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Abstract: We identify the ages that constitute critical periods in children's development towards their adult health status. For this we use data on families migrating into Sweden from countries that are poorer, with less healthy conditions. Long-run health is proxied by adult height. The relation between siblings' ages at migration and their heights after age 18 allows us to estimate the causal effect of conditions at certain ages on adult height. Moreover, we compare siblings born outside and within Sweden. We apply fixed-effect methods to a sample of about 9,000 brothers. We effectively exploit that for siblings the migration occurs simultaneously in calendar time but at different developmental stages (ages). We find some evidence for a critical period at age 9. The effects are stronger in families migrating from poorer countries but weaker if the mother is well-educated.

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1. Introduction

Recently, there has been an accumulation of evidence that conditions early in life influence health at higher ages. Children born under adverse economic and nutritional conditions or with a high disease exposure in the birth year have higher morbidity and mortality rates later in life.[1] Most of the evidence concerns conditions in utero or shortly after birth. Much less is known about conditions at other childhood ages. It is conceivable that the age interval from birth to adulthood contains so-called critical periods during which sub-optimal conditions have particularly adverse long-run implications for health later in life.[2] One reason for the focus on conditions around birth is that the physical development is strongest in the first birth year, so that the physical state later in childhood is partly determined by conditions earlier in life. Accordingly, conditions later in childhood may be confounded by conditions around birth.

More in general, the empirical analysis of long-run effects of early-life conditions is hampered by two constraints. First, the use of observations of health or mortality at high ages entails that only cohorts born a long time ago can be studied. Secondly, early-life conditions need to be exogenous, or otherwise one needs to deal with their endogeneity. The first constraint can be dealt with by using adult height as a proxy for health outcomes later in life insofar as the latter are affected by conditions before adulthood (Steckel, 1995, 2008, Silventoinen et al., 2006). An adult individual's height has been denoted "probably the best single indicator of his or her dietary and infectious disease history during childhood" (Elo and Preston, 1992). Adult height has therefore been widely utilized as a marker of secular trends and socioeconomic variations in childhood conditions (Silventoinen 2003; see also the literature discussion in Section 2).[3]

In this study, we explore the shift in living conditions for children who migrated to Sweden, a relatively wealthy nation in which people's stature and longevity are among the highest, and poverty rates among the lowest, in the world. Migration is a discrete event, potentially causing a permanent shift in the individual's living standards. We analyze the association between age at migration and subsequent adult height. To deal with the endogeneity of migration (which is the second of the two constraints mentioned above), we

[1] See the literature overview in Section 2.
[2] In the recent literature, the term "critical period" is sometimes replaced by "sensitive period".
[3] On an individual basis, height has also been found to be associated with a range of cognitive and economic outcomes later in adulthood, such as cognitive ability, education, earnings and social position (Marmot, 1995,

compare siblings in a family-fixed-effects framework. Family migration occurs for brothers usually at the same point in time but at different developmental stages, yielding the opportunity to consider critical periods during childhood for the development of adult stature. Comparing the adult height of brothers born outside and within Sweden allows us to also consider the role of early life conditions at the pre-natal stage. The fixed-effects approach effectively deals with the endogeneity of background characteristics. Brothers differ genetically, but their genes are sampled from the same ancestral "gene pool", implying that any genetic height variation is randomly distributed between them. Besides genetic factors, the fixed-effects approach also neutralizes additive height effects of other unobserved heterogeneity between families, like heterogeneity in terms of location, family structure, traditions, values norms, habits, wealth and household practices, all potentially being connected to age at migration and nutrition, disease load, and, ultimately, height. Consider, for example, the effect of conditions at ages 11 to 13. By comparing the adult height of two brothers who immigrated at ages 11 and 13, one can isolate the effect of exposure to Swedish living conditions at ages 11-13 (compared to conditions in the country of origin) on adult height and thereby on adult health. One might object that migration can be induced by a particularly bad health of the youngest brother, in which case the conditions for a fixed-effects approach based on all brothers would be invalid. We can deal with this by excluding the youngest brothers and perform the estimation with data from families with at least three sons.

The analysis is relevant from various points of view. The most general relevance concerns the identification of critical age intervals in human development. Knowledge of this has potentially important policy implications. If adverse conditions at a certain age before adulthood have particularly severe long-run effects on health (and thereby on economic outcomes like earnings) then the value of life is reduced for those affected, and this would increase the benefits of supportive policies for groups of individuals exposed to such conditions. Notice that the long-run effects of conditions during childhood on health in adulthood may be smaller than the instantaneous effects of current conditions, but the former exert their influence over a longer time span. Moreover, the presence of a time interval between childhood and the manifestation of the effect implies that there is a scope for identification and treatment of the individuals at risk. Specifically, young individuals exposed

Mackenbach, 1992, Meyer and Selmer, 1999, Abbott et al., 1998, Silventoinen et al., 1999, Rashad, 2008, Case and Paxson, 2008a, 2008b).

to adverse conditions at a critical age can be targeted for a screening of health markers and predictors, and those who have unfavorable test values are amenable to preventive intervention.

The analysis in this paper also has a more specific relevance concerning immigrant families. If they bring along children with ages just above a critical period, then such children will be at a higher risk of future health problems, and one may reallocate funding towards preventive health care for such children. Similarly, the analysis is relevant for adoption policies. Adopted children from poor countries with an adoption age above a critical period will also be at a higher risk of health problems, and therefore they may need special health care after adoption.

The data contain the full population of immigrants living in Sweden in the year 1999 who, in between 1984 and 1997, had been subjected to the mandatory enlistment test for military service. Specifically, we use merged registers from Statistics Sweden (including information on birth date, date of immigration, country of birth, and family structure, including birth order) and the Swedish National Service Administration ("Pliktverket" in Swedish). In principle, every male Swedish citizen enlists for the military when turning 18, and hence, we have a measure of their height/stature at this age.

Our data include the levels of education of the parents. Most likely, these capture economic well-being as well as health knowledge before migration. Interactions of the effect of age at migration with the region of birth and the parents' levels of education allow us to shed some light on whether effects of adverse conditions during critical periods can be offset by wealth or by health knowledge. The extent to which immigrants suffer from assimilation problems may depend on the interaction of cultural distance and age. By performing separate analyses by cultural distance we can, to some extent, separate such age-specific assimilation effects from the age effects due to critical periods. In all cases we control for birth-order effects.

The paper is organized as follows. In Section 2, we review the various bodies of literature that are connected to our study, notably the literature on long-run effects of early-life conditions and critical periods. Section 3 briefly describes the institutional context concerning military service and immigration in Sweden. In Section 4 we describe our data. Section 5 discusses the econometric methods we use and their underlying assumptions. We also provide a description in terms of treatment effects in a counterfactual framework. Section 6 presents our results. Section 7 concludes.

2. Related literature

2.1. Long-run effects of early-life conditions on health and mortality later in life

A number of bodies of work, from various disciplines, are relevant to our study. First, there is an expanding literature on the long-run effects of early-life conditions on late-life health outcomes. Secondly, there is a literature focusing on adult height as an outcome of events and conditions before adulthood. And third, there is a literature on the health of immigrants.

There are many surveys and meta-studies of the association between markers of early-life conditions (like birth weight) on the one hand, and health outcomes later in life on the other. For epidemiological and medical studies, see e.g. Poulter et al. (1999), Rasmussen (2001), and Huxley et al. (2007). Pollitt, Rose and Kaufman (2005) provide a survey and meta-study of the "life course" literature on causal pathways in which early-life socio-economic status (SES) is connected to morbidity and mortality later in life. Galobardes, Lynch and Davey Smith (2004) survey studies on early-life SES and cause-specific mortality in adulthood. See also Case, Fertig and Paxson (2005) and Case, Lubotsky and Paxson (2002), and references therein, for influential studies focusing on effects of economic household conditions early in life. Underlying explanations in this literature refer to nutrition, disease exposure, stress, and living conditions, as factors affecting the development of the child, and the extent to which these effects are exacerbated by schooling, career, family formation, and so on.

As noted in Section 1, almost all of this literature focuses exclusively on conditions at birth or shortly before birth as the starting point of a causal chain. Recently, interest has increased in long-run causal effects of conditions after birth. The survey in Eriksson (2007) focuses on medical early-life indicators measured after birth. Gluckman, Hanson and Pinal (2005) and Barker (2007) give overviews of the underlying medical mechanisms. We already pointed out that an association between conditions after birth and long-run outcomes can be confounded by conditions at birth and by unobserved individual or family characteristics. This poses a methodological challenge. In a study of the effects of conditions at ages 1-4 on over-all mortality later in life among those born in Denmark in 1873-1906, Van den Berg, Doblhammer and Christensen (2009) deal with this confounding issue by using the business cycle at ages 1-4 as exogenous idiosyncratic indicators of economic conditions at these ages.

