Early life undernutrition and adult height

Portrait, F. R.M.; van Wingerden, T. F.; Deeg, D. J.H.

published in
Economics and Human Biology
2017

DOI (link to publisher)
10.1016/j.ehb.2017.09.008

document version
Publisher's PDF, also known as Version of record

Economics and Human Biology 27 (2017) 339–348

Contents lists available at ScienceDirect

Economics and Human Biology

journal homepage: www.elsevier.com/locate/ehb

Early life undernutrition and adult height: The Dutch famine of 1944–45

F.R.M. Portrait[a,*], T.F. van Wingerden[a], D.J.H. Deeg[b]

[a] Department of Health Sciences, FALW, VU University Amsterdam, The Netherlands
[b] Department of Epidemiology and Biostatistics, Amsterdam Public Health Institute, VU University Medical Centre, Amsterdam, The Netherlands

ARTICLE INFO

Article history:
Received 25 September 2016
Received in revised form 23 September 2017
Accepted 29 September 2017
Available online 18 October 2017

Keywords:
Nutrition in early life
Adult height
Dutch famine
The Netherlands

ABSTRACT

Current research shows strong associations between adult height and several positive outcomes such as higher cognitive skills, better earning capacity, increased chance of marriage and better health. It is therefore relevant to investigate the determinants of adult height. There is mixed evidence on the effects of undernutrition during early life on adult height. Therefore, our study aims at assessing the impact of undernutrition during gestation and at ages younger than 15 on adult height.

We used data from the Longitudinal Aging Study Amsterdam. Exposure to undernutrition was determined by place of residence during the Dutch famine during World War II. Included respondents were born between 15 May 1930 and 1 November 1945 and lived in the northern part of the Netherlands during the famine period (n = 1008). Exposure data was collected using interviews and questionnaires and adult height was measured. Exposed and non-exposed respondents were classified in the age categories pregnancy- age 1 (n = 85), age 1–5 (n = 323), age 6–10 (n = 326) or puberty (age 11–15, n = 274). Linear regression analyses were used to test the associations of adult height with exposure. The robustness of the regression results was tested with sensitivity analyses.

In the models adjusted for covariates (i.e., number of siblings, education level of parents, and year of birth) and stratified by gender, adult height was significantly shorter for females exposed at ages younger than 1 (−4.45 cm [−7.44–−1.47]) or at ages younger than 2 (−4.08 cm [−7.20–−0.94]). The results for males were only borderline significant for exposure under age 1 (−3.16 [−6.82–0.49]) and significant for exposure under age 2 (−4.09 cm [−7.20–−0.96]). Exposure to the Dutch famine at other ages was not consistently significantly associated with adult height.

In terms of public health relevance, the study's results further underpin the importance of supporting pregnant women and young parents exposed to undernutrition.

1. Introduction

The current study aims at investigating the consequences of undernutrition in early life on adult height. A large body of evidence has consistently shown associations between tall height and several positive outcomes such as better health, higher cognitive skills, better earning capacity and increased chance of marriage (e.g. Deaton, 2008; Deaton and Arora, 2009). In particular, the pioneering study by Waaler reported strong negative associations between tall height and mortality in Norwegian males and females (Waaler, 1984). More recent studies also reported negative associations between adult height on the one hand and various chronic diseases and causes of death on the other hand (Batty et al., 2009; Silvertoinen et al., 1999). Furthermore, Case and Paxson (2008) also reported strong associations between tall height and higher occupational status and earnings. Case and Paxson suggested that these associations could be explained by higher cognitive ability, in addition to higher self-esteem or social dominance. The findings on socioeconomic status have been confirmed in more recent studies (e.g. Rietveld et al., 2015). Finally, taller individuals are more likely to marry and to have children than others (Fu and Goldman, 1996; Smith and Monden, 2012). These empirical results suggest that adult height plays an important role in many later life outcomes in terms of health and socio-economic status; the investigation of early life determinants of adult height is therefore of great interest to those seeking to emulate these conditions.

* Corresponding author at: Department of Health Sciences, W&N Building, VU University Amsterdam, De Boelelaan 1085, 1081 HV Amsterdam, The Netherlands.
E-mail address: f.r.m.portrait@vu.nl (F.R.M. Portrait).

https://doi.org/10.1016/j.ehb.2017.09.008
1570-677X/

Adult height results from a complex interplay of genetic and environmental factors such as availability of good-quality food, socio-economic conditions and exposure to diseases during early life (e.g. Silvertoinen, 2003; Bozzoli et al., 2009). Roughly 75% (in women) and 90% (in men) of adult height is inherited (Silvertoinen et al., 2003; McEvoy and Visscher, 2009; Yang et al., 2010), which implies that the influence of environmental factors is relatively limited. However, in historical as well as in developing populations, the heritability of height may have been much lower (Alter and Oris, 2008; Wells, 2011). Alter and Oris (2008) showed for instance that in nineteenth-century Belgium the correlation of brothers' heights was much smaller in deprived than in elite families. This may be explained by the fact that poorer people are less able to protect their children from (negative) environmental factors than wealthier parents.

Gestation and infancy, as well as puberty, are crucial periods of time with regard to physical developments including hormonal developments, body composition changes and physical growth (e.g. Silvertoinen, 2003; Siervogel et al., 2003; Kuh and Ben-Shlomo, 2004). Therefore, favorable environmental conditions during these periods of life are especially important to reach genetic height potential.

In particular, appropriate nutrition during early life is essential as growth is partly the result of the balance between energy from nutritional intake and energy needed for body maintenance and other bodily activities (e.g. Rogol et al., 2000). Studies have consistently shown the importance of nutrition at the (very) beginning of life in determining pubertal development and final body stature. For instance, the recent review of Soliman et al. (2014) underlines the importance of nutrition in early life in affecting (the onset and progression of) pubertal development, such as physical growth. Regarding adult height, Martorell (1995, 1999) found for instance that supplementing foods rich in energy, proteins and micronutrients during early childhood reduced the occurrence of "stunting", a term used to describe problematic, long-term reduced height of children in relation to their age. Stunting occurs mostly before the age of two and is often followed by "catch-up growth" (Martorell et al., 1994). Stunting is a direct cause of a short adult height (Dewey and Begum, 2011). Furthermore, the study of Alderman et al. (2006) showed the sizeable impact of pre-school malnutrition on height as a young adult. Also, two Chinese studies observed a remarkable drop in average height for cohorts born between 1956 and 1962 (Zheng-Wang and Cheng-Ye, 2005; Wang et al., 2010). The authors suggested that this was due to the severe famine that China experienced between 1959 and 1961. In contrast with these results, studies among individuals exposed to the famine during the Leningrad siege (1941–1944) showed no significant reduction in body height in exposed individuals during early life compared to non-exposed individuals. Average adult height of exposed females was even taller than that of non-exposed females (Koupil et al., 2007; Shestov et al., 2009).

A healthy diet may be even more important during puberty than in the very beginning of life, since puberty is characterized by increased needs in macro- and micronutrients including protein, calcium, zinc and iron (Klump, 2013; Soliman et al., 2014). To the best of our knowledge, there is limited evidence on the long-term effects of undernutrition during puberty on adult height. Most relevant research investigates the relationship between eating disorders and retarded growth. The findings on adult height are mixed, although the majority of the studies reported reduced adult height after severe eating disorders. For instance, the studies by Modan-Moses et al. (2012a, 2012b) Modan-Moses et al. reported incomplete catch-up growth in adolescent males (2012a) and females (2012b) with anorexia nervosa.

