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Adverse Effect of Fat Intake on Insulin Sensitivity and Associated Risk of Non-Communicable Diseases (NCD): A Review

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Abstract
The objective of this review was to address long term intake of dietary fat result fat induced Insulin resistance and other non communicable diseases with reference to hypertension, diabetes, obesity, cancer, cholesterol and cardio vascular diseases. Dietary fats, or lipids, are a macronutrient class that includes fatty acids, triglycerides, and cholesterol. Fatty acid quantity and quality also vary by their source, with important differences between meat, fish, and plant sources, as well as natural versus synthetic sources. This heterogeneity allows for food consumption choices to modulate the quantity and quality of fats that, in turn, influence metabolic and health outcomes. The dietary intake of fat has received considerable attention in the past few decades because of its link with an increased risk of coronary heart disease (for high intakes of saturated fatty acids and cholesterol) and obesity (for a high intake of total fat). Currently, several lines of evidence indicate that the type of fat is more important in decreasing metabolic and CVD risk than the total amount of fat in the diet. The scientific evidence is clear that a high-fat diet relates to chronic health problems such as heart disease, cancer, diabetes, and obesity. Therefore that, books, cookbooks, and magazine articles tout low-fat messages. Saturated fat or the bad fats are a significant risk factor for cardiovascular diseases (CVD) including heart disease and stroke through raising blood total cholesterol and LDL cholesterol levels. On the other hand, the good fats or the unsaturated fats – monounsaturated fats, omega 3 polyunsaturated fats (PUFA) and omega 6 polyunsaturated fats primarily protect against CVD through their effects on cholesterol i.e. lowering harmful LDL cholesterol and raising protective HDL cholesterol. Concerning Insulin sensitivity and resistance, it is important to understand TFA consumed during long periods could promote insulin resistance and have clinically relevant effects on diabetes risk. The reviews showed that Insulin resistance is associated with type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVD) and thus, Insulin resistance (IR) is considered the key mechanism unifying obesity, diabetes and heart disease. Globalization and Immigration also have contribution to increased consumption of high-fat, energy-dense diets, particularly rich in saturated fatty acids (SFAs). All age groups and all regions are affected by NCDs. Children, adults and the elderly are all vulnerable to the risk factors that contribute to noncommunicable diseases, whether from unhealthy diets, physical inactivity, exposure to tobacco smoke or the effects of the harmful use of alcohol. The situation is double burden for low-income and middle-income countries and is the most remaining public problem. This clearly shows that there must be global action for the prevention and control of noncommunicable diseases focussing on healthy diet and physical activity as a public health priority.
Keywords: dietary fat, Insulin resistance, NCD and globalization

INTRODUCTION

Dietary fats, or lipids, are a macronutrient class that includes fatty acids, triglycerides, and cholesterol. Fats supply fuel energy (9 kcal/g) and the essential fatty acids, linoleic and alpha-linolenic acids (Donahoo W, et al., 2008). Fats, therefore, are a key factor in the maintenance of caloric balance and body weight (J. Virtamo, et al., 2009). Specific fatty acids also serve as precursors for numerous biological pathways that influence inflammation, coagulation, and gene expression among other functions. Fat soluble vitamins (vitamins A, D, E, K) and carotenoids are absorbed and transported with fats (Dietary Guidelines Advisory Committee, 2003).

Fatty acids are bound to glycerol as triglycerides for transport and storage in the human body. Fatty acids are heterogeneous and classified based on their chain length, the number of double bonds, the position of the first double bond from the methyl end, and a *cis* versus *trans* configuration across a double bond (Siri-Tarino PW, et al., 2010). These heterogeneities are important determinants of the significant variation in biological effects of the different fatty acids. Fatty acid quantity and quality also vary by their source, with important differences between meat, fish, and plant sources, as well as natural versus synthetic sources (Hu FB, and Willett WC. 2001). This heterogeneity allows for food consumption choices to modulate the quantity and quality of fats that, in turn, influence metabolic and health outcomes (WHO/FAO, 2003).

Currently, several lines of evidence indicate that the type of fat is more important in decreasing metabolic and CVD risk than the total amount of fat in the diet (Bray GA, et al., 2002). Metabolic studies have established that it is the type of fat, rather than total fat intake that affects common intermediate risk factors, such as serum lipid and lipoprotein levels (Ravnskov U, et al., 2002). Results from controlled clinical trials and epidemiological studies have shown that replacing SFA with unsaturated fats is more effective in decreasing CVD risk than is reducing total fat intake overall (Ascherio A, 2002).

The dietary intake of fat has received considerable attention in the past few decades because of its link with an increased risk of coronary heart disease (for high intakes of saturated fatty acids and cholesterol) and obesity (for a high intake of total fat)(Astrup A, et al., 2000). Ingested fat is insoluble oil at body temperature, a reason why the basic process in fat absorption is the conversion of this oil into water-soluble compounds that can be efficiently absorbed (Field AE, et al., 2007).

Fat intake lowering keep saturated fatty acid, *trans* fatty acid, and cholesterol intakes as low as possible while consuming a nutritionally adequate diet (Tucker LA, and Kano MJ, 1999). Because In the absence of sufficient dietary fat, the body is apparently capable of synthesizing the saturated fatty acids that it needs from carbohydrates, and these saturated fatty acids are principally the same ones that are present in dietary fats of animal origin (Lichtenstein AH, and Schwab US. 2000). Saturated fat is a significant risk factor for cardiovascular diseases (CVD) including heart disease and stroke through raising blood total cholesterol and LDL cholesterol levels. The primary sources of dietary saturated fat are meat, dairy products, processed foods and palm oil. While availability of saturated fat in low and middle income countries is currently below 10% of energy, across all world regions, availability of dietary energy from total fat has been rising (Abbott WGH, et al., 2001). It is imperative that global targets and indicators for saturated fat are put in place to prevent high saturated fat intakes and high cholesterol becoming a widespread problem, particularly in low income countries.

Classification of fats

The good fats

The good fats include the unsaturated fats – monounsaturated fats, omega 3 polyunsaturated fats (PUFA) and omega 6 polyunsaturated fats (Lichtenstein AH, et al., 1994). These primarily protect against CVD through their effects on cholesterol i.e. lowering harmful LDL cholesterol and raising protective HDL cholesterol (Chiu CC, et al., 2012). Polyunsaturated fats lower both LDL and HDL, while monounsaturated fats lower LDL but tend to preserve HDL levels. Omega-3 fatty acids tend to increase HDL and lower triglycerides.

These types of fat melt at a lower temperature and therefore are much more likely to be oil at room temperature. Consuming these types of fats, in place of saturated fats, has been shown to reduce the risk for many of the outlined diseases mentioned below that are associated with high saturated fat intakes (Hooper L, 2001). Great sources include: olive oil, peanut oil and most nuts such as avocados, almonds and peanuts. Also, vegetable oils such as corn, sunflower and soybean oil are all primarily polyunsaturated and, two important unsaturated fatty acids, omega-3 and omega-6, are *essential* fatty acids as they cannot be made by the body and are vital in the diet- more on these special fats below (Hooper L. *et al*., 2004).

Omega 6 oils: A large body of evidence supports the health impact of linoleic acid (the principle omega 6 oil) including meta analyses of clinical trials, and long-term prospective cohort studies (Hooper L. *et al*., 2004). In a recent Science Advisory for the American Heart Association, reviewed the evidence of omega 6 oils and cardiovascular disease, and found agreement in the evidence from different types studies. A meta-analysis of RCTs which substituted saturated fats for omega 6 oils found a 24% decreased risk of CHD. Epidemiologically, substituting 10% of calories from saturated fat with omega 6 fats was associated with an 18mg/d decrease in LDL cholesterol (Hooper L. *et al*., 2004). While the Nurses Health Study found women with the highest intakes of omega 6 fats (7% of energy) had a 25% reduced risk of CHD compared to those in the lowest intakes (2.8% energy). As a guide, the American Heart Association recommends up to 10% omega 6 polyunsaturated fatty acids (PUFA) and up to 1.2% omega 3 oils (Chiu CC, et al., 2012). The rather dated UK COMA report cautiously recommends that 6.5% of total energy should come from omega 3 and omega 6 oils. Intake levels in the UK population average 5.8% of energy (Chiu CC, et al., 2012).