They find that the cycle at age 3 has a significant effect, in that those who reach the age of 3 during a recession have a significantly higher mortality rate later in life. This suggests that age 3 constitutes a critical period. Van den Berg and Gupta (2008), using data on individuals born in the Netherlands in 1815-1902, find that the average business cycle at ages 7-12 affects the individual mortality later in life for men (but not for women). Notice that the cycle at ages beyond infancy may influence schooling decisions and subsequent occupational hazards and thus have indirect long-run health effects. The use of adult height as an outcome precludes such indirect long-run effects.

Famines also provide natural experiments of nutritional deficiencies. Sparén et al. (2004) find that boys around age 9 who had been exposed to starvation during the Leningrad siege of 1941 have higher rates of cardiovascular morbidity and mortality much later in life. Not all famine studies have found significant long-run effects. Obviously, extremely adverse conditions at early ages generate strong dynamic selection effects that go in the opposite direction of the causal effect on health. Another problem with famine studies is that one needs to be able to rule out other cohort-specific effects, including cohort-specific secular long-run causal effects on society and the health of its population.

2.2. Determinants of adult height

We now briefly summarize literature concerning height as an outcome. We first mention some stylized facts on "normal" height development. Under reasonably good nutritional conditions and limited disease load, human height growth is rapid during infancy, slows down monotonically during childhood, and reaches a minimum until the adolescent growth spurt starts. Werner and Bodin (2006; Table IIa) plot growth velocity rates (GVR, cm/year) for boys in Sweden in 1981. In the first year of life, GVR is 25.4. It decreases to about 5 at ages 10-12. Subsequently, it slightly increases to 7.5 at age 14, and it declines to zero at the age of 18. Overall, average birth height was 51 cm, while average final height (at age 19, in 1981) was 180.4 cm. Reviewing the literature, Silventoinen (2003) finds that about 20% of the variation in adult height, in post-war western civilizations, is due to environmental factors, while the rest is attributable to heritable factors.

Across populations, environmental factors appear to account for most of the differences in average height (Steckel, 1995). The marked increase in body height in the developed world during the twentieth century occurred too rapidly to be attributable to genetic variation (Beard and Blaser, 2002). Moreover, despite ethnic diversities of average

adult heights in the world, studies indicate that the genetic height *potential* is rather uniformly distributed, with children of different ethnical origin growing up under good circumstances on average becoming approximately equally tall (see Steckel, 1995). It has been suggested that socio-economic variation in height depends on the social position of the family during childhood, the latter being a determinant of both early-life nutrition and social status of the offspring. Children of wealthier families are less often subject to nutritional deficiencies, and higher educated parents, especially mothers, are more able (and inclined) to apply new information on child-caring techniques. Upward social mobility is positively associated with height, while cognitive ability and IQ in childhood and adulthood have been positively linked to height in childhood, suggesting that socio-economic variation in height may also reflect inherent cognitive capabilities (Case and Paxson, 2008a, 2008b).

In Section 1 we already mentioned that adult height has been widely used in the literature as a marker of childhood conditions, and we cited studies showing that adult height is correlated to cognitive, economic, and health outcomes later in life. Height-growth velocity retardation is a bodily response to nutritional deficiencies during early life, childhood and adolescence. Natural experiments have been used to study the impact of extreme nutritional deficiencies at or before or after birth on cohort-average adult height. For example, Godoy et al. (2007) find that among native Amazonians, rainfall variability at ages 2-5 has a negative effect on adult height among women. Alderman, Hoddinott and Kinsey (2006) consider the effect of exposure to drought and war at ages 2 to 3 in Zimbabwe on height towards the end of adolescence. Like us, they also consider the difference between siblings' heights as the outcome of interest. They find significant effects of adverse conditions at ages 2 to 3.

Some famine studies consider adult height as an outcome measure. Stanner et al. (1997) do not find any effect on adult height of having been exposed to intra-utero starvation during the Leningrad siege of 1941-44. Susser and Stein (1994), using data on cohorts born around the Dutch winter famine 1944-45, find that adult stature is susceptible to the postnatal but not the prenatal environment. Gørgens, Meng and Vaithianathan (2007) provide evidence that the dynamic selection due to the famine may completely cancel any causal effect on height, using data of families that experienced the Chinese famine around 1960.

Very low birth weight, assumed to reflect adverse nutritional conditions in utero, is associated with short adult stature (Hack et al. 2003). This association between birth weight and adult height has also been found among monozygotic twins, who share the same genes but may experience different intrauterine environmental conditions (e.g. due to various

positions in the womb), indicating that nutrition in utero affects adult height (Black et al., 2007).

A number of studies have found that the height effects of adverse conditions in early life can be mitigated by improvements in conditions up to adulthood. Notice that for our strategy to identify critical periods of development it is necessary that adult height is responsive to improvements in conditions at positive ages. Children from poor countries who are adopted by rich countries commonly show substantial catch-up growth. For example, Rutter et al. (1998) compare Romanian adoptees in the U.K. to native adoptees, and they find complete height catch-up at age 4 among the former if they entered the U.K. before the age of 6 months. Catch-up among those entering between 6 months and 2 years of age is also impressive but smaller. This suggests that the age interval between 6 months and 2 years constitutes a critical period. The meta-analysis by Van IJzendoorn, Bakermans-Kranenburg and Juffer (2007) concludes that height catch-up among children adopted within their first year is virtually complete in the sense that they become as tall as children in the destination country or environment, whereas catch-up of older children is incomplete. This suggests that adult height may not be a good marker for critical periods in the first months after birth. Note that adoptee studies can not deal with the potentially endogenous selection of adoptees.

Height development catch-up may also occur if living conditions improve past the first few childhood years. Slave children in the US, who were among the shortest cohorts of children ever measured, possibly due to attenuated breastfeeding and limited food supply throughout their early childhood, displayed a strong increase in height growth past the age of 10 at which they entered the workforce, presumably because they started to receive better nutrition while working. Their ultimate adult height was only 1-2 cm shorter than contemporaneous Union Army troops (Steckel, 1987, 2008).

2.3. Health after immigration

In this subsection we cite some previous studies based on immigrants. Note that the adoptee studies mentioned in the previous subsection are also relevant here, to the extent that they consider cross-national adoptions.

A small number of anthropological studies, while focusing on different issues than we do, present results that can be related to ours.[4] Susanne (1979) reports adult height of a small sample of about 330 male individuals who migrated before age 25 from Italy to Belgium

[4] See Susanne, Vercauteren and Zavattaro (1998) for an overview of this literature.

(which was more wealthy). Age at migration is aggregated into the categories 0-<5, 5-<15 and 15-<25, and children born after migration are observed as well (siblings are not observed). The adult height of those who migrated after age 5 is significantly lower than of the others. There is no difference between those born after migration and those who migrated before age 5. Lasker and Evans (1961) consider the adult height of individuals who migrated from Mexico to the US and subsequently returned to Mexico while spending at least two years abroad. They distinguish between the age categories 0-<16 or 17-<26. The sample is very small (61 men and 35 women) and although the adult height differences between the two age categories are large for each gender, they are not significant.

Pak (2004) compares the height of adult escapees from North Korea to the height of adult South Koreans and shows that the divergence during the past 50 years moved in tune with the economic divergence of the two countries. Bates and Teitler (2008) provide a comprehensive literature overview of the health development of adult immigrants. They also list many studies showing that immigrant teenagers are more likely to develop health problems than natives. All studies mentioned in this subsection so far are potentially affected by the same selection problems as adoptee studies. In particular, they do not consider siblings.

Bates and Teitler (2008) also conjecture the existence of critical periods for cognitive and non-cognitive skill acquisition among adolescent immigrants. Identity formation and acquisition of a foreign language may be disproportionally difficult for adolescents beyond a certain threshold age. Bleakley and Chin (2010) survey the evidence for a threshold critical age of 12 for language acquisition. To prevent a misunderstanding of terminology, notice that this does not mean that age 12 is a critical age in our sense (i.e., in the sense that language acquisition would be harder if it does not occur at age 12), but rather that the age interval up to 12 is critical. Böhlmark (2008) provides some evidence for cognitive skill acquisition among immigrants in Sweden, by studying the effect of the number of years since immigration on the grades at graduation.[5] He infers from the estimation results that there is a threshold age around 10.

