -0.62; P = 0.001) and serum LDL cholesterol (r = -0.58; P = 0.019).

Table 4-4 Mean (± s.d.) plasma lipid concentrations at week 0 and 12 with each dietary intervention.

Lipid	Control group	Intervention group)
Total cholesterol		
Week 0 (mmol/L)	6.64 (± 0.8)	6.89 (± 1.1)
Week 12 (mmol/L)	6.29 (± 0.7)	6.10 (± 1.0)
Change %	-5.3 (± 11.5)	-11.5 (± 13.9)
LDL cholesterol (n = 17)		
Week 0 (mmol/L)	4.25 (± 0.6)	4.35 (± 0.8)
Week 12 (mmol/L)	4.06 (± 0.9)	3.82 (± 0.9)
^Change %	-4.5 (± 15.7)	-12.2 (± 13.1)
Triglyceride (n = 24)		
Week 0 (mmol/L)	2.40 (± 1.4)	2.28 (± 1.1)
Week 12 (mmol/L)	2.20 (± 1.3)	2.20 (± 0.9)
^Change %	-8.3 (± 28.7)	-3.5 (± 34.7)
HDL cholesterol (n = 19)		
Week 0 (mmol/L)	1.27 (± 0.3)	1.26 (± 0.3)
Week 12 (mmol/L)	1.29 (± 0.3)	1.27 (± 0.3)
^Change %	1.6 (± 8.7)	0.7 (± 13.1)
LDL:HDL ratio (n = 18)		
Week 0 (mmol/L)	3.37 (± 0.9)	3.49 (± 1.0)
Week 12 (mmol/L)	3.29 (± 1.1)	3.21 (± 1.1)
Change %	-2.4 (± 14.7)	-8.0 (± 10.0)

$^{*}P < 0.05$, $^{**}P < 0.01$, significantly different from baseline

$$\text{^Change \%} = \frac{\text{week 12} - \text{week 0}}{\text{week 0}} \times 100\%.$$

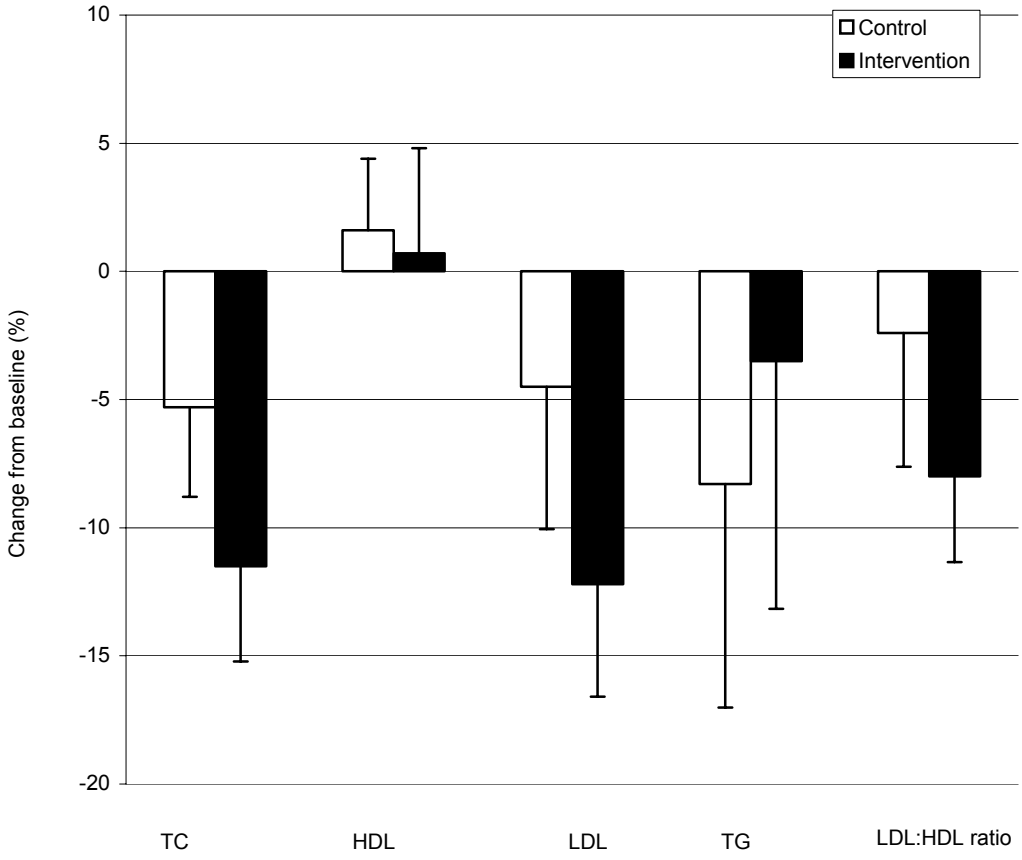

Figure 4-2 Mean (± SEM) percentage change in serum lipids from baseline to the end of the 12 week dietary intervention.

4.3.4 Clinical indexes

At the completion of the 12-week study 5 of 14 subjects in the intervention group achieved ≥ 15% change in total cholesterol compared to 0 of 11 in the control ($P < 0.05$). No other significant changes were observed between the groups. Both groups showed a mean weight change of < 1.0 kg and there were no significant differences in body weight from the baseline to the end of the study. The NNT was calculated to be 2.8.

4.4 Discussion

This present study is the first to investigate the utility of using plant sterol/stanol enriched margarine in a free-living clinical context. While evidence-based nutrition recommendations attempt to translate research data into nutrition care (344), research in clinical settings are paramount, as the applicability of controlled studies to the general community is often questioned (10). For example, in the effective management of hyperlipidaemia there are a number of dietary approaches that can achieve moderate results; these include reducing the saturated fat in the diet, increasing n-3 polyunsaturated diet and increasing dietary fibre (345). It is important that the introduction of novel dietary strategies do not adversely affect the dietary profile.

In conjunction with dietary advice consistent with the NCEP guidelines, the inclusion of ~1.5-2g/d of plant sterol in margarine did not adversely affect the dietary fat or plasma lipid profiles (Tables 4-2 and 4-4). Of particular interest was the increase in the proportion of fats coming from polyunsaturated fat in the intervention group. Intervention trials have shown that visible fats (such as margarine and nuts) are often excluded from the diet in preference to hidden fats that are often saturated (346). The net effect is a decrease in the P:S ratio. There is increasing evidence of the importance of polyunsaturated fats as an important dietary component in reducing the risk of CVD and the need to increase the P:S ratio is of importance (347). A serendipitous result of prescribing a daily dose of plant sterol/stanol enriched margarine was the delivery of extra n-3 polyunsaturated fatty acids. As plant sterols/stanols have an additive effect with

other dietary modifications (348), prescribing enriched margarine is an effective strategy to achieve both of these targets.

The dietary assessment measures had some limitations. In pursuit of designing a study resembling ‘real life’ conditions there was a trade off by un-blinding the DH, as the assessor was also the interventionist. The impact of this on the interpretation of data is marginal as it was demonstrated that there was a strong agreement between the DH and FR methods for dietary fats and sterol/stanols, indicating relative validity.

Although compliance to the dietary regimen is a key component of determining effectiveness, the ability to show the therapeutic benefit is also important. The mean percent reduction in total cholesterol and LDL cholesterol in the intervention group was ~11% and ~12% respectively compared to ~5% and ~4% in the control group. This difference however was not statistically significant either within groups or between groups over time, probably because the final sample size was significantly smaller (n = 25) compared to the original sample size power calculations (n = 80). The study was underpowered to reject the null hypothesis that is, there was not enough evidence to say there was a significant difference between the groups.

Results showed that dietary prescription of plant sterol/stanol margarines in conjunction with the NCEP guidelines for the management of blood lipids resulted in an intake of 1.3g sterol/day, whereas in the control group plant sterols reduced their intake by 0.4g/day. Although studies have shown that 2g/day of plant sterol is

the therapeutic optimal dose, the response curve is linear suggesting that an intake of around 1.5g/day is still clinically useful and the levels achieved would be predicted to achieve a change of ~10% in total serum cholesterol and LDL cholesterol (348).

A significant limitation to clinical practice in the dietary management of hyperlipidaemia is that despite studies in controlled environments achieving up to 15% reductions in total cholesterol, results in a free-living context have at best shown a 5% reduction (12). This study showed that 5 of 11 (36%) in the intervention group achieved a reduction in serum cholesterol levels ≥ 15% compared to none in the control. This has an important clinical implication, which can be summarised using the NNT statistic. Using this index one can predict that if clinicians routinely prescribe 2g/day of plant sterol/stanol margarine, then for every 2.8 patients given this advice, an extra patient will achieve cholesterol reducing effect ≥ 15%, compared to a more conventional approach to dietary therapy. However, these results can only be viewed as preliminary at this stage as the small sample size makes generalising beyond the bounds of this clinical context inappropriate. Further research is required in this area which would involve a larger sample, drawn over multiple clinical sites before conclusive evidence can be provided.

What has been known for many years regarding the efficacy of plant sterol/stanol enriched food products in the management of hyperlipidaemia can now be extrapolated into a free-living, clinical context. Plant sterol/stanol margarines provide a useful and significant adjunct to medical nutrition therapy. However,

while these positive results may be relevant to at-risk individuals, the utility of functional foods applied to a population approach needs to be verified. The following study investigated the effectiveness of n-3 enriched functional foods in a lower-risk group.

4.5 Acknowledgements

I would like to thank Pork Kembla Hospital Executive, the administrative team for their support in this project and the study participants for their enthusiasm. In-kind support (administration and clinic space) was provided by the Executive of Port Kembla Hospital.

CHAPTER 5

FUNCTIONAL FOODS ARE RELEVANT TO POPULATION BASED APPROACH TO MANAGING CHD RISK[3,4]

5.1 Introduction

Population intakes of fatty fish remain suboptimal and functional foods enriched with LCn-3 may be an effective population approach in managing this risk. Diets high in fish has been found in epidemiological studies to protect against heart disease (15-17). In addition, a number of experimental studies have demonstrated the efficacy of LCn-3, which is found in fish, in reducing triglycerides, blood pressure, arrhythmia and thrombotic risk (145, 349, 350), and it is this combined effect that is believed to be cardioprotective (147). This study was a 6-month

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[3] *A significant portion of this chapter has been submitted for publication in the following peer reviewed journal:*

Patch CS, Tapsell LC, Mansour J, Noakes M, Murphy KJ, Mori TA, Meyer BJ, Clifton PA, Puddey IB, Beilin L, Annison G, Howe PRC. (In Press) The use of novel foods enriched with long chain omega-3 fatty acids to increase dietary intake. *Journal of the American Dietetic Association*. 2005.

[4] CP was responsible for the critical discussions of the design of the study, entire dietary assessment, analysis of the dietary data and preparation of the manuscript; JM and KM were responsible for the coordination of the study, data collection and assessment, critical discussions in the analysis and discussions of the manuscript. MN and IP were responsible for critical discussions of the study design, critical discussions of the analysis and discussions of the manuscript. PH, BM, TM, GA & LT were responsible for the grant submission, study design and administration and, critical discussions of the analysis and discussions of the manuscript. In addition, LT supervised the dietary assessment.

nutritional intervention study investigating the effects of foods enriched with (LCn-3) on cardiovascular biomarkers. In order to substantiate the dietary effects of these processed enriched foods, evidence of actual changes in fatty acid intakes is required. In addition, the net effect on the whole diet needs to be considered in those who select these foods, given the importance of other dietary aspects on cardiovascular disease risk. For example, authorities recommend 25 - 30% of total calories from fat (< 7% saturated fatty acids (SFA), up to 10% polyunsaturated fatty acids (PUFA), and up to 20% monounsaturated fatty acids (MUFA)), 50 - 60% of total calories from carbohydrates (CHO), ~15% of total energy from protein (PRO), < 200mg cholesterol/day, fibre 20 –30g/day and total energy to be balanced with energy expenditure to maintain desirable body weight or prevent weight gain (14) and for a 8360kJ (2000kcal) diet, a daily intake of n-3 fatty acids of 0.65g eicosapentaenoic acid (EPA) + docosahexaenoic acid (DHA) and 2.22g α-linolenic acid (ALA), with the n-6:n-3 ratio reduced to substantially less than 10 (160). Therefore, in assessing dietary interventions, proper dietary assessment methodology is essential as a cornerstone to evaluating the overall dietary profile.

The DH is considered to be the gold standard of methods used to assess dietary intakes (307). It has an established place as a dietary assessment method and lends itself well to clinical practice (309). The DH method is also valuable in intervention trials where dietary manipulation is required at the time of assessment (310). However, as with all dietary assessment methods there are limitations, which include interviewer bias, inaccuracy of portion size reporting, errors in reporting of frequency, and a need for subjects to follow regular eating habits

(316). Due to the limitations of collecting intake data, the quality of dietary assessment methods needs to be assessed in the context of each study (310). This can be assessed using a number of methods such as reporting the degree of over- and underreporting, assessing bias to over- or under report and the relative validity between dietary intake data and erythrocyte fatty acid biomarker data. In this study we developed multiple strategies to determine dietary intake. The aim of this study was to evaluate the impact of incorporating LCn-3 enriched foods in the diets of individuals, whose usual diet was low in fish, making clear reference to the quality and validity of the dietary data.

5.2 Methods

5.2.1 Recruitment and screening

The study was approved by the Human Research Ethics Committees at the University of Western Australia, the University of Adelaide and the CSIRO (Appendix G). Ninety-four overweight men and women were recruited, age range between 20 to 65 years through local media advertisements at two sites – Perth and Adelaide (Australia). During a screening interview those who had plasma triglycerides < 1.6 mmol/L, a diagnosis of diabetes, recent symptomatic heart disease; angina pectoris; history of myocardial infarction or stroke; peripheral vascular disease; major surgery within the last 3-months; BP > 170/100 mmHg; liver or renal disease (plasma creatinine > 120 μmol/L); regular non-steroidal anti-inflammatory drug therapy; antihypertensive drugs, lipid lowering or other drugs affecting lipid metabolism; eating more than one fish meal per month or taking fish

oil supplements; or an inability to consume the test foods were excluded. All participants gave their written informed consent. Of the 94 volunteers who attended screening, 86 volunteers were randomly allocated to either the intervention group (n = 42) or the control group (n = 44). A block randomization scheme was used to balance groups according to baseline triglyceride concentration and body mass index (BMI).

5.2.2 Study procedures

The goal of the intervention was to consume 8 serves of study foods per day equivalent to 1g LCn-3. Each serve of the enriched foods provided 125mg EPA + DHA The objective was for subjects to replace current foods with study food equivalents. A variety of LCn-3 enriched foods and matching control foods, which included cheese spread, chocolates, instant oats, milk, dip (pepper, tzatziki, salsa), biscuits (Anzac and ginger), pancakes, muffins, bread, salad dressing and soup (chicken laksa and vegetable) were prepared by Goodman Fielder Ltd Sydney, Australia. The fatty acid composition of the n-3 enriched foods provided to the participants is detailed in Table 5-1. Intervention foods (enriched with LCn-3) and equivalent control foods (not enriched) were supplied to all subjects in unmarked packages with one of two codes. The content of the study foods was blinded to subjects as well as researchers. Subjects were provided with two weeks supply of study foods and instructed on how to incorporate at least 8 portions of study foods into their daily eating pattern (Appendix H), but were not prescribed menus. The background diet was *ad libitum*, however after the first month of the study, those subjects who gained weight were advised by a qualified dietitian on ways to reduce

their energy intake whilst accommodating the study foods. This was monitored throughout the study.

Table 5-1 Estimated fatty acid content of the study foods provided to subjects in the intervention and control groups

Food (Serve size)	*Group*	*Oleic (18:1n-9)*		*Linoleic (18:2n-6)*		*α-linolenic (18:3n-3)*		*Eicosapentaenoic (20:5n-3)*		*Docosahexaenoic (22:6n-3)*	
		mg/ serve	mg/ 100g	mg/ serve	mg/ 100g	mg/ serve	mg/ 100g	mg/ serve	mg/ 100g	mg/ serve	mg/ 100g
Biscuit[a]	Intervention	2.04	5.43	0.45	1.19	0.09	0.23	0.05	0.14	0.07	0.20
(2 biscuits ~ 40g)	Group	1.95	5.19	0.44	1.16	0.08	0.21	0.00	0.00	0.00	0.00
Bread	Intervention	0.53	0.63	0.71	0.85	0.06	0.07	0.05	0.06	0.07	0.09
(2 slices ~ 90g)	Group	0.44	0.52	0.70	0.83	0.05	0.06	0.00	0.00	0.00	0.00
Cheese spread	Intervention	2.30	9.19	1.20	4.80	0.29	1.15	0.05	0.21	0.07	0.30
(25g)	Group	2.21	8.84	1.19	4.76	0.28	1.12	0.00	0.00	0.00	0.00
Chocolate	Intervention	2.01	8.37	0.19	0.79	0.03	0.11	0.05	0.22	0.07	0.31
(25g)	Group	1.92	8.00	0.18	0.75	0.02	0.83	0.00	0.00	0.00	0.00
Dip[a]	Intervention	1.12	3.69	1.23	4.10	0.13	2.34	0.05	0.18	0.07	0.25
(30g)	Group	1.02	3.40	1.22	4.07	0.12	0.40	0.00	0.00	0.00	0.00
Egg	Intervention	2.41	2.68	1.06	1.18	0.63	0.70	0.00	0.00	0.15	0.17
(2 ~ 90g)	Group	4.35	4.83	1.00	1.11	0.02	0.02	0.00	0.00	0.04	0.04
Margarine	Intervention	5.53	27.65	2.19	10.96	0.97	4.85	0.05	0.24	0.07	0.36
(20g)	Group	5.42	27.10	2.18	10.90	0.96	4.80	0.00	0.00	0.00	0.00
Milk	Intervention	0.98	0.39	0.06	0.02	0.04	0.02	0.05	0.02	0.07	0.03
(250ml)	Group	0.89	0.36	0.05	0.02	0.03	0.01	0.00	0.00	0.00	0.00
Muesli	Intervention	0.95	2.11	0.91	2.02	0.07	0.15	0.05	0.12	0.07	0.16
(30g)	Group	0.86	1.91	0.90	2.00	0.06	1.33	0.00	0.00	0.00	0.00
Muffin	Intervention	0.23	0.46	0.39	0.78	0.07	0.14	0.05	0.11	0.07	0.15
(1 muffin ~ 75g)	Group	0.14	0.28	0.38	0.76	0.06	0.12	0.00	0.00	0.00	0.00
Oat cereal	Intervention	0.72	1.80	0.15	0.38	0.03	0.07	0.05	0.13	0.07	0.18
(30g)	Group	0.63	1.58	0.14	0.35	0.02	0.05	0.00	0.00	0.00	0.00
Pancake	Intervention	0.85	0.77	1.13	1.03	0.08	0.07	0.05	0.05	0.07	0.07
(3 pancake ~ 30g)	Group	0.76	0.69	1.12	1.02	0.07	0.06	0.00	0.00	0.00	0.00
Salad dressing	Intervention	5.17	12.94	4.95	12.37	1.13	2.82	0.05	0.13	0.07	0.18
(40ml)	Group	5.09	12.73	4.94	12.35	1.12	2.80	0.00	0.00	0.00	0.00
Salsa	Intervention	0.56	1.86	1.32	4.40	0.22	0.73	0.05	0.18	0.07	0.25
(25g)	Group	0.47	1.57	1.31	4.37	0.21	0.70	0.00	0.00	0.00	0.00
Soup[a]	Intervention	1.02	1.20	0.99	1.16	0.04	0.44	0.05	0.60	0.07	0.09
(1 sachet ~ 85g)	Group	0.94	1.10	0.98	1.15	0.03	0.04	0.00	0.00	0.00	0.00

[a]Mean value of two separate varieties within this category

At baseline, at 3-months and at the end of the 6-month intervention, fasting blood samples were taken by venipuncture for erythrocyte fatty acid and lipid compositional analysis. Height, weight, waist circumferences (at natural waist when clearly identifiable or midway between the lower rib and iliac crest) and hip circumference (at greater trochanter) were measured at each of the three time points.

5.2.3 Dietary Measurement

A modified Burke style DH (Appendix I) (351) was administered to each subject by trained dietitians using a standardised tool at baseline, 3-months and 6-months. Dietitians asked subjects to describe a typical day's intake, as well as amounts, frequencies and variations in consumption. Quantities were assessed using familiar volumes, dimensions or purchasing units, or with food models representing volumes and serving sizes. A standardised questionnaire was then used to gather information about the types of meals consumed, the frequency of meals, eating out and formal entertaining. A daily checklist of foods and brands was also used to identify or clarify intakes of fatty foods and foods containing n-3 fatty acids. Using each of these tools, a 7-day pattern of consumption was constructed. In addition, all subjects, prior to the DH at baseline, completed FR (Appendix J) after 3-months and 6-months. Subjects kept records of all foods consumed over a period of three days including two weekdays and one weekend day. All foods were either weighed by the subjects or listed in common serving or package sizes. Subjects were instructed to be as detailed as possible when reporting food items, weights and sizes. FR was checked with each subject by a different dietitian to that which

performed the DH. Analysis of the DH and FR were performed by an independent dietitian. Both methods were assessed against erythrocyte fatty acid levels with strong correlations (particularly with the DH) achieved for EPA, DHA, total LCn-3 and total n-3 at 3 and 6-month time points. As additional measure of compliance each subject was asked to record the number of study food portions consumed per day and the average weekly portions consumed were calculated for each subject. At the conclusion of the study participants were asked to rate the taste of each product on a 7-point Likert scale ranging from -3 (extremely dislike) to +3 (extremely like) (Appendix K).

DH and FR were entered into the Foodworks™ nutrient analysis software package (Xyris Software, Highgate Hill, Brisbane Australia, Professional Version 3.2,2002), using the Australian nutrient database AusNut Rev 0.14 (AusNut 2000, Department of Human Services and Health, Canberra) and the Australian Fatty Acids Rev 0.6 (Royal Melbourne Institute of Technology, 2002) to produce values for energy and macronutrient intakes and fatty acids. All study foods (controls and intervention) were entered into the Foodworks database and nutrient content analysed through recipe analysis using product information and analytical data from microencapsulated fish oil powder. Additional foods from interviews that were not found in the database, were substituted with equivalent food items (352).

5.2.4 Laboratory procedures[5]

Fasting venous blood was collected in EDTA tubes and centrifuged at 4000rpm for 10 minutes at 4°C to separate erythrocytes from plasma. Subsequently, erythrocytes were washed twice with isotonic saline and stored at -80°C prior to fatty acid extraction. Erythrocytes membrane lipids were extracted into methanol: toluene (4:1, by vol.) and transmethylated according to the method of Lepage & Roy (1986). Gas Chromatographic analyses of fatty acid methyl esters (FAME) were performed using a Shimadzu GC 20A (Shimadzu Corporation, Kyoto, Japan) fitted with a FID and a 50m BPX70 cross-linked 70% cyanopropyl polysilphenylene-siloxane capillary column (0.32 mm ID and 0.25 μm film thickness, SGE, Australia). Samples were injected at 125°C and held for 1 minute. Oven temperature was set to increase in a step-wise manner to a final temperature of 220ºC. The injector and detectors were maintained at 260ºC and hydrogen was used as the carrier gas. Peak areas and fatty acid percentages were quantified on an IBM compatible workstation using Shimadzu software. Individual fatty acids were identified by comparison with known FAME standards (Nuchek Prep Inc, USA). Results are expressed as percent of total fatty acids.

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[5] This analysis was conducted by researchers at the University of Adelaide. Results obtained were correlated with dietary measurements by CP.

5.2.5 Data Analysis

The statistical analysis focused on changes in dietary variables at three time points, baseline, 3-month and 6-month. Effects of treatment on dietary intake over time were assessed by repeated measures ANOVA based on an intention to treat model of analysis. Demographics and dietary intakes of treatment and control groups were compared using one-way ANOVA. Non-parametric analysis was conducted using the Kruskall-Wallis test.

Differences between DH and FR data were assessed using Student's *t*-tests and Pearson's correlation coefficients were estimated to view relationships. The plausibility of estimated intake (EI) was tested against energy expenditure (EE). As EE was unavailable, we used the EI:basal metabolic rate (BMR) ratio and compared this to EE:BMR or physical activity level (PAL). Comparing the sample EI: BMR ratio to the PAL level at which life is not sustained, the level of underreporting for both individuals and groups could be determined. The calculations were derived by Goldberg et al (353) and Black et al (354). Underreporting of energy was determined by taking a group mean reported energy intake (EI_{rep}) to basal metabolic rate (BMR) ratio and applying the Goldberg cut-off limits of 1.52 for diet histories and 1.50 for food records. These Goldberg cut-off were determined using the equation suggested by Black *et al* (354), using the group sample size, number of days dietary reporting as well as a physical activity level (PAL) value. The sample size of n = 86, 86 & 74 was used for the time points 0, 3 & 6-months respectively. The number of days was 3 for food records and 14 for diet history. Using the World Health Organisation (WHO) recommendations for

energy requirements (355) we made a judgment that a PAL of 1.55 would be used, based on self reported exercise frequency, duration and intensity.

To assess the quality of dietary data, data obtained from diet history interviews and food records were compared at 0, 3 and 6 months using Bland-Altman plots (343). The ranges for the limits of agreement were set at 840kJ, 2% energy and 1g for reported energy, macronutrients and total n-3 fatty acids respectively. Evidence of systematic bias was observed if a significant correlation was found between the difference (DH-FR) and mean (DH-FR/2) values. SPSS V11.0 (Version 11.0.0; 19/09/01, SPSS Inc., Chicago, IL, USA) was used for all statistical analyses, with a statistical significance level of $P < 0.05$.

