Original Article

Body Height and Incident Risk of Venous Thromboembolism
A Cosibling Design

Bengt Zöller, MD, PhD; Jianguang Ji, MD, PhD; Jan Sundquist, MD, PhD; Kristina Sundquist, MD, PhD

Background—Body height has been associated with an increased risk of venous thromboembolism (VTE), but the association can be confounded with shared familial factors (genetic/environmental). A cosibling design is useful for deeper understanding about the relationship between VTE and height.

Methods and Results—From Swedish national registry databases, we used a corelative design with full siblings alongside a general Swedish population sample. A cohort of male conscripts (n=1 610 870), born in 1951 to 1992 without previous VTE, was followed from enlistment (1969–2010) until 2012. Another cohort of first-time pregnant women (n=1 093 342) from the medical birth register, without previous VTE, was followed from first pregnancy (1982–2012) until 2012. Using the Multi-Generation Register, we identified all full-sibling pairs discordant for height. This cosibling design allowed for adjustment for familial factors (genetic/environmental). Compared with the tallest women (>185 cm) and men (>190 cm), there was a graded decreased risk by lower height for both men and women. The risk was lowest in women and men with the shortest stature (<155 and <160 cm, respectively): hazard ratios=0.31 (95% confidence interval, 0.22–0.42) and 0.35 (95% confidence interval, 0.22–0.55), respectively. There was a graded association also in the cosibling design comparing siblings with varying degree of discordance for height (reference was the taller sibling): ≥10 cm difference between brothers hazard ratios=0.69 (95% confidence interval, 0.61–0.78) and sisters hazard ratios=0.65 (95% confidence interval, 0.52–0.80), respectively.

Conclusions—Height is an independent predictor of VTE. The use of sibling pairs reduces the likelihood that familial confounding explains the results. The findings are important for the understanding of the pathogenesis of VTE. **(*Circ Cardiovasc Genet*. 2017;10:e001651. DOI: 10.1161/CIRCGENETICS.116.001651.)**

Key Words: embolism ■ epidemiology ■ genetics ■ risk factors ■ thrombosis

Greater height and long legs have been associated with an increased risk of incident venous thromboembolism (VTE), but results are divergent.[1–9] One study by Schmidt et al[10] found no association between VTE and height. Moreover, Braekkan et al[4] found an association between height and VTE only among men and not among women. Similarly, Flinterman et al[8] found only weak nonsignificant associations between height and VTE for women. This might be related to the fact that men are taller than women in general. An association between recurrent VTE and height has also been observed.[8,11] Still, a plausible explanation exists for an association between great height and increased VTE risk. Tall people and those with long legs may be subject to greater stasis in the legs as a consequence of greater hydrostatic pressure.[12–14]

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Height is highly heritable.[15] Thus, genetic variants could be related to an increased VTE incidence. However, the common genetic variants related to height are individually only weakly associated with height although they together explain a large part of the heritability of height.[16] A genetic risk score of 696 variants, however, could be worthwhile to determine whether it is associated with VTE.[17] In a modern Western society, 20% of the variation in body height is because of environmental causes.[18] In poorer societies, the influence of the environment might be even stronger. Important nongenetic factors affecting growth and adult body height are nutrition, diseases, socioeconomic status, and environmental factors in childhood.[18–20]

Observational studies have shown an association between body height and VTE,[1–9] but concerns remain about confounding variables. In the current study, we used a cosibling design.[21–24] Traditionally, in this approach, the association between an exposure and an outcome is compared in the general population and in full siblings. From the pattern of the associations in these 2 groups, it is possible to assess the degree to which the association observed in the population might be causal versus because of confounding from familial factors (genetic/environmental).[21–24] The use of sibling pair analysis reduces the influence of familial confounding. If

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a significant association between height and VTE was not detected in sibling pairs, it would weaken the argument that height is a causal risk factor for VTE. The aims of this study were to explore the association between height and VTE in a nationwide follow-up study using Swedish national registry databases to ascertain the risk of VTE and to further examine whether these associations persisted within nested sibling pairs.

