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Review Article

Role of Beta-Glucan in Diabetes Management

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ABSTRACT

Diabetes is a universal metabolic disorder prevalent in world and in India it comprises 7.8% of world diabetic population. Beta cells of islets of Langerhans of pancreas secrete insulin hormone which regulate the cellular intake of glucose in human body. Due to insufficient insulin secretion or insulin in sensitivity or any injury in pancreas impairs cellular glucose intake leads rise in blood sugar level. This condition is called as diabetes. Beta glucan found in foods like barley oats etc is a pro-glucagon molecule which exerts strong insulinotropic effects in vivo. It is a good alternative of diabetic medicine for diabetes patients.

Key words: Diabetes, Beta Glucan.

INTRODUCTION

The aetiology of diabetes in India is multi factorial and includes genetic factors coupled with environmental influences such as obesity associated with rising living standards, steady urban migration, and life style changes. More than 80% of people live in low and middle income countries. Pattern of diabetes incidence are related to the geographical distribution of diabetes in India. According to WHO (2014) in 2000 Indian diabetic population was 31.7 million and it is expected to be 79.4 till 2030. It contributes 7.8% (50.8 million) of world diabetic population. Prevalence of diabetes in rural population is one- quarter that of urban population for India and other Indian sub- continent country such as Bangladesh, Nepal, Bhutan and Sri Lanka.

Diabetes is a group of metabolic disorders resulting from a defect in insulin secretion, insulin action or both (ADA, 2000). These defects alter glucose uptake by cells and its use within them. Insulin facilitates glucose uptake and use. Diabetes involves an absolute or relative insulin arises when the pancreas fails to produce insulin due to destruction of the pancreatic beta cells usually resulting from an autoimmune disorder or deficiency occurs when insulin requirements are increased results in insulin resistance (Bowman and Russel, 2001). It is generally accepted that beta-cell failure is caused by insulin resistance. It was diagnosed that in diabetic patients β- cell function may have decreased by up to 80% because of increased oxidative stress is one of the mechanisms by which hyperglycaemia damages β- cell (DeFronzo *et al.*, 2008). Treatments with beta cell–preserving properties are therefore essential for the management of type 2 diabetes (Standl E., 2007). Glucagon like peptide is a fragment of proglucagon molecule (Drucker, 1990). it exerts strong insulinotropic effects in vivo (Orskov *et al.*, 1987). β - glucans, are frequently present in endosperm cell walls of cereals. Both soluble and insoluble beta glucans are

present in cereals (Ahlu walia and Ellish 1985)

DISCUSSION

Glucagon-like peptide-1- (7–36) amide (GLP-1), a potent gluco-incretin hormone (Fehmann *et al.*, 1995) secreted by the intestinal L-cells in response to fat meals and carbohydrates is a potentially important drug in the treatment of Type 2 diabetes in view of its ability to improve insulin secretion in subjects with impaired glucose tolerance and Type 2 diabetes mellitus (Drucker, 2001). A decline in insulin: glucagon ratio would favour mobilization of stored nutrients; increased hepatic glucose production from glycogen and from available amino acids would occur at the expense of protein synthesis, nitrogen balance would shift towards the negative with increased production of urea, and the release of free fatty acids and glycerol from adipose tissue would also increase. In other words, high insulin: glucagon ratio would favor nutrient storage and protein anabolism, while a low insulin: glucagon ratio would favor mobilization of nutrient stores and protein catabolism (Unger *et al.*, 1970).

