# The association between serum vitamin D status and gait ability in young adults.

## Jong-Hwan Park\*

Institute of Convergence Bio-Health, Dong-A University, Busan, Republic of Korea

#### Abstract

The purpose of the present study was to examine whether serum vitamin D status is associated with a change in gait speed in young adults. In this cross-sectional study, 60 young adults aged  $\geq$  19 years (36 female and 24 male participants) were recruited from the general population. Spatiotemporal parameters of gait were assessed by using a validated wireless inertial sensing device. The blood vitamin D status was classified as deficiency (<12.5 ng/ml), insufficiency ( $\geq$  12.5 ng/ml to <20 ng/ml), and sufficiency ( $\geq$  20 ng/ml). Linear increases in physical performance parameters such as grip strength, gait speed, stride length, and High-Density Lipoprotein Cholesterol (HDL-C) were found across the incremental categories of serum 25-hydroxyvitamin D (25(OH)D). A significant positive association was observed between serum 25(OH)D levels and HDL-C (r=0.759, P=0.001), grip strength (r=0.329, P=0.011), gait speed (r=0.873, P=0.001), stride length (r=0.690, P=0.001), and swing phase (r=0.322, P=0.013). A decline in gait speed was more likely to be associated with serum 25(OH)D deficiency (Odds Ratio: (OR), 13.1; 95% Confidence Interval (CI), 3.84-45.02; adjusted OR, 13.9; 95% CI, 3.61-53.7). To our knowledge, this cross-sectional study is the first to report that higher serum 25(OH)D levels are associated with gait ability and lipid parameters in young adults. Multiple regression analysis showed that vitamin D status is an independent predictive factor for a decline in gait speed.

Keywords: Serum 25-hydroxy Vitamin D, Gait speed, Physical performance.

Accepted on August 4, 2016

## Introduction

The incidence of serum 25-hydroxyvitamin D (25(OH)D) deficiency has gradually increased; vitamin D deficiency may be a direct risk factor in various metabolic diseases including obesity [1], diabetes [2], metabolic syndrome [3], and cardiovascular disease [4]. Sunlight exposure, diet, and supplements are well-known contributors to serum vitamin D status. Most of the vitamin D in the body is produced by cutaneous synthesis in response to sunlight exposure. However, the increasingly sedentary lifestyles of young adults, with more time spent indoors, lead to insufficient exposure to the sunlight that is needed for adequate cutaneous production of vitamin D [3].

Slow timed gait is a strong predictor of the onset of disability involving basic activities of daily living [5] and may represent an early stage in the process of progression of disability [6,7]. Moreover, a decline in gait speed is distinctly observed in those >60 years of age [8] and is caused by a decrease in physical function such as in the joint range of motion and a decrease in muscle strength due to inactivity [9-11]. A previous study reported that low serum vitamin D status is linked to a decline in the aging-related mobility [12].

Furthermore, observational studies have demonstrated clinical relationships between vitamin D serum status and physical

performance. The InCHIANTI (Invecchiare in Chianti) study demonstrated a significant association between low levels of vitamin D and poor physical performance as assessed through handgrip strength and a short physical performance battery test in 966 participants (435 men and 531 women) with a mean age of 75 years [13]. Participants with serum vitamin D levels of <25 nmol/L performed worse than those with a level >25 nmol/L. Muscle strength measured by means of a handgrip test was also significantly greater in subjects with vitamin D levels >50 nmol/L than in those with levels <50 nmol/L [13]. Although these studies showed an association between serum vitamin D status and physical performance in older adults, it remains unclear whether serum vitamin D status is associated with gait speed in young adults. It is important to address this issue because of the lifestyles of young adults involving insufficient physical activity and deficient exposure to sunlight due to insufficient outdoor activities.

Therefore, the purpose of the present study was to examine whether serum vitamin D status is associated with a change in gait speed in young adults.

