Effects of Two Pre-germinated Brown Rice of Thailand on Postprandial Blood Glucose and Insulin Responses in Type 2 Diabetes

Jariya Boonpattararuksa[1], Sriwatana Songchitsomboon[1,*], Pakinee Akkaravessapong[2], SunantaWongpiyachon[3], Angsutorn Wasusun[2], Surat Komindr[4]

Abstract

Effect of two pre-germinated brown rice (PGBR) of KhaoDawkMali105 (KDML105) and glutinous rice RD6 (GRD6) on postprandial blood glucose, insulin and lipid concentrations were compared with glucose solution (50 g in 150 mL of drinking water) in 11 subjects with type 2 diabetes. All rice samples contained

50 g of digestible carbohydrates. The study was single-blind, randomized block design in which subjects participated in three separate 4 hour meal tolerance test. On test day, they ingested each test rice sample with steamed garlic-chicken, following 150 mL of drinking water in 15 min. Venous blood sample were collected in fasting subjects and at 30, 60, 90, 120, 180 and 240 min after starting to eat. The incremental areas under the curve (IAUC) of postprandial plasma glucose (PPG) and serum insulin response, glycemic index (GI) and insulin index (II) values were determined. Results showed that PGBR-KDML105 had mean values of PPG at 30and 60 min significantly lower ($p<0.05$) than glucose at the same time whereas PGBR-GRD6 had mean values of PPG only at 60 min significantly lower ($p<0.05$) than glucose. Mean value of IAUC-glucose of PGBR-KDML105 were also lesser than PGBR-GRD6 but not significant. PGBR-KDML105 had a medium GI = 66.0, while PGBR- GRD6 had a high GI = 71.5 which not significantly different from PGBR-KDML105 but significantly lower ($p<0.05$) than glucose. There were no significant differences in mean values of IAUC-insulin, II and lipid profiles among two PGBR and glucose. In conclusion, consumption of PGBR-KDML105 will be more health benefits than PGBR-GRD6.

Keywords: Pre-germinated brown rice, glycemic index, type 2 diabetes, glucose, insulin

[1] Postgraduate Program of Nutrition, Faculty of Medicine at Ramathibodi Hospital, Mahidol University, BKK10400, Thailand

[2] Bureau of Rice Research and Development, Rice Department, Ministry of Agriculture and Cooperatives, BKK10900, Thailand

[3] Pathumthani Rice Research Center, Bureau of Rice Research and Development, Rice Department, Ministry of Agriculture and Cooperatives, Pathumthani12110, Thailand

[4] Division of Nutrition and Biochemical Medicine, Department of Medicine, Faculty of Medicine at Ramathibodi Hospital, Mahidol University, BKK10400, Thailand

* Corresponding author, e-mail: sriwatana.son@mahidol.ac.th

1. Introduction

Diabetes mellitus is a global health problem that its prevalence is projected to double between 2010 and 2030 (Shaw *et al*., 2010). For Thailand, Bureau of Non-Communicable Diseases (2009 and 2010), Ministry of Public Health found that the prevalence of diabetes in adults (aged >15 years) was 6.9 % (3.46 million people) and in 2011 death rates with diabetes estimated 7,625 people or 19 people a day. The development of type 2 diabetes (T2D) is caused by processes of destruction of the beta cells in the pancreas, characterized by insulin resistance which may be combined with relatively reduced insulin secretion that are causes of abnormally high glucose levels in the blood as hyperglycemia (Prato *et al*.,2002). The long-term complications include retinopathy, nephropathy, neuropathy, stroke and cardiovascular disease which are a major cause of death in individuals with diabetes (Grundy et al., 1999; Sowers

et al., 2001; Haffner *et al*., 1998). Thus, a goal in the management of diabetes is the regulation of blood glucose levels to achieve near-normal blood glucose, since improved blood glucose control is associated with reduction in development, and progression, of complications (Sheard *et al*., 2004). Especially, postprandial glucose control diet is necessary for these patients.

