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Pitout JDD, Church DL, Gregson DB, Chow BL, McCracken M, Mulvey M, Laupland KB (2007). Molecular epidemiology of CTXM-producing Escherichia coli in the Calgary Health Region: emergence of CTX-M-15-producing isolates. Antimicrob. Agents Chemother. 51: 1281-1286.

Pelczar JR, Harley JP, Klein DA (1993). Microbiology: Concepts and Applications. McGraw-Hill Inc., New York, pp. 591-603.

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Full Length Research Paper

A positive role for selenium in mitigating complications associated with fructose-induced metabolic syndrome in rats

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This study was designed to investigate the potential role of selenium on metabolic syndrome induced by fructose in rats. Thirty male Wister Albino rats weighing 185 to 225 g were used. The rats were randomly divided into 3 groups, 10 rats each. The first group received water only and set as a control (GI). The second group received 20% fructose in drinking water daily (GII). Third group (GIII) received fructose 20% and 0. 25 mg/0.5 ml distilled water/kg body weight per day selenium in drinking water. After 5 weeks of supplementation, rats were sacrificed; blood samples were obtained for different biochemical analysis including serum level of glucose; lipids profile were measured including total cholesterol, high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c) and triglyceride (TG). Liver function tests, alanine transaminase (ALT), aspartate transaminase (AST), albumin and total protein were carried out. Also, kidney function tests (creatinine, urea and uric acid) were estimated in serum. The present study revealed that selenium treatment mitigate the complications associated with metabolic syndrome. It is recommended that selenium may be an important part of an individual medical nutritional and lifestyle intervention.

Key words: Fructose, metabolic syndrome, selenium, glucose, lipids profile, liver functions, kidney functions.

INTRODUCTION

Unsuitable lifestyle and diets can cause many disorders. Epidemiological studies have demonstrated that atherosclerosis is caused from several conditions such as dyslipidemia, insulin resistance and hypertension which refer to the metabolic syndrome. Increasing incidence of diabetes with obesity is as a result of changes in nutrition and the sedentary lifestyles (Eriksen et al., 2011). Metabolic syndrome is usually associated with lipoprotein abnormalities which serve as risk factors for atherosclerosis (Avramoglu et al., 2003).

Fructose is one of the most common sweetener which causes weight gain when in high dietary intake (Abdulla et al., 2011). In human, fructose when absorbed is not changed in the small intestine followed by transportation to the liver, which metabolizes in liver and the remainder taken up by the kidney and adipose tissue (Cirillo et al., 2009). Fructose-fed rats developed features of metabolic syndrome (Johnson et al., 2010). One difference between fructose and glucose is that fructose raises serum uric acid. Elevated serum level of uric acid predicts the development of obesity and hypertension. This increased the possibility that uric acid may have a strong

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pathogenic role in metabolic syndrome (Johnson et al., 2010). There is evidence that treatment with antioxidants improves insulin resistance in fructose fed rats (Roncal et al., 2009).

Selenium (Se) is a trace element, it is toxic if taken in excess, creating an imbalance of nutrients in the diet. Selenium is an important element in many biochemical and physiological processes. Selenium is incorporated as seleno-cysteine at the active site of a wide range of selenoproteins. The biochemical role for Se is as a part of the active site of the enzymes including glutathione peroxidase which is known as an endogenous antioxidant enzyme (Michiels, 1994) and thioredoxin reductase (TR) that catalyzes the NADPH- dependent reduction. TR is a major redox protein for many enzymes/transcription factors. Also, selenium possesses a number of insulin like action (Shao et al., 2002). Also, the beneficial effects of Se as an antioxidant in biological systems have been returned to its ability to stabilize biomemberanes, to scavenge reactive oxygen species, and to reduce the peroxidation of unsaturated membrane lipids (Nandhini and Anuradha, 2004).

Because of the important role of oxidative stress in the development of insulin resistance, we examined the role of antioxidant micronutrient in a model of metabolic syndrome, the fructose-fed rat. Several reports suggest that high fructose diet leads to the metabolic changes observed in metabolic syndrome such as dyslipidemia, and insulin resistance are observed which are associated with a high risk of cardiovascular diseases (Khan et al., 2012). In this present study, we intended to investigate the preventive role of selenium on high fructose induced metabolic syndrome in male Wister rats.

