PROCEEDINGS B

rspb.royalsocietypublishing.org

Research

Cite this article: Stulp G, Barrett L, Tropf FC, Mills M. 2015 Does natural selection favour taller stature among the tallest people on earth? *Proc. R. Soc. B* **282**: 20150211. http://dx.doi.org/10.1098/rspb.2015.0211

Received: 30 January 2015
Accepted: 13 March 2015

Subject Areas:
behaviour, evolution

Keywords:
height, natural selection, The Netherlands, fertility, reproductive success, LifeLines

Author for correspondence:
Gert Stulp
e-mail: gert.stulp@lshtm.ac.uk

Electronic supplementary material is available at http://dx.doi.org/10.1098/rspb.2015.0211 or via http://rspb.royalsocietypublishing.org.

THE ROYAL SOCIETY
PUBLISHING

Does natural selection favour taller stature among the tallest people on earth?

Gert Stulp[1,2], Louise Barrett[3,4], Felix C. Tropf[2] and Melinda Mills[5]

[1]Department of Population Health, London School of Hygiene and Tropical Medicine, London WC1E 7HT, UK
[2]Department of Sociology, University of Groningen, Grote Rozenstraat 31, Groningen 9712 TG, The Netherlands
[3]Department of Psychology, University of Lethbridge, 4401 University Drive West, Lethbridge, Alberta, Canada T1 K 3M4
[4]Applied Behavioural Ecology and Ecosystems Research Unit, University of South Africa, Private Bag X6 Florida 1710, Johannesburg, South Africa
[5]Nuffield College/Department of Sociology, Manor Road, Oxford OX1 3UQ, UK

GS, 0000-0003-0173-5554; MM, 0000-0003-1704-0001

The Dutch are the tallest people on earth. Over the last 200 years, they have grown 20 cm in height: a rapid rate of increase that points to environmental causes. This secular trend in height is echoed across all Western populations, but came to an end, or at least levelled off, much earlier than in The Netherlands. One possibility, then, is that natural selection acted congruently with these environmentally induced changes to further promote tall stature among the people of the lowlands. Using data from the LifeLines study, which follows a large sample of the population of the north of The Netherlands ($n = 94\,516$), we examined how height was related to measures of reproductive success (as a proxy for fitness). Across three decades (1935–1967), height was consistently related to reproductive output (number of children born and number of surviving children), favouring taller men and average height women. This was despite a later age at first birth for taller individuals. Furthermore, even in this low-mortality population, taller women experienced higher child survival, which contributed positively to their increased reproductive success. Thus, natural selection in addition to good environmental conditions may help explain why the Dutch are so tall.

1. Background

When it comes to height, the Dutch have a remarkable history. In the mid-eighteenth century, the average height of Dutch (military) men was approximately 165 cm. This was well below the average for other European populations, and very much shorter than the average height of men in the United States, who towered over the Dutch by 5–8 cm [1–3]. Dutch men are now the tallest in the world, having grown by approximately 20 cm over the last 150 years [3,4]. By contrast, male height in the United States has increased by only 6 cm across the same time span. Equivalent differences in height are also observed between The Netherlands and other European countries. Indeed, it is notable that, while the secular trend in height has slowed or stopped in most North-European countries [5], it has continued for much longer among the Dutch [6,7], with the available evidence suggesting it has begun to slow only very recently [4].

The Dutch superiority in height has been attributed to various environmental factors, including nutrition, particularly the heavy consumption of dairy products [6,8], and low levels of social inequality, with the provision of high-quality, universal healthcare [1]. By contrast, the United States has experienced growing levels of social inequality over the last 150 years, despite equivalent (if not higher) levels of overall wealth compared with The Netherlands [2,9,10], and these may be responsible for the much smaller increase in average height.

Environmental differences may not be the whole story, however. Recently, Byars *et al.* [11] showed, on the basis of pedigree data, that natural selection was acting on height among United States women: shorter women had higher lifetime reproductive success than taller women, and their descendants were, on average, predicted to be slightly shorter than they would have been in the absence of selection. Stulp *et al.* [12,13] showed phenotypic associations between stature and reproductive success in the United States, such that shorter women had higher reproductive success than their taller counterparts [12], while average height men experienced greater reproductive success than taller or shorter men ([13]; findings that were replicated by [14] and [15]). These findings are interesting, not only because they suggest that selection has been acting on height in contemporary Western populations but also because they deviate from expectations with respect to expressed mate preferences, which favour taller men and average height women [16,17]. In addition, they show that environmental influences vary across populations: the factors that tend to push up average height clearly operate more strongly in The Netherlands than they do in the United States. Furthermore, it appears that, in the United States, natural selection is working against environmental factors to actively favour shorter stature.

It therefore becomes relevant to ask whether natural selection has also exerted an influence on stature among the Dutch, and whether selection pressures differ to those in the United States. Specifically, the recent large increase in height suggests the possibility that environmental and selective factors operate congruently among the Dutch, rather than antagonistically as seen in the United States. Here, we test the prediction that, in The Netherlands, taller men and women have experienced higher reproductive success. We examine how both male and female height are related to fertility (i.e. the number of children born) in a very large sample (approx. 90 000) of individuals from the Northern three provinces of The Netherlands. In doing so, we also address factors that potentially underlie any observed associations, including child survival, aspects of mate choice and partnership formation and the timing of births.

2. Material and methods

LifeLines is a multidisciplinary, prospective population-based cohort study examining the health and health-related behaviours of 167 729 persons living in the northeast region of The Netherlands. It employs a unique three-generation design and covers a broad range of investigative procedures in assessing the biomedical, socio-demographic, behavioural, physical and psychological factors which contribute to the health and disease of the general population, with a special focus on multimorbidity and complex genetics. In addition, the LifeLines project comprises a number of cross-sectional sub-studies that investigate specific age-related conditions. These include investigations into metabolic and hormonal diseases, including obesity, cardiovascular and renal diseases, pulmonary diseases and allergy, cognitive function and depression, and musculoskeletal conditions. The study design involves initial contact with a random sample of people aged between 25 and 50 years, who are invited to participate ($n = 54\,702$). Subsequently, their family members (if present) are invited to join the study (i.e. parents, partner, parents in law, children), resulting in the sampling of three generations across each family [18]. Here, we have used data collected during the first wave of the study ('Data Release 2013 I'; $n = 94\,516$), with all participants being between 18 and 93 years of age. All participants signed an informed consent form during the first visit at one of the LifeLines research locations.