The existence of an immigration age A, say $A=10$, after which cognitive skill acquisition is more difficult, might be an implication of a critical age for health development

[5] To our knowledge, Böhlmark (2008) is the only published study using a family-specific fixed effect in combination with migration data of children. His explanatory variable is the time from immigration to graduation. Åslund, Böhlmark and Nordström Skans (2009) considers measures of social integration.

just below *A*. This would be visible in our results. Alternatively, if there is an immigration age *A* after which skill acquisition becomes harder then this by itself may lead those who immigrate beyond age *A* to display more adverse health-related behavior, and to the extent that this behavior is ultimately revealed in adult height, such a causal pathway may also show up in our estimates. This pathway is not driven by economic, nutritional, or health differences between Sweden and the country of origin, but by cultural barriers. By stratifying the analyses by country of origin, we can deal with this. Immigrants from Nordic countries do not face a major change in social and cultural context, and they do not face the same level of difficulty in learning Swedish as immigrants from outside of Europe. Immigrants from Finland have learned at least some Swedish at school prior to migration. So we may assess whether jumps in the effect of age at migration on adult height vary with the social and cultural distance to Sweden.

3. Institutional context

3.1. Swedish society and health care

Sweden is a relatively wealthy country with 9.2 million inhabitants. Swedes are among the tallest in the world, especially the men in recent birth cohorts. Whereas the country fell in the OECD ranking of per capita GDP from 4 in 1970 to 12 in 2005, it has always had one of the 10 highest ranks in the UN Human Development Index (HDI) which incorporates life expectancy, adult literacy, educational attainment and GDP per capita into a composite measure. During the period 1960-75, the infant mortality rate per 1000 newborn decreased from 17 to 9, which was the lowest infant mortality rate in the OECD. Since then, infant mortality rates have declined further, to 2.4 in 2005, which is a level similar to Iceland, Finland and Japan.

Sweden, together with Denmark, has also had the lowest estimated poverty rates and Gini coefficients since OECD started to make comparisons in the 1970s. The health care system is based on universal compulsory national health insurance.[6] Health care fees are very low.[7] There is a co-payment for prescription drugs, but the annual expenditure per patient is

[6] Health care is provided by 20 county councils. The county councils finance care, mainly via local proportional income taxes, and they also produce almost all health care.

[7] Fees for primary health care as well as specialist consulting were about 50-100 SEK in 1990, rising to 100-250 (depending on council) in 1997 (exchange rate: about 6-8 SEK/USD 1990-97). From 1998, children's (up to 18

capped. The financial constraints on parents' demand for children's health care are therefore small. In school, children are served free lunches, typically based on potatoes, rice or pasta in combination with fish or meat.

3.2. Immigration

Post-war immigration to Sweden can be divided into two phases. The period 1945-1975 is dominated by labor migration of single men from other Nordic and from Southern European countries. Since then, refugee and tied-mover immigration have become more common, where the latter category consists of family-reunification immigration as well as immigration of family members who migrate simultaneously with the main immigrant. In 2000, 11.3% of the Swedish population was foreign born, of which one third had migrated from the Nordic countries, one third from other European countries, and one third from non-European countries. During the period 1980-2001, the Swedish admission board granted residence permits to about 730,000 people, of whom 8,000 were labor migrants, 275,000 were refugee migrants, and 354,000 were tied movers, while the remaining were guest students, adoptees and EU/EES–movers. In this study we focus on children in immigrant households, and these are tied movers. See Böhlmark (2008) and references therein for a description of the Swedish educational system and special arrangements for immigrant children.

The recorded date of immigration into Sweden is the date at which a residence permit is granted. This is not always the same as the date of migration from the source country. A family who immigrate as tied movers to a refugee and who immigrate simultaneously with the refugee might spend time in an asylum camp before receiving a residence permit. Rooth (1999) reports that this typically takes less than half a year. Immigration due to family reunification does not involve an intervening spell.

Individuals with a residence permit may apply for Swedish citizenship. Advantages of naturalization are that it gives the individual a passport with which he/she can travel abroad, that it leads to the certainty associated with the right to live and work in a rich and stable country, and that it gives the right to participate in elections. In our observation window, the time from the date of immigration until Swedish citizenship was about two years for families from Nordic countries and about 4 to 5 years for immigrants from other parts of the world.

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years of age) primary health has been free of charge. Maternal care during pregnancy is free of charge in most counties.

Some decide not to naturalize; this is particularly common if they migrate from other EU countries (see Section 4 for figures extracted from our data registers).

3.3. Military service

In principle, every male Swedish citizen enlists for the military when turning 18. Enlistment is mandatory. Individuals who become Swedish citizen after the age of 17 but before the age of 25 usually have to enlist as well. The Enlistment Office performs a series of tests, including a measurement of the individual's height.

Refusal to enlist leads to a fine, and eventually to imprisonment. Individuals are exempted from enlistment if they are imprisoned, if they have ever been convicted for heavy crimes (which mostly concerns violence-related and abuse-related crimes), or if they are in care institutions and are deemed to be unable to function in a war situation. During our study period, the annual cohort size of men turning 18 was about 50,000. Per cohort, around 1,250 (i.e., 0.25%) were exempted from enlistment. No information is available about the distribution of immigrants in the latter group.

4. Data and summary statistics

Our data integrate registers from Statistics Sweden, including the Register of the Total Population (RTB, with information on birth date, date of immigration, sex, country of birth, and parents' country of birth), the Multi Generation Register (Flergenerationsregistret, with identification of siblings on the mothers side through a family ID), and the Swedish National Service Administration, which contains every individual living in Sweden in the year 1999 who enlisted for the military between 1984 and 1997. For each of the individuals in the latter we observe the height at the date of enlistment, i.e. close to age 18. In our sample, 73 percent enlists at age 17 to 19, while 12 percent enlists after age 21.[8] Given the enlistment rules, our data only contain individuals born between 1956 and 1979. Data originating from the military service enlistment registry have been used in a number of studies, usually to establish associations with outcomes later in life, and usually focusing on native Swedish individuals.

[8] Since we find a positive correlation between age at immigration and age of enlistment, and since our measure of height may depend on at what age it is measured, we will control for age of enlistment in our regressions. We also perform sensitivity checks on whether the estimates change when we put various restrictions on the age of enlistment on our sample. Note that male growth usually ends by age 18.

See e.g. Magnusson, Rasmussen and Gyllensten (2006) who find a positive relation between height and educational achievement among adults.

Immigrants are identified from the registers as having been born abroad and having foreign-born parents. Siblings are identified by having the same mother. In our analyses, we focus on two overlapping samples. The first (smallest) one consists of those who immigrated to Sweden, who enlisted, and who have at least one brother who also enlisted. Moreover, we restrict the sample to brothers born in the same country.[9] We do not observe the height of those not becoming Swedish citizens, since those do not enlist.[10] We also restrict our sample to those who immigrated to Sweden before the age of 18.[11] Although those who become Swedish citizen after age 17 and before age 25 have to enlist up to the age of 25, the registers are sometimes inaccurate when it comes to assigning a person to a family for those immigrating at age 18 or older, i.e., as adults.

The RTB register contains 51,578 male individuals who were born abroad, immigrated before age 18, and belong to the birth cohorts 1956-1979. Of these, we observe the height for 37 percent, or 18,827 individuals, namely for those who became Swedish citizens and enlisted in 1984-1997 and who lived in Sweden in 1999. The share is relatively low for Nordic (34%), Western (22%) and Southern European immigrants (28%), but higher for Eastern European (47%) and non-European immigrants (Middle East 43%, Asia 52%, Latin America 43%, and Africa 26%). This reflects naturalization decisions by their parents as well as variation across entry year by region of origin. In the end, our sample of foreign-born brothers consists of 5,576 individuals, distributed across 2,524 families. The four most common countries of origin are Finland, Iran, Iraq, and Bosnia.

The data allow us to examine which family members naturalize. Among pairs of brothers having immigrated to Sweden before the age of 18 who are 25-40 years old in 2003 and who live in Sweden in 2003, both brothers were Swedish citizens in 76% of the cases, whereas none of them was in 8% and one of them in 16% of the cases. Among brother pairs of Swedish citizenship, in which both migrated before the age of 13, both enlisted in 70% of the cases, while none of them enlisted in 10% of the cases. This reflects the fact that some brothers who arrive at higher ages, such as 17, will pass the enlistment age before becoming Swedish citizens.

[9] For brothers born outside Sweden, being born in different countries is uncommon (we lose only 87 observations).

[10] However, typically, all siblings within a family have the same nationality.

Our second main sample adds families with both Swedish-born and foreign-born children. This increases the sample of families, for example because some families have two sons where one is born in Sweden and one is born outside Sweden. As a result, this sample consists of 8,691 brothers in 3,893 families.