The component of the diet that has the most influence on blood glucose is carbohydrate. Both the quantity and type or source of carbohydrate, include type of starch (amylose versus amylopectin) found in foods influence postprandial glucose level (Franz et al., 2002). For Thai people**,** rice (*Oryza sativa* L) is the main staple food that its carbohydrate contributed 50 to 60% of total energy intake. They consumed rice and other starch products (noodles, bread, taro, potato etc.) averaged 281 and 25 g/day, respectively (the IV National Food and Nutrition Survey, 1995). A recent prospective cohort study of Sun Q et al., (2010) found that people who ate white rice $\geq$ 5 servings/week have higher risk of T2D than who ate brown rice $\geq$ 2 servings/week among US adults. Meta-analysis and systematic review of Hu EA *et al*., (2012) showed clear relationships between higher white rice intake and risk of T2D in Asian populations that stronger than in western population. Therefore, both quantity and quality of rice is important for Thai people especial with T2D.

The glycemic index or GI (Jenkins et al., 1981) was used to rank carbohydrate exchanges according to their effect on postprandial plasma glucose. A lower glycemic index food suggests slower rate of digestion and absorption of carbohydrate in the food. So, consumption of low-GI rice may reduce incidence or slowing the progression of T2D. The wide variability in the GI of rice has been attributed to many factors such as the amylose content, degree of cooking & gelatinization, fiber content, post-harvest treatments (i.e. parboiling), physical size & form and the presence of fats & proteins (Panlasigui *et al*., 1991; Brand-Miller

et al., 1992; Wolever *et al*., 2006; Jenkins *et al*., 1988; Foster-Powell *et al*., 2002; Thorne *et al*., 1983; Owen *et al*., 2003). However, brown rice is not a popular choice for consumer because of harder to chew and not as tasty as white rice. Pre-germinated brown rice (PGBR) firstly innovative in Japan, produced by soaking brown rice (BR) in water to induce slight germination, the outer bran layer becomes soft and more prone to water absorption, making it easier to cook, overcame the problem which can be cooked and soft enough to chew even for children. Moreover, during germination, many nutrients in BR were increased in the bran layer and embryo such as GABA (gamma-aminobutyric acid), dietary fiber, vitamins and minerals (Kayahara *et al*., 2000a, 2004b; Banchuen *et al*., 2010). GABA is an amino acid decarboxylation derivative and a neurotransmitter in the brain.

The objectives of this study were to investigate postprandial blood glucose and insulin concentrationsin patients with T2D after ingesting two types of PGBR of KhaoDawkMali105 (KDML105) from Surin and glutinous rice RD6 (GRD6) from Chiang Mai province and to determine its glycemic index and insulin index. These two types of rice were selected because of popular among health-conscious consumers. From our knowledge, glycemic index of both type of rice have not been evaluated.

2. Materials and Methods

2.1 Participants

It is recommended that selection of a minimum of 10 subjects shall be recruited to determine GI of food [ISO 26642:2010(E)]. Eleven participants (both males and females) with T2D, aged 35–70 years, BMI < 35 kg/m^2 were recruited from the outpatient nutrition clinic at Ramathibodi Hospital for this study. Inclusion criteria were fasting plasma glucose (FPG) $\geq$ 126 and <180 mg/dL, not on insulin injection, not pregnant, not lactating, not change in drug and dose in 3-months before study. Participants had normal liver and kidney function. Exclusion criteria were a history or present of uncontrolled hypertension, cardiovascular disease, cancer, kidney disease, chronic liver disease and anemia or thyroid dysfunction; and/or use of medication affecting body weight within the previous 3 months. Ethical approval of the study was obtained from the Committee on Human Rights Related to Researches Involving Human Subjects, Faculty of Medicine, Ramathibodi Hospital, Mahidol University. All subjects gave written informed consent before participation.

2.2 Study design

This study is single-blind, randomized block design, controlled trial. On the morning of the study, all participants were come to the research unit at 7.00 am, after 8–10 hours of overnight fasting and withheld their drugs. Fasting and postprandial plasma glucose, serum insulin and lipid profile were collected for analysis at 0, 30, 60, 90, 120, 180, and 240 min.

On the day before test day, all participants ate same standard menu. Throughout the study, participants were instructed on weight-maintaining diabetes exchange diet. They were asked to not change their physical activity pattern. Their total energy intakes based on Harris-Benedict Equation and distribution of energy intake are 20% of protein, 30% of fat and 50% of carbohydrates.

