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IMPACT OF β-GLUCAN-BASED DIET IN ASSOCIATION WITH THE PREVENTION OF NIDDM IN ATHLETES IMPROVING CARDIOVASCULAR FUNCTIONING

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Abstract— β-glucan has immense potential in lowering the cholesterol and blood sugar levels in the body, and a β-glucan-rich diet may help in maintaining the glycemic index of individuals suffering from metabolic disorders. This study focuses on the prevention & treatment of diabetes mellitus and obesity. Four groups are involved in the study, comprising one control group (healthy subjects) and 3 treated groups (diabetic & non-obese subject, non-diabetic & obese subjects, and diabetic & obese subjects). The subject will be recommended to consume a β-glucan-rich diet for one year depending upon the voluntary availability of subjects. After the intervention period is done, various parameters comprising anthropometric, biochemical, clinical, and dietary assessments along with psychological parameters will be evaluated. This study will help evaluate and determine the effect of a high-carb, β-glucan-rich diet on the blood parameters and lipid profiles of individuals suffering from metabolic disorders.

Keywords*— *β-Glucan, metabolic syndrome, diabetes mellitus, obesity, nutritional intervention.

I. INTRODUCTION

Diabetes mellitus (DM), a metabolic disorder, is brought on by a failure in either the secretion or the action of insulin. In turn, a lack of insulin leads to chronic hyperglycemia with anomalies in the metabolism of proteins, lipids, and carbs. Insulin resistance associated with obesity is the best indicator of type 1 diabetes, other from HLA genotype, and it manifests prior to the development of chronic illness. (Baum et al. 1975; Johansson et al. 1994; Hypponen et al. 1999, 2000; Bruining 2000; Xu et al. 2007; Fourlanos et al. 2008).

Type 1 and type 2 diabetes mellitus are the two most prevalent forms, while there are others. Vascular or tissue damage brought on by diabetes can result in serious side effects such ulceration, retinopathy, neuropathy, nephropathy, and cardiovascular problems. (Bastaki, S. (2005). While the incidence of type 1 diabetes has stayed constant for high-risk HLA types over the past 20 years, it has increased by two, three, and seven times for medium-, low-, and very-low-risk HLA types, respectively. (Fourlanos et al. 2008b).

One of the well-known and extensively studied bioactive polysaccharides is β-glucan. B-glucan is a non-starch polymer composed of unbranched or branching β-D-glucose monomer units with a glyosidic link at β (1→3), (1→4), and/or (1→6).(Kaleem, 2018; Ahmad, A). The endospermic and aleuronic walls of cereal grains (oats, barley, rye, and millets) and the cell walls of different microorganisms (fungi, yeast, and bacteria) are commonly discovered to contain beta glucan. (Bernstein, A.M.; Titgemeier, B.; Kirkpatrick, K.; Golubic, M.; Roizen, M.F. 2013), (Fesel, P.H.; Zuccaro, 2016).

Low in fat and high in complex carbs is the Pritikin diet. Less than 10% fat, 10% to 15% protein, and 75% to 80% mostly complex carbohydrates make up the Pritikin diet. It also includes less than 25 mg of cholesterol day (for the regression diet) or less than 100 mg daily (for the maintenance diet). (Li, Z., & Heber, D. (2020).

By restricting all high-fat, calorie-dense meals, the Pritikin diet lowers cholesterol intake while simultaneously lowering total caloric intake. The detrimental effects of additional sugars in the diet have come to light more lately. (Li, Z., & Heber, D. (2020).

LACUNA

1. Not much evidence is available on the long term effect of β-glucan rich diet in the prevention of diabetes mellitus.
2. Little evidence is available on the nutritional adequacy of the High fibre diet.
3. No effect of β-glucan is clearly analysed in association with the prevention of metabolic syndromes including diabetes mellitus & obesity.

II. REVIEW OF LITERATURE

The human body's many systems and pathways work together to develop and sustain a healthy physiological state. At the core of these activities is the body's capacity to preserve homeostasis, or a constant, stable state. An imbalance in homeostasis causes an injury or pathological condition to occur in several organs. A person with diabetes mellitus has trouble controlling their blood glucose levels, which can result in a number of significant and minor issues. (Kaul, K., Tarr, J. M., Ahmad, S. I., Kohner, E. M., & Chibber, R. (2013).

