# FOOD AVAILABILITY AND FAT COMPOSITION OF DIET IN EASTERN PROVINCE, SAUDI ARABIA, WITH REFERENCE TO CORONARY HEART DISEASE

*By*

*AHMED ABDULLAH ALOBAID*

A thesis submitted in partial fulfilment of the
requirements of the University of North London
for the degree of Doctor of Philosophy

**To**

School of Life Sciences
University Of North London, London, United Kingdom

**MAY 1995**

The following images have been redacted at the request of the university:

# ABSTRACT

This presentation discusses the first study of its kind conducted in the Eastern Province in Saudi Arabia. The study population was selected from three areas, representing different socio-demographic conditions i.e. urban, rural and Bedouin areas. The aims were to provide information on food availability and the coronary heart disease mortality and its implications among the population, taking into consideration fat and fatty acid profile in the diet.

The data were collected in ***three*** approaches; (1) food availability survey based on household food purchasing for five consecutive days, (2) coronary heart disease proportion mortality survey based on the recorded deaths from six Ministry of Health (public) Hospitals from 1989 to 1990 and (3) the experimental work, involved the determination of fatty acid profile of foods identified in the first study.

Our results demonstrate that there are marked differences in food availability as well as dietary habits amongst the three groups studied. The fat and fatty acid profile of the analysed foods demonstrated that many Saudi foods contained a high amount of saturated fatty acids. The coronary heart disease proportional mortality of the study region was comparable to that in the UK. They also show a significant difference between Bedouin and non-Bedouin coronary heart disease proportional mortality, which may be due to dietary as well as non-dietary risk factors.

There were a number of inherent biases associated with the methods used in this study which made our data interpretation difficult. We hope that the present work will stimulate other researchers in this field.

# ACKNOWLEDGEMENTS

I wish to express my sincere gratitude to my understanding supervisor, Dr. Brian B. Bointon, Head of the School of Life Sciences, for his invaluable guidance and total support of this research. Without his advice, criticism, patience, kindness and unfailing encouragement, this work could not have been completed.

I am, ever, grateful to Dr. Margret Lawson, Senior lecturer, paediatrics and Nutrition Department, London Institute of Child Health, where a study such as this would not have been possible without the collaboration, assistance, experience and advice received from her.

Special thanks are due particularly to Professor Robert Gilchrist, Head of the Statistics and Operation Research Centre, for his tremendous advice and help in the data statistical analysis, which made this study very significant and worthwhile. Also I extend my thanks to Miss M. Sanderson, course tutor in Human Nutrition and Dietetics Department, for her assistance during the study. Deep appreciation is extended to Dr. M. Ghavami for his laboratory assistance.

Special appreciations must also go to my family for their patience, love and financial support throughout my graduate career.

I am grateful to the families who participated in this study, for their co-operation and patience they showed in keeping the detailed food records, which ensured the successful completion of this study.

The study could not have been undertaken without the help and support provided by the office of the Saudi Education Attache in England, in particular to its director Mr. Abdulla Al-Nasser, and King Fasal University, Nutrition and Food Science Department, in particular Dr. M. Al-Sharwy for his assistance in the survey field. Also I am grateful to the Ministry of Health and to the Health Affairs in the Eastern Region for permission to undertake this study, and to the hospital directors and to the staff of the Statistics Department at the Ministry of Health Hospitals, Eastern Province.

Among others, too numerous to mention, who helped me at various stages of the study, I would especially like to thank Mr. Abdelaziz Alobaid, Mr. Zuhair Olyabek, Mr. Hussein Youssef, Mr. Saeed Alobaid, Mr. Abdalaziz Albesher, Mr. Sami Albesher and to many individuals who have given me so much help.

Last but not least, I would like to thank the members of the staff of the library in the University of North London Library and the British Science Library for their tremendous cooperation.

## DEDUCATION


To Those I Love Most
My Parents, My Sisters
And To The Memories Of My Grandmother

## TABLE OF CONTENTS

## *CHAPTER 4*

## *SECOND STUDY : CORONARY HEART DISEASE SURVEY*



## *CHAPTER 5*

## *EXPERIMENTAL WORK: FAT AND FATTY ACID PROFILE*



## *CHAPTER 6*

## *GENERAL DISCUSSIONS AND CONCLUSIONS*

## LIST OF TABLES

# LIST OF FIGURES

# LIST OF APPENDICES

# LIST OF ABBREVIATIONS

| | |
|---|---|
| AA | Arachidonic Acid |
| ALA | Alpha-Linolenic Acid |
| BMA | British Medical Association |
| BMI | Body Mass Index |
| BSI | British Standards Institution |
| CHD | Coronary Heart Disease |
| COMA | Committee on Medical Aspects of Food Policy |
| CVD | Cardiovascular Disease |
| DCH | Dammam Central Hospital |
| EFA | Essential Fatty Acid |
| EP | Eastern Province |
| FA | Fatty Acid |
| FAME | Fatty Acid Methyl Esters |
| GCH | Gatif Central Hospital |
| GGH | Gatif General Hospital |
| HDL | High Density Lipoproteins |
| ICD | International Classification of Disease |
| IFFD | Intra-Family Food Distribution |
| JGH | Jubale General Hospital |
| KFH | King Fahad Hospital |
| KKH | King Khiald Hospital |
| LA | Linoleic Acids |
| LDL | Low Density Lipoproteins |
| MUFA | Mono-unsaturated Fatty Acids |
| MY | Myocardial Infarction |
| NACNE | Committee on Medical Aspects of Food Policy |
| P/S | Polyunsaturated to saturated fatty acids ratio |
| PG | Prostaglandins |
| PM | Proportional Mortality |
| PMR | Proportionate Mortality Ratio |
| PUFA | Polyunsaturated Fatty Acids |
| RAV | Dietary Reference Values. |
| RDA | Recommended Dietary Allowances |
| RNI | Recommended Nutrients intake |
| SA | Saudi Arabia |
| SAC | Society for Analytical Chemistry |
| SAFBS | Saudi Arabian Food Balance Sheets |
| SFA | Saturated Fatty Acid |
| SMR | Standardised Mortality Ratio |
| SR | Saudi Riyals |
| STCC | Serum Total Cholesterol Concentration |
| Tri | Triglycerides |
| TXA | Thromboxane |
| UFA | Unsaturated Fatty Acids |
| VLDL | Very Low Density Lipoproteins |

## GLOSSARY OF TERMS USED IN THE SURVEY

| | | |
|---|---|---|
| **Urban households** | : | Are those which are located in cities irrespective of size. |
| **Rural households** | : | Are those which are located on the outside, the boundaries of the cities. |
| **Bedouin households** | : | Are those which are located in the desert (HEJRA). |
| **Group (cashment)** | : | This referred to the urban, rural and Bedouin communities. |
| **Monthly income** | : | This includes all the income from the salaries of all the members of the household, but not from income, achieved from self employment. |
| **Household members** | : | This was defined as a group of people who regularly reside together in the same accommodation and who share the same catering arrangements, excluded are those who usually live in the house but for some reason or other are away during the survey period. This generally consists of the husband, wife, children and may also include some relatives or domestic servants. |
| **Household Head** | : | Is defined as the person chiefly responsible for the economic maintenance of the household, usually the father or the older son. |
| **Housewife** | : | Is defined as the person chiefly responsible for the meal preparation, cooking, usually the mother. |
| **Adult** | : | A person of 18 years of age or over. |
| **Child** | : | A person under 18 years of age. |

| | | |
|---|---|---|
| **Infant** | : | Child under one year old. |
| **Food Purchases** | : | This term used to donate all food coming into the home whether purchased or free. |
| **Proportional Mortality** | : | This terminology used to describe the recorded coronary heart disease deaths as a proportion of the number of adult deaths in each hospital in our study. |

Chapter One

# INTRODUCTION

Chapter 1

# INTRODUCTION

## 1.1 General Introduction

After the second world war cardiovascular disease (CVD), particularly coronary heart disease (CHD), became the leading public health problem in most of the industrialised countries and this group of diseases is also emerging as a prominent public health problem in developing countries (WHO, 1982). However, declining trends in CHD mortality have been observed in some industrialised countries (e.g. USA, Australia, Finland, New Zealand and Iceland), whilst CHD mortality is stable in some and rising in others (Uemura & Pisa, 1988). Nevertheless, there are enormous differences in CHD morbidity and mortality between these countries and within different population groups within each country. There has been a striking increase in CHD and the so-called "*the modern epidemic diseases*" in societies that have undergone dramatic changes in their life-style, in particular food habits. Notably, among such population are the Japanese (Yuichiro, 1992). Epidemiological studies over the past three decades have established a link between the symptoms of the disease and the various so-called "*risk factors*" which might cause the disease. It is generally accepted that there is no single cause of heart disease but there are many interrelated risk factors.

The Kingdom of Saudi Arabia has undergone an unprecedented change towards urbanisation and industrialisation. This has been accompanied by improved socio-economic status and change in the dietary habits of the people. The only survey in that period was carried out by Sebai (1983), in 1967 among three communities (settled, semi-settled and nomadic) living in Turaba in the Western Province. He reported that families in the settled area consumed more meat, milk, vegetable and fruit than families in the other two groups who lived mostly on rice, bread and dates. In his

follow up study in 1981, observed that the eating habits have changed since 1967, he found that rice and bread still constituted the staple diet. Whereas the average family meat was purchased more than before. An analysis of the Saudi Arabian Food Balance Sheets (SAFBS) No. 2 (CDESS, 1988), from 1974 to 1986, published by the Ministry of Agriculture and Water, indicated that the per capita availability of calories for consumption increased by 67%, fat by 179%, and protein by 64%, paralleled with an increase in consumption of oil and fat (319%), eggs (272%), chicken (128%), meat (114 %). In comparison, there was a slight increase in the use of cereal products (30%), fruit (49%), and vegetable (69%). This might suggest, given the existing etiological diet-CHD hypothesis, that there would follow an increase in the incidence of CHD in the Saudi Arabia (SA). However, to relate diet and CHD incidence among the Saudi is a great over-simplification for three main reasons. Firstly, CHD is a multifactorial disease and there are other environmental influences. Secondly, in SA, up to now there has been no data available, part of the previous two references, relating to the past or the present dietary habits of the population. Finally, there is no published data concerning CHD incidence in SA. Within any health program it is not until an adequate information system has been established, that understanding of the subject becomes possible. In view of these data shortages and emerging evidence concerning the relationship of CHD incidence and lifestyle changes, the importance of such a study in a country like SA is noted. It is the aim of this work to investigate food consumption and CHD in SA.

This introductory chapter sets the context for this study in terms of food consumption in SA, CHD in SA, and the aetiology of CHD in relation to diet. The particular problems of such research in SA are noted and a brief outline of the objectives of this research and a plan of the thesis are contained in this chapter.

## 1.2 Food Consumption

Information concerning the habitual dietary intake of populations is of great importance for both epidemiological research as well as for decision making in nutrition, food and

health fields. Indeed, the eating habits of many traditional societies are presently being influenced by the process of modernisation. SA is one such country that has experienced rapid modernisation in the last three decades. Little is known about the dietary consumption and pattern of the population in SA; even less is known about the fatty acids (FAs). Nevertheless, a few reports have been found which analyse one or two aspects of nutrition and/or food composition in small communities and using a few samples (Sawaya et al., 1987; Sawaya et al., 1984; Al-Jebrin et al., 1985ab). The SAFBS (previous reference) with all its limitations, is the only source available on food production, availability and consumption per person in terms of energy, protein and fat. This aggregate data, however, does not provide a complete picture of the adequacy of the diet within different socio-economic strata in the Saudi population or the seasonal fluctuations in food availability, but it may give a general idea of the major features of food consumption. For the nutritionist its greatest disadvantage is that food availability and actual consumption and distribution within the family, may be very different.

It can be seen from the above that the investigation of the food consumption concerning dietary habits and food availability in the SA is very important. Therefore, a food survey among the Saudi population in the Eastern Province of SA was carried out (***THE FIRST STUDY***). One of the serious difficulties faced by the author in this study was to find the suitable dietary survey method to be applied in our study population. A review of the literature led to the identification of the advantages and disadvantages associated with existing dietary methods. As a result, household food consumption based on the purchasing food method was decided to be the most suitable for the type of the population studied (the reasons are fully discussed in Chapter 3). One hundred private Saudi households, selected at random from urban, rural and Bedouin areas, participated in this study. The energy and nutrient (fat, protein, carbohydrate and fibre) were calculated using the food composition table "Food Composition Table for use in Bahrain" (Musaiger & Al-Dallal, 1985). The Nelson (1986), family intake distribution technique was used as no equivalent data is available

for the Saudi people. The results of that analysis were then compared with the recommended dietary intake (RNI), as suggested by FAO/WHO/UNU Expert Committee Report (1985).

## 1.3 Coronary Heart Disease (CHD)

CHD incidence index is considered a vital parameter in evaluating the level of changes in life-style in a country. This is because CHD, in part, is regarded as a disease of affluent lifestyle. Unfortunately, there is a great paucity regarding CHD information in Saudi Arabia. A thorough search of the Saudi Medical Bibliography between 1984 and 1987, and also the WHO annual mortality statistics failed to identify any published article relating to CHD mortality (Madkour & Kudwah, 1985). Furthermore, a Medline literature search back to 1970 did not reveal any pertinent publication. However, the mortality data available from the other Arab Gulf countries, which share similar tradition, culture and economic conditions, shows that diseases of the circulatory system are responsible for more than 30% of causes of death in these countries (Musaiger & Al-Dallal, 1983; Kohli & Al-Omaim, 1986).

A second objective of this study was to investigate the CHD mortality in EP (***THE SECOND STUDY***). This was done by carrying out a survey in the Ministry of Health (public) Hospitals, which are the main sources of mortality data in SA. The mortality data in six hospitals were reviewed and analysed. Note, however, this study cannot determine CHD incidence (this will be discussed later). The present data providees only the proportional mortalities (PMs); i.e the observed deaths from CHD.

## 1.4 Fat and Fatty Acids (FA) Profile and CHD

The CHD-lipid hypothesis has been and still is the leading hypothesis on aetiology; thus relates the incidence of CHD, through serum cholesterol concentrations, to the fat in the diet, and more particularly to the saturated fatty acid (SFA) content and the ratio of polyunsaturated to saturated fatty acids (P/S). In most countries, high intake of saturated fats is strongly correlated with high mortality from CHD. However, same

populations with a high level of consumption of SFAs may not have a high rate of CHD. Dramatic example of this come from studies in France. This paradox has been attributed, in comparison with the UK, to the higher consumption of wine, fresh fruit and lower intake of trans fatty acids from margarine, and massive obesity is less in France.

The focus of recent research by scientists in this field has been on the type of the FAs rather than the total dietary fat. Barr et al., (1992), concluded that reduction of dietary fat intake from 37 to 30% of calories did not lower plasma total and low density lipoprotein (LDL) cholesterol concentrations unless the reduction in total fat was achieved by decreasing SFA. A recent study by Morgan et al., (1993), reached the same conclusion. However, total dietary fat is also important as it could contribute to obesity, which is one of CHD risk factor. Recently a new hypothesis has emerged indicating that the susceptibility of LDL to oxidation should be considered an important risk factor of CHD (Salonen et al., 1992).

A comparison of trends in CHD mortality and food consumption in the USA between 1909 and 1980, showed that changes toward a diet lower in SFAs and cholesterol preceded the national decline in CHD mortality (Slattery & Randall, 1988), although the decline can be attributed only partly to these dietary trends.

Again in this aspect, there is no data available in SA for population FA intake nor data available for FA profile in Saudi food. Another problem faced by the author was that the food composition table used in this study although designed for use in a Middle Eastern countries, were not specific to SA and had no FA data.

Thus, the final stages of this study was to carry out an investigation concerning further analysis (***THE EXPERIMENTAL WORK***) of fat and the FA profile of the diets of the subjects studied. This study enabled us to accurately predict the fat in diet and also the FA profile for the three population groups. This was achieved as follows:

1. Food identified in the survey (***FIRST STUDY***) as a significant source of fat in the population's diet were sampled at the point of sale. The sample was then determined according to the number and type of source of each significant food item and the likely variability of the composition of the food item concerned.

2. The fatty acid spectrum was obtained by capillary gas chromatography of the prepared methyl esters.

3. A data base for the fat content and fatty acid composition of foods making a significant contribution to the food available of the Saudi population was then established.

4. The contribution of foods to the total fat availablehas been identified. This information was used in conjunction with the data described above to refine the data relating to fat available for consumption and to determine the fatty acid profile in the household diets.

5. A new comparison on the basis of the computed data obtained in **5** was made between different groups (i.e. urban, rural and Bedouin) studied with a further refinement relating to the type of fat consumed by these groups.

6. Total fat and energy availables of individual FA and the P/S ratio were studied along with data on the rates of CHD in different studies to examine the possible interactions and correlations between dietary, ethnic and lifestyle factors with CHD rates.

## 1.5 The Study Population

Every society has some type of social stratification within it. The Kingdom of Saudi Arabia can be conveniently divided into three clearly stratified groups; which are (1) urban, (2) rural, and (3) Bedouin. Their life-style is quite well defined in relation to

their traditions, cultures and social pattern. Individual variation within each mentioned group and within the person's own group may also be observed in relation to social class such as occupation, education, etc. It has been well documented that social, economic, cultural and other very important environmental factors are associated with the aetiology of particular diseases such as CHD (Crombie et al., 1989). Diet, in particular, is one of the most important of these factors in response to pathology of CHD. The World Health Organisation (WHO), and other major organisations believe that CHD mortality can be cut by at least half by changing to a healthy lifestyle. The Lifestyle Heart Trial in the USA, shows that comprehensive lifestyle changes may be able to bring about regression of coronary atherosclerosis after 1 year, without the use of lipid-lowering drugs (Ornish et al., 1990).

Therefore, an attempt has been made by this study to identify various socio-economic factors that might affect the nutritional status and CHD of the population studied by dividing the population into the three groups noted above. Discussion in this work evaluates differences between these groups.

## 1.6 Geographical Area of the Study

The study was carried out, in the Eastern Province (EP) of the Kingdom of Saudi Arabia, because:

1. It is the most industrialised, and has undergone radical changes within the last two decades.
2. All aspects of the life-style have undergone dramatic changes both in culture and in environmental.
3. The province has witnessed an intense emigration from within and outside the country. Studies in many countries have shown that migrations have a great impact on life style, particularly food habits.

Of course, the effect of the above changes could be positive and/or negative on the nutritional status of the community and therefore alter their state of health, as will be

examined in this study.

## 1.7 Scope and Objective of the Thesis

An assessment of the food consumption and CHD of the EP population is of particular interest, because of the reasons explained earlier. The current study is the first to describe the food consumption and fatty acid profile of foods commonly consumed in the EP (and the whole country), and there has been no detailed published study about CHD incidence in the Kingdom. In response to the paucity of data mentioned above and the requirements needed for accurate and reliable information in SA, and understanding the importantance of such information, this work has undertaken the task of investigating the prevalence of CHD in the EP of the Kingdom of Saudi Arabia, with particular reference to the consumption of fat and the FA profile of the diet.

***The main objectives of this study concern the following aspects:***

1. To investigate the food availability and dietary habits in the EP.

2. To investigate fat content and fatty acid profile of foods in the EP.

3. To estimate the proportionate deaths in the observed CHD and to investigate possible relationship between CHD and urbanisation in the EP.

Such information could shed light on the present concepts of the Saudi nutrition status and its related diseases, particularly CHD. Which may provide physicians, nutritionists and policy makers with a quick reference source on the state of food habits, nutrition and health in SA. Furthermore, it provides the foundations for other researchers to carry out more investigations in this area.

## 1.8 Structure of the Thesis

The thesis consists of six chapters, the initial stages which includes general background of various topics. The following chapters cover the food consumption

survey and the CHD mortality survey carried out and the experimental work.

***Chapter two:*** is a literature survey which gives a brief look on the geographical area of the study that includes history, economic as well as the social, food and nutrition situation and factors associated with it. These are studied in the light of the economic prosperity brought about by oil revenue and the consequent changes in the health education as well as social and dietary attitudes. It also reviews the pathogenesis, prevalence, mechanism, risk factors and the clinical feature of CHD. Furthermore, it discusses the CHD-lipid hypothesis in terms of the effect of different lipids and the aetiology and the pathology of the CHD.

***Chapter three:*** outlines details of the food consumption survey (***FIRST STUDY***). It presents the full detail of the method used in the survey to assess the nutrient available of the subjects and also the method used for estimating the intra-household food distribution. The difficulties encountered in the survey were also discussed. Finally, it presents the assessment of the result.

***Chapter four:*** covers details of the CHD mortality survey (***SECOND STUDY***) method, results and discussion of the surveys are described.

***Chapter five:*** outlines the fat and fatty acid profile of the common foods sampled from the first study (***EXPERIMENTAL WORK***). Details of experimental work; method, results and discussion are described.

***Chapter six:*** contains a summery of the results of Chapter 3, 4 and 5. It also covers details of the general discussions and conclusive remarks of the implication of this studies main findings. In addition, it outlines several areas of future research directions suggested on the basis and experience of this study.

Chapter Two

# GENERAL BACKGROUND

## 2.1 The Region Studied

2.1.1 Eastern Province
2.1.2 Area, Location and Climate
2.1.3 Population
2.1.4 Population Structure
2.1.5 Recent Economic Changes
2.1.6 Food Situation
2.1.7 Dietary Changes
2.1.8 Traditions and Social Activities
2.1.9 Intra-Family Food Distribution
2.1.10 Health Service

## 2.2 Coronary Heart Disease (CHD)

2.2.1 Definition
2.2.2 Prevalence
2.2.3 Coronary Arteries
2.2.4 Pathology
2.2.5 Mechanism
2.2.6 Plasma Lipoproteins
2.2.7 The Clinical Feature of CHD
2.2.8 Risk Factors
4.2.9 Major Risk Factors Associated with CHD in SA
4.2.10 Other CHD Risk Factors in SA
2.2.11 CHD and Dietary Prevention Guidelines

## 2.3 Dietary Lipids and CHD

2.3.1 The CHD-Lipid Hypothesis
2.3.2 Effects of Different Lipids in the Diet
2.3.3 Epidemiological Studies
2.3.4 Atherosclerosis and Lipid in Experimental Animals
2.3.5 Lipid Per-oxidation and Atherosclerosis
2.3.6 The Role of Anti-oxidants

Section 2.1

# THE REGION STUDIED

## 2.1.1 Eastern Province (EP)

The Kingdom of Saudi Arabia is divided into provinces called governorates (Mantikah); the largest of these are the Central, Western, and the Eastern (Figure 2.1). Each of these has a capital and a number of towns and villages linked to it. They are headed by a governor (Emir) who is the chief administrator according to government regulations. For the purpose of this research (see Chapter 1) only **EP**, Al-Mantika Al-Sharkiya, will be studied. It is well known in the world for its massive petroleum resources. Within "***a coast of black gold***" as it is called by Almuslim (1962), the development of the oil industry has brought even greater increases in prosperity to this province than those seen in the Kingdom as a whole. The fabric of life has altered, poor living conditions, shortage of natural resources, poor health services and sanitary conditions have been replaced by a high socio-economic standard of living and better health care facilities. A population of farmers and fishermen has become an industrial work force; a largely rural population has become urbanised, as cities such as Damman, Al-Kubar, Al-Gatif, Jubail and Hafar Al-Batin areas have grown from small villages to towns and cities. In addition, it is also considered to have an integrated economy, covering agriculture, trade, and industry. It also has a long historical background which dates back to ancient time, 3000BC. Evidence from excavation conducted by the antiquities department has revealed that the region was the first urbanisation settlement centre in the peninsula. The contemporary population of the region today are the descendants of ancient tribes. During its long history the province has been given several names, Al-Hasa, Al-Gatif, Khatt, Hajar and Delmon (Almuslim, 1962; AMME, 1977, 1978, 1982).

## 2.1.2 Area, Location and Climate

EP lies in the eastern part of the Kingdom on the Arabian Gulf coast. It is the largest province in the Kingdom, covering approximately 726,059 sq km. It is surrounded by desert; Al Dahna to the west and Rub-Alkhali to the south, and the Arabian Gulf to the east. The major districts are the Oases of Al-Hasa and Al-Qatif, famous for their production of dates, and the district of Hafr Al-Batin in the north where most of its

inhabitants are Bedouins.

EP climate is moderate in the winter, which starts from the end of December until mid April. Starting from May, the weather tends to get hot with temperatures of over 50°C (122°F) accompanied by humidity in coastal area and dry heat inland, with strong seasonal north west winds causing major sandstorms. The annual rainfall is irregular and averages between 4 inches (100mm) and 8 inches (200mm) in the north and even lower in the south.

The province has great graphical diversity, with vast deserts and long beaches in addition to mountainous regions. Huge sand dunes with altitudes exceeding tens of meters are features of the desert. Oases with highly fertile soil are another aspect of this area. Unfortunately most of these oases were effectively drowned by moving sands, Alrub Alkhali "*Empty Quarter*" is considered to be the greatest desert occupying two thirds of the Kingdom. Aldahna is another large desert in the area.

### 2.1.3 Population

The first census held in September 1974 showed the total population of the Kingdom to be circa 7 million of which about 47% were urban, 28% rural and 25% were Bedouins. EP's population was estimated to be around 1.5 million of which 66% were urban, 24% rural and 10% were Bedouins. More recently the UN estimated Saudi Arabia's population to have been around 11 million (mid-1984). The Saudi official estimate for mid-1989 is just over 14 million (MENA, 1993). The growth in the population is mainly due to an increase in the number of expatriates and the national population living longer due to the improved standard of living and health services. The only upto date data available on population distribution by age and sex is shown in Table 2.1, obtained from the 1974 nationwide survey. Almost half of the population are children under the age of 15 years. Together, women and children make up approximately three quarters of the population (MHE, 1981). On average the population consists of 53.2% male and 46.8% female. The age group 15-19 and above shows an unexpected drop in the female population when compared to male age groups (i.e. the number of males is higher than females population). This may be an error the source of which may be due to one of the followings:

1. Miscalculation.
2. The correct number of female members of the family was not given.

3. The correct age of female members of the family was not given.

### 2.1.4 Population Structure

The social and cultural structure of a community in SA is influenced by different traditions in various regions. Saudi people may be divided into two main demographic groups, a settled and a non-settled population.

The settled population in the Kingdom may be further sub-divided into two groups, urban and rural. Urbanisation is an important factor in determining population change in SA. In 1990 an estimated 77% of the population resided in the urban area compared with 39% in 1965, with an annual average growth of more than 5% (MENA, 1993). This increase in urbanisation is a result of large numbers of immigrants from rural and Bedouin areas seeking the high income and better social services available in the cities. The rural population, on the other hand, has undergone extraordinary changes over the years developing into a more urbanised way of life. This is mainly due to the availability of modern services as a result of the country's economic prosperity.

The Bedouin population may also be divided into two groups. Semi-settled (in oases and cities) and non-settled. Settlement (Hejar) areas were established 80 years ago by the late King Abdul Aziz, and have developed rapidly due to the improved economy of settled life and the encouragement of the government. Non-settled nomads migrate over the desert throughout the year. In 1967 the nomads were 30% of the Kingdom's population, but by 1981 this proportion had decreased to less than 25%. However, despite the rapid development of the Kingdom, Bedouin life has undergone little change over the years. Generally, desert inhabitants suffer from limited resources compared to other parts of the country.

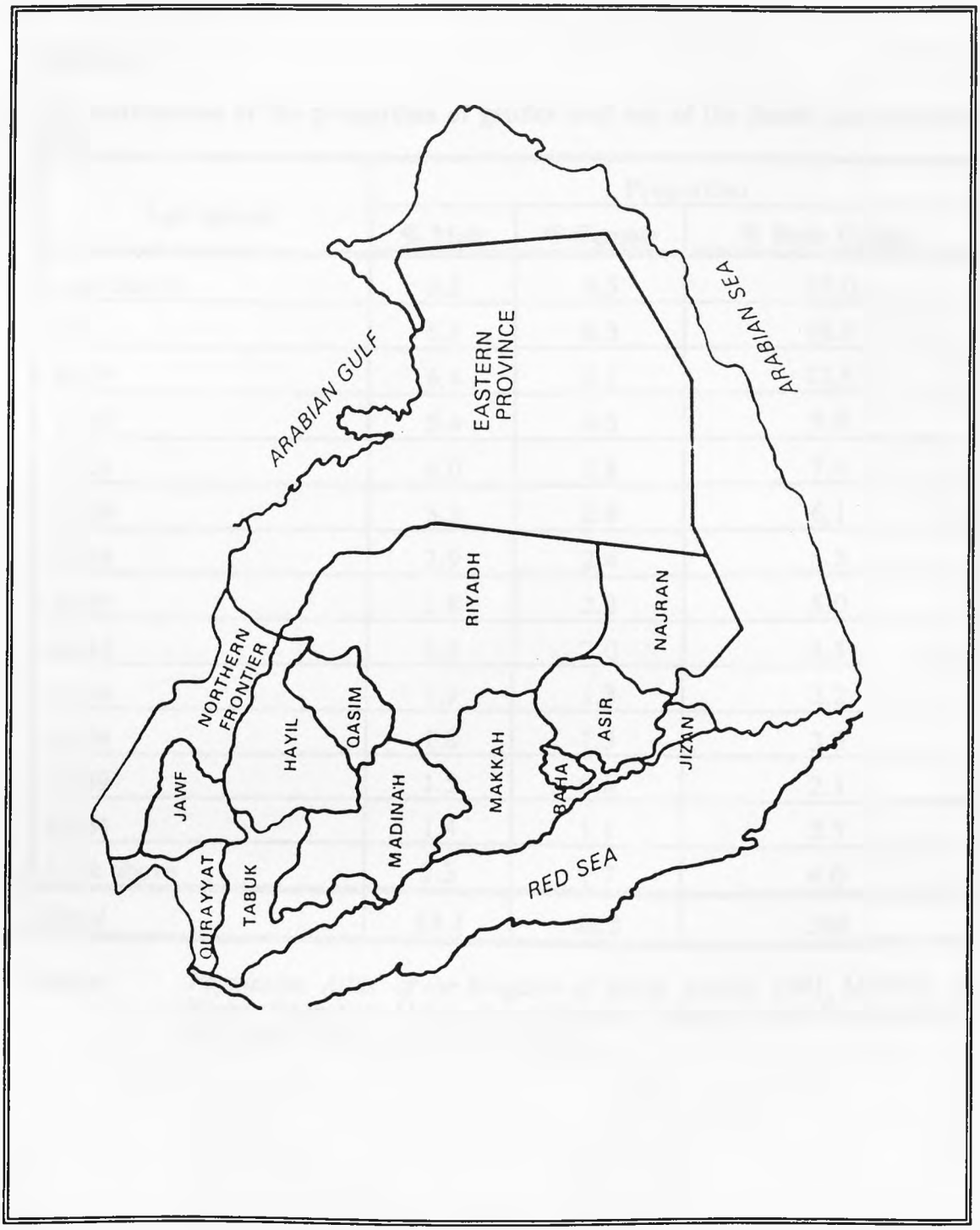



**Fig. 2.1**

**Kingdom of Saudi Arabia, Administrative Divisions**
**Source:** *H. Bendagiy. Atlas of Saudi Arabia; Copyright 1980, Oxford University Press.*

**Table 2.1**

**The distribution of the proportion of gender and age of the Saudi population in 1974**

| Age (years) | Proportion | | |
|---|---|---|---|
| | % Male | % Female | % Both Gender |
| Less than 5 | 8.5 | 8.5 | 17.0 |
| 5-9 | 8.5 | 8.3 | 16.8 |
| 10-14 | 6.4 | 6.1 | 12.5 |
| 15-19 | 5.4 | 4.5 | 9.9 |
| 20-24 | 4.0 | 3.8 | 7.8 |
| 25-29 | 3.3 | 2.8 | 6.1 |
| 30-34 | 2.9 | 2.4 | 5.3 |
| 35-39 | 2.8 | 2.2 | 5.0 |
| 40-44 | 2.5 | 2.0 | 4.5 |
| 45-49 | 1.9 | 1.3 | 3.2 |
| 50-54 | 1.8 | 1.5 | 3.3 |
| 55-59 | 1.5 | 0.6 | 2.1 |
| 60-64 | 1.4 | 1.1 | 2.5 |
| 65 & above | 2.3 | 1.7 | 4.0 |
| ***Total*** | ***53.2*** | ***46.8*** | ***100*** |

**Source:** *Population Atlas of the Kingdom of Saudi Arabia;* 1981, Ministry of Higher Education, University of Riyadh, College of Arts Department of Geography Saudi Arabia (in Arabic).

## 2.1.5 Recent Economy Changes

Since 1970, the development of the economy in the Kingdom has been guided by a series of five-year plans. These plans have played an essential role in guiding the economic and social development during the past two decades. The Kingdom's economy is basically an oil-based one. It produced 10.3% of the total world production of oil in 1990 making it the third biggest producer in the world and the biggest within the OPEC organisation. The oil industry alone supplied 72.5% of the gross national product (GNP). The Kingdom is economically stable and enjoys a strong foreign exchange position with a surplus balance of international payments. The GNP grew from 6,543 million Saudi Riyals (SR) in 1965 to 382 billion SR in 1979 and 553 billion SR in 1982. The per capita GNP per head reached $7,060 in 1990, compared with $6,430 in 1989. Development in the public sector has focused on health, agriculture, education, communication, water reserves and mineral deposits. In the private sector the effort has been to encourage private investment to attract outside expertise for industrialisation (MENA, 1993). In this context, the Kingdom of Saudi Arabia could be considered a typical example of a country that has experienced very rapid modernisation in a very short period of time. Such development has had both negative and positive side effects on the SA community.

*a. Positive side effects*:

Higher living standards, better health and housing services. Improvements have also occurred in other public services such as transport, education etc. In addition, a greater variety of food stuff is now available in local markets and there is widespread access to the latest in consumer product/electrical appliances.

*b. Negative side effects*:

This economic shift has resulted in rapid changes in lifestyle, which may lead to the emergence of affluence-related nutritional problems such as CHD, obesity, diabetes, etc.

## 2.1.6 Food Situation

*1. Food Policy*

The government recognises that the only way to reach self-sufficiency in food is by improving the agricultural sector. Over the past ten years agriculture has rapidly developed in the Kingdom. Moreover, in 1988, the agricultural sector became the

most important productive sector, second to petroleum, forming about 40% of non-oil exports. Despite the problems of poor climate, water shortage and the nature of Saudi land, during 1984 to 1988 there was an increase in some agricultural production. Exports have increased from 165m SR to 1220m SR. Wheat, eggs, poultry, dairy products, fish, dates and watermelon are the main products.

### *2. Major Agricultural Products*

#### a. Wheat:

Wheat is considered to be the most important crop, representing 95% of total cereal production in the Kingdom. In 1987 the Kingdom became the worlds sixth largest exporter of cereals. In 1981/1982 the Kingdom's wheat production was 417 thousand tons which has increased to 3.9m tons in 1990/91. The local demand is estimated at about 1m tons a year, thus enabling the Kingdom to export wheat (MENA, 1993).

#### b. Dates:

The Kingdom of Saudi Arabia is the world's leading producer of dates (MENA, 1993). In 1985/86 the area of date agriculture was estimated to be more than 6 thousand hectares, with a total production exceeding 459,829 thousand tons. This enabled the Kingdom to export dates to neighbouring countries. In addition, large quantities of dates are donated by the government to be distributed in famine stricken countries.

#### c. Meats, Poultry and Poultry Products:

The Kingdom is approaching self-sufficiency in white and red meats, which in 1988 and 1989 reached 75% and 72% respectively of the total demand. In 1973 the Kingdom's production of eggs was only 6 thousand tons, thus resulting in the Kingdom importing 42% of its production to meet demands. Since 1985 the Kingdom has exceeded production demand by more than 9% (MAWDESS, 1988; MP, 1990).

#### d. Dairy and Dairy Products:

The Kingdom has been self-sufficient in milk production since 1985 and some of its production is exported to neighbouring countries. In 1975 there were 8 dairy factories in the Kingdom. By the end of 1989 there were 55 factories producing 600 thousand tonnes of milk and dairy products, such as yogurt, white cheese, ice cream, cream and long-life milk etc. (RNP, 1991).

*4. Food Imports*

Despite all the efforts and development, the contribution of agriculture to gross domestic product does not exceed 2%. This may be because of the environmental problems and the rapid demand for food commodities in the area. Therefore SA, as other Arab Gulf countries, is mostly dependent on food imports. The value of food imports for the gulf countries was 8 billion dollars in 1990 exceeding the total value of foods imported in all other Arab countries by one-third (RNP, 1991). Bearing in mind that the population of the Gulf countries represents only 12% of all Arab countries.

## 2.1.7 Dietary Changes

Analysis of the SAFBS No. 2 (CDESS, 1988), reveal that the nature of the Saudi diet has changed since 1974. Comparison of the most recent data in the 1974/76 and 1983/86, show that the apparent consumption of almost all foods and nutrients had increased. The results are presented in two sections: food groups and nutrients.

*i. Trends in Food Consumption*

Foods were classified into 11 groups. Table 2.2 shows trend in food availability in SA of major food groups during the periods 1974-1976 and 1983-1986. The overall per capita consumption shows a 64% increase in total foods, of which a 58% increase in foods of plant origin and a 92% increase in that of animal origin.

*Cereal and Cereal Products*

In the period studied availability of cereal and cereal products increased by 30% upto 336 grams per capita per day in 1983/86, with rice and wheat-flours providing the major contribution to cereal availability. Rice constituted 31% of total cereal availability, up to 103 gram per capita per day in 1983/86; whilst the availability of wheat flour show a 45% increase by the time of the 1983/86 survey, representing about 58% of total cereal availability.

*Fruits*

The availability of fruit increased by 48.5% up to 427 gram per capita per day. Fresh fruit was preferred to prepared fruits such as canned fruit, 88% and 12%, respectively, in 1983/86, although the availability of prepared fruit has increased 3-times more than availability of fresh fruit over the period. The most common fruits consumed are dates, banana, orange, apple, grape and watermelon.

*Vegetables*

Vegetable availability shows a 69% increase, from 163 in 1974/76 up to 276 gram per capita per day in 1983/86. As with fruit, fresh vegetables were preferred to prepared products. Prepared vegetable such as frozen or canned showed a greater increase (157%) in the survey time than fresh vegetables (64% increase). The most vegetables consumed are tomato, lattice, cucumber, onions, okra, aubergine.

*Animal Food*

Animal foods included meat, fish, eggs, milk and dairy products. Chicken and red meat availability have increased by 281 and 114%, respectively, whilst fish availability has increased by 128% up to 21 gram per capita per day. Availability of eggs increased by 272% up to 25 gram per capita per day in 1983/86. Milk and dairy products showed the lowest increase of all food groups, just 21% up to 112 gram per capita per day. Fresh milk declined by 12% over the four study periods from 79.8 in 1974/76 to 69.9 gram per capita per day in 1983/86. Perhaps this was due to the facts that the food balance sheet specifically states the consumption of fresh milk, where as consumption of yoghurt is not taken into account. In SA, the consumption of yoghurt and yoghurt drink (Laban) is preferred over milk particularly when the whether is hot, chilled yoghurt drink is consumed on daily basis as can be seen from this study (see section 3.2).

*Oils and Fats*

The major change is reflected in oils and fat availability which have increased by 319% from 11 in 1974/76 to 46 gram per capita per day in 1983/86. Vegetables oil/fats showed a 376% increase and animal oil/fat 154% increase.

*Sugars*

Availability of total sugar (i.e. including sugar confectionary and honey) has increased by 73% by 1983/86 up to 83 gram per capita per day, but should be noted that there was an apparent increase by 1980/82 of 113% compared to 1974.

## 2. Trends in Nutrient Levels

Shifts in the kinds and amounts of food making up the Saudi diet are reflected in the levels apparent nutrients available for consumption (Table 2.3). Changes in energy-yielding nutrients, particularly in fat, is of special interest. The level of available food

energy has risen from 1807 to 3012 kcal per person per day, an increase of almost 67% in 12 years.

Figure 2.2 depicts trends in protein, fat and carbohydrate in SA from 1974 to 1986. Protein provided about 11% of energy throughout this period. In 1974/76, fat contributed only 16.7% of total energy intake, but that proportion had increased up to 28.5% by 1983/86. The share of carbohydrate has dropped from 71.9% to 60.4%. As can be seen from Figure 2.2, this has been accompanied by an equivalent increase in fat intake.

The amount of total apparent protein available rose from 51 to 84 gram per capita per day, an increase of 64%. The most significant change associated with protein availability is the shift in its sources. Animal products provided almost 45% in 1983/86, compared with 31% in 1974/76 (Table 2.3).

Nutrient fat has increased from 34 in 1974/76 to 95 grams per capita per day percent in 1983/86, a 179% increase. The total of intake of oil (plant origin) increased by 212%, from 16.7 to 52.1 gram per capita per day in 1983/86, while fat from meat tripled, from 7 to 21 gram per capita per day in 1983/86. The overall result of these increases is that the proportion of fat from animal sources dropped from 50.3 to 45.2%.

**3. Evaluation of the Adequacy of Dietary Intake**

Table 2.4 shows a comparison between the RDA as suggested by FAO/WHO/UNU Expert Conclusion Report (1985), with the available energy and protein in 1974/76 and 1983/86. The supply of energy in 1974/76 was 75% of RDA. In 1983/86 the energy supply was about 25% greater than the RDA. We belief that this changes in energy intake in SA are real, due to several important factors. These are significant changes in socio-economic conditions such as increased overall income, government stability, food availability, internal and external immigration and foreign influence. Protein supply was greater than the RDA by 21% in 1974/76 and 100% in 1983/86.

## 2.1.8 Traditions and Social Activities

Despite the uniform picture of social traditions in SA, major differences in certain social traditions between and within the different regions exists. The family in SA is the core of the community. The father of the family has the privilege of the final

decision in the ruling of the household. The man is responsible for the financial planning (buying channel) and monitoring the social activities and relationships of his family. The older son is the second most important member of the family after the father and if the father passes away he obtains the responsibilities of the father. This is mainly amplified by the social restriction that regulate the woman's activates. The housewife on the other hand is busy with her household activities, it is interesting that the daily meal is entirely her responsibility (cooking channel). She dictates the type of the meal to the extent that in certain families, the members may not be aware of the type of meal until it is served. Nevertheless, she could play a crucial role in determining the expenditure of the household. However, today in most urban families the woman has attained a greater influence in the family than in the countryside.

Generally the daily life of the Saudi family starts with a light breakfast with preparations for school and/or work. Work is interrupted at mid-day for prayer and lunch and on returning to work late afternoon. The lunch is served at mid-day and is the main meal of the day and the focal point of family reunion. In general the evenings consist of buying stocks of fresh foods, followed by a light supper and socialising with friends at home.

### 2.1.9 Intra-Family Food Distribution

There is no doubt that foods available for consumption between countries, and within a country as well as within a household, are not equally distributed among the population. Certainly, intra-family food distribution (IFFD) within a country and/or group has both a physiological and socio-cultural basis. The physiological (logic) basis is influenced by factors such as gender, age, health status and physical activity. Whereas the socio-cultural basis maybe influenced by three main factors as reported by Den Hartog (1972).

1. Social and economic position of members in their household and the society.
2. Social function of food in a household and the society.
3. Prevailing concept towards food.

Studies in various countries have found considerable inequality in the distribution of food among household members in relation to their nutritional needs. Some studies show that there is an age and/or gender discrimination within a family. This can favour

men consuming more than their share and younger members and women receiving less in proportion to their requirements (Flores et al., 1964; Valenzuela et al., 1979).

Unlike the majority of other Arab Gulf countries, most Saudi families segregate the sexes during eating, as well as the children, particularly in the presence of guests. Sebai (1984), and Serenius (1981), reported that in some rural areas of SA adult men eat together from one main dish leaving the leftovers for the women and the children. In Bahrain, Musaiger (1982), found that 72% of the family members ate together, and 13% of families separate the sex. In rural areas, 5% of the families separate the head of the household from other members. This phenomena of IFFD in the region may be due to several reasons, first, the culture and the religion, second, respect and appreciation from the wife towards the husband, who does the hard physical work and acts as protector of the household, third, altruism may play another role, particularly for children, fourth, expresses the women's affection and love towards their husbands and children. There is no doubt that this may affect the quality and quantity of diet presented to women and children, where the best of it is eaten by men. Unfortunately, the present study was not able to reveal patterns of IFFD in SA, there was an important limitation of the method used, the reasons will be discussed in later stages.

### 2.1.10 Health Service

All of the Saudi citizens and foreigner workers are covered by free health services, which means they have no financial constraints on medical consultations on continued treatment. This services should have contributed to reduction of mortality and morbidity of many diseases.

The health service in the Kingdom is provided by three main sectors, the Ministry of Health, other government organisations, and the private sector. The Ministry of Health has been responsible for all health services in the Kingdom since it was established in 1951. The Ministry of Health has established branches called "Health Authorities", which are responsible for health administration in the regional hospitals, clinics, and primary health centres. A total of 18 health authorities manage all the Ministry of Health facilities throughout the Kingdom. Almost 68% of hospital beds, 59% of physicians and 64% of nursing staff, are provided by the Ministry of Health. Table 2.5 shows the development in health services during the Fourth Plan, 1984/85 to 1988/89 (MHSA, 1987). During this period the number of hospital beds per

thousand population increased from 2.66 to 3.35. The number of physicians increased, so that the population per physician declined from 726 to 264 persons. The number of nurses also increased, with the population per nurse falling from 363 person to 264.

**Table 2.2**
**Trends in foods consumption, of major food groups during 1974-1976 to 1983-1986 (All quantities are in grams/capita/day)**

| Food Items | 74/76 (1) | 77/79 (2) | 80/82 (3) | 83/84 (4) | % increase 1:4 |
|---|---|---|---|---|---|
| **GRAND TOTAL** | **963.2** | **1250.3** | **1471.5** | **1577.7** | **64** |
| **VEGETABLE ORIGIN** | **795.8** | **1017.7** | **1202.4** | **1257.1** | **58** |
| **ANIMAL ORIGIN** | **167.4** | **232.6** | **269.1** | **320.6** | **92** |
| **Total CEREAL** | **258.4** | **326.6** | **355.7** | **335.9** | **30** |
| Rice | 71.5 | 92.2 | 104.9 | 102.9 | 44 |
| Flour | 134.2 | 169.6 | 186.6 | 194.4 | 45 |
| Others | 52.7 | 64.8 | 64.2 | 38.6 | -27 |
| **Total FRUITS** | **287.4** | **365.5** | **412.7** | **426.7** | **48.5** |
| Fresh fruits | 268.1 | 295 | 342.6 | 375.9 | 40 |
| Prepared fruits | 19.3 | 70 | 70.1 | 50.8 | 163 |
| **Total VEGETABLES** | **163.4** | **178.1** | **207.2** | **275.6** | **68.7** |
| Fresh vegetables | 155.7 | 156.1 | 186.4 | 255.8 | 64 |
| Prepared vegetables | 7.7 | 22 | 20.8 | 19.8 | 157 |
| Potatoes | 6.1 | 11.7 | 19.6 | 20.1 | 230 |
| **LEGUMES** | **7.4** | **11.8** | **19.1** | **20.6** | **178** |
| **NUTS** | **5.6** | **8.9** | **11.4** | **9.6** | **71** |
| **RED MEAT** | **29.6** | **33.9** | **41.3** | **63.4** | **114.2** |
| **CHICKEN** | **20.8** | **43.8** | **66.8** | **79.2** | **280.8** |
| **FISH** | **9.2** | **11.7** | **17.8** | **21** | **128.3** |
| **EGGS** | **6.8** | **11.8** | **16.4** | **25.3** | **272.1** |
| **Total MILK & DAIRY prod.** | **93.1** | **120** | **113.9** | **112.3** | **20.6** |
| Fresh milk | 79.8 | 94.5 | 80.3 | 69.9 | -12 |
| Dairy products (butter not inc.) | 13.3 | 25.5 | 33.6 | 42.4 | 219 |
| **Total OILS & FATS** | **11** | **26.4** | **39.3** | **46.1** | **319.1** |
| Veg. oil/fats | 8.2 | 22 | 33.7 | 39 | 376 |
| Animal oil/fats | 2.8 | 4.4 | 5.6 | 7.1 | 154 |
| **SUGAR** | **48.2** | **50.3** | **102.6** | **83.4** | **73** |

Data are compiled from Saudi Arabia Food Balance Sheets (No. 2) From (1974-1976) to (1983-1986).

**Table 2.3**

**Trends in consumption of total food energy, protein and fat from animal origin, Saudi diet per capita per day during 1974-1976 to 1983-1986**

| Years | Energy (kcal) | Protein (grams) | | | | Fat (grams) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Total | From animals origin | | | Total | From animals origin | | |
| | | | Total | % | ratio/energy | | Total | % | ratio/energy |
| 1974-1976 | 1807 | 51.3 | 15.7 | 30.6 | 3.5 | 33.6 | 16.9 | 50.3 | 8.4 |
| 1977-1979 | 2399 | 70.6 | 23.6 | 33.4 | 5.6 | 58.2 | 24.8 | 42.6 | 9.3 |
| 1980-1982 | 2867 | 77.7 | 29.3 | 37.7 | 4.1 | 76.7 | 32.8 | 42.8 | 10.3 |
| 1983-1986 | 3012 | 84.2 | 37.6 | 44.7 | 5.0 | 95 | 42.9 | 45.2 | 12.8 |

Data are compiled from Saudi Arabian Food Balance Sheets (No.2) From (1974-1976) to (1983-1986).

# Trends of intake of protein and fat

between 1974 to 1986

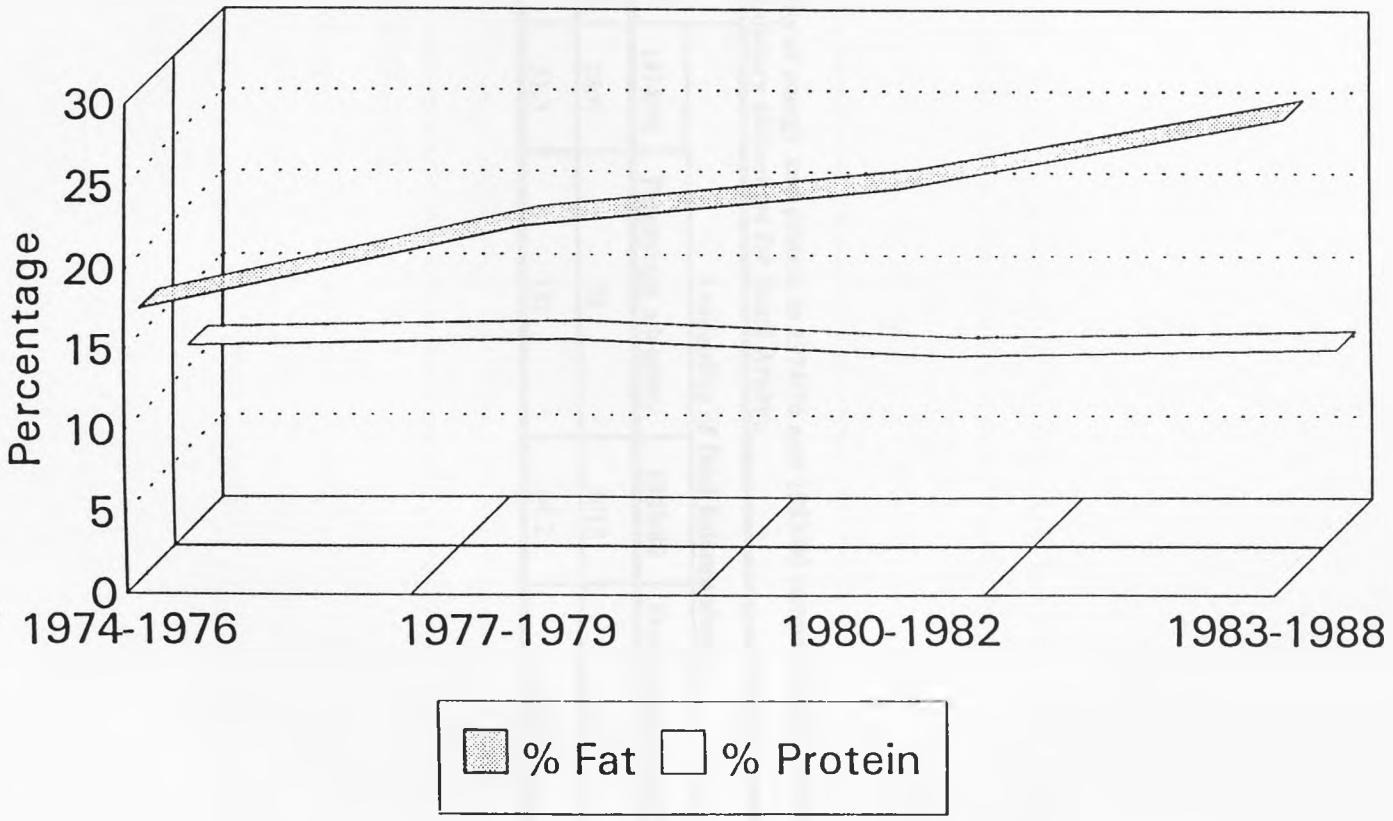



Fig. 2.2

**Table 2.4**
**Estimated availability of energy and protein in 1974/76 and 1983/86 compared with standard FAO recommended dietary allowances for Saudi Arabia.**

| | RDA | Availability of food balance sheet | | | |
|---|---|---|---|---|---|
| | | 1974/76 | Percentage adequacy | 1983/86 | Percentage adequacy |
| Energy | 2413 | 1807 | 75 | 3012 | 125 |
| Protein | 42 | 51.3 | 121 | 84.2 | 200 |

**Table 2.5**

**The development of the Kingdom's health services (hospitals, beds and primary health care) during 1984/85 and 1988/89.**

| | Year | | Increase (percent) |
|---|---|---|---|
| | 1984/85 No. | 1988/89 No. | |
| HOSPITALS: | | | |
| Ministry of Health | 86 | 162 | 88.4 |
| Other government agencies | 28 | 30 | 7.1 |
| Private sector | 31 | 55 | 77.4 |
| Total | 145 | 247 | 70.3 |
| HOSPITAL BEDS: | | | |
| Ministry of Health | 17,961 | 26,315 | 46.5 |
| Other government agencies | 5,432 | 6,577 | 21.1 |
| Private sector | 3,412 | 5,956 | 47.6 |
| Total | 26,805 | 38,848 | 44.9 |
| PRIMARY HEALTH CENTRES: | | | |
| Ministry of Health | 1,119 | 1,477 | 32.0 |
| Other government agencies | 287 | 298 | 3.8 |
| Private sector | 224* | 313* | 39.7 |
| Total | 1,630 | 2,088 | 28.1 |

* = Includes dispensaries and clinics.

Section 2.2

# CORONARY HEART DISEASE

## 2.2.1 Definition

Coronary heart disease (CHD) or ischaemic heart disease, is defined by the WHO as "*the cardiac disability, acute or chronic, arising from reduction or arrest of blood supply to the myocardium in association with disease processes in the coronary arterial system*" (Miller & Farmer, 1982). Recently, there is unclear universal definition of the ischaemia (Hearse 1994). Professor Rahimtoola (1994) suggested that ischaemia could be defined as "*a reduction of myocardial blood flow associated with perturbations of cardiac function (s) and/or take structure*". Also he added that this definition could be modified or expanded to take into consideration special situations such as "absolute", "relative", reduction of flow, anaemia etc.

CHD is a synonymous term for a group of diseases, which include myocardial infarction (MI), angina pectoris and sudden cardiac death, in addition to other manifestations which include non-fatal arrhythmias and cardiac failure.

## 2.2.2 Prevalence

CHD is the commonest cause of heart disease and the most important single cause of death in the affluent countries of the world. In 1991, a total of 28,730 English people under 65 years of age died from the consequences of CHD (TCPG, 1993). It should be kept in mind that the prevalence rate differ regarding the sex and age. The British Heart Study showed that by the age of 55-59 nearly one in three men had symptoms or signs of CHD. The annual death rate from this cause has, however, recently fallen substantially in a number of countries including USA, Australia and New Zealand, but little or none in some others such as the UK.

The cost, treatment and care of CHD is astronomical, e.g. in England and Wales, £550 million is spent annually on CHD treatment, £1,800 million annual value of lost production in 1986/87 (HEA, 1990); for USA, $88,000 million dollars for CHD in

1988. In developing countries the situation is worse since these countries cannot afford expenditure of these dimensions. They should therefore aim at reducing risk factors by promoting a healthy lifestyle. This applies to both developed and developing countries.

### 2.2.3 Coronary Arteries

The coronary network of arteries are END arteries; there is no anastomosis which helps to supply blood in case of segmental block of small branches to an area of the heart muscles. The two coronary arteries are located just above the left ventricle after the valve (Figure 2.3). Their branches are distributed in the heart muscles and terminate in the myocardium as "end arteries". The wall of the normal artery consists of three layers, namely intima, media and adventitia. Figure 2.4 shows a cross-section of a normal artery wall.