MATERIALS AND METHODS

Chemicals

Fructose was purchased from local markets. Selenium (Sigma-Aldrich,Castle Hill, Australia) was purchased from General Nutrition Centers (GNC) market**,** Jeddah, Saudi Arabia Shop Market. All chemicals used were of high analytical grade, product of Sigma (US), Merk (Germany) and BDH (England).

Animals

Thirty male Wister Albino rats of 12 weeks old, weighing 185 to 225 g were used at the start of experiment. The rats were purchased from King Fahad Medical Research Center in Jeddah. The Animals were kept in acrylic cages separately, and maintained on a constant 12 h light/12 h dark cycle with air conditioning and temperature ranging 20 to 22°C and humidity (60%). Rats were fed with standard pellet chow with free access to tap water *ad libitum* for one week before the experiment for acclimatization. Animal utilization protocols were performed in accordance with the guidelines provided by the Experimental Animal Laboratory and approved by the Animal Care and Committee of the King Abdulaziz University.

Induction of metabolic syndrome

Fructose-induced metabolic syndrome was induced in male Wister rats by adding fructose solution 20% (w/v) in tap water that was prepared every day.

Experimental design

Thirty rats were randomly divided into 3 groups of 10 rats each as follows:

1. The first group acted as control (C): Rats received standard rodent diet and tap water;
2. The second group acted as metabolic syndrome (F): Rats received standard rodent diet and tap water supplemented with 20% fructose;
3. The third group acted as treated fructose-fed (F-T): Rats received standard rodent diet and tap water supplemented with 20% fructose and selenium administered at the dose of 0.25 mg/0.5 ml distilled water/kg body weight per day by gavages. Body weight was measured every two weeks.

The experiment was carried out for 5 weeks. At the end of the treatment period and after an overnight fast (10 to 12 h), blood glucose concentrations were measured in tail vein blood using glucose meter (Mallick et al., 2007), all animals were scarified under light ether anesthesia. Blood samples were collected for serum separation. Serum was separated by centrifugation at 3,000 rpm. for 15 min and stored at -20°C for further analyses.

Liver indicator such as activity of alanine aminotransferase (ALT) (Bergmeyer et al., 1978), aspartate aminotransferase (AST) (Saris, 1978), albumin (Glick et al., 1986) and total protein (Lowry et al., 1951) were measured spectrophotometrically. Lipids profile including total cholesterol (Stein 1986), high density lipoprotein (HDL) (Burtis and Asshuood, 1999), low density lipoprotein (LDL) (Burtis and Asshuood, 1999) and triacylglycerol (TG) (Wahelfed, 1974) were also determined. Finally, kidney profile such as creatinine (Larsen, 1972), urea (Talke and Schubert, 1965), uric acid (Bulgar and Johns, 1941) were determined.

Statistical analysis

Data were analyzed by comparing values for different treatment groups with the values for individual controls. Results are expressed as mean ± standard error (SE). The significant differences among values were analyzed using analysis of variance (one-way ANOVA) followed by Bonferroni as a post-ANOVA test by SPSS version 15. For all analyses, p values ≤ 0.05 were considered significant. Correlation of variables was tested by the Pearson test.

RESULTS

The effects of fructose feeding and selenium supplementation on body weight in different groups for a period of 5 weeks have been shown in (Figure 1). The body weight of F-T rats group decreased significantly ($p < 0.01$) compared with those in C and F groups. In contrast, fasting serum levels of glucose, TG, LDL-c were significantly increased in F rats when compared with control ($p < 0.01$). Fasting serum levels of glucose, TG, LDL-c were significantly decreased in F-T rats compared with F group ($p < 0.01$). Moreover, fasting serum levels of