The two key variables in the empirical analyses – height and age at immigration – are objectively measured. Age at immigration is measured as the difference between the date of immigration, which is the first day of being granted a residence permit in Sweden, and the individual's birth date. We transform the difference into numbers of years. Recall from Section 3 that even among children who immigrate at the same time as their refugee parents, the time between entry into Sweden and the registration as immigrant is often less than half a year. There is no intervening spell for non-refugee households.

In the raw data, the average adult height is almost identical for those born in Sweden (177.71 cm) as for those who arrived before their first birthday (177.78 cm). Only relatively few (2%) of the children arrive before the first birthday. This may reflect that parents find it stressful to immigrate at the moment around birth of a baby, and hence, few choose to do so. The average adult height declines steadily as a function of age at immigration. At age 16 it equals 171.36 cm. However, this decline partly reflects the fact that children from European immigrants tend to be younger at immigration. Europeans may find it easier to plan their move according to the age and situation of their children. For individuals migrating from poorer countries or from war zones, the age at immigration may be less determined by the age of the children. Over-all, the most common immigration ages are 3 to 6. Immigration ages above 15 are relatively rare. The observed decline is stronger for immigrants from non-European countries than for those from Europe.

Table 1 presents summary statistics for the above-mentioned two full samples as well as for the subsamples defined by 5 country groups: Nordic, Europe (meaning Europe apart from the Nordic countries), Middle East, Asia (apart from the Middle East), and Latin America. The Nordic group consists for 89% of individuals from Finland. The European group mainly contains individuals from Eastern and Southern Europe. In the empirical analyses, the additive height effect of the country of origin is captured by the family-specific fixed effect (see Section 5). However, the effect of age at immigration may depend on the

[11] This restriction implies that we exclude 306 individuals above the age of 17 at immigration and who have at least one brother that is included in the data set being used.

region of origin. We therefore also present estimates for the two largest country groups: Nordic and Middle East.

5. Empirical model

We estimate versions of the following equation:

$$y_{if} = \alpha + \theta A_{if} + \delta X_{if} + \mu_f + \varepsilon_{if},$$

where y_{if} denotes the adult height in cm of individual i in family f, A_{if} is a vector of variables capturing the age at immigration of individual i, X_{if} is a vector of control variables, μ_f captures family-specific unobserved determinants, and ε_{if} is an individual-specific, i.i.d. error term. We will also estimate specifications where age at immigration enters linearly or as a piecewise linear function, and specifications that impose that adult height is non-increasing in age at immigration.

Recall that we are ultimately interested in the causal effect on adult height (and health) of an intervention in the life of an individual that consists of the assignment of good or bad living conditions in a specific age year. For each age year, we may construct a counterfactual-outcome framework in which an individual has different potential adult heights, depending on whether the assigned conditions are good or bad in that age year. The difference of the two potential outcomes is then assumed to equal the difference in potential outcomes between the case where the age year corresponds to the assigned age at immigration versus the case where immigration occurs at the subsequent age. A slightly less ambitious and less restrictive alternative approach is to start from the outset with potential adult heights defined by the assigned year of migration from bad to good conditions. In either case, the above equation captures the actual individual outcome in case conditions are good from the actual age at migration onwards. We exploit cross-individual variation in outcomes in order to relate actual outcomes to the causal effects of interest. Such inference needs to take into account that the actual treatment assignment process in the data is not randomized, in the sense that there are unobserved confounders that may influence the outcomes and the age at migration. By assuming that these factors are family-specific, we can remove them by fixed effects methods.

It is useful to discuss the latter in some more detail. The migration decision is taken at the family level, and the family members migrate at the same point in time. The family fixed effect μ_j will absorb all characteristics that are identical across members of the same family, such as country of migration, year of migration, origin, family structure, traditions, values norms, habits, wealth, household practices, neighborhoods, and family-level preferences. The estimates of θ are therefore not affected by factors at the family-level that are correlated with age at migration. (In OLS analyses across children from different families, such unobserved determinants of migration at the family level may confound the estimated effects of age at migration.) The fixed-effects approach also conveniently deals with selectivity of the observation window and the population from which we sample. For example, one may argue that our population is a selective sub-population of the population of all immigrants, because it only contains naturalized immigrants. To the extent that the association between naturalization and height is captured by the family fixed effect, our results can be generalized to the full population of immigrants.

As noted in the introduction, the migration decision may also be induced by a particularly bad health of one of the children, in which case the conditions for a fixed-effects approach based on all brothers would be invalid. We are able to deal with this by performing analyses with data from families with at least three sons, excluding the youngest (or other subsets of brothers).

Of course, age is an imperfect indicator of physical development. For a given age at immigration, different children may be in somewhat different developmental stages. In addition, as we have seen, there is some variation, ranging from zero to a few months, in the time between the moment of entering the country and the moment of receiving a residence permit. This is why we abstract from model specifications where age at immigration is measured at a finer level than the annual level, and why we do not consider non-parametric fixed-effect panel data model estimation methods (see e.g. Lee and Mukherjee, 2009, for such estimation methods).

With estimates based on the sample of siblings born outside Sweden, the age at immigration dummies in A_{if} range from age 1 to age 17, with age zero being the omitted reference category. With the sample that includes Swedish-born siblings, the omitted category is replaced by being born in Sweden, and we can identify the effect of immigration at age zero.

Since height is measured at about the same age for everyone, and since height does not change to any important extent after the age of 18, the age at test should be virtually orthogonal to our height variable. To be sure, however, we include a control variable measuring the exact age at which the measurement was taken.[12] In addition, since the siblings in our sample immigrate at the same point in time, any immigrant-cohort effects will be picked up by the family fixed effect.

A complication not solved by family fixed effects is that birth order may affect height. Since age at immigration is correlated with parity, ignoring this may induce a spurious negative correlation between age at immigration and height. A substantial literature suggests that parents often favor earlier-born children.[13] If this affects their height, then any negative effect of age at immigration on height would be underestimated. To address this concern we include an indicator of being first-born in our regressions. The effect is identified from the effect of the difference of age at migration because the former does not increase in the age difference.

We finish this section by addressing secular drifts in adult height in consecutive birth cohorts. Younger siblings benefit from a positive drift, and this could lead to over-estimation of the effect of living conditions during childhood on adult height. Moreover, if the drift is larger in the countries of origin than in Sweden then the height gap between birth in Sweden and birth in another country is on average smaller for those who migrate at a more recent point in time. As it turns out, in Sweden during our study period there has hardly been any drift. Garcia and Quintana-Domeque (2007) provide estimates for a range of European countries. In Sweden and Finland, the average height among men in adulthood is constant across the birth cohorts 1960-1980. However, in Southern European countries, there has been an increase of a few centimeters across these cohorts. Deaton (2007) provides estimates for female birth cohorts 1950-1980 in groups of countries across the world. It turns out that the adult-height increase is strongest in Southern European countries. In developing countries, the increase was less than a centimeter. We conclude that the drift is potentially only an issue for migrants from Southern European countries. Even then, it is not obvious that conditions in the local place of origin has been improving as much as average conditions in the country or

[12] In addition, we will conduct sensitivity analyses, putting different restrictions on what ages to include.

[13] For example, Horton (1988) found that later-born children in the Philippines received less nourishment than first-born, as assessed through children's height and weight. A number of studies have found that in developed countries and developing countries, later-born children are less likely to be vaccinated (Barreto and Rodrigues, 1992; Kaplan et al., 1992). First-borns often have higher educational achievements than subsequent siblings (Black et al., 2005; Conley and Glauber, 2006; Kantarevic and Mechoulan, 2006).

continent of origin. After all, the reason to migrate in the first place may be that conditions have not improved over time or have deteriorated. Notice also that difficulties to adjust to a Western lifestyle may cause us to under-estimate the effects. Finally, note that a drift is mostly a smooth process that cannot generate the discontinuities that we are after.

6. Results

6.1. Estimated coefficients for the baseline specifications

Table 2 gives the estimates of the baseline model specification, with family-specific fixed effects. The effect of age at immigration can be presented in various ways. Column (1) lists the estimates of the parameter vector θ, giving the reduction in adult height as a function of age at immigration, where the latter is indicated by dummy variables, the omitted reference category being "birth after immigration". Critical periods are identified by downward jumps in these coefficients as a function of age at immigration. Therefore, column (2) reports the estimated first-differences of consecutive elements of θ. Notice that specifications (1) and (2) fit the data equally well, but the parameterization in (2) enables more direct insights into the issues of interest.

