

The Life Cycle of Early Skill Formation

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I, Emma Tominey confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Abstract

This thesis focuses on two dimensions of the child production function - the technology of human capital formation and the role of specific family inputs into human capital. The first two chapters explore the technology by which inputs produce child human capital. Specifically, for given parental lifetime income, these ask whether the timing of income matters for later outcomes of the children. Two methodologies estimate the effect at different margins. Firstly in a fully flexible model, the relationship between parental income at child ages 0-5, 6-11 and 12-17 and subsequent child outcomes is estimated nonparametrically, allowing for complementarity across periods. Income aged 0-5 is as important in general as income at age 6-11 for child human capital formation. Complementarities exist between 0-5 and 6-11 for households with low permanent income, which are those likely to be credit constrained. Similarly, very strong complementarities are found between early years income and income during adolescence (age 12-17) for the group of poor parents. Chapter 3 analyses the role of permanent and transitory income shocks at different ages, upon adolescent human capital. Empirical results suggest the effect of permanent shocks declines across age. This is intuitive, given that a permanent shock changes household wealth and hence a shock at age 1 drives more future income realisations than a later shock. Transitory shocks on the other hand, have an increasing effect upon child outcomes across child age. Further, there is evidence of intrahousehold insurance against paternal transitory income shocks.

The final two chapters of the thesis look at parental inputs in the production function. Chapter 4 allows the life cycle of skill formation to begin pre-birth, by estimating the role of maternal smoking during pregnancy upon birth outcomes. Results suggest a large proportion of the correlation is explained by a maternal fixed effect. Finally, chapter 5 offers a cross country comparison of the similarities in child test score gaps, by a range of measures of family inequality. Despite wide institutional differences, this chapter estimates homogeneous correlates for maternal education, family size and child gender upon child achievement, but differences in the covariates of lone parenthood and ethnicity.

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Contribution to chapters

Chapters 3 and 4 are sole author papers.

Chapter 2 is coauthored with Dr Pedro Carneiro and Professor Kjell Salvanes

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Full stop. The end.

1 Introduction

The chapters in this thesis focus on the life cycle formation of human capital, from birth to early adulthood. Two dimensions of human capital formation are considered - the technology of human capital formation and the role of neo-natal and early childhood family inputs into human capital of children.

From the late 1950's, economists have been modelling the accumulation of human capital in a market setting, to explain the distribution of earnings. For example Mincer (1958, 1962) and Becker (1964, 1966) first formulate and Ben-Porath (1967) develops a production function for the development of human capital, where technology is determined by constraints and inputs, such as ability and family background, which drive the stock of human capital. The life cycle of skill formation was analysed, with a view to understanding the decision to forego earnings to acquire skills. Skills are generally developed early in a lifetime, either during schooling or initial years in the labour market, suppressing contemporaneous wages. Then, in the post-acquisition years, earnings rise across a lifetime, at a decreasing rate.

There have been departures from the production function for human capital accumulation, where economists allow for more dynamic skill formation. For example, more recent literature adds an additional dimension to the life cycle of skill formation, analysing how investment in human capital may have a return that varies across the lifetime of a child (see Cunha et al (2005), Cunha & Heckman (2006, 2007, 2008), Cunha et al (2006)). The first stage of this thesis offers empirical evidence as to the differential return of parental income across child age, in the child production function of human capital.

Additionally, Corman et al (1987) and Rosenzweig & Wolpin (1991) extend the human capital production function, by analysing the effect of neo-natal behaviours upon birth outcomes, in an attempt to understand the life cycle accumulation of skill formation as a process which starts before the birth of an individual. Todd & Wolpin (2007) provide a review of the different techniques that have been used empirically to address particular elements of the child production function. They formulate an estimator of the effect of inputs into child skill formation under different models and find that a production function which contains only contemporaneous inputs is rejected by the data, in favour for a more cumulative process of skill formation. The second stage of the thesis offers evidence in line with these papers, by firstly considering how neo-natal behaviour drives birth outcomes, when a mother fixed effect is allowed for. Secondly, the correlates of family inputs with early child outcomes is estimated for a range of countries, examining descriptively whether there are country dynamics in the way that inputs enter production functions.

The first two chapters explore the technology through which inputs across the child life cycle produce child human capital. Specifically, for given parental inputs into the child production function, these ask whether the timing of the inputs throughout childhood matter for the stock of human capital in adolescence and early adulthood.

It is possible to think of a model which could predict that income received at different stages of child development, would impact differentially upon the eventual stock of child human capital. In a simple model, parents choose consumption in each period, plus investment in child human capital to maximise their utility. The utility is a function of the stock of child human capital at the end of the periods of investment. A production function which converts investment into child human capital may allow for a return to investment that differs across child age. For example, Cunha & Heckman

(2008) note the presence of sensitive periods (in which the marginal return to investment is higher than in other periods) and critical periods (in which the marginal return to investment is greater than zero only in the critical period) in the development of certain skills. This means that parental investment may be empirically more productive in certain stages of human capital development than others.

However, it is the differential timing of parental income, not investment that is estimated in this thesis. Under perfect credit markets, even if the return to parental investment was relatively high in one period, parents could borrow or save to ensure that they invest optimally. Therefore, it is the interaction of differential returns to human capital investment with specific market failures, that gives rise to an effect of parental income that changes across the child life cycle. Chapters 2 and 3 consider different market failures to estimate the effect of the timing of parental income.

Chapter 2 considers the relationship differentially for liquidity constrained parents. When a liquidity constraint binds in a particular period, the parent will be unable to borrow sufficiently to optimally invest in the human capital of their child. Therefore, a return to income in one period may differ to the return in another.

Chapter 3 allows for uncertainty over income in each period, estimating the effect of income shocks upon child outcomes. With income uncertainty, the permanent income hypothesis predicts that an income shock will change investment by the annuity value of the shock, as it shifts household wealth. An earlier permanent income shock will be expected to have a greater impact therefore than a later shock, as the former alters investment for more periods of the child's lifetime. A transitory shock, on the other hand, is expected to have a smaller effect, which is constant across the age of the child.

In these two chapters, a Norwegian administrative data set provides information on annual household income in each year of a child's lifetime. The aim is to identify the effect of the timing of income upon a range of outcomes for the child aged between 16-30. Chapters 2 and 3 use two different methods of estimating the differential productivity of income across child age, which provide parameters at different margins.

Firstly, chapter 2 looks flexibly at the relationship between the timing of parental income and child human capital, in a fully nonparametric setting. Owing to the curse of dimensionality inherent in nonparametric analysis, child lifetime is aggregated into three periods - age 0-5, 6-11 and 12-17. A multivariate local linear kernel regression analysis identifies the effect of the differential timing of income in these three periods, exploiting movements across deciles of the income distribution. The parameter estimated here therefore concerns particularly large changes in income. In addition to evaluating the relative productivity of income in each of these three periods of lifetime, the fully flexible functional form of the production function enables an evaluation of potential dynamic complementarity in the return to income. Dynamic complementarity exists if the return to income in one period is increasing in the level of income in another period. No study to date has estimated both productivity of and complementarity in the return to the timing of income.

The chapter reports findings that early years income (age 0-5) is as important in general as income at age 6-11 for child human capital formation. However, there are complementarities across the two periods for households with low permanent income, which are those likely to be credit constrained. Similarly, very strong complementarities are found between early years income and income during adolescence (age 12-17), whereby even bundles of early and late income are optimal

to extreme bundles. Again, this is solely for the poorer groups of parents. For all other parents, income is more productive during adolescence than early childhood. These conclusions are replicated in a semiparametric analysis, where by a first stage controls parametrically for a wealth of parental controls and the second stage estimates the residual effect of the timing of income nonparametrically. Additionally, the results are robust to variation of bandwidth.

Chapter 3 exploits income variation within each period of the child's lifetime, rather than aggregating the effect to three stages of development. The idea is to estimate the stochastic process for household income and construct permanent and transitory shocks to identify the effect of the timing of income shocks in each period of a child's lifetime.

Chapter 3 estimates empirically the role of permanent and transitory income shocks realised across child age, upon a range of cognitive and noncognitive adolescent outcomes. The technique to decompose income shocks into permanent and transitory components relies upon the assumed income process. The first step therefore provides empirical evidence of the Auto-Regressive Moving Average (ARMA) transitory income process in Norway. Such evaluations have been carried out in many other countries, but not to date in Norway. Permanent income is assumed to follow a martingale and for transitory income, the model which best fits the data is an MA(1) or MA(2) process. Given this, the effect of permanent and transitory shocks are estimated using the derived moment conditions.

The results of chapter 3 suggest that transitory income shocks have a slightly larger effect when realised later in the life of a child than earlier. For most outcomes, there is a monotonically increasing relationship across time, such that earlier transitory income shocks have a relatively small effect on adolescent human capital.

An opposite relationship was established for permanent income shocks, that the earlier shocks have a greater impact. This is because a permanent shock will shift household wealth and therefore all future investment decisions, hence an earlier shock drives human capital investment for more periods than a later shock. These findings are robust to changes in the assumed income process, restricting the persistence of both the permanent and transitory components.

Additionally, the chapter explores potential intrahousehold insurance against transitory income shocks, by repeating the analysis on father's income alone, rather than household income. Child human capital may be insured against paternal income shocks, if the mother responds by raising her investment in the child, either by working more hours or increasing time spent with the child. The results suggest evidence of such insurance, as paternal transitory income shocks have a smaller coefficient than the household equivalent, especially during early years and later adolescence.

The final two chapters focus upon the role of specific inputs into the child production function. These two chapters explore the determinants to early human capital development, firstly by looking at how neo-natal behaviour drives outcomes at birth (Chapter 4) and secondly at how family characteristics from birth to age 9 drive early child test score outcomes in different countries (Chapter 5).

