Joint Effects of Obesity and Body Height on the Risk of Venous Thromboembolism

The Tromsø Study

Knut H. Borch, Cecilie Nyegaard, John-Bjarne Hansen, Ellisiv B. Mathiesen, Inger Njølstad, Tom Wilsgaard, Sigrid K. Brækkan

Objective—The goal of this study was to investigate the combined effect of obesity and body height on the risk of venous thromboembolism (VTE) in a prospective population-based study.

Methods and Results—Personal characteristics, including measures of obesity and body height, were collected in 26 714 men and women, aged 25 to 97 years, who participated in the Tromsø Study in 1994 to 1995. Incident VTE events were registered through September 1, 2007. There were 461 incident VTE events during a median of 12.5 years of follow-up. A tall stature was associated with increased risk of VTE in normal-weight (body mass index \( \leq 25 \text{ kg/m}^2 \)) and obese (body mass index \( \geq 30 \text{ kg/m}^2 \)) men, but not in women. The combination of obesity and tall stature synergistically increased the risk of VTE in both sexes. Tall (\( \geq 182 \text{ cm} \)), obese men had a 5-fold (multivariable hazard ratio 5.16; 95% CI 2.39 to 11.14) increased risk of VTE compared with normal-weight men with short (\( \leq 172 \text{ cm} \)) stature. Tall (\( \geq 168 \text{ cm} \)), obese women had an almost 3-fold (multivariable hazard ratio 2.89; 95% CI 1.31 to 6.35) increased risk of VTE compared with normal-weight, short (\( \leq 159 \text{ cm} \)) women.

Conclusion—The combination of obesity and a tall stature was associated with a substantially increased risk of VTE, especially in men, suggesting synergistic effects of obesity and height on risk of VTE in both sexes. (Arterioscler Thromb Vasc Biol. 2011;31:00-00.)

Key Words: obesity ■ pulmonary embolism ■ venous thrombosis ■ body height

Venous thromboembolism (VTE) is a collective term for deep vein thrombosis and pulmonary embolism. It is the third most common cardiovascular disease in the United States\(^1\) and is associated with severe complications and potentially fatal outcome.\(^2,3\) The overall incidence of VTE is 1 to 2 per 1000 person-years, but the incidence increases significantly with age.\(^4,5\) Venous thrombosis is a multicausal disease including both acquired and genetic factors.\(^6\) Despite the knowledge of several risk factors for VTE, 30% to 50% of the VTE events still have no obvious predisposing factors.\(^2,7\)

The prevalence of obesity is dramatically increasing in countries throughout the world, and obesity has become a particularly relevant public health challenge.\(^8\) Obesity is recognized as a strong and independent risk factor for VTE, and most studies have reported a 2- to 3-fold increased risk of VTE in obese compared with normal-weight subjects.\(^9-12\) Data from the Physicians’ Health Study indicate that body mass index (BMI) predicts VTE more strongly than coronary heart disease and stroke.\(^1\) The same study also reported an unexpected increased risk of VTE in tall men.\(^1\) This sex-specific association of increased VTE risk by greater body height has recently been confirmed by others.\(^13-15\)

Only a few previous studies have assessed the joint effect of obesity with other risk factors for VTE. These studies show that obesity combined with prothrombotic mutations (FV Leiden and prothrombin G20210A) is associated with a 6- to 8-fold increased risk of VTE compared with noncarriers with normal BMI\(^13\) and that use of oral contraceptives greatly enhances the risk of VTE in overweight\(^16\) and obese women,\(^13\) yielding a 24-fold higher risk in obese hormone users compared with nonusers with a normal BMI (<25 kg/m\(^2\)).

