Chapter-II

REVIEW OF LITERATURE

This chapter provides the brief overview of metabolic syndrome, its concept, definition, epidemiology, its relation to cardiovascular disease and their prevention & management through non pharmacological interventions. Related research and non-research literature were extensively reviewed to gain insight into the research area. The review was significant in conceptualizing the research, the development of intervention, the selection of tools and for discussing the results. The studies referred in this review are of diverse methodology and research designs, both observational (Descriptive and analytical studies) and interventional (randomized and non-randomized control trial) studies. The relevant literatures were searched using electronic database such as MEDLINE, Pub Med, EMBASE, Science Direct, Cochrane Library, Bio Med Central, CINAHL, Cochrane Collaboration, Campbell collaboration, Ind med, google scholar etc. During search, Boolean operators, word truncation and wildcards were used to combine the different words. Manual search of references were also performed from the retrieved articles in addition grey literature that was available in books and other sources were also used. The literature search for this was limited to clinical trials in humans and English language.

The review is organized in the subsequent pages under the following heads:

2.1. Concept, Definition, Epidemiology and pathophysiology of Metabolic Syndrome.

2.2. Metabolic Syndrome and Cardiovascular diseases.
2.3. Diet and Metabolic syndrome: A complex relationship

2.4. Prevention and management of metabolic syndrome.

2.5. Role of dietary fibre in metabolic syndrome

2.6. Barley (as a source of dietary fibre) its history and nutritional composition.

2.7. Effect of barley consumption on various risk factors related to cardiovascular diseases.

2.1 Concept Definitions, Epidemiology and Pathophysiology of Metabolic Syndrome

2.1.1 Concept & Origin of Metabolic Syndrome

The concept of the metabolic syndrome has existed for at least 80 years (Cameron et al 2004). This constellation of metabolic disturbances, all risk factors for cardiovascular disease, was first described in the 1920s by Kylin, a Swedish physician, as the clustering of hypertension, hyperglycemia, and gout (Kylin E et al 1923). Later, in 1947, Vague drew attention to upper body adiposity (android or male-type obesity) as the obesity phenotype that was commonly associated with metabolic abnormalities associated with type 2 diabetes and cardiovascular disease (Vague et al 1947). Over the past two decades, a striking increase in the number of people with the metabolic syndrome worldwide has taken place. This increase is associated with the global epidemic of obesity and diabetes (Zimmet P et al 2001). With the elevated risk not only of diabetes but also of cardiovascular disease from the metabolic syndrome, there is urgent need for strategies to prevent the emerging global epidemic. The metabolic syndrome is a master of disguise since it can present in various ways according to the different components that constitute the syndrome.
The metabolic syndrome is also known as syndrome X, (Reaven et al 1988) the insulin resistance syndrome, (De Fronzo et al 1999) and the deadly quartet (Kaplan NM 1989). The constellation of metabolic abnormalities includes glucose intolerance (type 2 diabetes, impaired glucose tolerance, or impaired fasting glycaemia), insulin resistance, central obesity, dyslipidaemia, and hypertension, all well documented risk factors for cardiovascular disease. These conditions co-occur in an individual more often than might be expected by chance. When grouped together, they are associated with increased risk of cardiovascular disease (Isomaa B et al 2001, Lakka HM 2002).

2.1.2 Defining the Metabolic Syndrome

While the concept of the metabolic syndrome was accepted, and even while controversies have raged about its cause, in 1998 that there was an initiative to develop an internationally recognised definition. In an attempt to achieve some agreement on definition, and to provide a tool for clinicians and researchers, a WHO consultation proposed a set of criteria (Alberti KG 1998). Subsequently, the National Cholesterol Education Program’s Adult Treatment Panel III (NCEP: ATP III) and the European Group for the Study of Insulin Resistance have formulated definitions. These definitions agree on the essential components-glucose intolerance, obesity, hypertension, and dyslipidaemia—but do differ in the detail and criteria. The WHO definition and that of the European Group for the Study of Insulin Resistance agree in that they both include either glucose intolerance or insulin resistance as an essential component (Alberti KG 1998).
However, for the NCEP ATP III (2001) definition, this criterion is not included. Additionally, the cut-off points for criteria of each component of the cluster and the way of combining them to define the metabolic syndrome differ between the definitions of the WHO and European Group for the Study of Insulin resistance and the definition of the NCEP ATP III. The WHO proposal was designed as a first attempt to define the syndrome. The report clearly stated that the definition would be modified as new information became available about the components and their predictive power. In retrospect, it is apparent that the WHO definition was better suited as a research tool whereas the NCEP ATP III definition was more useful for clinical practice. Clinicians prefer simple tools with which to assess patients and improve their management, and it is generally agreed that the NCEP ATP-III definition is simpler for practice. It requires only a fasting assessment of blood glucose, whereas the WHO definition can require an oral glucose tolerance test.

Furthermore, because an accurate assessment of insulin resistance requires a more complicated test (eg, the hyperinsulinaemic euglycaemic clamp technique), its application in an epidemiological or clinical setting is impractical, although the Homeostasis Model Assessment (HOMA) model could be used as an alternative method (Matthews et al 1985). Yet another attempt at a definition came from the American Association of Endocrinology (Einhorn D et al, 2002) who have referred to the cluster as the insulin resistance syndrome. They suggest that four factors should be the “identifying abnormalities” of the syndrome. These are elevated triglycerides, reduced HDL cholesterol, elevated blood pressure, and elevated fasting and post load (75 g) glucose. Obesity is not a component of their definition. Given the mounting
evidence that central obesity is a major risk factor for type 2 diabetes and cardiovascular disease this omission is rather surprising (Lemieux et al 2000).

Since several definitions of the syndrome are in use, it is difficult to compare prevalence and impact between countries. Fortunately, there is now a chance for a more rational approach. In May, 2004 a group of experts was convened by the International Diabetes Federation (IDF) to attempt to establish a unified definition for the metabolic syndrome and to highlight areas where more research into the syndrome is needed. A similar process has been initiated jointly by the National Heart, Lung and Blood Institute (NHLBI) and the American Heart Association. Further consideration of the definition by the ATP III panel is expected to follow.

Ultimately, the combined efforts of the IDF and NHLBI-American Heart Association will result in a new definition(s) of the metabolic syndrome that will be suitable for use in clinical practice worldwide. A major issue for the IDF consensus consultation was the fact that criteria used for obesity in Asian and other populations could be different from those used in the west.

More recently, a working party with representation from WHO (Geneva), the International Society for the Study of Obesity, and the International Obesity Task Force re-emphasised the fact that obesity-associated risk is a continuum and that there are interethnic differences in the relations between various obesity indices and the risks of cardiovascular disease (Lancet 2004). They noted that in urban Asians, the BMI range of 23–24 has an equivalent risk of type 2 diabetes, hypertension, and dyslipidaemia as a BMI of 25–29·9 in white people. This finding will probably be taken into account when the new IDF definition is published.
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<th>Table 2.1.1 Different Criteria Proposed for Clinical Diagnosis of Metabolic Syndrome</th>
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**Review of Literature**
2.1.3 Epidemiology of Metabolic Syndrome

It is difficult to determine the prevalence of metabolic syndrome worldwide because of the differing criteria used to identify the condition (Ford 2004). Nearly 25% of the world population has metabolic syndrome (Mishra et al 2010). Findings from NHANES III suggest that 24% of American men and 23% of American women, or about 47 million people, have metabolic syndrome.