The main aim of our study is therefore to enhance our insight into the consequences of undernutrition during gestation, the first year of life and puberty on adult height. In terms of public health relevance, the study's results further underpin the importance of supporting pregnant women and young parents exposed to undernutrition and of implementing programs preventing restrictive food-intake disorders during puberty. Undernutrition is still a worldwide problem predominantly in developing countries but also to a lesser extent in developed countries for instance in case of diseases such as anorexia nervosa (De Onis and Blossner, 2003; Soliman et al., 2014).

In this study, exposure to undernutrition is determined by experiencing the Dutch famine during World War II. Between November 1944 and May 1945, an estimated 20.000 à 25.000 Dutch citizens died as a result of undernutrition and other harsh conditions (niod.knaw.nl). Although the Allies had begun to liberate several parts of Europe, the majority of the Netherlands was still occupied by the Germans at that time. In September 1944, the Dutch government in exile set up a nation-wide railway strike to weaken the German war machine as much as possible and to stimulate resistance movements in the occupied part of the Netherlands. The German occupiers responded with a complete blockade of all food- and fuel transport to the west of the Netherlands. This resulted in extremely severe survival conditions in the urban areas of the west of the country (Lumey and Van Poppel, 1994, 2013). Average nutritional intakes declined from 1800 kilocalories per day per individual before May 1944 to 500 kilocalories in February 1945, when the famine conditions were at their most severe. Even pregnant and lactating women and infants under age 1 could not be protected at the peak of the famine (Roseboom et al., 2011). In the rest of the country, sufficient amounts of food were available in November 1944–May 45 (Lumey and Van Poppel, 1994). During the Dutch famine, inhabitants of the South of the Netherlands lived under very different circumstances from those of the West and North-East (niod.knaw.nl), because part of the Netherlands to the South of the large rivers (Rhine, Meuse and Waal) had already been liberated in September 1944, but was exposed to ongoing battles and bombing (Barnouw, 1999; De Jong, 1981). Soon after the liberation of the country in May 1945, the average food intake rose to a normal level of 2000 kilocalories per day (Lumey and Van Poppel, 1994).

The Dutch famine of 1944–45 is a very useful source of information on the long-term effects of undernutrition for several reasons. Firstly, the time period of exposure is clear and well-defined (November 1st, 1944–May 15th, 1945) and, secondly, sufficient amounts of food were available in the non-exposed parts of the country during this period, which provides ready control groups (Lumey and Van Poppel, 1994). Thirdly, the relatively short period of the Dutch famine also minimizes the risk of selection of healthier survivors, which otherwise may have masked long-term effects of famine exposure. Finally, many individuals born during the Dutch famine are still alive today, which makes it possible to trace them and to collect information on their current health and height and other individual characteristics.

2. Methods

2.1. Study sample

Our data come from the Longitudinal Aging Study Amsterdam (LASA) (Deeg et al., 2002). The LASA study follows a nationally representative sample of 3107 Dutch individuals aged 55–85 at baseline in 1992–93 (cohort I). The baseline was recruited from three broad, culturally distinct, geographical areas of the Netherlands (West, North-East and South), each area consisting of one large city and several smaller municipalities. At baseline, there was an oversampling of older men such that after five years equal numbers of men and women were expected to be alive in age

categories: 55–59; 60–64; 65–69; 70–74; 75–79; 80–85. Respondents are followed in measurement cycles on average every three years by trained interviewers. Interviews take place at the respondents' homes. In 2002-03, an additional cohort of 1002 individuals born in 1937–47 (cohort II) was recruited from the same sampling frame and added to the initial cohort in the measurement cycle of 2005–06 (Huisman et al., 2011). The LASA study follows respondents who migrate within the country in order to limit loss to follow-up. The first measurement cycles of cohorts I and II were the basis of this study, as explained below (n = 4109).

Firstly, we selected all 1492 individuals born between May 15th, 1930 and November 1st, 1945. This is because we were interested in both individuals exposed to the Dutch famine between conception and late puberty and same-age individuals who were not exposed. We return to this in the section "Exposure variables". Secondly, respondents who had missing information on their municipality of residence in 1944 (n = 17) or who reported living in a foreign country in 1944 (n = 97) were excluded since the environmental conditions in other countries were most likely not comparable to the ones in the Netherlands during this time period. Thirdly, to keep the heterogeneity between the exposed and control groups within limits, we excluded from our analyses all respondents who reported living in the South of the Netherlands in 1944 (n = 310). Fourthly, all respondents with missing information on the outcome variable were excluded (n = 60). This resulted in a study sample of 1008 respondents.

2.2. Outcome variable

The outcome variable "adult height" was measured using a stadiometer as the number of centimeters from the bottom of the feet (without shoes or socks) to the top of the head, with the entire body of the respondent placed in a straight, upright position as far as possible. The figures were rounded to two decimal points. Body height of older adults may change over time due to shrinking (Stewart et al., 1985), and for this reason we used height data from the first available measurement. Height data of cohort I respondents were derived from the 1992–93 measurement cycle (n = 461), and in case of missing information, available data from the cycles of 1995–96 (n = 4), 1998–99 (n = 9), and 2001–02 (n = 18) were used instead. Following the same approach, height data of cohort II respondents were derived primarily from the 2002–03 cycle (n = 499), and in case of missing information, data of the 2005–06 (n = 10) and of 2008–09 (n = 3) cycles were used. In case no valid measurement could be done (e.g. because the respondent had a scoliosis or did not want to remove his/her shoes; n = 4), respondents were still measured and imputation methods were used to approximate the true height (http://www.lasa-vu.nl/themes/physical/recodingheightandweight.htm).

2.3. Exposure variables

Exposure is as defined in Portrait et al. (2011), following the early approach by Lumey and Van Poppel (1994). Firstly, the 1008 respondents were divided into four groups depending on their age during the Dutch famine: (1) "gestation to one year of age" (i.e. born between May 15th, 1944 and November 1st, 1945; n = 85), (2) "early childhood", defined as two to less than six years of age (i.e. born between May 15th, 1939 and May 14th,1944; n = 323), (3) "late childhood", defined as six to less than eleven years of age (i.e. born between May 15th, 1934 and May 14th, 1939; n = 326) and (4) "puberty" defined as eleven to less than sixteen years of age (i.e. born between May 15th, 1930 and May 14th, 1934; n = 274).

We opted for these specific cut-off points for the following reasons. With respect to the first group, the relevant literature only considers as exposed to undernutrition during gestation the individuals who were in their mother's womb for at least 13 weeks of gestation during the Dutch famine (Lumey et al., 2007). Those born between February 15th, 1945 and May 15th, 1945 were exposed to undernutrition in their mother's womb and their first year of life. Because of the relatively large overlap and because of the small sample size, we considered the periods "gestation and first year of life" jointly. The second and third age categories cover early and late childhood respectively (Ki-Moon, 2007) and are included in the analyses as exposure to undernutrition during childhood may also have long-term consequences on height. Finally, the pubertal period is defined as starting at age eleven and ending at age fifteen.

Secondly, only respondents who lived in large cities in the West of the country (i.e. in Amsterdam, Rotterdam, The Hague, Utrecht, Leiden, Delft or Haarlem, see Appendix 1) during the Dutch famine were included in the four exposed groups (n = 352). We derived this information from the following question at baseline of each cohort: "Where did you live in 1944? Please specify the municipality, and, if needed, the country." The place of residence at birth was used for respondents born after 1944, namely born between January 1st, 1945 and November 1st, 1945 (n = 56).