Omega 3and Oily fish oils: Evidence for the protective benefits of oily fish on CVD is abundant (Mozaffarian, 2008) recently reviewed the evidence on omega 3 oils and CVD from observational studies, RCTs and experimental studies. Among the findings, a meta-analysis of over 15 cohort studies and RCTs found a 36% lower risk of CHD death in those who ate an average of 250mg per day (equivalent of 1 portion per week) compared with those who did not eat any omega 3 fats . The recommended intake amount is at least one portion of oily fish per week; UK intakes average 1/3 of a portion (Mozaffarian, 2008).

The bad fats

Saturated fats are usually solid at room temperature and are white or yellowish in color. They are found in animal products (dairy, meat, and poultry) and tropical oils such as coconut oil and palm kernel (mostly used in the food industry for baked goods). Excessive intakes of saturated fat are the principal cause of elevated LDL

levels in Western diets (Ascherio A, 2002).

UK intakes average at around 13% in adults and over 80% of the UK population exceed the maximum recommended intakes of 10% of food energy. Saturated fats are a much worse fat choice compared to unsaturated fatty acids (Hu, F.B., et al., 2001). Generally, they come from animal sources and tend to be solid at room temperature, such as the fat found on the edges of a steak. Vegetable sources of saturated fat include palm oil, palm kernel oil and coconut oil. It is well known that consuming large amounts of saturated fat is a positive risk factor in the development of hypertension, cancers and cardiovascular disease (Hu, F.B., et al., 2001).

Tran's fats: Tran's fats raise the bad LDL cholesterol and lower the good HDL cholesterol, and therefore damage health. Evidence from both RCTs and prospective studies have demonstrated harmful effects on CHD risk factors and outcomes (Louheranta AM, et al., 1999). A recent meta-analysis of randomised trails and prospective cohort studies found that replacing 1% of food energy from trans fats with plant-based unsaturated fats reduced CHD risk by 12% (Louheranta AM, et al., 1999). The UK Government recommends maximum trans fat intakes of up to 2% of total energy. Average intake levels for adults are 1.0% of energy, but a minority on low income consume up to 6% Trans-fatty acids, are called hydrogenated fats, are man-made and are probably more adverse to your health than even saturated animal fats(Bendsen, N. et al., 2011). Trans-fats are put into high fat foods that are required to have a long shelf life, such as snack foods found in convenience stores. Trans-fats are also found in many crackers, cookies, pre-packaged goods and universally in highly processed foods with a high fat content (Nishida C and Uauy R, 2009).

Strict avoidance of partially hydrogenated fats (often called "trans" fats) is crucial. These are chemically modified fats that are found in most margarines, vegetable shortening, and a large percentage of commercial baked goods (including most breads, crackers, cookies, pastries, etc.) and snack foods (Lichtenstein AH, et al., 2003). Everyone who cares about insulin resistance and protecting their cardiovascular system needs to read labels (look for the words "partially hydrogenated" followed by any oil) and try to avoid these artery-damaging and cell-stiffening fats to the greatest extent possible. These partially hydrogenated fats have a rigid structure. If you consume them, they will be incorporated into cell membranes (the outer layer of cells) and cause stiffening and dysfunction of both the membrane and the receptors within the cell membrane, including insulin receptors (Willett WC, et al., 2003). The result can be increased insulin resistance. Another variety of bad fats are oxidized fats (any oil that has been heated to very high temperatures, particularly when it is re-used over and over, and, thus, all the fried foods made with such oils). These oxidized fats cause cell membrane damage in a different way, and can also harm cellular and receptor function (Maron DJ, et al., 2001).

Controversy over saturated fat intake

Is dietary fat important? The question of whether dietary fat plays a role in the risk of obesity has produced a divergence of opinions. Some argue that dietary fat plays an important role in whether people readily become fat and others feel that it is unimportant. Whether fat plays a role or not, a chronic positive imbalance of calories over time is the primary cause of excess storage of body fat (Ravnskov U. 2002). Diet plays a role by providing the calories for the chronic imbalance of energy intake that slowly produces obesity. If a high amount of dietary fat enhances the risk of obesity, it is by providing calories in excess of what is needed. It is clear that no specific food, diet, or dietary pattern is essential to become obese. High-fat diets usually mean increased intakes of saturated fat.

Epidemiologic data suggest that saturated fats increase the concentration of LDL cholesterol in the bloodstream of some persons and that elevated cholesterol concentrations heighten the risk of heart disease (Ravnskov U. 1998). However, from a pragmatic food-choice perspective, it is impossible to achieve a nutritionally adequate diet that has no saturated fat. Controversy exists about the roles that dietary fat and cholesterol play in the risk of heart disease. Confounding factors influence the interpretation of results of epidemiologic studies. For example, over the years, France and Finland—populations that have similar intakes of cholesterol and saturated fat—have consistently had very different mortality rates from CAD. Some epidemiologic evidence suggests that consumption of high amounts of saturated fat and cholesterol lead to high blood cholesterol and thus to an increased risk of heart disease.

The logic that dietary fat is unhealthy is based on the fact that high intakes of saturated fat elevate blood cholesterol and thus increase the incidence of atherosclerosis, which then increases the risk of CAD (Lada AT, and Rudel LL. 2003). In the modern nutrition approach, saturated fatty acids, *trans* fatty acids, and dietary cholesterol have no known beneficial role in preventing chronic disease and are not required at any level in the diet. Both types of fat increase the risk of heart disease in some people by boosting the level of harmful, low-density cholesterol in the bloodstream; this occurs even with very small quantities in the diet (Mozaffarian D et al. 2010). Since there is no intake level of saturated fatty acids, *trans* fatty acids, or dietary cholesterol at which there is no adverse effect, no UL (*upper* limit) is set for them; instead, the recommendation is to keep their intake *as low as possible* (Dietz WH, et al., 2012).

Widely recommended as a way to reduce the risk of coronary heart disease because populations with

low intakes of saturated and total fat tend to be at low risk and because saturated fat increases low-density lipoprotein (LDL) cholesterol levels (G. Davey Smith. 2011). However, low-fat, high-carbohydrate diets also reduce high-density lipoprotein (HDL) cholesterol levels and raise fasting levels of triglycerides. Because low levels of HDL cholesterol and high levels of triglycerides independently increase risk, the value of replacing fat in general with carbohydrates has been questioned (UAUY-DAGACH R, 1999). Replacing saturated fat and trans unsaturated fat with unhydrogenated unsaturated fats has clear beneficial effects on blood lipids and thus provides an alternative strategy for reducing the risk of coronary heart disease (Donnelly JE, et al., 2008). Interventions to prevent and control the growing burden of NCDs are:

- reduction of saturated fat consumption : target intake of less than 10% of total dietary energy from saturated fat is recommended by WHO for the prevention of CVD
- reduction of salt consumption
- elimination of industrially produced trans fatty acids
- limit intake of free sugars
- corresponding increase in consumption of polyunsaturated fats up to 10% of total energy would be acceptable and increase consumption of fruits and vegetables
- achievement of a healthy weight
- practice of adequate levels of physical activity
- low-FAT, high-carbohydrate diets have been used

Insulin resistance and Fat intake

Insulin resistance is the experimental or clinical condition in which the hormone exerts a biological effect less than expected or Insulin resistance is the failure of insulin sensitivity (Simopoulos AP. 1994). Therefore, Insulin resistance, as a key mediator of metabolic syndrome, is thought to be associated with pathogenesis of aortic valve disease and altered left ventricular (LV) function and structure. However, in patients with aortic valve sclerosis (AVS), the association between insulin resistance and subclinical impairment of LV function is not fully elucidated (Simopoulos AP. 1994).