A priori it is hard to believe that postponing migration by one year can have a positive effect on adult health.[14] In column (3) we impose this monotonicity constraint that consecutive elements of θ are non-increasing, i.e., that their first differences are non-positive. We enforce this constraint through inequality (non-positivity) constraints on all the separate first-differences from column (2). The specification (3) is more parsimonious than the other two, because some of the first differences of the elements of θ are estimated to be on the boundary zero of the parameter space. The standard errors of the other coefficients in (3) are conditional on these first differences being equal to zero.

The coefficients in column (1) follow a decreasing pattern, and they are significantly different from zero at the 5% level for all immigration ages beyond age 1. At immigration

[14] Over-compensating refeeding after immigration following a short period of severe starvation might have a detrimental effect on adult health, and in such cases the over-all effect might even be worse than if the migration had taken place several years after the short period of starvation provided that the child survives the starvation (see Sparén, et al., 2004, for a discussion). Recent work by Kaati et al. (2007) and Kaati (2010) suggest that exposure to adverse economic and nutritional conditions around age 11 for boys triggers an epigenetic change that is transmitted to subsequent generations, leading to a lower mortality rate among grandsons when they reach advaced ages, but simultaneously increasing the diabetes risk in subsequent generations. They do not report effects on longevity of those who are directly exposed to the adverse conditions.

ages beyond 8, adult height is between 3.4 and 6.1 cm smaller than if the child is born in Sweden. Such differences are too large to be attributable to secular changes within the countries of origin.

The data fit of specification (3) is almost equal to the fit of the unconstrained specification (2). In particular, the p-values of the non-zero coefficients are barely affected. This suggests that the monotonicity constraint is satisfied. Figure 1 visualizes the age coefficients in column (3) by plotting the reduction in adult height as a function of age at immigration in comparison to adult height if birth takes place after migration. The coefficients for age 9 imply that adult height is relatively strongly dependent on whether one immigrates just before the age of 9 or whether one immigrates after this age. In specification (3), the adult height difference is 0.94 cm. This suggests a critical period in children's development at age 9. The estimated difference in adult height is significantly different from zero. Notice that the p-values in columns (2) and (3) apply to two-sided tests of the hypothesis of a zero coefficient. When positive coefficient values are ruled out then one-sided tests have to be used, so the p-values must be divided by 2, and the null hypothesis of a non-zero coefficient is rejected at lower test levels (e.g. at 2.5% instead of 5%).

The results in Table 2 also suggest critical periods at ages 0 and 6. The adult height effect of birth just before migration versus birth after migration is equal to 0.6 cm. The quantitative magnitudes of this jump and the jump for conditions at age 6 are smaller than for conditions at age 9. At age 5, the coefficient in columns (2) and (3) is almost significantly different from zero at the 10% level, while it is significantly negative at the 10% level.

At adolescent ages, the unconstrained coefficients in column (2) display an alternating pattern of positive and negative values. However, this pattern can be attributed to data limitations. First, the sample size decreases by age at immigration, and indeed, the positive coefficients are not significantly different from zero. A gradual decrease in adult height as a function of age at migration among adolescents is plausibly explained by the combined effects of nutrition, access to health care, language acquisition problems, and identity formation problems (recall the discussions in Section 2). Secondly, as age at immigration increases beyond age 15, the results are less reliable, as only the siblings who naturalize very fast are enlisted if they immigrated as a teenager.

To shed more light on the results, we estimate separate specifications for migrants from Nordic countries and for migrants from the Middle East. These are the two largest country groups, and they are quite different in terms of living conditions and distance to

Sweden, even though people able to migrate from the Middle East may more often come from the upper end of the income distribution in their country of origin. Estimates by other regions of origin display a reduction in adult height as a function of age at immigration, but the sample sizes are too small to derive more specific conclusions.

The separate estimates for the "Nordic" and "Middle East" subsamples are in Table 3. Clearly, in the Middle East subsample, the point estimates of θ are more negative, and their decrease as a function of age at migration is stronger, than in the Nordic subsample. More importantly, we find a critical period at age 9 in both subsamples, so the results confirm a critical period at age 9 regardless of the region of origin.

The results for the Nordic countries do not provide any evidence for any other critical period in childhood ages. In particular, there are no critical periods at ages 0, 5 or 6. The estimates for the Middle East do provide evidence for a critical period at ages 5 and 6, and at first glance they may also seem to provide evidence for critical periods at some other ages, but the standard errors of the coefficients at the latter ages are too large to reject the null hypothesis of absence of a critical period. We conclude that there is insufficient evidence for critical periods at ages 0, 5 and 6 that are independent of the country of origin. Incidentally, notice that the peculiar pattern at adolescent ages of the unconstrained coefficients in Table 2 as a function of age is not present for the Nordic countries, so this pattern is not replicated by region of origin. This confirms our conjecture that the pattern in Table 2 is a random sampling feature.

6.2. Discussion of the baseline results

It is interesting to connect our finding of a critical period at age 9 to the existing literature on nutrition and height and health later in life. As noted in Subsection 2.1, Sparén et al. (2004) identify a critical period around age 9, in the sense that a disruption of nutrition due to a famine at the onset of puberty causes cardiovascular problems much later in life.[15] Koupil et al. (2007) do not find an effect of this famine on height, but this may be because the famine was so severe that it generated a strong dynamic selection in height outcomes. The medical literature provides support for the existence of a critical period at age 9 for men. As documented and surveyed by Marshall and Tanner (1986), Gasser et al. (1994), and Zemel (2002), the earliest manifestation of puberty concerns the so-called "fat spurt" at age 9. In this

[15] Notice that our results are also consistent with the early small-sample studies of Lasker and Evans (1961) and Susanne (1979) which, taken together, suggested a critical period somewhere between ages 5 and 15.

spurt, the body collects resources in anticipation of the adolescent growth spurt. Sparén et al. (2004) argue that nutritional distortions and stress at this stage may lead to a permanent disruption of blood pressure regulation, leading to long-run cardiovascular health problems. Other studies have related the calcium intake at age 9 to adult height. Van Dusseldorp et al. (1996) consider children aged 9 in households that follow a strict macrobiotic diet. A moderate addition of dairy products to the daily diet of these children led to a significant catch-up growth. Our results confirm the importance of the quality and quantity of nutrition at age 9 for long-run health outcomes.

It is important to point out that a critical period at this age cannot be explained by institutional features of the Swedish school system. In this system, individuals used to enter primary school at age 7. The allocation into secondary-school trajectories that are defined by their level of complexity only takes place after age 14. This suggests that the critical period is not due to the importance of the corresponding school year in Sweden. This in turn lends credence to the view that our result captures a direct health effect on adult height (and, ultimately, adult health) instead of an indirect effect of health through educational attainment on adult height.

Notice also that age 9 does not correspond to ages at which the average bodily growth is highest. The same applies to ages 5/6. This means that the findings are not simply driven by a mechanical proportional decrease of height growth at these ages.

When using the full sample, we find evidence for a critical period at age 0, but this is not corroborated by analyses by region of origin. At the same time, the literature discussed in Subsection 2.1 demonstrates that conditions at birth do have important long-run health effects. We conclude that adult height does not do a good job in detecting the critical period around birth. In other words, it does not strongly reflect the causal long-run health effects of conditions close to the date of birth, so it is not a good marker for those conditions. This is in line with the studies on adoptees, mentioned in Subsection 2.2, which find that height catch-up after adoption very early in life is almost complete.

6.3. Sensitivity analyses

We address the sensitivity of the results with respect to the criteria for inclusion in the sample and the regression specification. First, we exclude Swedish-born siblings. Columns (1) and (2) in Table 4 provide the estimates corresponding to those in columns (1) and (3) in Table 2 for the full sample. Figure 2 visualizes the coefficients in column (2) of Table 4. (Notice that

the coefficients in the column (1) in Table 2 and in Figure 1 are differently normalized with respect to zero than those in column (1) in Table 4 and in Figure 2.) Clearly, the results are virtually identical. The critical periods are exactly like in Table 2. This means that the results with the full sample are not driven by migration that is induced by pregnancy of the mother around the time of migration.

Next, we compare family Fixed Effects (FE) regression results to those from the corresponding OLS regression (columns (1) and (3) of Table 4). The FE coefficients are typically larger in absolute value than the corresponding OLS coefficients, so, ignoring the self-selection into migration leads to smaller coefficients. Apparently, migrant families with relatively young sons on average have larger height. This is in line with the fact that families from Western Europe often have smaller children upon migration.