2.3 Test articles and test meals

All 2 pre-germinated brown rice will be prepared and packed under vacuum in plastic bags from Bureau of Rice Research Center and Development in Pathumthani. All vacuum bags ofbrown rice samples were kept in cold room (4°C) until study. The nutrient composition of the test meals was shown in Table 1. Each test meal contained PGBR samples equivalent to 50 g of carbohydrates or glucose solution (50 g glucose dissolves in 150 mL water), consumed with precise weight of steamed garlic-chicken. All test meals were prepared in the metabolic kitchen of the Division of Nutrition and Dietetics at Ramathibodi Hospital. Only after eating PGBR meals, participants were allowed to drink 150 mL of water. They were requested to finish their test meals in approximately 15 minutes.

Table 1 The nutrient composition of rice sample for test meals*

	PGBR-KhaoDawk Mali105†	**PGBR-glutinous rice RD6**
	unit/100 g dry wt rice	
Energy, kcal	357.08	352.78
Moisture, %	9.47	9.98
Ash, g	1.27	1.30
Protein, g	7.56	7.69
Fat, g	2.56	2.78
Total digestible Carbohydrate, g	75.95	74.25
Dietary fiber, g	3.09	4.00
Amylose, %	15.32	5.1

Note: *Source: Bureau of Rice Research Center and Development in Pathumthani

†PGBR; pre-germinated brown rice

2.4 Biochemical analysis

Blood sample was collected in the sodium fluoride tube (NaF) for determining plasma glucose. A clotted blood sample was collected for determining serum total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and insulin. All blood samples were immediately kept in an icebox after being drawn. Serum and plasma were frozen at -70˚C until analysis. Plasma glucose and lipid profile were performed on the Dimension© Clinical Chemistry System of Dade Behring Company. Serum insulin was analyzed by using an enzyme-linked immunosorbent assay technique. Anthropometric measurements and blood pressure were made in the fasting state.

2.5 Calculation of glycemic index and insulin index

Glycemic index (GI) was calculated from the incremental area under the curve (IAUC) of blood glucose response to 50 g of carbohydrate in test food, relative to the IAUC of blood glucose response to 50 g glucose as reference, taken by the same subject.

The IAUC of glucose and insulin response were calculated according to the method of Wolever and Jenkins (1986) ignoring values beneath basal level. The GI of each test food was calculated as followed. The insulin index (II) was calculated as same as GI.

$$GI = \frac{\text{IAUC of blood glucose response after eating food containing 50g CHO}}{\text{IAUC of blood glucose response after drinking 50g glucosessolution}} \times 100$$

2.6 Statistical Analysis

All data were presented as means ± SE. Comparison between time points were assessed using a general linear model (repeated measures analysis of Varian), compared main effects by Bonferroni and one-way analysis of variance (ANOVA) was used to compare IAUC values and GI between 2 types of test rice and glucose solution, post-hoc multiple comparisons carried out by Bonferroni using the Statistical Package for Social Science (SPSS) software for Windows version 17.0. Statistical significance was established at $p<0.05$.

3. Results and Discussion

A total of 11 participants (5 females, 6 males) were recruited for the study. Participant baseline characteristics are showed in the Table 2. The means (±SE) of age, body mass index and fasting plasma glucose (FPG) of participants were 58.1±2.9 years, 25.3±1.3 kg/m^2 and 138.8±4.4 mg/dL, respectively and had normal lipid levels. After ingestion, postprandial plasma glucose levels increased and reached the peak at 60 min and decrease gradually in all test meals. The PGBR-KDML105 had mean values of PPG at 30 and 60 min significantly lower

(p<0.05) than glucose at the same time whereas PGBR-GRD6 had mean values of PPG only at 60 min significantly lower (p<0.05) than glucose (Figure 1a). There were no significant differences in postprandial serum insulin among two PGBR and glucose at any time point (Figure 1b). Both PGBR-KDML105 and GRD6 had mean IAUC-glucose and GI significantly lower than glucose. Mean value of IAUC-glucose of PGBR-KDML105 were also lesser than PGBR-GRD6 but not significant (9,299.3±927.5 and 66.0±4.6 vs 9,998.2±925.5 and 71.5± 4.7, respectively). Significant differences in mean values of IAUC-insulin and then insulin index were not found (Table 3). There were no significant differences in postprandial TG including total cholesterol, HDL- C and LDL-C among two PGBR and glucose too (Figure 2).