The intima which lies immediately beneath the single layer of endothelium is the area where the lesions of atherosclerosis develop. It is composed of a few smooth muscle cells, collagen fibres and glucosaminoglycans. These provide a smooth surface for the blood to flow over. The internal elastic lamina is an incomplete layer of fibres of elastin, a protein secreted by the arterial smooth muscle cells. Normally the intima is very thin at birth, and childhood, and increases gradually in thickness with age.

The middle layer media contains mostly smooth muscle cells separated by small amounts of collagen, elastin and glucosamino glycans. Fibroblasts are not found in the intima or media. This provides the artery with the ability to keep a constant pressure as the heart pumps, and helps the blood on its way within the arteries.

The third layer, adventitia is the outermost coat and separated from the media by a loose elastin barrier, the external elastic lamina. It consists of fibroblasts, collagen and glucosamino glycans and in larger arteries is supplied by small blood vessels, the vase vasorum. It acts to fix the anterior layer to the tissues surrounding and provides the middle layer with a blood supply by tiny blood vessels (capillaries) and a group of nerve fibres to control the smooth muscles (Spector, 1980).

Although it may affect any artery, atherosclerosis has very serious consequences when it affects the coronary arteries. These arteries are more susceptible to damage

due to one and/or more of the following factors:

1. They are the only arteries in the body, which are in a state of constant motion. This may lead to an increased risk of minor damage to intima.

2. During systole there is no blood flow in the inner layers of the myocardium, which may lead to ischaemic damage.

3. The coronary artery is the first vessel to come from the heart and is therefore subjected to the highest pressure.

4. Because of their small size, are particularly susceptible to luminal narrowing at a relatively early stage (Becker & Anerson, 1983).

Thus, most of the epidemiological and experimental work has been concerned with atherosclerosis in the coronary rather than other arteries.

### 2.2.4 Pathology

CHD is usually the sequence of severe atherosclerosis, which is a long term process, and thrombosis, which is a more acute event. Atherosclerosis (Greek, athere-gruel or porridge; sclerosis-hardening) has been recognised in humans for thousands of years, going back to as far as the fifteenth century BC (Lebowitz, 1970; Vanden et al., 1986; Crawfor, 1977; Ball & Mann, 1990). The term "atherosclerosis" is given to the process whereby blood vessels become narrower and thick due to the deposition of lipid material and other substances in the intima. It is generally accepted that lesions of atherosclerosis occur principally within the innermost layer of the artery wall, the intima. The three different kinds of lesion include the fatty streak, the fibrous plaque and the advanced (complicated) lesion.

*1. Fatty streak*: The earliest sign of atherosclerosis plaque is a barely visible yellowish streak on the inner surface of the artery called a "*fatty streak*", which is composed of layers of macrophage foam cells, lipid-rich lesions and smooth muscle cells (Stary et al., 1994). The mechanism leading to accumulation of the fatty streak is unknown, but it may result from the uptake of modified LDLs that enter from blood or are modified within the arterial wall. This process may begin in childhood shortly

after birth in the aorta and develops in increasing numbers between the ages of 8 and 18 years. At about the age of 15 it appears in the coronary arteries and continues to increase in amounts in these vessels. Around the age of 25 more advanced lesions begin to develop, in which there is a high incidence of atherosclerosis and its clinical sequelae (Hurst et al., 1986). This suggests that dietary factor influence the extent but not the distribution of atherosclerotic lesions in the aorta. Recent evidence suggests that fatty streak progression can be reversible if its detected at early stage. This can be achieved by modification of lifestyle; include a low-fat, vegetarian diet, moderate exercise, stress reduction, quit smoking, etc., (D'Armiento et al., 1993; Piatti et al., 1993; Kowala et al., 1993; Ornish 1993; Ornish 1991; Ornish 1990; Ruderman & Schneider 1992). The Lifestyle Heart Trial in the USA, shows that comprehensive lifestyle changes may be able to bring about regression of coronary atherosclerosis after 1 year, without the use of lipid-lowering drugs (Ornish et al., 1990). Experiment study on animals, shows that a macroscopic reduction in fatty streak production in rabbits fed dietary assimilation of fish oil (2g/day) (Piatti et al., 1993). However, this is only effective before the onset of irreversible vascular alterations, i.e., before the fibrous plaque formation has become established (Davies 1992).

2. ***The fibrous plaque***: It is the major lesion of advancing atherosclerosis, and begins to develop around the age of 25. The fibrous plaque (atheroma) is grossly white-yellow, with a shaggy, crusted appearance (due to crystallisation of the cholesterol that is accumulating in the plaque and to dystrophic calcification) it is elevated and protrudes into the arterial lumen. These small fatty plaques occur in all populations, but, in populations with a high prevalence of CHD, gradually because more fibrous (sclerotic). Plaque develops in the main from fatty streaks, although this is uncertain (i.e. it may develop in areas where no fatty streaks exist). The most essential change that occurs which the arterial intima during the development of the fibrous plaque consists of the proliferation of smooth muscle cells which have migrated into the intima from the media. This proliferation is regarded as a response to intimal injury. Smooth muscle cells migrate into the intima from the media. The smooth muscle cells and the extracelluar matrix from the fibrous cap that covers a deeper deposit of cell debris and free extracelluar lipid.

3. ***The complicated lesion*** : An advanced state of the fibrous plaque can become altered due to haemorrhage, calcification and cell necrosis. This type of

lesion may lead to occlusive disease. In this stage the thickness of plaque increases which leads to reducing the size of the arterial lumen and also decreases the elastic properties of the artery. In the complicated lesion, the necrotic "lipid-rich core" increases in size and often becomes calcified. Thus the blood flow to the heart and to the conduction system of the heart reduces. The complications of atherosclerosis (decreased blood flow, thrombosis or embolism) begin to appear. Eventually, occlusion, superimposed thrombosis or ulceration of arteries with haemorrhage may be produced in the coronary arteries by atherosclerotic plaque. The atherosclerotic plaques that develop and obstruct blood flow in large arteries are analogous to boiler scale in water pipes. Atherosclerotic plaques are composed of many substances, but at least 20% of this plaque is lipid-cholesterol, triglyceride and phospholipid, which is derived from the blood lipid in a poorly understood manner. The plaque itself consists of mushy, yellowish material collected in the vascular wall. An advanced stage of the disease will involve deposits of calcium salts, which will decrease the elastic properties of the artery, bleeding into the plaque, necrosis of its constituent tissues, ulceration of the bulging intimal surface or even rupture of the plaque itself (Sector, 1980). Under a microscope the plaque is seen to contain unusual yellowish, foamy-looking cells. This foamy material is composed of various types of fat particles which are inside special scavenger cells. Chemically, the plaques contain crystals which, upon analysis, are shown to be cholesterol. It is worth noting that the development of atherosclerosis (Figure 2.5), and the composition of the lesion, varies from one anatomical site to another, depending upon the age, genetic and physiological status, and the risk factors of the individual. Therefore, the pathology of CHD implies that several factors interact to produce the disease.

### 2.2.5 Mechanism

The exact mechanism for the development of coronary atheroma is not fully understood, although various theories exist concerning that. Though damage to the arterial endothelium is generally accepted to be a prerequisite for the development of atherosclerosis. It is suggest that coronary atheroma begins with minute injury, mechanical and chemical, to the inner lining of the artery. The initial mechanical injury damage by tow distinct processes (Davies, 1994); one is superficial and due to endothelial denudation and the second one is due to deep intimal injury. Although both processes can result in minor or major thrombi, the latter one responsible for 3 times as many occluding thrombi than the first one. The chemical injury lipid infiltration

of the intima; build-up of fibrous plaque and then thrombosis and heart attack.

Under normal circumstances the arterial endothelial cells protect the intima and media by acting as a filtration barrier. These cells maybe damaged by several sources of injury (Table 2.6) including the effects of increased plasma concentrations of LDL, shear stress, immune injury, toxins, metabolites and hormones (Jackson, 1988; Becker & Anderson, 1983). Once endothelial injury occurs, this will lead to monocyte and platelet to emigrate form the blood into the intima at sites of injury to repair it. They play a major role as contributing factors in fibrous plaque formation by interacting with damaged LDL particles. Within the tissue, monocytes activate to become macrophages (Figure 2.6). Macrophages possess a scavenger receptor specific to modified lipid, mainly oxidised LDL, from bloodstream and when become overloaded they become foam cells (Wieland et al., 1993; Weinbaum 1993; Landers 1993; Kelley 1991). Moreover, a growth factor derived from the platelet cause arterial smooth muscle cells in the media layer to multiply and move outwards into the intima. When they migrate from the media into the intima, become undergo proliferation, they secrete collagen, mucopolysaccharide and elastin in the lesion leading to further development of atheroma. Dead cells, mainly foam cells, within the lesion give rise to the lipid rich base or core of the most advanced lesions. Fibrinogen from the plasma may also infiltrate the lesion, collagen fibres are laid down and a fibre fatty plaque is formed that reduces the lumen of the vessel. If the injury to lining is chronic then the process begin to build up. The results are damage and distortion of the inner arterial wall with greater tendency towards clot formation and narrowing of the channel through which blood can flow. In addition, the artery wall becomes thicker and less elastic. Symptoms do not arise, however, until the coronary arteries are markedly narrowed or until a clot, thrombus, forms or lodges at a site damaged by atheroma.

### 2.2.6 Plasma Lipoproteins

Lipoproteins are a combination of fat and protein which are synthesised to help the lipid transport, mainly triglycerides and cholesterol, to various body tissues. They are classified in terms of their hydrated density. They consist of four major families:

1. Chylomicrons (CM).
2. Lipoproteins of very low density (VLDL) or pre-B-lipoproteins.
3. Low density lipoproteins (LDL) or (B) lipoproteins.
4. High density lipoproteins (HDL) or (alpha) lipoproteins.

CM transport dietary fat and cholesterol, referred to as the "exogenous" lipid pathway, to the tissues where they are needed. In addition they act as a vehicle for fat soluble vitamins.

VLDL particles transport endogenous triglyceride. They are less rich in triacylglycerols than CM but contain relatively more cholesterol, phospholipid and protein than CM. Most of the triacylglycerol is carried by CM and VLDL and they are both broken down to yield fuel.

LDL particles are the major carriers of cholesterol in human plasma (60-70% of total cholesterol plasma). They are produced partly by synthesis from their component parts in the liver and by breakdown of other lipoproteins, such as VLDL in the vascular system. Recently, another lipoprotein has been identified called "*lipoprotein (a)*" or Lp (a). It is similar in structure to LDL but with a higher density (Grinstead & Ellefson, 1988). Its function is unknown but plasma concentrations exceeding 300mg/ml are associated with a two to three-fold increased risk for cardiovascular disease. Ernst et al., (1994) reported that an elevated Lp (a) level is an independent risk factor for CHD in hypercholestrolemic white men.

HDL particles act as cholesterol scavengers. When cholesterol has accumulated in other lipoprotein particles or cell membranes of body tissue, the HDL particles transfer this cholesterol to the liver where it is utilised. This process is referred to as reverse cholesterol transport. Several types of HDL have been discovered, $HDL_2$ and $HDL_3$, and may have different functions. $HDL_2$ is thought to be protective but $HDL_3$ is not (Klag, 1990). HDL concentrations may be affected by several factors such as age, gender obesity, physical activity, etc.

### 2.2.7 Clinical Features of CHD

The manifestations of obstruction due to atherosclerosis, inflammation, thrombosis, embolism, or external compression. This may lead to a variety of syndromes which can affect the same patient at different times. These *syndromes* can be summarised in the following stages:

1. Asymptomatic stage.
2. Stage of stable angina.
3. Stage of unstable angina.

4. Myocardial infarction.
5. Chronic "pump" failure.
6. Sudden death.

The commonest *early sign* of CHD is the following:

1. Pain in the chest.
2. Shortness of breath.
3. Palpitation and thumping hearts (tachycardia).
4. Dizziness and faintness.

### 2.2.8 Risk Factors

A useful result of all the research into the causes of CHD has been the identification of a number of factors (Table 2.7), which why been charchterise as multifactorial disease, that are commonly found in people who develop the disorder. These characteristics are known as "*risk factors*". Nevertheless, not everyone with these risk factors has CHD, but this may indicate that they have a greater chance of doing so. Also atherosclerosis can be present without any of these risk factors, as yet the cause is unknown. Some of these factors, however, cannot be altered, such as sex, age and family history of CHD. The other factors can be modified to some degree, such as high blood pressure, blood lipids, exercise, stress, smoking and diet. The later factors can be potential targets in the search for controlling the incidence of CHD.

### 2.2.9 Major Risk Factors Associated with CHD in SA

In this section we only examine the available data of CHD risk factors with reference to SA. This provides a context within which to view the data presented into the study and explores the implication of CHD in the region studied.

*Dietary factors*: There is an overwhelming evidence provided by both metabolic and epidemiological studies which indicates that certain dietary practices are likely to play etiologic role in CHD progress. The mechanisms by which dietary intake might modulate the morbidity and mortality from CHD has been related to their effects on plasma lipids and lipoproteins metabolism and platelet endothelial cell interactions. Also, by its association with increased blood pressure and enhance obesity and diabetics. These metabolic responses to diet differed from one type of nutrient to another. However, because of the interaction between dietary and other non-dietary

factors, specific causal factors cannot be firmly identified.

The major dietary factors strongly thought to be associated with CHD development is fat, although the magnitude of their effects in uncertain (see section 2.3). The quantity and quality of fat ingested has been suggested to influence via plasma LDL cholesterol concentrations. The mechanism of CHD is also influenced by other dietary factors such as total energy (Dattil & Etherton 1992; Morris et al., 1977), protein (Connor & Connor, 1990), carbohydrate (Katan et al., 1994), fibre, minerals (Anderson & Smith 1994; Willett 1990), antioxidants (Hoffman & Garewal 1995; Knekt et al., 1994; Hertog et al., 1993; Frankel et al., 1993), etc. It is not known how much of these dietary factors in SA influences the development of the CHD and other diet-related diseases. From the little available data, however, it is evident that the Saudi diet has progressively had undesirable changes over the last few years and the situation should be a cause for concern as this study demonstrate in the following chapters.

***Diabetes***: Diabetes has been known for thousands of years and occurs in various parts of the world. From the little data available it is evident that diabetes mellitus occurs at a high prevalence rate in SA. A recent study reported by Anokute (1990), shows a prevalence rate of 6% among adult males among Saud university students in Riyadh. A study in Riyadh has also reported similar high prevalence rates (6%) of diabetes Type II among adult males, municipal and government workers, over the age of 35 years (Bacchus et al., 1982). The complication of CHD and other health disorders associated with diabetes have been reported in many studies. The risk of CHD is 2-3 times higher in diabetics than non-diabetics (WHO, 1985). Unfortunately, there is no published data explaining the prevalence rate of diabetics among the Bedouin population in SA.

***Obesity***: Obesity is considered to be a big health hazard in industrialised communities. It is a product of welfare. Obesity is not only an aesthetic problem, but is goes together with higher morbidity and mortality. A number of diseases and health disorders have been associated with it such as diabetes, hypertension, cancer and heart disease. It was found that the number of deaths from CHD in obese subjects was 40%, more than that expected on the basis of normal mortality and from diabetes by 80-130% (Stock & Rothwell 1982). Hypertension is twice as common among the obese as

among lean persons.

Obesity has become the most prevalent nutrition disorder in the Gulf countries (Al-Awadi & Amine, 1989). It is more prevalent among adults, particularly females, because, in part, females do not participate in out-door activities. A recent study carried out on 1072 (477 male and 595 female) Saudi patients at King Fahd Hospital-EP shows that 51.5% of the men and 65.4% of the women were obese when weight for height was used as an index (Binhemed et al., 1991). A similar pattern was found among Kuwaiti and Bahraini adult females (52.5% and 40% respectively), (Al-Awadi & Amine, 1989). A recent study carried out among 300 diabetic Saudi women in the EP (their ages ranged from 20 to 65 years), showed 89.7% with of obesity (Binhemd, 1992). Similar studies by Kingston and Skoog (1986) showed that most of the patients with Type II diabetes were obese. Excess dietary intake, in particular dietary fat, accompanied with lack of physical activity may influence the obesity in SA especially among women. The prevalence of overweight and obesity in men and women in the UK, is 45% of men and 36% of women are over weight (BMI >25) and 8% of men and 12% of women are obese (BMI >30) (Gregory et al., 1990).

***Smoking***: Smoking has been described by overwhelm evidence as one of the major cause of several diseases mortality including CHD (Ben-Shlomo 1994; Castelli, 1990). People who smoke cigarettes have about twice the risk of heart attack than those who do not. In the UK smoking accounts for more than one third of all deaths in middle age (SSH 1991). The reason for the risk is the mix of nicotine, carbon monoxide and tar. Nicotine increases the work of the heart by stimulating the body to produce adrenalin which raises heart rate and blood pressure. Carbon monoxide reduce the supply of oxygen to the heart muscle. The tar can increase the clotting stickiness of blood and induce spasm in the muscular walls of arteries and heart. Also cigarette smoking is associated with increased fibrinogen levels (Eliasson et al., 1995). Another effect of cigarette smoking on CHD etiology might occur via depressed antioxidant nutrient status of the smokers compared with non-smokers. It has been proposed that different in diet between smokers and nonsmokers may partially explained the positive association between cigarette smoking and CHD and cancer (Thompson et al., 1993). Thompson et al., (1993) investigated the relationship between smoking habit and diet among 910 men and women. They found that smoker had different food choices containing higher SFA and lower PUFA than nonsmokers diets. Smokers have been

shown to have substantially lower serum concentrations of both bet-carotene and vitamin C than do non-smokers even allowing for differences in dietary intake. Hence suppression of concentrations of circulation antioxidants may be one of the pathways through which smoking increases cancer risk (Pamuk et al., 1994). This study indicates that low levels of circulating antioxidants through smoking exacerbates the risk already present because of low dietary intakes.

While cigarette consumption is decreasing in industrialised countries by about 1% year, in Asia and Latin America, it is exceeding the population growth by 7% and in Africa by 18% (Champman, 1988). Smoking become one of the habits of modern life in SA which has become widespread. It was found to be significantly more frequent among Saudi CHD patients Ahmed et al., (1993). A study of the smoking habits of 2264 male students at King Saud University in Riyadh, revealed that 37% of the students smoked and over half smoked more than 15 cigarettes per day. Thirty six percent started smoking between the ages of 10 and 15 years. In another study on the smoking habits of 698 physicians in Riyadh, it was found that 38% of the male physicians and 16% of the female physicians were smokers (Saeed et al., 1989). This may be compared with the USA where only 8% of the physicians and dentists smoked (Fortmann et al., 1989; Christen, 1984). The hookah pipe (sheesha) is common in SA; users smoke jurak, a mixture of tobacco and fruits cooked to produce a dark paste. Serum levels of carboxyhaemoglobin among jurak smokers were found to be significantly higher than those in cigarette smokers and non-smokers (Zahran et al., 1985; Zahran et al., 1982). Wealth and the poor education factors, we believe, contribute to the high prevalence of smoking among Saudi people. The present low price of a packet of cigarettes (20 cigarette) 4 SR (UK £1=7 SR) was even less than half that price not long ago. This compares with the UK where one packet of 20 cigarette is, on average, £2.20 almost four-fold greater than in SA. Furthermore, many reputable magazines and newspapers carry attractive, colourful advertisements for cigarette smoking, whereas, it is forbidden in the UK and some other western countries.

### 2.2.10 Other CHD Risk Factors in SA

Other risk factors thought to play a role in CHD mortality are as follows:

*Stress*: Stress has been defined by Jackson as "*an imbalance between the demands on an individual and the way the individual sees the demands*" (Jackson, 1988). Table

2.2 shows the idea of a coronary-prone personality and behaviour pattern, referred to as 'Type A' and the opposite personality pattern 'Type B'. It was developed by Freedman and Rosenman in the 1950s (see Dimsdale, 1988, for review). Data from the prospective Western Collaborative Group Study, which followed 3154 healthy men for 8.5 years, demonstrated a strong relationship between risk of developing CHD and a Type A behaviour pattern which was independent of other risk factors (Rosenmen et al., 1975). Men with the highest levels of life stress and social isolation have a 4-fold increase of early death compared with men with the lowest level (Bray & Ward, 1986). The people in SA are presently involved in a dramatic process of transition which might put considerable stress on them. Mahgoub et al., (1991), observed that anxiety and depression was significantly more frequent among Saudi patients with ulcers.

***Insufficient exercise***: In SA, there is no data available explaining the physical activity of the population. However, it is reasonable to assume that the level of physical activity has gradually diminished in SA due to the sedentary life-style, such as the introduction of cars and office jobs. Much has been written over the years about the benefits of physical activity for the body system. Exercise of the aerobic type, brisk walking, jogging, cycling and swimming, has a whole range of beneficial effects on the heart, joints, weight, etc. (Eriksen 1994; Saris, 1988; Leon, 1988). The British Health Education Authority recommended three 20 minute bouts of vigorous exercise per week. Exercise helps to reduce the blood pressure and increase the HDL blood level (Jackson, 1988). Epidemiologic evidence indicates that physically active individuals have a lower incidence of MI and mortality from coronary disease (Rigotti, 1983). There is also suggestive evidence that exercise may be beneficial in improving metabolic control in type II diabetes (Rauramea, 1984).

***Hereditary disorders***: The traditional Saudi family, has for generations followed the custom of marrying among close relatives. Hereditary factors play an important role in the prevalence of disorders such as CHD, anaemia, diabetes etc. A positive family history of CHD amongst first degree relatives, parents, siblings or children, carries an increased risk of developing CHD. The incidence may be as much as five times as high as that of people with a negative family history (Bray & Ward, 1986). The familial incidence of CHD is largely the result of genetic predisposition to diabetes, hypertension and hypercholesterolaemia. In addition to a genetic predisposition, there are environmental risk factors that tend to be frequently similar in families such as diet,

smoking, obesity and activity (Desmond, 1988).

***Hypercholesterolaemia***: Hypercholesterolaemia (high blood cholesterol) is a plasma cholesterol exceeding 6.5mmol/l '250mg/dl' as defined by the National Institute of Health Consensus Conference on Cholesterol (National Institutes of Health Consensus Conference, 1985). At this level the risk of CHD is about twice that of a the ideal target of 5.2mmol/l '200mg/dl' (Neaton et al., 1984). This elevation of the plasma cholesterol usually is due to an increase in the level of LDL. LDL concentrations in general are influenced both by genetics and dietary factors.

A study carried out in Riyadh (Inam et al., 1991), examined the serum total cholesterol concentration (STCC) over a 3 months period among Saudi patients in intensive care (N=8291, 50.5% were male and 44.5% female) at the Armed forces Hospital. The results showed that almost 38% of the patients studied had an STCC over 5.2mmol/l, 11% had values above 6.5mmol/l and 3.6% had a value over 7.5mmol/l. It also showed that female STCC levels were higher than males. This may be due to prevalence of obesity among the Saudi females. Comparing the mean STCC for age 40-64 years of Saudi males (5.25mmol/l) population to that reported from other countries (e.g. in the UK (6.38mmol/l), Finland (7.03mmol/l), and the USA (5.88mmol/l) the values were higher than the Saudi population, by up to 1.78mmol/l.

### 2.2.11 CHD and Dietary Prevention Guidelines

Steady accumulating evidence from many countries, makes it clear that reduction in the level risk factors known to affect the level of mortality from CHD can reduce the incidence of the disease. Two types of evidence suggest that CHD is, in principle preventable. First, there are large international differences in CHD mortality. Second, time trends: much of the rapid change in CHD mortality is likely to have resulted from change in incidence rates, so factors determining onset of disease must have changed. There are two types of method to prevent CHD. These are:

1. *Primary prevention*; is an attempt to prevent or reduce the occurrence of CHD either by identifying those at high risk, or by changing the habits of the whole population.

2. *Secondary prevention*; aims not only to lower the mortality figures of who

have recovered from infarction, but also to prevent other recurrences of the disease.

The present study was only concerned with the dietary aspects, in particular fat. There is now persuasive evidence that dietary intervention is causally related to atherosclerosis and its complications. The major risk factors that are believed to be responsive, have been discussed in earlier sections, to dietary intervention include: elevated plasma cholesterol and LDL cholesterol, elevated blood pressure and obesity.

In recent years, there have been numerous reports published by many health bodies all agreed, in principle, that the priority for CHD prevention has to be reduction of serum blood cholesterol level (WHO 1992). In the UK, for example, the three reports which have proved the most influential guidelines have been prepared by NACNE, the Committee on Medical Aspects of Food Policy (COMA), and British Medical Association (BMA) on diet in relation to CVD. Recently, COMA published a new report nutritional aspects of cardiovascular disease (DH 1994). Previous COMA report was on dietary reference values (DRVs) for Food Energy and Nutrients for the UK (DH 1991). The major nutrient recommendations from these tow reports can be summarised as follows:

1. Excess calories should be avoided.
2. Total fat should not exceeded 35% energy from food.
3. Saturates decrease to 10% energy from food.
4. MUFAs 12%.
5. PUFAs 6% (maximum 10%) of which:
   Linoleic acid (n-6) 1%.
   Linolenic (0.2%).
6. Trans fatty acids should provide no more than 2% of dietary energy.
7. Complex carbohydrate, plant origin. should increase to approximately 50%.
8. Decrease simple sugars intake.
9. Decrease sodium intake.
10. Increase in the average intake of potassium by the adult population to about 3.5 g/day (90mmol/day).
11. Excess alcohol should be avoided.

**Fig. 2.3**

**The anatomy of the coronary arteries**

*Source: Nutrition: Principles and Clinical Practice, by Hunt, S. M., et al., Copyright 1980, by John Wiley & Sons, Inc., USA.*

**Fig. 2.4**

**Cross-section of normal artery**

*Source: Processes in Pathology, by Taussig, MJ., Copyright 1979, by Blackwell Scientific Publ., UK.*

**Fig. 2.5**

**Progression of atherosclerosis**

*Source: Encyclopaedia of Natrural Medicine. Nurray M., and Pizzorno J. Copyright 1994, by Littel Brown and Company (UK) Limited. P. 159.*

**Fig. 2.6**

**Roal of oxidised LDL in formation of fibrous plague**

*Source: Brown A. (1992). Oxidative-modified lipoproteins in coronary heart disease. British Nutrition Foundation Bulletin. 17: 49-64.*

**Table 2.6**
**Major injury sources factors (for atherogenesis) damaged the endothelium cells**

| Factor | Type of damage |
|---|---|
| Hypertension | Haemodynamic injury, increased arterial well tension and permeability of the endothelium to lipids. |
| Diabetes | Hyperlipidaemia, increased lipid blinding in the arterial wall. |
| Cigarettes smoking | Toxic cell wall damage from carbon monoxide and platelet aggregation and lipid deposition in the arterial wall. |
| Obesity | Diabetes, hypertension, increased cardiac work. |
| Infective/immunological | Insult |

**Table 2.7**

**CHD major risk factors.**

| | |
|---|---|
| *a. Biological risk factors* | 1. Age.<br>2. Gender.<br>3. Blood pressure.<br>4. Serum cholesterol level.<br>5. Body build<br>6. Diabetes |
| *b. Behaviour risk factors* | 1. Dietary components.<br>2. Smoking.<br>3. Exercise. |
| *C. Environmental risk factors* | 1. Pollution.<br>2. Climate<br>3. Poor living conditions. |
| *d. Medical health services* | Advanced medical and surgical management. Which may influenced health related behaviours and eventually influence risk factors of individuals. |

Section 2.3

# DIETARY LIPIDS AND CHD

## 2.3.1 The CHD-Lipid Hypothesis

Lipids, particularly saturated fats, because of their alleged role in the etiology of CHD (lipid hypothesis), have received a large amount of attention, in relation to public health. Several possible mechanisms relating to fat distribution, metabolism, and CHD risk have been discussed. This relationship seems at present somewhat controversial because an increasing number of published reports have emphasised difficulties in the measurement of diet and in the analysis and interpretation of dietary data, and have expressed warning and criticism in the search for causal relationships especially when involving free living population groups (Gordon et al., 1984). The intra-country comparisons has not shown a strong relationship between saturated fats and CHD (Shekelle et al., 1985). Notwithstanding this, there is a marked variation amongst individuals in the response to the FA. In addition, wide variation exists in response to fat quality among human and animals used to model the human response. One possible explanation for the apparent variation between CHD risk and blood cholesterol concentration may be due to age, sex and genetic factors. The CHD-lipid hypothesis, however, can be summarised as follows:

1. The type of fat is very important in CHD incidence.

2. High dietary intake of fat (i.e. among population, not individual), in particular SFA, leads to a high concentration of plasma cholesterol (especially LDL).

3. A reduced fat ratio to energy intake (i.e. among population, not individual) leads to a lower concentration of plasma cholesterol.

4. High plasma cholesterol concentration leads to high CHD mortality.

5. Reducing the dietary intake of fat, in particular SFA, leads to a lower concentration of plasma cholesterol (especially LDL).

6. High P/S ratio leads to a lower concentration of plasma cholesterol.

7. Reducing plasma cholesterol concentration (LDL) leads to lower CHD morbidity and mortality.

### 2.3.2 Effects of Different Lipids in the Diet

***1. Saturated Fatty Acids (SFA)***

SFA have been identified as the major dietary factor that raises serum cholesterol concentrations. This recognition has led numerous international committees to recommended that people should reduce their intake of dietary SFA (see section 2.2.11). It is essential to realise that not all types of SFAs seemingly raise the cholesterol concentration to the same extent. FAs with a small-and medium chain length of C4 to C10 (e.g. coconut, butter fat), appear to have no effect on serum total cholesterol concentrations (Hegsted et al., 1965; Hashim et al., 1960). Early evidence suggested that the same holds true for stearic acid (C18:0) (Aherns et al., 1957), and this has been confirmed by Bonanme & Grundy, (1988). Also, earlier studies suggested that dietary myristic acid (C14:0) raises cholesterol concentrations in human about as much as does palmitic acid (16:0) (Keys et al., 1965) or even more cholesterol raising than palmitic (Hegsted et al., 1965). Recent study by Sundram et al., (1994) disputes this and suggests that a combination of lauric acid (C12:0) and myristic acid is more cholesterolaemia than palmitic acid. Lauric acid (C12:0) found to be increase LDL concentrations about two-thirds as much as did palmitic acid (Denke & Grundy 1992). This finding dismissed Hegsted et al., (1965) claimed that lauric acid increases cholesterol concentrations much less than do other long-chain FAs. Oleic acid (C18:1) generally has been considered to be a neutral FA, nether raising nor lowering cholesterol concentrations.

***2. Polyunsaturated Fatty Acids (PUFA) and Essential Fatty Acids (EFA)***

There are two series of the PUFA; n-3- PUFA and n-6 PUFA derived from alpha-linolenic (ALA) and linoleic acids (LA), respectively. PUFAs have an influence of double bond position; n-3 PUFA found to be more potent than n-6 PUFA in reducing plasma cholesterol concentration. Because of its availability to oxidative processes, he optimal amount of PUFA intake in the diet remains uncertain. Therefore, the COMA (DH 1991) recommended intakes of PUFAs should not provide more than 10% of total energy.

Eicosapentaenoic acid (EPA) "20:5n-3" and docohexaenoic acid (DHA) "22:6n-3", two primary example of n-3 FA. There is overwhelming evidence that n-3 PUFA, present in fish and fish products, are of value in the prevention and treatment of CVD risk factors and disease (Turini et al., 1994; Kumar & Das 1994; Semplicini & Valle 1994; Axelrod et al., 1994). Other health conditions can be improved by increasing the n-3 oils including rheumatoid arthritis (Kremer, 1991), psoriasis (Soyland et al., 1993), cancer cachexia (Tisdal & Dhesi, 1990), stroke risk (Keli et al., 1994) and also menstrual cramps and eczema. N-3 PUFA supplement found to be significantly increased $HDL_2$ cholesterol concentrations in health subjects and lower triacylgycerols levels (Puhakainen et al., 1995; Frankel et al., 1994). The positive effects of dietary PUFA supplementation are generally ascribed to regulation of in vivo eicosanoid production (Kinsella et al., 1990). Also several studies have shown that dietary supplementation with n-3 PUFA tends to depress thromboxane, prostaglandins (PGE2) and prostacylin (Kinsella et al., 1990; Fischer et al., 1986; Fisher & Weber 1984). Nevertheless, Kromhout et al., (1985) observed that intake of not fatty fish but also of lean varieties was inversely related to CHD mortality.

Fish with the highest amount of EPA/DHA n-3 series are; mackerel, salmon and sardine; other good sources are anchovy, herring, tuna and butterfish. Another sources other than fish are found in oils of certain plants such as flax seed, pumpkin seed, chia seed, rape seed and soybean. Dark green vegetables such as parsley, kale and collards are good sources as well. The low death rate from CHD among the Eskimos of Greenland and the Japanese has been associated with a high consumption of fish and seal, both rich in n-3 FA (Kazawa et al., 1982; Kromann & Green 1980). Both of these populations also have a low intake of SFAs, less than half of that traditionally consumed in industrial populations (Hirai et al., 1980; Bang et al., 1980). However, since high PUFAs are susceptible to oxidation, diets rich in PUFA could have potential atherogenic effects because they promote lipid per-oxidation as well be discussed latter in section 2.3.5.

Much of the effect of the EFAs is a result of their conversion into hormone-like substances in the body known as prostaglandins (PGs). PGs thought to play a role in the regulation and function of every organ and cell in the human body. There are many different PGs; the most relevant according to current research are those of the "E family" e.g. $PGE_1$, $PGE_2$ and $PGE_3$. Prostaglandins of the type $PGE_2$, in excess, can produce pain and inflammation and encourage blood to clot. In contrast, $PGE_1$ and

$PGE_3$ both have the heart and artery protecting values. $PGE_1$ is produced by the vascular endothelium and is anti-aggregation effects. One of the proposed mechanism whereby diets rich in the long-chain n-3 FAs inhibit platelet aggregation is via decreased production of the platelet PG thromboxane $A_2$ ($TXA_2$). $TXA_2$, a product of arachidonic acid (AA) metabolism, is a potent aggregatory eicosanoid. AA increases aggregation via conversion to $TXA_2$. In contrast, linolenic, linoleic and EPA acids inhibit the conversion of AA to $TXA_2$ (Parkash et al., 1994; Axelrod et al., 1994; Blair et al., 1993). Thus the balance of the EPA:AA ratio may be a marker of $TXA_2$ production and thrombotic potential.

### *3. Monounsaturated Fatty Acids (MUFA)*

Recent studies have shown that MUFA is as effective in lowering serum cholesterol and LDL-cholesterol levels when they replace SFAs in the diet (Baggio et al., 1988; Mensink & Katan 1987; Mattson & Grundy, 1985). Moreover MUFA also has additional advantages over the PUFA of not lowering blood levels of HDL and not raising plasma triacylglycerols. Not only that, but also, they are not as susceptible to oxidation as PUFA. Oleic acid (*cis*-isomer C18:1) is the most common member of the MUFA in the diet and is found in various plant oils ranging from 65-85% of the total fat.

### *4. Trans fatty acids*

With the advent of soft margarine made form PUFA-rich vegetable oils, the dietary intake of *trans*-fatty acids has increased. Such acids do not occur in nature but are the result of vegetable oil hydrogenation, partial or otherwise. Evidence is now building up of potential toxicity of these artefact. Foods that known as major sources of *trans*-isomers are; margarine, biscuits, white bread and cake. Recent studies indicated that *trans*-isomers could be implicated in the etiology of CHD (Ascheriod et al., 1994; Willett et al., 1993). Mensinck and Katan (1990), reported that a diet high in *trans* FA (34g/person/day) administered to human subjects increased total LDL cholesterol and reduced HDL cholesterol compared with a diet high in *cis* FA. Similar findings was observed by Kummerow (1986) and Mensinck et al., (1992) not only *trans* FA raise blood cholesterol level as much as saturates but in addition decrease the level of the HDL cholesterol.

### *5. Ratio of Polyunsaturated to Saturated Fatty Acids (P/S)*

The P/S ratio is widely used in relation to the prevalence of CHD between countries

and within countries, resulting in the observation that the lower the P/S ratio the greater the incidence of CHD. In Britain the official advisory committee on Diet and Cardiovascular Disease, Department of Health and Social Security (DHSS, 1984) specified a P/S ratio of 0.45 as an attainable figure. Several studies revealed that the plasma cholesterol level increased by lowering the P/S ratio and decreased as the consequence of increasing the P/S ratio (Schaefer et al., 1981; 1981; Berry et al., 1991).

### *6. Cholesterol*

There has been controversy surrounding the influence of dietary cholesterol on plasma concentration (Grundy & Denke, 1990). Some studies have shown that adding cholesterol from eggs (about 500mg cholesterol) to regular diets containing about 236mg dietary cholesterol, caused a uniform increase in plasma cholesterol levels regardless of initial plasma lipid concentration (Roberts et al., 1981). McGill (1979) concluded that a range of dietary cholesterol intake from 0 to 600mg/d resulted in a response of from 3 to 12 mg/dl serum total cholesterol per 100mg of dietary cholesterol per 1000kcal. An additional dietary cholesterol intake above 500mg/d did not produce an additional response; however if the diet contained saturated fat the response to dietary cholesterol was greater. Other studies however, failed to produce a significant change in plasma total cholesterol, when cholesterol in the form of eggs, was added to diets already containing moderate amounts of cholesterol (Slater et al., 1976; Kummerow et al., 1977; Porter et al., 1977; Flynn et al., 1979; Flaim et al., 1981). It appears that considerable variation exists between individual responses to the effect of dietary cholesterol intake on plasma cholesterol level. Some individuals showed a marked or moderate response, and others very little or no response. Other dietary variables such as saturation of fat, complex carbohydrates and fibre could have some interactions as well (Pyorala, 1987).

## 2.3.3 Epidemiological Studies

### *1. National Data*

The relationship between saturated fat intake and risk of CHD within the general population of a particular country is either non-existent or very weak (McGill, 1979). One reason for the lack of evidence for the role of fat in CHD within-country may be due to similarity in fat intake. Difficulties are encountered in the accurate assessment of individual FA intake which may explain the lack of clear association between saturated fat intake and risk of CHD. However, comparisons made between groups

with widely differing saturated fat intakes do show a relationship between saturated fat intake and CHD. The western diet is one in which total fat intake is high (>40% dietary energy) and SFA comprises a large proportion. This has led to international dietary recommendations advising a reduction in total fat with an increase in the proportion of PUFA intake. In Mediterranean countries, however, although the fat intake is frequently more than 40%, the incidence of CHD is much lower than would be expected from this factor alone. Vegans who consume no animal fats and whose diets contain 10% of energy from saturated fat have a lower incidence of CHD than lactovegetarians and omnivores (Phillips et al., 1978). The level of linoleic acid in adipose tissue is believed to be a good indicator of PUFA intake. Scottish men were reported to have lower linoleic acid levels in adipose tissue compared to Swedish men (Logan et al., 1984), and later studies in the Scottish populations revealed lower levels of linoleic acid in patients with angina than in unaffected controls (Woods et al., 1987). However, adipose tissue linoleic acid levels in smokers have been observed to be lower than in non-smokers, and the association between linoleic acid and heart disease may be obscured by the smoking habits of the population, which in itself is a major risk factor of CHD. Intakes of PUFA of marine origin have been reported as being negatively associated with risk of CHD and, recently, substituting meat with oily fish in the diets of survivors of myocardial infarction has been evaluated (Burr et al., 1989).

### *2. International Data*

A major epidemiological investigation relating intakes of SFAs to plasma cholesterol and CHD rate came from the well known "Seven Countries Study" (Keys, 1970). This study showed that the national percentage of energy derived from saturated fat was correlated with death from CHD and with serum cholesterol level. Later, much epidemiological evidence also showed that populations consuming a low cholesterol, low fat diet have less CHD, whereas in populations of the Western world, where the diet is concentrated in animal foods rich in cholesterol and saturated fat, the incidence of CHD is very high (Sanders, 1987; Technical Report Series, No.678, 1982). CHD mortality rates are highest in North America, Northern Europe and Australia, where most of the dietary fat is derived from meat and dairy products (Thom et al., 1985). In contrast, CHD mortality rate is low in Southern Mediterranean countries, where olive oil is the main source of dietary fat. Despite the higher mortality from other types of CVD such as strokes, mortality from CHD remains low in Japan and Greenland Eskimos. A decline in CHD mortality has been observed in several

countries over the past 20 years, notably in the United States, Finland and Australia. This may be a consequence of change in the quality of fat consumed. Populations migrating from an area of low incidence of CHD to one of high incidence acquire the disease pattern of the new country, as observed in the Japanese living in Hawaii and California, who have mortality rates from CHD similar to the Americans. Migration in the opposite direction to areas where incidence is low has been accompanied by a low risk of CHD; the incidence of CHD in second generation Jewish immigrants to Israel is lower than in New York. International comparisons show an association between fat consumption and CHD which is more evident when fat is expressed as a percentage of the energy intake. CHD has been called a '*disease of affluence*' and consumption of fat is known to increase with affluence.

### 2.3.4 Atherosclerosis and Lipid in Experimental Animals

It is well-known that atherosclerosis can be produced in many species of animals (e.g. rabbits, monkeys, pigs, dogs), experimentally, by feeding large amounts of cholesterol. During the last three decades many studies have shown that cholesterol and fat produces hypercholesterolaemia and atherosclerosis in many species of monkeys (Taylor, 1963). It has also been found that when rabbits, pigs and certain primates were fed diets containing a high proportion of saturated fat over a long period they developed lesions that are similar to those in humans (Wissler & Vesselinovitch, 1988). The severity of experimental atherosclerosis is influenced by the type rather than the quantity of fat in diet. When unsaturated vegetable oils such as sunflower oil are substituted for butter fat, the resulting atherosclerosis was less severe (Mendelsohn & Mendelsohn, 1989). Generally, saturated fats such as butter, tallow lard and hardened coconut oil have been found to be atherogenic, but palm oil and cocoa butter appear to be exceptions. MUFA such as oleic acid do not appear to be atherogentic in animals. PUFA from fish oils inhibit atherogenesis in pigs, dogs and monkeys, even in the presence of hyperchlesterolaemia (Weiner et al., 1986) but not in rabbits.

### 2.3.5 Lipid Per-oxidation and Atherosclerosis

The oxidation of atherosclerosis, played by the free radical process, known as "*lipid peroxidation*" is new hypothesis recently emerged and not fully understood. It has been suggested that oxidative modification of the LDL increases its atherogenicity (Esterbauer et al., 1992; Chislom 1991; Esterbauer et al., 1990). PUFAs in LDL lipids are more substrate for oxidations, but they protected from peroxidation as long as endogenous antioxidants are present. It is thought that there are many factors which

affect the potential of LDL to become oxidised; the most important are: (1) the availability of antioxidants, (2) oxidisability of FAs in the LDL particle and (3) the amount of peroxidants (Ashwell 1993). Witzum (1994) reported that the balance of antioxidant nutrients in the LDL cholesterol complex may be critical for its protection from free radical attack. Once they are oxidised, they produce a number of breakdown products such as aldehydes and conjugated dines. The interaction of these breakdown products likely cause for changes in receptor recognition, leading to uptake by scavenger receptors. It appears that this process occurs locally in diseased arteries. But the precise way in which per-oxidation is triggered is as yet uncertain. It seems likely that once the LDL has undergone exudative modification, it is then available for uptake by macrophages via a specific receptor, termed the 'scavenger receptor' (Sparrow et al., 1989; Duthie, 1989). Oxidised LDL is cytotoxic, capable of causing local cellular disruption to the generation of foam cells and it is "chemotactic" (i.e. it attracts additional circulating monocytes and it is also interacts with platelet) causing them to aggregate (Brow et al., 1983). This process is believed to promote the development of atheromatous plaque.

### 2.3.6 The Role of Anti-oxidants

There is growing evidence that antioxidants play an important role in the prevention of CHD (Hoffman & Garewal 1995; Hodis et al., 1995; Knekt et al., 1994; Hertog et al., 1993; Frankel et al., 1993) and also other diseases such as lung and colon cancers (Mayne et al., 1994; Blot et al., 1993) and rheumatoid arthritis (Heliovaara et al., 1994). The principal dietary antioxidant nutrients are vitamins E and C, beta-carotene, selenium and flavonoid. They acts as neutralising harmful substances known as free radicals which can damage the body cells, in particularly the cells lining blood vessels, and contribute to atherosclerosis. Free radical is any atom or molecule that contains one or more unpaired electrons. It is unbalanced, highly reactive substances which is produced in the body as a result of normal metabolic process can also be triggered by exposure to pollutants, cigarette smoking and radiation. Recent studies have shown a reduction in LDL oxidation after antioxidants supplementation. For example, Belcher et al., (1993) found that oral vitamin E supplementation increases LDL alpha-tocopherol content, increases LDL resistance to oxidation and decreases the cytotoxicity of oxidised LDL to cultured vascular endothelial cells.

The mechanism of antioxidant for its protective action may be due to that it prevents free radical from inducing oxidative damage to LDL (Steinberg et al., 1989).

Also may prevent of propagation once an oxidation has started by trapping free radicals and terminating the reaction. Yet there is no global answer given about the role of individual antioxidants in human tumorigenesis. Not all antioxidants have the same protective potential and efficacy in this respect will eventually depend on chemical structure and there interaction in the body. A wide variety of antioxidants is therefore required to protect against the large number of potentially harmful metabolites produced during everyday metabolism (Jocanovic et al., 1994; Shklar et al., 1993).

Chapter 3

# *FIRST STUDY: HOUSEHOLD FOOD CONSUMPTION SURVEY*

Section 3.1

# MATERIALS AND METHODS

## 3.1.1 Data Collection

Information about food consumption is particularly difficult to obtain. These difficulties vary from one society to another. Therefore, many different methods have been developed for the collection of the food consumption of individuals and groups and each has its advantages and disadvantages. Bingham (1991, 1987) has extensively and critically reviewed the dietary assessment methods with regard to their accuracy and limitations and recommendations. The choice of method is affected by several factors such as culture, tradition, the aim of the survey, cost and the time available (Wija, 1990; Nettleton, 1980; Pekkarinen, 1970). Therefore, food survey methodology needs to be related not only to the purpose of the study but also to the characteristics of the population studied. For example, in a group who eat from the same plate (e.g. the population in this study), a weighed survey based on the individual intake is difficult, if not impossible, and likely to be highly inaccurate as a measure of usual dietary intake.

After the evaluation of the advantages and disadvantages of the different methods available; household food consumption based on food purchasing records without the larder inventory, was the method used to carry out this study. This is the most suitable available method in societies like SA. A major obstacle was that the social structure/tradition in SA (see section 3.2.3), forced us to use a method which could not take into account the available larder stocks before and after the study period. The difficulties, advantages and limitations of this method are fully discussed in section 3.2.2. One of the limitations of this method which was the aggregate data of groups of people (based on household food purchase) rather than individuals intake, is that the relationships between diet and disease risk cannot be assessed at the level of individual on the basis of high or low consumption of a specific food or nutrient (Nelson in Maregette and Nelson, 1991). In addition, its inability to determine the food distribution within individual households. This method was attractive to us because it does not required all family members to participate, this was very important where there is a high level of literacy in our study population. In addition this method is

cheap in comparison with most of the food survey methods available and easy to administer.

The sampling frames and interviewing techniques were modelled (with adjustments) on those used in the National Food Survey (NFS) for the UK, by the of Department of Health, the Ministry of Agriculture, Fisheries and Food, and the Office Population Censuses and Surveys (MAFF, 1988).

It should be kept in mind that at preliminary stages of this study, it was planned to conduct a weighed survey for food prepared for cooking and household food purchase survey. However, at the end of the survey, the weighed method was omitted from the final analyses due to its limitation and shortcomings which will be discussed in more detail in the following section.

### 3.1.2 Pilot Study

In order to arrive at the proposed method, it was decided that the method would first be tested on a group of households. No attempt here was being made to quantify consumption. The main objectives were to obtain a clear overall picture of the population to be studied, assessing the level of co-operation, testing the methodology of selecting the sample size and the study period, testing procedures for taking samples and examining the quality of the dietary information recorded. Therefore, at the beginning of the study a pilot trial was carried out in London. Ten Saudi families from the Saudi Royal Embassy in the United Kingdom participated in this study.

The following are the main important points which have been taken into account in the planning of the food consumption survey in this study.

1. The survey period of 7-day was too long from the participants point of view. Therefore, the period was reduced to 5 days maximum to serve the purpose of this study. In addition, it was also time consuming for this study since it required more attention from each individual household.

   There are no "normal" shopping days for shopping in the region studied (i.e. household tend to buy food any time they need it), and food purchase is spread evenly throughout the week. However during the study period the survey was carried out on different days of the week.

2. It had been noticed that one visit to the household was not enough to obtain the accurate information required. Therefore, they were visited several times during the survey period to assist them in the recording.

3. It was noticed that a large sample was very difficult to cover in the present study, the reasons are fully explained in the discussion section, therefore a minimum of 100 households were selected to mirror the population percentage.

4. As had been expected, as a male, it was impossible to contact the housewife. Therefore, other methods of collection were not possible in these circumstances.

5. In SA the male is responsible for the purchase of food (buying channel) whereas the housewife is responsible for the household activity (cooking channel).

6. Food consumption by weights method whether for individual or for the household was difficult to be obtained by the households participating in the study.

7. The families were found to be more co-operative using food purchases rather than weights.

In the present study, unsuccessful attempt was made to validate the data obtained by using the weighing method in a pilot study. All families in this study (10 families) were asked to give an approximate weight of each food prepared for cooking. As expected, the majority (86%) of the families did not agree to do so. Those who did agree were unable to give necessary and reliable information. They could not distinguish between gram and kilo (e.g. food cooked during the survey period they wrote 1 or 2 grams of food (e.g. for the rice or vegetable) in weight for all the household member). This was due to the low level of people's understanding, in addition to the poor literacy, it was impossible to get reliable information. Therefore, this data was not considered in the analyses.

### 3.1.3 Food Records

Each family were required to keep a complete record (Appendix A) of the following information:

1. All raw foods obtained for family consumption for five consecutive days. These included: all foods purchased, plus other foods, such as food produced on the family farm, fishing, hunting and gifts, but not food eaten outside the home.

2. In an attempt to assess the dietary habits, families were required to keep a complete record of all foods and drinks served at each meal (including snacks), for five consecutive days. Foods eaten outside the home were not included. Each item of food consumed during the day was recorded and the total consumption of each item was obtained for the period of the study. For every intake of any food item one mark was recorded. Then the frequency of food consumption was divided in the three categories of "non or rarely eaten foods", "1-3" and "4-5". This is to show the overall picture of the raw data concerning the number of time food is served and no further data was generated from it.

3. In addition, they were asked to give the approximate weight of each food prepared for cooking. A note was made of the number of cans or glasses used to measure certain foods (e.g rice). The weight of other food items such as meat and chicken was estimated by the housewife.

4. To note the members of the household and any visitors present at each meal during the period of the survey.

Food eaten outside the home was not included although this food could potentially affect the quality of the total dietary intake of individual members. The study reported here is restricted to the dietary quality of foods in the household supply. The additional food consumption outside the home requires further data collection. However, this might not make any significant contribution to their intake since eating out in SA is not a common habit.

### 3.1.4 Interviews

An extended interview took place to explain the survey to each family and the way in which they should calculate and record the amounts of food entering the household. At the same interview, a short questionnaire containing the characteristics of the households in the sample (i.e. age, sex, income, education etc.) was completed.

Non-response problems were handled by randomly selecting a replacement household of an equivalent category. It was not possible to take details of these families who refuse to take part of the study. This type of sampling with replacement may introduce more bias to the results, so the sample not truly representative.

### 3.1.5 Socio-economic Characteristics

A number of socio-economic variables which may be related to food consumption were collected (Appendix B). These included age, sex, family size, income, education and occupation of the head of the household and the housewife. The households were asked if they have any of the following: housemaid, refrigerator, freezer, farm or animals. In addition they were asked if they buy food in bulk and their house status (i.e owns, rents or other).

Family sizes were arbitrarily classified into four categories according to their members numbers. These categories were as follows:

1. Small - those having 4 or less members.
2. Middle - those having 5 up to 10 members.
3. Large - those having 11 up to 16 members.
4. Very large - those having 17 members or more.

Household incomes, based on the monthly total family income, were arbitrarily classified into four classes, since there is no statistical data available on Saudi income classification. These income classes were as follows:

1. Low - those earning less than 3000 SR.
2. Middle - those earning 3000 up to 7000 SR.
3. High - those earning 7001 up to 11,000 SR.
4. Very high income - those earning more than 11,000 SR (Note. up to date, average UK currency £1 = 5.5 SR).

Education in SA is divided into four stages; primary education consists of 6 years: 3 years of elementary and 3 years of secondary schooling, followed by higher education. The following 3 categories were used to rank the respondent's education: no formal schooling (illiterate, read only and read and write); moderate level (elementary, primary and secondary schooling); and high level (Colleges and Universities).

### 3.1.6 Study Population and Sample Technique/Size

The study took place from August 1988 to December 1988. Because of social and cultural obstacles and time limitations and also the large area to be covered, the study sampled 100 private Saudi households selected at random. To obtain households representing a diversity of food consumption in the area studied, a sample from the three communities differing in their life-style (i.e. urban, rural and Bedouin) were selected. The sampling frame from the three communities were chosen based on the following criteria: urban 66%, rural 24% and Bedouin 10%, mirroring population distribution in the province according to the Population Atlas of the Kingdom of SA (MHE, 1981). Up to date, this is the only available data that studied the population distribution in SA.

The area survey locations were selected following random sample method. The sample stages obtained were as seen below (for more detailed, see Appendix L): At the initial stage and to ensure all areas of the province are covered, EP was divided into three zones; Al-Hasa, Dammam and Hafr Al-Batin (Figure 3.1), plus the Bedouin population. To ensure a representative sample from each zone, at least one city and village were selected (Figure 3.2). For the Bedouin area two Hejra sites, were selected, one in the north (Al-Graieh Alolia) and the other in the south (Yabreen).

***Urban***: The sample size was fixed at 66 households which were selected from the cities. The number of households selected from each city were determined by the population size of the city. Each city was divided into two sectors, corresponding to areas with old and new housing; approximately half the surveyed households were from old areas, half from new. Each sector was divided into a number of blocks and households were selected randomly from these. The sections chosen were proportional to their numbers in that area. Within each section an enumeration of the house was made. Each household was numbered in the selected block, then the houses selected randomly.

***Rural***: The sample size was fixed at 24 households selected from villages. At the first stage villages were divided into two groups according to the villages population size. One household was selected from small villages and two from larger ones. Within the village four sections were made (the village was divided into four quarters), one quarter was randomly chosen. Within the chosen quarter an enumeration of the

houses was made. Then the household (s) was/were chosen randomly.

***Bedouins***: The sample size was fixed at 10 households from two areas north and south. Five households were selected from each area, using records from primary and elementary schools. This might give a biased result as the more educated Bedouin families with children who can read might not be typical. But this is the only way to carry out the survey using a questionnaire, because at least one member of the household must be literate.

The sampling technique described is intended to give a representative sample of the population in the area to be investigated.

### 3.1.7 Data Handling

Evaluation of the data obtained from the subjects was calculated using the computer programme of Salford University "MICRODIET" system, 1988, Mark 7.10. A food composition table for use in Bahrain" (Musaiger & Al-Dallal, 1985), provided the data which was fed into the programme and used to calculate energy, protein, fat, carbohydrate and fibre intakes. A limited number of other foods (e.g. salt, sugar and flour), not listed in the Bahrain food composition tables, were obtained from "Food Composition Tables for use in the Middle East" (Pellet & Shadarevion, 1970) and "McCance and Widdowson's the Composition of Foods" (Paul & Southgate, 1992). The weights of foods were entered (as grams) from study records. Hard copies were produced, detailing foods weights, together with full nutritional break downs.

It is obvious from the forgoing that the food composition data, although produced to the Arabian Gulf, do not relate directly to SA. It is however, the only available published data. Possible inaccuracies introduced as a result of the use of non SA specific data will be examined at a later stage (see Chapter 5), as will the FA profiles of foods in diets.

In order to estimate intake per individual from the 5-day household food and drink purchase records, the following procedure was used:

1. Total household's nutrient intake was calculated from the food and drink which entered the house during the survey period. This was done by using Bahrain

food tables as mentioned earlier.

2. Then the total household's food purchased was divided by 5 to give a daily average.

3. The individual nutrient availability, then was estimated by using Nelson (1986) technique, in a semi-weighed study in the UK. The reason for the adaptation of Nelson's factors are detailed in section 3.2. Thus, family members were expressed as "*fractional man-values*" (i.e. male values) depending on age and sex (Table 3.1).