## **Materials and Methods**

A total of 69 young adults aged  $\geq$  19 years (40 female and 29 male participants) provided their written informed consent to

participate in this study. The participants were recruited from the general population (i.e., none of them were trained athletes competing in any sporting events, although some participants were recreationally active) of a university community. To support health promotion in the university community, we accepted all participants who enrolled in this study if they met the study criterion of having no difficulties in activities of daily living. However, in this cross-sectional study, nine participants were excluded from data analysis (but not for their feedback) for the following reasons: taking lipid- and/or glucoselowering medication (n=5) and performing strenuous physical activities in the 48 h preceding the blood collection (n=4). Thus, the data of 60 participants (36 female and 24 male participants between the ages of 19 and 25 years) were analysed and presented here. Approval was obtained from the ethics review board of our university. By using post-hoc sample size calculation, a minimum total sample size of 58 was determined.

 Table 1. Baseline characteristics of the participants by serum 25(OH)D categories.

Variables	Serum vitamin D status		
	Deficiency (<12.5 ng/ml, n=19)	Insufficiency ( $\geq$ 12.5 ~ <20 ng/ml, n=22)	Sufficiency (≥ 20 ng/ml, n=19)
Gender, male/female	5/14	8/14	11/8
Age (years)	20.8 ± 1.9	19.7 ± 1.7	19.5 ± 1.4
Body mass (kg)	59.8 ± 10.2	62.8 ± 10.5	63.3 ± 12.5
Height (m)	1.67 ± 0.08	1.69 ± 0.08	1.70 ± 0.10
BMI (kg/m <sup>2</sup> )	21.3 ± 2.4	21.9 ± 2.5	21.7 ± 2.5
Body fat (%)	22.2 ± 5.5	21.7 ± 4.9	20.8 ± 4.9
LBM (kg)	46.4 ± 8.3	49.3 ± 9.7	50.1 ± 10.2
SBP (mmHg)	111.6 ± 11.2	113.5 ± 13.0	119.1 ± 17.3
DBP (mmHg)	65.1 ± 6.7	69.4 ± 8.7	66.1 ± 10.4
TC (mg/dl)	165.8 ± 16.5	164.3 ± 9.1	147.5 ± 7.5**
TG (mg/dl)	88.7 ± 4.9	71.0 ± 8.6	63.7 ± 16.3**
HDL-C (mg/dl)	53.1 ± 5.1	65.3 ± 3.4	73.3 ± 10.2**
LDL-C (mg/dl)	79.3 ± 11.9	77.9 ± 7.8	69.8 ± 9.4**
Glucose (mg/dl)	67.2 ± 9.2	71.1 ± 10.5	68.9 ± 10.9
Grip strength (kg)	19.6 ± 4.8	71.1 ± 10.5	68.9 ± 10.9**
Gait speed (m/min)	68.1 ± 3.2	75.9 ± 2.3	79.6 ± 2.9**
Stride length (m)	1.22 ± 0.10	1.33 ± 0.09	1.43 ± 0.11
Stance phase (% of the gait cycle)	65.4 ± 6.2	63.9 ± 2.0	62.8 ± 1.5
Swing phase (% of the gait cycle)	34.9 ± 6.3	36.3 ± 1.6	37.5 ± 1.6

Values are mean ± SD. BMI: Body Mass Index; LBM: Lean Body Mass; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TC: Total Cholesterol; TG: Triglyceride; HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol.

Body weight and height were measured to the nearest 0.1 kg and 0.1 cm, respectively, by using a Venus 5.5 body composition analyser (Jawon Medical, Gyeongsan, Korea). Body Mass Index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Waist circumference was measured to the nearest 0.1 cm at the level of the umbilicus, by using a flexible plastic tape while the participants were in the standing position.

Arterial blood pressure was measured in the right arm in a seated position, by using a standard mercury sphygmomanometer (605P; Yagami Co. Ltd., Nagoya, Japan).

The participants sat on a chair for 5 min before measurement. Two measurements were taken at each time point, and the mean of these values was recorded.