D. S. Prasad et al (2011) conducted a cross-sectional study in urban Eastern India. This study was planned to determine the prevalence of metabolic syndrome and to identify predictors for the same. The results of the study revealed that a very high prevalence rate of 43.2% (n=509) of metabolic syndrome was reported in this community. In this study, metabolic syndrome rates are significantly higher among females with 52.2% (n=307) than in males at 34.2% (n=202). Further age-standardized prevalence rates of metabolic syndrome were 33.5% overall, 24.9% in males and 42.3% in females. Older age, female gender, general obesity, inadequate fruit intake, hypercholesterolemia, and middle-to-high socioeconomic status significantly contributed to increased risk of metabolic syndrome.

JL Patel et al (2013) conducted a retrospective cross-sectional study to know clinical profile of metabolic syndrome and its individual components in type 2 diabetes mellitus (T2DM) subjects and their asymptomatic first-degree relatives and to identify risk factors of glucose intolerance. This study reported that among T2DM subjects 90% were hypertensive, 85% had low HDL, 30% males and 80% females had central obesity, 85% had metabolic syndrome. Among asymptomatic first-degree relatives of T2DM subjects: 48.7% had metabolic syndrome; hypertension, low HDL,
central obesity, impaired glucose tolerance, T2DM were present in 52.5%, 68.7%, 48.7%, 26.2%, 35%, respectively. This study also concluded that in T2DM and their asymptomatic first-degree relatives, hypertension and low HDL were commonest components of metabolic syndrome, females were more obese. Glucose intolerance was significantly associated with other components of metabolic syndrome. Impaired fasting glucose, increased hip circumference and low HDL levels were risk factors for glucose intolerance.

Deepa et al (2007) conducted a study to compare the prevalence of metabolic syndrome (MS) using the World Health Organisation (WHO), Adult Treatment Panel III (ATPIII) and International Diabetes Federation (IDF) criteria of MS in an urban south Indian population, and their ability to identify coronary artery disease (CAD). They observed that MS was identified in 546 subjects (23.2%) by WHO criteria, 430 subjects (18.3%) by ATPIII criteria and 607 subjects (25.8%) by IDF criteria. Only 224 of these subjects were identified by all the three criteria. There was an increased risk of probable CAD in MS subjects diagnosed by WHO criteria (odds ratio (OR) 3.86, 95% Confidence Interval (CI), 2.37–6.29, p < 0.001), compared to ATPIII criteria (OR 2.19, 95% CI 1.30–3.67, p < 0.05) and IDF criteria (OR 1.90, 95% CI 1.16–3.12, p < 0.05). The WHO criteria marked out a much higher population for CAD risk compared to ATPIII and IDF criteria in males, but not in females.

Jagmeet G et al (2016) has planned a cross-sectional study in Mumbai, India with the objective to determine the prevalence of metabolic syndrome in apparently healthy adult male population on their anthropometric, biochemical, and clinical health markers. The results of the study indicated that the prevalence of Metabolic
syndrome was 40% with 82% of the population surveyed being overweight and obese and 70.3% of the population with waist circumference of ≥90 cm. It was observed that 36% of the subjects were pre hypertensives and 23.4% had systolic and/or diastolic blood pressures ≥140/90 mmHg. Almost 40% of the subjects had dysglycemia with 34% of the subjects with high triglycerides, 26% with high total cholesterol, 64% with raised serum low density lipoprotein cholesterol, and almost 66% with low serum high density lipoprotein cholesterol levels. The mean age of the subjects was 46 years. A significant positive correlation was observed between anthropometric and biochemical markers.

2.1.4 Pathophysiology of Metabolic Syndrome

1. Insuline Resistance: Although the pathogenesis of this syndrome is not fully elucidated, there is strong evidence that insulin resistance and the ensuing hyperinsulinemia play a major role in several of its metabolic hallmarks (Reaven et al 1996). Insulin resistance may obviously play a major role in the development of impaired glucose tolerance or diabetes mellitus (De Fronzo 1988). Hyperinsulinemia may contribute to dyslipidemia by increasing hepatic free fatty acid reesterification and VLDL triglyceride secretion (Parks & Hellerstein 2000). Hyperinsulinemia may simultaneously stimulate de novo formation and re-esterification of fatty acids in the adipose tissue, thus increasing adipose tissue triglycerides, which constitute the major source of circulating fatty acids. Increased plasma free fatty acids are generally observed in dyslipidemic patients. Such plasma fatty acid levels have been widely
documented as an important factor contributing to the development of insulin resistance.

**Figure: 2.1.1 Pathogenesis of Metabolic Syndrome (Insulin Resistance)**

PAI1=plasminogen activator inhibitor 1. FFA=free fatty acids.

2. **Obesity and increased Waist Circumference:** The WHO and ATP III definitions of metabolic syndrome both include abdominal obesity, but it is a necessary
requirement in the IDF. Despite the importance of obesity in the model, we should remember that patients of normal weight can also be insulin resistant. Those are called metabolically obese, normal-weight individuals, typically having increased amount of visceral adipose tissue.

3. Dyslipidemia: In general, with increases in free fatty acid flux to the liver, increased production of very low-density lipoproteins (VLDL) occurs. Under physiological conditions, insulin inhibits the secretion of VLDL into the systemic circulation. In the setting of insulin resistance, increased flux of free fatty acids to the liver increases hepatic triglyceride synthesis. Thus, hypertriglyceridemia is an excellent reflection of the insulin resistant condition and is one of the important criteria for diagnosis of the metabolic syndrome. The other major lipoprotein disturbance in the metabolic syndrome is a reduction in HDL cholesterol.

4. Glucose intolerance: The defects of insulin action in glucose metabolism include failure to suppress gluconeogenesis in the liver, and to mediate glucose uptake in insulin sensitive tissues (i.e. muscle and adipose tissue). To compensate for defects in insulin action, insulin secretion must be increased to sustain euglycaemia. If this compensation fails, defects in insulin secretion predominate and hyperglycaemia occurs.

5. Hypertension: The relation between insulin resistance and hypertension is well established. Several different mechanisms are proposed. First, insulin is a vasodilator when given intravenously to people of normal weight, with secondary effects on sodium reabsorption in the kidney. Hyperinsulinaemia may result in increased
sympathetic nervous system (SNS) activity and contribute to the development of hypertension.

### 2.1.5 Causes and Risk Factors

Metabolic syndrome is a challenging condition due to its varying definitions, cut-offs, and limited data for certain populations especially the young adults and elderly. It is not a discrete entity but consists of multitude of risk factors. The risk varies in an individual depending on the aggregation of metabolic and non-metabolic risk factors (Grundy et al 2005). According to the AHA statement (2005), atherogenic dyslipidemia, elevated blood pressure, and elevated plasma glucose are the most familiar risk factors. Abdominal obesity (Carr DB et al 2004) insulin resistance, along with physical inactivity (Park YW 1988), aging (Ford et al 2002) hormonal imbalance (Apridonidze 2004) and atherogenic diet (a diet rich in saturated fat and cholesterol) can exacerbate the risk for developing metabolic syndrome, and consequently CVD. Relationship of Metabolic syndrome with sex, age, degree of obesity and socio-economic status has also been confirmed (Buckland et al 2008). A solitary factor cannot purport to development of the syndrome; therefore, interaction amongst various domains relating to it, holds colossal worth that needs to be evaluated with the help of longitudinal studies (Misra A, Vikram NK 2008).

There are numerous risk factors for metabolic syndrome such as age, body weight, physical inactivity, blood pressure, cigarette smoking, alcohol intake, and dyslipidemia (Do et al 2000).

- **Obesity:** Obesity is an independent risk factor for CVD. According to the World Health Organisation there are currently more than 1 billion overweight
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adults, 300 million of whom are obese (Mackay & Mensah 2004). The INTERHEART study, which enrolled almost 30000 men and women in 52 countries, reported that a waist/hip ratio greater than the cut-off of 0.83 for women and 0.9 for men resulted in a 3-fold increase in population attributable risk for myocardial infarction (Yusuf et al 2004).