4. Visitors to each household were eliminated by calculating one third of the appropriate Nelson factors for each meal consumed at the household, and subtracting these figures from the total nutrients available for consumption by the family per day.

In order to assess the adequacy of food consumption for the population in the study, as there is no information available for the Saudi population RDA, the calculated energy and protein availability were compared with the RDA, as suggested by FAO/WHO/UNU Expert Conclusion Report (1985). The procedures used were as follows:

1. In order to estimate the total daily requirement for energy, the average basal metabolic rate (BMR) factor (of 1.8 BMR) was used from the FAO/WHO/UNU report. The basis of choosing a BMR of 1.8 was due to unavailability of data concerning Saudi Arabia population on this respects. Therefore, the average BMR recommended by WHO was used, and it is understood that the BMR requirements are different for the various age and gender groups. It was beyond the limitations of this work to collect information concerning the body weight of the participants.

2. Individual RDA according to the FAO/WHO/UNU was based on different body weight. Again here, because there was no information available for the subjects body weight, energy and protein requirements from FAO/WHO/UNU report were calculated by averaging the body weight in each age group from RDA, to meet with Nelson figures used in this study. For example, Nelson

considered adults in one age group starts from age 18 and above. Whereas WHO divided adults into 3 age groups; 18-30, 30-60 and over the age of 60 years. Therefore, the corresponding data for these age groups were aggregated and the average was used. The same procedure was followed in estimating energy and protein requirements of children and adolescents.

It should be noted that the physical state of the subjects studied, such as type of body weight, work, pregnancy, lactation or illness have not been taken into account, it was beyond the limit of this study to collect such information. Also, it should be noted that although the intake by different age group and sex were presented here, their intake variation has not been discussed. So the values presented were only for the purpose of general interest.

### 3.1.8 Statistical Analysis

In any modelling exercise, it is necessary to decide upon an appropriate possibility of distribution for the responses being modelled. The most basic assumption is to assume a Normal (Gausian) distribution for the responses. However, the present data were positive and skewed. Ad-hoc data transformations were used to determine the most appropriate distribution. The log transformation seemed most acceptable suggesting that the data might be assumed to follow a gamma distribution (see p 244 of Francis 1993). It is preferable to use the gamma assumption rather than the log transformation. The latter not only stabilises the variance but also forces a (log) relationship between the mean of the data and the predicting variables whereas the former allows the mean-linear predictor relation to be defined. It was thereby found that the gamma distribution is the most plausible candidate for energy and nutrient intakes (protein, fat, carbohydrate and fibre). The analyses were then carried out in GLIM, assuming a gamma distribution for these response variables.

Explanatory variables considered in the analysis were:

1. Area type (urban, rural and Bedouin)
2. Family income
3. Family size (total number of members)
4. Farm
5. Food in bulk
6. Freezer

7. House type (owns, rents or other)
8. Head of household's education
9. Head of household's occupation
10. Housemaid
11. Housewife's occupation
12. Housewife's education
13. Livestock
14. Number of females in the house
15. Number of males in the house
16. Refrigerator
17. Visitors

The main question addressed in the statistical analysis was: what factors influence food purchasing. Linear models were used to identify possible relationships. Variables which do not indicate that any statistically significant relationships have been removed. Predicted variables in the model included family nutrients availability and individual nutrients availability.

**Family nutrients availability**: Total household nutrients availability considering all family members.

**Individual nutrients availability**: Total household nutrients availability divided by the number of people in the household. It is understandable that this type of analysis give only crude measure since treat all family members (adult, children and gender) nutrients availability as one value, but it may give general indication about the socio-economic characteristics investigated here and food consumption.

Scaled deviance change was calculated by the change in deviance and divided by an estimate of the scale. The scaled deviance is a measure of goodness fit for generalise linear models. It generalise the concept of residual sum of square for normal (Gaussian) models (i.e. residual deviance divided by residual degrees of freedom). The scaled deviance is 2 $\log_e$ L, where L is the likelihood ratio statistical for testing the current model against a fully saturated model (i.e. a model with exact fit). The scaled

deviance is asymptotically distributed as a Chi-squared variate, with degrees of freedom given by the number of observations minus the number of fitted parameters. The changes in scaled deviance is used as a mean of comparing two (noted models), using degrees of freedom given by the differences in degrees of freedom between the models.

The lack of a larder inventory leads to data with highest variability, due to a number of reports of zero purchasing despite use of the food in question. We have no reason to suppose that there would be particular bias introduced by the 5 days in question. Overall, the study sampling means that our data has large overall variability, leading to more difficulty in finding significant effects which may exist but not be identifiably.

The analysis of the relationship between food availability and socio-economic characteristics was based on the food purchased by the household as a whole, as we did not have data to analyse each individual intake. It is known that this method may hide the true associations between factors observed in this population and consumption at individual level.

Thus, by this study one cannot be certain of the true association between the socio-economic variables observed and the nutrients availability, and caution must be made, in making a strong interpretation of any relationships.

**Fig. 3.1**

**Study area; zones and population density**

**Fig 3.2**

**Survey area; location**

**Urban** **Rural** **Bedouin**

**Table 3.1**
**Factors used to calculate distribution of nutrient "*intake*" within households (according to Nelson, 1986)**

| Age group (years) | | Food group | | | | |
|---|---|---|---|---|---|---|
| | | Energy | Protein | Fat | Carbohydrate | Fibre |
| Adult male | 18 & + | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| Adult female | 18 & + | 0.70 | 0.73 | 0.74 | 0.71 | 0.80 |
| Male youth | 11 - 17 | 0.91 | 0.84 | 0.89 | 0.99 | 1.03 |
| Female youth | 11 - 17 | 0.81 | 0.77 | 0.80 | 0.94 | 0.95 |
| Male child | 5 - 10 | 0.73 | 0.69 | 0.76 | 0.84 | 1.02 |
| Female child | 5 - 10 | 0.61 | 0.56 | 0.60 | 0.70 | 0.72 |
| Male infant | 1 - 4 | 0.50 | 0.52 | 0.55 | 0.52 | 0.56 |
| Female infant | 1 - 4 | 0.48 | 0.43 | 0.45 | 0.52 | 0.58 |

Section 3.2

# RESULTS AND DISCUSSIONS

## 3.2.1 Subject Studied

This survey has studied representative parts of the EP of SA including urban, rural and Bedouin areas. A total of 960 subjects in 100 families were studied. Only persons who were still living at home were included. For the purpose of the study, infants under one year old were excluded in the analysis. This was because they typically obtain most of their nutrient needs from their nursing mothers, there were, however, relatively few sample observations for this group (3%). Of these, 1.3% infants were bottle-fed, 1.3% breast-fed and 0.6% both. This reduced the subjects studied to 930. A total of 500 males (53.8%) and 430 females (64.2%) were identified. Table 3.2 shows the composition of the population participated in the survey according to their age, sex and group.

**Table 3.2**
**Composition of population studied by gender, age groups and area**

| Age group | Type of area | | | | | | All population | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | | | |
| | M | F | M | F | M | F | M | F | T | % |
| <1 | 17 | 6 | 2 | 1 | 2 | 2 | 21 | 9 | 30 | 3.1 |
| 1-4 | 45 | 37 | 13 | 11 | 1 | 5 | 59 | 53 | 112 | 11.7 |
| 5-10 | 56 | 55 | 17 | 13 | 10 | 3 | 83 | 71 | 159 | 16.0 |
| 11-17 | 69 | 40 | 27 | 18 | 11 | 4 | 107 | 62 | 169 | 17.6 |
| >=18 | 170 | 178 | 65 | 50 | 15 | 17 | 250 | 245 | 495 | 51.6 |
| **Total** | **357** | **316** | **124** | **93** | **39** | **31** | **520** | **440** | **960** | |

## 3.2.2 Shortcomings

Since this study is based on the consumption of food purchased for the family. The results of food availability and requirements of individual given must be questioned, several major shortcomings have to be recognised. Of course, it would have been preferable to survey the food consumption of individuals. However, the subjects studied were incapable of other dietary evaluation methods. That is to say, that the

assessment of individual dietary intakes by the weighing method, 24-hour recall and/or calibrated diaries with randomisation from population is very difficult, if not impossible. Neither is the selection from certain households feasible. As has been mentioned in section 3.1, because of socio-cultural obstacles in SA, in particular when it came to terms of food consumption, forced us to use a method which could not take into account the available larder inventory nor the individual intakes, which has major biases in this study. Some of these difficulties/obstacles and the advantages/ disadvantages of the method used are outlined below, which give some justification of our reasons behind using the current method:

1. High levels of illiteracy among family members, in particular the parents. This factor reduced the choice of the method adopted for the study to those which do not rely on the participants ability to read and write. However, if subjects who cannot read and write were excluded from the survey sample, the estimated nutrient intake may not be representative of the community.

2. Traditionally, in SA food is served in a single big round tray and the family member eat from it together (Figures 3.4a, b, c, d). This makes individual intake very difficult to assess.

3. The practice of the eating habit of using the hands (Figures 3.3a, b, c, d). This makes individual intake very difficult to assess.

4. Generally, SA people are reluctant to take part in surveys.

5. Most Saudi dishes are a mixture of several food items (Figures 3.3a, b, c, d). It is impossible for an individual to give an estimate of his/her consumption of the food or its ingredients (Al-Shoshan, 1993). This behaviour is also observed in other countries with broadly comparable food habit patterns, as for example in other Arab Gulf countries and Iran (Rahmanifar et al., 1990; Kamel & Martinez, 1984b).

6. Culturally, Saudi people, in particular women, might be embarrassed and/or not likely to show the amount of food they eat. This is because of their belief that this disclosure might cause health hazards such as feeling ill and losing

their appetite.

7. Traditionally, a Saudi family is an extended one (i.e. very large), and has a great number of visitors. This places some strain and difficulties on the participant and on the investigator if the weighing method for individuals were used.

8. The cost and time, where a large number of well trained teams are required. It is also very difficult to have female researchers in the cultural context of SA.

9. Sample sizes cannot be large enough, if individual cooperation is required, for the above reasons.

10. Random samples cannot be employed, if individual cooperation is required, again for the above reasons.

11. There are no food composition analyses for native Saudi dishes.

12. There is no RNI set for the Saudi Arabian population.

In conclusion, all methods which require the respondent to estimate his/her own intake of food, recalling or recording, are partially applicable in this population, bearing in mind tradition, eating patterns and habits. Therefore, any of these methods should aim at assessing the household's food consumption, and then estimating the individual's consumption. The selected procedure employed was therefore considered to be the most feasible method.

**The great advantage of this method is that**:

1. The degree of cooperation is higher, where it does not require much responsibility.

2. The ability to provide reliable and necessary information is higher, since it is not very complicated.

3. It does not invade the privacy of respondents, which in the context of SA is a

major factor.

4. It requires less subject motivation than other methods.

5. It is less likely to disturb the normal eating habits of the participants.

6. Random samples can be employed, in this respect and the results obtained can be generalised.

7. It does not require direct communication between the housewife and the investigator. In SA, household food purchase is mostly handled by the husband, and the housewife usually, plans, prepares and cooks the meals.

**Limitations of the method**:

One of the major limitations of this method was it is in ability to measure the individual intake.

**Other points should be taken into account when interpreting this data. These are:**

1. Calculations of apportioning food purchased to the various individuals within a family was made by using population models from another country. The observations made by Nelson (1986), were based on families living in the UK, where the life style is very different to that in SA. This may well lead to a inaccurate distribution of the available nutrients. Unfortunately, direct data relating to SA or indeed any part of the Middle East is not available. Examination of published values of intake in some developed and developing countries, indicated that results of the mean sharing indexes for energy individuals are very similar to these used in the present study (for review, see Wheeler, 1991). In these studies each households intake was expressed as a percentage of the head of the household's (adult male) formula 3.1.

**Formula 3.1**

$$\textit{Sharing index (family value)} = \frac{\textit{individual's intake}}{\textit{intake of (male) head of household}}$$

Since most studies related to under-developed countries, and none considered the

distribution of fat within the family, the Nelson factors were adopted as the best available. In addition, the Nelson study was well designed and investigated using 7 days semi-weighed records of food consumption, which is generally considered most accurate (Cameron & van Staveren 1988). Whereas, other studies used 24-hour recall or weighing technique. These methods are known for its limitation in measuring the usual diet of individuals.

2. Calculation of food nutritive value was made using food composition tables from another country which was presumed to be similar to SA, as there was no such table available for SA. In chapter 5 of this study, this contributes more to our knowledge of fats and oils with SA foods.

3. Calculation of energy and protein requirements were made using the FAO/WHO expert committee figures. The figures in the WHO/FAO/UNU report may be over or an underestimate of the true average Saudi population requirements and thus, the relationships between intake and recommendations given in this study should only be seen as a rough guide to adequacy of intake.

4. Foods eaten outside the home were not included although these foods potentially could affect the quality of the total dietary intake of individual members. The study reported here is restricted to the dietary quality of the foods as purchased and available for consumption from household supplies. The additional food consumption outside the home awaits further studies. Traditionally, in SA, however, eating out is very rare and restricted to special occasions or invitation. In the present study for example, only 4 families reported that they had one meal out. This consists an insignificant percentage so that can be neglected for the purpose of this study.

5. This consumption data is based on the foods purchased, but not necessarily the amounts consumed. The actual values would, therefore, have been lower had adjustments been made for edible waste.

In the absence of a more valid alternative, the remainder of this discussion assumes that food was distributed within the family in a proportional manner for each member according to energy and nutrient requirements for age, sex, physiological condition and activity.

**Figure 3.3a**

**Tradition Saudi eating habits (all members of a family eat from a common bowl using hand)**

**Figure 3.3b**

**Tradition Saudi eating habits (all members of a family eat from a common bowl using hand)**

**Figure 3.3c**

**Tradition Saudi eating habits (all members of a family eat from a common bowl using hand)**

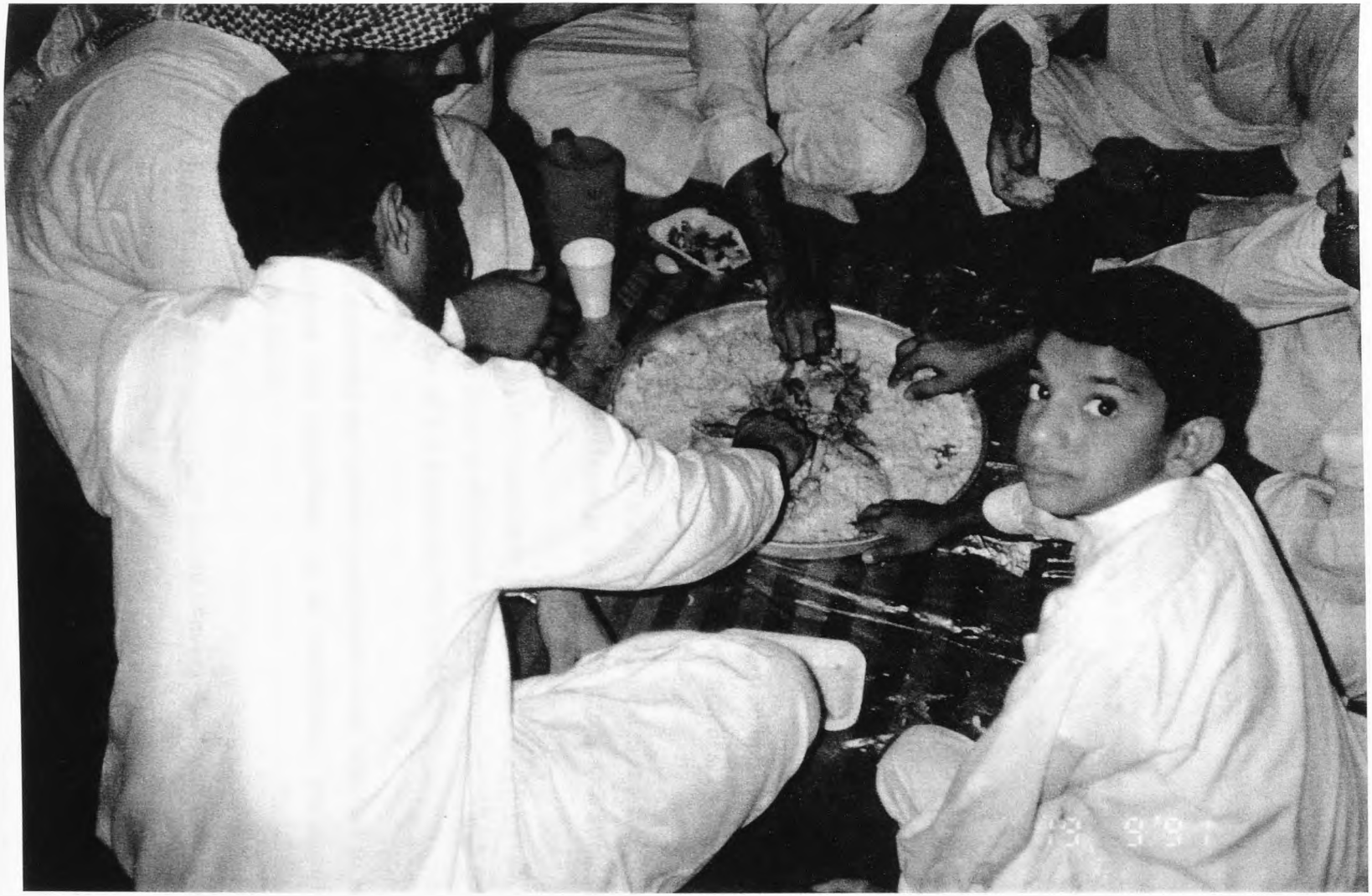


**Figure 3.3d**

**Tradition Saudi eating habits (all members of a family eat from a common bowl using hand)**

### 3.2.3 The Difficulties Encountered During the Survey

There is no doubt that field studies are associated with problems and hazards. Eventually, these problems may affect the study in terms of cost, time and reliability. As had been expected, this study has not been an easy one. Some of the major difficulties and problems encountered are discussed below:

1. As field studies are not common-place in SA, obtaining the co-operation of the people of all sectors of society created problems. Some families would not participate unless they were assured that their neighbours were also taking part.

2. Proper completion of the questionnaires, even among the educated people, was a time consuming step in the survey. This resulted in the necessity of daily visits to some of the families to explain certain points in the questionnaire, and to ensure the willingness to continue to participate in the survey.

3. The length of the survey period created the most complaints; this was also seen with the pilot survey in London among Saudi families.

4. Talking about the habits of the food consumption of the family created problems, as it is not common to discuss such things with strangers.

5. Hospitality is a genuine inheritance of the society. However this created problem in the time taken to distribute the questionnaire which resulted in the average distribution of 2-3 questionnaires weekly.

6. The culture demanded that certain participants were reluctant to answer some of the questions they considered to be confidential, particularly questions regarding the female.

7. Inability to judge the proportion weight of every food item they cooked, served and eaten by them.

9. Culture and religions beliefs were associated with restriction in the choice of the method used in this study. For example, it was impossible for the male investigator to communicate with the housewife. In this study, it was not

possible to have female interviewers.

8. Culturally, in SA the man is responsible for the financial planning (**buying channel**), whereas the female is responsible for the intra household activities (**cooking channel**). This was of a great advantage for this study.

In spite of all this, some families participated in the survey with great enthusiasm. It might be surprising that the response rate was very high (90%), perhaps because of the proposed method, which did not interfere directly with every household numbers, and not with females. This is a very important advantage of the method where another method for instant the semi-weighed method conducted in the UK by Nelson et al., (1985), the response rate was 73%.

### 3.2.4 Food Wastage

No allowance was made for food wastage, since no data was available in SA concerning this. Wenlock et al., (1980), found that food wastage among 1000 British households was significantly influenced by the size of the household. The larger the household the less wastage per person, no similar data is available for SA. In this study, however, even if it was assumed that 10 percent of food is lost during food processing and preparation, the analyses suggests that food available for consumption is still ample for meeting the dietary requirements of the population studied.

### 3.2.5 Dietary Record Period

Number of days for record-keeping are necessary for dietary investigations. This is due to day-to-day variations in the intake of individuals. Yet there is still debate among research workers on this subject. Some workers observed that a period of 7-day should be the minimum duration of a dietary survey (Thomson, 1985). Others are of the opinion that one week would be insufficient because of inter week variations (Walker, 1965; Yudkin, 1951). In contrast, some workers argue that a 3-day period is good enough (Hussain & Sarker, 1971). In conclusion, the length of time of any dietary survey is dependent on the type of nutrients to be investigated (Bingham, 1987). For example, if the energy yielding nutrients are assessed in a population, one to 7-day may be sufficient, whereas nutrients such as vitamins and minerals are needed for longer periods of up to 14-d. The previous studies are based on individual intake not on household consumption which is the subject of this study which may be different.

### 3.2.6 Meal Patterns

Data is presented in Tables 3.3-5 concerning the frequency of most food items commonly eaten at each meal during the study period. The frequency of consumption of these food items were assessed by the number of times a family included it in their meals during 5-day. In general, three meals were taken a day;

***Breakfast***: The most common breakfast amongst all groups consisted of tea mixed with milk and sugar accompanying bread with cheese, jam, peanut butter and/or eggs (Table 3.3). 31% of the sample reported had tea with bread only. This practice is more common among Bedouin than urban and rural groups. The type of bread preferred by the subjects was Arabic bread (pitta bread). Eggs were preferred boiled especially among Bedouin and rural groups, eggs were also sometimes consumed fried and/or mixed with tomatoes, oils and onions (Shakshoka). Sometimes foods such as liver, olives, fruits, vegetables, and chicken were consumed infrequently among all groups. Coffee, in particular Arabic coffee (diluted unsweetened), was also consumed. Breakfast cereals were unpopular among the Bedouin group, less so among the rural group, compared to the urban group.

***Lunch***: Lunch is the main meal in SA, is usually taken in the early afternoon. The main source of food at lunch is rice mixed with meat, chicken and/or fish accompanied by vegetables (Table 3.4). Rice is eaten either boiled, accompanying by stews (Marag; a mixed vegetables), or mixed with meat, chicken and/or fish (Kabsa), more than 65% of urban, 62% of rural and 60% of Bedouin's households consumed rice 1-3 times per week. Fruit is usually taken at the end of the meal. Laban (butter milk), is taken frequently at least 1-3 times per week by the majority of urban and rural groups, but was infrequent among Bedouins.

***Dinner***: The most common food items eaten by the majority of the subjects are presented in Table 3.5. A light meals was preferred by many families studied, about 43% of urban households, 42% of rural households and 50% of Bedouin households consumed a light average meals 1-3 times per week. A heavier meal was less frequently. Bread and tea only, were taken 1-3 times per week among 36% of Bedouin households and 21% of rural households and just 3% per week in urban households. Unlike rural and Bedouins, breakfast cereals were consumed 1-3 times per week by 4.5% of urban households. Salad was unpopular among Bedouin households.

***Snacks***: Eating snacks is an important indicator of food habits, particularly with children, because foods consumed as snacks contribute a significant proportion to the food intake (Bull, 1985). In SA eating snacks, broadly speaking, is not very important compared with the UK for instance. Very few families studied had snacks between meals, apart from when visitors were present, these snacks consisted of sandwiches and sometimes fruit, juices, tea and coffee. Al-Musharef (1990), unpublished thesis, found that only 33.5% of the mothers in EP reported to have one (27.8%) or two (5.7%) snacks during the day, while 66.5% never ate snacks. Al-Sudairy and Howard (1992), however found that eating between meals was a regular practice among the majority (72.3%) of 690 Saudi students studied aged 16-25 years.

The hot climate in SA, as other Gulf countries, encourages the people to drink of carbonated beverages and juices. Laban is the most popular drink, either consumed alone or with dates as a snack between meals and/or with main meals. Musaiger (1991), also reported that 47% of mothers in Oman drank laban daily.

**Table 3.3**
**The weekly frequency of selected food items eaten by the families at breakfast by groups (percent of total sample)**

| Food Items | Urban (n=66 households) | | | Rural (n= 24 households) | | | Bedouin (n= 10 households) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Never | 1-3 | 4-5 | None | 1-3 | 4-5 | None | 1-3 | 4-5 |
| Bread & tea only | 78.8 | 18.2 | 3 | 58.3 | 29.2 | 12.5 | 30 | 40 | 30 |
| Bread, cheese, jam & tea | 18.2 | 51.5 | 30.3 | 29.2 | 58.3 | 12.5 | 60 | 40 | 0 |
| Eggs, boiled | 39.4 | 53 | 7.6 | 37.5 | 58.3 | 4.2 | 30 | 60 | 10 |
| Eggs, fried | 42.4 | 51.5 | 6.1 | 87.5 | 12.5 | 0 | 100 | 0 | 0 |
| Breakfast cereal | 83.3 | 15.2 | 1.5 | 91.7 | 8.3 | 0 | 100 | 0 | 0 |

**Table 3.4**
**The weekly frequency of selected food items eaten by the families at lunch by groups (percent of total sample)**

| Food Items | Urban (n= 66 households) | | | Rural (n= 24 households) | | | Bedouin (n= 10 households) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Never | 1-3 | 4-5 | None | 1-3 | 4-5 | None | 1-3 | 4-5 |
| Rice, boiled | 66.7 | 31.6 | 1.5 | 41.7 | 41.7 | 16.6 | 90 | 10 | 0 |
| Rice & chicken | 22.7 | 66.7 | 10.6 | 37.5 | 62.5 | 0 | 30 | 60 | 10 |
| Rice & meat | 21.2 | 65.2 | 13.6 | 25 | 70.8 | 4.2 | 10 | 80 | 10 |
| Rice & fish | 43.9 | 56.1 | 0 | 70.8 | 29.2 | 0 | 80 | 20 | 0 |
| Rice & shrimp | 84.8 | 15.2 | 0 | 87.5 | 12.5 | 0 | 100 | 0 | 0 |
| Chicken, fried | 84.8 | 13.6 | 1.5 | 75 | 25 | 0 | 90 | 10 | 0 |
| Meat, fried | 97 | 3 | 0 | 91.7 | 8.3 | 0 | 100 | 0 | 0 |
| Fish, fried | 90.9 | 9.1 | 0 | 70.8 | 29.2 | 0 | 100 | 0 | 0 |
| Stew | 34.8 | 62.2 | 3 | 54.2 | 37.5 | 8.3 | 80 | 20 | 0 |
| Fruits (dates not included) | 34.8 | 51.5 | 13.6 | 75 | 25 | 0 | 100 | 0 | 0 |
| Laban (yoghurt) | 48.5 | 33.3 | 18.2 | 45.8 | 45.8 | 8.3 | 80 | 20 | 0 |
| Salad | 16.7 | 45.5 | 37.9 | 41.7 | 37.5 | 20.8 | 80 | 20 | 0 |
| Macaroni | 92.4 | 7.6 | 0 | 91.7 | 8.3 | 0 | 100 | 0 | 0 |
| Potatoes, fried | 86.4 | 13.6 | 0 | 85.8 | 4.2 | 0 | 100 | 0 | 0 |
| Eggs, boiled | 93.9 | 6.1 | 0 | 95.8 | 4.2 | 0 | 100 | 0 | 0 |

Table 3.5 The weekly frequency of selected food items eaten by the families at dinner by groups (percent of total sample)

| Food Items | Urban (n= 66 households) | | | Rural (n= 24 households) | | | Bedouin (n= 10 households) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Never | 1-3 | 4-5 | None | 1-3 | 4-5 | None | 1-3 | 4-5 |
| Rice, boiled | 80.3 | 19.7 | 0 | 87.5 | 12.5 | 0 | 80 | 20 | 0 |
| Rice & chicken | 78.8 | 19.7 | 1.5 | 91.7 | 8.3 | 0 | 90 | 10 | 0 |
| Rice & meat | 80.3 | 19.7 | 0 | 95.8 | 4.2 | 0 | 60 | 30 | 10 |
| Rice & fish/shrimp | 90.9 | 9.1 | 0 | 95.8 | 4.2 | 0 | 100 | 0 | 0 |
| Rice & shrimp | 95.5 | 4.5 | 0 | 100 | 0 | 0 | 100 | 0 | 0 |
| Eggs, boiled | 83.3 | 16.7 | 0 | 75 | 20.8 | 4.2 | 70 | 30 | 0 |
| Eggs, fried | 69.7 | 30.3 | 0 | 66.7 | 33.3 | 0 | 100 | 0 | 0 |
| Shackshuka (egg, vegetable) | 87.9 | 12.1 | 0 | 83.3 | 16.7 | 0 | 80 | 20 | 0 |
| Chicken & fried | 81.8 | 18.2 | 0 | 91.7 | 8.3 | 0 | 80 | 20 | 0 |
| Meat, fried | 90.9 | 9.1 | 0 | 95.8 | 4.2 | 0 | 80 | 20 | 0 |
| Fish, fried | 93.9 | 6.1 | 0 | 79.2 | 20.8 | 0 | 90 | 10 | 0 |
| Stew | 75.6 | 37.9 | 4.6 | 66.7 | 33.3 | 0 | 70 | 30 | 0 |
| Bread & tea | 97 | 3 | 0 | 79.2 | 20.8 | 0 | 70 | 30 | 0 |
| Jam, cheese & bread | 51.5 | 42.4 | 6.1 | 58.3 | 41.7 | 0 | 50 | 50 | 0 |
| Fruits (dates not inc.) | 60.6 | 37.9 | 1.5 | 75 | 25 | 0 | 80 | 20 | 0 |
| Yoghurt | 56.1 | 34.8 | 9.1 | 50 | 45.8 | 4.2 | 80 | 20 | 0 |
| Salad | 54.5 | 42.4 | 3 | 58.3 | 33.3 | 8.3 | 70 | 10 | 20 |
| Macaroni | 63.6 | 36.4 | 0 | 79.2 | 20.8 | 0 | 100 | 0 | 0 |
| Kebab | 90.9 | 9.1 | 0 | 100 | 0 | 0 | 100 | 0 | 0 |
| Breakfast cereal | 95.5 | 4.5 | 0 | 100 | 0 | 0 | 100 | 0 | 0 |
| Potatoes, fried | 69.7 | 28.8 | 1.5 | 54.2 | 45.8 | 0 | 80 | 20 | 0 |
| Foul | 84.8 | 15.2 | 0 | 83.4 | 16.6 | 0 | 70 | 30 | 0 |

### 3.2.7 Food Available For Consumption

This section examines food available for consumption among the families studied, expressed as grams per adult male value per week.

***Cereal and cereal products (Table 3.6)***: The diet of nearly all the subjects studied was basically a cereal diet, the main type of cereal being rice and bread. Rice is the staple food in SA and provides a substantial source of calories and protein in the diet. This is also found among other Asian countries such as Japan, China and India, etc. The results show that rice was taken almost every day, at least once by every family. Rice consumption was higher among Bedouin and rural groups than urban. This was due to the fact that these two groups, i.e. the Bedouin and the rural, eat rice twice a day compared with once in the urban community. A negative association between rice consumption and CHD was found among 8218 Puerto Rico men aged 45-64 year old (Garica-Palmieri et al., 1980). It has been found that the energy and carbohydrates, chiefly from rice and legumes, was lower in men who developed MI or who died of CHD. Bread and flour comes next to rice; bread is eaten mainly at breakfast and dinner. The result shows that the consumption of flour was high among the Bedouin group than the rural and the urban groups. This maybe due to the fact that the Bedouin groups made their own bread as it is more convenient for them.

**Table 3.6**
**Cereal and cereal products available for consumption in Saudi households (g/man values/week)**

| Food items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total cereal & cereal prod** | **2825** | **4559** | **5325** | **3470** |
| Rice | 1341 | 3457 | 3929 | 2082 |
| Bread | 924 | 862 | 496 | 868 |
| Flour | 404 | 145 | 900 | 393 |
| Others | 156 | 96 | 0 | 127 |

***Fruit and vegetables (Table 3.7)***: As may be expected, fruit and vegetable availability was high, being higher among the rural population than the urban and Bedouin. This may due to the facts that, the study took place during the summer, when a massive amount, and a variety of imported fruit and vegetables are available in the market at a reasonable price. It is worth noting, that changes in seasonal availability

of fruits and vegetables and some other foods might result in major changes in food purchasing patterns. It is quite clear that here, there is a need for more designed studies to be conducted during different seasons of the year.

The low availability of fruit and vegetables among the Bedouins in comparison with the urban and rural may due these people have to travel long distance to the nearest city to buy them. This is hard for them to make the journey in weekly bases.

The study shows that dates, oranges, apples, grapes and watermelon were consumed in large quantities (Table 3.7). Dates are the commonest fruit consumed, almost on a daily basis, with every meal and between meals as snacks and also represent for the guests. EP is well known for its massive production of this type of fruits which might explain its high availability. The result shows that dates availability was higher among the rural areas in comparison with the urban and the Bedouin (SD 1083.09). This could be due to bulk purchase and especially the fact that the study was carried out in the late summer which is the date seasons. Also, naturally, this is may demonstrate that dates are more popular fruits among the rural than other two groups.

A variety of vegetables are used in Saudi dishes such as potatoes, squash, aubergine, okra, tomatoes, etc. Tomatoes and onions were consumed in large quantities due to the practice of using them in almost all main dishes. Green leaves such as lettuce, radishes, basil and celery were also consumed, served raw as a side dish (salad). Several studies indicated that consumption of large quantities of vegetables lowered serum cholesterol levels (Anderson et al., 1990). Vegetables also provide substantial $\beta$-carotene, folate and unrefined carbohydrate, the last two of which are not available in substantial amounts from fruits (Kromhout et al., 1982).

***Legumes***: The results (Table 3.7) show that legumes were the lowest quantity of food to be purchased, although they are used in soups, stews and rice dishes. Perhaps this low purchase of legumes was due to large stocks, as they can last for a long time when they are dry. Other explanations, were due to the fact that legumes were not popular in the family's dishes. The most popular legumes are lentils, beans, black-eyed beans and chick-peas, usually cooked alongside meat. Legumes contain slowly digested and resistant starch and water-soluble fibre which can be of benefit to people with diabetes because of their low glycaemic index. The desirable weekly consumption of

legumes is unknown. However, vegetarians who eat legumes are regularly reported to have a lower cholesterol level, lower blood pressure and less heart disease and cancer, compared to those who do not eat them (Dwyer, 1988).

***Meat, poultry and fish (Table 3.8)***: Chicken/meat are the central items of most Saudi main dishes and consumed more frequently (at least once a day) by the majority of the families. Lamb was not as popular as chicken but more popular than beef, mutton and camel meat. In contrast with Kuwait, chicken was preferred to meat (Kamel & Martinez, 1984a), perhaps because it is cheaper than meat and preparation and cooking is more convenient. The type of red meat consumed in large proportions was lamb, whereas beef and camel meat (laham jazzer) was less common. The amount consumed depends on the affluence of the family and on the family size, although this needs to be investigated in a more detailed study. SAFBS show that red meat and chicken consumption has increased in 1983/86 by 114% and 281% respectively since 1974/76. These findings may raise serious questions about the high consumption of meat (red meat and poultry), because of its high content of SFAs. Slattery et al., (1991), observed in their study that people who consume very little meat had lower total cholesterol, LDL, cholesterol and triglyceride concentrations than this with a high meal consumption. Other studies showed serum cholesterol concentrations to be lower in vegetarians than non-vegetarians (Fonnebo, 1988). On the other hand, dietary fish oils rich in w-3 FAs were shown to primarily decrease plasma, VLDL and triglyceride concentrations (Harrise, 1989). Surprisingly, although the province studied is a coastal area and there is a fishing industry, fish was not popular among the majority of the subjects. Possible explanations for this, are that the community is ignorant of its benefit. Other reasons, maybe due to its high price compared with meat. The low consumption of fish among the Bedouin may be due to its unavailability. The findings of this study suggest that a programme to encourage fish consumption in the area should be carried out.

***Eggs (Table 3.8)***: Eggs are regarded as one of the best sources of high quality protein and balanced distribution of vitamins and minerals in relation to their low calorie content. This study shows that eggs are eaten at breakfast and in the evening as part of the dinner (Tables 3.3-5). On average egg consumption was 5 eggs/adult male/w. This is much higher than the UK 2.25 eggs per person (MAFF, 1991). Egg consumption among Bedouins was lower than that of the urban and rural population.

The unpopularity of egg among Bedouins may be due to the fact that they are unavailable to them in their local market and it is difficult to raise chickens in the desert.

***Milk and milk products (Table 3.9)***: Fresh milk was not frequently taken and this is also has been shown by the government food balance sheets. Kamel and Martinez (1984), also, found that milk consumption was low among the Kuwaiti population. Laban was more frequently consumed than fresh milk. The latter was mostly drunk in the Bedouin area where Laban is difficult to get. The most popular milk consumed by Bedouins was camel and goat's milk presumably reflecting the varieties of milk animals kept.

***Oils and fats (Table 3.9)***: The main type of fat used was vegetable oil; ghee and butter were less common. Although, traditionally people preferred animal fats for cooking, particularly with rice. Also, not so long ago, people in Bedouin and rural communities used to drink animal fats, especially in the morning. There is a general belief that drinking animal fats give them strength. However, this old habit is no longer practised. Again here, the low purchase of butter and ghee may be caused by large stocks. Another reason for this may be due to the lower prices of vegetable oils.

***Sugar (Table 3.9)***: The consumption of sugar was high, being higher among the Bedouin, which is a serious concern. There is no clear explanation for this, other than the people's habit which tend to have 'extra-sweet' test. In addition, this is may contributed by bulk purchasing.

**Table 3.7**
**Fruits and vegetables available for consumption in Saudi households (g/man value/week)**

| Food items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total fruits** | **3815** | **5915** | **3675** | **4287** |
| Dates | 338 | 2370 | 704 | 843 |
| Oranges | 491 | 939 | 712 | 730 |
| Apples | 646 | 911 | 630 | 705 |
| Bananas | 354 | 393 | 475 | 375 |
| Watermelon | 599 | 442 | 377 | 541 |
| Mandarin | 09 | 124 | 82 | 311 |
| Pomegranate | 151 | 207 | 164 | 165 |
| Lemon | 144 | 105 | 98 | 142 |
| Grapes | 355 | 242 | 434 | 337 |
| Juice (litre) | 50 | 104 | 0 | 57 |
| Other fruits | 109 | 78 | 0 | 80 |
| **Total vegetables** | **3233** | **3505** | **2672** | **3241** |
| Potatoes | 587 | 618 | 344 | 571 |
| Tomatoes | 806 | 727 | 606 | 768 |
| Greens, leaves | 285 | 115 | 224 | 240 |
| Squash | 295 | 173 | 115 | 249 |
| Onions | 388 | 922 | 606 | 533 |
| Okra | 100 | 100 | 327 | 123 |
| Cucumber | 430 | 407 | 0 | 383 |
| Others | 342 | 443 | 450 | 374 |
| **Legumes** | **47** | **31** | **74** | **46** |

**Table 3.8**
**Meat, poultry, fish and eggs available for consumption in Saudi households (g/man value/week)**

| Food Items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total meat & poultry** | **1429** | **1807** | **2488** | **1620** |
| Chicken | 862 | 875 | 1719 | 948 |
| Meat | 567 | 932 | 769 | 672 |
| **Fish** | **379** | **597** | **131** | **405** |
| **Eggs (n)** | **5.06** | **5.88** | **3.82** | **5.14** |

**Table 3.9**
**Milk, diary products, oils/fats, sugar and miscellaneous foods available for consumption in Saudi households (g/man value/week)**

| Food Item | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total milk & diary prod.** | **1051** | **1552** | **1319** | **1193** |
| Milk, fresh | 452 | 532 | 999 | 524 |
| Laban | 454 | 867 | 90 | 514 |
| Cheese | 81 | 95 | 25 | 79 |
| Others | 64 | 58 | 206 | 76 |
| **Total oils & fats** | **255** | **242** | **434** | **269** |
| Vegetable oils | 204 | 214 | 434 | 229 |
| Butter | 50 | 28 | 0 | 40 |
| **Sugar** | **1039** | **1450** | **2374** | **1264** |
| **Miscellaneous** | **47** | **15** | **70** | **42** |

### 3.2.8 Energy and Nutrient Availability

Table 3.10 shows a summary of the contribution of major food groups to total energy and other nutrient availability for urban, rural and Bedouin and for the sample as a whole (for more details see Appendices C-G). The nutritional value of the food brought into homes during the survey period is summarised in this section and compared with the FAO RNIs.

The percentage of RNI values for all nutrients investigated were found to be high among all groups studied. It is highly unlikely for this population, and indeed any population, to consume this amount of food, for example the observed carbohydrate availability was up to 1098g/adult man value/day. It is indicate that these results over estimate the actual intake. There were several likely potential biases inherent in the method used which attributed to invalidity of our data.

**The potential biases inherent in the dietary survey method we used:**

*1. Larder inventory*

This was a major bias in this study where we were unable to find out how much food was in stock before and after the survey period. It assumed that the collected data of purchased and free food are averaged over a large number of households may represent the intake on the individual level, provided that there is no systematic change in their general level of food stocks. In another word, the basic idea of household food survey based on food purchase is that the aggregation of the intake of group of people will balance out the individual intake, but we have no evidence it would balance out without more investigations.

*2. Food in bulk*

This was the second important bias, when people of buy their food in bulk for convenience. We recognised that it is unlikely that all foods purchased during the survey period were consumed at the same time. This is inevitable in a food purchasing survey of this type, spread over a relatively short period of time, and is caused by families stockpiling foods for later consumption. We assumed that the sum of the food purchased, plus the food produced and food gifts, may be equalled to the food consumed by the average family. This is clearly also dependent upon the assumption that the larder inventory at the end of the survey period does not differ greatly from the inventory at the start of the survey. There is no reason to believe that the

organisation of this study was such that it would lead to consistent sampling of period of over or under purchase stockpiling.

*3. Under or over-purchase*

The raw data (Appendix I) on which this study is based, shows great variability in the food purchased by individual families. In many cases, the food purchased was either far in excess of the amount of food which would have been expected to be consumed by that family, or in other cases far below. These are influenced by the factors outlined below:

a. The culture and tradition: it is important that large food stocks for unexpected guests at all the time.
b. In honour of the invited guests, other distinguished members of the community are always invited.
c. The habits of throwing a way extensive amount of cooked food leftovers.
d. The possibility of miscalculated food purchase by the subjects can not be overruled.
e. The possibility of exaggeration of food purchased. It is may that families would over-purchase to impress the investigator. Or perhaps would they under-purchase to get money or food supplements as has been observed in some developing countries.
f. Under-reporting is likely to occur since culturally it is considered improper to talk about the food one eats.
g. Under-reporting and/or over-reporting are more likely to occur; this could have arisen either from failure to report all food purchase, or because the subject could not remember the weight of the food they purchased.
h. Finally, under-purchase could be caused by large larder stock available during the study period, whereas over-purchase caused by food bought for future consumption.

*4. Use of Food Tables*

It is known that food composition tables used in one country are not applicable to other countries, as they may under- or over-estimate the actual food intake. As we have no alternative, the study was used food tables used on other country. Our fat and fatty analyses (see Chapter 5) shows that there was marked differences with calculated value reported from food table used.

*5. Sample errors*

In addition to the above problems, other issues which may cause some problem with our method defects, in particular the sample size and the study period. The sample size and the study period may not large/long enough to be absolutely confident of the estimate of dietary availability to the population sampled.

As a consequence of the above points, this study has not quoted, and is not in a position to discuss the nutritional status of the population studied. Also it should be kept in mind that the results are average energy and nutrients availability in the household and not the actual individual intake. Therefore the following discussions must be interpreted with caution for these reasons.

In spite of the biases of this method for assessment of an individual's food availability, a survey, such as the current one, yield relevant information on the supply of household food and nutrients, thereby providing (1) direct indicators of food sufficiencies at the household level and, (2) indirect evidence of potential nutritional deficiencies at the individual level. In SA where no studies are available in this respect, it provide (3) descriptive data on the food purchase customs of the population studied. Furthermore and for the most of all, this study may (4) highlights the need for further studies, and (5) gives encouragement to other researchers to carry out further investigations on this subject, building on the experiences outlined in this investigation.

**The nutrients investigated in this study:**

*1. Energy*

Table 3.11 shows the average energy availability recorded by urban, rural, and Bedouin and for the sample as a whole. The energy availability of all these groups is higher than the FAO RNI by more than 100%. Bedouin energy availability recorded (225% of RNI) was the highest, and next to it rural (203% of RNI) then the urban group (125% of RNI). Though there is no RNI for Saudi population it unlikely that energy requirements would be significantly different from the WHO/FAO values. If these energy values were truly representative of intakes then the population would be likely to be obese. The results indicate that some error has occurred either in collecting food availability data or estimating energy values.

The differences between the three communities studied may have arisen from the

differences in food purchasing habits. In addition, the differences in the habit of stock large amount of food for convenience and also for unexpected guests between these communities. An inverse relationship exists between energy intake and CHD, a diet containing more energy than is needed may lead to obesity and also reflects the physical activity and both have been related to the development of CHD.

The statistical analysis shows that the family's energy availability depends upon income and head of household's education level (Table 3.12). Table 3.13 shows the ***family's*** predicted energy availability: heads of households with a lower education level and low income have a higher energy availability than high earners in the same group. It also shows that a family with an educated head of household has a lower energy availability. Table 3.14 shows the result of modelling energy per ***individual*** (gamma errors, log link). The discrepant point 93 is weighted out, this is data point which has an unrealistic value and strongly influenced the results and therefore not included in the analyses. Energy per individual is dependent on the difference in area and income level. As shown in Table 3.15, the predicted individual energy availability increases with decreasing income in each area. Average energy availability per individual have using, (gamma errors, inverse link with point 93 weighted out), could best be modelled using area and head of household's education level (Table 3.16). Table 3.17 shows that predicted individual energy availability varies from area to area and head of household's education levels. In general, head of household's with secondary level education have the highest energy availability, whereas, the highest education level have the lowest.

The largest contribution to energy availability was from cereal and cereal products, which provides more than one third of the overall average energy. Within the cereal products category, rice provides 63%, bread provides 21% and 12% is contributed by flour. Sugar contributed quite substantial amounts of energy to the subjects diet; up to 21% among the Bedouin group. This figure exceeded their total meat contribution by almost 10%. Fruits 14%, meat and poultry 11% and oils and fats 8% were the next largest contribution to energy availability (Table 3.10). Then vegetables by 5% and milk and diary products by 4%. There was some variation between urban, rural and Bedouin in the sources of energy. Rural derived their energy from fruits (21%), whereas urban derived 11.5% and Bedouin derived 9%. On the other hand, rural derived energy from oils and fats (5.4%), where urban and Bedouin derived 9% each.

## Table 3.10

**Summary of the percent contributions made by main food groups to energy and nutrient content of the Saudi's diet, and the mean daily nutrient intake per man-value**

| Main Food Groups | Urban | | | | | Rural | | | | | Bedouin | | | | | All population | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Energy | Pr. | Fat | CHO | Fibre | Energy | Pr. | Fat | CHO | Fibre | Energy | Pr. | Fat | CHO | Fibre | Energy | Pr. | Fat | CHO | Fibre |
| Cereal & cereal prod. | 37.4 | 33.3 | 5.2 | 48.5 | 24.7 | 38.3 | 34.7 | 6.3 | 46.2 | 21.3 | 41.4 | 39.0 | 5.9 | 50.6 | 34.8 | 38.3 | 34.5 | 5.6 | 48.1 | 24.6 |
| Fruits | 11.4 | 4.1 | 2.1 | 15.8 | 40.8 | 21.3 | 6.6 | 3.2 | 26.9 | 58.4 | 9.2 | 3.3 | 1.6 | 12.0 | 38.7 | 14.1 | 4.7 | 2.3 | 18.8 | 46.5 |
| Vegetables | 5.5 | 5.3 | 2.6 | 6.0 | 31.4 | 3.9 | 4.3 | 1.8 | 3.9 | 19.8 | 2.9 | 3.4 | 1.2 | 2.8 | 25.0 | 4.6 | 4.8 | 2.2 | 4.8 | 26.8 |
| Meat & poultry | 12.0 | 35.2 | 33.5 | | | 9.9 | 30.9 | 38.0 | | | 10.8 | 40.7 | 31.6 | | | 11.2 | 34.7 | 34.4 | | |
| Fish | 1.2 | 9.1 | 0.3 | | | 1.3 | 11.2 | 0.4 | | | 0.3 | 2.3 | 0.1 | | | 1.1 | 8.8 | 0.3 | | |
| Eggs | 1.6 | 4.4 | 4.9 | | | 1.2 | 3.6 | 4.6 | | | 0.7 | 2.1 | 2.3 | | | 1.4 | 3.9 | 4.5 | | |
| Milk & diary prod. | 4.1 | 7.6 | 9.6 | 1.6 | | 4.4 | 8.6 | 13.5 | 1.7 | | 4.1 | 8.1 | 10.3 | 1.8 | | 4.2 | 7.9 | 10.7 | 1.6 | |
| Oils and Fats | 9.0 | | 41.1 | | | 5.4 | | 32.2 | | | 8.9 | | 46.2 | | | 7.9 | | 39.5 | | |
| Sugar | 16.7 | | | 27.1 | | 14.4 | | | 21.1 | | 21.3 | | | 32.3 | | 16.6 | | | 25.9 | |
| Miscellaneous | 1.0 | 1.1 | 0.8 | 1.1 | 3.2 | 0.09 | 0.2 | | 0.2 | 0.6 | 0.5 | 1.1 | 0.9 | 0.5 | 1.5 | 0.6 | 0.8 | 0.6 | 0.7 | 2.1 |
| Mean daily nutrient intake per man-value | 3509 kcal | 106 gram | 84 gram | 575 gram | 8.8 gram | 5694 kcal | 151 gram | 104 gram | 1033 gram | 15.7 gram | 6287 kcal | 167 gram | 133 gram | 1098 gram | 11.6 gram | 4285 kcal | 123 gram | 94 gram | 731 gram | 10.7 gram |

Pr. = Protein
CHO = Carbohydrate

**Table 3.11**
**Calculated average energy availability (kcal per day) by gender, age and area (based on adult males value)**

| | Male age groups | | | | Female age groups | | | |
|---|---|---|---|---|---|---|---|---|
| | > =18 | 11-17 | 5-10 | 1-4 | > =18 | 11-17 | 5-10 | 1-4 |
| All population | 4285 | 3899 | 3128 | 2142 | 2999 | 3471 | 2614 | 2057 |
| % RNI | 153 | 154 | 159 | 158 | 129 | 166 | 147 | 152 |
| Urban | 3509 | 3193 | 2562 | 1755 | 2456 | 2843 | 2141 | 1684 |
| % RNI | 125 | 127 | 130 | 130 | 106 | 136 | 121 | 125 |
| Rural | 5694 | 5181 | 4156 | 2847 | 3986 | 4612 | 3473 | 2733 |
| % RNI | 203 | 205 | 210 | 211 | 172 | 221 | 196 | 202 |
| Bedouin | 6287 | 5721 | 4590 | 3143 | 4401 | 5092 | 3835 | 3018 |
| % RNI | 225 | 227 | 232 | 233 | 189 | 244 | 216 | 224 |
| FAO RNI | 2800 | 2525 | 1975 | 1350 | 2324 | 2088 | 1775 | 1350 |

**Table 3.12**
**Average energy availability (kcal per day) per family. Increase in scaled deviance by removing each factor from the final model IN+EG.**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| IN | Income | 13.85 | 4 | < 0.01 |
| EG | Head of household's education level | 18.51 | 7 | < 0.01 |

**Table 3.13**
**The predicted energy (kcal per day) available per family (gamma errors, log link) displayed by income and head of household education level.**

| Head-of-household Education Level | Energy (kcal) | | Mean |
|---|---|---|---|
| | For very high income household | For others income household | |
| Primary & less | 19674 | 48431 | **43013** |
| Secondary | 17925 | 32373 | **26955** |
| College | 15793 | 29307 | **24802** |
| Missing | 0 | 28804 | **28804** |
| **Mean** | **18265** | **43401** | |

Others = Low, Medium and High income.
0 = zeros correspond to zero observed cases.

**Table 3.14**
**Average energy availability (kcal per day) per individual. Increase in scaled deviance by removing each factor from the final model AR+IN**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 5.4 | 2 | < 0.07 |
| IN | Income | 9.3 | 4 | < 0.05 |

**Table 3.15**
**The predicted energy availability (kcal per day) per individual (gamma errors, log link) displayed by income and area type.**

| Income category | Area type | | | Mean |
|---|---|---|---|---|
| | Urban | Rural | Bedouin | |
| Low | 4795 | 7285 | 10118 | **6097** |
| Medium | 4038 | 6136 | 8521 | **6033** |
| High | 4172 | 6340 | 8804 | **5409** |
| Very high | 2021 | 3071 | 4265 | **2308** |
| No information given | 2021 | 7418 | 10303 | **5765** |
| **Mean** | **3895** | **6514** | **8243** | |

**Table 3.16**
**Average energy availability (kcal per day) per individual. Increase in scaled deviance by removing each factor from the final model AR+EG**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 5.6 | 2 | < 0.06 |
| EG | Head of household's education level | 13.6 | 6 | < 0.06 |

**Table 3.17**
**The predicted energy availability (kcal per day) per individual (gamma errors, inverse link) displayed by head of household's education level and area type.**

| Guardian education level | Area type | | | Mean |
|---|---|---|---|---|
| | Urban | Rural | Bedouin | |
| a. No formal schooling: | | | | |
| 1- Illiterate | 3588 | 5001 | 3384 | **4045** |
| 2- Read only | 3377 | 4790 | 0 | **3554** |
| 3- Read & write | 5412 | 6825 | 5209 | **5908** |
| b. Moderate level: | | | | |
| 1- Elementary | 4704 | 0 | 4501 | **4684** |
| 2- Primary | 3394 | 4807 | 3191 | **3939** |
| 3- Secondary | 12544 | 13957 | 12341 | **12644** |
| c. High level: | | | | |
| 1- College & University | 3171 | 4584 | 2968 | **3431** |
| **Mean** | **4419** | **5568** | **7019** | |

0 = zeros correspond to zero observed cases.

***2. Protein***

Table 3.18 shows the average of protein and percentage of RNI for different communities by gender and age. On average, the daily protein availability recorded was 123g for an adult male, which provides 11.5% of total energy. The protein of the three groups is seen to be far greater than the RNI, with the Bedouin group at the top of the list. Their protein was almost 3.5 times (341%) RNI, while the corresponding figure for the rural group was just over 3 times (308%) RNI. Protein for the urban group is less than the other two but still at twice (216%) the FAO RNI. Protein excess may have a negative effect on health. In animal studies, it has been suggested that animal protein such as casein (from milk) is hypercholesterolaemic, while vegetable protein such as soy protein, has the opposite effect. In humans, with the difficulties in controlling all the variables, including fat and cholesterol content, it may be that the consumption of vegetable proteins has some hypercholesterolaemic action (Connor & Connor, 1990). Protein excess may also contribute to obesity in population.

Total family protein availability show no relationship across all families. But average protein availability per individual (gamma errors, log link with point 93 weighted out) seems to be dependent on area type and food in bulk (Table 3.19). In all areas, the predicted protein for individuals increases among families who bought their food in bulk (Table 3.20).

For the total population, the main sources of protein (Table 3.10) were meat and poultry (35%), with more than half of this (66%) derived from chicken. Next to it was cereal and cereal products (34.5%), of which 53.5% was derived from rice. Then comes fish (9%) and milk and dairy products (8%). There was little variation between different groups in the main sources of protein. For example, Bedouin obtain 2.3% protein from fish, whereas rural and urban obtain 11.2% and 9%, respectively. Bedouin derived 41% of protein from meat and poultry, whereas urban and rural derived 35% and 31%, respectively.

**Table 3.18**
**Calculated average protein availability (gram per day) by gender, age and area (based on adult males value)**

| | Male age groups | | | | Female age groups | | | |
|---|---|---|---|---|---|---|---|---|
| | **>=18** | **11-17** | **5-10** | **1-4** | **>=18** | **11-17** | **5-10** | **1-4** |
| **All population** | **123** | **103** | **85** | **64** | **89** | **94** | **69** | **53** |
| **% RNI** | **251** | **223** | **354** | **413** | **207** | **224** | **288** | **342** |
| Urban | 106 | 89 | 73 | 55 | 78 | 82 | 60 | 46 |
| % RNI | 216 | 192 | 304 | 355 | 181 | 195 | 250 | 297 |
| Rural | 151 | 127 | 104 | 78 | 110 | 116 | 85 | 65 |
| % RNI | 308 | 274 | 433 | 503 | 256 | 276 | 354 | 419 |
| Bedouin | 167 | 140 | 115 | 87 | 122 | 128 | 93 | 72 |
| % RNI | 341 | 302 | 479 | 561 | 284 | 305 | 488 | 465 |
| FAO RNI | 49 | 46.3 | 24 | 15.5 | 43 | 42 | 24 | 15.5 |

**Table 3.19**
**Average protein availability (gram per day) per individual. Increase in scaled deviance by removing each factor from the final model AR+FB.**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 5.46 | 2 | < 0.06 |
| FB | Food in bulk | 3.76 | 1 | < 0.05 |

**Table 3.20**
**The predicted protein availability (gram per day) per individual (gamma errors, log link) displayed by area type and food in bulk.**

| Food in bulk | Area type | | | Mean |
|---|---|---|---|---|
| | Urban | Rural | Bedouin | |
| Yes | 192.2 | 261 | 368 | **233.2** |
| No | 110.5 | 150 | 211.5 | **129.6** |
| **Mean** | **118** | **168.5** | **227** | |

### *3. Carbohydrate*

The average of total available carbohydrate recorded by the subjects was 698g/adult male/d. This represents 65% of the total energy. The Bedouin group have the highest carbohydrate availability followed by the rural and urban groups, respectively (Table 3.21). These results are in excess of the likely level of intake and would appear to be the major contribution factor in the over-estimation of energy intake (discussed in 3.2.8). Rice is a major provider of carbohydrate; it is also purchased in bulk and therefore difficult to quantify, and over-estimation of rice intake may confound estimates of the energy intake.