2.4. Additional covariates

Gender, socio-economic status (SES), season of birth, the total number of siblings and the degree of urbanization of the municipality of residence during the Dutch famine were tested for confounding. Firstly, males and females have different nutritional needs and there are large gender differences in adult height. Secondly, low-SES families may have had even less access to food during the Dutch famine than others and research consistently shows that individuals with a lower SES are on average shorter than others (Hamill et al., 1972; Cavelaars et al., 2000). SES was characterized by the highest level of education achieved by parents, using four dummy variables indicating: (1) primary education or lower, (2) lower vocational education, (3) secondary general level or medium vocational education, and (4) higher vocational or university. Thirdly, there is some evidence that individuals born in different seasons grow to significantly different adult heights (Day et al., 2015). Fourthly, the number of siblings during the Dutch famine may have influenced the amount of food available per child. The LASA data provides information on the total number of siblings actually born. This variable was used to characterize the number of siblings during the Dutch famine. In the analyses, we used four dummy variables indicating the quartiles of siblings even born. Fifthly, differences in the degree of urbanisation between the exposed and unexposed areas could bias the results due to different living conditions and socio-economic opportunities. We used zip-code categorical data from Statistics Netherlands (2004) to characterize the degree of urbanization of the municipality of residence in 1944 (1 ="$>$ 2500 addresses per km^2"to 5 ="$<$ 500 addresses per km^2"). Finally, our analyses were corrected for year of birth using a full set of dummies to correct for secular trends in height over time.

2.5. Statistical analysis

All statistical analyses were performed using STATA/SE 13.0. Firstly, descriptive analyses were used to characterize the study sample. Independent Sample T-tests were performed to investigate whether there was a (significant) difference in adult height between the exposed and the non-exposed groups.

Subsequently, several linear regression models were estimated to examine the relationship between exposure to undernutrition early in life and adult height. Before doing so, the outcome variable

"adult height" was tested for normality using a Q-Q plot and a steam leaf diagram, and all potential confounders were tested for linearity with the outcome variable using scatter plots. A basic regression model was estimated with only exposure to undernutrition, gender and dummies for year of birth included as independent variables. Next, all additional covariates were tested for confounding. Firstly, these variables were added separately to the basic regression model. When the regression coefficients of the exposure variables changed more than 10% after adding a variable and when the covariate was significantly associated with the outcome, this covariate was considered as a confounder and included in further models. Thereafter, a "forward selection procedure" was applied: of all remaining additional covariates, the variable with the lowest p-value was added to the regression model, and this step was repeated until all significant (p-value < 0.1) additional variables were included (Twisk, 2007). A level of statistical significance equal to 0.05 was used to test the main association (p-value < 0.05).

Empirical evidence shows differential effects on males and females of adverse exposure in early life on later health (Roseboom et al., 2011; Lumey and Van Poppel, 2013). Therefore, the previous analyses were also performed per gender.

In order to investigate the robustness of the results of the main analyses, various sensitivity analyses were performed. Firstly, in the analyses so far, the control group included unexposed individuals of all ages though the exposure group was divided into four age classes. We refined the previous analyses by re-estimating the models per age class. Secondly, we varied the cut-off points of the age at exposure categories and considered the youngest age at exposure during at least 13 weeks of gestation and the first two years of age. The window "gestation to age 2" is also considered as a crucial period to reduce the long-term consequences of undernutrition in early life as stunting often occurs before the age of 2 (Bryce et al., 2008; Black et al., 2008). Pubertal exposure was defined more liberally as being exposed between 10 and 15 years of age, since several studies consider age 10 as the onset of puberty (Marceau et al., 2011). Thirdly, the "borders" of geographical exposure to the Dutch famine were varied. In a recent publication, Lumey and Van Poppel (2013) state that the line between the famine stricken area and the non-stricken areas may be drawn roughly from the IJsselmeer coast around Elburg through Apeldoorn and Arnhem, following the major rivers to the west coast of the country, thus including the cities of Alkmaar, Amersfoort, Amstelveen, Zeist or Zaanstad. These cities are also located in the west of the Netherlands but are smaller and more peripheral than the cities in the narrower approach (see Appendix 1). These cities may also have been exposed to undernutrition, but to a lesser extent than the cities in the main analyses. In comparison with the earlier narrower definition, this includes a much larger area and therefore many more people who would be labeled as exposed in terms of location. Therefore, respondents who resided in these cities were also included in the exposed groups. Fourthly, we repeated the final main analyses using self-reported adult heights as outcomes. This was to investigate whether our results were sensitive to shrinking of individuals at older ages. Shrinking at old ages may indeed result in less variation in height across individuals and may therefore attenuate the differences in height between the exposed and unexposed individuals. The LASA respondents were asked to report their height in cm at age 25 (cohort I) and at age 40 (cohort II). This information was missing for 130 respondents of the study sample. Fifthly, to make the control and exposed groups even more comparable, we also re-ran all analyses including only individuals born in cities in the non-exposure group (in the North and East of the country). Finally, we re-estimated the main models after inclusion of an additional control group, namely those who lived in the South of the Netherlands during the famine.

3. Results

3.1. Descriptive analyses

Fig. 1 shows the distribution of adult height per gender. Adult height in centimeters was normally distributed as the results of the

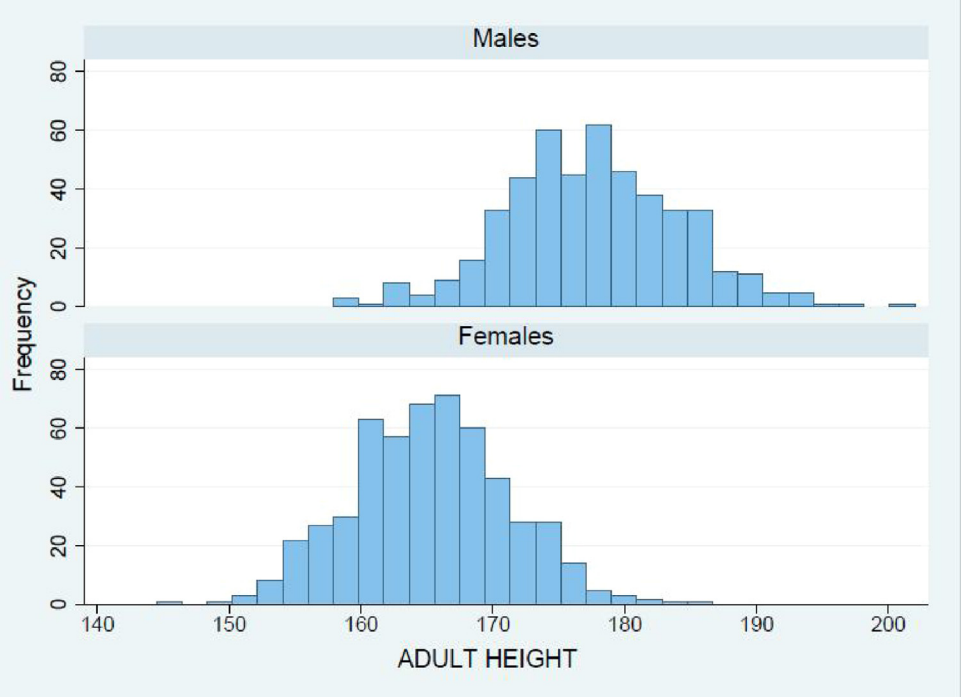

Fig. 1. Distribution of adult height per gender.

Q-Q plot and the steam leaf diagram, as well as the small difference shown between the mean adult height (171.0 cm) and the median of adult height (170.7 cm). As expected, females were on average shorter (165 cm) than males (177 cm).