Chapter 4 allows the life cycle of skill formation to begin pre-birth, by estimating the role of maternal neo-natal behaviour upon birth outcomes. Specifically, the chapter aims to estimate effect on child birth outcomes - birth weight, weeks of gestation, probability of having a low birth weight infant and probability of pre-term gestation - from maternal smoking during pregnancy, allowing for a maternal fixed effect. A large fraction of the correlation between maternal smoking and child birth

outcomes disappears once the fixed effect method is used. Indeed, the only remaining significant effect is for the birth weight outcome. This suggests that smoking during pregnancy is correlated with other behaviours, which independently lower birth outcomes. Exploring heterogeneity in the effect on birth weight, it is mothers who smoke for the entire nine months of gestation that suffer the harm, whereas there is an insignificant effect for mothers who chose to quit by month five. Additionally, there is evidence of potential complementarity in investment of human capital, as the impact on birth weight of smoking is much greater for low educated mothers.

Finally chapter 5 provides a cross country comparison of the similarities in test score gaps, by a range of measures of family inequality. Despite evidence that early childhood experience is an important determinant of child achievement, the empirical literature to date has not explored the empirical connections between test scores and their correlates across countries.

Heterogeneity may exist in correlates if there are differential coefficients on inputs in a child production function, or if test scores measured inconsistently across countries. On the other hand, if the same child production function translates inputs into skills, or budget constraints are similar for differently types of families, then the correlates will be similar. There is a need to ensure that inconsistent measurement of test scores is not driving the results, therefore the chapter compares datasets with identical measurements. The children of the 1958 UK National Child Development Study (CNCDS) were given identical tests to the children of the 1979 US National Longitudinal Survey of Youth (CNLSY). We are also able to study a wider group of countries, by repeating the analysis on the 35 participating countries in the 2001 Progress in International Reading Literacy Study (PIRLS) where children were again administered identical tests. For the PIRLS, the analysis groups countries by OECD and developing country status.

The second stage of this chapter is to consider how measured test score gaps relate to two factors considered in quite a lot of recent work on child development. These are non-cognitive traits of children (NC) and parental investments (PI). In a number of recent papers both of these have been shown to display significant correlations with test scores. But again, the question we ask is not just whether these two traits create channels through which test score gaps emerge, but whether they play a similar role in different countries, developing the Conditional Cross-Country Constancy and NC and/or PI Cross-Country Constancy Hypotheses.

Despite different institutional environments, we report evidence of some rather marked cross-country similarities in the association between early age test scores and their correlates, especially maternal education, family size, child gender and family earnings. This emerges from the detailed in-depth UK-US comparison. In the much broader comparison across the 35 countries participating in the 2001 Progress in International Reading and Literacy Study, around 50% of countries have correlates statistically different to the group mean, however family earnings have a similar coefficient on test scores only for a group of rich countries. Other correlates – like ethnicity and lone parenthood – do not show similarities (probably due to heterogeneous demographics across nations). Examination of associations with the measures of non-cognitive skills and parental investments reinforces findings for test score correlates displaying cross-country similarity in that complementarities with these factors are also seen in the data.

Importantly for the first three chapters, in chapter 5 Norway generally has correlates between family status and child achievement similar to the group mean of OECD or developed countries, suggesting that the results found in earlier chapters can be generalised to other countries.

3 The Timing of Parental Income on Child Outcomes: The Role of Permanent and Transitory Shocks.

Abstract

Does the timing of permanent and transitory shocks to parental income matter for child outcomes? Whilst the literature has linked the evolution of lifetime income to consumption behaviour, little is known about the response of child human capital. This chapter aims to fill the gap. The first step is to document the income process for the population of parents in a data set of Norway, for children born in 1970-1980. I assume that permanent income follows a random walk and the data indicates an MA(1) process for transitory income. Next, annual household income shocks are estimated as the deviation from the life cycle profile of income, allowing for a parental fixed effect and from this, moment conditions allow a decomposition of household income shocks into permanent and transitory components. The effect of these shocks across child age are then estimated upon the stock of adolescent human capital. I find that the effect of permanent income shocks declines across the age of the child between age 6 and 16 - that is a shock realised when the child is age 6 is more important than a shock realised at age 16, but the effect is constant across early years. When looking just at father's income however, the smooth decline in the effect starts from age zero, through to adolescent years. The difference is likely due to a change in maternal labour supply when children enter school which shifts household permanent income. The declining effect of permanent income shocks across age is intuitive, given that a permanent shock changes household wealth and hence a shock at age 1 drives more future income realisations than a later shock. Transitory income shocks to the household have a slightly increasing effect across child age for three outcomes - years of schooling, college attendance and ability- suggesting that it is the later shocks that matter most for child development. Additionally, there is some evidence that mothers insure the household against paternal transitory income shocks, especially at early ages and during late adolescence. Finally, the results prove robust to checks which vary the persistence of permanent and transitory income shocks.

3.1 Introduction

The extent to which consumers insure themselves against changes in income has been well documented in the economics literature¹¹, however little is known about how the evolution of household income drives the human capital of their children. The aim of this chapter is to fill the gap. Specifically, the question raised is whether there is a differential effect of permanent and transitory shocks to parental income realised at different points across a child's life, upon subsequent child human capital. This is the first paper to date which decomposes income shocks into permanent and transitory components and subsequently examines the effect upon child outcomes.

In the case of permanent income shocks, one would expect an early permanent income shock to have a larger effect on child outcomes than one realised later in the child's lifetime. The reason is

¹¹Deaton (1992), a good summary, describes how under the permanent income hypothesis, individuals smooth their consumption against transitory income shocks but adjust consumption to permanent income shocks. Blundell & Preston (1998), Attanasio *et al* (2002), Blundell *et al* (2008) examine consumption responses to income changes and Adda *et al* (2007) look at health responses to income shocks. Attanasio & Szekely (2004) find that durable consumption is particularly sensitive to wage changes.

that a permanent shock shifts household wealth for all future periods and therefore, *ceteris paribus*, a shock realised at birth will drive all future income realisations, whereas a shock at age 16 will drive far fewer.

There are at least two reasons to expect a parental transitory income shocks to have heterogenous effects at different stages of child development are that the ability of parents to insure children against transitory income shocks may differ across a lifetime, or that there is a return to parental investment in child human capital that varies across child age. The first mechanism relates specifically to the permanent income hypothesis (PIH). According to the PIH, fluctuations in parental investment should be smoothed against transitory income shocks, but respond to shocks that are permanent. Thus, there should be a constant, small effect of transitory shocks across child age. However, failure of the PIH¹² may lead to differential effects of transitory income shocks depending upon the child age at realisation. For example, younger parents may face binding liquidity constraints which relax as they and the child ages. Consequently, transitory income shocks realised early in the child's life will not be fully insured and will have a larger impact upon child human capital than later shocks.

Alternatively (or simultaneously), the return to parental investment may change across different stages of child development such that *ceteris paribus*, the effect of income shocks on human capital varies with the child age in which the shocks are realised. Cunha & Heckman (2008) and Cunha *et al* (2006) describe human capital as a function of parental investment made across child age. According to the authors, the child's early years are critical periods of investment (where the return equals zero in all other periods) in cognitive achievement. For example, the ability to learn a language falls sharply at the age of 5 and therefore failure to invest before this age will seriously damage cognitive skill acquisition. On the other hand, Cunha & Heckman (2008) find that noncognitive skills are more malleable, and therefore later investment will also yield a high return. Similarly to this, the results in chapter 2 of this thesis suggested that income realised during adolescence was more productive than income received during early years. Consequently, an empirical question to be addressed in this chapter is whether there are periods during the childhood in which income shocks are most productive in accumulating human capital.

No study to date has addressed how parental income shocks, realised at different stages of child development drive the stock of child human capital accumulated by adolescence. The first contribution of this chapter is to explore this question. The empirical literature has explored the differential effect of the timing of income levels¹³ and additionally there is a literature which evaluates whether parents insure their children (who are currently in the labour market) against risk, for example through financial transfers and cohabitation¹⁴. However, there is no literature assessing how parental income shocks realised when the child is growing up are insured against and how the ability to insure differs as the children age. An innovation of this chapter is to combine the methodology to decompose income shocks into permanent and transitory components with the literature on the life cycle formation of child human capital, in order to understand the extent to which child human capital is insured against parental shocks to income.

One potential reason why literature has not examined this issue is a lack of adequate data. The data in this chapter takes the population of around 600,000 Norwegian children, born in the 1970s

¹²Empirical failures note excess sensitivity of consumption to predictable income changes (Flavin, 1981) and excess smoothness of consumption to permanent income changes (Campbell & Deaton, 1989).

¹³see chapter 2 of the thesis and additionally Levy & Duncan (2000), Jenkins & Schluter (2002)

¹⁴See for example Rosenzweig & Wolpin (1993), Hayashi *et al* (1996), Becker *et al* (2004) and Kaplan (2007)

and tracked through to 2006, which provides in depth information on annual household income plus a range of cognitive and non-cognitive adolescent outcomes, including years of schooling, dropping out from high school, university attendance and ability and health test scores from an ability test for males. Thus, the second contribution of the chapter is to add to the literature on the technology of skill formation, assessing whether the effect of the timing of income shocks upon cognitive outcomes is different to the effect upon noncognitive skills.

A third contribution is to understand intrahousehold insurance against shocks to paternal income. A shock to the income of the father of a household can be insured by potential behavioural responses of the mother. For example, she can maintain constant investment in child human capital by raising her time input, or work more hours to raise her financial input. This question is explored by comparing an analysis of household income shocks to paternal shocks.

The methodology used in this chapter is as follows. A structural model of the parental income process and child human capital formation decomposes income shocks into permanent and transitory components, and evaluates the effect of these shocks upon child development. An important first step therefore is to gather empirical evidence as to the income process that is applied to the structural model. Permanent income is assumed to follow a random walk and the evidence presented suggests that transitory income is best described by an MA(1) process. Next, deviations of household income from the life cycle trend are predicted in each year of child life. Moment conditions from the income process allow a decomposition into permanent and transitory income shocks from early childhood to adolescence for each cohort within a labour market¹⁵. Finally, the effect of both types of shock, realised across child age, is estimated on the eventual stock of adolescent human capital. I allow for an initial condition in household income and a parental fixed effect in child human capital to be correlated. The identification assumption is that second order moments of the permanent and transitory income shocks differ across child age, cohorts and labour markets, but that the effect of these shocks upon child outcomes is homogeneous.