5.3 Results

Eighty six men and women commenced the trial. The intervention and control groups were well matched for weight, height, BMI, age, daily total energy, protein, fat and carbohydrate intake (Table 5-2). There were no withdrawals at 3-months whereas at 6-months 12 withdrew (8 in the intervention and 4 in the control group), leaving a group randomisation of 36 in the control group and 38 in the intervention group. Body weight increased significantly in both groups at the end of 6-months by 2.7kg $\pm$ 4.7 in the intervention group and 1.4kg $\pm$ 2.4 in the control group ($P < 0.001$), with no difference detected between the groups.

Table 5-2 Baseline characteristics

	Control (n = 22 M, 22 F)	**Intervention (n = 19 M, 23 F)**	**P-value**
Age (y)	50.2 ± 9.4	50.4 ± 14.5	0.943
Height (cm)	170.4 ± 9.8	172.3 ± 10.9	0.379
Weight (kg)	94.6 ± 17.2	93.0 ± 17.6	0.667
BMI (kg/m^2)	31.9 ±4.4	31.2 ± 5.2	0.242
Waist-to-hip ratio	0.91 ±0.08	0.93 ±0.09	0.495
Total Energy (kJ/day)	11244.2 ±3136.6	11657.9 ±3805.8	0.574
Protein (g/day)	115.0 ±31.6	117.3 ±33.2	0.746
Fat (g/day)	96.6 ±34.0	103.6 ±34.3	0.333
Carbohydrate (g/day)	322.9 ±105.0	337.6 ±135.5	0.565

5.3.1 Dietary data

Dietary intakes of macronutrients and key fatty acids were estimated using the DH interview data (Table 5-3). Protein as a percent of energy intake, alcohol and fibre intake decreased over the course of the study, but there were no significant differences between the two groups. There were no significant changes in total fat (%E), SFA (%E) and MUFA (%E) after 6-months but there was a significant difference between the groups at each time point. Dietary intake of PUFA (%E) increased in the treatment group (≈12%) whereas it decreased in the control group (≈5.5%). AA intake was significantly different between groups, but this did not change over time, nor was there a significant interaction between the two groups.

There were significantly different changes in intake between the control and intervention for ALA (≈75%, 25%; $P < 0.01$), EPA (≈490, -58%; $P < 0.001$), DHA (≈370%, -60%; $P < 0.001$), LCn-3 (≈365%, -55%; $P < 0.001$), n-6:n-3 ratio (≈-51%, -11%; $P < 0.001$) and total n-3 (≈122%, 9%; $P < 0.001$) respectively.

Table 5-3 Subjects' daily intake as estimated by diet history analysis Mean (± s.d.)

	Baseline	3 months	6 months	P-value
Total energy (kJ)				0.910 [a]
Intervention	11716.0 ±3912.0	11132.6 ±3107.8	11473.7 ±2764.7	0.760 [b]
Control	10946.6 ±2922.7	11539.7 ±2703.2	11268.0 ±2592.0	0.419 [c]
Carbohydrate (% E)				0.845 [a]
Intervention	45.54 ±6.02	46.11 ±4.81	44.74 ±5.86	0.115 [b]
Control	46.97 ±6.44	47.21 ±5.76	47.44 ±6.44	0.448 [c]
Protein (%E)				0.050 [a]
Intervention	16.56 ±2.58	15.70 ±2.18	16.14 ±2.10	0.774 [b]
Control	16.60 ±2.53	15.54 ±1.78	15.90 ±2.18	0.622 [c]
Alcohol (%E)				0.071 [a]
Intervention	7.96 ±15.18	7.35 ±13.45	6.77 ±14.41	0.437 [b]
Control	11.03 ±15.14	9.61 ±14.74	8.85 ±11.62	0.595 [c]
Fibre (g)				0.038 [a]
Intervention	33.33 ±11.8	28.00 ±8.5	29.92 ±8.3	0.428 [b]
Control	32.33 ±7.9	32.50 ±9.6	30.44 ±8.3	0.542 [c]
Cholesterol (mg)				0.071 [a]
Intervention	360.5 ±325.9	392.3 ±159.8	402.0 ±141.2	0.722 [b]
Control	324.9 ±122.3	342.2 ±102.0	383.8 ±112.8	0.330 [c]
Fat (% E)				0.613 [a]
Intervention	33.20 ±5.80	32.79 ±5.58	34.00 ±5.07	0.005 [b]
Control	30.58 ±5.86	30.98 ±5.53	30.62 ±5.54	0.656 [c]
SFA (% E) [‡]				0.509 [a]
Intervention	12.93 ±3.03	12.70 ±1.46	12.86 ±2.26	0.012 [b]
Control	11.29 ±3.26	11.92 ±2.28	11.87 ±2.31	0.400 [c]
MUFA (% E)				0.084 [a]
Intervention	12.45 ±2.89	12.66 ±2.00	13.41 ±2.47	0.049 [b]
Control	11.48 ±2.92	12.18 ±2.77	12.05 ±3.08	0.659 [c]
PUFA (% E)				0.466 [a]
Intervention	5.03 ±1.56	5.53 ±0.91	5.65 ±1.32	0.046 [b]
Control	5.07 ±1.59	5.12 ±1.37	4.79 ±1.18	0.055 [c]

PUFA:SFA ratio				0.412 [a]
Intervention	0.413 ±0.17	0.440 ±0.09	0.451 ±0.13	0.499 [b]
Control	0.500 ±0.27	0.442 ±0.12	0.419 ±0.14	0.025 [c]
n-6:n-3 ratio				0.000 [a]
Intervention	8.88 ±4.2	3.43 ±0.8	4.31 ±1.6	0.000 [b]
Control	8.57 ±3.6	6.40 ±1.6	7.59 ±2.4	0.001 [c]
LA				0.722 [a]
Intervention	12.92 ±5.59	12.79 ±4.46	14.07 ±5.16	0.470 [b]
Control	12.46 ±5.56	13.52 ±4.83	11.93 ±4.36	0.336 [c]
AA				0.261 [a]
Intervention	0.14 ±0.08	0.10 ±0.06	0.14 ±0.07	0.035 [b]
Control	0.14 ±0.12	0.14 ±0.08	0.18 ±0.07	0.247 [c]
ALA				0.000 [a]
Intervention	1.34 ±0.69	2.60 ±1.19	2.33 ±1.18	0.011 [b]
Control	1.25 ±0.60	2.11 ±1.09	1.56 ±0.65	0.011 [c]
EPA (mg)				0.000 [a]
Intervention	73.0 ±45.8	474.0 ±173.6	428.2 ±140.0	0.000 [b]
Control	83.0 ±70.1	49.0 ±48.7	35.3 ±28.6	0.000 [c]
DHA (mg)				0.000 [a]
Intervention	144.0 ±120.1	775.1 ±296.2	675.7 ±239.8	0.000 [b]
Control	186.6 ±166.7	94.3 ±78.8	80.5 ±74.8	0.000 [c]
LCn-3 (mg)				0.000 [a]
Intervention	258.2 ±179.7	1339.6 ±503.0	1195.7 ±401.0	0.000 [b]
Control	313.4 ±248.9	177.6 ±137.3	146.3 ±119.2	0.000 [c]
Total n-3 (g)[§]				0.000 [a]
Intervention	1.59 ±0.67	3.94 ±1.55	3.53 ±1.48	0.000 [b]
Control	1.57 ±0.63	2.28 ±1.16	1.71 ±0.68	0.000 [c]
Total n-6 (g)				0.695 [a]
Intervention	13.08 ±5.62	12.94 ±4.52	14.25 ±5.18	0.346 [b]
Control	12.63 ±5.62	13.68 ±4.87	12.14 ±4.39	0.484 [c]

Values significant at $P < 0.05$; [a] significant differences observed over time, [b] significant differences observed between treatment group, [c] significant difference with time and treatment group.

[‡] SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated acids; LA, Linoleic acid, AA; Arachidonic acid; ALA, α –linolenic acid; EPA eicosapentaenoic acid; DHA, docosahexaenoic acid. § Includes ALA + EPA + DPA (Docosapentaenoic acid, 22:5 n-3) + DHA.

5.3.2 Relative Validity

Energy reported using DH correlated significantly with that reported in the FR ($r = 0.55$, $P < 0.001$) at baseline. Significant correlations were also found between the DH and FR for all macronutrients and fatty acids (with the exception of PUFA (%E) and n-6:n-3 ratio at baseline) at all time points, including protein (%E), carbohydrate (%E), fat (%E), SFA (%E), MUFA (%E) and PUFA (%E) as well as the P:S ratio, n-6 PUFA, n-3 and EPA + DHA. Correlation coefficients are listed in Table 5-4.

Table 5-4 Correlation coefficients of dietary variables using the diet history and 3-day food record

	Baseline	3 month	6 month
Total energy (kJ)	0.55**	0.70**	0.64*
Carbohydrate (% E)	0.56**	0.55**	0.48**
Protein (%E)	0.24*	0.34**	0.48**
Alcohol (g)	0.71**	0.86**	0.71**
Fibre (g)	0.49**	0.49**	0.42**
Fat (% E)	0.50**	0.49**	0.38**
SFA (% E)‡	0.59**	0.35**	0.35**
MUFA (% E)	0.36**	0.40**	0.29*
PUFA (% E)	0.18	0.52**	0.33**
PUFA:SFA ratio	0.60**	0.38**	0.32**
n-6 (g)	0.40**	0.47**	0.22
n-3 (g)	0.28**	0.65**	0.62**
EPA + DHA (g)	0.35**	0.82**	0.71**
n-6:n-3 ratio	0.13	0.68**	0.59**

Pearson's correlation coefficient; * P < 0.05; **P < 0.01.

‡ SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated acids; EPA eicosapentaenoic acid; DHA, docosahexaenoic acid.

5.3.3 Underreporting

Underreporting was more common in FR than DH. However, both methods reported plausible energy intakes when compared to the Goldberg cut-off at all time points (Table 5-5).

Table 5-5 Group mean reporter EI_{rep}:BMR and Goldberg cut-off as an indicator of under reporting

Record	Time-point (month)	Group Mean EI_{rep}:BMR	Goldberg cut-off
DH	0	1.84	1.52
FR	0	1.60	1.50
DH	3	1.89	1.52
FR	3	1.65	1.50
DH	6	1.83	1.52
FR	6	1.57	1.50

5.3.4 Bias

No systematic bias was detected using Bland-Altman plots for all macronutrients and fatty acids, and at all time points. Only baseline data for total fat and LCn-3 are presented (Figures 5-1 & 5-2). The DH data compared well with FR as the mean difference between the two methods was close to zero for each variable, and the range for the limits of agreement lay within the acceptable values. Site differences for each dietary assessment method were determined and showed that BMI was significantly different between the sites at baseline (P = 0.024) and 3-months (P =

0.048). After adjusting for BMI, only site differences were observed for the 3-month assessment of total n-6 (P = 0.031) and total n-3 (P = 0.034) using the DH.

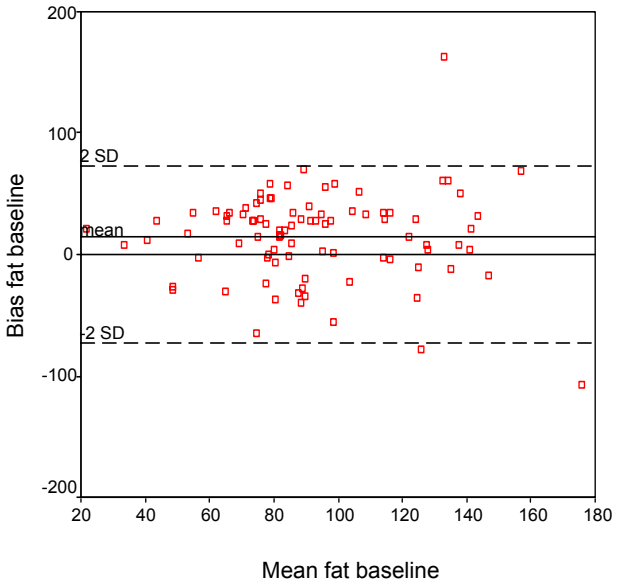

Figure 5-1 Bias of fat versus mean fat (g/day) intake at baseline

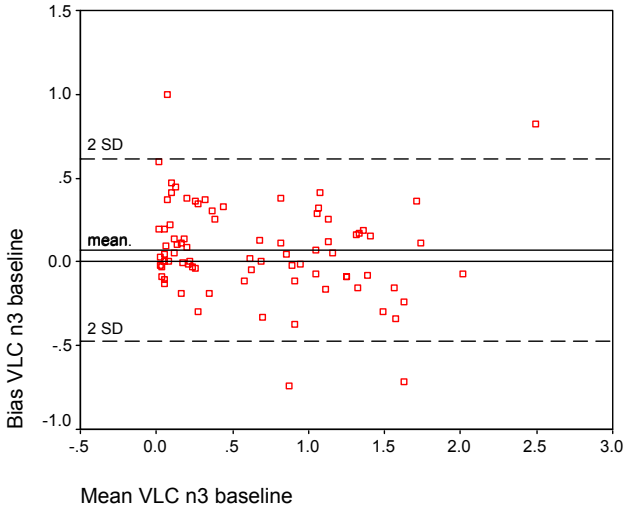

Figure 5-2 Bias of LCn-3 versus mean intake (mg/day) at baseline

5.3.5 Compliance

Daily log books revealed the consumption of study foods decreased over time (Figure 5-3). There was no significance between the groups, with both groups consuming on average between 6 and 7.5 serves of enriched foods per day (~750mg – 940mg LCn-3 per day)

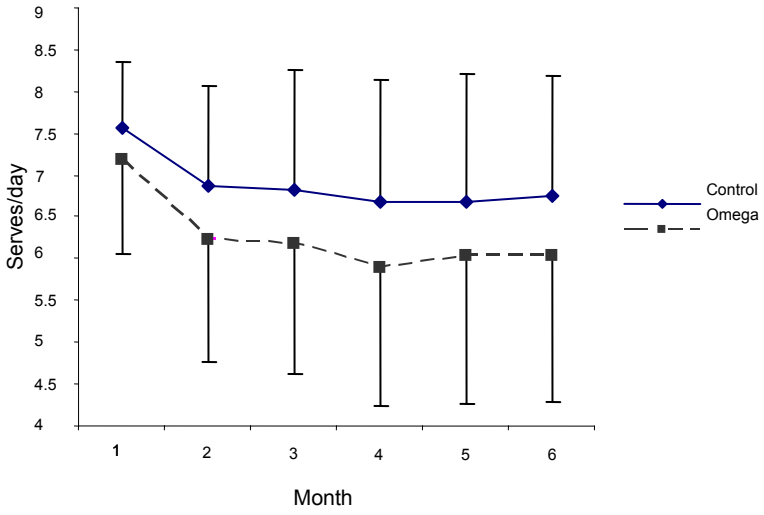

Figure 5-3 Daily mean (± s.d.) number of study food serves consumed

Values significant at $P < 0.05$; * significant differences observed over time, but not between groups using repeated measures ANOVA.

5.3.6 Food pattern analysis

Table 5-6 shows the contribution of different food groups to the overall LCn-3 intake. After 6-months, the major sources of LCn-3 in the intervention group were enriched milk, cereal and bread. All other enriched products combined contributed

a further 28.2% of the total LCn-3. In contrast, the major sources of LCn-3 in the control group were fish (fatty and white) (approximately 1 serve per week) and meat.

Table 5-6 Major food group contributors to overall LCn-3 intake (% contribution to total LCn-3)

Baseline (n = 86)		**6-month (n = 74)**	
Control	**Intervention**	**Control**	**Intervention**
Fatty fish (36.7%)	Fatty fish (52.3%)	Fatty fish (32.4%)	Enriched milk (13.8%)
Chicken (19.3%)	Meat (12.6%)	White fish (22.8%)	Enriched cereal (12.1%)
Meat (14.8%)	White fish (12.1%)	Meat (19.3%)	Enriched bread (11.3%)
			Fatty fish (9.9%)
			Enriched biscuit (8.1%)
			Enriched chocolate (7.5%)
			Enriched dip (6.4%)
			Enriched margarine (6.3%)
Total - 70.8%	**Total - 77.0%**	**Total - 74.5%**	**Total – 75.4%**
LCn-3 intake – 0.3g/d	LCn-3 intake – 0.3g/d	LCn-3 intake – 0.1g/day	LCn-3 intake – 1.2g/day

5.3.7 Food preferences

At the conclusion of the study, participants in the intervention group rated the oats and milk slightly favourable (1.4 and 0.5 respectively; items scored between -3 to +3) whereas they rated the bread as relatively unfavorable (0.3) which was significantly different ($P < 0.05$) from the control group (1.1). Apart from the salad

dressing, all other food items were rated as tasting favorable with no reported significant differences between the groups (Figure 5-4).

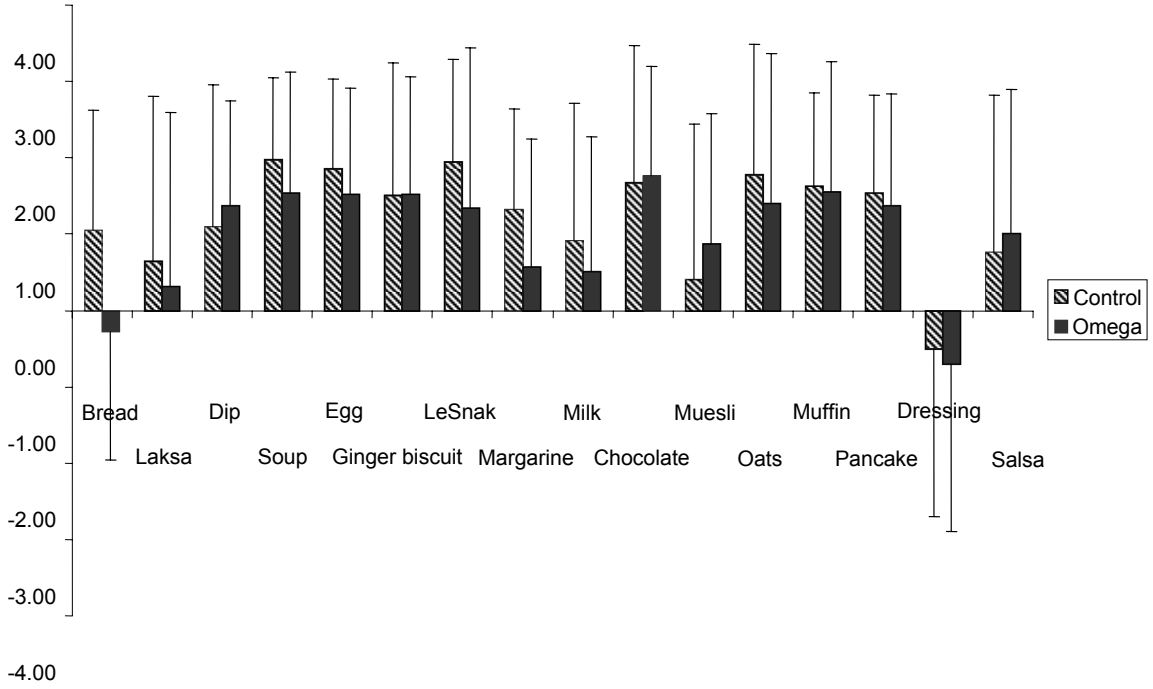

Figure 5-4 Mean (± s.d.) taste ratings of study foods (scores ranged from unfavourable (-3) to favourable (+3))

5.3.8 Dietary Biomarkers

There were significant increases in the proportions of EPA, DHA, LCn-3 and total n-3 in the intervention group by 96%, 79%, 59% and 25% respectively (Table 5-7). A significant difference in the proportion of n-6 between the groups was observed at 3-months, but not baseline or at 6-months.

Table 5-7 Plasma fatty acid biomarkers at each time point for each group (% total fatty acids)[†]

	Baseline	3 months	6 months	P-value
LA[‡]				*0.058
Intervention (n=34)	6.72 (1.37)	6.93 (0.732)	7.00 (0.71)	**0.144
Control (n=40)	6.98 (1.22)	7.24 (0.97)	7.27 (0.99)	***0.951
AA				*0.254
Intervention (n=34)	10.20 (4.61)	11.08 (2.89)	11.61 (2.33)	**0.138
Control (n=40)	11.64 (2.98)	12.24 (2.23)	11.49 (3.69)	***0.162
ALA				*0.429
Intervention (n=28)	0.390 (1.582)	0.110 (0.026)	0.111 (0.026)	**0.305
Control (n=31)	0.100 (0.030)	0.111 (0.028)	0.136 (0.076)	***0.278
EPA				*0.000
Intervention (n=35)	0.594 (0.386)	0.984 (0.382)	1.165 (0.338)	**0.000
Control (n=40)	0.617 (0.240)	0.619 (0.176)	0.683 (0.286)	***0.000
DHA				*0.000
Intervention (n=35)	3.314 (1.339)	5.058 (1.438)	5.927 (1.178)	**0.000
Control (n=40)	4.109 (1.394)	4.044 (0.913)	3.921 (1.078)	***0.000
LCn-3				*0.000
Intervention (n=35)	5.933 (2.185)	8.339 (2.101)	9.411 (1.870)	**0.000
Control (n=40)	6.824 (2.126)	6.864 (1.418)	6.708 (1.695)	***0.000
Total n-3				*0.022
Intervention (n=35)	8.93 (4.85)	10.39 (2.60)	11.16 (1.81)	**0.001
Control (n=40)	8.64 (1.86)	8.70 (1.17)	8.45 (1.55)	***0.007
Total n-6				*0.087
Intervention (n=35)	20.76 (6.40)	21.96 (3.48)	22.15 (2.68)	**0.007
Control (n=40)	22.91 (4.86)	24.00 (3.24)	24.02 (4.39)	***0.839

[†] Mean (s.d.)

[‡]LA, Linoleic acid, AA; Arachidonic acid; ALA, α – linolenic acid; EPA eicosapentaenoic acid; DHA, docosahexaenoic acid. *significant differences observed over time, ** significant differences observed between treatment groups, *** significant difference with time and treatment groups. Two-way repeated-measures ANOVA.

5.3.9 Relationship between dietary intake and dietary biomarkers

The correlation coefficients (r) between dietary intake as recorded by DH and erythrocyte membrane was significant at 3-months and 6-months for EPA, DHA, LCn-3 and total n-3 (Table 5-8). The correlation coefficients between the FR and erythrocyte membrane EPA, DHA and LCn-3 showed a similar trend, but weaker relationship. In addition the total n-3 was only correlated at 6-months and there was a small but significant correlation between dietary intake recorded by FR and erythrocyte membrane linoleic acid (LA) at 3-months.

Table 5-8 Pearson's correlation between reported dietary intake and equivalent plasma fatty acid biomarkers[†]

Variable	Baseline	3 months	6 months
LA[‡]			
Diet history	0.024 (0.825)	0.084 (0.445)	0.124 (0.328)
Food record	-0.016 (0.883)	0.335 (0.002)***	0.080 (0.506)
AA			
Diet history	0.110 (0.318)	0.172 (0.117)	0.070 (0.582)
Food record	-0.001 (0.995)	0.115 (0.308)	-0.097 (0.420)
ALA			
Diet history	-0.052 (0.672)	0.022 (0.842)	0.036 (0.785)
Food record	0.154 (0.203)	0.002 (0.985)	-0.041 (0.739)
EPA			
Diet history	0.123 (0.259)	0.463 (0.000)***	0.743 (0.000)***
Food record	-0.131 (0.228)	0.374 (0.001)***	0.555 (0.000)***
DHA			
Diet history	0.151 (0.166)	0.418 (0.000)***	0.663 (0.000)****
Food record	0.095 (0.384)	0.288 (0.010)**	0.497 (0.000)***
LCn-3			
Diet history	0.101 (0.354)	0.421 (0.000)***	0.641 (0.000)***
Food record	0.050 (0.645)	0.305 (0.006)***	0.450 (0.000)***
Total n-3			
Diet history	-0.164 (0.132)	0.341 (0.001)**	0.515 (0.000)***
Food record	-0.018 (0.871)	0.202 (0.720)	0.266 (0.240)*

[†] Mean (s.d.). [‡]LA, Linoleic acid, AA; Arachidonic acid; ALA, α – linolenic acid; EPA eicosapentaenoic acid; DHA, docosahexaenoic acid. * P < 0.05; **P < 0.01; ***P < 0.001

5.4 Discussion

This study demonstrated that replacement of foods with n-3 enriched equivalent foods for 6 months significantly increased total n-3, LCn-3, EPA and DHA intake as verified by analysis of FR and DH completed by participants. These findings may be important as an increased n-3 fatty acid intake has been associated with a substantially reduced risk of death in subjects with existing coronary heart disease (CHD) (82, 108) and epidemiological evidence has demonstrated a reduced risk of CHD mortality in those who eat fish regularly (79, 95, 99, 100). With a large number of the population unable to consume enough of these essential fatty acids from fish alone (18), enriched processed foods may have positive public health implications. In addition to the total intake of LCn-3, the ratio of n-6:n-3 is also important as it may be associated with improved health outcomes (347, 356) whereas excess amounts of LA and associated eicosanoids may promote thrombosis and inflammation (160). This study showed a reduction of the ratio of n-6:n-3 to 3:1 and increased the daily intake of n-3 in the intervention group. In addition there was no alteration of the PUFA, MUFA, SFA or the PUFA:SFA ratio which are also important when considering the overall dietary affect on heart health risk (14).

