Effects of vitamin D supplementation and circuit training on indices of obesity and insulin resistance in T2D and vitamin D deficient elderly women

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INTRODUCTION

The prevalence of diabetes in recent times has doubled compared to ten years ago. Diabetes is spreading like an epidemic around the world, and it is estimated that the number of diabetics will increase to 531 million persons by 2035 [1]. In recent years, vitamin D has attracted attention as an important factor that can improve diabetes. It has been reported that the deficiency of vitamin D in the blood is greatly associated with various metabolic factors related to diabetes such as hyperglycemia and insulin resistance [2]. The deficiency of vitamin D has been implicated as the predictive factor for the occurrence of diabetes [3] and it has been reported that increasing the vitamin D concentration in the blood has an effect on maintaining glucose homeostasis by increasing insulin sensitivity [4].

A number of previous studies associated with diabetes have reported that regular physical activity increases the physical strength of diabetic patients, helps control blood glucose, and prevents the progression from impaired glucose tolerance to type 2 diabetes [5]. Due to this connection, the American Diabetes Association (ADA) recommends aerobic exercise of medium intensity such as 150 minutes of walking per week, or 75 minutes of high intensity aerobic exercise per week for patients with type 2 diabetes [6]. However, according to a study by Plotnikoff et al. [7], only 28% of diabetic patients...
Effects of vitamin D and exercise on obesity and diabetes

perform regular physical activity. The reason for the low exercise practice rate of diabetic patients despite clear recommendations and instructions for aerobic exercise is that for an individual with high obesity, arthritis, disability, or a person with severe diabetic complications, although they understand the advantage of aerobic exercise, it is hard to perform even low intensity exercise such as light walking of about 20-30 minutes a day. In the event that the practice of aerobic exercise decreases and the prevalence of diabetes increases despite the knowledge that aerobic exercise is an effective method, it is considered that the development of exercise training that can be used as a substitute is required. Circuit training, which has generated high interest and develops various physical strength elements simultaneously for short periods, may be a suitable alternative.

As mentioned previously, it is judged that the intake of vitamin D and circuit training will be effective for the improvement of diabetes in elderly women with both diabetes and vitamin D deficiency. However, studies to verify the effectiveness of the complex treatment remain insufficient. Therefore, the authors of this paper intended to investigate the effect of the implementation of vitamin D intake for 12 weeks in addition to circuit exercise training on the body composition, abdominal fat, blood lipid, and insulin resistance index of elderly women with Type 2 diabetes and symptoms of vitamin D deficiency.

**METHODS**

**Research Subjects**

The subjects of this study were 52 persons exhibiting symptoms of diabetes with fasting blood glucose higher than 126 mg·dl⁻¹ and vitamin D deficiency symptoms with 25(OH)D₃ less than 15 ng·ml⁻¹. The subjects were elderly women aged 65 and over located in Y-si in G-do. From a total of 60 subjects selected in the early study, 15 people each were initially assigned to (1) the vitamin D intake and circuit training group, (2) the vitamin D intake group, (3) the circuit training group, and (4) the control group. However, because 2 people in the vitamin intake group, 4 people in the circuit training group, and 2 people in the control group dropped out, only the results for 52 people were included in the final analysis.

The researchers explained the purpose and procedures of this study to the subjects through an orientation before conducting the pretest, and took measures to begin the study after receiving test consent forms from the subjects who wanted to voluntarily participate. All plans for this study were approved by the Seoul National University Bundang Hospital Institution Review Board before beginning the study. The physical characteristics of the subjects are shown in Table 1.

**Measurement Items and Methods**

**Physique and Body Composition**

Height was measured using a normal manual extensometer (Samhwa, Korea). Weight was measured using an electronic balance (CAS-150kg, DW-150, Korea), and the body mass index (BMI) was calculated. Body composition was measured using DXA (dual energy X-ray absorptiometry, Hologic, QDR-4500W, USA), and the measured variables were fat mass, fat free mass, and percent body fat.