Table 2 Baseline characteristics of the subjects

Parameter	Subject (n = 11)		
	Mean		SE
Age, year	58.1	±	2.9
Height, cm	1.62	±	0.02
Body weight, kg	68.0	±	5.1
BMI, kg/m^2	25.3	±	1.3
Body fat, %	26.0	±	1.9
Waist circumference, cm	88.3	±	4.2
Hip circumference, cm	95.3	±	3.2
Systolic BP, mmHg	124.7	±	4.4
Diastolic BP, mmHg	76.6	±	2.8
FPG, mg/dL	138.8	±	4.4
Serum insulin, µIU/mL	10.9	±	0.9
Total cholesterol, mg/dL	176.8	±	4.4
HDL-cholesterol, mg/dL	51.4	±	1.6
LDL-cholesterol, mg/dL	98.1	±	4.0
Triglyceride, mg/dL	136.6	±	8.1

Table 3 Means ± SE of the incremental area under the curve of plasma glucose(IAUC-glucose) and insulin response (IAUC-insulin), glycemic index (GI) and insulin index (II)

Type of rice	IAUC-glucose mg.hr/dL	GI	IAUC-insulin μIU.hr/mL	II
Glucose	13808.2 ± 522.4	100	4397.2 ± 782.6	100
PGBR-GRD6	9998.2 ± 925.5*	71.5 ± 4.7*	4270.1 ± 783.3	99.8 ± 14.2
PGBR-KDML105	9299.3 ± 927.5*	66.0 ± 4.6*	4244.4 ± 885.2	95.2 ± 8.2

Note: *Significantly different from glucose, $p<0.05$

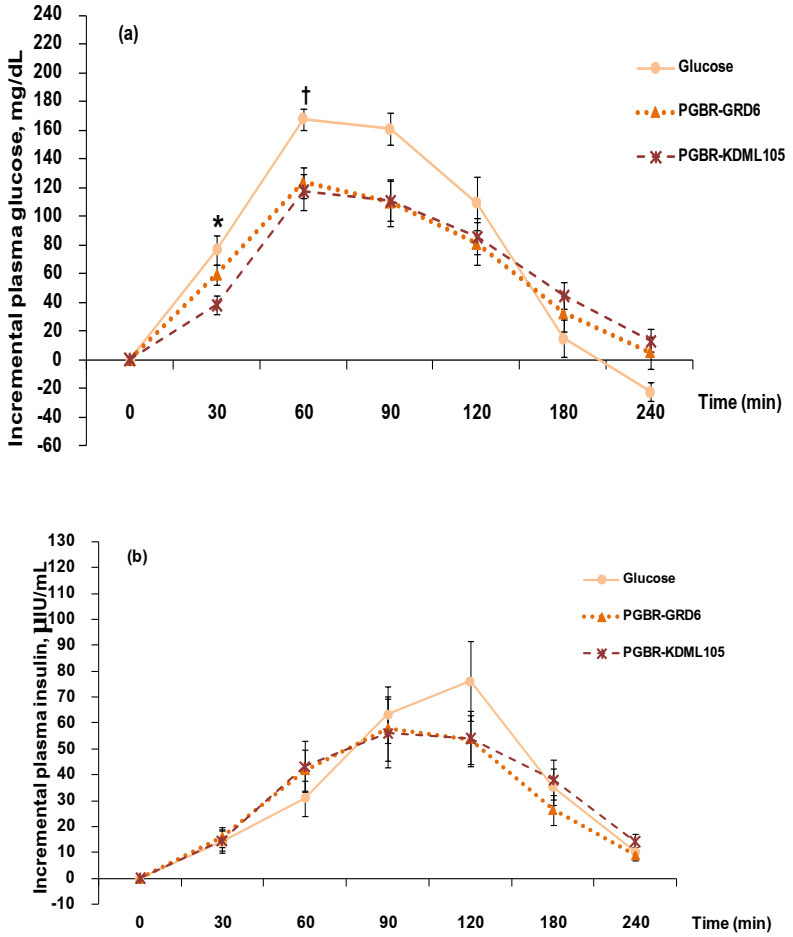

Figure 1 Means ± SE of incremental plasma glucose (a) and plasma insulin response (b) after ingestion PGBR-KhaoDawk Mali105, PGBR-glutinous RD6 and glucose solution