Trans fatty acids (TFA) could affect cell membrane functions, and may therefore influence peripheral insulin sensitivity and the risk of developing type 2 diabetes (Rivellese AA, and Lilli S, 2003). It is important to understand whether low amounts of TFA consumed during long periods could promote insulin resistance and have clinically relevant effects on diabetes risk. Data from controlled intervention studies examining the effects of TFA on insulin sensitivity and type 2 diabetes are reviewed. The results show no consistent effect of TFA on insulin sensitivity in lean healthy subjects, but there is some evidence that TFA could impair insulin sensitivity more than unsaturated fat in subjects with insulin resistance or type 2 diabetes (Rivellese AA, and Lilli S, 2003).

In particular, conjugated TFA, i.e. certain isomers of conjugated linoleic acid (CLA), impair insulin sensitivity and could promote metabolic disorders. The effect of CLA (trans10cis12) on insulin sensitivity and lipid peroxidation is the most dramatic adverse effect described for a dietary fatty acid (Moloney F, et al., 2004). CLA isomers are found in relatively low amounts, but long-term exposure may, in theory, have unwanted health effects. The mechanisms of CLA effects are still not completely understood, but may involve increased oxidative stress and inflammation, as well as endothelial dysfunction and direct down-regulating effects on transcription factors required for optimal insulin sensitivity. As mentioned above, oxidative stress and inflammation could be involved in promoting insulin resistance in humans supplemented with high doses of CLA TFA isomers (Jung MY, and Ha YL. 1999). A recent study in mice supported such a hypothesis, demonstrating increased insulin resistance and signs of local inflammation in adipose tissue, as well as induction of cytokine gene expression after treatment with trans10cis12-CLA. This shows that insulin resistance is fat induced health problem (Jung MY, and Ha YL. 1999).

In another case, weight gain and obesity can develop insulin resistance syndrome: this is because Obesity and weight gain have been associated independently with hypertension, hyperinsulinemia, and dyslipidemia(abnormal amount of lipids in the blood); however, prior research has not looked at the relation between weight gain from early adulthood to middle age and the development of this cluster of risk factors, known as insulin resistance syndrome(Rivellese AA, and Lilli S. 2003). Yet, insulin resistance is strongly related to several classic cardiovascular risk factors such as hyperglycemia, obesity, high triglycerides, low HDL cholesterol, hypertension and microalbuminuria. Several routes seem to link insulin resistance to cardiovascular disease, one is going through classic risk factors (e.g.,diabetes, dyslipidemia and hypertension), another one is going through non-classic risk factors (e.g. coagulation and fibrinolytic abnormalities) (American Diabetes Association, 2000).

Syndromes of Insulin resistance (IR)

Insulin resistance is associated with type 2 diabetes mellitus (T2DM), cardiovascular diseases (CVD), both independently and in association with the insulin resistance syndrome (Ferrannini E, et al., 1999). Decreased

insulin sensitivity has been documented in those known to be at risk for T2DM, such as normoglycaemic first-degree relatives of patients with T2DM, or women with history of gestational diabetes2,3 (Ferrannini E, et al., 1999).

It is now well established that generalized obesity and abdominal obesity (excess subcutaneous and intra-abdominal fat) are associated with insulin resistance. Other important factors contributing to insulin resistance include accumulation of hepatic fat and intra-myocellular lipids, both of which can exist independent of generalized adiposity. People diagnosed insulin resistance have the following signs (Kaplan NM, 1992).

- High blood sugar. When people have insulin resistance, glucose builds up in the blood instead of being absorbed by the cells, leading to type 2 diabetes or prediabetes.
- Brain fogginess and inability to focus
- Intestinal bloating – most intestinal gas is produced from carbohydrates in the diet, mostly those that humans cannot digest and absorb.
- Sleepiness, especially after meals.
- Weight gain, fat storage, difficulty losing weight – for most people, excess weight is from high fat storage; the fat in IR is generally stored in and around abdominal organs in both males and females. It is currently suspected that hormones produced in that fat are a precipitating cause of insulin resistance.
- Increased blood triglyceride levels.
- Increased blood pressure. Many people with hypertension are either diabetic or pre-diabetic and have elevated insulin levels due to insulin resistance. One of insulin's effects is to control arterial wall tension throughout the body.
- Increased pro-inflammatory cytokines associated with cardiovascular disease.
- Depression. Due to the deranged metabolism resulting from insulin resistance, psychological effects, including depression, are not uncommon.
- Increased hunger.

In addition to the above, Insulin resistance (IR) is considered the key mechanism unifying obesity, diabetes and heart disease (Maron DJ et al., 2000). In the course of months or years, IR is followed by the increase in β-cell insulin secretion and by several complications known as the insulin resistance syndrome, which is associated with dyslipidemia (abnormal amount of lipids in the blood), hypertension, hyperglycemia and cardiovascular disease (Vessby B. 2000). Thus, efforts are continuously underway to prevent obesity in the population. Although weight gain is ultimately the result of an overall positive energy balance, the environmental and genetic interplay that accounts for the dramatic rise in obesity is not fully understood.

Although the traditional weight loss approach advises a high carbohydrate low fat diet, a very low carbohydrate and high fat diet has been suggested to have greater effectiveness in weight loss and metabolic improvement. One potential problem associated with chronic ingestion of a low-carbohydrate diet is that it usually contains a high percentage of fat to compensate for carbohydrate-calorie reduction, and most in the form of saturated fat (Lovejoy JC. et al., 1998). High-saturated fat diet in humans is known to be associated with high risk of diabetes and cardiovascular disease.

In most people with T2DM, insulin resistance is generally present for many years before the diagnosis. Despite possible influences from genetic and perinatal factors, diet and physical activity are likely to have greater and often overriding influence in pathogenesis of the insulin resistance syndrome and T2DM (Reaven GM 1993). This review aims to critically analyse influence of dietary fat on the insulin resistance.

Risk Factors for Insulin Resistance

- Being overweight or obese.
- Leading a sedentary lifestyle.
- Being over age 40.
- Being in one of the following ethnic groups: Latino or Hispanic American, African American, Native American, Asian American, Pacific Islander.
- A history of glucose intolerance or gestational diabetes.
- Existing type 2, hypertension, or cardiovascular disease.
- Elevated triglycerides/low HDL-cholesterol.

The consequences of insulin resistance:

Globalization and global market have contributed to increased consumption of high-fat, energy-dense diets, particularly rich in saturated fatty acids (SFAs). Polyunsaturated fatty acids (PUFAs) regulate fuel partitioning within the cells by inducing their own oxidation through the reduction of lipogenic gene expression and the enhancement of the expression of those genes controlling lipid oxidation and thermogenesis (Perrotti N, et al., 2000). Moreover, PUFAs prevent insulin resistance by increasing membrane fluidity and GLUT4 transport. In contrast, SFAs are stored in non-adipocyte cells as triglycerides (TG) leading to cellular damage as a sequence of

their lipotoxicity.