Considering these previous reported synergistic effects, it is likely that the combination of obesity and body height influences the overall risk of VTE in individual patients. Thus, the joint effects of the 2 anthropometric measures need to be properly addressed. We used data from a prospective, general population–based cohort study to assess the joint effect of obesity and body height on the risk of VTE and to investigate whether the combined effect of the 2 was synergistic or purely additive.
Methods

Study Population

Participants were recruited from the fourth survey of the Tromsø Study, conducted in 1994 to 1995, a single-center, prospective, population-based study, with repeated health surveys of inhabitants in Tromsø, Norway. All inhabitants aged ≥25 years old were invited, and 27 158 participated (77% of those invited). Data were collected by physical examination, blood samples, and self-administered questionnaires. The study was approved by the regional committee for research ethics. Subjects without written consent to medical research (n=300), subjects not officially registered as inhabitants of the municipality of Tromsø at baseline (n=43), subjects with known history of prior VTE (n=47), and subjects with missing BMI values (n=54) were excluded from the study. Thus, the total study population consisted of 26 714 subjects. A subgroup of these, namely all subjects aged 55 to 74 years and 5% to 10% random samples from the other age groups, was invited to a more extensive second screening visit (n=6753). Measures of waist circumference (WC) were available in 6741 subjects attending this second visit. Incidence VTE events among the study participants was recorded from the date of enrollment (1994 to 1995) through the end of follow-up, September 1, 2007.

Outcome Registration

All first lifetime events of VTE during follow-up were identified by searching the hospital discharge diagnosis registry, the autopsy registry, and the radiology procedure registry at the University Hospital of North Norway from date of enrollment (1994 to 1995) to September 1, 2007, as previously described. The medical records for each potential VTE case were reviewed by trained personnel. For subjects derived from the hospital discharge diagnosis registry and the radiology procedure registry, an episode of VTE was recorded when all 4 of the following criteria were met: (1) objectively confirmed by diagnostic procedures, including compression ultrasonography, venography, spiral computed tomography, perfusion ventilation scan (high or moderate probability for pulmonary embolism), pulmonary angiography, or autopsy; (2) the medical record indicated that a physician had made a diagnosis of deep vein thrombosis or pulmonary embolism; (3) signs and symptoms consistent with deep vein thrombosis or pulmonary embolism were present; and (4) treatment with anticoagulants (heparin, warfarin), thrombolytic therapy, or vascular surgery was required. For patients derived from the autopsy registry, a VTE event was recorded when the autopsy record indicated VTE as cause of death or as a significant condition contributing to death.

Baseline Measurements

Body height and weight were measured with subjects wearing light clothing and no shoes. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m2). WC was measured at the umbilical line. Information on self-reported diabetes, current smoking, and prior cardiovascular disease was collected from self-administered questionnaires.

Statistical Analysis

Statistical analysis was carried out using SPSS version 15.0 (SPSS Inc, Chicago, IL). Age-adjusted baseline differences between groups were tested by analysis of covariance for continuous variables and by logistic regression of dichotomous variables.

BMI was categorized according to the WHO definition17 into normal-weight (BMI <25 kg/m2), overweight (BMI 25 to 29.9 kg/m2) and obese (BMI ≥30 kg/m2), and WC was categorized into abdominally normal (WC <80 cm in women and <94 cm in men), abdominally overweight (WC 80 to 87.9 cm in women and 94 to 101.9 cm in men) and abdominally obese (WC ≥88 cm in women and ≥102 cm in men).18 Body height was categorized into short stature (≤159 cm in women and ≤172 cm in men), normal stature (160 to 167 cm in women and 173 to 181 cm in men), and tall stature (≥168 cm in women and ≥182 cm in men). A total of 9 body habitus categories were generated by combining BMI or WC with body height categories. Sex-specific Cox proportional hazard regression models were used to calculate age-adjusted and multivariable adjusted hazard ratios (HR) with 95% confidence intervals (CI) for VTE by categories of body height within each BMI stratum and WC stratum. In the multivariable models, age, diabetes mellitus, smoking, and estrogen supplementation (women only) were included as covariates. To assess the joint effect of BMI and body height on risk of VTE, the other 8 constellations of BMI/body height were compared with those with normal weight and short stature (BMI <25 kg/m2, and body height ≤159 cm in women/≤172 cm in men). Similarly, we assessed the joint effect of body height/WC on the risk of VTE by comparing constellations of body height/WC with abdominally normal subjects of short stature (WC ≤80 cm in women/≤94 cm in men and body height ≤159 cm in women/≤172 cm in men). The proportional hazard assumption was verified by evaluating the parallelism between the curves of the log-log survivor function for different categories of the variables. Biological interaction and synergism was evaluated using the Rothman synergy index19 to determine whether the joint effects from body height and obesity on the risk of VTE exceeded the sum of effects from each factor alone. Synergy index=(RRab−1)/(RRa+RRb−2).
RRab is the relative risk of the joint exposure group; RRa and RRb are relative risks for the exposure of obesity and taller body height, respectively. A value greater than 1.0 suggests that the effect of the joint exposures of 2 risk factors is greater than the sum of the separate effects.