Furthermore Buteau *et al.* (2004) investigated on whether glucagon-like peptide-1 protects beta cells against cell death induced by elevated glucose and/or non-esterified fatty acids. Human islets and INS832/13 cells were cultured at glucose concentrations of 5 or 25 mmol/l in the presence or absence of palmitate. Apoptosis was evaluated by monitoring DNA fragmentation and chromatin condensation. Wild-type and protein kinase B mutants were over expressed in INS832/13 cells using adenoviruses. Nuclear factor-κB DNA binding was assayed by electrophoretic mobility shift assay. Results revealed that in human pancreatic beta cells and INS832/13 cells demonstrate a potent protective effect of glucagon-like peptide-1 on beta cell gluco-, lipo- and glucolipotoxicity. This effect is mediated via protein kinase B activation and possibly its downstream target nuclear factor- B. Glucagon-like peptide-1 (7- 36) amide (glucagon-like insulinotropic peptide, or GLIP) is a gastrointestinal peptide that potentiates the release of insulin in physiologic concentrations. We compared the effect of an infusion of GLIP twofold with the effect of an infusion of saline, on the meal-related release of insulin, glucagon, and somatostatin in normal subjects, nine obese patients with non- insulin - dependent diabetes mellitus. The blood glucose concentrations in the patients with diabetes were controlled by closed- loop insulin - infusion system (artificial pancreas) during the infusion of each agent, allowing measurement of the meal -related requirement for exogenous insulin. Result demonstrates that in the normal subjects, the infusion of GLIP significantly lowered the meal- related increase in the blood glucose concentration and the plasma concentration of insulin and glucagon. GLIP has an antidiabetogenic effect, and it may therefore be useful in the treatment of patients with NIDDM. Shah. et al (2013) studied that the hypothesis that a lack of suppression of glucagon causes postprandial hyperglycemia in subjects with type 2 diabetes. Nine diabetic subjects ingested 50 g glucose on two occasions. On both occasions, somatostatin was infused at a rate of 4.3 nmol/kg·min, and insulin was infused in a diabetic insulin profile. On one occasion, glucagon was also infused at a rate of 1.25 ng/kg·min to maintain portal glucagon concentrations constant (nonsuppressed study day). On the other occasion, glucagon infusion was delayed by 2 h to create a transient decrease in glucagon (suppressed study day). Glucagon concentrations on the suppressed study day fell to about 70 ng/L during the first 2 h, rising thereafter to approximately 120 ng/L. In contrast, glucagon concentrations on the nonsuppressed study day remained constant at about 120 ng/L throughout. The decrease in glucagon resulted in substantially lower ($P < 0.001$) glucose concentrations on the suppressed compared with the

nonsuppressed study days (9.2 ± 0.7 *vs.* 10.9± 0.8 mmol/L) and a lower ($P < 0.001$) rate of release of glucose from glycogen (labeled by infusing galactose). On the other hand, flux through the hepatic UDP-glucose pool (and, by implication, glycogen synthesis), measured using the acetaminophen glucuronide method, did not differ on the two occasions. Result showed that lack of suppression of glucagon contributes to postprandial hyperglycemia in subjects with type 2 diabetes at least in part by accelerating glycogenolysis. These data suggest that agents that antagonize glucagon action or secretion are likely to be of value in the treatment of patients with type 2 diabetes.

Likewise Degn. et al (2004) reported that once-daily dosing with the GLP-1 derivative liraglutide for 1 week in individuals with type 2 diabetes results in 1) substantially decreased glucose concentration lasting 24 h and is ascribable to a relative increase in insulin secretion and decrease in glucagon concentration; 2) reduced EGR, as a result of diminished GLY; and 3) substantially improved AIR and maximal insulin secretory capacity. During longer lasting treatment, these promising antidiabetic effects may be further supplemented by an increase in -cell mass and the benefit achieved by weight loss. Drucker *et* al (1986) investigated that insulin secretion is controlled by a complex set of factors. Although blood glucose levels serve as the major stimulus of insulin secretion in mammals, insulin release is also modulated by amino acids, catecholamines, glucagon, and other, intestinal hormones. The identification of factors that modulate insulin production has engendered much interest because of their potential importance in the altered dynamics of insulin secretion in response to glucose characteristic of maturity-onset diabetes mellitus. Decoding of the glucagon gene has uncovered two additional glucagon-like peptides encoded in proglucagon, the polypeptide precursor of glucagon. One of these peptides, glucagon-like peptide I, is processed from proglucagon in two forms, of 31 and 37 amino acids. We report that the smaller of the two glucagon-like peptides potently increases cAMP levels, insulin mRNA transcripts, and insulin release in cultured rat insulinoma cells. These results indicate that glucagon-like peptide I is a physiologic modulator of insulin gene expression.

