Height and the disease environment, the impact of mortality on height in the Netherlands 1850-1940

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Adult height and infant mortality are both considered to be good indicators of health and living standards of a population (Steckel, 2009). Over the past century stature and mortality rates developed hand in hand since both indicators are intertwined. On the one hand, childhood diseases are accepted as a part of the equation that determines adult height: energy from nutrition minus energy loss trough labour, stress and diseases (Floud et al., 2011). On the other hand, improved nutrition might build resistance against infectious diseases (McKeown, 1976). This poses a problem to study the direct effect of the disease environment (regime of all diseases that prevail in a population) on height, as they are both influenced by confounding factors, e.g. rising real wages. The aim of this paper is to study the influence of the disease environment between 1850 and 1940 in the Netherlands on height to contribute to the understanding of the mechanism that is at play.

The disease environment can be studied in two ways. First the severity of the disease environment can be measured through mortality rates. Often infant mortality rates (IMR) are used (Bozzoli, Deaton, & Quintana-Domeque, 2009; Hatton, 2011; Öberg, 2015; Spijker, Cámara, & Blanes, 2012). These provide the most sensitive proxy of the disease environment as the youngest of a population are also the most vulnerable. Furthermore, as Hatton (2013) argues, the disease environment that kills infants, might make older children chronically ill, diverting energy from growth to fighting diseases. Second different diseases might result in greater energy loss. Unfortunately the occurrence of diseases is rarely systematically documented in historical populations. Although not all diseases that impact height also result in early death, studying causes of death might prove to be our best proxy for the composition of the disease environment.

The most pronounced relation between height in mortality rates is found for West- and South-European countries in the second half of the twentieth century (Schmidt, Jørgensen, & Michaelsen, 1995). Yet, the relation was also found in countries that generally have a lower income (Coffey, 2015). Still no relation was found for Brazil (De Oliveira & Quintana-Domeque, 2014), and also in Sub-Saharan Africa a reduction of IMR did not result in increased stature (Akachi & Canning, 2010). Furthermore, for historical populations, Öberg (2015) found a significant but very weak relation and Baten & Blum (2000) found that economic factors predict height better than mortality rates.

Such discrepancies in results might help us to unravel the underlying mechanisms. So far, various explanations have been provided. Bozzoli et al. (2009) attribute the changing relationship between disease and height to the prevalence of scarring (stunting) over selection (of the strongest and tallest) below an IMR of 100, whereas above this threshold selection might cloud or dominate the effect. This would explain the puzzling situation in Sub-Saharan countries where mortality rates are declining, but this is not reflected in rising stature. Akachi and Canning (2010) provide an alternative explanation in that this might be related to interventions that prevent infant death, but that nothing has changed regarding the nutritional situation or with regard to childhood morbidity.

All of the afore mentioned studies test the impact of IMR on height in the critical first year of life (Barker & Osmond, 1986) as living conditions at this stage have a strong impact on terminal adult height (Cole 2003). However, Hatton (2011) found no effect in the first year of

life for British schoolchildren in the first half of the twentieth century. Yet an accumulation of the disease environment, measured between the ages of two and six resulted in a shorter height. Recently Depauw and Oxley (2018) moved this accumulation effect to span the years in adolescence during which the pubertal growth spurt occurs and found an effect of IMR on heights of Belgium prisoners in the nineteenth century. Already, similar results were found for the nineteenth century Netherlands (de Beer 2001).

Another complicating factor using IMR, is that it is an average for a population. Recent research has shown that infant mortality is clustered in certain high risk families (Van Dijk, 2019) that is not directly captured by other factors such as socioeconomic status. This does not directly imply that morbidity, and therefore the effect of the disease environment on height is also clustered. To gain a better understanding it is necessary to move beyond the aggregated death statistics and examine the mechanism at a household level.

Data

The heights data stem from relatively unbiased conscription records (Quanjer & Kok 2020) that were linked to the Historical Sample of the Netherlands database (HSN) (Mandemakers, 2000). This allows for a complete life course reconstruction of all research persons including the a household reconstruction and therefore also sibling mortality. Furthermore, by the creation of the Male Kin Heights (MKH) database (Mandemakers, 2018), heights of brothers were added allowing for a multilevel analyses with observations nested in one family. The MKH is biased towards larger families and to the provinces of Noord-Holland and Friesland that allowed for the digital linkage of conscripted brothers. The other limitation is that by using conscription records, the analyses is limited to men only.