I find a strong and significant effect of the initial condition on child outcomes, of up to 0.94 standard deviations. Thus, there is significant dispersion in outcomes for the sample of Norwegian children, determined at the start of their lifetime. For all outcomes, the effect of a household permanent income shock is significant and approximately constant between ages 1-6 and then declines thereafter. One explanation for the divergence of results is that at age 7 in Norway, children enter the schooling system. This may change maternal labour supply and cause a shift in permanent income in the household. By focusing just on paternal income, which is less sensitive to child schooling, this effect is removed. Indeed, in general, permanent shocks to paternal income have a large effect early in the lifetime of the child and this effect falls smoothly across child age, to zero during the later adolescent years, as would expected.

In terms of transitory income shocks, for the outcomes education, college attendance and ability, transitory income shocks have an effect which *increases* across child age. A shock realised in early childhood has a smaller effect than a late transitory income shock. For the other two outcomes - drop out from high school and health, there is a flat relationship. Whilst it is not possible to compare the return to parental investment into child human capital, this suggests that there are important returns to investment in later years, as the effect of shocks during these years are non-negligible and

¹⁵The method is a slight adaptation to Moffitt & Gottschalk (1995), Meghir & Pistaferri (2004) and Blundell *et al* (2008) and similar to Jappelli & Pistaferri (2008)

in some cases, higher than the effect of early shocks.

Heterogeneity in the manifestation of transitory income shocks on child outcomes was also observed when mother's income was excluded from the analysis. The effect at all years, but particularly the early childhood and during adolescent, was much lower for paternal income shocks. This is suggestive of intrahousehold insurance against income shocks.

The chapter is structured as follows. Section 3.2 describes the Norwegian data, section 3.3 maps the income process for the sample of parents in Norway. The empirical strategy is pursued in section 3.4, section 3.5 discusses the results and section 3.6 the robustness checks. Finally, section 3.7 concludes.

3.2 Data

The Norwegian register and administrative data provides information for the analysis. Annual information is recorded for the population of Norway on a range of variables, linking across generations of the same family their records from birth, education, labour market and marriage market status. Table 1 displays the summary statistics for the data, containing 616,210 children born to 399,603 mothers. The chosen sample contains the population of children born in Norway between 1970-1980.

It is possible to define a wide range of child human capital outcomes, recorded during their adolescence. Educational status is measured as late as 2006, meaning that the youngest children in the sample are aged 26 by this time and likely to have completed their education. Three education variables are defined for the analysis. Firstly, years of completed education is recorded for the full sample, with a mean value of 12.53 years. Secondly, a focus on the bottom of the educational distribution records a dummy variable equal to one if the child dropped out of high school before receiving a certificate for vocational or academic education. Without this certificate, students' future paths are restricted and for example, they will not be able to attend university. 26% of students in the sample are recorded as dropout students¹⁶. The final educational record is attendance at college/university, which applies to 37% of students.

Additional human capital measures come from tests, when the male child sample are around age 18. Military service is compulsory in Norway for males, who take tests including a measure of ability and health for entry to the army. The ability score is a composite score from arithmetic, word similarities and figures tests. Sundet *et al* (2004, 2005) detail the tests. The arithmetic and word tests are most similar to the Wechsler Adult Intelligent Scale (WAIS) and the figures test to the Raven Progressive matrix, which are approved by psychologists as measures of ability. The continuous scores are banded into 9 point scale, with a mean of 5.09. Also measured in the Armed Forces Qualifying Test is an indicator of physical health. The score is again on a 9 point scale, with 9 indicating perfect health. As the mean value of the score is 8.35, it is clear that this test in fact records perfect physical health for the majority of the sample (85%)¹⁷.

Often the literature on human capital relies on such test scores as measures of child human capital achievement. For example, both Cunha & Heckman (2008) and Todd & Wolpin (2003) use test scores from the National Longitudinal Study of Youth, many papers including Blanden

¹⁶this outcome is referred to as dropping out of high school, for simplicity

¹⁷Note, I created a dummy variable equal to one if individuals scored 9 and zero otherwise. The results for this outcome were almost identical to the 9 point scale

et al (2007) measure child achievement from the cognitive tests taken from the UK National Child Development Survey. Whether these tests are a reliable and generalisable measure of human capital is debatable. On the one hand, they offer a measure which is closer to ability than tests from an education system, where child outcomes are closely linked to paternal socio-economic status. On the other hand, test scores measure human capital with a degree of measurement error and comparability across different tests taken in different countries is not widely understood. Chapter 5 explores this issue further. Therefore, it is interesting itself to compare the results across school outcomes and test score achievement.

I link the child unique identifier from the educational data sets to the mother and father from the birth certificate and match income and years of education for the mother and father from 1967-2006. Income is deflated to 2000 prices and household income calculated as the sum of paternal and maternal income if both parents are known, or one parent otherwise. For ease of comparability, the income measures are converted to pound sterling. Parental education is defined as the maximum years of schooling between these years. The parental age at birth is calculated and marital status information is available for all relevant years of the sample from which an indicator variable is created to equal one if the parents are married in a particular year and zero otherwise.

The paternal identifier is linked to the municipality of residence in each year. If a paternal identifier is missing, the maternal identifier is used instead. There are around 450 municipalities in Norway. However, it is the local labour market identifier that is used in the analysis, so as to appropriately group areas by something similar to a travel-to-work-area (TTWA). Geographers in Norway have defined 90 labour markets in Norway. From the sample of parents contained in our data set, the labour market size varies between 1,000 and 65,000 households. For a large majority of children in the sample (78%), the labour market observed when the child is born is identical to that at age 16. I keep only these children in our sample, so as to be able to define the local labour market of the child as being constant across the lifetime of the child.

3.3 Income Process in Norway

In the empirical section below, the effects of transitory and permanent income shocks across child age are identified for a particular income process. Permanent income is assumed to follow a martingale and transitory income follows an ARMA(p,q) process. This section aims to infer the correct income process using very detailed administrative income data for the population of Norwegian parents, from 1967 to the present. Meghir & Pistaferri (2004) and Blundell *et al* (2008) suggest that in the US, a permanent transitory model of income is appropriate, whereby permanent income is a martingale and transitory income serially uncorrelated or a first order Moving Average process (MA(1)). In the UK, Dickens (2000) estimates a random walk in age for permanent income and a serially correlated transitory component. Bonhomme & Robin (2009b) model income in France as a (deterministic component plus) a fixed effect and first order Markov process for transitory income. In Norway the income process is as yet unknown, warranting further investigation before making assumptions in the empirical model.

Similarly to the aforementioned papers, I assume a permanent component to income and estimate the income process for transitory income. For the bulk of the chapter, income is defined across each year of the child's lifetime. However, in the current analysis a panel of income is constructed for each household across time, from 1967-2004. In the sample there is one observation per household

in each time period, for those who had a child between 1970-1980, hence this is a sample of parents. Household income is calculated as the sum of paternal and maternal income, deflated to 2000 prices.

Two methods are used to understand the time series properties of the income process. First, the variance of income is plotted across the life cycle for the sample of mothers and fathers. If a random walk describes permanent income, the variance of income will be an increasing function of age as each period a new permanent shock hits different parents and persists for the remaining lifetime. Figures 1a) and 1b) plot the variance of income for the mothers and fathers respectively. For the mothers, there is a clear increasing relationship in the variance of earnings across age for the middle periods. During the early years in the labour market and around retirement, the relationship differs. The same is true of fathers, except for some outliers in the 40s. Of course, there are other reasons why variance of income may increase across time, however this evidence does not rule out a random walk permanent component to income.

The second methodology employed, following MaCurdy (1982), seeks to understand the ARMA transitory income process. Consider the model $\ln w_{it} = Z'_{it}\varphi + P_{it} + v_{it}$ where P and v are the permanent and transitory components respectively to log income ($\ln w$) for individual i in period t . Z denotes a set of covariates and φ a vector of coefficients. Permanent income follows a martingale, hence $P_{it} = P_{it-1} + \zeta_{it}$ where ζ denotes the independently and identically distributed (iid) permanent income shock. This section estimates the ARMA(p,q) process for transitory income. In a general model, transitory income is given by $v_{it} = -\sum_{j=1}^p a_j v_{it-j} + \sum_{j=0}^q m_j \varepsilon_{it-j}$ where $m_0 = 1$.

a_t and m_t are the lag coefficients and equal zero if transitory income is iid. ε denotes the transitory income shock to the level of transitory income (v). The orders p and q of the AR and MA components are to be established empirically. The equation can be expressed in terms of the lag parameter L , by $a(L)v_{it} = m(L)\varepsilon_{it}$ where L is the lag operator and $a(L)$, $m(L)$ are lag polynomials. $a(L) = 1 + \sum_{j=1}^p a_j L^j$, $m(L) = \sum_{j=0}^q m_j L^j$ and $L^j x_{it} = x_{i(t-j)}$.

In order to analyse the persistence of the transitory income component, separately to the permanent component, I follow MaCurdy (1982), Meghir & Pistaferri (2004) and Blundell *et al* (2008) and estimate the residuals from first differences in income $\Delta \ln w_{it} = \Delta Z'_{it}\varphi + \zeta_{it} + \Delta v_{it}$, where $\Delta x_t = x_t - x_{t-1}$. The first differenced residuals are described by $Dv_{it} = (1 - L)v_{it}$. The ARMA(p, q) process of the first differenced disturbances is now given by $a(L)Dv_{it} = m(1 - L)\varepsilon_{it}$. This shows that the order of the AR process is the same as in the levels, however first differencing estimates the MA process to the order $q + 1$.

The first stage is to estimate residuals from a system of equations of log wage first differences in period t for individual i . Estimating in a system of equations allows for serial correlation for individuals across years. The controls (Z) are a quadratic in parental age, parental education, marital status and municipality of residence. The second stage is to calculate the autocovariances of residuals (γ) at different lags (k), by calculating $E(v_t v'_{t-k}) = \gamma_k + \omega_t$ where ω is the error in the autocovariance process and $k = \{1, \dots, 8\}$. Autocorrelations at different lags will provide some information to ascertain the ARMA process for transitory income.

