



Utilization of *Amorphophallus Oncophyllus* for Decreasing Blood Sugar on Hyperglycemic Rat

Mardiah¹, Siti Irma Rahmawati^{1,2}

¹ Magister of Food Technology, Graduate School of Djuanda University, Bogor, Indonesia

² Research Center for Biotechnology, Indonesian Institute of Sciences, Cibinong, Indonesia

Abstract:

There are many significant achievements in treatment and preventive measures, the prevalence on diabetes has risen exponentially. An increasing number of people are using dietary and herbal supplements. A glucomannan has been purified partially from *Amorphophallus oncophyllus* (in Indonesia called iles-iles or porang) using combination between alcohol 95% and heating at 55°C. Twenty rats (170-210 gram) were fed stock diet for one week and induced with alloxan 20 mg/kg weight to become hyperglycemic rats. After treated with alloxan, blood sugar of those rats was increased to become around 400 mg/dl to 600 mg/dl. Four groups of five animals were then fed one of the three diets: basal; basal with glucomannan 2.5%; basal with glucomannan 5%; and basal with glucomannan 10% for three weeks period. After 15 days feeding with glucomannan the blood sugar of those rats was decreased from 492 mg/dl and became around 250 mg/dl compared to the rats without feeding glucomannan which still high (470 mg/dl). The effect was also shown on the amount of langerhans island and β cells which were 15.7; 33.3; 37.3 and 45.7 for 0; 2.5%; 5% and 10% adding glucomannan respectively.

Keywords: *Amorphophallus oncophyllus*, Glucomannan, Blood sugar, Hyperglycemic

INTRODUCTION

Diabetes is a major health problem in North America reaching epidemic proportions. In the past decade, the United States has seen a dramatic 33% rise in diabetes coupled to increases in obesity and inappropriate lifestyle [1,2]. This increase in diabetes has occurred in spite of major inroads in understanding the pathophysiology and treatment of this insidious disease. Current therapies seem to be insufficient to prevent diabetic complications in type 2 diabetes, with a two- to fourfold likelihood for developing cardiovascular events [3]. Because of these limitations, there is a continuous need for the development of novel health promotion strategies and therapeutic modalities.

Dietary fiber, although not always defined as such, has been consumed for centuries and has been recognized as having health benefits. Fiber intake through the consumption of foods rich in this dietary component, such as fresh vegetables, fruits, whole grains, and nuts, is associated with reductions in plasma and LDL-cholesterol, attenuating glycemic and insulin response, increasing stool bulk, and improving laxation [4].

Glucomannan is a neutral polysaccharide that is extracted from the tuber of *Amorphophallus* sp. and consists of β -1,4-linked D-mannose and D-glucose. Glucomannan is regarded as noncalorie food in, and one of the primary benefits is the content of indigestible dietary fiber, the role of which has been demonstrated in weight reduction, modification of carbohydrate metabolism in diabetics, and cholesterol reduction. There is a long history of *Amorphophallus* tubers being used as a cure for certain diseases in China and Japan [5].

EXPERIMENTAL SECTION

Materials:

The tubers of *Amorphophallus oncophyllus* was bought from Saradan East Java. *Sprague-Dawley* rats were

bought from Faculty of Veterinary-IPB. Alloxan was bought from Pharmacia. The slices of tubers was then powdered and further purified by dispersed in distilled water (30 ml/gram) with heated at 50°C with stirring at room temperature for 1 h. Solution than mixed with two times weight of 96 wt % ethanol and then drying using cabinet drier at 60°C for 48h.

Sixteen male *Sprague-Dawley* rats were obtained at 3–4 weeks of age and randomly assigned to four cages (six rats per cage). The animals were maintained under controlled environmental conditions and fed a stock laboratory rodent diet for 1 week. Those rats were starved for 1 day and induced with alloxan (80 mg/kg body weight) to made it as hyperglycemic rats. Each cage was then assigned to one diet (basal, glucomannan (2.5%, 5% or 10 %)) for 16 days period and were given food and water daily, made available *ad libitum*. Body weights and blood sugar were recorded every 3 days. At the end of the experimental period, all animals were killed. The pancreas was immediately removed from each animal and cut mid-way to separate the proximal and distal ends. Samples were immediately frozen and stored at -20°C until analysis. 1 cm long regions of the pancreas tissues were taken, slit longitudinally and fixed in 10% neutral buffered formalin. The tissue was dehydrated by washing in a series of alcohol dilutions, embedded in paraffin wax and 5 μm through sections were cut. Two adjacent sections per slide were obtained for apoptosis analysis.