Spatiotemporal parameters of gait were assessed by using a validated [14] wireless inertial sensing device (BTS Bioengineering, Milan, Italy) attached to the subject's waist with a semi-elastic belt, covering the L4-L5 intervertebral space, in order to acquire acceleration values for three anatomical axes: the anteroposterior, medial-lateral, and vertical axes. The subjects were asked to walk along an 8 m pathway at a self-selected speed as naturally as possible. The

collected data were transmitted via Bluetooth to a personal computer and processed using dedicated software (BTS G-Walk).

Grip strength was measured by using a dynamometer (Takei TKK 5401; Takei Scientific Instruments Co. Ltd., Tokyo, Japan). The maximum strength (in kg) in two attempts with the dominant hand was used.

After a 48 h period of physical activity avoidance and fasting (i.e., overnight fast of at least 10 h), venous blood samples were taken from an antecubital vein. The blood samples were analysed for glucose, serum Total Cholesterol (TC), Triglyceride (TG), High-Density Lipoprotein Cholesterol (HDL-C), and Low-Density Lipoprotein Cholesterol (LDL-C). For TC, TG, HDL-C, and LDL-C, samples were collected in tubes containing clotting activators for the isolation of serum. Thereafter, samples were allowed to clot for 45 min at room temperature and then centrifuged at 3000 rpm for 10 min at 4°C. After separation, serum was dispensed into plain microtubes and stored at -80°C for later analysis. Concentrations of serum TC, TG, HDL-C, LDL-C, and plasma glucose were determined according to standard laboratory methods.

The serum vitamin D levels were determined by using a LIAISON 25(OH) vitamin D direct competitive chemiluminescence immunoassay on an automated analyser (DiaSorin S.P.A, Saluggia, Italy). The blood vitamin D status was classified as deficiency (<12.5 ng/ml), insufficiency ( $\geq$ 12.5 ng/ml to <20 ng/ml), and sufficiency ( $\geq$  20 ng/ml) [3].

Data were analysed by using the IBM SPSS Statistics version 22.0 software package for Windows (SPSS Inc., Chicago, IL, USA). Linear trends were assessed with the Kruskal-Wallis test (two-tailed) for the median value in each category of serum vitamin D status. Spearman correlation coefficients were calculated between serum vitamin D and lipid parameters, and between serum vitamin D and physical fitness parameters. Partial correlation coefficients were also calculated between serum vitamin D and both lipid and physical fitness parameters, with adjustment for sex. Forced-entry adjusted logistic regression was conducted to estimate the Odds Ratios (ORs) for gait speed with no adjustment (Model 1) and with other potential confounders such as sex and Lean Body Mass (LBM) (Model 2). Statistical significance was accepted at the 5% level. The results are presented as mean  $\pm$  standard deviation (SD).

## Results

Table 1 shows the physical and biochemical characteristics and the physical performance of the study participants. There were linear decreases in TC, TG, and LDL-C, and a linear increase in the HDL-C categories across serum 25(OH)D levels. Linear increases in physical performance parameters such as grip strength, gait speed, and stride length were found across the incremental categories of serum 25(OH)D. There was also a linear increase for HDL-C across the incremental serum 25(OH)D categories. No significant linear increases or decreases were found in body mass, height, BMI, body fat, LBM, systolic blood pressure, and diastolic blood pressure across the incremental serum 25(OH)D categories.

Table 2 shows the partial correlation coefficients between serum 25(OH)D levels and the parameters measured in this study. Serum 25(OH)D was significantly correlated with TC (r=-0.656, P=0.001), TG (r=-0.654, P=0.001), HDL-C (r=0.829, P=0.001), and LDL-C (r=-0.376, P=0.003). Serum 25(OH)D levels were also significantly correlated with grip strength (r = 0.431, P = 0.001), gait speed (r=0.873, P=0.001), and stride length (r=0.669, P=0.001). After adjusting for sex, serum 25(OH)D remained inversely correlated with TC (r=-0.582, P=0.001), TG (r=-0.599, P=0.001), LDL-C (r=-0.407, P=0.001), and stance phase (r=-0.296, P=0.023). A significant positive association was also observed between serum 25(OH)D levels and HDL-C (r=0.759, P=0.001), grip strength (r=0.329, P=0.011), gait speed (r=0.873, P=0.001), stride length (r=0.690, P=0.001), and swing phase (r=0.322, P=0.013).