The disease environment is defined by a crude death rate (CDR) per year per municipality that was calculated using the Historical Database of Dutch Municipalities (Boonstra 2016). The CDR captures the yearly fluctuations, whereas the IMR on the municipal level are only available for different multiyear periods (Ekamper & Van Poppel, 2008). Yet, both rates show a correlation of 0.85 and the IMR will also be used to test the robustness of the models.

Method

In line of the Akachi & Canning (2010) hypothesis, the disease environment only impacts heights in a state of long endured misery. On the other hand short mortality peaks, killing the weakest and scarring the healthiest, would exercise less or no effect on height. This will be tested, taking into account the limited information available for this historical period, at the municipal and household level. First the overall disease environment is established to proxy the chronic disease load and living conditions. At the municipal level this is done by the average CDR in early childhood between the time *in utero* and age 4 and during adolescence between ages 14 and 19. At the household level the overall child mortality rate (CMR) is calculated which is the share of brothers and sisters that do not reach the age of 15. Second mortality peaks are defined at the municipal level as a yearly CDR peak that is one standard deviation higher than the five year moving average for this municipality. These peaks are tested at the same age ranges as described above. Within the household the occurrence of a sibling death is used to define a mortality peak.

To test the effect mortality has on height OLS regression models will be used. Furthermore multilevel models with observations nested in families and decades will be employed. These models will be controlled for socioeconomic background and birth region as was done in Quanjer & Kok (2019). Also birth decade will be used as a control variable.

Results

The main findings of the effect of disease environment at the municipal level on height are presented in table 1. A significant negative effect of CDR on height was found both in the very first years of life as during the late teenage years. If we translate these effect sizes to the reduction of average CDR from 25 to 10 per 1000, this would imply a height gain between 1,4 and 2,3 centimetre for the period under observation. The later timed CDR seems to have a stronger effect on heights. Living through a municipal mortality peak also reduced heights although the effect was small and not significant.

Table 1. The effect of the municipal disease environment on height.

	HSN OLS models				*Family multilevel models*			
CDR early life	-0.79 **	-	-	-	-1.12 ***	-	-	-
CDR adolescence	-	-1.11 **	-	-	-	-1.28 ***	-	-
No mortality peak	-	-	*Ref.*	*Ref.*	-	-	*Ref.*	*Ref.*
Mortality peak early life	-	-	-1.30	-	-	-	-0.48	-
Mortality peak adolescence	-	-	-	-1.33	-	-	-	-2.63

Significance codes: 0 *** 0.001 ** 0.01 * 0.05. All models are controlled for SES, birth region and birth cohort/year.

The findings of the effect of disease environment at the household level on height are presented in table 2. The CMR has a strong effect on height of more than 2,5 centimetre, yet this effect disappears in the family multilevel model. Those research persons who experienced a sibling death in the first years of life are significantly scarred with regard to their height. This is not the case for those who experience a sibling death in adolescence.

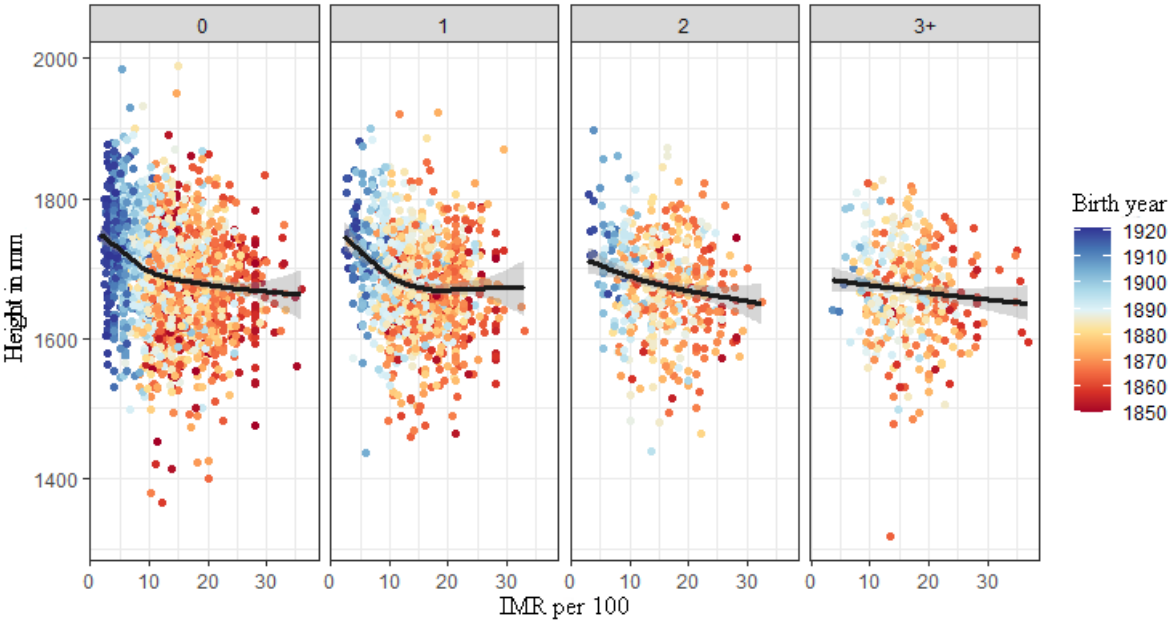
Table 2. The effect of the household disease environment on height.