RESULT AND DISCUSSION

Rats made hyperglycemic by intravenous injections of Alloxan were killed after 16 days, and the pancreas, was examined histologically. The marked lesions were found in the islets of Langerhans of the pancreas. In animals there was degranulation and shrinkage of the β -cells, and the β -cells were reduced in numbers (Fig. 1 on the left panel). Cytoplasmic vacuolation was obvious, and though

some glycogen was demonstrable in the cells. Quantitative and qualitative abnormalities of β -cell function precede the development of type 2 diabetes. Once established, type 2 diabetes is characterized by diminished insulin secretion, decreased β -cell mass and proteinaceous infiltration of islets with amyloid deposits [6]. In figure 1 on the right panel shown that there were significant improvements in increasing number of β -cells after treated by feeding glucomannan.

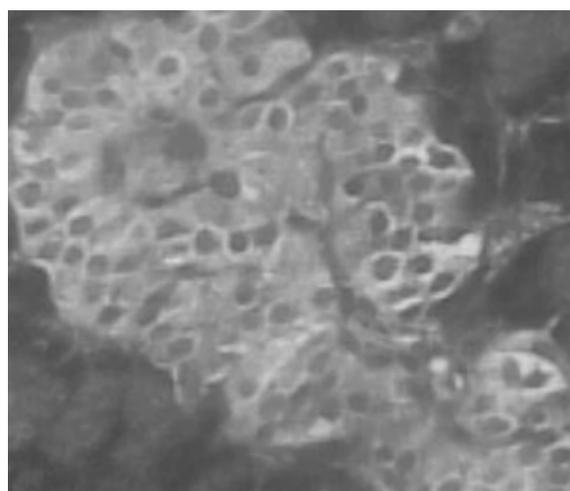
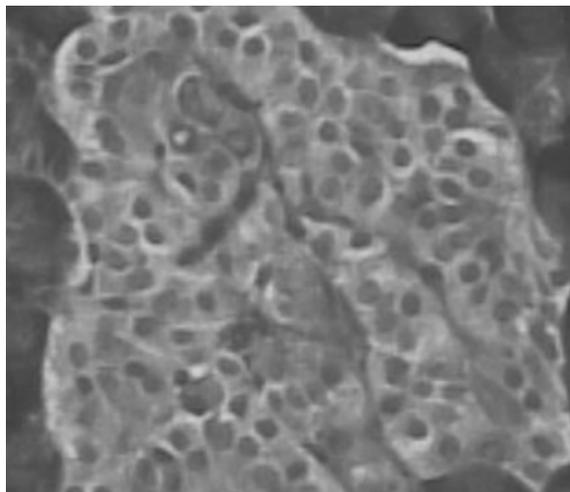


Figure 1. A figure of a haematoxylin and eosin specimen of the pancreas taken from a hyperglycemic rat (Sprague dawley). The left panel shows control hyaline degeneration of the islands of Langerhans and the right panel was specimen of hyperglycaemic rat after feeding with 10% glucomannan.

In Alloxan diabetic rats, All of those group of mice after injected with Alloxan have high blood sugar level (above 200 mg/dl). After oral administrations of the glucomannan in doses of 0.5 g (2.5%); 1g (5%) and 2g (10%) for sixteen days caused significant reduction in blood sugar levels. The glucose level started decreasing from the third day and continued up to seventh day after drugs administration, as shown on figure 2. It seems that