In the data that we received, family members and spouses could not be linked to one another. Thus, we cannot avoid a certain amount of pseudoreplication owing to the similarity between family members and spouses. Although not ideal, we believe that this does not constitute a major problem for our analyses because: (i) given the long generation time of humans, there is unlikely to be much overlap across generations because we only include only men and women above the age of 45, and those born within a specific time period (1935–1967); and (ii) we are primarily interested in how selection acts on height within each sex, given that previous research suggests different pathways by which the sexes achieve higher reproductive success (e.g. [17]). While genotype information is available for a subsample of the LifeLines study, the selection of data for the current study means that the sample available for genetic analyses is rather small (approx. 2000 per sex), and would be severely underpowered for any statistical approach using non-family members, particularly for the bivariate model [19].

(a) Selection of sample

As fertility-related traits fluctuate across time (see the electronic supplementary material, figures S1 and S2), we stratified birth year into 5-year cohorts (e.g. 1935–1939, 1940–1944, ..., 1965–1967). We excluded birth cohorts prior to 1935 owing to insufficient sample size (excluding 846 cases). We also included only those men and women who were 45 years of age and older (as virtually all of these have completed reproduction), which further restricted our sample to the birth year, 1967. Many women show perimenopausal symptoms at this age accompanied by higher levels of sterility [20]. In this sample only 0.1% of women reproduced above the age of 45. Although men are physically able to reproduce to a very late age, we observed that only 1.7% of men in our sample had their last child after the age of 45.

Finally, we included only heterosexual couples, and only those individuals who were born in The Netherlands and whose parents were also born in The Netherlands. Cases in which fertility variables were inconsistent, where relevant information was missing, including values for height, or when the age at first birth was younger than 16, were excluded from the analyses, leaving a sample of $n = 42\,612$ (see the electronic supplementary material, table S1 for the number of cases that were dropped owing to each restriction).

(b) Fertility variables

Data are available for the sex, birth and death dates for up to six biological children of the respondent and his or her current partner. These same questions were also asked for up to six biological children produced within a previous partnership. We included fertility data only for those respondents who gave full information on the birth-date and sex of all their children. Based on these variables, we computed the total number of children ever born, the total number that survived to the age of 18, and the age at first and last birth. We excluded all those cases in which respondents reported an age at first birth younger than 16 (to exclude errors in reporting for very young ages at first birth).

Respondents were also asked about relationship duration, which we used to calculate the age at onset of the current relationship. We excluded those cases in which the age at the start of the relationship was reported as being younger than 16. From this variable, we calculated the timing of the first birth with the current partner from the start of the relationship. The age at the start of the relationship does not necessarily correspond to the age at the start of an individual's first relationship, nor the first successful (reproductive) union. We excluded cases in which the age at first birth with the current partner was younger than the age reported at the beginning of the current relationship as a means to exclude reporting errors, but, by necessity, this will also exclude any individuals who experience their first birth with a previous partner. This in turn means that, when examining the timing of the first birth from the onset of the current relationship, we could only calculate values for those who had experienced a first birth with their current partner.

We reran analyses pertaining to the age at onset of the current relationship and the time to first birth, including only those individuals that had never been divorced, widowed, and who did not have children with any previous partners. Results did not differ (see the electronic supplementary material, tables S6 and S10).

We also included only those women who reached menarche between 8 and 20 years of age. Very late menses may signal potential problems with ovarian function, which would have a subsequent effect on fertility-related traits. Something similar may also be true for very young and old ages at menopause, hence we restricted age at menopause to between 30 and 60. Although these cases are of interest given that age at menses and menopause are plausibly related to fertility outcomes, we cannot exclude the possibility of reporting errors, and our sample is therefore conservative.

(c) Mate choice variables

Marital status was coded as Married/Registered partnership (79.9%), Living together (6.6%), Living alone (4.0%), Widow (2.9%), Divorced (4.4%), Living apart together (2.0%) and Other (0.3%). From these, we created a variable 'ever had a partner', which coded all categories except 'Living alone' as 1 (96.0%), with 'Living alone' coded as 0 (4.0%; we excluded the 'Other' category). Our second mate choice variable concerned current relationship status. We deduced whether a participant was in a current relationship from the survey question concerning the sex of their partner, i.e. we included only those men and women that reported an opposite-sex partner, as this was the only means available to assess current status. Those that did not report on the sex of the partner were not considered to be in a current relationship. Based on this classification, 87.6% of participants currently had a partner, whereas the rest did not.

(d) Other variables of interest

A number of our analyses control for education, income and health, as these are often considered to be important confounds of height [12,13,21–25]. Health, education and income were provided at the time of the interview. Such measures cannot, therefore, be taken as indicative of childhood environment, and so our analyses do not control for any environmental influences that affect early growth and subsequent adult height. The homogeneity of the population with respect to health differentials, and the wide provision of healthcare, suggest any such influence is likely to be small. Instead, our analyses control for health, education and income in adulthood as possible mediators of the influence of adult height on fertility. Education was partitioned into nine categories: No education, Primary education, Lower or preparatory vocational education, Lower general secondary education, Intermediate vocational education or apprenticeship, Higher general secondary education or pre-university secondary education, Higher vocational education, University and Other education. We excluded those that reported 'Other' ($n = 1801$; 1.9%). Net income per month (in euros) was divided into the following categories: Don't know, Rather not say, less than 750 euros, 750–1000, 1000–1500, 1500–2000, 2000–2500, 2500–3000, 3000–3500, more than 3500. Some respondents filled in an older questionnaire that meant they could not be placed into one of the above categories. For our analyses, we included only those who reported their income (79.2%). Self-perceived health was rated as either Excellent, Very good, Good, Not so good or Poor. Self-reported health is a valid general assessment of one's health status, covering largely physical and functional aspects of health, but is not connected to any specific illness, and has been shown to predict mortality and morbidity and to have a high test–retest reliability in a number of studies. This variable has, furthermore, been recommended for comparative research by the World Health Organization and many researchers have followed this advice (see [26] and references therein). Height was measured by researchers, and the measured height was subsequently verified by the respondent. Height was missing for only 29 individuals.