Statistical analysis shows that carbohydrate availability seems to be dependent on the difference in income (Table 3.22). It shows that families with high income have lower carbohydrates availability (Table 3.23). The analysis per individual (using gamma errors, log link with point 93 weighted out), depends on area, number of males in the household and income (Table 3.24). It was found that as the number of males in the household increases the amount of carbohydrate available for consumption per head decreased (Table 3.25).

Almost half the carbohydrate (48%) was derived from cereal and cereal products, with rice contributing 65% and bread 21% of the total cereal and cereal products. The second major source was sugar providing 26% of total carbohydrates. Fruits provided 19%, and more than half of this was from dates (55%). As shown in Table 3.10, there was some variation in sources of carbohydrate recorded by different groups. Fruits provided 27% of carbohydrate for rural group, 16% for urban and 12% for Bedouin. Sugar provided 32%, 27% and 21% of carbohydrate for Bedouin, urban and rural, respectively.

**Table 3.21**
**Calculated average carbohydrate availability (gram per day) by gender, age and area (based on adult males value)**

| | Male age groups | | | | Female age groups | | | |
|---|---|---|---|---|---|---|---|---|
| | > =18 | 11-17 | 5-10 | 1-4 | > =18 | 11-17 | 5-10 | 1-4 |
| **All population** | **731** | **724** | **614** | **380** | **519** | **687** | **512** | **380** |
| Urban | 575 | 569 | 483 | 299 | 408 | 540 | 402 | 299 |
| Rural | 1033 | 1023 | 868 | 537 | 734 | 971 | 723 | 537 |
| Bedouin | 1098 | 1087 | 922 | 571 | 780 | 1032 | 769 | 571 |

**Total 3.22**
**Average carbohydrate availability (gram per day) per family. Increase in scaled deviance by removing each factor from the final model IN.**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| IN | Income | 11.07 | 4 | < 0.025 |

**Table 3.23**
**The predicted carbohydrate availability (gram per day) per family (gamma errors, log link) displayed by income**

| | Family's income level | | | | |
|---|---|---|---|---|---|
| | Low | Medium | High | V. high | N.I.G. |
| Mean | 7447 | 7959 | 7440 | 2607 | 6851 |

N.I.G. = No information given.

**Table 3.24**
**Average carbohydrate availability (gram per day) per individual. Increase in scaled deviance by removing each factor from the final model AR+M+IN**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 6.89 | 2 | < 0.03 |
| M | Number of males | 4.10 | 1 | < 0.04 |
| IN | Income | 10.79 | 1 | < 0.001 |

**Table 3.25**
**The predicted carbohydrate availability (gram per day) per individual (gamma errors, log link) displayed by area type, income and number of males in the household.**

| Area/income | No of males in the household | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 17 | Mean |
| **Urban:** | | | | | | | | | | | | |
| 1- Low | 0 | 932 | 0 | 691 | 625 | 625 | 0 | 0 | 0 | 0 | 0 | 795 |
| 2- Medium | 1030 | 932 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1005 |
| 3- High | 0 | 932 | 763 | 691 | 691 | 0 | 0 | 0 | 0 | 420 | 0 | 771 |
| 4- V. high | 398 | 0 | 295 | 267 | 267 | 242 | 219 | 198 | 0 | 162 | 81 | 257 |
| 5- N.I.G. | 1030 | 932 | 763 | 691 | 691 | 625 | 566 | 512 | 464 | 420 | 0 | 660 |
| **Rural:** | | | | | | | | | | | | |
| 1- Low | 0 | 0 | 0 | 1223 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1223 |
| 2-Medium | 1649 | 0 | 0 | 1223 | 1107 | 1002 | 0 | 820 | 0 | 0 | 0 | 1160 |
| 3- High | 0 | 0 | 1351 | 1223 | 0 | 0 | 906 | 820 | 743 | 0 | 0 | 992 |
| 4- V. high | 0 | 0 | 0 | 0 | 0 | 387 | 350 | 0 | 0 | 0 | 0 | 369 |
| 5- N.I.G. | 1649 | 1493 | 1351 | 1223 | 1107 | 1002 | 906 | 0 | 0 | 0 | 0 | 1213 |
| **Bedouin:** | | | | | | | | | | | | |
| 1- Low | 0 | 0 | 0 | 1639 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1639 |
| 2- Medium | 0 | 0 | 1811 | 1639 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1697 |
| 3- High | 0 | 0 | 1811 | 0 | 1484 | 0 | 0 | 0 | 0 | 0 | 0 | 1647 |
| 4- V. high | 0 | 0 | 700 | 0 | 573 | 0 | 0 | 0 | 0 | 0 | 0 | 637 |
| 5- N.I.G. | 0 | 2001 | 0 | 1639 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1820 |
| **Mean** | **1027** | **1206** | **890** | **1068** | **781** | **636** | **524** | **510** | **603** | **368** | **81** | |

N.I.G. = No information given.
0 = zeros correspond to zero observed cases.

### *4. Dietary fibre*

The food tables used in this study was estimated as crude fibre instead of dietary fibre. It has been suggested that crude fibre should be multiplied by a factor of 3.9 to give an estimation of dietary fibre (Ahrens and Boucher 1978). The average dietary fibre intake for the whole population (i.e. almost 43g/day) was higher than that now advocated by the Department of Health for a British Public (18 to 30g/d: DHRHS, 1991). The rural groups had more dietary fibre than urban and Bedouin groups. Possibly because they may have more access to fruit and vegetables. However, the surprising finding was that the Bedouin groups had more dietary fibre than the urban group. The reasons for this may be due to the fact that they eat more cereals. Numerous studies have indicated that dietary fibre is a protective factor against a variety of diseases, in particular CHD.

Fibre was dependent upon area and number of females in the family (Table 3.27). It shows that family consumption of fibre increased as the number of females increased (Table 3.28). For example, families with 18 females in their mean fibre availability were 354.3g/family/day. In contrast, in families with 1-3 females their mean fibre availability was 83.5g/family/day. Perhaps female spend most of their time at home and have more women visitor and customary the provide fruits for their gusts. Otherwise this is difficult to explain, as it would be expected that fibre availability would increase with increasing energy availability. Fibre per individual (analysing using gamma errors, inverse link again with point 93 weighted out) seems to be dependent on area type and food in bulk (Table 3.29). Again, as the predicted individual fibre increased when families bought their food in bulk (Table 3.30). The mean fibre available in the household was higher among rural and Bedouin groups than the urban group.

Table 3.10 shows the variation between the different groups studied and the main sources of fibre. Cereal and cereal products provided 35%, 25% and 21% of fibre recorded for Bedouin, urban and rural groups. The rural group obtained 58.5% of total fibre recorded from fruits, whereas urban and Bedouin groups provided 41% and 38.5%, respectively. Other important variations were among vegetables; urban groups provided a higher proportion (31.5%) than Bedouin (25%) and rural (20%). For the whole population, almost half (46.5%) of fibre recorded was derived from fruits; dates

contributed 45.5% of the total fruit availability. Second was vegetables (27%); tomatoes and potatoes were the most individual vegetable contributing 21% and 10% of total fibre provided by vegetables. Third was cereal and cereal products (24.5%), of which 61% was provided by rice and 21% by bread.

**Table 3.26**
**Calculated average fibre availability (gram per day) by gender, age and area (based on adult males value)**

| | Male age groups | | | | Female age groups | | | |
|---|---|---|---|---|---|---|---|---|
| | > =18 | 11-17 | 5-10 | 1-4 | > =18 | 11-17 | 5-10 | 1-4 |
| **All population** | **10.7** | **11** | **10.9** | **6** | **8.5** | **10.1** | **7.7** | **6.2** |
| Urban | 8.8 | 9.1 | 9 | 4.9 | 7 | 8.4 | 6.3 | 5.1 |
| Rural | 15.7 | 16.2 | 16 | 8.8 | 12.5 | 14.9 | 11.3 | 9.1 |
| Bedouin | 11.6 | 12 | 11.9 | 6.5 | 9.3 | 11.1 | 8.4 | 6.8 |

**Total 3.27**
**Average fibre availability (gram per day) per family. Increase in scaled deviance by removing each factor from the final model AR+F.**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 6.22 | 2 | < 0.05 |
| F | Number of female | 6.55 | 1 | < 0.01 |

**Table 3.28**
**The predicted fibre availability (gram per day) per family (gamma errors, log link) displayed by area and number of females in the household.**

| Number of females in the household | Area type | | | Mean |
|---|---|---|---|---|
| | Urban | Rural | Bedouin | |
| 1 | 56.70 | 106.46 | 89.22 | **82.43** |
| 2 | 63.15 | 118.57 | 99.38 | **81.53** |
| 3 | 70.34 | 132.07 | 110.69 | **88.53** |
| 4 | 78.34 | 147.10 | 123.29 | **105.67** |
| 5 | 87.26 | 163.85 | 0 | **103.67** |
| 6 | 97.19 | 182.49 | 0 | **116.88** |
| 7 | 108.25 | 203.27 | 0 | **155.76** |
| 8 | 120.58 | 226.40 | 0 | **141.74** |
| 9 | 134.30 | 0 | 0 | **134.30** |
| 10 | 149.59 | 0 | 0 | **149.59** |
| 11 | 166.61 | 0 | 0 | **166.61** |
| 18 | 354.34 | 0 | 0 | **354.34** |
| **Mean** | **89.4** | **148.2** | **110.1** | |

0= zeros correspond to zero observed cases.

**Table 3.29**
**Average fibre availability (gram per day) per individual. Increase in scaled deviance by removing each factor from the final model AR+FB**

| Factor removed | | Scaled deviance change | d.f. | P-value |
|---|---|---|---|---|
| AR | Area | 5.7 | 2 | < 0.06 |
| F | Food in bulk | 7.2 | 1 | < 0.007 |

**Table 3.30**
**The predicted fibre availability (gram per day) per individual (gamma errors, log link) displayed by area type and food in bulk.**

| Food in bulk | Area type | | | Mean |
|---|---|---|---|---|
| | Urban | Rural | Bedouin | |
| Yes | 22.0 | 39.9 | 37.5 | **29.9** |
| No | 9.2 | 16.6 | 15.6 | **11.5** |
| **Mean** | **10.3** | **20.5** | **17.8** | |

### *5. Fat and fatty acids*

The findings in this study (Table 3.31) show that fat intake among the Bedouin community (138g) was higher than rural (109g) and urban (88g). However, although the urban group had the lowest fat intake, the ratio to total energy (21.5%) was the highest, and the lowest rate was among the rural group of only 16%. In the UK, the figure 41.3% (MAFF, 1991) is much higher than this study. It should be noted that the effect of over purchasing of cereal in our study was the apparent reduction of the estimate of energy percentage from fat (see Table 3.10). The contribution of fat to the energy content of the diet has been of growing concern over recent years, particularly in relation to incidence of CHD. The Royal College of Physicians and British Cardiac Society recommended that the dietary energy contribution for fat be reduced to 35%. This figure has also been recommended by the COMA panel (DHRHS, 1991), in its recent report. The FA profile in the Saudi diet and the P/S ratio will be investigated in Chapter 5.

More than one third (39.5%) of total fat was derived from oils and fats (Table 3.10). The Bedouins derived 46% of their total fat availability from oils and fats while the corresponding figures for urban and rural groups were 41% and 32%. A variety of vegetable oil products contribute to this total, such as palm, corn, and olive oils. Next to it, also with more than one third (34.5%) of total fat, was meat and poultry. Milk and milk products also contributed significantly to fat with higher education levels (almost 11% of total fat). While milk and milk products; milk (whole, powder and evaporated) provided 46.5%, cheese provided 31% and yogurt 19%. Another contribution was from cereal and cereal products (5.5%), of which 56% was provided from rice, 4.5% of total fat availability derived from eggs with a higher level for urban (almost 5%) and rural groups (4.7%) than Bedouin (2.5%).

**Table 3.31**
**Calculated average fat availability (gram per day) by gender, age and area (based on adult males value)**

| | Male age groups | | | | Female age groups | | | |
|---|---|---|---|---|---|---|---|---|
| | >=18 | 11-17 | 5-10 | 1-4 | >=18 | 11-17 | 5-10 | 1-4 |
| **All population** | **94** | **83** | **71** | **51** | **69** | **75** | **56** | **42** |
| **ratio/energy** | **20** | **19** | **20** | **21** | **21** | **20** | **19** | **18** |
| Urban | 84 | 75 | 64 | 46 | 62 | 67 | 51 | 38 |
| ratio/energy | 21.5 | 21 | 22.5 | 24 | 23 | 23 | 21 | 20 |
| Rural | 104 | 93 | 79 | 57 | 77 | 83 | 63 | 47 |
| ratio/energy | 16 | 16 | 17 | 18 | 17 | 16 | 16 | 15.5 |
| Bedouin | 133 | 118 | 101 | 73 | 98 | 106 | 80 | 60 |
| ratio/energy | 19 | 19 | 20 | 21 | 20 | 19 | 19 | 18 |

### *6. Vitamins and minerals*

Vitamins and minerals were not analyzed in this study because of incomplete data in the food table used. Intake levels however, are expected to be quite satisfactory, particularly since high levels of fruit were consumed. Nonetheless, some studies reported that vitamins deficiency is common in SA. For example, in spite of abundant sunlight, vitamin D deficiency is reported to be common in SA (Woodhouse & Norton, 1982; Sedrani, 1986). Woodhouse and Norton (1982), concluded that this was as a result of a poor dietary intake. In addition, this may be attributed to the low exposure to sunlight due to over dressing and remaining indoors. It is quite obvious that there is a need for research measuring vitamin and minerals intake among the population.

## 3.2.9 Socio-economic Characteristics

Individual food consumption and nutrient intake may be influenced by a host of social, psychological, sensory and economic factors. The search for the link factor-(s) between socio-economic status and food consumption has long been considered important in determining the nutritional status of the population and in the prevention of dietary intake related diseases. For example, if dietary fat is high in the community, it is necessary to determine which socio-economic factors influence that dietary behaviour.

Some socio-economic variables investigated in this study failed to show any relationship with the household's food availability. As explained in section 3.2.8, there is a critical aspect concerning the analysis, we cannot dismiss the possibility that the dietary survey method used may over-or-under-estimate any true associations or distort the results. Some reservations should be made in order to avoid misinterpretation of the following findings. For example, although we found income had an effect on family and individual energy and carbohydrate available, the lack of information on income for 41% of the families meant that the analysis was restricted to certain types of families in which the major source of income from employment, and families in which the major source of income is other than employment (e.g. self employment).

Therefore, we do not infer from our limited data that any of the associations observed were causal. It may be that if the larder before and after the survey measure the relationship with socio-economic factors could be better explored.

Even given these limitations, there are several explanations that can be proposed for the following findings, but all of them must be regarded as suggested directions for future research. They are as follows:

*a. Factors which had effect upon food availability in this study were:*

1. Area type (urban, rural and Bedouin)
2. Family income
3. Food in bulk
4. Number of females in the house
5. Number of males in the house
6. Head of household's education

*b. Factors which had no effect upon food availability in this study were:*

1. Family size (total number of members)
2. Farm
3. Freezer
4. House type (owns, rents or other)
5. Head of household's occupation
6. Housemaid
7. Housewife's occupation
8. Housewife's education
9. Livestock
10. Refrigerator
11. Visitors

The result shows that the family's fibre-availability as well as the individual's energy, protein, carbohydrate and fibre availability were affected by the area in which the participants live. This may be due to lifestyle and social differences between these communities studies. This may also be due to the reflection of the price differences in food stuff, communication, etc, which effects the family's food intake. For example, in the urban area, families with low income had a mean carbohydrate-availability which was much lower, up to two times, than rural and Bedouin groups in the same income category (see Table 3.25).

***Family size***: Family size varied from 2-37 persons (Table 3.32). 51% were

classified as moderate; 29% as large, 12% as small and 8% as very large families. The median and range of household size was 9 (3-37), 9 (2-16) and 7 (3-10) for the urban, rural and Bedouin sample, respectively. Average household sizes among urban families were larger than for rural and Bedouin households. Similar findings observed by Abu Baka and Al-Suleiman (1990), in EP, show that 21% of the mothers had 7-9 children and 7% had 13-15 children. This contrasts with the UK, where the average household consists of 3 people and some households only have one adult. This is due to the fact that, traditionally, in SA most families are extended. Some households included up to three generations, and accordingly had a high number of members. Secondly, the majority get married when they are young. The above study reported that 40% of the housewives in Qatif oasis get married before the age of 14 years. Thirdly, with polygamy being legal in SA, there were a number of cases where the head of the household had up to four wives.

While the literature appears to support the effect of family size on the household food consumption, it is clear that the relationship can not be a simple one. Family size is interrelated with a number of other characteristics of the household, which include family income, food availability, education of the head of the households, household composition, etc. Each of these characteristics, individually, is considered to be an important factor in determining the quality and quantity of the household diets.

It would be expected in this study that as family size increased total family food availability would be increased. The statistical analysis shows that the total family size had significant relation with the total family energy availability. The analysis indicate that every one increase in family size was accompanied by an increased of 4256 kcal/person/day in total family energy availability. When we plot the data (Figure 3.4) using Nelson adults equivalent, it also does suggest a positive relationship between family units and total family energy intake, that as family units (adult male equivalent) increased the total family energy availability increased by 3779 kcal/adult man/day. Note that the statistical line in figure 3.4 goes through the origin. Valenzuela et al., (1979), conducted dietary survey among 97 Filipino families. They measured in their survey the individual intake by food weighing method and found that an increase in family size had a significant negative effect on calorie and vitamin A intake per individual. But the effect was less among those large families, which had more food

resources. It is important to note, however, that we are comparing food availability with an individual weight intake and may be not valid comparison. The effects noted by Valenzuela was thought to be related to individual intake being reduced as per capita income for the family was decreased by increasing family size. It is also, one should be wary of generalisations about the finding in this study and the previous references as it came from developing country, whereas food shortage and poor wages are exits. What is true in developing countries is not necessarily true in affluent countries. It may be reasonable to suggest that large family size does cause high family food availability in our study, whilst individual male equivalent intake remains static, but in actual fact both are probably indicative of income levels and other factors mentioned above.

***Visitors***: Traditionally, in SA household's visitors are more frequent on daily basis. Table 3.33 shows the distribution of the families' visitors during the 5-day studied. There was 527 visitors, 335 (63.6%) male and 192 (36.4%) female. It is not totally clear why the family's visitors had no effect with family nutrients availability investigated here. Maybe the family's visitors have had an effect on other family expenditure rather than foods. More studies in this area are needed.

***Family income***: Since all the household members share in the food expenditure, the family income has been calculated as one unit for each household (i.e. income from the salaries of all the members of the family). Table 3.34 shows the family income distribution. Those who are self-employed usually do not allocate a fixed salary for themselves and therefore they could not provide the necessary information requested. In total, 41% did not give information concerning their income, 23% were considered to be very high earners of which 19% were urban and only 2% for each rural and Bedouin. 6% of households were classified as low income category (4% urban and 1% for each rural and Bedouin). Among the Bedouin sample there was one family without income where the father died and the family were dependent on social and relative help.

Income is considered to be one of the most important factors in determining food consumption in both quality and quantity. In SA, wealth, prosperity and high income have resulted in unique changes in lifestyle, particularly in food availability (see Chapter 2). As SAFBS show, there has been a drastic change in food consumption patterns; diet has changed to a more diversified one, meat is consumed more frequently

as well as eggs, oils, vegetables and fruits. For the reasons discussed earlier, estimating the proportion of the family budget allocated to purchase of foods was beyond the limits of this study. The results show a significant relationship between income and family/individuals energy and carbohydrate availability. Households with the lowest income had the highest energy and carbohydrates availability. The increase in the carbohydrate availability of families who are less well off may have to do with food prices of cereal products, their main food consumption commodity, which contain high levels of carbohydrates and are cheaper than meat. Kamel and Martinez (1984b) found that families with high incomes consume more animal protein foods and fruits. The present study shows no significant relationship between income and family protein availability, which is perhaps surprising given Kamel and Martinez (1984b) findings. Several possible reasons for this lack of relationship might be put forward; for example, some families do not want to disclose their income to outsiders, therefore, they might be under-reporting their actual income. Or there maybe some families who belong to high income groups in those "No information given" category, who were 41% of total households. In our analyses we assumed that families with non-reported income may have similar description as the families who reported there income. This may not reflect the correct results but there is no other way to obtain the exact data and therefore results.

Obviously, the relationship between income and dietary quality may vary from household to household, and from place to place. The study of household food consumption in relation to income must be viewed not as closed, but as open with other household characteristics such as education and family size, including gender and age of the household members. Also there are variations in the way in which household income is controlled. Other studies in other part of SA, for example, might find different relationships between income and diet.

***Education status***: Table 3.35 shows the educational status of the parents of the families studied. Almost half of the head of the households and 70% of the housewives had no formal schooling, although in every household at least one person had some school education. In general, urban and rural inhabitants were more educated than Bedouins. Education only recently became available to Bedouin, and it is younger people who have had the opportunity to attend school. The low education level among

mothers was also observed by another study in the area, where they found that almost 74% of mothers were illiterate (Abu Baker & Al-Suleiman, 1990).

No relationship between the household's education and family nutrients availability investigated here. In contrast, many studies have reported that educated mothers provided better diets for their families. In Bahraini for example where there is no much strict on the female, Musaiger (1986) demonstrated that educated mothers provided better diets for their families than those who were less well educated. The findings in this study could be due to the method used of measuring intake by food purchase. As has been explained earlier, household food purchased is a male responsibility rather than the housewife. Therefore, household education is likely to relate husband education. Poly educated women could marred any body, because man are not restricted on their future wife's education. The result might be not surprising as we are not excepted any relationship between household and food availability.

The heads of household education had its effect on the family's energy availability. Table 3.17 shows that families with head of households with 9 years of schooling, (secondary), had the highest energy availability per individual. Whereas, families whose head of households had above 9 years of schooling (Colleges and Universities), had less per individual of energy availability. This being lower among the Bedouin group, is probably due to the fact that only one family with this category was in this group. However, the overall results may be surprising knowing the facts that better education, in particular nutrition education, is one development measure which may be employed to improve the diet among the nutritionally disadvantaged. The MPHS (1982), reported that a literate and better educated population consumed a greater average number of servings than illiterates and less educated. The reflecting results of this study and the above mentioned survey may, in part, be due to the different survey methods used. The MPHS was an individual study based on 24-hour recall questionnaires, whereas the current study was based on household foods purchased. Perhaps more research in this field might give us more insight, to establish if the findings is true or it was because of the biases in the method.

Normally we expect good relation between good health and education, but in SA not apply because, generally speaking, health education in SA is very poor and far

from adequate, despite the efforts made by the government towards health services. A survey of primary schools in the Gasim region showed that health education knowledge amongst the students about the relationship between health and diet was very poor (Sinha et al., 1985). Although, there are nutrition departments in the main hospitals, their main activities are catering, supervision of kitchens and preparation of patients food. A study carried out by Abdullah and his co-workers (1982), indicates that malnutrition exists in SA and is a serious public problem. They concluded their findings: "*... this is probably due to poor education and infection rather than due to poverty*".

***Occupation status***: Table 3.36 shows the occupation status of the head of household and the housewife of the families studied. The majority of the housewives did not work outside the home, except 5 who worked as teachers. This is not surprising, since in SA the father and husband has the career or job, earning income, whilst the wife and mother is economically dependent on her male, confined to home and child rearing. Traditionally, it is immoral to let the female contribute to the household spending, particularly food stuffs. Even, broadly speaking, if the housewife is working she usually spends her earnings on herself (e.g. buying clothes, etc). Among the husbands in the samples, 13 persons did not work since they were retired. The majority of the head of households worked in government offices or as industrial workers. The self-employed people, worked in agriculture, shepherds and/or in the trade sector. The rural and Bedouin occupation patterns were shown to have shifted from traditional agriculture/shepherds and fishing, to office jobs in the government or private sector. The results show that only 16.5% of the rural subjects had farms, whereas in the urban area 24% had farms. This can be explained by two theories: firstly, the family could migrate from the villages and still have their farm and/or, secondly, this may due to the fact this area has undergoing development from rural to city area. Finally, as explained earlier, the province studied is an agricultural area. Although, this province is known as a fishing area, only one family claimed to do fishing as an occupation. The result shows that the head of households occupation had no relationship with family availability of any nutrients investigated. Again, as explained earlier for education, other family members' occupation may exert an influence. Working housewives also show no relationship with the family availability of any nutrients investigated. This is due to the fact that only a very small number

(5%) of the subjects wives had jobs outside the home.

***Household particulars***: Table 3.37 shows the raw data for the household particulars for different groups studied;

***Food in bulk***: The majority of the families (89%) buy their food in bulk either weekly, such as vegetables and fruits, or monthly, such as meat, rice, sugar, flour etc. Other food stuff, such as milk and milk products and bread, are bought either daily or every two days. This factor was one of the main obstacles on the study to obtain reliable results. The statistical analyses show that food in bulk was related to protein and fibre availability to individuals. Individuals protein availability increased when the families bought their food in bulk. This could be explained by the fact that these families may tend to buy large quantities of meat and poultry, which contain high proteins. Again here, as the families buy their food in bulk the individuals availability of fibre increased. This might be true as the families might buy large quantities of fruit and vegetables.

***Farms***: 25% of the sample had farms. Fishing and bee-keeping are not a significant source of either food subsistence or finance. Only one family had hives and three families fished. Several investigations demonstrated that energy intake is higher among children whose families produce more of their own food (Kaiser & Dewey, 1991). The present study failed to show any relationship whether a family had a farm or not and their energy and other nutrients availability investigated. However, given the fact that only that 25% of the families in the study reported have farm, the lack of a relationship between families's energy or other nutrients availability and ownership of farm is not surprising. Another explanation is that these families may keep farms just for prestige and pleasure and not as an income subsidy. It may also be due to the nature of this area, which characteristically has a very hot climate, which makes growing vegetables and fruits very difficult and not economical.

***Livestock holding***: Animals were kept by 28% of the families. The kind of animal owned by the families varied from hens and pigeons to sheep, cows and camels. Among rural and Bedouin inhabitants, naturally, animals were kept for both food subsistence and cash yielding activities, as well as for prestige; where some of those

in the urban areas kept pigeons for hobbies. However, the keeping of animals shows no significant relationship with the family nutrients availability. This is not surprising among the urban population where their main job is office work, but may surprising to find among the rural and the Bedouin populations, where the owner of livestock is the simple of power and wealth. This shows one of many changes the area experienced, the shift from traditional occupations to work in government offices and private sectors.

***Housemaid***: 19% of the households have a housemaid. All of them lived in the urban area, except one in the Bedouin area. Housemaids had no effect on the family's availability of any nutrients investigated, although all these housemaids came from overseas. Rufaida et al., (1994) found that the number of servants was negatively associated with the BMI among Saudi females in Jeddah. One may expect to see some food consumption patterns change among these families who have housemaids. The facts that, in SA, these servants do not have say in the household affair. Once may not expecting any relationship or at least significant. Perhaps more detail studies is recommended to investigated the family food habits before and after they have the servant. This may help to see if these foreign housemaids have helped in changing family's food habits.

***Household status***: Table 3.38 shows the distribution of the household status among different groups studied. The majority (70%) of the families were household owners, whereas 11% of the families had a rented house and 19% had a government free interest loan. The result shows that no relationship between family nutrients availability and the family's household status. One of the possibilities to explain the findings, is that families who own their homes may be with low income compared, therefore even they have no extra expenditure for rent or loan, they cannot afford to buy large amounts of food. Another, is the opposite, i.e. families who own their homes, may be rich, but they spend their money in other commodities rather than food. These two explanations can also be applied to families who live in rented homes or live in loan houses. Also there is a possibility that a finding effect could not be measured, this being due to the fact that the majority (70%), of the families investigated owned their households. Whereas only 11% households were rented and 19% were government free interest loan.

***Refrigerators***: Refrigerators and freezers are tools of modern society which has its effect on the community food consumption. All the households who participated in this study had refrigerators and 77% had freezers. So a finding effect could not be measured and, with such a small number without freezers, a freezer effect was most unlikely.

Obviously, from the primitive discussions, there appears to be no single key variable that provides a complete understanding of food consumption and socio-economic variables in this population and indeed any other population. There is much to be learned about the factors mentioned in this study, and many other which are not mentioned here, which may influence the pattern of food consumption in SA. More studies are needed to determine the actual impact of each of these factors.

**Table 3.32**
**Family size**

| Family category | Family members range | Area type | | | All population |
|---|---|---|---|---|---|
| | | Urban | Rural | Bedouin | |
| | | n= 66 | n= 24 | n= 10 | n= 100 |
| Small | 2-4 | 7 | 4 | 1 | 12 |
| Moderate | 5-10 | 32 | 10 | 9 | 51 |
| Large | 11-16 | 19 | 10 | - | 29 |
| Very large | >=17 | 8 | - | - | 8 |

**Table 3.33**
**Composition of visitors studied by gender, age groups and area**

| Age group | Type of are | | | | | | All population | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | | | |
| | M | F | M | F | M | F | M | F | T | % |
| <1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1-4 | 12 | 4 | 3 | 3 | 2 | 0 | 17 | 7 | 24 | 4.6 |
| 5-10 | 21 | 16 | 6 | 5 | 2 | 3 | 29 | 24 | 53 | 10.1 |
| 11-17 | 17 | 11 | 13 | 8 | 19 | 5 | 49 | 24 | 73 | 13.9 |
| >=18 | 127 | 98 | 54 | 17 | 59 | 22 | 240 | 137 | 377 | 71.5 |
| **Total** | **177** | **129** | **76** | **33** | **82** | **30** | **335** | **192** | **527** | |

M = male, F = female, T = total.

**Table 3.34**
**Household income distribution by area**

| Income category | Income range | Area type | | | Total |
|---|---|---|---|---|---|
| | | Urban | Rural | Bedouin | |
| | | n=66 | n=24 | n=10 | n=100 |
| Low | <3000 | 4 | 1 | 1 | 6 |
| Medium | 3000-7000 | 4 | 5 | 3 | 12 |
| High | 70001-11,000 | 10 | 6 | 2 | 18 |
| Very high | >11,000 | 19 | 2 | 2 | 23 |
| No information given | | 29 | 10 | 2 | 41 |

**Table 3.35**
**Education of the householder (H) and the housewife (W)**

| Education category | Area type | | | | | | All population | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | |
| | H | W | H | W | H | W | H | W |
| **No formal schooling:** | | | | | | | | |
| Illiterate | 18 | 38 | 10 | 15 | 2 | 6 | 30 | 59 |
| Read only | 7 | 3 | 1 | 1 | - | 2 | 8 | 6 |
| Read and write | 6 | 3 | 4 | 2 | 1 | - | 11 | 5 |
| Total | 31 | 44 | 15 | 18 | 3 | 8 | 49 | 70 |
| **Moderate level:** | | | | | | | | |
| Elementary | 59 | 6 | - | - | 1 | 1 | 10 | 7 |
| Primary | 5 | 2 | 4 | 2 | 1 | 1 | 10 | 5 |
| Secondary | 4 | 29 | 1 | 4 | 3 | - | 8 | 13 |
| Total | 68 | 37 | 5 | 6 | 5 | 2 | 28 | 25 |
| **High level:** | | | | | | | | |
| College & university | 16 | 5 | 4 | - | 1 | - | 21 | 5 |

**Table 3.36**
**Occupation of the householder (H) and the housewife (W)**

| Occupation category | Area type | | | | | | All population | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouins | | | |
| | H | W | H | W | H | W | H | W |
| Not employed | 11 | 61 | 1 | 24 | 1 | 10 | 13 | 95 |
| Government & company employed | 31 | 5 | 15 | - | 8 | - | 54 | 5 |
| Self employed | 23 | - | 8 | - | - | - | 31 | - |

**Table 3.37**
**Household particulars**

| | Area type | | | | | | All population | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | |
| | No | % | No | % | No | % | No | % |
| Refrigerator | 66 | 100 | 24 | 100 | 10 | 100 | 100 | 100 |
| Freezer | 53 | 80 | 15 | 62.5 | 9 | 90 | 77 | 77 |
| Animal | 15 | 23 | 8 | 33.3 | 5 | 50 | 28 | 28 |
| Fish | 3 | 4.5 | - | - | - | - | 3 | 3 |
| Farm | 16 | 24 | 4 | 16.5 | 5 | 50 | 25 | 25 |
| Bee-keeping | 1 | 1.5 | - | - | - | - | 1 | 1 |
| Food in bulk | 60 | 91 | 20 | 83.5 | 9 | 90 | 89 | 89 |
| Housemaid | 18 | 27 | - | - | 1 | 10 | 19 | 19 |

**Table 3.38**
**Households status of 100 Saudi families**

| | Area type | | | | | | All population | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | |
| | No | % | No | % | No | % | No | % |
| Own | 44 | 66.7 | 19 | 79.2 | 7 | 70 | 70 | 70 |
| Rent | 8 | 12.1 | 2 | 8.3 | 1 | 10 | 11 | 11 |
| Loan | 14 | 21.2 | 3 | 12.5 | 2 | 20 | 19 | 19 |

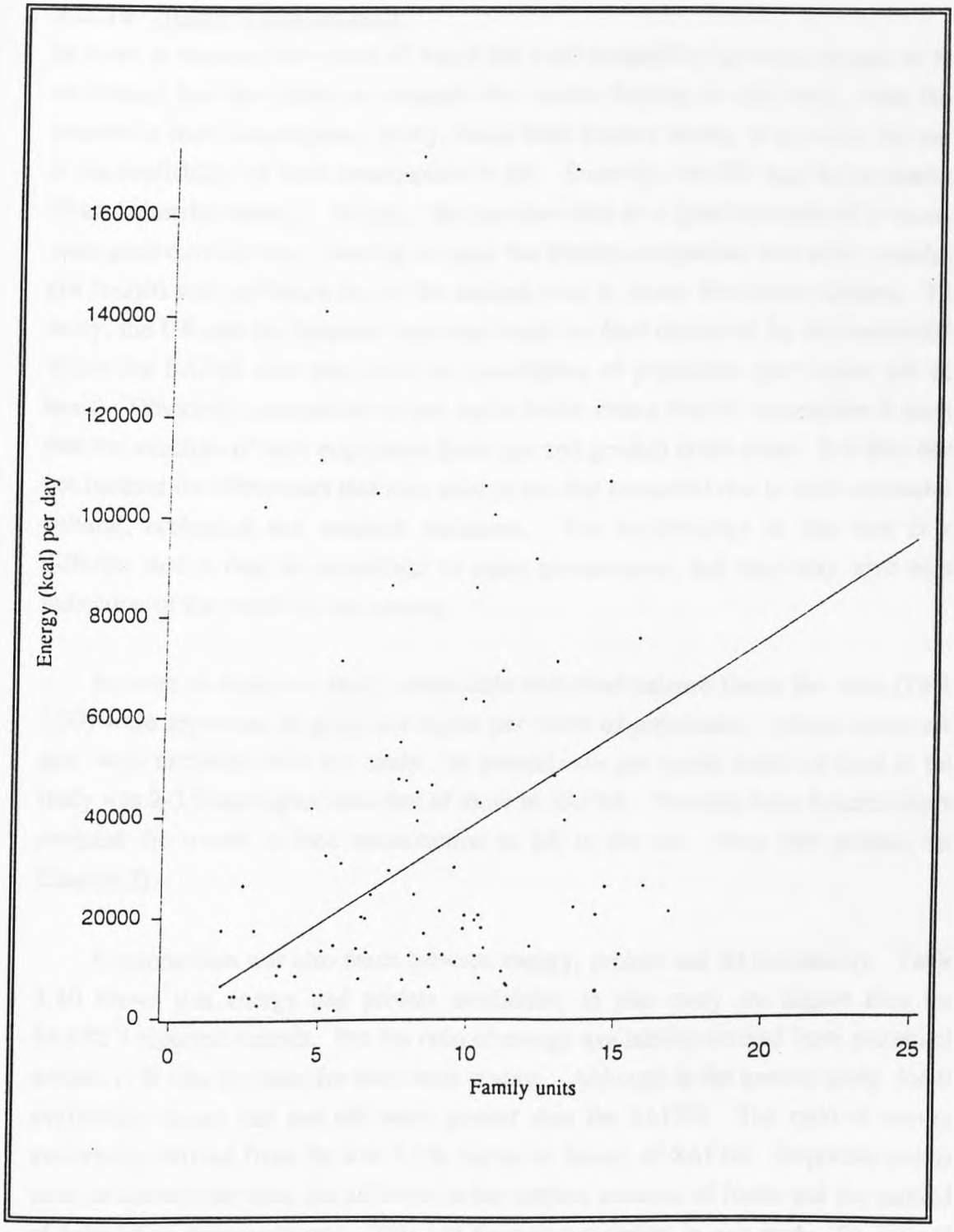



**Fig 3.4**

**Correlation line between family units and total family energy availability**

### 3.2.10 Study Comparison

In order to examine the extent of which the food availability has seen changes in SA, an attempt has been made to compare the current findings in this study, with three sources in food consumption; firstly, Saudi food balance sheets, to examine the trend in the availability of food consumption in SA. Secondly, the UK data as an example of an industrial country. Finally, the Japanese data as a good example of a country undergone development. Bearing in mind that dietary comparison with other countries are fraught with problems due to the method used to assess the dietary intakes. This study, the UK and the Japanese data were based on food consumed by the households. While the SAFBS data was based on assumption of population (per capita per day basis). Obviously, comparison of per capita intake means that the assumption is made, that the structure of each population (both age and gender) is the same. It is also does not indicate the differences that may exist in the diet consumed due to socio-economic, cultural, ecological and seasonal variations. The methodology in this case is so different that it may be unrealistic to make comparisons, but they may give some indication of the trends in the country.

In order to make our study comparable with food balance sheets the data (Table 3.39) were expressed as gram per capita per week of population. Infants under one year were excluded from our study. In general, the per capita intake of food in this study was 2-3 times higher than that of those in SAFBS. The data from balance sheets revealed the trends in food consumption in SA in the last years (for review, see Chapter 2).

A comparison was also made between energy, protein and fat availability. Table 3.40 shows that energy and protein availability in this study are higher than the SAFBS's reported records. But the ratio of energy availability derived from protein of around 11% was the same for both these studies. Although in the present study foods availability except fats and oils were greater than the SAFBS. The ratio of energy availability derived from fat was 7.4% higher in favour of SAFBS. Important points must be considered here; the different in the nutrient contents of foods and the method of calculating these nutrients. This has been demonstrated in our study, fat content analyses (see Chapter 5). Therefore, caution must be taken when interpreting this data.

Rapid industrialisation in SA during 1960s and 1970s resulted in massive rural-urban as well as Bedouin-urban migration. Also considerable numbers of foreign workers have been attracted to the country. Accurate information on their numbers is scarce, it was estimated to be 4.5m in 1985, however, their numbers have declined down gradually. There is no doubt, that an intense international migration as well as indigenous migration had a great impact on the lifestyle of the population, particularly food habits. The adoption/availability of new foods means the adoption of new food habits. Considering these changes, it can be said that the food habits of Saudi became more or less westernised after the oil discovery. There are a wide range of foods are available in the Saudi market. In Bahrain, which experience the same phenomenon, it has been found that there were more than 1900 different kinds of processed foods available on the market imported from 38 countries. Bearing in mind that Bahrain is a very small market in comparison with SA.

As shown in the results (Table 3.39), the diet of EP population resembles the UK in some respects and Japanese in other respects. Cereal and cereal products and fish were almost three times as high than in the UK. Whereas, among the Japanese, cereal was almost twice as high, but fish consumption in Japans was three time as high than EP. Rice is the staple food in the Far East and the Arab Gulf countries. In this study rice consumption was found to be higher than that in Japan. Egg consumption in this study and in Japan were much higher than the UK. Fat and oil consumption were quite similar in this study and the UK, but two and half times as high as in Japan. Also meat consumption was quite similar with the UK, twice as much as Japan.

**Table 3.39**
**Comparison of average foods available for consumption in this study with Saudi-food balance sheets in 1983-1986 (All quantities are in grams per capita per week)**

| Food Items | Present study | SAFBS 1983/86[1] | UK[2] 1991 | Japan[3] |
|---|---|---|---|---|
| Cereal (inc rice) | 3273 | 2351 | 1796 | 2115 |
| (rice) | 1964 | 720 | | 1462 |
| Vegetables (inc potatoes) | 3097 | 1921 | 2730 | 2701 |
| (potatoes) | 538 | 131 | 1183 | 429 |
| Fruits | 4043 | 2631 | 1175 | 965 |
| Meat & poultry | 1528 | 1065 | 1194 | 483 |
| Fish | 382 | 147 | 178 | 649 |
| Eggs (n.) | 4.8 | 3.5 | 2.25 | 5.62 |
| Milk & dairy products | 1125 | 786 | 275 | 851 |
| Fats & oils | 254 | 323 | 307 | 92 |
| Sugars | 1192 | 540 | 206 | 75 |

Sources:

1 = Ministry of Agriculture and Water, department of Economic Studies and Statistics. Saudi Arabian Food Balance Sheets from (1974-1976) to (1983-1986), No. 2. Saudi Arabia.

2 = Ministry of Agriculture Fisheries and Food. Household Food Consumption and Expenditure 1991. Annual Reports of the National Food Survey Committee, HMSO, London, 1992.

3 = Matsumura M and Ryley J. Thirty foods a day-is this the well-balanced way. BNF Nutrition Bulletin, 1991; 16: 83-101.

Table 3.40
Comparison of energy, protein and fat availability in present study with SAFBS in 1983/86. (All figures presented as gram per capita per day).

| | Present study | | SAFBS (1983/86)[1] | | UK 1991[2] | |
|---|---|---|---|---|---|---|
| | | ratio/energy | | ratio/energy | | ratio/energy |
| Energy (calories/capita/day) | 4034 | | 3012 | | 2190 | |
| Fat (gram/capita/day) | 89.5 | 20 | 95 | 28.4 | 100.5 | 41.3 |
| Protein (gram/capita/day) | 114 | 11.3 | 84.2 | 11.2 | 74 | 13.5 |

Sources:

1 = Ministry of Agriculture and Water, department of Economic Studies and Statistics. Saudi Arabian Food Balance Sheets from (1974-1976) to (1983-1986), No. 2. Saudi Arabia.

2 = Ministry of Agriculture Fisheries and Food. Household Food Consumption and Expenditure 1991. Annual Reports of the National Food Survey Committee, HMSO, London, 1992.

Chapter Four

# SECOND STUDY: CORONARY HEART DISEASE URVEY

Section 4.1

# MATERIALS AND METHODS

## 4.1.1 Data Collection

Unlike many developed countries, vital statistics and data on morbidity and mortality and epidemiology of health hazards are greatly deficient in SA and/or not available. Perhaps there are three main reasons for this; (1) there is no clear organisation in the health statistics departments and/or, (2) there is ignorance about the importance of such information. In addition, (3) the lack of well trained Saudi health personal.

*Death Certificates Data*: According to the WHO, death certificates are the most commonly used source to estimate the frequency of diseases in populations. This method may be expected to provide a useful indication of total mortality data among large populations. Not long time ago, a death certificate was not required in SA for burial and therefore people buried their dead without notifying the public security/health authorities. Since 1989, according to Civil Registration Law, the registration of all births and deaths are compulsory. No burial can take place without a death certificate. Therefore, any family, upon the death of another family member, must notify the public security or the health authority before they bury their dead, i.e they must take the deceased to the hospital to establish the reason for death and obtain the permission for burial. An attempt was therefore made at preliminary stages of this study, to review the death certificates at the Ministry of Health Registration Department. However, at the early stage of the survey, the method was omitted from the survey due to its liability and deficiency. Much vital information is missing and/or not filled in with accuracy (e.g. cause of death, age, marital status, place of living and type of job etc). We believe more time is needed to see the full implementation and organisation of the

system. In this study, there were no means of selecting an unbiased sample, as no (even vaguely) accurate sampling frame existed. In addition, there is no data available in this respect. Therefore the hospitals are in the meantime, the only source available with more accurate and complete information on mortality, compared to death certificates.

***Hospitals Mortality Data***: Our definition of the hospital mortality is any deaths occurred during the study period within the hospital and/or at home and the deceased brought to the hospital for clinical examination as this required by the law before given buried's permission. It is understood that this type of data generated from hospitals has its limitations and shortcomings for several reasons which will be fully explained in section 4.2. The data were collected from six Ministry of Health (public) hospitals in EP were studied (see Table 4.1; Figure 4.1). These hospitals were selected because they are the main referral hospitals in the province. The data were based on the information gathered from two sources within the hospitals.
These are:

1. The death registration statements.
2. The files of the deceased patients (patient charts).

These two sources were used because they represent the only complete and reliable data from the hospitals. The data had to be collected in different ways, depending on the format of information provided by the different hospitals. For example, only one hospital provided information on patients death according to patients charts, while other hospitals were only prepared to make the data available on the death registration statements; the patients charts in these hospitals could not be used.

***CHD Recorded Deaths***: The hospitals had recorded deaths using the codes of the International Classification of Diseases (ICD, 9th edition, WHO, 1977). The hospitals records treating codes 410-414 as CHD were used in this study. These were included; acute myocardial infarction (410), other acute and subacute forms of CHD (411), old

myocardial infarction (412), angina pectoris (413) and other form of chronic CHD (414)). For the purpose of this study, sudden death and heart failure were also classified as CHD. Mortality data was collected for the two years 1409 and 1410 (Aug/1988 -Aug/1989 and Aug/ 1989-Aug/1990). Some hospitals could not provide data for one to two months of this period. Infants under one year, non-Saudi nationals and deaths due to unknown causes were excluded from the analysis, in order to obtain more reliable data. CHD mortality was recorded, together with explanatory information. In one hospital (King Fahd), it was possible to collect information on social group (i.e. whether the patient was from an urban, rural, or Bedouin environment). But it was not possible to collect information on age for this hospital. In the other five hospitals, CHD mortality could be collected together with data on age and gender.

***Proportional Mortalities (PMs)***: The data presented here considering the CHD mortality as what we shall call proportional mortalities (PMs), which is obtained by dividing the number of deaths from a given cause (i.e. CHD) by the total number of deaths in the year (Formula 4.1). This has been used to emphasise the importance of the contribution of one cause-specific mortality to overall mortality.

Formula 4.1

$$\frac{\text{the deaths assigned to the disease in a certain year}}{\text{the total deaths in the population in the same year}} \times 100$$

There appears to be some uncertainty when using the proportional mortality ratio (PMR) terminology by different people (Wickham, ed Maregette & Nelson 1991; Pertri 1987; Breslow & Day 1987; Morton & Hebel 1984; Mould 1983). For example, Pertri (1987) and Morton & Hebel (1984) used the PMR to describe the previous formula (Formula 4.1). Whereas Maregette and Nelson (1991) used the same terminology for different formula (Formula 4.2). Therefore, for the purpose of the

present study and to avoid any confusion we use "***proportional mortality***" terminology to describe our data (Formula 4.1). It is understood that this type of analyses is inadequate as a substitute for rates or standardised mortality ratios (SMRs). One of its limitation is that the PM for one condition may be distorted by high or low rates of another condition. Whereas standardised mortality rate is an average death rate in which allowance is made for the age and gender composition of the population. Thus, the SMR compares the observed number of deaths (either from a specific disease or for all causes) which occurred in a designated group with that of a standard population.

In the absence of a knowledge of the age distribution of the Saudi population needed, the rate standardisation could not be used. In order to overcome this obstacle we used PMR formula 4.2 used by Wickham (Maregette and Nelson (1991) to standardise our population study using data from the UK as standard population. The results given by this method (which we call PMR, following Wickham) is almost similar to those produced using SMRs (Roman et al., 1984). The idea of PMR analysis is to compare the fraction of cohort deaths due to a specified cause with the corresponding fraction for the general population or among subgroups in the study group. This type of analysis have been used considerably in descriptive cancer epidemiology when, in the absence of corresponding census data, they may draw to unusual or contrasting patterns of cancer occurrence (Parkin 1986).

Formula 4.2

$$PMR = \sum_{j=1} (d_j) / \sum_{j=1} (t_j \times D_j/T_j) \times 100$$

$$= d / \sum_{j=1} (t_j \times D_j/T_j) \times 100$$

$t_j$ = Total deaths observed.
$d_j$ = CHD deaths observed.
$T_j$ = Total UK deaths.
$D_j$ = CHD UK deaths.

It should be emphasise here that we are studying PMs, it is not an incidence nor prevalence rate, as there is no data available. Therefore, one must be cautious about making any comparison between PMs and incidence data.

### 4.1.2 Statistical Analysis

A standard technique for analysing data such as the present one, is to consider the number of CHD deaths as being a Binomial random variable, representing the number of CHD deaths as a proportion of the total number of deaths, (PM). These CHD counts are to be modelled in terms of the explanatory varieties. For example, in Table 4.5, gender and group (urban/rural/Bedouin) have been used as possible explanatory variables. Similarly, in Table 4.2-4, gender and hospital also have been used as Binomial explanatory variables. As mentioned earlier, the data for Table 4.2-4 was collected for both 1989 and 1990, so there is a further factor which it may call year. The data has been analysed assuming the usual logistic regression model (Collett 1991); the analysis being carried out in GLIM3.77 (Payne, 1985). Logistic regression is widely used as the standard technique for the modelling of count data bounded above. So-called quantal response, e.g. the number of insects for a group of size $n$ who die after exposure to a certain dose of insecticide. Probit analysis was used historically but logistic regression is now generally preferred for the modelling socio-demographic/socio-medical data and, in any case, gives very similar results. Ordinary regression is not appropriate as it does not use appropriate assumptions and, moreover, can lead to predicted probabilities outside the range zero to one. There is a large literature on logistic regression in such cases. A recent comprehensive treatment is in 'Modelling Binary Data', by D Collett, 1991. The use of logistic regression allows one to screen the set of available explanatory varieties to find which of them explains the random variation in the data, and to select a well fitting model for the observed responses. It is simple to test for interactions between effects, as well as looking for 'main' effects. The usual goodness of fit measure was employed, namely the deviance (see, e.g Payne et al, 1985). For logistic regression, the changes in deviance can be treated as approximately following a chi-squared distribution.

**Table 4.1**
**Hospitals which supplied the data for this study.**

| Hospitals | Location | No. of beds | Type of community |
|---|---|---|---|
| Dammam Central Hospital (DCH) | Dammam | 446 | urban |
| Gatif Central Hospital (GCH) | Gatif | 234 | mixed |
| Gatif General Hospital (GGH) | Gatif | 52 | mixed |
| *Al-Jubail General Hospital (JGH) | Al-Jubail | 200 | mixed |
| King Fahd Hospital (KFH) | AL-Hofuf | 770 | mixed |
| King Khalid Hospital (KKH) | Hafr AlBatin | 284 | Bedouins |

* JGH provided data for this study only for 1990.

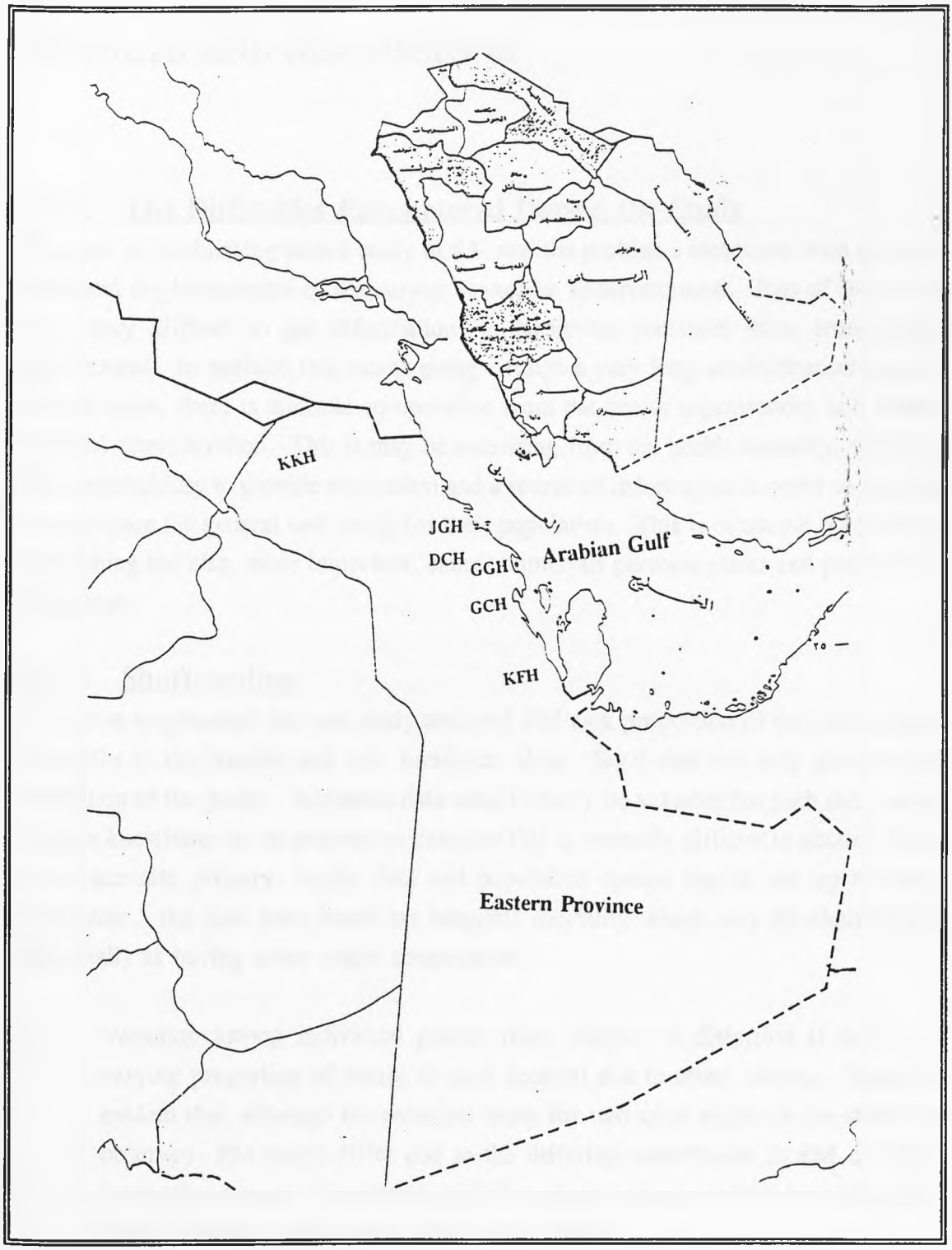



**Figure 4.1**

**The location of the hospitals which supplied the data for this study**

# RESULTS AND DISCUSSIONS

## 4.2.1 The Difficulties Encountered During the Study

The task of establishing such a study in SA, and the problems associated with the study field and implementation of the survey cannot be underestimated. Part of this is that it is very difficult to get information, in particular statistical data, from central government. In addition this means going through a very long administration process. Furthermore, there is minimal co-operation from the health organisations and indeed, hospital administration. This is may be surprising from the health authority, who have the responsibility to provide motivation and a source of information in order to motivate and enhance the general well being for their population. This is of course not just time consuming but also, more important, causes numerous personal stress and pressure for the author.

## 4.2.2 Shortcomings

It is to be emphasised that our study analysed PM as a proportion of the total number of deaths in the hospital and not incidence data. Such data can only give a crude indication of the deaths. Incidence data would clearly be valuable but such data would require knowledge of the population base and this is currently difficult to obtain. There is no accurate primary health data and population census data is not up to date. Moreover, the data were based on hospitals mortality which may be characterised informally as having seven major components:

1. Variation among individual patient risks; subject to distortion if there is a varying proportion of deaths in each hospital due to other causes. Thus it is evident that, although the recorded death for two areas might be the same, the observed PM might differ due to the differing contribution to PM of deaths from other causes. Conversely, differing death rates might lead to similar PM, due to fortuitous death rates from other causes.