The hallmark of type 1 diabetes mellitus (T1DM), a chronic autoimmune illness caused by an insulin shortage brought on by the loss of pancreatic islet β-cells, is hyperglycemia, or high blood glucose. (SEARCH Study Group. (2004). (Gepts, W. (1965). (Eisenbarth, G. S. (1986). (Atkinson, M. A., Eisenbarth, G. S. & Michels, A. W. (2014).

However, the liver, skeletal muscle, and fat are the three target tissues that are most important in relation to glycaemia. (Saltiel and Kahn 2001). These tissues are the sites of insulin's metabolic action as well as the primary causes of insulin resistance. (Kahn and Flier 2000; Shulman 2000; Qatanani and Lazar 2007). Because insulin cannot stop hepatic gluconeogenesis while lipid production remains unaffected, insulin resistance in the liver results in elevated fasting serum glucose, a critical clinical criteria for the diagnosis of type 2 diabetes. (Brown and Goldstein 2008).

Hyperlipidemia, hyperglycemia, and compensatory hyperinsulinemia are the outcomes of insulin resistance in skeletal muscle and adipose tissue, which presents as increased lipolysis and glucose intolerance, respectively. (Kahn and Flier 2000; Shulman 2000). It is thought that environmental stimuli cause an autoimmune response against pancreatic beta cells in a genetically predisposed person, leading to type 1 diabetes. The major histocompatibility complex on chromosome 6—which includes the protective DR2-DQ6 allele and the risky DR3-DQ2 and DR4-DQ8 alleles—is the greatest genetic component of the risk of diabetes. Relatives of people with diabetes have a higher risk of developing the condition due to the hereditary component of risk. (Cooke, D. W., & Plotnick, L. (2008).

Insulin-sensitizing drugs and treatments (such exercise and weight loss) can prevent, delay, and even partially reverse type 1 illness. (Kjems et al. 2003; Miller and Silverstein 2006; Kilpatrick et al. 2007; Moon et al. 2007; Neovius et al. 2008).

Insulin resistance, decreased insulin production, or a combination of the two causes type 2 diabetes mellitus (T2DM), which is characterized by dysregulation of protein, lipid, and carbohydrate metabolism. (DeFronzo, R. A (2015). Type 2 diabetes is influenced by both genetic and environmental factors. The pathophysiological changes, which progressively compromise blood glucose homeostasis and lead to the formation of micro- and macrovascular problems, are characterized by β-cell failure, insulin resistance, and chronic inflammation. (DeFronzo, R. A. (2009). (DeFronzo, R. A. (2010).

T2DM is heritable and tends to cluster in families. (Hemminki, K., Li, X., Sundquist, K. & Sundquist, J. (2010). When the mother has type 2 diabetes, the likelihood of developing the condition is higher than when the father has. (Groop, L. et al. (1996). A BMI of ≥30 or a non-normal fasting glucose level of >5.5 mmol l–1 are also significantly associated with an elevated risk of developing type 2 diabetes. (Lyssenko, V. et al. (2005)

Together with a genetic predisposition, (Morris, A. P. et al. (2012). Obesity and physical inactivity cause insulin resistance, which stresses β cells and eventually results in a drop in insulin secretion and β cell failure (Morris, A. P. et al. (2012). (DeFronzo, R. A. (2009), (Abdul-Ghani, M. A., Tripathy, D. & DeFronzo, R. A. (2006). (DeFronzo, R. A. & Abdul-Ghani, M. A. (2011). (Kahn, S. E., Cooper, M. E. & Del Prato, S. (2014), (Shulman, G. I. et al. (1990). Insulin resistance occurs many years before type 2 diabetes. (DeFronzo, R. A. (2009), (Abdul-Ghani, M. A., Tripathy, D. & DeFronzo, R. A. (2006), (Gulli, G., Ferrannini, E., Stern, M., Haffner, S. & DeFronzo, R. A. (1992), (Martin, B. C. et al. (1992).

The body needs glucose and energy from digestible polysaccharides for regular metabolic functions. (Ferretti, F.; Mariani, M. 2017), (Qiao, F.; Liu, Y.K.; Sun, Y.H.; Wang, X.D.; Chen, K.; Li, T.Y.; Li, E.C.; Zhang, M.L.2017) . The β-glucan's solubility profile is essential for the molecules' rheological, nutritional, and sensory applications. (Temelli, F.; Bansema, C.; Stobbe, K, 2004.). β-glucans were discovered to be the most efficient polysaccharide immunostimulants against cancer and viral illnesses. (G. D. Brown and S. Gordon, 2003).