Note that neither the OLS nor the FE estimation is able to deal with self-selection due to a particularly bad health of their smallest children. To investigate whether immigration is driven by adverse health of the youngest or oldest brother in the family, we replicate the analyses excluding the youngest and/or oldest brother at the moment of migration. This entails that we discard data from families with only one or two brothers at the moment of migration. It turns out that the results (available upon request) are very similar to those presented in this paper. Excluding the youngest brother at the moment of migration results in a slightly smaller (in absolute value) decline of adult height as a function of age at immigration. This is in agreement with the hypothesis that an exceptionally weak youngest child in the family may trigger a migration. However, the results on critical periods are the same as in our baseline analyses.

Column (4) of Table 4 displays the FE estimates if the effect of age at immigration is assumed to be linear. This shows that the average height loss per year spent in adverse conditions compared to Sweden equals 0.25 cm. Notice that it is not clear whether we should contrast the linear model to the model with age-specific coefficients. The literature on which we build leads us to focus on hypotheses concerning the presence of critical periods. The models that we estimate do not rule out critical periods at any age. Accordingly, we estimate a large number of coefficients to capture age at immigration. Alternatively, we could have looked for significance in more parsimonious specifications, notably specifications that are piecewise constant or piecewise linear in the age at immigration, where the intervals in which the effect is smooth in age at immigration are larger than a year. In general, in the case of piecewise constant specifications with a discontinuity at one age, the size of the jump is

significantly negative. In the case of a piecewise linear specification it sometimes is significant and it sometimes is not.

One concern with the sample used for Table 2 is that it includes mothers who re-married to Swedish men and had additional children with them. In such a case, some siblings do not have the same father, introducing a source of heterogeneity that the fixed effects estimator may not be able to deal with. However, the results do not change if we omit siblings whose father was born in Sweden (Table 5, column (2)).

Another concern is that early-life experiences among those born after migration may depend on the amount of time between migration and birth. In order to check the sensitivity of the results for this, we re-estimated the models with cases where the sibling was born within at most one year after migration (Table 5, column (3)). Moreover, we estimate models where the explanatory variables include binary indicators for the number of years between migration and subsequent birth. In all cases, our main results are robust. In addition, adult height does not depend in any significant and/or systematic way on the number of years between migration and subsequent birth.

As noted above, some individuals in our sample enlist at ages over 18. As a sensitivity check we re-estimate the model with various age-at-enlistment restrictions. It turns out that the results are robust with respect to this. We also re-estimate the models with a sample in which the height distribution is trimmed at both sides with 1% (i.e. at 163 cm and 186 cm). This results in a decrease in the number of observations to 4,901. Again, the results are not affected by this.

Yet another concern is that height growth does not always terminate upon age 18. It is conceivable that the individuals who experienced adverse conditions at age 9 take longer to reach their adult height. In that case we may find a causal effect of conditions at age 9 on height at age 18, but there may be no causal effect of conditions at age 9 on the realized adult height at, say, age 25, and then the conditions at age 9 would perhaps not exert a long-run effect on health later in life. However, in general, height growth after age 18 has been found to be negligible (see e.g. Marshall and Tanner, 1986). The average growth after age 18 is of a smaller order of magnitude than our estimated effect of conditions at age 9. Moreover, as we have seen, the “age at enlistment test” coefficient for adult height is always insignificantly different from zero. The same applies if we estimate separate coefficients for each enlistment age above 18: if we control for age at migration then age at enlistment is not related to height.

6.4. Interactions with parents' education and age at migration, and year of migration

In Subsection 6.1 we allowed the effect of age at migration (i.e., the effect of adverse conditions at a certain age) on adult height to vary with the global region of origin. It is conceivable that the former effect also varies with characteristics of the parents and the year of migration. Highly-educated parents may cope better with adverse economic conditions in the country of origin, both because they know more about health risks and because they may have a higher income. Parents arriving at lower ages may more easily adapt to Swedish culture and take greater advantage of the improved living conditions. For some countries, such as Finland, the difference in economic conditions compared to Sweden has been narrowing over time, and the effect of age at migration could therefore decrease over time.

To proceed, we consider the following four interaction variables: father's level of education, mother's level of education, mother's age at migration, and year of migration. Due to missing observations on parental education, the sample size is reduced to 3,308. For reasons of conciseness, we only consider specifications in which age at migration itself is included linearly rather than by way of 17 or 18 coefficients, since otherwise the number of interaction terms would exceed 65. The results are in Table 6. They show that the effect of adverse conditions is smaller if the parents have higher education. This interaction effect is strongly significant for the mother's education, and the size of the effect is also larger for the mother's education. This is in line with previous results, suggesting that the mother's education is more important for the health of the child than the father's education (see e.g. Strauss and Thomas 1995).

To the extent that parental education influences health behavior of the parents towards the children, these results suggest that our findings do not reflect a biological causal pathway only. Rather, the parents' behavior may be able to influence the detrimental effects of adverse conditions.

The mother's age at migration does not affect the results so far. The same applies to the calendar year of migration.

7. Conclusions

This paper introduces a new approach to identify critical periods in childhood and adolescence for health later in life. By exploiting the differences in ages at immigration

across brothers in the same family, and by comparing brothers born outside and within Sweden, we are able to assess the causal effect of conditions at given pre-adult ages on later life health.

Adult height decreases with exposure to adverse conditions during childhood and adolescence. The higher the age at which conditions improve, the lower the adult height. Adverse conditions at age 9 have a particularly strong effect on adult height. This applies to migrants from global regions that are widely different in terms of economic conditions. The importance of age 9 is corroborated by some existing literature on nutrition and height and health later in life. It can not be explained by institutional features of the Swedish school system. This in turn suggests that our result captures a direct health effect on adult height (and, ultimately, adult health) instead of an indirect effect of health through educational attainment on adult height. Analogously, the absence of critical periods at ages where major transitions in educational trajectories take place (e.g. from primary school to high school) suggests that the experience of such a transition in the country of destination does not play a major role in health outcomes later in life.

At the same time, a high level of the mother's education reduces the effect of adverse conditions during childhood on adult height. To the extent that the mother's education influences health behavior towards the children, this suggests that our findings do not reflect a biological causal pathway only, but rather, that the parents' behavior may be able to influence the detrimental effects of adverse conditions.

We do not detect effects of conditions close to the date of birth. Apparently, adult height does not reflect causal long-run health effects of conditions close to the date of birth. This is in accordance to adoptee studies claiming complete height catch-up if adoption takes place within 6 months after birth, although those studies are sensitive to selection bias.

The results suggest that it is particularly important to take care of living conditions of children at age 9. Failure to do so may result in higher long-run health care costs than failure to focus on conditions at other childhood ages.

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Table 1. Descriptive statistics.

Variable	Pooled sample	Sweden	Nordic	Europe	Middle East	Asia	Latin America	Pooled sample of sibs born outside Sweden
Adult height in cm	175.56	177.72	177.62	177.15	173.54	169.56	172.32	174.43
	(6.88)	(6.48)	(6.33)	(6.67)	(6.11)	(6.21)	(6.15)	(6.79)
Age at immigration	4.68	-3.12	3.75	6.05	9.27	8.88	7.71	7.62
	(5.85)	(2.75)	(3.14)	(4.54)	(4.43)	(4.33)	(4.29)	(4.55)
First-born sibling	.31	.014	.45	.54	.29	.27	.34	.31
Age at military test	19.26	18.60	19.32	19.61	19.70	19.03	19.11	19.45
	(1.77)	(1.02)	(1.68)	(2.10)	(2.03)	(1.61)	(1.84)	(1.89)
Fraction in data	1	0.20	0.28	0.13	0.23	0.09	0.074	0.64
Observations	8691	1746	2404	1150	2006	741	644	5576

Note: Standard deviations in parentheses

Table 2. Fixed-effect analysis of adult height in cm as a function of age at migration.