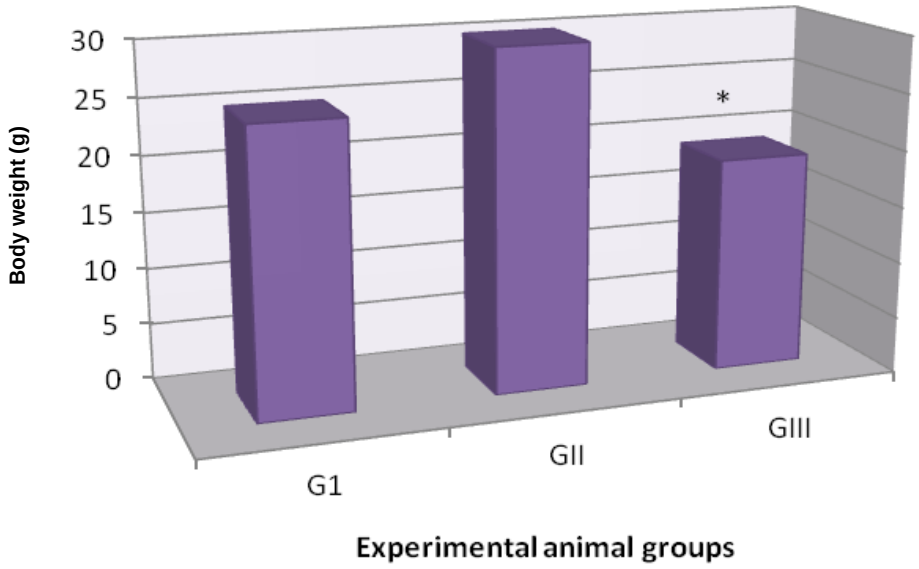

Figure 1. Effects of fructose feeding and selenium on body weight in experimental groups for the period of 5 weeks. GI = control rats, Group II = fructose-fed rats, GIII = fructose fed selenium treated rats. *p < 0.05 compared to control rats.

Table 1. Fasting serum glucose and lipid profile levels in experimental rats.

Parameters (mg/dl)	Control (n = 10)	Fructose (n = 10)	F-T (n = 10)
Glucose	86.1±3.8	176±14 [a]	97±2.0 [b]
Total cholesterol	78.3±7.0	96.3±3·0[a]	74.8±3.3[b]
HDL	43.6±3.0	34.6±2.1[a]	38.6±0.42
LDL	132±5.4	208±24 [a]	79.5±5.3[a,b]
Triglycerides	44.5±5.3	93.3±2.7[a]	42.6±1.9[b]

Values are means ± SE. [a]P < 0.05, compared to control rats. [b]P < 0.05, compared to fructose fed rats. Control: rats received standard diet, Fructose: rats received standard diet and 20% fructose in tap water, F-T: rats received standard diet + 20% fructose in tap water + selenium.

HDL significantly decreased in the F group when compared with control rats ($p < 0.01$), but fasting serum levels of HDL increased in F-T rats in comparison with the F group ($p < 0.01$) (Table 1).

AST, ALT, total protein and albumin serum levels were measured to assess liver function. As can be observed in Table 2, no significant alterations in activity of AST, total protein and albumin serum levels are detected in the animal groups treated with high fructose and selenium during 5 weeks when compared with the control group while serum ALT showed statistical significant difference ($p < 0.05$). The effects of fructose feeding and selenium on kidney function parameters are shown in Table 3. There was no significant difference in serum urea between groups. Serum uric acid levels were elevated in fructose-fed rats compared with control rats which demonstrate that fructose feeding induce early features of metabolic syndrome in rats. Treatment with selenium was effective at lowered uric acid.

To investigate further the role of uric acid, we examined the Pearson's correlation coefficients between uric acid and other factors in experimental rats. Serum uric acid most strongly positively correlated with serum TG ($r = 0.856$), glucose ($r = 0.738$), and LDL-c ($r = 0.808$), while uric acid is negatively correlated with serum HDL-c ($r = -0.253$) and ALT ($r = -0.047$) (Table 4).

DISCUSSION

In this study, we examined the positive role of selenium in the treatment of metabolic syndrome in fructose-fed rats. Treatment of fructose-fed rats with selenium showed significant reduction in serum glucose level to near the normal values. Previous reports indicate that selenium activate a key important protein involved in the insulin-signal cascade (Stapleton, 2000). Also, selenium cause partial restoration of mRNA levels of glucokinase and

Table 2. The levels of indicator of liver function in experimental rats.