Note: *Significantly different from PGBR- KhaoDawk Mali105 (KDML105), $p<0.05$

†Significantly different from PGBR- KhaoDawk Mali105 and PGBR-glutinous RD6 (GRD6), $p<0.05$

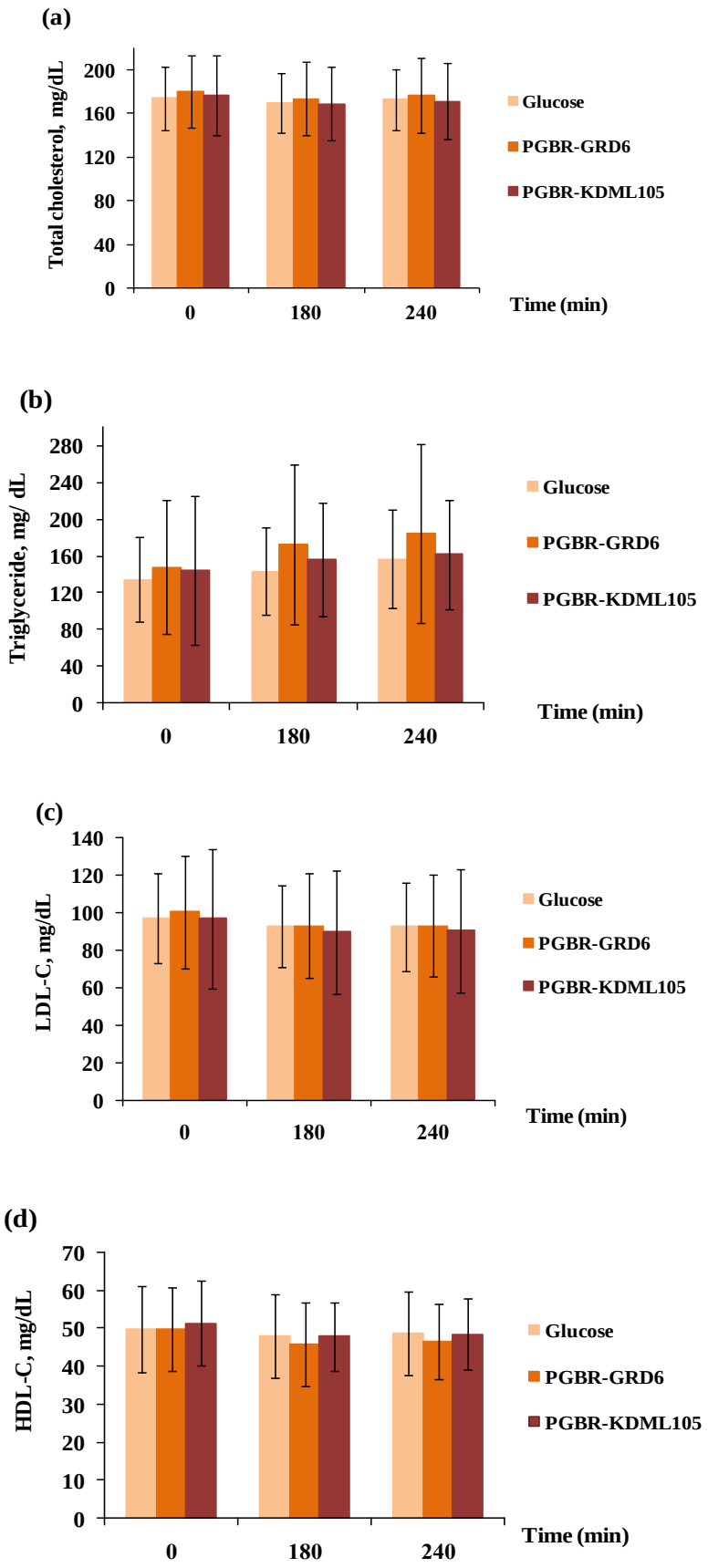

Figure 2 Means ± SE of serum total cholesterol (a), triglyceride (b), LDL-cholesterol (c) and HDL-cholesterol (d) at baseline, 180 and 240 min after ingestion of the test meals