Methods

This study was approved by the Ethics Committee of Lund University, Sweden. Data used in this study represented information on individuals registered as residents of Sweden who either belonged to a cohort of male conscripts (n=1 610 870) born in 1951 to 1992 with no history of previous VTE or to a cohort of first-time pregnant women from the medical birth register with no history of previous VTE with first pregnancy (n=1 093 342) in 1982 to 2012.[25–32] It included individual-level information on the following: enlistment, births, age, height, weight, body mass index (BMI), sex, educational attainments, hospital and outpatient diagnoses, dates of hospital admissions, date of emigration, and date and cause of death. The data sources were multiple national Swedish data registers, including the Swedish National Population and Housing Census, the Total Population Register, the Swedish Hospital Register (the Hospital Discharge Register and the Hospital Outpatient Register), Military Conscription Register, Medical Birth Register, and the Multi-generation Register.[25–32] These registers were provided to us by Statistics Sweden and the National Board of Health and Welfare. Using the Swedish Hospital Register, we identified hospital inpatients and outpatients, with diagnoses of VTE in Sweden, between January 1, 1969, and December 31, 2012. Data were linked using the individual personal identification numbers that are assigned to all persons in Sweden for their lifetime. These numbers were replaced with serial numbers to maintain anonymity. The serial numbers were used to check that each individual was only entered once for his or her first diagnosis of VTE diagnosis (inpatient or outpatient and death registers). The follow-up ran from January 1, 1969 (men) or 1982 (women) until a VTE event, death, emigration, or the end of the follow-up on December 31, 2012, whichever came first. This register does not include data for patients treated at primary healthcare centers, but diagnosis of VTE is not done in primary healthcare in Sweden.

Outcome Variable and Ascertainment of Cases

Cases of VTE, classified according to the World Health Organization *International Classification of Diseases* (*International Classification of Diseases Eighth Revision*, *Ninth Revision*, and *Tenth Revision*), were identified in the Hospital Discharge Register and the Outpatient Register 2001 to 2010 (Table I in the Data Supplement). The definition of VTE has been the same over time although the diagnostic methods may have changed (ie, more computed tomographic scans and ultrasound examinations). All VTE cases in Sweden are initially diagnosed and treated in the hospital specialist clinics (outpatient or inpatient care). The Swedish Hospital Discharge Register has ≈90% overall validity or positive predictive values.[27,29] The positive predictive values for cardiovascular disorders, such as VTE, myocardial infarction, and stroke, are ≈90% to 95%.[3,27,29] Negative predictive values for common cardiovascular diagnosis vary between 95.0% and 99.8%.[27]

In a Swedish study of men with VTE, hospital records were available for 304 cases (1970–1998).[3] A total of 289 of 304 (95%) cases, with diagnosed VTE, were judged to be diagnosed correctly.[3] Only 12 (3.9%) cases were not diagnosed with an objectively verified method but were treated with oral anticoagulation because of strong clinical probability. In total, 277 (91%) cases were objectively diagnosed with methods, such as phlebography, ultrasound, computed tomographic scan, and pulmonary scintigraphy.[3] The Outpatient Register has not previously been validated for VTE. Recently, Abdul Sultan et al[33] found that only 57% of pregnant VTE cases first recorded as outpatients were accompanied by anticoagulant prescriptions, whereas this proportion was much lower than those cases first recorded in the inpatient register (91%). Moreover, in a previous study of ours, we used Anatomical Therapeutic Chemical Classification System codes for anticoagulant drugs to validate the whole outpatient and inpatient registers with prescription of anticoagulant drugs after VTE diagnosis.[34] The prescription register is only available from July 2005. Positive predictive values for inpatient diagnosis of VTE were similar to published data by Rosengren et al[3] and Abdul Sultan et al.[33,34] An outpatient diagnosis of VTE had less validity, similar to Abdul Sultan et al,[33] but the presence of VTE diagnosis on 2 occasions in outpatients was associated with high positive predictive values (ie, anticoagulant prescription), similar to what was observed in the study by Rosengren et al.[3,34] We, therefore, classified only outpatient diagnosis of VTE if diagnosis had occurred twice in outpatients while 1 event of VTE in inpatients is enough to be classified as VTE.

Main Predictor Variable

Height (cm) and weight (kg) and BMI were obtained from the Swedish military conscription register for the male cohort at the time of enlistment.[32] Height and weight and BMI were obtained from the Swedish Medical birth register for the female cohort.[30,31] We used height and weight register at the start of the first pregnancy.