See the electronic supplementary material, table S2 for descriptive statistics of the entire sample.

(e) Analysis

We used Poisson, logistic and linear regression, depending on the distribution of our dependent variable. For our 'timing' measures (e.g. age at first and last birth, age at menses), we used linear regressions, but survival analyses produced very similar results (not reported here).

To correct for any secular changes in the dependent variables over time (e.g. changes in fertility and age at first birth; see the electronic supplementary material, figure S1), we included birth cohort as a factor in all our analyses (i.e. the seven different 5 year cohorts). To correct for the secular trend in height over time, we calculated a running average of height (5 year bins) across time for each sex, and we did the same for the standard deviation of height. Thus, the mean and the standard deviation of height in a given year, e.g. 1950, are represented by the mean and s.d. of heights within that year plus the two previous and two subsequent years (1948–1952). We standardized an individual's height by subtracting the running average height for the birth year of the respondent, and dividing it by the running s.d. Thus, a Z-score of 1.0 indicates an individual who is one standard deviation above average height for their sex within a given birth year (see the electronic supplementary material, table S3 and figure S2). We included an interaction term between height and birth cohort to examine whether height was differentially related to the dependent variable across birth cohorts. In only four (out of 51) analyses was a significant interaction found (electronic supplementary material, tables S4–S14). For those cases where there was a significant interaction, it was never the case that the relationship changed in terms of sign and remained significant (i.e. only the magnitude of the effect of height changed, with some cohorts displaying significant effects of height and others showing no effect). We do not discuss these interactions further.

To assess whether there were any nonlinear patterns with respect to the effects of height on any of the dependent variables, we included a quadratic term for height, while always including a linear term for height, even when this was non-significant. In what follows, if neither height nor height^2 was significant in the analyses, we present the model with both variables. To examine the optima of curvilinear effects, we obtained a 95% confidence interval for the optimum by simulating the model that included both the linear and quadratic term over 1000 iterations (using the 'simulate()' function in R), and refitting the statistical model. Through this method, 1000 parameter estimates and hence optima are generated, from which we could determine a 95% range, which we used as our confidence interval.

To provide a measure of effect size that was comparable across the different measures and sexes, we estimated the smallest (minimum) and largest (maximum) predicted value (based on the parameter estimates in the electronic supplementary material, tables S4–S14) within -2 and $+2$ s.d. of the height range. We expressed the ratio between this maximum and minimum (maximum/minimum) in percentages. This measure of 'effect size' is not comparable with more traditional effect size estimates. All analyses were performed in R [27].

3. Results

See table 1 and figure 1 for an overview of the results, and the electronic supplementary material, tables S4–S14 for parameter estimates of all analyses.

Table 1. Summary of results.

variable	men			women		
	effect of height[a]	optimum (CI_{95})[b]	effect size (min–max)[c]	effect of height[a]	optimum (CI_{95})[b]	effect size (min–max)[c]
ever-born children	curvilinear	1.016 (0.504, 2.242)	11.6% (2.14–2.39)	quadratic	−0.081 (−1.163, 0.768)	3.0% (2.29–2.35)
ever-born with controls[d]	curvilinear	0.630 (0.060, 2.224)	7.3% (2.20–2.36)	quadratic	−0.139 (−0.990, 0.585)	3.8% (2.27–2.35)
no. children after firstborn	no effect		2.7% (2.47–2.54)	no effect		1.1% (2.50–2.53)
ever had child	curvilinear	1.265 (0.688, 2.803)	4.5% (0.89–0.93)	quadratic	−0.388 (−1.420, 0.194)	1.5% (0.92–0.93)
ever had 2nd child	positive		2.9% (0.92–0.95)	no effect		0.6% (0.93–0.93)
proportion surviving	no effect			curvilinear	1.811 (0.421, 6.692)	0.7% (0.98–0.99)
surviving children	curvilinear	0.996 (0.506, 2.146)	12% (2.11–2.36)	quadratic	0.058 (−0.493, 0.601)	3.8% (2.24–2.33)
has current partner	curvilinear	1.808 (0.381, 10.951)	2.4% (0.93–0.96)	quadratic	−0.354 (−1.297, 0.153)	2.5% (0.86–0.88)
ever had partner	curvilinear	1.188 (0.423, 3.934)	1.1% (0.97–0.99)	curvilinear	−0.772 (−2.419, −0.204)	1.1% (0.97–0.98)
age in relationship	curvilinear	−0.582 (−2.202, −0.0001)	2.8% (26.76–27.50)	quadratic	−0.759 (−5.692, 2.399)[e]	2.0% (24.55–25.05)
no. children with current partner given age when in relationship[f]	positive		7.4% (2.05–2.20)	positive		6.7% (2.19–2.33)
age at first birth	curvilinear	−0.771 (−4.685, 0.175)	1.4% (27.46–27.84)	curvilinear	−3.452 (−14.495, −1.730)	4.8% (24.25–25.40)
age at last birth	no effect		0.9% (32.31–32.60)	curvilinear	−2.705 (−10.120, −1.351)	3.3% (29.25–30.22)
time until birth child since onset relationship[f]	no effect		4.2% (3.74–3.90)	positive		16.9% (3.04–3.55)
age at menses				positive		5.3% (12.87–13.55)
age at menopause				positive		1.2% (49.36–49.95)
reproductive span[g]				no effect		0.1% (36.42–36.47)

[a]The effect of height on the dependent variable. 'Curvilinear' indicates a nonlinear pattern with an optimum significantly different from the average height (with both the estimates of height and $height^2$ significant), whereas 'quadratic' refers to a nonlinear pattern with an optimum around average height (with only $height^2$ significant).

