THE RISING BURDEN OF NON-COMMUNICABLE DISEASE AND FOOD AS MEDICINE.

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INTRODUCTION

As opposed to communicable diseases, non-communicable diseases (NCDs) are those diseases that cannot be transmitted from one person to another. These conditions which are chronic (cause a protracted period of impaired health), cause dysfunction or diminution of the quality of life and develop over relatively long periods of time. They usually persist for the life-time of the person affected and so place a massive burden on the individual's resources and that of the health system. The most common of these NCDs are cancer, respiratory diseases (chronic obstructive airway disease and asthma), cardiovascular diseases (heart attack and stroke) and diabetes as they account for approximately 70% of all deaths worldwide and 82% of all NCD deaths[1]. Cardiovascular disease (CVD) is the most common cause of NCD deaths (17.5 million annually), followed by cancer (8.2 million), respiratory diseases (4 million) and diabetes (1.5 million)[2,3].

According to WHO estimates, 38 million people now die from NCDs annually and of these, 75% (28 million) occur in low and middle income countries[1], a very steep rise from just under 40% in 1990[4]. About 16 million NCD deaths occur in people aged less than 70 years and 82% of the these "premature" deaths occur in low- and middle-income countries[2]. NCDs account for 48% of the healthy life years lost (Disability adjusted life years ? DALYs) worldwide versus 40% for communicable diseases, maternal and perinatal conditions and nutritional deficiencies, and 1% for injuries[1].

It is estimated that over the next 20 years, NCDs will cost more than US$ 30 trillion, representing 48% of global GDP in that time, and push millions of people below the poverty line[5]. By contrast, mounting evidence suggests that NCDs are preventable, meaning millions of deaths can be averted and economic losses reduced by billions of dollars if extra attention is paid to preventive measures such as healthy eating habits. A recent World Health Organization report states that population-based life-style adjustment measures aimed at reducing tobacco and harmful alcohol use, promoting healthy diet and improving physical inactivity, are estimated to cost US$ 2 billion per year for all low- and middle-income countries, which in fact translates to less than US$ 0.40 per person[5].

Healthy eating patterns are reported to result in lower circulating concentrations of inflammatory markers. A diet consisting of whole grains, vegetables and fruits, and fish will lower chronic low-grade inflammation. Meals rich in advanced glycation end products (AGEs) enhance oxidative stress and inflammation as do, saturated fatty acids and trans-mono unsaturated fatty acids. Poly unsaturated fatty acids especially of long chain n-3 variety are antinflammatory[6]

Inflammatory cells produce soluble mediators, such

as metabolites of arachidonic acid, cytokines and chemokines, which act by recruiting more inflammatory cells and producing reactive oxygen species (ROS). ROS can both induce changes in transcription factors, such as nuclear factor kappa B (NFKB), signal transducer and activator of transcription 3 (STAT3), hypoxia-inducible factor-1α (HF-1α), activator protein-1 (AP-1), nuclear factor of activated T-cells (NFAT) and NF-E2 related factor-2 (NrF2), which mediate immediate cellular stress responses, induction of cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS). This sustained inflammatory/oxidative environment is self-perpetuating and can lead to endothelial dysfunction, vascular remodelling, altered tone and vascular inflammation. It may also result in the introduction of gene mutations and structural alterations of DNA that damage DNA, blockage of intercellular communication and modification of second messenger systems leading to cell proliferation or a decreased apoptosis, among other changes characteristic of chronic disease[7,8,9]

SOCIO-ECONOMIC AND PUBLIC HEALTH IMPACT OF NCDS

People of all ages and in all countries of the world, including children, adults and the elderly are all susceptible to the risk factors that lead to NCDs as these diseases are associated with ageing, rapid unplanned urbanization and globalisation of unhealthy lifestyles[1]. NCDs are commonest in people aged 40 and over but there are worryingly increasing reports of type 2 diabetes occurring more frequently in children in the Africa sub region. The WHO reports that this previously rare situation is now so common that in some countries in the region, children and adolescents are now responsible for approximately half of all newly diagnosed cases of type 2 diabetes[10].

About half of all those aged 25 years and older in sub-Saharan Africa already suffer from hypertension, an established precursor for strokes and heart attacks[1] and according to the Commission on Ending Childhood Obesity (ECHO) overweight prevalence in children aged less than 5 years has risen from 4.8% to 6.1% between 1990 and 2014 with the absolute number of children so affected in the same period, increasing from 31 million to 41 million. For lower middle-income countries however, the number of overweight children has more than doubled from 7.5 million to 15.5 million, over the same period. In Africa, the number of children under 5 years of age who are overweight has nearly doubled from 5.4 million to 10.3 million since 1990[11].

Although Communicable diseases and other conditions still predominate in sub-Saharan Africa, NCDs are projected to be the leading cause of death by 2030. Almost 30% of NCD deaths in low- and middle-income countries occur in people under age 60, at the peak of their economic productivity compared to only 13% in high-income countries. Also, in sub-Saharan Africa, people die on average 10 years earlier than in developed countries[10].

Non-communicable diseases are closely associated with poverty because they markedly raise household costs associated with treatment of these diseases that endure for the life of the sufferers. Additionally, the rising burden of these diseases will negate any poverty alleviation measures the Government may put in place. The result is that poor members of our society suffer a steady deterioration in their health and die earlier than the more affluent members of society mainly because they are more likely to seek quick but potentially harmful solutions to their health conditions such as herbal medicine, to use tobacco and alcohol and to consume unhealthy food.

The massive cost of NCDs resulting from lengthy and expensive treatments involving antihypertensives, antidiabetics and antineoplastic drugs, and the loss to death of breadwinners is dragging a large proportion of Nigeria's population into poverty yearly, and preventing development.

RISK FACTORS FOR NCDs

The risk factors for NCDs can be described as modifiable behavioural risk factors or metabolic or physiological risk factors. The very high rate of poverty in many countries in sub-Saharan Africa leads to high rates of infectious diseases and leads to non-communicable disease risk factors such as drinking, smoking and poor diet, driving a double burden of disease. Tobacco use, physical inactivity, unhealthy diet and the harmful use of alcohol are considered the most important modifiable behavioural factors that increase the risk of NCDs by the World Health Organization[12].

