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The Scourge of Asian Flu: *in utero* Exposure to Pandemic Influenza and the Development of a Cohort of British Children

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The Scourge of Asian Flu: *in utero* exposure to pandemic influenza and the development of a cohort of British children*

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Abstract

This paper examines the impact of *in utero* exposure to the Asian influenza pandemic of 1957 upon physical and cognitive development in childhood. Outcome data is provided by the National Child Development Study (NCDS), a panel study of a cohort of British children who were all potentially exposed in the womb. Epidemic effects are identified using geographic variation in a surrogate measure of the epidemic. Results indicate significant detrimental effects of the epidemic upon birth weight and height at 7 and 11, but only for the offspring of mother's with certain health characteristics. By contrast, the impact of the epidemic on childhood cognitive test scores is more general: test scores are reduced at the mean, and effects remain constant across maternal health and socioeconomic indicators. Taken together, our results point to multiple channels linking foetal health shocks to childhood outcomes.

JEL Classification: I12, I29, D13
Keywords: NCDS; Foetal Origins; Birth Weight; Influenza

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1 Introduction

The foundations for life-long health and human capital formation are laid in the womb. As the foetus develops, it is subject to a range of environmental influences, only some of which the mother can control. At the extreme, intrauterine exposures to certain maternal diseases or drugs can lead to substantial physical and mental impairments at birth[1]. But more commonly, episodes or events have impacts that are mild, remain unnoticed, or only emerge in later life. The original Barker or foetal origins (FO) hypothesis proposed a link between foetal nutrition and certain diseases in adulthood (Barker, 1992). However, this definition has subsequently expanded to include a broader range of both potential hazards and relevant outcomes. (Jaddoe & Witteman, 2006)

The FO hypothesis is hard to test, and effects difficult to identify; the substantial scope for omitted variables and non-random selection almost always preclude the use of non-experimental data. As a result, much of the empirical economics literature has used natural experiments to generate sharp, random shocks to foetal health conditions, either across space or cohorts[2]. Examples include disease outbreaks (Almond, 2006; Almond & Mazumder, 2005), *in utero* exposure to radioactive emissions (Almond, Edlund, & Palme, 2008; Otake & Schull, 1998) famine (Almond et al., 2007; Banerjee et al., 2007), fasting (Almond & Mazumder, 2008), and policy induced changes in alcohol consumption (Nilsson, 2008). This approach has successfully established long run effects of foetal health shocks. However, data constraints have often restricted researchers to a limited number of adult outcomes, recorded in repeated cross-sections; it is thus hard to assess the importance of the FO hypothesis for the broader economics literature, or to use results to inform policy.

This paper examines the physical and cognitive development of a cohort of British children, *in utero* during the 1957 Asian influenza pandemic. Effects of exposure on outcomes at birth, 7 and 11 are identified using geographical variation in the intensity of the influenza outbreak. The data provide a unique concurrence of a foetal health shock and a detailed panel of individuals, each of whom were potentially exposed. As such, we can go beyond what is usually possible in the foetal origins literature, and address two sets of questions. First, did Asian influenza have significant effects upon childhood physical and cognitive development? Second, how do any effects change over time, and can we learn anything about the mechanisms behind the estimated relationships? The first of these questions is in accord with the existing foetal origins literature, and could

[1]For example, contracting rubella during the first trimester leads to congenital rubella syndrome, whilst excessive consumption of alcohol can lead to foetal alcohol syndrome.

[2]Omitted variables and selection mechanisms, if they do exist, should not bias estimates, as are they are assumed uncorrelated with the change in foetal conditions

be achieved using a repeated cross-section; the second relies on the structure of the data.

The NCDS an ongoing cohort study that follows all 17,400 individuals born in Great Britain in one week in March 1958. The first wave was conducted at birth, the second at age 7, and the third at age 11[3]. These waves include detailed information on the health and development of the child, the socioeconomic background and health of the parents, and interviews with teachers and schools where applicable. Outcomes at birth are measured by birth weight and gestation. At 7 and 11, the outcomes used are cognitive test scores and height in metres. Asian influenza struck Great Britain between September and November 1957, when the majority of NCDS cohort were in their second trimester[4]. Influenza exposure is measured by official pneumonia notifications per 100,000 population during the epidemic period, in each child's local authority of birth, a surrogate for the local influenza infection rate (Hunter & Young, 1971).

Most existing evidence on the impacts of *in utero* exposure to pandemic influenza comes from the devastating Spanish flu of 1918-19. This literature typically identifies effects by using across-cohort comparisons of adult outcomes. Perhaps the most comprehensive study on Spanish flu is provided by Almond (2006), who exploits information on quarter of birth, documented by the 1960, 1970 and 1980 US censuses. He finds that cohorts exposed during the first or second trimester of pregnancy[5] were significantly less likely to complete high school, had consistently lower earnings, received higher welfare payments, had higher rates of incarceration, and were more likely to experience a work limiting disability than would have been predicted from trend[6]. These results are consistent with medical evidence on the vulnerable periods for cerebral development, and with the remainder of the empirical FO literature (see, for example Otake and Schull (1998); Almond et al. (2008)).

This paper adds to and supports Almonds work, by providing results for outcomes at birth and in early childhood. But, our more substantive and novel contributions stem from the panel structure of the NCDS. Two issues are addressed in detail: first, the extent to which the impacts of the epidemic on childhood outcomes are captured by birth weight; and second, the role of maternal health in mediating the effects of influenza. The principal aim is to gain some information on the mechanisms, which might link foetal health shocks to subsequent outcomes.

There is a large literature that estimates the effects of birth weight upon subsequent physical and cognitive development (e.g., Black et al., 2007; Conley & Bennett, 2000;

[3]There have since been six subsequent followups: in 1974, 1981, 1991, 2000, 2004 and 2008

[4]The epidemic peaked in the week ending the 19th of October 1957, when 90% of cohort members were between 17 and 23 weeks in gestation

[5]Exposed in the fall of 1918, and born in the first two quarters of 1919

[6]Similar patterns are found for health outcomes in the US (Almond & Mazumder, 2005), and for data from Taiwan and Brazil (Jen, 2008; Nelson, 2003)

Currie & Moretti, 2007; Currie & Hyson., 2001). However, a surfeit of potential omitted variables makes the mechanisms very hard to identify. Some studies have used birth weight as a proxy for the quality of the intrauterine environment and parental investment prior to birth (e.g., Conley & Bennett, 2000; Currie & Moretti, 2007). We test whether birth weight does indeed capture any effects of the foetal health shock, Asian flu, upon subsequent developmental outcomes. The role of maternal health is particularly salient for the economic literature, for two reasons: first, a mothers health represent one possible channel for the intergenerational transmission of disadvantage; second, advice for, and policies towards, pregnant women have important implications for the health and productivity of populations in generations to come. The main findings are as follows.

First, Asian influenza had a significant and negative effect on birth outcomes, but only for the offspring of mothers with certain health characteristics. There is no effect of the epidemic upon mean birth weight, intrauterine growth, or gestation. However, interactions between the epidemic and two indicators of maternal health, pre-pregnancy smoking and adult height, show negative effects on birth outcomes confined to particular groups of women: those that smoked before pregnancy, and those 61 inches or shorter. This pattern of heterogeneity is consistent with medical evidence on the possible relationships between influenza and foetal nutrition.

Second, the epidemic had significant effects on mean test scores at 7 and 11: a one standard increase in epidemic intensity reduces test scores by 0.06 standard deviations at 7, and 0.05 at 11. The effects are general, and do not vary with cohort member characteristics, including the maternal health measures and socioeconomic background. Negative effects of influenza upon child height at 7 and 11 are only present for smokers, thus replicating results for birth weight. The differing patterns of results for birth weight and test scores are consistent with two separate transmission mechanisms linking maternal influenza to child outcomes: (1) maternal influenza can reduce birth weight, but only where the symptoms are plausibly very severe, or where the mother's nutritional stores cannot compensate for a temporary interruption in her diet; (2) the inflammation and hyperthermia that always accompany influenza may impair cognitive development, when exposure occurs during the second trimester; good maternal health and nutrition provides little or no protection.

Third, the impacts of the epidemic on childhood height and test scores do not operate through, and are not captured by, birth weight. The association between birth weight an later physical and cognitive outcomes is well documented by the medical and social science literatures, but remains poorly understood[7]. Our results suggest that birth weight

[7]For example Richards et al. (1998), Currie & Hyson (2001), Black et al. (2007), Behrman & Rosenzweig (2004), and Conley & Bennett (2000), find significant relationships between birth weight, cognitive development and educational attainment. See Almond et al. (2005) and Godfrey & Barker (2001) for

should not be viewed as a catchall measure of influences on development prior to birth.

Fourth, maternal smoking plays a critical role in shaping whether influenza affected physical development, from birth onwards. The effect of the epidemic upon birth weight and child height is negative only where mothers smoked prior to pregnancy; the relationship continues when height is measured in adolescence and adulthood. This result is consistent with medical evidence that suggests smoking increases the rate of infection from influenza, induces more severe symptoms, and causes a higher incidence of complications (Arcavi & Benowitz, 2004; Rasmussen et al., 2008). Pregnant women are already warned not to smoke during pregnancy; our results act to further reinforce this message.

The structure of the paper is as follows. Section 2 details the Asian Influenza pandemic and examines the possible links between maternal influenza and foetal health. Section 3 describes the data. Section 4 presents the results for birth weight. Section 5 presents the results for child outcomes at 7 and 11. In section 6, we discuss our results and their implications for government policy and the economics literature. Further results and robustness tests are provided in the appendix.

2 Background

2.1 The Epidemic

The Asian influenza pandemic of 1957-58 was the second of three twentieth century influenza pandemics. It was far milder than its predecessor, the catastrophic Spanish influenza of 1918-19, but claimed more lives than the Hong Kong influenza of 1968-9[8]. Each of these pandemics occurred when a new form of the influenza A virus was introduced into the human population from a non-human - and in general, avian - host.

In non-pandemic years, influenza infection rates fluctuate with the seasons, claiming several hundred thousand of lives worldwide each year. Pandemic influenza spreads in waves, without apparent regard for climate or season, and the death toll usually runs into the millions. Whilst seasonal flu takes the heaviest toll on those under 2 and over 65, victims of pandemic include high numbers of older children and prime aged adults[9].

