

# Lactase persistence and milk consumption are associated with body height in Swedish preadolescents and adolescents

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## Abstract

**Background:** Body height is a classic polygenic trait. About 80%–90% of height is inherited and 10%–20% owed to environmental factors, of which the most important ones are nutrition and diseases in preadolescents and adolescents.

**Objective:** The aim of this study was to explore potential relations between the LCT (lactase) C>T-13910 polymorphism, milk consumption, and body height in a sample of Swedish preadolescents and adolescents.

**Design:** In a cross-sectional study, using a random sample of preadolescents and adolescents ( $n=597$ ), dietary intakes were determined. Anthropometric measurements including sexual maturity (Tanner stage) and birth weight were assessed. Parental body height and socio-economic status (SES) were obtained by questionnaires. Genotyping for the LCT C>T-13910 polymorphism that renders individuals lactase persistent (LP) or lactase non-persistent (LNP) was performed by DNA sequencing. Stepwise backward multivariate linear regression was used.

**Results:** Milk consumption was significantly and positively associated with body height ( $\beta=0.45$ ; 95% CI: 0.040, 0.87,  $p=0.032$ ). Adjustments were performed for sex, parental height, birth weight, body mass index (BMI), SES, and Tanner stage. This model explains 90% of the observed variance of body height (adjusted  $R^2=0.89$ ). The presence of the -13910 T allele was positively associated with body height ( $\beta=2.05$ ; 95% CI: 0.18, 3.92,  $p=0.032$ ).

**Conclusions:** Milk consumption is positively associated with body height in preadolescents and adolescents. We show for the first time that a nutrigenetic variant might be able to explain in part phenotypic variation of body height in preadolescents and adolescents. Due to the small sample size further studies are needed.

Keywords: LCT-13910 C>T polymorphism; body height; milk consumption; parental body height

Received: 1 May 2011; Revised: 7 July 2011; Accepted: 11 August 2011; Published 6 September 2011

Body height is a classic polygenic trait and 80%–90% heritable (1, 2). Many genetic variants might contribute to phenotypic variation of body height (3). About 180 genomic regions contributing to height have been detected by genome-wide association analysis. Altogether these loci account for about 10% of phenotypic variation for height (3). In modern western societies, about 10%–20% of variation in body height is due to environmental factors. The most important environmental factors affecting body height are nutrition and diseases (4).

We focus on potential associations between body height and the LCT (lactase) C>T-13910 polymorphism that renders individuals lactase persistent (LP) or lactase non-persistent (LNP) and, thus, may modulate milk consumption. Milk consumption is one of the most studied nutritional factors in relation to height and growth rate in humans. Already in the early 1900s, very well-performed experimental studies showed that cow's milk supplementation in schoolchildren led to an increased longitudinal growth (5, 6).

Today, a century later and in a very different socio-economic setting, the long-term effect of childhood milk consumption on growth is still debated. There is, however, considerable evidence that milk stimulates longitudinal growth in certain populations, even in recent studies (7–10). Non-caloric components of milk, especially insulin-like growth factor I (IGF-I), are widely held to account for the growth stimulating effect of cow's milk in industrialised countries (7, 11–16).

Swedish preadolescents and adolescents are of specific interest for the study of potential links between milk consumption and body height on account of the high prevalence of LP (17, 18) due to their highest consumption of milk and milk products in Europe (19) and in the world. Surprisingly, given the large volume of publications related to milk intake, body height, and growth in preadolescents and adolescents, the publications that show corrections for parental body height are very scarce.

The aim of this study is to explore if there is a relation between the LCT C > T-13910 polymorphism, as well as actual milk intakes and body height. For this purpose we used two age groups, 9- to 10-year-old preadolescents and 15- to 16-year-old adolescents before and after initiation of the pubertal growth spurt. These age groups were sampled because the beginning of a pubertal growth spurt or onset of puberty can vary individually and between gender, in timing and acceleration of growth (20). Furthermore, adolescents' daily pattern may vary more than that of preadolescents since younger children have less control over their own diets (9).

We thus hypothesised that the LCT C > T-13910 polymorphism could contribute with a small effect by allelic heterogeneity to phenotypic variation in height.