About 6 million deaths every year are attributable to tobacco use, including those resulting from the effects of exposure to second-hand smoke. This figure is projected to increase to 8 million by 2030. Inadequate physical activity is responsible for approximately 3.2 million deaths annually with harmful use of alcohol (which is defined as the presence of physical and/or psychological complications)[2], contributing another 3.3 million deaths annually. One million, seven hundred thousand deaths due to cardiovascular diseases were directly attributable to excess salt or sodium intake in 2010[3].

The risky behaviours described above lead to four major metabolic/physiological changes that elevate the risk of NCDs and they include: high blood pressure, overweight/obesity, raised blood glucose levels (hyperglycaemia) and raised levels of fat in the blood (hyperlipidaemia). Of these, the leading metabolic risk factor globally is high blood pressure (responsible for 18% of deaths worldwide), followed by overweight/obesity and hyperglycaemia[12].

PATHOLOGICAL BASIS OF NON-COMMUNICABLE DISEASES

The Role of Reactive Oxygen Species in the Development of NCDs

Oxidative stress is defined as a disequilibrium between production of free radicals and reactive metabolites, also called oxidants or reactive oxygen species (ROS) and their neutralisation by protective agents called antioxidants. During regular metabolic processes in the body, aerobic cells produce ROS such as superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), hydroxyl radical (OH^-), and organic peroxides as normal products of the reduction of molecular oxygen[8,9].

All non-communicable diseases are linked in the sense that they are all associated with an increased inflammatory response, observed long before overt disease manifests. The sources of inflammation are varied and numerous and include microbial and viral infections, exposure to allergens, radiation and toxic chemicals, autoimmune and chronic diseases, obesity, consumption of alcohol, tobacco use, and a high calorie diet. In general, the longer the inflammation persists, the higher the likelihood of developing chronic disease (NCD)[7].

Ingestion of excess calories causes the mitochondria to be overloaded with glucose and fatty acids and an overproduction of acetyl coenzyme A (acetyl coA)[13], an important enzyme in cellular metabolism. Elevated levels of acetyl coA causes an increase in reduced nicotinamide adenine dinucleotide (NADH) generation from the tricarboxylic acid (TCA) cycle. The excess NADH increases electron production by Complex I of the mitochondrial electron transport chain and elevates membrane potential sufficiently to stall the activity of Complex III, resulting in a longer half-life for coenzyme Q. The longer half-life of coenzyme Q means it is available for a longer time to cause an increased reduction of oxygen to superoxide (O_2^-)[14].

Superoxide is unstable and is quickly converted to hydrogen peroxide (H_2O_2) in the mitochondria by superoxide dismutase. H_2O_2 undergoes a Huber-Weiss or Fenton reaction to produce a highly reactive hydroxyl radical (HO^-), which can oxidise mitochondrial proteins, DNA, lipids and amplify the effects of the superoxide-initiated oxidative stress[15,16]. ROS also activate redox-sensitive transcription factors which set off inflammatory cascades and cause the generation of more ROS (Figure 1).

Inflammatory cells produce soluble mediators, such as metabolites of arachidonic acid, cytokines and

chemokines, which act by further recruiting inflammatory cells and producing more reactive oxygen species (ROS). ROS can both induce changes in transcription factors, such as nuclear factor kappa B (NFKB), signal transducer and activator of transcription 3 (STAT3), hypoxia-inducible factor-1α (HF-1α), activator protein-1 (AP-1), nuclear factor of activated T-cells (NFAT) and NF-E2 related factor-2 (NrF2), which mediate immediate cellular stress responses, induction of cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS). This sustained inflammatory/oxidative environment is self-perpetuating and can lead to endothelial dysfunction, vascular remodelling, altered tone and vascular inflammation. They may also result in the introduction of gene mutations and structural alterations of DNA that damage DNA, blockage of intercellular communication and modification of second messenger systems leading to cell proliferation or a decreased apoptosis, among other changes characteristic of chronic disease[7,8,9].

The impact of oxidative stress on pancreatic β cells is a diminished expression of glucose transporter type 4 (GLUT 4), which over time can support the onset of type 2 diabetes mellitus. In endothelial cells, oxidative stress causes the formation of peroxynitrite radicals which decrease nitric oxide levels and result in defective endothelial dependent vasodilation, which over time leads to cardiovascular disease[6].

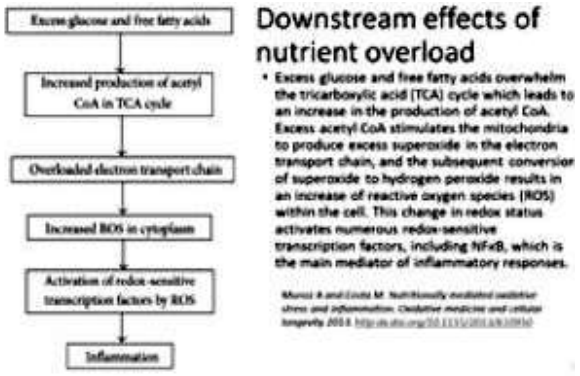


Figure 1: Downstream Effects of Nutrient Overload

PREVENTION AND CONTROL OF NON-COMMUNICABLE DISEASES

As described earlier, these conditions seem to be linked by a common pathological process and so solutions apply to all conditions. For a resource-poor country like ours, emphasis has to be placed on prevention. The WHO recommends that individuals adopt the following simple and inexpensive lifestyle adjustment measures:

1. Achieve and maintain a healthy body weight
2. Be physically active, engaging in regular moderate-intensity exercises for at least 30 minutes day on most days of the week, with a minimum of 120 minutes weekly.
3. Eat a healthy diet consisting of three to five servings of fruits and vegetables a day and a low content of sugar, salt, saturated fats and trans-fatty acids.
4. Avoid smoking or the use of tobacco in any form
5. Avoid harmful use of alcohol
6. Receive regular medical checks, including fasting blood sugar, blood lipids, PSA checks, mammograms etc.

Governments can help with fiscal policies that place a huge tax on the manufacture, importation and sale of sugary and high energy drinks such that their retail price goes up at least 20%. The reasoning is that low-income individuals who suffer most from the consequences of consuming these products are more likely to be dissuaded from purchasing them if their costs go up[10]. The WHO recommends that the revenues derived be invested in subsidising the cultivation of and cost of fruits and vegetables and by so doing encourage the consumption of fruits and vegetables. Additionally, fast food outlets should be made to use only oils that are free of saturated and trans fatty acids[10,12,17].

The most effective of these measures at ensuring that future generations are protected from non-communicable diseases is a change in our food culture. I will therefore focus the remainder of this paper in describing what constitutes a healthy diet.