Detailed analysis of the dietary data indicated a sense of confidence in these findings. Strong positive correlations were found between both measures of dietary intake for energy and major macronutrient intakes reported using DH and FR and within the range of coefficients found in other studies of similar-aged subjects (309,

357, 358). The weakest, although still significant, correlation was found in reported intakes of PUFA. It can be speculated that this weak correlation was due to over-reporting of PUFA in the DH as this was a focal point of questioning. Subjects were asked specific questions about intakes of foods that were good sources of PUFA, and may have over-reported their intakes as they interpreted these to be healthy or desirable foods (359). Although significant correlations can suggest that two dietary assessment methods possess relative validity, there is still considerable unexplained error within each of the methods. Correlations are used to demonstrate the strength of association between two methods, but provide no guide as to the accuracy or precision of the methods (359).

Both over- and underestimation has been reported in other studies (307). The level of underreporting in this study was considerably smaller than, or similar to, that found in previous studies (309, 314, 315, 357, 360-362). The proportion of under-reporters was considerably less in the DH than the FR, indicating that the DH is a better method of estimating energy intake in this study population. Under-consumption may be more likely than underreporting in the FR, since recording all food consumed for more than one day is known to modify behaviour to reduce intake (307). However, while some studies show that the DH over-estimates intake relative to the FR, others have shown using metabolic studies that both the DH and the FR underestimated intake (313). Rothenberg *et al* (1998) showed that energy expenditure reported in activity diaries matches energy intake reported in DH, but doubly labeled water tests concluded that both must have been underreported (363). This may explain why we observed a significant weight gain over the study

period with no apparent increase in reported energy intake. Both methods may have overestimated intake of LCn-3. A dose response study into EPA + DHA intake and subsequent rises in erythrocyte fatty acids found that this index rose from 4.7 ± 0.9% to 7.9 ± 1.7% with supplementation of 0.5g/day ($P < 0.001$), to 9.9 ± 2.9% with 1g/day ($P < 0.001$), and 11.6 ± 2.4% with 2g/day ($P = 0.02$) (9). Based on the estimated dietary intake of 1.2g/day an expected increase in erythrocyte fatty acid level of > 8% would be expected, whereas an increase of 7.1 ± 1.9% was reported. Results from the food logs also suggest that the intake of LCn-3 may have been lower.

There are other components of error relevant to the DH and FR in an intervention trial in addition to under-reporting. These include random error, error in portion size estimates and method-related bias resulting in consistent over- or under-representations (359). Method-related bias was addressed using Bland-Altman plots. No systematic bias was found in this study, indicating that any misreporting was random in nature and does not indicate specific error in reporting of macronutrients in the DH relative to the FR. This may in part be attributed to standard study food portion sizes as well as using well-trained research dietitians to collect the data. In addition, apart from n-6 and n-3 intake at 3-months, there were no significant differences between the sites in reported intake using the DH after adjusting for BMI. Others have reported that the diet history is subject to observer bias, with the bias appears to be macronutrient specific with energy, fat and SFA consistently underreported (310).

The question of validity can be addressed by comparing both dietary assessment methods to fatty acids accumulated in the erythrocytes. The correlation between the dietary intakes measured by DH of EPA, DHA, LCn-3 and Total n-3 and the equivalent erythrocyte fatty acid was stronger that that observed using the FR. This relationship ranged from 0.34-0.46 at 3-months to 0.52-0.74 at 6-months for the DH and 0.29-0.37 at 3-months to 0.27-0.55 at 6-months for the DH. This is the first study to investigate the relationship of the DH with erythrocyte fatty acids. Parra *et al* (2000) exploring the relationship between FFQ and usual dietary patterns in Mexican women reported modest correlations with erythrocyte ALA, DHA and EPA against their dietary counterparts (r = 0.32, 0.35 and 0.36 respectively) (322). Others have investigated the relationship between FR and serum fatty acids for LCn-3 with reported correlations of between 0.49-0.75 (364, 365), and between FFQ and serum fatty acids for LCn-3 (r = 0.49-0.51) (366, 367). Results at 3 and 6-months are comparable with these studies; however no significant correlations at baseline were found. Study design and population characteristics may explain these differences. Anderson *et al*, reported strong correlations on LCn-3 at baseline using a larger sample than ours (n = 579) men and women (365), whereas cross sectional studies reporting strong correlations were conducted in Japan where fish intake is habitually high (364, 366, 367). In this study, baseline habitual intake of LCn-3 (~200mg/day) was not correlated to the equivalent erythrocyte fatty acid, however once the LCn-3 enriched study foods were added to the diet this relationship emerged. Finally, there is an apparent anomalous correlation with LA to the equivalent fatty acid in the erythrocyte membrane at 3-months as reported by the FR which may be explained by the difference in dietary

assessment methods or a statistical artifact. Interestingly, this result was mirrored by an equally anomalous absence of correlation for total n-3 using the same assessment method at the same time point. It is important to be aware that the biochemical marker used can be confounded by the fact that they reflect relative percentage and not absolute amounts (322). The method of collection, sampling site and analytical technique may also influence the results (322).

Through the consumption of enriched novel foods an increase in EPA + DHA levels from 4.09% at baseline to 7.10% was observed. Harris and von Shacky propose that a combined EPA + DHA level of ≥ 8% is associated with the greatest cardioprotection, compared to ≤4% (9). However, an unintended negative impact of this study was the increase in body weight in the intervention group. Weight gain has the potential to worsen all elements of the cardiovascular risk profile, including blood pressure, dyslipidemia, glucose tolerance, left-ventricular hypertrophy, hyperuricaemia, and fibrinogen levels (368). As the design of this study was to simply replace food items in an *ad libitum* fashion, 'whole of diet' advice was not provided upfront, and only discussed once any weight gain was detected. A whole of diet approach considers advice of the number of portions of all food groups and may be necessary to ensure energy balance as well as controlling for the background diet in supplemental studies (369). The finding reinforces the need for caloric consideration in the design of novel foods as well as education about how the whole diet may emerge when such foods are incorporated.

This study also provides an insight into how consumers may use novel enriched foods. Although providing a wide range of food products, we found that staple

foods such as milk, cereal and bread were the most significant contributors to the overall LCn-3 intake in the intervention group. This finding is consistent with qualitative research suggesting that consumers prefer staple foods as potential vectors for this functional ingredient (370). Of particular note was the finding that participants in the intervention group rated the taste of the bread as being unfavourable, with comments suggesting that it tasted ‘fishy’. Despite this, the enriched bread was the third most significant contributor to the increase in LCn-3 intake and suggests that improving the taste of this product has the potential for it to be an important vector for delivery of LCn-3.

In conclusion, the dietary intakes of LCn-3 were significantly increased, reaching recommended targets (145), through the use of enriched novel foods in overweight subjects. The validity of the dietary data was confirmed; however unintended weight gain has the potential to nullify any positive cardiovascular benefits of an increase in dietary LCn-3. The ability to extrapolate results from these studies to a population approach is limited by the controlled nature of the study design. Therefore, the compliance levels observed in this study may not be achieved outside of the confines of such a controlled environment highlighting the need to study consumer behaviour. Therefore, an investigation into consumer beliefs and attitudes about n-3 functional foods will be presented.

5.5 Acknowledgements

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CHAPTER 6

CONSUMER BELIEFS ABOUT FUNCTIONAL FOODS INFLUENCE UPTAKE[6]

6.1 Introduction

For n-3 functional foods to be effective in the primary prevention of CHD widespread consumer acceptance and dietary inclusion would be required. To date the understanding of consumer beliefs and attitudes towards this concept has received very little research effort. This is confounded by the fact that there is no universally accepted definition for functional foods and they seem to fall into a grey area, somewhere between foods and medicines, with the precise boundaries on either side far from clear (371). From the viewpoint of nutrition education, the functional food category does not fit straightforwardly into any of the long-established food educational models and there are concerns relating to consumer acceptability and perception (25). With evidence showing that food choice affects dietary quality as measured by intake (372, 373), it seems appropriate that greater

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[6] *A significant portion of this chapter is about to be published in the following peer reviewed journal:*

Patch CS, Tapsell LC, Williams PG. Overweight consumers' salient beliefs on omega-3 enriched functional foods in Australia's Illawarra region. Journal of Nutrition Education and Behavior. 2005;37:83-89.

CP was responsible for the design of the study, organisation and leading the study, data collection and analysis, critical discussion of the analysis and preparation of the manuscript. LT and PW were responsible for critical discussions of the study design, critical discussions of the analysis and discussions of the manuscript.

attention be given to the factors that facilitate or constrain the use of functional foods.

The field of behavioural psychology suggests that the best predictor of human behaviour is a person's conscious decision to perform the behaviour. The Theory of Planned Behaviour (TPB) provides a model that can help explain consumers' purchase intentions to use functional food products. A person's behavioural intention is determined by the attitude they hold towards the behaviour, the degree of social pressure felt by the person to perform or not perform the behaviour, and the degree of control that the person feels they have over performing the behaviour (19). These three factors in turn are determined by a number of beliefs and subsequently how each is evaluated. It has been demonstrated that the TPB, or modified versions of it, is applicable in explaining consumers' food choice (20-22), including supplement use (23) and genetically modified products purchasing (24). However, to date this model has not been applied to understanding consumer behaviour regarding the use of functional food products.

Focus groups can be used to access a diverse range of opinions and viewpoints and determine salient beliefs. These may then be tested on a larger sample in quantitative surveys to access their generalisability. A focus group discussion is defined as "a carefully planned discussion designed to obtain perceptions on a defined area of interest in a permissive, non threatening environment" (329). This approach is suggested by Ajzen & Fishbein (1980) as the method of choice to determine salient beliefs about the topic area of interest (19.). In the present study, the utility of the method was strengthened by focusing on specific products rather

than investigating functional food products in general (19.). In addition, consumers' attitude toward functional food could be expected to be influenced by general attitudes such as attitude toward technology or food neophobia (374), beliefs about the production process, perceived healthiness (288) and beliefs about the quality of the resulting product. This is particularly relevant as the range of n-3 functional foods entering the market place is increasing.

The aim of this study was to gain an understanding of the salient beliefs underlying Australian consumer attitudes and purchase intentions with regard to n-3 enriched functional foods. Using focus group discussions, consumer beliefs and attitudes were determined for the purpose of developing a survey instrument to test the generalisability of these opinions in a larger population sample.

6.2 Methods

Focus group interviews examining salient beliefs and attitudes regarding functional foods were conducted with a cross-section of adult consumers in the Illawarra region of New South Wales (NSW), Australia. Compared to the NSW average, the population residing in the Illawarra is more socially disadvantaged and hospitalisation rates from coronary heart diseases in the Illawarra were 20% higher in men and 14% higher in women than the state average in 2001 (375). The study employed a qualitative research design, with overweight participants recruited via media release (Appendix L). This group reflects a potential target population for functional foods enriched with n-3 due to their likelihood having a high cardiovascular risk.

6.2.1 Participants

Participants were recruited through general advertising in local media. Fifty potential participants expressed interest in the study and all were invited to attend one of six focus group discussions. An information sheet and a consent form were mailed to each potential participant. Each participant was offered a small payment (AUS$20) to offset any inconvenience that might have resulted from involvement in the study. Approval for the conduct of the study was provided by the University of Wollongong / Illawarra Area Health Service Human Research Ethics Committee (Appendix M).

6.2.2 Focus group procedure

The focus groups were conducted between July and August 2002. All groups were conducted in a community centre located in the Illawarra region. Focus groups were audio taped and later transcribed to allow systematic analysis of the discussion. Each focus group ran for approximately one hour and was conducted by a moderator and an observer. To ensure consistency between the groups the same trained moderator ran each group and all groups were monitored by an independent observer for quality assurance purposes (329). An interview guide was developed by the research team (CP, LT, PW) following the general guidelines provided by Krueger (1994) (329). Using the TPB theoretical framework, 10 questions were devised to determine consumer awareness, beliefs, attitude and barriers to using n-3 fatty acid functional foods (Figure 6-1). The question guide was pre-tested using undergraduate nutrition students and reviewed by the

research team. At the beginning of each session instructions were provided to the group, the purpose of the interviews was outlined and it was emphasised that there were no right or wrong answers. During the sessions, participants were encouraged to speak until no more views were expressed, and then the moderator probed and clarified points. In short, probes were primarily used to encourage participants to clarify meanings or explain points of incongruence in their answers. To introduce the concept of functional food, sample products were provided as prompts for discussion (Questions 1 – 5)-, Flora's *Pro-activ*® and Meadow Lea's *Logicol*™ (margarines with added plant sterols), Yakult's fermented milk drink (a probiotic beverage) and Buttercup's *Wonder White*® bread (with high-amylose resistant starch). Specific n-3 enriched food products were used as prompts for discussion (Questions 6 – 10) - Tip Top's *Up*™ bread and Meadow Lea's *Hi Omega*® margarine (both enriched with n-3 fatty acids).

1. Have you seen these foods and what do you think of them? [Probe: Has anyone used them and why? If no one has, do you know anyone who has? Do you think they are useful and necessary?]

2. What do you see as the difference between these foods today and the foods of yesteryear? [Probe: Do you think we should be going back to foods with fewer additives or less technology?]

3. Do you think you can improve your health by choosing foods with health benefits? Why?

4. How would you decide if you were going to use these products?
[Probe: Would you be influenced by other people's view –professional, family, and friends?]

5. Why wouldn't you use these products?
[Probe: Cost factors, availability in supermarket]

6. What do you think about this new margarine/bread (Meadow Lea n-3 and Tip Top Up)?
[Probe: Who do you think should use this?]

7. What health conditions should we be designing new foods for?
[Probe: Would it be better to add these health-promoting ingredients to food or make them into a supplement? Have you taken supplements before?]

8. We all buy food for lots of different reasons; do you specifically buy foods for your health? Why?
[Probe: increase performance, reduce cholesterol, bowel health]

9. Do you believe what you read on the labels about the products?
[Probe: Do you think they will do what they claim to do? What else would help convince you that they really work?]

10. If you were convinced that they work would you still buy these products?
[Probe: explore the 'it depends' issues]

Figure 6-1 Moderator question guide for salient beliefs focus groups

6.2.3 Data Analysis

Each focus group discussion was tape-recorded and transcribed, producing on average 30 pages of transcript. A computer software package for qualitative research analysis, NVivo 2.0 (2002, QSR International Pty Ltd), was used for data management and coding (334). Using the TPB model, transcribed conversation was categorised into one of the three areas of belief related to behaviour: *behavioural beliefs*, which are assumed to influence attitudes toward the behaviour; *normative beliefs*, which constitute the underlying determinants of subjective norms; and *control beliefs,* which provide the basis for perceptions of behavioural control. Content analysis was carried out and sub-categories were developed to capture the emerging themes. An iterative process of subcategorisation was conducted as suggested by Knodel (1993) (376). After the preliminary analysis by a single coder, two other members of the research team assessed the categorisation of conversation as well as the content of each category and sub-category. All three members of the team discussed areas of disagreement and a consensus was reached. Because the purpose of this study was exploratory, with an aim to elicit salient beliefs about functional foods, quantification (e.g. frequency counts) was not undertaken; rather, examples from the discourse are presented to illustrate the interpretive frames that emerged, along with the corresponding themes (377).

6.3 Results/Discussion

Of the 109 people who responded to media advertisements 50 agreed to attend a focus group; however 8 people failed to attend on the day leaving a final number of 42. Of those who originally expressed interest and subsequently declined to participate, the major reason cited was that they expected to be involved in a dietary intervention trial. Six focus groups were scheduled and conducted at the same venue. Each focus group included between 6-9 participants and lasted between 50 and 80 minutes. The mean age of participants was 48 years with the majority being female (65%). Fifty seven percent of participants were married, 27% single and 16% living in a live in relationship. Group characteristics are shown in Table 6-1.

Table 6-1 Focus group characteristics

	Focus group number						
	1	2	3	4	5	6	Total
Number of participants	9	9	6	7	6	5	42
Gender (n)							
Males	6	0	2	2	3	0	13
Females	3	9	4	5	3	5	29
Age (years)	49	42	47	50	51	51	48 (Mean)
Marital status (n)							
Single	2	3	3	2	0	1	11
Live in relationship	2	2	0	0	2	0	6
Married	5	4	3	5	4	4	25
Focus group duration (min)	50	80	60	65	60	55	-

6.3.1 Salient Behavioural beliefs

Table 6-2 Categories of salient behavioural beliefs related to the use of n-3 enriched functional foods

Category	Sample Quote	Speaker
Provide long term health benefits	But do you think, if we started eating healthy, things like this [omega enriched bread], as young as [name] is, then it would make a difference over time, instead of starting when you're older!	Group3, F
Help to manage chronic disease	I've got heart disease and n-3 oil is good if you have heart disease.	Group 1, F
	Well as you get older the fish oil definitely helps your arthritis. In a lot of people the fish oil helps arthritis.	Group3, M
	Because I heard it's good for asthma, arthritis…It's for a whole range [of conditions] not just for lowering cholesterol. So I am pretty happy that these are on the market now that I can give to him.	Group 2, F
	Well I know that it's a very good brain food and it keeps us functioning and the brain needs n-3. But also possibly, people that suffer or a suffering from Alzheimer's.	Group 4, M
Otherwise deliver the health benefits of fish	Because of the fact that I've got two boys that won't eat tuna - one of them will eat a bit of fish - and this is a way of supplementing that then [yeah].	Group 4, F
Have an advantage over supplement	Put it this way, I would rather not take it in tablets whatsoever, I'd rather not take any stuff like that in tablets, my wife does, but none of my children [do].	Group 5, F
May undermine the concept of a healthy diet	But we get two different messages. We get a message that everyone should be eating a balanced diet and exercise and the other message were getting is, hey, you don't need to, [because you can take this instead] yeah, because you can take this instead. So many people won't eat a balanced diet.	Group 3, F
May be at risk of overexposure or inadequate intake of a nutrient	If they're putting in enough to be beneficial [n-3], you could get to a point where you are taking too much…particularly if it's being put into nearly every food or if you can never get to a stage where you are taking too much, therefore they are not putting in enough to be useful.	Group 1, F
	I take my own supplements and that way I would know how much I am taking. I am not having my food doctored up and not knowing exactly the levels I am taking.	Group 1, F

Categories of statements displaying behavioural beliefs, or outcomes beliefs relating to using n-3 enriched foods, are outlined in Table 6-2. Most participants indicated they believed that n-3 fats were good for them, often describing them as being good for general health. In addition, there was strong agreement that most people, from children to the elderly, could gain health benefits from including more n-3 fats in the diet. When participants were asked about their beliefs about eating a diet high in n-3 (or foods high in n-3 or foods enriched with n-3) a most prevalent view was that they had a role in the prevention and treatment of heart and vascular disease. There was confusion about the mechanism of action, with a number of participants stating that n-3 fatty acids were important in lowering cholesterol; however this did not detract from the believed importance of this nutrient in the diet. In addition a number of participants were strong advocates of the benefits in ameliorating inflammatory diseases, most notably arthritis. The importance of n-3 fatty acids in improving visual function and asthma was a belief held by relatively few participants. There was a belief expressed by some individuals that n-3 fatty acids had both a brain health promoting role, as well as a therapeutic role in the treatment of Alzheimer's disease.

There was a commonly expressed view that the health benefits of dietary n-3 fats were only achievable when they were included in the diet over a long period of time. However, there was also a prevailing belief that the ongoing use of these products was largely determined by how effective these products were for the individual; that is, did they alleviate symptoms or make the individual feel better and did these benefits take long to be observed? Many people expressed the view

that functional foods enriched with marine n-3 fatty acids were a good way of delivering this nutrient in the diet, particularly as an alternative to fish, and more specifically, a fish alternative for children. In general, participants saw functional foods as a logical dietary option (e.g. fibre is good for you and children prefer white bread, therefore white high fibre bread is a sensible innovation). Also, these foods were seen to overcome other barriers to good nutrition such as access to fresh fish products.

Research in the US has suggested that CHD remains the largest health concern for consumers. Moreover, after CHD, Americans were concerned with eyesight, cancer and joint pain (378). Research has shown positive associations with beliefs about n-3 intakes and all of these health concerns (123, 379-381). These results reflect this, with participants acknowledging the wide range of health benefits of n-3 fatty acids. Despite this, participants still demonstrated some reservation over whether these health benefits could be extrapolated to novel foods enriched with this ingredient.

Some participants raised the concern that n-3 enriched functional foods may not contain enough of the functional ingredient to be effective and the dosage would need to be clearly stated for use. This was the reason that supplements had greater appeal for some over functional foods. However, the opposite was apparent when considering the nutritional needs of children. Functional foods were seen as a more practical and appropriate option than supplements for children and adolescents and the elderly, a finding that has also been reported elsewhere (382).

From the literature it is known that companies selling functional food products and nutritional supplements share a similar market place and therefore compete for market share. Reports have indicated that the market for ‘natural’ or ‘organic’ produced foods equals the growth of functional foods and may exceed it when there is a reported food scare (383). It is also known that the perceived wholesomeness of the overall product is also an important factor in food purchase behaviour (384). Previous research into health behaviours shows that consumers regard “healthy” eating as eating natural foods and “unhealthy” eating in terms of eating manufactured or processed food (384) and our study suggests this may also be the case when considering functional foods. Participants indicated that foods that were not perceived as being healthy (e.g. chocolate or soft drink) were regarded as less acceptable carriers for n-3 fatty acids despite acknowledging the logical argument for the contrary – a finding which has been reported elsewhere (382).

Overall this data showed a strong support for the concept of functional food products as a convenient and logical way to achieve a healthy diet. Some participants expressed concerns that functional foods had the potential to undermine a person’s ability to achieve a healthy diet by creating confusion about what constitutes a healthy food and that a total diet approach was preferred. Concerns were also raised about the risk of overexposure of active ingredients. For example, if recommended daily doses were provided in a typical number of daily serves, an excessive intake of the food may lead to overexposure and potentially undesirable side effects. Overall though, functional foods were viewed as an

important part of a total diet approach by providing consumers with more healthy food options.

6.3.2 Salient Normative beliefs

Table 6-3 Categories of salient normative beliefs related to the use of n-3 enriched functional foods

Category	Sample Quote	Speaker
Normative beliefs generated with reference to:		
Family members	With different products, we just see if it's one the family will like	Group 4, F
Friends	It's my best mate that told me about fish oil for arthritis.	Group 3, M
Medical practitioner	If a doctor or medical person said that it would improve my health, I'd have a serious look at it. But advertising [by] itself wouldn't influence me.	Group 1, F
Nutritionist	It's not just these people [food industry] making the claim, it's the nutritionists and all that across the country and across the world are making the same claim.	Group 2, F
Scientists	And the other thing too is who did the research? Because if they do their own research it doesn't mean that it's honest.	Group 5, F
Food Industry	There are lots of loopholes in the labelling of foods they [Food industry] can get around the legislation.	Group 5, F

Table 6-3 outlines the major normative beliefs affecting the use of n-3 fatty acid enriched functional foods as expressed by participants. Communication between family members was important in spreading the word about new or novel foods entering the market. Furthermore, the incorporation of these foods into the diet depended on overall acceptance by the family unit. Similarly the opinions

expressed by friends appeared to be an important factor in at least exposing consumers to novel food products. Medical and health professional opinion was also an important factor in influencing participants' behaviour in using functional foods as a part of an overall health strategy, with nutritionists being regarded as an independent credible source of information. Participants considered the independence of health professional advice as being very important along with a proven scientific basis to the claims, even though they found it difficult to interpret and evaluate scientific results. There was a general belief that scientists and scientific evidence was inconsistent, confusing and probably biased. These beliefs were amplified when related to functional food products and industry-sponsored research. In general participants were very skeptical of advertising campaigns as a result of being more informed and less inclined to be coerced into purchasing. Also, there was apparent distrust of labelling and the legislation controlling claims on food packages.

The normative influences on participants' use of functional foods such as family, friends, food companies, health professionals and scientists revealed in our study are consistent with the results of other studies that have investigated other more general health related behaviours (23). The high level of trust in the opinion of nutritionists compared to scientists and medical practitioners, when it came to matters of nutrition, was of interest. The major factor contributing to this view was their perceived level of independence. Conversely, food companies and their advertising were not valued very highly. However, there was acknowledgement of the need to inform the market of new food innovations through advertising.

Participants place trust in family and friends and their advice affects willingness to try a new product.

6.3.3 Salient Control beliefs

Table 6-4 Categories of salient control beliefs related to the use of n-3 enriched functional foods

Category	Sample Quote	Speaker
Salient control beliefs based on perceptions of:		
Availability at the supermarket and lack of control over purchasing	I do most of my shopping at Woolies or Coles, and if it's not there I'm not going to buy it am I!	Group 3, M
Taste/Texture	No! [I would not eat these products] because of my fish hate, I'd be turned off [laughter] Supposing I didn't have that fish taste maybe yes I'd buy it.	Group 1, F
Lack of clarity in dosage and effect	I'd like to know the difference. If I was going to start taking some n-3 tablets…I want to know what my benefits are going to be. If I don't feel any different next week, how long will it take?	Group 5, F
Lack of time to read labels	I honestly don't have time to get there and read labels and decipher all this stuff as you say and every time you buy a product…	Group 1, F
Cost	…a discussion came up about them [functional foods] at work day, and one of the comments was the cost, because they are expensive and a lot of people because of their financial situation have to take that into consideration [mmm]…I think with the food supply that if it is generally proved to do what they claim then the next avenue to go would be to lower them down to everybody's reach. If that's their claim, that they're doing it not for profit or only partially for profit and for the good of peoples health and the average family, they should lower it down to everybody's reach	Group 1, M

Stated control beliefs, or factors out of the control of participants that could be barriers to the purchase of functional foods, are listed in Table 6-4. It has been shown that socially disadvantaged families disproportionately live in areas where there are fewer large supermarkets (385). Other food outlets typically stock a limited range of foods where prices are higher thus limiting access to functional foods (385). However, there was little evidence that this is a real concern held by the participants. Likewise, lack of control over purchasing is another potential barrier, but only minor reference was made to this as a legitimate concern for individuals within the groups. Taste and texture was fundamentally important to all food purchasers. There was no evidence to suggest that participants would trade taste for health and they expressed the view that if the tastes weren't at least equal to comparable products then they would not buy them, which is consistent with many studies investigating dietary habits (386). Some participants expressed the view that a major barrier that prevented them from purchasing functional food products was the fact that they had little time during shopping to decipher the positive and negative attributes of various products and to determine the appropriate place for them within their own diet. Of all the discussion areas, cost of products generated the most discussion as well as consistent attitudes. Most participants believed that foods promoted as having health benefits cost more. This cost was not always seen as worth the investment, particularly if the health claims were not believed or ambiguous.