There is good evidence that insulin resistance can contribute to cardiovascular disease also independently of classic risk factors (Kelishadi R et al., 2009). Several cross-sectional studies documented that insulin resistance, as assessed by various techniques, is related to coronary, carotid or peripheral vascular disease in both non diabetic and diabetic subjects, also independently of classic risk factors. In a wide angle perspective we might consider insulin resistance as a common denominator and probably a central causal disorder affecting several metabolic parameters like glucose, lipids, urate but also adversely affecting blood pressure, fibrinolysis and coagulation, oxidative stress, endothelial function and inflammation. Many literature data, indeed, support the conclusion that insulin is causally related to many non-traditional risk factors. Therefore, Insulin resistance seems to be a gateway to several physiological mental disturbance which ultimately impact on the arterial wall: it worsens the profile of risk because it increases glucose, lipids and blood pressure, it favors a prothrombotic state through abnormalities in the coagulation and the fibrinolysis, it deteriorates the endothelial function, it promotes monocyte transmigration, LDL oxidation, foam cell formation, inflammatory molecules synthesis and release, vascular smooth cell proliferation and migration and plaque (Kelishadi R et al., 2009).

Non-communicable diseases and fat consumption

Noncommunicable diseases (NCDs) are currently the leading global cause of death worldwide. In 2008 of the 57 million deaths that occurred globally, 36 million – almost two thirds – were due to NCDs, comprising mainly of cardiovascular diseases, cancers, diabetes and chronic lung diseases (WHO, 2011). The combined burden of these diseases is rapidly increasing in lower-income countries. It causes costs of personal medical care costs for diagnosis, procedures, and drugs and inpatient and outpatient care; non-medical costs, such as the costs of transportation for treatment and care; nonpersonal costs like those associated with information, education, communication and research; and income losses. About one fourth of the global NCD-related deaths occur before the age of 60 (Adeyi O., et al., 2007).

A large proportion of NCDs are preventable. They share modifiable behavioural risk factors such as tobacco use, unhealthy diet, lack of physical activity, and the harmful use of alcohol. These risk factors lead to overweight and obesity, raised blood pressure and raised cholesterol (WHO. 2009). If no action is taken, over the next three decades, the cost of NCD burden will amount to trillions of dollars of lost resources. Feasible and cost-effective interventions to reduce the burden and impact of NCDs exist, and sustained action to prevent risk factors and improve health care can avert millions of preventable premature deaths (Abegunde DO, et al., 2007).

> Premature death (before age 60 years) or living long term with an NCD or related disability has socioeconomic consequences and constitutes a double burden to sustainable social and economic development *(4)*. Reduced income and early retirement caused by NCDs can lead individuals and households into poverty. At the societal level, in addition to surging health care costs are increased demands for social care and welfare support as well as the burdens of the impact of absenteeism from school or work, decreased productivity and employee turnover *(5)*.

The exposures for NCDs:

- Fat intake
- Low fruit and vegetable intake
- Overweight and obesity
- Physical inactivity
- Raised blood glucose
- Raised Blood pressure
- Raised total cholesterol
- Salt/sodium intake
- Tobacco
- Alcohol

Greater consumption of energy rich foods containing high trans fat, saturated fat and w-6 fat and refined carbohydrates in conjunction with physical inactivity are known to enhance all these biomarkers which have adverse proinflammatory effects resulting into NCDs (Paoletti R. 2011). In almost all the countries of the world, NCDs are the major health issue for men and women, and are a serious issue for all health-care systems. NCDs also account for half of all global disability (Nabel EG, et al., 2012).

Non-communicable diseases, particularly cardiovascular disease (**CVD**) and type 2 diabetes have traditionally been considered diseases of the developed world, associated with affluence, over-consumption of energy-dense foods, and lack of physical activity. With an increase in economic prosperity, many populations appear to increase their consumption of animal products, and it is inviting to associate increased susceptibility to

NCD with increased intake of such foods (WH O., 2011).

However, it is important to recognize that such diseases occur throughout the global population (WHO, 2011). In fact, death from NCD in a range of countries, indicate that, on an age-standardized basis, death from CVD and diabetes is greatest in those countries where consumption of animal products is lowest (India and Kenya) compared with those with a high intake (UK and US). WHO, 2012, Comparing mortality data between countries should be undertaken with caution as mortality, as opposed to prevalence of the disease, may be severely influenced by differences in health care availability. However, such NCD are multi-factorial, and at worst, consumption of animal products is only one of a range of factors that influence the impact of such diseases on morbidity and mortality (Poortinga W, 2007).

In general, such studies are difficult to perform on free-living populations and are prohibitively expensive, due to the length of follow-up required to record sufficient disease incidence. However, substitution of SFA with unsaturated fatty acids has repeatedly been shown to reduce CVD morbidity and mortality. For example, a review by Hooper et al. (2011), which included 65,508 participants from 48 randomly controlled trials concluded that long-term (greater than two years) reduction of SFA intake was associated with a significantly (14%) reduced cardiovascular risk when it was substituted with unsaturated fatty acids rather than replaced with carbohydrates. Similar analyses appear to confirm the deleterious effect of trans fatty acids (Poortinga W, 2007). Many countries develop policies to reduce TFA derived from hydrogenated vegetable fats from our diets, animal products (specifically those derived from ruminant animals) have become a significant source of residual intakes.

The link between intake of animal products and risk of developing CVD has largely been attributed to the relatively large contribution such foods make to our intake of saturated fatty acids (**SFA**). Meat, meat products, milk, and dairy products contribute approximately half of the intake of SFA in the UK. While it is generally accepted that the quantity and composition of dietary fat can affect plasma lipoprotein cholesterol concentrations (Mensink et al., 2003), the impact of dietary fat on actual CVD morbidity and mortality has continued to be a topic of debate. The link between diet and development of disease can be explored in a number of ways (UN, 2012).

Vulnerable groups affected by NCDs

All age groups and all regions are affected by NCDs. NCDs are often associated with older age groups, but evidence shows that more than 9 million of all deaths attributed to noncommunicable diseases (NCDs) occur before the age of 60. Of these "premature" deaths, 90% occurred in low- and middle-income countries (Abegunde DO et al 2007). Children, adults and the elderly are all vulnerable to the risk factors that contribute to noncommunicable diseases, whether from unhealthy diets, physical inactivity, exposure to tobacco smoke or the effects of the harmful use of alcohol. Poverty is closely linked with NCDs. The rapid rise in NCDs is predicted to impede poverty reduction initiatives in low-income countries, particularly by forcing up household costs associated with health care. Vulnerable and socially disadvantaged people get sicker and die sooner than people of higher social positions, especially because they are at greater risk of being exposed to harmful products, such as tobacco or unhealthy food, and have limited access to health services (Abegunde DO et al 2007).

Fat intake and Blood glucose level

Blood glucose levels are affected differently depending on whether you eat foods containing carbohydrates, proteins, fats, or a combination of these three (American Diabetes Association, 2000). Carbohydrates will cause blood glucose to rise the most and the most quickly. Liquids that contain carbohydrates (like milk and juice) will cause blood glucose to rise faster than solids that contain carbohydrates (like bread). Because of the impact that they have on blood glucose levels, carbohydrates are the most important macronutrient for people with diabetes to monitor (Hanefeld M, et al., 1991).

The amount of food that we eat also impacts blood glucose levels. Eating more food, or bigger portions, will cause your blood glucose levels to rise more than eating smaller portions (Madigan C, et al., 2000). Since carbohydrates affect blood glucose levels the most, the amount of carbohydrate that we eat each day is very important in controlling our blood glucose levels.

The process of digestion results in the release of glucose (a simple sugar) into the bloodstream. Normally, the pancreas then produces sufficient amounts of the hormone insulin to transport the glucose into the body's cells, thus maintaining a healthy blood sugar level (Nettleton JA, and Katz R, 2005). However, in some people the cells develop a decreased sensitivity to insulin, usually referred to as insulin resistance. This results in what is called glucose intolerance (a decreased ability to remove sugar from the blood). The body attempts to compensate by producing higher and higher amounts of insulin (shown in blood tests by high insulin levels, or hyperinsulinemia), but with serious insulin resistance, this may be inadequate to maintain a normal blood sugar. (Nettleton JA and Katz R, 2005). When the blood sugar remains too high (called hyperglycemia), the person will be diagnosed with diabetes.