The risk of infection, the severity of symptoms and death rates from influenza are all elevated during pregnancy. This heightened vulnerability can be attributed to physiolog-

links between birth weight and short run and long run health.

[8]The US Dept. of Health and Human Services estimate that Spanish flu had a worldwide death toll of 40 million, Asian flu 1-2 million and Hong Kong flu 0.7 million (US Department of Health and Human Services, 2008)

[9]In non-pandemic years those under 65 account for between 10 and 20 % of all influenza deaths, as compared 36% during Asian Flu, 48% during Hong Flu and 99% during Spanish Flu (Simonsen et al., 1998)

ical changes in a woman's cardiovascular, respiratory and immune systems (Rasmussen et al., 2008; Goodnight & Soper, 2005). Those with underlying medical conditions, such as asthma, or those with weak respiratory systems due to smoking, are at a particularly high risk (Arcavi & Benowitz, 2004; Rasmussen et al., 2008; Goodnight & Soper, 2005). Influenza rarely crosses the placenta to infect the foetus. Any negative effects on foetal development therefore operate through secondary responses, such as inflammation or an interruption in maternal nutrition (Rasmussen et al., 2008). However, the precise mechanisms remain poorly understood.

Asian influenza hit Great Britain[10] between June 1957 and April 1958, with cases concentrated between September and November, henceforth known as the "epidemic period". In 1957 alone, the epidemic was responsible for 30,000 excess deaths in England and Wales, of which 6,716 were directly from influenza[11]. According to official estimates, at least 7.5 million people in England and Wales (or 17% of the population) suffered some level of incapacitation during the main epidemic period[12]. Cases were concentrated amongst those aged between 4 and 39, with the highest incidence amongst those of school age[13]. There were no significant differences in infection rates by sex. The relationship between weekly sickness benefit figures indicates that the majority of cases lasted between one and two weeks. (Woodall et al., 1958; Ministry of Health, 1960)

Influenza was not a notifiable disease in 1957, so the total incidence can only be estimated using proxy measures. We adopt the approach taken by Hunter and Young (1971), and use official pneumonia notifications made to the Registrar Generals of England & Wales, and of Scotland[14]. Pneumonia is closely clinically related to influenza, an attack of influenza may result in pneumonia, and quarterly trends in pneumonia notifications closely replicate influenza deaths. Figure 1 shows the timing and number of pneumonia notifications made during the epidemic period.

The effect of the epidemic is identified using the spatial variation in pneumonia notifications per hundred thousand population, by local authority of birth. Figure 2 shows the incidence of the epidemic at the more aggregated county level, in England and Wales. The epidemic was more prevalent in the north, but there were significant variations within

[10]England, Scotland and Wales. The United Kingdom officially includes England, Scotland, Wales and Northern Ireland.

[11]Although this total is not negligible, excess mortality during the Spanish Flu stood at 200,000, with 150,000 deaths attributed to influenza.

[12]Official estimates were compiled by piecing together claims for sickness benefits and reports from medical officers around the country (Ministry of Health, 1960)

[13]Infection rates amongst those age 5-14 approached 50%. For women of child-bearing age the infection rate was around 30%, but there no figures for the infection rate amongst pregnant women (Woodall et al., 1958). Deaths from pneumonia amongst women of child-bearing age were ten times higher than the year before, although absolute number remained small at 344.

[14]Scotland has a separate Registrar General and a separate set of Returns. Pneumonia was a notifiable disease until 1968, meaning that doctors were legally obliged to report cases to the relevant authorities

regions. Local authority level data reveals further variation within counties.

As previously explained, the Spanish Influenza pandemic of 1918-19 has supplied the bulk of the evidence on the long run effects of *in utero* exposure to pandemic influenza. Asian influenza was mild in relation to its Spanish antecedent; the identification of any effects may therefore be more difficult. Furthermore, the NCDS does not allow for the across cohort comparisons used by the existing studies, as all subjects in the data were born in the same week. We must instead rely on geographical variation in severity, which will be subject to more measurement error than month or quarter of birth. Where our advantage lies is in the ability to follow the outcomes of the same individual from birth. As such, we can examine the impact of the epidemic upon early outcomes, and study the dynamics and mechanisms behind estimated effects.

2.2 *In Utero* Exposure to Influenza and Child Outcomes - Possible Channels

2.2.1 Maternal health shocks, influenza and birth weight

Birth weight is used by medical, scientific, and social science literatures as a proxy for the quality of conditions for growth and development in the womb (Conley & Bennett, 2000; Currie & Moretti, 2007). It is an object of interest only in so far as it may linked - by cause or correlation - to subsequent outcomes, whether they be health or socioeconomic (Jaddoe & Witteman, 2006, p. 93). There is nothing inherently good about heavier births[15].

Birth weight is determined by two factors: gestation and intrauterine growth. A baby born before 37 weeks is defined as premature, and is at risk of health complications caused by incomplete foetal development[16]. In most cases, the cause of a premature birth is unknown: gestation is unresponsive to policy interventions, such as improving access to healthcare or providing nutritional supplements, and is not that sensitive to maternal behaviour (Kramer, 1987; Frisbie et al., 1997; Goldenberg & Rouse, 1998; Godfrey & Barker, 2001). Some studies have found a relationship between pandemic influenza and premature birth, but this appears to stem from the impact of complications, such as pneumonia, rather than the virus itself (Rasmussen et al., 2008).

Impeded intrauterine growth (IUG), or foetal malnutrition occurs when the birth weight of a child born at term falls below a certain threshold; this, by definition, is caused

[15]Indeed, very high birth weight has been found to increase the risk of breast cancer and to impair cognitive development (Santos et al., 2008; Cesur & Rashad, 2008).

[16]For example, the critical periods are at around 30 weeks for the muscles and 34 weeks for the kidneys. (Barker, 2009)

by insufficient nutrient uptake in the womb[17]. The foetal origins hypothesis postulates that foetal under-nutrition can lead to "reprogramming" or altered gene expression, which has permanent impacts upon an individual's physiology. Consequences include a higher risk of stokes, cardiovascular disease and diabetes in adult life[18]. IUG has proved responsive to policy interventions designed to improve maternal nutrition during pregnancy and is negatively affected by smoking. (Kramer, 1987; Frisbie et al., 1997; Goldenberg & Rouse, 1998; Godfrey & Barker, 2001)

The nutrients a foetus receives from its mother come from two sources: her diet during pregnancy, and the stock of nutrients stored in her liver and the tissues of her body. Barker (2009) claims that it is latter, the mother's nutrient reserves, that are of primary importance for foetal growth; this, he suggests, can be proxied by her height. Nutrients are delivered to the baby through the placenta; the efficiency of these transfers can vary with placental size, which is in turn affected by maternal nutrition.

Maternal influenza could affect foetal nutrition flows in at least three ways. First, through suppressing appetite and reducing nutritional intake; the consequences of which may be more severe in pregnant women, due to biochemical changes in their bodies (Metzger et al., 1982)[19]. Secondly, by increasing excretion rates, interfering with the absorption of fats, proteins, and other essential nutrients. Lastly, by inducing fever, which acts to accelerate energy consumption and nutrient loss. Fever elevates the basal metabolic rate, increasing energy expenditure even in a resting state, with a one centigrade elevation in body temperature associated with a 10% increase in energy, protein and micronutrient requirements. The rate of illness-induced nutritional depletion depends on the characteristics of the mother; in particular, the diminution is more rapid where pre-infection nutrient intake was low (Scrimshaw, 1977, p. 1538). (Tomkins et al., 1994; Edwards, 2007; Scrimshaw, 1977)

2.2.2 Maternal health shocks and brain development

The human brain is more susceptible to tetrogenic insults than most other embryonic and foetal structures; vulnerability is highest between 8 and 25 weeks in gestation, when the brain is developing (Nyagu et al., 2002, p. 202). This is strongly reflected in empirical

[17]The most precise measure uses weight per cubed squared, to take account of variation in the length of babies. Barker (2009) specifies a threshold of 26 kg/metre cubed. However, where such measurements are unavailable, weight is adjusted for gestation. Precise weight thresholds vary from paper to paper (Martorell & Gonzalez-cossio, 1987)

[18]See Jaddoe & Witteman (2006) for hypothesis on the precise mechanisms that link nutrition to foetal reprogramming

[19]Metzger et al. (1982) examine the biochemical profiles of pregnant and non-pregnant women following a 12 hour fast. Pregnant women were found to experience "accelerated starvation", with significantly higher levels of ketones and lower levels of plasma glucose and alanine than non-pregnant controls.

results of the FO literature: intrauterine health shocks, such as influenza or exposure nuclear radiation, do depress cognitive development, but only when experienced during the vulnerable period (Almond, 2006; Otake & Schull, 1998; Almond et al., 2008). The period from 8 to 25 weeks is of particular relevance to this study, as over 99% of the NCDS cohort were at this stage when the epidemic peaked in their local authority birth[20].

Cebrogenesis, "the birth of the brain", takes place over two critical periods. The first occurs between 8-15 weeks, when the proliferation of neuronal (nerve cell) elements hit its peak and there is substantial migration of neurones to different parts of the developing brain. A foetal health shock during this period has the most severe and pronounced effects upon cognitive development (Otake & Schull, 1998).

The second critical period occurs between 16 and 25 weeks in gestation: rapid neurone differentiation sees neurones developing different, and specific, biochemical and physiological properties; synaptogenesis, or the creation of new synapses (the structures that enable the transmission of nerve signals) hits its peak; the architecture of the brain begins to form; and, neuronal pruning takes place, eliminating more than 50% of neurones prior to birth (Otake & Schull, 1998; Nyagu et al., 2002). Some studies (e.g., O'Callaghan et al., 1991) have posited that exposure to influenza at this stage interrupts the neuronal pruning process, increasing the child's susceptibility to schizophrenia in adulthood. Possible causes of this interruption are influenza-induced hyperthermia and inflammation (Rasmussen et al., 2008).

The majority of NCDS intrauterine exposures to Asian influenza will have occurred during the second period of cebrogenesis. The epidemic peaked in the week ending the 19th October, when 95% of the NCDS cohort were between 16 and 25 weeks in gestation. Assuming a two-week infection window either side of the peak implies that a minimum of 80% of exposures would have occurred at between 16 and 25 weeks; a three-week window reduces this to a minimum 60%. The remainder of exposures would have occurred almost exclusively between 8 and 15 weeks[21]. The path of foetal development and empirical evidence from the Spanish influenza pandemic thus suggest that the cognitive development of the NCDS cohort could have been affected by Asian flu.