Age	(1) estimate element of θ	(2) estimate 1st diff. of θ w.r.t. age-1	(3) 1st diff. of θ with monotonicity constraint
Zero	-0.556	-0.556**	-0.435**
	(0.37)	(0.27)	(0.17)
One	-0.358	0.199	0.000
	(0.26)	(0.31)	
Two	-0.666**	-0.308	-0.249
	(0.27)	(0.24)	(0.22)
Three	-0.949***	-0.283	-0.288
	(0.29)	(0.24)	(0.24)
Four	-1.080***	-0.131	-0.139
	(0.31)	(0.25)	(0.25)
Five	-1.499***	-0.418	-0.420
	(0.34)	(0.26)	(0.26)
Six	-2.200***	-0.701**	-0.612**
	(0.36)	(0.29)	(0.24)
Seven	-1.975***	0.225	0.000
	(0.39)	(0.29)	
Eight	-2.207***	-0.232	-0.134
	(0.40)	(0.30)	(0.27)
Nine	-3.421***	-1.214***	-0.936***
	(0.43)	(0.33)	(0.28)
Ten	-2.809***	0.612	0.000
	(0.45)	(0.33)	
Eleven	-3.867***	-1.058***	-0.424*
	(0.47)	(0.33)	(0.24)
Twelve	-3.172***	0.696	0.000
	(0.51)	(0.33)	
Thirteen	-4.398***	-1.227***	-0.653**
	(0.52)	(0.34)	(0.25)
Fourteen	-3.848***	0.550	0.000
	(0.58)	(0.37)	
Fifteen	-5.108***	-1.260***	-0.876***
	(0.63)	(0.40)	(0.29)
Sixteen	-4.807***	0.301	0.000
	(0.71)	(0.42)	
Seventeen	-6.087***	-1.280**	-1.105**
	(0.77)	(0.51)	(0.44)
Age at test	0.0424	0.0424	0.060
	(0.061)	(0.048)	(0.048)
First born	0.621***	0.621***	0.641***
	(0.15)	(0.11)	(0.11)
Observations	8691	8691	8691
Sibling fixed effects	Yes	Yes	Yes

Notes: (1): FE regression with reference category "born in Sweden". (2): reparameretized FE NLS. First element is w.r.t. "born in Sweden". (3): monotonicity constraint imposed on (2).
*** p<0.01, ** p<0.05, * p<0.1. Note that these refer to two-sided tests; in (2) and (3) the p-values should be halved for one-sided tests. Standard errors in parentheses. Robust standard errors in (2) and (3).

Table 3. Fixed-effect analysis of adult height in cm as a function of age at migration, for migrants from Nordic countries and from the Middle East.

	Nordic: estimate element of θ	Middle-East: estimate element of θ
Zero	-0.285	-0.170
	(0.43)	(1.41)
One	0.467	-1.417*
	(0.32)	(0.79)
Two	0.307	-2.587***
	(0.36)	(0.74)
Three	0.0472	-2.010***
	(0.40)	(0.65)
Four	-0.293	-1.759***
	(0.46)	(0.66)
Five	0.474	-3.158***
	(0.52)	(0.66)
Six	-0.413	-4.136***
	(0.59)	(0.68)
Seven	0.236	-3.642***
	(0.68)	(0.67)
Eight	0.812	-3.082***
	(0.80)	(0.69)
Nine	-1.850**	-4.996***
	(0.84)	(0.70)
Ten	-0.951	-3.899***
	(0.94)	(0.73)
Eleven	-2.313*	-4.944***
	(1.28)	(0.72)
Twelve	0.246	-4.683***
	(1.58)	(0.76)
Thirteen	0.474	-6.136***
	(1.48)	(0.81)
Fourteen	-1.280	-4.737***
	(1.99)	(0.87)
Fifteen	-2.496	-6.224***
	(3.14)	(0.96)
Sixteen	-2.627	-5.751***
	(3.20)	(1.09)
Seventeen	-0.731	-7.401***
	(2.98)	(1.18)
Age at test	0.0430	-0.0744
	(0.101)	(0.112)
First born	0.0818	0.320
	(0.249)	(0.296)
Observations	3482	2173

Notes: FE regressions with reference category “born in.Sweden”. *** p<0.01, ** p<0.05, * p<0.1. Standard errors in parentheses.

Table 4. Adult height in cm as a function of age at migration. Sample of siblings born outside Sweden.

	(1) estimate element of θ	(2) 1st diff. of θ with monotonicity	(3) estimate element of θ	(4) estimate element of θ
One	-0.0333	0.000	-0.548	
	(0.62)		(0.64)	
Two	0.333	0.000	-0.265	
	(0.60)		(0.62)	
Three	-0.0798	-0.159	-0.211	
	(0.60)	(0.21)	(0.62)	
Four	0.117	0.000	-0.559	
	(0.62)		(0.62)	
Five	-0.363	-0.403	-0.669	
	(0.62)	(0.24)	(0.62)	
Six	-0.958	-0.523**	-1.016	
	(0.64)	(0.25)	(0.63)	
Seven	-0.731	0.000	-1.015	
	(0.65)		(0.64)	
Eight	-0.964	-0.150	-1.000	
	(0.66)	(0.28)	(0.64)	
Nine	-2.233***	-0.967***	-1.266*	
	(0.68)	(0.28)	(0.65)	
Ten	-1.561**	0.000	-1.562**	
	(0.69)		(0.65)	
Eleven	-2.476***	-0.255	-1.718***	
	(0.70)	(0.24)	(0.66)	
Twelve	-1.763**	0.000	-1.344**	
	(0.74)		(0.68)	
Thirteen	-2.993***	-0.646**	-2.359***	
	(0.76)	(0.26)	(0.68)	
Fourteen	-2.376***	0.000	-1.266*	
	(0.80)		(0.71)	
Fifteen	-3.629***	-0.827***	-2.745***	
	(0.86)	(0.29)	(0.75)	
Sixteen	-3.230***	0.000	-3.164***	
	(0.92)		(0.79)	
Seventeen	-4.566***	-1.136**	-2.887***	
	(0.98)	(0.45)	(0.85)	
Age at immigration				-0.251***
				(0.039)
Age at test	0.0217	0.0558	-0.0929*	-0.0221
	(0.074)	(0.057)	(0.056)	(0.066)
First born	0.476***	0.531***	0.454**	0.511***
	(0.18)	(0.13)	(0.19)	(0.18)
Constant			177.2***	
			(1.16)	
Observations	5576	5576	5576	5576
Sibling fixed effects	Yes	Yes	No	Yes

Notes: (1): FE regression with reference category "age 0 at immigration". (2): reparameretized FE NLS with monotonicity constraint. First element is w.r.t. "age 0 at immigration". (3): OLS version of (1) with country fixed effects. (4): FE regression with linear effect of age at immigration.

*** p<0.01, ** p<0.05, * p<0.1. Note that these refer to two-sided tests; in (2) p-values should be halved for one-sided tests. Standard errors in parentheses. Robust standard errors in (2).

Table 5. Sensitivity analysis with respect to births after immigration.

	(1)	(2)	(3)
Zero	-0.556	-0.327	-0.540
	(0.37)	(0.40)	(0.50)
One	-0.358	-0.0957	-0.569*
	(0.26)	(0.27)	(0.34)
Two	-0.666**	-0.588**	-0.620*
	(0.27)	(0.28)	(0.33)
Three	-0.949***	-0.794***	-1.107***
	(0.29)	(0.30)	(0.34)
Four	-1.080***	-0.828**	-0.909**
	(0.31)	(0.32)	(0.36)
Five	-1.499***	-1.275***	-1.599***
	(0.34)	(0.34)	(0.38)
Six	-2.200***	-2.074***	-2.186***
	(0.36)	(0.37)	(0.40)
Seven	-1.975***	-1.815***	-1.964***
	(0.39)	(0.40)	(0.42)
Eight	-2.207***	-1.936***	-2.223***
	(0.40)	(0.42)	(0.44)
Nine	-3.421***	-3.306***	-3.420***
	(0.43)	(0.43)	(0.46)
Ten	-2.809***	-2.770***	-2.797***
	(0.45)	(0.46)	(0.48)
Eleven	-3.867***	-3.764***	-3.818***
	(0.47)	(0.48)	(0.50)
Twelve	-3.172***	-3.077***	-3.060***
	(0.51)	(0.51)	(0.54)
Thirteen	-4.398***	-4.317***	-4.352***
	(0.52)	(0.53)	(0.55)
Fourteen	-3.848***	-3.773***	-3.777***
	(0.58)	(0.58)	(0.60)
Fifteen	-5.108***	-5.058***	-4.993***
	(0.63)	(0.64)	(0.67)
Sixteen	-4.807***	-4.705***	-4.677***
	(0.71)	(0.72)	(0.75)
Seventeen	-6.087***	-6.129***	-5.966***
	(0.77)	(0.78)	(0.81)
Age at test	0.0424	0.0523	0.0219
	(0.061)	(0.061)	(0.068)
First born	0.621***	0.638***	0.636***
	(0.15)	(0.16)	(0.16)
Observations	8691	8137	6820
Sibling fixed effects	Yes	Yes	Yes

Notes: etimated elements of θ from FE regressions with reference category "born in Sweden". (1) replicates (1) in Table 2. In (2), cases with Swedish-born fathers are excluded. In (3), Swedish-born siblings are only included if the mother had been in Sweden at most one year after migration. *** p<0.01, ** p<0.05, * p<0.1. Standard errors in parentheses.

Table 6. Interactions between the adult height effect of age at immigration and pre-migration family characteristics.

