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Glycemic control of type 2 diabetic patients after short-term zinc supplementation*

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Abstract

This study was carried out to determine whether a short-term zinc supplementation contributes to beneficial changes in glycemic control among type 2 diabetic patients. Seventy-six diabetic subjects and 72 normal adults participated in this study. Subjects were divided into supplemented and control groups. Forty-four diabetic patients and 34 normal subjects were supplemented with 50 mg zinc daily as zinc gluconate for 4 weeks. Zinc status was assessed from fasting plasma levels and urinary excretion. The effects of zinc supplementation on fasting blood glucose, HbA_{1c}, insulin, and C-peptide were measured at the beginning of the study and after 4 weeks of supplementation. The changes in glycemic control indicators were compared between diabetic groups, classified by baseline HbA_{1c} levels, and by diabetic duration. At baseline, the incidence of marginal zinc deficiency in the diabetic group, as determined by plasma zinc level, was approximately twice as high as in the normal adult group. The changes of HbA_{1c} concentration, and fasting blood glucose following supplementation were not statistically significant in diabetic subjects. In normal subjects, a significant decrease of HbA_{1c} occurred only in the zinc supplemented group. No significant changes were observed for serum insulin and C-peptide in diabetic as well as normal subjects. However, when the changes were compared by baseline HbA_{1c} level, we found that diabetic subjects with $HbA_{1c} \geq 7.5\%$ showed significantly improved levels of HbA_{1c} and fasting glucose after Zn supplementation. While such improvement in fasting blood glucose was significant among diabetics with shorter diabetic duration, significant levels of increase in serum insulin and C-peptide were observed in zinc supplemented subjects with longer diabetic duration. Fasting blood glucose was significantly decreased, whereas serum insulin and C-peptide were increased in diabetics with marginal zinc status. Therefore, we suggest that Zn supplementation for a short-term period may improve glycemic control in diabetic patients with higher HbA_{1c} levels and marginal zinc status.

Key Words: Diabetes, zinc supplementation, diabetic duration, glycemic control

Introduction

Zinc is involved in numerous metabolic pathways as an cofactor for more than 300 enzymes (Rink & Kirchner, 2000). It also plays an important role for insulin action, carbohydrate and protein metabolism (Chausmer, 1998). Insulin, which contains a variable number of zinc atoms, are stored in β-cells of the pancreas and released into the portal venous system at the time of β-cells degranulation. Type 2 diabetes mellitus is a metabolic disorder with hyperglycemia as the dominant feature (Chausmer, 1998).

Several investigators have shown the perturbation of zinc metabolism in diabetics (Golik *et al.*, 1993; Kinlaw *et al.*, 1983; Levine *et al.*, 1983; Walter *et al.*, 1991). It has been suggested that hyperzincuria and impaired absorption are major causes of zinc deficiency among diabetics (Cunningham *et al.*, 1994; Heise *et al.*, 1988; Kazi *et al.*, 2008; Salgueiro *et al.*, 2001; Walter *et al.*, 1991). Clinical manifestations including delayed wound healing, decreased cell immunity and deterioration of taste acuity in this population have been also reported (Keen & Gershwin, 1990; Kinlaw *et al.*, 1983; Salgueiro *et al.*, 2001).

It was suggested that the marginal zinc deficiency may be related with impaired immunity and taste (Salgueiro *et al.*, 2001). Hence, zinc supplementation is receiving growing attention as a way to improve the immune function as well as the marginal zinc deficiency (Anderson *et al.*, 2001; Faure *et al.*, 1995).

Few studies have been conducted on the effects of zinc supplementation on hyperglycemia of diabetics and their results are inconsistent. Zinc supplementation has improved fasting insulin level and fasting glucose in genetically obese mice models (Chen *et al.*, 1998; Simon & Taylor, 2001). Improved fasting glucose levels up to 30% in patients with cirrhosis through supplementation of zinc for 2 months has been reported (Marchesini *et al.*, 1998). Supplementation of 30 mg zinc for 3 months in type 2 diabetics showed a decrease in HbA_{1c} concentration. Meanwhile, others reported that neither fasting glucose nor HbA_{1c} levels was changed by a short-term supplementation with zinc for type 1 diabetics (Cunningham *et al.*, 1994; de Sena *et al.*, 2005).