Fat intake and blood pressure

Blood pressure is the force of blood against artery walls. Blood pressure rises and falls during the day. But when it stays elevated over time, then it's called high blood pressure (Singh RB, et al., 2000). High blood pressure is dangerous because it makes the heart work too hard, and the high force of the blood flow can harm arteries and organs such as the heart, kidneys, brain, and eyes. High blood pressure often has no warning signs or symptoms. Once it occurs, it usually lasts a lifetime. If uncontrolled, it can lead to heart and kidney disease, stroke, and blindness. Blood pressures can be reduced with an eating plan that is low in saturated fat, cholesterol, and total fat and that emphasizes fruits, vegetables, and fat-free or low-fat milk and milk products (Reaven GM, et al., 2006). This eating plan—known as the Dietary Approaches to Stop Hypertension, DASH eating plan that includes whole grain products, fish, poultry, and nuts. The DASH eating plan follows heart healthy guidelines to limit saturated fat and cholesterol.

High blood pressure can be controlled if you take these steps:

- Maintain a healthy weight.
- Be moderately physically active on most days of the week.
- Follow a healthy eating plan, which includes foods lower in sodium.
- If you drink alcoholic beverages, do so in moderation.
- If you have high blood pressure and are prescribed medication, take it as directed.

Fat intake and diabetes

In the EU, 32 million people suffer from the diabetes, with about a further 6 million unaware that they are living with it. This figure is set to rise by 25 per cent to about 40 million by 2030. Worldwide, around 350 million people have diabetes according to the World Health Organization, and more than 900 million are expected to be diagnosed with, or as having high risk of developing, type 2 diabetes within the next two decades (Diabetes Control and Complications, 2003). Moreover, in Europe, type 2 diabetes is likely to reduce life expectancy by up to 10 years. The disease contributes to coronary heart disease, stroke, peripheral vascular disease and end-stage renal disease, making it the fifth leading cause of death worldwide. It is even worse for type 1 suffers whose lifespan can be cut by over 20 years.

High-fat diets are known to lead to a positive fat balance and consequently to adipose mass accumulation (Brown S, et al., 1996). Also, these diets do not seem to stimulate fat oxidation rate in the same way in obese and lean subjects. In addition, the type of dietary fat also seems to be a determinant in body fat increase. For example, diets rich in saturated lipids can increase body fat stores to a greater extent when compared to omega-6- and omega-3-rich diets (Diabetes Prevention Program: 1999). On the other hand, aerobic exercise training improves body composition, aerobic fitness, leptinemia, intramuscular lipid accumulation, and insulin sensitivity in obese individuals. However, few studies relate the possible interactions of different types of dietary fat and exercise. The objective of this review is to connect the interactions of dietary fat, different fatty acids and exercise to metabolic parameters normally impaired in obesity. Due to its direct association with various chronic diseases, including coronary heart disease, diabetes, colon carcinoma and gallstones, and because of its epidemic nature in developed societies, obesity is a very important public health problem (American Diabetes Association, 2002). Societies and individuals consume an extraordinary amount of resources in an attempt to prevent and treat obesity, usually with little success. The only reasonable treatment of overweight or obesity is the voluntary limitation of energy intake in the form of high-calorie foodstuffs associated with increased energy expenditure by physical exercise, but this is more easily said than done. Due to its high energy content, dietary fat is frequently a source of undesirable calories, and a lifelong adherence to a relatively low-fat, high-complex carbohydrate diet provides the best opportunity for prevention and treatment of obesity (reviewed in (Dabelea D, et al., 2001).

In front of the difficulty in voluntarily omitting fat from the diet, the food industry has, in past decades, developed the technology to substitute it, producing processed foodstuffs that are low in fat but preserve the flavor and texture of high-fat ones. Although each source of dietary fat is composed of hundreds of different complex triglycerides, every one of them tends to have a stereospecific structure (individual fatty acids are located in particular positions of the three available ones in the glycerol molecule) that might affect metabolic disposition in a variable manner, from intra alumenal hydrolysis to adipose tissue deposition. The transformation of naturally occurring triglycerides into molecules structured in such a way as to limit their intestinal absorption is another fertile field of alimentary research, with objectives similar to those of fat replacers (Tuomilehto J et al. 2001).

There also are natural products with the ability to interfere with triglyceride absorption, such as the chitosans, polyaminosaccharide molecules derived from crustacean and fungal chitins, already approved for use as food additives or supplements in several countries (Knowler WC, et al., 2002). Chitosans posses both cationic groups that can bind fatty acids and bile acids through ionic bonds, and hydrophobic domains that can trap neutral fats such as triglycerides and cholesterol, with the effect of promoting fecal fat excretion. There are

preliminary evidences that chitosans may potentiate the weight loss induced by a hypocaloric diet in obese subjects. All persons with diabetes and pre-diabetes need to attain and maintain optimal metabolic outcomes, including:

- Blood glucose levels in the normal range or as close to normal as is safely possible to prevent or reduce the risk for complications of diabetes
- Lipid and lipoprotein profile that reduces the risk for macro vascular disease
- Blood pressure levels that reduce the risk for vascular disease
- improve health through healthy food choices and physical activity
- address individual nutritional needs, taking into consideration personal and cultural preferences and lifestyle while respecting the individual's wishes and willingness to change

Fat intake and Obesity

Epidemiological, experimental, and clinical evidence shows that obesity and weight gain are associated with increased risk for cardiovascular diseases (CVDs) and events and type 2 diabetes, and with risk factors for these disorders, including hypertension, glucose intolerance, abnormal amount of lipids in the blood and insulin resistance, i.e resistance to insulin stimulated glucose uptake appears (Tremblay A, et al., 1998).

Weight fluctuates to some degree throughout one's life and is known to increase with age. Physical inactivity and high calorie intake from high fat are among the most important behavioural risk factors. Not only total calorie intake, but also the dietary pattern and specific nutrients may influence risk factors such as inflammation and fat distribution associated with insulin resistance and diabetes (Hubert HB, et al., 2003).

Weight loss may reduce these risk factors and could delay or prevent the onset of CVD, type 2 diabetes, and other diseases associated with increased weight. Beyond health problems, overweight and obesity impose high costs in health expenditure in countries. For individuals at risk for diabetes, to decrease the risk by encouraging physical activity and promoting food choices that facilitate moderate weight loss or at least prevent weight gain. Excess body fat is perhaps the most notable modifiable risk factor for the development of type 2 diabetes. It is estimated that the risk of type 2 diabetes attributable to obesity is as much as 75% (Singh RB, et al., 2007).

In the pharmacological field, there is the potential to reduce the intestinal disposition of dietary fat by interference with one or more of the metabolic processes implicated in fat digestion and absorption (Rodriguez LM, and Castellanos VH, 2000). Pancreatic lipase inhibition is the only therapeutic modality that has been tried in humans and has demonstrated a reasonable efficacy to promote weight loss by way of an induced fat malabsorption.

Investigators emphasize that the difference in the prevalence of obesity in some population groups is related to environmental factors, especially diet and reduction of physical activity. These aspects, interacting or not with genetic factors, could explain at least in part the excess body fat observed in large proportions worldwide(Foreyt JP, and Goodrick GK, 1993). An excess of body fat tissue may be related not only to energy intake and energy expenditure in humans, but also to the type of diet, especially high-fat diets (HFD), which may lead to various metabolic alterations such as hyperphagia in humans reduced lipolytic activity in fat tissue, reduction in leptin secretion and/or sensitivity, hypothalamic neuron apoptosis, impairment of mitochondrial metabolism insulin resistance, and obesity (Kendall A,et al., 1999).