**Abdominal Fat**

Abdominal computerized tomography (CT) was performed in the abdomen using ECLDS (Hitachi, Japan). A cross-sectional area of 10mm thickness around the L4-L5 vertebrae was measured with the subject in a supine position, and the abdominal fat area value was obtained using the skeletal radiograph (120Kv, 200mAs). The total fat area (TFA), visceral fat area (VFA), and subcutaneous fat area (SFA) of the abdomen were obtained using this value.

**Blood Analysis**

All subjects arrived at 8:00 am on the day of the test in

| Variables          | Groups (n) | (Mean ± SE) |
|--------------------|------------|-------------|
|                     | D+T (15)   | T (13)      | D (11)    | CON (13) | p  |
| Age (yrs)           | 69.53 ± 0.84 | 68.54 ± 1.18 | 73.27 ± 2.06 | 70.08 ± 1.37 | .114 |
| Height (cm)         | 153.09 ± 0.66 | 152.78 ± 1.25 | 154.12 ± 0.79 | 153.14 ± 1.56 | .345 |
| Body weight (kg)    | 58.59 ± 1.35 | 59.55 ± 2.12 | 57.13 ± 1.52 | 57.13 ± 1.95 | .724 |
| BMI (kg·m⁻²)        | 25.02 ± 0.59 | 25.51 ± 0.77 | 24.08 ± 0.73 | 23.72 ± 0.68 | .244 |
| Fasting glucose (mg·dl⁻¹) | 132.67 ± 5.37 | 127.00 ± 4.34 | 145.09 ± 9.84 | 128.54 ± 7.88 | .294 |
| 25(OH)D₃ (ng·ml⁻¹)  | 11.91 ± 1.66 | 13.05 ± 1.43 | 10.44 ± 1.80 | 11.66 ± 2.80 | .849 |

* D+T: vitamin D intake + Circuit Training group; T: Circuit Training group; D: vitamin D intake group; CON: Control group
the clinical laboratory with fasting state of more than 12 hours. After maintaining the subjects in a stable state for 30 minutes, 20ml of venous blood was sampled from the antecubital vein using an anticoagulant treated syringe. The sampled blood was placed in a tube that was not treated for anticoagulation, and was then centrifuged at 3,000rpm using a centrifugal separator for 10 minutes. After extracting the serum from the cellular components, the serum was put in a storage tube and stored in the refrigerator at -70℃ until analysis.

Blood Lipid

The triglyceride (TG), total cholesterol (TC), HDL-C, and LDL-C concentrations were analyzed using the serum sample. The TG was analyzed with an automatic analyzer (Olympus AU 2700, Olympus, Japan) using the enzymatic option with glycerol blank and sample. The TC was analyzed by the chemical reaction principle using enzyme. The HDL-C analysis measured the color intensity from a color producing reaction after generating hydrogen peroxide using a direct assay. The LDL-C analysis measured the color intensity from a color producing reaction after generating hydrogen peroxide using a direct assay.

Insulin Resistance

The fasting plasma glucose concentration and the fasting insulin concentration were measured from the serum sample. Blood glucose was analyzed using the Automatic Analyzer (Olympus AU 2700, Olympus, Japan) using hexokinase, and insulin was analyzed using an immunoassay equipment (Elecsys, Roche, Swiss) as an electrochemiluminescence immunoassay (ECLIA). In addition, HOMA-IR (homeostasis model for insulin resistance) was calculated using the following equation in order to evaluate insulin resistance [9]. HOMA-IR = [insulin (uU/ml) × blood glucose(mmol/l)] / 22.5

Vitamin D Intake Method and Circuit Training

Vitamin D Intake Method

In order to precisely investigate the effect of vitamin D intake, vitamin D was distributed using the double blind method. The subjects of ①, the vitamin D intake and circuit training group, and ②, the vitamin D intake group were allowed to ingest vitamin D every day for 12 weeks of treatment. The amount of intake was defined as 1,200 IU per day by considering the amount of intake (800-1,000IU/day) and upper limit (< 2,400IU /day) recommended for general adults and diabetic patients [10]. On the other hand, tablets made of cellulose were provided to ③, the circuit training group, and ④, the control group, and the tablets were designed so they were not externally distinguishable from the vitamin D tablets.