## Methods

The random sample comprises 267 Caucasian preadolescents (121 girls, 146 boys) and 330 Caucasian adolescents (164 girls, 166 boys) of a population belonging to the Swedish part of the European Youth Heart Study (EYHS). Genetic descent was self-reported or reported by the parents. The EYHS is a cross-sectional school-based study of risk factors for future cardiovascular disease among preadolescents (9–10 years old) and adolescents (15–16 years old). The mean ages in the Swedish sample for preadolescents and adolescents were 9.6 years and 15.6 years, respectively. Sampling procedures and participation rates have been described previously (21). Height, weight, and birth weight were measured by internationally accepted standardised procedures (22). Body mass index (BMI) was calculated as  $\text{weight/height}^2$  ( $\text{kg/m}^2$ ).

Identification of sexual maturity was assessed according to Tanner and Whitehouse (1976). A researcher of the same gender as the child recorded the pubertal stage after brief observation. On account of ethical reasons the

direction of one school preferred not to take part in the assessment of sexual maturity. Fifty subjects did therefore not participate in this assessment (3 preadolescent girls, 7 preadolescent boys, 25 adolescent girls, and 15 adolescent boys). These subjects did not enter final analysis.

The consumption of milk was assessed by an interviewer-mediated 24-hour recall. In preadolescents, a qualitative food record completed the day before the interview with the help of parents served as a checklist for the data obtained during the recall. A food atlas was used to estimate portion sizes. Dietary data were processed by StorMats (version 4.02, Rudans Lättdata, Sweden) and analysed using the Swedish National Food database (version 99.1). As part of a broad-ranging questionnaire, parents of the participants were asked if their child had a chronic illness or adhered to a special diet.

Since under-nourishment is practically non-existent in a nutritionally replete population such as Sweden and milk intake accounts in part for daily energy intake, the BMI was taken as proxy for energy intake. Furthermore, BMI is traditionally used to validate energy intake data (23).

For the genetic analysis of LP and LNP, genomic DNA was isolated from EDTA whole blood samples from the individuals with the QIAamp DNA Blood Mini Kit spin procedure. The DNA fragment spanning the -13910-C/T polymorphic site was amplified using a biotinylated forward-primer (5'-GGGCTGGCAATACAGATAA-GATA-3') and an unbiotinylated reverse-primer (5'-AGCAGGGCTCAAAGAACAATCTA-3'). The applied sequencing primer was: 5'-CTTTGAGGCCAGGG-3'. Sequencing was performed using a PSQ96 SNP reagent Kit and a PSQ 96MA system (Pyrosequencing AB) PSQ 96MA 2.0.1 software. The procedure has been previously described in detail (24, 25).

For the determination of socio-economic status (SES) we used the dichotomous variable, below versus above the mean income level in the catchment areas of the sample (below or above the mean in their municipality) on account of the relatively equal distribution of income in Sweden (26). For four subjects, data for SES were missing.

Statistical analyses were carried out using SPSS 15 (SPSS, Inc., Chicago, IL). Continuous variables were checked for normality. The data are presented as means and standard deviations or frequencies and percentages. Student's *t*-test was used to determine differences in milk intake (g/d) between LP and LNP subjects. Milk intake (g/d) was somewhat skewed to the right and therefore split in quintiles of milk intake. Stepwise backward multiple linear regression analysis was performed in order to study the relationship between milk intake in quintiles and body height (cm) after adjustments for sex, birth weight (g), father's and mother's height (cm), BMI, Tanner stage (1–5), and SES.

The study was approved by the Research Ethics Committees of Örebro County Council and Huddinge University Hospital. Parents and 15-year-olds gave specific written informed consent to participate in the study.

## Results

The characteristics of the sample are summarised in Table 1. The variables included in the backward multivariate regression model were: milk intake, LCT C>T-13910 polymorphism (LP =CT, TT vs. LNP =CC), sex, birth weight, father's and mother's height, BMI, Tanner stage, and SES. Characteristics of preadolescents and adolescents by quintile of milk intake are given in Table 2.

Fifty-six (9%) of the subjects of the whole sample were LNP. Where LNP may restrict the intake of milk in these subjects and can thereby have an effect on the dependent variable, body height as well as the exposure variable, i.e. milk intake. Milk intake tended to be lower in LNP compared to LP but the difference did not reach statistical significance ( $p = 0.067$ ).