As an adjunct to the TPB model utilised in this analysis, participants' views about which were the potential products that could best be used as carriers of n-3 fatty

acids were also investigated. The responses were consistent, with participants framing the importance of the wholesomeness of the products as an important factor. Also, participants felt that is was better for everyday products such as milk, bread, margarine and yoghurt to be the carriers of this functional ingredient, although participants also expressed the belief that snack and occasional foods would also be appropriate carriers of functional food ingredients. Further probing revealed that although this may be logical (people don't eat enough n-3 fat but people drink diet coke, therefore the addition of n-3 fatty acids in diet coke is logical), overall participants were suspicious of this strategy and viewed it as a gimmick, rather than a legitimate health proposition. Participants believed that the overall health proposition of the functional foods should be consistent with other health messages, i.e. low fat, low salt, low sugar.

6.3.4 General beliefs about functional foods

There was no evidence of negative salient beliefs related to healthiness of functional foods, which was surprising. Compared to conventional foods, functional foods are often processed with a relatively high degree of technological manipulation, which has parallels with the manufacturing of genetically modified (GM) foods. Attitudinal studies into consumer views of GM foods have indicated that this is a factor attributable to the general resistance to use of these products (387). Furthermore interactions between enrichments and product types of functional foods were found to be important determinants of consumers' perceptions of the healthiness in a cross-national study of Danish, Finnish and American consumers (288). The authors of that study described prevailing cultural

values of ‘mastery’ or ‘harmony’ that help to explain these attitudes (288). People or cultural values that can be described as high in mastery emphasise active mastery of the social and natural environment through self-assertion. These people tend to view functional foods positively (288). On the other hand, ‘harmony’ refers to values that co-exist with nature and deference of the technological manipulation of natural resources, therefore would most likely oppose the use of functional foods. It is possible that participants in our study were not aware of the level of technical manipulation involved in the development of these products – as there was national media coverage from the market release of several n-3 enriched products in Australia preceding the interviews which may have attributed to an understanding and acceptance of the concept– or that Australians in general (or at least those that self selected for focus group interviews) have a value system that can be described as high in ‘mastery’.

In the past food purchasing behaviour has formed the focus of many promotion and health education campaigns with the aim of improving food and nutrient intakes (388). With the emergence of functional foods there is a need to develop education campaigns and design behaviour change models to ensure that those segments of the population who can benefit the most from these novel products actually use them (389). To this end understanding consumer beliefs and attitudes toward functional foods is critical to both commercial success and achieving positive health outcomes from the introduction of enriched functional foods. However, more targeted research is required, as these results alone provide limited value in guiding education and marketing disciplines alike. In this study we

uncovered the salient beliefs in relation to n-3 enriched functional foods from a self-select Australian sample and therefore caution is required in interpreting these results. The nature of this focus group study was exploratory and these results were next used to expand on the TPB theoretical model to help examine consumer behaviour in relation to the use of n-3 enriched products. These salient beliefs formed the basis for question development for a quantitative survey tool which was used in the following study.

6.4 Acknowledgements

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CHAPTER 7

ATTITUDES AND INTENTIONS TOWARDS PURCHASING FUNTIONAL FOODS PREDICT UPTAKE[7]

7.1 Introduction

Although understanding consumer beliefs and attitudes provides interesting insights, it is the ability to predict purchasing behaviour that will ultimately assist in the promotion of this primary prevention strategy. Functional food products are distributed through traditional food markets and to be effective must first be purchased, and then incorporated into an individual's eating pattern. This presents unique challenges to health educators when considering the ability and potential of functional foods to deliver population health benefits. Efforts to increase intake depend, in part, on understanding the factors determining selection and intake. With this knowledge health promotion strategies can be targeted to maximise their effectiveness.

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[7] *A significant portion of this chapter is about to be published in the following peer reviewed journal:*

Patch CS, Tapsell LC, Williams PG. (In press) Attitudes and intentions towards purchasing novel foods enriched with omega-3 fatty acids. *Journal of Nutrition Education & Behavior*. 2005.

CP was responsible for the design of the study, organisation and leading the study, data collection and analysis, critical discussion of the analysis and preparation of the manuscript. LT and PW were responsible for critical discussions of the study design, critical discussions of the analysis and preparation of the manuscript.

The reasons for consuming functional foods are likely to be multifactorial, with a combination of social, psychological, knowledge based and economic factors. Earlier market segmentation studies in the US described the average functional food user to be female, well educated, with a higher income, aged between 35-55 years old and actively interested in health as a result of illness (390). More recently, Bech-Larsen and Grunert (2003) examined attitudes relating to perceived healthiness of functional foods across different cultures (288). They found that different processing methods, the use of different health claims, types of enrichments and types of products were determinants of consumers' acceptance of functional foods. Some studies have investigated demographic characteristics of users and non-users, however, they note that characteristics are specific to the functional food under study (391). Overall, research into factors that determine choice of functional food is limited and no studies to date have investigated the specific factors affecting the use of foods enriched with n-3 fatty acids.

One model that has been used to explain health related behavior in the past is the Theory of Planned Behavior (TPB) (19). It has been applied to food-related behaviors such as supplement use (23), genetically modified foods (387), dairy product intake (326) and organic vegetable consumption (392). According to the theory, behavior is directly predicated by intention to perform this behavior, as well as control factors, when behavior is not under complete volitional control. Intention in turn is determined by attitudes (A) toward the behavior, subjective norms (SN) and perceived control over the behavior (PBC). Intention can be viewed as the conscious plan to carry out a particular behavior and the motivation to perform it.

Each of these determinants is formed from a set of referent beliefs. Attitudes are determined by a behavioural belief about performing a particular behavior. This is the combination of strength of the belief (BS) combined with the belief that performing a particular behavior will result in the outcome (OE). Also, Subjective Norm is determined by the social pressure and beliefs held by significant others, or normative beliefs (NB), combined with the motivation of the individual to comply with this pressure (MC). Perceived behavioural control is determined by the likelihood of various factors outside of direct control to facilitate or inhibit a certain behavior. Therefore, a person who has a positive attitude toward a behavior, perceives social pressure to perform the behavior and believes that they have control over their decision to perform the behavior is more likely to carry out that behavior. The aim of this study was to identify the nature, strength and relative importance of influences on intention to purchase foods that are enriched with n-3.

7.2 Methods

7.2.1 Questionnaire development

Survey questions were developed using results obtained from focus group interviews, as recommended by Ajzen & Fishbein (1980) (19). The details of those interviews are described in Chapter 6 (370). Each focus group was tape-recorded, transcribed and content analysed using NVivo 2.0 (2002, QSR International Pty LTD). Sub-categories were developed to capture emerging themes. These formed the basis of the questions used in the questionnaire. A draft of the questionnaire was pre-tested on 10 subjects who were representative of the study sample.

Comments attained resulted in minor changes being made to the final questionnaire (Appendix N). Example foods enriched with n-3 fatty acids were included in the instructions on filling out the questionnaire.

Behaviour (B) - Use of foods enriched with n-3 was assessed 2 weeks after implementation of the questionnaire. A question was asked: *Did you eat any of the following foods high in n-3 over the previous 2 weeks (canola margarine, enriched bread, milk, enriched eggs, enriched bread etc)?*

Intention (I) - The dependent variable was measured by a global question using a seven point bipolar differential scale as suggested by Ajzen and Fishbein (1980) (19). Respondents could choose extremely unlikely, quite unlikely, slightly unlikely, neither, slightly likely, quite likely or extremely likely (+3 to -3) to the statement: *I intend to eat one or more foods with added omega-3 oils over the next two weeks*.

Attitude (A) - The independent variable, attitude, was also directly measured by a global question using a seven point bipolar differential scale. Respondents could choose favourable to unfavourable (+3 to -3) responses to the statement: *Overall, my attitude toward my eating foods with added omega-3 oils over the next two weeks is.*

Belief strength (BS) - Eleven independent variable statements were used to assess belief strength towards purchasing foods enriched with n-3 fatty acids: provide long-term health benefits, importance of obtaining the health benefits, may be at risk of inadequate intake of nutrient, may be at risk of over exposure of this nutrient, importance of scientific proof, developing new ways to make it easier to

choose a diet high in n-3, provide heart health benefits, provide brain health benefits, provide eye health benefits, improve arthritis and improve asthma. Each statement was anchored on a bipolar differential 7-point scale ranging from extremely important to extremely not important.

Outcome evaluation (OE) - The TPB assumes that having a positive attitude towards a behavior is based on believing that the behavior will be likely to lead to positively evaluated outcomes, or will be unlikely to lead to negatively evaluated outcomes. Therefore for each of the BS there was an equivalent outcome evaluation statement. Each statement was anchored on a bipolar differential 7-point scale ranging from extremely likely to extremely unlikely.

Beliefs relating to n-3 functional foods were transformed into consequences of purchasing by multiplying the belief strength by the outcome evaluation (BS X OE) and a mean computed (possible range +9 to –9) and reflect an overall behavioural belief scale.

Subjective Norm (SN) - The TPB assumes that normative factors are based on perceptions of whether specific significant others believe you should perform the behavior or not (Normative Belief), and the motivation to comply with the wishes of these significant others. Therefore beliefs relating to n-3 functional foods were transformed into consequences of purchasing by multiplying the normative belief by the motivation to comply and a mean was computed (possible range +9 to –9). This reflects an overall subjective norm scale (SN). The independent variable, subjective norm, was also measured by a global question using a seven point

bipolar differential scale. Respondents could choose extremely likely to extremely unlikely (+3 to -3) to the statement: *most people who are important to me think I should eat foods with added omega-3 fats.*

Normative beliefs (NB) - Groups or individuals whose views might influence functional food use were also explored. Seven normative beliefs (NB) were assessed: family, friends, medical practitioners, nutritionists, scientists and food industry. For example one statement is *"most members of my family think that I should eat foods with added omega-3 oils"* (extremely likely – extremely unlikely). Normative belief statements were scored from +3 to –3.

Motivation to comply (MC) - Motivation to comply with the beliefs of significant others was also determined. For each of the NB there was a question relating to the motivation to comply. For example one statement is *"Generally speaking, I want to do what my family thinks I should do."* Motivations to comply statements were scored from +3 to –3. Each statement was anchored on a bipolar differential 7-point scale ranging from extremely likely to extremely unlikely.

Perceived Behavioural Control (PBC) – Factors which might facilitate or inhibit functional food use, are termed control beliefs (CB). Included in our questionnaire were availability at the supermarket, control over shopping, cost, taste, time, and suitability for the family. In addition a global question of control was included: *"how much control do you have over whether you do or do not eat foods with added omega-3 oils"* (seven point scale: complete control - very little control). Unlike other determinants of intention, CB does not correspond with measures of intention. The

TPB assumes that the higher CB would be found in those intending to purchase functional foods. In this case it was expected that a high CB would result in both intention to purchase or not to purchase functional foods, whereas a lower CB would result in no intention to purchase. For this reason CB was transformed into a binomial scale. The transformation involved estimating whether motivation was either positive or negative from the determinant variables, therefore placing PBC as either (a) having control over purchasing or not purchasing, or (b) no control.

7.2.2 Sample

This study was conducted in the Illawarra region of New South Wales, Australia. At the time of the study health claims on food products were prohibited, however a regulatory system for health claims is currently being developed. Two sub-samples were surveyed via questionnaire: general consumers who responded to media advertisement and subjects with Type 2 diabetes participating in an intervention trial. These sub-samples were selected as we wanted to investigate if there were differences in intention and attitude between those with an existing disease (in this case type 2 diabetes mellitus) and those without, as suggested by Childs (1997) (390). General consumers were recruited from advertisements in the local media. Potential participants phoned in their contact details to a study-specific answering service and these calls were returned and a brief outline of the study was explained. They were screened and excluded if they were undergoing treatment for chronic illness. Those expressing interest were sent an information sheet and consent form to sign and return. Once consent was obtained a questionnaire was

sent via post. Questionnaires not returned after 2 weeks were followed up via telephone communication.

Subjects were recruited from advertisements in the local media and on local institutional email networks (University and Technical College). Potential participants phoned in their contact details to a study specific answering service and these calls were returned with a screening questionnaire. Inclusion criteria were: aged 35-75yrs, diagnosed with type 2 diabetes mellitus for at least one year and generally well. Exclusion criteria were: on insulin therapy (or with HbA1c >9%), BMI > 35kg/m^2, with major debilitating illness, food allergies or food habits inhibiting their participation in the study, illiteracy and inadequate conversational English. Subjects at the first clinic appointment of this 6-month trial completed the questionnaire. Approval for the conduct of the study was provided by the University of Wollongong / Illawarra Area Health Service Human Research Ethics Committee (Appendix O).

Using the database of eligible consumers responding to the local media advertisement, 134 questionnaires were sent out. After 3 weeks (including a reminder call after 2 weeks) 79 were returned representing a response rate of 61%. Fifty-five adults with type-2 diabetes who had volunteered separately for participation in a dietary intervention trial were asked to fill out the questionnaire and five declined to participate. The final sample size of the two sub-samples was 129.

7.2.3 Data Analysis

Internal consistency reliability of the scales used to measure the variables was determined by calculating the Chronbach's alpha and questions with an alpha < 0.3 were deleted. All variables that constituted attitude were included in the analysis, and the alpha value was 0.89. The questions relating to the influence of friends and food companies were deleted as they showed a low level of internal reliability (0.222 and 0.204 respectively). Chronbach's alpha for SN in the final analysis was 0.77. Control over food choice also showed a low level of internal reliability and once deleted the Chronbach's alpha value for PBC increased from 0.71 to 0.74.

There were no significant differences between the two sub-samples in both demographic parameters and measured variables (apart from the proportion reporting a chronic illness); therefore the results were combined for analysis. Respondents were dichotomised into intenders (47.7%) and non-intenders (52.3%) (Positive scores were classified as intenders; neutral and negative scores were classified as non-intenders). Independent t-tests where used to compare mean scores of TPB components and other continuous variables. Non-parametric data were compared using Mann Whitney *U*-tests. Correlations between the variables were measured using both Pearson's and Spearman's correlation coefficients. The influence of the TPB components of use of n-3 enriched functional foods was investigated by multiple logistic regressions. The determinants of intention as per TPB were investigated using multiple linear regressions. According to the TPB, Attitude, Subjective Norm and Perceived Behavioural Control are direct

determinants of intention. The first step involved entering global measures of attitude, subjective norm and perceived behavioural control into the model. The second step involved the addition of the attitudinal beliefs, normative beliefs and control beliefs. This allows us to determine if beliefs are mediated through attitude. Additional demographic variables (age, income and education) were added to the model to investigate any associations. Estimates were computed at the overall means for the data set. All analyses were carried out using SPSS for Windows version 7.0 (SPSS Inc.)

7.3 Results

The mean (± s.d.) age of the participant sample was 53 (± 12.8) years (range 17 – 80 years) and the mean body mass index (BMI) 28.6 (± 6.6) kg/m^2. Two thirds of the participants were female (66.7%) and the majority of subjects were currently in a relationship with no children under the age of 18 living at home (75.2%). Ninety four percent of participants were the main shopper in the household, or shared the shopping in the household. Income range was relatively evenly distributed between the five income brackets – 22.6% earned < $20K per year, 19% earned between $20K-$40K per year, 13.9% earned between $41K-$60K per year, 16.1% earned between $61K-$80K per year and 12.4% earned between $81K-$100K per year. The smallest number (7.3%) earned greater than $101K per year. The highest level of education of participants was 0.8% finished primary school, 31% finished high school, 24.8% finished technical college and 38.8% graduated from University.

Spearman's or Pearson's correlation between the various TPB components and those above 0.3 are reported. Attitude (r = 0.56; P = 0.01) and Subjective Norm (r = 0.41; P = 0.01) were correlated to Intention. In turn, each set of beliefs was correlated with the corresponding global measure. For example BB X OE correlated strongly with attitude (r = 0.75; P = 0.01) and NB X MC correlated strongly with Subjective Norm (r = 0.48; P = 0.01). Perceived behavioural control beliefs did not correlate with any of the variables under investigation.

There were no differences between the two sub-samples in intention to use functional foods or any of the determinant variables. Table 7-1 summarises the differences between intenders and non-intenders of n-3 enriched functional foods. Perceived behavioural control and control beliefs were not different between intenders and non-intenders. Those who intended to use these products had a more positive attitude toward them and perceived normative pressure to use them. Similarly scores on belief items also showed the same trend. There were no difference in age, income or education between the intenders and non-intenders.

Table 7-1 Comparison of variable rating scores between those intending or not intending to use n-3 enriched products in the following two weeks [mean ± s.d.]

	Intenders (n = 61 [47.7%])	Non Intenders (n= 67 [52.3%])	Significance [a] (P- value)
Attitude[b]	2.2 (0.6)	-0.5 (1.5)	0.00
Behavioural beliefs (BS x OE) [c]	4.2 (1.9)	1.6 (2.5)	0.00
Subjective Norm [b]	0.8 (1.4)	-0.2 (1.5)	0.00
Normative beliefs (NB x MC) [c]	1.9 (2.0)	0.7 (1.6)	0.00
Perceived behavioural control [b]	2.2 (1.1)	1.9 (1.4)	0.13
Control Beliefs (CB)[b]	0.5 (1.1)	0.4 (1.2)	0.95

[a]Student's t- test between intenders and non-intenders.

[b]Scores are from +3 to –3

[c]Scores are from +9 to –9

The results (Table 7-2) showed that the first step intentions were a significant predictor of n-3 enriched food use, with overall 78.5% of subjects correctly classified as users or non-users based on intentions and PBC. Twenty-nine percent of the variation in behaviour was explained by the regression (R^2 = 0.298; P < 0.001). The odds of using n-3 functional foods increased 0.75 fold for every increase of one unit in intention score. PBC was not a significant predictor of use. At the second step, the addition of attitude and SN the predictive power slightly decreased and none of the variables were significant. Thus as predicted by the TPB, the effects of attitude and subjective norm on behaviour were mediated by intention. At the third step, the predictive power of the model decreased to 75.3% with none of the variables being significant. Thus as predicted by TPB, the belief components have no unmediated effects on behaviour.

Table 7-2 Logistic regression of behaviour onto TPB components[a,b]

Variable[c]	Odds ratio	95% CI	Significance
Step 1			
Intention	0.75	0.14	P = 0.04
Perceived behavioural control (PBC)	1.03	0.26	P = 0.92
Step 2			
Intention	0.65	0.32	P = 0.65
Perceived behavioural control (PBC)	1.03	0.26	P = 1.03
Attitude	1.33	0.39	P = 1.33
Subjective Norm	0.84	0.26	P = 0.84
Step 3			
Intention	0.51	0.38	P = 0.78
Attitude	1.03	0.28	P = 0.93
Subjective Norm	1.66	0.44	P = 0.25
Perceived behavioural control (PBC)	0.77	0.29	P = 0.36
Behavioural beliefs (BS X OE)	0.99	0.18	P = 0.97
Normative beliefs (NB X OE)	0.60	0.31	P = 0.09
Control beliefs (CB)	1.25	0.21	P = 0.28

[a] Non significant variables were retained in the model to determine the effect of the additional variables when existing TPB variables were taken into account; [b] $R^2 = 0.298$ ($P < 0.001$);[c] Percent correctly classified: step 1 78.5%; step 2 = 77.9%; step 3 = 75.3%;

A further question is the extent to which intentions are predictable. By entering the direct predictors of intention (attitude, subjective norm, PBC) at the first step and all the other beliefs at the second we are able to test whether the effects of belief components are mediated by other TPB variables. At step 1 in the development of the linear regression model of intention, attitude was significantly positively associated with intention, whereas SN and PBC was not significant (Table 7-3). Thus stronger intentions to use n-3 enriched foods were associated with having

positive attitudes toward the use of these foods. Overall the equation accounted for 72.4% of the variance of intentions ($P < 0.001$). Assumptions of multiple regressions were validated using residual plots. At step 2, the belief variables explained a marginal amount of variation in intentions 72.5% ($P <0.001$). Thus as predicted by TBP, the effect of belief components have no unmediated effects. Therefore, the next phase was to examine how beliefs about the consequences of using n-3 enriched foods were associated with intention to use.

Table 7-3 Linear regression of intentions onto TPB components[a]

Variable	*B*	Beta	SE	Significance
Step 1				
Attitude	1.01	0.85	0.07	P < 0.001
Subjective Norm	0.02	0.01	0.07	P = 0.82
Perceived behavioural control (PBC)	0.02	0.01	0.08	P = 0.87
Step 2				
Attitude	0.97	0.81	0.07	P < 0.001
Subjective Norm	-0.05	-0.04	0.09	P = 0.57
Perceived behavioural control (PBC)	-0.01	-0.01	0.09	P = 0.88
Behavioural beliefs (BS X OE)	0.06	0.07	0.06	P = 0.32
Normative beliefs (NB X OE)	0.08	0.07	0.06	P = 0.22
Control beliefs (CB)	0.00	0.00	0.09	P = 1.00

[a] *B* = regression coefficient; SE = standard error; R^2 = 0.724 (P < 0.001) for Step 1, R^2 = 0.725 (P < 0.001) for Step 2.

Based on the dichotomised categorisation of self-reported intention to use, differences in responses to each belief question between intenders and non-intenders were examined using independent t-tests (Table 7-4).

Table 7-4 Mean scores for belief items: comparison of intenders and non intenders [mean ± s.d.] (n = 126)

Variable	Intenders	Non-intenders	Intenders	Non-Intenders	Intenders	Non-intenders
	Belief strength (BS)[a]		***Outcome evaluation(OE)[a]***		***BS X OE[b]***	
Importance of general health	2.85 (0.40)	2.74 (0.82)	2.1 0(0.65)†††	0.92 (1.32)	6.08 (2.15)†††	2.48 (3.9)
Importance of health benefits n-3	2.48 (0.57)†††	1.43 (1.33)	2.15 (0.64)†††	0.92 (1.41)	5.49 (2.40)†††	2.05(3.19)
Contain enough n-3 to be useful	2.41 (0.70)†††	1.28(1.46)	1.7 (0.10)††	0.48 (1.45)	4.42 (2.99)†††	1.35 (2.94)
Little risk of over exposure	1.32 (1.81)	1.16 (1.69)	0.98 (1.72)	0.42 (1.60)	1.21 (4.45)	-0.09 (3.58)
Food development based on science	2.82 (0.50)	2.52 (1.19)	1.80 (1.14)††	1.05 (1.44)	5.14 (3.37)†††	2.43 (4.41)
New ways to make n-3 intake easy	2.43 (0.62)†††	1.51 (1.20)	1.69 (1.09)†††	0.45 (1.6)	4.34 (3.03)†††	1.59 (3.12)
Improving heart health	2.85 (0.36)	2.76 (0.46)	1.97 (0.62)†††	0.86 (1.40)	5.64 (2.02)†††	1.97 (4.10)
Brain health	2.82 (0.39)	2.69 (0.55)	1.32(1.32)††	0.63 (1.39)	3.73 (3.95)††	1.78 (4.01)
Eye health	2.79 (0.45)	2.68 (0.58)	0.98 (1.32)	0.55 (1.36)	2.81 (3.77)	1.54 (3.90)
Improving Arthritis	2.74 (0/48)	2.68 (0.53)	1.39 (1.10)††	0.63 (1.39)	3.98 (3.09)††	1.86 (3.88)
Asthma	2.83 (0.38)	2.69 (0.50)	0.91 (1.26)††	0.23 (1.40)	2.67 (3.58)††	0.85 (3.67)
	Normative beliefs(NB)[a]		***Motivation to comply(MC)[a]***		***NB X MC[b]***	
Family	0.75 (1.41)†††	-0.56 (1.60)	0.56 (1.76)	0.18 (1.70)	1.88 (2.89)†	0.51 (3.20)
Friends	0.53 (1.39)†††	-0.52 (1.51)	-0.03 (1.68)	-0.38 (1.52)	1.05 (2.92)	0.97 (3.01)
Dietitians	1.47 (1.24)†	0.92 (1.52)	1.80 (0.93)†††	0.98 (1.57)	3.14 (3.05)†	1.83 (2.90)
Doctors	1.26 (1.36)†	0.69 (1.44)	1.80 (1.01)†	1.34 (1.36)	3.12(2.91)††	1.33 (2.90)
Scientists	1.43 (1.33)††	0.78 (1.39)	1.30 (1.13)††	0.63 (1.41)	2.43 (2.91)††	0.88 (2.50)
Food companies	1.14 (1.60)	1.20 (1.40)	-0.72 (1.64)	-1.09 (1.63)	-0.47 (4.10)	-1.42 (3.92)
	Control beliefs(CB)[a]					
Availability at the supermarket	-0.25 (1.77)†	0.19 (1.5)				
Control over purchase	1.16 (2.25)††	1.51 (1.68)				
Cost	0.28 (1.93)	0.06 (1.87)				
Taste/texture	0.49 (1.76)	0.52 (1.66)				
Time required to find	0.64 (1.85)	0.22 (1.73)				
Suitability for family	0.38 (1.88)	0.04 (1.91)				

† P < 0.05; †† P < 0.01; ††† P < 0.001 equal variances not assumed; [a] Items scored between +3 to -3; [b] Items scored between +9 to –9

7.3.1 Behavioural beliefs

Intenders differed from non-intenders in 3 behavioural beliefs; “importance of the health benefits of omega-3 fats”, “that foods enriched with omega-3 contain enough of this nutrient to be of benefit” and “how important is it that we make new ways to make omega-3 intake easy”. When considering how each group evaluated these outcomes on n-3 enriched food intake, intenders differed significantly in 9 of the 11 beliefs. With the multiplicative measure of belief strength and outcome evaluation (BS X OE) there was a significant difference between intenders and non-intenders for the 9 beliefs. These results suggest that intenders are more likely to believe that eating n-3 enriched products specifically will provide a variety of health benefits despite the fact there was little difference between how intenders and non-intenders rated the overall importance of these effects.