Results

There were 461 validated incident VTE events during a total of 288,974 person-years of follow-up (median 12.5 years). The overall crude incidence rate of VTE was 1.6 per 1000

Table 1. Baseline Characteristics Across Categories of BMI (Tromsø Study, 1994 to 2007)

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Category of Body Height</th>
<th>Persons at Risk</th>
<th>Events</th>
<th>Age-Adjusted HR (95% CI)</th>
<th>Multivariable HR (95% CI)‡</th>
</tr>
</thead>
</table>
| Women (242 events) | BMI <25.0 kg/m² | <25 to 29.9 kg/m² | ≥30 kg/m² | P for trend |</table>

RRab is the relative risk of the joint exposure group; RRa and RRb are relative risks for the exposure of obesity and taller body height, respectively. A value greater than 1.0 suggests that the effect of the joint exposures of 2 risk factors is greater than the sum of the separate effects.

Results

There were 461 validated incident VTE events during a total of 288,974 person-years of follow-up (median 12.5 years). The overall crude incidence rate of VTE was 1.6 per 1000

Table 2. Sex-Specific HR for VTE With 95% CI by Categories of BMI and Categories of Body Height

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Category of Body Height</th>
<th>Persons at Risk</th>
<th>Events</th>
<th>Age-Adjusted HR (95% CI)</th>
<th>Multivariable HR (95% CI)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women (242 events)</td>
<td>BMI &lt;25.0 kg/m²</td>
<td>&lt;25 to 29.9 kg/m²</td>
<td>≥30 kg/m²</td>
<td>P for trend</td>
<td></td>
</tr>
</tbody>
</table>
person-years. Sex-specific, age-adjusted incidences of VTE by various constellations of BMI/body height are shown in Figure 1.

Baseline characteristics across categories of BMI are presented in Table 1. In women, 59% were considered normal weight, 29% overweight, and 12% obese. The corresponding figures in men were 46%, 45%, and 10%, respectively. Age, proportion of diabetics, and number of subjects with prior cardiovascular disease increased significantly across categories of BMI in both sexes, whereas the proportion of smokers decreased \( (P<0.001) \). The proportion of women on hormone therapy decreased across BMI categories \( (P=0.001) \).

Sex-specific HR for VTE by constellations of body height and BMI are shown in Table 2. Table 2 shows both the impact of body height within each BMI stratum and the joint effects of BMI and body height, using normal-weight subjects with a short stature as the reference group. In women, comparison of the upper versus lower category of body height within each BMI stratum did not yield significant differences in risk of VTE in either age-adjusted or multivariable analyses (Table 2). However, a nonsignificant trend of increasing VTE risk by increasing body height was observed within the overweight (multivariable HR 1.32, 95% CI 0.71 to 2.47) and obese (HR 1.69, 95% CI 0.73 to 3.89) groups. In contrast, linear trends of increased VTE risk by increasing body height were found within all categories of BMI for men in multivariable analysis \( (P \text{ for trend } 0.01 \text{ in normal-weight, 0.05 in overweight, and 0.04 in obese subjects, respectively} \text{ (Table 2). Normal-weight, tall (≥182 cm) men had a 2-fold increased risk of VTE (HR 2.33, 95% CI 1.20 to 4.53) compared with normal-weight, short (≥172 cm) men. Similarly, obese, tall men had an almost 3-fold increased risk (HR 2.90, 95% CI 1.09 to 7.70) compared with obese men of short stature. Comparison of all constellations of BMI/body height using normal-weight, short women as reference group, revealed an almost 3-fold higher risk of VTE in obese, tall women (multivariable HR 2.89, 95% CI 1.31 to 6.35) (Table 2 and Figure 2). In men, the risk of VTE was more than 5-fold increased in those who were obese with tall stature compared with those of normal weight with short stature (multivariable HR 5.16, 95% CI 2.39 to 11.14) (Table 2 and Figure 2). A synergy index of 2.0 in women and 1.7 in men suggested that the joint effect of obesity and tall stature was greater than the sum of the individual effects in both sexes. In women, further calculations revealed that 34% of the cases in the joint exposure group were attributable to biological interaction between obesity and tall stature (ie, attributable to mechanisms in which both factors play a causal role). The corresponding number in men was 33%.