Potential Confounders

Adjustments were made for potential confounders: BMI, age at first pregnancy (women only because men were all aged 18–20 years at enlistment), education, year of enlistment (men) or year of first pregnancy (women), and family history of VTE 1969 to 2012 (Table I in the Data Supplement) in first-degree relatives (parent/sibling). Education was used as a proxy for socioeconomic status. Education was classified as completion of compulsory school or less (≤9 years), practical high school (10–12 years), or theoretical high school or college (≥13 years). BMI was calculated by dividing weight (kg) by the square of height (m). In Tables II and II in the Data Supplement, the associations between height and potential confounders are presented.

Statistical Analysis

The log-rank test was used to examine the difference of VTE-free survival curve. Cox regression was used to calculate hazard ratios (HRs)[35] and 95% confidence intervals (CIs) for subsequent VTE in patients with different height and was controlled for potential mediators or confounders by the selected demographic characteristics (age [continuous variable], education level, BMI, family history of VTE [parent/sibling], and year of first pregnancy as a continuous variable [women] or enlistment [men]). We censored individuals (ie, treated them as no longer under observation or at risk of the study outcome) at the time of death from any cause, at the end of the follow-up period (December 31, 2012), or at the time of emigration. The proportional hazards assumption was tested using Schoenfeld residuals and by plotting the log of the negative log of the survival function versus the log of time.[36] A scaled version of the Schoenfeld residual at time k for a particular covariate p will approximate the change in the regression coefficient at time k. We used scatter plot smooths to explore the scaled Schoenfeld residuals' relationship with time, and we found that the smooths seem mostly flat at 0, suggesting that the coefficient for age does not change over time and that proportional hazards hold for this covariate. We also tested for interactions between variables, but no significant interaction was observed. Using the Swedish Multi-Generation Register, we identified all full-sibling pairs with a maximum of 5 cm height difference, between 5 and 9 cm height difference, and with ≥10 cm height difference. Using stratified Cox proportional hazards models, we performed an analysis of these full-sibling pairs. Stratification was used to compare sibling pairs. This was done in SAS using the strata statement (SAS version 9.2, SAS Institute, Cary, NC).[37] The corelative design allows us to contrast the rates of VTE in relatives with different levels of height difference. Reference is the taller siblings. The stratified Cox

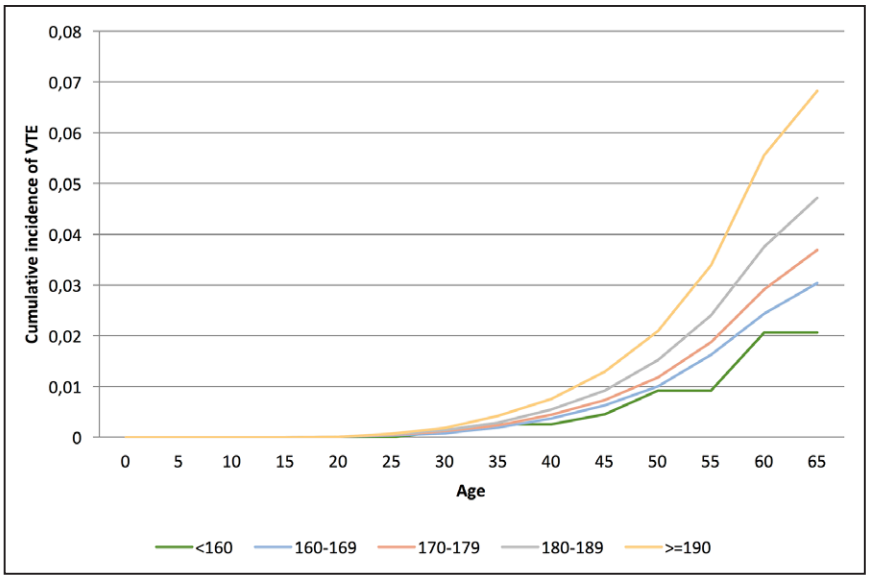

Figure 1. Cumulative incidence of venous thromboembolism (VTE) by height in male conscripts in Sweden.

proportional hazards model provides a HR for VTE that is adjusted for familial confounding (genetic/environmental).[21–24,37] Cumulative 5-year incidence rates were calculated by lifetest in SAS. The data were transferred to Excel, and Figures 1 and 2 were created in Excel. Data are accurate to 2 decimals places. All analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC).