**Table 2.** Correlations between serum 25(OH)D and both lipid and physical fitness parameters.

Variable	Unadjusted	Adjusted
	r	r
TC (mg/dl)	-0.656**	-0.582**
TG (mg/dl)	-0.645**	-0.599**
HDL-C (mg/dl)	0.829**	0.759**
LDL-C (mg/dl)	-0.376**	-0.407**
Glucose (mg/dl)	0.018	-0.039
Grip strength (kg)	0.933**	0.914**
Gait speed (m/min)	0.873**	0.873**
Stride length (m)	0.669**	0.690**
Stance phase		
(% of the gait cycle)	-0.226	-0.296*
Swing phase		
(% of the gait cycle)	0.206	0.322*

Spearman correlations were used to calculate coefficients of correlation between vitamin D levels, and lipid and physical fitness parameters.

Statistical analysis was conducted by using partial correlations, with adjustment for  $\ensuremath{\mathsf{sex}}$ 

Values are mean ± SD. BMI: Body Mass Index; LBM: Lean Body Mass; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TC: Total Cholesterol; TG: Triglyceride; HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol.

The ORs for declines in gait speed are presented in Table 3 according to the serum 25(OH)D levels. In Model 1 (unadjusted), gait speed decline was more likely to be associated with serum 25(OH)D deficiency (OR, 13.1; 95% Confidence Interval [CI], 3.84-45.02). In Model 2 (adjusted for sex and LBM), gait speed decline was still more likely to be

associated with vitamin D deficiency (OR, 13.9; 95% CI, 3.61-53.7).

 Table 3. OR (95% CI) of gait speed decline according to serum 25(OH)D level.

Gait speed				
	Model 1	Model 2		
	OR (95% CI)	OR (95% CI)		
Vitamin D level				
Sufficiency	Ref.	Ref.		
Deficiency	13.1 (3.84-45.02)	13.9 (3.61-53.7)		
OR: Odds Ratio; 0	CI: Confidence Interval.			
Model 1 is not adj	usted.			

Model 2 is adjusted for gender and lean body mass.

# Discussion

To the best of our knowledge, the present study is the first to report the relationship between serum vitamin D status and gait ability in young adults. The main finding of the present study is that serum 25(OH)D levels were positively correlated with gait speed, suggesting that physically active young individuals, at least in our study population, had higher 25(OH)D levels.

In the present study, we observed that gait speed decline was more likely to be associated with serum 25(OH)D deficiency in voung adults. This is significant because a gait speed <0.8 m/s is an important predictor of survival [15,16]. Our findings are consistent with the data from a previous study examining the relationship between serum vitamin D and gait speed in older adults [12]. However, our study is not consistent with a study of elderly subjects that demonstrated that vitamin D insufficiency was not related to physical performance, as assessed on the basis of gait speed and handgrip strength [17]. This discrepancy may be due to differences in the age of participants between the studies, as the serum vitamin D levels were higher in the present study than in the previous study. We also observed that serum 25(OH)D levels were positively correlated with grip strength. In another study including young women 19-29 years old, there was a correlation between vitamin D level and handgrip strength in both the dominant and nondominant arms [18]. Thus, it seems reasonable to speculate that serum vitamin D sufficiency partially reflects the increased risk of a decline in physical performance in young adults.