FOOD AS MEDICINE

Food is defined simply as any substance that can be metabolised by an animal to give energy and build tissue. It is divided into macronutrients and micronutrients. Macronutrients are the classes of chemical compounds humans consume in largest quantities and which provide bulk energy. These are fat, protein and carbohydrate. Micronutrients are substances needed only in small quantities for normal body function and include vitamins and minerals.

Evolution of the human diet

The human diet has evolved through four stages, namely: the Miocene to early Pleistocene era, the Paleolithic era, the Neolithic era, and the Industrial Revolution. In the Miocene to early Pleistocene era, diets consisted of foliage, leafy vegetables, fruits, seeds and nuts and supplied high amounts of fiber, plant sterols, and vegetable proteins.

The diet of the Paleolithic man (Caveman) was not much different as it also consisted mainly of plant food but in addition, contained large amounts of animal protein derived from lean meat and seafood. The Paleolithic period ended about 10,000 years ago with the emergence of agriculture during the Neolithic period.

The Neolithic era was characterized by starchy foods in the form of grains and legumes as the main dietary staples. The diet also contained dairy products as well as vegetable oils such as olive oil. Neolithic diets were sources of large amounts of fiber, vegetable protein, and plant sterols.

The Industrial Revolution brought about the most significant change in the human diet, introducing the convenience and prepackaged foods including canned meats, condensed canned soups, hydrogenated vegetable oils, and refined grains, including white flour. A major consequence of the Industrial Revolution which has taken place over the past 200 years is a human diet rich in high glycaemic index carbohydrate sources (carbohydrate sources that cause a rapid rise in blood sugar), animal products, meat, saturated fat, and dietary cholesterol but deficient in legumes, vegetables, fruits and nuts[18].

The changes in the human diet over these past 10,000 years, it is now widely held, has adversely affected a number of dietary markers of health, including the amount of glucose in blood, fatty acid composition, macronutrient composition, micronutrient composition, acid/base balance, sodium/potassium ratio, and fiber content. It is also believed that these adverse effects have occurred because the changes that have taken place in our environment, including dietary and lifestyle shifts, have happened at a rate faster than the human genome could adapt to, and so humans are still biologically adapted to the environment of their ancestors[19]. This slow adaptation to modern diets (the basis of the paleolithic diet) has resulted in a multitude of chronic diseases in modern man, which our ancestors did not have.

Food as a remedy for illness or food as medicine became popular in the mid-1970s following publications by the gastroenterologist Walter Voegtlin and others. These scientists believed that adopting a nutritional plan based on the presumed ancient diet of the caveman of the paleolithic era that is known variously as paleolithic, caveman, stone age or hunter-gatherer diet, and that consisted mainly of wild plants and animals, protects against chronic disease. One of the major proponents of this diet, a Swedish scientist called Staffan Lindeberg did a number of studies, now collectively known as the Kitava Study, on the non-westernised population of Kitava, one of the Trobriand Islands of Papua New Guinea. These studies conducted between 1989 and 1993, found that the people of Kitava apparently did not suffer from stroke, ischaemic heart disease, diabetes, obesity or hypertension[20]. From about 1999, several doctors and nutritionists have argued in favour of a return to a paleothic diet or preagricultural diet and have formulated diets from modern foods that mimic the nutritional characteristics of the ancient paleothic diet[21].

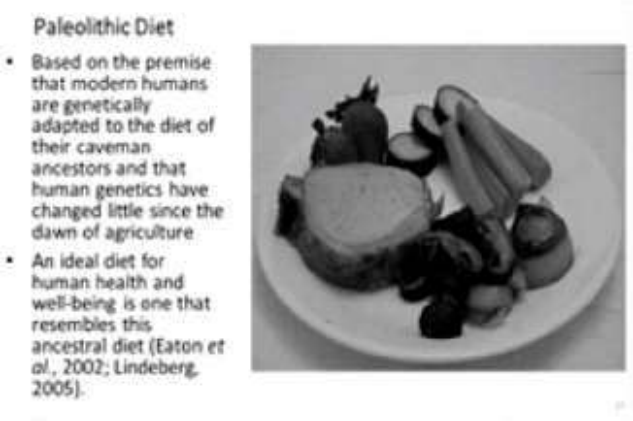


Figure 2: Paleolithic Diet

Mediterranean Diet

The Mediterranean diet is a modern nutritional recommendation inspired by the traditional dietary patterns of Southern Italy, Greece and specifically the Greek island of Crete and parts of the Middle East (Morocco).

In addition to regular physical activity, the Mediterranean diet emphasises abundant plant foods and fresh fruits as the typical daily dessert, olive oil as the principal source of fat, dairy products (principally cheese and yoghurt) and fish and poultry consumed in low to moderate amounts, zero to four eggs consumed weekly, red meat consumed in low amounts and wine consumed in low to moderate amounts. Total fat in this diet is 25% to 35% of calories, with saturated fats at 8% or less of calories.

The highlights of this diet include high olive oil consumption; high consumption of legumes; high consumption of unrefined cereals; high consumption of fruits; high consumption of vegetables; moderate consumption of dairy products (mostly cheese and yoghurt); moderate to high consumption of fish; low consumption of meat and meat products and moderate wine (particularly red wine)[25] (Ellen Gouch, 2005). Red wine contains flavonoids with powerful antioxidant properties[26] (Martinez-Gonzalez et al., 2010).

I shall in the remaining part of this paper, outline the potential benefits and adverse effects of ingested food and food supplements; and present evidence that components of the diet of early man when consumed as functional foods (foods that are consumed as part of the normal diet and contain biologically active components which can enhance health or reduce the risk of disease) today will reduce the risk of obesity, stroke, ischaemic heart disease, diabetes, cancer and osteoporosis.