2. Variation due to variable quality of medical care in the hospital. It may mean that most patients were taken to the hospitals that treated them effectively, that

is a good overall medical care system.

3. Variation due to the hospital's admissions policy e.g. the DCH hospital does not admit infants under one year.

4. Variation due to the basis of the referral to the hospitals between groups studied. There is no information about available for us about this, e.g. what is the basis for the Bedouins referrals. Our impression is that they are unlikely to be referred just for minor complains. This is because its inconvenience for them to travel, considering the long uncomfortable distance. Also this might very much depends on the doctor diagnosis.

5. Diagnostic inaccuracy for deceased who died at home and were brought to the hospital to establish the reason for death. These may be not seen by a qualified person/doctor to establish the exact nature of death.

6. All deaths may be not reported to the hospital. Again here there is no data available of the number of people who die at home. As has been mentioned in section 4.1, Since 1989 the government made it compulsory that any one who dies at home must be taken to hospital for examination before burial. But it is not known how many Bedouin or non-Bedouin, comply to this law. But we presume all groups have reported the death to the hospitals in the same manner, as we have no evidence otherwise.

7. Random variation, attributable to purely random process (random sources).

With all the limitations outlined above, this study indicates some hypotheses with policy implications; it is hoped that it might see the official collection of more comprehensive data to enable such hypotheses to be investigated more fully. A comprehensive nationwide census to record CHD deaths annually in SA, might be one of the best methods to monitor the CHD. It is a very difficult task and does require much experience, time, means and most of all dedication.

### 4.2.3 Study Outcomes

The data presented here was derived from recorded deaths among in-and-out-patients

in six Ministry of Health Hospitals, EP. The overall results show that almost 26% (n=540) of total deaths were recorded as CHD deaths, 359 male CHD deaths representing 27% of total male deaths, and 181 female deaths representing 23.5% of total female deaths. The observed PM in present hospitals are not dissimilar to incidence data in other Arab Gulf countries which share similar social, economic and geographical conditions. Musaiger and Aldallal (1983), found that diseases of the circulatory system were responsible for about 26.5% of deaths in Bahrain, with CHD and hypertension being the leading cause. In Kuwait more than one-third (37%) of the current mortality rate is due to cardiovascular disease (Kohli and Al-Omaim 1986). It is to be stressed that we are not considering here the death rate due to CHD as a percentage of the total population at risk (incidence data), as accurate data on the total number of people at risk in each hospital area is not available.

### 4.2.4 Data Description

The six hospitals studied have catchment areas which differ considerably (Table 4.1). One hospital (DCH) might be considered to have a mainly urban catchment; one (KKH) has a mainly Bedouin catchment whilst the others have a population of urban, rural and Bedouin patients. The death registration statements of all the hospitals were obtained, with the exception of KFH where medical files were used.

The analysis of death data may be carried out in one of two ways because of the classification of this data in "**known**" and "**unknown**" death. One approach was to treat the "known-death" and "unknown-death" data separately. This will determine the minimum PM due to CHD in population but not including any possible cases from the "unknown-death". The other method was to estimate the PM with respect to the total death (i.e. both the known and unknown-death). In both approaches the true PM due to CHD in the population is underestimated.

It is recognised that calculations based only on those deaths from registration statements not classified as unknown, may underestimate the true PM of death due to CHD in the population as a whole and in each hospital. The "known-death" data was used since it will give a definite indication of the cause of death from the data available. This will set the minimum PM. The data of "unknown-death" was not included in the above analysis since it will result in uncertainty in final discussions. The effect of the "unknown-death" is that, in real terms, the minimum PM is enhanced depending on the

nature of the death. This method was used since it gave clear data for the health policy making in this field. The proportions of unknown causes of death in all hospitals were approximately 7%. The exception was GGH (50%), where the majority of the patients had died at home and the cause of death was not determined. However, this hospital was excluded from the analysis.

Tables 4.2-4 shows a summary description of the data reviewed (Appendix M, give full description of the data). Overall, 3276 deaths were examined. Infants (under one year old) (n= 915) deaths and those due to unknown causes (n= 275), which make up 36.3% of the total recorded deaths, were excluded. This reduced the deaths studied to 2086: i.e. 63.6% of the total registered deaths. A total of 1317 male adults (63%) and 769 female adults (37%) were identified. Table 4.5-7 shows the full distributions of total deaths and CHD deaths studied by age and gender. The KFH is a hospital in which all three lifestyle groups (urban/rural/Bedouin) are represented. This allowed comparisons between these three groups to be conducted.

### 4.2.5 Description of Distribution of PM at King Fahd Hospital

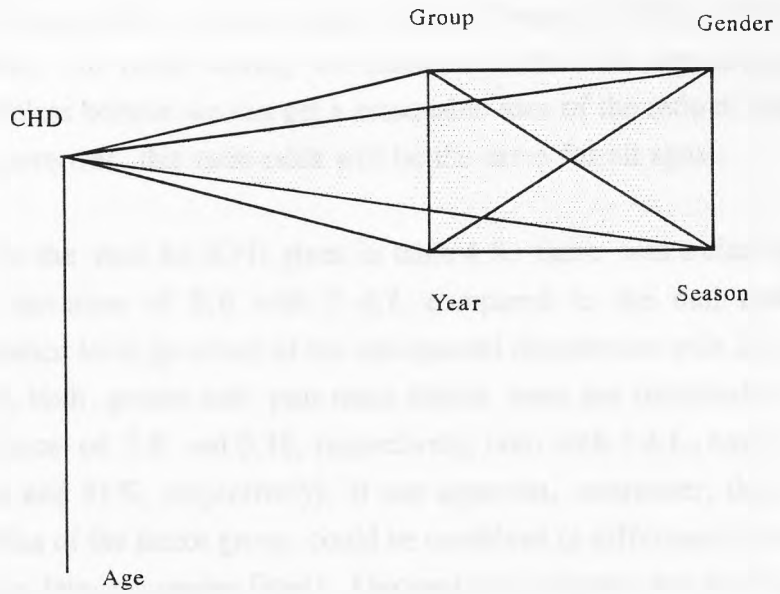
Table 4.8 refers entirely to the King Fahd Hospital. This hospital is located in the Al-Hasa oasis of EP at Al-Hofof city and serves a population of approximately 500,000 inhabitants from different lifestyle groups. The medical records of all Saudi deceased patients (N= 1188) in that hospital during two years 1409 and 1410 (Aug/1988-Aug/1989 and Aug/1989- Aug/1990) were retrospectively reviewed. Deaths due to unknown causes and infant deaths represent almost 36%; they were excluded. This reduced the deaths studied to 763: i.e. 64% of the total registered deaths. A total of 447 male adults (58.5 %) and 316 female adults (41.5%) were identified. Of all adult registered deaths, 244 (32%) died in the hospital from CHD. Of these 244 deaths, 152 (62% of CHD deaths) were male and 92 (38% of CHD deaths) were female.

***Statistical Analysis of Table 4.8 (King Fahad Hospital)***

The first discussion concerns Table 4.8. We find out that we have no data on the age of the patients at KFH. Age is clearly an important factor in CHD. Following Guo and Geng (1995), we can consider the logistic regression of CHD on group, gender, season, without reference to age, provided that age is conditionally independent of group, gender, year and season, given CHD, as illustrated in Diagram 4.1.

**Diagram 4.1**

**The interaction between CHD, age, gender, group, season and year**




The result of Guo and Geng shows us that we can find the size of the effect of group, gender, year and season from logistic regression of CHD on these variables alone, provided the above conditional independence holds. Moreover, assuming there is no unobserved confounding variable (e.g. smoking) relating age to the other explanatory factor; this is an untested assumption. In our case, we have no direct information for KFH as to whether age is indeed conditionally independent of the other explanatory variables, given CHD. However, using an "*ecological*" approach, we tested this conditional independence hypothesis in the large data set, for the other 4 hospitals where age data was available (see section 4.2.6). We treated hospital as a surrogate variable for "group" (grouping the hospitals into 3 similar trends, and used a season variable with 2 levels (summer, winter). Fitting a log-linear model using Glim4, we tested the model;

CHD * age * hospital * gender * season * year

**against the model**;

CHD * age + CHD * hospital * gender * season * year.

This test gave a difference in scaled deviance of 279 with 230 d.f., indicating that we would accept the conditional independence hypothesis on the 1% level, although not at a 5% level. Thus we feel that, fortunately, we can perhaps obtain some idea of the size of the gender, group, season, year effects in KFH, without having age data available. In other words, we cannot ascertain the age effects in KFH but we nevertheless believe we can get a reasonable idea of the ratio of odds for each variable (and, moreover, this ratio odds will be the same for all ages).

For the data for KFH given in table 4.8, there was a clear group main effect (a scaled deviance of 8.6 with 2 d.f. compared to the null model, i.e. the 1.3% significance level (p-value) of the chi-squared distribution with 2 d.f.). Having allowed for this, both gender and year main effects were not statistically significant (changes in deviance of 2.8 and 0.18, respectively, both with 1 d.f., having significance levels of 11% and 81%, respectively). It was apparent, moreover, that the urban and rural categories of the factor group could be combined (a difference in deviance of only 0.17 with one less parameter fitted). Our analysis indicated that there was no year, gender or season effect for KFH, but there was a group effect (i.e. a difference between urban/rural and Bedouins). The ratio of estimated odds for urban/rural compared to Bedouins is 0.4948/0.1579 = 3.1, indicating a much higher CHD PM for urban/rural than for Bedouins. Moreover, our analysis indicating that this ratio of odds is perhaps found to be the same for all ages, and appears the same for males and female, for both years and for both summer and winter.

### 4.2.6 Statistical Analysis of Table 4.5-7 (All Hospitals)

The data of Table 4.2-4 can, generally, be expanded to include age and month of year as shown in Table 4.5-7. However, age data was **not** available for KFH, so the following analysis of Table 4.5-7 excludes this hospital. The expanded data set consists of 1782 observed cells (see, Appendix M). A first point to notice was that GGH has very poor data records, with 50% of deaths being unclassified. This hospital is therefore excluded from the analyses.

The analyses of Table 4.5-7 is based upon possible gender, age, month, year and hospital effects. There was a significant gender main effect (a change in scaled deviance of 11.1 with 1 d.f., i.e. the 0.1% level), contrary to the conclusion for Table 4.8 (which is only for KFH). However, it should be noted, that these data does not

have he benefit of having the factor group, which was the key explanatory variable of Table 4.8. It is therefore possible that, by ill-chance, a differing proportion of males/females in each (unobserved) group might be causing this supposed gender effect (i.e that CHD appears to be related to group, whereas in reality CHD is conditionally independent of group, given gender). Data on the lifestyle groups would be needed to get a firm view on this. Without the benefit of such grouping data, we have concluded that the data appears to indicate evidence of a possible gender effect. The data also shows strong evidence of a difference between the CHD PM in the 4 hospitals here considered (i.e. excluding KFH). However, there is no significant difference between GCH, KKH and JGH (change in scaled deviance of 1.8 with 1 d.f., i.e. the 18% level of a chi-squared distribution with 1 d.f.); i.e. the hospital KKH with a mainly Bedouin catchment had PM similar to the more mixed GCH and JGH. There was no evidence of a difference between years (main effect having a scaled deviance of 1.0 with 1 d.f., i.e. p=0.31). The 4 winter months (November to February) were similar to each other, but had higher CHD proportion than the 8 summer months (March to October).

Table 4.9 shows the predicted percentage of CHD mortality for the 4 hospitals which had complete age, month, year and gender data, but excluding GGH where records were poor. Table 4.9 displays the fitted PM from our best model (i.e. the 'main effects' model, including gender and using the reduced level factors for 'age', 'months' and 'hospital' and omitting the insignificant year effect, there were no significant interaction effects). Table 4.9 shows an average over the two years of the study. Table 4.9 uses combined hospitals GCH, KKH and JGH as we found no significant interaction effects between them. Also, Table 4.9 uses combined age groups 35-54 and 55-74, as we found no statistically significant difference between 35-44 and 45-54, or between 55-65 and 65-74 (a change in scaled deviance of 6.6 with 5 d.f., i.e. p= 0.25). As remarked above, we combined months which are not statistically different (a scaled deviance change of 7.2 with 10 d.f., i.e. the 70% significance level), to give a 'season' factor with levels corresponding to 'winter' and 'summer'.

For all ages, the proportion of the male patients recorded as dying from CHD, as opposed to other causes of death is higher than the proportion of female deaths, i.e. given a death, the probability of the death being recorded as CHD is higher for men

two to three times that of women up to the age of 45 than. Other studies have reported that younger women are relatively protected, but it tends to show up some 10 years later (Kaplan et al., 1984). However, in the older age groups (by age 70) in both sexes, the incidence is approximately equal (Desmond, 1988). These differences in the incidence between men and women may be attributed to the differences in hormonal factors (Jackson, 1988), and women have relatively higher levels of HDL cholesterol. In addition, males had higher blood pressure and more extensive atherosclerotic lesions at the carotid bifurcations than females (Kaplan et al., 1984).

The CHD PM is considerably higher in winter than in summer. The urban hospital has notably high CHD proportions. The findings agreed with other studies (Fox, 1987; Scragg et al., 1990). Fox (1987), indicated three factors which might contribute to the increase in CHD mortality in winter. These are; (1) increase spread of infection, (2) increase in pneumonia and (3) ambient temperature itself.

The mainly Bedouin KKH does not show significantly lower proportions of CHD than GCH and JGH, so no clear 'Bedouin' effect is noticeable here, unlike our results for KFH.

In Table 4.10, it presents estimated CHD PM for males and females of differing ages from the four hospitals (excluding GGH and KFH). In this table, 6 age groups were used, to correspond to the PM data available for the UK. CHD PM in Saudi have been estimated in each hospital from a model assuming the rates to differ for all 6 age categories (although as remarked above, some could be combined).

From Table 4.10, it may be observed that there is a general tendency for CHD PM to increase with age e.g. 15% of deaths of men in the age group 35-44 were estimated as CHD rising steadily to 35% of the 65-74 year old men. This may be associated with an increase in the prevalence of risk factors such as high blood pressure, blood cholesterol concentration (Beaglehole, 1991). It has been demonstrated that LDL cholesterol level is higher in older people than in younger ones, and higher in males than in females (Burton & Foster, 1988). Men increase LDL cholesterol at about 20 mg/dl/10 years of age, whilst women increased about 3 mg/dl (Jacobs, 1988). Other studies have shown that total cholesterol and triglycerides (Tri), LDL cholesterol and Tri/HDL cholesterol ratio increases with age (Cuesta et al., 1989). While other studies

provide evidence that CHD begins very early in life (Klevay, 1987; Garcia, 1990; Ball et al., 1981). This study shows that after the age of 74 the death rate seems to drop in both sexes but it drops more sharply among females than males. 24% of male deaths over 75 years old are estimated to be CHD. There is possibility of artificial cohort effects. Since all deaths, including CHD, occurred before age 74 and only the reminder occurs after. For females, however, 10% of the deaths of those in the age group 35-44 are estimated to be CHD, rising to 24% of 65-74 years olds. 16% of the over 75 year old female deaths are from CHD. These results may be compared with UK CHD PM data, as given in Table 4.10. The similarity in the patterns may be observed. The overall predicted PMs in Table 4.10, aggregated over age, are somewhat different from those the UK PM data.

The Expected number of death in Saudi age group, is given by $t_j \times D_j/T_j$, as shown in table 4.11. The overall PMR is given by $\Sigma\, d/\, \Sigma\, (t_j \times D_j/T_j) \times 100$. The PMR for a given age is given by $d_j/\, (t_j \times D_j/T_j) \times 100$ (Table 4.12). *Note: $t_j$= Total deaths observed, $d_j$ = CHD deaths observed, $T_j$ = Total UK deaths, $D_j$ = CHD UK deaths.*

Table 4.12 illustrate the CHD PMR by different age groups (i.e comparing Saudi to the UK). It shows that the CHD PMR for male increased by age, whereas female CHD PMR shows the opposite characteristics. This apparent difference could be due, in part, to the low number of CHD deaths among the low age group in our study, although the trend seems indication of some difference between the UK and Saudi Table 4.11). The difference between the PMR for men and women seems potentially interesting. However, overall, the PMR is 1.02 for males, and 1.09 for females, indicating not much overall difference between Saudi and the UK, despite an apparent difference of different ages.

In conclusion, the data discussed above shows two major points that; first, the observed CHD PM amongst the Bedouin was the lowest in comparison with non-community. The variation observed may be due to social/cultural/economic/ environmental differences between the social groups using these hospitals. But, the reasons for the existence of these differences cannot fully be explained by this study. The results from this study (see chapter 3) shows marked differences in the dietary habits and food availability between these communities. This may indicated that dietary

habits differences may have some explanation for our finding. Indeed, it is well known that in population, whose diet contains high calories and more fat, such as the industrial countries e.g. the UK., CHD mortality exists in far higher rates whereas lower rate is found with opposite dietary habits. Other factors such as smoking, blood pressure, physical activity, unemployment, etc., which may also contribute to our finding. Unfortunately, there is no published data regarding these aspects among these population to give a more clear picture. Although we recognise of the important of the factors mentioned above, but it was not possible for us to collect this information. This because of the large scale of the project, time and the means required. Future studies in this area are needed. However, in comparison with other populations from other countries, the Saudi Bedouin and the Aborigines (e.g. Australia Aborigines, American Indians) may be compared on the basis that these two ethnic population share the same lifestyle (lived as nomadic hunter-gatherers). O'Dea (1991), characterised the Aborigines lifestyle as high physical activity and a diet of low energy density (low fat, high fibre). When the Aborigines made the transition from traditional hunter-gatherer lifestyle to a westernised lifestyle, they have been shown to develop high prevalence rates of CHD, obesity, diabetes and hypertension compared with the non-Aborigines (Thomson, 1991). It is worth noting, that the experience of the Bedouin people here has not been the same as that of the Aborigines. The Aboriginal people have been colonised by another country, which led to dispossession, deprivation, their culture demolished and they are viewed as subhuman (Kunitz, 1990). This may give some explanation to their high CHD rate where they are under stress and emotion. Whereas, the Bedouin in our study have their full right and freedom and enjoy a very relaxed life. The result is suggests an interesting hypothesis for future evaluation.

Secondly, the overall observed CHD PM shows a similarity with the UK CHD PM. Again here there are many reasons for this apparent similarity with the UK CHD PMs which can not be fully explained by this study. If we take diet as, which is an important factor for CHD, one example to explain our findings. The diet-heart hypothesis assumes that pathological changes leading eventually to CHD begin early life and continue to develop thereafter in populations consuming high SFA diets. The estimates for latency to the exposure to dietary intake and the development of the disease range from a few years to decades. Of course such latency period and its effect to develop the disease may vary between individual and may be affected by the presence of other risk factors. From the observation made in this chapter and in the

second study, of what seems to be high CHD PM. We have believe that in SA, diet, in part, paly major role in CHD deaths. That to say, long term of such dietary behaviour (see Chapter 3 and 5) have possible interaction with the observed CHD mortality in this country. Only further investigations can arrive at a final conclusion. The available epidemiological data from other countries who witnessed a rather similar dietary and related lifestyle changes as SA, show an increased in chorionic diseases such as CHD (Yuichiro 1992; King & Rewers 1991; Walker 1989; Egusa et al., 1984; Hiral et al., 1986; Palgi 1981). Japan may be regarded as a classic example of the lowest CHD incidence in the world, however, for the last two decades the country witnessed a gradual increase of this disease. Yuichiro (1992) and Hiral et al., (1986), gave evidence that these changes in the CHD incidence were results of the Japanese population becoming more affluent and adopting negative dietary habits, in particular increase intake of meats, dairy products and eggs, and decreased intake of cereals and potatoes, hence the SFA intake increased and the ratio of P/S has decreased. Palgi (1981) suggested that dietary changes in Israel accounted for 42 to 57% of the increase in CHD, when total daily fat rose steadily from 25.5% in 1949-1950 to 33.2% of total calories in 1976-1977.

**Table 4.2**
**Summary of the recorded deaths reviewed (included and excluded deaths in the study) by hospital and gender during 1989.**

| HOSPITALS | OVERALL RECORDED DEATHS | INCLUDED DEATHS | | EXCLUDED DEATHS | | |
|---|---|---|---|---|---|---|
| | | Total | CHD | Unknown deaths | <1 year | Total |
| **DCH:** | | | | | | |
| Male | 170 | 158 | 52 | 12 | 0 | 12 |
| Female | 78 | 74 | 22 | 4 | 0 | 4 |
| Total | 248 | 232 | 74 | 16 | 0 | 16 |
| **GCH:** | | | | | | |
| Male | 145 | 91 | 12 | 11 | 43 | 54 |
| Female | 98 | 59 | 6 | 7 | 32 | 39 |
| Total | 243 | 150 | 18 | 18 | 75 | 93 |
| **GGH:** | | | | | | |
| Male | 73 | 28 | 7 | 38 | 7 | 45 |
| Female | 54 | 21 | 8 | 28 | 5 | 33 |
| Total | 127 | 49 | 15 | 66 | 12 | 78 |
| **KFH:** | | | | | | |
| Male | 375 | 239 | 81 | 6 | 130 | 136 |
| Female | 258 | 168 | 51 | 3 | 87 | 90 |
| Total | 633 | 407 | 132 | 9 | 217 | 226 |
| **KKH:** | | | | | | |
| Male | 175 | 102 | 24 | 9 | 64 | 73 |
| Female | 99 | 34 | 6 | 9 | 56 | 65 |
| Total | 274 | 136 | 30 | 18 | 120 | 138 |
| **OVERALL:** | | | | | | |
| **Male** | **938** | **618** | **176** | **76** | **244** | **320** |
| **Female** | **587** | **356** | **93** | **51** | **180** | **231** |
| **Total** | **1525** | **974** | **269** | **127** | **424** | **551** |

**Table 4.3**
**Summary of the recorded deaths studied (included and excluded deaths in the study) by hospital and gender during 1990.**

| HOSPITALS | OVERALL RECORDED DEATHS | INCLUDED DEATHS | | EXCLUDED DEATHS | | |
|---|---|---|---|---|---|---|
| | | Total | CHD | Unknown deaths | <1 year | Total |
| **DCH:** | | | | | | |
| Male | 137 | 128 | 34 | 9 | 0 | 9 |
| Female | 82 | 77 | 19 | 8 | 0 | 8 |
| Total | 222 | 205 | 53 | 17 | 0 | 17 |
| **GCH:** | | | | | | |
| Male | 194 | 136 | 29 | 15 | 43 | 58 |
| Female | 146 | 91 | 11 | 10 | 45 | 55 |
| Total | 340 | 227 | 40 | 25 | 88 | 113 |
| **GGH:** | | | | | | |
| Male | 70 | 35 | 15 | 32 | 3 | 35 |
| Female | 64 | 25 | 9 | 33 | 6 | 39 |
| Total | 134 | 60 | 24 | 65 | 9 | 74 |
| **JGH:** | | | | | | |
| Male | 66 | 48 | 11 | 4 | 14 | 18 |
| Female | 38 | 19 | 2 | 3 | 16 | 19 |
| Total | 104 | 67 | 13 | 7 | 30 | 37 |
| **KFH:** | | | | | | |
| Male | 332 | 208 | 71 | 1 | 123 | 124 |
| Female | 234 | 148 | 41 | 1 | 85 | 86 |
| Total | 566 | 356 | 112 | 2 | 208 | 210 |
| **KKH:** | | | | | | |
| Male | 250 | 144 | 23 | 21 | 85 | 106 |
| Female | 135 | 53 | 6 | 11 | 71 | 82 |
| Total | 385 | 197 | 29 | 32 | 156 | 188 |
| **OVERALL:** | | | | | | |
| **Male** | **1049** | **699** | **183** | **82** | **268** | **350** |
| **Female** | **702** | **413** | **88** | **66** | **223** | **289** |
| **Total** | **1751** | **1112** | **271** | **148** | **491** | **639** |

**Table 4.4**
**Summary of the recorded deaths studied (included and excluded deaths in the study) by hospital and gender during 1989 and 1990.**

| HOSPITALS | OVERALL RECORDED DEATHS | INCLUDED DEATHS | | EXCLUDED DEATHS | | |
|---|---|---|---|---|---|---|
| | | Total | CHD | Unknown deaths | <1 year | Total |
| **DCH:** | | | | | | |
| Male | 307 | 286 | 86 | 21 | 0 | 21 |
| Female | 163 | 151 | 41 | 12 | 0 | 12 |
| Total | 470 | 437 | 127 | 33 | 0 | 33 |
| **GCH:** | | | | | | |
| Male | 339 | 227 | 41 | 26 | 86 | 112 |
| Female | 244 | 150 | 17 | 17 | 77 | 94 |
| Total | 583 | 377 | 58 | 43 | 163 | 206 |
| **GGH:** | | | | | | |
| Male | 143 | 63 | 22 | 70 | 10 | 80 |
| Female | 118 | 46 | 17 | 61 | 11 | 72 |
| Total | 261 | 109 | 39 | 131 | 21 | 152 |
| **JGH:** | | | | | | |
| Male | 66 | 48 | 11 | 4 | 14 | 18 |
| Female | 38 | 19 | 2 | 3 | 16 | 19 |
| Total | 104 | 67 | 13 | 7 | 30 | 37 |
| **KFH:** | | | | | | |
| Male | 707 | 447 | 152 | 7 | 253 | 260 |
| Female | 492 | 316 | 92 | 4 | 172 | 176 |
| Total | 1199 | 763 | 244 | 11 | 425 | 436 |
| **KKH:** | | | | | | |
| Male | 425 | 246 | 47 | 30 | 149 | 179 |
| Female | 234 | 87 | 12 | 20 | 127 | 147 |
| Total | 659 | 333 | 59 | 50 | 276 | 326 |
| **OVERALL:** | | | | | | |
| **Male** | **1987** | **1317** | **359** | **158** | **512** | **670** |
| **Female** | **1289** | **769** | **181** | **117** | **403** | **520** |
| **Total** | **3276** | **2086** | **540** | **275** | **915** | **1190** |

**Table 4.5**
**Summary of the recorded deaths studied by hospitals, age and gender in 1989.**

| Hospital | | Male | | | | | | Female | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
| DCH | ALL | 36 | 7 | 18 | 28 | 36 | 33 | 8 | 5 | 5 | 13 | 19 | 24 |
| | CHD | 0 | 2 | 7 | 12 | 17 | 14 | 1 | 0 | 2 | 6 | 6 | 7 |
| GCH | ALL | 27 | 1 | 4 | 13 | 22 | 24 | 17 | 2 | 5 | 6 | 10 | 19 |
| | CHD | 0 | 0 | 1 | 0 | 9 | 2 | 0 | 0 | 0 | 0 | 4 | 2 |
| GGH | ALL | 18 | 0 | 1 | 1 | 1 | 7 | 5 | 1 | 1 | 1 | 2 | 11 |
| | CHD | 0 | 0 | 0 | 1 | 0 | 6 | 0 | 0 | 0 | 0 | 1 | 7 |
| KKH | ALL | 26 | 7 | 8 | 15 | 19 | 27 | 6 | 4 | 2 | 2 | 9 | 11 |
| | CHD | 0 | 2 | 1 | 4 | 10 | 7 | 0 | 1 | 0 | 1 | 2 | 2 |
| Total | ALL | 107 | 15 | 31 | 57 | 78 | 91 | 36 | 12 | 13 | 22 | 40 | 65 |
| | CHD | 0 | 4 | 9 | 17 | 36 | 29 | 1 | 1 | 2 | 7 | 13 | 18 |

ALL = Total recorded deaths
CHD = Coronary heart disease deaths

Table 4.6
Summary of the recorded deaths studied by hospitals, age and gender in 1990.

| Hospital | | Male | | | | | | Female | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
| DCH | ALL | 36 | 9 | 11 | 12 | 30 | 30 | 11 | 2 | 6 | 18 | 11 | 29 |
| | CHD | 1 | 1 | 6 | 6 | 11 | 9 | 0 | 0 | 1 | 8 | 2 | 8 |
| GCH | ALL | 22 | 6 | 9 | 22 | 42 | 35 | 18 | 2 | 6 | 14 | 18 | 33 |
| | CHD | 0 | 1 | 1 | 7 | 13 | 7 | 1 | 1 | 1 | 1 | 3 | 4 |
| GGH | ALL | 9 | 1 | 1 | 4 | 9 | 11 | 6 | 2 | 1 | 3 | 3 | 10 |
| | CHD | 0 | 1 | 1 | 2 | 7 | 4 | 0 | 0 | 1 | 3 | 3 | 2 |
| JGH | ALL | 12 | 3 | 4 | 6 | 7 | 16 | 4 | 0 | 0 | 6 | 3 | 6 |
| | CHD | 0 | 0 | 1 | 1 | 3 | 6 | 0 | 0 | 0 | 1 | 0 | 1 |
| KKH | ALL | 45 | 10 | 11 | 18 | 15 | 45 | 11 | 0 | 7 | 8 | 11 | 16 |
| | CHD | 0 | 1 | 1 | 4 | 6 | 11 | 0 | 0 | 2 | 2 | 2 | 0 |
| Total | ALL | 124 | 29 | 36 | 62 | 103 | 137 | 50 | 6 | 20 | 49 | 46 | 94 |
| | CHD | 1 | 4 | 10 | 20 | 40 | 37 | 1 | 1 | 5 | 15 | 10 | 15 |

ALL = Total recorded deaths
CHD = Coronary heart disease deaths

Table 4.7
Summary of the recorded deaths studied by hospitals, age and gender in 1989 and 1990.

| Hospital | | Male | | | | | | Female | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
| DCH | ALL | 72 | 16 | 29 | 40 | 66 | 63 | 19 | 7 | 11 | 31 | 30 | 53 |
| | CHD | 1 | 3 | 13 | 18 | 28 | 23 | 1 | 0 | 3 | 14 | 8 | 15 |
| GCH | ALL | 49 | 7 | 13 | 35 | 64 | 59 | 35 | 4 | 11 | 20 | 28 | 52 |
| | CHD | 0 | 1 | 2 | 7 | 22 | 9 | 1 | 1 | 1 | 1 | 7 | 6 |
| GGH | ALL | 27 | 1 | 2 | 5 | 10 | 18 | 11 | 3 | 2 | 4 | 5 | 21 |
| | CHD | 0 | 1 | 1 | 3 | 7 | 10 | 0 | 0 | 1 | 3 | 4 | 9 |
| JGH | ALL | 12 | 3 | 4 | 6 | 7 | 16 | 4 | 0 | 0 | 6 | 3 | 6 |
| | CHD | 0 | 0 | 1 | 1 | 3 | 6 | 0 | 0 | 0 | 1 | 0 | 1 |
| KKH | ALL | 71 | 17 | 19 | 33 | 34 | 72 | 17 | 4 | 9 | 10 | 20 | 27 |
| | CHD | 0 | 3 | 2 | 8 | 16 | 18 | 0 | 1 | 2 | 3 | 4 | 2 |
| Total | ALL | 231 | 44 | 67 | 119 | 181 | 228 | 86 | 18 | 33 | 71 | 86 | 159 |
| | CHD | 1 | 8 | 19 | 37 | 76 | 66 | 2 | 2 | 7 | 22 | 23 | 33 |

ALL = Total recorded deaths
CHD = Coronary heart disease deaths

**Table 4.8**
**The distribution of deaths at King Fahd Hospital Al-Hofof, by gender and group during 1989 and 1990.**

| GROUP | 1989 deaths | | 1990 deaths | |
|---|---|---|---|---|
| | Total | CHD | Total | CHD |
| URBAN: | | | | |
| Male | 145 | 50 | 121 | 39 |
| Female | 100 | 31 | 97 | 30 |
| Total | 245 | 81 | 218 | 69 |
| RURAL: | | | | |
| Male | 78 | 29 | 72 | 29 |
| Female | 62 | 19 | 44 | 11 |
| Total | 140 | 48 | 116 | 40 |
| BEDOUIN: | | | | |
| Male | 16 | 2 | 15 | 3 |
| Female | 6 | 1 | 7 | 0 |
| Total | 22 | 3 | 22 | 3 |
| OVERALL: | | | | |
| **Male** | **239** | **81** | **208** | **71** |
| **Female** | **168** | **51** | **148** | **41** |
| **Total** | **407** | **132** | **356** | **112** |

Table 4.9
**The predicted proportion of CHD as a percentage of total deaths displayed by age, gender, season and hospital.**

| AGE GROUP/MONTHS | HOSPITALS | | | |
|---|---|---|---|---|
| | GCH, KKH, JGH | | DCH | |
| | Male | Female | Male | Female |
| < 35 | | | | |
| Summer | 0.7 | 0.4 | 1.5 | 0.1 |
| Winter | 1.1 | 0.6 | 2.3 | 1.4 |
| 35-54 | | | | |
| Summer | 15.9 | 9.9 | 28.4 | 18.8 |
| Winter | 22.5 | 14.4 | 37.8 | 26.1 |
| 55-74 | | | | |
| Summer | 26.0 | 17.0 | 42.4 | 30.0 |
| Winter | 35.0 | 24.0 | 53.0 | 39.7 |
| 75 & + | | | | |
| Summer | 18.3 | 11.6 | 32.0 | 21.5 |
| Winter | 25.6 | 16.7 | 41.9 | 29.6 |

Table 4.10
**CHD PM as a percentage of total deaths reported in the UK and PM as recorded by this study in four Saudi hospitals.**

| AGE GROUP | Saudi Arabia (PM) | | | United Kingdom (PM) | | |
|---|---|---|---|---|---|---|
| | Male | Female | Overall | Male | Female | Overall |
| <35 | 1.1 | 0.6 | **0.9** | 1.3 | 0.7 | **1.1** |
| 35-44 | 15.1 | 9.5 | **12.5** | 20.7 | 5.2 | **14.7** |
| 45-54 | 24.2 | 15.9 | **21.0** | 33.2 | 11.2 | **24.7** |
| 55-64 | 29.1 | 19.6 | **26.0** | 36.3 | 20.3 | **30.1** |
| 65-74 | 34.7 | 24.0 | **32.0** | 34.3 | 26.5 | **31.1** |
| 75 & + | 23.7 | 15.5 | **21.4** | 27.5 | 24.5 | **25.7** |

1= Data for Saudi Arabia represent year 1989 and 1990.
2= Data for United Kingdom represent year 1990.

Table 4.11
The expected CHD mortality for Saudi by age and gender standardise UK PM.

| Gender | | Age group | | | | | |
|---|---|---|---|---|---|---|---|
| | | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
| Males | *CHD* | 1 | 8 | 19 | 37 | 76 | 66 |
| | *Expected CHD* | 3.10 | 9.12 | 22.27 | 43.19 | 62.00 | 62.60 |
| Females | *CHD* | 2 | 2 | 7 | 22 | 23 | 33 |
| | *Expected CHD* | 0.58 | 0.94 | 3.68 | 14.41 | 22.79 | 38.89 |
| Both gender | *CHD* | 3 | 10 | 26 | 59 | 99 | 99 |
| | *Expected CHD* | 3.52 | 9.10 | 24.74 | 57.24 | 82.93 | 99.35 |

Table 4.12
The PMR for Saudi by age group and gender (comparing to the UK).

| Gender | Age group | | | | | |
|---|---|---|---|---|---|---|
| | < 35 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
| Males | 32.24 | 87.72 | 85.31 | 82.24 | 122.58 | 105.44 |
| Females | 347.32 | 212.83 | 189.99 | 152.64 | 100.90 | 84.86 |
| Both gender | 85.16 | 109.83 | 105.10 | 103.08 | 119.38 | 99.65 |

Chapter Five

# EXPERIMENTAL WORK: FAT AND FATTY ACID PROFILE

## 5.1 MATERIALS AND METHOD

5.1.1 The Aims
5.1.2 Sample Collection
5.1.3 Laboratory Procedures
5.1.4 Fatty Acid Values

## 5.2 RESULTS AND DISCUSSIONS

5.2.1 Study Outcomes
5.2.2 Food Analysis
5.2.3 Study Comparisons
5.2.4 Fat and Fatty Acids

Section 5.1

# MATERIALS AND METHODS

## 5.1.1 The Aims

The role of dietary fatty acids (FA) and their influence on the plasma FAs and serum lipid levels is well established (reviewed in Chapter 2). In SA, there is a paucity of published information concerning the fat content and FA profile for foods. Furthermore, the calculation of FA profile from the food tables (Musaiger & Al-Dallal, 1985), used in this study was not possible as values were not available. In addition, there is no such information available in this respect from neighbouring countries. Therefore, it was necessary for this study to analyses a number of foods and commercially available fats/oils to provide the first data on FA profiles. Thus, foods identified in the food consumption survey (i.e. the first study) as significant sources of oils/fats in the EP diet were sampled, and brought over to the UK for the analysis (Table 5.1). The FA profile identified in this study will be used to calculate the FA profile in the Saudi diet. For the purpose of this study, the food analysed here was limited on raw foods.

## 5.1.2 Sample Collection

Samples of foods were collected at the point of sale by visiting various areas. The samples of each item were analysed for (1) grammes of fat/100g of food; (2) the FA profile and hence the contribution of SFA and USFA; and (3) the P/S ratio.

The samples underwent through four stages of handling procedures. Great care was taken to avoid/minimize food spoilage, as a result of chemical reaction involved in the processes of aging and decay, through the action of microorganisms, or through a combination of both, until they are ready for laboratory analyses.

***a. First stage involved collecting the samples from the survey area***

The food collected is divided into two categories:

1. ***Fresh food***, which included meat, chicken and milk, bought from the local butchers. In SA animals are slaughtered in abattoirs under government supervision and sold at the same day. A resident vet ensures that the meat is fit for consumption before dispatch.

   The samples purchased were immediately housed in an ice-cooled food container and transferred to freezers within 2 hours of sampling. The samples were then stored in the freezer. Milk was collected from the local farm early in the morning, directly after the cows were milked. The milk was immediately labelled and bottled and in a similar way.

2. ***Manufactured food***, included chicken, processed milk, cheese, butter and oils. Frozen chickens were bought from the local supermarket and transported in cooled food containers for storage within 2 hours. Processed milk and cheese were also bought from the supermarket and stored at the home refrigerator. The temperature in such refrigerator is usually 5°C, which was judged to be sufficient to chill the food and maintain the quality of the food prier to analysis.

***b. Second stage involved the transport of the collected sample from SA to London***

During transport the samples were kept cold to maintain the original nature of the samples. The samples were stored in ice-cooled containers and during the flight from SA to UK the containers were kept in the aircraft refrigerators. On arrival, a substantial amount of ice was still present in the containers which indicates that condition in which the samples were transported was satisfactory.

***c. Third stage involved analysing the samples***

In London the samples were immediately stored in refrigerate/freezer, in the same manner as in the first stage, until they were analysed. In the analyses we began with

the milk, because of its short storage life. Then meat and chicken were analysed followed by cheese. Finally, oils and butter were analysed.

***d. Fourth stage involved fat and fatty acids extraction***

Laboratory analyses in the University of North London.

## 5.1.3 Laboratory Procedures

### 5.1.3.1 Choice of Analytical Method

***a. Determination of oil/fat***

Total fat contents for the foods were determined by the approbate method recommended for each food (Egan et al., 1990). The Werner-Schmid and Rose-Gottlieb for dairy products and the method described by Society for Analytical Chemistry (SAC, 1974) for meat and chicken. These method are known to give closet to total fat extractions (James 1995; Egan et al., 1990). The methods procedures are given in full detail in the Appendix N. Werner-Schmid method involves treatment of the food with hot concentrated hydrochloric acid to release fat bound to protein. Followed by extraction of the fat with ether or diethyl ether and petroleum ether. The solvent is then removed and fat residue weighed. Rose-Gottlieb method involves dissolution of non-fat solid in ammonia rather than the acid used in Werner-Schmid method. The fat is then extracted using solvents similar to those used in Werner-Schmid method.

***b. Determination of fatty acids profile***

Gas-liquid chromatography (GLC) was used for the determination of component FAs. The GLC used was a Carlo Erba (HRGC 5160 Mega Series, NFC-500) gas chromatography occupied with flame ionisation detector. Table 5.2 shows the GLC conditions employ in the analyses.

The extracted fat was saponified and the FAs were converted into their methyl esters according to Christie (1982): 10 to 50mg of oil was dissolved in 1ml dry toluene in a stoppered tube, 2ml of 0.5N sodium methoxide in anhydrous methanol was added

and the tube heated in a water bath at 50°C for 15 minutes. The contents were cooled and neutralised with 0.1 ml glacial acetic acid. About 5ml distilled water was added and the esters extracted twice, each time with 5ml of hexane. The hexane extract was separated, dried over anhydrous sodium sulphate, filtered and finally the solvent was removed under reduced pressure on a rotary film evaporator.

Methods used are routinely operated in the laboratory which participates in the FA profiles scheme to validate its analytical methods by compiling sample of known composition.

### 5.1.3.2 Sample Preparation

The whole of each sample was completely homogenised to ensure that sample from the prepared homogenised could be taken to be representative of the whole. Particular car was taken to avoid fat separation. Subsamples were taken for fat determinations. The remain of the homogenise having return under refrigeration until satisfactory result for that sample has been obtained.

### 5.1.3.3 Analysis and Reproducibility

Fat determination by the most appropriate method was carried out by triplicate on each sample. Provided the result a greed within $\pm$ 5%, the determination was accepted and the average included. Where the disagreement was great than $\pm$ 5%, the determination was repeated. In order to evaluate day to day variability, the same sample was analysed on three separate occasions to confirm that the results are consistent.

The GCL and analytical conditions used in fatty acid methyl esters (FAME) profile determinations were contributed by the use of standard mixture of FAMEs. Individual esters was shown to have characteristic retention times which allowed the identification of the various peaks. At the time of the work the GCL was in routine use for FA determinations so that the identification of the peaks was largely routine.

### 5.1.4 Fatty Acid Values

The values of total FAs in the fat were calculated using conversion factors used by Paul and Southgate (1992). These conversion factors applied were the following:

| | |
|---|---|
| Milk and milk products | 0.945 |
| Poultry | 0.945 |
| Lamb | 0.910 |
| Fats and oils | 0.956 |

Evaluation of the data obtained from the analysis was calculated using the computer programme of Salford University "MICRODIET" system, 1988, Mark 7.10. The data calculated by experiment were fed into the programme and used to calculate FA availability, using the same technique as in the first study. The data was expressed in three groups of FAs; saturated, PUSFA and MUSFA.

**Table 5.1**

**Foods analysed for their fat content and fatty acids profile**

| CHEESE | MILK and DIARY products | MEATS | OILS and FATS |
|---|---|---|---|
| La vache quirit, cream | Sheep, fresh | Chicken, fresh | Al-Arabi (palm) |
| La vache quirit, spread | Goat, fresh | Chicken frozen (Radwa, Saudi brand) | Nakheel (palm) |
| Kraft, cream | Cow, fresh | Chicken, frozen (Doux, french brand) | Mazola (corn) |
| Kraft, slices | **PROCESSED MILK:** | Lamb, fresh | Afia (corn) |
| Kraft, cheddar | Um Al-Hiaran, camel | | Sasso (olive) |
| Puck, toast slices | Asafi | | Al-Wazir (olive) |
| Puck, burger slices | Nadic | | Altayeb (olive) |
| Puck, cream | Nada | | Golden chair (ghee) |
| Puck, danish mazzarel | Al-Matrood | | Danish Lurpak (butter) |
| Al-Marai, burger slices | Yogurt | | |
| Al-Marai, cream | | | |
| Al-Marai, slices | | | |
| Anchor, slices | | | |
| Kiri, cream | | | |
| Farmer, cream | | | |

**Table 5.2**

**The gas-liquid chromatography conditions**

| | |
|---|---|
| Column (silicone) | 50m * 0.32mm i.d. |
| Packaging | SP 2340 |
| Carrier gas | Hydrogen |
| Temperature (oven) | Initial 80°C<br>Increasing 15°C/min<br>Final 180°C |
| Detector: flame ionisation detector (FID) | 300°C |
| Injector (Direct on column) | 90°C |

Section 5.2

# RESULTS AND DISCUSSIONS

## 5.2.1 Study Outcomes

One of the problems which faced this study was the use of food tables from another country. In addition, this table was incomplete for many nutrients and in particular for fatty acids (FAs). The fat and FA availability as shown in this chapter, were derived from the experimental work carried out in this study, and the following discussion regarding the FAs was also based upon the analysis carried out.

In the earlier study reported in Chapter 3, fat and other nutrient available for consumption investigated were based on food consumption tables on used in Bahrain. The values for fat available were updated as a result of this evaluation of the fat content of these foods contributing as significant proportion of the total fat available in the Saudi diet.

## 5.2.2 Food Analysis

Appendix O shows the FA composition for the foods analysed (fatty acids g/ 100g total fatty acids). The FA composition for the foods analysed (fatty acids g/ 100g food) is shown in Appendix P.

Table 5.3 shows the average fat content for the foods analysed. Table 5.4 shows FA profile and P/S ratio for the foods analysed (fatty acids are given as percentage of all fatty acids).

**1. Milk and Dairy Products**

*Milk*: All dairy products analysed were Saudi manufactured (see section 2.1.6). On average, total fat for cows milk was 3.3%, close to the figure 3.5%, in Bahrain food

tables used in this study (Table 5.3). The corresponding figure for the UK is 3.8%. Goats milk total fat was 2.5%, this was approximately half that from the UK and two thirds less than the Bahrain food tables.

Saturates in processed cows milk were found be higher than unprocessed milk, by about 17% of total FA. This slightly surprising result could be due to the difference in the type of food fed to the animals used for small scale fresh milk production compared to that fed to intensively housed animals used to produce milk for processing. It is also possible that different breeding cows were used for these operations. The major USFA of all type of milk analysed, but with different percentage, was oleic FA. It is understandable that the fat from every animal has a slightly different FA profile. In comparison with the UK, the P/S ratio for cows milk was higher in this study by 0.01, whereas it was the same for goats milk (Paul & Southgate, 1988). This differences would obviously cause significant errors in the estimation of fat availability as calculated in Chapter 3.

*Cheese*: Several type of cheese were analysed in this study, as cheese was so popular in the community, and is taken as much as twice a day in the breakfast and in the dinner (see Chapter 3). The majority of the cheeses analysed were imported and some were Saudi manufactured. There was no difference in total fat content between the Saudi cheese manufactured and the imported one. For example, "Al-Marai" burger, slices cheese (Saudi brand) total fat was 22%, similar to the "Puck" imported from West Germany. The correspondent figure in this type of cheese, from Bahrain food tables was higher by 3.4%. Other type of cheeses such as cream cheese, were almost the same with the Bahrain food tables. The total fat content among cheese products ranged from 19.5% to 33%. "Puck" cream cheese (31%) and "kraft" cream cheese (31%) had the highest fat content, whereas white cheese (19.5%) had the lowest.

On average fat in cheese contained 69% saturates of total FAs, the major SFAs were found to be palmitic, myristic, and stearic. Unsaturate represent 31% of total

FA, of which 29.9% was monounsaturates and 1.6% polyunsaturated, the major USFA was oleic. The P/S ratios were varied from 0.01 to 0.04.

**2. Meat and Poultry**

***Chicken***: Three types of chickens were analysed; local market, Saudi manufacture (Radwa brand), and imported chicken (Doux brand, French). "Doux" chicken was popular among the Bedouin households. One reason for that may be due to the unavailability of fresh chicken to this group as is the case among the urban and rural area. There were no differences between the two type of Saudi chicken in total fat content. But there was a difference in fat content between the Saudi chicken and the imported type, by almost 6%, the latter being higher. A marked difference between chicken from Bahrain food tables and the analysed chicken was found, the difference was on average 7% higher in the chicken analysed in this study. It is possible that the chickens analysed for the Bahrain food tables were indigenous, from chickens rather than the battery chickens encountered in this study. Difference in methodology (see later) may also contribute to this difference. The correspondent figures from the UK (17.6%) were comparable to the analysed imported chicken. Fresh chicken contained more saturates (36%) than frozen chicken, the major SFA, was found to be palmitic and stearic. The major USFA was oleic and linoleic The P/S ratio for imported chicken was much higher than the local chicken.

***Lamb***: As discussed in Chapter 3, lamb is preferred to beef among this population. The average total fat content in lamb was 25%, comparable, to the figures from Bahrain food tables. In the UK, lamb contains 28% of total fat. On average, lamb contained 52.4% saturates; monounsaturates and polyunsaturated were 46% and 1.7%, respectively. The major SFAs were palmitic, stearic and myristic, the major monounsaturate was oleic and polyunsaturated was linoleic

**3. Oils and fats**

Ten brands, of vegetable oils and ghee and butter commonly consumed were analysed

for their FA profile. Two brands were Saudi manufactured; Al-Arabi and Nakheel brand, and the rest were imported. Table 5.4 shows the average FS profile for the oil analysed.

Butter has been demonstrated to be neutral on serum cholesterol and HDL cholesterol in man (Hayes et al., 1991). As shown in Chapter 3, the consumption of butter among this population was lower than the UK. Also other characteristics for this population (regarding this respect) in comparison with the UK, was that they did not consume margarine, whereas, in the UK it comes as part of their daily meal. Margarine is a major source of dietary trans acid in terms of contents and amounts. This type of isomer is related to pathogenesis of atherosclerosis (see Chapter 2). FA analyses for the most popular ghee and butter in this study, shows the average SFA was 69% of total FAs. It also contained trans MUSFA up to 3.7% and P/S ratio of 0.02.

### 5.2.3 Study Comparisons

Table 5.5 shows a comparison of the percentage of total fat in chicken, lamb and milk found in this study, Bahrain and the UK. Bahraini chicken has the lower percentage of fat (5.8%) following by the present study 10.7%, and the UK having the highest at 17.7%. Whereas, total fat from the imported and local Saudi chicken were similar to the UK chicken. The UK lamb was higher than the present study and the Bahraini lamb by about 5%, whereas the present study was the lowest (25%).

Comparisons between the major FAs was also made between SA (present study) and the UK (Table 5.6). The overall data shows some similarity, however, there are some marked difference in the FAs observed as follows:

1. ***Chicken***; palmitic and stearic acids were lower by 1.2 and 2.4%, respectively, in SA chicken than in UK food tables. Oleic and linoleic acid were higher by 5.5 and 3.4%, respectively, in SA chicken than in the UK.

2. ***Lamb***; overall, there was not much difference between lamb in SA and the UK except palmitic was higher in SA lamb by 2.8%. Whereas, stearic was higher in the UK (1.7%) than SA. Apart from that, linolenic acid in the UK has 2.5% while SA lamb had no detectable linolenic acid.

3. ***Milk***; the marked difference was the palmitic acid. The content of palmitic acid in cows milk in SA was more than that in the UK, with 9.1%. Palmitoleic, oleic and linoleic acids were higher in Saudi's cows milk than the UK milk, with up to 1.7%. Whereas myristic, stearic and linolenic acids were lower, with up to 1.4%.

4. ***Oils***; with the palm and olive oils there was not much difference between the two countries, except that linoleic acid in the Saudi palm oil was higher by almost 3% than the UK food table. However, a marked difference was observed among the corn and soya bean oils content. In SA, linoleic acid content in corn and soyabean oils were higher than the UK, with 9.8 and 5.3%, respectively. But oleic acid was less in SA, with 3.4 and 3.3%, respectively. Palmitic acid in corn oil was also less in SA (by 2.3%) than the UK.

This difference in fat and FA profile may, in part, be due to the analytical method used to determine the fat. For example, fat contents reported in the Bahrain food tables were determined by the Soxhlet method, whereas, the method used to obtain the by Paul and Southgate was "the standard procedure for extracting fat in each class of foods stuffs", generally involving acid hydrolysis in the cases discussed above. In the present study, acid hydrolysis procedures described by Egan et al., (1990) were used (Appendix N). FA profile varies according to the diet fed to the animal. It also varies between plant species and depends on the environmental conditions.

### 5.2.4 Fat and Fatty Acids Availability

This section considers the fat availability and the percentage of FA in the Saudi

household diet for adult males from the different groups studied. These calculations were from foods analysed in this study.

Table 5.7 shows the distribution of the average daily availability of fat recorded by the subjects. In comparison with Table 3.31 in Chapter 3, the results indicated that the calculated fat availability for adult males was higher (up to 11.3%) than that calculated from the food consumption table used in Bahrain. It is appears that the study of fat availability discussed in chapter 3 has underestimated the actual fat availability estimated, and so consequently will under estimate the actual energy availability as well. Obviously, there is a difference between the fat content of individual foods from different countries and since the foods analysed in this study were those making average contribution of the fat content of the Saudi diet the higher fat content as reported in this study are sufficient to explain the apparently increased fat availability compared to Chapter 3. This shows that food composition tables used in one country are not applicable to other countries, as they may under-or-overestimate the actual food intake. This highlight the need for set up food composition table for use in SA containing raw foods as well as native Saudi dishes.

Table 5.8 summarises FA availability, as shown in the table, the percentage of FAs per adult males per day, varied amongst the groups studied. Caution should be observed when comparisons are made between the results in Table 5.7 and Table 5.8. This is because the latter table was based on total fat in a food, whereas, the result in Table 5.8 was based only on FAs. Thus the sum of the FA availability in Table 5.8 is lower than the fat availability in Table 5.7, since the total availability of Fas amount by definition be somewhat less than availability of fat.

As discussed in Chapter 2, the P/S ratio is widely used in relation to the prevalence of CHD in a population. A low P/S ratio in the diet is thought to increase the risk of CHD. In the UK the official advisory committee on Diet and Cardiovascular Disease, Department of Health and Social Security (DHSS, 1984)

specified a P/S ratio of 0.45 as an attainable figure. The results in this study show that the P/S ratio among the urban group was greater than that for the rural group and equal to the target of the British recommendation. Whereas, among the rural group it was below the 0.45 UK target, by 0.06. On the other hand the Bedouin group figures were higher than this, by about 0.13. The recent dietary survey of British adults, shows that the average P/S ratio for men was 0.40 (Gregory et al., 1990). So that our findings shows that the current Saudi diet appears to be closer to the UK target in this respect than the diets in the UK itself.

As expected in all groups the major SFAs were found to be palmitic and stearic. The major nonunsaturated fatty acid was oleic acid. Recent work has suggested that intake of mono-unsaturated FAs may also have a protective effect against CHD.

Table 5.9 shows the major food sources contributing to the fat availability for different groups studied. The major FAs which are thought to have positive or a negative influences on the pathology of CHD incidence have been identified. In the case of palmitic acid, for urban families, 38% of total palmitic acid availability is derived from meat and poultry and 32% from vegetable oil. These figurers were 43% and 27% for rural families and 39% and 42% for Bedouin families, respectively. Obviously, the variation in the proportion of the FA availability by the groups studied is mainly due to the type of food consumed. For example, the results from the first study shows that Bedouin families consume chicken (Doux type), which has a higher P/S ratio than the chicken consumed by the other groups. This may help to explain the apparently high P/S ratio for this groups in this group in the diets as a whole.

The role of dietary FAs and their influence on the plasma FAs and serum total and LDL-cholesterol levels is well established (see Chapter 2). Mortality from CHD increases with increases in the serum total and LDL-cholesterol levels. A considerable body of experimental evidence has shown, that blood cholesterol concentration can be modulated in the individual by modification of the type of dietary FAs and by

medications.

The result from this study shows that fat content and FAs profile of many foods analysed did differ, either relatively or significantly, from the Bahraini food tables used. It would seem that most Saudi foods are higher in fat content than the reference food table from Bahrain and that from the UK, significant of differences in FA profile between SA and the UK were also found. It is therefore becomes apparent that mean fat availability calculated from food tables used in Bahrain in the first study indicate under-reporting. The present results highlights the effect of substituting analysed food from another table used in another countries. This emphasises the need for food composition tables for use in SA. If meaningful estimates of dietary intake in SA are to be obtained. Another important point should be mentioned here, is that ready made foods for native Saudi dishes were not investigated by this study. We are well aware of there important contributions to fat and FA composition in the Saudi diet. But it was not possible for us to carry the analyses as our goal was to investigate the raw foods coming into the house only. Another study is required in this area to investigate these dishes; the ingredients, preparing and cooking method.