[20]Even allowing for a two week infection window either side of the peaks, over 90% would have been exposed at this stage

[21]Authors calculations. Minimum number of infections using a two week window are calculated by assuming that a local authority peak at 16 or 17 of gestation means exposure at week 14 or 15, and a peak at week 24 or 25 means exposure at week 26 or 27.

3 Data

3.1 The National Child Development Study

The National Child Development Study (NCDS) is a ongoing longitudinal cohort study, which follows all those born in England, Scotland and Wales between the 3rd and 9th March 1958, a total of around 17,400 births. The public use data files are augmented by local authority identifiers for the first four waves under special license. Great Britain is divided into 172 local authorities; populations range from 18,400 (Shetland) to 5.5 million (London), with a median of 134,000. Observations are dropped entirely if birth weight is missing, reducing the total sample to 16,765[22].

The NCDS began with the Perinatal Mortality Survey of 1958 (PMS), which was designed to examine the social and obstetric factors associated with stillbirth and death in early infancy. There have been eight subsequent followups, the most recent of which was completed in 2008[23]. We use data from the first three waves, collected in 1958, 1965 and 1969 when the cohort members were 0, 7 and 11. These waves contain detailed information on both parents and the children. Parental data includes health status, behaviour, anthropometric measures and socioeconomic background variables. Cohort member data comprises of a rich set of health outcomes and anthropometric measurements, cognitive test scores and teacher behavioural and aptitude assessments. Participation fell from 16,765 in 1958 to 14,358 in 1965 and 14,069 in 1969[24].

3.2 The Epidemic

In absence of mother specific information on infection or exposure, the epidemic is measured at the finest level of geographical aggregation possible given the data: the local authority of birth[25]

[22]Those with missing birth weight (the"BW missin" group) did not die, as they are present in later waves, and are spread across 124 of the 172 local authorities. In the 1958 survey, this group has a higher rate of non-response to other survey questions. However, where background characteristics are recorded, they are not significantly different from the rest of the sample. In the 1965 and 1969 surveys, responses and non-responses of the BW missing group do not differ from the rest of the sample. The propensity to provide incomplete information therefore appears specific to the 1958 survey, not to the cohort members in question.

[23]In an effort to ensure that the sample remained representative, the cohort was augmented with immigrants born in the relevant week in waves 1 to 3. These observations are not included in the sample, as they were not exposed to Asian Influenza at the same time as the British- born cohort

[24]This attrition was not additive: 15265 participated in at least one of the 1965 and 1969 waves. The birth weight of those that did not participate in each test was on average lower than those that did. However, the differences are not significant

[25]As part of the PMS, the mothers were asked whether they had certain illnesses when pregnant, including influenza. This data does not appear in the files held by the UK data archive or the Centre for Longitudinal Studies.

The epidemic is measured by the ratio of total number of pneumonia notifications during the epidemic period (September-November 1957) to population in local authority of birth[26]. This acts as a measure for the likelihood that the cohort child's mother was infected by influenza, or indirectly affected by the infection rate in the local area. The epidemic peaked in the week ending 19th of October, when 90% of the NCDS children were between 17 and 23 weeks in gestation. Pneumonia notifications are given at a local authority level, by week and by quarter, in the Registrar General's Returns for England and Wales, and in the corresponding Returns for Scotland[27]. Local authority population estimates are provided by the same source, as of 1st June 1957. Deaths from pneumonia or influenza are not given by week or quarter until 1958.

Further figures are collected on the number of pneumonia notifications in the same period in 1955 and 1956. Totals from these two years are averaged and divided by local authority population estimates for 1956. Pre-epidemic intensity captures the underlying level of non-pandemic pneumonia in each local authority. Other local authority control variables come from the 1956 Registrar Generals' Returns and the 1951 census. The descriptive statistics of all epidemic measures and local authority controls are described in Table 1.

Asian influenza increased the mean, median, and interquartile range of local authority pneumonia notifications by a factor of 4. As indicated in Table 1, there was an average of 39.7 pneumonia notifications per 100,000 population during the epidemic period; this compares to an underlying rate of just 9.64[28]. There is a high degree of correlation be-

[26]The assumptions made when using pneumonia notifications to proxy for influenza infectious rates are as follows:

1. Physicians diagnoses were correct.
2. Reportage was complete.
3. Errors would occur randomly if some diagnoses were erroneous or some reportage were incorrect, and thus relative differences between local authorities would not be invalidated.
4. Acute pneumonia is clinically associated with influenza in an acceptable parameter relationship. (Hunter & Young, 1971) propose that the ratio of pneumonia notifications to influenza infections is 1 to 417.
5. Subclinical cases of influenza need not be taken into account (Hunter & Young, 1971, p. 642)
6. Any difference between local authority of birth and local authority of exposure is random with respect to the epidemic.

If assumptions 1 and 2 fail to hold, but measurement error is uncorrelated to local authority variation in cohort member outcomes, the estimated effect of the epidemic will be attenuated towards zero.

[27]See England and Wales. Office of Population Censuses and Surveys (1957); England and Wales. Registrar General (1957) for citation

[28]The median and interquartile range of *Epidemic* are 29.1 and 29.3, as compared to 7.24 and 7.17 for *PreEpidemic*. Both *Epidemic* and *Pre-Epidemic* have large standard deviations (36.7 and 10.4). Glasgow is an outlier in both *Epidemic* and *Pre-Epidemic*. Dundee is an outlier in the *Epidemic* measure, but *Pre-Epidemic* value falls within the normal range

tween *Epidemic* and *PreEpidemic* (0.68), in part because *Epidemic* will include those who would have contracted pneumonia in absence of the epidemic. However, the correspondence between *PreEpidemic* pneumonia levels and the ratio of *Epidemic* to *PreEpidemic* is low (0.2). Areas with a high underlying rate of pneumonia therefore did not experience proportionately more notifications during the epidemic period

As might be expected, the epidemic was more prevalent in densely populated local authorities, where households live in cramped conditions, where the average socioeconomic status was low, and where the underlying health of the population was relatively poor. The highest correlate is the proportion of households living in crowded conditions, at 0.47. To ensure that results are not biased by unobserved local authority characteristics, all variables in Table 1 will be used as controls in all but the most descriptive specifications.

3.3 Birth Weight

Birth weight is the earliest outcome measure and is used for two purposes: first, as an indicator for whether the effects of the epidemic can be detected at birth; second, to assess whether outcomes measured at birth do capture the effects of foetal health shocks on subsequent development. If the effects of the epidemic operate through, or are captured by, birth weight, the same individuals should experience both depressed birth weight and reduced cognitive test scores. Birth weight is measured in ounces, with a mean of 116.2 (7.3 lbs or 3.3kg) and a standard deviation of 20.4 (1.27lbs or 0.58kg).

As previously discussed, birth weight is determined by gestation and intrauterine growth. Gestation in days is defined for 89.5% of the sample, distributed with a mean of 280 and a standard deviation of 14. Intra-uterine growth can be calculated by adjusting birth weight for gestation[29].

3.4 Outcomes at 7 and 11

In both 1965 and 1969, the NCDS cohort were set a series of cognitive tests,. The tests we use are Draw a Man Score aged 7 and the General Test Score (non-verbal) taken at age 11. These tests are chosen for having a larger range than other tests at the same ages, and distributions which are approximately normal. However, the tests are not of the same form; there is therefore no way to distinguish changes in performance over time from differences in the test[30].

[29]Those with missing gestation have a mean birth weight of 114.4 oz and a standard deviation of 21.4; significantly lower than for the rest of the sample. Parents of these cohort members are, on average, less educated and of a lower social class.

[30]The NCDS does administer reading and maths tests in all three waves, but the distribution of results differ substantially from test to test and rarely approximate the normal. The non-verbal General Test has a verbal counterpart, but the scores are very similar and results do not change. Of those who

For the Draw a Man test, children were asked to draw a man, and were graded on detail and accuracy. The General Test was designed to test general ability through verbal and non-verbal reasoning. The absolute test scores have no economic interpretation and are thus normalised, so as to be distributed with mean zero and standard deviation 1. Child heights, in meters, are measured in the same years as the tests and provide a comparison between physical and cognitive development.

4 Birthweight

4.1 Empirical Method

The effect of the epidemic on the birth weight of child i, in local authority l, is estimated using the following linear specification:

$$BW_{il} = \alpha + \beta Epidemic_l + \theta PreEpidemic_l + \gamma^T \mathbf{LA_l} + \chi^T \mathbf{X_{il}} + \varepsilon_{il} \quad (1)$$

where $Epidemic_l$ represents the number of pneumonia notifications per hundred thousand population in the child's local authority of birth; $PreEpidemic_l$, the same measure in the two years previous; $\boldsymbol{LA}_l$, local authority characteristics; and, $\boldsymbol{X}_{il}$ child-level characteristics at birth. The error term, ε_{il}, is assumed to be conditionally uncorrelated with $Epidemic_l$, and is clustered at the local authority level. Tables will include both estimated effect, $\hat{\beta}$, and the implied effect size, $\hat{\beta}[\frac{sd(Epidemic_l)}{sd(BW_{il})}]$[31]. The baseline specification only considers the effects of the epidemic at the mean; interactions between $Epidemic_l$ and maternal characteristics are subsequently added to allow for heterogeneity across cohort members.

Local authority controls, vector $\boldsymbol{LA}_l$ and $PreEpidemic_l$, are vital to ensure that $\hat{\beta}$ is not simply reflecting the correlation between the intensity of the epidemic and local authority characteristics associated with poor birth outcomes. The controls, described in Table 1, attempt to capture features of the local environment or population that could increase the rate of infection or make symptoms more severe. $PreEpidemic_l$ controls for the underlying rate of pneumonia in the area, so that $\hat{\beta}$ estimates the effect of the excess notifications during the epidemic period.

The vector $\boldsymbol{X}_{il}$ represents a rich set of parental and background controls for each child, including the social class and schooling of both parents, mother's age and its square,

participated in the 1965 wave, 95% took the Draw A Man test. Of those who participated in the 1969 wave, 92% took the General Test.