[b]Confidence interval determined through simulating the data (see text).

[c]Our measure of the size of the effect; smallest (minimum) and largest (maximum) predicted value within −2 and +2 standard deviations of the height range; the percentage reflects maximum/minimum. This is not a 'traditional' effect size.

[d]Controlled for education, health and income.

[e]The effect of height is weak and marginally significant, which is why the optimum cannot be established very accurately.

[f]This 'effect size' was established for the median age in the relationship (22 for women, 25 for men).

[g]Difference between age at menses and age at menopause.

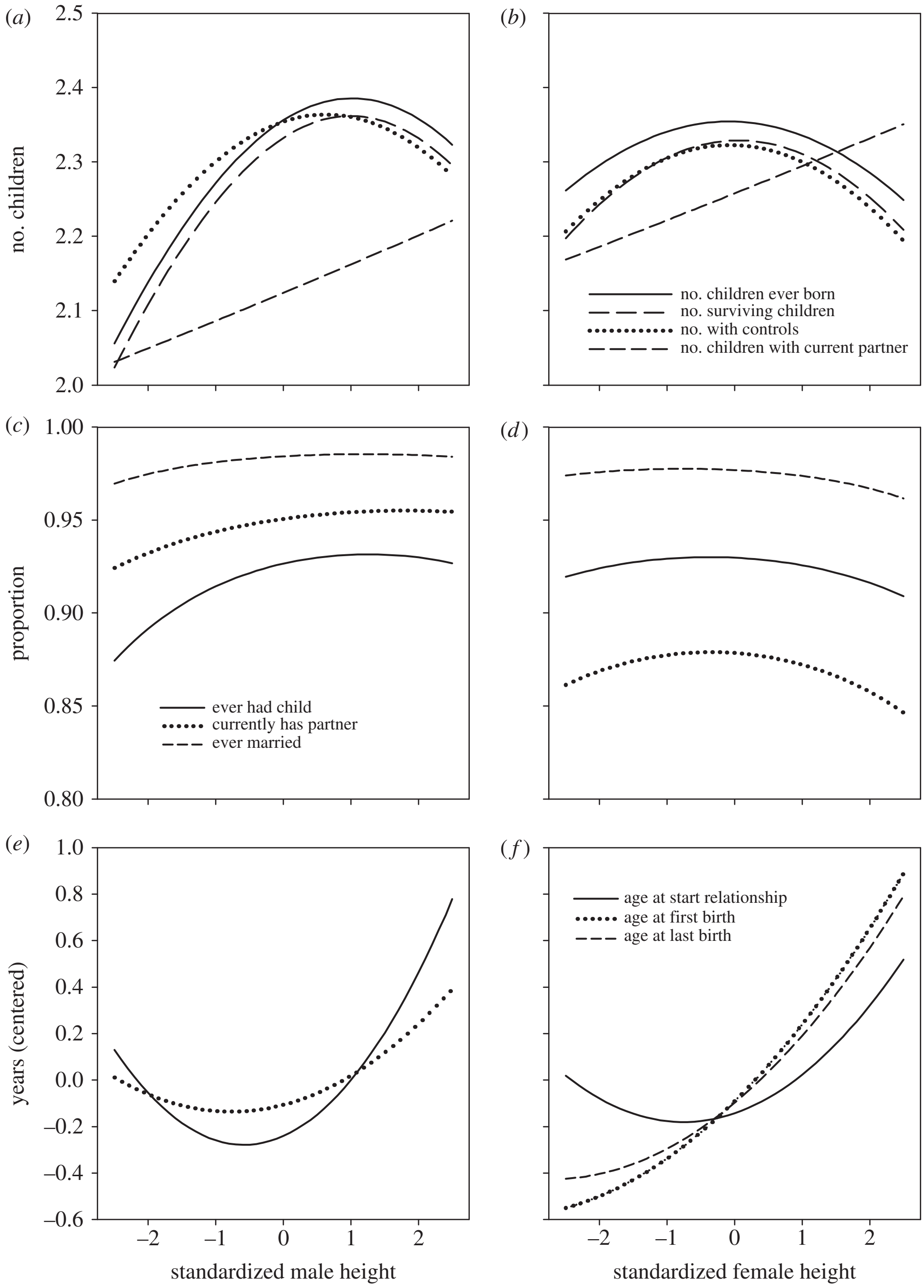

Figure 1. Model predictions for the effect of (*a*,*c*,*e*) male and (*b*,*d*,*f*) female height (standardized) on several variables. The height range between −2.5 and 2.5 standard deviations is plotted. Models evaluated for birth cohort 1950–1954. The number of children with current partner (*c* and *d*) is evaluated at the median age at the start of the relationship. Health, education and income are control variables in *a* and *b*.

(a) Height and reproductive success in men

For men, there was a curvilinear effect of height on reproductive success, with taller than average men producing both a larger number of ever born children and more surviving children (table 1 and figure 1*a*). There was no effect of height on child mortality nor on the proportion of surviving offspring (table 1). Controlling for education, income and health slightly attenuated the effect of height on reproductive success, suggesting that the relationship was partially mediated by these factors (table 1, figure 1*a*). Shorter men were also likely to have lower fertility because of a reduced likelihood of currently being in a relationship and ever having a partner (table 1 and figure 1*c*); controlling for these mate choice variables in both cases decreased the effect size of height on fertility, suggesting partial mediation (electronic supplementary material, table S7). Taller men were also more likely to have a second child compared with shorter men (table 1), however, which indicates that relationship status was not the sole cause of higher fertility among taller men. Taller men started their current relationships at a later age than both average height and shorter men (the latter starting their relationships at a later age than average height men; figure 1*e*), which might account for the levelling-off of fertility for very tall men. This is countered, however, by the positive relationship between height and the number of children produced within a man's current relationship (figure 1*a*). Indeed, very tall men were likely to do better reproductively than men of only above average height within their current relationship. It is also notable that taller men achieve their higher reproductive success despite a later age at first birth (figure 1*e*).