Following Granger & Newbold (1986), if the process is a pure MA process, $v_t = \sum_{j=0}^q m_j \varepsilon_{t-j}$ with $m_0 = 1$, the autocovariance is as follows

$$E[v_t v_{t-k}] = E(\varepsilon_t + m_1 \varepsilon_{t-1} + \dots + m_q \varepsilon_{t-q}, \varepsilon_{t-k} + m_1 \varepsilon_{t-k-1} + \dots + m_q \varepsilon_{t-k-q})$$

$$\begin{aligned}
&= \sigma_\varepsilon^2 [m_k + m_1 m_{k+1} + \dots + m_{q-k} m_q] && \text{if } |k| \leq 0 \\
&= 0 && \text{otherwise}
\end{aligned}$$

Autocovariances drop sharply to zero for $k > q$.

On the other hand, if the process is a pure AR process $v_t = \sum_{j=1}^p a_j v_{t-j} + \varepsilon_t$ then autocovariances are given by

$$\begin{aligned}
E(v_t v_{t-k}) &= E\left(\left(\sum_{j=1}^p a_j v_{t-j} + \varepsilon_t\right) v_{t-k}\right) \\
&= E\left((a_1 v_{t-1} + \dots + a_p v_{t-p} + \varepsilon_t)(a_1 v_{t-k-1} + \dots + a_p v_{t-k-p} + \varepsilon_{t-k})\right) \\
&= \sum_{j=1}^p a_j \lambda_{k-j}
\end{aligned}$$

The autocovariances of an AR process at $k > p$ gradually fall to zero.

For each value of k , the autocovariances are estimated in a system of equations and the coefficient on the autocovariance is constrained to be constant in each regression. Two potential difficulties with estimating the autocovariances are firstly that the residuals are estimated in a first stage and secondly that there may be serial correlation across time for individuals. However, MaCurdy (1981) notes that using a seemingly unrelated regression procedure to estimate autocovariances will result in parameters and test statistics that are asymptotically valid.

The results are reported in Table 2, where the autocovariances at different lags are restricted to take the same value across all years of data. The autocovariances are initially negative at one lag but fall close to zero after the first lag, although it remains significant. Again, between lags 2 and 3 there is another sharp drop in the autocovariances and after lag 3, they are no longer significant. This is suggestive of a low order MA process, of the order of 2 or 3 in differences, or of order 1 or 2 in levels.

In conclusion, permanent income will follow a random walk and transitory income an MA process where I will estimate the model initially for a first order process and test the robustness of results to a second order process. This is the similar income process found in the studies mentioned above, suggesting a similar income process in Norway as in the UK and the US.

It is impossible to exactly identify the income process, therefore I additionally test for the sensitivity of the main results to the income process assumed by allowing for less persistence in the permanent component than a martingale.

3.4 Empirical Strategy

3.4.1 Income Process

Log wages ($\ln w$) for individual i in period t are modelled as a linear function of a permanent and a transitory component (denoted P and v respectively) and a set of covariates (Z)

$$\ln w_{it} = Z'_{it} \varphi_t + P_{it} + v_{it} \tag{1}$$

where $i = \dots, N$ and $t = 1, \dots, T$. Permanent income follows a martingale (equation 2) and transitory income is a serially correlated MA(1) process (equation 3), where ζ and ε denote the

permanent and transitory income shocks respectively and θ the first order MA coefficient. Note that section 3.3 provided evidence to suggest that this is a good representation of the true income process for the sample of Norwegian parents.

$$P_{it} = P_{it-1} + \zeta_{it} \quad (2)$$

$$v_{it} = \theta\varepsilon_{it-1} + \varepsilon_{it} \quad (3)$$

Both permanent and transitory shocks are assumed to have a mean of zero and be uncorrelated with each other, $E(\zeta_{it}) = E(\varepsilon_{it}) = E(\zeta_{it}\varepsilon_{it}) = 0$; $t = 1, \dots, T$, $i = 1, \dots, N$.

Define y as log income with the effect of the covariates removed in a first stage, $y_{it} = \ln \hat{w}_{it} - Z'_{it}\varphi_t = P_{it} + \theta\varepsilon_{it-1} + \varepsilon_{it}$. Substituting in for the permanent income component gives

$$y_{it} = P_{i0} + \sum_{s=1}^t \zeta_{is} + \theta\varepsilon_{it-1} + \varepsilon_{it} \quad (4)$$

Income in period t is the sum of P_0 , the initial level of permanent income, representing an unobservable endowment, or initial condition, current permanent and transitory shocks, all past permanent shocks and transitory shocks at one lag.

3.4.2 Child Human Capital Production Function

Appendix 3.1 details a model whereby a stock of child human capital (h) accumulates at the end of a lifetime of parental investment. Equation (A5) showed the stock of human capital in the final period, T , to be a function of permanent and transitory shocks in each period of life. The model saw parents optimising levels of parental investment and consumption to maximise their utility, which is a function of the child's stock of human capital in period T , hence human capital has a subscript i relating both to child and parent. For ease, let $h_{iT} = e_{iT} - Z'_{iT}\delta$; where e is the raw measure of human capital. Child human capital is modelled as the sum of parental income in each period, where coefficients vary for permanent and transitory components, a set of parental traits Z , a child level idiosyncratic error u_{iT} and initial endowment, μ_{i0} (for example genes or parental unobservable characteristics).

$$h_{iT} = \sum_{t=0}^T \beta_t^P P_{it} + \sum_{t=0}^T \beta_t^T v_{it} + \mu_{i0} + u_{iT} \quad (5)$$

Repeatedly substituting for P_{it} and substituting for v_{it} gives

$$h_{iT} = \sum_{t=0}^T \beta_t^P \left(P_{i0} + \sum_{s=1}^t \zeta_{is} \right) + \sum_{t=0}^T \beta_t^T (\theta\varepsilon_{it-1} + \varepsilon_{it}) + \mu_{i0} + u_{iT} \quad (6)$$

I make the assumption that the income shocks are uncorrelated with the child idiosyncratic term, u and that u is mean zero; $E(u_{iT}\varepsilon_{it}) = E(u_{iT}\zeta_{it}) = E(u_{iT}) = 0$; $t = 1, \dots, T$, $i = 1, \dots, N$. However, $E(P_{i0}\mu_{i0}) \neq 0$. Both P_{i0} and μ_{i0} cannot be separately observed, as they are initial conditions causing an identification problem, the consequences of which can be seen in the identification section below.

3.4.3 Identification

Cohort-Local Labour Market Level Analysis It is possible to identify the timing of parental income by exploiting variation in the second order moments of income across cohorts of children in different labour markets. This method is similar to Blundell *et al* (2008) and Adda *et al* (2006), both of whom used time variation in variance of shocks.

Meghir & Pistaferri (2004) show that it is possible to identify the moments of the income process using information on income alone. Given the income process above, the covariance matrix of income at different lags is given at a cohort and labour market level (c) by

$$\begin{aligned} cov(y_{it,c}, y_{it-s,c}) &= \begin{cases} \sum_{l=1}^{t-s} \sigma_{\zeta_l,c}^2 + \sigma_{P_0,c}^2 + \theta^2 \sigma_{\varepsilon_{t-1,c}}^2 + \sigma_{\varepsilon_t,c}^2 & \text{if } s = 0 \\ \sum_{l=1}^{t-s} \sigma_{\zeta_l,c}^2 + \sigma_{P_0,c}^2 + \theta \sigma_{\varepsilon_{t-1}}^2 & \text{if } s = 1 \\ \sum_{l=1}^{t-s} \sigma_{\zeta_l,c}^2 + \sigma_{P_0,c}^2 & \text{if } |s| > 1 \end{cases} \end{aligned} \quad (7)$$

where $\sigma_{\zeta_t}^2$ and $\sigma_{\varepsilon_t}^2$ denote the variance of permanent and transitory shocks in period t , respectively and the subscript c denotes the labour market. All variance terms are identified, with the exception of $\sigma_{\varepsilon_T}^2, \sigma_{\zeta_T}^2$, which are not separately identifiable. For this reason, an additional year of data is included at the end of the time series.

The covariance matrix between income in each year of the child's lifetime and human capital is given below.

$$\begin{aligned} cov(y_{it}, h_{iT}) &= \begin{cases} \left(\sum_{l=0}^T \beta_l^P \right) \sigma_{P_0,c}^2 + \sum_{s=1}^t \left(\sum_{l=s}^T \beta_l^P \right) \sigma_{\zeta_s,c}^2 + \theta \left(\beta_{t-1}^T + \theta \beta_t^T \right) \sigma_{\varepsilon_{t-1}}^2 \\ \quad + \left(\beta_t^T + \theta \beta_{t+1}^T \right) \sigma_{\varepsilon_t}^2 + \sigma_{\mu_0 P_0} & \text{if } t = 1, \dots, T-1 \\ \left(\sum_{l=0}^T \beta_l^P \right) \sigma_{P_0,c}^2 + \sum_{s=1}^t \left(\sum_{l=s}^T \beta_l^P \right) \sigma_{\zeta_s,c}^2 + \theta \left(\beta_{t-1}^T + \theta \beta_t^T \right) \sigma_{\varepsilon_{t-1}}^2 \\ \quad + \beta_t^T \sigma_{\varepsilon_t}^2 + \sigma_{\mu_0 P_0} & \text{if } t = T \end{cases} \end{aligned} \quad (8)$$

where $\sigma_{\mu_0 P_0}$ denotes the correlation between initial condition in income (P_0) and child human capital (μ_0). As noted above, the two initial conditions cannot be separately identified and consequently, β_0^P is not identified. However, all other parameters are identified. θ is estimated empirically.

I exploit variation in the variance of shocks across cohorts and labour markets for identification. It allows me to estimate parameters on the income shocks that differ across years of the child's lifetime. To be specific, the inherent identification assumption is that second order moments of the permanent and transitory income process differ across cohorts and labour markets, but that the effect of these shocks upon child outcomes is homogeneous.

Note that measurement error is omitted from the model to date. Meghir & Pistaferri (2004) estimate that between a quarter and a third of the transitory income shock variation is due to measurement error in the Panel Study of Income Dynamics (PSID). However, the bias is likely to be smaller in the current sample, as income is recorded from administrative data. However, zero measurement error is very unlikely and future research is planned to incorporate it into the analysis.

Parametric Analysis A second method allows identification of the parameters of the child human capital production function without analysing at the level of the cohort and labour market, by exploiting higher order moments than the second order. Rather than estimate the distribution of the income shocks, a method similar to Cunha *et al* (2006) would assume a distribution, then estimate the effect of permanent and transitory income shocks using simulated maximum likelihood method. This is the future research strategy.