The numbers in the exposure and non-exposure groups and the height characteristics per exposure group are reported in Table 1. Firstly, of all 1008 respondents, 352 lived in the urban west area of the Netherlands, 209 lived in the rural west area and 477 lived in the north/west region during the Dutch famine. Out of the 352 respondents who lived in the urban west area during this period, 26 were exposed to the famine during "gestation to age one" and 98 were exposed during "puberty". The remainder of these 352 respondents in the urban west was exposed during early childhood (n = 116) or late childhood (n = 112). Secondly, average adult height was not significantly shorter in those exposed to the famine during "gestation to age one" (170.8 cm) or during "puberty" (170.6 cm) than when not exposed (171.0 cm). Most importantly, females exposed during gestation or at ages younger than 1 had a significantly shorter height than females of the same birth cohort who were unexposed (p-value = 0.01).

Characteristics of the entire study sample per status of exposure are reported in Table 2. In our sample, the majority of the respondents was male (52%) in the exposed group while the majority of the respondents not exposed was female (56%). The mean levels of attained education by the respondents' parents were primary education or below (57.6%), lower vocational – secondary general level (15.9%) and medium vocational (16.2%). Respondents who were exposed during "gestation to age 1" had parents with slightly higher education levels than respondents who were exposed during "puberty" or who were not exposed (p-value $\leq$ 0.01). Education level was higher in the group that was exposed than in the group that was not exposed (64.4% versus 44.0% primary school or less). In addition, the exposed respondents had fewer siblings (on average 3.8) than those who were not exposed (on average 4.4).

3.2. Association of exposure to the Dutch famine in early life and adult height

The associations of all relevant additional variables with adult height proved to be linear, which implies that these variables were qualified as independent variables for linear regression analyses.

Table 3 shows the results of the first linear regression analysis with adult height as the dependent variable and the exposure variables as independent variables. Since the distribution of gender was very different in the exposed group compared to in the unexposed group and because of secular trends in height, the variables characterizing gender and the dummies for year of birth were already included in the basic model. Because the Breusch-Pagan test showed evidence of heteroskedasticity (*p-value = 0.016*), we computed White heteroskedasticity-corrected standard errors. In the basic model, women were on average 12.35 cm shorter than men (t-value = −31.2). The coefficient of the variable characterizing exposure during "gestation to age 1" was significantly different from 0 (−3.23, t-statistic = −2.90), whereas the coefficients of exposure during other age groups were not significantly different from 0 at the conventional statistical level of 5%. The coefficients of the year of birth dummies showed a significant increase in average height of about 3.4 cm between 1930 and 1945.

Subsequently, the covariates "number of siblings" and "highest education level of the parents" were included in the regression model. We again computed robust standard errors since the p-value of the Breusch-Pagan test was equal to 0.006. The dummy variables for "season of birth" and for "degree of urbanization" were excluded from the final analyses since neither of them was significantly related to adult height or changed the coefficients of the exposure variables with more than 10% (largest t-statistic in absolute value equal to 0.88).

A smaller family was significantly associated with an increase of adult height, namely having less than two siblings was associated with an increase of adult height equal to 1.39 cm (t-statistic = 2.34). Similarly, a higher parental education level was significantly associated with a higher adult height, namely children of parents who went to high schools or university were on average 1.74 cm taller than those with parents with primary education or less (t-statistic = 2.08). After inclusion of these confounders in the regression model, the coefficient of the exposure status before age 1 became larger (−3.70, t-statistic = −3.10). Being exposed during "early childhood" and "puberty" remained not associated with adult height. Notably, the coefficient of exposure during "late childhood" (exposure at age 6–10) became significant (equal to −1.34 cm and t-statistic = −2.0). Note that the results were highly similar when all potential confounders (namely number of sibling,

Table 1
Number respondents and height characteristics per exposure group during the Dutch famine.

	Age of respondents during the Dutch famine				
	Gestation to age 1	Early childhood	Late childhood	Puberty	Total
Place of residence during Dutch famine		Age 1–5	Age 6–10	Age 11–15	
# Urban west Netherlands (exposed)	26	116	112	98	352
# Rural west Netherlands (unexposed)	17	64	74	54	209
# North/East Netherlands (unexposed)	42	143	140	122	477
Height characteristics					
Mean (SD) height exposed individuals:					
Both genders	170.8 (8.1) p-value =0.15*	171.8 (9.0) p-value =0.93*	171.0 (8.3) p-value =0.94*	170.6 (9.0) p-value =0.24*	171.0 (8.8)
Males	177.1 (4.9) p-value =0.19*	177.9 (6.8) p-value =0.48*	176.6 (6.0) p-value =0.09*	176.6 (6.8) p-value =0.53*	177.0 (6.4)
Females	163.4 (3.4) p-value =0.01*	165.5 (6.2) p-value =0.81*	165.2 (6.1) p-value =0.96*	163.9 (5.8) p-value =0.52*	164.8 (5.9)
Mean (SD) height unexposed individuals:					
Both genders	173.4 (8.4)	171.7 (9.3)	170.9 (9.0)	169.3 (8.1)	171.0 (8.9)
Males	179.7 (6.5)	178.6 (6.8)	178.5 (7.0)	175.8 (5.8)	178.0 (6.7)
Females	168.2 (5.8)	165.7 (6.6)	165.3 (5.4)	164.5 (5.9)	165.5 (6.0)
Total	85	323	326	274	1008

* P-values Independent samples T-tests exposed versus unexposed category per age group during the Dutch famine.

Table 2
Descriptive characteristics of entire study sample and by exposure status to the Dutch famine.

	Exposed during gestation to age 1	Early childhood	Late childhood	Puberty		
		Exposed at age 1–5	Exposed at age 6–10	Exposed at age 11–15	Unexposed	Total
Number of respondents	26	116	112	98	656	1008
Year of birth (#,%)						
1930–35	0	0	39 (34.0)	98 (100.0)	250 (38.1)	387 (38.3)
1936–40	0	43 (37.0)	73 (66.0)	0	210 (32.0)	326 (32.3)
1941–45	26 (100.0)	73 (63.0)	0	0	196 (29.8)	295 (29.2)
Female (#,%)	12 (46.1)	57 (49.1)	55 (49.1)	46 (46.9)	366 (55.9)	536 (53.1)
	p-value = 0.33*	p-value = 0.18*	p-value = 0.18*	p-value = 0.10*		
Highest education achieved by parents (#, %)						
Primary education or below	6 (23.0)	52 (46.0)	44 (39.3)	53 (54.1)	419 (64.4)	574 (57.6)
Lower vocational –Secondary general level	6 (23.0)	18 (15.9)	27 (24.1	19 (19.4)	89 (13.7)	159 (15.9)
Medium vocational	7 (26.9)	28 (24.8)	30 (26.8)	14 (14.3)	83 (12.8)	162 (16.2)
High schools – university	7 (26.9)	15 (13.2)	11 (9.8)	10 (10.2)	59 (9.1)	102 (10.2)
Others/missing	0 (0.0)	2 (0.0)	0 (0.0)	2 (0.0)	6 (0.1)	10 (0.1)
	p-value = 0.00*	p-value = 0.00*	p-value = 0.00*	p-value = 0.21*		
Mean number of siblings born [in total] (SD)	3.0 (1.8)	3.0 (2.5)	3.7 (3.0)	3.8 (3.1)	4.4 (2.9)	4.1 (2.9)
	p-value = 0.01*	p-value = 0.00*	p-value = 0.02*	p-value = 0.07*		

* P-values Independent samples T-test or Chi-test exposed versus unexposed category.

Table 3
Results from linear regression analysis with adult height as dependent variable and four exposure groups to the Dutch famine (with and without confounders).