Type 2 diabetic patients are characterized by reduced secretion of insulin and increased insulin resistance, resulting in elevated blood glucose levels (Roth & Kirchgessner, 1981; Salgueiro *et*

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al., 2001). It has not been clearly elucidated whether zinc deficiency is a consequence of hyperglycemia or, alternatively, whether zinc deficiency contributes to the pathogenesis of diabetes.

The National Health and Nutrition Examination Survey for Koreans conducted in 2005 showed that the prevalence of diabetes was 9.0% for male adults and 7.2% for female adults (Ministry of Health & Welfare, 2006). Previous studies indicated that marginal zinc deficiency is more prevalent among diabetic adults compared to the normal adult population (Lee *et al.*, 2005; Yoon & Lee, 2007). Therefore, we intended to determine whether zinc supplementation for a short-term period could improve the levels of fasting glucose and HbA_{1c} as well as the insulin levels in Korean type 2 diabetics. Moreover, this study aimed to find out whether the changes in glycemic control by zinc supplementation are associated with diabetic duration or zinc status.

Materials and Methods

Subjects

This study was conducted on 76 type 2 diabetic adults attending local clinics and public heath centers. Characteristics of the diabetic subjects have been reported elsewhere (Yoon, 2008). For comparison, 72 apparently healthy normal adults living in the same city were also recruited during the same period. Informed consent was obtained from each subject before the beginning of the study. Exclusion criteria of the subjects were pregnancy, lactation, consuming mineral supplements or prescribed drugs for diseases. The subjects were divided into zinc supplementation groups and control groups. Among diabetic participants, 44 subjects took zinc supplements. Among 72 normal adults, 32 subjects took zinc supplements.

Methods

1) Interview questionnaire

Information on age, education levels, diabetic duration, drug usage, diabetic complications was interviewed by a trained dietitian.

2) Anthropometric Assessment

Height, weight, circumferences of waist and hip, and body fat were measured from each subject. BMI (body mass index) was calculated with measured height and weight. Body fat content and percentage body fat were assessed using the Bio-electrical Impedance Fatness Analyzer (HBF-300, OMRON Corporation, Japan).

3) Biochemical Assessment

Overnight fasting blood samples were collected either into evacuated heparin coated tubes or into serum tubes and were stored at -70℃. HbA_{1c}, fasting blood glucose, serum insulin, and C-peptide were measured as the indicators of glycemic regulation using routine laboratory methods (Roussel *et al.*, 2003). Plasma level of zinc was assessed by atomic absorption spectrometry (Lee *et al.*, 2005). Urine samples for 24 hours were collected for one day from each subject. Urinary zinc was analyzed by Inductively Coupled Plasma Emission Spectroscopy (ICP) (Jobin Yvon, 38 plus, France). We followed the criteria for marginal zinc deficiency from the previous studies (King & Keen, 1994).

Zn supplementation

Selection of chemical form, dose level, and duration of zinc supplementation in this study were based on the reports of other investigators (Anderson *et al.*, 2001; Bonham *et al.*, 2003; Chandra, 1984; Roussel *et al.*, 2003). Capsules containing 50 mg of zinc gluconate (Labcatal, Pharmaceutical), reported as the safe level for supplementation for 3 months, were given to the diabetic and normal subjects for 4 weeks. Each subject was encouraged to take the supplement everyday. Compliance with the supplementation trial was confirmed at the end of the study. Subjects who reported consuming less than 80% of the total amounts were excluded from the data analysis.

Data Analysis

The Statistical Analysis System (SAS) Package was used to analyze the relationship of glycemic control status and zinc supplementation. Data were presented as mean ± SD. The statistical significance of the differences between the diabetic and the normal subjects was tested by the Student's *t* test. The changes in glycemic indicators were compared between the two diabetic patients groups, first classified by baseline HbA_{1c} levels ($<7.5\%$ vs $\geq 7.5\%$), then by zinc status (marginal vs normal), and, lastly, by diabetic duration (<4 years vs ≥ 4 years). Differences in the distribution of variables between the two diabetic groups were tested by the Student's *t* test. The effects of zinc supplementation on hyperglycemia of diabetics were tested by paired t-test. When any variables show significant differences between supplemented and non-supplemented groups at baseline, analysis of covariance (ANCOVA) was used to determine the effects of supplementation.