Table 5.3

**Average fat content for the foods analysed**

| Food Item | Total Fat |
|---|---|
| **MILK & DAIRY PRODUCTS:** | |
| 1. Cheese | |
| *Cream* | 29 |
| *Slices* | 22.9 |
| 2. Milk (fresh) | |
| *Sheep* | 2.6 |
| *Goats* | 2.5 |
| *Cows* | 3.5 |
| 3. Milk (processed) | |
| *Camels* | 2.6 |
| *Cows* | 3.3 |
| 4. Yogurt | 2.9 |
| **Meat & poultry:** | |
| 1. Chicken | |
| *Fresh* | 10.7 |
| *Frozen (imported)* | 16.4 |
| *Frozen (local)* | 10.7 |
| 2. Lamp | 25 |

**Table 5.4**
**Fatty acid compsition (gram fatty acids/100 gram total fatty acids)**

| Food Item | Saturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 | Total % |
| **MILK & DAIRY PRODUCTS:** | | | | | | | | | | | |
| 1. Cheese | | | | | | | | | | | |
| *Cream* | 0 | 0.3 | 0.7 | 2.5 | 4.2 | 13.5 | 0.9 | 35.6 | 0.6 | 11.4 | 69.7 |
| *Slices* | 0.1 | 0.3 | 0.5 | 2.0 | 4.2 | 13.7 | 1.0 | 34.0 | 0.5 | 12.2 | 68.4 |
| 2. Milk (fresh) | | | | | | | | | | | |
| *Sheep* | 0 | 0 | 0 | 2.2 | 3.2 | 15.0 | 0 | 46.5 | 0 | 4.8 | 71.6 |
| *Goats* | 0.3 | 0.1 | 0.9 | 5.2 | 3.8 | 13.4 | 0.3 | 38.2 | 1.2 | 6.9 | 70.2 |
| *Cows* | 0.1 | 0 | 0 | 0.5 | 1.2 | 6.0 | 2.0 | 28.8 | 1.0 | 5.0 | 44.6 |
| 3. Milk (processed) | | | | | | | | | | | |
| *Camels* | 0 | 0 | 0.1 | 0.1 | 1.1 | 12.2 | 1.2 | 33.8 | 0.7 | 6.8 | 56.1 |
| *Cows* | 0.2 | 0.1 | 0.4 | 1.8 | 2.8 | 10.0 | 0.8 | 35.1 | 0.6 | 10.0 | 61.8 |
| 4. Yogurt | 0 | 0.3 | 0.8 | 2.1 | 3.3 | 11.4 | 1.1 | 32.5 | 0 | 9.7 | 61.2 |
| **Meat & poultry:** | | | | | | | | | | | |
| 1. Chicken | | | | | | | | | | | |
| *Fresh* | 0 | 0 | 0 | 0 | 0 | 0.8 | 0 | 29.9 | 0 | 45.0 | 35.7 |
| *Frozen (imported)* | 0 | 0 | 0 | 0 | 0 | 0.5 | 0 | 20.2 | 0 | 4.7 | 25.5 |
| *Frozen (local)* | 0 | 0 | 0 | 0 | 0 | 0.5 | 0 | 26.3 | 0 | 4.4 | 31.3 |
| 2. Lamp | 0 | 0 | 0 | 0 | 0 | 4.5 | 0 | 27.0 | 1.8 | 19.2 | 52.4 |
| **Oil & fat:** | | | | | | | | | | | |
| 1. Palm oil | 0 | 0 | 0 | 0 | 0.1 | 1.0 | 0 | 40.6 | 0 | 3.4 | 45.1 |
| 2. Corn oil | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11.2 | 0 | 1.5 | 12.8 |
| 3. Olive oil | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12.9 | 0 | 2.3 | 15.2 |
| 4. Ghee | 0 | 0.7 | 0.6 | 2.1 | 3.0 | 11.7 | 0.9 | 34.6 | 0.7 | 13.8 | 68.0 |
| 5. Butter | 0 | 0.4 | 0.6 | 1.7 | 3.8 | 12.4 | 0.8 | 37.0 | 0.6 | 11.4 | 68.7 |

**Table 5.4** *Continued*

| Food Item | Unsaturated | | | | | | | | | | | | P/S ratio |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 | Total % | |
| **MILK & DAIRY PRODUCTS:** | | | | | | | | | | | | | |
| 1. Cheese | | | | | | | | | | | | | |
| *Cream* | 0.3 | 0.5 | 1.4 | 0 | 0.5 | 2.1 | 0.1 | 2.3 | 21.4 | 1.3 | 0.3 | 30.1 | 0.03 |
| *Slices* | 0.3 | 0.6 | 1.5 | 0 | 0.6 | 2.0 | 0 | 2.6 | 22.9 | 0.9 | 0.4 | 31.6 | 0.02 |
| 2. Milk (fresh) | | | | | | | | | | | | | |
| *Sheep* | 0.7 | 01.8 | 0 | 0 | 2.0 | 0 | 0 | 22.0 | 1.8 | 1.8 | 0 | 28.4 | 0.03 |
| *Goats* | 0.4 | 0.7 | 1.7 | 0.4 | 0.7 | 2.8 | 0.4 | 0.9 | 19.4 | 1.9 | 0.6 | 29.8 | 0.03 |
| *Cows* | 0.5 | 0 | 1.7 | 0 | 1.8 | 6.8 | 0.8 | 16.3 | 25.0 | 2.6 | 0 | 55.4 | 0.06 |
| 3. Milk (processed) | | | | | | | | | | | | | |
| *Camels* | 0.4 | 0.9 | 1.4 | 0.3 | 0.5 | 13.7 | 0.5 | 4.0 | 20.9 | 1.3 | 0 | 43.9 | 0.02 |
| *Cows* | 0.1 | 0.3 | 1.2 | 0 | 1.2 | 2.8 | 0 | 2.7 | 26.8 | 3.1 | 0.1 | 38.2 | 0.05 |
| 4. Yogurt | 0.2 | 0.5 | 1.7 | 0.3 | 1.4 | 3.7 | 0.4 | 3.4 | 24.3 | 2.2 | 0.8 | 38.8 | 0.05 |
| **Meat & poultry:** | | | | | | | | | | | | | |
| 1. Chicken | | | | | | | | | | | | | |
| *Fresh* | 0 | 0 | 0 | 0 | 0 | 8.9 | 0 | 0 | 45.0 | 10.4 | 0 | 64.3 | 0.29 |
| *Frozen(imported)* | 0 | 0 | 0 | 0 | 0 | 2.9 | 0 | 0 | 44.9 | 25.6 | 1.2 | 74.6 | 1.05 |
| *Frozen (local)* | 0 | 0 | 0 | 0 | 0 | 7.8 | 0 | 0 | 46.1 | 14.8 | 0 | 68.8 | 0.47 |
| 2. Lamp | 0.7 | 0 | 0 | 0 | 0 | 2.1 | 0 | 2.1 | 41.0 | 1.7 | 0 | 47.6 | 0.03 |
| **Oil & fat:** | | | | | | | | | | | | | |
| 1. Palm oil | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 43.6 | 11.3 | 0 | 54.9 | 0.25 |
| 2. Corn oil | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 26.6 | 59.8 | 0.8 | 87.2 | 4.74 |
| 3. Olive oil | 0 | 0 | 0 | 0 | 0.3 | 0.7 | 0 | 0 | 72.4 | 11.2 | 0.1 | 84.8 | 0.71 |
| 4. Ghee | 0 | 0 | 1.4 | 0 | 0.4 | 2.4 | 0 | 1.9 | 23.8 | 0.9 | 0.6 | 32.0 | 0.02 |
| 5. Butter | 0 | 0 | 1.2 | 0 | 0.4 | 2.3 | 0 | 1.9 | 23.8 | 1.7 | 0 | 31.3 | 0.02 |

**Table 5.5**
**Comparison of total fat from this study with other data form Bahrain and UK (gram fat/100g food)**

| | Chicken | | Lamb | Milk | Oils | | |
|---|---|---|---|---|---|---|---|
| | Local | Imported | | | Palm | Corn | Olive |
| Present study | 10.67-10.73 | 16.40 | 25 | 3.5 | 99.9 | 99.9 | 99.9 |
| Bahrain[a] | 5.8 | | 26.8 | 3 | 99.9 | | 99.9 |
| United Kingdom[b] | 3.5-17.7 | | 30.5 | 3.8 | | | |

**a** = The data were compiled from "Food Composition Tables for use in Bahrain", by Musaiger AO., & Aldallal ZS. 1985, Ministry of Health, Bahrain.

**b** = The data were compiled from McCance and Widdowson's "The Composition of Foods" 5th revised edition, by Paul & Southgate. 1992, London, H.M. Stationary Office.

*Table 5.6*
*Compareson of major fatty acids from this study with British data[a] (gram fatty acids/100 gram total fatty acids)*

| Fatty acids | Chicken | Lamb | Milk | Oils | | | |
|---|---|---|---|---|---|---|---|
| | | | | Palm | Corn | Soyabean | Olive |
| Lauric 12:0 | | | | | | | |
| Present study | 0 | 0 | 2.8 | 0.1 | 0 | 0 | 0 |
| United Kingdom[a] | 0 | 0 | 3.5 | 0.2 | 0 | 0 | 0 |
| Myristic 14:0 | | | | | | | |
| Present study | 0.6 | 4.4 | 10.0 | 1.0 | 0 | 0 | 0 |
| United Kingdom[a] | 1.3 | 5.4 | 11.2 | 1.1 | 0 | 0.2 | 0 |
| Palmitic 16:0 | | | | | | | |
| Present study | 25.5 | 27.0 | 35.1 | 40.6 | 11.2 | 10.8 | 12.9 |
| United Kingdom[a] | 26.7 | 24.2 | 26.0 | 41.5 | 14.0 | 10.0 | 12.0 |
| Stearic 18:0 | | | | | | | |
| Present study | 4.7 | 19.2 | 10.0 | 3.4 | 1.5 | 3.0 | 2.3 |
| United Kingdom[a] | 7.1 | 20.9 | 11.2 | 4.3 | 2.3 | 4.0 | 2.3 |
| Palmitoleic 16:1 | | | | | | | |
| Present study | 6.6 | 2.1 | 4.0 | 0 | 0 | 0 | 1.0 |
| United Kingdom[a] | 7.2 | 1.3 | 2.7 | 0.3 | 0.3 | 0.2 | 1.0 |
| Oleic 18:1 | | | | | | | |
| Present study | 45.3 | 43.1 | 29.5 | 43.6 | 26.6 | 21.7 | 72.4 |
| United Kingdom[a] | 39.8 | 38.2 | 27.8 | 43.3 | 30.0 | 25.0 | 72.0 |
| Linoleic 18:2 | | | | | | | |
| Present study | 16.9 | 1.7 | 3.1 | 11.3 | 59.8 | 57.3 | 11.2 |
| United Kingdom[a] | 13.5 | 2.5 | 1.4 | 8.4 | 50.0 | 52.0 | 11.0 |
| Linolenic 18:3 | | | | | | | |
| Present study | 0.4 | 0 | 0.1 | 0 | 0.8 | 7.1 | 0.1 |
| United Kingdom[a] | 0.7 | 2.5 | 1.5 | 0.3 | 1.6 | 7.4 | 0.7 |

[a] = The data were compiled from Paul & Southgate. McCance and Widdowson's "The Composition of Foods" 4th revised edition, London, H.M. Stationary Office.

**Table 5.7**
**The effects of substituting analysed fat in this study with the calculated fat availability reported in Chapter 3 (gram/male value/day) by gender, age and area**

| | Male age group | | | | Female age group | | | |
|---|---|---|---|---|---|---|---|---|
| | >=18 | 11-17 | 5-10 | 1-4 | >=18 | 11-17 | 5-10 | 1-4 |
| **All population** | **101** | **90** | **77** | **56** | **75** | **81** | **61** | **46** |
| **Ana/Cal %** | **+ 7.5** | **+ 8.4** | **+ 8.5** | **+ 9.8** | **+ 8.7** | **+ 8** | **+ 8.9** | **+ 9.5** |
| Urban | 91 | 81 | 69 | 50 | 68 | 73 | 55 | 41 |
| Ana/Cal % | + 8.3 | + 8 | + 7.8 | + 8.7 | + 9.7 | + 9 | + 7.8 | + 7.9 |
| Rural | 111 | 99 | 84 | 61 | 82 | 89 | 66 | 50 |
| Ana/Cal % | + 6.7 | + 6.5 | + 6.3 | + 7 | + 6.5 | + 7.2 | + 4.8 | + 6.4 |
| Bedouin | 148 | 131 | 112 | 81 | 109 | 118 | 89 | 66 |
| Ana/Cal % | + 11.3 | + 11 | + 10.9 | + 11 | + 11.2 | + 11.3 | + 11.3 | + 10 |

*Ana/Cal %* = Analysed fat in this study/fat availability calculated from Bahrain food compassion table (see Table 3.31).

Table 5.8
Fatty acids composition in the average Saudi household diet (gram/adult males/day).

| Fatty acids | Urban | | Rural | | Bedouin | | All | |
|---|---|---|---|---|---|---|---|---|
| | g/d | % | g/d | % | g/d | % | g/d | % |
| TOTAL | | | | | | | | |
| Saturated | 33.7 | 41.3 | 40.3 | 41.3 | 49.3 | 36.9 | 36.7 | 40.7 |
| Unsaturated | 48.0 | 58.7 | 57.3 | 58.7 | 84.2 | 63.1 | 53.6 | 59.3 |
| MUSFA | 32.8 | 40.1 | 41.5 | 42.5 | 55.6 | 41.6 | 37.0 | 41.0 |
| PUSFA | 15.2 | 18.6 | 15.8 | 16.2 | 28.6 | 21.4 | 16.6 | 18.4 |
| P/S ratio | 0.45 | | 0.39 | | 0.58 | | 0.45 | |
| SATURATED: | | | | | | | | |
| Lauric 12:0 | 0.8 | 2.3 | 0.6 | 1.5 | 0.9 | 1.8 | 0.7 | 2.0 |
| Myristic 14:0 | 2.8 | 8.2 | 3.3 | 8.2 | 3.0 | 6.2 | 2.9 | 7.9 |
| Palmitic 16:0 | 22.4 | 66.3 | 26.0 | 64.6 | 34.6 | 70.1 | 24.4 | 66.4 |
| Stearic 18:0 | 6.7 | 20.0 | 8.8 | 21.8 | 9.1 | 18.5 | 7.4 | 20.3 |
| Others | 1.1 | 3.3 | 1.5 | 3.8 | 1.7 | 3.4 | 1.3 | 3.4 |
| UNSATURATED: | | | | | | | | |
| *a. Mono-unsaturated* | | | | | | | | |
| Palmitoleic 16:1 | 1.9 | 3.9 | 2.5 | 4.3 | 2.9 | 3.4 | 2.1 | 3.9 |
| Oleic 18:1 | 30.5 | 63.6 | 38.3 | 67 | 52.2 | 62.0 | 34.4 | 64.2 |
| Others | 0.4 | 0.8 | 0.7 | 1.2 | 0.5 | 0.6 | 0.5 | 0.9 |
| *b. Poly-unsaturated* | | | | | | | | |
| Linoleic 18:2 | 14.69 | 30.6 | 15.1 | 26.3 | 27.9 | 33.1 | 16.1 | 29.9 |
| Linolenic 18:3 | 0.4 | 0.8 | 0.5 | 0.8 | 0.7 | 0.8 | 0.4 | 0.8 |
| Others | 0.1 | 0.3 | 0.2 | 0.4 | 0.1 | 0.1 | 0.2 | 0.3 |

*Table 5.9*
***The major foods contribute to selected fatty acids, as percentage, in Saudi diet.***

| Fatty acids | Area type | | | | | | All populations | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | |
| | Food items | % | Food items | % | Food items | % | Food items | % |
| Lauric 12:0 | Milk & milk products | 35.72 | Milk & milk products | 76.71 | Milk & milk products | 49.01 | Milk & milk products | 44.96 |
| | Coconut | 32.62 | Butter | 21.61 | Coconut | 48.44 | Coconut | 28.36 |
| | Butter | 30.14 | | | Butter | | Butter | 25.01 |
| Myristic 14:0 | Milk & milk products | 33.04 | Milk & milk products | 45.94 | Milk & milk products | 47.25 | Milk & milk products | 37.88 |
| | Meat | 28.55 | Meat | 34.96 | Meat | 33.75 | Meat | 30.76 |
| | Butter | 27.34 | Butter | 12.75 | Vegetable oil | 8.59 | Butter | 20.73 |
| | Vegetable oil | 4.81 | Vegetable oil | 3.52 | Chicken | 5.60 | Vegetable oil | 4.86 |
| | | | | | Coconut | 4.72 | | |
| Palmitic 16:0 | Vegetable oil | 31.88 | Vegetable oil | 26.45 | Vegetable oil | 42.37 | Vegetable oil | 31.99 |
| | Meat | 21.79 | Meat | 28.76 | Meat | 18.44 | Meat | 23.03 |
| | Chicken | 15.95 | Milk & milk products | 15.99 | Chicken | 20.72 | Chicken | 16.12 |
| | Milk & milk products | 11.14 | Chicken | 13.97 | Milk & milk products | 11.40 | Milk & milk products | 12.55 |
| | Butter | 10.07 | Butter | 4.87 | Eggs | 2.14 | Butter | 7.41 |
| | Eggs | 4.36 | Eggs | 4.37 | | | Eggs | 4.06 |
| Stearic 18:0 | Meat | 50.81 | Meat | 57.23 | Meat | 49.03 | Meat | 52.34 |
| | Milk & milk products | 11.71 | Milk & milk products | 16.19 | Milk & milk products | 15.52 | Milk & milk products | 13.79 |
| | Butter | 10.32 | Chicken | 7.89 | Vegetable oil | 15.84 | Butter | 7.49 |
| | Vegetable oil | 10.12 | Vegetable oil | 8.05 | Chicken | 14.93 | Vegetable oil | 10.24 |
| | Chicken | 10.08 | Butter | 4.46 | Eggs | 2.63 | Chicken | 10.06 |
| | Eggs | 4.69 | Eggs | 4.20 | | | Eggs | 4.31 |
| Palmitoleic 16:1 | Chicken | 45.92 | Chicken | 35.65 | Chicken | 60.52 | Chicken | 45.07 |
| | Meat | 21.44 | Meat | 31.61 | Meat | 20.21 | Meat | 23.78 |
| | Milk & milk products | 14.84 | Milk & milk products | 19.03 | Milk & milk products | 14.97 | Milk & milk products | 13.26 |
| | Butter | 9.01 | Eggs | 6.77 | Eggs | 3.80 | Butter | 6.44 |
| | Eggs | 7.62 | Butter | 3.86 | | | Eggs | 6.89 |

Table 5.9 *Continued*

| Fatty acids | Area type | | | | | | All population | |
|---|---|---|---|---|---|---|---|---|
| | Urban | | Rural | | Bedouin | | | |
| | Food items | % | Food items | % | Food items | % | Food items | % |
| Oleic 18:1 | Vegetable oil | 31.64 | Vegetable oil | 31.02 | Vegetable oil | 42.66 | Vegetable oil | 33.11 |
| | Meat | 24.30 | Meat | 29.84 | Meat | 18.59 | Meat | 24.88 |
| | Chicken | 21.43 | Chicken | 17.39 | Chicken | 25.15 | Chicken | 20.94 |
| | Milk & milk products | 6.67 | Milk & milk products | 7.02 | Milk & milk products | 7.15 | Milk & milk products | 7.61 |
| | Butter | 5.13 | Eggs | 4.44 | Eggs | 2.12 | Butter | 3.65 |
| | Eggs | 4.78 | Butter | 2.3 | | | Eggs | 4.31 |
| Linoleic 18:2 | Vegetable oil | 66.51 | Vegetable oil | 60.73 | Vegetable oil | 70.98 | Vegetable oil | 66.01 |
| | Chicken | 13.79 | Chicken | 13.73 | Chicken | 14.59 | Chicken | 13.91 |
| | Rice | 4.49 | Rice | 11.37 | Rice | 6.99 | Rice | 6.40 |
| | Bread | 3.57 | Bread | 3.29 | | | Bread | 3.09 |
| Linolenic 18:3 | Vegetable oil | 31.01 | Vegetable oil | 22.63 | Vegetable oil | 35.16 | Vegetable oil | 29.47 |
| | Chicken | 20.10 | Milk & milk products | 21.90 | Chicken | 22.61 | Chicken | 19.43 |
| | Milk & milk products | 11.88 | Chicken | 16.02 | Milk & milk products | 18.37 | Milk & milk products | 15.44 |
| | Bread | 8.73 | Meat | 13.29 | Flour | 7.11 | Bread | 6.92 |
| | Flour | 5.66 | Bread | 6.18 | | | Flour | 4.83 |

Chapter Six

# GENERAL DISCUSSIONS AND CONCLUSIONS

Section 6.1

# SUMMARY OF THE RESULTS

## 6.1.1 First Study (chapter 3)

The investigations carried out during the first study of this work revealed important conclusions, which have social implications for the EP population. These are:

1. The pattern of food availability revealed the following important findings:
   a. Households with higher income had lower food availability.
   b. Households who usually buy their food in bulk had higher food availability.
   c. Households with head of the household with a higher education level have a lower energy purchase over the week considered.

2. The meal pattern data has indicated that three-daily-meals were common amongst all the groups studied. However since this was qualitative data, further investigation to determine the composition of the food consumed was highly needed.

3. The diet in the area is cereal based, the main staples being rice, accompanied by meat, fruit and vegetables. Vegetable oil was commonly used for cooking etc.

4. Fresh foods such as meat, fruit and vegetables were highly used.

5. The study demonstrated that the consumption of five traditional items (rice, meat, chicken, vegetable soup and dates) were constant in all groups studied.

## 6.1.2 Second Study (chapter 4)

The investigations carried out in the second study has produced the following results:

1. It is revealed that CHD PM as possible cause of death was high among the non-Bedouin in comparison with the Bedouin population.

2. The proportion of CHD recorded deaths PM among males was higher than females.

3. The proportion of recorded CHD deaths (PM) generally increased, with increasing age, (except for the very old).

4. CHD PM is higher in winter.

5. Observed CHD PM in this study indicated a similar pattern to the UK CHD PM data.

### 6.1.3 Third Study (chapter 5)

The investigation of fat and fatty acid profile obtained from the experimental work shows the following important points:

1. **Fatty acid availability (gram/adult/day):**
   a. SFAs was lower among the Bedouin group than the urban and rural groups, by about 4.5%.
   b. In comparison, lauric and myristic acids were lower among the Bedouin group than the urban and the rural groups.
   c. The palmitic acid was higher among the Bedouin group than the urban and the rural groups.

2. **Fat and fatty acid profile of the foods analysed:**
   **a. CHEESE:**
   1. Some type of cheese contains up to 33% of total fat.
   2. Some type of cheese contains up to 8% *cis* MUSFAs.
   3. The P/S ratio ranging from 0.01 to 0.04.
   4. The major FAs of all cheese were: myristic, palmitic, stearic and oleic.

   **b. MILK:**
   1. Sheep, goats and camels milk were low in total fat (around 2.5%) comparable to cows milk 3.5%.
   2. "Nada" and "Al-Matrod" types were the highest of SFA (up to 2.3% of total FA).

3. The rest of the milk types were on the same levels of SFA (less than 2%).
4. The P/S ratio was high among cows milk more than 0.05, in comparison to "Al-M Matrod" type (0.03).
5. The camels milk (Um Al-Hiaran) type was the lowest P/S ratio (0.02).

**c. POULTRY:**

1. Fat contents of imported chicken (Doux brand) was higher than locally produced by almost 6%.
2. "Doux" brand was also the highest of the P/S ratio (1.05).

**d. OIL AND FAT:**

1. "Nakheel" and "Al-Arabi" palm oil types were the highest of SFA (up to 47% of total FA).
2. "Afia" oil type was the lowest SFA (12.3% of total FA) and also contains the highest MUFAs (81% of total FA).
3. "Nakheel" and "Al-Arabi" palm oil types were the lowest P/S ratio (down to 0.22).
4. "Mazola" and "Afia" corn oil and "Calaira" soya bean oil were the highest P/S ratio up to 5.0.

Section 6.2

# GENERAL DISCUSSION

The Saudi government has for many years led a successful policy to improve the standard of living and well-being of their population. But despite this successful policy, the approach on health awareness and education is very weak and almost non-existent, i.e. what is needed is a strong and effective prevention policy. This has led to the lack of epidemiological studies related to factors associated with chronic diseases, inadequate health information scheme, inadequate health and nutrition education, absence of a food and nutrition policy and insufficient information on prevention and management of chronic diseases. This is in a country which has enjoyed prosperity through economic growth and fast development.

The Kingdom of Saudi Arabia is a developing one which had experienced a remarkable change in various aspects of the lifestyle in the last three decades. Such dramatic increases in the gross domestic product, wealth, prosperity and income distribution, foreign influence, food availability in the market with affordable price, etc. There has been a drastic change in food consumption patterns and dietary habits as a results of the high income and associated prosperity. Examining the food consumption for the whole country as obtained from the SAFBS (see chapter 2), the availability of energy supplies/calories does not necessarily reflect the amount consumed but indicate the trends for the entire country. It shows a marked increase in almost all food commodities during a short period of time, from 1974 to 1986. During this period there was an increase in the consumption of (1) fat and meat (particularly poultry and lamb), fish and dairy products; (2) refined sugar; (3) a favourable rise in the consumption of fruits and vegetables. The (4) increase of the contribution of fat to the percentage of calories available has been associated with the decline of the percentage of calories from carbohydrate. Although there is no data for SFAs, but the rise in the consumption of fat, meat and diary products may reflect an undesirable increase in the SFAs. The full impact of the above mentioned changes on the health and nutritional status of this population is unknown.

The aims of this study, therefore, were to investigate the relationship between the

food availability their fat content, the fatty acid profile and coronary heart disease proportionate deaths in the Eastern Province.

In order to achieve this goal, the information was obtained from data collected in this study in different stages. First, a food availability survey based on household food purchasing for five consecutive days. Second, CHD PM survey based on recorded deaths from six Ministry of Health (public) Hospitals from 1989 to 1990. Third, the experimental work, involved the determination of fat and fatty acid profile of foods identified in the first study.

The study succeeded in exploring the variations among the three different communities in the area (i.e. urban, rural and Bedouin) with reference to the investigation mentioned above. To date, this study is the first effort of its kind to assess food availability and coronary heart disease mortality in SA. Nevertheless, the major disappoint of the study was its inability to determine the individual intake which may give us a clearer picture of a possible relationship between diet and CHD among the population studied. As discussed in the previous chapters, there were a number of inherent biases associated with the methods used in this study which were enhanced by the social and cultural habits. The major bias in the survey which had important effects on the results was inability of the study to undertake a larder inventory before and after the survey. Another disappointment of our study is that we were unable to study the CHD incidence as such data would require knowledge of the population base and this is currently difficult to obtain. There is no accurate primary health data and population census data is not up to date. Therefore, this study analysed PM as a proportion of the total number of deaths in the hospital. Such data can only give a crude indication of differences in CHD mortality between Bedouins, urban and rural Saudis. In addition, this type of data may have some other limitations as has been fully discussed in section 4.3.3. Moreover, the data available from these hospitals was only for a short period of time (two years), which did not allow us to assess the trend changes. We acknowledge that this weakness outlined above were important and made our data interpretation difficult. Nevertheless, the findings highlight significant problems which should be tackled when further research is considered in the future.

Our results demonstrate that the CHD PM was comparable with that in the UK. They also show a significant difference between Bedouin and non-Bedouin CHD PM, which may be due to dietary as well as non-dietary risk factors. It is difficult for us,

on the basis of the current study, to explain this. Furthermore, the observed differences may also lie in the biases in the method used in this study (see section 4.3.3). Whatever the explanation, it remains that the CHD PM differences between the population studied and the similarity of percentage of CHD PM with the UK are an attractive hypothesis with as yet only scanty data to support or refute it. Further investigations may arrive at a final conclusion.

The results of this study in chapters 3 and 5 may provide some indication of western diet influence which is characterised for its high fat content and hence SFA as a result of daily consumption of animal meats and dairy products. The fat and FA profile of the analysed foods demonstrated that many Saudi foods contained a high amount of SFAs. In comparison with diets from the UK and Japan (Table 3.40), reveals that the Saudi diet resembles the UK in some respect and Japanese in other respects. The epidemiological studies have clearly established a strong correlation between the dietary pattern and the mortality of CHD. Our findings regarding the food availability and FAs profile and the observed CHD PM as well as the government food balance sheets may lead us to the prediction that CHD will rise in the future if current dietary consumption patterns continue. The fact that several social, environmental and dietary factors are associated with CHD occurrence means that a multi-factorial intervention programme should be considered in order to prevent and control the CHD in this country. Diet is undoubtedly only one factor involved in the CHD aetiology, but is not sufficient to explain the whole picture of the CHD mortality. We have strong believe that the causation of this disease in SA is not only the high intake of foods rich in fat but rather results from a mixture of several factors such as ignorance, sedentary lifestyle, socio-economic factors, lack of physical activity as well as increasing in smoking. Since this study is limited in its objective further in depth studies is highly recommended. Future researches should not focus only on diet but also on other CHD risk factors such as diabetes, hypertension, smoking, lack of exercise, stress of modern life, etc., to determine the true prevalence of the disease and factors associated with its prevalence among these population. Section 6.3 summarise several area of recommended studies.

As far as the public is concerned, the lack of awareness and shortage of information are the major stumbling blocks. Therefore, it is the major responsibility of the health professionals, academics, scientists and above all the media to advocate and dramatise these stark realities, with facts, figures and other documentation, and

bring them to the attention of government authorities, planners and decision makers so that they will be sensitized enough to draw the government support for actions. It is through these channels that lay down the foundation for the development of guidelines and public policies in order to alleviate the disastrous consequences of these chronic disease due to our sedentary lifestyle, overconsumption and affluent diet. There is no doubt that changing the behaviour and culture in SA, and indeed any society, is not an easy task but it needs hard and continuous innovative, devising strategies whereby these harmful food habits can be changed.

The change needs support from food and nutrition planning, food policy and marketing system. The change also needs effort from the food industry, agriculture, public health and other related departments. Therefore, an integrated approach is suggested in nutrition intervention activities. The first step, if intervention strategies is to be successful over the long term, is by bringing public attention towards healthy eating habits, through mass education. We believe that getting the right, healthy eating massage across does start at school. It is vital that, from an early age, children understand the nutritional values of basic foods, the need for a balanced diet and why it is important. Second, encourage the food industry to make available products that conform with healthy food, especially by reducing saturated fat in their products.

Section 6.3

# CONCLUSIONS AND RECOMMENDATIONS

To conclude, it seems imperative to ask what the study has shown and in what way it adds to our understanding of the food availability, fatty acid profile and CHD mortality in SA. Various important points have already been discussed throughout this thesis.

There is very limited information available on food consumption and food compositions in SA. In addition there limited data on general health in SA, in particularly chronic diseases. Our special concern is the government ignorance and the lack of understanding amongst the general population, considering the single biggest factors in rising of the life-threatening conditions in SA. It is about time, and in fact a little too late, that the government through a concerted effort and the cooperation of other government sectors, organise the development of major guidelines and public health policies concerning the consequences of excessive caloric intake, in particularly SFA, smoking, lack of physical activity, etc. Extensive studies are required to determine the exact prevalence, incidence, types, nature, complications, genetic, etiology, treatment and prognosis of these chronic health hazards. To concluded, in the hope of achieving that, a number of recommendations for further investigation/action are suggested on the basis and experience of this study.
These are:

1. The government food balance sheets shows that food is considerably in excess of requirements. Further, the present study also show the same results. Further studies are required to assess the Saudi's nutrition intakes.

2. Further studies should be carried out to develop improved method for collecting reliable quantitative data on habitual food consumption and individual intake.

3. How family food is distributed within the home should be studied to assess the age/sex discrimination among the family which might put certain family member at health risk.

4. Estimates of the RNI for the Saudi population groups is also needed, taking into account body weight, dietary and availability of nutrients in local foods.

5. More work needed in respect to dietary fat contents of the Saudi diet.

6. There is an urgent need to set up food composition tables with local foods and native dishes for use in SA.

7. In-depth studies of the effects of changing lifestyle and diet on the CHD incidence/prevalence/mortality in the Kingdom of Saudi Arabia are needed.

8. Alternative CHD risk factors, other than diet, are needed to be studied in more details to explain the prevalence of CHD in SA. This is should be possible to assess risk factors by selecting an unbiased sample (case control study) of the population. Then the risk factors could be assessed and compared between the cases and this sample (controls).

9. Further research should focus on the genetic aspects and its contribution to the CHD among the Saudi.

10. Since obesity is common among the Saudi population, more studies are required to delineate the extent of the obesity and its impact in CHD and other chronic diseases on national level.

11. We observed that the Bedouins in KFH were recorded as dying from CHD less often than other Saudis seems particularly worthy of future attention. More detailed, extensive and comprehensive data is needed to confirm this hypothesis and what lesson should be learned from their lifestyle.

12. Anti-smoking campaigns must receive more attention, e.g. banning cigarettes from local publications, replaced by anti-smoking adverts and band smoking from school and public places. In addition, the price of cigarettes should be increased.

13. Designing and implementing a health education policy is needed and should be focusing on the lifestyles and related diseases. For example, emphasis needs

to be placed on the immediate benefits of adopting a healthy diet, stop smoking, etc.

14. Investigating how the family income is spent, in particular on food.

15. The lack of qualified and trained staff to collect the dietary intake, make it important for a national training programme in the nutrition.

16. This study revealed that there is an urgent need for the improvement of the existing vital registration system in SA. In addition, greater efforts are needed to collect accurate information on the cause of death in order to plan programmes to minimise such health hazards in SA.

# *LIST OF PUBLICATIONS*

1. Ahmed Abdulla Alobaid, BSc; Robert Gilchrist, PhD; Brian Bointon, PhD. Coronary Heart Disease Mortality In The Eastern Province of Saudi Arabia in 1989 and 1990. *Annals of Saudi Medicine Journal.* 1994; Vol. 14, No. 5: 387-391.

2. "Food Consumption Survey In The Eastern Province Of Saudi Arabia: (I) Energy, fat, protein, carbohydrate and fibre intakes". To be published.

3. "Food Consumption Survey In The Eastern Province Of Saudi Arabia: (II) The Dietary Habits in the Urban, Rural and Bedouin Communities". To be published.

4. "Food Consumption Survey In The Eastern Province Of Saudi Arabia: (iii) Household socio-economic Characteristics and its influence on the Saudi household's food availability. To be published.

5. "Evaluation of the Apparent Dietary Intake of the Saudi Population, 1974-1986". To be published.

# *REFERENCES*

Abdullah M.A. Swailem A. and Taha S.A (1982). Nutrition status of preschool children in central Saudi Arabia. *Ecology of Food and Nutrition*, **12**: 103-107.

Abu Bakr S. and Al-Suleiman S. (1990). Attitudes towards fertility control in the Gatif region, Eastern Province, Saudi Arabia. *Saudi Medical Journal*, **11**, (1): 64-68.

Aherns E.H., Hirsh J., Insull W., Tsaltas T.T., Blomstrand R. and Peterson M.L. (1957). The influence of dietary fats on serum-lipid levels in man. *The Lancet*, **1**: 943-953.

Ahmed A.F., Abdelsalam S.A., Mohammed M.E. and Gadri M.A. (1993). A case-control study of the incidence of coronary heart disease risk factors in Saudis at Almadina Almounawarh. *Saudi Medical Journal*. **14** (2): 146-151.

Ahrenc E.H. and Boucher C.A. (1978). The composition of a simulated American diet. *Journal of American Dietetic Association*, **73**: 613-620.

Almuslim M.S. (1962). *The Black Gold Coast*. Alhayat Press, Beirut (in Arabic).

Al-Awadi F. and Amine E. (1989). Overweight and obesity in Kuwait. *Journal of Royal Society of Health*, **5**: 175-177.

Al-Jebrin A., Sawaya W.N., Salji J.P., Ayaz M. and Khalil J.K. (1985a). Chemical and nutritional quality of some Saudi Arabian dishes based on cereal and legumes: I. Proximate composition, amino acid contents and nutritive value. *Ecology of Food and Nutrition*, **17**: 157-164.

Al-Jebrin A., Sawaya W.N., Salji J.P., Ayaz M. and Khalil J.K. (1985b). Chemical and nutritional quality of some Saudi Arabian dishes based on cereal and legumes: II. Mineral and vitamin contents. *Ecology of Food and Nutrition*, **17**: 352-354.

Al-Musharef S.A. (1990). *Maternal food habits and infant feeding practice in Saudi Arabia*. King's College London (KQC), London, Department of Food and Nutrition Sciences, (PhD. thesis).

Al-Sudairy A. and Howard K. (1992). Dietary habits of technical and vocational student in Riyadh, Saudi Arabia-Part 1 meal skipping. *Journal of Royal Society of Health*, 112 (5): 217-218.

Al-Sudairy A. and Howard K. (1992). Dietary habits of technical and vocational student in Riyadh, Saudi Arabia-Part 11 eating between meals. *Journal of*

*Royal Society of Health*, 112 (6): 271-272.

Anderson T.W., Deakins D.A., Floore T.L. Smith B.M. and Whitis S.E. (1990). Dietary fibre and coronary heart disease. *Food Science and Nutrition*, **29** (2): 95-147.

Anokute C.C. (1990). Epidemiologic studies of diabetes mellitus in Saudi Arabia- Part 1- screening of 3158 males in King Saud University. *Journal of Royal Society of Health*, **110** (6): 201-203.

Antiquities and Museums, Ministry of Education (1982). *Atlal*. The Journal of Saudi Arabia Archaeology Department, Riyadh, The Kingdom of Saudi Arabia, **6**: 95-105 (in Arabic).

Antiquities and Museums, Ministry of Education (1978). *Atlal*. The Journal of Saudi Arabia Archaeology Department, Riyadh, The Kingdom of Saudi Arabia, **2**: 7-29, 109-123 (in Arabic).

Antiquities and Museums, Ministry of Education (1977). *Atlal*. The Journal of Saudi Arabia Archaeology Department, Riyadh, The Kingdom of Saudi Arabia, **1**: 9-18, 25-35 (in Arabic).

Ascherio A., Hennekens C.H., Buring J.E., Master C., Stampfer M.J. and Willett W.C. (1994). Trans-fatty acids intake and risk of myocardial infarction. *Circulation*. **89** (1): 94-101.

Ashwell M., (1993). *Diet and Heart Disease, A Round Table of Factors*. The British Nutrition Foundation. UK.

Axelrod L., Camuso J., Williams E., Kleinman K., Briones E., Schoenfeld D. (1994). Effects of a small quantity of omega-3 fatty acids on cardiovascular risk factors in NDDM - a randomized, prospective, double-blind, controlled study. *Diabetic Care*. **17** (1): 37-44.

Bacchus R.A., Bell J.L., Madkour M. and Kilshaw B. (1982). The prevalence of diabetes mellitus in male Saudi Arabs. *Diabetologia*, **23**: 330-332.

Baggio G. Pagnan A., Muraca M., et al., (1988). Olive oil enhanced diet: effect on serum lipoprotein levels and biliary cholesterol saturation. *American Journal of Clinical Nutrition*. **47**: 960-964.

Ball K.P., Fleming J., Fowler T.J., James I., Maidment G. and Word C. (1981).

*Handbook of Cardiac Care*. M.T.P. Press Ltd., UK: 103.

Ball M., and Mann J. (1990). *Lipids and Heart Disease: A Practical Approach*. Oxford University Press, UK: 20-21.

Bang A., Hamazaki T., Terano T., et al., (1980). Eicosapentaenoic acid and platelet function in Japanese. *Lancet*. **2**: 1132-1133.

Barr S.L., Ramakrishnan R., Johnson C., Holleran S., Dell R.B. and Ginsberg H.N. (1992). Reducing total dietary fat without reducing saturated fatty acids does not significantly lower plasma cholesterol concentrations in normal males. *American Journal of Clinical Nutrition*, **55**: 675-681.

Beaglehole R. (1991). Coronary heart disease and elderly people, *British Medical Journal*, **303**: 69-70.

Becker A.E. and Anderson R.H. (1983). *Cardiac Pathology: An Intergrated Text and Colour Atlas*. Gower Med., Pub., Lit. London: 3.2.

Belcher J.D., Balla J., Balla G., Jacobs DR. Jr, Gross M., Jacob H.S. and Vercellotti G.M. (1993). Vitamin E, LDL, and endothelium, brief oral vitamin supplementation prevent oxidized LDL-mediated vascular injury in vitro. *Arterioscler Thromb*. **13**: 1779-1789.

Ben-Shlomo Y., et al., (1994). What determines mortality risk in male former cigarette smokers? *American Journal of Public Health*. **84** (8): 1235-1242.

Berry E.M., Eisenberg S., Haratz D., Friedlander Y. and Norman Y. (1991). Effects of diets rich in monounsaturated fatty acids on plasma lipoproteins-the Jerusalem Nutrition Study: high MUFAs vs high PUFAs. *American Journal of Clinical Nutrition*, **53**: 899-907.

Bingham S.A. (1987). The dietary assessment of individuals; methods, accuracy, new techniques and recommendations. *Nutrition Abstracts and Reviews* (Series A), **57** (10): 705-742.

Bingham S.A. (1991). Limitations of the various methods for collecting dietary intake data. *Annals of Nutrition and Metabolism*. **35**: 117-127.

Binhemd T.A. (1992). Diabetes mellitus: knowledge, attitude, practice and their relation to diabetes control in female diabetics. *Annals of Saudi Medicine*, **12** (3): 247-251.

Binhemed T., Larbi E.B. and Abosood G. (1991). Obesity in a primary health care centre: A retrospective study. *Annals of Saudi Medicine*, **11**: 163-166.

Blair J.A., Parkash C., Phillips M.A., Dougherty R.M., Iacono J.M. (1993). Dietary modification of W-6 fatty acid intake and its effect on urinary eicosanoid excretion. *American Journal of Clinical Nutrition*. **57**: 154-160.

Blot W.J., Li J.Y., Taylor P.R., Guo W., Dawsey S., Wang G.Q., Yang C.S. Zheng S.T., Gail M., Li G.Y., et al., (1993). Nutrition intervention trails in Linxian China: supplementation with specific vitamin/mineral combination, cancer incidence and disease-specific mortality in the general population. *Journal of Nat Cancer Inst*. **85** (18): 1483-1492.

Bonanome A. and Grundy S.M. (1988). Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *New England Journal of Medicine*, **318**: 1244-1248.

Bray C. and Ward C. (1986). *Ischaemic Heart Disease*. Update Pub., Ltd., UK: 7-8.

Breslow N.E. and Day N.E. (1987). *Statistical Methods in Cancer Research Volume II - The Design and analysis of Cohort Studies*. International Agency for Research on Cancer. IARC Scientific Publication No. 82.

Brown M.S. and Goldstein J.L. (1983). Lipoprotein catabolism in the macrophage: implications for cholesterol deposition in atherosclerosis. *Annual Review of Biochemistry*. **52**: 223-261.

Bull N.L. (1985). Dietary habits of 15-25 year olds. Human Nutrition: *Applied Nutrition*, **39A** (1): 1-68.

Burr M.L., Gilbert J.F., Holliday R.M., Elwood P.C., Fehily A.M., Rogers S., Sweetnam P.M. and Deadman N.M. (1989). Effects of changes in fat, fish and fibre intakes on deaths and myocardial infarction: diet and infarction trial (DART). *The Lancet*, **2** (8666): 757-761.

Burton B.T. and Foster W.R. (1988). *Human Nutrition*. Formerly the Heinz Handbook of Nutrition. 4th ed. H J Heinz Co., USA. 47-171, 371.

Cameron M.E. and Van Staveren W.A. (1988). *Manual on methodology for food consumption studies*. Oxford medical publications, Oxford.

Castelli W.P. (1990). Diet, smoking and alcohol: influence on coronary heart disease risk. *American Journal of Kidney Disease*, **16** (4): 41-46.

Central Department of Economic Studies and Statistics (1988): *Saudi Arabian Food Balance Sheets from 1974-1976 to 1983-1986.* Ministry of Agriculture and Water, Safir Press, Riyadh, Kingdom of Saudi Arabia, **2**.

Champman S. (1988). Fiddling while tobacco burn: sixth world conference on smoking and health. *British Medical Journal*, **296**: 39-40.

Chislom G.M. (1991). Cytotoxicity of oxidized lipoproteins. *Curr Opin Lipidol.* **2**: 311-316.

Christen A. (1984). Survey of smoking behaviour and attitudes of 630 American dentists: current trends. *Journal of American Dentils Association*, **109**: 271-272.

Christie W.W. (1982). *Lipids Analysis: isolation, separation, identification and structural analysis of lipids*. 2nd edn. Pergamon Press, UK.

Collett D. (1991). *Modelling Binary Data.* Chapman and Hall, London.

Connor W.E. and Connor S.L. (1990). Diet, atherosclerosis and fish oil. *Advances in Internal Medicine*, **35**: 139-172.

Crawfor T. (1977). *Pathology Ischaemic Heart Disease*. Butterworths and Co., Pub., Ltd., London: 28-34.

Crombie I.K., Kenicer M.B., Smith W.C.S. and Tunstall-Pedoe H.D. (1989). Unemployment, socio-environmental factors, and coronary heart disease in Scotland. *British Heart Journal*, **61**: 172-177.

Cuesta C., Sanchez-Muniz F.J., Garcia-La-Cuesta A., Garrido R., Castro A., San-Felix B. and Domingo A. (1989). Effects of age and cigarette smoking on serum concentrations of lipids and apolipoproteins in a male military population. *Atherosclerosis*, **80** (1): 33-39.

D'Armiento F.P., Di-Gregorio F., Ciafre S.A., Posca T., Liguori A., Napoli C., Colasanti P., Cali A. and Vecchione R. (1993). Histological findings and evidence of lipid conjugated denes and maloyldialdehyde in human fetal aortas. *Acta Paediatr.* **82** (10): 823-828.

Dattil A.M. and Etherton P.M. (1992). Effects of weight reduction on blood lipids and lipoproteins. *American Journal of Clinical Nutrition*. **56**: 320-328.

Davies M. (1992). Atherosclerosis - can regression be achieved? *Cardiology in Practice*. **10**: 4-5.

Davies M.J. (1994). Pathology of arterial thrombosis. *British Medical Bulletin*. **55** (4): 789-802.

Den Hartog A.P. (1972). Unequal distribution of food within the household. *Nutrition News*, **10**: 8-17.

Denke M.A., Grundy S.M (1992). Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. *American Journal of Clinical Nutrition*. **56**: 895-898.

Department of Health (1991). *Dietary Reference Values for Food Energy and Nutrients for the UK*. COMA Report on health and social subjects, No. 41. London. HMSO.

Department of Health (1994). *Nutritional Aspects of Cardiovascular Disease*. COMA Report on health and social subjects, No. 46. London. HMSO.

Desmond G.J. (1988). *Cardiology*. 5th ed. Bailliere Tindall. London: 139, 143.

Dimsdale J.E. (1988). A perspective on Type A behaviour and coronary disease. *New England Journal of Medicine*, **318**: 110-112.

Duthie G.G. (1989). Oxidants, antioxidants and cardiovascular disease. *Nutrition Research Reviews*. **2**: 51-62.

Dwyer J.T. (1988). Health aspects of vegetarian diets. *American Journal of Clinical Nutrition*, **48** (3): 712-738.

Egan H., Kirk R.S. and Sawyer R. (1990). *Pearson's Chemical Analysis of Foods*. 8th Ed., Churchill Livingston, UK: 404, 438, 458, 499.

Egusa G., Bennett P.H., Kirk A., Tylor R. and Howard B.V. (1984). Hperlipemia and arteriosclerotic cardiovascular disease in the Polynesian population of Raratonga. *Atherosclerosis*. 53: 241-254.

Eid N., Al-Hooti S., Bourisly N., Khalafaw M., Elimam A. and Al-Gayear M. (1983). Nutritional status in Kuwait. I. Nutritional assessment of school children. *Kuwait Institute for Scientific Research*, Kuwait.

Eliasson M., Asplund K., Evrin P. and Lundblad D. (1995). Relationship of cigarette smoking and snuff dipping to plasma fibrinogen, fibrinolytic variables and serum insulin. The North Sweden MONICA study. *Atherosclerosis*. **113**: 41-53.

Eriksen W. (1994). The role of social supports in the pathogenesis of coronary heart disease. A literature review. *Family Practice*. **11** (2): 201-209.

Ernst J., Schaefer et al., (1994). Lipoprotein (a) levels and risk of coronary heart disease in men. The lipid research Clinics coronary primary prevention trial. *Journal A M A*. **271**: 999-1003.

Esterbauer H., Dieber-Rothender M., Waeg G., Strigl G., Jurgens G. (1990). Biochemical, structural and functional properties of oxidized low-density lipoprotein. *Chem Research Toxicology*. **3**: 77-92.

Esterbauer H., Gebicki J., Puhl H., Jurgens G. (1992). The role of lipid peroxidation and antioxidants in oxidative modification of LDL. *Free Radical Biology and Medicine*. **13**: 341-390.

Fatain M.S. and Baig M.H.A. (1983). A survey over a period of three years of glucose tolerance test at King Abdulaziz University Hospital, Jeddah (Abstract). 8th Saudi Med Conference, King Khalid Military Academy, Riyadh, Saudi Arabia 30 Oct-4Nov.,

Findanza F. and Albert A.F. (1967). Dietary surveys in connection with the epidemiology of the heart disease: reliability, sources of variation and other data from nine surveys in Italy. *VOEDING*, **28** (6): 244-252.

Fischer S., Weber P.C. and Dyerberg J. (1986). The prostacyclin/thromboxane balance is favourably shifted in greenland eskimos. *Prostaglandins*. 32: 235-241.

Fisher S. and Weber P.C. (1984). Thromboxane (TX) $A_3$ and prostaglandins (PG)$I_3$ are formed in man after dietary eicosapentaenoic acid: identification and quantification by capillary gas chromatography-electron impact mass spectrometry. *Biomed Mass Spectrum*. 12: 470-476.

Flaim E., Ferreri L.F., Thye F.W., Hill J.E. and Ritchey S.J. (1981). Plasma lipid and lipoprotein cholesterol concentrations in adult males consuming normal and

high cholesterol diets under controlled conditions. *American Journal of Clinical Nutrition*, **34**: 1103-1108.

Flores M., Garica B., Florez Z. and Lara M.Y. (1964). Annual patterns of family and children's diet in three Guatemalan Indian communities. *British Journal of Nutrition*, **18**: 281-293.

Flynn M.A., Nolph GB., Flynn TC., Kahrs R. and Hrause G. (1979). Effect of dietary egg on human serum cholesterol and triglycerides. *American Journal of Clinical Nutrition*, **32**: 1050-1057.

Fonnebo V. (1988). The Tromso Heart Study: diet, religion, and risk factors for coronary heart disease. *American Journal of Clinical Nutrition*, **48**: 826-829.

Fortmann S.P., Sallis J.F., Magnus P.M. and Farquar J.W. (1989). Attitudes and practices of physicians regarding hypertension and smoking. The Stanford Five City Project. *Preventive Medicine*, **14**: 70-80.

Fox K.M. (1987). *Ischaemic Heart Disease.* MTP Press Ltd., UK.: 33-44.

Francis B., Green M., Payne C., et. al., (1993). *The Glim System, Release 4 Manual.* Clarendon Press, UK: 244.

Frankel E.N., Kanner J., German J.B., Parks E., Kinsellaa J.E., (1993). Inhabitation of oxidation of human low-density lipoprotein by phenolic substances in red wine. *Lancet.* **341**: 454-457.

Frankel E.N., Park E.J., Xu R., Schneeman B.O., Davis P.A. and German J.B. (1994). Effect of N-3 fatty acid-rich fish oil supplementation on the oxidation of low density lipoproteins. *Lipids.* **29** (4): 233-236.

Garcia-Palmieri M.R., Sorlie P., Tilloston J., Costas R., Codero E. and Rodriguez M. (1980). Relationship of dietary intake to subsequent coronary heart disease incidence: the Puerto Rico heart health program. *American Journal of Clinical Nutrition*, ; **33**: 1818-1827.

Gordon T., Fisher M. and Rifkind B. (1984). Some difficulties inherent in the interpretation of dietary data from free-living populations. *American Journal of Clinical Nutrition*, **39**: 152-156.

Gregory J., Foster K., Tyler H., and Wiseman M.J. (1990). *The Dietary and Nutritional Survey of British Adults.* HMSO., London.

Grinstead G.F. and Ellefson R.D. (1988). Heterogeneity of lipoprotein Lp(a) and apolipoprotein (a). *Clinical Chemistry*, **34** (6): 1036-1040.

Grundy S.M. and Denke M.A. (1990). Dietary influences on serum lipids and lipoproteins. *Journal of Lipid Research*, **31**: 1149-1172.

Guo J. and Geng Z (1995). Collapsibility of logistic regression coefficients. *Journal of the Royal Statistical Society B*. **57** (1): 263-268.

Harris W.S. (1989). Fish oils and plasma lipid and lipoprotein metabolism in humans: a critical review. *Journal of Lipid Research*, **30**: 785-807.

Hashim S.A., Artega A. and van Itallie T.B. (1960). Effect of a saturated medium-chain triglyceride on serum-lipids in man. *The Lancet*, **1**: 1105-1108.

Hayes K.C., Pronczuk A., Lindsey S. and Diersen-Schade D. (1991). Dietary saturated fatty acids (12:0, 14:0, 16:0) differ in their impact on plasma cholesterol and lipoprotein in non human primates. *American Journal of Clinical Nutrition*, **53**: 491-498.

Health Education Authority (1990). Diet and cancer: Briefing paper. *Health Education Authority*. London.

Hearse D.J. (1994). Myocardial ischaemia: can we agree on a definition for the 21st century? *Cardiovascular Research*. **28**: 1737-1744.

Hegsted D.M., McGandy R.B., Myers M.L. and Stare F.J. (1965). Quantitative effects of dietary fat on serum cholesterol in man. *American Journal of Clinical Nutrition*, **17**: 281-295.

Heliovaara M., Knekl P., Aho K., Aaram R.K., Alfthan G. and Aromaa A. (1994). Serum antioxidants and risk of rheumatoid arthritis. *Annals of the Rheumatic Diseases*. **53** (1): 51-53.

Hertog M.G.L., Feskens E.J.M., Hollman P.C.H., Katan M.B., Kromhout D., (1993). Dietary antioxidant flavonoids and the risk of coronary heart disease: the Zutphen Elderly Study. Lancet. **342**: 1007-1011.

Hirai H.O., Dyerberg J., Sinclair H.M., (1980). The composition of the Eskimo food in North Western Greenland. *American Journal of Clinical Nutrition*. **33**: 2657-2661.

Hodis H.N., Mack W.J., LaBree L., et al., (1995). Serial coronary angiographic evidence that antioxidant vitamin intake reduces progression of coronary artery atherosclerosis. *JAMA*. **273** (23): 1849-1854.

Hoffman R.M. and Garewal H.S. (1995). Antioxidants and the prevention of coronary heart disease. *Arch International Medicine*. **155** (3): 241-246.

Hurst J.W., Logue B.R., Schlant R.C., Wallance A.G. and Wengen N.K. (1986). *The Heart*. 6th Ed., McGraw - Hill, Inc., USA: 801-803.

Hussain M. and Sarkar T.U. (1971). A comparative study of record and recall method of dietary survey in West Pakistan. *Journal of Pakistan Medicine Association*, **21**: 208-211.

Inam S., Cumberbatch M. and Judzewitsch R. (1991). Importance of cholesterol screening in Saudi Arabia. *Saudi Medical Journal*, **12** (3): 215-220.

Jackson G. (1988). *Practical Management of Ischaemic Heart Disease*. Martin Dunitz. UK: 7-22.

Jacobs J.R., Burke G.L., Liu, K., Cutter, G., Hughes G., Hulley S. and Sidney S. (1988). Relationships of low density lipoprotein cholesterol with age and other factors. A cross-sectional analysis of the cardiac study. *Annals of Clinical Research*, **20** (1-2): 32-38.

James C.S. (1995). *Analytical Chemistry of Foods*. Blackie Academic and Professional pub., UK: 46-51.

Jocanovic S.V., et al., (1994). Flavonoids as antioxidants. *Journal of the American Chemical Society*. **116** (11): 4846-4851.

Joint FAO/WHO/UNU report (1985). *Energy and protein requirements*. World Health Organisation, Geneva: 133-137.

Kaiser L., and Dewey K.G. (1991). Household economic strategies, food resource allocation, and intrahousehold patterns of dietary intake in rural Mexico. *Ecology of Food and Nutrition*, **25**: 123-145.

Kamel B.S. and Martinez O.B. (1984a). Food habits and nutrient intake of Kuwaiti males and females. *Ecology of Food and Nutrition*, **15**: 261-272.

Kamel B.S. and Martinez O.B. (1984b). Food availability for Kuwaiti and non-Kuwaiti households. *Ecology of Food and Nutrition*, **15**: 191-201.

Kaplan J.R., Adams M.R., Clarson T.B. and Kontnik D.R. (1984). Psychosocial influences on female protection among cynomolgus macaques. *Atherosclerosis*, **53** (3): 283-295.

Katan M.B., Zock P.L. and Mensink R.P. (1994). Effects of fats and fatty acids on blood lipids in humans. *American Journal of Clinical Nutrition*. **60**: 1017S-1022S.

Kazawa Y., Niskizawa M., Suzuki M., et al., (1982). Eicosapentaenoic acid of serum lipids of Japanese islanders with low incidence of cardiovascular diseases. *Journal of Nutrition Society Vitaminol (Toko)*. **28**: 441-453.