	Basic Model		Model with confounders	
Independent variables	Coefficient [95% CI]	t-stat.	Coefficient [95% CI]	t-stat.
Non-exposed	–	–	–	
Exposed during gestation to age 1	−3.23 [−5.42–−1.04]***	−2.90	−3.70 [−6.04 – −1.36]***	−3.10
Exposed in early childhood (age 1–5)	−0.52 [−2.01–0.90]	−0.69	−0.98 [−2.53–0.55]	−1.25
Exposed in late childhood (age 6–10)	−0.89 [−2.25–0.45]	−1.30	−1.34 [−2.65 – −0.02]*	−2.00
Exposed during puberty (age 11–15)	−0.21[−1.70–1.28]	−0.28	−0.37 [−1.81–1.23]	−0.37
Females	−12.35 [−13.13–−11.57]***	−31.16	−12.31 [−13.09 – −11.53]***	−30.85
Number of siblings ever born:				
0–1			1.39 [0.22–2.57]**	2.34
2–3			0.79 [−0.25–1.83]	1.49
4–5			0.97 [−0.18–2.11]*	1.65
More than 5 (reference category)			–	–
Highest education level parents:				
Primary or less (reference category)			–	–
Lower vocational –Sec. general level			0.02 [−1.02–1.06]	0.03
Medium vocational			0.27 [−0.81–1.35]	0.49
High schools – university			1.74 [0.10–3.38]**	2.08
Dummy Year of birth 1931	−0.22 [−2.52–2.07]	−0.19	−0.22 [−2.53–2.08]	−0.19
Dummy Year of birth 1932	−0.94 [−3.37–1.48]	−0.78	−0.54 [−3.01–1.93]	−0.43
Dummy Year of birth 1933	1.11 [−1.22–3.44]	0.95	1.08 [−1.23–3.39]	0.91
Dummy Year of birth 1934	2.06 [−0.36–4.50]*	1.73	2.31 [−0.09–4.70]**	1.89
Dummy Year of birth 1935	1.49 [−1.00–3.98]	1.23	1.48 [−0.97–3.93]	1.18
Dummy Year of birth 1936	2.68 [0.11–5.25]**	1.98	2.97 [0.42–5.52]**	2.29
Dummy Year of birth 1937	1.92 [−0.68–4.54]	1.59	2.17 [−0.44–4.78]	1.63
Dummy Year of birth 1938	0.65[−1.76–3.07]	0.55	0.79 [−1.62–3.21]	0.64
Dummy Year of birth 1939	1.90 [−0.52–4.33]	1.54	1.94 [−0.48–4.37]	1.57
Dummy Year of birth 1940	2.24 [−0.27–4.76]*	1.85	2.39 [−0.09–4.89]*	1.88
Dummy Year of birth 1941	2.00 [−0.62–4.62]	1.60	2.29 [−0.33–4.93]*	1.71
Dummy Year of birth 1942	2.25 [−0.35–4.86]*	1.85	2.50 [−0.10–5.11]*	1.88
Dummy Year of birth 1943	1.62 [−1.11–4.36]	1.31	1.84 [−0.88–4.58]	1.33
Dummy Year of birth 1944	3.26 [0.50–6.03]**	2.40	3.15 [0.37–5.92]**	2.23
Dummy Year of birth 1945	3.38 [0.72–6.04]**	2.57	3.43 [0.73–6.13]**	2.50
Constant	176.3 [174.3–178.4]***	170.84	175.4 [173.2–177.5]***	161.5
Number individuals	1008		978	
R−squared	0.51		0.52	

* Significant at 10% confidence.
** Significant at 5% confidence.
*** Significant at 1% confidence.

highest parental education, season of birth and degree of urbanization of municipality of residence) were included in the models.

The results per gender are reported in Table 4. Although the direction of the associations was similar for both genders, exposure during gestation to age 1 appeared to be only significant for females (t-value = −2.94; the coefficient for males was only significant at the 10% level). However, the confidence intervals for males and females showed large overlaps, which may indicate that the difference in statistical significance may be due to the relative small sample size. These results hold true when all potential confounders were included in the analyses. The coefficients of the year of birth dummies showed a significant increase in average height of about 5 cm for males between 1932 and 1945 (the coefficient of the year dummies were jointly significant) and of about 4.2 cm for females between 1930 and 1945. Furthermore, daughters in smaller families (≤3 siblings) were on average taller than daughters in larger families. The directions of the associations were similar for sons, though no coefficient was statistically significant at conventional level. In addition, sons of more educated parents were on average significantly taller than sons of less educated parents. Noteworthy, the associations were only significant for the highest educated parents, namely for those who went to high schools or university. The associations were not significant for daughters. Finally, note that, after we stratified per gender, the R-squares were considerably lower.

3.3. Sensitivity analyses

Six sensitivity analyses were performed in order to investigate how robust the regression results were for variations in the cut-off points of the exposure variables (results not shown). The first sensitivity analysis in which the analyses were performed per age group confirmed to a large extent the main results. The second analysis (including confounders) used exposure during "gestation to age 2" instead of during "gestation to age 1" and exposure at ages 10–15 instead of exposure at ages 11–15 to characterize the pubertal period. The results show that females and males exposed before age 2 were significantly shorter than those unexposed (−4.08 cm, t-statistic = −2.57 for females and −4.09 cm, t-statistic = −2.81 for males). The results regarding exposure during puberty

Table 4
Results from linear regression analysis with adult height as dependent variable and four exposure groups to the Dutch famine, confounding variables and dummy variables for year of birth as independent variables (per gender).

	MALES		FEMALES	
Independent variables	Coefficient [95% CI]	t-statistic	Coefficient [95% CI]	t-statistic
Non-exposed	–		–	
Exposed during gestation to age 1	−3.16 [−6.82–0.49]*	−1.70	−4.45 [−7.44 – −1.47]***	−2.94
Exposed in early childhood (age 1–5)	−1.71 [−4.08–0.65]	−1.42	−0.31 [−2.36–1.74]	−0.30
Exposed in late childhood (age 6–10)	−1.70 [−3.79–0.38]	−1.61	−0.85 [−2.56–0.84]	−0.99
Exposed during puberty (age 11–15)	−0.14 [−2.44–2.16]	−0.12	−0.06 [−2.09–1.95]	−0.07
Number of siblings ever born:				
0–1	1.46 [−0.37 – 3.30]	1.56	1.24 [−0.24 – 2.72]*	1.65
2–3	0.39 [−2.08 – 1.29]	−0.46	1.61 [0.28 – 2.94]**	2.38
4–5	1.31 [−0.44 – 3.08]	1.47	0.67 [−0.87 – 2.22]	0.86
More than 5 (reference category)	–	–	–	–
Highest education level parents:				
Primary or less (reference category)	–	–	–	–
Lower vocational –Sec. general level	0.38 [−1.18–1.95]	0.48	−0.29 [−1.69–1.10]	−0.42
Medium vocational	1.29 [−0.48–3.06]	1.43	−0.59 [−1.90–0.71]	−0.90
High schools – university	2.86 [0.41–5.31]**	2.30	0.87 [−1.32 – 3.07]	0.78
Dummy Year of birth 1931	0.12 [−3.32–3.57]	0.07	−0.89 [−3.86–2.07]	−0.60
Dummy Year of birth 1932	−2.57 [−6.31–1.16]	−1.35	0.89 [−2.31–4.10]	0.55
Dummy Year of birth 1933	−0.36 [−3.93–3.21]	−0.20	2.15 [−0.90–5.21]	1.39
Dummy Year of birth 1934	0.67 [−2.91–4.26]	0.37	3.65 [0.50–6.80]**	2.28
Dummy Year of birth 1935	1.86 [−1.82–5.56]	0.99	1.51 [−1.71–4.74]	0.92
Dummy Year of birth 1936	2.65 [−1.03–6.34]	1.41	3.04 [−0.50–6.58]*	1.69
Dummy Year of birth 1937	1.76 [−2.37–5.89]	0.84	2.63 [−0.48–5.75]*	1.66
Dummy Year of birth 1938	−0.64 [−4.52–3.23]	−0.33	1.98 [−1.08–5.05]	1.27
Dummy Year of birth 1939	2.29 [−1.29–5.87]	1.26	1.62 [−1.58–4.82]	1.00
Dummy Year of birth 1940	0.96 [−2.99–4.92]	0.48	3.64 [0.51–6.78]**	2.29
Dummy Year of birth 1941	2.32 [−1.79–6.44]	1.11	2.57 [−0.84–5.99]*	1.48
Dummy Year of birth 1942	2.17 [−1.51–5.86]	1.16	2.79 [−0.88–6.46]*	1.49
Dummy Year of birth 1943	1.70 [−2.25–5.67]	0.85	2.03[−1.62–5.69]	1.09
Dummy Year of birth 1944	1.45 [−2.64–5.54]	0.70	4.48 [0.72–8.24]**	2.34
Dummy Year of birth 1945	2.35 [−1.76–6.48]	1.12	4.22 [0.72–7.73]**	2.37
Constant	176.0 [172.9–179.1]***	111.1	162.4 [159.7–165.1]***	117.1
Number individuals	457		522	
R-squared	0.07		0.06	