- **Physical Inactivity:** The literature consistently indicates that a sedentary lifestyle increases the risk of developing several chronic diseases and conditions, while regular physical activity enhances overall health (Belahsen & Rguibi 2006). Physical activity includes any bodily movements produced by skeletal muscles that result in energy expenditure, covering daily walking activities at work and structured exercise training (Pettman et al 2008). To reduce the risk of CVD, people need at least 150 minute of moderate-intensity aerobic physical activity per week or at least 90 min of vigorous aerobic exercise per week (Franzini and Franz 2008). There is evidence to suggest that abdominal obesity is an independent risk factor for the development of diabetes, hypertension, hyperlipidemia and cardiovascular disease in individuals with BMI < 35 (Carr & Brunzell, 2004). Abdominal obesity is also associated with the metabolic syndrome. Measurement of waist circumference is the simplest clinical method to detect abdominal obesity.

- **Blood Pressure:** High blood pressure (BP) is one of the most prevalent cardiovascular risk factors and the single greatest contributor to cardiovascular disease worldwide (opez et al 2006). High BP commonly clusters with other cardiovascular risk factors, such as metabolic syndrome (Malik et al 2004).
Lifestyle factors that may lower blood pressure are sodium restriction, weight reduction or physical activity programs, and a reduction of excessive alcohol intake (Watkins 2003).

- **Smoking:** Cigarette smoking is an avoidable risk factor for CVD. An estimated 34.7% of all deaths resulting from cigarette smoking are related to CVD. Strong evidence links consumption of tobacco with increases in low-density lipoprotein cholesterol (LDL-C) oxidation, platelet aggregation, and endothelial impairment (Bloomer 2007). Smoking increases the risk for developing atherosclerosis, hypertension, and stroke, and it is the most important preventable cause of premature death (AHA 2008).

- **Alcohol Intake:** Moderate alcohol consumption, typically defined as up to 2 drinks per day for men and 1 drink per day for women, has been consistently associated with lower risk of coronary heart disease in observational studies. At least 2 meta-analyses have come to consistent conclusions about the magnitude of this association (Corrao et al 2000) and it is further supported by the established beneficial effects of moderate drinking on high density lipoprotein cholesterol and other cardiovascular risk factors (Watzl et al 2002). However, excess in alcohol consumption has detrimental health effects on blood pressure and triglyceride levels (Wakabayashi 2009).

2.2 Metabolic Syndrome and Cardiovascular Diseases

It is estimated that CVD will be the largest cause of disability and death in India, with about 2.6 million are predicted to die due to coronary heart disease (CHD),
which constitutes 54.1% of all CVD deaths in India by 2020 (NNMB Technical report 2017). In addition, CHD in Indians has been shown to occur prematurely, that is atleast a decade or two earlier as compared to those in developed countries. Hypertension is important risk factors for CVD alongside overweight and obesity and is a major public health problem in developing countries around the world. Metabolic Syndrome is a complex web of metabolic risk factors that are linked with a 2-fold risk of CVD and a 5-fold risk of diabetes (NCEP ATP-III 2002, Sattar N et al 2003). Individuals with MS have a 30%–40% likelihood of developing CVD within 20 years (NNMB Technical report 2017). There is strong evidence to suggest that metabolic syndrome doubles the risk for cardiovascular disease and raises the risk for diabetes and mortality fivefold over a 5- to 10-year period (Greenstone 2008).

Gami et al (2007) performed a meta-analysis of longitudinal studies that assessed any cardiovascular event outcomes or mortality in people with three or more coronary risk factors (regardless of whether this was termed the metabolic syndrome) compared with people without the diagnosis of metabolic syndrome. A total of 37 cohort studies from 1971 to 1997 including 172,573 individuals were included. This meta-analysis showed patients with metabolic syndrome had a relative risk (RR) of cardiovascular events and death of 1.78 (95% confidence interval [CI] 1.58, 2.00). The association was stronger in women (RR 2.63 vs. 1.98, p = 0.09), in studies enrolling lower risk individuals (RR 1.96 vs. 1.43, p = 0.04), and in studies using the WHO definition (RR 2.68 and 2.06 vs. 1.67 for National Cholesterol Education Program definition and 1.35 for other definitions; p = 0.005). The association remained after adjusting for traditional cardiovascular risk factors (RR 1.54, 95% CI, 1.32, 1.79). The population of patients in this trial was probably heterogeneous in that
the review included studies of overweight patients aged over 18 years. There was little
description of the patients in the individual studies.

Mottillo et al (2010) conducted a systematic review and meta-analysis of the
cardiovascular risk associated with the metabolic syndrome as defined by the 2001
National Cholesterol Education Program (NCEP) and 2004 revised National
Cholesterol Education Program (NCEP) definitions. Among 87 studies, which
included 951,083 patients (NCEP: 63 studies, 497,651 patients; NCEP: 33 studies,
453,432 patients). When both definitions were pooled, the metabolic syndrome was
associated with an increased risk of cardiovascular disease (CVD) (relative risk [RR]:
2.35; 95% confidence interval [CI]: 2.02 to 2.73), CVD mortality (RR: 2.40; 95% CI:
1.87 to 3.08), all-cause mortality (RR: 1.58; 95% CI: 1.39 to 1.78), myocardial
infarction (RR: 1.99; 95% CI: 1.61 to 2.46), and stroke (RR: 2.27; 95% CI: 1.80 to
2.85). Patients with the metabolic syndrome, but without diabetes, maintained a high
cardiovascular risk. The metabolic syndrome is associated with a 2-fold increase in
cardiovascular outcomes and a 1.5-fold increase in all-cause mortality. Studies are
needed to investigate whether or not the prognostic significance of the metabolic
syndrome exceeds the risk associated with the sum of its individual components.

Galassi et al (2006) conducted a meta-analysis and included a total of 21
prospective cohort studies. The RR of cardiovascular disease associated with the
metabolic syndrome in each study and overall is > 1.0 and all but five were
statistically significant. Individuals with the metabolic syndrome had an overall RR of
cardiovascular disease of 1.61 (95% CI, 1.42, 1.83) compared with individuals
without the metabolic syndrome, Prevalence of the metabolic syndrome varied from
8.8% to 92.3%. However, the highest prevalence of the metabolic syndrome (75.6–92.3%) was observed in the three studies in which all study participants had type 2 Diabetes.

Katzmarzyk et al (2006) compared the predictive ability of the NCEP-R and IDF metabolic syndrome criteria for mortality risk, and examined the effects of waist circumference on mortality within the context of these criteria. Their sample included 20,789 White, non-Hispanic men aged 20–83 from the Aerobics Center Longitudinal Study. “The study concluded that waist circumference is a valuable component of metabolic syndrome. However, the change in the IDF definition of metabolic syndrome of an elevated waist circumference warrants caution given that a large proportion of men with normal waist circumference have multiple risk factors and an increased risk of mortality”.

2.3 Diet and Metabolic Syndrome: a Complex Relationship

Though, the “Stone Age/Cave Man” diet of our hunter gatherer ancestors was far healthier from what we eat today; experience of alternate periods of abundance and scarcity during that time, led to an adaptive trait to survive the situation resulting in quick insulin release and lipid synthesis during starvation, to provide energy. However, with the advent of agriculture and urbanization, current diets have changed extremely from being healthy to those that are rich in saturated fat and cholesterol, which coupled with sedentary lifestyle leads to rapid glucose release and eventually positive energy balance. Therefore, it emerges that faulty diet is a major root-cause of the problem and is the preventive measure that needs to be taken to ameliorate metabolic syndrome or at least reduce its prevalence.
The type of diet consumed also has various implications on metabolic syndrome. A vegetarian diet consists of mostly food items that are from plants and plant products and does not have items resulting from death of animals. However, they may consume dairy products, milk and eggs (Teixeira et al 2007). The vegetarian dietary pattern can further be subdivided into lacto-ovo vegetarian (consuming milk, dairy products and eggs), lacto vegetarian (consuming milk and dairy products) and vegans (consuming no animal product). This vegetarian dietary pattern has shown to lower the risk of metabolic syndrome.