Results

Baseline characteristics of the participants such as age, diabetic duration, are shown in Table 1. Details on the characteristics of the diabetic subjects were described in previous reports (Yoon, 2008). Among diabetic participants, 44 subjects were provided with zinc supplement, while 32 subjects participated as the control group. Among the 72 normal adults, 32 subjects took

Table 1. Baseline characteristics of the subjects

Variables	Diabetic Supplement (n=44)	Diabetic Control (n=32)	Nondiabetic Supplement (n=32)	Nondiabetic Control (n=40)
Age (years)	58.7±10.1	58.5±11.7	53.1±11.7	49.6±10.7
Height (cm)	161.8 ± 8.88	161.7 ± 8.38	163.0 ± 7.16	160.0 ± 7.99
Weight (kg)	63.9 ± 9.97	62.7 ± 9.61	62.7 ± 7.75	60.5 ± 9.22
Waist (cm)	84.3 ± 11.17	84.7 ± 8.87	84.5 ± 6.78	82.6 ± 8.58
Hip (cm)	97.3 ± 5.46	96.2 ± 6.52	95.4 ± 5.55	95.6 ± 5.25
WHR	0.87 ± 0.10	0.88 ± 0.06	0.89 ± 0.06	0.86 ± 0.06
BMI (kg/㎡)	24.3 ± 2.86	23.9 ± 3.09	23.6 ± 2.12	23.6 ± 2.61
Body fat (kg)	18.3 ± 4.98	17.4 ± 5.10	18.9 ± 4.47	17.3 ± 4.09
Body fat ratio (%)	27.8 ± 6.11	27.5 ± 5.89	28.6 ± 5.18	27.84 ± 5.35
Diabetic duration (yrs)	5.38 ± 3.61	6.51 ± 7.77	-	-

Values are mean ± SD.
All average value are not significantly different among diabetic group and normal group.

Table 2. Effects of zinc supplementation on improvement of zinc deficiency

Variables	Diabetic supplement (n=44)	Diabetic Control (n=32)	Normal Supplement (n=32)	Normal Control (n=40)
Marginal plasma Zn deficiency (%)				
Before	41.9	46.9	22.6	24.3
After	22.5	43.8	19.4	19.4
Marginal urinary Zn deficiency (%)				
Before	33.3	20.0	31.9	32.3
After	0.00	33.3	6.25	40.0

zinc supplement. The average age of the zinc supplemented diabetics was 58.7 ± 10.1 years and that of the diabetic control group was 58.5 ± 11.7 years. The average age of the zinc supplemented normal adults group was 53.1 ± 11.7 years and that of the diabetic control group was 49.6 ± 10.7 years. There were no significant differences in baseline physical characteristics such as age, height, weight, body mass index (BMI), circumferences of waist and hip, or body fat (%) between the diabetic and the non-diabetic adults groups. All of these variables were not significantly different between the supplemented and the control group in diabetic or non-diabetic subjects. Diabetic duration was 5.38 ± 3.61 years for the diabetic supplemented group, 6.51 ± 7.77 years for the diabetic control group. All the physical characteristics were similar between subgroups of the subjects classified by diabetic duration.

Table 2 shows the incidence of marginal zinc deficiency at baseline by plasma zinc level and urinary excretion. In the diabetic group, more than 40% of the subjects had marginal zinc deficiency by plasma zinc level. By contrast, the incidence of marginal zinc deficiency was 22.6% in the normal supplemented, and 24.3% in the normal control adults. After 4 weeks of zinc supplementation, incidence of marginal zinc deficiency decreased to 22.5% in the diabetic supplement group. When the incidence of zinc deficiency was evaluated by urinary zinc level, 33.3% of the diabetic supplement group, and 20% of the diabetic control group were diagnosed to have marginal zinc deficiency at baseline. More than 30% of normal adults were found to have marginal zinc deficiency by urinary zinc excretion.

Table 3. Effects of zinc supplementation on indices of glycemic control

Variables	Diabetic Supplement (n=44)	Diabetic Control (n=32)	Nondiabetic Supplement (n=32)	Nondiabetic Control (n=40)
HbA_{1C} (%)				
Before	7.62 ± 1.25	7.87 ± 1.39	5.84 ± 0.41	5.69 ± 0.43
After	7.53 ± 1.19	7.85 ± 1.63	5.67 ± 0.42	5.64 ± 0.39
P value	NS	NS	**	NS
Fasting glucose (mg/dl)				
Before	143.0 ± 47.48	149.0 ± 66.96	91.2 ± 12.13	89.7 ± 13.73
After	131.5 ± 44.51	135.5 ± 41.76	95.5 ± 18.99	85.9 ± 8.88†
P value	0.068	NS	NS	*
Insulin (uIU/ml)				
Before	9.1 ± 4.40	13.7 ± 5.60	9.5 ± 3.38	10.0 ± 6.01
After	11.3 ± 3.62	12.1 ± 6.91	11.3 ± 7.38	8.5 ± 3.83†
P value	0.068	NS	0.087	*
C-peptide (ng/mg)				
Before	1.84 ± 0.62	2.14 ± 1.86	1.87 ± 0.77	1.80 ± 0.79
After	2.01 ± 0.85	2.23 ± 1.60	1.99 ± 0.96	1.65 ± 0.51
P value	0.06	NS	NS	NS