[31] This signifies the effect of a one standard deviation increase in $Epidemic_l$ on birth weight, in standard deviations, and will be used to gauge the magnitudes of the estimated effects and to make comparisons with the existing literature.

the mother's height, the tenure of accommodation and number of persons per room, and whether the mother smoked before becoming pregnant[32]. These characteristics are assumed to be predetermined, and thus unaffected by the epidemic; but, could plausibly influence the probability of being infected. Whereas $\boldsymbol{LA}_l$ captures the characteristics of the child's area, $\boldsymbol{X}_{il}$ captures the characteristics of the children in the NCDS cohort in that area. Child-level information will later be exploited to heterogeneous effects across parental characteristics.

The coefficients estimated in (1) should represent a lower bound of the true effects of Asian flu. $Epidemic_l$ is measured at a local authority level; the estimated coefficients are thus a weighted average of the children of mothers who were affected by the epidemic and those who were not. In the foetal origins literature, it is common to approximate effects on treated individuals by multiplying estimates by the inverse of the population exposure rate (e.g., Almond, 2006; Banerjee et al., 2007). However, there is no reliable information on Asian influenza infection rates amongst pregnant women[33]. Moreover, any adjustment factor would not account for heterogeneity across mothers, or any spillovers upon those not infected. Estimates are therefore left unadjusted.

4.2 Results

4.2.1 Baseline Results and Heterogeneity

Table 2 presents the estimated impacts of Asian influenza upon birth outcomes. The first two columns consider the effect of the epidemic upon mean birth weight, in ounces. In column 1, which includes a full set of local authority controls, $\hat{\beta}$ is negative and significant at the 1% level. However, when child level controls are introduced, in column 2, the coefficient is halved and is no longer significant. A large proportion of this change can be explained by the introduction of a control for maternal height. The pattern is the same for both gestation and IUG[34].

The epidemic, as measured by our local authority proxy, has no effect on mean birth weight. However, this summary statistic could mask significant and important heterogeneities in impacts, across groups or categories of cohort members. Holding the local authority epidemic constant, such heterogeneity could arise for two reasons: differences in the rates of infection of pregnant women; or, variation in the severity of the symptoms

[32]The summary statistics and details of these variables are contained in the appendix, Table 6

[33]The Ministry of Health (1960) estimate that a third of women of child-bearing age contracted flu during the epidemic period. However, as detailed in section 2, infection rates amongst pregnant women are typically higher than amongst non-pregnant women of the same age

[34]There is no statistical difference between the mean birth weight of the NCDS cohort, and that of the British Cohort Study, born 12 years later, a result consistent with an absence of an effect of the epidemic on mean birth weight.

and the detrimental effects on foetal development. In general, it is not possible to distinguish between the two, and it is not the aim of this paper to do so. The intention is instead to assess whether Asian influenza had an effect on the birth weight of any groups of cohort members, and to evaluate whether any patterns are consistent with the evidence on foetal malnutrition presented in section 2.2.1.

A priori, two sets of characteristics appear most likely to mediate the effect of the epidemic upon birth weight: the cohort members socioeconomic background, which captures the level of resources available to the parents; and the mothers health, which is critical to the quality of the intrauterine environment. To test for heterogeneity across these characteristics, $Epidemic_l$ is interacted with sets of child-level indicators for each. Results point to the primary importance of maternal health, which provides the focus for the last 4 columns of Table 2.

Columns 3 to 5 show the effect of the epidemic interacted with two indicators for mothers health: her height and pre-pregnancy smoking behavior. Both of these characteristics are pre-determined, and are thus left unaltered by the epidemic. Maternal height can be used as at proxy for life long nutrition, and thus the resources from which the foetus can draw upon (Barker, 2009). Smoking is an established cause of restricted IUG, and all pregnant women are advised not to partake (National Health Service, 2009). In column 3, the dependent variable is birth weight in ounces. In columns 4 and 5, birth weight is broken down into its constituent parts: intrauterine growth and gestation.

The results in column 3 indicate that Asian influenza did significantly reduce birth weight, but only for those born to certain categories of mothers: in particular, women who were short (60 inches or 152 cm and under) or who smoked prior to pregnancy[35]. For short mothers the implied effect of the epidemic was to reduce birth weight by 0.04 standard deviations, relative to children of mothers 65-66 inches tall. The implied effects on birth weight when mothers smoked prior to pregnancy were -0.03 for heavy smokers and -0.02 for light smokers. Both sets of interaction coefficients are monotone, with the absolute magnitude of effects falling as cigarette consumption decreases and height increases. These are precisely the relationships predicted by the medical evidence presented in section 2.2.1. Taken together, all epidemic measures are jointly significant at the 1% level.

Column 4 controls for gestation, so that the dependent variable now represents intrauterine growth. The epidemic measures remain jointly significant at the 1% level. However, the negative effect of the epidemic upon birth weight is confined just to the children of heavy smokers. The effect of the epidemic varies with maternal height at the 6% level,

[35]Approximately 11% percent of the NCDS mothers were 60 inches tall or less. Forty percent of mothers smoked prior to pregnancy, evenly divided between heavy and light smokers

but no individual height category is significant. In column 5, the effect of the epidemic upon gestation varies significantly with maternal height, but not with maternal smoking. For a cohort member whose mother was 61 inches or shorter, a one standard deviation in the epidemic reduces gestation by 0.8 days or 0.06 standard deviations, relative to a mother 65-66 inches tall.[36]

There is further variation in epidemic effects by maternal blood pressure and maternal weight: birth weight is reduced where mothers weighed 8 stones or less in 1958, or suffered from preeclampsia during pregnancy. These variables cannot be assumed as exogenous from the epidemic; results are thus harder to interpret and therefore not shown. However, the estimates are consistent with the pattern of significant negative epidemic effects when higher infections rates were plausibly higher, or where there is evidence that symptoms could have been more severe.

In contrast to maternal health, there is no significant interaction between the epidemic and socioeconomic variables, such as mother's education or father's social class. Equally the effect of the epidemic does not vary other local authority characteristics, such as population density, or with the cohort member's gestation. Block bootstrapped quantile regressions indicate that there is no quantile of the birth weight distribution where the effects of the epidemic is significant at the 5% level.

4.2.2 Robustness

The controls in columns 1 to 5 limit any bias that could arise from underlying, pre-existing, local authority ($\boldsymbol{LA}_l$) or cohort member background characteristics ($\boldsymbol{X}_{il}$). However, at least two potential sources of bias still remain. First, the epidemic could have altered cohort composition in a way that is non-random way, through increasing rates of stillbirth and abortion or changing gestation patterns. In the appendix, we examine aggregate evidence on the number of live births, the still birth rate, and the number of maternal deaths, and conclude that any substantive change in cohort composition is highly improbable. Second, all other influences on foetal health that occurred during the cohort's gestation period (May 1957- March 1958) are left unobserved, and would enter (1) through ε_{il}; our results could be biased or spurious if such shocks were correlated with $Epidemic_l$. The possible role of contemporaneous shocks is considered in the last column of Table 2

Column 6 adds two further sets of local authority variables to baseline specification

[36]The results in columns 4 and 5 are not entirely comparable to those in column 3, as the sample has changed due the missing values for gestation. When the specification in column (3) includes only those with non-missing gestation, the effect of the epidemic varies with height, but no interaction is individually significant

(1): region of birth fixed effects[37], and (log) distance from Windscale. The introduction of region of birth fixed effects acts to control for geographical variation in foetal conditions, both before and during pregnancy. The estimated effects of Asian flu now reflect intra-regional variation in $Epidemic_l$; any biasing contemporaneous shock would thus have to be correlated locally rather than on more broad geographical measures. Distance from Windscale is a direct measure of potential exposure to an alternative contemporaneous shock. On the 10th of October 1957, at the peak of the influenza epidemic, a fire started at the Windscale nuclear plutonium factory in the far north west of England. A significant quantity of radioactive material was emitted before the fire was extinguished on the 12th of October, with Iodine-131 and Plutonium-210 the main particles of concern[38]. The new controls do not change the estimated effect shown in column 3, and the Windscale measure is not significant.

5 Outcomes at 7 and 11

A child's endowments at birth - its health and its ability to learn - are a function of the complex interplay of genetics, the intrauterine environment, and parental investments made prior to birth (Heckman, 2007). How health and human capital then evolve depends upon the interaction between these endowments, the child's environment, and the sequence of subsequent investments. Two sets of questions have received considerable attention: first, how initial endowments, often measured by birth weight, translate into subsequent outcomes (Currie & Hyson., 2001; Black et al., 2007); second, whether parents compensate or accentuate initial differences (Heckman, 2007; Becker, 1962; Becker & Tomes, 1976; Cunha et al., 2005). The critical difficulty is that initial endowments are an unobservable function of a series of unobservables: genetics, the foetal environment and prenatal investments.

This section examines the impact of Asian flu on height and test scores at 7 and 11. As in section 4, we first establish whether significant effects exist and test for heterogeneity by

[37]there are 11 regions of birth, with a minimum of 10, and a maximum of 32 local authorities per region. The median number of local authorities per region is 14

[38]The most up to date estimates for emissions are as follows: Iodine-131, approximately 1800TBq, with a range of uncertainty of 900-3700TBq; Caesium-137, 90-350TBq; and, Plutonium-210, 42Tbq with a range of 14-110TBq (Garland & Wakeford, 2007). Initial public health concerns focused upon Iodine-131. A milk ban was put in place in the immediate aftermath of the fire, covering 200 square miles. The concern was that the consumption of contaminated milk could lead to thyroid cancer. By November 23rd, the ban had been completely lifted. The incident was rated as a level 5 accident on the International Nuclear Event Scale and was considered as the world's worst nuclear accident until 3 Mile Island in 1979. However, emissions of Iodine-131 were 1000 times less than at Chernobyl some 30 years later. Even the most up to date estimates place an upper bound on cumulative fatalities of 200, tiny in comparison to an annual death toll from cancer in the UK of 100,000 plus. The carcinogenic impact of Plutonium-210, is now thought more important than that of I-131(Arnold, 1995)

maternal health. We then address two issues pertinent for the human capital and health economics literature: whether the impact of influenza upon child outcomes operates through birth weight; and, the role played by parental investment in mediating the long-run effects of the foetal health shock.