Table 3 shows the sex-specific HR for VTE by constellations of WC and body height in the subgroup of subjects who attended the second screening visit \( (n=6741) \). Consistently, greater body height was significantly associated with VTE within categories of WC in men but not in women (Table 3). Analyses of the joint effects of WC/body height showed that abdominally obese (WC ≥102 cm) men had a 5-fold increased risk of VTE compared with short (≤172 cm), abdominally normal (WC <94 cm) subjects \( (HR 5.07, 95\% \text{ CI } 2.26 \text{ to } 11.37) \). Correspondingly, tall (≥182 cm), abdominally obese (WC ≥102 cm) women had a 2.4-fold increased risk of VTE compared with abdominal normal (WC <80 cm) women with a short (<160 cm) stature (multivariable HR 2.37, 95% CI 0.98 to 5.72) (Table 3). Correspondingly, tall (≥182 cm), abdominally obese (WC ≥102 cm) men had a 5-fold increased risk of VTE compared with short (≤172 cm), abdominally normal (WC <94 cm) subjects \( (HR 5.07, 95\% \text{ CI } 2.26 \text{ to } 11.37) \).

**Discussion**

Obesity is a well-known independent risk factor for VTE in both men and women, with a 2- to 3-fold increased risk in obese compared with normal-weight subjects. \( ^{9-12,17} \) Recently, greater body height has been acknowledged as a risk factor for VTE, \( ^{1,14} \) and the association is apparently restricted to males. \( ^{13,15} \) In the present study, a tall stature was a significant risk factor for venous thrombosis in men but not in women, and the association was independent of body composition expressed by BMI or WC. The intriguing question of this study was whether the combination of obesity and tall stature would produce a synergistic effect on the risk of VTE in men or women. Compared with those who were short with normal weight, the combination of a tall and obese habitus was associated with 3-fold and 5-fold increased risks of VTE in
women and men, respectively. A synergy index of $>1$ suggested that the joint effect of obesity and tall stature was greater than the additive sum of the individual components in both men and women.

The individual underlying pathophysiological mechanisms by which obesity and body height increases the risk of venous thrombosis are not fully understood. Greater body height is associated with higher resting venous pressure,$^{20–22}$ and venous stasis has been proposed as a possible mechanism for the observed VTE risk in tall men. Abdominal obesity is associated with raised intraabdominal pressure and reduced venous blood flow velocity, which may render blood more susceptible to thrombosis.$^{23,24}$ A recent study showed that lower limb venous flow parameters differed significantly between healthy obese and nonobese subjects, suggesting a mechanical role of abdominal adipose tissue, potentially leading to elevated risk of VTE.$^{25}$ On the other hand, visceral adipose tissue is highly metabolic active, releasing increased amounts of proinflammatory, proatherogenic, and prothrombotic substances, such as interleukin-6, tumor necrosis factor-$\alpha$, tissue factor, and plasminogen activator inhibitor-$1,26,27$ which may also contribute to thrombosis risk. Interestingly, plasminogen activator inhibitor-$1$ was recently shown to be associated with increased risk of VTE even after adjustment for BMI.$^{28}$

In our study, one third of the cases in the joint exposure group were attributable to biological interaction between obesity and body height. Biological interaction indicates a mechanism in which both factors play a causal role, ie, that the 2 factors are biologically dependent on one another to cause the disease. Thus, it may be speculated that the increased risk of VTE in tall, obese subjects may be caused by extensive stasis induced by the combined presence of tall stature and obesity, or that stasis induced by tall stature potentiates the effect of circulating prothrombotic substances released from adipose tissue. Additional mechanistic studies are required to elucidate both the individual and combined mechanisms of obesity and body height on the development of VTE.