The ability of plant β-glucans (referred to as "β-glucans" in the following sections) to form very viscous solutions in the human stomach is thought to be the basis for their health advantages. These benefits include reducing postprandial insulin and glucose responses, improving feelings of fullness, and lowering cholesterol. Beta glucan can form very viscous solutions because it is a straight, unbranched, non-starchy polymer composed of glucose molecules with β (1–4) and β (1–3) linkages. (P. J. Wood and M. U. Beer, 1998.)

Several soluble fibers, including guar gum, psyllium, and β-glucan, improve insulin sensitivity and decrease postprandial glucose and insulin responses in both diabetics and non-diabetics. (H. Hanai, M. Ikuma, Y. Sato et al. 1997.), (I. Thorsdottir, H. Andersson, and S. Einarsson, 1998), (J. W. Anderson, L. D. Allgood, J. Turner, P. R. Oeltgen, and B. P. Daggy, 1999), (M. Sierra, J. J. Garcia, N. Fernandez et al., 2001), (M. Sierra, J. J. Garc´ıa, N. Fernandez et al., 2002), (K. S. Juntunen, L. K. Niskanen, K. H. Liukkonen, K. S. Poutanen, J. J. Holst, and H. M. Mykkanen, “Postprandial glucose, insulin, and incretin responses to grain products in healthy subjects,”

American Journal of Clinical Nutrition, vol. 75, no. 2, pp. 254–262, 2002), (M. Alminger and C. Eklund-Jonsson, 2008).

Soluble fibers have been shown to have the most beneficial impact on cholesterol metabolism. According to a meta-analysis, soluble fibers such as pectin, psyllium, oat bran, and guar gum are equally effective at reducing plasma total and LDL cholesterol levels. (L. Brown, B. Rosner, W. W. Willett, and F. M. Sacks, 1999). When added to a diet low in saturated fat and cholesterol, soluble fibers decreased LDL cholesterol levels by 5–10% in patients with diabetes and hypercholesterolemia. (L. Brown, B. Rosner, W. W. Willett, and F. M. Sacks, 1999). (M. Sierra, J. J. Garc´ıa, N. Fernandez et al, 2002). There have been no documented negative effects on humans after consuming a diet high in β-glucan from barley or oat flour or their extracts. (J. Hallfrisch and K. M. Behall, 2003).

In patients with metabolic syndrome, the Pritikin Program (Aventura, FL) offers a special chance to assess the clinical consequences of an exercise regimen and an ad libitum, very-low-fat diet (10%–15% of total calories). The Pritikin Longevity Program's short-term treatment, which consists of a very-low-fat, low-sodium, high-fiber diet and exercise, concurrently improves the majority of metabolic CHD risk factors and lowers the prevalence of the metabolic syndrome in patients who already have it. (Sullivan, S., & Samuel, S. (2006). When combined with daily exercise and weight loss, the Pritikin high-complex carbohydrate, high-fibre, low-fat diet has been demonstrated to be very effective in lowering fasting glucose and removing the need for medication in a large number of NIDDM patients. (Barnard, R. J., Lattimore, L., Holly, R. A., Cherny, S., 1982).

Many NIDDM patients seem to respond well to the Pritikin high-complex-carbohydrate, high-fibre, low-fat diet when paired with regular exercise and sensible weight management. With long-term adherence to the regimen, the majority of patients are able to stop either insulin injections or oral hypoglycemic medications. Massey, M. R., Cherny, S., O'Brien, L. T., Barnard, R. J., & Pritikin, N. (1983).

III. AIMS & OBJECTIVES

Primary:

To study the effect of β-glucan-rich diet on the reduction of weight & diabetes on the control & target groups before and after the study.

Secondary:

1. To assess the effect of β-glucan-rich diet on the nutritional status of the control and target groups before and after the study.

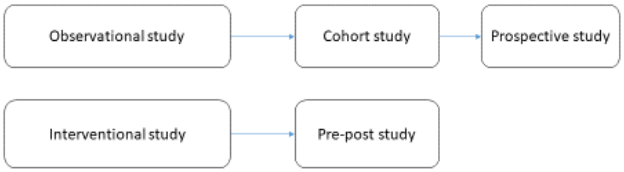
2. To assess the quality of life and psychological behaviour of the control & target groups.