Many epidemiological studies showed that postprandial glycemic control is important in avoiding microvascular and macrovascular complications (Klein, 1995; Adler *et al.,* 1997; Groeneveld *et al*., 1999; Stratton *et al*., 2000 and Ceriello *et al.,* 2004). Database from DECODE study (1999) showed that an increase of 1.0 mmol/L in postprandial blood glucose resulted in a 7 % increase in total mortality over a 5 to 10 year period and abnormalities in 2 hour glucose levels were better predictors of all causes mortality than fasting glucose alone. Several epidemiological studies also demonstrated an association between hyperinsulinemia and coronary heart disease (CHD) (Fontbonne *et al*., 1991; McKeigue *et al.*, 1993 and Despres *et al.,* 1996). So, results from this study suggested that consumption of PGBR-KDML105 is more health benefits for patients with diabetes than PGBR-GRD6 because it raised IAUC of glucose and insulin lesser although data not statistical significantly different. Postprandial lipidemia after ingestion of studied PGBR were evaluated too because increased postprandial triglyceride levels is associated with the development of atherosclerosis and macrovascular complications (Ahmad et al., 2005; Teno et al., 2000 and Noedestgaard et al., 2007). However, there were no significant differences in postprandial lipid profiles between two types of rice.

Trinidad et al (2012) found that Sinandomeng with the lowest amylose content (AC) had a high GI = 75, while PSBRc10 with the highest AC had a low GI = 50. Sinandomeng with a low dietary fiber (DF) had GI = 75, while its brown rice had GI = 55 which suggested that AC and DF content of rice have significant role on GI of cooked milled and brown rice varieties. Moreover, several studies also showed an inverse relationship between the amylose content and glucose response (Goddard *et al*., 1984; Behall et al., 1988), since amylose was more slowly digested and absorbed than amylopectin. This fact could explained why PGBR of GRD6 with lower AC (5%) had GI value greater than PGBR of KDML105 (AC =15%). Limited data exist on the GI values of PGBR other than white rice and brown rice. Therefore the values reported in this study are the first report of PGBR planted in Thailand. From our study, PGBR of KDML105 had medium GI = 66 and PGBR of GRD6 had high GI which higher than PGBR of Japonica variety reported by Ito et al., 2005 (GI values of 56.9 and 54.4). To our knowledge, this is only one study that had been published.

Hsu et al (2008) reported that after intake of PGBR of Japonica (from the study of Ito) for 6 weeks, patients with impaired fasting glucose or T2D had fasting blood glucose, fructosamine, serum total cholesterol and triacylglycerol levels favorably improved (p<0.01), but not on white rice diet (crossover study). In addition, Hagiwara et al (2004) reported that feeding streptozotocin-induced diabetic rats with PGBR caused significantly improved glycemia after 7 weeks and reduced levels of PAI-1, triglycerides, and lipid peroxides had correlated well with amounts of dietary fiber in the PGBR. Because of many previous studies (Fengfeng *et al*.,

2013) including the aforesaid studies reported that pre-germinated brown rice play an important role in preventing some chronic disease such as diabetes, cardiovascular disease, Alzheimer's disease, and cancer, etc. Therefore, further studies are need for development of Thai pre-germinated brown rice with low GI (<55) for Thai people and also people around the world since Thailand is the world's leading rice exporter.

4. Conclusion

In conclusion, the study in type 2 diabetic subjects found that PGBR KhaoDawk Mali105 had medium GI = 66 while PGBR of glutinous rice RD6 had high GI = 71.5. This information could help diabetic and health-conscious consumers to make a good choice for their health.

Acknowledgements

This study was supported by grant from Bureau of Rice Research and Development, Rice Department, Ministry of Agriculture and Cooperatives, Thailand

References

Adler, A.I., E.J. Ahroni, V. Stensel, R.C. Forsberg, and D.G. Smith. 1997. Risk factors for diabetic peripheral sensory neuropathy. Results of the Seattle prospective diabetic foot study. Diabetes Care 20:1162–7.