5.1 Empirical Method

The baseline specification for estimating the effect of the epidemic upon childhood outcomes, in this example test scores at 7, is as follows:

$$TestScr7_{il} = \alpha + \beta_1 Epidemic_l + \theta_2 PreEpidemic_l + \gamma_1^T \mathbf{LA_l} + \chi_1^T \mathbf{X_{il}} + \phi_1^T \mathbf{P7_{il}} + \epsilon_{1il} \quad (2)$$

where,

$$\epsilon_{1il} = \tau_{\mathbf{il}}^{\mathbf{T}} \mathbf{g_{il}} + \omega_{\mathbf{il}}^{\mathbf{T}} \mathbf{q_{il}} + e_l + e_i \quad (3)$$

Test scores of child i, in local authority l, are determined by genetic endowments and parental investments since conception. Neither will be perfectly observed, so are proxied by observable parental characteristics, $\boldsymbol{X}_{il}$[39], and observable childhood investments up to the age of 7, $\boldsymbol{P7}_{il}$[40]. The error term, ϵ_{1il}, can be decomposed into unobserved genetic and environmental factors, $\mathbf{g}_{il}$, unobserved parental investments, $\boldsymbol{q}_{il}$, and local authority and individual idiosyncratic error terms, e_l and e_i.

The $\hat{\beta}_1$ coefficient estimates whether the epidemic had an effect upon average child outcomes, but provides very limited information on the impact of influenza on an individual cohort member. As discussed in section 4.1, $Epidemic_l$ is a local authority proxy for the prevalence of Asian flu; $\hat{\beta}_1$ thus underestimates the effects upon those directly exposed, by a factor that is unknown. In addition, any impact of $Epidemic_l$ on childhood outcomes could operate through a number of different channels, which (2) cannot untangle. There may be a direct negative effects upon health and human capital endowments. However, the epidemic could also alter the quantity or efficiency of observed and unobserved parental investment, which would generate a correlation between $Epidemic_l$ and $\boldsymbol{q}_{il}$ or τ_{il}.

The role of birth weight in explaining or capturing the effect of the epidemic on childhood outcomes, can be examined by comparing (2) to the following specification, which adds a birth weight control:

[39] In theory, the relevant observable characteristics, $\boldsymbol{X}_{il}$ in the birth weight equation will not be the same as in the test score equation. However, in this case the vector of observable characteristics are always measured at baseline; contemporaneous test score background characteristics could have been affected by the epidemic, and baseline characteristics could affect both birth weight and test scores

[40] At age 11, we include parental investment measures at both 7 and 11.

$$TestScr7_{il} = \alpha + \beta_2 Epidemic_l + \mu_2 BirthWeight_{il} + \theta_2 PreEpidemic_l + \gamma_2^T \mathbf{LA_l} + \chi_2^T \mathbf{X_{il}} + \phi_2^T \mathbf{P7_{il}} + \epsilon_{2il} \quad (4)$$

The addition of $BirthWeight_{il}$ will affect $\hat{\beta}_2$ to the extent that birth weight was a source of correlation between $Epidemic_l$ and ϵ_{1il}. Child outcomes are, as an empirical regularity, a positive function of birth weight, and the epidemic on the birth weight of certain cohort members: $\hat{\beta}_2$ should be thus be (weakly) less than $\hat{\beta}_1$. If any effect of the epidemic operates entirely through birth weight, $\hat{\beta}_2$ should be zero. In subsequent specifications, (2) and (4) are augmented with interactions between $Epidemic_l$ and maternal height and smoking. The aim is to assess possible heterogeneity by maternal health, and to examine the role of birth weight for those groups identified as affected in section 4.

5.2 Results

Table 3 shows the effects of the birth weight and the epidemic upon test scores of cohort members, aged 7 and 11. The "baseline" columns correspond to (2), and the "+ BW" columns to (4). The "+ interactions" and "+ interactions & BW" columns augment (2) and (4) with dummies for mother's height and pre-pregnancy smoking. Gestation is not included, as has no effect upon test scores and contains a number of missing observations; $BirthWeight_{il}$ thus represents an aggregate of gestation and intrauterine growth.

In contrast to the results for birth weight, the epidemic has a significant negative effect on the mean of both test scores. The "baseline" specifications indicate implied effects of 0.07 at 7 and -0.05 at 11, significant at the 1% and 5% levels respectively. These effects remain unchanged in the "+BW" columns. The impact of the epidemic upon mean test scores therefore does not operate through birth weight[41]. Quantile regressions reveal that the epidemic acted on the middle and upper parts of the conditional test score distributions: the $Epidemic_l$ coefficients are significant between quantiles 0.55 and 0.95 at age 7 and 0.36 and 0.7 at age 11[42]. Birth weight itself has a positive and significant effect on both test scores, with an implied effect of 0.08 for both tests; this replicates the findings of previous papers to have used the NCDS (Currie & Hyson., 2001).

The "+ interactions" and "+ interactions & BW" columns evaluate the role of maternal health in mediating the effects of the epidemic upon tests scores. In section 4, maternal height and pre-pregnancy smoking were critical in determining the effects of the epidemic on birth weight. However, this pattern is not replicated in the results for

[41] Equally, there is no significant interaction between birth weight and $Epidemic_l$

[42] Graphs of quantile regression coefficients are contained in the appendix, Figure 4

test scores: 7 of the 8 interaction terms in Table 3 are not significant and have large ρ-values, whilst the joint significance of the maternal height at 7 appears spurious[43]. For both test scores, results are invariant to the inclusion of birth weight as a control. The effect of the epidemic therefore does not operate through birth weight, even for groups identified as affected in section 4. Thus, whilst the effects of the epidemic on birth weight are specific to certain cohort members, impacts on test scores are generalized: not contingent on maternal health characteristics, independent of birth weight, and significant at the mean and upper portions of the conditional test score distribution.

The striking differences in the patterns of results for test scores, in Table 3, and birth weight, in Table 2, are consistent with two counterfactual explanations (i) that the foetal health shock impaired cognitive development of some cohort members without affecting observable physical development (ii) that the foetal health shock had a latent effect, which only became apparent as the cohort aged. In Table 4, we assess the impact of the epidemic on a key measure of childhood development, child height. The specifications in columns 1 and 3 correspond to (4), but with child height in meters as the dependent variable. Columns 2 and 4 add interactions between $Epidemic_l$ and the two maternal health characteristics.

The effects of $Epidemic_l$ upon mean height are very small and not significant: results consistent with (i), but not with (ii). However, interactions added in columns 2 and 4 indicate significant negative impacts where the mother smoked prior to pregnancy. The same relationship continues when height is measured at 16, and in adulthood. Our results thus suggest that the combination of influenza and smoking can have lasting deleterious effects on physical development. However, the effects on height do not appear to operate through birth weight, as results are invariant to using $BirthWeight_{il}$ as a control.

The differing patterns of results for physical and cognitive development appear consistent with two separate channels of transmission. For physical development, the effect is negative only where the mother's nutritional stores are insufficient to compensate for temporary disruptions in her dietary intake, or where symptoms were plausibly severe. The relationship between influenza and cognitive development is different, and is thought to stem from inflammation: a general symptom of illness, affecting everyone with flu. The ongoing swine flu pandemic has demonstrated that influenza can swiftly sweep through populations of healthy adults and school age children. The heterogeneity in epidemic effects seen for birth weight therefore appears more attributable to differences how a mother's body responds to influenza, rather than to disproportionately higher infection rates. If this is the case, there is no reason to expect that our results for the effects of Asian flu on birth weight and cognitive development should be linked.

[43]No interaction coefficient is individually significant and the coefficients are not monotone

5.3 Possible Mechanisms

Asian influenza had significant negative effects upon mean test scores at both 7 and 11, which does not vary with maternal health indicators. However, outcomes at 7 and 11 are a function not just of endowments at birth, but on their interaction with the subsequent series of investments made by parents, carers and schools. If parents are able to observe the effects of a foetal health shock, how they respond could influence the degree and nature of effects of subsequent effects. (Heckman, 2007; Becker & Tomes, 1976).

To test the role of parents in mediating the long run effects of the foetal health shock, (2) is augmented with interactions between $Epidemic_l$ and proxies for parental investment. Father's social class and mother's education proxy for parental resources; contemporaneous teacher reports on parental interest in cohort members education act as surrogates for human capital investment, with resources held constant. If parents are able to compensate, the effects of the epidemic should be lower for parents who invest more in their children. The opposite would be true if the epidemic affected the human capital production function, making investments less effective. However, results suggest that neither case holds: there is no consistent pattern of heterogeneity for any of the parental investment measures[44]. This finding reinforces the key result from Table 3, that the effect of Asian influenza on cognitive development was general, and invariant to maternal health and cohort member background characteristics. The independent effects of socioeconomic status upon childhood outcomes are in keeping with Currie and Hyson. (2001), who find that the impacts of birth weight and measures of SES on NCDS academic achievements are quite separate from one another.

The measures of parental investment we use may well be incomplete or measured with error, so that estimated effects are attenuated towards zero. A role for unobserved investment in shaping the long run effects of intrauterine influenzal exposure, is thus impossible to rule out. However, it does seem that any behavioral responses were not overwhelming. One explanation is that impact of epidemic were not observable. Epidemic effects were not manifest in childhood physical characteristics, and are driven by the middle and upper portions of the conditional test score distributions. Whether parents observe or respond to small differences in child endowments within this range is debatable.

6 Discussion

Results in sections 4 and 5 indicate that Asian influenza did have a significant effect on the childhood outcomes of the NCDS cohort. However, the patterns of results for

[44]The estimated $Epidemic_l$ coefficients remain identical to those in Table 3, and any heterogeneity detected is inconsistent, either within or between tests.

physical and cognitive development are very different. The impacts of influenza upon physical development, at birth and in childhood, appear contingent upon certain health characteristics: the epidemic reduces birth weight where mothers are short or smoked prior to pregnancy; child height is depressed when mothers smoked. By contrast, the estimated effects of the epidemic upon test scores are general: present at the mean and middle to upper parts of the conditional test score distribution, and invariant to birth weight, maternal health, socioeconomic status or parental investment. The key implications of our results are as follows.

First, the effects of Asian flu appear to operate through two distinct channels. Physical development growth is impeded where mothers are unable to compensate for interruptions in nutrition, or when symptoms were plausibly more severe. The effects on cognitive development are more general, and may be related to the inflammation that always accompanies an influenza infection. A given foetal health shock therefore does not necessarily affect all those exposed identically. Individual treatment effects, recovered by multiplying estimated coefficients by the cohort exposure rate, should thus be viewed and interpreted with caution.