Values are mean ± SD.
* p<0.05, **p<0.01 by paired t-test between before and after treatment within group
† p<0.05 by ANCOVA between supplement and control group

Table 3 compares the baseline results of HbA_{1c}, fasting blood glucose, insulin, and C-peptide, which are possible indicators of glycemic control and insulin resistance, in diabetic and non-diabetic subjects. All of these variables at baseline were not significantly different between the supplemented and the control groups in diabetic or non-diabetic subjects. The fasting blood glucose and HbA_{1c} in the diabetic group were significantly higher than the values of the normal group. However, there were no significant differences in insulin and C-peptide levels between the two groups. Serum levels of insulin and C-peptide in diabetic subjects were within normal ranges (insulin : 2~25 uIU/mL and C-peptide : 0.48~3.30 ng/mL). The average HbA_{1c} of diabetics was higher than the normal range (4~6%), however, it did not belong to the level of action suggested for the treatment (>8.0%) (Palumbo, 2001).

The representative long term indicator, HbA_{1c} did not change by 4 weeks zinc supplementation as is shown in Table 3. While HbA_{1c} of the diabetic group slightly decreased from 7.62% to 7.53% following zinc supplementation, without statistical significance, no changes occurred in diabetic control groups. By contrast, a significant decrease of HbA_{1c} was observed for zinc supplemented normal adults after 4 weeks of supplementation. While the fasting plasma glucose level decreased from 143.1 mg/dL to 131.5 mg/dL (p=0.068) in diabetic patients, serum levels of insulin as well as C-peptide were slightly increased after zinc supplementation (p=0.068, p=0.06).

When the effect of zinc supplementation on glycemic control was further analyzed by the baseline HbA_{1c} level of the diabetic subjects (HbA_{1c}<7.5% vs HbA_{1c}≥7.5%) as shown in Table 4, significant decreases (p<0.05) of fasting glucose and HbA_{1c} were

Table 4. Effects of Zn supplementation on indices of glycemic control by HbA_{1c} level

Variables	Diabetic Supplement		Diabetic Control	
	HbA_{1c}		HbA_{1c}	
	7.5% ≤ (n=22)	< 7.5% (n=22)	7.5% ≤ (n=18)	< 7.5% (n=14)
HbA_{1c} (%)				
Before	8.57 ± 1.03	6.67 ± 0.52§§§	8.77 ± 1.16	6.71 ± 0.49§§§
After	8.30 ± 1.18	6.79 ± 0.60†	8.78 ± 1.55	6.65 ± 0.63
P-value	*	0.069	NS	NS
Glucose (mg/dl)				
Before	169.7 ± 49.47	116.4 ± 26.02§§§	177.5 ± 75.87	112.3 ± 24.27§§
After	150.7 ± 46.89	113.1 ± 33.83	153.94 ± 40.75	112.0 ± 30.33
P-value	*	NS	NS	NS
Insulin (uIU/ml)				
Before	9.5 ± 5.64	8.7 ± 2.73	12.1 ± 10.66	13.7 ± 5.60
After	11.7 ± 0.08	10.8 ± 3.15	10.3 ± 4.95	14.48 ± 8.44
P-value	0.095	***	NS	NS
C-peptide (ng/mg)				
Before	2.01 ± 0.75	1.67 ± 0.42	1.80 ± 1.16	2.56 ± 2.48
After	2.23 ± 0.92	1.80 ± 0.75	1.91 ± 0.96	2.64 ± 2.14
P-value	0.079	NS	NS	NS

Values are mean ± SD.
* p<0.05, ** p<0.01, *** p<0.001 by paired t-test between before and after treatment within group
† p<0.05 by ANCOVA between supplement and control group
§ p<0.05, §§ p<0.01 §§§ p<0.001 by student t- test