Overall then, taller than average men in our sample experienced higher fertility, partly because they were more likely to be in a (current) relationship, and also because they were able to produce more offspring within those relationships than shorter men, despite beginning their reproductive careers later. The levelling-off of the relationship at the very tallest heights may be owing to entering into a reproductive partnership at an older age, which then influences age at first birth.

(b) Height and reproductive success in women

For women, the effect of height on reproductive success was quadratic, with women of average height giving birth to more children and producing more surviving children (table 1 and figure 1*b*). Controlling for education, income and health attenuated this relationship only very slightly (figure 1*b*), suggesting these factors have a lesser role to play in mediating this relationship compared with men. Again, in contrast to men, height also had a significant curvilinear effect on the probability of losing a child and also on the proportion of children surviving (table 1). Shorter women were more likely to experience the death of a child, and hence taller women had a higher proportion of surviving children.

Average height women were more likely to have a partner than women of shorter or taller heights, which may partly explain their higher fertility (table 1 and figure 1*d*). Indeed, as with our male sample, controlling for partnership status attenuated the effect of height on fertility, indicating partial mediation (electronic supplementary material, table S11). Also similar to men, height actually had a positive effect on fertility within a current partnership (figure 1*b*), with taller women producing more offspring than average height or shorter women. This increase in female reproductive output was achieved despite a later age at first birth, and a longer interval between the onset of the current relationship and starting a family (table 1 and figure 1*f*). Taller women also had a later age at last birth, which may have compensated, at least partly, for this late start (table 1 and figure 1*f*).

For women, it was also possible to assess age at sexual maturity as well as menopausal age, both of which were positively related to height (table 1). There was, however, no overall effect of height on the overall length of women's reproductive lifespan (i.e. the period between the onset of menstruation and its termination at menopause). Controlling for the age of menses did not affect the relationship between height and fertility in any meaningful way (electronic supplementary material, table S13).

In summary, then, average height women experienced higher fertility than shorter or taller women, and this effect was partly mediated by their greater likelihood of currently being in a relationship. Within an established relationship, however, taller women experienced higher reproductive success, and did so despite a longer interval between relationship formation and starting a family, and a later age at first birth. Taller women also experienced a later age at sexual maturity, which may have contributed to this later age at first birth.

4. Discussion

Our results suggest that average height women have higher fertility, compared with both shorter and taller women, and that taller men have higher fertility compared with shorter men. It therefore seems plausible to suggest that natural selection may have acted on the Dutch population, and helped drive the Dutch toward taller heights. Examining the effect sizes in table 1 (see also figure 1), however, two things are apparent: first, effect sizes are very low. Owing to our large sample, we were able to identify highly significant, but very small effects. Second, for our more direct measures of fertility (e.g. childlessness, surviving children), the effects of height are stronger for men than for women. With respect to the number of surviving children, for example, the difference between the most successful man (as predicted by the statistical model, and within the −2 and 2 s.d. of the height continuum) and the least successful man was approximately 0.24 of a child (i.e. 11.2% difference in magnitude). For women, this difference was only 0.09 of a child (3.8%). Phenotypic selection in this sample is therefore stronger on men than on women.

Although our findings are suggestive of selection on height, and are in line with genetic evidence regarding past selection on stature in Northern Europe [28], we do not present direct evidence for natural selection. The phenotypic correlation we observe can be caused by both environmental and/or genetic factors, and we can infer the degree of natural selection only to the extent to which genetic effects on both traits correlate [29]. Nevertheless, our current knowledge of the genetic architecture of both human height and fertility support an evolutionary interpretation. The largest part of the variation in human height (approx. 80%) in Western populations is due to genetic differences between individuals, as shown using both family designs (e.g. [30–32]), as well as molecular genetic information on genetic variation within families [33]. Genetic variation in common single nucleotide polymorphisms measured in unrelated individuals also supports this interpretation [34]. Studies by both biologists and demographers also show a moderate genetic component to fertility traits, such as number of children ever born and the age at first birth, which explains up to 40–50% of the variance in these traits [11,35–38]. Such findings were also observed for a historical Dutch population [39]. Moreover, studies have observed a genetic correlation between female height and age at first birth [14,40].

At present, then, we only have a phenotypic relationship, but this finding is interesting to consider: why should average height women and taller men be reproductively more successful in this population? Taking women first, one reason why average height women do better is that shorter women in our sample showed higher rates of child mortality, despite this being a low-mortality population overall (findings very much in line with non-Western populations: [41–43], and a previous study on a Western population [12]). Thus, although average height women experienced a later age at first birth than shorter women, this may be offset by the small reproductive disadvantage suffered by shorter women. At the other end of the height spectrum, and similar to men, taller women actually had higher fertility with their current partner, but tended to form partnerships later in life compared with shorter women, and taller women were also less likely to be partnered overall. Given that both taller men and women started their current relationships at a later age, our findings are also consistent with studies demonstrating assortative mating for height (e.g. [44]). Although taller women show lower fertility compared with average height women, this does not seem to reflect differences in fecundity per se, but more likely relates to other decision-making processes (such as the decision to continue into higher education) that postpone fertility [20,45,46].