Rizzo et al (2011) studied various dietary patterns and looked at their association with Metabolic syndrome and its risk. Among subjects (n=773) of 30-94 years of age, dietary intake was assessed using a quantitative self-administered food frequency questionnaire and classified them into three categories – as vegetarian (consuming meat, poultry or fish <1 time in a month), semi-vegetarian (consuming fish at any frequency but other meats <1 time in a month), and non-vegetarians (consuming red meat or poultry ≥1 time/month). BMI was lowest in vegetarians and highest in non-vegetarians. Metabolic syndrome was classified according to the ATPIII criteria, with maximum number of components (i.e.5) for non-vegetarians. TG, FBG, BP, WC and BMI was significantly lower in vegetarians as compared to non-vegetarians (p<0.05). Metabolic syndrome was highest in non-vegetarians (39.7%), followed by semi-vegetarians (37.6%) and vegetarians (25.2%); with vegetarians having a lower odds for developing the syndrome (OR-0.44; p<0.001). Similar finding were observed in case of male and female vegans and lacto-ovo vegetarians; with significantly lower BMI, TG level, TC/HDL ratio among vegans as compared to lacto-ovo vegetarians (p<0.05). Vegans also demonstrated more
favorable serum lipid profile as compared to lacto-ovo vegetarians.

De Baise et al (2007) tried to compare levels of TG, LDL-cholesterol and HDL-cholesterol between vegetarians (lacto-ovo vegetarians [n=19], lacto vegetarians [n=17] and vegans [n=18]) and omnivores (n=22). For the same, blood samples were drawn from 76 adults and it was observed that there was a significant difference for these biochemical parameter values among all groups except for HDL-cholesterol. HDL/TC ratio was significantly higher in vegans (p<0.01), while TC and LDL-cholesterol values were highest for omnivores as compared to vegans (p<0.001). Cardiovascular risk [125] was also ascertained in a cohort of 201 individuals (35-64 years) in Brazil after classifying them as vegetarians (n=67) and omnivores (n=134). Levels of BP, FBG, TG, TC and LDL-cholesterol were significantly lower among vegetarians (p<0.001) except for HDL-cholesterol which showed no difference. Overall, vegetarians had a significantly lower cardiovascular risk (p<0.001) compared to omnivores whose diet consisted of animal protein and more fat.

Various studies related specifically to the Indian dietary pattern have shown risk for metabolic syndrome and eventually CVD (Daniel et al 2011). Whereas consumption of dairy foods, fried snacks and sweets consumption was associated positively with abdominal obesity; inverse relationship was observed with vegetables and pulses consumption for DM and hypertension. Vegetable and mustard oil consumption has also been proven beneficial (Rastogi et al 2004). The risk factors were more prevalent among the South Indians than North Indians (Kinra S 2010).
2.4 Prevention and Management of the Metabolic Syndrome

Given the central role played by insulin resistance in the pathogenesis of the syndrome, all strategies which improve insulin sensitivity are thought to be effective in preventing or improving the metabolic syndrome. Combining a heart healthy diet pattern and regular physical activity with even a small amount of weight loss (7-10%) in overweight person can reverse the metabolic syndrome. The primary approach is to reduce the major risk factors for cardiovascular disease: stop smoking and reduce LDL cholesterol, blood pressure and glucose levels to the recommended levels by:

- Weight loss to achieve a desirable body weight (BMI less than 25 kg/m2).
- Increased physical activity, with a goal of at least 30 minutes of moderate-intensity activity on most days of the week.
- Healthful eating habits that include reduced intake of salt, saturated fat e.g. butter, ghee, coconut, coconut oil and coconut milk, palm oil, fatty meats, Trans fat e.g. vanaspati (Dalda), partially hydrogenated oils used in snacks and dietary cholesterol e.g. egg yolks, meat, dairy.

2.4.1 Effect of Diet Counseling

Thompson et al (2007) revised Cochrane Review on the effects of dietary advice given by a dietitian compared with another health professional or the use of self-help resources in reducing blood cholesterol in adults. They studied 38 trials in which 17,871 healthy adults were randomly assigned to receive dietary advice or no dietary advice. The dietary improvements recommended to the people in the intervention groups centered largely on the reduction of salt and fat intake and an
increase in the intake of fruit, vegetables, and fiber (Thompson et al 2003). Participants receiving advice from dietitians experienced a greater reduction in blood cholesterol than those receiving advice only from doctors (-0.25 mmol/L [95% CI, -0.37, -0.12 mmol/L]). There was no statistically significant difference in change in blood cholesterol between dietitians and self-help resources (-0.10 mmol/L [95% CI, -0.22, 0.03 mmol/L]). No statistically significant differences were detected for secondary outcome measures between any of the comparisons with the exception of dietitian versus nurse for HDL-C, where the dietitian group showed a greater reduction (-0.06 mmol/L [95% CI, -0.11, -0.01]) and dietitian versus counselor for body weight, where the dietitian group showed a greater reduction (-5.80 kg [95% CI, -8.91, -2.69 kg]).

Dansinger et al (2007) in a meta-analysis of 46 trials of dietary counseling revealed a maximum net treatment effect of -1.9 (95% CI, -2.3 to -1.5) BMI units (approximately -6%) at 12 months. These studies were identified through a previously published systematic review: MEDLINE and the Cochrane Central Register of Controlled Trials from 1997 through July 2006. The authors included only weight loss studies with a dietary component. Different analyses suggested that calorie recommendations, frequency of support meetings, inclusion of exercise, and diabetes may be independent predictors of weight change. Studies were generally of moderate to poor methodological quality. They had high rates of missing data and failed to explain these losses.

Rees K et al (2007) included Forty-four trials with 52 intervention arms (comparisons) comparing dietary advice with no advice were included in the review;
18,175 participants or clusters were randomised. Twenty-nine of the 44 included trials were conducted in the USA. Dietary advice reduced total serum cholesterol by 0.15 mmol/L (95% CI 0.06 to 0.23) and LDL cholesterol by 0.16 mmol/L (95% CI 0.08 to 0.24) after 3 to 24 months. Mean HDL cholesterol levels and triglyceride levels were unchanged. Dietary advice reduced blood pressure by 2.61 mm Hg systolic (95% CI 1.31 to 3.91) and 1.45 mm Hg diastolic (95% CI 0.68 to 2.22) and 24-hour urinary sodium excretion by 40.9 mmol (95% CI 25.3 to 56.5) after 3 to 36 months but there was heterogeneity between trials for the latter outcome. Dietary fiber intake increased with advice by 6.5 g/day (95% CI 2.2 to 10.82), while total dietary fat as a percentage of total energy intake fell by 4.48% (95% CI 2.47 to 6.48) with dietary advice, and saturated fat intake fell by 2.39% (95% CI 1.4 to 3.37). Two trials analysed incident cardiovascular disease (CVD) events (TOHP I/II). Follow-up was 77% complete at 10 to 15 years after the end of the intervention period and estimates of event rates lacked precision but suggested that sodium restriction advice probably led to a reduction in cardiovascular events (combined fatal plus non-fatal events) plus revascularisation (TOHP I hazards ratio (HR) 0.59, 95% CI 0.33 to 1.08; TOHP II HR 0.81, 95% CI 0.59 to 1.12). Dietary advice appears to be effective in bringing about modest beneficial changes in diet and cardiovascular risk factors over approximately 12 months, but longer-term effects are not known.