The main strengths of our study are the temporal sequence of exposure and outcome, large number of participants

Table 3. Sex-Specific HR for VTE With 95% CI by Categories of WC and Categories of Body Height

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Category of Body Height</th>
<th>Persons at Risk</th>
<th>Events</th>
<th>Age-Adjusted HR (95% CI)</th>
<th>Multivariable HR (95% CI)‡</th>
<th>Age-Adjusted HR (95% CI)</th>
<th>Multivariable HR (95% CI)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Women (112 events)</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>WC &lt;80 cm</td>
<td>115 to 159 cm</td>
<td>400</td>
<td>9</td>
<td>0.76 (0.29 to 1.96)</td>
<td>0.76 (0.29 to 1.98)</td>
<td>0.70 (0.27 to 1.83)</td>
<td>0.71 (0.27 to 1.83)</td>
</tr>
<tr>
<td></td>
<td>160 to 167 cm</td>
<td>562</td>
<td>8</td>
<td>0.53 (0.66 to 4.27)</td>
<td>0.51 (0.66 to 4.07)</td>
<td>0.38 (0.05 to 3.05)</td>
<td>0.38 (0.05 to 3.01)</td>
</tr>
<tr>
<td></td>
<td>168 to 199 cm</td>
<td>175</td>
<td>1</td>
<td>1.09 (0.46 to 2.60)</td>
<td>1.11 (0.46 to 2.66)</td>
<td>1.05 (0.44 to 2.50)</td>
<td>1.05 (0.44 to 2.50)</td>
</tr>
<tr>
<td></td>
<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.4</td>
<td></td>
<td>0.94 (0.37 to 2.37)</td>
<td></td>
</tr>
<tr>
<td>WC 80 to 87.9 cm</td>
<td>115 to 159 cm</td>
<td>359</td>
<td>9</td>
<td>1.06 (0.62 to 1.79)</td>
<td>1.04 (0.61 to 1.78)</td>
<td>2.09 (1.00 to 4.39)</td>
<td>2.09 (0.99 to 4.40)</td>
</tr>
<tr>
<td></td>
<td>160 to 167 cm</td>
<td>480</td>
<td>12</td>
<td>1.27 (0.58 to 2.78)</td>
<td>1.26 (0.58 to 2.76)</td>
<td>1.89 (0.65 to 5.54)</td>
<td>1.90 (0.67 to 5.35)</td>
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<tr>
<td></td>
<td>168 to 199 cm</td>
<td>168</td>
<td>6</td>
<td>3.24 (1.24 to 8.50)</td>
<td>3.24 (1.24 to 8.46)</td>
<td>3.31 (1.30 to 8.73)</td>
<td>3.37 (1.30 to 8.73)</td>
</tr>
<tr>
<td></td>
<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.7</td>
<td></td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>WC $\geq$88 cm</td>
<td>115 to 159 cm</td>
<td>456</td>
<td>24</td>
<td>1.27 (0.58 to 2.78)</td>
<td>1.26 (0.58 to 2.76)</td>
<td>1.28 (0.59 to 2.80)</td>
<td>1.31 (0.60 to 2.85)</td>
</tr>
<tr>
<td></td>
<td>160 to 167 cm</td>
<td>598</td>
<td>32</td>
<td>1.41 (0.58 to 3.39)</td>
<td>1.38 (0.57 to 3.33)</td>
<td>2.84 (1.40 to 5.78)</td>
<td>2.89 (1.42 to 5.89)</td>
</tr>
<tr>
<td></td>
<td>168 to 199 cm</td>
<td>210</td>
<td>11</td>
<td>1.81 (0.67 to 4.91)</td>
<td>1.72 (0.63 to 4.67)</td>
<td>3.24 (1.02 to 9.72)</td>
<td>3.26 (1.02 to 9.74)</td>
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<tr>
<td></td>
<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.7</td>
<td></td>
<td>0.8</td>
<td></td>
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<tr>
<td><strong>Men (111 events)</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WC &lt;94 cm</td>
<td>140 to 172 cm</td>
<td>620</td>
<td>11</td>
<td>1.27 (0.58 to 2.78)</td>
<td>1.26 (0.58 to 2.76)</td>
<td>1.28 (0.59 to 2.80)</td>
<td>1.31 (0.60 to 2.85)</td>
</tr>
<tr>
<td></td>
<td>173 to 181 cm</td>
<td>705</td>
<td>15</td>
<td>2.10 (0.98 to 4.51)</td>
<td>2.19 (1.02 to 4.70)</td>
<td>2.84 (1.40 to 5.78)</td>
<td>2.89 (1.42 to 5.89)</td>
</tr>
<tr>