ANNUAL report 2008.Bureau of Non-Communicable Diseases, Department of Disease Control Ministry of Publc Health. http;//www.ncd.ddc.moph.go.th

Ahmad, J., B. Hameed, G. Das, M.A. Siddiqui, and I. Ahmad. 2005. Postprandial hypertriglyceridemia and carotid intima-media thickness in north Indian type 2 diabetic subjects. Diabetes Res ClinPract 69: 142–50.

Banchuen, J., P. Thammarutwasik, B. Ooraikul, P. Wuttijumnong, and P. Sirivongpaisal. 2010. Increasing the bio-active conpounds contents by optimizing the germination conditions of Southern Thai Brown Rice. SongklanakarinJ.Sci.Technol 32(3): 219–230.

Behall, K.M., D.J. Scholfield, and J. Canary.1988.Effect of starch structure on glucose and insulin responses in adults.Am J ClinNutr 47:428–432.

Brand-Miller, J.C., E. Pang, and L. Bamall.1992. Rice: A high or low glycaemic index food. Am J ClinNutr 56: 1034–1036.

Ceriello, A., M. Hanefeld, L. Leiter, L. Monnier, A. Moses, and D. Owens.2004. Postprandial Glucose Requlation and Diabetic Complications. Arch Inter Med 164:2090–5.

Despres, J.P., P. Lamarche, P. Mauriege, B. Cantin, G.R. Dagenais, et al. 1996. Hyperinsulinemia as an independent risk factor for ischemic heart disease.N ENGL J MED 334:952–7.

Fengfeng, W.U., Y. Na, T. Alhassane, J. Zhengyu, and X. Xueming. 2013. Germinated brown rice and Its role in human health. Critical Reviews in Food Science and Nutrition 53(5): 451–463.

Fontbonne, A., M.A. Charles, N. Thibult, J.L. Richard, J.R. Claude, et al. 1991. Hyperinsulinemia as a predictor of coronary heart disease mortality in healthy populations: the Paris Prospective Study, 15 year follow-up. Diabetologia 34:356–61.

Foster-Powell, K., S.H.A. Holt, and J.C. Brand-Miller. 2005. International table of glycaemic index and glycaemic load values: 2002. Am J ClinNutr 76: 5–56.

Franz, M.J., J.P. Bantle, C.A. Beebe, J.D. Brunzell, J.L. Chiasson, and A. Garg. 2002. Evidencebased nutrition principles and recommendation for the treatment and prevention of diabetes and related complications. Diabetes Care 25:148–98.

Goddard, M.S., M.S.G. Young, and R. Marcus.1984.The effect of amylose content on insulin and glucose responses to ingested rice.Am J ClinNutr 39: 388–392.

Groeneveld, Y., H. Petri, J. Hermans, and M.P. Springer. 1999. Relationship between blood glucose level and mortality in type 2 diabetes mellitus: a systematic review. Diabet Med 116:2–13.

Grundy, S.M., I.J. Benjamin, G.L. Burke, A. Chait, R.H. Eckel, and B.V. Howard.1999. Diabetes and cardiovascular disease: a statement for healthcare professionals from The American Heart Association Circulation 100: 1134–46.

Haffner, S.M. 1998. Management of dyslipidemia in adults with diabetes. Diabetes Care 21: 160–78.

Hagiwara, H., T. Seki, and T. Ariga. 2004. The effect of pregerminated brown rice intake on blood glucose and PAI-1 levels in streptozotocin-induced diabetic rats. Bioscience, Biotechnology and Biochemistry 68(2): 444–447.

Hu, E.A., A. Pan, V. Malik, and Q. Sun. 2012.White rice consumption and risk of type diabetes: meta-analysis and systematic review. Br Med 344: e1454.

Hsu, T.F., M. Kise, M.F. Wang, Y. Ito, M.D. Yang, and H. Aoto. 2008. Effects of Pre-germinated brown rice on blood glucose and lipid levels in free-living patients with impaired fasting glucose or type 2 diabetes. J NutrSciVitaminol 54: 163–168.

Ito, Y., A. Mizukuchi, M. Kise, H. Aoto, S. Yamamot, and R. Yoshihara. 2005. Postprandial blood glucose and insulin responses to pre-germinated brown rice in healthy subjects. J Med Invest 52(3-4): 159–64.