Parameters	Control (n = 10)	Fructose (n = 10)	F-T (n = 10)
ALT (U/L)	56.5±1.9	74.8±4.3[a]	67.8±2[a]
AST (U/L)	71.6±2	76.6±3.8	67.6±5.7
Albumin (g/dl)	4.4±0.09	4.5±0.05	4.4±0.14
Total protein (g/dl)	7.4±0.14	7.3±0.06	7.5±0.16

Values are means ± SE. [a]P < 0.05, compared to control rats. Control: rats received standard diet, Fructose: rats received standard diet and 20% fructose in tap water, F-T: rats received standard diet + 20 % fructose in tap water + selenium.

Table 3. The levels of kidney function parameters in experimental rats.

Parameters	Control (n=10)	Fructose (n=10)	F-T (n=10)
Creatinine (mg/dl)	0.54±0.016	0.45±0.01[a]	0.42±0.01[a]
Urea (mg/dl)	22.5±0.99	24.3±1.6	22.8±1.2
Uric acid (mg/dl)	0.76±0.04	1.5±0.11[a]	0.66±0.07[b]

Values are means ± SE. [a]P < 0.05, compared to control rats. [b]P < 0.05, compared to fructose-fed rats. Control: rats received standard diet, Fructose: rats received standard diet and 20% fructose in tap water, F-T: rats received standard diet + 20 % fructose in tap water + selenium.

Table 4. Pearson correlation coefficient (r) in experimental rats.

Parameters	Total cholesterol	Triglycerides	HDL	LDL	ALT	Glucose
Glucose	0.476*	0.846**	0.487*	0.636**	0.153	-
Creatinine	- 0.301	-0.197	0.613**	0.224	-0.587*	-0.361
Albumin	0.566*	0.164	-0.290	0.055	0.112	0.125
Weight gain	0.199	0.14	-0.075	0.483*	0.087	0.213
Uric acid	0.421	0.856**	-0.253	0.808**	-0.047	0.738**

*p < 0.01 **p < 0.001.

pyruvate kinase enzymes. It also decreases the elevated level of mRNA and the activity of phosphoenol pyru-vate carboxy kinase enzyme (Becker et al., 1996). Finally, it has the ability to maintain the expression of the lipogenic enzymes, glucose-6-phosphate dehydrogenase (G6PDH) and fatty acid synthase (FAS) (Berg et al., 1995).

Selenium is a crucial factor in maintaining antioxidant defense and it can also act as a pro-oxidant properties. Selenium supplementation was able to reverse features of metabolic syndrome in fructose-fed rats (Surai, 2002). The mechanism by which selenium exerts its beneficial changes on physiological functions may be through Se-containing proteins. Because of it being an integral part of the active site of a widely dispersed enzyme glutathione peroxidase, it helps to save low levels of cellular peroxides that damage cell membranes (Purshad and Natt, 2007).

Results of this study showed that fructose-fed rats could induce dyslipidemia in fasting rats. Previous studies in rats showed the same results (Shahraki et al., 2011). Fructose feeding stimulates the hepatic production of triglycerides by stimulating de novo fatty acid synthesis and by promoting the re-esterification of circulating non-esterified fatty acids. Increasing the levels of triglycerides and fatty acids reached the muscle and interferes with the utilization of glucose and impairs insulin action (Haidari et al., 2002).

There is strong evidence that uric acid may have a pathogenic role in metabolic syndrome. Hyperuricemia has been found to predict the development of obesity and diabetes (Nakagawa et al., 2006). Most authors consider hyperuricemia in metabolic syndrome to be the consequence of elevated serum insulin levels which stimulate renal reabsorption of uric acid (Facchini et al., 1991).

Although the role of uric acid in the metabolism of triglycerides is still unknown, uric acid might be involved in either the reduction or increase production of clearance of triglycerides. This decrease in fructose-fed rats has been attributed to a reduction in lipoprotein lipase activity in endothelial cells. Also, the increase in de novo purine synthesis in fructose-fed rats may be linked to hepatic

fatty acid synthesis, resulting in increase production of triglycerides (Nakagawa et al., 2006).

Finally, selenium supplementation to fructose-fed rats prevents not only oxidative stress but also renal structural injury as well (Reddi and Bollineni, 2001). Also, the ameliorating effect of selenium on the kidney and liver of fructose-fed rats may be due to its antioxidant as well as its insulin mimetic action which influence polyol pathway, thus selenium treatment may delay or prevent the development of metabolic features (Treska et al., 2002).