<td></td>
<td>182 to 207 cm</td>
<td>187</td>
<td>7</td>
<td>2.37 (0.98 to 5.71)</td>
<td>2.37 (0.98 to 5.72)</td>
<td>5.08 (2.27 to 11.35)</td>
<td>5.07 (2.26 to 11.37)</td>
</tr>
<tr>
<td></td>
<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.7</td>
<td></td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>WC 94 to 101.9 cm</td>
<td>140 to 172 cm</td>
<td>339</td>
<td>9</td>
<td>1.41 (0.58 to 3.39)</td>
<td>1.38 (0.57 to 3.33)</td>
<td>2.49 (1.17 to 5.26)</td>
<td>2.44 (1.15 to 5.19)</td>
</tr>
<tr>
<td></td>
<td>173 to 181 cm</td>
<td>526</td>
<td>25</td>
<td>2.84 (1.40 to 5.78)</td>
<td>2.89 (1.42 to 5.89)</td>
<td>4.08 (1.82 to 9.15)</td>
<td>4.07 (1.81 to 9.14)</td>
</tr>
<tr>
<td></td>
<td>182 to 207 cm</td>
<td>212</td>
<td>6</td>
<td>2.49 (1.17 to 5.26)</td>
<td>2.44 (1.15 to 5.19)</td>
<td>5.08 (2.27 to 11.35)</td>
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<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.7</td>
<td></td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>WC $\geq$102 cm</td>
<td>140 to 172 cm</td>
<td>178</td>
<td>7</td>
<td>2.05 (0.79 to 5.29)</td>
<td>2.11 (0.89 to 5.27)</td>
<td>2.81 (0.99 to 7.38)</td>
<td>2.74 (0.99 to 7.36)</td>
</tr>
<tr>
<td></td>
<td>173 to 181 cm</td>
<td>398</td>
<td>18</td>
<td>2.13 (0.84 to 5.35)</td>
<td>2.11 (0.84 to 5.33)</td>
<td>2.81 (0.99 to 7.38)</td>
<td>2.74 (0.99 to 7.36)</td>
</tr>
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<td></td>
<td>182 to 207 cm</td>
<td>168</td>
<td>13</td>
<td>2.23 (0.90 to 5.79)</td>
<td>2.21 (0.90 to 5.79)</td>
<td>2.81 (0.99 to 7.38)</td>
<td>2.74 (0.99 to 7.36)</td>
</tr>
<tr>
<td></td>
<td>$P$ for trend</td>
<td></td>
<td></td>
<td>0.07</td>
<td></td>
<td>0.03</td>
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</tr>
</tbody>
</table>

*Impact of body height within each WC stratum. Ref indicates reference value.
†Joint effects of WC and body height using normal-weight subjects with a short stature as the reference group (the Tromsø Study, 1994 to 2007).
‡Multivariable model adjusted for age, diabetes, smoking, and estrogen supplementation (in women).
recruited from a general population with high attendance rate, and long-term follow-up. The incidence of VTE events in our study was similar to that in previous reports.29 A single hospital serves the entire Tromsø population, which enhances the probability of a complete VTE registry and proper validation of the VTE events. The study has some limitations. Modifiable risk factors, such as BMI, may be a potential limitation of cohort studies when the time between exposure assessment and disease manifestation is very long. An individual’s BMI is likely to increase during follow-up,30 which implies that our risk estimates may be underestimated. Body height is normally constant over time and is therefore unlikely to produce this kind of bias.

The combination of obesity and a tall stature was associated with a substantially increased risk of VTE, especially in men, and suggests synergistic effects of obesity and body height on risk of VTE in both sexes. Our findings suggest that both body height and obesity should be considered when assessing risk of VTE.

Disclosures
None.

References
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