The higher reproductive success of tall women within established partnerships, combined with findings on age at menopause and menses fit well, at least superficially, with life-history theory and the trade-off between growth and reproduction [14,47,48]. Taller women invest more in growth, reach menses later and have a later age at first birth, possibly because they reach peak fertility later than women who experience early menarche. As already noted, a later age at first birth among taller women also seems to reflect a lower likelihood of being in a relationship. Once such women establish a relationship and begin reproducing, however, they appear to compensate for their late start, and are more reproductively successful than shorter women. This is partly because they continue reproducing to a later age. This suggests that taller women, having invested in early growth, are able to reap the benefits of greater productivity and higher fecundity in later life, as life-history theory suggests [48]. Tellingly, however, these benefits only become apparent once a partnership is formed. Thus, although typical life-history trade-offs may be present, these are heavily contingent on the likelihood and timing of pair formation, and the advent of reproduction. This probably explains why average height women, who are both more likely to be partnered and less likely to suffer the child mortality disadvantage of shorter women, display the highest overall reproductive success.

It is possible that similar life-history trade-offs may affect men's fertility, but these are likely to be less stringent than in females, as the costs of reproduction are lower for men, and life-history processes may therefore operate differently between the sexes. It is also the case that selection pressures on height can differ between the sexes, where sexual selection via female choice exerts more of an influence on male height than does male choice of female partners (for further discussion of these points, see [3]). This is not to say that height does not influence female mating success, only that the effects are known to be stronger in males (see [17,49] and [3,16] for reviews). Indeed, there is a striking similarity between the selection pressures identified in this study, and relationships found between height and attractiveness in both laboratory and field settings [3,16,17], where taller men and average height women are, on average, preferred by the other sex. Thus, direct preferences for taller height among men, and a combination of preference for average height and life-history trade-offs among women may account for the patterns seen here.

It is important to remember, though, that the variables used here (e.g. timing of reproductive partnership formation) are not very accurate measures of attractiveness or mate choice. Indeed, the finding that taller men establish partnerships at a later age and have a later age at first birth, suggests that treating such measures as proxies for attractiveness may be flawed. This is complicated by the fact that we could only assess reproductive timing and success within current relationships; men may have experienced previous (reproductive) relationships, prior to their current relationship. In addition, factors unrelated to partnership formation or attractiveness undoubtedly underpin many of the patterns we observe here. Height may therefore act as an index for differing life goals and choices (for example, taller individuals often receive more education and higher incomes [22,23], which may influence both partnership and family formation decisions in distinctive ways [20]).

Another question our study raises is why the Dutch pattern differs from that of the United States where shorter women and average height men have higher fertility [11,12]. Although we can only speculate, one plausible reason may be that, because shorter height is associated with a much earlier age at marriage and first birth in the United States, the increased length of the reproductive period contributes to higher reproductive success, despite shorter women suffering higher rates of child mortality. In turn, the higher reproductive success of shorter women may explain why average height men, who are more likely to be paired with shorter women [44], have higher fertility in the United States [50]. Given that, in The Netherlands, within a current partnership, average height women and taller women display higher fertility than shorter women, it would follow that men partnered to such women, who are themselves likely to be taller than average, will reap reproductive benefits from their spouses. Future studies can test this hypothesis by examining the simultaneous effects of spousal heights on a couples' fertility. Of course, there may be many other ecological differences between the United States and The Netherlands that may favour taller or shorter stature (see [3] for further discussion), and there is no reason to expect that selection pressures will be consistent across populations, or across time [51]. Furthermore, biases in sampling or response rates, and accuracy of reporting, also hinder accurate comparison between the two populations (see also [15]).

Finally, it is important to emphasize again that our effect sizes are very small, and there are many other factors that explain why people have children [46]. Indeed, it is remarkable that height shows any influence at all, given the vagaries of the mate choice and partnering processes. What this study also makes clear, however, is the complexity of the relationship between height and reproductive success, even if we ignore these other factors. Our study thus provides an excellent illustration of why, even for a seemingly straightforward 'biological' trait like height, sociocultural factors that influence the likelihood and timing of reproduction are influential, in addition to physiological and life-historical considerations. Despite this complexity, our results nevertheless suggest the possibility that natural selection acts on height in The Netherlands, and may partly explain why the Dutch, apparently not yet tall enough, keep growing taller.

Ethics statement. The procedures have been put in place by the LifeLines Scientific Board and local ethic committees.

Data accessibility. The LifeLines data is available by contacting the LifeLines Research Office (https://www.lifelines.nl/lifelines-research/access-to-lifelines/application-process). The data cannot be released without assessment by the LifeLines Scientific Committee with transfer agreements since the phenotypic data can be sensitive and has the potential in some cases to lead to the identification of individuals involved in the study.

Acknowledgements. The authors wish to acknowledge the services of the LifeLines Cohort Study, the contributing research centres delivering data to LifeLines and all the study participants. We thank the editor and reviewers for their helpful and constructive comments on a previous draft of the manuscript.

Funding statement. The LifeLines Cohort Study and generation and management of GWAS genotype data for the LifeLines Cohort Study is supported by The Netherlands Organization of Scientific Research NWO (grant no. 175.010.2007.006), the Economic Structure Enhancing Fund (FES) of the Dutch government, the Ministry of Economic Affairs, the Ministry of Education, Culture and Science, the Ministry for Health, Welfare and Sports, the Northern Netherlands Collaboration of Provinces (SNN), the Province of Groningen, University Medical Center Groningen, the University of Groningen, Dutch Kidney Foundation and Dutch Diabetes Research Foundation. G.S. is supported by an NWO Rubicon fellowship, and L.B. is supported by the NSERC Discovery Grant and Canada Research Chair Programs. M.M. and F.T. are supported by a grant from the NWO (VIDI grant no. 452-10-012 to M.M.) and the European Research Council via an ERC Consolidator Grant SOCIOGENOME (615603 awarded to M.M.).