Circuit Training

The exercise training conducted in this study consisted of 10 kinds of exercise methods: push-up with knees against the floor, running on the spot, squats, walking in place, good morning exercise, step box, stair-climb, side lunge, high jump with open arms, and leg-lift. The circuit training program lasted for 12 weeks, and aerobic exercises were alternated with weight bearing exercises. This program was presented by Rasmussen et al. [11]. The 10 kinds of movements above were performed without any break time in 1 set, which lasted for 1 minute. The process was controlled so that 60 to 80% HRR (heart rate reserve) was maintained during the exercises, and every 4 weeks, the exercises were performed while monitoring for the target heart rate by wearing a heart rate meter (Polar Electro DY, Finland). The exercise frequency was 3 times a week for the 1st-6th weeks, and 4 times a week for the 7th-12th weeks. The circuit training program applied in this study is the same as shown in Table 2.

Data Processing Method

The results obtained in this study were analyzed using SPSS PC for Windows (version 21.0). The descriptive statistics quantity was presented as the mean and the standard error of the mean (SE). In order to simultaneously analyze the average difference of the dependent variables between the four groups and between two tests, repeated two-way ANOVA was used. If the main effect of the group, the main effect of the test, or the interaction between the groups and the tests were significant, the difference between two tests within each group was verified using one-way ANOVA. The least significant difference (LSD) was calculated for post verification, and the difference between two tests within the same group was verified by paired t-test. In order to determine the rate of change of the pre-test and post-test, delta % [(posttest - pretest) / pretest × 100] was presented, and the significant level (α) of all statistical analysis was 0.05.

**Table 2. Circuit Training Program**

| Stage         | Mode/set | Duration | Intensity  |
|---------------|----------|----------|------------|
| Warm-up       | Stretching | 10 min   | HRR 60~80% |
| Main Exercise | 1~6 week  | 2 set 25 min | 1 set(10 min) |
|               | 7~12 week | 3 set 40 min | Rest(5 min) |
| Cool-Down     | Stretching | 10 min   | 3 times / 1-6 week |
|               |          |          | 4 times / 7-12 week |
RESULTS

Changes in Body Composition

The main effect of the test on weight, fat mass, percent body fat, and BMI were significant, but significant changes in fat mass were not observed in all groups (Table 3).

Changes in Abdominal Fat

The main effect of the test as well as the interaction between the group and the test were significant. However, significant changes were not observed in all variables associated with abdominal fat in the vitamin D intake group and the control group (Table 4).

Table 3. Changes in body composition during 12 weeks of interventions (Mean ± SE)