3.5 Results

3.5.1 Variance of income shocks

Table 3 reports estimates of the variance of the initial level of permanent income and per period transitory and permanent income shocks. A Diagonally weighted minimum distance procedure generates the estimates, details of which are in Appendix 3.2.

Variances of the permanent and transitory income shocks and of the initial permanent income level are estimated within each cohort (from 1970-1980) and labour market (of which there are 90) and across the age of the child. This gives in total 35,640 estimates of the variance particular components of income. For ease of explication the table describes the sample statistics for the variances, listing the mean, standard deviation, minimum and maximum across the 990 cohort-labour market cells. A potential worry with aggregating to the level of the cohort-labour market, is that much of the variation in income shocks may exist across, not within labour markets or cohorts. If this were the case, the final estimates of the effect of income shocks on child human capital will not be representative to the population as a whole. Therefore, in the final column, the variance was calculated for the entire sample, allowing comparison of cohort-labour market level variances with population variances.

The first row of Table 3 shows that the variance of log initial permanent income is high, at 0.0477. This means that, even controlling for parental education, a polynomial of age and marital status, the initial condition in permanent income has a standard deviation of 0.2184. This is reassuring for generalisation of this study to other countries, as Norway is often considered to have very little inequality. There is a deal of variation across labour markets, with up to 0.1449 standard deviations in log income. The population level variance is similar to the labour market mean, at 0.0679, suggesting that exploiting labour market differences in inequalities of shocks for identification is reasonable.

The variance of transitory shocks at ages 0-17 are reported in the next rows. These tend to be fairly stable until the final years of child age when they fall although recall from above that it is not possible to identify the final variance of transitory or permanent shocks in the final period. The variance of permanent shocks is much smaller, as would be expected. Permanent shocks last for a lifetime, therefore a small shock can be very important. The variance on average ranges between 0.0051 and 0.0140 across the years. Again, the cohort-labour market variances resemble closely the population variances.

3.5.2 Effect of shocks on adolescent outcomes

The truly innovative aspect of this paper is the application of methodology which decomposes shocks into transitory and permanent components and subsequently estimates the effect of the shocks upon

child outcomes. This section documents the results, examining whether the realisation of transitory and permanent income shocks will have a heterogeneous effect upon child outcomes, depending upon the age of the child at realisation. The variances of transitory and permanent income shocks across child age are applied to equation (8) to estimate the effect of the income shocks upon child human capital outcomes. The form of the human capital production function in equation (6) allows the effect of income shocks to vary across child age. Before estimating this complex model, it is interesting to restrict the coefficients to instead be homogenous across child age, estimating the following function

$$h_{iT} = \delta_0 P_{i0} + \delta_1 \zeta_{it} + \delta_2 \varepsilon_{it} + \mu_{i0} + u_{iT}$$

A panel data is constructed at the cohort - labour market - child age level. Regression results are reported in Table 4. Note that as permanent income shocks do not exist in period 0, this year is dropped from the regressions, resulting in 16830 observations (90 labour markets, 11 cohorts, 17 years of child age). The dependent variables have been standardised such that the coefficients are expressed in standard deviations of the outcomes. Two different functional forms are estimated. In columns 1, 3, 5, 7 & 9, the human capital outcomes are estimated as a linear function of the initial level of permanent income (P0), a transitory income shock and a permanent income shock. Columns 2, 4, 6, 8 & 10 include an interaction term of permanent (transitory) shocks with child age thus allowing some heterogeneity in the effect of income shocks across child age.

For all outcomes, row 1 shows that there is a strong and significant coefficient on the initial level of permanent income. This is independent to inclusion of the set of controls. income is logged, thus the coefficient is interpreted as a doubling of P0 raises years of schooling, probability of dropping out of high school, college attendance, ability and health by up to 1.155, 0.197, 0.150, 0.737 and 0.175 standard deviations respectively. The first column of data for each outcome shows that the effect of transitory income shocks is to improve child human capital¹⁸ (columns 1, 3, 5, 7 & 9), however once the interaction between transitory shocks and child age is added, the level effect becomes negative or insignificant. The interaction between transitory income shock and child age is positive - suggesting that initially there is a negative effect of transitory shocks which is increasing across child age. Permanent shocks have a larger effect on child outcomes, as would be expected from the permanent income hypothesis. The interaction between permanent shocks and child age is negative, suggesting a declining impact of permanent shocks across child age, although the interaction term is insignificant.

Next, the fully flexible model is estimated and the age specific coefficients on the permanent and transitory income shocks and initial permanent income are reported in Table 5. The coefficients and standard errors have been adjusted to represent standard deviations in the dependent variable. The results are easier to see in graphical form. Figures 2a-2j plot the coefficients across child age, again in standard deviations of the child outcome. Note that the estimates are plotted only up to age 16, as it is not possible to separately identify a transitory shock from a permanent shock in the final period of observation.

Figure 2a plots the coefficients on permanent income shocks realised in every year of the child's lifetime, upon years of completed child schooling. Additionally, the effect of the log initial level of permanent income (P0) is plotted. This is positive, with a coefficient of 0.906, suggesting that a doubling of initial permanent income raises schooling by nearly a whole standard deviation, or 2.34

¹⁸drop out is a negative human capital indicator, hence the sign of the coefficient is the opposite to other outcomes

years. As I interpret the initial permanent income level as the initial condition, or parental fixed effect, this is large and statistically significant.

Turning now to the effect of permanent income shocks, the effect of shocks falls between ages 2-4, flattens out for the mid-childhood ages of 6-10 (with the exception of age 7) and declines again to zero up to age 16. A permanent shock realised during early years has a greater effect on child's schooling than a shock realised at age 16. This is intuitive, given that the early permanent shock shifts household wealth forever and therefore drive income realisations for all future periods. With reference to the discussion of the optimal timing of investment, Cunha & Heckman (2008) suggest neurological arguments lead to the return to parental investment early in a lifetime being higher than later in a lifetime. In this chapter, a permanent income shock realised early in a child's lifetime has a larger effect on schooling but mainly because the effect of the shock lasts for a lifetime.

Figure 2b plots the estimates of the effect of transitory income shocks in each year of the child's lifetime. The transitory income shocks are logged, hence interpretation of the coefficient at age 1 is that a doubling of transitory income shock raises schooling by 0.4 standard deviations, or around a year. This effect upon schooling tends to increase slightly across child age. From Table 5, the effect of doubling the transitory shock at age 1 and age 16 is to raise schooling by 0.427 and 0.915 standard deviations respectively. This suggests that positive transitory income shocks are slightly more important in raising child education when they are realised during adolescence, than during the early years. The magnitude of the coefficients on transitory income shocks upon child outcomes is lower than for permanent shocks, which is consistent with the permanent income hypothesis. In the consumption literature, a permanent income shock should change current consumption by the annuity value of the shock, whereas only a proportion $1/T$ of the transitory shock is consumed. Interestingly, the coefficients of the permanent and transitory income shocks converge towards the final period, as a permanent shock in the final period of human capital investment should drive human capital to the similar degree as a transitory income shock in that period.

The estimates of income shocks upon the probability of a child dropping out from high school are plotted in Figures 2c and 2d. In this case, the human capital measure is indicating a negative achievement, which is why the coefficients are negative. Doubling the initial level of income lowers the probability of a child dropping out of high school by 0.6795 standard deviations. A positive permanent income shock lowers the probability of dropping out from high school. This effect again is initially increasing in magnitude, but then declines across the age of the child from age 6. Thus as with the years of schooling, there is a slightly different pattern for the early years. The effect of transitory income shocks again are slightly larger early in a child's life, although the confidence intervals do not reject a linear relationship. The conclusion for this outcome is that household transitory income shocks lower the probability of dropping out of high school by around 0.4-0.85 standard deviations throughout the lifetime of the child.

When looking at Figure 2e, a log increase in the initial level of permanent income increases college/university attendance by 0.8549 standard deviations - a substantive effect. Similarly to the other outcomes, the effect of permanent income shocks upon college is initially flat, then declines across time. A different effect of transitory income shocks is seen on college attendance. The curve initially increases, suggesting that a transitory shock at age 1 will drive college attendance by a lesser extent than a shock at age 6. From 6-16, there is a flat relationship with a coefficient of around 0.6 standard deviations.

The final two outcomes - ability and health - refer to results for tests similar to those often used in the literature on human capital accumulation, when information on schooling outcomes are not available. Log initial permanent income raises achievement of ability and health by 0.71 and 0.64 standard deviations respectively. The effect of permanent income shocks, whilst generally declining like the other outcomes, are noisy for both outcomes. It could be that scores aggregated to a 9 point scale do not provide enough variation to adequately estimate robust effects of the timing of permanent income shocks. On the other hand, the noise could be generated by the measurement error in the variable itself, as an imprecise measure of human capital.

There is a definite increasing relationship between the effect of transitory income shocks upon ability across age, whereas for health, similarly to the drop out outcome, the pattern is flatter.

To summarise, there is a large coefficient on the initial level of permanent income, or parental fixed effect, suggesting significant heterogeneity in child outcomes which is determined at the birth of the child. This is interesting, as often Norway is considered a very equal country, with a compressed income distribution. However, the evidence suggests large variance in outcomes driven by a family initial condition. Permanent income shocks drive child human capital significantly. There was noted a declining relationship across child age, such that shocks realised at age 1 will have a larger effect than those realised at ages 2, 3 etc., up until adolescence where a permanent income shock often has an insignificant effect on child human capital. This pattern is to be expected, as permanent income shocks last for a lifetime, hence a shock realised at age 1 will last for the entire childhood, rather than a shock realised towards the period of adolescence. However, often the patterns vary from this rule when the shocks are realised early in the child's lifetime. It is at age 7 that children start school in Norway, so this jump may well represent a change in maternal labour supply which shifts permanent income. This raises the question of whether the shocks as estimated in this chapter, are truly shocks, or unanticipated by the households themselves. If the shock just picks up an expected change in labour supply, then the resulting change in child outcomes (through adjusted parental investment) would not be as the model predicted. One way to overcome this problem would be to run the analysis solely on paternal income, which fluctuates less around child schooling. These results are reported in the following section.