2.4.2 Dietary Strategies (Current Therapeutic Options)

The National Cholesterol Education Program’s Adult Treatment Panel III (ATP III) (2001) has developed guidelines for reducing the risk of CVD which strongly urge lifestyle modification, including dietary changes, as the foundation and
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Initial intervention for persons at risk for CVD. An important component of the lifestyle modification is a ‘heart-healthy’ diet, which specifically includes a recommendation for consumption of at least 5–10 gm viscous soluble fiber (VSF) per day. In addition to reducing saturated fat and cholesterol intake, and increasing unsaturated fat intake, the importance of other dietary approaches, such as increasing the intake of viscous soluble fibers has become increasingly recognized.

2.5 Dietary Fibre and Metabolic Syndrome

Dietary fiber is a collective term for a variety of plant substances that are resistant to digestion by human gastrointestinal enzymes. Current evidence suggests that dietary fiber that is rich in whole and unrefined grains is protective and plays an important role in preventing or delaying the onset of chronic diseases and disorders such as coronary heart disease, (Leu S et al 2000, Truswell AS 2002) diabetes mellitus, (Meyer et al 2000) cancer, and colon dysfunction (Schweizer 1991).

The CARDIA (The Coronary Artery Risk Development in Young Adults) (2010) conducted a multicenter population-based cohort study carried out over 10 years, examined 2909 young individuals to determine the relationship between total dietary fiber intake and plasma insulin concentrations, weight and other cardiovascular diseases risk factors. After adjusting for BMI and multiple dietary (total energy, fat, alcohol intake) and potential non-dietary confounders (gender, education, physical activity, basal body weight, tobacco use), the study reported an inverse association between total fiber intake, plasma insulin concentrations and body weight gain suggesting that fiber may play an important role in the prevention of insulin resistance and obesity. Individuals consuming higher amounts of fiber have a lower weight gain.
compared to those consuming lower amounts, independently of the level of total fat consumed. However, it has yet to be established whether body weight gain can be modulated by fiber intake or whether such observations are a consequence of a reverse causation bias or other uncontrolled factors. Controlled trials analyzing the effects of fiber on body weight change are required.

Liu et al (2003) showed in the Nurses’ Health Study cohort that women in the highest quintile of dietary fiber intake had 49% lower risk of major weight gain than did women in the highest quintile. However, it is important to emphasize that these findings may reflect a healthy dietary pattern rather than fiber intake per se.

Chandalia et al (2000) also demonstrated that high fiber diets contributed to better metabolic control in thirteen Type 2 DM diabetic patients. In a cross-over study, patients were randomized to a diet containing a moderate amount of fiber (8 g of soluble fiber and 16 g insoluble fiber) or to a diet containing a high amount of fiber (25 g of soluble fiber and 25 g insoluble fiber). Plasma glucose concentrations were significantly lower for the high fiber diet than for the low-fiber diet.

**Dietary fiber are classified in to two types:**

- **Insoluble Dietary Fiber:** Cellulose, lignin, some pectins, and some hemicelluloses are insoluble fibers. Vegetables and cereal grains are especially rich in insoluble fiber, with the highest amounts in wheat and corn. Insoluble fiber is responsible for increased stool bulk and helps to regulate bowel movements.
- **Soluble Dietary Fiber**: The natural gel-forming fibers, such as β-glucans, gums, mucilages (e.g. psyllium), pectins, and some hemicelluloses are soluble. Foods rich in soluble fiber are dried beans, oats, barley, and some fruits and vegetables. The mean total daily fiber intake amongst adults in most industrialized countries is well below 25 g, the minimal amount recommended by various health organizations. Of total dietary fiber intake, approximately 20% is soluble and 80% is insoluble.

![Figure 2.5.1](image)

**Figure: 2.5.1** Potential effects of DF consumption. Colonic fermentation with the production of SCFA can be observed with most types of DF to some extent, but it tends to be more pronounced with soluble DF in naturally available foods.
2.5.1 Soluble Fiber (β-glucans)

β-Glucan is a viscous soluble fiber found in cereals, in particular oats and barley, as well as in yeast, bacteria, algae, and mushrooms. β-glucans are non-starch polysaccharides composed of glucose molecules in long linear glucose polymers with mixed β-(1→4) and β-(1→3) links with an approximate distribution of 70% to 30%.

![Figure: 2.5.2 Structure of β-glucan](image)

This specific chemical structure is responsible for physical properties, such as viscosity and solubility, as well as the potential to influence cholesterol metabolism.


2.5.2 How β-Glucans Works

Several of the principal benefits of soluble fiber in metabolic syndrome patients are indisputably due to its effect on carbohydrate absorption. Viscous fibers such as psyllium, β-glucans, and pectin may form a gel in the small intestine, which
acts to delay nutrient absorption, slowing the delivery of glucose into the bloodstream and reducing the need for insulin. These fibers’ ability to lower postprandial glycemia and insulinemia, as well as cholesterol, has been established in numerous studies, but long-term effects are less well known. Bacteria ferment beta-glucans in the intestinal tract, producing short-chain fatty acids. These may stimulate insulin release from the pancreas and alter glycogen breakdown by the liver and therefore play a role in glucose metabolism and protect against insulin resistance.

2.5.3 Source of β-glucan

To our knowledge, barley contains huge amount of dietary fiber (soluble (β-glucan) and insoluble) (Wursch P et al 1997) is an affluent source of vitamins & mineral especially B vitamins and trace minerals (Slavin J et al 1997).

2.6 Barley (As a Source of Dietary Fiber) its History and Nutritional Composition.

Barley is primarily a cereal grain popularly known as (jau) in India. It is the fourth most important cereal crop after rice, wheat and maize and it is actually one of the oldest consumed grains in the world. It was a staple grain for peasants during medieval times for centuries and today is still included in the diet of many European, African, and Middle Eastern nations that have been eating barley for thousands of years. In a 2007 ranking of cereal crops grown around the world, barley was listed as the fourth largest produced grain worldwide, with about 136 million tons of barley produced every year. Barley is also called the “King of Cereals” for some impressive health benefits.
Barley is widely used for food and fodder. Major uses of barley are in the beer industry, food processing and feed manufacturing industries. With the rising demand for beer, the demand for barley is also picking up. Also, more than 90% of the world malt production comes from barley. Globally, some of the major producers of barley are the European Union, Russia, Ukraine, Canada, Australia and USA, forming about 75% of the world production.

In India, the production has been quite stable with Rajasthan and Uttar Pradesh forming the largest producers of it. Saudi Arabia is the largest importer of barley in the world. In terms of derivatives trading, it's the second mostly traded coarse cereal after corn in the global market.

