

Relationship between Dietary Pattern And Level of Antioxidant Enzymes in Diabetic Patients

Maigari, F.U.^{1*}, Shu'aibu Isa², Alkali, Y.S¹., Maigari, M.U³
, Sadau Y¹., Musa Abubakar⁴

¹College Of Medical Sciences, Gombe State University,

²Department Of Microbiology, Gombe State University,

³Federal College Of Education (T)Gombe, ⁴Ministry Of Health, Gombe.

Abstract

Background: Diabetes mellitus is a medical condition affecting many with incidence rapidly growing worldwide. Approximately, 7.1 million Africans were said to be suffering from diabetes at the end of 2000, a figure that was expected to rise to 18.6 million by 2030 (Wild *et al.*, 2004).

Materials and Methods: This study determined the relationship between the dietary pattern and level of antioxidant enzymes of some diabetic patients attending the Federal Teaching Hospital Gombe. The patients are registered with the hospital and have their glycated haemoglobin level above normal and their informed consent was sought. The hospital also gave an ethical clearance for the research.

Results and Conclusion: There was no significant difference in the levels of glycated hemoglobin, superoxide dismutase, catalase, GPx and malondialdehyde levels ($p > 0.05$) against the breakfast levels of the study subjects. The results also showed no significant difference ($p > 0.05$) in the mean levels of the antioxidant enzymes of superoxide dismutase, catalase and GPx in the pattern of lunch of the subjects but a significant difference exists in the level of glycated hemoglobin and malondialdehyde ($p < 0.05$). With respect to dinner consumption among the study subjects, a significant difference in the levels of glycated hemoglobin and GPx also exist. It can be concluded from the study that relationship between the level of antioxidant enzymes of GPx, superoxide dismutase and catalase with the selected breakfast and lunch pattern of is not to a significant level, however level of GPx is significantly affected by eating rice during dinner.

Keywords: Diabetes mellitus, Dietary pattern, Antioxidant enzymes, Glycated haemoglobin, Diabetic subjects.

I. Introduction

The term 'oxidative stress' is used when the body's natural defence mechanism are exceeded by the production of deleterious reactive oxygen species (ROS), resulting in damage to susceptible cell components such as DNA, proteins and lipids. Features of the brain that cause it to be particularly sensitive to oxidative stress include a high rate of oxidative metabolic activity, relatively low levels of antioxidant enzymes (e.g. catalase, glutathione peroxidase), a high concentration of unsaturated fatty acids, large iron and copper stores, and a low mitotic index (Lynch *et al.*, 2000).

Superoxide dismutase is one of the key antioxidant enzymes which provide an essential defence against oxygen toxicity to the cell. Superoxide dismutase is the antioxidant enzyme that catalyses the conversion of O_2^- to O_2 and to the less reactive species H_2O_2 . There are three forms of SOD in humans (Landis and Tower, 2005); CystolicCu,Zn-SOD, Mitochondrial Mn-SOD, Extracellular SOD EC- SOD.

Catalase is a heme containing redox enzyme, found in high concentration in the peroxisomes. The enzyme is present in the cells of plants, animals and aerobic bacteria (Mates *et al.*, 1999). The enzyme very efficiently promotes the conversion of H_2O_2 to water and molecular oxygen.

Glutathione metabolism is one of the most essential of antioxidative defence mechanisms. GPx acts in conjunction with the tripeptide glutathione (GSH), which is present in cells in high concentration (micro molar). The substrate for the catalytic reaction of GPx is H_2O_2 , or organic peroxide ROOH. GPx decomposes peroxides to water (or alcohol) while simultaneously oxidising GSH.

The incidence of diabetes, especially type 2, is rapidly growing in the world. In 1985, an estimated 30 million people suffered with this chronic disease, which, by the end of 2006, had increased to 230 million, representing 6% of the world population. Of this number, 80% is found in the developing world (Roglic *et al.*, 2005). It is estimated that during the next 35 years, diabetic world-wide prevalence will reach 25%, with India being the hardest hit. For a long time, Africa was considered safe from many of the diseases that are called "diseases of affluence," which plague the Western world. Similarly, there was a time when Africa was thought to be a continent, relatively free of diabetes mellitus illnesses. Indeed, from 1959 to the mid-1980s, medical statistics showed that the prevalence rate of diabetes in Africa was equal to or less than 1.4%, with the exception of South Africa, where the rate was estimated to be as high as 3.6% in 2001 (Motala, 2002; Motala *et al.*, 2003;

Rheeder, 2006). But, by 1994, the continent-wise prevalence of diabetes mellitus stood at 3 million and was then predicted to double or triple by the year 2010 (Sobngwi *et al.*, 2001; International Diabetes Federation, 2003). Approximately, 7.1 million Africans were said to be suffering from diabetes at the end of 2000, a figure that was expected to rise to 18.6 million by 2030 (Wild *et al.*, 2004).