7.3.2 Normative beliefs

Normative beliefs differed between intenders and non-intenders for 5 of the 6 variables. Differences were observed for family members, friends, dietitians, doctors and scientists with more positive ratings for intenders. This was consistent when considering motivations to comply as well as the multiplicative value of NB X MC, with all variables except for family members (MC) differing. There were no differences between the groups in the normative belief relating to food companies, with both groups responding negatively when rating motivation to comply with their recommendations.

7.3.3 Control beliefs

Only 2 out of the 6 control beliefs differed significantly between intenders and non-intenders: availability in the supermarket and control over purchasing. Interestingly, intenders did not perceive availability as being a barrier to purchase (indicated by the negative response), whereas non-intenders believed that this was a barrier.

Non-intenders were more likely to believe that a lack of control over purchasing was a significant reason for not purchasing these products.

7.4 Discussion

Using a questionnaire based on the TPB, the model explained 72.5% of the variance of intention to use n-3 enriched foods. This result is comparable with other studies investigating intentions to use genetically modified foods and supplements, using the TPB, which have reported R^2 = 0.35 and 0.75 respectively (23, 387). These results, in light of this earlier work, highlight the importance of studying specific food products, processing techniques and health effects in order to understand the complex nature of food selection. For example, whilst these results are valid in understanding consumer behavior in selecting n-3 enriched foods, they do not translate to an understanding of the use of fish oil supplements, nor n-3 enriched products enriched using GM technology.

These findings into the determinants of intention extend on previous work using the TPB. In relation to n-3 enriched foods, attitude was found to be the sole determinant of intention to eat these products and was the sole significant predictor. It was found that both normative beliefs and control were not significant determinants of intention to eat these products in this study. It is apparent from the literature that the relative importance of these factors is variable and is dependent on the behavior, demographic and particular food under investigation. For example, dairy product use by the elderly is predicted by both attitudes and control beliefs, whereas the use of genetically modified foods is predicted by attitude, control beliefs as well as subjective norms (326, 387). This adds weight to the importance of investigating single action, specific behaviors as suggested by Azjen and Fishbein (19).

It appears that selection of n-3 enriched foods remains largely a personal choice. Although marketers, health professionals and family members may suggest the use of these novel products, ultimately use is an individual decision having minimal

influence from normative factors. Similarly as perceived behavioural control was not predictive of intention to eat n-3 functional foods in our sample; it can be speculated that selection of these products is under volitional control, although control factors may act directly on behavior. In summary, the selection of n-3 functional foods appears to be a cognitive process based on underlying beliefs, leading to overall attitudes which in turn have a significant impact on food choice.

The incorporation of functional foods into the diet has the potential to improve individual and population health (393). A survey of American dietitians found that over three quarters had recommended the use of functional foods in the last year (394). Results from this study can be used in a practical sense with the upstream determinants of attitude providing nutrition educators with cognitive targets that can be used to improve the specificity and assist in the development of nutrition education programs. Intenders had a greater belief in the importance of n-3 fats and their associated health benefit and a belief that it is important to provide these novel foods on the market, but insisted it was important that they contain enough of the active ingredient to be useful. Most significant was the belief that eating foods enriched with n-3 fats specifically would lead to a number of health benefits. Subjects believed in a wide range of health benefits attributed to n-3 enriched foods despite varying degrees of empirical scientific evidence to support these claims. However, both intenders and non-intenders alike rated the importance of these health parameters similarly. This would suggest that changing individual beliefs about the importance in health issues alone will not translate into the use of these foods. Efforts to influence consumers may best be channeled into demonstrating and communicating a cause and effect relationship between a specific product and a health parameter or benefit. Therefore, the role of health claims might be important in promoting these foods. However, more research is required to determine the effectiveness of this approach in increasing the use of omega-n-3 functional foods.

There are a number of limitations to this study. Despite the lack of differences between our two sub-samples, the combined sample was not representative of the general population and was both time and context specific. In addition, these study participants were more likely to be interested in nutrition than the general population. Intention to use n-3 functional food was high (54%) and may have been a result of the recent product launches which coincided with the study. Also, this study provides a static view of attitudes towards n-3 enriched foods and follow-up over time, as more of these foods enter the market, would be useful. Another limitation was that the questionnaire did not account for more general beliefs to do with healthfulness, naturalness and altruism, which previous studies have been shown to be significant determinants of GM food selection (387). However, these issues did not emerge as salient beliefs from earlier focus group work (370). This implies a more general limitation to the TPB.

In summary, to be initially effective in maintaining and encouraging positive intentions and behaviour, a likely strategy for promoters of n-3 enriched functional foods would be to direct their promotions towards changing attitude, and specifically belief in the effectiveness of enriched products in achieving specific health benefits. Independently, environmental factors are also important in determining the attitudes of consumers towards n-3 functional foods. For example, consumer beliefs can be affected by the way n-3 functional foods are portrayed in the media, which in turn is a product of the level of stakeholder agreement or disagreement. The final study of this thesis investigated the stakeholder beliefs and attitudes that may impact on the consumer environment.

7.5 Acknowledgements

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CHAPTER 8

STAKEHOLDER BELIEFS AND ATTITUDES DISPLAY MIXED IDEOLOGIES THAT MAY HAVE AN IMPACT ON THE CONSUMER ENVIRONMENT[8]

8.1 Introduction

To ensure the success of functional foods in the market place, an active collaboration of all stakeholders in the promotion of these novel foods is required to ensure a positive consumer attitude (395). McConnon *et al* (2002) have argued that a dissonance in ideology, beliefs, views and motivations between stakeholders could lead to a consumer lack of confidence in this concept, and ultimately undermine the potential for both commercial success and public health gain (25). To increase a favourable public perception they propose that the future of functional foods is dependent upon communication and trust between the stakeholders and warn how misperception can threaten their potential (25). The real challenge is to create an environment for the development and promotion of functional foods through collaboration between the various stakeholders (294).

The area of functional foods has attracted interest from a wide range of stakeholders, including government personnel, consumers, and members of the food industry, scientists and health professionals. The rising popularity of functional foods has resulted from the concomitant advances in food technology, nutrition science and consumer awareness in health and wellbeing (396, 397). The

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[8] *A significant portion of this chapter has been submitted to the following peer reviewed journal:*

Patch CS, Tapsell LC, Williams PG. (Submitted 19 November 2004) Australian stakeholder beliefs and attitudes about functional foods. *Food Policy*.

CP was responsible for the design of the study, organisation and leading the study, data collection and analysis, critical discussion of the analysis and preparation of the manuscript. LT and PW were responsible for critical discussions of the study design, critical discussions of the analysis and preparation of the manuscript.

landscape of the food supply is changing at an unprecedented rate, with the emergence of novel functional foods in most food categories thus creating many business opportunities for the food industry (383, 398). Equally as exciting is the advanced emphasis on preventing chronic illness and disease as well as promoting better health and wellbeing through an improved food supply (399).

Because the topic of functional foods is relatively new, there remains confusion amongst stakeholders surrounding this concept (395). Areas of confusion and debate include the lack of a clear definition and categorisation of functional foods, the lack of inclusion of functional foods in established nutrition education models as well as concerns relating to consumer perceptions and acceptability (25), social and cultural differences (288) and regulatory concerns about health claims (25, 400). This confusion amongst stakeholders has the potential to erode consumer confidence and dissipate the potential benefits if not managed effectively (27). There is evidence to suggest that with appropriate legislation, good scientific support and effective marketing, it is quite possible that any general consumer skepticism can be overcome (401). Many countries either have health claim legislation in place or are currently in the process of review (402). In addition, attempts are being made at an international level by the *Codex Alimentarius* Commission to establish guidelines for health claims about foods (403). However, there is continuing disagreement in these policy developments which is often fuelled by the interests of a range of stakeholders who seek to exert control over the marketing and product landscape of the food supply (26).

Stakeholders can be conceptualised as networks of human beings, actors or agents with diverse interests and somewhat bound by their role (404, 405). This metaphor highlights the problematic notion of rational decision-making as the sole basis for matters such as functional food evaluation, strategic direction and policy development. For consumer interests to be placed at the centre of policy direction it is essential that stakeholders come to an agreement free from restraint due to their roles. It has been suggested that this emancipation can be achieved by

recognising and challenging ideology (405). In this sense ideology can be defined as ideas that are so commonplace and familiar that they are taken for granted as objective fact or truth (406). Ideology becomes a coherent and widely accepted set of ideas that permeate a group and has a role in shaping political activity, often in an unconscious way (407). By surfacing and challenging underlying assumptions and making ideologies explicit, emancipation may be possible (405). The aim of this qualitative investigation was to explore the beliefs and attitudes and ideologies of different stakeholders toward the development and communication of functional foods and attempt to categorise underlying ideological positions.

8.2 Method

8.2.1 Participants

Semi-structured interviews were undertaken by using purposive sampling. Twenty potential participants were identified from a database of contacts to the recently created National Centre of Excellence in Functional Foods (NCEFF) (408). This centre was funded by the Australian government to provide leadership for functional foods in Australia, with an emphasis on integrating science and business interests. Twenty individuals representing general practitioners (GPs), nutritionists, industry, media, regulatory bodies and authoritative health organisations from various States were contacted initially via e-mail and an expression of interest to participate was requested. Quotas were set for each category, priority lists compiled (Table 8-1) and each individual contacted via e-mail. For those interested, an information sheet and a consent form were mailed to each potential participant, and interviews were conducted at a mutually convenient time.

Forecasted quotas were not reached for the GPs and journalists. The interviewees included two nutrition researchers, two food industry marketers, two public health nutritionists, one general practitioner, two clinical dietitians, two nutrition consultants, one prominent spokesperson, one journalist, one research consortium executive, two food policy experts, one food legislation enforcer and one food

industry lobbyist. The proportions of females and males were 50% each and all States and Territories (except Northern Territory) were represented.

Table 8-1 Profile of interviewees

Stakeholder group	Quota	State/territory	Gender
General practitioners	2	South Australia, Queensland	Male, Female
Nutrition Researchers	2	Western Australia, Victoria	2 Males
Food Industry Marketers	2	Western Australia, New South Wales	Male, Female
Public Health Nutritionist	2	Queensland, Victoria	2 Males
Clinical Dietitian	2	New South Wales, Victoria	2 Females
Industry consultants	2	New South Wales	Male, Female
Prominent spokesperson	1	New South Wales	Female
Journalist	2	Queensland, New South Wales	2 Female
Research Director	1	New South Wales	Female
Policy expert	2	Victoria, New South Wales	Male, Female
Industry lobbyist	1	Australian Capital Territory	Male
Regulatory enforcer	1	New South Wales	Male
Total	**20**		**10 Males; 10 Females**

Details of the participants and organisations have been suppressed in order to maintain confidentiality in view of the sensitive issues involved. Approval for the conduct of the study was provided by the University of Wollongong / Illawarra Area Health Service Human Research Ethics Committee (Appendix P).

8.2.2 Study context

In qualitative research, transparency of the study context is an important aspect of validity (409). The study context comprised the current regulatory framework, the

positions of the authors and those of the stakeholders interviewed. The authors did not receive financial gain from the study. One of the authors (CP) was in receipt of an Australian Research Council scholarship through a linkage grant co-funded by Goodman Fielder Ltd, Sydney, Australia with the scholarship provided by government funds. In addition the Smart Foods Centre is part of a consortium that forms the National Centre of Excellence in Functional Foods that has received Federal Government funding. LT is the Director of this Centre and PW is the Regulatory Affairs Cluster coordinator within this organisation. All three authors hold Accredited Practicing Dietitian status. In December 2003, the Australian and New Zealand Food Regulation Ministerial Council agreed to a policy guideline on nutrition, health and related claims. The policy guideline provided the policy principles to underpin the regulation of nutrition, health and related claims which included the regulatory system. At the time this study was conducted, the initial assessment report on a proposal for Nutrition, Health and Related Claims (Proposal P293) was drafted by Food Standards Australia New Zealand (FSANZ) and released for public comment (410).

To enhance the quality and credibility of qualitative analyses there is a particular need to highlight the professional and social pressures on the author which may have an effect on the reporting of these results (409). Further, as this study interprets attitudes and beliefs from the perspective of ideology, it is important to state the author's own political ideology. To date our research is based on the belief that a functional-food led intervention is effective in ameliorating many life-style diseases. Our own liberal view transcends a purely scientific view that tends towards promoting functional food within the food supply, but does not preclude objectivity at the expense of ideology.

8.2.3 Semi-structured interview procedure

The interviews were conducted during the months of April to June 2004. Interviews were pre-arranged and carried out over the telephone (except for two face to face interviews given the proximity with the interviewer's location). Interviews were

audio taped and later transcribed to allow systematic analysis of the discussion. An interview guide was developed following the general guidelines provided by Krueger (329). Using the theoretical framework proposed by McConnon *et al* (2002) (25), as well as reviewing the literature pertinent to this area (26, 27), ten open-ended questions were devised to determine stakeholder awareness, beliefs and attitudinal issues regarding the development and promotion of functional foods (Figure 8-1). After a definition of functional foods was provided, interviewees were asked for their views on the processing and production of functional foods, the ability of functional foods to improve health, evidence supporting functional foods and resultant claims, the regulatory environment, intellectual property and propriety, and finally, the perceived roles of stakeholders. During the interview, participants were encouraged to speak until no more views were expressed, and then the moderator probed and clarified points. In short, probes were primarily used to encourage participants to clarify meanings or explain points of incongruence in their answers.

General impression about these foods

1. Given the definition of functional foods we have provided;

We have seen the emergence of a number of foods with added health benefits such as foods enriched with plant sterols and foods enriched with fish oils…what do you think about these?

[Probe: Is this something that we should be doing to our food supply?]

Processing/Production

2. What do you see as the important factors when considering the development and promotion of functional foods in the food supply?

[Probe: Environmental concerns, production/processing technologies etc[

Ability to achieve health benefits

3. Do you believe that functional foods have the potential to improve health or promote health?

4. Do you see any alternative to the promotion of functional foods in the food supply in improving public health?

[Probe: Do you see this as a realistic approach? What do you consider as being the ideal balance between functional foods and the alternative approach?[

Determining evidence

5. What do you see as the type of scientific evidence that would be required to substantiate claims for functional foods?

[Probe: Consider draft discussion paper on levels of evidence.]

6. Do you believe that consensus can be reached about the scientific evidence about the effect of a food?

Regulatory environment

7. Do you believe that consensus can be reached about the scientific evidence about the process of approval or regulation regarding functional foods and claims?

8. Which body should ultimately be the authority for substantiating, authorisation of claim wording?

[Probe: Should this be a national body, 'competent' body or self-regulation? [

9. Who do you see as being responsible for post-launch surveillance of these novel products?

Intellectual property

10. Should evidence for a health claim be available to the public? How could the intellectual property of the claimant be protected? Do you think that this is an important issue?

Perceived role of stakeholder

11. What do you see as your role in the area of functional foods and claims about these foods?

Figure 8-1 Moderator question guide for stakeholder interviews

8.2.4 Data Analysis

Each interview was tape-recorded and transcribed, producing on average 6 pages of transcript. A computer software package for qualitative research analysis, NVivo 2.0 (2002, QSR International Pty Ltd), was used for data management and coding (334). Content analysis was carried out by CP and sub-categories were developed to capture the emerging themes. An iterative process of subcategorisation was conducted as suggested by Knodel (376). After the preliminary analysis, other members of the research team (LT and PW) assessed the content of each category and sub-category. Because the purpose of this study was exploratory, with an aim to elicit salient beliefs about functional foods, data were presented as examples from the interview discourse that illustrated the interpretive frames that emerged, and the corresponding themes (377). The analysis attempted to represent the full spectrum of beliefs within the study sample.

8.3 Results/Discussion

From the initial sampling frame (n = 20), 16 key stakeholders agreed to participate (representing a participation rate of 80%). None of the initially targeted GP's or journalist's agreed to participate. A further five people were approached from each of these two groups with only one from each group agreeing to participate, resulting in a final number of 18 interviews.

As a way of introduction into the interview participants were asked about their overall impression of functional foods. As would be expected diverse views were expressed ranging from positive and supportive of functional foods to negative in a number of areas (Table 8-2)

Table 8-2 Views expressed on functional foods

Supportive views	Non-supportive views
• Functional foods have a role where there is strong evidence	• Functional foods are problematic due to conflicting industry and health goals
• Functional foods are a necessary and logical development of the food supply	• Functional foods may be seen as a magic bullet approach to a very complex public health issue
• Functional foods provide innovative solutions to deliver health benefits to the community	• There is ambiguity over the definition of functional foods
• Functional foods provide the potential to increase the healthy choices of the population	• The success of functional foods will ultimately be determined by consumer acceptance
	• There is potential for functional foods only to be available to those who can afford them

The notion that functional foods are at odds with public health goals is one that has been expressed in the literature. Some believe that functional foods will undermine the most important determinants of public health by making them out of reach of those that need it most (389, 411) because of issues such as price (412), and access (412). Like the assumption that functional foods will contribute to public health gains, neither is substantiated by strong evidence. Both of these positions need to be tested, monitored and reviewed.

A range of views were expressed on matters concerning processing and production, achievement of health benefits, determining evidence, regulatory issues and intellectual property (Table 8-3 to 8-7). With respect to processing and production, concerns about the use of GM technology, shelf stability, safety and environmentally sound practices (Table 8-3) have been found by others in relation to personal predictors of consumers' food and health concerns and are generally well described factors (413). However, the notions that these foods should be produced with a justifiable health need in mind as well as the need for functional ingredients to be bioavailable appear to be unique to functional foods. Whilst

stakeholders expressed concerns regarding the scientific validity of functional food claims, consumers are more concerned with honesty in food labelling and disease concerns in general (413). In this way a scientific paradigm expressed by stakeholders is expressed in a different way to consumer concerns. The link between the two creates a challenging territory for those working in the field of functional foods.

Table 8-3 Key issues identified by stakeholders relating to processing and production of functional foods

Major sub-category	Exemplar Quote
Technological considerations (e.g. GM technology)	*It's [GM technology] an area where you should look at research and what can be done with it*
Justifiable need	*Well I would want to approach it [functional food production] from a public health perspective*
Shelf stability	*They've got to be shelf stable*
Safety	*No dispute over safety, that has to be there*
Bioavailability	*My perspective is that if these products are informed by clinical trials and they've got some powerful evidence that they have an impact on, say biomarkers in a certain population group, then you would expect that when it's produced it actually retains the qualities it was tested for*
Environmentally sound practices	*…much more consideration of the whole food chain. What might turn out to be the most important functional food might be organically grown.*

The ability of functional foods to deliver health benefits lies at the heart of their production and promotion. Those who held the belief that these foods would have a positive impact on public health also believed that this impact would only be in certain individuals who also had a specific medical condition and would be conditional upon there also being an education program about use of these foods (Table 8-4). Others believed that the negative impacts outweighed the potential public health gains. For example the risks of unintended overexposure, misleading and confusing labelling and inappropriate advertising were all seen as having a potentially negative effect on public health. Other stakeholders appeared less concerned, indicating that energy balance is the current key issue for promoting widespread public health, so unless functional foods could address this issue, their effects would be insignificant. Some believed cost barriers and the inability to prove the benefit would undermine the widespread use of these foods. A final view expressed was that the effort (human and financial) invested in the research, development, promotion and regulation of these novel foods would be better spent

in alternative public health campaigns such as mass education, mandatory fortification where there is a demonstrated benefit, promotion of minimally processed foods and promoting more politically risky approaches such as taxing ‘unhealthy’ foods.

Despite the assertion that the production and promotion of functional foods has the potential to promote public health benefits (266, 269), these data suggest large dissention in particular stakeholder categories. These respondents believe that functional foods are best targeted to those with specific health concerns rather than mass markets, a point discussed in the position paper of the American Dietetic Association (414) and an Australian review paper on dietetics and functional foods (415). This issue is clearly debatable, making further research specifically addressing effectiveness of a functional food led intervention of benefits desirable.

Table 8-4 Stakeholder beliefs about a functional food-led intervention and the promotion of public health benefits

Major sub-category	Minor sub-category	Exemplar Quote
Positive impact	In conjunction with education	*'[regarding plant sterol margarines] I doubt that it will have an impact unless a person at the same time reduced the saturated fat in the diet, knows what cholesterol is and gets it monitored, measures their blood pressure and tries to increase exercise and lose weight'*
	For certain individuals with specific medical concern	*'I believe that in certain circumstances some of them may help individuals. I am not convinced that they will have a wide spread public health benefit'*
Neutral (insignificant impact)	Energy balance is the real concern	*'The modern lifestyle diseases are largely related to too much food and not enough exercise. I am not sure that any sort of functional food will address that'*
	Those in need are least likely to use due to cost barriers	*' I suspect that the population groups that are likely to use these things are least likely to need them'*
	Unable to prove or disprove the public health benefit	*'Whether they [functional foods] have a protective effect for the whole of the population and one which is unproblematic, I think has never really been tested'*
Negative impact	Unintended exposure of added functional ingredient	*'…I think plant sterols [products] is a good one because we know that there are a number of groups that might not benefit from the consumption of these foods'*
	Misleading and confusing labelling	*'…I've written a bit about food labelling and there's a lot of room to interpret. For instance what 'lite' might mean'*
	Misleading marketing and promotion	*'Well the negatives from my perspective are it the information that's used to promote these foods is misleading or inaccurate so that consumers get the wrong message'*
	Promoted in inappropriate context	*'I think that on that issue that to me is the way in which food is developed as a drug for a specific*

		group. And is then promoted broadly to people that do not have a particular problem'
Lost opportunity cost - alternative Public health strategies	Mass education (dietary guidelines and fruit/veg messages)	*'I think it is a fantastic alternative to treating people when they are sick…I think it is under the umbrella of wellness'*
	Mandatory fortification where benefit clear	*'I mean if it is established that calcium enrichment is important than it should be in all milk'*
	Promotion of minimally processed foods	*'My personal belief is that as the first line of defense I would be promoting natural foods first'*
	Political approaches (such as taxing of unhealthy foods)	*'I'd be arguing for a more public health model which is look at dismantling the barriers to people choosing healthy food'*

Because the most distinctive feature of functional foods is their claimed “health benefit”, health claims are often used to distinguish between functional foods and ordinary foodstuffs (27) and the establishment of a legal framework for the management of claims is seen as imperative. Stakeholders identified a range of regulatory issues and the organisations for dealing with them that should be responsible (Table 8-5). The views on the latter ranged from a competent body to a suitable government agency such as FSANZ or a body set up by government, models described to varying degrees in other countries (333). There was less variation in the opinions about an appropriate body to authorise wording of health claims with most believing this should lie under the auspices of FSANZ. However, there was a strong view from some that there should be involvement from communications and marketing experts to help establish and manage the process. Views about who should be responsible for post-launch surveillance were diverse. In part this was due to the expressed lack of experience and knowledge in this area. One statement was made that the problem with post-launch surveillance lies with the current legislation that the health authorities in each state are working with, not the absence of mandate. In fact it was suggested that the current regulatory structure, which now primarily deals with food safety, could easily be extended (given appropriate guidelines and funding) to the monitoring of functional foods.

Table 8-5 Opinions on the regulatory environment for substantiation, claim wording and monitoring of health claims

Major sub-category	Organisation responsible
Authority for substantiation	Food Standards Australia New Zealand (FSANZ)
	Government body
	Independent body
	Authoritative body
	Competent body
Authority for wording of claims	Food Standards Australia New Zealand (FSANZ)
	Expert panel (communication experts)
Post-launch surveillance	Food Standards Australia New Zealand (FSANZ)
	State/Territory health authorities
	Australia Competition and Consumer Commission
	Consumer Association
	Australia Food & Grocery Council and peers
	Independent body
Removal of regulatory hurdles	

Despite there being insufficient data on the possible influences of health claims on eating behaviour (416) there is a demand for health claims, particularly from the food industry (27), whereas others warn of the potential threat they may pose to public health (417). Not surprisingly then, in this study, a range of stakeholder beliefs regarding the level of evidence required for the substantiation health claims was found (Table 8-6). Putting the relative merits to one side, it was widely accepted by participants that evidence is required to support health claims. However, details regarding the level of evidence required and the type of evidence varied. It was apparent from the interviews that a clearly articulated structured approach to the assessment of evidence is warranted. The consideration of functional foods within the context of the total diet of the intended population was also regarded as an important consideration. Participants expressed the need to consider the totality of the evidence relevant to the proof of claim. Policy related to the establishment of evidence for health claims needed to address the challenge of determining the appropriateness of extrapolating evidence from a controlled research environment to the general population, an issue already debated in the literature (10).