Keli S.O., et al., (1994). Fish consumption and stroke risk. *Stroke*. **25**: 328-332.

Kelley J.L. (1991). Macrophage scavenger receptor (s) for modified low density lipoproteins. *Progress in Lipid Research*. 30: 231-235.

Keys A., Anderson J.T., Grande F. (1965). Serum cholesterol response to changes in the diet. IV. Particular saturated fatty acids in the diet. *Metabolism*. **14**: 776-787.

King H. and Rewers M. (1991). Diabetes in adult is now a third world problem. *Bull World Health Organisation*. **6**: 543-548.

Kingston M. and Skoog W.C. (1986). Diabetes in Saudi Arabia. *Saudi Medical Journal*, **7**: 130-142.

Kinsella J.E., Lokesh B. and Stone R.A. (1990). Dietary n-3 polyunsaturated fatty acids and amelioration of cardiovascular disease: possible mechanisms. *American Journal of Clinical Nutrition*. 52: 1-28.

Kissebah A.H., Freedman D.S. and Peiris A.N. (1989). Health risks of obesity. *American Journal of Clinical Nutrition*, **73**: 111-138.

Klag M.J. (1990). Alcohol and cardiovascular disease: a review of the evidence. Cholesterol et Prevention Primaire, CIDIL, Paris: 31-46.

Klevay L.M. (1987). Ischaemic heart disease. A major obstacle to becoming old.

*Clinical Geriatr of Medicine*, **3** (2): 361-372.

Knekt P., Peunanen A., Jarvinen R., Seppanen R., Heliovaara M. and Aromaa A. (1994). Antioxidant vitamins and coronary heart disease mortality in a longitudinal study population. *American Journal of Epidemiology*. **139** (12): 1180-1189.

Kohli KL., and Al-Omaim M. (1986). Mortality level, trends and differentials in Kuwait, 1957-1983. *Population Bulletin of ESCWA*, **28**: 91-123.

Kowala M.C., Mazzucco C.E., Hartl K.S., Seiler S.M. Warr G.A., Abid S. and Grove R.I. Prostcyclin agonists reduce early atherosclerosis in hyperlipidemic hamsters. Octimibate and BMY 42393 suppress monocyte chemotaxis, macrophage factor production. *Arterioscler Thromb*. **13** (3): 435-444.

Kremer J.M. (1991). Clinical studies of omega-3 fatty acid supplementation in patients who have rheumatoid arthritis. *Rheumatic Disease Clinics of North America*. **17**: 391-402.

Kromann N., and Green A., (1980). Epidemiological studies in the Upernavik district. Greenland: incidence of some chronic disease 1950-1974. *Acta Med Scand*. **208**: 401-406.

Kromhout D., Bosschieter E.B. and DeLezenne Coulander C. (1982). Dietary fibre and 10-year mortality from coronary heart disease, cancer and all causes: the Zutphen study. *The Lancet*, **2**: 518-521.

Kromhout D., Bosschieter E.B. and Coulander C. d L. (1985). The inverse relation between fish consumption and 20-year mortality from coronary heart disease. *New England Journal of Medicine*. **312** (19): 1205-1209.

Kucera B. and McIntosh W.A. (1991). Family size as a determinant of children's dietary intake: A dilution model approach. *Ecology of Food and Nutrition*, **26**: 127-138.

Kumar K. V., and Das U.N., (1994). Lipid peroxides and essential fatty acids in patients with coronary heart disease. *Journal of Nutritional Medicine*. **3**: 33-37.

Kummerow FA., Dim Y., Hull MD., Pollard M., Ilinov P., Dorrossiev D and Valek J. (1977). Influence of egg consumption on the serum cholesterol leve in human subjects. *American Journal of Clinical Nutrition*, **30**: 664-673.

Kunitz S.J. (1990). Public policy and mortality among indigenous populations of Northern America and Australasia. *Population and Development Review*, **16**: 647-672.

Landers S.C. and Lewis J.C. (1993). acLDL binding and endocytosis by macrophages and macrophage foam cells in situ. *Exp Mol Pathol.* **59** (1): 38-50.

Leibowitz J.O. (1970). *The History of Coronary Heart Disease*. William Clowes and sons, Ltd., London: 1.

Leon A.S. (1988). Physiological interactions between diet and exercise in the etiology and prevention of ischaemic heart disease. Ann Clin Research, **20** (1-2): 114-120.

Madkour M.M. and Kudwah A.J. (1985). *Saudi Medical Bibliography*. Churchil Livingston, Edinburgh, **1-3**.

Mahgoub O.M., Abdel-Hafeiz H.B., Al-Quorain A., Al-Idrissi H., Al-Ghassab G. and Absood G. (1991). Life events stress in Saudi peptic ulcer patients of the Eastern Province. *Annals of Saudi Medicine*, **11** (6): 669-674.

Maregette B.M. and Nelson M. (1991). *Design Concepts in Nutritional Epidemiology*. Oxford University Press. USA: 311-312.

Mattson F.H. and Grundy S.M. (1985). Comparisons of the effects of dietary saturated, monounsaturated and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. *Journal of Lipid Research*, **26**: 194-202.

Mayne S.T., Janerich D.T., Greenwald P., Chorost S., Tucci C., Zaman M.B., et. al., (1994). Dietary Beta-carotene and lung cancer risk in US non-smokers. *Cancer Inst.* **86**: 33-38.

McGill H.C. (1979). The relationship of dietary cholesterol to serum cholesterol concentration and to atherosclerosis in man. *American Journal of Clinical Nutrition*, **32**: 2664-2702.

Mensink R.P. and Katan M.B. (1990). Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *New England Journal of Medicine*, **323**: 439-445.

Mensink R.P. and Katan M.B. (1987). Effect of monounsaturated fatty acids versus complex carbohydrates on highdensity lipoproteins in healthy men and women.

*Lancet*. **1**: 122-125.

Mensinck R.P., Zock P.L., Katan M.B., Hornstra G., (1992). Effect of dietary cis and trans fatty acids on serum lipoprotein (a) levels in humans. *Journal of Lipid Research*. **33**: 1493-1501.

Miller D.L. and Farmer R.D.T. (1982). *Epidemiology of Disease*. Blackwell Scientific Pub., UK:

Ministry of Agriculture, Fisheries and Food (1988). *The National Food Survey, Form S900*, London.

Ministry of Agriculture Water, Department of Economic Studies and Statistics (1988). *Agricultural Sector Development "Graphical Indicators"*, The Kingdom of Saudi Arabia, **3** (in Arabic).

Ministry of Higher Education, University of Riyadh, College of Arts Department of Geography (1981). *Population Atlas of the Kingdom of Saudi Arabia*. Saudi Arabia (in Arabic).

Ministry of Planning (1990). *Fifth Development Plan 1990-1995*. Kingdom of Saudi Arabia.

Ministry of Health, Saudi Arabia (1987). *Annual Health Report*, Riyadh, the Kingdom of Saudi Arabia (in Arabic).

Morgan S.A. and Sinclair A.J. (1993). Effect on serum lipids of addition of safflower oil or olive oil to very low fat diets rich in lean beef. *Journal of the American Dietetic Association*, **93** (6): 644-648.

Morton R.F. and Hebel J.R. (1984). *A study Guide to Epidemiology and Biostatistics*. 2nd ed. University Park Press, USA: 23-24.

Mould R.F. (1983). *Cancer Statistics*. Adam Hilger Ltd., UK: 139-142.

Musaiger A.O. and Sungpuag P. (1985). Composition of mixed dishes commonly consumed in the Arabian Gulf states. *Ecology of Food and Nutrition*, **16**: 153-160.

Musaiger A.O. and Aldallal Z.S. (1983). *Some Factors Influencing Cause of Death in Bahrain (1976-1980)*. Ministry of Health, Bahrain: 1-7.

Musaiger A.O. and Al-Dallal Z.S. (1985). *Food Composition Tables for Use in Bahrain*. Ministry of Health. Bahrain.

Musaiger A.O. (1986). Education and food habits of Bahraini housewives. *Journal of Royal Society of Health*, **3**: 85-86, 89.

Musaiger A.O. (1982). Factors influencing food consumption Bahrain. *Ecology of Food and Nutrition*, **12**:39-48.

Musaiger A.O. (1991). Food consumption patterns of mothers in Oman (a preliminary study). *Journal of Royal Society of Health*, **111** (1): 8-9.

Musaiger A.O. (1981). Food habits in Bahrain, dietary practices and social occasions. Ministry of Health, Bahrain (in Arabic).

National Institutes of Health Consensus Conference (1985). Lowering blood cholesterol to prevent heart disease. *The Journal of the American Medical Association*, **253**: 2080-2090.

Neaton J.D., Kuller L.H., Wentworth D. and Borhani N.O. (1984). Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. *American Heart Journal*, **108**: 759-769.

Nelson M. (1986). The distribution of nutrient intake within families. *British Journal of Nutrition*, **55**: 267-277.

Nestel P.J. (1987). Polyunsaturated fatty acids (n3-, n-6). *The American Journal of Clinical Nutrition*, 45: 1161-1167.

Nettleton P.A. and Nelson M. (1980). Dietary survey methods, 2. A comparison of nutrient intakes within families assessed by household measures and the semi-weighed method. *Journal of Human Nutrition*, **34**: 394-354.

Norel S.E, Ahlbom A., Feychting M. and Pederson N.L. (1986). Fish consumption and mortality from coronary heart disease. *British Medical Journal*, **293**: 426.

Ornish D. (1993). Can lifestyle changes reverse coronary heart disease? *World Review of Nutrition and Dietetics*, **72**: 38-48.

Ornish D. (1991). *Program for Reversing Heart Disease*. Ballantine Books, New York.

Ornish D., Brown S.E., Scherwitz L.W., Billings J.H., Armstrong W.T., Ports T.A., McLanahan S.M., Kirkeeide R.L., Brand R.J. and Gould K.L. (1990). Can lifestyle changes reverse coronary heart disease: the Lifestyle Heart Trial. *The Lancet*, **336** (8708): 129-133.

O'Dea K. (1991). Westernisation, insulin resistance and diabetes in Australian Aborigines. *The Medical Journal of Australia*, **155**: 258-264.

Palgi A. (1981). Association between dietary changes and mortality rates: Israel 1949 to 1977; a trend-free regression model. *The American Journal of Clinical Nutrition*, 34: 1569-1583.

Pamuk E.R., Byers T., Coates R.J., Vann J.W., Sowell A.L., Gunter E.W. and Glass D. (1994). Effect of smoking on serum nutrient concentrations in African-American women. *American Journal of Clinical Nutrition*. **59**: 891-895.

Parkash C., Nelson G.J., Wu M-M., Schmidt P.C., Phillips M.A., Blair J.A. (1994). Decreased systemic thromboxane $A_2$ biosynthesis in normal human subjects fed a salmon-rich diet. *American Journal of Clinical Nutrition*. **60**: 369-373.

Parkin D.M. ed. (1986). *Cancer Occurrence in Developing Countries*. Lyon, International Agency for Research on Cancer. IARC Scientific Publication No. 75.

Paul A.A. and Southgate D.A.T. (1992). *McCance and Widdowson's, the Composition of Foods*. HMSO, London.

Payne C. (1985). The Glim System. Release 3.77. NAG, Oxford.

Pekkarinen M. (1970). Methodology in the collection of food consumption data. *World Review of Nutrition and Dietetics*, **12**: 145-171.

Pellet P.L. and Shadarevion S. (1970). *Food composition Tables for Use in the Middle East*. American University of Beirut, Lebanon.

Pertri A. (1987). *Lecture Notes on Medical Statistics*. 2nd ed., Blackwell Scientific Pub., UK: 206.

Piatti G., Borella F., Berti M.A. and Braga P.C. (1993). Delaying effects of dietary

eicosapentaenoic-docosahexaenoic acids on development of "fatty streaks" in hypercholesterolaemic rabbits. A morphological study by scanning electron microscopy. *Drugs Exp Clin Research.* **19** (4): 175-181.

Pocock S.J., Cook D.G., Shaper A.G., Phillips A.N. aand Walker A.N. (1987). Social class differences in ischaemic heart disease in British men. *The Lancet*, **2**: 197-201.

Porter M.W., Yamanaka W., Carlson S. and Flymm M.A. (1977). Effects of dietary egg on serum cholesterol and triglyceride of human males. *The American Journal of Clinical Nutrition*, **30**: 490-495.

Puhakainen I., Ahola I. and Yki-Jarvinen H. (1995). Dietary supplementation with n-3 fatty acids increases gluconeogenesis from glycerol but not hepatic glucose production in patients with non-insulin-dependent diabetes mellitus. *American Journal of Clinical Nutrition.* 61: 121-126.

Pyorala K. (1987). Dietary cholesterol in relation to plasma cholesterol and coronary heart disease. *The American Journal of Clinical Nutrition*, **45**: 1176-1184.

Rahimtoola S.H. (1994). The definition of ischaemia. *Cardiovascular Research.* **28**: 1745-1746.

Rahmanifar A. and Bond JT. (1990). Food consumption, iron intake and dietary patterns of urban pregnant women from different socioeconomic population in central Iran. *Ecology of Food and Nutrition*, **24**: 97-114.

Rao M. (1987). Ecological factors and nutritional status of nursery school children in rural Dharwad, India. *Ecology of Food and Nutrition*, **19**: 323-332.

Rauraman R. (1984). Relationship of physical activity, glucose tolerance, and weight management. *Preventive of Medicine*, **13** (1): 37-46.

Rigotti NA., Thomas G.S. and Leaf A. (1983). Exercise and coronary heart disease. *Annual Review of Medicine*, **34**: 391-412.

Riyadh News Papers (1991). 22nd Aprel.

Riyadh News Papers (1991). No. 8463.

Renaud S. and Lorgeril M. (1992). Wine, alcohol, platelet, and the French paradox for

coronary heart disease. *Lancet*, **339** (8808): 1523-1526.

Roberts S.L., McMurray M.P. and Connor W.E. (1981). Does egg feeding (i.e., dietary cholesterol) affect plasma cholesterol levels in humans? The results of a double-blind study. *The American Journal of Clinical Nutrition*, **34**: 2092-2099.

Roman E., Beral V. and Inskip H. (1984). A comparison of standardised and proportional mortality rations. *Statistics in Medicine*. **3**: 7-14.

Rosenman R.H., et al (1975). Coronary Heart Disease in Western Collaborative Group Study: final follow-up experience of 8.5 years. *The Journal of the American Medical Association*, **233**: 872-877.

Ruderman N.B. and Schneider S.H. (1992). Diabetes, exercise and atherosclerosis. *Diabetes Care*. **15** (11): 1787-1793.

Saeed A.A.W., Taha A.M. and Al-Shahran A.H. (1989). Smoking habits of physicians in Riyadh, Saudi Arabia. *Saudi Medical Journal*, **10** (6): 508-511.

Salonen J.T., Yla-Herttuala S., Yamamoto R., Yla-Herttuala S., Yamamoto R., Butler S., Korpela H., Salonen R., Nyyssonen K., Plainski W. and Witzyum J.L. (1992). Autoantibody against oxidised LDL and progression of carotid atherosclerosis. *The Lancet*, **339**: 883-887.

Sanders T.A.B. (1988). Essential and trans fatty acids in nutrition. *Nutrition Research Reviews*, **1**: 57-78.

Saris W.H. (1988). The assessment and evaluation of daily physical activity in children. A review. *Acta Paediatrica Scandinavica*, **318**: 37-48.

Sawaya W.N., Al-Jebrin A., Salji J.P., Ayaz M. and Khalil J.K. (1985). Cholesterol contents and fatty acid composition of selected Saudi Arabian Dishes. *Nutrition Reports in International*, **31** (3): 593-600.

Sawaya W.N., Al-Jebrin A., Salji J.P., Ayaz M. and Khalil J.K. (1986). Nutritional evaluation of selected meat based on Saudi Arabian dishes. *Ecology of Food and Nutrition*, **18**: 171-182.

Sawaya W.N., Khalil J.K., Khatchadourian J.K. and Al-Mohamed M.M. (1984). Nutritional evaluation of various breads consumed in Saudi Arabia. *Nutrition Reports in International*, **29**: 1161-1170.

Sawaya W.N., Tannous R.I. and Othaimeen A.I. (1987). Nutrients intake of Saudi infants and pre-school children by meal of the day. *Nutrition Reports in International*, **35** (2): 233-250.

Schaefer E.J., Levy R.J., Ernst N.D., Van Sant F.D. and Brewer H.B (1981). The effects of low cholesterol, high polyunsaturated fat, and low fat diets on plasma lipid and lipoprotein cholesterol levels in normal and hypercholesterolemic subjects. *The American Journal of Clinical Nutrition*, **34**: 1758.

Scragg R., (1990). Myocardial infarction is inversely associated with plasma 25-hydroxyvitamin $D_3$ levels: a community-based study. *International Journal of Epidemiology*, **19**: 559-563.

Sebai A.Z. (1983). *The Health of the Family in a Changing Arabia: A case study of primary health care*. 3th ed., Tihama Pub. Saudi Arabia.

Secretary of State for Health (1991). *Health of the Nation*. London. HMSO.

Sedrani S.H. (1986). Are Saudi at risk of developing vitamin D deficiency. *Saudi Medical Journal*, **7** (5): 427-432.

Semplicini A., and Valle R., (1994). Fish oils and their possible role in the treatment of cardiovascular diseases. *Pharmacology and Therapeutics*. **61** (3): 385-397.

Serenius F. (1981). Health and nutritional status in rural Saudi Arabia. *Saudi Medical Journal*, **2** (1): 10-22.

Shaper A.G. (1987). Environmental factors in coronary heart disease: diet. *Europe Heart Journal*, **8** (E): 31-38.

Shekelle R.B. and Stamler J. (1989). Dietary cholesterol and ischaemic heart disease. *The Lancet*, **1** (8648): 1177-1179.

Shklar G., Schwartz J., Trickler D. and Cheverie S.R. (1993). The effectiveness of a mixture of beta-carotene, alpha-tocopherol, glutathione and ascorbic acid for cancer prevention. *Nutrition Cancer*. **20** (2): 145-151.

Sinha NP., Khan M.Y. and Elsaiyad E.M. (1985). Health education programme and it's evaluation in Saudi Arabia. *Journal of Royal Society of Health*, **5**: 163-164.

Slatter G., Mead J., Dhopeshwarkar G., Robinson S and Alfin-Slater R.B. (1991). Plasma cholesterol and triglycerides in men with added eggs in the diet. *Nutrition Reports International*, **14**: 249-260.

Slatter G., et al (1991). Plasma cholesterol and triglycerides in men with added eggs in the diet. *Nutrition Reports International*, **14**: 249-260.

Slattery M.L. and Randall D.E. (1988). Trends in coronary heart disease mortality and food consumption in the United States between 1909 and 1980. *The American Journal of Clinical Nutrition*, **47**: 1060-1067.

Slattery M.L., Jacobs D.R., Hilner J.E., Caan B.J., van Horn L., Bragg C., Manolio T.A., Kushi L.H. and Liu K. (1991). Meat consumption and its associations with other diet and health factors in young adults: the CARDIA study. *The American Journal of Clinical Nutrition*, **54**: 930-935.

Soyland E., Funk J., Rajka G., et al. (1993). Effect of dietary supplementation with very-long-chain n-3 fatty acids in patients with psoriasis. *New England Journal of Medicine*. **328**: 1812-1816.

Sparrow C.P., Parthasarathy S. and Steinberg D. (1989). A macrophage receptor that recognize oxidised low density lipoprotein but not acetylated low density lipoprotein. *Journal of Biological Chemistry*. **264**: 2599-2604.

Spector W.G. (1980). *An Introduction to General Pathology*. 2ed ed., Churchil Livingston, UK: 128.

Stallones R.A. (1983). Ischaemic heart disease and lipids in blood and diet. *Annual Review of Nutrition*, **3**: 155-185.

Stary H.C., Schwartz C.J., Wagner W.D. and Wissler R.W. (1994). A definition of inial, fatty streak, and intermediate lesions of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Atherosclerosis, American Heart Association. *Arterioscler Thromb*. **14** (5): 840-856.

Steinberg D. and workshop participants (1992). Antioxidants in the prevention of human atherosclerosis- summery of the proceedings of a National Heart, Lung and Blood Institute workshop, September 5-6, 1991, Bethesda, Maryland. *Circulation*, **85**: 2338-2344.

Steinberg D., Parthasarathy S., Carew T.E., Khoo T.C., Witztum J.L. (1989). Beyond cholesterol: modifications of low-density lipoprotein that increase its atherogenicity. *New England Journal of Medicine*. **320**: 915-924.

Stock M. and Rothwell N. (1982). *Obesity and Leanness Nastic Aspects*. John Wiley & Sons, Inc., USA: 78.

The Coronary Prevention Group (1993). *Coronary Heart Disease Statistics 1991*. British Heart Foundation Statistics Database. UK., 1991.

The Meddle East and North Africa, Regional Survey of the World 1991/1992 (1993). 38th Ed., London/Europa Pub. Ltd., London: 763-797.

Thomas L.H., Winter J.A. and Scott R.G. (1983). Concentration of trans unsaturated fatty acids in the adipose body tissue of decedents dying of ischaemic heart disease compared with controls. *Journal of Epidem Common Health*, **37**: 22-24.

Thomson N.J. (1991). Recent trends in Aboriginal mortality. *The Medical Journal of Australia*, **154**: 235-239.

Thompson R.L., Pyke S., Scott E.A., Thompson S.G. and Wood D.A. (1993). Cigarette smoking, polyunsaturated fats, and coronary heart disease. *Ann N Y Acad Sci*. **686**: 130-138.

Tisdal M.J., and Dhesi J.K., (1990). Inhabitation of weight loss by w-3 fatty acids in an experimental cachexia model. *Cancer Research*. **50**: 5022-50-26.

Turini M.E., Powell W.S., Behr S.R., Holub B.J., (1994). Effects of a fish-oil and vegetable-oil formula on aggregation and ethanolamine-containing lysophospholipid generation in activated human platelet and on leukoriene production in stimulated neutrophils. *Journal of Clinical Nutrition*. **60**: 717-724.

Uemura K. and Pisa Z. (1988). Trend in cardiovascular disease mortality in industrialised countries since 1950. *World Health Stat Q*; **41**: 155-78.

Valenzuela R.E., Florenico C.A. and Guthrie H.A. (1979). Distribution of nutrients within the Filipino family. *Nutrition Reports International*, **19** (4): 573-581.

Van Staveren W.A. and Burema J. (1990). Dietary methodology: implication of errors in the measurement. *Proceedings of the Nutrition Society*, **49**: 281-287.

Walker W.J. (1989). Changing U.S. lifestyle and declining vascular mortality - a retrospective. *New England Journal of Medicine*. **308**: 649-651.

Walker A.R.P. (1975). Sucrose, hypertension and heart diseases (letter). *American Journal of Clinical Nutrition*, **28**: 195-196.

Wenlock R.W., Buss D.H. and Derry B.J. (1980). Household food wastage in Britain. *British Journal Nutrition*, **43**: 53-70.

Wheeler E.F. (1991). Intra-household food and nutrient allocation. *Nutrition Research Reviews*, **4**: 69-81.

Wieland E., Brandes A., Armstrong V.W. and Oellerich M. (1993). *Eur Journal Chem Clin Biochem*. **31** (11): 725-731.

Willett W.C. (1990). Nutritional Epidemiology. Oxford University Press. New York.

Witzum D.L., (1994). The oxidation hypothesis of atherosclerosis. *Lancet*. **344**: 793-795.

Weinbaum S. and Chien S. (1993). Lipid transport aspects of atherogenesis. *Journal Biomech Eng*. **115** (4B): 602-610.

Willett W.C., Stampfer M.J., Manson J.E., Colditz G.A., Speizer F.E., Rosner B.A., Sampson L.A., Henneken C.A., (1993). Intake of trans fatty acids and risk of coronary heart disease among women. *The Lancet*. **341**: 581-585.

Woodhouse N.J.Y. and Norton W.L. (1982). Low vitamin D levels in Saudi Arabians. *King Faisal Specialist Hospital Medical Journal*, **2** (3): 127-131.

Woodward M., Shewry M.C., Smith W.C.S. and Tunstall-Pedoe H. (1990). Coronary heart disease and socio-economic factors in Edinburgh and North Glasgow. *The Statistician*, **39**: 319-329.

World Health Organisation (1985). Joint FAO/WHO/UNU report. *Energy and protein requirements*. Geneva: 133-137.

World Health Organisation (1984). *World Health Statistics Annual*, Geneva: 190-350.

World Health Organisation (1977). *International Classification of Diseases*. Manual of the international statistical clarification of disease, injuries and causes of death. World Health Organisation, Geneva, **1**: 259-282.

Yudkin J. (1951). Dietary surveys: variation in the weekly intake of nutrients. *British Journal of Nutrition*, **5**: 177-194.

Yuichiro G. (1992). Cardiovascular system and nutrition in the aging process. *Nutrition Reviews*, **50** (12): 398-401.

Zahran F.M., Ardawi M.S.M and Al-Fayez S.F. (1985). Carboxyhaemoglobin concentrations in smokers of sheesha and cigarettes in Saudi Arabia. *British Medical Journal*, **291**: 1768-1770.

Zahran F.M., Yousef A. and Baig M.H. (1982). A study on carboxyhaemoglobin level in cigarette and sheesha smokers in Saudi Arabia. *American Journal of Public Health*, **72**: 722-724.

# *APPENDICES*

# APPENDIX A

## HOUSEHOLD FOOD CONSUMPTION SURVEY

### Dear Resident (s)

This survey is an important study concerning the food consumption and dietary habits in the area. Therefore, the information you give about your daily food consumption (i.e. purchases and eaten) is a great contribution to achieve this work, and the result will be of significant benefits to our society.

You have been selected to contribute among many families who were chosen from all over the Eastern Province where this survey takes place.

Any information given will be treated in strict confidence and used for statistical purposes only. It will not be used in any way in which it can be identified with you. Your co-operation will give the actual image of the responsibility you have toward others and will be highly appreciated by us.

*The researcher*

### Note:

**If you have any inquiry about the survey, please do not hesitate to contact me on the following telephone number any time: . . . . . .**

APPENDIX (A)

**FOOD SURVEY**
**Household Food Consumption**
(Form A)

*To be completed at first interview*

---

A) Region ☐ B) Area ☐

C) Area type ☐ D) Serial No. Of Household ☐

E) Date of first interview ☐☐☐

---

F) Do you have a refrigerator?

Yes ☐ No. ☐

G) Do you have a deep freezer?

Yes ☐ No. ☐

H) Do you grow any of your vegetables or fruit?

Yes ☐ No. ☐

*If 'yes'*

a) Where ?

Garden ☐ Farm ☐

b) Have you a store of home grown vegetables or fruits ?

| | Yes | No |
|---|---|---|
| Dates | Y | X |
| Tomatoes | Y | X |
| Onions | Y | X |
| Okra | Y | X |
| Beans | Y | X |
| Any other specify | Y | X |
| | ..... | |
| | ..... | |
| | ..... | |
| | ..... | |
| None at all | Y | X |

I) Do you keep hens, goats, cows or camels ?

Yes ☐ No. ☐

If 'yes' are they kept for eggs, meat, milk or others?

Egg ☐

Meat ☐

Milk ☐

Others ☐

J) Do you have a store of honey from your own hives ?

Yes ☐ No. ☐

K) Do you catch any fish to eat ?

Yes ☐ No. ☐

L) Do you buy any food in bulk as a store ?

Yes ☐ No. ☐

*If 'yes'*

*Which food do you buy in this way ?*

Rice ..... 1
Meat ..... 2
Flour ..... 3
Oil ..... 4
Potatoes ..... 5
Tomatoes ..... 6
Onions ..... 7
Dates ..... 8
Any other ..... 9
specify .....
.....
.....
.....
.....

M) Is this house

Owns ☐ Others ☐

N) The total family monthly income!

| | |
|---|---|
| <3000 ..... | 1 |
| 3,001 to 5,000 ..... | 2 |
| 5,001 to 7,000 ..... | 3 |
| 7,001 to 9,000 ..... | 4 |
| 9,001 to 11,000 ..... | 5 |
| 11,001 to 13,000 ..... | 6 |
| 13,001 to 15,000 ..... | 7 |
| 15,001 to 17,000 ..... | 8 |
| 17,001 to 19,000 ..... | 9 |
| 19,001 to 21,000 ..... | 10 |
| 21,001 to 23,000 ..... | 11 |
| 23,001 to 25,000 ..... | 12 |
| 25,001 to 29,000 ..... | 13 |
| >= 30,000 ..... | 14 |
| No. information Gevin ..... | 15 |

---

FOR FAMILY THAT HAVE BABY UNDER ONE YEAR

O) What type of milk you give your baby

1 Breast-fed
2 Bottle-fed (specify) .....
3 Both

If 'No.2' How much per day?
.....
.....
.....

| Age year (s) | Sex M | Sex F | Marital status | Relationship to head of the household | Education | Occupation | Monthly income Salary | Monthly income Others |
|---|---|---|---|---|---|---|---|---|
| Adult 18 & + | | | | | | | | |
| Youth 11-17 | | | | | | | | |
| Child 5-10 | | | | | | | | |
| Infant 1-4 | | | | | | | | |
| Infant <1 | | | | | | | | |
| Total | | | Note:- | | | | | |
| | | | | | | | Total | |

APPENDIX (B)

**FOOD SURVEY**
**HOUSEHOLD FOOD CONSUMPTION**

All the particulars you give on this form will be treated in STRICT CONFIDENCE.
Please do not put your name or address on it.

*How to use this booklet*

1) There are two pages for each day.
2) On the first of the two pages please write down all the items of food coming into the house on that day.
3) On the second page please show what was eaten at each meal and who ate the meal.

Region

Area

Area type

Serial number

Household Composition

| SEX | AGE |
|---|---|
| | |
| | Total persons |

Visitor Composition

| SEX | AGE |
|---|---|
| | |
| | Total persons |

## REMINDER ABOUT RECORDING WHAT YOU BUY

**Please follow up all the points:**

- It will help us very much if you make sure that you write down all the foods you purchased on the day you buy it.

- In each case we should like to know, if possible, the exact amount or weight (in kilos and grams or litres, and the number of items) for example, if you buy four containers of fresh milk each of which is one litre, write in the table 4 x 1 litre.

- Please put down details of the type of food, for example if you buy meat, state whether it is mutton, goat, beef. For bread, state if it is Arabic bread, large or small, or foreign bread, and so on with other type of the food.

- The name of package products is very useful. For example, fresh milk Almaraiy product and so on with other type of the food.

- The more details you can give, the more helpful it is to us.

- An example of the detail we would like to know, if possible, is given opposite.

- We realise that you may not buy the same items as we have listed.

***We have simply shown as many example of different foods as we can so that you are clear about the details we need.***

## FOOD COMING INTO THE HOUSE

DAY .....

| GRAMS KILOS OR LITRES | DESCRIPTION OF FOOD Please describe item in full and give brand. Use one line for each item. | PLEASE LEAVE BLANK | |
|---|---|---|---|
| | | Food code | Food quantity |
| 1 lit | milk (Al-Marai products) | | |
| 2 kg | Meat (lamb), raw, Fresh | | |
| 6X500 | Chicken, raw Fresh | | |
| 2 X 30-g | Butter (... products) | | |
| 25X250g | Orange, Juices, Canned | | |

## HOME GROWN FOOD, GIFTS

| GRAMS KILOS OR LITRES | DESCRIPTION OF FOOD | SOURCE Garden, farm, employer own business. | PLEASE LEAVE BLANK | |
|---|---|---|---|---|
| | | | Food code | Food quantity |
| 2kg | Potatoes | Farm | | |
| 1 kg | Onions | Garden | | |
| 500g | Dates, Fresh | Friend | | |

## REMINDER ABOUT RECORDING WHAT YOU SERVED EVERY DAY AT HOME

- **Note in section (A)**

Please write a detailed description of each meal served at home.

- **Section (B)**

Record the total number of people eating it (member of the family + visitors).

- **Section (C)**

Record the number of visitors who were served and also their age and sex.

- **Section (D)**

The age and sex every member of the family who did not have their meal at home, also note the place where they had it, (i.e. whether at work, at school, with friends, etc).

- **Section (E)**

Record the age and sex of any member of your household has a packed meal in section (F).

- **Section (G)**

Record the age and sex of any member of the family who do not have their meal at home for any reason such as young age, illness, etc.

*Please see example opposite.*

**MEALS**

| | 1 | 2 | 3 | 4 |
|---|---|---|---|---|
| | Breakfast | Mid-day meal | Evening meal | Other food drink or snacks. |
| (A) What did you serve? | Eggs boild<br>Arabaic bread<br>Tea.<br>Sugar | Rice<br>Meat beef<br>Dates<br>Salad<br>Tea | Chicken, Fried<br>Dates<br>Salal<br>Bread.<br>Tea. | Tea<br>Milk<br>Orange |
| (B) How many of the people on the front of this book ate the meal home? | Number of people 8 | Number of people 6 | Number of people 5 | Number of people 2 |
| (C) Did you have visitors for this meal?<br>MALE = M<br>FEMALE = F | Visitors to breakfast<br>SEX: M, F<br>AGE: 40, 35 | Visitors to mid-day meal<br>SEX<br>AGE | Visitors to evening meal<br>SEX<br>AGE | Visitors to snacks<br>SEX<br>AGE |
| (D) Did anyone obtain a meal out? (For which you did not provide the food) Where was it eaten? | Breakfast out<br>S \| A \| Where | Mid-day meal out<br>S \| A \| Where | Evening meal out<br>S \| A \| Where | Snack out<br>S \| A \| Where |
| (E) Did anyone take a packed meal from home to eat out?<br>MALE = M<br>FEMALE = F | Packed breakfast<br>SEX \| AGE | Packed mid-day meal<br>SEX \| AGE | Packed evening meal<br>SEX \| AGE | Packed Snacks<br>SEX \| AGE |
| (F) Contents of a packed meal? | | | | |
| (G) Who did not have a meal<br>MALE = M<br>FEMALE = F | | | M = 30 | M = 30<br>F = 40<br>M = 12<br>N = 16 |

# Appendix C

## Contribution made by food groups to energy content of the Saudi's diet

| Food Items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total cereal & cereal prod.** | **37.44** | **38.26** | **41.36** | **38.25** |
| Rice | 18.19 | 30.06 | 31.01 | 24.06 |
| Bread | 10.51 | 6.04 | 3.16 | 8.08 |
| Flour | 5.77 | 1.28 | 7.19 | 4.59 |
| Others | 2.25 | 0.89 | 0 | 1.51 |
| **Total fruits** | **11.43** | **21.29** | **9.20** | **14.14** |
| Dates | 3.62 | 16.27 | 5.11 | 7.72 |
| Oranges | 1.05 | 0.92 | 0.63 | 0.95 |
| Apples | 1.66 | 1.44 | 0.91 | 1.48 |
| Bananas | 1.44 | 0.99 | 1.08 | 1.25 |
| Other fruits | 3.65 | 1.66 | 1.47 | 2.73 |
| **Total vegetables** | **5.52** | **3.85** | **2.86** | **4.63** |
| **Total meat & poultry** | **12.02** | **9.93** | **10.74** | **11.19** |
| Meat | 7.10 | 6.86 | 5.25 | 6.76 |
| Chicken | 4.92 | 3.08 | 5.49 | 4.43 |
| **Total fish** | **1.16** | **1.26** | **0.25** | **1.06** |
| **Eggs** | **1.64** | **1.17** | **0.69** | **1.36** |
| **Total milk & diary products** | **4.10** | **4.42** | **4.13** | **4.20** |
| **Total oils and Fats** | **9.04** | **5.37** | **8.91** | **7.89** |
| **Sugar** | **16.69** | **14.35** | **21.34** | **16.64** |
| **Miscellaneous** | **0.96** | **0.09** | **0.52** | **0.64** |

# Appendix D

## Contribution made by food groups to protein of the Saudi's diet

| Food Items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total cereal & cereal prod.** | **33.33** | **34.71** | **39.01** | **34.47** |
| Rice | 13.68 | 24.94 | 25.77 | 18.47 |
| Bread | 10.31 | 6.80 | 3.56 | 8.42 |
| Flour | 6.76 | 1.72 | 9.68 | 5.71 |
| Others | 2.58 | 1.26 | 0 | 1.86 |
| **Total fruits** | **4.06** | **6.56** | **3.26** | **4.67** |
| Dates | 0.80 | 4.16 | 1.36 | 1.83 |
| Other fruits | 3.26 | 2.40 | 1.90 | 2.84 |
| **Total vegetables** | **5.31** | **4.25** | **3.43** | **4.76** |
| **Total meat & poultry** | **35.18** | **30.92** | **40.68** | **34.69** |
| Meat | 11.15 | 13.67 | 9.86 | 11.69 |
| Chicken | 24.03 | 17.25 | 30.82 | 23.00 |
| **Total fish** | **9.06** | **11.21** | **2.28** | **8.78** |
| **Eggs** | **4.41** | **3.62** | **2.14** | **3.89** |
| **Total milk & diary prod.** | **7.58** | **8.55** | **8.10** | **7.92** |
| Milk, whole | 2.15 | 1.79 | 3.06 | 2.17 |
| Milk, powder | 0.74 | 2.52 | 4.60 | 1.75 |
| Laban | 1.98 | 2.67 | 0.25 | 1.94 |
| Cheese | 2.39 | 1.16 | 0.19 | 1.75 |
| Others | 0.32 | 0.42 | 0 | 0.31 |
| **Miscellaneous** | **1.08** | **0.19** | **1.10** | **0.83** |

# Appendix E

**Contribution made by food items to carbohydrate content of Saudi's diet.**

| Food Items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total cereal & cereal prod.** | **48.52** | **46.17** | **50.63** | **48.06** |
| Rice | 25.56 | 36.80 | 39.04 | 31.19 |
| Bread | 13.39 | 6.97 | 3.75 | 9.88 |
| Flour | 6.75 | 1.35 | 7.84 | 5.15 |
| Others | 2.82 | 1.05 | 0 | 1.83 |
| **Total fruits** | **15.76** | **26.93** | **11.98** | **18.84** |
| Dates | 5.07 | 20.64 | 6.66 | 10.37 |
| Oranges | 1.51 | 1.20 | 0.85 | 1.31 |
| Apples | 2.25 | 1.77 | 1.14 | 1.93 |
| Bananas | 2.29 | 1.42 | 1.61 | 1.91 |
| Other fruits | 4.64 | 1.9 | 1.72 | 3.32 |
| **Total vegetable** | **5.95** | **3.92** | **2.83** | **4.83** |
| Potatoes | 2.55 | 1.50 | 0.78 | 1.95 |
| Tomatoes | 0.80 | 0.40 | 0.31 | 0.60 |
| Other vegetables | 2.60 | 2.01 | 1.74 | 2.29 |
| **Total milk & diary prod.** | **1.56** | **1.69** | **1.79** | **1.63** |
| **Sugar** | **27.12** | **21.12** | **32.29** | **25.93** |
| **Miscellaneous** | **1.10** | **0.18** | **0.49** | **0.71** |

# Appendix F

## Contribution made by food groups to fibre content of the Saudi's diet

| Food items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Cereal & cereal prod.** | **24.74** | **21.27** | **34.78** | **24.63** |
| Rice | 11.70 | 17.09 | 26.06 | 15.05 |
| Bread | 6.53 | 3.46 | 2.74 | 5.09 |
| Flour | 3.53 | 0.72 | 5.97 | 2.84 |
| Others | 2.98 | 0 | 0 | 1.66 |
| **Total fruits** | **40.80** | **58.40** | **38.68** | **46.53** |
| Dates | 9.87 | 40.66 | 18.68 | 21.21 |
| Oranges | 3.85 | 3.09 | 3.08 | 3.51 |
| Apples | 6.57 | 5.26 | 4.88 | 5.95 |
| Bananas | 3.09 | 1.95 | 3.15 | 2.71 |
| Grapes | 2.59 | 1.00 | 2.40 | 2.03 |
| Watermelon | 3.73 | 1.48 | 1.76 | 2.76 |
| Other fruits | 11.11 | 4.97 | 4.74 | 8.36 |
| **Total vegetables** | **31.43** | **19.77** | **25.00** | **26.81** |
| Potatoes | 3.43 | 2.04 | 1.52 | 2.76 |
| Tomatoes | 7.07 | 3.60 | 4.02 | 5.57 |
| Other vegetables | 20.93 | 14.14 | 19.46 | 18.48 |
| **Miscellaneous** | **3.18** | **0.56** | **1.54** | **2.12** |

# Appendix G

## Contribution made by food groups to fat content of the Saudi's diet

| Food Items | Urban | Rural | Bedouin | All population |
|---|---|---|---|---|
| **Total cereal & cereal prod.** | **5.19** | **6.31** | **5.86** | **5.57** |
| Rice | 2.24 | 4.68 | 4.18 | 3.13 |
| Bread | 1.49 | 1.13 | 0.53 | 1.26 |
| Flour | 0.81 | 0.24 | 1.15 | 0.71 |
| Others | 0.66 | 0.26 | 0 | 0.46 |
| **Total fruits** | **2.14** | **3.23** | **1.6** | **2.34** |
| **Total vegetables** | 2.56 | **1.77** | **1.20** | **2.17** |
| **Total meat & poultry** | **33.53** | **38.03** | **31.60** | **34.42** |
| Meat | 25.18 | 31.15 | 20.98 | 26.13 |
| Chicken | 8.35 | 6.88 | 10.62 | 8.28 |
| **Total fish** | **0.26** | **0.40** | **0.06** | **0.27** |
| **Eggs** | **4.87** | **4.58** | **2.34** | **4.45** |
| **Total milk & diary prod.** | **9.63** | **13.50** | **10.32** | **10.72** |
| Milk, whole | 2.26 | 2.16 | 3.19 | 2.37 |
| Milk, powder | 0.98 | 3.82 | 6.04 | 2.41 |
| Laban | 1.97 | 3.06 | 0.25 | 2.01 |
| Cheese | 3.68 | 3.85 | 0.84 | 3.33 |
| Others | 0.73 | 0.61 | 0 | 0.60 |
| **Total oils and fats** | **41.07** | **32.15** | **46.16** | **39.48** |
| Vegetable oils | 34.11 | 24.31 | 42.68 | 32.78 |
| Olive oils | 0 | 4.67 | 3.48 | 1.68 |
| Butter | 6.96 | 3.17 | 0 | 5.02 |
| **Miscellaneous** | **0.75** | **0.02** | **0.86** | **0.58** |

# Appendix I

**Household energy and other ntreints intake invstigated (grams per household per**

## *1. Urban Households* :

| *Male* | *Female* | *Total* | *Energy* | *Proten* | *Fat* | *CHO* | *Fibre* |
|---|---|---|---|---|---|---|---|
| 8 | 7 | 15 | 21989 | 1594 | 575 | 2471 | 55 |
| 6 | 7 | 13 | 91833 | 3236 | 3003 | 12638 | 28 |
| 8 | 10 | 18 | 5052 | 187 | 89 | 823 | 51 |
| 8 | 6 | 14 | 13699 | 409 | 301 | 2368 | 77 |
| 7 | 5 | 12 | 7672 | 259 | 210 | 1136 | 77 |
| 7 | 5 | 12 | 16669 | 568 | 656 | 2026 | 33 |
| 4 | 3 | 7 | 4484 | 123 | 310 | 295 | 2 |
| 5 | 8 | 13 | 20247 | 1061 | 519 | 2605 | 25 |
| 6 | 3 | 9 | 19284 | 989 | 354 | 2879 | 95 |
| 7 | 2 | 9 | 45126 | 1433 | 3466 | 1856 | 94 |
| 3 | 8 | 11 | 100658 | 2023 | 1559 | 20097 | 21 |
| 2 | 2 | 4 | 26353 | 1778 | 1445 | 1371 | 20 |
| 4 | 1 | 5 | 111468 | 2511 | 1051 | 23531 | 12 |
| 8 | 6 | 14 | 20461 | 1043 | 360 | 3127 | 56 |
| 3 | 4 | 7 | 12955 | 371 | 161 | 2448 | 81 |
| 1 | 2 | 3 | 17514 | 903 | 279 | 2792 | 10 |
| 7 | 6 | 13 | 42835 | 906 | 257 | 9098 | 14 |
| 7 | 5 | 12 | 12380 | 413 | 116 | 2322 | 88 |
| 5 | 5 | 10 | 5948 | 256 | 41 | 1128 | 16 |
| 19 | 18 | 37 | 16089 | 363 | 170 | 3072 | 15 |
| 5 | 4 | 9 | 63190 | 694 | 262 | 15191 | 37 |
| 8 | 9 | 17 | 10444 | 419 | 209 | 1627 | 55 |
| 3 | 3 | 6 | 13758 | 1500 | 439 | 846 | 25 |
| 10 | 6 | 16 | 44122 | 1330 | 1343 | 6556 | 21 |
| 11 | 8 | 19 | 121746 | 4329 | 3051 | 19105 | 26 |
| 10 | 9 | 19 | 21025 | 1054 | 240 | 3523 | 11 |
| 6 | 4 | 10 | 6497 | 437 | 160 | 687 | 7 |
| 4 | 4 | 8 | 104961 | 2736 | 2561 | 17597 | 31 |
| 9 | 6 | 15 | 58602 | 487 | 1335 | 11692 | 80 |
| 8 | 4 | 12 | 12124 | 310 | 634 | 1248 | 22 |
| 10 | 11 | 21 | 26145 | 1508 | 461 | 3688 | 23 |
| 2 | 6 | 8 | 19778 | 902 | 808 | 2074 | 89 |
| 2 | 5 | 7 | 86571 | 1086 | 3979 | 12190 | 51 |
| 1 | 2 | 3 | 4335 | 124 | 34 | 834 | 22 |
| 2 | 2 | 4 | 3991 | 201 | 66 | 607 | 28 |
| 3 | 2 | 5 | 8360 | 410 | 175 | 1239 | 24 |
| 4 | 6 | 10 | 3300 | 219 | 36 | 515 | 4 |
| 7 | 5 | 12 | 39345 | 850 | 2586 | 2955 | 15 |
| 8 | 9 | 17 | 5233 | 168 | 156 | 766 | 11 |
| 3 | 3 | 6 | 12282 | 411 | 130 | 2267 | 10 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 4 | 1 | 5 | 2511 | 132 | 84 | 296 | 2 |
| 6 | 7 | 13 | 26028 | 393 | 95 | 5707 | 22 |
| 3 | 2 | 5 | 4499 | 155 | 214 | 475 | 12 |
| 5 | 3 | 8 | 17698 | 1057 | 298 | 2606 | 95 |
| 3 | 5 | 8 | 30845 | 1228 | 922 | 5368 | 46 |
| 4 | 3 | 7 | 47821 | 1385 | 393 | 9538 | 86 |
| 6 | 3 | 9 | 47468 | 259 | 97 | 12047 | 29 |
| 3 | 5 | 8 | 20101 | 366 | 1370 | 1544 | 21 |
| 3 | 4 | 7 | 1317 | 35 | 16 | 249 | 11 |
| 4 | 5 | 9 | 55202 | 752 | 3975 | 4176 | 77 |
| 2 | 2 | 4 | 13379 | 706 | 841 | 675 | 43 |
| 3 | 2 | 5 | 8545 | 344 | 683 | 214 | 3 |
| 3 | 6 | 9 | 168000 | 998 | 1535 | 111 | 40 |
| 2 | 3 | 5 | 3152 | 88 | 25 | 618 | 38 |
| 7 | 5 | 12 | 113260 | 2678 | 14 | 20675 | 14 |
| 4 | 3 | 7 | 9001 | 597 | 230 | 1039 | 57 |
| 4 | 2 | 6 | 1864 | 1504 | 447 | 2094 | 88 |
| 5 | 3 | 8 | 24560 | 1368 | 821 | 2679 | 15 |
| 1 | 2 | 3 | 4367 | 189 | 376 | 784 | 38 |
| 7 | 4 | 11 | 86086 | 2243 | 266 | 18232 | 18 |
| 11 | 7 | 18 | 12481 | 803 | 412 | 1299 | 48 |
| 8 | 3 | 11 | 3393 | 616 | 392 | 7172 | 10 |
| 6 | 3 | 9 | 7423 | 255 | 93 | 1379 | 15 |
| 1 | 3 | 4 | 32376 | 1088 | 1228 | 4086 | 12 |
| 6 | 5 | 11 | 51366 | 1403 | 162 | 10444 | 17 |
| 5 | 4 | 9 | 52474 | 1457 | 308 | 10642 | 89 |

## *1. Rural Households :*

| *Male* | *Female* | *Total* | *Energy* | *Proten* | *Fat* | *CHO* | *Fibre* |
|---|---|---|---|---|---|---|---|
| 4 | 2 | 6 | 11231 | 338 | 82 | 2363 | 23 |
| 8 | 6 | 14 | 168830 | 2593 | 2796 | 32557 | 78 |
| 3 | 3 | 6 | 41904 | 1159 | 265 | 8585 | 14 |
| 6 | 6 | 12 | 17573 | 915 | 403 | 4833 | 22 |
| 7 | 5 | 12 | 10849 | 438 | 153 | 1865 | 42 |
| 3 | 4 | 7 | 71502 | 1640 | 306 | 15468 | 15 |
| 2 | 2 | 4 | 10421 | 715 | 270 | 1237 | 76 |
| 4 | 8 | 12 | 69346 | 1856 | 2422 | 10175 | 80 |
| 8 | 8 | 16 | 48268 | 1034 | 1149 | 8477 | 12 |
| 5 | 2 | 7 | 63749 | 2436 | 1766 | 8998 | 30 |
| 7 | 5 | 12 | 20282 | 1083 | 584 | 2604 | 28 |
| 1 | 1 | 2 | 6362 | 379 | 218 | 705 | 13 |
| 9 | 6 | 15 | 106985 | 2410 | 2291 | 19472 | 20 |
| 2 | 1 | 3 | 17437 | 555 | 776 | 2002 | 59 |
| 7 | 3 | 10 | 24518 | 2015 | 1056 | 1592 | 25 |
| 7 | 5 | 12 | 29865 | 1868 | 1510 | 2017 | 36 |
| 6 | 4 | 10 | 89865 | 1697 | 972 | 18155 | 38 |
| 1 | 1 | 2 | 102206 | 1889 | 1517 | 19977 | 40 |
| 5 | 4 | 9 | 46554 | 1506 | 437 | 9093 | 15 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 4 | 1 | 5 | 14418 | 611 | 1080 | 472 | 9 |
| 6 | 2 | 8 | 71196 | 676 | 117 | 1734 | 11 |
| 6 | 7 | 13 | 75660 | 916 | 418 | 18028 | 37 |
| 7 | 4 | 11 | 11486 | 834 | 361 | 1164 | 14 |
| 6 | 3 | 9 | 12426 | 473 | 422 | 1682 | 64 |

## *1. Bedouin Households:*

| *Male* | *Female* | *Total* | *Energy* | *Proten* | *Fat* | *CHO* | *Fibre* |
|---|---|---|---|---|---|---|---|
| 4 | 3 | 7 | 141122 | 2735 | 1485 | 29128 | 26 |
| 3 | 2 | 5 | 42059 | 1187 | 1281 | 6349 | 14 |
| 3 | 4 | 7 | 171678 | 2884 | 2393 | 24048 | 27 |
| 5 | 3 | 8 | 106391 | 2824 | 2042 | 19551 | 20 |
| 5 | 2 | 7 | 7644 | 215 | 31 | 1594 | 20 |
| 4 | 3 | 7 | 21173 | 688 | 1650 | 782 | 46 |
| 2 | 1 | 3 | 39312 | 1448 | 422 | 7366 | 68 |
| 4 | 4 | 8 | 6147 | 332 | 142 | 847 | 11 |
| 5 | 5 | 10 | 14036 | 639 | 961 | 652 | 25 |
| 4 | 4 | 8 | 29394 | 1019 | 1063 | 3982 | 37 |

# Appendix J

**Composition of the household's members participating in the study by sex and age in the URBAN area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U1H1N | 3 | 3 | 2 | 0 | 0 | 8 | 2 | 2 | 2 | 1 | 0 | 7 | 15 |
| U2H2N | 4 | 1 | 1 | 0 | 0 | 6 | 3 | 1 | 2 | 0 | 1 | 7 | 13 |
| U3H3N | 4 | 2 | 0 | 1 | 1 | 8 | 5 | 3 | 1 | 1 | 0 | 10 | 18 |
| U4H4N | 3 | 2 | 1 | 2 | 0 | 8 | 3 | 1 | 1 | 1 | 0 | 6 | 14 |
| U5H5N | 5 | 0 | 1 | 1 | 0 | 7 | 3 | 1 | 1 | 0 | 0 | 5 | 12 |
| U6H6N | 1 | 2 | 2 | 1 | 1 | 7 | 2 | 1 | 2 | 0 | 0 | 5 | 12 |
| U7H7N | 2 | 0 | 1 | 1 | 0 | 4 | 2 | 0 | 0 | 1 | 0 | 3 | 7 |
| U8H8N | 4 | 0 | 0 | 1 | 0 | 5 | 7 | 0 | 0 | 1 | 0 | 8 | 13 |
| U9H9N | 4 | 2 | 0 | 0 | 0 | 6 | 2 | 1 | 0 | 0 | 0 | 3 | 9 |
| U10H10N | 5 | 2 | 0 | 0 | 0 | 7 | 2 | 0 | 0 | 0 | 0 | 2 | 9 |
| U11H11N | 3 | 0 | 0 | 0 | 0 | 3 | 4 | 1 | 2 | 1 | 0 | 8 | 11 |
| U12H12N | 1 | 0 | 0 | 0 | 1 | 2 | 2 | 0 | 0 | 0 | 0 | 2 | 4 |
| U13H13N | 4 | 0 | 0 | 0 | 0 | 4 | 1 | 0 | 0 | 0 | 0 | 1 | 5 |
| U14H14N | 4 | 2 | 0 | 1 | 1 | 8 | 4 | 1 | 1 | 0 | 0 | 6 | 14 |
| U15H15N | 1 | 0 | 1 | 1 | 0 | 3 | 2 | 1 | 0 | 1 | 0 | 4 | 7 |
| U16H16N | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 2 | 3 |
| U17H17N | 4 | 2 | 1 | 0 | 0 | 7 | 3 | 0 | 2 | 1 | 0 | 6 | 13 |

**Appendix J** ***continued*** **...**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U18H18N | 2 | 2 | 2 | 1 | 0 | 7 | 3 | 1 | 0 | 1 | 0 | 5 | 12 |
| U19H19N | 2 | 2 | 1 | 0 | 0 | 5 | 3 | 0 | 2 | 0 | 0 | 5 | 10 |
| U20H20N | 3 | 6 | 3 | 5 | 2 | 19 | 6 | 3 | 7 | 2 | 0 | 18 | 37 |
| U21H21N | 4 | 1 | 0 | 0 | 0 | 5 | 2 | 1 | 1 | 0 | 0 | 4 | 9 |
| U22H22N | 4 | 1 | 1 | 2 | 0 | 8 | 3 | 1 | 4 | 1 | 0 | 9 | 17 |
| U23H23O | 1 | 0 | 2 | 0 | 0 | 3 | 1 | 0 | 0 | 2 | 0 | 3 | 6 |
| U24H24O | 4 | 3 | 2 | 1 | 0 | 10 | 6 | 0 | 0 | 0 | 0 | 6 | 16 |
| U25H25O | 5 | 2 | 2 | 1 | 1 | 11 | 6 | 0 | 1 | 1 | 0 | 8 | 19 |
| U26H26O | 5 | 1 | 2 | 2 | 0 | 10 | 3 | 3 | 1 | 1 | 1 | 9 | 19 |
| U27H27O | 2 | 2 | 0 | 2 | 0 | 6 | 2 | 1 | 0 | 1 | 0 | 4 | 10 |
| U28H28O | 2 | 0 | 0 | 2 | 0 | 4 | 2 | 0 | 1 | 1 | 0 | 4 | 8 |
| U29H29O | 4 | 1 | 3 | 1 | 0 | 9 | 3 | 0 | 2 | 1 | 0 | 6 | 15 |
| U30D1N | 4 | 3 | 1 | 0 | 0 | 8 | 4 | 0 | 0 | 0 | 0 | 4 | 12 |
| U31D2N | 6 | 0 | 3 | 1 | 0 | 10 | 5 | 0 | 3 | 3 | 0 | 11 | 21 |
| U32D3N | 2 | 0 | 0 | 0 | 0 | 2 | 5 | 1 | 0 | 0 | 0 | 6 | 8 |
| U33D4N | 1 | 0 | 1 | 0 | 0 | 2 | 2 | 0 | 2 | 1 | 0 | 5 | 7 |
| U34D5N | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 2 | 3 |
| U35D6N | 1 | 0 | 0 | 1 | 0 | 2 | 1 | 0 | 0 | 1 | 0 | 2 | 4 |