Table 5. Effects of Zn supplementation on indices of glycemic control by diabetic duration

Variables	Diabetic Supplement		Diabetic Control	
	Diabetic duration		Diabetic duration	
	4 years ≤ (n=24)	< 4 years (n=13)	4 years ≤ (n=9)	< 4 years (n=12)
HbA_{1c} (%)				
Before	7.90 ± 1.17	7.30 ± 1.30	8.26 ± 1.10	7.72 ± 1.50
After	7.69 ± 1.19	7.34 ± 1.20	8.16 ± 0.78	7.73 ± 1.87
P-value	0.076	NS	NS	NS
Fasting glucose (mg/dl)				
Before	145.4 ± 46.34	140.3 ± 49.87	177.4 ± 84.76	137.9 ± 56.98
After	133.6 ± 46.90	129.1 ± 42.67	152.4 ± 37.85	129.0 ± 42.12
P-value	NS	*	NS	NS
Insulin (uIU/ml)				
Before	8.6 ± 2.79	9.7 ± 5.80	13.8 ± 3.68	12.8 ± 6.50
After	11.9 ± 3.20	10.6 ± 4.02	10.1 ± 4.60	12.92 ± 7.57
Significance	***	NS	NS	NS
C-peptide (ng/mg)				
Before	1.89 ± 0.65	1.78 ± 0.59	1.37 ± 0.58	2.44 ± 2.11
After	2.12 ± 0.78	1.88 ± 0.93	1.38 ± 0.65	2.56 ± 1.74
P-value	*	NS	NS	NS

Values are mean ± SD.
* p<0.05, ** p<0.01, *** p<0.001 by paired t-test between before and after treatment within group

found in the higher HbA_{1c} group after 4 weeks of supplementation. HbA_{1c} level changed from 8.57% to 8.30% and fasting glucose changed from 169.7 mg/dL to 150.7 mg/dL. Marginal changes

Table 6. Effects of Zn supplementation on indices of glycemic control by plasma Zn status

Variables	Diabetic Supplement		Diabetic Control	
	Plasma Zn		Plasma Zn	
	Marginal (n=18)	Normal (n=25)	Marginal (n=15)	Normal (n=17)
HbA_{1c} (%)				
Before	7.54 ± 1.08	7.76 ± 1.34	8.03 ± 1.58	7.73 ± 1.23
After	7.39 ± 1.08	7.70 ± 1.25	8.06 ± 1.91	7.67 ± 1.37
P-value	NS	NS	NS	NS
Fasting glucose (mg/dl)				
Before	126.6 ± 38.2	157.4 ± 49.4§	161.3 ± 79.2	138.2 ± 54.2
After	112.2 ± 40.5	147.7 ± 42.1	143.5 ± 42.2	128.7 ± 41.4
P-value	*	NS	NS	NS
Insulin (uIU/ml)				
Before	9.4 ± 2.55	8.9 ± 5.47	11.8 ± 4.43	13.4 ± 11.2
After	11.3 ± 2.88	11.4 ± 4.18	11.7 ± 8.03	12.5 ± 5.97
P-value	*	*	NS	NS
C-peptide (ng/mg)				
Before	1.63 ± 0.33	2.00 ± 0.74	1.55 ± 0.86	2.65 ± 2.34
After	1.99 ± 0.68	2.05 ± 0.99	1.95 ± 2.04	2.47 ± 1.09
P-value	*	NS	NS	NS

Values are mean ± SD.
* p<0.05, ** p<0.01 by paired t-test between before and after treatment within group
§ p<0.05 by student t-test between supplement and control group

of insulin and C-peptide were also shown in higher HbA_{1c}.

In order to figure out whether glycemic control indicators respond to the zinc supplementation differently by diabetic duration, we compared the changes of HbA_{1c}, fasting glucose, insulin and C-peptide by the diabetic duration period as shown in Table 5. The fasting glucose was significantly reduced, after supplementation, among diabetics with less than 4 years of duration (Table 5). Meanwhile, insulin and C-peptide were significantly increased by supplementation in diabetics with more than 4 years of duration ($p<0.001$, $p<0.05$). Marginal improvement with supplementation was observed for HbA_{1c} ($p=0.076$). The changes of glycemic control indicators after zinc supplementation was compared by plasma zinc levels of the subjects as presented in Table 6. When we compared the HbA_{1c}, fasting plasma glucose, insulin and C-peptide between the two groups classified by baseline plasma level of zinc, significant changes in fasting blood glucose, insulin and C-peptide were observed for zinc supplemented diabetics with lower zinc status ($p<0.05$).