Conclusion

Fructose produced metabolic syndrome may result in setting of energy restriction. Furthermore, we demonstrated that the selenium treatment is recommended for more investigation in diabetic patients in future study.

REFERENCES

Abdulla MH, Sattar MA, Johns EJ (2011). The relation between Fructose-Induced Metabolic Syndrome and Altered Renal Haemodynamic and Excretory Function in the Rat. Int. J. Neph. pp. 1-17.

Avramoglu RK, Qiu W, Adeli K (2003). Mechanisms of metabolic dyslipidemia in insulin resistant states: Deregulation of hepatic and intestinal lipoprotein secretion. Front Biosci. 8:d464-476.

Becker DJ, Reul B, Ozcelikay AT, Buchent JP, Henquin JC, Brichard SM (1996). Oral selenate improves glucose homeostasis and partly reverses abnormal expression of liver glycolytic and gluconeogenic enzyme in diabetic rats. Diabetologia 39:3-11.

Berg EA, Wu JY, Campbell L, Kagey M, Stapleton SR (1995). Insulin like effect of vanadate and selenate on the expression of glucose-6-phosphate dehydrogenase and fatty acid synthase in diabetic rats. Biochimie 77:919-924

Bergmeyer HU, Bernt E, Grassl M, Michel G (1978). Glucose-6-phosphate dehydrogenase. In UH Bergmeyer, Methods of Enzymatic Analysis, Verlag Chemie Weinheim, Academic Press. New York pp. 458-459

Bulgar HM, Johns HE (1941). The determination of plasma uric acid. J. Biol. Chem. 140:427

Burtis CA, Asshuood ER (1999). Tietz text book of clinical chemistry. 3rd, Phladelphia, USA. W.P Saunders-Co, PA. pp. 39-72.

Cirillo P, Gersch MS, Mu W. Scherer PM, Kim KM Gesualdo L, Henderson GN, Johnson RJ, Sautin YY (2009). Ketohexokinase-Dependent Metabolism of Fructose Induces Proinflammatory Mediators in Proximal Tubular Cells. J. Am. Soc. Nephrol. 20:545–553

Eriksen MB, Minet AD, Glintborg D, Gaster M (2011). Intact primary mitochondrial function in myotubes established from women with PCOS. J. Clin. Endocrinol. Metab. 96(8):E1298-1302.

Facchini F, Chen YD, Hollenbeck CB, Reaven GM (1991). Relationship between resistance to insulin-mediated glucose uptake, urinary uric acid clearance, and plasma uric acid concentration. JAMA 266:3008–3011

Glick MR, Ryder KW, Jackson SA (1986). Graphical Comparisons of Interferences in Clinical Chemistry Instrumentation. Clin. Chem. 32(3):470–475.

Haidari M, Leung N, Mahbub F, Uffelman KD, Kohen-Avramoglu R, Lewis GF, Adeli K (2002). Fasting and postprandial overproduction of intestinally derived lipoproteins in an animal model of insulin resistance. Evidence that chronic fructose feeding in the hamster is accompanied by enhanced intestinal de novo lipogenesis and ApoB48-containing lipoprotein overproduction. J. Biol. Chem. 277(35):31646-31655.

Johnson RJ, Sanchez-Lozada LG, Nakagawa T (2010). The Effect of Fructose on Renal Biology and Disease. J. Am. Soc. Nephrol. 21:2036-2039.

Khan MS, Dilawar S, Ali I, Rauf N (2012). The possible role of selenium concentration in hepatitis B and C patients. Saudi J. Gastroenterol. 18(2):106-110.

Larsen K (1972). Creatinine assay by a reaction-kinetic approach. Clin. Chem. Acta 41:209-217.

Lowry OH, Roesebrough NS, Farr A L, Randall R J (1951). Protein measurement with folin phenol reagent. J. Biol. Chem. 193:265-275.

Michiels C, Raes M, Toussaint O, Remacle J (1994). Importance of Se glutathione peroxidase, catalase, and Cu/Zn-SOD for cell survival aganist oxidative stress. Free Radic. Biol. Med. 17:235-348.