Analysis was performed in a backward multivariate linear regression model. The studied exposure variables in this model were milk intake in quintiles and the LCT C>T-13910 polymorphism, and the outcome variable was body height. The model explained 90% of the observed variance of body height in preadolescents and adolescents (adjusted  $R^2 = 0.89$ ). Non-significant variables were removed in a stepwise manner by elimination when  $p(F) \geq 0.10$ . Model 1 included the variables: milk intake in quintiles, LCT-13910 C>T polymorphism, sex, birth weight, parents' height, Tanner stage, BMI, and SES. In a first step the variable BMI was eliminated and in a second step SES (Table 3). Milk intake (g/d) in quintiles

remained significantly different ( $\beta = 0.46$ ; 95% CI: 0.040, 0.87 and  $p = 0.032$ ) in the final model. In addition, the LCT C>T-13910 polymorphism (LP vs. LNP), remained significant in the final model ( $\beta = 2.05$ ; 95% CI: 0.18, 3.92 and  $p = 0.032$ ), showing a positive association of LP with height.

## Discussion

We found that milk intake and LCT C>T-13910 polymorphism, known to modulate milk consumption, were significant contributors to the observed variance of body height in the studied population. We primarily did not expect to find our hypothesis confirmed in a nutritionally replete country with already one of the highest intakes of milk and dairy products per capita in the world. The LCT C>T-13910 polymorphism might contribute with a small effect to phenotypic variation in height. As far as we know no genome wide association studies or meta-analysis have been performed previously addressing the question whether LP or LNP affects body height.

Increasing evidence suggests that cow's milk consumption exerts a positive effect on longitudinal growth in preadolescents and adolescents, for instance, by affecting the IGF-I-axis (8, 11, 27–30). But not all studies show a positive effect of milk on height, especially in Nordic countries, where average milk intake per capita traditionally is high (31).

The LCT C>T-13910 polymorphism, known to modulate milk intake (32, 33), remained significant in the final model. By including the LCT C>T-13910 polymorphism in our model, we subtract for hidden heritability in an already predominantly LP population (18). In a recent prospective study, evidence of an association

**Table 1.** Basic characteristics of covariates to body height in the study population of Swedish preadolescents and adolescents

LCT-13910 C>T genotypes	LP (n = 540)	LNP (n = 56)
Height, cm		
(preadolescents, adolescents)	139 ± 6, 171 ± 9	140 ± 5, 172 ± 10
Milk intake, g/d	571 ± 399	472 ± 374
BMI, kg/m <sup>2</sup>	19.1 ± 2.9	19.0 ± 2.8
Birth weight, g	3,511 ± 582	3,389 ± 585
Father's height, cm	180 ± 6	180 ± 7
Mother's height, cm	166 ± 6	166 ± 7
Tanner stage, 1–5 <sup>a</sup>		
(1), (2), (3), (4), (5)	185 (38%), 47 (10%), 8 (2%), 80 (16%), 173 (34%)	18 (34%), 6 (11%), 2 (4%), 8 (15%), 173 (34%)
Socio-economic status		
(below mean), (above mean) <sup>b</sup>	235 (48%), 258 (52%)	20 (39%), 32 (61%)
Girls/boys	258 (48%), 282 (52%)	27 (48%), 29 (52%)

<sup>a</sup>50 missing subjects.

<sup>b</sup>4 missing subjects.

**Table 2.** Variables associated to body height by quintiles of milk intake

Milk intake g/d, Quintiles	Quintile 1 (n=119)	Quintile 2 (n=120)	Quintile 3 (n=119)	Quintile 4 (n=120)	Quintile 5 (n=119)
Girls, boys	70 (65%), 37 (35%)	63 (55%), 52 (45%)	66 (53%), 59 (47%)	54 (44%), 70 (57%)	32 (25%), 94 (75%)
Height, cm					
(preadolescents, adolescents)	139 ± 6, 169 ± 8	138 ± 6, 169 ± 8	139 ± 6, 171 ± 9	139 ± 6, 172 ± 10	140 ± 6, 174 ± 8
BMI, kg/m <sup>2</sup>	19 ± 2.8	19 ± 3.1	19 ± 2.9	19 ± 3.0	19 ± 2.8
Birth weight, g	3,481 ± 550	3,487 ± 546	3,516 ± 583	3,409 ± 670	3,597 ± 543
Father's height, cm	180 ± 7	179 ± 7	180 ± 7	181 ± 7	180 ± 5
Mother's height, cm	167 ± 6	166 ± 6	166 ± 5	166 ± 6	166 ± 6
Tanner stage, 1–5 <sup>a</sup>					
1	31 (33%)	40 (38%)	44 (38%)	52 (45%)	37 (32%)
2	9 (10%)	16 (15%)	15 (13%)	8 (7%)	5 (4%)
3	0	1 (1%)	5 (4%)	2 (2%)	2 (2%)
4	17 (18%)	25 (23%)	11 (10%)	17 (15%)	18 (16%)
5	37 (39%)	25 (23%)	41 (35%)	35 (31%)	54 (46%)
Socio-economic status <sup>b</sup>					
(below mean), (above mean)	45 (45%), 56 (55%)	59 (54%), 50 (46%)	47 (43%), 62 (57%)	47 (41%), 69 (59%)	58 (52%), 53 (48%)
LCT C > T-13910					
(CC), (CT+TT)	12 (11%), 95 (89%)	14 (12%), 101 (88%)	14 (11%), 111 (89%)	9 (7%), 115 (93%)	7 (6%), 118 (94%)