The balance between fat consumption and oxidation rates is not so tightly regulated and depends on the type of fatty acids (Wing RR and Hill JO, 2000). On the other hand the balance of each nutrient seems to involve a rigorous control to adjust its intake to its oxidation. An increase in carbohydrate and/or protein consumption is accompanied by increased oxidation rates of both nutrients. Therefore, obesity is associated with high-fat diets (HFD), and studies have indicated a possible "anti-obesity" effect attributed to polyunsaturated fatty acids (PUFA. This may be due to their greater oxidative rates when compared to saturated fatty acids (SFA) (Purnell JQ, et al., 1999). Several investigators have shown that FA oxidation increases directly. Rats submitted to a high saturated fat (HSF) diet for 7 weeks (58% total caloric intake) developed greater adiposity when compared to a high-omega 3 diet. Surprisingly, the omega 3 group also showed a significant decrease in adiposity when compared to the control low-fat diet - 10% of total energy intake (Ditschuneit HH, et al., 1999). This indicates that the composition of fat in the diet is more important than the amount of fat. Unsaturated fatty acids seem to have the opposite effect through the following mechanisms: decreased energy intake and/or increased energy expenditure through the activation of mitochondrial uncoupling proteins, reduced lipid uptake by white adipocytes by suppression of lipoprotein lipase, increased lipid catabolism enhancing.

In general, to prevent and treat the chronic complications of diabetes the following are time tasted approaches; modify nutrient intake and lifestyle as appropriate for prevention and treatment of obesity, dyslipidemia(abnormal amount of lipids in the blood), cardiovascular disease, hypertension, and nephropathy (de Leiva A, 2004). For youth with type 1 diabetes, to provide adequate energy to ensure normal growth and development, and to integrate insulin regimens into usual eating and physical activity habits. For youth with type

2 diabetes, to facilitate changes in eating and physical activity habits that reduces insulin resistance and improve metabolic status. For pregnant or lactating women, to provide adequate energy and nutrients needed for optimal outcomes (Goldstein DJ. 1992), for older adults, to provide for the nutritional and psychosocial needs of an aging individual. Nutritional recommendations to monitor diabetes and obesity complications are presented in the next table.

Fat intake and cardio vascular disease (CVD)

Cardiovascular disease (CVD) is a leading cause of death in most countries. Reducing saturated fatty acid (SFA) intakes is still at the heart of dietary recommendations to reduce CVD, mainly because of its effect on blood cholesterol (Jakobsen M U et al., 2009). This view has recently been challenged. First, a review of epidemiological studies failed to conclude that SFAs are associated with an increased risk of CVD. Second, the validity of meta-analyses of clinical trials showing that CVD can be prevented by replacing SFAs with polyunsaturated fatty acids (PUFAs) has been questioned (Hu FB, et al., 2001). Third, it has been claimed that the effect of diet on a single biomarker (such as plasma cholesterol) is insufficient evidence to assess CVD risk. Fourth, the hypothetical protective effect of omega-6 PUFAs has been said to be considerably exaggerated because of the failure to draw a line between the trials that selectively increased omega-6 PUFAs and those that substantially increased omega-3 PUFAs - known to PUFAs to replace SFAs (Kris-Etherton PM, et al. 2001). Finally, clinical and epidemiological studies exploring the dietary fat issue failed to provide a clear biological understanding of the effect of the various dietary fats on the risk of CVD.

Fat intake and cancer

In animal studies, omega-6 PUFAs have a strong mammary tumor-enhancing effect. In order to exert their carcinogenic effects; they must first undergo an oxidative metabolization, mainly through the lipoxygenase and cyclooxygenase pathways (Cockbain AJ, et al., 2012). The main substrate of these oxidative pathways is arachidonic acid, which is mainly produced from dietary linoleic acid, the most common omega-6 PUFAs in Western foods and cooking fats. Several recent epidemiologic studies have found a positive association between dietary omega-6 PUFAs and breast cancer risk. Certain analyses took into account a genetic predisposition related to omega-6 metabolism. To determine whether 5-lipoxygenase (LOX)-mediated dietary omega-6 metabolism might influence breast cancer risk, investigators examined genetic variants of the LOX enzyme in combination with linoleic acid intakes (Heidemann C et al., 2008). They found that women with a genetic aberration affecting the LOX enzyme whose diet provided a high level of omega-6 (linoleic acid) had a significantly increased breast cancer risk. However, when women with the same high-risk genetic profile had a diet lower in linoleic acid, their genotype had no significant effect on their breast cancer risk.

This demonstration that a diet-gene interaction increases the risk of cancer may explain why some previous studies were inconsistent or conflicting. Other recent studies have shown interactions between heterocyclic amines and omega-6 PUFAs on the one hand (Popp-Snijders C et al., 2001), and between omega-3 and omega-6 on the other hand in determining the risk of invasive breast cancer. Other factors, such as the obesity status were shown to affect the association between dietary PUFAs and breast cancer risk. Finally, the food sources of omega-3 and omega-6 PUFAs, as well as their relative amounts in the diet of individuals, appear to be very important for breast cancer risk.

Thus, there are several recent and concordant studies that strongly suggest that dietary omega-6 PUFAs – the consumption of which is encouraged worldwide to decrease blood cholesterol - increase the risk of breast cancers (Popp-Snijders C et al., 2001). In the same line of reasoning, it is important to recall that the most frequently prescribed cholesterol-lowering drugs (including statins) increase the blood concentration of arachidonic acid, the main omega-6 PUFA in cell membranes. Also, studies have suggested that low cholesterol and/or cholesterol-lowering are associated with an increased risk of cancers. Thus, despite the fact that many confounders tend to obscure the effects of cholesterol-lowering drugs on the clinical occurrence of cancers, the association of high intake of omega-6 and statins both aimed at reducing blood cholesterol to prevent CVD - may add up to increase cancer risk, in particular breast cancer risk (Vessby B, et al., 2001).

Fat intake and cholesterol

Cholesterol, a waxy fat-like substance, is found in everybody cell. Cholesterol is necessary for nerve cell function, sex hormone production and the production of vitamin D from sunlight exposure of the skin. Humans can generally make all the cholesterol they need without food sources. Cholesterol is only found in animal (Shekelle RB, et al., 2001).

Tissue cholesterol occurs primarily as free (unesterified) cholesterol, but is also bound covalently to fatty acids as cholesteryl esters and to certain proteins. Free cholesterol is an integral component of cell membranes and serves as a precursor for steroid hormones such as estrogen, testosterone, and aldosterone, as well as bile acids (Rivellese AA, et al., 2003).

Cholesterol plays an important role in steroid hormone and bile acid biosynthesis and serves as an integral component of cell membranes. An increased concentration of cholesterol in the blood (*i.e.*, hypercholesterolemia) is widely recognized as a risk factor for coronary artery disease. Reducing plasma levels of total and low-density lipoprotein (LDL) cholesterol by diet, drugs or lifestyle modification is thus of principal importance in treating and preventing cardiovascular disease (Nicolosi RJ. 1997). In humans, blood cholesterol is derived from two sources. It is either absorbed from the diet by the intestine, or it is synthesized from precursor molecules in the liver. Given the capability of all tissues to synthesize sufficient amounts of cholesterol for their metabolic and structural needs, there is no evidence for a biological requirement for dietary cholesterol. Therefore, neither an adequate intake nor a Recommended Dietary Allowance is set for cholesterol (Asztalos B, 2000).