Nakagawa T, Hu H, Zharikov S, Tuttle KR, Short RA, Glushakova O, Ouyang X, Feig DI, Block ER, Herrera-Acosta J, Patel JM, Johnson RJ (2006). A causal role for uric acid in fructose induced metabolic syndrome. Am. J. Physiol. Renal. Physiol. 290:F625–F631.

Nandhini AT, Anuradha CV (2004). Hoe 140 abolishes the blood pressure lowering effect of taurine in high fructose fed rats." Amino Acids 26:299–303.

Purshad RK, Natt JK (2007). Effects of monosodium glutamate on food acceptance and toxicity of selenium in rats. Ind. J. Exp. Biol. 45:802-806.

Reddi AS, Bollineni JS (2001). Selenium defficient diet induces renal oxidative stress and injury via TGF- beta-1 in normal and diabetic rats kidney. Kidney Int. 59:1342-1353.

Roncal CA, Reungjui S, Sanchez-Lozada LG, Mu W, Sautin YY, Nakagawa T, Johnson RJ (2009). Combination of captopril and allopurinol retards fructose-induced metabolic syndrome. Am. J. Nephrol. 30:399–404.

Shahraki MR, Harati M, Shahraki AR (2011). Prevention of High Fructose Induced Metabolic Syndrome in Male Wistar Rats by Aqueous Extract of Tamarindus Indica Seed. Acta Medica Iranica 49(5):277-283.

Shao L, Tanaka T, Gribi R, Yu J (2002). Thioredoxin-related regulation of NO/NOS activities. Ann. N. Y. Acad. Sci. 962:140-156.

Stapleton SR (2000). Selenium: An insulin mimetic. Cell Mol. Lif. Sci. 57:1874-1879.

Surai PF (2002). Selenium in poultry nutrition – 1: Antioxidant properties deficiency and toxicity. World's Poult. Sci. 58:333-347.

Talke H, Schubert GE (1965). Enzymatishe Harnstoffbestimmung in Blut und serum in optischen test nach Warburg. Klin Wschr 41:174.

Treska V, Kuntscher V, Hasman D, Neprasová P, Kobr J, Racek J, Trefil L, Hes O (2002). Importance of selenium for the influence of ischemia-reperfusion syndrome after kidney transplantation from a non-heart beating donor in a pig model. Transplant Proc. 34(8):3057-3059.

Wahelfed AW (1974). Method of Enzymatic Analysis. Academic press, New York 5:1831-1835.

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Short Communication

Mineral nutrients of 'pazhaya sadham': A traditional fermented food of Tamil Nadu, India

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Traditionally, in Tamil Nadu, the fermented rice is a desirable staple food. It is prepared by overnight soaking of cooked parboiled rice in water. The main complementary role of fermentation is the bioenhancing or bioavailability of essential nutrients especially minerals through the enzymatic reduction of Phytate. In the present study, essential trace elements such as Calcium, Magnesium, Iron, Sodium, Potassium and Selnium were determined by using Inductively coupled plasma optical emission spectrometry (ICP-OES) method. The minerals such as Calcium (9.23 mg/100 g) and Sodium (17.18 mg/100 g) are rich in the sample Source 2. The determination of the presence of selenium (0.2 to 0.3 mg/100 g) in this fermented rice can be explained as a preventive mechanism to cancer as natural source of mineral availability.

Key words: Traditional food, phytic acid, cancer.

INTRODUCTION

Fermentation is one of the oldest and most economical methods of producing and preserving food (Billings, 1998; Chavan and Kadam, 1989). Cereal grains are considered to be one of the most important sources of dietary proteins, carbohydrates, vitamins, minerals and fibre for people all over the world. However the nutritional quality of cereals and the sensorial properties of their products are sometimes inferior or poor in comparision with milk and milk products. The reasons behind this are the lower protein content, the deficiency of certain essential amino acids (Lysine), the low starch availability, the presence of determined antinutrients phytic acid, tannins and poly phenols and the coarse nature of the grains (Chavan and Kadam, 1989).