By far the most investigated diet component is cow milk protein and its effect on auto-immunity or diabetes outcome; several reviews are available (Kolb and Pozzilli, 1999; Schrezenmeir and Jagla, 2000). Human milk contains approximately four times more insulin as cow milk. This has prompted the suggestion that human insulin should be added to infant formulas to increase the oral immune tolerance to insulin and possibly prevent diabetes (Shehadeh *et al.*, 2001).

II. Methodology

This study was conducted in Gombe State. It is one of Nigeria's 36 states with an area, having its state capital Gombe. Subjects for this study were drawn from Diabetic patients attending the Medical clinic of Federal Teaching Hospital Gombe (FTHG). The subjects were registered with the clinic and their informed consent was sought. The patients were previously diagnosed with diabetes for more than a year, and they were treated for the disease but still had their glycated haemoglobin above normal range. They were free from other diseases and chronic diabetic complications. The institutional ethical board granted the approval of the research protocol.

The procedure for estimation of Glycated haemoglobin was as described by Gonen and Rubenstein (1978), plasma malondialdehyde was measured by the method of Ohakawa *et al.* (1979), Superoxide dismutase (SOD) activity was measured according to the method of Maier and Chan (2002), Reagents for catalase activity were obtained from Cell Biolabs, Inc USA and the method for assay of the enzyme activity in the diabetic patients and apparently healthy subjects employed was as described by Zamocky and Koller (1999). Glutathione peroxidase (GPx) activity was measured according to the method of Paglia and Valentine (1967) using reagents purchased at Cayman, MI, USA.

III. Results

There was no significant difference in the levels of glycated hemoglobin, superoxide dismutase, catalase, GPx and malondialdehyde levels ($p > 0.05$) against the breakfast levels of the study subjects (table 1). The number of subjects consuming tea and bread were highest (N=50) and has a mean level of glycated hemoglobin (6.57 ± 1.263), superoxide dismutase (0.19 ± 0.228), catalase (44.06 ± 14.707), GPx (87.87 ± 31.703), malondialdehyde ($7.81 \pm 5.242 \text{ nmol/min/L}$).

Table 2 shows the frequency and mean \pm standard deviation of subjects against their lunch consumption. The table showed no significant difference ($p > 0.05$) in the mean levels of the antioxidant enzymes of superoxide dismutase, catalase and GPx in the pattern of lunch of the subjects. However, there is significant difference in the levels of glycated hemoglobin and malondialdehyde ($p < 0.05$). The mean levels of glycated hemoglobin (7.36 ± 0.31) was highest in the subjects that consume pasta (N=5) with the lowest level (6.19 ± 1.372) in the subjects that consume rice (N=50). From the table, the subjects that consumed rice (N=50) have the lowest level of malondialdehyde ($6.42 \pm 5.004 \mu\text{mol/L}$) with those consuming Tuwo (N=5) having the highest level of malondialdehyde ($14.09 \pm 5.357 \mu\text{mol/L}$).

There was a significant difference in the levels of glycated hemoglobin and GPx in the different pattern of dinner consumption among the study subjects (table 3). The level of GPx is higher in the subjects (N=16) that consume Rice (7.19 ± 0.223) and lowest among the subjects (N=26) that consumed Pasta (6.13 ± 1.435). The mean level of GPx was significantly highest in subjects (N=16) that consumed rice (110.79 ± 44.929) with the lowest level of GPx in subjects (N=66) that consumed Tuwo (84.13 ± 28.470). From the table, there was no significant difference ($p < 0.05$) in the mean levels of superoxide dismutase, catalase and malondialdehyde in relation to the dinner pattern of the subjects.

Table 1: Breakfast of Subjects against Glycated Hb, Antioxidant Enzymes and MDA Levels

	Breakfast		Mean \pm Std. Dev
		N	
Glycated Hb	Koko/Kosai	28	6.41 \pm 1.345
	Tea/ Bread	50	6.57 \pm 1.263
	Tuwo	39	6.85 \pm 0.966
	Chips	3	5.39 \pm 1.649
	Total	120	6.59 \pm 1.212
SOD(u/ml)	Koko/Kosai	28	0.19 \pm 0.025
	Tea/Bread	50	0.19 \pm 0.228
	Tuwo	39	0.19 \pm 0.026
	Chips	3	0.21 \pm 0.173
	Total	120	0.19 \pm 0.024
Catalase(u/ml)	Koko/Kosai	28	46.94 \pm 14.195
	Tea/Bread	50	44.06 \pm 14.707
	Tuwo	39	44.69 \pm 12.887