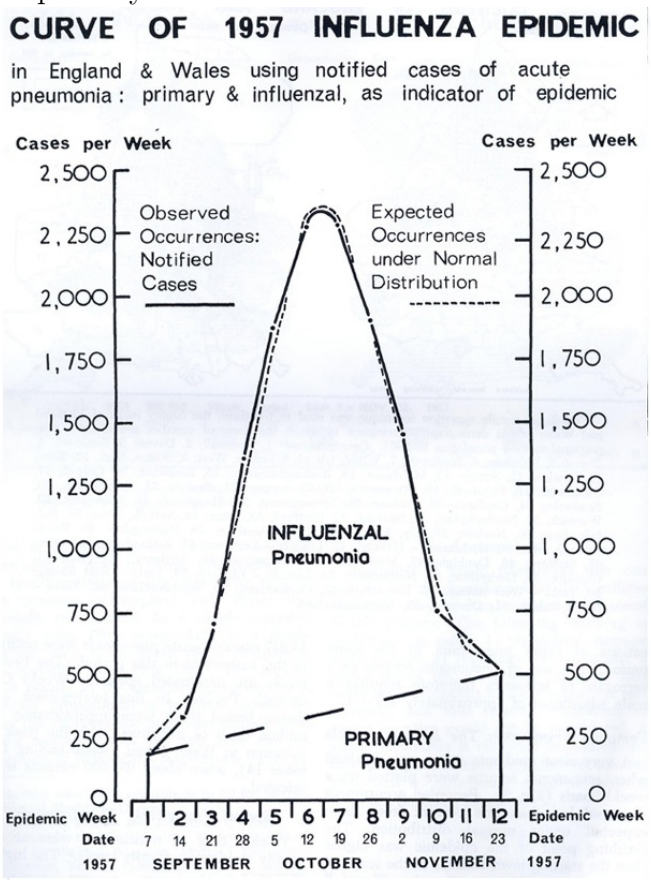
Second, birth weight does not capture the effect of the epidemic upon child height or test scores. Care should therefore be taken when interpreting estimates of "birth weight" effects. A childs weight at birth, particularly below 2.5kg, remains an important correlate with later life outcomes. However, it does not represent a catch-all measure of all influences on health and development prior to birth. The precise mechanisms behind the relationship between birth weight and subsequent child development remains poorly understood, and are a matter for the medical profession.

Third, maternal smoking plays the most consistent and robust role in mediating the effect of the epidemic upon physical growth. The negative interaction between smoking and influenza emerges at birth, and continues into adulthood. Whether these results are attributable to smoking during pregnancy or to the damage caused by previous smoking behavior, is not possible to determine using the NCDS data. Due to the adverse effects upon intrauterine growth, pregnant women are already urged to qui1t smoking. Given the ongoing swine flu epidemic, our results perhaps provide one more reason to give up. Women who are small and light might also consider maintaining or increasing their dietary intake, if they are pregnant or trying to conceive.

Influenza pandemics have occurred three times per century since the 17th century (World Health Organisation, 2009). It is yet unclear whether the measures taken by national governments and the WHO will succeed in limiting infection and death rates from swine flu, and any subsequent pandemics. The British government has announced that pregnant women are to be amongst the first to receive the swine flu vaccine, once it

becomes available. Our results only serve to reinforce the prudence of this measure for the health of both mothers and babies.

Figure 1: The Curve of the 1957 Influenza Epidemic in England and Wales: notified cases of acute pneumonia - primary and influenzal



Source: Taken from Hunter &Young (1971) Fig 2 p. 640.. Epidemic curve of acute pneumonia, England and Wales 1957. Twelve weeks are indicated. Pneumonia is taken as a surrogate of influenza in the ratio of of approximately 1:417

Figure 2: The Spatial Timing and Incidence of the Epidemic

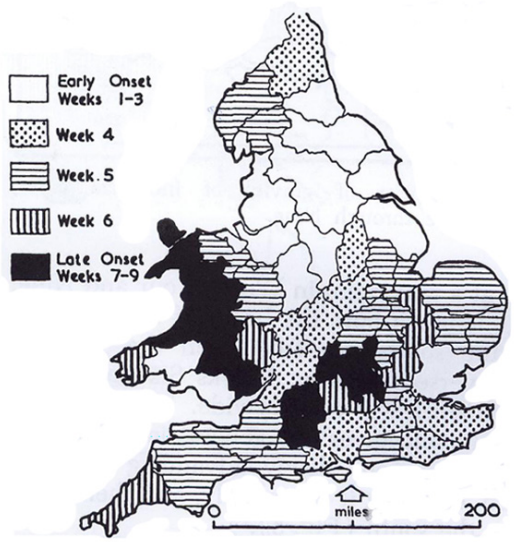


Figure A: Spatial Timing of the Epidemic

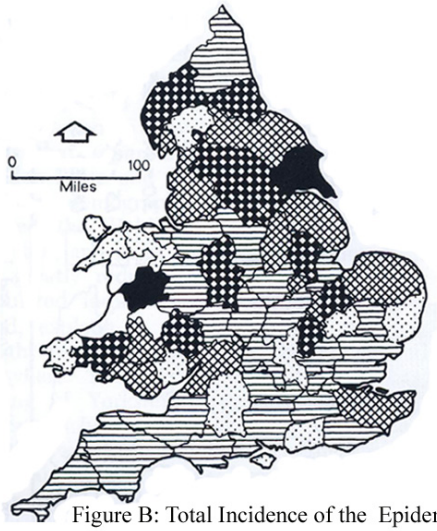
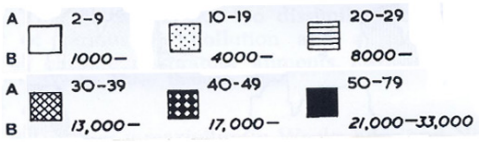


Figure B: Total Incidence of the Epidemic



A- Pneumonia notifications per 100,000 population
B - Estimates of influenza infection rates per 100,000

Notes: Figure A reproduces Figure 7, Onset by Areas, Hunter & Young (1971) p.645. Weeks as described by Figure 1. Figure B reproduces Fig 4, Total Incidence in the 12 Week Epidemic Period, Hunter & Young (1971) p.642

Table 1: Summary Statistics of Pneumonia Notifications and Local Authority Characteristics

Variable	Obs	Mean	Std. Dev.	Min	Max	Correl with *Epidemic*
Pneumonia notifications per hundred thousand population (September - November 1957) ($Epidemic_l$)	172	39.71	36.72	0.89	261.72	1.00
Average Pneumonia notifications per hundred thousand population, September - November 1955-6 ($Pre\text{-}Epidemic_l$)	172	9.64	10.95	0.00	106.69	0.68
Still births/Total Births (1956) ($Stillbirth\ Rate_l$)	172	0.02	0.01	0.01	0.09	-0.05
Deaths from TB per 1000 population (1956) ($DRTB_l$)	172	0.12	0.06	0.00	0.35	0.02
% Households living with $>$ 1 person per room ($\%\ Crowded_l$)	172	0.20	0.10	0.08	0.51	0.47
Percentage of male working population unskilled ($\%\ Unskilled_l$)	172	0.13	0.04	0.07	0.30	0.22
Percentage of men leaving school aged 16 or older ($\%\ Leaving\ school\ post\ 16_l$)	172	0.13	0.05	0.04	0.45	-0.20
Population in thousands/square km ($Pop\ Density_l$)	172	1.81	1.96	0.01	7.91	0.25

Notes: $Epidemic_l$, $Pre\text{-}Epidemic_l$, $Stillbirth\ Rate_l$ and $DRTB_l$ are calculated using the Registrar General's Returns for England & Wales, and for Scotland. Epidemic is calculated by dividing the total number of pneumonia notifications in a local authority in September, October and November 1957 by the local authority population, as estimated in June 1957. $Pre\text{-}Epidemic_l$ takes an average of the number of pneumonia notifications in a local authority in the same periods in 1956 and 1955, and divides by the population in 1956. $\%\ Crowded_l$, $\%\ Unskilled_l$, and $\%\ Leaving\ school\ post\ 16_l$ are calculated using the 1951 census 10% sample tables. $Pop\ Density_l$ is calculated using local authority geographic areas, available from UKBORDERS, and the 1956 population estimates from the Registrar's Returns

Table 2: Birth Weight in Ounces and the Intensity of the Epidemic:

	Birth weight (Oz)				Gestation (Days)	Birth Weight (Oz)
	1) LA controls	2) LA + Child Controls	3) BW Interactions	4) IUG Interactions	5) Gestation Interactions	6) Robustness
Epidemic	-0.0185***	-0.00903	0.00317	-0.00179	0.0064	0.00582
	[0.00702]	[0.00752]	[0.0107]	[0.00942]	[0.00575]	[0.0103]
Epidemic - Maternal Health Interactions						
Smoking Prior to Pregnancy (No-Smoker Omitted)						
Smoking <10/Day x Epidemic			-0.0101*	0.00123	-0.0123	-0.0104*
			[0.00568]	[0.00628]	[0.00965]	[0.00563]
Smoking 10+/day x Epidemic			-0.0142**	-0.0181***	-0.00533	-0.0144**
			[0.00574]	[0.00641]	[0.00660]	[0.00582]
Mother's Height in inches - 65/66 inches omitted.						
Epidemic x <=60 (<=154cm)			-0.0191**	-0.00737	-0.0166	-0.0203***
			[0.00793]	[0.00619]	[0.0108]	[0.00767]
Epidemic x 61 (154-156cm)			-0.00988	-0.00414	-0.0168**	-0.0101
			[0.00847]	[0.00755]	[0.00707]	[0.00859]
Epidemic x 62 (156-159cm)			0.00644	0.000225	0.00209	0.00573
			[0.00818]	[0.00754]	[0.00519]	[0.00831]
Epidemic x 63 (159-161cm)			-0.0036	-0.00275	-0.00536	-0.0041
			[0.00880]	[0.0102]	[0.00803]	[0.00873]
Epidemic x 64 (161-164 cm)			-0.00392	0.00429	-0.00757	-0.0045
			[0.00828]	[0.00636]	[0.00520]	[0.00852]
Epidemic x >=67 (>169cm)			-0.00932	0.00178	-0.00405	-0.0097
			[0.0203]	[0.0162]	[0.0116]	[0.0205]
Local Authority Controls	Yes	Yes	Yes	Yes	Yes	Yes
Child Level Controls	Yes	Yes	Yes	Yes	Yes	Yes
Implied Epidemic Effects	-0.0403	-0.0197				
	[0.0153]	[0.0164]				
Gestation				0.705***		
				[0.0119]		
Log Distance from Windscale						0.855
						[0.761]
Birth region FE						Yes
P-value: No Effect of the Epidemic	0.0094	0.2318	0.0041	0.0004	<0.0001	0.0026
Heterogeneous Effects of the Epidemic						
P-value: No diff by prior smoking behaviour			0.0389	<0.0001	0.4469	0.037
P-value: No diff by maternal height			0.0047	0.0564	0.0019	0.0031
Observations	16765	16765	16765	15005	15005	16765
R-squared	0.004	0.082	0.083	0.321	0.03	0.084

Notes: *** denotes significance at 1%, ** at 5%, and * at 10% level. The dependent variable is birth weight in ounces. Observations are at the cohort member level. Robust standard errors are clustered at the local authority level. $Epidemic_l$ represents pneumonia notifications per 100,000 population in LA of birth Sept-Nov1957. Local authority controls include: $Pre\text{-}Epidemic_l$, an average of notifications per 100,000 population in the same periods in 1955 and 1956; the rate of still birth, death rates from TB, and population density in 1956; the %s of household living with > 1 person per room, unskilled men of working age, men leaving school aged 16+, in 1951. See Table 1 for a descriptive statistics. Child-level controls include social class and schooling of both parents, mother's age and its square, the mother's height, the tenure of accommodation and number of persons per room, and whether the mother smoked before becoming pregnant. Summary statistics are presented in Table 6. The implied effects size of epidemic denotes the effect of a one standard deviation in epidemic intensity on birth weight, in standard deviations.