<sup>a</sup>50 missing subjects.<sup>b</sup>4 missing subjects.

between increased infant size at birth and cow milk consumption during pregnancy was found (34). It is known that LP subjects consume on average more milk than LNP subjects. Where LP might thus lead to an increased susceptibility towards already prenatally programmed increased body sizes, mediated by the higher

capacity of LP mothers to consume milk and dairy products.

Our study accounts for parental height as regards milk intake and its potential effects on body height and growth in preadolescents and adolescents. Despite the circumstance that body height is a highly heritable complex trait; corrections for parental height continue to be an exception, also shown in recent studies (31). Even twin growth studies have been performed without consideration of parental height (35). It has been demonstrated that at the current stage a simple prediction based on phenotype of relatives obviously outperforms sophisticated genomic predictions as regards body height (36). Limitations of this study are the sample size, and the limits inherent to cross-sectional studies as regards causal inference.

Cow's milk is an evolutionary food constructed to promote growth and development in calves and appears to affect growth in human beings also (13, 14, 28). Milk has become a normative food for preadolescents and adolescents beyond weaning age, even in Asian populations and developing countries, which traditionally did not have home fare based on domesticated dairy animals (8, 37). The long-term effects of this changed nutritional normative are yet poorly understood, even in Western countries.

We conclude that actual milk intake and the genetic LP trait is positively associated with body height in preadolescents and adolescents. Nevertheless, higher milk consumption in childhood might exert both negative and

**Table 3.** Main effect by quintiles of milk intake and LCT-13910 C > T (exposure variables) on body height in Swedish preadolescents and adolescents (n=542); stepwise backward multiple linear regression models were used

Dependent variable: height (cm)	$\beta$	95% CI of $\beta$	P	R <sup>2</sup>
Model 1 <sup>a</sup>				
Quintiles of milk intake (g/d) <sup>2</sup>	0.45	0.34–0.86	0.034	0.89
LCT-13910 C > T (LP vs. LNP)	2.01	0.14–3.89	0.035	
Model 2 <sup>b</sup>				
Quintiles of milk intake (g/d)	0.45	0.35–0.87	0.034	0.89
LCT-13910 C > T (LP vs. LNP)	2.00	0.13–3.87	0.036	
Model 3 <sup>c</sup>				
Quintiles of milk intake (g/d)	0.46	0.40–0.87	0.032	0.89
LCT-13910 C > T (LP vs. LNP)	2.05	0.18–3.92	0.032	

<sup>a</sup>Included variables in Model 1: birth weight, sex, mother's and father's height, BMI, Tanner stage, SES.<sup>b</sup>Excluded variable: BMI ( $p[F] \geq 0.10$ ).<sup>c</sup>Excluded variables: SES ( $p[F] \geq 0.10$ ).

Missing subjects: 54.



positive effects, since greater height has been associated with higher risk of some cancers (29, 38–41).

### Conflict of interest and funding

This study was supported by grants from Örebro Läns Landstings Forskningskommitté and by Nyckelfonden, Örebro, Sweden. The authors declare no conflicts of interest.

### Acknowledgements

We thank the Stockholm County Council, Sweden, for creating and guaranteeing the study base. Expert statistical advice was provided by Mr. Anders Magnuson, Clinical Epidemiology and Biostatistics Unit, Örebro University Hospital, Sweden.

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