	Chips	3	56.87±7.422
	Total	120	45.26±13.917
GPx(nmol/min/ml)	Koko/Kosai	28	96.42±28.696
	Tea/ Bread	50	87.87±31.703
	Tuwo	39	92.73±33.478
	Chips	3	65.37±11.483
	Total	120	90.88±31.454
Malondialdehyde (µmol/L)	Koko/Kosai	28	6.88±4.147
	Tea/Bread	50	7.81±5.242
	Tuwo	39	8.51±4.599
	Chips	3	8.19±6.028
	Total	120	7.83±4.790

Values are mean ± standard deviation

N= number of subjects

Values with * are significantly different at p< 0.05.

Table 2: Lunch of Subjects against Glycated Hb, Antioxidant Enzymes and MDA Levels

		N	Mean± Std. Dev
Glycated Hb	Couscous	20	6.65±1.165
	Pasta	45	6.92±0.972
	Rice	50	6.19±1.372
	Tuwo	5	7.36±0.31*
	Total	120	6.59±1.216
SOD(u/ml)	Couscous	20	0.19±0.019
	Pasta	45	0.19±0.026
	Rice	50	0.19±0.021
	Tuwo	5	0.18±0.049
	Total	120	0.19±0.002
Catalase(u/ml)	Couscous	20	43.82±16.469
	Pasta	45	43.13±12.888
	Rice	50	47.53±13.988
	Tuwo	5	47.53±10.598
	Total	120	45.26±13.911
GPx(nmol/min/ml)	Couscous	20	85.19±40.019
	Pasta	45	92.48±34.654
	Rice	50	92.81±25.558
	Tuwo	5	79.98±15.131
	Total	120	90.88±31.454
Malondialdehyde (µmol/L)	Couscous	20	8.37±4.242
	Pasta	45	8.46±4.068
	Rice	50	6.42±5.004
	Tuwo	5	14.09±5.357
	Total	120	7.83±4.790*

Values are mean ± standard deviation

N= number of subjects

Values with * are significantly different at p< 0.05.

Table 3: Dinner of Subjects against Glycated Hb, Antioxidant Enzymes and MDA Levels

	Dinner	N	Mean±Std. Dev
Glycated Hb	Couscous	12	6.53±1.237
	Pasta	26	6.13±1.435
	Rice	16	7.19±0.223
	Tuwo	66	6.64±1.209
	Total	120	6.59±1.216*
SOD(u/ml)	Couscous	12	0.191±0.023
	Pasta	26	0.20±0.020
	Rice	16	0.18±0.020
	Tuwo	66	0.19±0.026
	Total	120	0.19±0.024
Catalase(u/ml)	Couscous	12	46.68±13.000
	Pasta	26	49.52±12.271
	Rice	16	40.04±12.829
	Tuwo	66	44.59±14.662
	Total	120	45.26±13.911
GPx(nmol/min/ml)	Couscous	12	82.35±25.868
	Pasta	26	99.72±24.617
	Rice	16	110.79±44.929
	Tuwo	66	84.13±28.470

	Total	120	90.88±31.454*
Malondialdehyde (µmol/L)	Couscous	12	7.82±6.089
	Pasta	26	5.81±4.159
	Rice	16	8.09±2.846
	Tuwo	66	8.56±5.002
	Total	120	7.83±4.790

Values are mean ± standard deviation

N= number of subjects

Values with * are significantly different at p< 0.05

Table 34: In between meals Consumption of Subjects against glycated hemoglobin, antioxidant enzymes and malondialdehyde Levels

		N	Mean± Std. Dev
Glycated Hb	water	47	6.45±1.339
	tea nd bread	26	7.02±0.840
	juice	27	6.29±1.326
	Pasta	20	6.76±1.056
	Total	120	6.59±1.216
SOD(u/ml)	water	47	0.19±0.027
	tea nd bread	26	0.19±0.025
	juice	27	0.19±0.023
	Pasta	20	0.19±0.179
	Total	120	0.19±0.024
Catalase(u/ml)	water	47	46.71±15.243
	tea nd bread	26	42.67±13.968
	juice	27	44.18±14.976
	Pasta	20	46.66±8.230
	Total	120	45.26±13.911
GPx(nmol/min/ml)	water	47	79.17±20.635
	tea nd bread	26	101.68±40.353
	juice	27	88.86±29.256
	Pasta	20	107.09±32.717
	Total	120	90.88±31.454*
Malondialdehyde (umol/L)	water	47	8.64±5.638
	tea nd bread	26	8.15±3.501
	juice	27	6.57±4.927
	Pasta	20	7.21±3.603
	Total	120	7.83±4.790

Values are mean ± standard deviation

N= number of subjects

Values with * are significantly different at p< 0.05.