There is much evidence to indicate a positive linear trend between cholesterol intake and low density lipoprotein cholesterol concentration, and therefore increased risk of coronary heart disease (CHD). A Tolerable Upper Intake Level is not set for cholesterol because any incremental increase in cholesterol intake increases CHD risk. Because cholesterol is unavoidable in ordinary diets, eliminating cholesterol in the diet would require significant changes in patterns of dietary intake (Gotto AM Jr. 2002). Such significant adjustments may introduce undesirable effects (e.g., inadequate intakes of protein and certain micronutrients) and unknown and unquantifiable health risks. Nonetheless, it is possible to have a diet low in cholesterol while consuming a nutritionally adequate diet (Gotto AM Jr. 2002).

Although fatty acids are required for intestinal micelle formation, there is no strong evidence that fat content (or other dietary constituents such as fiber) has a significant effect on cholesterol absorption.

Health Impact of Urbanization and Immigration

Globally, people migrate from rural area to the cities in search of work, more income, and better life. Practically, due to this migration they walk less, ride more, watch more television, and eat a diet higher in fat and sugars when they live in the city (WHO, 2004). They have less access to home-grown and local vegetables and are more likely to eat fast foods, as well as fried foods and sugar drinks widely accessible from vendors on the street (Hu FB, 2008).

Thus, around the world obesity and diabetes are climbing to epidemic proportion, even in countries previously characterized by scarcity. Likewise, people from low-income and minority communities, as well as immigrants from the developing world, Explanations limited to lifestyle factors such as diet and exercise are inadequate to explain the universality of what can be called a *syndemic*, a complex and widespread phenomenon in population health produced by multiple reinforcing conditions. Underlying the problem are complex Factors such as —genetic, physiological, psychological, familial, social, economic, and political (Wing RR, et al., 2001).

These interacting factors include events occurring during fetal life, maternal physiology and life context, genotype, the nutritional transition, health impact of urbanization and immigration, social attributions and cultural perceptions of increased weight, and changes in food costs and availability resulting from globalization. Better appreciation of the complexity of causation underlying the worldwide epidemic of obesity and diabetes can refocus the work of clinicians and researchers to work at multiple levels to address prevention and treatment for these conditions among vulnerable populations (Singh RB, et al., 2007).

Mechanisms that used to improves NCDs

Role of exercise

Physical activity and dietary patterns are risk behaviours that travel across countries and are transferable from one population to another like an infectious disease, affecting disease patterns globally (WHO, 2009, 2012). Study conducted on n-3 and n-9 unsaturated fatty acids and exercise on 16 male subjects in an attempt to improve exercise performance,

insulin sensitivity and fat oxidation. They used an iso energetic diet enriched with fish and olive oils containing 34% fat (12% SFA, 12% MUFA, 5% PUFA) and an exercise protocol consisting of a 10-day gradual endurance training protocol (80% VO2 max). Training significantly increased time to exhaustion and improved insulin sensitivity (Pavlou KN, et al., 1999). Those effects, however, were of similar magnitude in the unsaturated fat group and in controls. However, fat oxidation tended to increase in the unsaturated fat group, but not in controls. Aerobic exercise has been used in weight loss programs for a long time. A study conducted in 1998 by Sial et al. evaluated the role of aerobic physical activity in fat oxidation in sedentary obese individuals submitted to a 16-week exercise program. Before starting the program, fat oxidation was measured in the first minute of exercise and the value obtained was reduced by 50% after 4 months of exercise. However, fat oxidation after the first 10 min of exercise was doubled in the exercised group compared to the sedentary individuals (Feskens EJ, et al., 2001).

Role of Dietary improvements

Strict avoidance of partially hydrogenated fats (often called "trans" fats) is crucial. These are chemically modified fats that are found in most margarines, vegetable shortening, and a large percentage of commercial baked goods (including most breads, crackers, cookies, pastries, etc.) and snack foods. Everyone who cares about insulin resistance and protecting their cardiovascular system needs to read labels (look for the words "partially hydrogenated" followed by any oil) and try to avoid these artery-damaging and cell-stiffening fats to the greatest extent possible. These partially hydrogenated fats have a rigid structure (Boylan S, et al., 2009). If you consume them, they will be incorporated into cell membranes (the outer layer of cells) and cause stiffening and dysfunction of both the membrane and the receptors within the cell membrane, including insulin receptors. The result can be increased insulin resistance. Another variety of bad fats are oxidized fats (any oil that has been heated to very high temperatures, particularly when it is re-used over and over, and, thus, all the fried foods made with such oils). These oxidized fats cause cell membrane damage in a different way, and can also harm cellular and receptor function.

Instead of these damaging fats, stick with the fats Mother Nature made, especially the monounsaturated fats like olive oil. If flavor is an issue to you in terms of oil choices, note that olive oil is available in the more full-flavored green varieties, as well as in lighter versions that have little or none of the usual olive taste. For baking or other cooking where you don't want the traditional olive oil flavor, the latter would be a good choice. There are other monounsaturated oils that would be good choices, including walnut oil and almond oil. However, some of the other monounsaturated oils are less desirable. For example, canola oil is heavily processed (using heat and chemicals) in ways that make it less appealing for those seeking the best oil for cardiovascular health (Storm H, et al., 1997).

Increasing the intake of fiber by eating more whole-grain foods (like brown rice instead of white, whole-grain breads and crackers and pastas instead of white, and so on), beans, and fruits and vegetables is important, as is using healthful fats (mostly monounsaturated fats like olive oil or walnut oil or almond oil, as well as walnuts, almonds, flaxseed, pumpkin seeds, and pecans) (McCall DO et al., 2009). Increasing your fiber intake by consuming more dark green vegetables (spinach, Swiss chard, broccoli, mustard greens, kale, and so on) rather than focusing on an increase in starchy grain foods will have a more positive influence on regulating blood sugars (McCall DO et al., 2009). One multi-center study found that both a higher intake of polyunsaturated fats (like corn oil and safflower oil and all the foods made with such polyunsaturated fats) and a lower intake of dietary fiber were strongly associated with insulin resistance. One dietary change that may be particularly important is to aim for foods lower on the "glycemic index" scale. The glycemic index ranks foods based on how they affect blood sugar levels by measuring how much blood sugar increases in the two or three hours after eating them. Since foods that are high in fat or protein have much less effect on blood sugar, the glycemic index concept is generally only used for foods high in carbohydrates. Foods that have a low glycemic index are broken down more slowly, giving the body a better chance to maintain normal blood sugar.

4. CONCLUSION

Whether fat plays a role or not, a chronic positive imbalance of calories over time is the primary cause of excess storage of body fat. The dietary intake of fat has link with an increased adverse effects such as Insulin resistance-hypertension, diabetes, obesity, cancer, cholesterol and cardio vascular diseases. According to metabolic studies, the type of fat, rather than total fat intake that affects common intermediate risk factors and risk of coronary heart disease is assoaciadet with high intakes of saturated fatty acids and cholesterol and obesity is associated with for a high intake of total fat. This is because Saturated fat or the good fats is a significant risk factor for cardiovascular diseases (CVD) including heart disease and stroke through raising blood total cholesterol and LDL cholesterol levels. On the other hand, the good fats or the unsaturated fats – monounsaturated fats, omega 3 polyunsaturated fats (PUFA) and omega 6 polyunsaturated fats. In the modern nutrition approach, saturated fatty acids, *trans* fatty acids, and dietary cholesterol have no known beneficial role in preventing chronic disease and are not required at any level in the diet. Both types of fat increase the risk of heart disease in some people by boosting the level of harmful, low-density cholesterol in the bloodstream; this occurs even with very small quantities in the diet. Since there is no intake level of saturated fatty acids, *trans* fatty acids, or dietary cholesterol at which there is no adverse effect, no UL (*upper* limit) is set for them; instead, the recommendation is to keep their intake *as low as possible* while consuming a nutritionally adequate diet, as many of the foods containing these fats also provide valuable nutrients. Replacing saturated fat and trans unsaturated fat with unhydrogenated unsaturated fats has clear beneficial effects on blood lipids and thus provides an alternative strategy for reducing the risk of coronary heart disease.