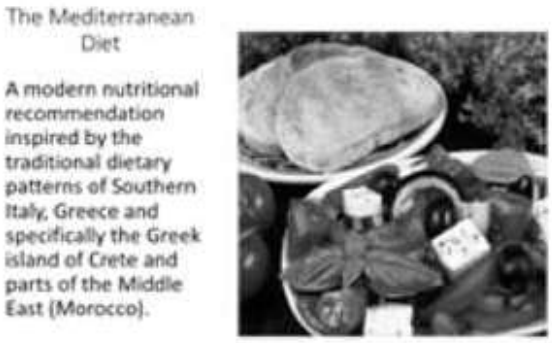


Figure 3: Mediterranean Diet

Evidence that food can be medicine

In the early 1950s the Rockefeller Foundation appointed Dr Leland Albaugh Field Director to investigate causes of high rates of death on the Greek Island of Crete. After systematically reviewing every aspect of life on Crete and comparing it to life in mainland Greece and America, Dr Albaugh reported that the rates of death in Crete were high, but the primary problem was poor public health infrastructure, poor access to good quality medical care and the availability of drugs to combat infections such as malaria, typhoid and dysentery. Importantly however, his data also showed that the adults of Crete had significantly better rates of heart disease than in America with 30% fewer deaths related to cardiovascular disease and just less than 30% deaths from cancer. Dr Albaugh's research showed that although Americans and the adults of Crete consumed similar daily calories, the food groups and nutrients for the main calories were different for the Crete and American adults (Table 1). Although the

significance of this finding was lost on the investigators at the time, it has now become the basis of a very popular functional diet called the Mediterranean diet.

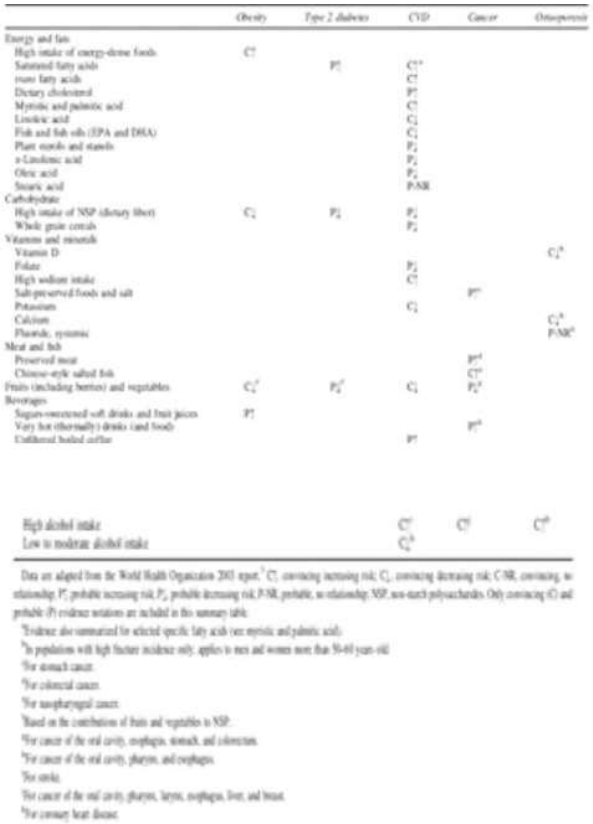
Table 1:
Sources of calories consumed by percentage in Crete Greece and United States in 1948.

Food Group	Crete Fall 1948	Greece 1948-1949	USA 1948-1949
Cereals	39	61	25
Potatoes	4	2	3
Sugar and honey	2	4	15
Pulses and nuts	7	6	3
Vegetables and fruits	11	5	6
Meat, fish and eggs	4	3	19
Dairy products	3	4	14
Oils and fats	29	15	15
Wine, beer & spirits		not given	not given
Total calories per person per day	2,547	2,477	3,129
Sources of protein in the diet, by percentage			
Animal protein	24	19	66
Vegetable protein	76	81	34

Emphasis is the author's.

A World Health Organisation (WHO) report published in 2003[27] (Table 2) summarised the links between diet and obesity, diabetes, cardiovascular disease (CVD), cancer and osteoporosis.

Table 2:
Summary of links between diet and obesity,diabetes, CVD, cancer & Osteoporosis



EVIDENCE OF HEALTH PROMOTING EFFECTS OF PALEOLITHIC AND MEDITERRANEAN DIETS

The nature of the Caveman and Mediterranean diets may have stimulated the design of therapeutic diets to manage chronic diseases in this and other countries[28,29,30]. Results from studies in human beings involving interventions using the Paleolithic or Mediterranean diet have been promising. For instance, a short term intervention trial using the Paleolithic diet by Osterdahl et al.,[29] in healthy human volunteers showed decreases in mean weight, body mass index, waist circumference, and systolic blood pressure. Additionally, dietary intake of fat, antioxidants such as vitamins C and E, and potassium-sodium ratio all showed favourable changes. Participants had adverse effects attributed to low levels of calcium because the Paleolithic diet excludes dairy products such as milk.

Another randomized, controlled, cross-over clinical study in the primary care setting, compared the Paleolithic diet with a commonly prescribed diet for type 2 diabetes. The Paleolithic diet resulted in lower mean values of HbA1c, triacylglycerol, diastolic blood pressure, body mass index, waist circumference and higher values of high density lipoprotein when compared to the Diabetes diet. Also, glycemic control and other cardiovascular factors were improved in both diets without significant differences[19]. It is also important to note that the Paleolithic diet was lower in total energy, energy density, carbohydrate, dietary glycemic load and glycemic index, saturated fatty acids and calcium, but higher in unsaturated fatty acids, dietary cholesterol and some vitamins.

The beneficial health effects of the Mediterranean diet are thought to be due to the eating of small portions, daily exercise and the emphasis on freshness, balance and pleasure in food. It has for instance been reported in the Seven Countries Study that Cretan men on a traditional Mediterranean diet consisting mostly of olive oil, large amounts of fruit and vegetables, fish and a moderate amount of dairy foods and wine, had exceptionally low death rates from heart disease, despite moderate to high intake of fat. The Lyon Diet Heart Study made some modifications to the Cretan diet by replacing olive oil with rapeseed (canola oil) increasing vitamin C-rich fruit and bread by 20% and decreasing processed and red meat. The researchers found that on this modified diet, mortality from all causes in the participants who had all survived a first heart attack, was amazingly reduced by 70%[31].

The British Medical Journal published a study in 2009 which reported that components of the Mediterranean diet, such as high vegetable consumption and low meat and meat product consumption, are more significantly associated with low risk of mortality than other components, such as cereal consumption and fish consumption[32].

As can be seen, all the health promoting nutritional plans described so far, contain fruits and vegetables and so before we make further progress let me quickly outline the health benefits of fruits and vegetables.

The culinary definition of vegetable is an edible plant or part of a plant but usually excludes seeds and most, sweet fruits. This usually means the leaf, stem, or root of a plant. Although botanically, a fruit is the ovary of a flowering plant, for culinary purposes, it is any edible part of a plant with a sweet flavor.