	HSN OLS models			*Family multilevel models*		
CMR household	-26.95 ***	-	-	-9.39	-	-
No sibling death occurred	-	*Ref.*	*Ref.*	-	*Ref.*	*Ref.*
A sibling death occurred between age -1 and 5	-	-7.33 **	-	-	-5.17 *	-
A sibling death occurred between age 14 and 19	-	-	-5.11	-	-	0.23

Significance codes: 0 *** 0.001 ** 0.01 * 0.05. All models are controlled for SES, birth region and birth cohort/year.

The disease environment does not split itself along the lines of a household doorstep. Family members interact with the outside world and bring home diseases. Figure 1 tries to capture this interaction between the household and municipal disease environment. To allow for a comparison with Bozzoli et al. (2009) IMR was used. The figure shows a similar threshold as was mentioned in the article for the ‘healthier’ households. The relation in the ‘unhealthy’ households seems to be more linear, although the interaction was not significant in a regression model.

Figure 1. IMR per 100 in adolescence relation with height for HSN RPs per birth year displayed per number of diseased siblings.



Discussion & Conclusion

This paper has shown that especially a constantly bad disease environment has an impact on height. Short mortality peaks have little influence although at the household level, experiencing a sibling death at a very young age is likely to reduce conscription height. Still this leaves open the first question raised about the intertwined relation of height and mortality. Might other factors such as wealth influence these results. To test the robustness of the results, GNP levels were used as a control. However, since this is a blunt instrument, aggregated at the national level, municipal tax revenues per capita, available for the period 1859-1889, were also employed to proxy wealth at a local level. To account for economies of scale in large population centres, this variable was controlled for the fact if a municipality was an urban centre. None of these controls changed the outcomes of the models in a significant way, which advocates for the robustness of the mortality effect on height. Furthermore switching between CDR and IMR resulted in similar outcomes as the IMR dominated the CDR figures during this era.

What also stands out is the reduction of the CMR effect in the household using the multilevel model. This is not so much related to the biases described in the data section, but can be attributed to age differences between brothers. If the multilevel analysis was limited to brothers that were born no more than five years apart, the effect was -24.84, similar to that in table 2. The effect reduction in table 2 is therefore explained by brothers born far apart in time, causing the family CMR not to reflect the disease environment of at least one of them.

Finally it needs to be noted that heights of conscripted men are not the same as terminal adult heights during this period of research. Bad living conditions delay growth which can be made up for after the age of twenty (Thompson et al. 2020). As a result, the effect sizes found might also be an overestimation of the true effect on adult height, as a CDR at age 15 may only stunt growth in the short run. The findings of Depauw and Oxley (2018) might be reassuring, as they also found an effect of IMR in adolescence for full grown prisoners.

Returning to the frameworks introduced in the introduction. Figure 1 shows a similar 100 per 1000 IMR threshold as Bozzoli et al. (2009) which cannot be attributed to confounding

factors such as wealth as this was controlled for in a robustness check. Most likely medical intervention might bring down IMR to certain extend, but only a structural epidemiological transition is capable of bringing the IMR beyond the threshold, which brings us to the Akachi framework. At the municipal level only the long term disease environment had an impact on height, but on the household level, sibling death in early childhood did result in a shorter stature. Therefore a historical study into medical interventions with regard to infant mortality is needed to unravel the pathway to better living standards for everyone.

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