Natural fermentation of cereals leads to a decrease in the levels of Carbohydrate as well as some non-digestible poly and oligosaccharides. Certain amino acids may be synthesized and the availability of B group vitamins may be improved. Fermentation also provides optimum pH conditions for enzymatic degradation of phytate which is present in cereals in the form of complexes with polyvalent cations such as iron, zinc, calcium, magnesium and proteins. Such a reduction in phytate may increase the amount of soluble iron, zinc and calcium several folds (Haard et al., 1999; Khetar and Chauhan, 1990; Nout and Motarjemi, 1997).

The bioavailability of minerals from foods is defined as the proportion of the minerals that can be absorbed and utilized within the body (Larsson et al., 1997; Lestienne et al., 2005a, b, c). Solubility of minerals, pH of intestinal lumen, dietary factors and residence time at the absorption site influences the bioavailability of minerals (Larsson et al., 1997). The study was carried out to investigate the mineral content of the fermented rice from two different house hold preparations by using ICP-OES method.

MATERIALS AND METHODS

The samples were collected from local houses in the District Thanjavur, Tamil Nadu. The traditional method of preparation in household is presented in Figure 1.

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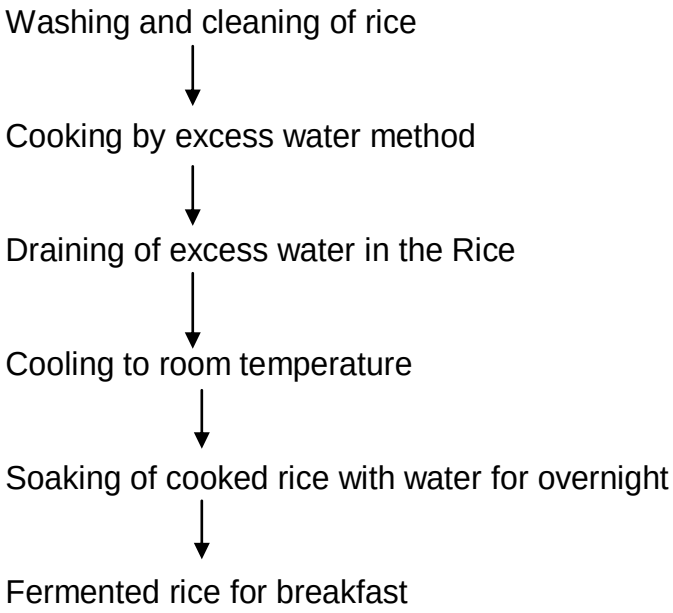

Figure 1. The traditional method of preparation.

Table 1. Instrumental conditions of ICP-OES.

Parameter	Setting
RF power	1100 W
Nebulizer flow	0.950 L/min
Auxiliary flow	1.0 L/min
Plasma flow	15 L/min
Sample flow	1.0 mL/min
Source equilibration time	15 s
Viewing height	15 mm
Background correction	Manual selection of points
Measurement processing mode	Area
Auto integration	1 s minimum to 50 s maximum
Read delay	45 s
Rinse delay	45 s
Number of replicates	3

Table 2. Mineral analysis by ICP-OES in fermented rice.

S/No	Minerals	Source 1	Source 2
1	Calcium (mg/10 g)	8.2±0.21	9.23±0.27
2	Iron (mg/1 g)	0.20±0.03	0.24±0.04
3	Potassium (mg/10 g)	2.93±0.06	3.57±0.38
4	Magnesium (mg/100 g)	2.77±0.23	3.02±0.26
5	Sodium (mg/100 g)	13.63±0.40	17.18±0.50
6	Selenium (mg/100 g)	0.02±0.01	0.03±0.01

Values are given as mean ± Standard deviation of the *triplicate* samples.

Mineral analysis by ICP-OES method

The fermented rice was homogenized with mortar and pestle and 4 g of homogenized sample was ashed with muffle furnace at 550°C for 3 h and it was cooled for 30 min. After cooling, the ash was dissolved with 30 ml of HCl with distilled water (1:1) and kept in sand bath for 15 min to reduce the volume of the solution to 5 to 8 ml and the sample solution was filtered through a Whattman No. 1 filter paper and made up to 25 ml in standard flask. The mineral contents were determined by using ICP-OES method (Perkin Elmer DV-2000). The sample was digested in triplicate and analysed using the following conditions in ICP-OES (Table 1).