Table 8-6 Categories of evidence identified as required for substantiation of health claims for functional foods

Major sub-category	Minor sub-category	Exemplar Quote
Cochrane-type review	Established evidence	*'What I have a lot of respect for are Cochrane reviews and the reason I have a lot of respect for those is that they give you the criteria under which they are reviewing'*
Food modelling studies		*'Food is not drugs. There is a whole lot of contingencies associated with this and the main one is the reference to the whole diet and the variation that occurs within usual dietary pattern and somehow we need to get a grip on that as well'*
Totality of the evidence		*'certainly human experimental research is essential but then there is the issue in terms of substantiating the link between the claim and the functioning of that particular food'*
Effectiveness trials		*'The testing and the efficacy of those foods are often undertaken through clinical trials. Often using fairly small groups and then through really a slight of hand they are then promote it to wider populations outside the clinical trials themselves'*
Bioavailability		*'…but its important that it actually works in that product because it might be that one product would bind the active ingredient and that it would not be bio-active anymore'*

There was unanimous agreement that evidence used to support a health claim should be available to the public, however the timing of this availability was an issue (Table 8-7). In order to gain first to market benefits, some suggested that the evidence should only be made available once communication commenced. This raises the larger issue of claimant protection. There were disparate views ranging from the belief that claimant protection only served the interest of industry, whilst undermining public health goals, to a fundamental requirement of the legislation to support innovation. In addition, some believed that while patenting legislation, trade marks and fair trading legislation provide some protection for innovation, procedural changes to the review process would assist claimant protection. Disparity between stakeholders emerged on the necessity of claimant protection. Some stakeholders believed that protection was unnecessary and that the benefit for the greater good (through decreased competition and increased process) would

be compromised if there was significant claimant protection, whereas others believed that company investment should be protected to ensure continuing investment in innovation. One way to view this disparity is to investigate these beliefs as a function of ideology. According to Eccleshall *et al* (2003) (407), recurrent controversies through several centuries of Western thought reveal a polarity that extends from extreme “left-wing” humanism to an extreme “right wing” normative position. Here the desire for a greater good can be viewed as a humanist feature and claimant protection of rights as a normative feature.

Table 8-7 Categories of responses on intellectual property

Major sub-category	Minor sub-category	Exemplar Quote
Evidence available to the public	Evidence provided to the public unconditionally	*'Absolutely, because for people like me who are interested in it you want more information'*
	Evidence provided to the public conditionally	*'I think [evidence available to the public] once a company starts to talk to consumers about it'*
Claimant protection	Unnecessary	*'…the public health outcome is the greater good and if there is a copycat product out there in the market then there is a greater opportunity for consumers to take advantage of these benefits [bringing cost down] we should support that'*
	Covered under IP regulation	*'…I think in terms of intellectual property there are things like patenting or identifying an actual process of manufacturing [which] could potentially be patented, rather than the actual ingredient or formulation'*
	Need to protect investment and promote innovation	'*if you were sinking hundreds of millions of dollars which some of them do, it is reasonable that if you're first to market you should get some protection'*
	Change of FSANZ act on public comment	*'… a company produces a new product but they're restricted from putting it on the market until they go through a rigorous consultation process where all their IP is made publicly available before the product is launched'*
	Has potential to undermine Public Health goals	*'Public health [and the public] would probably benefit more if it [IP]was accessible to anybody or any company so it can be more widely distributed for cheaper'*

8.4 Conclusion

The findings of this research need to be interpreted in the context of the limited sample frame of stakeholders, with the omission of 'average consumers' noted. There is also solely an Australian perspective on an issue that is legislated in partnership with New Zealand. A cautionary note is also required on the examination of ideology. In an effort to draw out insights of ideology from beliefs expressed by participants the clear link between stakeholder views and the

ideologies they represent needs to be considered in terms of everyday realities. For example, to assume that the ideology of a normative position can be drawn neatly from the beliefs expressed by those who represent the food industry is to reduce the complexity of both normative ideology and food industry politics to banal caricatures. Also, despite the characterisation of the humanist and the normative orientations as opposites, at an individual level they are often orthogonal, that is, both can be held by the same person when considering different aspects of the topic of functional foods.

This research has revealed that there are a number of areas of agreement between stakeholder positions. Stakeholders in this study agreed that there was a role for functional foods in specific cases where the evidence supported the use. There were consensus that these functional foods should be safe, stable, and the bioactive agent available in the products. There was also agreement amongst stakeholders that regulation was required, and there was a need for evidence to substantiate claims. One feature of this research is that it also started to distinguish areas of disagreement. The most significant incongruity between stakeholders was related to the beliefs about the process of substantiation and regulation of health claims, as well as the evidence required for substantiating these claims. Interestingly the areas of disagreement are often characterised by the lack of clear scientific evidence available to inform stakeholder views. Examples included the beliefs expressed that plant sterol foods are only effective with adjunct education, contrasted with an alternative belief that they may be harmful for certain sub-groups. Stakeholders thus sometimes rely on pre-existing ideology to form beliefs and attitudes and this is in stark contrast to the stated beliefs regarding evidence for claims. We have categorised these ideologies as examples of “humanism” vs. “normative”. Emancipation from these ideologies is required to ensure consumer interests, from both a public health and market demand perspective, remain at the heart of decision making on functional foods.

In the formulation of regulation in relation to functional foods it may be tempting to hold off decision making until conclusive evidence is available. With this being unlikely, the process is inevitably political, and in making political decisions the concept of emancipation from ideologies is a very useful one. The clarification of differences and their ideological bases can help find common ground between stakeholders and lead to different approaches to the problem. The issue of political uncertainty thus can be managed by transparency of decision making (333). In countries like Australia where current legislation is under review, it is important that policy is founded on dialogue that is responsible and reflective, genuinely inclusive and free from domination from any one stakeholder group.

The effectiveness of the functional food concept as a means for primary prevention of CHD ultimately relies on consumer acceptance and the main priority of all stakeholders should be the protection of consumer interests. Differences in beliefs, attitudes and ideologies of stakeholder groups potentially distract from this goal. When stakeholders are relying on ideology rather than evidence they do not always make this explicit and sometimes people do not use evidence to support their arguments in a rigorous way. It is suggested that the next step from this exploratory work should be to focus on the identified areas of disagreement and attempt to examine in more detail those issues using a scientific approach. Particularly there needs to be an understanding about what evidence there is (or is lacking) about how consumers are likely to understand and use health claims. Also there needs to be agreement from stakeholders with differing views about what the key consumer questions are and what study methods would be appropriate to answer them. Some consensus meetings to do this might help inform national research priorities.

8.5 Acknowledgements

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CHAPTER 9

CONCLUSION

9.1 Summary

This research set out to examine the effectiveness of a functional food-led intervention in the primary prevention of CHD by addressing the question: 'can the use of functional foods be used to help overcome a person's natural aversion to dietary change in a way that results in an observable health benefit?' Analysis of a variety of data collected through dietary trials, focus group interviews, surveys and in-depth interviews suggest that it can. In the context of clinical practice where patients present with clear diagnosis of hypercholesterolaemia, the dietary prescription of plant sterol enriched margarine leads to clear improvements in biochemical markers, without compromising the whole of the dietary profile. However, targeting individuals with identified risk factors is only one part of the solution, targeting healthy populations is also an important aspect of primary prevention. As the chapter on increasing LCn-3 through enriched foods indicates, functional foods can overcome LCn-3 deficiency as indicated by dietary analysis and erythrocyte fatty acid fractions, but further benefits may be masked by the unintended weight gain. It is important to stress that an attempt to investigate the biological effects still leaves us wondering about the true 'effectiveness' of such an approach. The chapters on consumer beliefs and attitudes not only provide evidence for a relationship between beliefs, intention and behaviour, but also demonstrate a complex picture of consumer use of n-3 enriched foods that involves cognitive processes above and beyond taste and convenience which are often regarded as primary determinants of food related behaviour. Factors affecting beliefs about the effects of LCn-3 are not clearly understood, but it is believed that negative publicity as a result of conflict between stakeholders is an important factor. The final chapter on stakeholders argued that significant incongruence in beliefs and attitudes lie not in the notion of functional foods *per se*, but the way in

which they are promoted and advertised. Effective health claim legislation lies at the heart of functional food promotion. If policy fails to consider consumers' interests the potential public health gains could be negated through diminished consumer confidence caused by conflict between stakeholder groups.

9.2 Theoretical significance

The findings of this study support the hypothesis that a functional food-led intervention can be an effective preventative health strategy, but a note of caution is required at this point lest their significance be extrapolated inappropriately. More specifically, a functional food-led intervention using a single food, in a clinical context, with the support of a health professional, supports the hypothesis that this approach was more effective than usual practice. However, there are three main considerations which may limit the applications in healthy populations. Firstly, the nutritional composition of the vector or carrier of the functional ingredient needs to be considered. It may be the case that vectors are high in fat and energy concentration which may contribute to weight gain, which is counterproductive to all nutrition interventions. Secondly, concentrations of the functional ingredient must be incorporated into the vector in large enough quantities to be consumed readily, otherwise an excess in energy intake is required to consume the desirable intake. A final cautionary note concerns the way the functional food will be consumed and therefore the effect on other foods in the diet and the ultimate diet matrix. Subtle shifts in individual foods in the diet can lead to a dietary imbalance when the entire diet profile is considered. This is exemplified by the importance of the n-6 to n-3 ratio on CHD risk independent on total n-3 intake.

Notwithstanding the difficulties mentioned above, this research supports the claims of behavioural theorists who argue that the merits of the Theory of Planned Behaviour can be applied to food related behaviour, including the use of functional foods enriched with LCn-3. Given the complex nature of human behaviour, this gives us a method of prediction that again supports the theory that this approach will be supported through individual changes in diet.

The significance of this research is not, however, confined to the narrow realm of utility of behavioural theory. Identifying stakeholder belief incongruence also illuminates the wider theoretical issue of social power. This confrontation usually takes place around the formation of health claim legislation. Underlying the arguments are significant differences in ideology between the profit motives of the food industry and the public health motives of health professionals. The environmental context is an essential part of the epidemiological triad and is essential in the effectiveness of primary prevention interventions (418).

9.3 Limitations and areas for further research

As the cautionary note above suggests, there are some limitations to this study, the principal one being, as with all primary prevention interventions, the lack of definitive evidence for the effect on mortality and morbidity in the population. In low-risk populations without identifiable risk factors the likelihood of clinical events is infrequent, necessitating a larger study group to achieve statistical power in detection. Even when investigating at-risk individuals, large, expensive trial designs are required to show any positive effect. This raises the question of the degree to which conclusions about the effectiveness of this functional food approach can be applied to primary prevention based on causal inference. In the absence of definitive clinical trials cohort studies and population follow up may be the only way to determine the ultimate effectiveness of this approach.

Another limitation concerns the quality of evidence obtained from behavioural research. At best, models that have attempted to explain human behaviour have only managed to explain up to 30% of the behaviour under investigation. Therefore, most of the reasons for human behaviour cannot be explained through questionnaire sampling and quantitation. Compared to the precision of biochemical measurements, one cannot be as confident of conclusions based on these sources.

Although pre-clinical research into functional ingredients is well established, research into the application of this knowledge is in its infancy and further research is warranted. Not only is this needed to offset some of the limitations, but also to pursue some important theoretical questions arising from this research. Concerning dietary trials three studies are suggested. First, conducting an intervention in patients within a clinical context, again using plant sterols, but this time incorporating functional foods to reach n-3 targets. In addition this would need to include a larger sample which would probably require a multi-centre trial. A second trial, again using healthy subjects, could be designed to incorporate LCn-3 enriched functional foods but with greater emphasis placed on achieving fatty acid targets with fewer foods (higher LCn-3 concentrations). By basing the intervention on staple foods, and considering the energy and fat contribution to the overall diet, the likelihood of observing positive effects in cardiovascular biomarkers would be improved. In addition, due to the wide inter-individual variation in biomarkers (such as lipid and lipoprotein) responses to dietary change, and the impact of the presence of hypo and hyper-responders, which is related to genetic variation, methodologies from the fields of nutrigenomics and nutriproteinomics will emerge as an important component of this research (419). Thirdly, the logical conclusion to these studies is to test the effectiveness of a portfolio approach to dietary interventions incorporating functional foods – i.e. low fat, high sterol, high cereal fibre, high folate, high n-3 fatty acids, low GL. This has also been suggested by others (281).

Also, further research is required to pursue the nature of human behaviour in relation to the use of these novel foods. Having identified cognitive targets for intervention, research efforts could be focused on evaluating the effect that both education campaigns and health claim on labels have on the beliefs, intentions and ultimately behaviour of consumers. Also, this model could be extended to include a wider range of psychosocial variables to build a more comprehensive picture of the factors affecting consumer behaviour in relation to these foods. Finally, the approach used in this research focused specifically on n-3 enriched functional

foods. It would be worth researching the utility of this model with other functional food ingredients, thus devising a range of product specific behavioural models relating to functional food choice.

9.4 Policy implications

CHD remains a significant cause of chronic disease, our scientific understanding of nutrition is expanding, innovation in the food supply leading to novel foods is increasing and governments around the world are managing this through legislative changes. These are immovable facts. It is clear that despite the lack of definitive evidence on the effectiveness of primary prevention strategies, action is required. It is necessary for the practical immediate application of the existing knowledge regarding functional foods. Given the current legislative changes in Australia it is timely that we look at the policy implications of this research. At the centre of these suggestions is the consideration of consumer interests. As argued earlier, despite the best scientific and technologic intent, without consumer confidence the potential health benefits will be eroded.

The scientific design of these dietary trials provides a good model for the substantiation of health claims. Often the launch of a functional food is preceded by a body of animal, mechanistic, epidemiological, clinical trials. However, as argued earlier the translation of results in the controlled context does not necessarily translate into the free-living context. This is a point that is often overlooked. In establishing evidence for the substantiation of health claims consideration should be given to the demonstration of the effect in the proposed food, where trials in a ‘free-living’ context and with due consideration given to the effect on the whole diet.

With the knowledge that we can never replicate the true nature of human behaviour in a study design, it is important to gather intelligence on consumer beliefs and attitudes. For example, the widespread backlash against the introduction of GM foods might have largely been averted by an understanding of consumer attitudes. This has meant that the potential gains obtained from this

technology will not be realised for a much longer time frame. Traditionally much information about consumer views has been gathered by food companies with very little being published and peer reviewed. It is in the interest of all stakeholders that this information be available and should constitute part of the evidence required when evaluating health claims.

Methods of communicating the functional efficacy of foods may be needed to provide end users with a more accurate and complete view of the health effects of foods than can be provided by health claims or food composition alone in view of the complexity of the whole diet and whole foods (420). The health related effects of foods are often so dependent on food properties that the effects cannot be adequately represented by food composition alone (420). As a result, nutrient data can be unhelpful, or even misleading to end users, and there is a need for measures of functional efficacy that can be used to accurately guide evidence-based food choices for health (421).

Thus the balance between education and confusion is a fine one, and one that requires continual assessment. Stringent monitoring and disincentives for thin or misleading claims is one measure. Another is the research of consumer reactions to claims built from widespread intelligence gathering of consumers. What is suggested here is not that dissimilar to what is already known to industry but not in the scientific domain. Equally important is a regulatory environment that supports innovation and building incentives that reward innovation and protect industry investment needs to be considered.

A functional food-led intervention is no longer a futuristic approach to ameliorating lifestyle diseases. A new paradigm for diet, nutrition and lifestyle can be developed utilising the social capital available within the interested stakeholders. Through the development of scientifically based new functional foods, as well as education of the public about their role in a healthy diet, the potential to reduce the burden of lifestyle disease is great. The introduction of functional foods into the market place must be accompanied by extensive evaluation of functional foods, from both a

scientific effectiveness standpoint as well as a consumer standpoint. It is in the interest of all that regulatory policy should be founded on dialogue that is responsible and reflective, genuinely inclusive and free from domination by any single stakeholder group. In addition there is a responsibility of all stakeholders to advise on public health policy and the best ways of translating new science into practical information to ensure that these new foods enter the market in an equitable way that supports the health of both high-risk individuals and the population at large.

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APPENDIX A

STEROL/STANOL STUDY RECRUITMENT ADVERTISEMENT

WANTED

Patients with hyperlipidaemia who are considered candidates for cholesterol lowering medication.

The joint ethics committee of the Illawarra Area Health Service and the University of Wollongong has approved a dietary intervention 'field' trial to be held at Port Kembla Hospital. The aim of the study is to measure the effectiveness of a novel dietary strategy in improving dietary compliance and improving biochemical parameters in patients with hyperlipidaemia.

How to access the study

- Patients can be referred to the Port Kembla Dietitian clinic; Phone outpatients on 42238202
- Patients who do not fit the inclusion criteria or do not wish to participate in the study will undergo routine nutritional assessment and counselling
- Written consent will be obtained prior to commencement of the study and participation is voluntary
- In referring patients to the clinic, GP's are asked to ensure information is included on serum lipids, routinely assessed in clinical practice: total serum cholesterol; LDL cholesterol; HDL cholesterol; triglycerides. This information will be required at first referral and again after 3 months follow up.

Cost

- Free

Who will be conducting the research

- Mr Craig Patch, a qualified clinical Dietitian and PhD student
- supervised by Associate Professor Linda Tapsell from the Department of Biomedical Science at the University of Wollongong

Benefits to General Practitioners

- Nutritional assessment and counselling of patients considered candidates for cholesterol lowering medication
- Individual patient progress reports provided to the GP
- Results of study circulated to the members of the Illawarra Division of General Practice

For further information contact:

- Craig Patch, Dietitian (PhD student), Port Kembla Hospital; phone: 42238166; e-mail: patchc@iahs.nsw.gov.au
- Associate Professor Linda Tapsell, Director Smart Food Centre, Department of Biomedical Science, University of Wollongong; phone: 42213152; e-mail: linda_tapsell@uow.edu.au

APPENDIX B

STEROL/STANOL STUDY LETTER TO TARGETED GP'S

Dr **Frank Goderie**
Woonona Medical Practice
380 Princess Hwy
Woonona 2517

10 May 2002

Dear Dr Goderie

Re: Patients with hyperlipidaemia wanted

As part of our efforts to develop quality dietetic services, we have recently started a dietary lipid clinic at Port Kembla Hospital. This not only extends our services to the community, but also enables us to evaluate models of effective dietetic practice in lowering blood lipids. We feel this service is also important to general practitioners, in view of the recent budget announcement aimed at raising prescriber awareness on trialing dietary therapy prior to commencement of lipid-lowering medication.

Patients referred to our clinic for lipid management will be invited to participate in a research trial. This study involves a prospective, randomised, dietary intervention trial. *The aim of the study is to measure the effectiveness of a novel dietary strategy in improving dietary compliance and thereby biochemical parameters in patients with hyperlipidaemia*. We will be comparing dietary prescription of plant-sterol enriched margarines with standard dietary practice. Patients that decline will receive dietary assessment and counselling as per standard practice.

Dietary intake patterns will be monitored using research methodologies, and clinical outcomes will include dietary and weight change and biochemical parameters, routinely provided in referrals by GP's in clinical practice include: total serum cholesterol; LDL cholesterol; HDL cholesterol; triglycerides. The joint ethics committee of the Illawarra Area Health Service and the University of Wollongong has approved the research.

Details of the study as well as access details are attached. Please feel free to refer any of your patients with hyperlipidaemia.

Regards

Craig Patch MBA APD
PhD Candidate
Smart Foods Centre
University of Wollongong
Northfields Av, Wollongong NSW 2522. Australia

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Fax: +61 2 42214844
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APPENDIX C

STEROL/STANOL STUDY PATIENT CONSENT AND INFORMATION FORM

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE INFORMATION FORM

The efficacy of functional food prescription in free-living subjects with Hyperlipidaemia, Craig Patch APD

The positive aspects of nutrition and heart health are very well established. A lot of media attention is focussed on telling people what to eat; however the information is often confusing. Helping people achieve an optimal dietary intake- that is good for your heart- is the primary role of a dietitian. This study aims at investigating the best strategy for achieving a healthy diet. In taking part of this study you will undergo the following procedure. The first interview will involve the dietitian collecting information about your usual food intake via a dietary history and a 3-day food record; Instructions measuring equipment (scales, measuring spoons, measuring cups) will be provided. This will be followed up with dietary advice and counselling based on the National Heart Foundation's dietary guidelines. You will be allocated into one of two groups. One month after your first appointment you will be reviewed and further dietary advice will be provided. At three-months and six-months, a follow-up dietary history and 3-day food record will again be performed. Your GP will also follow your progress at this stage and will order a blood test. A phone call reminder will be made one week prior to remind to start your food record and a time will be made for a follow-up appointment. Dietary advice will be provided as necessary and follow-up negotiated. Extensive work is involved in participating in the study.

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE CONSENT FORM

The efficacy of functional food prescription in free-living subjects with Hyperlipidaemia, Craig Patch APD

This project is being conducted as part of a Doctor of Philosophy (PhD). The research is supervised by Assoc Prof Linda Tapsell, Smart Food Centre, University of Wollongong.

Your participation in this research is voluntary, you are free to refuse to participate and you are free to withdraw from the research at any time. Your refusal to participate or withdrawal of consent will not affect your treatment in any way/your relationship with Port Kembla Hospital, the Department of Nutrition & Dietetics or your relationship with the University of Wollongong. All results and information is confidential and your name will not be used. All data will be collected by me and stored (confidentially) in the department. If you would like to discuss this research further please contact Craig Patch on 4223 8166 or Assoc Prof Linda Tapsell on 4221 3152. If you have any enquires regarding the conduct of the research please contact the Secretary of the University of Wollongong, Human Research Ethics Committee on 4221 4457.

I,.....(Participant's name) consent to participate in the research conducted by Craig Patch as it has been described to me in the information sheet. I understand that the data collected will be used for research and I consent for the data to be used in this manner.

Signed Date

..../...../.....

APPENDIX D

STEROL/STANOL – CONTROL & INTERVENTION MEAL PLANS

Eating for a Healthy Heart
Suggested Meal Plan

BREAKFAST
Breakfast cereal with Low fat milk
Fresh, tinned, dried fruit or juice
Toast with margarine with a scrape of Peanut butter, honey etc

LUNCH
Wholemeal bread or roll or flat bread with Margarine
Add salad or vegetables with a low fat or olive oil or avocado
Add a small serve of lean meat, chicken or fish (twice a week)
Finish meal with fruit (fresh, dried, tinned or pureed)

EVENING MEAL
An optional start to the meal - soup (broth, vegetable or creamed - use low fat evaporated milk instead of cream)
Select starchy vegetables (potato, sweet potato or corn), rice, pasta or legumes (eg kidney beans, lentils)
Add salad or vegetables with a low fat or olive oil or avocado
Add a medium serve of lean, eats (fat trimmed from pork, lamb or beef) or skinless poultry (chicken or turkey) or fish, fresh or tinned (eg Tuna in water, spring water or drained of oil) or egg or reduced fat cheese.
Finish meal with fruit based dessert- fresh or tinned served with low fat ice cream or yoghurt
When dining out, limit deep-fried, battered or pastry based foods

AT MID MEAL SNACKS
Choose from a small tub of low fat yoghurt fruit or plain, or a piece of fruit, or a slice of raisin toast with Margarine or a glass of low fat milk or a couple of plain biscuits, scones etc.

Department of Nutrition & Dietetics
Port Kembla Hospital
PO Box 21 Warrawong 2502

Smart Foods Centre
ARC Key Centre of Teaching and Research
University of Wollongong

APPENDIX E

STEROL/STANOL STUDY DIET HISTORY QUESTIONNAIRE

The University of Wollongong
Metabolic Research Centre
Illawarra Dietary Intervention Studies

Name of study: The Efficacy of functional food prescription in free-living subjects with hyperlipidaemia

Research Diet History

Participant code:

Part1: General Questions

Client code: Interviewer: Date: Trial:

Initial Interview

1. Age: Sex: M/F Ht: Wt:
2. Previous medical history
3. Reason for admission
4. On average, how would you describe your activity level?
   Sedentary Light Light-Mod Mod Mod-heavy Heavy
5. Frequency of formal aerobic exercise (eg walking, cycling, classes):
   Nil 1-2/7 3-4/7 5-6/7 >6/7
6. Have you altered your usual diet as a result of any medical condition? Yes/No
   If yes, please describe these changes:
7. Reasons for changes:
8. Have you had dietary advice in the past? Yes/No
   If yes, by whom?
9. Are you on any Cholesterol lowering medication? Yes/ No
   If yes, what type?

Outline of Questionnaire

Diet History Protocol

Part 1: General Questions

Part 2: Food Intake Overview

Part 3: Meals Prepared in the Home

3.1 Breakfast

- 3.1.1 Breakfast cereals/porridge
- 3.1.2 Bread/toast/muffins
- 3.1.3 Hot/cooked dishes
- 3.1.4 Tea/coffee
- 3.1.5 Other foods
- 3.1.6 Variation

3.2 Morning Snack

3.3 Light Meal (Lunch)

- 3.3.1 Sandwiches/rolls
- 3.3.2 Salads
- 3.3.3 Soups
- 3.3.4 Hot meals (home-prepared)
- 3.3.5 Take-away
- 3.3.6 Tea/coffee
- 3.3.7 Other foods
- 3.3.8 Variation

3.4 Afternoon Snack

3.5 Main Meal (Dinner)

- 3.5.1 'Meat & 3 veg' style meals
- 3.5.2 Mixed dishes
- 3.5.3 Other foods
- 3.5.4 Tea/coffee
- 3.5.5 Desserts
- 3.5.6 Variation

3.6 Evening Snack

Part 4: Take-away/Restaurant Foods

Part 5: Individual Food Items

Part 6: Food Preparation Practices

Part 7: Notes

Diet History Protocol

The purpose of the diet history is to obtain an account of a person's usual food intake. Structurally it takes the form of a description of meals consumed throughout the day, with a food frequency cross-check (Burke, 1947). One way of looking at the first component is that of a story with a beginning (usually breakfast) and an end (usually supper). Using the narrative approach to taking the history means that the participant is given the opportunity to finish her/his story first before she/he is asked and more questions. In this way the flow of the participants information-giving is not interrupted and she/he is able to mention aspects which are seen as relevant in this context. Additional comments (not necessarily on food per se) made during this description may provide some insights for further questions or discussion later on. In introducing the diet history, reference is made to the notion of '**usual**', meaning within the last couple of months, and of a **time sequence** for the description, such as the duration of the day. Participants are asked to provide a general description and then point out variations to the pattern.