We also showed that the serum 25(OH)D level was negatively correlated with serum TG and plasma TC concentrations. Indeed, TC and TG are strong independent risk factors for cardiovascular disease [19], suggesting that serum 25(OH)D sufficiency might reduce the risk of cardiovascular disease by keeping TC and TG concentrations low. The present study also found that the serum 25(OH)D level was positively correlated with the serum HDL-C concentration. These observations are consistent with the data from a review article [20] indicating

that individuals with higher vitamin D levels have significantly higher concentrations of HDL-C than those with lower levels.

Because of the design of the present study, it was not possible to determine the mechanisms by which the serum 25(OH)D level affects gait ability and lipid parameters. Nonetheless, the current understanding has highlighted the importance of a direct effect of vitamin D on muscle strength and function in explaining this association. Because vitamin D has an effect on type 2 muscle fibers, it was tempting to postulate a protective effect of vitamin D against falls through improvement in muscle function [21,22]. Vitamin D also has some antiatherogenic effects; inhibiting the formation of foam cells and cholesterol uptake by macrophages and enabling HDL transport [23]. Lower serum 25(OH)D was associated with the metabolic syndrome and its components, especially the HDL-C concentration [24]. In addition, the role of vitamin D supplementation therapy in improving cardiovascular outcome in patients with low levels of vitamin D remains to be determined [25].

The present study had limitations. First, owing to its crosssectional design, the present findings are inherently limited in their ability to eliminate causal relationships between serum vitamin D status and gait ability. Second, the sample size was small, which limits our ability to determine the significance of the results. Therefore, additional studies with larger sample sizes are required to clarify the relationships observed in this study. Finally, the study lacks information about sunlight exposure, indoor or outdoor activity patterns, dietary vitamin D or calcium, and menstrual cycle for female participants.

# Conclusions

In conclusion, the present cross-sectional study is the first to report that higher serum 25(OH)D levels are associated with gait ability and lipid parameters in young adults. Multiple regression analysis showed that vitamin D status is an independent predictive factor for a decline in gait speed. Our data suggest that decline of gait ability may be prevented by ensuring vitamin D sufficiency. It would be interesting to investigate whether dietary interventions can be used to slow the progression of frailty in adults.

# Acknowledgments

This work was supported by the Dong-A University research fund.

## References

- Pereira-Santos M, Costa PR, Santos CA, Santos DB, Assis AM. Obesity and vitamin D deficiency: is there an association? Obes Rev 2016; 17: 484.
- 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. J Clin Endocrinol Metab 2007; 92: 2017-2029.

- Ha CD, Han TK, Lee SH, Cho JK, Kang HS. Association between serum vitamin D status and metabolic syndrome in Korean young men. Med Sci Sports Exerc 2014; 46: 513-519.
- Mozos I, Marginean O. Links between Vitamin D Deficiency and Cardiovascular Diseases. Biomed Res Int 2015; 2015: 109275.
- Onder G, Penninx BW, Ferrucci L, Fried LP, Guralnik JM, Pahor M. Measures of physical performance and risk for progressive and catastrophic disability: Results from the Womens Health and Aging Study. J Gerontol A Biol Sci Med Sci 2005; 60: 74-79.
- Guralnik JM, Ferrucci L, Simonsick EM, Salive ME, Wallace RB. Lower-extremity function in persons over the age of 70 years as a predictor of subsequent disability. N Engl J Med 1995; 332: 556-561.
- Verbrugge LM, Jette AM. The disablement process. Soc Sci Med 1994; 38: 1-14.
- Lauretani F, Russo CR, Bandinelli S, Bartali B, Cavazzini C. Age-associated changes in skeletal muscles and their effect on mobility: an operational diagnosis of sarcopenia. J Appl Physiol 2003; 95: 1851-1860.
- 9. Bohannon RW. Comfortable and maximum walking speed of adults aged 20-79 years: reference values and determinants. Age Ageing 1997; 26: 15-19.
- 10. Sturnieks DL, St George R, Lord SR. Balance disorders in the elderly. Neurophysiol Clin 2008; 38: 467-478.
- Buchner DM, Cress ME, Esselman PC, Margherita AJ, de Lateur BJ. Factors associated with changes in gait speed in older adults. J Gerontol A Biol Sci Med Sci 1996; 51: M297-302.
- Kositsawat J, Barry LC, Kuchel GA. C-reactive protein, vitamin D deficiency, and slow gait speed. J Am Geriatr Soc 2013; 61: 1574-1579.
- Houston DK, Cesari M, Ferrucci L, Cherubini A, Maggio D. Association between vitamin D status and physical performance: the InCHIANTI study. J Gerontol A Biol Sci Med Sci 2007; 62: 440-446.
- 14. Bugane F, Benedetti MG, Casadio G, Attala S, Biagi F, Manca M, Leardini A. Estimation of spatial-temporal gait parameters in level walking based on a single accelerometer: Validation on normal subjects by standard gait analysis. Comput Methods Programs Biomed 2012; 108: 129-137.
- 15. Abellan van Kan G, Rolland Y, Andrieu S, Bauer J, Beauchet O, Bonnefoy M, Cesari M, Donini LM, Gillette Guyonnet S, Inzitari M, Nourhashemi F, Onder G, Ritz P, Salva A, Visser M, Vellas B. Gait speed at usual pace as a