2.6.1 Nutritional Composition of Barley

According to the FDA, barley's soluble fiber reduces the risk of coronary heart disease and can lower cholesterol. But that's not all that this amazing food does! Barley also contains insoluble fiber, which reduces the risk of Type 2 diabetes and colon cancer and a range of important vitamins and minerals: fiber, selenium, B vitamins, copper, chromium, phosphorus, magnesium, niacin, and more. And when compared to many other grains, even other ancient whole-grains, barley is lower in fat and calories, but higher in dietary fiber and certain trace minerals. For example a one-cup serving of cooked barley has less calories, but more fiber, than an equal serving of quinoa, brown rice, amaranth, sorghum, millet or wild rice.
<table>
<thead>
<tr>
<th>Nutritional value per 100 g (3.5 oz)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>1,473 kJ (352 kcal)</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>77.7 g</td>
</tr>
<tr>
<td>Sugars</td>
<td>0.8 g</td>
</tr>
<tr>
<td>Dietary fiber</td>
<td>15.6 g</td>
</tr>
<tr>
<td>Fat</td>
<td>1.2 g</td>
</tr>
<tr>
<td>Protein</td>
<td>9.9 g</td>
</tr>
<tr>
<td><strong>Vitamins</strong></td>
<td></td>
</tr>
<tr>
<td>Vitamin (A)</td>
<td>13 μg</td>
</tr>
<tr>
<td>Thiamine (B1)</td>
<td>0.191 mg</td>
</tr>
<tr>
<td>Riboflavin (B2)</td>
<td>0.114 mg</td>
</tr>
<tr>
<td>Niacin (B3)</td>
<td>4.604 mg</td>
</tr>
<tr>
<td>Pantothenic acid (B5)</td>
<td>0.282 mg</td>
</tr>
<tr>
<td>Vitamin B6</td>
<td>0.26 mg</td>
</tr>
<tr>
<td>Folate (B9)</td>
<td>23 μg</td>
</tr>
<tr>
<td><strong>Minerals</strong></td>
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<tr>
<td>Calcium</td>
<td>29 mg</td>
</tr>
<tr>
<td>Iron</td>
<td>2.5 mg</td>
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<tr>
<td>Magnesium</td>
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<tr>
<td>Manganese</td>
<td>1,322 mg</td>
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<tr>
<td>Phosphorus</td>
<td>221 mg</td>
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<tr>
<td>Potassium</td>
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<tr>
<td>Sodium</td>
<td>9 mg</td>
</tr>
<tr>
<td>Zinc</td>
<td>2.13 mg</td>
</tr>
<tr>
<td><strong>Other constituents</strong></td>
<td></td>
</tr>
<tr>
<td>Water</td>
<td>10 g</td>
</tr>
</tbody>
</table>

USDA Nutrition Database 2010

- Units: μg = micrograms  mg = milligrams  IU = International units
2.6.2 Health Benefits of Barley

Barley and other whole grain foods have rapidly been gaining popularity over the past few years due to the various health benefits they can offer. Whole grains are important sources of dietary fiber, vitamins, and minerals that are not found in refined or "enriched" grains. Refining grains removes the bran, germ, and most of their fiber and nutrients. Choosing whole grains over their processed counterparts can help reduce the risk of obesity, diabetes, heart disease, cancer, and other chronic health problems.

Although barley may not be as popular as other whole grains like oats, wheat, or even grain-of-the-moment quinoa, barley has some impressive health benefits. A very high fiber content, vitamins and minerals, antioxidants, heart health and diabetes protection are just some of the barley nutrition benefits that make it one of the best whole grain choices. People following a high-barley diet experienced decreases in cholesterol and increased stool volume in a study published in "Nutrition" in 2003. Barley contains the same type of soluble fiber as oats, called β-glucan, which can help fill you up so you eat less and improve your cholesterol levels, according to an article published in "Comprehensive Reviews in Food Science and Food Safety" in July 2012. Eating foods containing 3 to 6 grams of soluble fiber per day is recommended to experience these benefits.

Barley contains both type of fiber soluble and insoluble fiber. Insoluble fiber makes up the cell walls of plants, and soluble fiber is found inside the cells. Soluble fiber forms a gel with water and slows the passage of food from the stomach, and insoluble fiber softens the stool and makes it more bulky, helping it move through
your digestive tract. Of the 6 grams of fiber in barley, 1.5 grams are soluble and 4.5 grams are insoluble.

Recently, the **US Food and Drug Administration (FDA), (2001)** allows the health claim statement that, depending on the beta-glucan content, consumption of soluble fiber from barley in a diet may reduce the risk of CVD. The high viscosity of -glucan may be particularly effective at reducing postprandial glycemia and several trials using oat or barley products reported significant reductions in glycemic response (Holm J et al 1992, Wood PJ et al 1994, Hallfrisch J et al 1995). Among participants in the National Health and Nutrition Examination Survey (NHANES) I Follow-up Study, each 5 g/d increase in viscous soluble fiber intake diminished the risk of CVD by 6% and coronary heart disease by 8%. The magnitude of the cholesterol-lowering effect in the relevant studies was variable. When only the higher-quality studies using barley grain products (no extracts) were taken into account (Anonymous 2005, Behall 2004a, Behall 2004b, Rondanelli 2011, Shimizu 2008, Sundberg 2008) the reduction in total cholesterol levels ranged from -0.06 to -0.50 mmol/L (-1.1% to -7.5%) while the reduction in LDL-cholesterol levels ranged from 0 to -0.32 mmol/L (0% to -8.5%).

**2.7 Effect of barley consumption on various risk factors related to cardiovascular diseases**

Jue Li et al (2003) conducted a study to observe the effects of high barley (high-fiber diet) intake on glucose tolerance, lipid metabolism, and bowel function in healthy women. Their study reported that the barley intake significantly lowered plasma total and low-density lipoprotein cholesterol concentrations and reduced
plasma triacylglycerol concentration. Barley intake also increased stool volume. There was no significant difference in glucose tolerance between diet regimens. This study demonstrated that barley intake has beneficial effects on lipid metabolism and bowel function and suggests that the intake of a high-fiber food, i.e., barley, should be recommended to prevent chronic diseases.

Another study published by Geraldine et al (2003) to investigated whether a β-glucan form of barley can favorably modify cholesterol and other markers of CVD and diabetes risk. They reported that there was no significant change in total (0.08 mmol/L, 1.3%), LDL (0.15 mmol/L, 3.8%), or HDL (0 mmol/L) cholesterol or in triacylglycerol (0.18 mmol/L), fasting glucose (0.05 mmol/L), or postprandial glucose when analyzed between treatments (P > 0.05; ANOVA). So, the study concluded that the effect of β-glucan–enriched barley on lipid profile was highly variable between subjects, and there was no evidence of a clinically significant improvement in CVD risk across this group of mildly hyperlipidemic men.

Esmailzadeh et al (2005) conducted a cross-sectional study in Tehran to evaluate the relationship between whole-grain intakes, metabolic syndrome and metabolic risk factors in Tehranian adults. The results of the study indicated that the both men and women reported higher intakes of refined grain than of whole grains. Compared with subjects in the lower quartile category, those in the upper category of whole-grain intake had lower prevalence of metabolic risk factors. Conversely, those in the higher category of refined-grain intake had higher prevalence of metabolic risk factors, except for diabetes. After controlling for confounders, a significantly decreasing trend was observed for the risk of having hypertriglyceridemia (odds ratios
among quartiles: 1.00, 0.89, 0.74, 0.61, respectively), hypertension (1.00, 0.99, 0.93, 0.84) and metabolic syndrome (1.00, 0.84, 0.76, 0.68). Higher consumption of refined grains was associated with higher odds of having hypercholesterolemia (1.00, 1.07, 1.19, 1.23), hypertriglyceridemia (1.00, 1.17, 1.49, 2.01), hypertension (1.00, 1.22, 1.48, 1.69) and metabolic syndrome (1.00, 1.68, 1.92, 2.25). Whole-grain intake is inversely and refined-grain intake is positively associated with the risk of having metabolic syndrome. Recommendations to increase whole-grain intake may reduce this risk.

Kristen et al (2008) planned a study to investigate the physiological effects of two concentrated barley β-glucans on cardiovascular disease (CVD) endpoints and body weight in human subjects. Their study reported that the only difference between treatments in lipid outcomes at week 6 was a reduction of the cholesterol/HDL ratio in the low-MW group and a small increase in the high-MW group. No changes were found in blood pressure, glucose, insulin, and gastrointestinal symptoms. Body weight decreased from baseline to 6 weeks in the high-MW group while body weight increased in the low-MW group. Levels of hunger decreased slightly in the low-MW group and decreased significantly in the high-MW group (P=0.02). The study also concluded supplementation with isolated barley β-glucans of different molecular weights had small effects on cardiovascular disease markers. Molecular weight of the barley fiber did alter effects on body weight with the high-MW fiber significantly decreasing body weight.