A. oncophyllus has antihyperglycaemic activity in Alloxan induced diabetic rats.

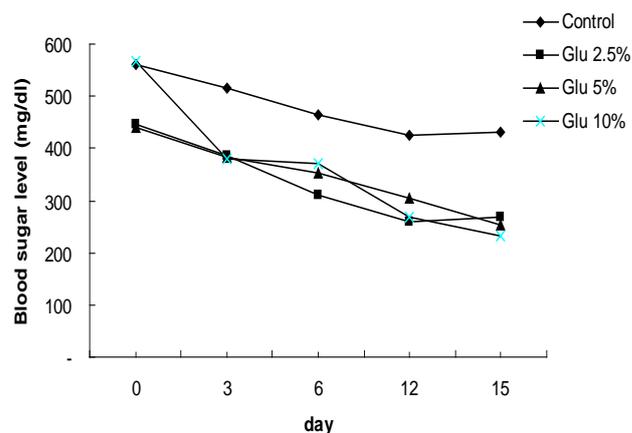


Figure 2. Graphics of blood sugar level on 4 groups of mice which injected with Alloxan. Those mice were than treated with 0%, 2.5%, 5% and 10% glucomannan

Table 1. Table of blood sugar level on 4 groups of mice which injected with Alloxan. Those mice were than treated with 0%, 2.5%, 5% and 10% glucomannan

Group	Blood sugar level (Mg/dl)				
	day				
	0	3	6	12	15
Control	560	517	464	426	430
TG 2,5%	447	387	311	259	270
TG 5%	440	383	352	306	254
TG 10%	566	379	370	267	232

TG : Glucmannan powder

The mechanism of glucomannan improves metabolic control and it is hypothesized that the gel forming glucomannan, increases the viscosity of the digesta slowing the rate of food absorption in the small intestine, thereby decreasing postprandial glucose and insulin surges. This in turn may result in a long-term improvement in peripheral insulin sensitivity. The attenuated increased insulin sensitivity [7] might reduce blood pressure by influencing sodium absorption in the distal tubule, increasing sympathetic nervous system activity and decreasing peripheral vascular resistance [8]. Such an improvement in insulin sensitivity might have been mediated by sustained slowed absorption during the glucomannan treatment. The mechanisms for each and their long-term effects are areas requiring more study. Other avenues of investigation include exploring ways to enhance the metabolic effects of glucomannan through modulation of its rheological characteristics, development of new products, and initiation of longer-term studies.

Finally, the isolation and optimization of active components from *Amorophallus* sp. specific to various physiologic variables will provide much interesting work for years to come.

CONCLUSIONS

Our preliminary data indicate that glucomannan from *Amorpophallus oncophyllus* may have therapeutic promise in the treatment of diabetes. These shows by increasing the amount of β -cell in langerhans island and decreasing of blood sugar level after treated by feeding glucomannan on hyperglycaemic rats.

ACKNOWLEDGEMENT

This research is supported by grant from Direktorat Jenderal Pendidikan Tinggi (DIKTI) through Hibah Fundamental fund. Also, the authors are thankful to Dr. Reki Wicaksono Ashadi, M.Agr. for his guidance during the research.

REFERENCES

- [1] Morkdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS. 2000. Diabetes trends in the U.S.: 1990–1998. *Diabetes Care* 23: 1278–1283.
- [2] Sorensen, TI. 2000. The changing of lifestyle of the world. *Diabetes Care* 23(Suppl 2): B1–B24.
- [3] Haffner SM, Lehto S, Ronnema T, Pyorala K, Laakso M. 1998. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med* 339: 229–234.
- [4] Schneeman BO. 1999. Fiber, inulin and oligofructose: similarities and differences. *J Nutr* 129:1424S-1427S.
- [5] Vladimir V., Sievenpiper, J.L., Zheng Xu., Evelyn Y. Y. W., Alexandra L. J., Uljana Beljan Z., Lawrence A. L., Robert G. J., and Mark P. S. 2001. Konjac-Mannan and American Ginseng: Emerging Alternative Therapies for Type 2 Diabetes Mellitus. *Journal of the American College of Nutrition*, Vol. 20, No. 90005, 370S-380S
- [6] Dornhorst, A. 2001. Abnormalities of b-cell function in the development and progression of type 2 diabetes. *Pract Diab Int January/February 2001 Vol. 18 No. 1 Supplement*.
- [7] Anderson JW, Tietzen-Clark J. 1986. Dietary fiber: hyperlipidemia, hypertension, and coronary heart disease. *Am J Gastroenterol* 81: 907–919, 1986.
- [8] Modan M, Halkin H, Almog S, Lusky A, Eshkol A, Shefi M, Shitrit A, Fuchs Z. 1985. Hyperinsulinemia. A link between hypertension, obesity and glucose intolerance. *J Clin Invest* 75: 809–817.