predictor of adverse outcomes in community-dwelling older people an International Academy on Nutrition and Aging (IANA) Task Force. J Nutr Health Aging 2009; 13: 881-889.

- 16. Studenski S, Perera S, Patel K, Rosano C, Faulkner K. Gait speed and survival in older adults. JAMA 2011; 305: 50-58.
- 17. Mathei C, Van Pottelbergh G, Vaes B, Adriaensen W, Gruson D. No relation between vitamin D status and physical performance in the oldest old: results from the Belfrail study. Age Ageing 2013; 42: 186-190.
- von Hurst PR, Conlon C, Foskett A. Vitamin D status predicts hand-grip strength in young adult women living in Auckland, New Zealand. J Steroid Biochem Mol Biol 2013; 136: 330-332.
- Despres JP, Lamarche B, Mauriege P, Cantin B, Dagenais GR. Hyperinsulinemia as an independent risk factor for ischemic heart disease. N Engl J Med 1996; 334: 952-957.
- 20. Halfon M, Phan O, Teta D. Vitamin D: a review on its effects on muscle strength, the risk of fall, and frailty. Biomed Res Int 2015; 2015: 953241.
- 21. Ceglia L, Harris SS. Vitamin D and its role in skeletal muscle. Calcif Tissue Int 2013; 92: 151-162.
- 22. Korkmaz N, Tutoglu A, Korkmaz I, Boyaci A. The Relationships among Vitamin D Level, Balance, Muscle Strength, and Quality of Life in Postmenopausal Patients with Osteoporosis. J Phys Ther Sci, 2014, 26: 1521-1526.
- 23. Oh J, Weng S, Felton SK, Bhandare S, Riek A, Butler B, Proctor BM, Petty M, Chen Z, Schechtman KB, Bernal-Mizrachi L, Bernal-Mizrachi C. 1,25(OH)2 vitamin D inhibits foam cell formation and suppresses macrophage cholesterol uptake in patients with type 2 diabetes mellitus. Circulation 2009; 120: 687-698.
- 24. Maki KC, Rubin MR, Wong LG, McManus JF, Jensen CD, Marshall JW, Lawless A. Serum 25- hydroxyvitamin D is independently associated with highdensity lipoprotein cholesterol and the metabolic syndrome in men and women. J Clin Lipidol 2009; 3: 289-296.
- Ciccone MM, Zito A, Dentamaro I, Vestito D, Scicchitano P. Vitamin D deficiency and cardiovascular diseases. G Ital Cardiol 2015; 16: 16-20.

#### \*Correspondence to

Jong-Hwan Park

Institute of Convergence Bio-Health, Dong-A University

Republic of Korea