| Variables          | Groups       | Pre       | Post      | Δ%      | p       |
|--------------------|--------------|-----------|-----------|---------|---------|
| Body weight (kg)   | D+T          | 58.59 ± 1.35 | 57.38 ± 1.32*** | -2.06 | Group .826 |
|                    | T            | 59.55 ± 2.12 | 58.40 ± 2.18*   | -1.93 | Test .000*** |
|                    | D            | 57.13 ± 1.52 | 56.46 ± 1.66*   | -1.18 | Group*Test .127 |
|                    | CON          | 57.13 ± 1.95 | 56.90 ± 2.14    | -0.41 |         |
| Fat free mass (kg) | D+T          | 36.22 ± 0.69 | 36.75 ± 0.72    | 1.45  | Group .757 |
|                    | T            | 36.61 ± 1.11 | 36.33 ± 1.18    | -0.78 | Test .488 |
|                    | D            | 35.40 ± 0.81 | 35.42 ± 0.90    | 0.05  |         |
|                    | CON          | 35.58 ± 0.70 | 35.74 ± 0.82    | 0.43  | Group*Test .249 |
| Fat mass (kg)      | D+T          | 19.69 ± 0.91 | 18.71 ± 0.94**  | -4.94 | Group .830 |
|                    | T            | 20.35 ± 1.14 | 19.37 ± 1.24**  | -4.82 | Test .000*** |
|                    | D            | 18.79 ± 1.24 | 18.38 ± 1.28    | -2.16 |         |
|                    | CON          | 18.87 ± 1.35 | 18.05 ± 1.42**  | -4.37 | Group*Test .327 |
| %body fat (%)      | D+T          | 33.98 ± 0.96 | 32.57 ± 0.97**  | -8.86 | Group .895 |
|                    | T            | 34.39 ± 0.98 | 33.35 ± 1.25*   | -3.04 | Test .000*** |
|                    | D            | 33.31 ± 1.50 | 32.79 ± 1.56    | -1.56 |         |
|                    | CON          | 33.09 ± 1.38 | 31.95 ± 1.54**  | -3.44 | Group*Test .448 |
| BMI (kg·m⁻²)       | D+T          | 25.02 ± 0.59 | 24.50 ± 0.57*** | -2.08 | Group .361 |
|                    | T            | 25.51 ± 0.77 | 24.99 ± 0.74*   | -2.04 | Test .000*** |
|                    | D            | 24.08 ± 0.73 | 23.80 ± 0.80    | -1.16 |         |
|                    | CON          | 23.72 ± 0.68 | 23.62 ± 0.75    | -0.42 | Group*Test .118 |

Table 4. Changes in abdominal fat area during 12 weeks of interventions (Mean ± SE)

| Variables (cm²)  | Groups       | Pre       | Post      | ΔΔ%     | p       |
|------------------|--------------|-----------|-----------|---------|---------|
| TFA (cm²)        | D+T          | 274.30 ± 15.96 | 249.37 ± 17.57*** | -9.09 | Group .840 |
|                  | T            | 295.20 ± 20.22 | 265.87 ± 19.29**  | -9.94 | Test .016*  |
|                  | D            | 276.22 ± 25.72 | 282.06 ± 28.72  | 2.11  |         |
|                  | CON          | 257.12 ± 20.89 | 263.62 ± 23.99  | 2.53  |         |
| VFA (cm²)        | D+T          | 106.88 ± 9.95 | 97.41 ± 8.74    | -8.86 | Group .426 |
|                  | T            | 93.46 ± 7.85 | 86.00 ± 8.28    | -7.98 | Test .206 |
|                  | D            | 113.48 ± 13.77 | 115.50 ± 15.31  | 1.78  |         |
|                  | CON          | 99.78 ± 8.60 | 103.02 ± 10.05  | 3.25  | Group*Test .114 |
| SFA (cm²)        | D+T          | 167.43 ± 13.19 | 151.97 ± 14.75*  | -9.23 | Group .445 |
|                  | T            | 202.71 ± 18.24 | 179.87 ± 18.52*  | -11.27 | Test .032*  |
|                  | D            | 162.75 ± 17.05 | 166.56 ± 17.65  | 2.34  |         |
|                  | CON          | 157.51 ± 16.11 | 160.59 ± 17.36  | 1.96  | Group*Test .023*  |

# D+T: vitamin D intake + Circuit Training group; T: Circuit Training group; D: vitamin D intake group; CON: Control group

* Significantly different between pre- and post-test, * p < .05; ** p < .01
Table 5. Changes in blood lipids during 12 weeks of interventions (Mean ± SE)