* Significant at 10% confidence.
** Significant at 5% confidence.
*** Significant at 1% confidence.

remained insignificant. The third sensitivity analysis used a different definition of exposure in terms of location. The results of the regression analysis (after inclusion of confounders) confirmed again the main results. In the fourth sensitivity analysis, we used self-reported adult height (mean = 172.7 cm; SD = 8.52) instead of measured height at older ages (n = 918). In the analyses stratified by gender, the regression coefficients of exposure during "gestation to age 1" were again relatively large for females and males (-4.52 and −4.04 respectively) and significant (t-statistic = −2.77 for females and t-statistic = −2.03 for males). The coefficients for exposure during early childhood, late childhood and puberty were not significant. Fifth, only including cities in the analyses did not change the main results either. Finally, the main results were only slightly affected by the inclusion of those who lived in the South of the Netherlands during the famine. The results of the analyses (including confounders) regarding females remained highly similar (−4.53 cm and t-statistic = −2.94) to the ones of the main analyses. This was also the case for males (−3.11 cm and t-statistic = −1.73).

4. Discussion and conclusion

The results from our study showed that females exposed during "gestation to age one" appeared to be shorter as adults (on average −4.45 cm) than those who were not exposed. The same was true for those exposed during "gestation to age two" (on average −4.08 cm). The results on females were confirmed in all sensitivity analyses. The results regarding males may seem slightly less convincing. However, the coefficients were also relatively large (on average −3.50 cm), and significant for exposure at ages under 2 (t-statistic = −2.81) and for self-reported height (t-statistic = −2.03). Moreover, the confidence intervals for both genders largely overlapped, which indicates no significant differences in the effect on height of exposure to famine in early life between genders. Exposure during infancy, childhood and puberty was not associated with a shorter adult height. Confounding variables in these relationships were gender, the number of siblings and the highest education level of the parents.

Our results on exposure during "gestation to age 1" and "gestation to age 2" agree with several studies reporting shorter adult height after exposure to undernutrition at the very beginning of life (e.g. Martorell 1995, 1999; Alderman et al., 2006). The fact that the relationships remained significant when including less affected geographical areas seems to indicate that even a smaller nutritional shock may trigger the loss in achieved adult height. Furthermore, the positive associations between parental education and height were only significant for males and the negative associations between family size and height was only significant for daughters. These differences in results may be explained by the gender bias against women in many parts of the world, and perhaps even in mid-20th century Netherlands (as described by Amartya Sen in his seminal paper of 1998, see also McNay et al., 2005). Therefore, women may have had less access to food and care and may have received less attention than men. Moreover, it could be that even higher educated parents provided less support to their daughters than to their sons.

The literature provides limited evidence of the long-term effects of undernutrition during puberty on adult height. The most relevant research investigates the relationships between eating disorders and retarded growth and shows mixed evidence (e.g. Rozé et al., 2007; Soliman et al., 2014). Our study provides evidence that exposure to undernutrition that is severe but of limited duration during puberty does not significantly decrease adult height.

These results may be explained using two theoretical models on (poor) nutrition in early life and health effects in later life. Firstly, the critical period model suggests that the specific period of exposure to undernutrition is most important for long-term effects on the development of an individual and that there would be no effects of exposure on development just before or after this critical period (Ben-Shlomo and Kuh, 2002). The critical period model that is currently considered most prominent is the fetal origins hypothesis ('Barker hypothesis', Barker, 2012). This hypothesis postulates that environmental conditions in utero are the most important for long-term outcomes. Secondly, the accumulation of risk model assumes that it is not one single period of exposure that has long-term effects on later life outcomes, but exposures during gestation, childhood, adolescence and adulthood are all of importance (Kuh and Ben-Shlomo, 2004). Therefore, the effects of exposures accumulate during life and together with the degree of exposure determine the seriousness of the long-term effects on later life outcomes. On the one hand, the results of our study contribute evidence to the critical period model. Respondents exposed during "gestation to age 1 or 2" might have been stunted during the famine period and might have experienced a period of catch up growth after the end of the Dutch famine. However, if catch-up growth occurred, it did not fully compensate for their relatively short height, which is consistent with the results reported by Coly et al. (2006). On the other hand, the positive cohort effects for females in 1944–45 compared to those born in 1930, are not likely to be the results of better conditions in very early life, since this was a period of war. Improved conditions later in life could be a more plausible explanation. This would point towards the accumulation model.

Finally, it is interesting to see that the coefficients for exposure during "gestation to age one" and during "late childhood" became larger and/or significant after inclusion of the variables "highest education level of the parents" and "number of siblings". This may be explained as follows. Table 2 reports significant differences in education level of the parents and in the number of children between the exposed, more urban, areas and the unexposed, more rural areas. These differences should have resulted in a shorter average height in the unexposed areas since taller adult height is positively associated with higher socioeconomic status (Case and Paxson, 2008). However, because of the Dutch famine, the individuals in the more urban (exposed) areas were as short as in the more rural (unexposed) areas and the height difference only showed up after inclusion of the socioeconomic covariates. With respect to the number of siblings, lower education parents had on average more children than others, and we may speculate that the higher the number of siblings, the smaller the food rations per child would be.

5. Strengths & limitations

A limitation of our study is that the findings might be partly biased by selection effects. Many years have passed since the Dutch famine, which implies that only the healthiest exposed individuals were still alive during the LASA measurements. It is plausible that many potential respondents could not participate because of health-related impairments, and others may have already died before the LASA study started. This implies that our results might underestimate the true relationship between exposure to undernutrition early in life and height. This selection effect is possibly stronger in people exposed during puberty than in people exposed at younger ages, since the respondents in the pubertal group were older at the time of measurement.