IV. MATERIALS & METHODS

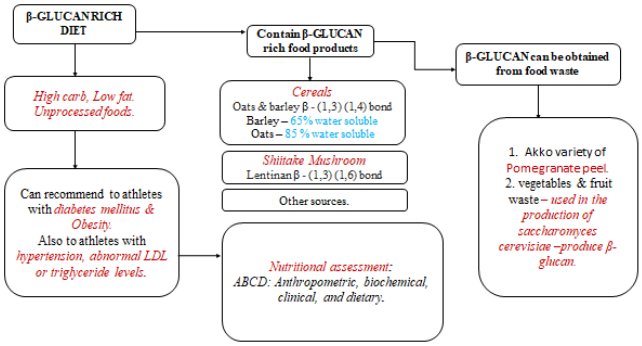
Place of study

The study will be done at hospital and sports centre.

Study design



Study Timetable/Flowchart:



Study population

A number of athletes, both males & females who are suffering from diabetes mellitus & obesity, aged in the range from 25 to 50 years, will be included in the study.

Number of population: 500 (50 for each group).

Number of groups: 4

Groups	Control group	Target group 1	Target group 2	Target group 3
Subjects	Healthy subjects	Diabetic & non – obese subject	Non – diabetic & obese subject	Diabetic & obese subject
Prescribed diet	High carb, fibre & low fat β-glucan rich	High carb, fibre & low fat β-glucan rich	High carb, fibre & low fat β-glucan rich	High carb, fibre & low fat β-glucan rich
Prescribed food items	β- glucan rich food products. Food products formulated with beta glucan along with the beta glucan rich food items already available. (Maize, rye, sorghum, seaweed, mushrooms, barley, oats etc.)	β- glucan rich food products. Food products formulated with beta glucan along with the beta glucan rich food items already available. (Maize, rye, sorghum, seaweed, mushrooms, barley, oats etc.)	β- glucan rich food products. Food products formulated with beta glucan along with the beta glucan rich food items already available. (Maize, rye, sorghum, seaweed, mushrooms, barley, oats etc.)	β- glucan rich food products. Food products formulated with beta glucan along with the beta glucan rich food items already available. (Maize, rye, sorghum, seaweed, mushrooms, barley, oats etc.)

Study duration

The duration of the study will be 2 – 2.5 years.

Inclusion criteria:

a) For control group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: should be healthy having BMI range from 18 - 22.9.
4. Stage of diabetes mellitus: should be healthy having a blood sugar level less than 140 mg/dL (7.8 mmol/L).

b) For diabetic & non - obese group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: should be healthy having BMI range from 18 - 22.9.
4. Stage of diabetes mellitus: should be having:
   a) FPG 100–125 mg/dL (5.6–6.9 mmol/L)
   b) 2-h PG 140 –199 mg/dL (7.8–11.0 mmol/L)
   c) A1C 5.7– 6.4% (39–47 mmol/mol) or ≥ 10 % increase in A1C.

c) For non - diabetic & obese group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: should be healthy having BMI ≥ 30.
4. Stage of diabetes mellitus: should be healthy having A blood sugar level less than 140 mg/dL (7.8 mmol/L).

d) For diabetic & obese group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: should be healthy having BMI ≥ 30.
4. Stage of diabetes mellitus: having:
   a) FPG 100–125 mg/dL (5.6–6.9 mmol/L)
   b) 2-h PG 140 –199 mg/dL (7.8–11.0 mmol/L)
   c) A1C 5.7– 6.4% (39–47 mmol/mol) or ≥ 10 % increase in A1C.

Exclusion criteria

a) For control group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: having BMI ≥ 30.
4. Stage of diabetes mellitus: having:
   a. FPG 100–125 mg/dL (5.6–6.9 mmol/L)
   b. 2-h PG 140 –199 mg/dL (7.8–11.0 mmol/L)
   c. A1C 5.7– 6.4% (39–47 mmol/mol) or ≥ 10 % increase in A1C.

b) For diabetic & non - obese group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: having BMI ≥ 30.
4. Should be healthy having a blood sugar level less than 140 mg/dL (7.8 mmol/L).
5. Possess following medical conditions:

   Coeliac disease, thyroid disorder: hypothyroidism and hyperthyroidism, High blood pressure, cardiovascular disease (CVD), coronary heart disease (CHD), stroke, peripheral arterial disease, and cardiomyopathy. Nerve damage (neuropathy), Kidney damage (nephropathy), Eye damage (retinopathy), Skin and mouth conditions, Hearing impairment, Alzheimer's disease, Depression.