Apples like most fruits are naturally low in fat and calories, are filling and provide essential vitamins and minerals, fibre and other substances that are important for good health. A regular size apple has between 70-100 calories. Eating an apple when craving for candy or chocolate or other junk food such as doughnuts has a mellowing effect on blood sugar that suppresses the desire, since apple itself contains sugar but gives you only a quarter of the calories.

It is important to note however, that with fruits and vegetables, variety is just as important as quantity and that no single fruit or vegetable provides all the nutrients required to be healthy. The largest and longest running clinical trial to date, conducted as part of the Harvard-based Nurses' Health Study and Health Professionals follow-up study, involving 110,000 men and women whose health and dietary habits were monitored for 14 years, found that the higher the average daily intake of fruits and vegetables, the lower the risk of developing cardiovascular disease[33]. Combining the findings of this study with many other long-term studies in the United States and Europe and examining the effects on stroke and coronary vascular disease separately, the researchers found that individuals who ate more than 5 servings of fruits and vegetables a day, had approximately 20% lower risk of coronary heart disease and stroke when compared to people who ate less than 3 servings a day[34,35].

The Optimal Macronutrient Intake Trial for Heart Health (OmniHeart), a follow-up study to the Dietary Approaches to Stop Hypertension (DASH) study, which examined the effects of a diet rich in fruits, vegetables, and low-fat dairy products and which restricted the amount of saturated and total fat, found that blood pressure was lowered even more (DASH lowered systolic blood pressure by about 11 mmHg and diastolic blood pressure by about 6 mmHg) when some of the carbohydrate was

replaced with healthy unsaturated fat or protein[36]. A meta-analysis of studies and observational studies[37] has also found that consumption of a vegetarian diet was associated with lower blood pressure.

The link between fruits and vegetables and cancer is weak. In the Nurses' Health Study and the Health Professionals Follow-up Study for instance, over a 14-year period, men and women with the highest intake of fruits and vegetables (8+ servings a day) were just as likely to have developed cancer as those who ate the fewest daily servings (under 1.5)[33]. This is not surprising in the light of a very recent study which found that two thirds of all cancers are due to random genetic mutations[38]. A team of scientists from the Johns Hopkins Kimmel Cancer Center studied mutations that drive abnormal cell growth in 32 different types of cancer by developing a mathematical model for assessing the role of genetic copying errors in the development of cancer, using DNA sequencing and epidemiological data. Their results explain why people who lead a healthy life and have no family history of cancers may get the condition.

Several studies have reported [33,39,40, 41] that in men and women who were free of major chronic diseases, greater consumption of whole fruits – especially blueberries, grapes, and apples – is associated with a lower risk of type 2 diabetes but that greater consumption of fruit juice is associated with a higher risk of type 2 diabetes.

The indigestible fibre content of fruits and vegetables absorbs water and expands as it passes through the digestive system which can calm symptoms of an irritable bowel and, by triggering regular bowel movements, relieve or prevent constipation[42]. Diverticulosis may also be prevented by this bulking and softening action of insoluble fiber by decreasing pressure inside the intestinal tract[43]. Finally, it has been reported that eating fruits and vegetables can keep your eyes healthy, and may help prevent two common aging-related eye diseases—cataracts and macular degeneration—which afflict millions of Americans over age 65[44,45,46,47]. Lutein and zeaxanthin, in particular, seem protective against cataracts[48].

Antioxidant Supplements

It is thought that oxidation of low density lipoprotein in the blood contributes to heart disease, and initial observational studies found that people taking vitamin E supplements had a lower risk of developing heart disease. As a direct result, at least seven large clinical trials have been conducted to test the effects of antioxidant supplementation with vitamin E, in doses ranging from 50 to 600 mg per day. None of these studies found a statistically significant effect of vitamin E on overall number of deaths or on death due to heart disease. So, despite the clear role of oxidative stress in cardiovascular disease, controlled studies using antioxidant vitamins have observed no reduction in either the risk of developing heart disease, or the rate of progression of existing disease[49,50,51]

While some level of antioxidants, vitamins and minerals in the diet are required for good health, there is considerable doubt as to whether antioxidant supplements are beneficial or harmful and if they are actually beneficial, which antioxidants are needed and in what amounts. It is believed that excessive antioxidant levels may inhibit recovery and adaptation mechanisms and may also prevent many of the health gains that normally come from exercise such as increased insulin sensitivity.

Processed Foods

Refining is one of the measures by which processed foods are made. Refined flour has the brown husk of the wheat grain removed, leaving the white, refined starch found in white bread, white rice, pasta, biscuits and many other junk foods. Without the fibrous husk, refined starches are broken down quickly into glucose which is readily absorbed into the blood stream, causing glucose levels to rise quickly (hyperglycaemia) and inducing higher than normal levels of insulin secretion (hyperinsulinaemia) to bring it under control. Both these effects increase the risk of obesity and hypertension. Eating whole grains, such as, whole wheat bread, cereals, brown rice and barley with the bran surrounding intact, on the other hand, leads to a much slower absorption of sugar into the blood stream than occurs with refined starches and reduces the risk of obesity.

Additionally, refining destroys and devitalizes most good foods. Healthy unsaturated fatty acids are lost

during the milling process. Half the vitamin E is destroyed when the wheat germ and bran are removed. Refining wheat into flour removes between 50 and 93% of wheat's magnesium, zinc, chromium, manganese and cobalt. Approximately 50% of calcium, 70 percent of phosphorous, 80 percent iron, 50% potassium, 65% of copper, 80% of thiamin, 60% of riboflavin, 75% of niacin, 50% of panthotenic acid and about 50% of pyridoxine is lost[52].

Fast foods or junk foods are harmful because they are made from processed white flour and oils rich in trans-fats. Trans-fats are formed by the process of hydrogenation of vegetable oils to convert the readily oxidisable fatty acids such as linoleic acid into oxidation-resistant fatty acids. Oxidation of fatty acids makes vegetable oils rancid but the trans-fats (as opposed to cis-fats) that result from hydrogenation are unnatural and harmful. Trans-fats raise LDL- or bad cholesterol and lower HDL- or good cholesterol. Scientists do not yet know why, but the addition of hydrogen to oil increases blood cholesterol more than do other types of fats. It is believed that the addition of hydrogen to oil makes the oil more difficult to digest and the body recognises trans-fats as saturated fats. Junk foods (meat pies, doughnuts, sausages, and others foods prepared with hydrogenated oils including fried rice) are thus very high calorie (energy dense) meals which contain more fat, cholesterol, salt and sugar, fewer vitamins, minerals and other nutrients, than fresh food. Several studies have shown that apart from their nutritional deficiencies, fast foods promote weight gain. In addition, junk food consumption alters brain activity in a manner similar to addictive drugs like cocaine and heroin, leading to compulsive eating for pleasure[52].