Table 3: Cohort Member Test Scores Aged 7 and 11, the Epidemic and Birth Weight

	Draw A Man Score Aged 7				General Test Score Aged 11			
	Baseline	+ BW	+interactions	+interactions & BW	Baseline	+ Birth weight	+interactions	+interactions & BW
Epidemic	-0.00148***	-0.00145***	-0.00136*	-0.00137*	-0.000974**	-0.000951**	-0.00107**	-0.00109**
	[0.000523]	[0.000527]	[0.000694]	[0.000697]	[0.000387]	[0.000391]	[0.000494]	[0.000496]
Birth Weight		0.00391***		0.00392***		0.00404***		0.00405***
		[0.000513]		[0.000511]		[0.000446]		[0.000447]
Local Authority Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Child-level controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Maternal Smoking x Epidemic			Yes	Yes			Yes	Yes
Maternal Height x Epidemic			Yes	Yes			Yes	Yes
Implied Epidemic Effects	-0.066	-0.0649			-0.0488	-0.0476		
	[0.0234]	[0.0236]			[0.0185]	[0.0188]		
Implied Birth Weight Effects		0.08				0.08591		
		[0.0105]				[0.0095]		
P value: No difference by Maternal Smoking			0.6806	0.7587			0.4745	0.4032
P value: No difference by Maternal Height			0.0435	0.0339			0.6694	0.583
Observations	13669	13669	13669	13669	12987	12987	12987	12987
R-squared	0.074	0.079	0.075	0.08	0.24	0.245	0.241	0.246

Notes: *** denotes significance at 1%, ** at 5%, and * at 10% level. The dependent variables normalised Draw a Man Score in columns 1-4, and normalised General Test Score (non-verbal) in columns 5-8. Robust standard errors are clustered at the local authority level. $Epidemic_l$ represents pneumonia notifications per 100,000 population in LA of birth Sept-Nov 1957. Local authority controls include: $Pre\text{-}Epidemic_l$, an average of notifications per 100,000 population in the same periods in 1955 and 1956; the rate of still birth, death rates from TB, and population density in 1956; the %s of household living with > 1 person per room, unskilled men of working age, men leaving school aged 16+, in 1951. See Table 1 for a descriptive statistics. Child-level controls include social class and schooling of both parents, mother's age and its square, the mother's height, the tenure of accommodation and number of persons per room, and whether the mother smoked before becoming pregnant. Summary statistics are presented in Table 6. Columns 1 and 2 include parental investment measures from 1965, whilst columns 3 and 4 include measures from 1965 and 1969. These measures encompass: teacher assessments as to whether each parent was interested in their child's education, in both 1965 and 1969; and, the frequency with which each parent read to their child, in 1965. See Table 7 for summary statistics. The implied effects size of epidemic denotes the effect of a one standard deviation in epidemic intensity on outcomes, in standard deviations.

Table 4: Cohort Member Test Scores and Heights Aged 7 and 11, Birth Weight and the Epidemic

	Age 7		Age 11	
Birth Weight	0.0586***	0.0585***	0.0702***	0.0700***
	[0.00322]	[0.00322]	[0.00374]	[0.00374]
Epidemic	-0.00171	-0.000115	-0.000436	0.00423
	[0.00456]	[0.00536]	[0.00316]	[0.00440]
Smoking < 10/Day x Epidemic		-0.00450***		-0.00570***
		[0.00150]		[0.00198]
Smoking 10+/day x Epidemic		-0.00623***		-0.00716**
		[0.00203]		[0.00307]
Implied Effect of Epidemic	-0.0129		-0.00212	
	[0.0344]		[0.0197]	
Implied Effect of Birth Weight	0.2026		0.1996	
	[0.0111]		[0.0106]	
P-value: No difference in epidemic effects by:				
Pre-pregnancy maternal smoking		0.0002		0.0002
Maternal Height		0.279		0.2482
LA and Child Level Controls	Yes	Yes	Yes	Yes
Observations	12750	12750	11679	11679
R-squared	0.203	0.204	0.205	0.206

Notes: *** denotes significance at 1%, ** at 5%, and * at 10% level. In columns 1 and 2, the dependent variable is child's height in metres at age 7; the dependent variables in columns 3 and 4 is child height in metres at 11. Robust standard errors are clustered at the local authority level. $Epidemic_l$ represents pneumonia notifications per 100,000 population in LA of birth Sept-Nov 1957. Local authority controls include: $Pre\text{-}Epidemic_l$, an average of notifications per 100,000 population in the same periods in 1955 and 1956; the rate of still birth, death rates from TB, and population density in 1956; the %s of household living with > 1 person per room, unskilled men of working age, men leaving school aged 16+, in 1951. See Table 1 for a descriptive statistics. Child-level controls include social class and schooling of both parents, mother's age and its square, the mother's height, the tenure of accommodation and number of persons per room, and whether the mother smoked before becoming pregnant. Summary statistics are presented in Table 6. Columns 1 and 2 include parental investment measures from 1965, whilst columns 3 and 4 include measures from 1965 and 1969. These measures encompass: teacher assessments as to whether each parent was interested in their child's education, in both 1965 and 1969; and, the frequency with which each parent read to their child, in 1965. See Table 7 for summary statistics.The implied effects size of epidemic denotes the effect of a one standard deviation in epidemic intensity on height, in standard deviations.

Appendix

Asian Influenza and Cohort Composition

Asian influenza could have altered the composition of the NCDS cohort in two ways: by increasing rates of foetal loss, through spontaneous abortion of maternal death prior to viability; or, by inducing higher rates of prematurity[45]. The evidence presented in Section 2 indicates that influenza significantly elevates the risk of both (Rasmussen et al., 2008). If any changes in composition are non-random with respect to unobserved child characteristics that influence cohort member outcomes, results in sections 4 and 5 would be biased.

Figure 3 shows the changes in maternal deaths from 1950-1966 from selected causes. There is a peak in the number of pneumonia death of women of childbearing age in 1957, caused by Asian influenza. The number of deaths is ten times higher than the year before, however the absolute number is just 344. Even if all of these women were pregnant and due in the NCDS week, which is highly improbable, 344 only amounts to 2% of the total NCDS sample.

Rates of foetal loss and prematurity are harder to measure. Table 5 shows the still birth rate and the number of live births, in the first and second quarters of 1956 to 1959. If the epidemic increased foetal loss or prematurity, still birth rates should be relatively higher, and live births relatively lower in first half of 1958. However, still birth rates were lower in Q1 and Q2 of 1958 than in 1956 or 1957, whilst live births were higher. Monthly live birth figures show live births were higher than the previous year in February, March and April of 1958. Small changes in gestation are impossible to rule out, as live birth figures are only given by the month. However, the distribution of gestation does not differ markedly from that of the 1970 birth cohort followed by the British Cohort Study. Furthermore, results in section 4 suggest that any effects on gestation were confined to certain sub-sections of the population.

A review of the aggregate data thus provides no evidence to suggest that the epidemic affected cohort composition to a degree large enough to bias results in sections 4 and 5. However, it should remember that the NCDS represents a particular cohort, exposed between 16 and 25 weeks in gestation. This cohort less vulnerable to spontaneous abortions than those exposed in the first 15 weeks (Nyagu et al., 2002; Otake & Schull, 1998). The increase in Q2 births between 1957 and 1958 is smaller than increases in other years. These children would have been in the early stage of gestation during the epidemic. The results of this study may therefore have been very different had the cohort been born in

[45]There could be no effect on fertility patterns, as all cohort members were in the womb by the time the epidemic struck

early summer.