Age at immigration	-1.014***
	(0.24)
Age at test	0.152**
	(0.067)
First born	0.440**
	(0.19)
Age at immigration* father's education	0.0278*
	(0.015)
Age at immigration* mother's education	0.0515***
	(0.019)
Age at immigration* mother's immigration age	0.000632
	(0.0049)
Age at immigration* year of immigration	-0.00538
	(0.0061)
Observations	3308

Notes: sample of siblings born outside Sweden. FE regression with linear specification in age at immigration. *** p<0.01, ** p<0.05, * p<0.1. Standard errors in parentheses.

Figure 1. Adult height difference (in cm) relative to immigration after birth.
FE panel data estimates with monotonicity constraint; Table 2(3).

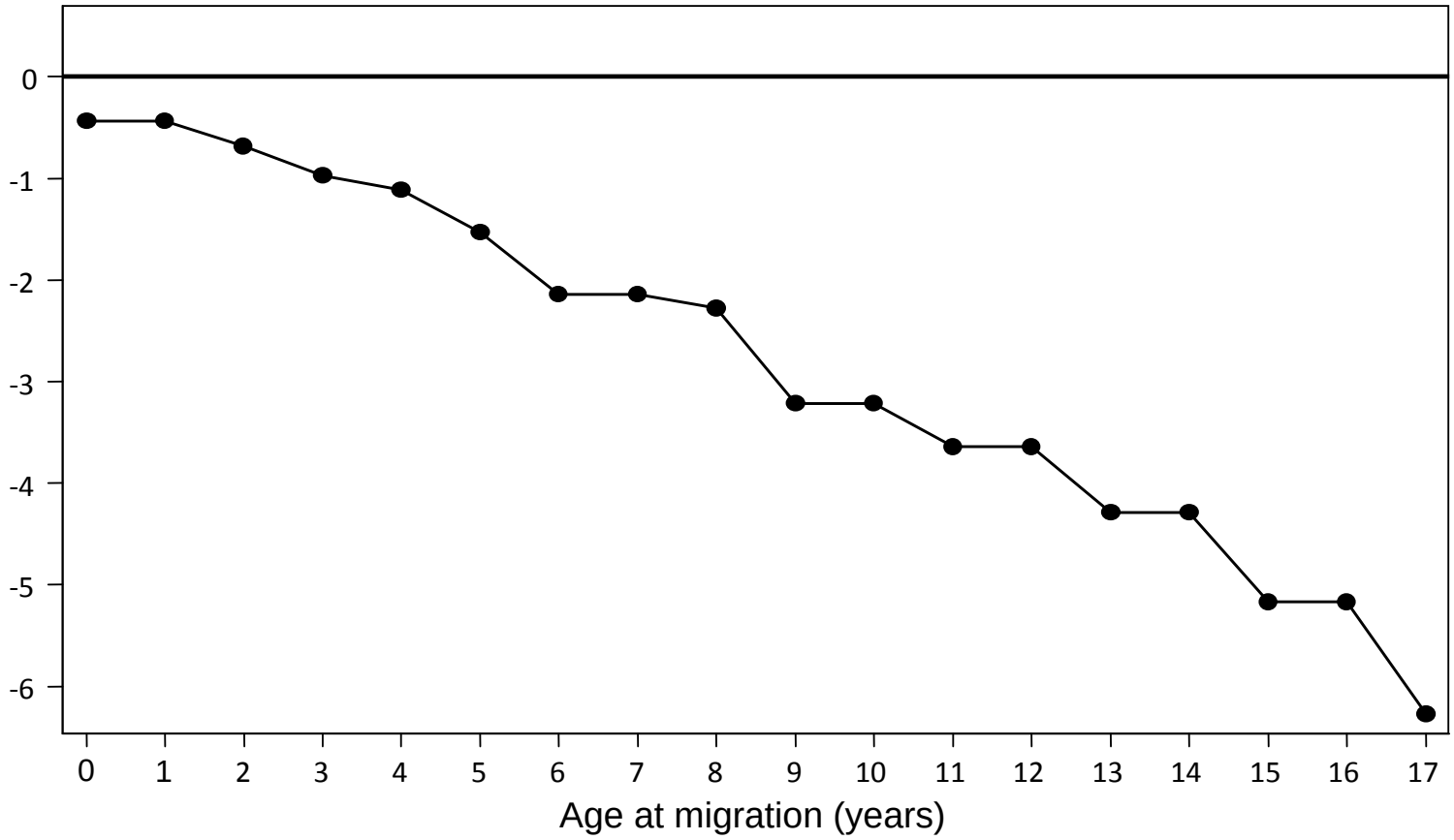
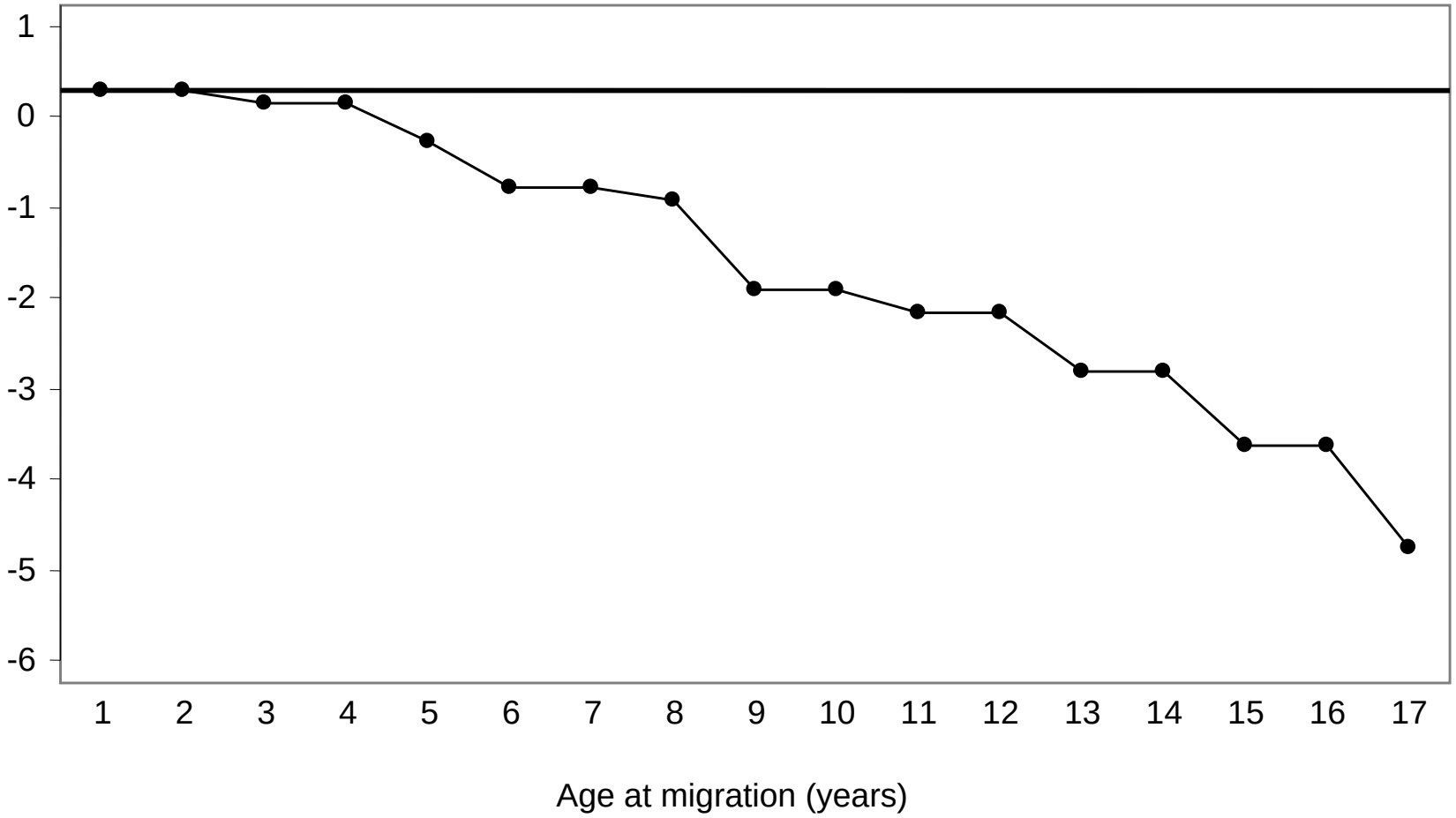


Figure 2. Adult height difference (in cm) relative to immigration at age 0.
FE panel data estimates with monotonicity constraint; Table 4(2).



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