Insulin resistance is the experimental or clinical condition in which the hormone exerts a biological effect less than expected or Insulin resistance is the failure of insulin sensitivity. Trans fatty acids (TFA) could affect cell membrane functions, and may therefore influence peripheral insulin sensitivity and the risk of developing type 2 diabetes.

In addition, conjugated linoleic acid (CLA) impair insulin sensitivity and could promote metabolic disorders, long-term exposure of conjugated linoleic acid (CLA) also have unwanted health effects and dysfunction insulin sensitivity by increasing oxidative stress and inflammation. When people have insulin resistance, glucose builds up in the blood instead of being absorbed by the cells, leading to type 2 diabetes or prediabetes. Insulin resistance affects the body in many ways and the most common diagnosed are: Intestinal bloating – most intestinal gas is produced from carbohydrates in the diet, mostly those that humans cannot digest and absorb, Increased blood triglyceride levels, Sleepiness, especially after meals, Depression due to the deranged metabolism resulting from insulin resistance, Increased blood pressure. Many people with hypertension are either diabetic or pre-diabetic and have elevated insulin levels due to insulin resistance. One of insulin's effects is to control arterial wall tension throughout the body, and Weight gain, fat storage, difficulty losing weight – for most people, excess weight is from high fat storage; the fat in IR is generally stored in and around abdominal organs in both males and females. It is currently suspected that hormones produced in that fat are a precipitating cause of insulin resistance.

In the Europe, 32 million people suffer from the diabetes, with about a further 6 million unaware that they are living with it. This figure is set to rise by 25 per cent to about 40 million by 2030. Worldwide, around 350 million people have diabetes according to the World Health Organization, and more than 900 million are expected to be diagnosed with, or as having high risk of developing, type 2 diabetes within the next two decades. The only reasonable treatment of overweight or obesity is the voluntary limitation of energy intake in the form of high-calorie foodstuffs associated with increased energy expenditure by physical exercise. Moreover, Due to its high energy content, dietary fat is frequently a source of undesirable calories, and a lifelong adherence to a relatively low-fat, high-complex carbohydrate diet provides the best opportunity for prevention and treatment of obesity. Such types of weight loss may reduce these risk factors and could delay or prevent the onset of CVD, type 2 diabetes, and other diseases associated with increased weight. The food sources of omega-3 and omega-6 PUFAs, as well as their relative amounts in the diet of individuals, appear to be very important for breast cancer risk. Thus, there are several recent and concordant studies that strongly suggest that dietary omega-6 PUFAs – the consumption of which is encouraged worldwide to decrease blood cholesterol which increase the risk of breast cancers.

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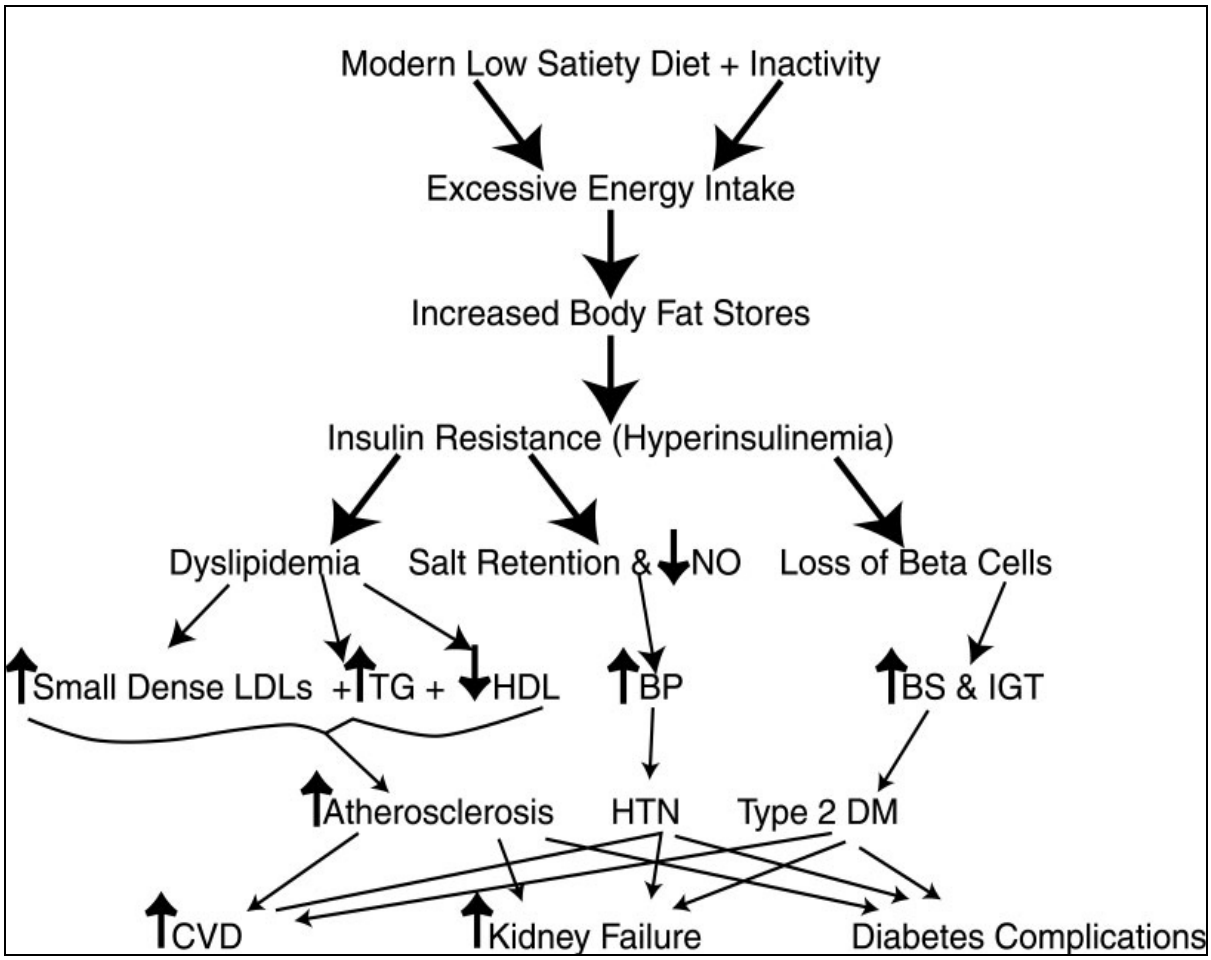
KEY FACTS IN THIS REVIEW

- Types of dietary fat intake are associated with doss effect of coronary heart diseases and other lifestyle diseases such as obesity, insulin resistance, diabetes, hypertension, cholesterol and inflammation.
- The burden and costs of NCD diseases in low-income and middle-income countries is the most remaining public problem
- Long term consumption of industrial and ruminant trans fatty acid and saturated fat diet are the most causes of coronary heart diseases and other NCDs
- Alteration of dietary fat intake of polyunsaturated and monounsaturated fat in place of saturated fat is a good f strategy to prevent weight gain and related health complications.
- Dietary supplementation of omega-3 polyunsaturated fatty acids improves insulin sensitivity in non-insulin-dependent diabetes.
- Globally physical inactivity and prevalence of NCDs are double burden of diseases during transition in a developing economy.
- WHO currently working on Action plan as the global strategy for the prevention and control of non communicable diseases focussing on Diet and Physical Activity as a public health priority.

Exercise and the consumption of higher amounts of unsaturated fats (MUFA and PUFA) are of primordial importance in the treatment of NCDs



Source: Ferrannini E, et al., 1999.
Figure 1: Insulin Resistance and its metabolic sequences

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