What should we eat?

We could adopt a nutritional plan that is based on the Paleolithic diet or the Mediterranean diet, the components of which I have shared with you. However, doing so may pose significant challenges. For instance, the Paleolithic diet derives most of its macronutrients from wild game animal protein and we cannot very well, go out and catch an antelope or a grass cutter for lunch or dinner every time we are hungry. The Caveman diet also lacks Vitamin D and calcium because of the exclusion of dairy products. Some fears have also been expressed about the high salt content of some foods included in the Mediterranean diet, such as olives, salt-cured cheeses, anchovies, capers, salted roe, and salads dressed with extra virgin olive oil. More importantly, most of these diets are foreign to us and so the components are not readily available[52]

It may therefore, not be practical to convert solely to one or the other. It may however be easier and of greater value to us to evolve a diet plan that includes the health promoting components of these diets as functional foods and adopting a healthy lifestyle, to prevent chronic disease. The chart below, published by the Harvard School of Public Health summarises what we should be eating, drinking and doing[53].

Avoid junk food as much as possible. I say this because it may be near impossible to completely avoid these highly attractive and palatable dishes. Also avoid carbonated and sweetened soft drinks such as coke, sprite, malt drinks, alcohol (except moderate quantities of red wine) and tobacco. These things give you so much unwanted calories and actually contain harmful substances such as trans fatty acids and refined sugar. In place of food made out of white flour - white bread, cakes, biscuits, white spaghetti eat whole grains such as whole wheat bread, brown rice and whole grain pasta.

Use healthy oils such as olive oil and canola oil. Both of these oils are heart healthy and can be used for baking and cooking. However, olive oil is a wholly natural oil made by pressing ripe olives at cold temperatures to preserve the nutritional content and collecting their juices. Olive oil contains polyphenols, antioxidants and omega-3 fatty acids. Canola oil on the other hand is made from a hybrid of the rapeseed plant, and is a vegetable oil. Some people have reservations about canola oil because it is manufactured at high temperatures using processes that may involve the use of toxic chemicals such as hexane. It is de-gummed, deodorised, bleached and further refined at high temperatures. It is believed that these high temperatures may change the omega-3 content and significantly increase its content of trans fatty acids and saturated fats. Canola oil is however undoubtedly good for your heart and is endorsed by the British Heart Foundation.

Olive oil is available in the market in different forms. Extra virgin olive oil is the best and is collected from the first pressing of the olive. It contains the most nutrients. Virgin olive oil is collected from the second pressing. Oils derived from subsequent pressing are used to make light and pure olive oils, which may undergo further processing. It is recommended we eat fruits and vegetables with every meal, very day. Fill half your plate with vegetables at very meal and eat fruits and vegetables at snack time too. You should have at least 5 portions of fruits and vegetables each day. A portion of fruits or vegetable is: a dessert bowl of salad; a handful of grapes, cherries or berries; a glass of fruit juice (150 mls); two tablespoons of beans and pulses (lentils, kidney beans etc). Note that however much of fruit juice or beans and lentils you eat, you can only count these as one portion per day. One apple, one orange, one mango, banana, pear or other medium sized fruit; half a grape or avocado; one slice of a large fruit e.g. water melon or pineapple. To find out more about this or how much you need to eat for your weight, sex and physical activity level, please visit: http://www.cdc.gov/nutrition/everyone/fruitsvegetables /howmany.html

Eat healthy protein such as that derived from fish, poultry, beans and nuts. Limit red meat to small portions of pasture-raised beef and wild game meat. Remember that although the Caveman ate large amounts of meat, he was highly mobile (physically active) and also consumed large amounts of monounsaturated and polyunsaturated fatty acids, n-3 fatty acids and fruits and vegetables, all of which are believed to mitigate the adverse effects of high protein intake. Drink water, tea and coffee (with very little or no sugar), red wine, and freshly squeezed orange or some other fruit juice (at least one 150 ml glassful daily). Water contains no calories and the others all contain high amounts of natural antioxidants that have been proven scientifically to be beneficial. Avoid alcohol in the form of beer and spirits, sugary drinks (what we call mineral and malt drinks, including the diet versions.

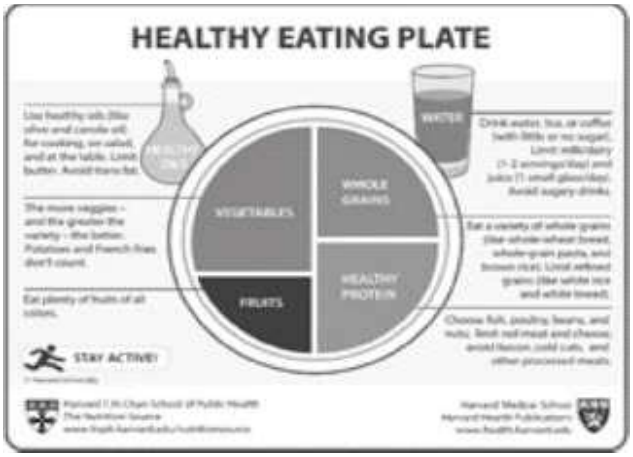
Finally, you must find time to exercise. It is recommended that you aim to take 10,000 steps a day but any amount of exercise daily is helpful. There are devices called pedometers that help you keep count of how many steps you have taken daily. The advantage in wearing them every day when you step out of your house is that you are consciously trying to meet a target and so you walk when you do not have to drive, you walk on the spot in your office while reading papers etc. You can get more information at www.cdc.gov/nutrition/everyone/basics/foodgroups.html and www.cdc.gov/nutrition/everyone/fruitsvegetables/index.html

How much should we eat?

You can get fat eating a perfectly healthy diet and so how much you eat is very important, whether it be fruits or vegetables alone or a Paleolithic or Mediterranean type diet. How much we should eat depends on many factors, including how tall we are, how old we are, whether we are male or female, our general state of health, what jobs we do, what leisure time activities we partake in, genetics, body size, body composition, and what medications we may be on. As a general rule, if we consume more than we use up by way of calories we gain weight and if the converse happens, we lose weight. There are certain other factors which may give you extra wiggle room on the amount you can eat: for example, if your food contains a lot of fiber you can usually eat more calories than if you eat food with a very low fiber content[53].