Transitory income shocks have an effect which is generally constant, or increasing across age such that shocks realised during early childhood have a smaller effect on outcomes than those realised during adolescence. This fits in with results of Chapter 2 of the thesis.

3.5.3 Paternal Income

It is interesting to repeat the above analysis, looking at paternal income shocks rather than shocks to the sum of maternal and paternal income. The relationship between both permanent and transitory income shocks and child outcomes across the age of the child, may differ somewhat to the previous estimates.

In terms of permanent income, recall in the results above that the effect of permanent income shock tended to decline from the age of 6 or 7 until age 16, but that there was a slightly different pattern for the early years. As Norwegian children start school at age 7, the difference in the effect could be caused by a change in maternal labour supply which shifts permanent income. Figures 3a-3j plot the effect of permanent and transitory shocks to paternal income upon child outcomes, across child age. These correspond to the coefficients reported in Table 6. Indeed, Figures 3a, c,

e & g show that when only paternal income is considered, there is an effect of permanent income shocks which tends to fall from age 1. In 3i there is a noisy relationship for health however. In general now, permanent shocks have a large effect early in the lifetime of the child and this effect falls smoothly across child age, to zero during the later adolescent years.

It may be the case that the effect of paternal transitory income shocks upon child human capital differs from that of household transitory income shocks. A mother may insure the family from a negative paternal income shock, by increasing her investment in the child. This could take two forms, increasing either the number of hours she works, or the amount of time she spends with the child. If this were the case, then a household income shock, the sum of maternal and paternal income, would have a larger effect than a paternal shock alone, as the effect of the latter could be reduced by the behaviour of the mother. The figures show evidence which would fit with the idea of maternal insurance against paternal income shocks.

Firstly Table 6 reports magnitudes for the coefficients of paternal transitory income shocks that are lower than the household equivalents of Table 5. For example, a 1 log point change in the paternal transitory income shock at age 1 raises education by 0.204 standard deviations, whereas for a household transitory shock, the coefficient is 0.427. Additionally, for years of schooling and college attainment, in figures 3b and 3f respectively, there is an inverse-u shaped curve in the effect of transitory shocks across child age. The effect of transitory shocks initially increases across child age between age 1-6, flattens out for the middle years, then falls again from age 11-16. That is, there is a larger difference between the effect of transitory shocks for the early years and the teenage years. These patterns fit with the idea that mothers are available to spend more time with children before they start school or during adolescence, to smooth parental investment in child human capital despite paternal transitory income shocks.

3.6 Robustness Checks

A structural model for income generates the results presented above. Section 3.3 undertook tests to ascertain the correct income process for the sample of parents in Norway. However, it is worthwhile changing the particular form of the income process, to understand the sensitivity of the results to the particular choice. In the bulk of the chapter, permanent income follows a random walk and transitory income a MA process of order 1. Section 3.6.1 relaxes the assumption of a unit root in permanent income and Section 3.6.2 allows transitory income to follow an MA(2) process.

3.6.1 Relaxing the Assumption of a Unit Root in Permanent Income

This chapter made the common assumption that permanent income followed a random walk, or has a unit root so $\rho = 1$ in the model $P_{it} = \rho P_{it-1} + \zeta_{it}$; $0 \leq \rho \leq 1$. In order to test whether the results are sensitive to this particular functional form assumed, a different model is estimated which allows a coefficient on lagged permanent income to differ from zero. As a robustness check, the model above is estimated, with $\rho = 0.9$, so that the permanent income shocks enter future income realisations, but die out across time. Repeatedly substituting for permanent income leads to the following income process rather than equation (4)

$$y_{it} = \rho^t P_{i0} + \sum_{j=1}^t \rho^{t-j} \zeta_{ij} + \theta \varepsilon_{it-1} + \varepsilon_{it} \quad (9)$$

The results, available upon request, show no significant difference once the assumption of martingale is relaxed. The findings are robust to a different specification for permanent income.

3.6.2 Assuming MA(2) Process for Transitory Income

Section 3.3 estimated a process for transitory income that was described by an MA(1) or an MA(2). This section tests the sensitivity of the effect of permanent and transitory income upon child human capital to the order of the MA process, by extending to a second order process. That is, $v_{it} = \varepsilon_{it} + \theta_1\varepsilon_{it-1} + \theta_2\varepsilon_{it-2}$. The covariance matrices of income becomes

$$\begin{aligned} cov(y_{it}, y_{is}) = & \begin{aligned} & \sum_{l=1}^{t-s} \sigma_{\zeta_{l,c}}^2 + \sigma_{P_{0,c}}^2 + \theta_1^2 \sigma_{\varepsilon_{t-1,c}}^2 + \theta_2^2 \sigma_{\varepsilon_{t-2,c}}^2 + \sigma_{\varepsilon_{t,c}}^2 & \text{if } s=0 \\ & \sum_{l=1}^{t-s} \sigma_{\zeta_{l,c}}^2 + \sigma_{P_{0,c}}^2 + \theta_1 \sigma_{\varepsilon_{t-1,c}}^2 + \theta_1 \theta_2 \sigma_{\varepsilon_{t-2,c}}^2 & \text{if } s=1 \\ & \sum_{l=1}^{t-s} \sigma_{\zeta_{l,c}}^2 + \sigma_{P_{0,c}}^2 + \theta_2 \sigma_{\varepsilon_{t-2,c}}^2 & \text{if } s=2 \\ & \sum_{l=1}^{t-s} \sigma_{\zeta_{l,c}}^2 + \sigma_{P_{0,c}}^2 & \text{if } |s|>2 \end{aligned} \end{aligned} \quad (10)$$

Again, allowing such a change leads to exactly the same conclusions in the bulk of the chapter. For household income shocks, permanent income shocks have an effect that declines across child age from 6 onwards and coefficients on transitory income shocks increase across child age for schooling, college attendance and ability, but are flat for high school drop out and health. The conclusion from this section is that the results of this chapter are robust to two quite substantial changes in the assumed income process generating the estimates for the variance of shocks, which are used to identify the role of permanent and transitory income shocks upon child outcomes.

3.7 Conclusion

In this chapter, I have estimated the income process in Norway, for the population of parents having children born in the 1970s. Similarly to studies of other countries, Norwegian individuals' income process is best described by the sum of a deterministic, permanent and transitory component where permanent income is a martingale and transitory income follows an MA(1). Given this model for income, the next stage was to estimate annual deviations of log household income from a life cycle profile, and decompose these into yearly permanent and transitory income shocks. The effect of the shocks was estimated upon a range of cognitive and non-cognitive child outcomes, to understand in which stages of child development the income shocks drive human capital achievement. There is evidence that permanent income shocks have a stronger effect on child outcomes early in life, and the effect falls to zero as the child ages. There was a slight divergence in this pattern for early shocks, before the child started schooling. Running the analysis using just father's income led to a large effect of an age 1 permanent shock, which declined smoothly across age. This result was expected and mechanical, because a permanent shock drives the household wealth and should therefore drive human capital investment. Therefore, a positive early shock will raise this investment for more periods than a later shock.

In terms of transitory income shocks, for three outcomes - years of schooling, college attendance and ability, there is an increasing relationship with the effect of the shock across child age. For high school drop outs and health, the relationship was flat. Some evidence of intrahousehold insurance

against transitory income shocks was observed, as the effect of a paternal income shock was smaller in magnitude than for a shock to the entire household, suggesting the mother may respond to paternal income shocks by increasing investment in children.

There is a question of generalisability to address, as Norway is richer and has a lower level of inequality than average. However, as the results still pointed to an effect of income shocks on child outcomes, even this government is not fully insuring the households against income shocks. Therefore, whilst a future research agenda is to carry out the same analysis on countries with a less supportive welfare state, this chapter still provides evidence of a lack of full insurance against household income fluctuations, albeit for a sample of households with access to relatively a generous government insurance mechanism.

Finally, future research is planned to extend this chapter in an important way. The assumption that income is linear in the human capital production function is empirically rejected by the analysis in Chapter 2, which finds strong evidence of dynamic complementarities in the return to income. An extension would therefore allow a nonlinear human capital production function, either by identifying off higher order moments (see Bonhomme & Robin, 2009a) or by moving away from a nonparametric estimation of the second order moments of the income process and estimating through a simulated maximum likelihood methodology (see Cunha *et al*, 2006).

Table 1: Sample Descriptives

Variable	N	Mean	Std. Dev.	Min	Max
Child's education	616210	12.53	2.34	5	21
Drop out High School	616210	0.26	0.44	0	1
College	616210	0.37	0.48	0	1
Ability (males)	290883	5.09	1.76	1	9
Health (males)	311163	8.35	1.65	1	9

Table 2: Autocovariances of residuals from log income differences $\Delta \ln w_{it} - \Delta Z'_{it}\beta$

Lag	$k=0$	$k=1$	$k=2$	$k=3$	$k=4$	$k=5$	$k=6$	$k=7$	$k=8$
Autocovariance	0.0610**	-0.0145**	-0.0041**	-0.0028**	-0.0009	-0.0011	-0.0001	-0.0005	0.0012
Standard error	(0.0044)	(0.0018)	(0.0012)	(0.0010)	(0.0011)	(0.0010)	(0.0010)	(0.0011)	(0.0009)

Table 3: Variances of Initial Permanent Income, Transitory and Permanent Income Shocks