c) For non - diabetic & obese group:

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
3. Stage of obesity: should be healthy having BMI range from 18 - 22.9.
4. Stage of diabetes mellitus: having:
   d. FPG 100–125 mg/dL (5.6–6.9 mmol/L)
   e. 2-h PG 140 –199 mg/dL (7.8–11.0 mmol/L)
   f. A1C 5.7– 6.4% (39–47 mmol/mol) or ≥ 10 % increase in A1C.

d) **For diabetic & obese group:**

1. Age: range from 25 - 50 years.
2. Gender: Both male and female.
5. Stage of obesity: having BMI range from 18 - 22.9.
3. Stage of diabetes mellitus: having a blood sugar level less than 140 mg/dL (7.8 mmol/L).

e) Individuals Possess following medical conditions:

Coeliac disease, thyroid disorder: hypothyroidism and hyperthyroidism, High blood pressure, cardiovascular disease (CVD), coronary heart disease (CHD), stroke, peripheral arterial disease, and cardiomyopathy. Nerve damage (neuropathy), Kidney damage (nephropathy), Eye damage (retinopathy), Skin and mouth conditions, Hearing impairment, Alzheimer's disease, Depression.

Sample size calculation

Slovin's Formula:

$$n = \frac{N}{1 + Ne^2}$$

Where:

- n = Number of samples,
- N = Total population and
- e = Error tolerance (level)

n = N / (1 + N e2) =

Putting the value as:

N (total population) = 500

E (error tolerance) = 0.05

We get the sample size = 222.

Discontinuation of the Study:

Possible reasons can be:

1) Organisation is inadequate.
2) Funding does not match the incurring costs.
3) Target patients are so rare that it is impracticable to recruit the required sample.

V. METHODOLOGY

1. INTERVENTION:

The typical diet of the control and target groups is changed to a high-carb, high-fiber, low-fat diet that is rich in β-glucan. Foods high in β-glucan will be suggested depending on the subjects' preferences and dislikes, and physical activity will be suggested if necessary.

NUTRITIONAL ASSESSMENT:

	Pre - assessment	**Observation period of one year: β- glucan rich diet will be recommended**	**Post - assessment**
Anthropometric assessment	Height, sitting height & Weight measurement. Calf (CC), mid upper arm (MUAC), chest (CCN), waist (WC) and hip (HC) circumferences measurement. Calf (CSK), biceps (BSK), triceps (TSK), sub-scapular (SBSK) and supra-iliac (SISK) skinfold measurement. Bi - epicondylar diameter of humerus (BDH), bi-condylar diameter of femur (BDF), bi-acromial diameter (BAD) and bi-iliac diameter (BID) measurement. Body mass index (BMI) measurement: $BMI = \frac{Weight\ (kg)}{\{Height\ (m)\}^2}$	Recommend the β- glucan rich diet. Diet along with the β- glucan rich food products. High complex carbohydrate, fibre & low fat food products and food products formulated with beta glucan along with the beta glucan rich food items already available. (Maize, rye, sorghum, seaweed, mushrooms, barley, oats etc.)	Height, sitting height & Weight measurement. Calf (CC), mid upper arm (MUAC), chest (CCN), waist (WC) and hip (HC) circumferences measurement. Calf (CSK), biceps (BSK), triceps (TSK), sub-scapular (SBSK) and supra-iliac (SISK) skinfold measurement. Bi - epicondylar diameter of humerus (BDH), bi-condylar diameter of femur (BDF), bi-acromial diameter (BAD) and bi-iliac diameter (BID) measurement. Body mass index (BMI) measurement: $BMI = \frac{Weight\ (kg)}{\{Height\ (m)\}^2}$
Biochemical assessment	Measurement of: BUN level lactate acid level creatine kinase level lactic dehydrogenase level total cholesterol level HDL level LDL level Triglycerides level Non esterified fatty acid level	Recommend the β-glucan rich diet.	Measurement of: BUN level lactate acid level creatine kinase level lactic dehydrogenase level total cholesterol level HDL level LDL level Triglycerides level Non esterified fatty acid level
Clinical assessment	Nutrient deficiency assessment. Assessment of Clinical signs include the symptoms such as: a. Pallor: on the palm of the hand or the conjunctiva of the eye. b. Pitting oedema c. Bitot's spot on the eye. d. Visible wasting etc.	Recommend the β- glucan rich diet.	Nutrient deficiency assessment. Assessment of Clinical signs include the symptoms such as: a. Pallor: on the palm of the hand or the conjunctiva of the eye. b. Pitting oedema c. Bitot's spot on the eye. d. Visible wasting etc.
Dietary assessment	Assessment include: Duplicate diet approach Food consumption record 24-Hour dietary recall Dietary record Dietary history Food frequency questionnaire	Recommend the β- glucan rich diet.	Assessment include: Duplicate diet approach Food consumption record 24-Hour dietary recall Dietary record Dietary history Food frequency questionnaire
Psychological assessment	Assessment include: a) Norm-referenced psychological tests. b) Informal tests and surveys. c) Interview information. d) Medical records, medical evaluation, and observational data.	Recommend the β- glucan rich diet.	Assessment include: a) Norm-referenced psychological tests. b) Informal tests and surveys. c) Interview information. d) Medical records, medical evaluation, and observational data.
Behavioural Assessment	Include: a) Direct assessment b) Analog assessment c) Indirect Assessment d) Idiographic assessment e) Contextual assessment	Recommend the β- glucan rich diet.	Include: f) Direct assessment g) Analog assessment h) Indirect Assessment i) Idiographic assessment j) Contextual assessment
Assessment of quality of life	Assessment of: ***Social or cultural*:** Access to care, societal stigma, and support. ***Coping:*** Ability to withstand stress, psychological or Physical ***General health perceptions:*** Self-rating, worry, concern ***Expectations/satisfaction:*** Satisfaction with functioning ***Social:*** Work and daily role ***Psychological:*** Distress (anxiety, depression, loss of behavioural and emotional control) Well-being (positive affect, emotional ties, life satisfaction) ***Cognitive:*** Memory, alertness, reasoning ***Physical:*** Activity restrictions, fitness ***Signs:*** Objective clinical findings directly observable ***Symptoms:*** Subjective evidence indirectly observable Physiologic Diagnosis and severity: Laboratory measures, pathology.	Recommend the β- glucan rich diet.	Assessment of: ***Social or cultural*:** Access to care, societal stigma, and support. ***Coping:*** Ability to withstand stress, psychological or Physical ***General health perceptions:*** Self-rating, worry, concern ***Expectations/satisfaction:*** Satisfaction with functioning ***Social:*** Work and daily role ***Psychological:*** Distress (anxiety, depression, loss of behavioural and emotional control) Well-being (positive affect, emotional ties, life satisfaction) ***Cognitive:*** Memory, alertness, reasoning ***Physical:*** Activity restrictions, fitness ***Signs:*** Objective clinical findings directly observable ***Symptoms:*** Subjective evidence indirectly observable Physiologic Diagnosis and severity: Laboratory measures, pathology.

2. DETECTION OF HEART FUNCTION
Using Electrocardiogram and Echocardiogram

3. DETECTION OF CARDIAC MUSCLE ACTIVITY
Using Electromyography

EXPECTED OUTCOMES

We will be able to find out the:

1. The impact and benefits long term effect of β-glucan-rich diet in metabolic syndromes including diabetes mellitus & obesity.
2. Effect of β-glucan-rich diet on the reduction of weight & diabetes on the athletes.
3. Impact of High fibre diet on overall cardiovascular health & function.
4. Effect of β-glucan-rich diet on the nutritional status of the athletes.
5. Impact of β-glucan-rich diet on the quality of life and psychological behaviour of athletes.

VI. CONCLUSION

The use of the β-glucan-rich high-complex carbohydrate, high-fibre, low-fat diet in combination with daily exercise and weight loss is highly effective in reducing fasting glucose and eliminating the need for medication in many non-insulin-dependent diabetes mellitus (NIDDM) athletes. And it also appears to be an effective long-term means of treatment for many NIDDM athletes. Also, High fibre diet having β- glucan rich foods have an impressive impact on cardiovascular functioning.

ETHICAL CONSIDERATIONS

1. **Voluntary participation**: All the participants are free to opt out of the study at any point of time.

2. **Informed consent:** Participants will be aware of the purpose, risks, benefits and funding behind the study before they agree or disagree to join.

3. **Anonymity:** Identity of all the participants will be recorded.

4. **Confidentiality**: Information of all the participants is kept hidden and anonymized.

5. **Potential for harm:** All the Physical, social, psychological types of harm will be avoided.

6. **Results communication:** All the work will be free from plagiarism and represent accurately with the results.

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Conflicts of Interest: The authors and co-authors have declared no conflict of interest.

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