Figure 3: Deaths of Women of Child-Bearing Age by Selected Causes, Indexed to 1950 (1950-1966)

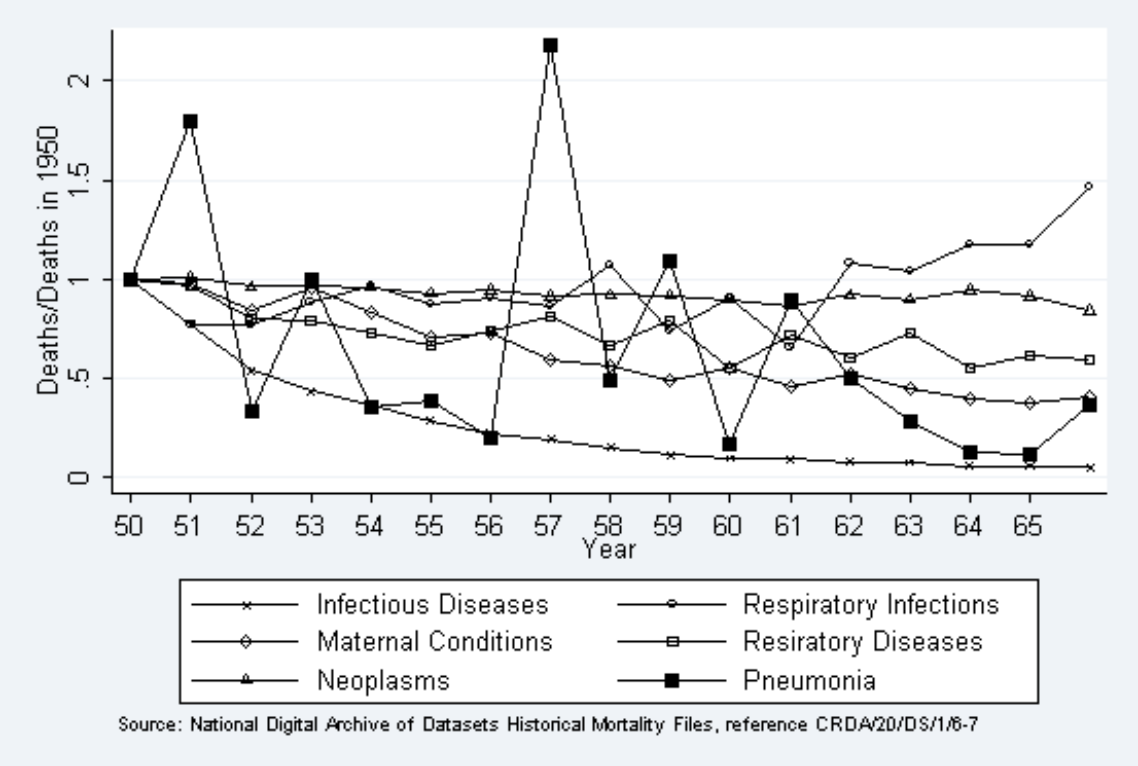


Table 5: Still Birth Rates and Live Births - First Two Quarters, 1956-1959

Year	Still Birth Rate[1]		Live Births[2]	
	Q1	Q2	Q1	Q2
1956	23.4	22.6	178893	177375
1957	22.6	22.5	181794	187797
1958	21.4	21.4	189942	189019
1959	21.4	21	193202	194103

Notes: Q1 runs from 1st January to 31st March, and Q2 from 1st April to 31st June.
[1]Registrar General's Quarterly Returns 1955-9.
[2]Office for National Statistics (ONS). Dataset PBH22A Live Births: 1938-2004: Month of Occurrence

Child-Level Summary Statistics

Table 6: Summary statistics of Child-level Characteristics used as Controls for Full Specifications ($\boldsymbol{X}_{il}$)

Variable	Mean	Std. Dev.
Mother's Age at Birth	27.433	5.775
Dummy Variables		
Mother's Education		
Left School at Minimum School Leaving Age	0.748	0.434
Left School after Minimum Age	0.248	0.432
Information Missing	0.003	0.058
Father's Education		
Left School at Minimum School Leaving Age	0.604	0.489
Left School after Minimum Age	0.18	0.384
Information Missing	0.216	0.412
Father's Social Class		
Class I	0.043	0.202
Class II	0.123	0.328
Class III Non-Manual	0.091	0.287
Class III Manual	0.482	0.5
Class IV	0.115	0.319
Class V	0.093	0.29
N/A	0.055	0.227
Mother's Father's Social Class		
Class I	0.023	0.149
Class II	0.13	0.336
Class III	0.427	0.495
Class IV	0.125	0.331
Class V	0.116	0.32
N/A/Retired/Dead	0.18	0.384
Number of Persons Per Room		
Up to 1	0.661	0.473
1–1.5	0.171	0.376
1.5–2	0.095	0.293
2–2.5	0.018	0.132
Over 2.5	0.028	0.165
N/A	0.028	0.164
Housing Tenure		
Owner Occupier	0.343	0.475
Council Renting	0.326	0.469
Privately Renting	0.099	0.299
Other	0.233	0.423
Mother Smoked Before Pregnancy		
No	0.59	0.492
Yes - Less than 10 per day	0.216	0.412
Yes - 10 or More Per day	0.194	0.396
Mother's Weight in 1958 (inches)		
<=60	0.109	0.311
61	0.101	0.301
62	0.159	0.366
63	0.141	0.348
64	0.163	0.370
65-66	0.180	0.385
67+	0.106	0.308
Missing	0.040	0.196
N	16765	

Source: Author's calculations using University of London. IoE (2008). The mean of each dummy variable represents proportion falling into that category dummy

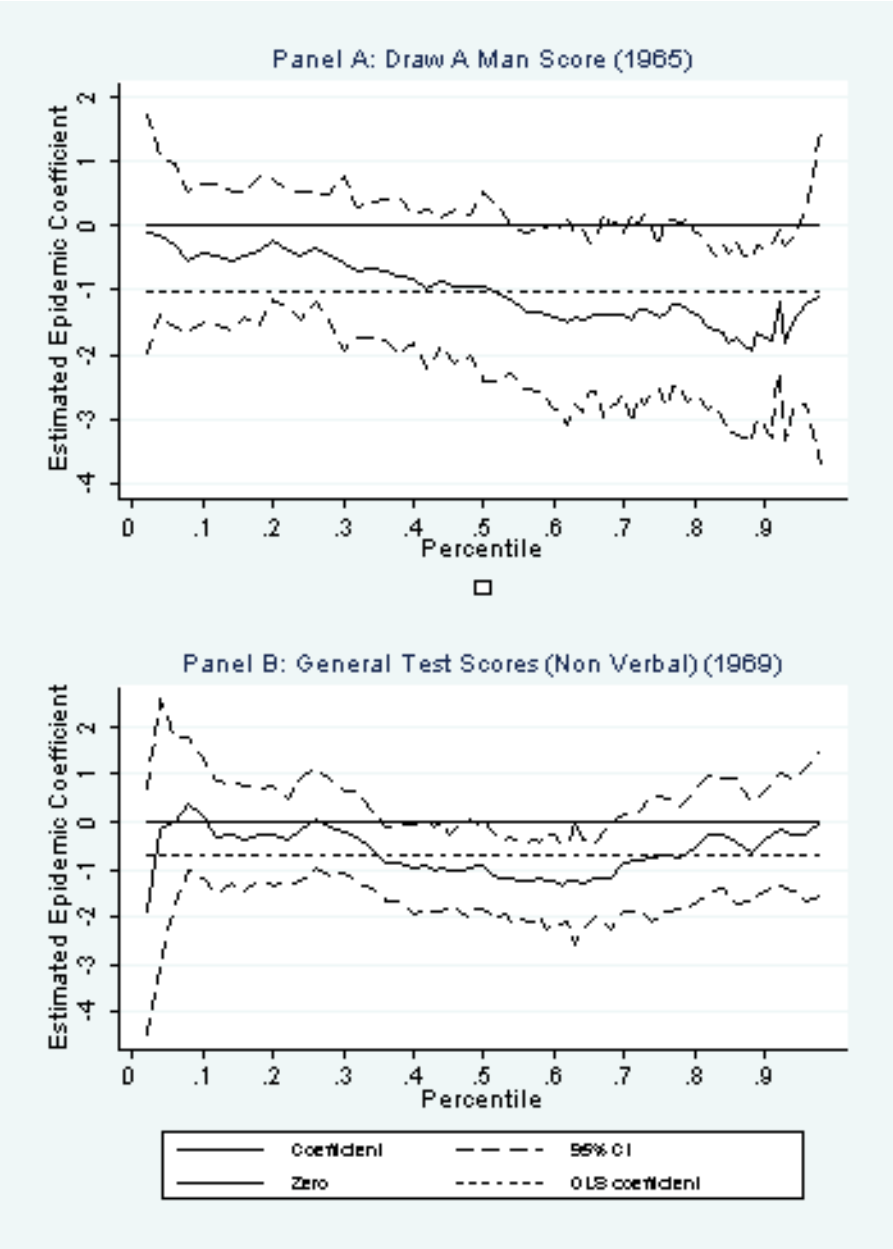
Table 7: Summary Statistics of Variables Measuring Parental Interest and Input in Cohort Members' Education ($\boldsymbol{P}_{il}$)

Variable	Mean	Std. Dev.
Teacher Assessments of Parental Interest in their Child's Education		
MOTHER, 1965		
Some Interest	0.330	0.470
Little Interest	0.123	0.329
Very Interested	0.298	0.457
Can't Say	0.023	0.149
Over Concerned	0.084	0.277
Can't Say/Na	0.142	0.350
MOTHER, 1969		
Some Interest	0.269	0.444
Little Interest	0.105	0.307
Very Interested	0.269	0.444
Can't Say	0.029	0.168
Over Concerned	0.086	0.280
Can't Say/Na	0.241	0.428
FATHER, 1965		
Some Interest	0.185	0.388
Little Interest	0.129	0.335
Very Interested	0.204	0.403
Can't Say	0.009	0.097
Over Concerned	0.330	0.470
Can't Say/Na	0.142	0.350
FATHER, 1969		
Some Interest	0.194	0.395
Little Interest	0.130	0.336
Very Interested	0.203	0.402
Can't Say	0.019	0.137
Over Concerned	0.180	0.384
Can't Say/Na	0.274	0.446
Parents Read to the Child, 1965		
MOTHER		
Yes, Every Week	0.393	0.488
Yes, Occasionally	0.282	0.450
Hardly Ever	0.129	0.335
Inapplicable/Na	0.053	0.224
Missing	0.142	0.350
FATHER		
Yes, Every Week	0.281	0.449
Yes, Occasionally	0.276	0.447
Hardly Ever	0.221	0.415
Inapplicable/Na	0.079	0.270
Missing	0.142	0.350
N	16765	

Source: Author's calculations using University of London. IoE (2008) All variables are dummies so the mean represents proportion falling into that category dummy.

Quantile Regressions

Figure 4: Quantile Estimates - The Distribution of Effects of the Epidemic upon Draw A Man Test Scores



Notes: Panel A plots the $Epidemic_l$ coefficients from quantile regressions, which estimate the conditional effect of $Epidemic_l$ upon raw Draw a Man Score. Panel B plots the corresponding $Epidemic_l$ coefficients for raw non-verbal General test scores. In both panels A and B, specifications include a full set of local authority and child-level level controls. See Tables 1 and 6, for descriptions. Panel A includes parental investment measures from 1965: teacher assessments of the interest each parent has in their child's education, and how frequently parents read to their child. Panel B includes all parental investment measures from 1965, plus teacher assessments of each parents interest in their child's education. See Table 7 for descriptive statistics. All standard errors are block bootstrapped at the local authority level.

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