Put simply, how much you eat is very closely linked to the types of foods you eat and your lifestyle. Also, how much each one of us should eat depends on what our aims are: to maintain our body weight, lose or gain weight, or prepare for some sports event. However, any focus on food quantity intake is closely linked with calorie consumption.

Calories are a measure of how much energy there is in the food we eat. By understanding calories, you can work out how much food you need to eat. Different foods have varying number of calories per gram of weight. There are resources on the internet to help you work out how much calories you should be consuming.



CONCLUSION

Non-communicable diseases long considered a result of the ageing process, are increasingly more prevalent globally with a disproportionately greater burden in Africa and other low- to middle-income countries. Worryingly, these diseases are now a lot more common in children. To curb this rising burden, which is capable of dragging many households into poverty and stalling development, Nigerians need mainly to modify their diet, exercise more, avoid harmful alcohol use and tobacco, altogether. We in the medical community have a massive responsibility to reduce the burden of NCDs and to help reshape food systems in line with the UN declared Decade of Action of Nutrition 2016-2025.

REFERENCES

1. World Health Organisation. World Health Statistics 2015. WHO Press, Geneva. Available at http://apps.who.int/iris/bitstream/10665/170250/1/9789240694439_eng.pdf?ua=1&ua=1 Accessed November 2, 2016
2. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H *et al*. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*, 2012; 380(9859):2224-2260.
3. Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, Lim S *et al*.; Global Burden of Diseases Nutrition and Chronic Diseases Expert Group. Global sodium consumption and death from cardiovascular causes. N Engl J Med. 2014;371(7):624- 34. doi:10.1056/NEJMoa1304127.
4. Murray CJL and Lopez AD. Measuring the global burden of disease. N Engl J Med 2013; 369: 448-457. DOI: 10.1056/NEJMra1201534.
5. Bloom DE, Cafiero ET, Jané-Llopis E, Abrahams-Gessel S, Bloom LR, Fathima S, *et al*. (2011). The Global Economic Burden of Noncommunicable Diseases. Geneva: World Economic Forum, 2011. Available at http://www3.weforum.org/docs/WEF_Harvard_HE_GlobalEconomicBurdenNonCommunicableDiseases_2011.pdf Accessed November 5, 2016
6. Munoz A and Costa M. Nutritionally mediated oxidative stress and inflammation. Oxidative Medicine and Longevity 2013; Article ID 610950. Available at http://dx.doi.org/10.1155/2013/610950.
7. Reuter S, Gupta SC, Chaturvedi MM, Aggarwal BB. Oxidative stress, inflammation and cancer: How are they linked? Free Radic Bio Med, 2010; 49(11): 1603-1616.
8. Touyz RM. Reactive Oxygen species and angiotensin II signalling in vascular cells – implications in cardiovascular disease. Brazillian Journal of Medical Biological Research, 2004; 37: 1263-1273.
9. Valko M, Lieberfritz D, Mancol J, Cronin MTD, Mazur M, Tesler J. Free radicals and antioxidants in normal physiological functions and human disease. The International Journal of Biochemistry and Cell Biology 2007, 39(1): 44-84.
10. World Health Organisation. WHO factsheet updated January 2015. WHO Press, Geneva. Available at http://www.who.int/mediacentre/factsheets/fs355/en/
11. World Health Organisation. Report of the Commission on ending childhood obesity. WHO Press 2016. Available at http://apps.who.int/iris/bitstream/10665/204176/1/9789241510066_eng.pdf
12. World Health Organisation. Non-communicable diseases and their risk factors/ prevention of non-communicable diseases. WHO Press, Geneva, October 2016. Available at http://www.who.int/ncds/prevention/en/ Accessed November 5, 2016

13. Monnier L, Mas E, Ginetetal C. Activation of oxidative stress by acute glucose fluctuations compared with sustained chronic hyperglycemia in patients with type 2 diabetes. Journal of the American Medical Association 2006; 295(14):1681–1687.
14. Ceriello A and Motz E, "Is oxidative stress the pathogenic mechanism underlying insulin resistance, diabetes, and cardiovascular disease? The common soil hypothesis revisited," Arteriosclerosis, Thrombosis, and Vascular Biology 2004; 24(5): 816–823.
15. Ott M, Gogvadze V, Orrenius S, and Zhivotovsky B. Mitochondria, oxidative stress and cell death. Apoptosis 2007;12(5): 913–922.
16. Yin F, Sancheti H, and Cadenas E. Mitochondrial thiols in the regulation of cell death pathways. Antioxidants & Redox Signaling 2012; 17(12): 1714–1727.
17. World Health Organisation. The Global Action Plan for the Prevention and Control of NCDs 2013–2020. WHO Press, Geneva. Available at https://ncdalliance.org/sites/default/files/rfiles/NCD%20Alliance%20-%20Global%20NCD%20Action%20Plan.pdf
18. Jew S, AbuMweis SS, Jones PJH. Evolution of the human diet: linking our ancestral diet to modern functional foods as a means of chronic disease prevention. Journal of Medical Food 2009; 12(5): 925-934.
19. Frassetto LA, Schloetter M, Mietus-Synder M, Morris RC and Sebastian A. Metabolic and physiologic improvements from consuming a paleolithic hunter-gatherer type diet. European Journal of Clinical Nutrition 2009; 63(8):947-955.
20. Lindeberg S, and Lundh B. Apparent absence of stroke and ischaemic heart disease in a traditional Melanasian island: a clinical study in Kitava. J Intern Med 1993; 233:269-275
21. Eaton SB, Cordain L. Evolutionary aspects of diet: old genes, new fuels. World Rev Nutr Diet 1997; 81:26– 37.
22. Lindbergh S (2005). Paleolithic diet (Stone Age diet). Scandinavian Journal of Food and Nutrition 49(2): 75-77.
23. Eaton SB, Cordain L and Lindeberg S. Evolutionary health promotion: a consideration of common counterarguments. Prev Med 2002; 34:119-123.
24. Strohle A, Wolters M, Hahn A. Carbohydrates and the diet—atherosclerosis connection—more between earth and heaven. Comment on the article "The atherogenic potential of dietary carbohydrate." Preventive Medicine 2007; 44 (1): 82-84.
25. Robinson KM. The Mediterranean diet. WebMD. Available at http://webmd.com/diet/a-z/themediterranean-diet. Accessed March 2017
26. Martínez-González MA, García-Arellano A, Toledo E, Salas-Salvadó J, et al., for the PREDIMED study investigators. A 14-Item Mediterranean Diet Assessment Tool and Obesity Indexes among High-Risk Subjects: The PREDIMED Trial 2010. http://www.plosone.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.004 3134&representation=PDF Accessed January 2014.
27. World Health Organisation. Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO Expert Consultation. WHO Technical Report Series 916, 2003. Geneva.
28. Jonson PM, Kenny PJ. Addiction-like reward dysfunction and compulsive eating in obese rats: role for dopamine D2 receptors. Nature Neuroscience 2010; 13 (5): 635-641.
29. Osterdahl M, Kocturk T, Koocheck A, Wandell PE. Effects of a short-term intervention with a Paleolithic diet in healthy volunteers. Eur J Clin Nutr 2008; 62:628-685.
30. Lindberg S, Jonsson T, Granfeldt Y et al. A Paleolithic diet improves glucose tolerance more than a Mediterranean-like diet in individuals with ischaemic heart disease. Diabetologia 2007; 50:1795-1807.
31. Kris-Etherton P, Eckel RH, Howard BV, St. Jeor S et al., for the Nutrition Committee, Population Science Committee and Clinical Science Committee of the American Heart Association. Circulation 2001; 103: 1823-1825.
32. Trichopoulou A, Bamia C, Trichopoulos D. Anatomy of health effects of Mediterranean diet: Greek EPIC propective cohort study. BMJ 2009; 338:b2337 Available at www.bmj.com/content/338/bmj.b2337.
33. Hung HC, Joshipura KJ, Jiang R, Hu FB et al. Fruit and vegetable intake and risk of major chronic disease. J Natl Cancer Inst 2004; 96(21): 1577-1584.