Results

HR for VTE According to Height in Male Conscripts

Characteristics of the male conscript cohort are shown in Table 1. The total follow-up for male conscripts was 41 346 944 person-years. Table 1 shows the crude and adjusted HR results for VTE in male conscripts at enlistment between 1969 and 2010. The adjusted HR decreased with 65% for men shorter than 160 cm compared with the tallest men (≥190 cm). The 95% CI were narrow. The HRs showed a clear gradient with decreased HR for VTE incidence for every 10 cm of decreased height compared with the tallest men. This is also evident in Figure 1, which shows the cumulative incidence of VTE by height in male conscripts in Sweden. The incidence rates for VTE were 26.6, 35.4, 41.4, 51.3, and 69.5 per 100 000 person-years. Only small differences in HRs were observed for the crude and adjusted models. In the adjusted model, the results were independent of enlistment year (not shown in table), BMI, family history of VTE (parent/sibling), and educational attainment (Table 1).

HR for VTE According to Height in Women Pregnant at Least Once

Characteristics of the female first pregnant cohort are shown in Table 2. The follow-up for first pregnant women was 14 351 631 person-years. Table 2 presents the crude and adjusted HR results for VTE in women at the start of the first pregnancy between 1982 and 2012. The HR decreased with 69% for women shorter than 155 cm compared with the tallest women (≥185 cm). The 95% CIs were narrow. The HRs showed a clear gradient with decreased HR for VTE incidence for every 10 cm of decreased height compared with the tallest women. This is also evident in Figure 2, which shows the cumulative incidence of VTE by height in first-time pregnant women in Sweden. The incidence rates for VTE were 65.9, 74.1, 94.1, 128.3, and 216.2 per 100 000 person-years. In the adjusted model, the results were independent of age at first birth, year of first birth, BMI, family history of VTE (parent/sibling), and educational attainment.

Table 1. Crude and Adjusted Hazard Ratios for Associations Between Body Height and Other Factors and Subsequent Risk of VTE Among Swedish Men

				Crude				Adjusted*			
Characteristics	Individuals	VTE	%	HR	95% CI		*P* Value	HR	95% CI		*P* Value
Height											
<160	2579	18	0.7	0.35	0.22	0.56	<0.0001	0.35	0.22	0.55	<0.0001
160–169	102 802	961	0.9	0.46	0.43	0.50	<0.0001	0.48	0.45	0.52	<0.0001
170–179	729 308	7875	1.1	0.55	0.52	0.58	<0.0001	0.57	0.54	0.60	<0.0001
180–189	679 368	8827	1.3	0.70	0.67	0.74	<0.0001	0.72	0.68	0.76	<0.0001
≥190	96 813	1634	1.7	1.00	Reference			1.00	Reference		

CI indicates confidence interval; HR, hazard ratio; and VTE, venous thromboembolism.
*Adjusted model is adjusted for year of enlistment, body mass index, family history of VTE (parent/siblings), and education.

Table 2. Adjusted Hazard Ratios for Associations Between Body Height and Other Factors and Subsequent Risk of VTE Among Swedish Women

Characteristics	Individuals	VTE	%	Crude HR	95% CI		*P* Value	Adjusted* HR	95% CI		*P* Value
Height											
<155	35 901	294	0.8	0.30	0.22	0.42	<0.0001	0.31	0.22	0.42	<0.0001
155–164	389 812	3839	1.0	0.34	0.25	0.46	<0.0001	0.36	0.27	0.49	<0.0001
165–174	564 228	7033	1.2	0.43	0.32	0.58	<0.0001	0.47	0.35	0.63	<0.0001
175–184	101 604	1583	1.5	0.60	0.44	0.81	0.0008	0.63	0.46	0.85	0.0022
≥185	1797	44	2.4	1.00	Reference			1.00	Reference		

CI indicates confidence interval; HR, hazard ratio; and VTE, venous thromboembolism.
*Adjusted model is adjusted for age at first birth, year of first birth, body mass index, family history of VTE (parent/siblings), and education.