Glucagon like peptide 1 (GLP1) (736 amide) is a physiological incretin hormone that is released after nutrient intake from the lower gut and stimulates insulin secretion at elevated plasma glucose concentrations. It was seen that exogenous GLP1 (736 amide) is an effective means of normalizing fasting plasma glucose concentrations in poorly controlled Type 2 diabetic patients. The glucose dependence of insulinotropic actions of GLP1 (736 amide) appears to be retained in such patients (Nauck *et al.,* 1993). Hallfrisch and Behall (2013) studied that various grains and grain products effective in improving insulin resistance or lowering glycemic index. The composition of the grain, including particle size, amount and type of fiber, viscosity, amylose and amylopectin content all affect the metabolism of carbohydrates from grains. Increasing whole grain intake in the population can result in improved glucose metabolism and delay or reduce the risk of developing type 2 diabetes mellitus. Whole grains provide a substantial contribution to the improvement of the diets. A number of whole grain foods and grain fiber sources are beneficial in reduction of insulin resistance and improvement in glucose tolerance. In this context Pi-Sunyer, (1995) studied that fiber is present in the carbohydrate component of the diet, the main source of which is cereals. But processed cereals tend to have a high GI. However, some cereals, such as barley or fractions of oat bran, are particularly high in the soluble fiber i.e. β-glucan taken in a meal increases the viscosity of the meal bolus once it has reached the small intestine, where absorption of nutrients occurs. The high viscosity delays absorption. When (β-

glucan is part of the food matrix, it seems to exert a more effective delaying action. With a concentration β-glucan of around 8-10% in a cereal, a 50% reduction in glycemic peak can be expected. Diabetic individuals should benefit from diets that are high in soluble fiber (β- glucan) which, as a component of oats and barley, can be incorporated into breakfast cereals and other products.

Further, Maki *et al* (2007) studied the effects of consuming foods containing oat β-glucan on carbohydrate homeostasis and biomarkers of oxidative stress. Changes from baseline to week 12 in mean peak insulin and incremental area under the insulin curve differed significantly between groups (P=0.037 and 0.034, respectively), with the β-glucan group showing declines and the control group remaining essentially unchanged. Blood pressure responses were not significantly different between groups overall. it was concluded that trial suggest beneficial effects of foods containing β-glucan from oats on carbohydrate metabolism in subjects.

Depression of the glycemic index by high levels of β-glucan fiber in two functional foods tested in type 2 diabetes was explored by Jenkins *et al* (2002) to determine the extent to which β-glucan reduces the glycemic index (GI) of oat products and whether high levels of β-glucan impair palatability. Sixteen volunteers with type 2 diabetes (10 men, six women, 612 y, body mass index 292kg=m2, HbA1c 7.40.4%) were recruited and in random order, 50 g available carbohydrate portions of: white bread; a commercial oat bran breakfast cereal (4.4g% b-glucan); and a prototype b-glucan-enriched breakfast cereal and bar, both high in b-glucan (8.1 and 6.5g% b-glucan, respectively) and sweetened with fructose. Capillary blood samples were taken fasting and then 30, 60, 90, 120, 150 and 180 min after the start of the meal. Palatability was recorded. The glycemic indices of the prototype b-glucan cereal (means.e.m.; 52±5) and b-glucan bar (43±4.1) were significantly lower than the commercial oat bran breakfast cereal (86±6) and white bread (100; P <0.05). All foods were highly palatable and not significantly different. It was found that the GI of the test foods used in this study decreased by 4.0±0.2 units per gram of b-glucan compared to estimate of 3.8±0.6. It was concluded that addition of β-glucan predictably reduces the GI while maintaining palatability. In a 50 g carbohydrate portion each gram of β-glucan reduces the GI by 4 units, making it a useful functional food component for reducing postprandial glycemia.

CONCLUSION

Researches results discussed above showed that beta-glucan is a component which posses nutraceutical properties in foods like barley, oats, etc. can be promoted as an alternative of medicine for diabetic patients. With proper exercise, life style changes and dietary habit diabetes can be managed.

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