34. He FJ, Nowson CA, Lucas M and MacGregor GA. Increased consumption of fruits and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. J Hum Hypertens 2007; 717-728.
35. He FJ, Nowson CA and MacGregor GA Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. Lancet 2006; 367(9577): 1320-1326.
36. Appel, LJ, Sacks FM, Carey VJ, Obarzanek E et al., Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. JAMA, 2005. **294**(19): p. 2455-64.
37. Yokoyama Yoko, Nishimura Kunihiro, Barnard Neal D, Takegami Misa, Watanabe Makoto, Sekikawa Akira, Okamura Tomonori, Yoshihiro Miyamoto. Vegetarian Diets and Blood Pressure: A Meta-analyisis. JAMA Intern Med. 2014;174(4):577-587.
38. Tomasetti C, Li L, Vogelstein B. Stem cell divisions, somatic mutations, cancer etiology and cancer prevention. Science 2017; 335(6331:1330-1334. Available at http://science.sciencemag.org/content/355/6331/1330 . Accessed April 6, 2017.
39. Muraki I, Imamura F, Manson JE, Hu FB et al., Fruit consumption and risk of type 2 diabetes: results from three prospective longitudinal cohort studies. BMJ, 2013. **347**: p. f5001.
40. Bazzano LA, Li TY, Joshipura KJ, and Hu FB. Intake of fruit, vegetables, and fruit juices and risk of diabetes in women. Diabetes Care, 2008. **31**(7): p. 1311-7.
41. Mursu J, Virtanen JK, Tuomainen TP, Nurmi T and Voutilainen S. Intake of fruit, berries, and vegetables and risk of type 2 diabetes in Finnish men: the Kuopio Ischaemic Heart Disease Risk Factor Study. Am J Clin Nutr, 2014. **99**(2): p. 328-33.
42. Lembo, A. and M. Camilleri, Chronic constipation. N Engl J Med, 2003. **349**(14): p. 1360-8.
43. Aldoori WH, Giovannucci EL, Rockett HR, Sampson L, Rimm EB and Willet WC. A prospective study of dietary fiber types and symptomatic diverticular disease in men. J Nutr, 1998. **128**(4): p. 714-9.
44. Brown L, Rimm EB, Seddon JM, Giovannucci EL et al. A prospective study of carotenoid intake and risk of cataract extraction in US men. Am J Clin Nutr, 1999. **70**(4): p. 517-24.
45. Christen WG, Liu S, Schaumberg DA and Buring JE. Fruit and vegetable intake and the risk of cataract in women. Am J Clin Nutr, 2005. **81**(6): p. 1417-22.
46. Moeller SM, Taylor A, Tucker KL, McCullough ML et al. Overall adherence to the dietary guidelines for americans is associated with reduced prevalence of early age-related nuclear lens opacities in women. J Nutr, 2004. **134**(7): p. 1812-9.
47. Cho E, Seddon JM, Rosner B, Millet WC and Hankinson SE. Prospective study of intake of fruits, vegetables, vitamins, and carotenoids and risk of age-related maculopathy. Arch Ophthalmol, 2004. **122**(6): p. 883-92.
48. Christen WG, Liu S, Glynn RJ, Gaziano JM and Buring JE et al. Dietary carotenoids, vitamins C and E, and risk of cataract in women: a prospective study. Arch Ophthalmol, 2008. **126**(1): p. 102-9.
49. Vivekenantham DP, Rem MS, Supp SK, Hsu A, Topol EJ (2003). Use of antioxidant vitamins for the prevention of cardiovascular disease: meta-analysis of randomised trials. Lancet, 361(9374): 2017-2023.
50. Lee IM, Cook NR, Gaziamo JM (2005). Vitamin E in the primary prevention of cardiovascular disease and cancer: the Women's Health Study: a randomised controlled trial. JAMA, 294(1): 56-65.
51. Sesso HD, Buring JE, Christen WG (2008). Vitamins E and C in the prevention of cardiovascular disease in men: the physicians Health Study II randomised controlled trial. JAMA, 300(18): 2123-2133.
52. Siminialayi IM. That it may be well with us: food as medicine and poison. Inaugural lecture series no 109, August 27 2014, University of Port Harcourt, Nigeria. Available at www.uniport.edu.ng/files/.../109%20Inaugural%20Lecture%20Siminialayi.pdf
53. Harvard T.H. Chan School of Public Health. The healthy eating plate and healthy eating pyramid. Accessed November 5, 2016. Available at https://www.hsph.harvard.edu/nutritionsource/healthy-eating-plate/