A second limitation is that the exposure variables used in our study may not measure the actual exposure to undernutrition during the Dutch famine. Exposure to the Dutch famine is defined using the municipality of residence in 1944 as no information was available on the extent to which respondents actually suffered

from undernutrition during the famine. Some respondents may have had better access to food than others (for instance on the black market or because of their social network or profession). However, there is historical evidence that the very large majority of the population living in the large cities of the West of the Netherlands suffered severely from the famine (Lumey and Van Poppel, 2013). Furthermore, the municipality of residence in 1944 is self-reported and thus to some extent subject to recall bias. However, we believe that most respondents knew where they were living during this exceptional and tragic period in Dutch history (Draaisma, 2004).

The sample size of the exposed group "gestation to age 1" may be a third limitation of the study. The sample included very few respondents exposed during gestation, and although these were grouped with respondents exposed during their first year of life, the group exposed during "gestation to age 1" included only 26 respondents. This also prevented us from analyzing the effects on adult height of undernutrition during gestation and during the first year of life separately. Another limitation is that the variable which describes "the total number of the respondents' siblings born" may not be a precise representation of the number of siblings who were actually alive during the Dutch famine. Since several siblings may already have died before this exposure period, there may be an overestimation of the actual number of siblings during this period. Finally, the variable "highest education level of the parents" was used to describe the potentially confounding effects of parental socio-economic factors. However, socio-economic status may be described by many other variables such as income or wealth (Grundy and Holt, 2001). Unfortunately, these variables were not available for the respondents' parents in the LASA data.

Finally, there is evidence suggestive of a negative effect of maternal smoking on heights of their offspring (Koch et al., 2012; Martínez-Mesa et al., 2012). However, very few women born at the beginning of the 20th century smoked (Barendregt et al., 1997). Because of that, we believe that not correcting for maternal smoking behavior did not significantly affect our results.

This study has a number of clear strengths: to the best of our knowledge, this is the first study on the relationship between undernutrition and adult height during the Dutch famine of 1944–1945. As described in the introduction, the Dutch famine is a highly appropriate historical period for the identification of the long-term effects of undernutrition, as it can be considered a natural experiment. A second strength is that we performed several sensitivity analyses which confirmed most of our results. For all respondents, a minimum of 13 weeks of exposure during the Dutch famine is used as an inclusion criterion, as defined in relevant research (Lumey et al., 2007). Another strength is that adult height was measured in two ways: firstly with a stadiometer using data at the earliest measurement for which it was available, as individuals are likely to shrink when getting older and secondly, using self-reports of adult height.

In conclusion, our study shows that exposure to undernutrition during "gestation to age 1 or 2" was significantly associated with a shorter adult height for both genders. Results of this study are valuable in the field of (under)nutrition and health research. Tragedies like the Dutch famine of 1944–45 may not happen again in a country such as the Netherlands, but undernutrition is still a major issue in developing countries worldwide.\

Acknowledgment and ethics approval

The Longitudinal Aging Study Amsterdam is largely supported by a grant from the Netherlands Ministry of Health Welfare and Sports, Directorate of Long-Term Care.

The LASA study is conducted in line with the Declaration of Helsinki, and was approved by the medical ethics committee of the VU University Medical Center. All study participants signed an informed consent.

Appendix A.

References

Alderman, H., Hoddinott, J., Kinsey, B.H., 2006. Long term consequences of early childhood malnutrition. Oxford Econ. Pap. 58 (3), 450–474.
Alter, G., Oris, M., 2008. Effects of inheritance and environment on the heights of brothers in nineteenth-century Belgium. Hum. Nat. 19, 44–55.
Barendregt, J.J., Bonneux, L.B., van der Maas, P.J., 1997. The health care costs of smoking. N. Engl. J. Med. 337, 1052–1057.
Barker, D.J.P., 2012. Development origins of chronic disease. Public Health 126 (3), 185–189.
Barnouw, D.C., 1999. The Dutch Famine. Uitgeverij Verloren, Hilversum (in Dutch.
Batty, G.D., Shipley, M.J., Gunnell, D., Huxley, R., Kivimaki, M., Woodward, M., Smith, G.D., 2009. Height, wealth, and health: an overview with new data from three longitudinal studies. Econ. Hum. Biol. 7 (2), 137–152.
Ben-Shlomo, Y., Kuh, D., 2002. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int. J. Epidemiol. 31 (2), 285–293.
Black, R.E., Allen, L.H., Bhutta, Z.A., Caulfield, L.E., De Onis, M., Ezzati, M., et al., 2008. Maternal and child undernutrition: global and regional exposures and health consequences. Lancet 371 (9608), 243–260.
Bozzoli, C., Deaton, A., Quintana-Domeque, C., 2009. Adult height and childhood disease. Demography 46 (4), 647–669.
Bryce, J., Coitinho, D., Darnton-Hill, I., Pelletier, D., Pinstrup-Andersen, P., 2008. Maternal and child undernutrition study group. Lancet 371 (9611), 510–526.
Case, A., Paxson, C., 2008. Stature and status: height, ability, and labor market outcomes. J. Polit. Econ. 116 (3), 499–532 University of Chicago Press.
Cavelaars, A.E.J.M., Kunst, A.E., Geurts, J.J.M., Crialesi, R., Grötvedt, L., Helmert, U., Lahelma, E., Lundberg, O., Mielck, A., Rasmussen, N., Regidor, E., Spuhler, T., Mackenbach, J.P., 2000. Persistent variations in average height between countries and between socio-economic groups: an overview of 10 European countries. Ann. Hum. Biol. 27 (4), 407–421.
Coly, A.N., Milet, J., Diallo, A., Ndiaye, T., Bénéfice, E., Simondon, F., et al., 2006. Preschool stunting, adolescent migration, catch-up growth, and adult height in young Senegalese men and women of rural origin. J. Nutr. 136 (9), 2412–2420.
Day, F.R., Forouhi, N.G., Ong, K.K., Perry, J.R.B., 2015. Season of birth is associated with birthweight, pubertal timing, adult body size and educational attainment: a UK Biobank study. Heliyon 1 (2) .
De Jong, L., 1981. The Netherlands during the Second World War: 1939e1945, part 10B, first half. 's-Gravenhage: Staatsuitgeverij. (in Dutch).
De Onis, M., Blossner, M., 2003. The world health organization global database on child growth and malnutrition: methodology and applications. Int. J. Epidemiol. 32 (4), 518–526.
Deaton, A., Arora, R., 2009. Life at the top: the benefits of height. Econ. Hum. Biol. 7 (2), 133–136.