Appendix J *contuined*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U36D7N | 1 | 0 | 1 | 1 | 0 | 3 | 1 | 0 | 1 | 0 | 0 | 2 | 5 |
| U37D8N | 3 | 1 | 0 | 0 | 0 | 4 | 5 | 0 | 0 | 1 | 0 | 6 | 10 |
| U38D9N | 4 | 1 | 0 | 1 | 1 | 7 | 5 | 0 | 0 | 0 | 0 | 5 | 12 |
| U39D10N | 3 | 3 | 1 | 0 | 1 | 8 | 4 | 1 | 3 | 1 | 0 | 9 | 17 |
| U40D11N | 1 | 0 | 1 | 1 | 0 | 3 | 2 | 0 | 0 | 0 | 1 | 3 | 6 |
| U41D12N | 1 | 0 | 1 | 1 | 1 | 4 | 1 | 0 | 0 | 0 | 0 | 1 | 5 |
| U42D13N | 2 | 3 | 0 | 0 | 1 | 6 | 3 | 2 | 1 | 1 | 0 | 7 | 13 |
| U43D14N | 1 | 0 | 1 | 1 | 0 | 3 | 1 | 0 | 1 | 0 | 0 | 2 | 5 |
| U44D15N | 4 | 1 | 0 | 0 | 0 | 5 | 2 | 1 | 0 | 0 | 0 | 3 | 8 |
| U45D16O | 2 | 0 | 1 | 0 | 0 | 3 | 2 | 0 | 1 | 2 | 0 | 5 | 8 |
| U46D17O | 3 | 1 | 0 | 0 | 0 | 4 | 2 | 1 | 0 | 0 | 0 | 3 | 7 |
| U47D18O | 4 | 1 | 1 | 0 | 0 | 6 | 2 | 1 | 0 | 0 | 0 | 3 | 9 |
| U48D19O | 2 | 1 | 0 | 0 | 0 | 3 | 5 | 0 | 0 | 0 | 0 | 5 | 8 |
| U49D20O | 2 | 0 | 1 | 0 | 0 | 3 | 2 | 1 | 1 | 0 | 0 | 4 | 7 |
| U50J1 | 1 | 1 | 1 | 1 | 0 | 4 | 1 | 2 | 1 | 1 | 0 | 5 | 9 |
| U51J2 | 1 | 0 | 0 | 0 | 1 | 2 | 1 | 0 | 0 | 1 | 0 | 2 | 4 |
| U52J3 | 1 | 0 | 0 | 1 | 1 | 3 | 1 | 0 | 1 | 0 | 0 | 2 | 5 |
| U53G1 | 2 | 1 | 0 | 0 | 0 | 3 | 6 | 0 | 0 | 0 | 0 | 6 | 9 |

Appendix J *contuined*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U54G2 | 1 | 0 | 1 | 0 | 0 | 2 | 1 | 0 | 1 | 1 | 0 | 3 | 5 |
| U55G3 | 3 | 1 | 3 | 0 | 0 | 7 | 3 | 1 | 0 | 1 | 0 | 5 | 12 |
| U56G4 | 3 | 1 | 0 | 0 | 0 | 4 | 3 | 0 | 0 | 0 | 0 | 3 | 7 |
| U57G5 | 1 | 0 | 2 | 1 | 0 | 4 | 1 | 1 | 0 | 0 | 0 | 2 | 6 |
| U58G6 | 4 | 0 | 0 | 1 | 0 | 5 | 2 | 1 | 0 | 0 | 0 | 3 | 8 |
| U59G7 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 2 | 3 |
| U60B1 | 1 | 2 | 2 | 1 | 1 | 7 | 2 | 2 | 0 | 0 | 0 | 4 | 11 |
| U61B2 | 6 | 2 | 2 | 0 | 1 | 11 | 3 | 1 | 1 | 1 | 1 | 7 | 18 |
| U62B3 | 2 | 2 | 2 | 1 | 1 | 8 | 1 | 1 | 0 | 1 | 0 | 3 | 11 |
| U63B4 | 2 | 2 | 0 | 2 | 0 | 6 | 2 | 0 | 1 | 0 | 0 | 3 | 9 |
| U64B5 | 0 | 1 | 0 | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 0 | 3 | 4 |
| U65B6 | 1 | 1 | 1 | 2 | 1 | 6 | 1 | 0 | 3 | 1 | 0 | 5 | 11 |
| U66B7 | 1 | 1 | 1 | 2 | 0 | 5 | 3 | 0 | 1 | 0 | 0 | 4 | 9 |
| TOTAL | 170 | 69 | 56 | 45 | 17 | 357 | 178 | 40 | 55 | 37 | 6 | 316 | 673 |

Appendix J *contuined*

**Composition of the household's members participating in the study by sex and age in the RURAL area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >=18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| R1H1S | 2 | 2 | 0 | 0 | 0 | 4 | 1 | 0 | 1 | 0 | 0 | 2 | 6 |
| R2H2S | 4 | 2 | 1 | 1 | 0 | 8 | 1 | 1 | 3 | 1 | 0 | 6 | 14 |
| R3H3S | 1 | 0 | 2 | 0 | 0 | 3 | 2 | 1 | 0 | 0 | 0 | 3 | 6 |
| R4H4B | 4 | 1 | 1 | 0 | 0 | 6 | 3 | 2 | 0 | 1 | 0 | 6 | 12 |
| R5H5B | 2 | 3 | 1 | 1 | 0 | 7 | 2 | 2 | 1 | 0 | 0 | 5 | 12 |
| R6H6B | 3 | 0 | 0 | 0 | 0 | 3 | 3 | 0 | 0 | 1 | 0 | 4 | 7 |
| R7H7B | 1 | 0 | 0 | 1 | 0 | 2 | 1 | 0 | 0 | 1 | 0 | 2 | 4 |
| R8H8B | 3 | 1 | 0 | 0 | 0 | 4 | 3 | 2 | 2 | 0 | 1 | 8 | 12 |
| R9H9B | 4 | 3 | 1 | 0 | 0 | 8 | 3 | 2 | 2 | 1 | 0 | 8 | 16 |
| R10D1S | 3 | 0 | 1 | 1 | 0 | 5 | 2 | 0 | 0 | 0 | 0 | 2 | 7 |
| R11D2S | 5 | 1 | 1 | 0 | 0 | 7 | 3 | 2 | 0 | 0 | 0 | 5 | 12 |
| R12D3S | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 2 |
| R13D4B | 4 | 2 | 1 | 2 | 0 | 9 | 5 | 1 | 0 | 0 | 0 | 6 | 15 |
| R14D5B | 1 | 0 | 0 | 1 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 3 |
| R15D6B | 2 | 3 | 1 | 1 | 0 | 7 | 2 | 1 | 0 | 0 | 0 | 3 | 10 |
| R16D7B | 5 | 0 | 2 | 0 | 0 | 7 | 3 | 0 | 0 | 2 | 0 | 5 | 12 |

Appendix J *continued*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| R17D8B | 1 | 3 | 2 | 0 | 0 | 6 | 2 | 0 | 2 | 0 | 0 | 4 | 10 |
| R18D9B | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 2 |
| R19B1 | 2 | 0 | 1 | 1 | 1 | 5 | 2 | 0 | 0 | 2 | 0 | 4 | 9 |
| R20B2 | 3 | 1 | 0 | 0 | 0 | 4 | 1 | 0 | 0 | 0 | 0 | 1 | 5 |
| R21B3 | 4 | 2 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 0 | 2 | 8 |
| R22B4 | 2 | 2 | 0 | 2 | 0 | 6 | 3 | 3 | 1 | 0 | 0 | 7 | 13 |
| R23B5 | 3 | 1 | 2 | 1 | 0 | 7 | 1 | 1 | 0 | 2 | 0 | 4 | 11 |
| R24B6 | 4 | 0 | 0 | 1 | 1 | 6 | 2 | 0 | 1 | 0 | 0 | 3 | 9 |
| TOTAL | 65 | 27 | 17 | 13 | 2 | 124 | 50 | 18 | 13 | 11 | 1 | 93 | 217 |

Appendix J *continued*

**Composition of the household's members participating in the study by sex and age in the BEDOUIN area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| B1S1 | 1 | 1 | 2 | 0 | 0 | 4 | 2 | 0 | 0 | 1 | 0 | 3 | 7 |
| B2S2 | 0 | 2 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 3 |
| B3S3 | 1 | 1 | 2 | 0 | 0 | 4 | 2 | 1 | 0 | 0 | 1 | 4 | 8 |
| B4S4 | 4 | 1 | 0 | 0 | 0 | 5 | 2 | 1 | 0 | 1 | 1 | 5 | 10 |
| B5S5 | 2 | 2 | 0 | 0 | 0 | 4 | 2 | 1 | 1 | 0 | 0 | 4 | 8 |
| B6N1 | 1 | 0 | 2 | 1 | 0 | 4 | 1 | 0 | 1 | 1 | 0 | 3 | 7 |
| B7N2 | 2 | 0 | 1 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 0 | 2 | 5 |
| B8N3 | 1 | 2 | 0 | 0 | 0 | 3 | 2 | 0 | 1 | 1 | 0 | 4 | 7 |
| B9N4 | 2 | 0 | 1 | 0 | 2 | 5 | 1 | 1 | 0 | 1 | 0 | 3 | 8 |
| B10N5 | 1 | 2 | 2 | 0 | 0 | 5 | 2 | 0 | 0 | 0 | 0 | 2 | 7 |
| **TOTAL** | **15** | **11** | **10** | **1** | **2** | **39** | **17** | **4** | **3** | **5** | **2** | **31** | **70** |

# Appendix K

**Composition of the household's visitors in the study by sex, age in the URBAN area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > =18 | 11-17 | 5-10 | 1-4 | <1 | Total | > =18 | 11-17 | 5-10 | 1-4 | <1 | Total | |
| U1H1N | 0 | 0 | 0 | 2 | 0 | 2 | 2 | 1 | 3 | 0 | 0 | 6 | 8 |
| U2H2N | 1 | 1 | 0 | 0 | 0 | 2 | 6 | 1 | 0 | 0 | 0 | 7 | 9 |
| U3H3N | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 0 | 3 | 4 |
| U4H4N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U5H5N | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| U6H6N | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| U7H7N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U8H8N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U9H9N | 5 | 1 | 0 | 0 | 0 | 6 | 1 | 0 | 0 | 0 | 0 | 1 | 7 |
| U10H10N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U11H11N | 1 | 1 | 2 | 0 | 0 | 4 | 3 | 1 | 1 | 0 | 0 | 5 | 9 |
| U12H12N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U13H13N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U14H14N | 3 | 0 | 0 | 0 | 0 | 3 | 12 | 0 | 0 | 0 | 0 | 12 | 15 |
| U15H15N | 5 | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 5 |
| U16H16N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Appendix K *continued*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U17H17N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U18H18N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U19H19N | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 1 | 3 |
| U20H20N | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| U21H21N | 1 | 1 | 2 | 0 | 0 | 4 | 2 | 2 | 1 | 2 | 0 | 7 | 11 |
| U22H22N | 5 | 0 | 2 | 0 | 0 | 7 | 1 | 0 | 1 | 0 | 0 | 2 | 9 |
| U23H23O | 5 | 0 | 0 | 0 | 0 | 5 | 2 | 0 | 0 | 0 | 0 | 2 | 7 |
| U24H24O | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| U25H25O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U26H26O | 7 | 0 | 2 | 0 | 0 | 9 | 0 | 0 | 0 | 0 | 0 | 0 | 9 |
| U27H27O | 0 | 9 | 0 | 1 | 0 | 10 | 3 | 0 | 1 | 0 | 0 | 4 | 14 |
| U28H28O | 2 | 0 | 1 | 0 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 1 | 4 |
| U29H29O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U30D1N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U31D2N | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 2 | 2 |
| U32D3N | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 |
| U33D4N | 0 | 2 | 2 | 1 | 0 | 5 | 3 | 0 | 2 | 0 | 0 | 5 | 10 |
| U34D5N | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |

Appendix K *contuined*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U35D6N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U36D7N | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| U37D8N | 5 | 0 | 0 | 0 | 0 | 5 | 8 | 0 | 0 | 0 | 0 | 8 | 13 |
| U38D9N | 6 | 0 | 3 | 0 | 0 | 9 | 6 | 0 | 1 | 0 | 0 | 7 | 16 |
| U39D10N | 6 | 0 | 0 | 0 | 0 | 6 | 0 | 0 | 0 | 0 | 0 | 0 | 6 |
| U40D11N | 9 | 0 | 0 | 0 | 0 | 9 | 6 | 0 | 0 | 0 | 0 | 6 | 15 |
| U41D12N | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U42D13N | 9 | 0 | 0 | 0 | 0 | 9 | 9 | 0 | 0 | 0 | 0 | 9 | 18 |
| U43D14N | 4 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| U44D15N | 3 | 0 | 3 | 3 | 0 | 9 | 3 | 0 | 2 | 0 | 0 | 5 | 14 |
| U45D16O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U46D17O | 3 | 0 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 0 | 2 | 5 |
| U47D18O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U48D19O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U49D20O | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U50J1 | 2 | 0 | 0 | 1 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 1 | 4 |
| U51J2 | 5 | 1 | 0 | 0 | 0 | 6 | 4 | 0 | 0 | 0 | 0 | 4 | 10 |
| U52J3 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 3 | 3 |

Appendix K *contuined*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| U53G1 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| U54G2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U55G3 | 5 | 0 | 0 | 0 | 0 | 5 | 3 | 0 | 2 | 1 | 0 | 6 | 11 |
| U56G4 | 9 | 0 | 3 | 3 | 0 | 15 | 4 | 0 | 0 | 0 | 0 | 4 | 19 |
| U57G5 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 2 |
| U58G6 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U59G7 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U60B1 | 3 | 0 | 0 | 1 | 0 | 4 | 4 | 0 | 0 | 1 | 0 | 5 | 9 |
| U61B2 | 4 | 0 | 0 | 0 | 0 | 4 | 0 | 2 | 0 | 0 | 0 | 2 | 6 |
| U62B3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U63B4 | 3 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| U64B5 | 3 | 0 | 0 | 0 | 0 | 3 | 5 | 0 | 0 | 0 | 0 | 5 | 8 |
| U65B6 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| U66B7 | 1 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 2 | 3 |
| **TOTAL** | **127** | **17** | **21** | **12** | **0** | **177** | **98** | **11** | **16** | **4** | **0** | **129** | **306** |

Appendix K *contuined*

**Composition of the household's visitors in the study by sex, age in the RURAL area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | >= 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| R1H1S | 0 | 0 | 3 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 0 | 1 | 4 |
| R2H2S | 7 | 0 | 0 | 0 | 0 | 7 | 0 | 0 | 0 | 0 | 0 | 0 | 7 |
| R3H3S | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R4H4B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R5H5B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R6H6B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R7H7B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R8H8B | 5 | 0 | 0 | 0 | 0 | 5 | 1 | 1 | 0 | 0 | 0 | 2 | 7 |
| R9H9B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R10D1S | 3 | 3 | 1 | 2 | 0 | 9 | 4 | 0 | 2 | 2 | 0 | 8 | 17 |
| R11D2S | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R12D3S | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R13D4B | 3 | 1 | 1 | 0 | 0 | 5 | 4 | 0 | 1 | 0 | 0 | 5 | 10 |
| R14D5B | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 2 |
| R15D6B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R16D7B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Appendix K *continued*

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| R17D8B | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R18D9B | 0 | 1 | 0 | 1 | 0 | 2 | 2 | 0 | 1 | 0 | 0 | 3 | 5 |
| R19B1 | 8 | 0 | 0 | 0 | 0 | 8 | 2 | 0 | 0 | 0 | 0 | 2 | 10 |
| R20B2 | 1 | 1 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 3 |
| R21B3 | 13 | 0 | 0 | 0 | 0 | 13 | 1 | 4 | 0 | 0 | 0 | 5 | 18 |
| R22B4 | 7 | 7 | 1 | 0 | 0 | 15 | 1 | 2 | 1 | 0 | 0 | 4 | 19 |
| R23B5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| R24B6 | 6 | 0 | 0 | 0 | 0 | 6 | 1 | 0 | 0 | 0 | 0 | 1 | 7 |
| TOTAL | 54 | 13 | 6 | 3 | 0 | 76 | 17 | 8 | 5 | 3 | 0 | 33 | 109 |

Appendix K *continued*

**Composition of the household's visitors in the study by sex, age in the BEDOUIN area.**

| Household code | Male | | | | | | Female | | | | | | All |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | > = 18 | 11-17 | 5-10 | 1-4 | < 1 | Total | |
| B1S1 | 6 | 2 | 0 | 0 | 0 | 8 | 4 | 1 | 0 | 0 | 0 | 5 | 13 |
| B2S2 | 8 | 5 | 0 | 1 | 0 | 14 | 5 | 0 | 1 | 0 | 0 | 6 | 20 |
| B3S3 | 13 | 4 | 0 | 0 | 0 | 17 | 1 | 0 | 0 | 0 | 0 | 1 | 18 |
| B4S4 | 9 | 1 | 0 | 0 | 0 | 10 | 4 | 1 | 0 | 0 | 0 | 5 | 15 |
| B5S5 | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| B6N1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| B7N2 | 12 | 0 | 0 | 0 | 0 | 12 | 0 | 0 | 0 | 0 | 0 | 0 | 12 |
| B8N3 | 4 | 0 | 0 | 1 | 0 | 5 | 2 | 1 | 2 | 0 | 0 | 5 | 10 |
| B9N4 | 3 | 7 | 2 | 0 | 0 | 12 | 6 | 2 | 0 | 0 | 0 | 8 | 20 |
| B10N5 | 2 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| **TOTAL** | **59** | **19** | **2** | **2** | **0** | **82** | **22** | **5** | **3** | **0** | **0** | **30** | **112** |

# Appendix L

## Household Food Consumption Survey : Additional Information

***Population Sample Design***: A random sample of 100 private Saudi households in the Eastern Province were taken. The sample was divided into three groups; 66% urban, 24% rural and 10% Bedouin area.

The survey areas were selected following a multistage random sampling method. These are as followed:

1. The first stage involves the division of the province into four zones. This was done to ensure that the sampling frame covers the whole the area.

   These zones were:

   a. Al-Hassa zone
   b. Al-Dammam zone
   c. Hafr Al-Batin zone
   d. Bedouin zone

2. The second stage was the selection of cities and villages within each zone. This was achieved by an enumeration of the cities and the villages in the area. Then the city/village were selected randomly.

3. The third stage was the selection of area in each city/village chosen. This was achieved by the following:

   a. In case of the cities; each city was divided into two sectors, corresponding to areas with old and new housing.

   b. In case of the villages; villages were divided into two categories according to the villages population size (i.e. small and large). One household was selected from small villages and two from larger ones

4. The fourth stage was the selection of the division within each city/village selected from the third stage.

   a. In the cases of the city; each household were numbered in the selected block, then the houses selected randomly.

   b. In the cases of the village; the village was divided into four quarters, one quarter was randomly chosen. Within the chosen quarter an

enumeration of the houses were made. Then the household(s) was/were chosen randomly.

5. The final stage was the selection of the Bedouin area. Two Hejra sites, were selected, one in the north (Al-Graieh Alolia) and the other in the south (Yabreen). Five households were selected from each area, using records from primary and elementary schools.

The number of households selected from each city were determined by the population size of the city.
For example:

| | | |
|---|---|---|
| City | : | Dammam |
| Total population | : | 174,000 |
| The average family number | : | 7 |
| Number of households | : | |

$$\frac{174,000}{7} = 24,857$$

Mathematical equation:

$$\frac{\text{household number in city}}{\text{Total number of the households in the urban}}$$

Then the sample size of the Dammam city is

$$= \frac{24857}{81173} = 20.21 \text{ household}$$

Therefore, the sample size for Dammam city will be 20 households.

# Appendix M

## The expanded mortality data set

**Months available from hospitals searched in the study**

| HOSPITALS | Year 1 1989 | Year 2 1990 |
|---|---|---|
| 1- GCH | 1-12 | 1-12 |
| 2- DCH | 1-12 | 1-10 |
| 3- GGH | 1-12 | 1-10 |
| 4- KKH | 1-9, 12 | 1-10 |
| 5- JGH | * | 1-11 |
| 6- KFH | 1-12 | 1-12 |

* = missing

**GCH CHD 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(DCH) CHD 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 | 6 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 1 | 1 | 1 | 5 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 2 |
| 0 | 0 | 0 | 0 | 0 | 1 | 3 | 3 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 1 | 0 | 0 | 1 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 4 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 2 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |

**(GGH) CHD 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(KKH) CHD 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 4 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 2 | 0 | 0 | 2 | 1 |
| 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 |

### (JGH) CHD 1989

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

### (GCH) CHD 1990

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 4 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |

**(DCH) CHD 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 2 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 3 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 |
| 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 2 | 0 | 1 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(GGH) CHD 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 0 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(KKH) CHD 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(JGH) CHD 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(GCH) All deaths 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 3 |
| 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 3 |
| 1 | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 2 |
| 1 | 2 | 1 | 0 | 0 | 0 | 2 | 3 | 3 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 3 | 1 | 0 | 0 | 0 | 3 | 1 | 0 |
| 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| 1 | 1 | 1 | 1 | 0 | 1 | 2 | 1 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 |
| 1 | 0 | 2 | 0 | 0 | 0 | 2 | 2 | 0 |
| 2 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 0 |
| 2 | 2 | 1 | 1 | 0 | 1 | 0 | 5 | 4 |
| 0 | 1 | 1 | 0 | 0 | 2 | 0 | 3 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 1 | 0 | 0 | 0 | 0 | 1 | 2 | 2 | 2 |
| 1 | 3 | 0 | 0 | 1 | 0 | 0 | 2 | 2 |
| 0 | 1 | 0 | 2 | 0 | 1 | 0 | 1 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 |
| 0 | 1 | 0 | 0 | 0 | 1 | 0 | 3 | 3 |
| 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 3 |

**(DCH) All deaths 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 2 | 1 | 2 | 1 | 1 | 4 | 4 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 |
| 0 | 0 | 0 | 2 | 0 | 0 | 2 | 4 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 4 | 3 |
| 0 | 1 | 0 | 0 | 0 | 2 | 0 | 0 | 1 |
| 1 | 1 | 2 | 1 | 0 | 2 | 1 | 3 | 6 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 4 | 3 |
| 0 | 0 | 2 | 0 | 0 | 1 | 2 | 1 | 3 |
| 0 | 0 | 2 | 0 | 0 | 1 | 0 | 2 | 2 |
| 0 | 1 | 5 | 1 | 3 | 1 | 2 | 8 | 1 |
| 0 | 0 | 1 | 0 | 2 | 0 | 2 | 3 | 2 |
| 0 | 1 | 1 | 0 | 0 | 2 | 3 | 4 | 4 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 6 |
| 1 | 0 | 2 | 3 | 0 | 0 | 3 | 2 | 6 |
| 0 | 0 | 1 | 0 | 0 | 1 | 4 | 1 | 1 |
| 0 | 0 | 2 | 0 | 1 | 2 | 5 | 3 | 2 |
| 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 3 |
| 0 | 1 | 2 | 1 | 0 | 1 | 4 | 1 | 2 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 2 | 3 |
| 0 | 0 | 2 | 1 | 0 | 4 | 0 | 2 | 0 |
| 0 | 0 | 0 | 1 | 0 | 0 | 0 | 2 | 2 |
| 0 | 0 | 0 | 0 | 1 | 4 | 4 | 0 | 0 |
| 0 | 0 | 0 | 0 | 2 | 0 | 1 | 4 | 0 |

**(GGH) All deaths 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 3 |
| 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 1 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**(KKH) All deaths 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 3 | 0 | 0 | 2 | 4 | 4 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 |
| 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 3 |
| 0 | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 2 | 3 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 3 | 2 | 0 | 2 | 2 | 0 | 1 |
| 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 2 |
| 1 | 0 | 0 | 1 | 1 | 0 | 2 | 4 | 6 |
| 0 | 0 | 0 | 0 | 1 | 0 | 1 | 2 | 1 |
| 0 | 0 | 1 | 2 | 1 | 1 | 0 | 4 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 5 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 1 | 0 | 0 | 4 | 1 | 1 | 3 | 0 | 2 |
| 0 | 0 | 2 | 1 | 0 | 1 | 0 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 2 | 0 | 4 | 0 | 1 | 3 | 4 |
| 0 | 0 | 0 | 0 | 1 | 1 | 1 | 0 | 2 |

**(JGH) All deaths 1989**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |

**(GCH) All deaths 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 1 | 0 | 2 | 0 | 0 | 1 | 0 | 4 | 2 |
| 0 | 1 | 0 | 0 | 0 | 1 | 2 | 0 | 2 |
| 1 | 0 | 1 | 0 | 0 | 2 | 1 | 3 | 3 |
| 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 2 |
| 0 | 1 | 0 | 0 | 0 | 1 | 2 | 5 | 1 |
| 2 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 5 |
| 0 | 1 | 0 | 0 | 2 | 1 | 1 | 3 | 3 |
| 2 | 0 | 0 | 0 | 0 | 1 | 3 | 1 | 2 |
| 0 | 0 | 1 | 2 | 0 | 1 | 6 | 6 | 5 |
| 1 | 0 | 1 | 0 | 0 | 1 | 1 | 2 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 3 | 5 |
| 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 |
| 0 | 0 | 0 | 0 | 2 | 0 | 3 | 8 | 2 |
| 0 | 1 | 0 | 0 | 1 | 0 | 1 | 4 | 4 |
| 3 | 0 | 0 | 1 | 0 | 0 | 1 | 3 | 4 |
| 1 | 0 | 0 | 1 | 0 | 1 | 1 | 3 | 5 |
| 0 | 1 | 1 | 2 | 1 | 0 | 0 | 2 | 4 |
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 3 | 4 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 1 | 1 | 0 | 0 | 4 | 2 | 2 |
| 0 | 1 | 1 | 0 | 0 | 0 | 2 | 2 | 3 |
| 0 | 0 | 0 | 0 | 0 | 3 | 1 | 0 | 3 |
| 0 | 1 | 0 | 1 | 0 | 0 | 1 | 1 | 0 |
| 0 | 1 | 1 | 0 | 1 | 0 | 1 | 3 | 1 |
| 0 | 0 | 0 | 0 | 1 | 1 | 2 | 0 | 0 |

**(DCH) All deaths 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 1 | 0 | 2 | 2 | 1 | 2 | 1 | 0 | 4 |
| 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 2 | 3 | 1 | 0 | 2 | 4 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 1 |
| 0 | 0 | 3 | 2 | 1 | 3 | 1 | 8 | 4 |
| 0 | 0 | 0 | 0 | 0 | 2 | 2 | 3 | 2 |
| 0 | 1 | 0 | 1 | 0 | 1 | 2 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 5 | 1 | 2 |
| 0 | 2 | 1 | 0 | 0 | 0 | 1 | 1 | 2 |
| 0 | 3 | 1 | 1 | 1 | 2 | 2 | 2 | 3 |
| 1 | 0 | 0 | 2 | 0 | 2 | 2 | 3 | 4 |
| 0 | 0 | 0 | 1 | 0 | 1 | 2 | 3 | 8 |
| 0 | 0 | 3 | 0 | 1 | 3 | 0 | 2 | 6 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 0 | 0 | 2 | 1 | 1 | 0 | 1 | 7 | 3 |
| 1 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 4 |
| 0 | 0 | 0 | 1 | 2 | 0 | 1 | 1 | 3 |
| 0 | 0 | 1 | 0 | 1 | 1 | 1 | 2 | 3 |
| 0 | 0 | 1 | 5 | 2 | 0 | 1 | 3 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 3 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |

**(GGH) All deaths 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 0 | 2 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| 0 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 2 |
| 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 0 | 1 | 0 | 0 | 0 | 1 | 2 | 1 | 2 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 3 |
| 1 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 3 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |

**(KKH) All deaths 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 1 | 0 | 0 | 0 | 2 | 2 | 1 | 2 |
| 1 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 2 |
| 0 | 1 | 1 | 0 | 1 | 0 | 1 | 1 | 4 |
| 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 1 | 1 | 1 | 2 | 1 | 0 | 2 |
| 1 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 1 |
| 0 | 0 | 0 | 2 | 0 | 1 | 5 | 2 | 4 |
| 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 2 |
| 2 | 0 | 2 | 1 | 1 | 1 | 3 | 1 | 4 |
| 1 | 0 | 1 | 0 | 0 | 0 | 0 | 3 | 1 |
| 0 | 1 | 3 | 1 | 1 | 3 | 1 | 1 | 4 |
| 0 | 0 | 0 | 0 | 0 | 2 | 2 | 0 | 3 |
| 2 | 1 | 3 | 4 | 1 | 1 | 1 | 2 | 8 |
| 0 | 1 | 0 | 0 | 0 | 2 | 1 | 0 | 1 |
| 0 | 3 | 4 | 0 | 2 | 0 | 1 | 5 | 9 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0 | 1 | 3 | 2 | 2 | 1 | 2 | 1 | 6 |
| 1 | 0 | 0 | 0 | 0 | 2 | 1 | 0 | 1 |
| 1 | 3 | 1 | 0 | 1 | 0 | 1 | 1 | 2 |
| 0 | 1 | 0 | 0 | 0 | 0 | 0 | 3 | 2 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |

**(JGH) All deaths 1990**

| 1-4 | 5-14 | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65-74 | 75 & + |
|---|---|---|---|---|---|---|---|---|
| 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 1 | 2 | 0 | 1 | 0 | 0 | 1 |
| 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| 1 | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 0 | 0 | 1 | 2 | 0 | 1 | 0 | 1 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 |
| 0 | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 1 |
| 1 | 0 | 1 | 0 | 0 | 0 | 2 | 1 | 1 |
| 0 | 0 | 1 | 0 | 0 | 1 | 0 | 3 | 4 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 1 | 0 | 3 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 3 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |

# Appendix N

## Determination method for oil/fat Contents

*a.* *Rose-Gottlib procedure*:

**i. Food samples**

*MILK*:

1. GOAT MILK, fresh, Saudi.
2. SHEEP MILK, fresh, Saudi.
3. CAMEL MILK, pasteurised and homogenised. Saudi.
4. COW MILK, fresh, Saudi.
5. ALSAFI MILK, cow milk, pasteurised and homogenised. Saudi.
6. NADIC MILK, pasteurised and homogenised. Saudi.
7. NADA MILK, pasteurised and homogenised. Saudi.
8. ALMATROOD MILK, pasteurised and homogenised. Saudi.

*CHEESE*:

1. LA VACHE QUIRIT, processed cream cheese spread. France.
2. PUCK, processed spread. Australia.
3. ALMARAI, Saudi.
4. LA VACHE QUIRIT, processed spread. France.
5. KRAFT, cream spread. Australia.
6. KIRI, cream spread. France.
7. PUCK MOZZARELLA, Copenhagen.
8. FARMER, Danish feta cheese, Denmark.

**ii. Procedure**

1. Weight 10g of the sample in the tube.
2. Add 1ml of 0.88 ammonia solution and mix.
3. Then add 10ml of alcohol (95%) and mix well.
4. Add 25ml peroxide-free diethyl ether, stopper the tube and shake vigorously for 1min.
5. Add 25ml for light petroleum (b.p. 40-60 C°) and shake vigorously for 30sec.
6. Then separate it.
7. Then add two successive lots of 5ml of mixed "ethers" and transfer (without shaking) to the flask.
8. Then repeat the extraction (with 15ml of ether and 15ml of light petroleum).
9. Distil off the solvents from the flask.
10. Dry the fat, cool and weigh.

*b. Werner-Schmid procedure*

**i. Food samples**

1. PUCK, Toast slices. processed cheddar. Copenhagen.
2. PUCK, Burger cheese slices, cheddar cheese, Saudi.
3. KRAFT, Slices, processed. Australia.
4. ALMARAI, Burger cheese slices, cheddar. Saudi.
5. ALMARAI, Toast cheese slices, cheddar. Saudi.
6. ANCHORE, Slices, processed cheddar. New Zealand.
7. YOGURT, Saudi.

**ii. Procedure**

1. Weight 1-2g of sample.
2. Using a rood, macerate thoroughly with a few drops of 0.88 ammonia.
3. Then add 10ml diluted hydrochloric acid (7 volume conc HCL + 3 volume water).
4. Heated careful until all the particles have dissolved.
5. Cool slightly.
6. Add 10ml alcohol, mix, then cool and transfer the liquid down into an extraction tube.
7. Add ether (total 25ml).
8. Then, shake the tube vigorously for 1min.
9. Then, add 25ml light petroleum.
10. Shake again and transfer the separated extract in to a weighed flask.
11. Repeat the extraction 3 more times.
12. Distil off the solvents from the flask.
13. Dry the fat, cool and weight.

*c. Total fat in the meat and chicken procedure*

**iii. Procedure**

1. Boil 3-5g of sample with 50ml 4M hydrochloric.
2. Dilute and extract the fat with light petroleum or n-hexane using the "wash bottle" technique.
3. Collect the extract in a weighed flash and remove the solvent by evaporation.
4. Dry and weight.

## Methylation Method for Fat Composition of Oil and Fat

**The following procedure was used:**

1. Weigh sample containing 10-50mg of oil into a dry stoppered tube.
2. Dissolve in 1ml dry toluene and shake it.
3. Add 2ml sodum methoxide solution and shake it.
4. Stopper the tube and heat in water bath at 50°C for 15 to 20 minutes.
5. Cool and neutralise with 0.1ml of glacial acetic acid.
6. Add about 4 to 5ml of H2O acid.
7. And, add about 10ml hexan to extract.
8. Drys the extract over anhydrous Na2So4, leave it for 5 minutes.
9. Filter and evaporate the filterate to dryness on a rotary evaporator at 35°C.
10. Redissolve in pure hexane for GLC.
11. Use 1ml hexane for each 20mg of original oil.

# Appendix O

**Fatty acid composition for the food analysis (fatty acids g/ 100g/ total fatty acids)**

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| La Vach quirit, cream | 0.29 | 0.33 | 0.80 | 2.52 | 4.27 | 13.99 | 0.91 | 34.60 | 0 | 11.18 |
| Puck, spread | 0 | 0.17 | 0.62 | 1.73 | 4.36 | 13.04 | 0.83 | 38.18 | 0.56 | 11.22 |
| Kraft, slices | 0.46 | 0.21 | 0.84 | 2.82 | 4.94 | 14.96 | 1.00 | 31.93 | 0.58 | 13.23 |
| Puck, Toast-slices | 0 | 0.18 | 0.72 | 2.56 | 4.23 | 13.92 | 1.09 | 34.81 | 0.83 | 10.33 |
| Kraft,slices | 0 | 0 | 0 | 1.81 | 3.62 | 14.04 | 0.94 | 35.83 | 0.81 | 12.56 |
| Puck, Burger-slices | 0.28 | 0.28 | 0.72 | 2.29 | 4.94 | 14.96 | 1.00 | 31.93 | 0.58 | 13.23 |
| Al-Marai, Burger-slices | 0.00 | 0.58 | 0.12 | 1.13 | 3.62 | 12.44 | 0.88 | 34.52 | 0.63 | 12.13 |
| Al-Marai, toast-slices | 0.00 | 0.00 | 0.15 | 1.05 | 3.62 | 12.77 | 0.91 | 33.10 | 0.00 | 14.36 |
| Anchor, slices | 0.00 | 0.40 | 0.47 | 1.85 | 4.55 | 13.40 | 0.86 | 33.80 | 0.65 | 12.45 |
| La Vach quirit, spread | 0.00 | 0.21 | 0.68 | 1.94 | 3.77 | 13.17 | 0.96 | 33.95 | 0.71 | 11.60 |
| kiri, spread | 0.00 | 0.15 | 1.03 | 3.64 | 4.70 | 13.28 | 0.89 | 33.01 | 0.68 | 10.97 |
| Puck, Mazzorella | 0.00 | 0.17 | 0.63 | 2.18 | 4.42 | 12.67 | 0.75 | 35.16 | 0.62 | 11.77 |
| Farmer, cream | 0.00 | 0.21 | 0.76 | 2.80 | 4.27 | 12.96 | 0.90 | 35.70 | 0.70 | 10.08 |
| Kraft | 0.00 | 0.17 | 0.64 | 2.54 | 3.75 | 12.85 | 0.94 | 32.98 | 0.86 | 12.43 |

**Appendix O** *Continued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 |
| La Vach quirit,spr. | 0.37 | 0.63 | 1.40 | 0 | 1.66 | 0.53 | 0 | 1.67 | 22.47 | 1.61 | 0.76 |
| Puck, spread | 0.30 | 0 | 1.12 | 0 | 0.27 | 1.83 | 0 | 1.94 | 22.06 | 1.77 | 0 |
| Kraft, slices | 0.44 | 0.66 | 1.46 | 0 | 0.48 | 1.10 | 0 | 3.50 | 20.68 | 0.70 | 0 |
| Puck, toast-slices | 0.29 | 0.54 | 1.50 | 0.24 | 0.72 | 2.70 | 0 | 2.07 | 21.43 | 1.17 | 0.65 |
| Kraft, slices | 0.47 | 0.71 | 1.54 | 0 | 0.74 | 2.52 | 0 | 2.30 | 21.26 | 0.87 | 0 |
| Puck,burger, slices | 0.41 | 0.55 | 1.51 | 0 | 0 | 1.97 | 0 | 1.28 | 22.62 | 1.30 | 0.00 |
| Al-Marai, byrger, slices | 0.23 | 0.40 | 1.34 | 0 | 0.40 | 2.25 | 0 | 2.55 | 24.79 | 1.23 | 0.76 |
| Al-Marai, toast, slices | 0.33 | 0.54 | 1.44 | 0 | 0.51 | 1.74 | 0 | 2.83 | 26.65 | 0.00 | 0.00 |
| Anchor, slices 10 | 0.28 | 0.62 | 1.46 | 0 | 0.56 | 2.25 | 0 | 3.59 | 20.96 | 0.90 | 0.95 |
| La Vach quirit, spread | 0.32 | 0.69 | 1.45 | 0 | 0.43 | 2.04 | 0.24 | 2.67 | 22.82 | 1.56 | 0.79 |
| Kiri, spread | 0.28 | 0.58 | 1.42 | 0 | 0.42 | 1.97 | 0.00 | 3.06 | 21.88 | 1.17 | 0.87 |
| Puck, Mazzoella | 0.12 | 0.29 | 0.92 | 0 | 0.62 | 2.40 | 0.00 | 1.89 | 23.72 | 1.67 | 0.00 |
| Farmer, cream | 0.23 | 0.49 | 1.46 | 0 | 0.30 | 2.22 | 0.17 | 1.07 | 23.30 | 2.38 | 0.00 |
| Kraft | 0.35 | 0.67 | 1.73 | 0.22 | 0.80 | 2.62 | 0 | 3.19 | 21.41 | 0.96 | 0.89 |

**Appendix O** *continued*

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| **Milk:** | | | | | | | | | | |
| Goat, fresh | 0.34 | 0.11 | 0.89 | 5.17 | 3.78 | 13.39 | 0.34 | 38.15 | 1.15 | 6.90 |
| Cow, fresh | 0.09 | 0 | 0 | 0.48 | 1.18 | 6.03 | 1.99 | 28.75 | 1.03 | 5.01 |
| Sheep | 0 | 0 | 0 | 2.20 | 3.17 | 15.02 | 0 | 46.45 | 0 | 4.80 |
| Camel | 0 | 0 | 0.13 | 0.11 | 1.13 | 12.21 | 1.23 | 33.79 | 0.7 | 6.84 |
| Asafi | 0 | 0 | 0.17 | 1.09 | 2.17 | 9.2 | 0.52 | 34.18 | 0.78 | 11.81 |
| Nadec | 0.43 | 0.13 | 0.51 | 1.54 | 1.99 | 7.86 | 0.6 | 35.86 | 0.58 | 8.83 |
| Nada | 0.54 | 0.16 | 0.58 | 2.37 | 3.64 | 11.25 | 0.87 | 33.46 | 0.73 | 10.26 |
| AL-Matrood | 0 | 0 | 0.47 | 2.16 | 3.31 | 11.52 | 1.19 | 36.91 | 0.34 | 8.94 |
| Yogurt | 0 | 0.29 | 0.76 | 2.05 | 3.31 | 11.36 | 1.14 | 32.53 | 0 | 9.72 |
| **Meat & Poultry:** | | | | | | | | | | |
| Lamb | 0 | 0 | 0 | 0 | 0 | 4.38 | 0 | 27 | 1.8 | 19.23 |
| Chicken fresh | 0 | 0 | 0 | 0 | 0 | 0.83 | 0 | 29.88 | 0 | 4.97 |
| Doux, chicken | 0 | 0 | 0 | 0 | 0 | 0.52 | 0 | 20.2 | 0 | 4.73 |
| Rudwa, chiken | 0 | 0 | 0 | 0 | 0 | 0.52 | 0 | 26.31 | 0 | 4.42 |

**Appendix O** *contunued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 |
| **Milk:** Goat, fresh | 0.37 | 0.69 | 1.71 | 0.41 | 0.73 | 2.75 | 0.44 | 0.86 | 19.41 | 1.86 | 0.55 |
| Cow, fresh | 0.53 | 0 | 1.66 | 0 | 1.79 | 6.84 | 0.77 | 16.28 | 24.98 | 2.59 | 0 |
| Sheep | 0.73 | 0 | 1.84 | 0 | 0 | 2.02 | 0 | 0 | 21.95 | 1.82 | 0 |
| Camel | 0.38 | 0.90 | 1.43 | 0.31 | 0.53 | 13.65 | 0.51 | 3.95 | 20.90 | 1.3 | 0 |
| Asafi | 0 | 0 | 0.90 | 0 | 3.10 | 3.48 | 0 | 2.62 | 27.13 | 2.85 | 0 |
| Nadec | 0.1 | 0.23 | 0.74 | 0 | 0.8 | 2.5 | 0 | 3.03 | 30.23 | 3.54 | 0.4 |
| Nada | 0.19 | 0.44 | 1.64 | 0 | 0.39 | 2.51 | 0 | 2.21 | 25.14 | 3.62 | 0 |
| AL-Matrood | 0 | 0.39 | 1.51 | 0 | 0.42 | 2.87 | 0 | 2.59 | 24.76 | 2.26 | 0 |
| Yogurt | 0.22 | 0.52 | 1.71 | 0.26 | 1.38 | 3.71 | 0.35 | 3.35 | 24.31 | 2.22 | 0.81 |
| **Meat & Poultry:** Lamb | 0.72 | 0 | 0 | 0 | 0 | 2.06 | 0 | 2.08 | 41.04 | 1.69 | 0 |
| Chicken fresh | 0 | 0 | 0 | 0 | 0 | 8.91 | 0 | 0 | 44.98 | 10.43 | 0 |
| Doux, chicken | 0 | 0 | 0 | 0 | 0 | 2.91 | 0 | 0 | 44.93 | 25.55 | 1.16 |
| Rudwa, chiken | 0 | 0 | 0 | 0 | 0 | 7.84 | 0 | 0 | 46.1 | 14.81 | 0 |

**Appendix O** *continued*

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| **Vegetable oils:** | | | | | | | | | | |
| Al-Arabi (palm) | 0 | 0 | 0 | 0 | 0 | 0.97 | 0 | 39.15 | 0 | 3.46 |
| Nakheel (palm) | 0 | 0 | 0 | 0 | 0.16 | 0.96 | 0 | 42.06 | 0 | 3.39 |
| Calaira (soya bean) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10.81 | 0 | 3.0 |
| Mazola (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11.28 | 0 | 1.46 |
| Afia (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11.20 | 0 | 1.62 |
| Sasso (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11.6 | 0 | 2.25 |
| Al-Wazir (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9.58 | 0 | 2.74 |
| Altayeb (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 17.57 | 0 | 1.78 |
| Golden chair (ghee) | 0 | 0.72 | 0.64 | 2.04 | 3.0 | 11.69 | 0.89 | 34.56 | 0.7 | 13.77 |
| Danish Lurbak (butter) | 0 | 0.43 | 0.58 | 1.68 | 3.8 | 12.36 | 0.84 | 37.01 | 0.56 | 11.43 |

**Appendix O** *continued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 |
| **Vgetable oils:** | | | | | | | | | | | |
| Al-Arabi (palm) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 43.38 | 12.59 | 0 |
| Nakheel (palm) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 43.36 | 10.07 | 0 |
| Calaira (soyabean) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 21.73 | 57.34 | 7.12 |
| Mazola (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 27.11 | 59.23 | 0.91 |
| Afia (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 26.17 | 60.30 | 0.71 |
| Sasso (olive) | 0 | 0 | 0 | 0 | 0.94 | 0 | 0 | 0 | 76.46 | 8.75 | 0 |
| Al-Wazir (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 80.84 | 6.84 | 0 |
| Altayeb (olive) | 0 | 0 | 0 | 0 | 2.22 | 0 | 0 | 0 | 59.99 | 18.05 | 0.39 |
| Golden chair (ghee) | 0 | 0 | 1.4 | 0 | 0.42 | 2.39 | 0 | 1.89 | 24.37 | 0.91 | 0.61 |
| Danish Lurpak (butter) | 0 | 0 | 1.15 | 0 | 0.44 | 2.32 | 0 | 1.94 | 23.79 | 1.65 | 0 |

# Appendix P

**Fatty acids composition for the foods analysis (fatty acids g/ 100g/ food)**

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| La Vach quirit, cream | 0.07 | 0.08 | 0.18 | 0.58 | 0.98 | 3.23 | 0.21 | 7.98 | 0 | 2.58 |
| Puck, spread | 0 | 0.05 | 0.19 | 0.54 | 1.36 | 4.08 | 0.26 | 11.94 | 0.13 | 3.51 |
| Kraft, slices (6) | 0.09 | 0.04 | 0.17 | 0.57 | 1.00 | 3.04 | 0.20 | 6.49 | 0.12 | 2.69 |
| Puck, Toast-slices (5) | 0 | 0.04 | 0.16 | 0.57 | 0.94 | 3.09 | 0.24 | 7.73 | 0.18 | 2.29 |
| Kraft,slices (4) | 0 | 0 | 0 | 0.54 | 1.06 | 4.15 | 0.28 | 10.60 | 0.24 | 3.72 |
| Puck, Burger-slices (7) | 0.06 | 0.06 | 0.15 | 0.47 | 0.90 | 3.01 | 0.22 | 7.35 | 0.12 | 2.16 |
| Al-Marai, Burger-slices 8 | 0 | 0.12 | 0.02 | 0.23 | 0.75 | 2.59 | 0.18 | 7.18 | 0.13 | 2.52 |
| Al-Marai, toast-slices 9 | 0 | 0 | 0.04 | 0.25 | 0.87 | 3.05 | 0.22 | 7.91 | 0 | 3.43 |
| Anchor, slices (10) | 0 | 0.09 | 0.10 | 0.40 | 0.99 | 2.91 | 0.19 | 7.35 | 0.14 | 2.71 |
| La Vach quirit, spread 11 | 0 | 0.05 | 0.17 | 0.48 | 0.93 | 3.26 | 0.24 | 8.41 | 0.18 | 2.87 |
| kiri, spread (12) | 0 | 0.04 | 0.30 | 1.07 | 1.38 | 3.89 | 0.26 | 9.67 | 0.20 | 3.21 |
| Puck, Mazzorella (13) | 0 | 0.04 | 0.13 | 0.45 | 0.92 | 2.63 | 0.16 | 7.31 | 0.13 | 2.45 |
| Farmer, cream (14) | 0 | 0.04 | 0.14 | 0.51 | 0.78 | 2.38 | 0.16 | 6.54 | 0.13 | 1.85 |
| Kraft (15) | 0 | 0.04 | 0.15 | 0.58 | 0.85 | 2.91 | 0.21 | 7.48 | 0.20 | 2.82 |

**Appendix P** *Continued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 |
| La Vach quirit | 0.09 | 0.15 | 0.32 | 0 | 0.38 | 0.12 | 0 | 0.39 | 5.18 | 0.37 | 0.18 |
| Puck, spread | 0.09 | 0 | 0.35 | 0 | 0.08 | 0.57 | 0 | 0.61 | 6.90 | 0.55 | 0 |
| Kraft, slices 6 | 0.09 | 0.13 | 0.30 | 0 | 0.10 | 0.22 | 0 | 0.71 | 4.20 | 0.14 | 0 |
| Puck, toast, slices | 0.06 | 0.12 | 0.33 | 0.05 | 0.16 | 0.60 | 0 | 0.46 | 4.76 | 0.26 | 0.14 |
| Kraft, slices (4) | 0.14 | 0.21 | 0.46 | 0 | 0.22 | 0.75 | 0 | 0.68 | 6.29 | 0.26 | 0 |
| Puck, burger,slices | 0.08 | 0.11 | 0.31 | 0 | 0 | 0.41 | 0 | 0.26 | 4.66 | 0.27 | 0 |
| Al-Marai, byrger, slices 8 | 0.05 | 0.08 | 0.28 | 0 | 0.08 | 0.47 | 0 | 0.53 | 5.15 | 0.26 | 0.16 |
| Al-Marai, toast, slices (9) | 0.08 | 0.13 | 0.34 | 0 | 0.12 | 0.42 | 0 | 0.68 | 6.37 | 0 | 0 |
| Anchor, slices 10 | 0.06 | 0.13 | 0.32 | 0 | 0.12 | 0.49 | 0 | 0.78 | 4.56 | 0.20 | 0.21 |
| La Vach quirit, spread | 0.08 | 0.17 | 0.36 | 0 | 0.11 | 0.51 | 0.06 | 0.66 | 5.65 | 0.39 | 0.20 |
| Kiri, spread (12) | 0.08 | 0.17 | 0.42 | 0 | 0.12 | 0.58 | 0 | 0.90 | 6.41 | 0.34 | 0.25 |
| Puck, Mazzorella | 0.02 | 0.06 | 0.19 | 0 | 0.13 | 0.50 | 0 | 0.39 | 4.93 | 0.35 | 0 |
| Farmer, cream | 0.04 | 0.09 | 0.27 | 0 | 0.05 | 0.41 | 0.03 | 0.20 | 4.27 | 0.44 | 0 |
| Kraft | 0.08 | 0.15 | 0.39 | 0.05 | 0.18 | 0.59 | 0 | 0.72 | 4.86 | 0.22 | 0.20 |

Appendix P *continued*

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| **Milk:** | | | | | | | | | | |
| Goat, fresh | 0.01 | 0 | 0.02 | 0.12 | 0.09 | 0.32 | 0.01 | 0.90 | 0.03 | 0.16 |
| Cow, fresh | 0 | 0 | 0 | 0.02 | 0.04 | 0.20 | 0.07 | 0.95 | 0.03 | 0.17 |
| Sheep | 0 | 0 | 0 | 0.05 | 0.08 | 0.36 | 0 | 1.12 | 0 | 0.12 |
| Camel | 0 | 0 | 0 | 0 | 0.03 | 0.29 | 0.03 | 0.81 | 0.02 | 0.16 |
| Asafi | 0 | 0 | 0.01 | 0.03 | 0.07 | 0.28 | 0.02 | 1.05 | 0.02 | 0.36 |
| Nadec | 0.01 | 0 | 0.02 | 0.05 | 0.06 | 0.25 | 0.02 | 1.12 | 0.02 | 0.28 |
| Nada | 0.02 | 0 | 0.02 | 0.07 | 0.11 | 0.34 | 0.03 | 1.01 | 0.02 | 0.31 |
| AL-Matrood | 0 | 0 | 0.02 | 0.07 | 0.11 | 0.39 | 0.04 | 1.24 | 0.01 | 0.30 |
| Yogurt | 0 | 0.01 | 0.02 | 0.06 | 0.09 | 0.31 | 0.03 | 1.11 | 0.02 | 0.31 |
| **Meat & Poultry:** Lamb | 0 | 0 | 0 | 0 | 0 | 1.00 | 0 | 6.14 | 0.41 | 4.37 |
| Chicken fresh | 0 | 0 | 0 | 0 | 0 | 0.08 | 0 | 3.03 | 0 | 0.50 |
| Doux, chicken | 0 | 0 | 0 | 0 | 0 | 0.08 | 0 | 3.13 | 0 | 0.73 |
| Rudwa, chiken | 0 | 0 | 0 | 0 | 0 | 0.05 | 0 | 2.65 | 0 | 0.45 |

**Appendix P** *contunued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T 18:1C | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | | 18:2 | 18:3 |
| **Milk:** Goat, fresh | 0.01 | 0.02 | 0.04 | 0.01 | 0.02 | 0.06 | 0.01 | 0.02 | 0.46 | 0.04 | 0.01 |
| Cow, fresh | 0.02 | 0 | 0.05 | 0 | 0.06 | 0.23 | 0.03 | 0.54 | 0.83 | 0.09 | 0 |
| Sheep | 0.02 | 0 | 0.04 | 0 | 0 | 0.05 | 0 | 0 | 0.53 | 0.04 | 0 |
| Camel | 0.01 | 0.02 | 0.03 | 0.01 | 0.01 | 0.33 | 0.01 | 0.10 | 0.50 | 0.03 | 0 |
| Asafi | 0 | 0 | 0.03 | 0 | 0.10 | 0.11 | 0 | 0.08 | 0.83 | 0.09 | 0 |
| Nadec | 0 | 0.01 | 0.02 | 0 | 0.02 | 0.08 | 0 | 0.09 | 0.94 | 0.11 | 0.01 |
| Nada | 0.01 | 0.01 | 0.05 | 0 | 0.01 | 0.08 | 0 | 0.07 | 0.76 | 0.11 | 0 |
| AL-Matrood | 0 | 0.01 | 0.05 | 0 | 0.01 | 0.10 | 0 | 0.10 | 0.83 | 0.08 | 0 |
| Yogurt | 0.01 | 0.01 | 0.05 | 0.01 | 0.04 | 0.10 | 0.01 | 0.09 | 0.67 | 0.06 | 0.02 |
| **Meat & Poultry:** Lamb | 0.16 | 0 | 0 | 0 | 0 | 0.47 | 0 | 0.47 | 9.34 | 0.38 | 0 |
| Chicken fresh | 0 | 0 | 0 | 0 | 0 | 0.90 | 0 | 0 | 4.56 | 1.06 | 0 |
| Doux, chicken | 0 | 0 | 0 | 0 | 0 | 0.45 | 0 | 0 | 6.96 | 3.96 | 0.18 |
| Rudwa, chiken | 0 | 0 | 0 | 0 | 0 | 0.79 | 0 | 0 | 4.65 | 0 | 0 |

**Appendix P** *continued*

| Food item | Saturated | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 4:0 | 6:0 | 8:0 | 10:0 | 12:0 | 14:0 | 15:0 | 16:0 | 17:0 | 18:0 |
| Oils and fats: | | | | | | | | | | |
| Al-Arabi (palm) | 0 | 0 | 0 | 0 | 0 | 0.93 | 0 | 37.39 | 0 | 3.30 |
| Nakheel (palm) | 0 | 0 | 0 | 0 | 0.15 | 0.92 | 0 | 40.17 | 0 | 3.24 |
| Calaira (soya bean) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10.32 | 0 | 2.87 |
| Mazola (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10.77 | 0 | 1.40 |
| Afia (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10.70 | 0 | 1.55 |
| Sasso (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11.08 | 0 | 2.15 |
| Al-Wazir (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9.15 | 0 | 2.62 |
| Altayeb (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 16.78 | 0 | 1.70 |
| Golden chair (ghee) | 0 | 0.68 | 0.60 | 1.92 | 2.83 | 11.03 | 0.84 | 32.61 | 0.66 | 12.99 |
| Danish Lurbak (butter) | 0 | 0.37 | 0.50 | 1.45 | 3.27 | 10.63 | 0.72 | 31.84 | 0.48 | 9.83 |

**Appendix P** *continued*

| Food item | Unsaturated | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 14:1T | 14:1C | 15:1T | 15:1C | 16:1T | 16:1C | 17:1 | 18:1T | 18:1C | 18:2 | 18:3 |
| Oils and fats: | | | | | | | | | | | |
| Al-Arabi (palm) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 41.86 | 12.02 | 0 |
| Nakheel (palm) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 41.41 | 9.62 | 0 |
| Calaira (soya bean) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 20.75 | 54.76 | 6.80 |
| Mazola (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 25.89 | 56.57 | 0.87 |
| Afia (corn) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 24.99 | 57.59 | 0.68 |
| Sasso (olive) | 0 | 0 | 0 | 0 | 0.90 | 0 | 0 | 0 | 73.02 | 8.36 | 0 |
| Al-Wazir (olive) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 77.21 | 6.53 | 0 |
| Altayeb (olive) | 0 | 0 | 0 | 0 | 0 | 2.12 | 0 | 0 | 57.29 | 17.24 | 0.37 |
| Golden chair (ghee) | 0 | 0 | 1.32 | 0 | 0.40 | 2.26 | 0 | 1.78 | 22.99 | 0.86 | 0.58 |
| Danish Lurpak (butter) | 0 | 0 | 0.99 | 0 | 0.38 | 2.01 | 0 | 1.67 | 20.47 | 1.42 | 0 |