	Cohort-Labour Market Variances				Total sample
	Mean	Standard Deviation	Min	Max	Variance
$\sigma_{P_0}^2$	0.0477	0.0210	0.0000	0.2196	0.0679
$\sigma_{\varepsilon_0}^2$	0.1036	0.0576	0.0061	0.4686	0.1144
$\sigma_{\varepsilon_1}^2$	0.0745	0.0461	0.0011	0.5873	0.0961
$\sigma_{\varepsilon_2}^2$	0.0801	0.0533	0.0000	0.3831	0.0989
$\sigma_{\varepsilon_3}^2$	0.0785	0.0581	0.0000	0.6326	0.0979
$\sigma_{\varepsilon_4}^2$	0.0870	0.0695	0.0000	0.6320	0.1020
$\sigma_{\varepsilon_5}^2$	0.0947	0.0760	0.0000	0.5527	0.1020
$\sigma_{\varepsilon_6}^2$	0.1006	0.0880	0.0000	1.0031	0.1004
$\sigma_{\varepsilon_7}^2$	0.1061	0.0860	0.0000	0.6306	0.0996
$\sigma_{\varepsilon_8}^2$	0.1104	0.0957	0.0000	0.8505	0.0990
$\sigma_{\varepsilon_9}^2$	0.1153	0.1040	0.0000	1.0270	0.0955
$\sigma_{\varepsilon_{10}}^2$	0.1008	0.0855	0.0000	0.6066	0.0917
$\sigma_{\varepsilon_{11}}^2$	0.1085	0.0908	0.0000	0.6728	0.0877
$\sigma_{\varepsilon_{12}}^2$	0.1048	0.0961	0.0000	0.6830	0.0811
$\sigma_{\varepsilon_{13}}^2$	0.0913	0.0909	0.0000	0.7582	0.0713
$\sigma_{\varepsilon_{14}}^2$	0.0763	0.0822	0.0000	0.9546	0.0656
$\sigma_{\varepsilon_{15}}^2$	0.0560	0.0721	0.0000	0.5826	0.0585
$\sigma_{\varepsilon_{16}}^2$	0.0369	0.0724	0.0000	1.1912	0.0437
$\sigma_{\varepsilon_{17}}^2$	0.0307	0.0393	0.0000	0.2899	0.0291
$\sigma_{\zeta_1}^2$	0.0051	0.0077	0.0000	0.0527	0.0035
$\sigma_{\zeta_2}^2$	0.0064	0.0087	0.0000	0.0732	0.0098
$\sigma_{\zeta_3}^2$	0.0063	0.0089	0.0000	0.0697	0.0076
$\sigma_{\zeta_4}^2$	0.0060	0.0097	0.0000	0.1357	0.0069
$\sigma_{\zeta_5}^2$	0.0055	0.0085	0.0000	0.0772	0.0064
$\sigma_{\zeta_6}^2$	0.0057	0.0092	0.0000	0.1068	0.0048
$\sigma_{\zeta_7}^2$	0.0055	0.0085	0.0000	0.0618	0.0042
$\sigma_{\zeta_8}^2$	0.0062	0.0118	0.0000	0.2162	0.0044
$\sigma_{\zeta_9}^2$	0.0058	0.0100	0.0000	0.0835	0.0048
$\sigma_{\zeta_{10}}^2$	0.0068	0.0127	0.0000	0.0990	0.0057
$\sigma_{\zeta_{11}}^2$	0.0074	0.0156	0.0000	0.1449	0.0053
$\sigma_{\zeta_{12}}^2$	0.0096	0.0189	0.0000	0.1342	0.0069
$\sigma_{\zeta_{13}}^2$	0.0124	0.0301	0.0000	0.2593	0.0076
$\sigma_{\zeta_{14}}^2$	0.0140	0.0346	0.0000	0.3224	0.0102
$\sigma_{\zeta_{15}}^2$	0.0111	0.0271	0.0000	0.3431	0.0028
$\sigma_{\zeta_{16}}^2$	0.0086	0.0252	0.0000	0.2613	0.0000
$\sigma_{\zeta_{17}}^2$	0.0307	0.0393	0.0000	0.2899	0.0337

Table 4: Estimates of the Effect of The Timing of Household Transitory Shocks, Initial Permanent Income (P0) and Permanent Shocks upon Child Outcomes: With Restrictions on Coefficients Across Child Age

	Education		Dropout		College		Ability		Health	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
P0	1.135*** (0.142)	1.155*** (0.141)	-0.195*** (0.019)	-0.197*** (0.019)	0.146*** (0.033)	0.150*** (0.032)	0.726*** (0.143)	0.737*** (0.144)	0.173* (0.104)	0.175* (0.103)
Transitory	0.303*** (0.030)	-0.127** (0.049)	-0.047*** (0.007)	0.011 (0.011)	0.054*** (0.006)	-0.022*** (0.008)	0.171*** (0.026)	-0.097** (0.046)	0.022 (0.022)	-0.040 (0.029)
Transitory*age		0.048*** (0.004)		-0.006*** (0.001)		0.008*** (0.001)		0.029*** (0.004)		0.007*** (0.002)
Permanent	0.440*** (0.060)	0.234 (0.212)	-0.081*** (0.013)	-0.037 (0.040)	0.037*** (0.011)	-0.025 (0.040)	0.238*** (0.073)	0.430* (0.248)	0.042 (0.048)	0.262 (0.172)
Permanent *age		-0.003 (0.016)		-0.001 (0.003)		0.001 (0.003)		-0.024 (0.018)		-0.018 (0.012)
N	16830	16830	16830	16830	16830	16830	16830	16830	16830	16830

Note: N=90 labour markets * 11 cohorts * 17 years of child age (permanent shocks are absent for age 0). Standard errors in parentheses. Dependent variables have been standardised such that the coefficients are expressed in standard deviations of the outcomes. The coefficients on transitory (permanent) income shocks are restricted to be constant across child age in the first specification, and include an additional term of the interaction between the shock and child age in the second.

Table 5: Estimates of the Effect of The Timing of Household Transitory Shocks, Initial Permanent Income (P0) and Permanent Shocks upon Child Outcomes: Without Restrictions on Coefficients Across Child Age

Outcome	Child Age																
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
Education	P0	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	0.9406 (0.0186)	
	Transitory	0.0427 (0.0103)	0.0487 (0.0086)	0.0530 (0.0077)	0.0504 (0.0068)	0.0714 (0.0062)	0.0585 (0.0061)	0.0594 (0.0057)	0.0714 (0.0057)	0.0598 (0.0053)	0.0637 (0.0065)	0.0816 (0.0056)	0.0607 (0.0060)	0.0598 (0.0063)	0.0632 (0.0071)	0.0534 (0.0082)	0.0915 (0.0112)
	Permanent	0.5671 (0.0786)	0.6103 (0.0688)	0.5226 (0.0650)	0.4171 (0.0620)	0.4308 (0.0654)	0.6551 (0.0641)	0.4308 (0.0679)	0.4457 (0.0585)	0.5543 (0.0577)	0.3372 (0.0453)	0.3782 (0.0414)	0.2966 (0.0541)	0.1453 (0.0212)	0.1026 (0.0180)	0.1252 (0.0315)	0.0180 (0.0364)
Dropout	P0	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	-0.6795 (0.0178)	
	Transitory	-0.0400 (0.0106)	-0.0468 (0.0089)	-0.0425 (0.0079)	-0.0357 (0.0071)	-0.0641 (0.0065)	-0.0427 (0.0063)	-0.0416 (0.0059)	-0.0500 (0.0059)	-0.0398 (0.0055)	-0.0475 (0.0063)	-0.0568 (0.0058)	-0.0432 (0.0060)	-0.0502 (0.0063)	-0.0532 (0.0069)	-0.0500 (0.0078)	-0.0852 (0.0112)
	Permanent	-0.4273 (0.0757)	-0.4432 (0.0689)	-0.3477 (0.0661)	-0.3045 (0.0611)	-0.3114 (0.0666)	-0.5068 (0.0655)	-0.3636 (0.0666)	-0.3682 (0.0575)	-0.4091 (0.0577)	-0.2591 (0.0455)	-0.2659 (0.0409)	-0.2166 (0.0330)	-0.166 (0.0206)	-0.0980 (0.0178)	-0.1434 (0.0305)	-0.0732 (0.0368)
College	P0	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	0.8549 (0.0187)	
	Transitory	0.0267 (0.0106)	0.0568 (0.0087)	0.0511 (0.0077)	0.0494 (0.0069)	0.0582 (0.0063)	0.0617 (0.0062)	0.0577 (0.0057)	0.0615 (0.0056)	0.0608 (0.0053)	0.0621 (0.0065)	0.0826 (0.0055)	0.0553 (0.0058)	0.0511 (0.0063)	0.0540 (0.0070)	0.0432 (0.0080)	0.0747 (0.0109)
	Permanent	0.5134 (0.0798)	0.5619 (0.0709)	0.4319 (0.0663)	0.4781 (0.0637)	0.4781 (0.0657)	0.6522 (0.0643)	0.4120 (0.0690)	0.4010 (0.0584)	0.4980 (0.0571)	0.3019 (0.0447)	0.3063 (0.0401)	0.2622 (0.0333)	0.1080 (0.0211)	0.1113 (0.0181)	0.0672 (0.0302)	-0.0211 (0.0355)
Ability	P0	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	0.7114 (0.0252)	
	Transitory	0.0306 (0.0148)	0.0370 (0.0124)	0.0210 (0.0109)	0.0264 (0.0098)	0.0580 (0.0090)	0.0642 (0.0089)	0.0394 (0.0085)	0.0306 (0.0082)	0.0304 (0.0076)	0.0291 (0.0091)	0.0468 (0.0079)	0.0483 (0.0082)	0.0414 (0.0091)	0.0531 (0.0095)	0.0480 (0.0107)	0.0784 (0.0150)
	Permanent	0.6892 (0.1068)	0.6136 (0.0943)	0.5659 (0.0926)	0.7330 (0.0885)	0.3659 (0.0920)	0.5841 (0.0886)	0.4409 (0.0943)	0.6989 (0.0784)	0.3091 (0.0790)	0.2381 (0.0653)	0.1875 (0.0597)	0.1653 (0.0444)	0.1273 (0.0290)	0.0124 (0.0252)	0.1125 (0.0424)	-0.0558 (0.0489)
Health	P0	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	0.0642 (0.0216)	
	Transitory	-0.0002 (0.0133)	0.0026 (0.0112)	-0.0076 (0.0099)	-0.0039 (0.0091)	0.0032 (0.0082)	0.0026 (0.0080)	0.0101 (0.0079)	0.0013 (0.0075)	-0.0042 (0.0064)	0.0024 (0.0075)	0.0023 (0.0069)	-0.0163 (0.0072)	-0.0152 (0.0079)	-0.0022 (0.0081)	-0.0164 (0.0087)	0.0035 (0.0121)
	Permanent	0.0453 (0.0903)	0.0297 (0.0794)	0.1242 (0.0770)	0.0921 (0.0715)	0.0344 (0.0721)	0.0104 (0.0764)	0.2006 (0.0788)	0.0891 (0.0642)	0.0532 (0.0624)	0.0927 (0.0510)	0.0388 (0.0474)	0.0057 (0.0358)	0.0197 (0.0228)	-0.0068 (0.0202)	0.0371 (0.0337)	-0.0482 (0.0392)

Note: standard errors in parentheses. Coefficients and standard errors represent standard deviations in dependent variables. P0 denotes initial level of permanent income. Estimates using household income. DWMD estimates. Permanent income follows a martingale and transitory income a MA(1) process. Education denotes years of schooling, dropout indicates leaving school at the compulsory age and college attendance at college/university. Ability and health were measured for males in the Armed Forces Test

Table 6: Estimates of the Effect of The Timing of Paternal Transitory Shocks, Initial Permanent Income (P0) and Permanent Shocks upon Child Outcomes.