Jenkins, D.J.A., T.M.S. Wolever, R.H. Taylor, M.R.C.P.H. Barker, and S.R.D.H. Fielden. 1981. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J ClinNutr 34: 362–6.

Jenkins, D.J.A., T.M.S. Wolever, andA.L. Jenkins.1988. Starchy foods and glycaemic index. Diabetes Care 11: 149–159.

Kayahara, H., and K. Tukahara. 2000. Flavor, health and nutritional quality of pre-germinated brown rice. In Proceedings of the International Chemical Congress of Pacific Basin Societies in Hawaii 2000.

Kayahara, H. 2004. Germinated brown rice.In Proceedings of the Department of sciences of Functional Foods.Shinshu University, Japan.

Klein, R. 1995. Hyperglycemia and microvascular and macrovascular disease in diabetes. Diabetes Care 18:258–68.

McKeigue, P.M., J.E. Ferrie, T. Pierpoint, and M.G. Marmot.1993.Association of early-onset coronary heart disease in South Aisan men with glucose intolerance and hyperinsulinemia.Circlation 87:152–61.

Nordestgaard, B.G., M. Benn, P. Schnohr, and A. Tybjaerg-Hansen. 2007. Nonfasting triglyceridees and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA 298: 299–308.

Owen, B., and T.M. Woleve. 2003. Effect of fat on glycaemic responses in natural subjects: A dose-response study. Nutr Res 23(10): 1341–1347.

Panlasigui, L.U., L.U. Thompson, B.O. Juliano, C.M. Perez, S.H. Yiu, and G.R. Greenberg. 1991. Rice varieties with similar amylose content differ in starch digestibility and glycaemic response in humans. Am J ClinNutr 54: 871–877.

Prato, S.D., P. Marchetti, and R.C. Bonadonna. 2002. Phasic insulin release and metabolic regulation in type 2 diabetes. Diabetes 51: 109–16.

Shaw, J.E., R.A. Sicree, and P.Z. Zimmet. 2010. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res ClinPract87: 4–14.

Sheard, N.F., N.G. Clark, J.C. Brand-Miller, M.J. Franz, F.X. Pi-Sunyer, and E. Mayer-Davis. 2004. Dietary Carbohydrate (Amount and Type) in the Prevention and Management of Diabetes. Diabetes Care27(9): 2266–71.

Sowers, J.R., M. Epstein, and E.D. Frohlich. 2001. Diabetes, Hypertension and Cardiovascular Disease: an update. Hypertension 37: 1053–9.

Stratton, I.M., A.I. Adler, H.A.W. Neil, D.R. Matthews, and S.E. Manley. 2000. Association of glycaemia with macrovascular and microvascular complications of type 2diabetes (UKPDS 35): prospective observational study. BMJ 321:405–12.

Sun, Q., D. Splegelmen, R.M. Van Dam, M.D. Holmes, V.S. Malik, and W.C. Willet. 2010. White rice, brown rice, and risk of type 2 diabetes in US men and women. Arch Intern Med 170(11): 961–9.

Omori, The DECODE Study Group. 1999. Glucose tolerance and mortality: comparison of WHO and American Diabetes Association diagnostic criteria. Lancet 354:617–21.

The IVNational Food and Nutrition Survey 1995. Bureau of nutrition, Department of health, Ministry Of Public Health.

The International Organization for Standardization (ISO 26642:2010(E)). Food products-Determination of the glycaemic index (GI) and recommendation for food classification.Fist edition 2010-10-01.

Thorne, M.J., L.U.Thompson, and D.J.A. Jenkins.1983. Factors affecting starch digestibility and the glycaemic response with special reference to legumes. Am J ClinNutr 38: 481–488.

Thrinidad, T.P., A.C. Mallillin, R.R. Encabo, R.S. Sagum, A.D.R. Felix, and B.O. Joliano. 2012. The effect of apparent amylose content and dietary fibre on the glycemic response of different varieties of cooked milled and brown rice. IJFNS 64(1):89–93.

Wolever, T.M.S., and D.J.A. Jenkins.1986.The use of the glycemic index in predicting the blood glucose response to mixed meals.Am J ClinNutr 43: 167–172.

Wolever, T.M.S., 2006. The glycaemic index-a physiological classification of dietary carbohydrate. Wallingford: CABI Publishers.