| Variables | Groups | Pre | Post | ΔΔ% | P     |
|-----------|--------|-----|------|-----|-------|
| TC (mg·dl⁻¹) | D+T    | 191.73 ± 7.31 | 167.80 ± 6.16** | -12.48 | Group .140 |
|           | T      | 180.77 ± 10.48 | 167.46 ± 8.31* | -7.36 | Test .001** |
|           | D      | 199.55 ± 9.43  | 195.09 ± 9.38  | -2.24 |       |
|           | CON    | 192.85 ± 8.74  | 199.54 ± 10.91 | 3.47  | Group*Test .000** |
| TG (mg·dl⁻¹) | D+T    | 146.13 ± 14.07 | 121.07 ± 14.14* | -17.15 | Group .255 |
|           | T      | 112.46 ± 13.82 | 109.46 ± 14.03 | -2.67 | Test .731 |
|           | D      | 133.27 ± 19.67 | 127.55 ± 17.07 | -4.29 |       |
|           | CON    | 125.38 ± 14.24 | 150.85 ± 16.43 | 20.31 |       |
| HDL-C (mg·dl⁻¹) | D+T   | 53.60 ± 2.49a | 58.13 ± 2.69** | 8.45 | Group .337 |
|           | T      | 49.77 ± 1.80a | 53.08 ± 2.13** | 6.65 | Test .006** |
|           | D      | 56.45 ± 3.57ab | 48.09 ± 3.03** | -14.81 |       |
|           | CON    | 62.08 ± 2.67b  | 51.85 ± 2.89** | -16.48 | Group*Test .001** |
| LDL-C (mg·dl⁻¹) | D+T   | 104.80 ± 5.03  | 94.60 ± 5.14** | -9.73 | Group .057 |
|           | T      | 98.77 ± 6.60  | 90.23 ± 6.06* | -8.65 | Test .252 |
|           | D      | 113.18 ± 7.24  | 116.91 ± 6.29* | 3.3  |       |
|           | CON    | 107.31 ± 5.00  | 114.15 ± 6.54* | 6.37 | Group*Test .001** |

# D+T: vitamin D intake + Circuit Training group; T: Circuit Training group; D: vitamin D intake group; CON: Control group

Table 6. Changes in insulin resistance indices during 12 weeks of intervention (Mean ± SE)

| Variables      | Groups | Pre  | Post  | Δ%    | p   |
|----------------|--------|------|-------|-------|-----|
| Fasting insulin (µU·ml⁻¹) | D+T    | 5.48 ± 1.09 | 3.52 ± 0.55 | -35.77 | Group .771 |
|               | T      | 5.52 ± 0.84  | 4.83 ± 0.67  | -12.5  | Test .488 |
|               | D      | 5.40 ± 1.18  | 4.97 ± 0.90  | -7.96  |       |
|               | CON    | 4.69 ± 0.87  | 6.06 ± 0.97  | 29.21  | Group*Test .275 |
| Fasting glucose (mg·dl⁻¹) | D+T    | 132.67 ± 5.37 | 118.47 ± 5.14 | -10.7  | Group .205 |
|                | T      | 127.00 ± 4.34 | 122.46 ± 5.84 | -3.57  | Test .291 |
|                | D      | 145.09 ± 9.84 | 143.82 ± 11.82 | -0.88  |       |
|                | CON    | 128.54 ± 7.88 | 133.77 ± 10.91 | 4.07  | Group*Test .228 |
| HOMA-IR | D+T    | 1.80 ± 0.36  | 1.03 ± 0.17  | -42.78 | Group .664 |
|          | T      | 1.72 ± 0.26  | 1.50 ± 0.22  | -12.79 | Test .439 |
|          | D      | 1.83 ± 0.37  | 1.71 ± 0.34  | -6.56  |       |
|          | CON    | 1.54 ± 0.31  | 2.00 ± 0.41  | 29.87  | Group*Test .187 |

# D+T: vitamin D intake + Circuit Training group; T: Circuit Training group; D: vitamin D intake group; CON: Control group

**Changes in Blood Lipids**

The main effect of the test as well as the interaction between the group and the test in the TC (p < .001) and HDL-C (p < .001) analyses were significant. The interaction between the group and the test in the TG (p = .031) and LDL-C (p = .001) analyses was also significant (Table 5).

**Changes in Insulin Resistance Index**

The interaction between the group and the test in the insulin, blood glucose, and HOMA-IR analyses were not statistically significant, but they tended toward improvement in the vitamin D intake group and the circuit training group (Table 6).