Discussion

The Korean National Health and Nutrition Examination Survey indicated that the prevalence of diabetes mellitus has continuously increased in Korea (Ministry of Health & Welfare, 2006). Accordingly, public awareness on dietary practice to control the blood glucose level is growing, because maintaining the blood glucose in the normal range is essential to prevent or reduce the risk for diabetic complication (Kim *et al.*, 1998; Franz *et*

al., 2003). In this study, the average level of HbA_{1c} for diabetic subjects (7.62%, 7.87%) were higher than normal range (5.0~6.0%), whereas that of normal subjects were 5.84% and 5.69. Fasting glucose level of diabetics were above normal range (70~120 mg/L). Both HbA_{1c} and fasting glucose were significantly higher in diabetics than normal subjects ($p<0.001$). These results indicated that most of diabetic participants in this study were not under a tight glycemic control. Most of the diabetic subjects did not participate in a diabetic management program or visit doctors regularly. It seemed that they were not aware of the importance of dietary practice to control blood glucose level. Thus, inappropriate food choices might have resulted in the deterioration of zinc status.

As hyperzincuria has been frequently reported among diabetics, which might be potential indications of zinc deficiency, zinc supplementation has been suggested to ameliorate the metabolic disturbance of diabetics (Salgueiro *et al.*, 2001). Baseline levels of insulin and C-peptide in diabetic subjects were within normal range. Our study showed that serum insulin and C-peptide were significantly increased after zinc supplementation in patients with more than 4 years history of diabetics. Previous investigators have hypothesized that zinc enhances tyrosine kinase phosphorylation in the insulin signal transduction from in vitro studies (Simon & Taylor, 2000). However, it is hard to conclude from our results that the changes of insulin level following 4 weeks of supplementation reflect an improvement in insulin resistance. This is because we did not collect the data on insulin receptor and/or the changes of insulin-stimulated enzyme activities.

In this study, marginal changes of blood glucose level ($p=0.068$) following supplementation was observed in diabetic subjects. An animal study showed that dietary zinc supplementation attenuated fasting hyperglycemia whereas a marginally Zn-deficient diet exacerbated fasting hyperglycemia in db/db mice (Simon & Taylor, 2001).

HbA_{1c} concentration of diabetic patients did not change significantly after 4 weeks of zinc supplementation. Considering the fact that HbA_{1c} reflects 2-3 months of glycemic control, our results are not unexpected. Our findings on the changes of HbA_{1c} after 4 weeks of supplementation are consistent with the previous studies. It has been reported that zinc supplementation did not improve the level of HbA_{1c}, while it was beneficial to maintaining immunity in type 2 patients (Niewoehner *et al.*, 1986). Others also found that six months of zinc supplementation (=30 mg/day) did not modify the level of HbA_{1c} nor glucose homeostasis significantly (Anderson *et al.*, 2001; Roussel *et al.*, 2003).

When we attempted to compare the magnitude of the supplementation effects by the status of glycemic control or diabetic duration, we observed that more significant effects occurred in less controlled groups or those with a longer medical history of diabetes. Higher levels of HbA_{1c} and fasting glucose with longer diabetic duration have been noted by other investigators (Rho & Ko, 1997). Therefore, we suggest that a short-term zinc supplementation may be effective on glycemic control for diabetic patients with higher HbA_{1c}% concentration and shorter history of diabetes.

Significant changes in fasting blood glucose, insulin and C-peptide were observed for zinc supplemented diabetics with lower zinc status ($p<0.05$) in our study. A recent randomized, clinical trial reported that 3 months of zinc supplementation (30 mg/day) for type 2 diabetics may have beneficial effects in elevating serum zinc level, and improving their glycemic control as shown by decreasing their HbA_{1c}% concentration (Al-Maroof & Al-Shabatti, 2006).

In summary, our study indicated that significant improvement of fasting glucose as well as HbA_{1c} were observed in zinc supplemented diabetic patients with shorter diabetic duration, poorer glycemic control, and marginal zinc status. However, further investigation is needed before a firm conclusion could be drawn for the relationship between zinc supplementation and glycemic control. At present, more attention must be paid to improving the zinc status and glycemic control of Korean diabetic patients in order to prevent or minimize complications.

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