IV. Discussion

The different lunch pattern of the study subjects in relation to glycated hemoglobin and malondialdehyde differed significantly (p<0.05). The study found that those subjects consuming Tuwo during lunch (table 28) had a higher level of glycated hemoglobin (7.36±1.372) and malondialdehyde (14.09±5.357µmol/L). Most of the subjects consume the tuwo with different kinds of soups which may have high lipid content that may be susceptible to lipid peroxidation.

The dinner pattern of the subjects showed significant rise in the level of glycated hemoglobin (table 29) with those consuming rice having a higher level of glycated hemoglobin (7.19±0.223). The level of GPx in the subjects was also significantly higher (110.79±44.929nmol/min/ml). The study found an increase in the level of GPx in diabetic subjects, with higher level of glycated hemoglobin indicating a poor management of blood sugar.

Subjects consuming rice (table 32) during dinner had a significantly higher content of Cu (105.57±14.362µg/dl) (p<0.05). From the food composition table (FAO, 2012) rice whole, polished and boiled has a high retention factor for copper. Serum copper level in the study subjects may be implicated due to diabetic condition since increase in serum copper was found to be higher in diabetic subjects as reported by Maigari *et al.* (2015).

The result showed a significant difference ($p < 0.05$) in the mean level of GPx in those subjects consuming Pasta in between their meals. Level of the antioxidant enzyme GPx was found to be high in diabetics (Maigari *et al.*, 2015)

References

- [1]. Gonen, B. And Rubenstein, A.H. (1978). Haemoglobin A1 and diabetes mellitus. *Diabetologia* 15:1-8
- [2]. International Diabetes Federation. (2003) *Diabetes Atlas*. 3rd ed. Belgium: IDF, Brussels.
- [3]. Kolb, H. and Pozzilli, P. (1999). Cow's milk and type I diabetes: the gut immune system deserves attention. *Immunol Today* 20: 108–10.
- [4]. Landis, G.N. and Tower, J. (2005) Superoxide dismutase evolution and life span regulation, *Mech. Ageing Dev.* 126:365–379.
- [5]. Maier, C.M. and Chan, P.H. (2002). Role of superoxide dismutases in oxidative neurodegenerative disorders. *The Neuroscientist* 8 (4): 323-334.
- [6]. Mates, J.M., Perez-Gomez, C. and De Castro, I.N. (1999). Antioxidant enzymes and human diseases, *Clin. Biochem.* 32:595–603.
- [7]. Maigari, F.U., Atiku, MK, Wudil, AM and Goje, LJ (2015) Determination of trace elements level in diabetes mellitus patients attending the Federal Teaching Hospital, Gombe. *International Journal of Scientific Innovation and Sustainable Development* 5(2)232-236.
- [8]. Motala, A.A. (2002). Diabetes trends in Africa. *Diabetes Metab Res Rev.* 18:S14–20.
- [9]. Motala, A.A., Omar, M.A.K., Pirie, F.J. (2003). Epidemiology of type 1 and type 2 diabetes in Africa. *J Cardiovasc Risk.* 10:77–83.
- [10]. Ohakawa H, Oshishi N. and Yagi K. (1979). "Assay for Lipid Peroxidation in Animal Tissue by Thiobarbituric Acid Reaction. *Anal. Biochem.* 75:351-358.
- [11]. Paglia, D.E., and Valentine, W. N. (1967) Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J. Lab. Clin. Med.* 70: 158-169.
- [12]. Rheeder, P. (2006). Type 2 diabetes: The emerging epidemic. *South Afr Fam Pract.* 48:20.
- [13]. Roglic, G., Unwin, N., Bennett, P.H., Mathers, C., Tuomilehto, J., Nag, S. (2005). The burden of mortality attributable to diabetes: Realistic estimates for the year 2000. *Diabetes Care.* 28:2130–5.
- [14]. Schrezenmeir, J., and Jagla, A. (2000). Milk and diabetes. *J Am Coll Nutr* 19 (Suppl. 2): 176S–190S.
- [15]. Sobngwi, E., Mauvais-Jarvis, F., Vexiau, P., Mbanya, J.C., Gautier, J.F. (2001). Diabetes in Africans: Part 1: Epidemiology and clinical specificities. *Diabetes Metab.* 27:628–34
- [16]. Wild, S., Roglic, G., Green, R., and King, H. (2004) Global prevalence of diabetes: Estimates for year 2000 and projections for 2030. *Diabetes Care* 27:1047-1053.