References

1. De Beer H. 2004 Observations on the history of Dutch physical stature from the late-Middle Ages to the present. *Econ. Hum. Biol.* **2**, 45–55. (doi:10.1016/j.ehb.2003.11.001)
2. Komlos J, Baur M. 2004 From the tallest to (one of) the fattest: the enigmatic fate of the American population in the 20th century. *Econ. Hum. Biol.* **2**, 57–74. (doi:10.1016/j.ehb.2003.12.006)
3. Stulp G, Barrett L. In press. Evolutionary perspectives on human height variation. *Biol. Rev.* (doi:10.1111/brv.12165)
4. Schönbeck Y, Talma H, van Dommelen P, Bakker B, Buitendijk SE, HiraSing RA, van Buuren S. 2013 The world's tallest nation has stopped growing taller: the height of Dutch children from 1955 to 2009. *Pediatr. Res.* **73**, 371–377. (doi:10.1038/pr.2012.189)
5. Garcia J, Quintana-Domeque C. 2007 The evolution of adult height in Europe: a brief note. *Econ. Hum. Biol.* **5**, 340–349. (doi:10.1016/j.ehb.2007.02.002)
6. Fredriks AM, van Buuren S, Burgmeijer RJF, Meulmeester JF, Beuker RJ, Brugman E, Roede MJ, Verloove-Vanhorick SP, Wit J-M. 2000 Continuing positive secular growth change in the Netherlands 1955–1997. *Pediatr. Res.* **47**, 316–323. (doi:10.1203/00006450-200003000-00006)
7. Danubio ME, Sanna E. 2008 Secular changes in human biological variables in Western countries: an updated review and synthesis. *J. Anthropol. Sci.* **86**, 91–112.
8. De Beer H. 2012 Dairy products and physical stature: a systematic review and meta-analysis of controlled trials. *Econ. Hum. Biol.* **10**, 299–309. (doi:10.1016/j.ehb.2011.08.003)
9. Steckel RH. 1995 Stature and the standard of living. *J. Econ. Lit.* **33**, 1903–1940.
10. Steckel RH. 2002 Health, nutrition and physical well-being. In *Historical statistics of the United States: millennial edition* (eds SB Carter, SS Gartner, MR Haines, AL Olmstead, R Sutch, G Wright), Bd. New York: Cambridge University Press.
11. Byars SG, Ewbank D, Govindaraju DR, Stearns SC. 2010 Natural selection in a contemporary human population. *Proc. Natl Acad. Sci. USA* **107**, 1787–1792. (doi:10.1073/pnas.0906199106)
12. Stulp G, Verhulst S, Pollet TV, Buunk AP. 2012 The effect of female height on reproductive success is negative in Western populations, but more variable in non-Western populations. *Am. J. Hum. Biol.* **24**, 486–494. (doi:10.1002/ajhb.22252)
13. Stulp G, Pollet TV, Verhulst S, Buunk AP. 2012 A curvilinear effect of height on reproductive success in human males. *Behav. Ecol. Sociobiol.* **66**, 375–384. (doi:10.1007/s00265-011-1283-2)
14. Stearns SC, Govindaraju DR, Ewbank D, Byars SG. 2012 Constraints on the coevolution of contemporary human males and females. *Proc. R. Soc. B* **279**, 4836–4844. (doi:10.1098/rspb.2012.2024)
15. Stulp G, Kuijper B, Buunk AP, Pollet TV, Verhulst S. 2012 Intralocus sexual conflict over human height. *Biol. Lett.* **8**, 976–978. (doi:10.1098/rsbl.2012.0590)
16. Courtiol A, Raymond M, Godelle B, Ferdy J-B. 2010 Mate choice and human stature: homogamy as a unified framework for understanding mating preferences. *Evolution* **64**, 2189–2203. (doi:10.1111/j.1558-5646.2010.00985.x)
17. Stulp G, Buunk AP, Kurzban R, Verhulst S. 2013 The height of choosiness: mutual mate choice for stature results in suboptimal pair formation for both sexes. *Anim. Behav.* **86**, 37–46. (doi:10.1016/j.anbehav.2013.03.038)
18. Stolk RP, Rosmalen JG, Postma DS, de Boer RA, Navis G, Slaets JP, Ormel J, Wolffenbuttel BH. 2008 Universal risk factors for multifactorial diseases: LifeLines: a three-generation population-based study. *Eur. J. Epidemiol.* **23**, 67–74. (doi:10.1007/s10654-007-9204-4)
19. Visscher PM, Hemani G, Vinkhuyzen AAE, Chen G-B, Lee SH, Wray NR, Goddard ME, Yang J. 2014 Statistical power to detect genetic (co)variance of complex traits using SNP data in unrelated samples. *PLoS Genet.* **10**, e1004269. (doi:10.1371/journal.pgen.1004269)
20. Mills M, Rindfuss RR, McDonald P, te Velde E, Force, on behalf of the E. R. and S. T. 2011 Why do people postpone parenthood? Reasons and social policy incentives. *Hum. Reprod. Update* **17**, 848–860. (doi:10.1093/humupd/dmr026)
21. Silventoinen K, Lahelma E, Rahkonen O. 1999 Social background, adult body-height and health. *Int. J. Epidemiol.* **28**, 911–918. (doi:10.1093/ije/28.5.911)
22. Judge TA, Cable DM. 2004 The effect of physical height on workplace success and income: preliminary test of a theoretical model. *J. Appl. Psychol.* **89**, 428–441. (doi:10.1037/0021-9010.89.3.428)
23. Silventoinen K, Krueger RF, Bouchard TJ, Kaprio J, McGue M. 2004 Heritability of body height and educational attainment in an international context: comparison of adult twins in Minnesota and Finland. *Am. J. Hum. Biol.* **16**, 544–555. (doi:10.1002/ajhb.20060)
24. Persico N, Postlewaite A, Silverman D. 2004 The effect of adolescent experience on labor market outcomes: the case of height. National Bureau of Economic Research Working Paper Series No. 10522.
25. Stulp G, Buunk AP, Verhulst S, Pollet TV. 2013 Tall claims? Sense and nonsense about the importance of height of US presidents. *Lead. Q.* **24**, 159–171. (doi:10.1016/j.leaqua.2012.09.002)
26. Prag P, Mills M, Wittek R. 2013 Income and income inequality as social determinants of health: do social comparisons play a role? *Eur. Sociol. Rev.* **30**, 218–229. (doi:10.1093/esr/jct035)
27. R Development Core Team. 2008 *R: a language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.
28. Turchin MC, Chiang CW, Palmer CD, Sankararaman S, Reich D, Hirschhorn JN. 2012 Evidence of widespread selection on standing variation in Europe at height-associated SNPs. *Nat. Genet.* **44**, 1015–1019. (doi:10.1038/ng.2368)
29. Morrissey MB, Kruuk LEB, Wilson AJ. 2010 The danger of applying the breeder's equation in observational studies of natural populations. *J. Evol. Biol.* **23**, 2277–2288. (doi:10.1111/j.1420-9101.2010.02084.x)
30. Silventoinen K. 2003 Determinants of variation in adult body height. *J. Biosoc. Sci.* **35**, 263–285. (doi:10.1017/S0021932003002633)
31. McEvoy BP, Visscher PM. 2009 Genetics of human height. *Econ. Hum. Biol.* **7**, 294–306. (doi:10.1016/j.ehb.2009.09.005)
32. Visscher PM, McEvoy B, Yang J. 2010 From Galton to GWAS: quantitative genetics of human height. *Genet. Res.* (*Camb*). **92**, 371–379. (doi:10.1017/S0016672310000571)
33. Visscher PM, Medland SE, Ferreira MAR, Morley KI, Zhu G, Cornes BK, Montgomery GW, Martin NG. 2006 Assumption-free estimation of heritability from genome-wide identity-by-descent sharing between full siblings. *PLoS Genet.* **2**, e41. (doi:10.1371/journal.pgen.0020041)
34. Yang J *et al*. 2010 Common SNPs explain a large proportion of the heritability for human height. *Nat. Genet.* **42**, 565–569. (doi:10.1038/ng.608)
35. Kohler H-P, Rodgers JL, Christensen K. 1999 Is fertility behavior in our genes? Findings from a Danish twin study. *Popul. Dev. Rev.* **25**, 253–288. (doi:10.1111/j.1728-4457.1999.00253.x)
36. Kohler H, Rodgers JL, Christensen K. 2002 Between nurture and nature: the shifting determinants of female fertility in Danish twin cohorts. *Biodemogr. Soc. Biol.* **49**, 218–248. (doi:10.1080/19485565.2002.9989060)
37. Milot E, Mayer FM, Nussey DH, Boisvert M, Pelletier F, Réale D. 2011 Evidence for evolution in response to natural selection in a contemporary human population. *Proc. Natl Acad. Sci. USA* **108**, 17 040–17 045. (doi:10.1073/pnas.1104210108)