**DISCUSSION**

**Changes in Body Composition**

It has been reported that the concentration of serum vitamin D has an effect on muscular activity, and also affect the number and size of muscular fibers [12]. It has been reported that in a study investigating the relationship between vitamin D and fat free mass, involving the intake of 1,000 IU of vitamin D a day over 2 years, the size and number of type II muscle fibers increased significantly [13]. Bunout et al. [14] have reported that in a study comparing exercise training and vitamin D intake (400IU/day) over 9 months in elderly...
worn, the fat free mass and the muscle strength in the exercise training group significantly increased, but there were no significant changes in fat free mass and muscle strength in the vitamin D intake group. As described above, contradictory results have been reported for the effect of vitamin D intake on fat free mass. In this study, the change in fat free mass in all groups was not significant. It is judged that the 12 week period of vitamin D intake applied for the elderly human subjects was too short to affect the fat free mass, and it is expected that if the subjects took vitamin D over a longer period in the future, significant changes in fat free mass will be more apparent.

It has been reported that unlike vitamin D intake, regular physical activity effects positive changes in body composition [15]. Exercise training targeting patients with diabetes increase their health level and has positive effects on body composition [5]. On the other hand, there have also been research results that significant changes in body composition were not apparent despite participation in exercise training. Kadoglou et al. [16] have reported that in a study that implemented aerobic exercise at 50-75% of VO2max for 6 months targeting patients with diabetes, a positive change in insulin resistance-related variables was shown, but there was no significant change in body composition. Accordingly, it is considered that exercise at less than medium strength for short periods is somewhat insufficient to induce a change of body composition. Therefore, this study suggests that in the case of diabetic patients, exercise at more than medium strength would be effective to change the body composition.

According to weight and BMI results obtained in this study, the most significant reduction was in the group with vitamin D intake combined with circuit training. However, it is difficult to determine whether a synergistic effect of vitamin D intake and circuit training for body composition was apparent. The mechanism through which the increase in the concentration of vitamin D in the body affects body composition is also difficult to explain, but it is interpreted that the vitamin D receptor (VDR) is activated by the action of 25(OH), and has a positive effect on body composition while improving the homeostasis control ability of calcium [2].

Changes in Abdominal Fat

It is known that accumulation of abdominal fat is the main cause of insulin resistance and metabolic syndrome. It has been reported that the accumulation of abdominal fat increases the secretion of adipocytokine, and has a negative effect on diabetes, hypertension, and cardiovascular diseases [17]. In previous studies, it was reported that vitamin D deficiency often appears in cases of obesity, and the concentration of 25(OH)D shows a negative correlation with abdominal fat [18]. However, there were no significant changes in abdominal fat-related variables in the vitamin D intake group of this study. Similarly, Wamberg et al. [19] have reported that as a result of obese middle-aged women taking vitamin D at 7,000 IU per day over 26 weeks, their blood 25(OH)D concentrations increased to a sufficient level, but there were no significant changes in subcutaneous fat mass and visceral fat mass.

It has been reported that abdominal fat tissue is significantly reduced by exercise training that increases physical activity. In this regard, Bacchi et al. [20] have reported that for middle-aged men and women with diabetes conducting aerobic exercise using a treadmill or cycle at 60-65% of HRR, 60 minutes per day, 3 times per week for 16 weeks, the abdominal visceral fat area and subcutaneous fat area were significantly reduced. In this study, the TFA and SFA were significantly reduced only in two groups conducting circuit training, and these results are similar to the results of previous studies that conducted aerobic exercise. Collectively, the abdominal fat results indicate that the increase of fat metabolism and the reduction of abdominal fat are effectively accomplished when combining exercise that increases direct physical activity with vitamin D intake rather than only independent treatment of vitamin D intake.

