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Original article

## Sex Differences in the Impact of Thinness, Overweight, Obesity, and Parental Height on Adolescent Height



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### ABSTRACT

**Purpose:** The secular trend of increasing weight may lead to a decline in height gain compared with the genetic height potential. The impact of weight on height in healthy male and female adolescents compared with their genetic height was assessed.

**Methods:** Height and weight were measured in Israeli adolescent military recruits aged 16–19 years between 1967 and 2013. The study population comprised 355,229 recruits for whom parental height measurements were documented. Subjects were classified into four body mass index percentile groups according to the U.S. Centers for Disease Control and Prevention body mass index percentiles for age and sex: <5th (underweight), 5th–49th (low-normal), 50th–84th (high-normal), and ≥85th (overweight-obese). Short stature was defined as height ≤ third percentile and tall stature as height ≥ 90th percentile for age and sex.

**Results:** Overweight-obese females had a 73% increased risk for short stature (odds ratio [OR]: 1.73, 95% confidence interval [CI] = 1.51–1.97,  $p < .001$ ). Conversely, underweight females had a 56% lower risk of short stature (OR: .44, 95% CI = .28–.70,  $p = .001$ ) and a twofold increased risk for being tall (OR: 2.08, 95% CI = 1.86–2.32,  $p < .001$ ). Overweight-obese males had a 23% increased risk of being short (OR: 1.23, 95% CI = 1.10–1.37,  $p < .001$ ). Underweight females were on average 4.1 cm taller than their mid-parental height.

**Conclusions:** Overweight-obese males and females had an increased risk of being short, and underweight females were significantly taller compared with their genetic height. The significantly

### IMPLICATIONS AND CONTRIBUTION

Overweight-obese females and males have an increased risk of being short compared to those with normal weight. A greater influence of body mass index on height was observed in females. Underweight females were significantly taller compared to those with normal weight, and taller than their expected genetic height.

**Conflicts of Interest:** The authors have no conflicts of interest to disclose.

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increased height among underweight healthy females may reflect a potential loss of height gain in overweight-obese females.

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Height is an inherited trait which is also substantially influenced by environmental factors during fetal life, childhood, and adolescence. About 60% to 80% of the difference in height between individuals is determined by genetic factors [1], whereas 20% to 40% can be attributed to environmental factors such as socioeconomic circumstances, parental education, illnesses, and nutrition. Poor nutrition is the single most common cause of growth retardation worldwide [2,3], and improvement of nutrition has resulted in improved growth. The global trend toward increasing height, which began in the middle of the last century, was observed in both developed and developing countries [4,5].

The impact of nutrition on growth however is a double-edged sword, and there is a strong biological basis for the association between obesity and growth restriction. The increase in height is regressive in the United States, where there is currently a negative secular trend [6]. Americans, who were the tallest nation in the world, were overtaken first by the Dutch and subsequently by most other Western and Northern European populations [7]. It has been suggested that the initial rapid tempo of growth of the U.S. children which was associated with high energy balance, led to higher body mass index (BMI) values which in turn resulted in decreased growth during adolescence [6]. Indeed, comparing linear growth profiles and BMI values revealed that although U.S. children are born with greater length than Dutch children, they experience an earlier onset and a shorter period of puberty [7]. As a consequence of these growth patterns, 20-year-old Dutch males and females are more than 5 cm taller than U.S. adults [7].

It appears that there is a significant impact in the balance between underweight and overweight on linear growth. Using a national database of Israeli adolescent military inductees for whom both paternal and maternal height measurements were documented, we aimed to assess the influence of body weight on adolescent height, accounting for genetic height potential.

## Patients and Methods

### Study population

One year before compulsory military service, Israeli adolescents undergo a thorough medical evaluation performed by military physicians who also review the inductees' medical records and refer them for any further investigations deemed necessary as previously reported [8]. Between 1967 and 2013, 2,605,792 Israeli adolescents were assessed at ages 16–19 years. For the purpose of this study, we initially excluded 715,652 subjects who were classified as not fit for combat service, as we considered that chronic endogenous conditions may be a principle confounder affecting both growth and height at age 17 years. The main reasons for exclusion were respiratory disorders (11%) and gastrointestinal disorders (9%); however, obese, lean, short, or tall inductees who do not have comorbidities are included in the study cohort. Of the remaining 1,133,070

male and 757,070 female adolescents with unimpaired health, we identified a subgroup of 355,229 subjects (185,788 males and 169,441 females) with documented paternal and maternal height and weight measured at their military service medical examinations at ages 16–20 years. Our data enables us to link parent and identify their children. This subgroup comprised the study population. The Israel Defense Forces Medical Corps Institutional Review Board approved this study and waived the requirement for informed consent.

### Data collection

Data obtained included age, year of birth, BMI percentile groups, socioeconomic status (SES), years of education, country of origin, cognitive function, and parental height. Weight and height were measured (barefoot and in underwear) by trained medics. Measurements were recorded and rounded to the nearest .5 kg for weight and 1 cm for height. BMI was calculated (weight in kilograms divided by height squared in meters). Short stature was defined as height  $\leq$  third percentile and tall stature as height  $\geq$  90th percentile for age and sex, according to the U.S. CDC-established height percentiles for age (by month) and sex [9]. In the absence of national Israeli anthropometric data, the CDC data were selected by the Israel Ministry of Health as the routine reference for anthropometric data for children [10]. To assess the difference between measured height and genetic height potential we used the corrected mid-parental height (MPH) [11]. MPH in centimeters was calculated as follows: for males = (father's height + [mother's height + 13])/2 and for females = ([father's height - 13] + mother's height)/2 [11]. The difference between the measured height and expected height (MPH) in centimeters was calculated.

### Confounding variables

Education level was considered as a categorical variable and was divided into low and high levels at a threshold of 11 full years of school education. This cutoff represents the maximum potential school instruction at the time of weight and height assessment. SES groups, based on the place of residence obtained from the Israeli Ministry of Interior, were categorized on a scale of 1–10 according to the Israeli Central Bureau of Statistics' scoring system. This score stratifies all municipalities into 10 decile groups taking into account multiple variables that might affect SES [12]. SES was stratified into 3 groups: low (SES = 1–4), medium (SES = 5–7), and high (SES = 8–10), as reported previously [13]. Country of origin, classified by the father's or grandfather's country of birth, was categorized into five geographical areas: former Union of Soviet Socialist Republics (USSR) countries, Asia (non-USSR), Africa (excluding South Africa), Western (comprised non-USSR Europe, North and South America, South Africa, Australia, and New Zealand), and Israel. Birth countries were similarly classified [14]. Cognitive function was assessed by the general intelligence score (GIS), conducted

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