RESULTS AND DISCUSSION

The mineral analysis of fermented rice showed that it was rich in sodium, potassium and calcium (Table 2). It also contains iron and magnesium which is the essential elements of our living system. It also contains the trace element selenium in the amount of 0.02 to 0.03 mg/100 g. Selenium is an essential element for normal development, growth and metabolism because of its role in the regulation of thyroid hormones (Gladyshev, 2006). The study showed that source 2 has more amount of mineral content than source 1. The fermented rice could serve as a mineral rich breakfast and it contains essential minerals for the metabolic activities of our normal body functioning.

REFERENCES

Billings T (1998). On fermented foods. Available: http://www.living-foods.com.

Chavan JK, Kadam SS (1989). Critical reviews in food science and nutrition. Food Sci. 28:348-400.

Gladyshev VN (2006). Selenoproteins and selenoproteomes. In: Hatfield DL, Berry MJ, Gladyshev VN, eds. Selenium: Its molecular biology and role in human health. 2nd ed. New York: Springer pp. 99-114.

Haard NF, Odunfa SA, Lee CH, Quientero–Ramirez R, Lorence-Quinones A, Wacher-Radarte C (1999). Fermented cereals. A global perspective. FAO Agric. Serv. Bull. No. 138.

Larsson M, Minekus M, Havenaar R (1997). Estimation of the bioavailability of iron and phosphorus in cereals using a dynamic *in vitro* gastrointestinal model. J. Sci. Food Agric. 74:99–106.

Lestienne I, Besancon P, Caporiccio B, Lullien-Pellerin V, Tre`che S (2005a). Iron and zinc *in vitro* availability in pearl millet flours (*Pennisetum glaucum*) with varying phytate, tannin, and fiber contents. J. Agric. Food Chem. 53(8):3240–3247.

Lestienne I, Icard-Vernie`re C, Mouquet C, Picq C, Tre`che S (2005b). Effects of soaking whole cereal and legume seeds on iron, zinc and phytate contents. Food Chem. 89(3):421-425.

Lestienne I, Mouquet-Rivier C, Icard-Vernie`re C, Rochette I, Tre`che S (2005c). The effects of soaking of whole, dehulled and ground millet and soybean seeds on phytate degradation and Phy/Fe and Phy/Zn molar ratios. Int. J. Food Sci. Technol. 40(4):391-399.

Nout MJR, Motarjemi Y (1997). Assessment of fermentation as a household technology for improving food safety: A joint FAO / WHO workshop. Food Control 8:221-226.

UPCOMING CONFERENCES

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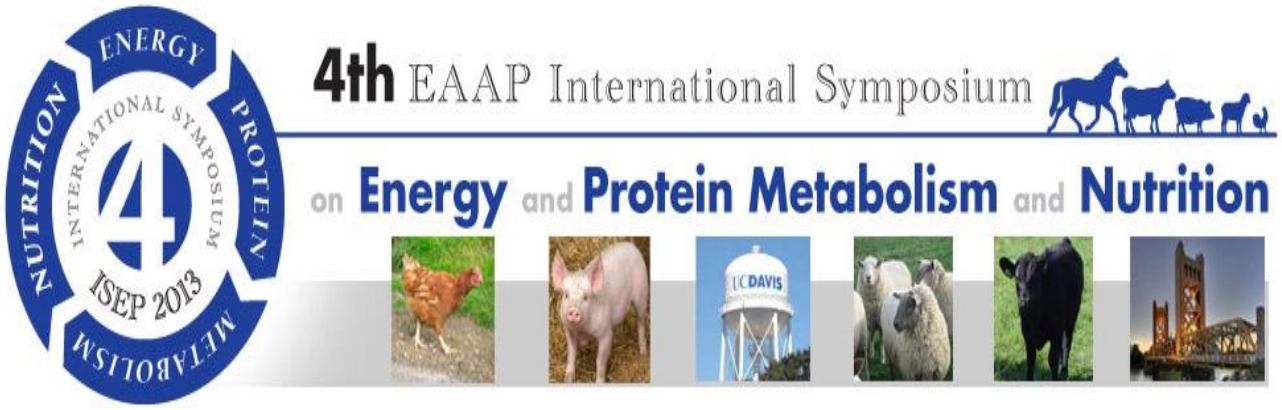

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