Changes in Blood Lipids

It has been reported that vitamin D plays an essential role in the homeostasis control of calcium. Vitamin D was closely connected to the metabolic mechanism of calcium, and increased blood calcium concentration has an effect on blood lipid concentration and insulin resistance [19]. In general, it has been reported that intake of vitamin D decreases LDL-C and TG [21], and increases HDL-C [22]. In this study, blood lipid analysis of the vitamin D intake group showed that HDL-C was significantly decreased, which contrasted with the above study. Heikkinen et al. [23] have reported that as a result of menopausal women taking 300 IU of vitamin D every day for 3 years, their TG and LDL-C concentrations were significantly increased, while TC and HDL-C concentrations were significantly reduced. Major et al. [24] also reported that in obese middle-aged women taking 600 mg of calcium and 200 IU of vitamin D per day for 15 weeks, TC, LDL-C, and HDL-C concentrations were significantly reduced. Unlike the changes in blood lipid according to general estimates of vitamin D intake, the study that reported
the decrease of HDL-C also reported that recording the dietary daily log by a recall method can be inaccurate even when the dietary intake was prepared. It was therefore concluded that difficulty with dietary intake control and alcohol intake may have an effect on blood lipid.

In this study, the change in blood lipid was clearly shown in the two groups undertaking circuit training. Sung & Bae [25] reported that in diabetic elderly men participating in walking exercise at 65-75% of HRmax for 50 minutes a day, 3 times a week for 24 weeks, TC and TG were significantly reduced. Greene et al. [26] also reported that obese middle-aged men and women participating in endurance exercise at 70% of VO2max, 3 times per week for 12 weeks had significant increases in HDL-C and significant decrease in LDL-C levels. The circuit training performed in this study is the form involving aerobic exercise. Since the result of this study showed that the effect of fat mass and abdominal fat on the blood lipid of the two groups that underwent circuit exercise were significantly reduced, it is considered that circuit training is an effective exercise to induce positive changes in blood lipid.

Changes in Insulin Resistance Index

In recent years, a number of studies to improve insulin resistance targeting diabetic patients have reported differences in the insulin resistance index according to vitamin D concentration [27]. Talaei et al. [28] reported that comparing insulin resistance indices according to the concentration of blood 25(OH)D in middle-aged women with diabetes showed significant differences in blood glucose, insulin, and HOMA-IR between people who are normal (>30 ng·ml⁻¹), people who have insufficiency (<30 ng·ml⁻¹), and people with deficiency (<20 ng·ml⁻¹). In this regard, VonHurst et al. [29] have reported that as a result of taking 4,000 IU of vitamin D every day for 26 weeks, the insulin and blood glucose levels of patients with diabetes was significantly reduced. These results suggest that vitamin D intake increases the secretion and sensitivity of insulin, and has an effect on the maintenance of homeostasis [4].

It has been reported that regular physical activity is the most effective method for improving insulin resistance [30]. Umpierre et al. [31] reported that conducting a meta-analysis of 23 research results that involved an aerobic exercise showed that aerobic exercise is effective in improving insulin resistance, regardless of type, strength, time and frequency of training. It could be concluded from these results that aerobic exercise has a positive effect on the insulin resistance index by inducing the oxidation of fatty acids as well as an increase in insulin sensitivity.

Based on the insulin resistance index obtained in this study, it was determined that the results were not statistically significant. The group that combined vitamin D intake and circuit training showed the most positive changes, and the reduction of fat mass and abdominal fat had an effect on improving the insulin resistance indices. It was determined that combining the two kinds of treatments rather than a single treatment involving only vitamin D intake or exercise training is more effective.

CONCLUSIONS

From the results of this study, it was concluded that vitamin D intake and circuit training for 12 weeks would have a positive effect on the abdominal fat and blood lipid of elderly women with vitamin D deficiency and type 2-diabetes. In particular, the most important information obtained in this study is that in situations where vitamin D intake is combined with exercise training, the improvements in body composition, abdominal fat, blood lipid, and insulin resistance index are greater than situations with one single treatment.

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REFERENCES

[1] Ramachandran A, Snehalatha C, Ma RC. Diabetes in South-East Asia: An update for 2013 for the IDF Diabetes Atlas. Diabetes Research and Clinical Practice, 2014;103(2):231-237.
[2] Pittas AG, Lau J, Hu FB, Dawson-Hughes B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. The Journal of Clinical Endocrinology and Metabolism, 2007;92(6): 2017-2029.
[3] Scragg R, Sowers M, Bell C. Third national health and nutrition examination survey: Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the third national health and
nutrition examination survey. Diabetes Care, 2004; 27(12):2813-2818.