Interview schedule

- Explain the purpose of the interview. Advise the participant that you are seeking a description of usual eating patterns and suggest she/he start with the beginning of the day.
- If the participant begins with the first meal of the day and uses time refernces or meal sequences of the day to progress with the description do not interrupt the story, merely indicate that you are listening (nod, write, say 'hmm' or 'yes').
- If the participant stops at intervals along the way waiting for you to respond, provide narrative support to continue e.g., 'was that all for breakfast', 'do you have anything after that?'
- If the participant responds with 'it depends' be sure to encourage all possible variations on that topic (usually a meal description).
- If the participant says 'probably' in defining amounts of foods, use visual aids to support this estimation process (e.g. food portion photo book).
- If the participant goes into explanations for why/how they consume certain foods acknowledge it in a supportive non-judgemental way, but keep the account on track.
- When the participant has reached the end of the day, look at what you have noted and identify areas you need more detail on. This will depend on the purpose for taking the history. Ask specific strategic questions, noting details within body of questionnaire (Parts 3 & 4).
- Summarise the overall pattern of the diet and ask whether there is a great deal of variation in this pattern. Note the variation.
- Proceed with a food frequency checklist and questions of food preparation (Parts 5 & 6).
- Ask the participant if there is anything else he/she would like to add to what they have told you and if they think you have a true reflection of their usual eating patterns.

Prepared by: Associate Professor Linda Tapsell APD, University of Wollongong Metabolic Research Centre.
Updated February 1999.

rt1: General Questions

ent code: Interviewer: Date: Trial:

itial Interview

. Age: Sex: M/F Ht: Wt:

. Previous medical history

. Reason for admission

. On average, how would you describe your activity level?

Sedentary Light Light-Mod Mod Mod-heavy Heavy

. Frequency of formal aerobic exercise (eg walking, cycling, classes):

Nil 1-2/7 3-4/7 5-6/7 >6/7

. Have you altered your usual diet as a result of any medical condition? Yes/No
If yes, please describe these changes:

. Reasons for changes:

. Have you had dietary advice in the past? Yes/No
If yes, by whom?

. Are you on any Cholesterol lowering medication? Yes/ No
If yes, what type?

Review Interview

1. Ht: Wt:

2. Have you recently been admitted to hospital?

3. On average, how would you describe your activity level?
Sedentary **Light** **Light-Mod** **Mod** **Mod-heavy** **Heavy**

4. Frequency of formal aerobic exercise (eg walking, cycling, classes):
~~Nil~~ ~~1-2/7~~ ~~3-4/7~~ ~~5-6/7~~ ~~>6/7~~

Part 3: Meals Prepared in the Home

3.1 Breakfast Foods

How often do you eat breakfast? _____

Comments:

3.1.1 Breakfast Cereals/Porridge

Type	Frequency	Serving size

Milk Type: _____ Serving size: _____ mls/cups

Sugar/Sweetener: _____ Serving size: _____ tsp

3.1.2 Bread/Toast/Muffins (eg, white, wholemeal, soy & linseed, raisin, crumpets, croissants)

Type	Frequency	Serving size

Spreads (PUFA/MUFA margarine or butter)

Type: _____ Portion/sl: _____ tsp

Toppings (eg jam, honey, peanut butter, cream cheese, cheese etc)

Type	Frequency	Serving size

3.1.3 Hot/Cooked Dishes (eg baked beans, pancakes, bacon, eggs - include cooking method)

Type	Frequency	Serving size

Type of oil/fat used in cooking: _____

3.1.4 Tea & Coffee

Frequency: _____

Milk Type: _____ Serving size: _____ mls/cups

Sugar/Sweetener: _____ Serving size: _____ tsp

3.1.5 Other Foods

Type	Frequency	Serving size

3.1.6 Variation (eg. weekend variation)

3.2 Morning Snack Foods

Type	Frequency	Serving size

3.3 Light Meal (Lunch) Foods

The light meal is defined in this form as a sandwich, soup, salad or other smaller amounts of food. If the midday meal is the main meal of the day (often a cooked meal), enter under section 3.6.

What types of foods do consume for your light meal? _____

_____

_____

3.3.1 Sandwiches/Rolls

Type of Bread/Roll	Frequency	Serving size

Spreads (PUFA/MUFA margarine or butter)
Type: _____ Portion/sl: _____ tsp

Fillings/Toppings

Type	Frequency	Serving size

3.3.2 Salads (eg. mixed green, tabouleh, coleslaw, potato, bean etc.)

Type	Frequency	Serving size

Dressings
Type: _____ Frequency: _____ Portion: _____ ml

Protein Foods (eg. meat, fish, chicken, cheese, egg)

Type	Frequency	Serving size

Bread with Salad

Type	Frequency	Serving size

Spreads (PUFA/MUFA margarine or butter)
Type: _____ Portion/sl: _____ tsp

3.3.3 Soups (eg. minestrone, pea & ham, pumpkin, vegetable)

Type	Frequency	Serving size

Bread with Soup

Type	Frequency	Serving size

Spreads (PUFA/MUFA margarine or butter)
Type: _____ Portion/sl: _____ tsp

3.3.4 Hot Meals (home prepared)

Type	Frequency	Serving size

3.3.5 Take-away Foods

Type	Frequency	Serving size

3.3.6 Tea & Coffee

Frequency: _____

Milk Type: _____ Serving size: _____ mls/cups

Sugar/Sweetener: _____ Serving size: _____ tsp

3.3.7 Other Foods

Type	Frequency	Serving size

3.3.8 Variation (eg. weekend variation)

3.4 Afternoon Snack Foods

Type	Frequency	Serving size

3.5 Main Meal (Dinner) Foods

3.5 Main Meal (Dinner)
What types of foods do consume for your main meal? _____
_____
_____
_____

3.5.1 'Meat and 3 Veg' Style Meals
This meal type refers to whole pieces of meat/fish eg steak, sausages, fish fillet, chicken schnitzel, vegetarian 'meats'

Type of meat/fish/chicken	Cooking method	Frequency	Serving size

Accompaniments
Include: potato (boiled, mashed, wedges, chips), pasta, rice, vegetables, salad, breads & spreads, sauces, gravies

Type of accompaniment	Cooking method	Frequency	Serving size

3.5.2 Mixed Dishes

This meal type refers to dishes such as casseroles, stews, pasta/rice based dishes and stirfrys eg. Stroganoff, risotto, beef stirfry, lasagne, spaghetti bolognaise.

Include all ingredients and quantities used in each dish.

Type of main dish	Cooking method	Frequency	Serving size

Accompaniments

Include: potato (boiled, mashed, wedges, chips), pasta, rice, vegetables, salad, breads & spreads, sauces, gravies

Type of accompaniment	Cooking method	Frequency	Serving size

3.5.3 Other Foods (eg Quiche, ricotta & spinach triangles)

Type	Cooking method	Frequency	Serving size

3.5.4 Tea & Coffee

Frequency: _____

Milk Type: _____ Serving size: _____ mls/cups

Sugar/Sweetener: _____ Serving size: _____ tsp

3.5.5 Desserts (eg fruitpie/crumble, cheesecake, icecream)

Type	Frequency	Serving size

3.3.8 Variation (eg. weekend variation)

3.6 Evening Snack Foods

Type	Frequency	Serving size

Part 4: Takeaway/Restaurant Meals

4.1 McDonalds

Type	Frequency	Serving size

4.2 Kentucky Fried Chicken (KFC)

Type	Frequency	Serving size

4.3 Pizza (pan/thin, size, topping, side dishes eg garlic bread/salad)

Type	Frequency	Serving size

4.4 'Fish and Chips'

Type	Frequency	Serving size

4.5 Asian Restaurant

Type	Frequency	Serving size

4.6 Other Takeaway/Restaurant Foods (eg kebabs, hamburger, meat pie)

Type	Frequency	Serving size

APPENDIX F

STEROL/STANOL STUDY FOOD RECORD QUESTIONNAIRE

The University of Wollongong
Metabolic Research Centre
Illawarra Dietary Intervention Studies

Name of study: The Efficacy of functional food prescription in free-living subjects with hyperlipidaemia

3-Day Food Record

Please record everything that you eat and drink for 3 days (two consecutive weekdays and one weekend day)

Participant code:

THE UNIVERSITY OF WOLLONGONG
METABOLIC RESEARCH CENTRE
ILLAWARRA DIETARY INTERVENTION STUDIES

FOOD RECORD/DIARY

We need as much detail as possible so these diaries can be of use to estimate your usual consumption. Please help by checking that you have supplied the following information as you record each meal.
Measuring cups, spoons and small kitchen scales are supplied to you, so please use them to record amounts.
A sample record is enclosed as an example of one volunteer's diary.

Breakfasts

Cereal – type, brand, amount. Measure amount milk, sugar added. Also type of milk.

Toast/Bread – which type, brand is useful.

Spreads – measure your usual spread by the teaspoon as it goes on – this can be your reference each day. This amount may vary for crumpets/toast, compared to bread as hot toast/crumpets soak up more butter/margarine.

Toppings – did you have jam, honey, etc., amount/teaspoons if possible.

Tea/Coffee – how do you have it? Measure your usual dash of milk once to see how much you add. Tell us type of milk and amount of sugar added, if any.

Fruit/Fruit Juices – amounts. Please specify brand of juice and whether 100%, or if it is a fruit drink (not juice).

Morning/Afternoon Teas – record how many cups of tea, coffee, and amount and type of milk. Biscuits, etc. – how many, type.

Lunches

Sandwiches – type of bread, spread used (may need to ask if you are using canteen). Was spread usual amount you would use? What type of fillings – estimate number of slices of meat, cheese. If it is a salad sandwich, what is actually in there – beetroot, meat, etc. as well?

Take-aways – try to estimate quantities as best you can. Include type of cooking oil.

Evening Meals

Raw-weight of cooked meat. If it is a meal for 4, estimate how much of the meal you ate (i.e. 1/3, ¼).

- How was meat cooked?
- How much, and what type of oil/margarine was used?
- Did you eat fat on meat?
- Did you eat skin on chicken?
- What vegetables, salad, rice, pasta was served with meat and how much?
- What salad dressings, butter, cream was added?

Desserts

- Brand of ice-cream. How many scoops or tablespoons?
- Fruit/tinned fruit – quantities?
- Yoghurt – brand, amount?
- Cheeses – what types, how much?
- Commercial products – brands?
- Home-cooked goods – i.e. apple crumbles, etc. Recipe would be helpful.

Drinks - Remember to record wine, soft drinks, juices, etc.

Also remember that we are not judging you by what you eat. The records are needed because this is a scientific project. To make it a valid study we need the truth, so don't be afraid to write down everything you eat and drink.

THE UNIVERSITY OF WOLLONGONG
METABOLIC RESEARCH CENTRE
ILLAWARRA DIETARY INTERVENTION STUDIES

SAMPLE FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR WEEKDAYS AND WEEKEND DAYS

DAY: THURSDAY EXAMPLE ONLY
DATE: 29/4/99

TIME OF DAY	TIME	DESCRIPTION OF AMOUNT OF FOOD OR DRINK	HOW PREPARED/ COOKED
BREAKFAST	8a.m.	¾ cup Special K	
		1 cup Lite-White Milk	
		1 Buttercup Wonder White toast	spread with 1 teaspoon Canola marg
		1/2 teaspoon Vegemite	
MORNING TEA/ SNACKS	10a.m.	4 Cruskit Biscuits	spread with 4 teaspoons peanut butter
		Cup of tea	with 2 tablespoons Lite White Milk

SAMPLE FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR WEEKDAYS AND WEEKEND DAYS

DAY: THURSDAY EXAMPLE ONLY
DATE: 29/4/99

TIME OF DAY	TIME	DESCRIPTION OF AMOUNT OF FOOD OR DRINK	HOW PREPARED/ COOKED
LUNCH	12.30	2 sandwiches – 4 slices wholemeal bread filled with 2 slices Soccerball ham ½ avocado 1 small tomato ½ lettuce leaf 5 slices cucumber	spread with total of 3 teaspoons Canola marg
AFTERNOON TEA/SNACKS	2p.m.	1 banana	
	3p.m.	40g dry roasted peanuts glass of cordial (diet)	

SAMPLE FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR . WEEKDAYS AND . WEEKEND DAYS

DAY: THURSDAY EXAMPLE ONLY
DATE: 29/4/99

TIME OF DAY	TIME	DESCRIPTION OF AMOUNT OF FOOD OR DRINK	HOW PREPARED/ COOKED
DINNER	6.30pm	1 x cod fillet (120g cooked)	fried in 1 tablespoon Canola oil
		2 medium potatoes (200g cooked)	boiled
		¾ cup broccoli ½ cup carrot	steamed
		1 slice white bread	spread with ¾ teaspoon Canola marg
SUPPER/ SNACKS	8.30pm	Tinned peaches in natural juice (3 halves)	
		1 banana	
		2 scoops Dairy Bell ice-cream	
	9.15pm	Cup of coffee 1 granita biscuit	with 2 tablespoons whole milk

THE UNIVERSITY OF WOLLONGONG
METABOLIC RESEARCH CENTRE
ILLAWARRA DIETARY INTERVENTION STUDIES

FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR WEEKDAYS AND WEEKEND DAYS

DAY: DATE:			
TIME OF DAY	TIME	DESCRIPTION OF AMOUNT OF FOOD OR DRINK	HOW PREPARED/ COOKED
BREAKFAST MORNING TEA/ SNACKS			

FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR 5 WEEKDAYS AND 2 WEEKEND DAYS

DAY: DATE:			
TIME OF DAY	**TIME**	**DESCRIPTION OF AMOUNT OF FOOD OR DRINK**	**HOW PREPARED/ COOKED**
LUNCH			
AFTERNOON TEA/ SNACKS			

FOOD RECORD

PLEASE RECORD EVERYTHING THAT YOU EAT AND DRINK FOR WEEKDAYS AND WEEKEND DAYS

DAY: DATE:			
TIME OF DAY	**TIME**	**DESCRIPTION OF AMOUNT OF FOOD OR DRINK**	**HOW PREPARED/ COOKED**
D I N N E R			
S U P P E R / S N A C K S			

APPENDIX G

OMEGA STUDY INFORMATION FORM AND CONSENT

ersity Department of Medicine
ical Research Foundation Building
el 4, Rear 50 Murray Street
n, Western Australia 6000

Dr Trevor A Mori
Telephone 61 8 9224 0273
Facsimile 61 8 9224 0246
Email tmori@cyllene.uwa.edu. au

SUBJECT INFORMATION SHEET

DEVELOPMENT AND EVALUATION OF NOVEL FOODS ENRICHED WITH VERY LONG CHAIN OMEGA-3 FATTY ACIDS

Purpose of the Study:

There is considerable evidence that omega-3 fats found mainly in fish and fish oils have wide-ranging health benefits. Consequently, the National Heart Foundation of Australian, health authorities and nutritionists, now recommend that we eat more omega-3 fats. Increased consumption of omega-3 fats may reduce heart disease and stroke by improving blood pressure and heartbeat disturbances, by reducing blood clotting, and favourably altering blood fats (i.e. reducing triglycerides and increasing HDL-cholesterol, the "good" fraction of cholesterol).

Most Australians consume less omega-3 fats than recommended by health authorities. This means that the majority of Australians do not receive the potential benefits of dietary omega-3 fats. While fish remains the best source of omega-3 fats, Australians consume relatively little fish. One possible reason for the failure to achieve the dietary recommendations for omega-3 fats is the limited range of readily available foods that can deliver these nutrients. A potential solution is the development of novel foods enriched with omega-3 fats. We propose to develop foods enriched in omega-3 fats and test the effectiveness of these foods on heart risk reduction.

Subjects and Groups:

40 non-smoking men and women, aged 20-65 years, who are overweight (i.e. have a body mass index > 25 kg/m^2, a waist circumference > 100 cm, a waist/hip ratio > 1), and have elevated blood triglycerides (>1.6 mmol/L), but are otherwise healthy, will be recruited from the general community. People not eligible include those with diabetes, recent symptomatic heart disease, a history of heart disease or stroke, peripheral vascular disease, major surgery within the last 3 months, high blood pressure (>170/100 mmHg), liver or kidney disease, regular users of non-steroidal anti-inflammatory drugs, drugs for high cholesterol or high blood pressure control, and those eating more than one fish meal per week or regularly taking fish oil supplements.

Procedures:

A. At the beginning of the study volunteers will attend the University Department of Medicine Research Unit at Royal Perth Hospital (Murray St) for 1 visit (approx 30-45 minutes).

<u>At the screening visit volunteers will:</u>

1. Complete a lifestyle questionnaire;
2. Complete a brief medical history questionnaire;
3. Have height, body weight and waist circumference recorded;
4. Have blood pressure measured sitting, at 1 min intervals for 5 minutes;

1. Provide a small blood sample (~10 ml). A blood donation at the blood bank is ~500 ml. This procedure may cause mild bruising at the site where the blood is taken.

B. Volunteers who meet the entry criteria will be invited to participate in the study. Over a 2 week period they will be asked to attend our Department for 2 visits (approx 30-45 minutes each). At each visit, volunteers will be required to fast (water is allowed) for 12 hours prior to their visit. They will:

1. Maintain usual diet and drinking habits;
2. Have measurements of body weight taken at each visit;
3. Complete a lifestyle questionnaire detailing alcohol consumption and physical activity at one visit;
4. On one occasion, complete 24 hour dietary diaries on 3 separate days of the week;
5. Provide a small blood sample (~50 ml) at one visit;
6. Have blood pressure measured by an automated recorder at each visit. Volunteers will be rested for 10 minutes, then blood pressure and heart rate will be measured every 2 minutes for 20 minutes while lying down and every minute for 5 minutes while standing.
7. Undertake assessment of blood vessel function. This is a non-invasive test that is similar to having your blood pressure taken. The test measures stiffness of the blood vessels determined from an artery in the arm.

C. Volunteers will then be randomly allocated (i.e. by chance) to one of two groups, either: (1) a treatment group, that will be consuming a range of omega-3 fatty acid enriched foods (the total amount of fish oil-derived omega-3 fats consumed will be approximately 1g per day), or (2) a control group, that will consume the same quantity of test foods as the treatment group, but versions of these foods will not contain omega-3 fatty acids. Low fat, moderate sodium intakes will be advised for both groups, in keeping with Australian dietary guidelines. Individual dietary intake will be determined at baseline ensuring minimal change from usual eating patterns but accommodating the test foods. A list of foods will be provided to enable variety within constraints of desired macronutrient consumption patterns. All participants will be asked to continue their usual lifestyle and physical activity throughout the 6-month intervention period.

D. At the end of 3 and 6 months, volunteers will be asked to attend our Department for 2 visits (approx 30-45 minutes each) during which they will have all measurements as outlined in B.2-7 above repeated.

Participants are free to withdraw from the study at any time and their records will be destroyed, unless otherwise agreed by the participant. Any medical treatment needed during the trial will not be prejudiced. All personal information collected in this study will be kept strictly confidential. Your participation in this study does not prejudice any right to compensation, which you may have under statute or common law. The Human Research Ethics Committee at the University of Western Australia has given approval for this study. Any concerns regarding the project can be directed to the Secretary, Human Research Ethics Committee, Registrar's Office, University of Western Australia, 35 Stirling Highway, Crawley, WA 6009 (telephone number 9380-3703). Any questions concerning the study can be directed to Dr Trevor Mori or Professor Ian Puddey of the Department of Medicine, Royal Perth Hospital on Telephone: 9224 0273 or 9224 0252.

versity Department of Medicine
ical Research Foundation Building
el 4, Rear 50 Murray Street
h, Western Australia 6000

Dr Trevor Mori
Telephone 61 8 9224 0273
Facsimile 61 8 9224 0246
Email tmori@cyllene.uwa.edu.au

CONSENT FORM

DEVELOPMENT AND EVALUATION OF NOVEL FOODS ENRICHED WITH VERY LONG CHAIN OMEGA-3 FATTY ACIDS

1. I have read a summary of the study and its nature has been fully explained to me. I consent to take part.

2. During screening I will be required to attend the University Department of Medicine Research Unit at Royal Perth Hospital (Murray St) for 1 visit (approx 30-45 minutes). I will be asked to:
(a) Complete a lifestyle questionnaire;
(b) Complete a brief medical history questionnaire;
(c) Have height, body weight and waist circumference recorded;
(d) Have blood pressure measured sitting at 1 min intervals for 5 minutes;
(e) Provide a small blood sample (~10 ml). A blood donation at the blood bank is ~500 ml. This procedure may cause mild bruising at the site where the blood is taken.

3. If eligible for participation in the study, I will over a 2 week period be asked to attend the UDM Research Unit at RPH for 2 visits (approx 30-45 minutes each) during which I will:
(a) Maintain usual diet and drinking habits;
(b) Have measurements of body weight taken, at each visit;
(c) Complete a lifestyle questionnaire detailing alcohol consumption and physical activity, at one visit;
(d) On one occasion, complete 24 hour dietary diaries on 3 separate days of the week;
(e) Provide a small blood sample (~50 ml) at one visit;
(f) Have blood pressure measured by an automated recorder at each visit. I will rest for 10 minutes, and then blood pressure and heart rate will be measured every 2 minutes for 20 minutes while lying down and every minute for 5 minutes while standing.
(g) Undertake assessment of blood vessel function at each visit. This is a non-invasive test that is similar to having blood pressure taken. The test measures stiffness of the blood vessels determined from an artery in the arm.

I understand that I will be randomly allocated (i.e. chance) to one of 2 groups, a control group or a treatment group. I may be required to either: (1) consume a range of omega-3 fatty acid enriched foods (the total amount of fish oil-derived omega-3 fats consumed will be approximately 1g per day), or (2) consume the same quantity of test foods as the treatment group, but versions of these foods will not contain omega-3 fatty acids. Low fat, moderate sodium intakes will be advised for both groups, in keeping with Australian dietary guidelines. My dietary intake will be determined at baseline ensuring minimal change to my usual eating

3. patterns, but accommodating the test foods. I will receive a list of foods to enable variety within constraints of desired macronutrient consumption patterns. I will be asked to continue my usual lifestyle and physical activity throughout the 6-month intervention period.

4. At the end of 3 and 6 months, I will be asked to attend the University Department of Medicine Research Unit at Royal Perth Hospital for 2 visits (approx 30-45 minutes each) during which I will have all measurements as outlined in 3 (a)-(g) above repeated.

I have read the Subject Information Sheet and the information above, and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising that I may withdraw at any time without reason and without prejudice, or without prejudice to my future medical treatment.