Subanalysis of Thrombosis in the Legs (Venous Thrombosis), Pulmonary Embolism, and Other Venous Locations (Others)

Among men, a graded association with height was observed for both pulmonary embolism and venous thrombosis of the legs and venous thrombosis at locations other than the legs and lungs (others; Table III in the Data Supplement). Others include migrating thrombophlebitis, cerebral vein thrombosis, portal vein thrombosis, Budd–Chiari syndrome, vena cava thrombosis, renal vein thrombosis, and other rare types of venous thrombotic events (*International Classification of Diseases* codes are defined in Table III in the Data Supplement). Among women, only venous thrombosis of the legs was significantly associated with height. No significant associations were observed with pulmonary embolism and venous thrombosis at other locations.

Corelative Analysis in Full Siblings

In the sibling pair analysis, the taller sibling was reference. A strong and graded association was observed among both women and men although among women a significant association was noted only for a difference of ≥10 cm (HR for sisters=0.65; 95% CI, 0.52–0.80; *P*<0.001; Table 3). In general, the 95% CIs were narrow because of the large sample sizes. Height difference between brothers ≥10 cm was associated with a HR of 0.69 (95% CI, 0.61–0.78; *P*<0.001). A strong association with VTE was also observed for as little as 5 to 9 cm among male full-sib pairs (HR=0.80; 95% CI, 0.73–0.87; *P*<0.001) but only borderline significant in females full-sib pairs (HR=0.90; 95% CI, 0.79–1.01; *P*=0.07). The association between VTE and difference of 10 cm was not weaker than among the general population sample. As there was a strong association between height and VTE in the sibling pair analysis, when adjusting for familial confounding, we did not perform any analysis of half-siblings and first cousins.

Discussion

The present study shows a strong association between VTE and height among both men and women. Thus, the association of height on VTE is not limited to men but is also valid for women. The present study is the largest epidemiological study and the only nationwide study of VTE and height, as well as the first

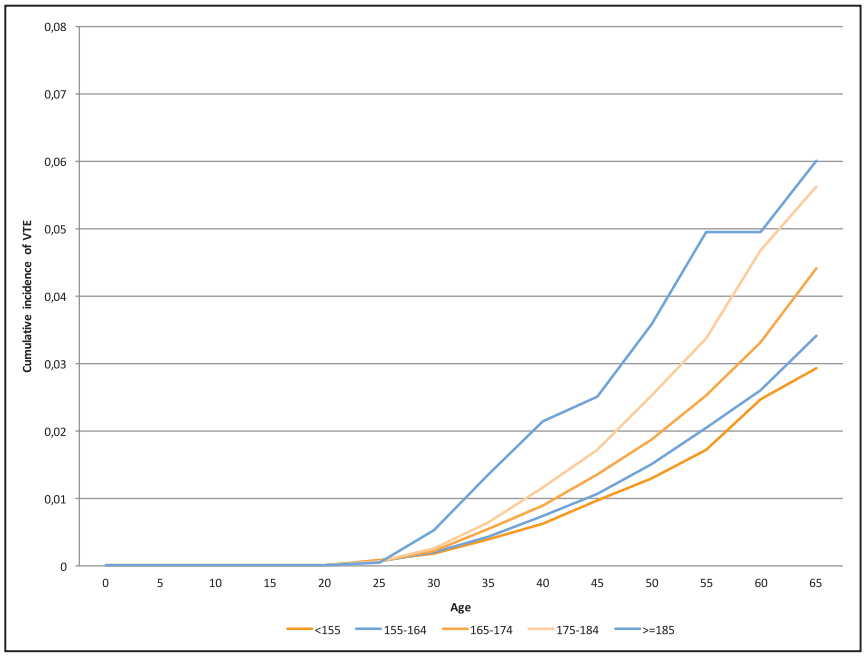

Figure 2. Cumulative incidence of venous thromboembolism (VTE) by height in first-time pregnant women in Sweden.