[4] Delvin EE. Importance of vitamin D in insulin resistance. Bulletin de l’Académie Nationale de Médecine, 2010; 195(4-5), 1091-1102.

[5] Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud’homme D, Fortier M, Reid RD, Tulloch H, Coyle D, Phillips P, Jennings A, Jaffey, J. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. Annals of Internal Medicine, 2007;147(6):357-369.

[6] American Diabetes Association. Standards of medical care in diabetes-2013. Diabetes Care, 2013;36(1):11-66.

[7] Plotnikoff RC, Taylor LM, Wilson PM, Courneya KS, Sigal RJ, Birkett N, Raine K, Svenson, LW. Factors associated with physical activity in Canadian adults with diabetes. Medicine and Science in Sports and Exercise, 2006;38(8):1526-1534.

[8] Eves ND, Plotnikoff RC. Resistance training and type 2 diabetes: Considerations for implementation at the population level. Diabetes Care, 2006; 29(8): 1933-1941.

[9] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia, 1985;28(7):412-419.

[10] The Korean Nutrition Society. Dietary reference intakes for Koreans. First revision. Seoul: Hanareum planning; 2010.

[11] Rasmussen CR, Kreider C, Kerksick B, Campbell B, Slonaker M, Greenwood J, Baer E, Pfau M, Grimstvedt C, Wilborn A, Thomas L, Autrey T, Magrans B, Marcello C, Mulligan D, Rohle L, Taylor A, Vacanti S, Ounpraseuth P, Casey R. Effects of the Curves? Fitness and Weight Loss Program on Markers of Health. Medicine and Science in Sports and Exercise, 2004;36(5):106-112.

[12] Hamilton B. Vitamin D and human skeletal muscle. Scandinavian Journal of Medicine and Science in Sports, 2010;20(2):182-190.

[13] Sato Y, Iwamoto J, Kanoko T, Satoh K. Low-dose vitamin D prevents muscular atrophy and reduces falls and hip fractures in women after stroke: a randomized controlled trial. Cerebrovascular Diseases, 2005;20(3): 187-192.

[14] Bunout D, Barrera G, Leiva L, Gattas V, de la Maza MP, Avendaño M, Hirsch S. Effects of vitamin D supplementation and exercise training on physical performance in Chilean vitamin D deficient elderly subjects. Experimental Gerontology, 2006;41(8):746-752.
program on behavioral and biochemical aspects in elderly people with type II diabetes. Nursing and Health Sciences, 2012;14(4):438-445.

[26] Greene NP, Martin SE, Crouse, SF. Acute exercise and training alter blood lipid and lipoprotein profiles differently in overweight and obese men and women. Obesity (Silver Spring), 2012;20(8):1618-1627.

[27] Petersen JL, McGuire DK. Impaired glucose tolerance and impaired fasting glucose-a review of diagnosis, clinical implication and management. Diabetes Disorder Research, 2005;2(1):9-15.

[28] Hyppönen E, Power C. Vitamin D status and glucose homeostasis in the 1958 British birth cohort: the role of obesity. Diabetes Care, 2006;29(10):2244-2246.

[29] Talaei A, Mohamadi M, Adgi Z. The effect of vitamin D on insulin resistance in patients with type 2 diabetes. Diabetology and Metabolic Syndrome, 2013;5(1):8-12.

[30] Sukala WR, Page R, Lonsdale C, Lys I, Rowlands D, Krebs J, Leikis M, Cheema BS. Exercise improves quality of life in indigenous Polynesian peoples with type 2 diabetes and visceral obesity. Journal of Physical Activity and Health, 2013;10(5):699-707.

[31] Umpierre D, Ribeiro PA, Kramer CK, Leitão CB, Zucatti AT, Azevedo MJ, Gross JL, Ribeiro JP, Schaan BD. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis. Journal of the American Medical Association, 2011;305(17):1790-1799.