J. Tovar (2014) conducted an interventional study to examine the effects of a diet enriched with whole-grain kernel-based barley, brown beans and chickpeas on
cardio-metabolic risk-associated parameters in a healthy cohort of mature overweight women. A total of forty-six overweight women (50–72 years, BMI 25–33 kg/m² and normal fasting glycaemia) participated in a randomised cross-over intervention comparing a diet rich in kernel-based barley products, brown beans and chickpeas (D1, diet 1 (functional diet)) with a control diet (D2, diet 2 (control diet)) of similar macronutrient composition but lacking legumes and barley. D1 included 86 g (as eaten)/d brown beans, 82 g/d chickpeas, 58 g/d whole-grain barley kernels and 216 g/d barley kernel bread. Both diets followed the Nordic Nutrition Recommendations, providing similar amounts of dietary fiber (D1: 46·9 g/d; D2: 43·5 g/d), with wheat-based products as the main fiber supplier in D2. Each diet was consumed for 4 weeks under-weight-maintenance conditions. Both diets decreased serum total cholesterol, LDL-cholesterol and HDL-cholesterol levels, but D1 had a greater effect on total cholesterol and LDL-cholesterol levels (P<0·001 and P<0·05, respectively). D1 also reduced apoB (P<0·001) and g-glutamyl transferase (P<0·05) levels, diastolic blood pressure (P<0·05) and the Framingham cardiovascular risk estimate (P<0·05). D1 increased colonic fermentative activity, as judged from the higher (P<0·001) breath hydrogen levels recorded. In conclusion, a specific barley/legume diet improves cardiometabolic risk-associated biomarkers in a healthy cohort, showing potential preventive value beyond that of a nutritionally well-designed regimen.

Ripple et al (2009) conducted a systematic review study to determine the association between consumption of barley and changes in plasma lipids in healthy and hypercholesterolemic men and women. This study found 8 trials (n = 391 patients) of 4 to 12 weeks’ duration evaluating the lipid-reducing effects of barley. The use of barley significantly lowered total cholesterol (weighted mean difference
Results of the study concluded that the Barley-derived β-glucan appears to beneficially affect total cholesterol, LDL-cholesterol, and triglycerides, but not HDL-cholesterol.

Vasanthan et al (2002) conducted a study in Canada to investigate the effect of extrusion cooking of barley flour (waxy and regular starch types), under different temperature/moisture combinations, on the TDF, IDF, SDF, β-glucan and resistant starch contents of barley flour. Furthermore, an attempt will be made to rationalize the changes in various dietary fibre fractions during extrusion cooking. The study reported that Barley grains, Phoenix and CDC-Candle, were extruded in a twin-screw extruder at 90–140°C and 20–50% moisture level. Effects of extrusion conditions on total (TDF), soluble (SDF), and insoluble dietary fibre (IDF) were determined. The content of SDF and TDF increased upon extrusion cooking of both types of barley flours. The changes in IDF content were found to be variety-dependent. Only a minor decrease in IDF content of CDC-Candle barley was found, but an increase in IDF content of Phoenix was observed at all extrusion temperatures. The increase in SDF, in both barleys, could be due to the transformation of some IDF into SDF during extrusion and the formation of additional SDF by trans glycosidation. The increase in IDF in Phoenix flour could be due to the formation of retrograded amylose [resistant starch (RS3)] during extrusion cooking and subsequent cooling.
M. Oscarsson et al (1996) conducted an experimental study to examine the range of variation in chemical composition of barleys represented by single samples of a number of genotypes, selected on the basis of their different characteristics—covered and naked types with different starch composition, protein content and β-glucan content in wort or grain. The chemical compositions of the samples were analysed, focusing particularly on dietary fibre components. Covered and naked barley genotypes differed in their average contents of non-starch polysaccharides, Klason lignin and ash. High amyllose types had higher total β-glucan values (6.3% and 7.9%) than waxy types (5.4%–5.8%). The content of extractable β-glucan was only slightly correlated with total β-glucan content. On average, 44% of the xylose residues in water extractable arabinoxylans were substituted with arabinose residues. The presence of considerable amounts of xylopyranose substituted only at O-2 was detected in water extractable arabinoxylans.

Bourdon et al (1999) conducted an interventional study to evaluate the postprandial glucose, insulin, lipid, and cholecystokinin responses in healthy men to complete test meals containing β-glucans from barley. This study done with design: One of the meals was high in fiber (15.7 g) and the other meal was low in fiber (5.0 g). The low-fiber meal contained pasta made with wheat flour. The high-fiber meals contained pasta prepared by replacing 40% of the wheat with 2 types of barley flour: barley naturally high in β-glucan and the other flour enriched in β-glucan during processing. The results of the study indicated Plasma glucose and insulin concentrations increased significantly after all meals but the insulin response was more blunted after the barley-containing meals. The test meals were low in fat (25% of energy) but elicited an increase in plasma triacylglycerol and cholecystokinin.
Cholecystokinin remained elevated for a longer time after the barley-containing meals. After the low-fiber meal, plasma cholesterol concentrations did not change significantly; however, 4 h after the barley-containing meals, the cholesterol concentration dropped below the fasting concentration and was significantly lower than that after the low fiber meal.

K Minehira and L Tappy (2002) conducted a study entitled: “Dietary and lifestyle interventions in the management of the metabolic syndrome: present status and future perspective” to review the mechanisms underlying the metabolic syndrome, or syndrome X, in humans, and to delineate dietary and environmental strategies for its prevention. They evaluated that Hyperinsulinemia and insulin resistance play a key role in the development of the metabolic syndrome. Strategies aimed at reducing insulin resistance may be effective in improving the metabolic syndrome. They include low saturated fat intake, consumption of low-glycemic-index foods, physical exercise and prevention of obesity. Results suggested for future research, in particular the genetic basis of the metabolic syndrome and the interorgan interactions responsible for insulin resistance, is needed to improve therapeutic strategies for the metabolic syndrome. This study also concluded that Carbohydrate was more slowly absorbed from the 2 high-fiber meals. Consumption of the barley-containing meals appeared to stimulate reverse cholesterol transport, which may contribute to the cholesterol-lowering ability of barley.

Giacco et al (2014) performed an interventional study in Italy. This study was designed to evaluate the effects of a 12-week intervention comparing a whole-grain based diet to a refined cereal-based diet on postprandial glucose, insulin and lipid
metabolism in individuals with metabolic syndrome, and no weight loss. In this study 61 men and women age range 40 to 65 years, with the metabolic syndrome were recruited to participate in this study using a parallel group design. After a 4-week run-in period, participants were randomly assigned to a 12-week diet based on whole-grain products (whole-grain group) or refined cereal products (control group). Blood samples were taken at the beginning and end of the intervention, both fasting and 3 h after a lunch, to measure biochemical parameters. Generalized linear model (GLM) was used for between-group comparisons. Overall, 26 participants in the control group and 28 in the whole-grain group completed the dietary intervention. Drop-outs (five in the control and two in the whole-grain group) did not affect randomization. After 12 weeks, postprandial insulin and triglyceride responses (evaluated as average change 2 and 3 h after the meal, respectively) decreased by 29% and 43%, respectively, in the whole-grain group compared to the run-in period. Postprandial insulin and triglyceride responses were significantly lower at the end of the intervention in the whole-grain group compared to the control group (p=0.04 and p=0.05; respectively) whereas there was no change in postprandial response of glucose and other parameters evaluated.