Table 3. Cosiblings With Different Height and Risk of VTE

	HR	95% CI		*P* Value
Men (reference is the taller siblings)				
Height difference between brothers				
<5 cm	0.94	0.88	1.01	0.08
5–9 cm	0.80	0.73	0.87	<0.0001
≥10 cm	0.69	0.61	0.78	<0.0001
Overall	0.85	0.81	0.89	<0.0001
Women (reference is the taller siblings)				
Height difference between sisters				
<5 cm	0.97	0.88	1.07	0.49
5–9 cm	0.90	0.79	1.01	0.07
≥10 cm	0.65	0.52	0.80	<0.0001
Overall	0.90	0.84	0.97	0.003

Full model is adjusted for age at first birth, height, body mass index, and education. CI indicates confidence interval; HR, hazard ratio; and VTE, venous thromboembolism.

using a cosibling design. Our study also shows that the association between height and VTE is graded and valid for different manifestations of VTE. Most importantly, use of sibling pair analysis, which reduces the influence of familial confounding, showed a strong association between VTE and height. The present study does not argue against that the association between height and VTE may be causal. Moreover, the present study is in agreement with a recently published Mendelian randomization study that indicates that tall height is a risk factor for VTE.[38] This suggests a robustness of our study design (nationwide cosibling study). The association between height and VTE may partly contribute to several phenomena. Postural effects on circulation have been suggested to be the result from the distension of vessels (particularly veins) subsequent to changes in gravitational pressure of blood.[39] For instance, the low incidence of VTE in children could partly be related to height, with expected lower effect of gravitational pressure of blood, although of course hormonal influence is also important.[40] The gravitational pressure of blood in the arms is also expected to be lower than in the legs and may explain the much lower incidence of deep venous thrombosis in the arms than in the legs. The same is true on the gravitational pressure of blood in visceral veins and veins of the brain, which are all rare manifestations of VTE.[41] The present study showed, among women, only an association with venous thrombosis of the legs indicating that the stasis theory (in Virchow triad) could be true. However, among men, all types of VTE were associated with height, suggesting a systemic hypercoagulability induced by height indicating that other mechanisms might also be involved.

The present study has several strengths. These include nationwide coverage in a country of high medical standards supervised by the Swedish National Board of Health and Welfare, together with diagnoses of patients by specialist physicians during examinations in clinics. Data in the Swedish registers are almost complete. In 2001, personal identity numbers were missing for only 0.4% of hospitalizations and main diagnoses for 0.9% of hospitalizations.[26]

A limitation is that we did not have access to individual-level data for childhood and parent lifestyle factors, such as smoking, diet, and physical activity in. However, we adjusted for educational level that is related to several lifestyle factors. Education is also known to be associated with height, but still there was an association between height and VTE.[19,42] A weakness is that the hospital register was not nationwide until 1987 and that the hospital outpatient register is only from 2001. However, this is most likely a nondifferential bias on association between height and VTE. In addition, this has probably not affected the association between height and VTE because, previously, most patients with VTE were treated as inpatients. The definition of VTE is the same over time although the diagnostic methods may have changed. However, the results are adjusted for year of enlistment (men) or year of first birth (women) to account for any potential differences related to time period.

In conclusion, body height is an independent predictor for VTE both among men and women. The use of sibling pairs reduces the likelihood that familial confounding explains the results.

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Disclosures

None.

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CLINICAL PERSPECTIVE

Body height has been associated with an increased risk of venous thromboembolism (VTE), but the association can be confounded with shared familial factors (genetic/environmental). A cosibling design is useful for deeper understanding about the independent relationship between VTE and height. A nationwide Swedish cohort of male conscripts (n=1 610 870) and another cohort of first-time pregnant women (n=1 093 342) from the medical birth register, without previous VTE, were followed until 2012. Compared with the tallest women (>185 cm) and men (>190 cm), there was a graded decreased risk by lower height for both men and women. The risk was lowest in women and men with the shortest stature (<155 and <160 cm, respectively): hazard ratios=0.31 (95% confidence interval, 0.22–0.42) and 0.35 (95% confidence interval, 0.22–0.55), respectively. There was a graded association also in the cosibling design comparing siblings with varying degree of discordance for height. Height is an independent predictor of VTE for both men and women. The use of sibling pairs reduces the likelihood that familial confounding explains the results. The findings are important for the understanding of the pathogenesis of VTE. In clinical practice, height is an easy accessible risk factor that might be useful to include in risk assessment.