38. Kohler H-P, Rodgers JL, Miller WB, Skytthe A, Christensen K. 2006 Bio-social determinants of fertility. *Int. J. Androl.* **29**, 46–53. (doi:10.1111/j.1365-2605.2005.00606.x)
39. Bras H, Van Bavel J, Mandemakers K. 2013 Unraveling the intergenerational transmission of fertility: genetic and shared-environment effects during the demographic transition in the Netherlands, 1810–1910. *Hist. Fam.* **18**, 116–134. (doi:10.1080/1081602X.2013.803491)
40. Silventoinen K, Helle S, Nisén J, Martikainen P, Kaprio J. 2013 Height, age at first birth, and lifetime reproductive success: a prospective cohort study of Finnish male and female twins. *Twin Res. Hum. Genet.* **16**, 581–589. (doi:10.1017/thg.2012.150)
41. Monden CWS, Smits J. 2009 Maternal height and child mortality in 42 developing countries. *Am. J. Hum. Biol.* **21**, 305–311. (doi:10.1002/ajhb.20860)
42. Özaltin E, Hill K, Subramanian SV. 2010 Association of maternal stature with offspring mortality, underweight, and stunting in low- to middle-income countries. *J. Am. Med. Assoc.* **303**, 1507–1516. (doi:10.1001/jama.2010.450)
43. Lawson DW, Alvergne A, Gibson MA. 2012 The life-history trade-off between fertility and child survival. *Proc. R. Soc. B* **279**, 4755–4764. (doi:10.1098/rspb.2012.1635)
44. Stulp G, Buunk AP, Pollet TV, Nettle D, Verhulst S. 2013 Are human mating preferences with respect to height reflected in actual pairings? *PLoS ONE* **8**, e54186. (doi:10.1371/journal.pone.0054186)
45. Deady DK, Law Smith MJ. 2006 Height in women predicts maternal tendencies and career orientation. *Pers. Individ. Dif.* **40**, 17–25. (doi:10.1016/j.paid.2005.06.014)
46. Balbo N, Billari FC, Mills M. 2013 Fertility in advanced societies: a review of research. *Eur. J. Popul.* **29**, 1–38. (doi:10.1007/s10680-012-9277-y)
47. Stulp G, Barrett L. In press. Fertility theory: theory of life history evolution. In *The international encyclopedia of the social and behavioral sciences*. London, UK: Elsevier Ltd.
48. Stearns SC. 1992 *The evolution of life histories*. Oxford, UK: Oxford University Press.
49. Courtiol A, Picq S, Godelle B, Raymond M, Ferdy J-B. 2010 From preferred to actual mate characteristics: the case of human body shape. *PLoS ONE* **5**, e13010. (doi:10.1371/journal.pone.0013010)
50. Stulp G, Mills M, Pollet TV, Barrett L. 2014 Non-linear associations between stature and mate choice characteristics for American men and their spouses. *Am. J. Hum. Biol.* **26**, 530–537. (doi:10.1002/ajhb.22559)
51. Siepielski AM, DiBattista JD, Carlson SM. 2009 It's about time: the temporal dynamics of phenotypic selection in the wild. *Ecol. Lett.* **12**, 1261–1276. (doi:10.1111/j.1461-0248.2009.01381.x)