Another study done by Rajesh et al (2015) to evaluate the effect of barley flour and Oat on body weight and associated metabolic disorder in over weight human volunteers. The study participants comprised of 48 healthy, over weight (defined as BMI>23/m²) human volunteers divided in two groups, of age group 20-60 years, in a dosage of 150 g, in a form of chapatti once in a day for two months. Anthropometrics, hematological and subjective parameters were carried out on in the beginning and also at the end of the trial. The single blind trial was conducted for 2 months. Significant
Review of Literature

Improvement in body weight, BMI, BMR, visceral fat, body fat, lipid profile, skin fold thickness was observed in both barley flour group and oat flour group, but barley flour group is found to be more effective than oat flour group. Barley flour in the form of chapatti to overweight human volunteers shown favorable impact on body weight and a variety of parameter characteristic of the metabolic syndrome.

Shimizu et al (2008) conducted a study in Japan with the aim to investigate whether the consumption of a diet in which high-β-glucan barley replaced rice would reduce the visceral fat area as well as the serum low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) in hypercholesterolemia Japanese men. A randomized, double-blinded, placebo-controlled intervention study was conducted in 44 men with a body mass index (BMI) >22 kg/m2. The subjects were randomly assigned to groups consuming either rice (placebo group) or a mixture of rice and pearl barley with a high β-glucan content (test group, 7.0 g β-glucan per day) for 12 weeks. Blood samples were taken, and CT scan obtained before the trial and every four weeks during the trial. The pearl barley intake significantly reduced serum concentrations of LDL-C (P=0.041) and TC (P=0.037) during the trial. Significant differences between the test and placebo groups were found for the visceral fat (P=0.039), BMI (P=0.015), and waist circumference (P=0.011) at the end point. The consumption of pearl barley with a high β-glucan content reduces not only LDL-C but also visceral fat area.

K M Behall et al (2004) conducted a study with the objective to examine the effects on CVD risk factors of the consumption of various amounts of β-glucan from barley, a grain not frequently consumed by Americans, in a controlled whole-grain
diet in mildly hypercholesterolemia men and women. They reported that total cholesterol was significantly lower when the diet contained 3 or 6 g β-glucan/d from barley than when it contained no β-glucan; the greatest change occurred in the men and postmenopausal women. HDL and triacylglycerol concentrations did not differ with the 3 amounts of dietary β-glucan. Large LDL and small VLDL fractions and mean LDL particle size significantly decreased when whole grains were incorporated into the 3 diets. Large LDL and large and intermediate HDL fractions were significantly higher, mean LDL particle size was significantly greater, and intermediate VLDL fractions were significantly lower in the postmenopausal women than in the other 2 groups. A group-by-diet interaction effect was observed on LDL fractions and small LDL particle size.

McIntosh et al (1999) conducted a study to examine the influence of two sources of dietary fiber (non starch polysaccharides, NSP) on blood lipids and glucose concentrations. Barley contains 3gm β-glucan as a source of soluble dietary fiber (DF) whereas wheat contains the largely insoluble cellulose and hemicellulose fiber. Total dietary fiber increased from a previous intake of 21-38 g/day during the period of study for the two groups. Consumption of barley relative to wheat foods was associated with a significant fall in both plasma total cholesterol (6%, P < 0.05) and in low-density-lipoprotein cholesterol (7%, P < 0.02) whereas triglyceride and glucose concentrations did not change significantly. It is concluded that barley dietary fiber is more effective than wheat dietary fiber at lowering blood cholesterol in hypercholesterolemia men.
Another study conducted by Delaney et al (2003) to compare the effects of concentrated β-glucan from oats and barley on plasma lipids and lipoprotein and hepatic cholesterol, fecal excretion of neutral sterols and total bile acids, and antiatherogenic properties in hamsters consuming a hypercholesterolemia diet (HCD). This study discuss the antiatherogenic properties of β-glucan concentrates from oats and barley were evaluated in Syrian golden F1B hamsters consuming a semi-purified hypercholesterolemia diet (HCD) containing cholesterol (0.15 g/100 g), hydrogenated coconut oil (20 g/100 g) and cellulose (15 g/100 g). After a 2-wk lead-in period, control hamsters were fed the HCD, whereas experimental hamsters consumed HCD formulated to include β-glucan (2, 4, or 8 g/100 g) by addition of β-glucan concentrate prepared from oats or barley at the expense of cellulose. Compared with control hamsters, dose dependent decreases that were similar in magnitude in plasma total and LDL cholesterol concentrations were observed in hamsters fed β-glucan from either source at wk 3, 6 and 9. Compared with controls, liver cholesterol concentrations were also reduced (P <0.05) in hamsters consuming 8 g/100 g oat or barley β-glucan. In agreement with previously proposed mechanisms, total fecal neutral sterol concentrations were significantly increased (P<0.05) in hamsters consuming 8 g/100 g barley or oat β-glucan. Aortic cholesterol ester concentrations were significantly reduced (P < 0.05) in hamsters fed 8 g/100 g β-glucan from barley or oats. Although aortic total cholesterol and cholesterol ester concentrations were significantly correlated with LDL cholesterol (r = 0.565, P<0.004 and r = 0.706, P<0.0001, respectively), this association could explain only half of the variability. This study demonstrated that the cholesterol-lowering potency of β-glucan is approximately identical whether its origin was oats or barley.
Keenan et al (2007) conducted an interventional study to evaluate the efficacy of a diet augmented with food products (cereal and juice beverage) that were enriched with BBG to increase their VSF content. In a 10-week blinded controlled study, subjects were randomized to one of four treatment groups or control. Treatment groups included either high molecular weight (HMW) or low molecular weight (LMW) BBG at both 3 and 5 g doses. Treatment was delivered twice per day with meals in the form of two functional food products: a ready-to-eat cereal and a reduced-calorie fruit juice beverage. Levels of total cholesterol, LDL-C, HDL-cholesterol (HDL-C), and TAG were determined at baseline and after 6 weeks of treatment. The study group comprised 155 subjects. All treatments were well tolerated and after 6 weeks of treatment the mean LDL-C levels fell by 15% in the 5 g HMW group, 13% in the 5 g LMW group and 9% in both the 3 g/d groups, versus baseline. Similar results were observed for total cholesterol. HDL-C levels were unchanged by treatment. Concentrated BBG significantly improves LDL-C and total cholesterol among moderately dyslipidaemic subjects. Food products containing concentrated BBG should be considered an effective option for improving blood lipids.

K.M. Behall et al (2006) conducted a study at Bloomberg. This study was planned to compare the effects of predominantly insoluble fiber (whole wheat and brown rice) and soluble fiber (barley) in a whole-grain diet on blood pressure. Subjects (7 men, 9 premenopausal women and 9 postmenopausal women) consumed a controlled Step I diet for 2 weeks; then about 20% of energy was replaced with whole wheat/brown rice, barley, or half wheat-rice/half barley, for 5 weeks each. Blood pressure was determined weekly and weight daily before breakfast. Urinary excretions of minerals that might affect blood pressure and urea nitrogen were
determined each period. Systolic pressure was lower after the wheat/rice and half-and-half diets. Diastolic and mean arterial pressures were reduced by all whole-grain diets. No differences were observed in urinary measurements. In a healthful diet, increasing whole-grain foods, whether high in soluble or insoluble fiber can reduce blood pressure and may help to control weight. Consumption of a healthful diet high in fiber from wholegrain foods lowers systolic and diastolic blood pressure in mildly hypercholesterolemia men and women whether sources are barley (soluble fiber), whole wheat and brown rice (insoluble fiber), or a combination of these wholegrain foods.