Deaton, A., 2008. Height, health, and inequality: the distribution of adult heights in India. Am. Econ. Rev. 98 (2), 468–474.
Deeg, D.J.H., van Tilburg, T., Smit, J.H., de Leeuw, E.D., 2002. Attrition on the longitudinal aging study Amsterdam: the effect of differential inclusion in side studies. J. Clin. Epidemiol. 55, 319–328.
Dewey, K.G., Begum, K., 2011. Long-term consequences of stunting in early life. Matern. Child Nutr. 7 (s3), 5–18.
Draaisma, D., 2004. Why Life Speeds up as You Get Older? How Memory Shapes Our Past. Cambridge University Press, Cambridge.
Fu, H., Goldman, N., 1996. Incorporating health into models of marriage choice: demographic and sociological perspectives. J. Marriage Fam. 58 (3), 740–758.
Grundy, E., Holt, G., 2001. The socioeconomic status of older adults: how should we measure it in studies of health inequalities? J. Epidemiol. Community Health 55 (12), 895–904.
Hamill, P., Johnston, F., Health, Lemeshow S., 1972. Weight of children: socioeconomic status. Vital Health Stat. 119, 1–87.
Huisman, M., Poppelaars, J.L., Van der Horst, M.H.L., Beekman, A.T.F., Brug, J., Van Tilburg, T.G., Deeg, D.J.H., 2011. Cohort profile: the longitudinal aging study Amsterdam. Int. J. Epidemiol. 40, 868–876.
Ki-Moon, B., 2007. Children and the Millennium Development Goals: Progress Towards a World Fit for Children. UNICEF, New York.
Klump, K.L., 2013. Puberty as a critical risk period for eating disorders: a review of human and animal studies. Horm. Behav. 64 (2), 399–410.
Koch, S., Vilser, C., Groß, W., Schleußner, E., 2012. Smoking during pregnancy – risk for intrauterine growth retardation and persisting microsomia. Z. Geburtshilfe Neonatol. 216 (2), 77–81.
Koupil, I., Shestov, D.B., Sparén, P., Plavinskaja, S., Parfenova, N., Vågerö, D., 2007. Blood pressure, hypertension and mortality from circulatory disease in men and women who survived the siege of Leningrad. Eur. J. Epidemiol. 22 (4), 223–234.
Kuh, D., Ben-Shlomo, Y., 2004. A Life Course Approach to Chronic Disease Epidemiology. Oxford University Press, Oxford, U.K.
Lumey, L.H., Van Poppel, F.W.A., 1994. The Dutch famine of 1944–45: mortality and morbidity in past and present generations. Soc. Hist. Med. 7 (2), 229–246.
Lumey, L.H., Van Poppel, F.W.A., 2013. The Dutch Famine of 1944–45 as a laboratory: changes in the early life environment and adult height. Early Life Nutrition and Adult Health and Development, .
Lumey, L.H., Stein, A.D., Kahn, H.S., van der Pal-de, K.M., Blauw, G.J., Zybert, P.A., et al., 2007. Cohort profile: the dutch hunger winter families study. Int. J. Epidemiol. 36 (6), 1196–1204.
Marceau, K., Ram, N., Houts, R.M., Grimm, K.J., Susman, E.J., 2011. Individual differences in boys' and girls' timing and tempo of puberty: modeling development with nonlinear growth models. Dev. Psychol. 47 (5), 1389.
Martínez-Mesa, J., Menezes, A.M., González, D.A., Horta, B.L., Matijasevich, A., Gigante, D.P., Hallal, P.C., 2012. Life course association of maternal smoking during pregnancy and offspring's height: data from the 1993 Pelotas (Brazil) birth cohort. J. Adolesc. Health 51 (December (6 Suppl.)), S53–7.
Martorell, R., Khan, L.K., Schroeder, D.G., 1994. Reversibility of stunting: epidemiological findings in children from developing countries. Eur. J. Clin. Nutr. 48 (1), S45–57.
Martorell, R., 1995. Results and implications of the INCAP follow-up study. J. Nutr. 125 (4 Suppl), 1127S–1138S.
Martorell, R., 1999. The nature of child malnutrition and its long-term implications. Food Nutr. Bull. 20 (3), 288–292.
McNay, K., Humphries, J., Klasen, S., 2005. Excess mortality in nineteenth-century England and Wales: a regional analysis. Soc. Sci. Hist. 29 (4), 49–681.
Modan-Moses, D., Yaroslavsky, A., Novikov, I., Segev, S., Toledano, A., 2012a. Stunting of growth as a major feature of anorexia nervosa in male adolescents. Pediatrics 111, 270–276.
Modan-Moses, D., Yaroslavsky, A., Kochavi, B., Toledano, A., Segev, S., Balawi, F., Mitrany, E., Stein, D., 2012b. Linear growth and final height characteristics in adolescent females with anorexia nervosa. PLoS One 7 (9), e45504.
Portrait, F., Teeuwiszen, E., Deeg, D., 2011. Early life undernutrition and chronic diseases at older ages: the effects of the Dutch famine on cardiovascular diseases and diabetes. Soc. Sci. Med. 73 (5), 711–718.
Rietveld, C.A., Hessels, J., van der Zwan, P., 2015. The stature of the self-employed and its relation with earnings and satisfaction. Econ. Hum. Biol. 17, 59–74.
Rogol, A.D., Clark, P.A., Roemmich, J.N., 2000. Growth and pubertal development in children and adolescents: effects of diet and physical activity. Am. Soc. Clin. Nutr. 72 (2), 521–528.
Roseboom, T.J., Painter, R.C., van Abeelen, A.F., Veenendaal, M.V., de Rooij, S.R., 2011. Hungry in the womb: what are the consequences? Lessons from the Dutch famine. Maturitas 70 (2), 141–145.
Rozé, C., Doyen, C., Le Heuzey, M., Armogum, P., Mouren, M., Léger, J., 2007. Predictors of late menarche and adult height in children with anorexia nervosa. Clin. Endocrinol. 67 (3), 462–467.
Sen, A., 1998. Mortality as an indicator of economic success and failure. Econ. J. 108, 1–25.
Shestov, D.B., Koupil, I., Plavinskaja, S., Parfenova, N., Danziger, P.D., Vågerö, D., 2009. Cancer mortality in women and men who survived the siege of Leningrad (1941–1944). Int. J. Cancer 124 (6), 1416–1421.
Siervogel, R.M., Demerath, E.W., Schubert, C., Remsberg, K.E., Chumlea, W.C., Sun, S., et al., 2003. Puberty and body composition. Horm. Res. 60, 36–45.
Silvertoinen, K., Lahelma, E., Rahkonen, O., 1999. Social background, adult body height and health. Int. J. Epidemiol. 28, 911–918.
Silvertoinen, K., Sammalisto, S., Perola, M., Boomsma, D.I., Cornes, B.K., Davis, C., et al., 2003. Heritability of adult body height: a comparative study of twin cohorts in eight countries. Twin Res. 6, 399–408.
Silvertoinen, K., 2003. Determinants of variation in adult body height. J. Biosoc. Sci. 35 (2), 263–285.
Smith, J., Monden, C., 2012. Taller Indian women are more successful at the marriage market. Am. J. Hum. Biol. 24, 473–478.
Soliman, A., De Sanctis, V., Elalaily, R., 2014. Nutrition and pubertal development. Indian J. Endocrinol. Metab. 18 (Suppl. 1), S39–S47.
Statistics Netherlands, 2004. Kerncijfers postcodegebieden. .
Stewart, A.W., Jakson, R.T., Ford, M.A., Beaglehole, R., 1985. Underestimation of relative weight by use of self-reported height and weight. Am. J. Epidemiol. 125 (1), 122–126.
Twisk, J.W.R., 2007. Inleiding in de toegepaste biostatistiek. Elsevier Gezondheidszorg, Maarssen.
Waaler, H.T., 1984. Height, weight and mortality, the norwegian experience. Acta Med. Scand. 679 (Suppl), 1–56.
Wang, Y., Wang, X., Kong, Y., Zhang, J.H., Zeng, Q., 2010. The great Chinese famine leads to shorter and overweight females in Chongqing Chinese population after 50 years. Obesity 18 (3), 588–592.
Wells, J.C.K., 2011. Re-examining heritability: genetics, life history and plasticity. Trends Endocrinol. Metab. 22 (10), 421–428.
Yang, J., Benyamin, B., McEvoy, B.P., Gordon, S., Henders, A.K., Nyholt, D.R., Madden, P.A., Heath, A.C., Martin, N.G., Montgomery, G.W., Goddard, M.E., Visscher, P.M., 2010. Common SNPs explain a large proportion of the heritability for human height. Nat. Genet. 42, 565–569.
Zheng-Wang, B., Cheng-Ye, J., 2005. Secular growth changes in body height and weight in children and adolescents in Shandong, China between 1939 and 2000. Ann. Hum. Biol. 32 (5), 650–665.