I understand that all information provided is treated as strictly confidential and will not be rele

I agree that research data gathered for the study may be published provided my name or other

_____ _____

Signature of Participant Date

_____ _____

Signature of Investigator Date

The Human Research Ethics Committee at the University of Western Australia requires that all participants are informed that, if they have any complaint regarding the manner, in which a research project is conducted, it may be given to the researcher or, alternatively to the Secretary, Human Research Ethics Committee, Registrar's Office, University of Western Australia, 35 Stirling Highway, Crawley, WA 6009

APPENDIX H

OMEGA STUDY DIET SHEET AND OMEGA LOG

Study Diet Sheet

Diet guidelines

- Eat your usual diet and substitute the test foods in to your usual eating pattern.
- Consume a total of ***at least*** **8 omega** points per day (You may have additional test foods as desired)
- Limit to one fish meal per week – this includes tuna sandwiches and anchovies on pizza
- Avoid omega enriched foods other than the study foods
- Foods are only to be consumed by study participants
- Try not to overeat so that you do not put on weight

Consume your usual diet to include:

Suggested Omega Plan

		Omega points
Breakfast	Muesli or oats plus 250ml milk or 2 Toast with 4 tsp spread or 1 Toast with 2tsp spread plus 2 eggs or 3 pancakes with 20g spread	**2**
Morning Tea	Le Snak or muffin or Biscuits or dip or 250 ml milk	**1**
Lunch	2 slices bread with 20g spread plus Egg or soup or Caesar dressing or 250ml milk	**3**
Afternoon Tea	Le Snak or muffin or 2 Biscuits or dip or 250ml milk or 3 pancakes	**1**
Dinner/Supper	Caesar dressing or 2 chocolates or Le Snak or dip or 250ml milk	**1**

Omega point system

Test food	Serve size	Omega points
Bread	2 slices	1
Spread	20g (4 tsp)	1
Milk	250ml	1
Cereal	1 packet	1
Soup	1 packet	1
Biscuits	2 biscuits (1pkt)	1
Eggs	2 eggs	1
Le Snak	1 serve (dip)	1
Chocolate	2 serve	1
Dip	1 serve	1
Muffin	1 (as per directions)	1
Pancake	3 (as per directions)	1
Caesar Dressing	40ml (2Tbs)	1

Smart Foods Centre
ARC Key Centre of Teaching and Research
University of Wollongong

Additional information

Food item	Serve size = 1 omega point	Mon __/__/__	Tue __/__/__	Wed __/__/__	Thu __/__/__	Fri __/__/__	Sat __/__/__	Sun __/__/__
Bread	2 slices							
Margarine	20g (4tsp)							
Cereal	1 packet							
Milk	250ml							
Biscuits	2 biscuits							
Soup	1 packet							
Eggs	2 eggs							
Le Snak	1 packet (dip)							
Chocolate	2 items							
Dip	1 packet							
Muffin	1 as per directions							
Pancake	3 as per directions							
Dressing	40ml (2Tbs)							

APPENDIX I

OMEGA STUDY DIET HISTORY QUESTIONNAIRE

Code:
Date:
Site:

diet history questionnaire

Return to:
Smart Foods Centre
University of Wollongong
Northfields Av, Wollongong NSW 2522. Australia
Phone: +61 2 42214232
Fax: +61 2 42214844

Smart Foods Centre
ARC Key Centre of Teaching and Research
University of Wollongong

Interviewer: _____

DOB: _____ Age: _____

Ht: _____cm Weight: _____

BMI: _____ _ BMR: _____

Medications: _____

History of health conditions: _____

Supplements: _____

Physical activity level: _____

Core Food Choices: Please indicate the ***type*** of foods you select in these categories

Food group	Type	Food group	Type
Milk (full fat, skim)		Spread (margarine etc)	
		Oils (olive, canola)	
Bread (white, grain)			
		Drinks (sweetening)	

Part 1: Breakfast

How often do you eat this meal?_____Home_____Away_____

Breakfast Cereals/Porridge		
Type	**Amount**	**Frequency**
Milk with cereal		
Sugar with cereal		

Toast/Bread/Muffins etc (including toppings)		
Type	**Amount**	**Frequency**
Spread with toast		
Topping on toast		

Eggs and other cooked dishes		
Type	**Amount**	**Frequency**
Oil/fat		

Other Foods (including drinks, fruit, yoghurt)		
Type	**Amount**	**Frequency**

Part 2: Light Meal. **Lunch or Dinner** (Circle)

How often do you eat this meal?_____Home_____Away_____

Sandwiches/Rolls		
Description (include all components)	**Amount**	**Frequency**
Spreads		
Added salt		

Salads		
Description (include all components)	**Amount**	**Frequency**
Dressings		

Soups		
Description (include all components)	**Amount**	**Frequency**
Soup mix		
Added salt		
Bread		

Other foods (including drinks, fruit, takeaway meals, cakes)		
Description	**Amount**	**Frequency**

Part 3: Main Meal. **Lunch or Dinner** (Circle)

How often do you eat this meal?_________________________Home_______Away___________

Main dishes (include all components)	Amount	Frequency
Oils/spreads		
Dressings		
Sauces		

Other foods (include drinks, bread, side dishes, desserts, fruit)		
Type	**Amount**	**Frequency**

Part 4: Foods consumed outside meals

How often do you eat snacks?_____________________Home_______________Away_________

Type	Amount	Frequency

Part 5: Food Frequency Checklist

Food category	Amount	Frequency
Bread/crumpet		
Biscuits		
Crispbreads/crackers		
Cakes/scones/muffins/pastries		
Pancakes		
Beans/legumes		
Fruit		
Fruit juice		
Soft drinks/cordials		
Chocolate/lollies		
Chips		
Alcohol		
Milk		
Yoghurt		
Ice cream		
Cheese		
Dip/cream cheese/cheese spread		
Soy milk		
Soy yoghurt		
Eggs/omega eggs		
Salmon/tuna (fresh/canned)		
Sardines/Mackerel		
White fish varieties		
Oysters		
Walnuts		
Pecans		
Other nuts		
Seeds		

Part 6: Food Preparation Practices

6.1 Butter/Margarine
What type do you usually use?
Butter
Dairy blend
Margarine - polyunsaturated, regular
Margarine - polyunsaturated, reduced fat
Margarine - monounsaturated, regular
Other_____

6.2 Oil/Fat in cooking
What type of oil/fat do you use in cooking?

Butter	Olive oil
Dairy blend	Canola oil
Margarine - polyunsaturated, regular	Soybean oil
Margarine - polyunsaturated, reduced fat	Gold'n Canola
Margarine - monounsaturated, regular	Other _____

6.3 Fat on Meats/Chicken
How much fat is trimmed from meat before cooking/eating?
a) None
b) 25%
c) 50%
d) 75%
e) All
How much of the skin on chicken to you remove before cooking/eating?
a) None
b) 25%
c) 50%
d) 75%
e) All
Other, please specify: _____

APPENDIX J

OMEGA STUDY 3-DAY FOOD RECORD QUESTIONNAIRE

Code:
Date:
Site:

3-day food record

Study centre to return to:
Craig Patch MBA APD
PhD Candidate
Smart Foods Centre
University of Wollongong
Northfields Av, Wollongong NSW 2522. Australia
Phone: +61 2 42214232
Fax: +61 2 42214844
Mobile: 0408 266832
Email: csp03@uow.edu.au

Smart Foods Centre
ARC Key Centre of Teaching and Research
University of Wollongong

food record (Base) 2040

Breakfast and Morning Tea Date:_____

Checklist	Food	Amount
Cereal		
Bread		
Eggs		
Cream cheese		
Milk drinks		
Soup mix		
Caesar dressing		
Biscuits		
Le Snak		
Dip		
Muffins		
Pancakes		
Chocolate		

Lunch and Afternoon Tea Date:_____

Checklist	Food	Amount
Cereal		
Bread		
Eggs		
Cream cheese		
Milk drinks		
Soup mix		
Caesar dressing		
Biscuits		
Le Snak		
Dip		
Muffins		
Pancakes		
Chocolate		

Dinner and Supper Date:_____

Checklist	Food	Amount
Cereal		
Bread		
Eggs		
Cream cheese		
Milk drinks		
Soup mix		
Caesar dressing		
Biscuits		
Le Snak		
Dip		
Muffins		
Pancakes		
Chocolate		

APPENDIX K

OMEGA STUDY FOOD PREFERENCE QUESTIONNAIRE

Code:
Date:
Site:

Food preference questionnaire

Directions

Thank you for completing this questionnaire. This should only take a couple of minutes. Please answer all questions. Please do not identify your name on any part of the questionnaire.

For each study food listed answer the five questions (a,b,c,d & e).

Margarine:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Bread:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

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Milk:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Muesli:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Oat temptations (Apple & Sultana):

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Ginger biscuit:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Chicken Laksa soup:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Creamy Vegetable soup:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Eggs:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Le Snak:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Mint Chocolate:

a. Please rate the taste of the study food:

Dislike ☐ ☐ ☐ ☐ ☐ ☐ ☐ Like
extremely quite slightly neither slightly quite extremely

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Salsa dip:

a. Please rate the taste of the study food:

Dislike							Like
☐ extremely	☐ quite	☐ slightly	☐ neither	☐ slightly	☐ quite	☐ extremely	

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes ☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or ☐ add this product to your normal diet

Cracked pepper dip:

a. Please rate the taste of the study food:

Dislike							Like
☐ extremely	☐ quite	☐ slightly	☐ neither	☐ slightly	☐ quite	☐ extremely	

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes ☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or ☐ add this product to your normal diet

Orange & Poppy seed muffin:

a. Please rate the taste of the study food:

Dislike							Like
☐ extremely	☐ quite	☐ slightly	☐ neither	☐ slightly	☐ quite	☐ extremely	

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes ☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or ☐ add this product to your normal diet

Pancake:

a. Please rate the taste of the study food:

Dislike							Like
☐ extremely	☐ quite	☐ slightly	☐ neither	☐ slightly	☐ quite	☐ extremely	

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Salad Dressing:

a. Please rate the taste of the study food:

Dislike							Like
☐ extremely	☐ quite	☐ slightly	☐ neither	☐ slightly	☐ quite	☐ extremely	

b. Comment

_____

c. I consume this food ____ times a week

d. Do you intend to continue to consume this product over the next three months? ☐ Yes
☐ No

e. For you to be able to include this product in your diet did you: ☐ substitute a similar product already eaten, or
☐ add this product to your normal diet

Thank you for completing this questionnaire

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APPENDIX L

FOCUS GROUP MEDIA RELEASE

NEWS RELEASE

RESEARCHERS CONCERNED THAT CONSUMERS DO NOT EAT ENOUGH FISH

Research suggests that Australians are not eating enough fish. Fish, in particular deep-sea varieties, are high in omega-3 polyunsaturated fats, which offer a range of heart health benefits.

Craig Patch – PhD student with the Smart Foods Centre – says the wide-ranging health benefits of fish oil, which is high in omega-3 polyunsaturated fats, are well documented in both in the prevention and treatment of cardiovascular disease.

Mr Patch states "Studies reveal that Australians consume on average 1.2 serves per week of fish with nutrition authorities now recommending up to 3 serves per week. There is a potential to improve heart health if we can get more people to eat marine omega-3 fats."

"But not everyone can eat enough fish. Obviously factors such as food preferences and disposable income affect fish consumption. However, factors such as depleting fish stocks and distribution across an expansive continent have led to the proposed development of range of foods enriched with fish oil," said Mr Patch

Researchers at the University of Wollongong in collaboration with Goodman Fielder, University of Adelaide and University of Western Australia are now developing and testing a range of novel food products enriched with fish oil in order to deliver the health benefits of fish to a wider number of consumers.

Dr Peter Williams, Head of the consumer insights program of the Smart Foods Centre said the introduction of entire ranges of functional food products has brought with it a whole new set of issues. "We need to understand what consumers think about new foods with added health benefits and how to incorporate them into the diet."

"We are conducting focus group research to find out what Wollongong consumers think about these proposed changes to the food supply and how they would want new foods to be labelled and advertised," said Dr Williams

If you are interested in participating or wish to find out about other studies conducted by the Smart Foods Centre please phone 42214600 for details on how you can be involved.

APPENDIX M

FOCUS GROUP SUBJECT CONSENT AND INFORMATION FORMS

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE
INFORMATION FORM

Consumer salient beliefs and attitudes with regard to functional foods and food labelling Craig Patch APD, Peter Williams PhD APD, Linda Tapsell PhD APD, Chung Sau Chan MS Student

(Research supported financially by Goodman Fielder foods and the Australian Research Council)

The positive aspects of nutrition and health are well established. In response the f industry has launched a range of products with added nutrients beyond the nor ingredients with health promoting affects eg margarine to lower cholesterol. Th products are termed 'functional foods'. The properties of these products are adverti via the food label. It is uncertain how the availability of functional foods or f labelling claims affect food choices and it is important to find out what consumers th **The aim of this study is to investigate the views of consumers toward both functio foods and food labels.**

In taking part of this study you will required to attend a focus group interview with 8 people. This will take approximately one and a half hours. You will be allocated to of two groups: one will encourage discussion about functional foods and the other encourage discussion about low fat food labelling.

All conversations will be tape recorded and later transcribed for analysis. So information about your age, name and marital status will be collected for purpose analysis. All comments made during the focus groups will be treated as confider and will not be individually identified. While we can ensure confidentiality, the ex of confidentiality within the focus group cannot be ensured. Record of focus gro discussions on food labelling will be summarised in a report to Goodman Fiel (Sponsor) foods and published as a scientific paper. Results from discussions functional foods will be used in the development of a questionnaire to be usec subsequent research.

We ask consent of all participants to be contacted in the future to participate in a s administered questionnaire survey. All participants will be reimbursed $20.00 for ti travel costs and inconvenience. This payment does not depend on contin participation.

If you would like to discuss this research further please contact Craig Patch on 42214 or Dr Peter Williams on 4221 4085. If you have any enquires regarding the conduc the research please contact the Secretary of the University of Wollongong, Hur Research Ethics Committee on 4221 4457.
Thank you for considering this request.
Craig Patch

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE
CONSENT FORM

Consumer salient beliefs and attitudes with regard to functional foods and food labelling Craig Patch APD, Peter Williams PhD APD, Linda Tapsell PhD APD, Chung Sau Chan M Student

(Research supported financially by Goodman Fielder foods and the Australian Research Council)

This project is being conducted as part of a Doctor of Philosophy (PhD). Dr P Williams and Assoc Prof Linda Tapsell, Smart Food Centre, University of Wollong supervise the research.

Your participation in this research is voluntary, you are free to refuse to particip and you are free to withdraw from the research at any time. Your refusal to particip or withdrawal of consent will not affect your treatment in any way your relations with Port Kembla Hospital, the cardiac rehabilitation team or your relationship with University of Wollongong. All results and information is confidential and your na will not be used. All data will be collected by me and stored (confidentially) in department.

I,.....(Participant's name) consent participate in the research conducted by Craig Patch. Specifically I consent to:

- Participation in a 1.5hrs focus group of 8-10 people at the University Wollongong.
- The focus group will be audio taped, analysed and reported upon.
- My identity will remain confidential to the researchers.

I understand that the data collected will be used for research and I consent for the d to be used in this manner.

Signed Date

..../...../.....

Consent to be contacted for subsequent survey

I,.....(Participant's name) consent to be contacted via the phone by Craig Patch to be involved in a survey as it has b described to me in the information sheet. I understand that this does not compel m participate in the survey.

Signed Date

..../...../.....

APPENDIX N

FUNCTIONAL FOOD BELIEF QUESTIONNAIRE

Code:
Date:
Site:

functional food belief questionnaire

Return to:
Craig Patch MBA APD
PhD Candidate
Smart Foods Centre
University of Wollongong
Northfields Av, Wollongong NSW 2522. Australia
Phone: +61 2 42214232
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Smart Foods Centre
ARC Key Centre of Teaching and Research
University of Wollongong

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Part A – General details

Directions

Thank you for completing this questionnaire. This should only take 10-15 minutes. Please answer all questions. Please do not identify your name on any part of the questionnaire.

Once completed please return to the study center.
Part A - Please indicate your personal details by ticking (✔) a box.

1. **Please indicate your age:**
   - ☐ 25 or under
   - ☐ 26-35
   - ☐ 36-45
   - ☐ 46-55
   - ☐ 56-65
   - ☐ 66 or over

2. **Please indicate your gender:**
   - ☐ Male
   - ☐ Female

3. **Please indicate your marital status:**
   - ☐ Single/divorced/separated
   - ☐ Married/de facto

4. **Do you have Children aged under 18 years old living with you?**
   - ☐ No
   - ☐ Yes

5. **Which best describes your shopping practices:**
   - ☐ I am the main shopper
   - ☐ I share shopping with my partner
   - ☐ I do not shop for food

6. **What is your employment status?**
   - ☐ In paid work
   - ☐ Un paid work

7. **Please indicate the income range of your household:**
   - ☐ Less than $20,000 per year
   - ☐ $20,000-$40,000 per year
   - ☐ $41,000-$60,000 per year
   - ☐ $61,000-$80,000 per year
   - ☐ $81,000- $100,000 per year
   - ☐ greater than $101,000 per year

8. Please indicate your highest level of education:

☐ Primary school
☐ High school
☐ TAFE
☐ University
☐ Other Please specify _____

9. Please provide the best estimate of your height and weight:

Weight ____
Height ____

10. Please indicate any chronic condition(s) you have:

☐ Diabetes ☐ High blood pressure ☐ Asthma
☐ Lung disease ☐ Heart disease ☐ Cancer
☐ Arthritis or other rheumatic disease
☐ Other Please specify _____

Part B

Directions

In this part of the questionnaire we are mainly concerned with your views toward foods with added omega 3 oils. Some examples of these products include *Meadow Lea's Hi-Omega margarine* and *Tip Top's UP* bread.

In the questionnaire you are about to fill out we provide both statements and questions which we would like you to rate on a scale with seven boxes; you are to make a tick (✓) in the place that best describes your opinion.

For example, if you were asked to rate "the weather in Wollongong" on such a scale and you think the weather in Wollongong is *extremely good*, and then you would place your mark as follows:

The weather in Wollongong is.

bad ☐ ☐ ☐ ☐ ☐ ☐ ☑ good
extremely quite slightly neither slightly quite extremely

If you think the weather in Wollongong is *quite bad*, then you would place your mark as follows:

The weather in Wollongong is.

bad ☐ ☑ ☐ ☐ ☐ ☐ ☐ good
extremely quite slightly neither slightly quite extremely

11. I intend to eat one or more foods with added omega-3 oils over the next two weeks:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

12. Overall, my attitude toward my eating foods with added omega-3 oils over the next two weeks is:

unfavourable ☐ ☐ ☐ ☐ ☐ ☐ ☐ favourable
extremely quite slightly neither slightly quite extremely

13. I believe that improving general health is:

Not important ☐ ☐ ☐ ☐ ☐ ☐ ☐ important
extremely quite slightly neither slightly quite extremely

14. I believe that obtaining the health benefits of omega-3 oils is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

15. How important is it for foods that have added omega-3 oils to contain enough of this nutrient to provide a health benefit?

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

16. When eating foods with added omega-3 oils how important is it that there is little risk of overexposure of this nutrient?

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

17. How important is it for food companies to base new food development on good scientific evidence?

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

18. I believe that developing new ways to make it easier to choose a healthy diet high in omega-3 oils is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

19. I believe that improving heart health for the general population is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

20. I believe that improving brain health for the general population is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

21. I believe that improving eye health for the general population is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

22. I believe that improving arthritis for the general population is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

23. I believe that improving asthma for the general population is:

Not important	☐	☐	☐	☐	☐	☐	☐	important
	extremely	quite	slightly	neither	slightly	quite	extremely	

24. My eating foods with added omega-3 oils will improve my general health:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

25. My eating foods with added omega-3 oils makes it easy to obtain the health benefits of this nutrient:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

26. Foods with added omega-3 fats contain enough of this nutrient to provide a health benefit:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

27. My eating foods with added omega-3 oils puts me at risk of overexposure of this nutrient in the diet:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

28. Foods with added omega-3 oils have been developed and based on a good understanding of the scientific evidence:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

29. My eating foods with added omega-3 oils is an easy way to choose a healthy diet:

unlikely	☐	☐	☐	☐	☐	☐	☐	likely
	extremely	quite	slightly	neither	slightly	quite	extremely	

30. My eating foods with added omega-3 oils will improve my heart health:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

31. My eating foods with added omega-3 oils will improve my brain health:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

32. My eating foods with added omega-3 oils will improve my eye health:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

33. My eating foods with added omega-3 oils will protect me from or improve arthritis:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

34. My eating foods with added omega-3 oils will protect me from or improve asthma:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

35. Most members of my family think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

36. My close friends think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

37. Most dietitians think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

38. Most doctors think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

39. Most scientists think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

40. Most food companies think that I should eat foods with added omega-3 oils:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

41. Most people who are important to me think I should eat foods with added omega-3 fats:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

42. Generally speaking, I want to do what my family thinks I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

43. Generally speaking, I want to do what my close friends think I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

44. Generally speaking, I want to do what the most dietitians think I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

45. Generally speaking, I want to do what the most doctors think I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

46. Generally speaking, I want to do what the most scientists think I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

47. Generally speaking, I want to do what the food companies think I should do:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

48. How much control do you have over whether you do or do not eat foods with added omega-3 oils?

Very little control ☐ ☐ ☐ ☐ ☐ ☐ ☐ complete control
extremely quite slightly neither slightly quite extremely

49. Even if I wanted to eat foods with added omega-3 oils they are often not available where I buy food:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

50. Even if I wanted to eat foods with added omega-3 oils I have no control over what is bought from the supermarket:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

51. Even if I wanted to eat foods with added omega-3 oils the cost prevents me from buying them:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

52. Even if I wanted to eat foods with added omega-3 oils the taste and/or the texture prevents me from buying them:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

53. Even if I wanted to eat foods with added omega-3 oils the time required searching for these foods prevents me from buying them:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

54. Even if I wanted to eat foods with added omega-3 oils if they were not suitable for the entire family I would not buy them:

unlikely ☐ ☐ ☐ ☐ ☐ ☐ ☐ likely
extremely quite slightly neither slightly quite extremely

Thank you for your participation.
Please return the questionnaire to the study center on your next visit.

APPENDIX O
VALIDATION OF F2BQ INFORMATION FORM AND CONSENT

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE INFORMATION FORM

Development and validation of a questionnaire predict the use of functional foods - Self administered questionnaire

Craig Patch APD, Peter Williams PhD APD, Linda Tapsell PhD APD, Chung Sau Chan MSc student

(Research supported financially by the Australian Research Council)

The positive aspects of nutrition and health are well established. There are a number of products that claim to have health benefits. The latest range of products has nutrients in addition to the normal ingredients and claim to have a health promoting effects eg lowering cholesterol. These products are termed 'functional foods'.

The aim of this study is to examine consumer beliefs and use of functional foods .

Specifically we will ask you to complete a self -administered questionnaire. This will take approximately 20 -30 minutes. Questions will be on beliefs toward food and health as well as current use of specific functional foods. All questionnaires will be anonymous and results used for research purposes only. The questionnaire should be returned as soon as possible using the self -addressed envelope provided. If we do not receive this within two weeks or we need to clarify answers we will contact you by phone.

If you would like to discuss this research further please contact Craig Patch on 42214232 or Dr Linda Tapsell on 4221 3152. If you have any enquires regarding the conduct of the research please contact the Secretary of the University of Wollongong, Human Research Ethics Committee on 4221 4457.

Thank you for considering this request.

Craig Patch

UNIVERSITY OF WOLLONGONG & ILLAWARRA AREA HEALTH SERVICE
CONSENT FORM

Development and validation of a questionnaire predict the use of functional foods
Self administered questionnaire

Craig Patch APD, Peter Williams PhD APD, Linda Tapsell PhD APD, Chung Sau Chan MSc student
(Research supported financially by the Australian Research Council)

This project is being conducted as part of a Doctor of Philosophy (PhD). The research is supervised by Assoc Prof Linda Tapsell, Smart Food Centre, University of Wollongong.

Your participation in this research is voluntary; you are free to refuse to participate and you are free to withdraw from the research at any time. Your refusal to participate or withdrawal of consent will not affect your treatment in any way/your relationship with the University of Wollongong. All results and information is confidential and your name will not be used. All data will be collected by Craig Patch and stored in secure, confidential holdings within the Department of Biomedical Sciences.

I,.....(Participant's name) consent to participate in the research conducted by Craig Patch. Specifically, I consent to:

- Completing a self-administered questionnaire on functional foods received in the mail.
- Returning this in the stamp self-addressed envelope provided.
- Being contacted by phone by Craig Patch if any questionnaires are not received within 2 weeks or if there is a need to clarify any of my responses.

I understand that the data collected will be used for research and I consent for the data to be used in this manner.

_____
Signed Date

..../...../.....

APPENDIX P

STAKEHOLDER STUDY INFORMATION SHEET AND CONSENT FORM

UNIVERSITY OF WOLLONGONG

INFORMATION FORM

Determining stakeholder beliefs and attitudes towards the development of and communication about novel foods designed to improve or enhance health (functional foods).

Craig Patch APD, Linda Tapsell PhD APD, Peter Williams PhD APD

(Research supported financially by an Australian Research Council Linkage grant with Goodman Fielder Foods being the industry partner)

The positive aspects of nutrition and health are well established. In response the food industry has launched a range of products with added nutrients beyond the normal ingredients with health promoting affects eg margarine to lower cholesterol. These products are termed 'functional foods'. To this end the Australia New Zealand Food Regulation Ministerial Council agreed to a range of policy initiatives, which included Nutrition, Health and Related Claims to be permitted on food labels. In addition, the existing code on novel foods will be reviewed. This is a policy shift in Australia and there are implications for Public Health.
Regulatory bodies often consult stakeholders on policy options, however there are no studies reporting beliefs and attitudes of Australian stakeholders towards functional foods. Identifying contrasting beliefs regarding this issue is important if we are to understand how we can use the food supply to improve the health of the population.

The aim of this study is to investigate views of stakeholders toward the development and promotion of functional foods and whether this is an appropriate strategy for improving population health through the food supply.

In taking part of this study you will be required to attend a one-off interview. This will take approximately one hour. All conversations will be tape recorded and later transcribed for analysis. Some information about your age, name and workplace will be collected for purposes of analysis. All comments made during the interview will be treated as confidential and will not be individually identified. Results from discussions will be published in peer reviewed journal articles. We ask consent of all participants and signature on the attached form is required.

If you would like to discuss this research further please contact Craig Patch on 42214232 or Prof Linda Tapsell, on 4221 3152 or Dr Peter Williams on 4221 4085. If you have any enquires regarding the conduct of the research please contact the Secretary of the University of Wollongong, Human Research Ethics Committee on 4221 4457.

Thank you for considering this request.

Craig Patch

UNIVERSITY OF WOLLONGONG
CONSENT FORM

Determining stakeholder beliefs and attitudes towards the development and communication of novel foods designed to improve or enhance health (functional foods).

Craig Patch APD, Linda Tapsell PhD APD, Peter Williams PhD APD
(Research supported financially by an Australian Research Council Linkage grant with Goodman Fielder Foods being the industry partner)

This project is being conducted as part of a Doctor of Philosophy (PhD). Prof Linda Tapsell and Dr Peter Williams, National Centre of Excellence in Functional Foods (NCEFF), University of Wollongong supervise the research.

Your participation in this research is voluntary, you are free to refuse to participate and you are free to withdraw from the research at any time. Your refusal to participate or withdrawal of consent will not affect your treatment in any way your relationship with the NCEFF, the Smart Foods Centre or your relationship with the University of Wollongong. All results and information is confidential and your name will not be used. All data will be collected by me and stored (confidentially) in the department.

I,.....(Participant's name) consent to participate in the research conducted by Craig Patch. Specifically I consent to:

- Participation in a 1 hr interview group either via telephone of mutually agreeable venue.
- The interview will be audio taped, analysed and reported upon.
- My identity will remain confidential to the researchers.

I understand that the data collected will be used for research and I consent for the data to be used in this manner.

_____
Signed Date

..../...../.....