Body Height and Incident Risk of Venous Thromboembolism: A Cosibling Design

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SUPPLEMENTAL MATERIAL

Supplementary Table 1. ICD codes (International Classification of Disease) used to define venous thromboembolism (VTE).			
	ICD-8	ICD-9	ICD-10
Cerebral vein thrombosis	321	437G	I63.6, I67.6
Venous thrombosis of the lower extremities	451	451	I80
Portal vein thrombosis	452	452	I81
Other venous embolism or thrombosis	453	453	I82
Pulmonary embolism	450	415B, 416W	I26

Supplementary Table 2. Association of height with potential confounders in males.

Male height	<160	160-169	170-179	180-189	190+	P value
BMI						
Normal	0.15	6.31	45.28	42.22	6.04	
Overweight	0.18	6.91	45.38	41.95	5.58	
Obese	0.38	6.71	44.74	41.32	6.85	<0.0001
Family history of VTE						
No	0.17	6.48	45.53	41.93	5.89	
Yes	0.12	5.56	43.21	44.12	6.99	<0.0001
Years of education						
<10	0.26	8.91	50.34	36.43	4.06	
10-12	0.18	7.04	46.79	40.61	5.38	
>=13	0.10	4.77	41.79	45.93	7.41	<0.0001

Numbers in the table are percentages. P-values were calculated with chi2-test.

Supplementary Table 3. Association of height with potential confounders in females.

Female height	**<155**	**155-164**	**165-174**	**175-184**	**185+**	**P value**
BMI						
Normal	3.03	35.00	52.26	9.54	0.16	
Overweight	3.89	37.05	50.21	8.69	0.17	
Obese	4.33	38.75	48.47	8.27	0.18	<0.0001
Family history of VTE						
No	3.45	36.17	51.15	9.07	0.16	
Yes	1.48	30.09	56.49	11.68	0.25	<0.0001
Years of education						
<10	7.88	44.76	41.68	5.57	0.11	
10-12	3.23	37.28	51.06	8.30	0.14	
>=13	2.39	32.30	54.17	10.94	0.20	<0.0001
Age at first birth						
<25	3.84	39.86	49.21	6.98	0.11	
25-29	2.91	34.87	52.58	9.47	0.16	
30+	3.26	33.42	52.36	10.76	0.20	<0.0001

Numbers in the table are percentages. P-values were calculated with chi2-test.

Supplementary Table 4. Hazard ratios for associations between height and subtype of VTE: venous thrombosis of the legs (VT), pulmonary embolism (PE), and venous thromboembolism in other sites than the legs and lungs (others)

	VT of lower extremities				PE				Others			
Characteristics	HR	95%CI		P value	HR	95%CI		P value	HR	95%CI		P value
Men												
<160	0.32	0.17	0.59	0.0003	0.23	0.07	0.71	0.01	0.66	0.27	1.60	0.35
160-169	0.48	0.43	0.53	<.0001	0.42	0.35	0.49	<.0001	0.61	0.50	0.74	<.0001
170-179	0.56	0.52	0.60	<.0001	0.51	0.46	0.57	<.0001	0.73	0.64	0.83	<.0001
180-189	0.72	0.67	0.77	<.0001	0.66	0.60	0.74	<.0001	0.80	0.70	0.92	0.00
>=190	1.00	Reference			1.00	Reference			1.00	Reference		
Women												
<155	0.25	0.17	0.38	<.0001	0.41	0.16	1.03	0.06	0.47	0.17	1.31	0.15
155-164	0.29	0.20	0.43	<.0001	0.56	0.23	1.35	0.20	0.47	0.17	1.25	0.13
165-174	0.39	0.27	0.57	<.0001	0.71	0.30	1.71	0.45	0.56	0.21	1.50	0.25
175-184	0.57	0.39	0.83	0.0036	0.83	0.34	2.02	0.69	0.73	0.27	1.96	0.53
>=185	1.00	Reference			1.00	Reference			1.00	Reference		

VT=Venous thrombosis of the legs: ICD-8 code 451, ICD-9 code 451, and ICD-10 code I80. PE=pulmonary embolism: ICD-8 code 450, ICD-9 codes 415B, 416W, and ICD-10 code I26. Others=VTE in sites other than legs and lungs: ICD-8 codes 321, 452, 453, ICD-9 codes 437G, 452, 453, and ICD-10 codes I63.6, I67.6, I81, I82.