Outcome	Child Age																
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
Education	P0	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	0.8855 (0.0185)	
	Transitory	0.0204 (0.0084)	0.0253 (0.0072)	0.0427 (0.0071)	0.0388 (0.0061)	0.0603 (0.0059)	0.0513 (0.0055)	0.0517 (0.0049)	0.0598 (0.0047)	0.0573 (0.0046)	0.0483 (0.0043)	0.0590 (0.0041)	0.0500 (0.0045)	0.0371 (0.0043)	0.0299 (0.0046)	0.0304 (0.0046)	0.0420 (0.0061)
	Permanent	0.4662 (0.0709)	0.5218 (0.0641)	0.3654 (0.0547)	0.3457 (0.0568)	0.2521 (0.0487)	0.3662 (0.0470)	0.3278 (0.0521)	0.2650 (0.0453)	0.3791 (0.0466)	0.3077 (0.0385)	0.2410 (0.0346)	0.2265 (0.0287)	0.1594 (0.0183)	0.1081 (0.0169)	0.0449 (0.0215)	-0.0009 (0.0253)
Dropout	P0	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	-0.6273 (0.0170)	
	Transitory	-0.0311 (0.0085)	-0.0336 (0.0073)	-0.0348 (0.0070)	-0.0318 (0.0062)	-0.0530 (0.0060)	-0.0414 (0.0055)	-0.0380 (0.0050)	-0.0461 (0.0047)	-0.0398 (0.0046)	-0.0334 (0.0042)	-0.0425 (0.0042)	-0.0357 (0.0045)	-0.0307 (0.0043)	-0.0270 (0.0045)	-0.0295 (0.0046)	-0.0441 (0.0061)
	Permanent	-0.3750 (0.0664)	-0.3841 (0.0623)	-0.3091 (0.0555)	-0.2220 (0.0550)	-0.2432 (0.0491)	-0.2818 (0.0468)	-0.2545 (0.0502)	-0.2116 (0.0439)	-0.2773 (0.0459)	-0.2864 (0.0377)	-0.1757 (0.0332)	-0.1534 (0.0277)	-0.1152 (0.0178)	-0.0573 (0.0166)	-0.0577 (0.0208)	-0.0011 (0.0259)
College	P0	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	0.7976 (0.0184)	
	Transitory	0.0139 (0.0085)	0.0180 (0.0072)	0.0408 (0.0070)	0.0337 (0.0061)	0.0507 (0.0059)	0.0478 (0.0056)	0.0476 (0.0048)	0.0511 (0.0046)	0.0549 (0.0046)	0.0467 (0.0043)	0.0564 (0.0040)	0.0436 (0.0043)	0.0319 (0.0042)	0.0253 (0.0045)	0.0253 (0.0045)	0.0344 (0.0059)
	Permanent	0.3812 (0.0714)	0.5619 (0.0648)	0.3239 (0.0549)	0.4429 (0.0584)	0.2358 (0.0491)	0.4275 (0.0472)	0.3481 (0.0520)	0.2358 (0.0449)	0.3746 (0.0454)	0.2820 (0.0368)	0.1796 (0.0331)	0.2034 (0.0278)	0.1185 (0.0181)	0.1205 (0.0169)	0.0282 (0.0203)	-0.0142 (0.0242)
Ability	P0	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	0.7125 (0.0233)	
	Transitory	0.0165 (0.0116)	0.0217 (0.0102)	0.0299 (0.0097)	0.0330 (0.0085)	0.0429 (0.0083)	0.0510 (0.0077)	0.0290 (0.0070)	0.0286 (0.0064)	0.0239 (0.0063)	0.0179 (0.0061)	0.0272 (0.0057)	0.0345 (0.0060)	0.0317 (0.0061)	0.0322 (0.0060)	0.0381 (0.0063)	0.0434 (0.0081)
	Permanent	0.3028 (0.0915)	0.5449 (0.0818)	0.3597 (0.0761)	0.4449 (0.0767)	0.4432 (0.0670)	0.3733 (0.0631)	0.3915 (0.0705)	0.3688 (0.0597)	0.3324 (0.0614)	0.2000 (0.0529)	0.2222 (0.0472)	0.1466 (0.0372)	0.1028 (0.0246)	0.0293 (0.0234)	0.0326 (0.0283)	-0.0458 (0.0338)
Health	P0	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	0.0679 (0.0207)	
	Transitory	0.0015 (0.0105)	-0.0079 (0.0090)	-0.0055 (0.0088)	0.0045 (0.0079)	0.0012 (0.0075)	-0.0019 (0.0069)	0.0107 (0.0065)	0.0037 (0.0059)	-0.0029 (0.0054)	0.0000 (0.0051)	0.0005 (0.0050)	-0.0055 (0.0053)	0.0003 (0.0052)	0.0106 (0.0052)	-0.0020 (0.0052)	0.0082 (0.0066)
	Permanent	0.0298 (0.0800)	0.1194 (0.0727)	-0.0304 (0.0661)	-0.0377 (0.0661)	0.0642 (0.0553)	0.0265 (0.0574)	0.0988 (0.0606)	0.0453 (0.0515)	0.0517 (0.0500)	0.0417 (0.0431)	0.0259 (0.0395)	-0.0110 (0.0307)	-0.0063 (0.0200)	-0.0190 (0.0193)	0.0484 (0.0233)	-0.0284 (0.0273)

Note: standard errors in parentheses. Coefficients and standard errors represent standard deviations in dependent variable. P0 denotes the initial level of permanent income. DWMD estimates. Permanent income follows a martingale and transitory income a MA(1). Education denotes years of schooling, dropout indicates leaving school at the compulsory age. College is attendance at college/university. Ability and health measured for males in the Armed Forces Test.

Figure 1a

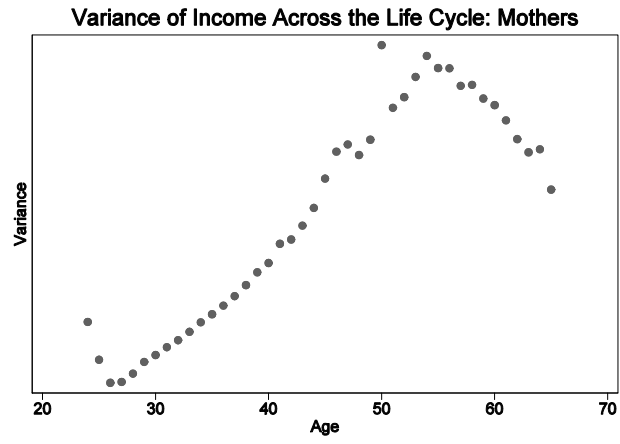
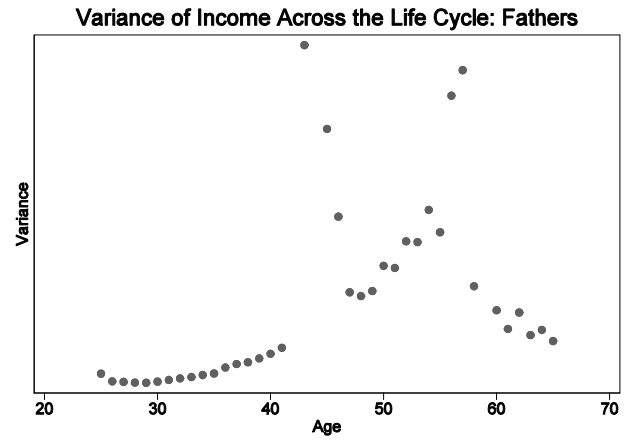


Figure 1b



Figures 2a-2j. The Effect of Household Permanent and Transitory Income Shocks at ages 1-16 Upon Child Human Capital Outcomes.

Figure 2a

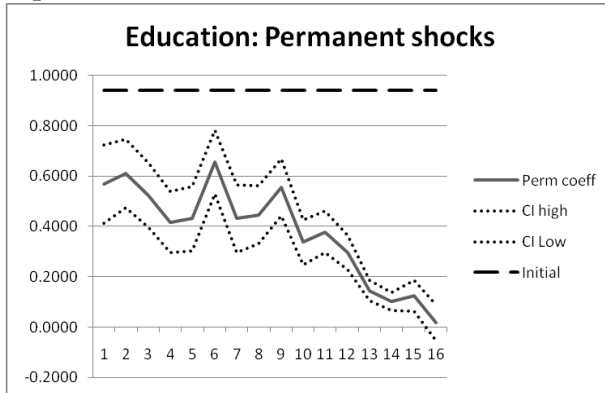


Figure 2b

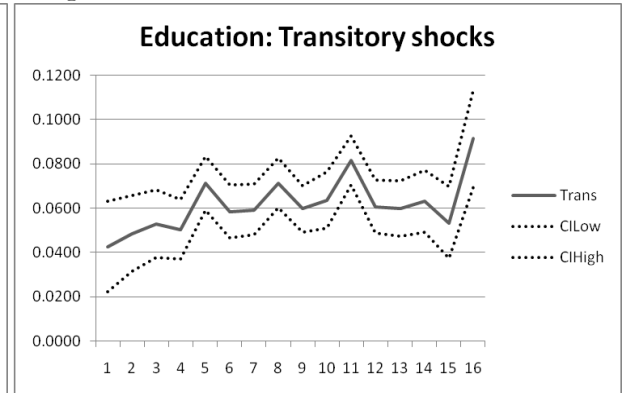


Figure 2c

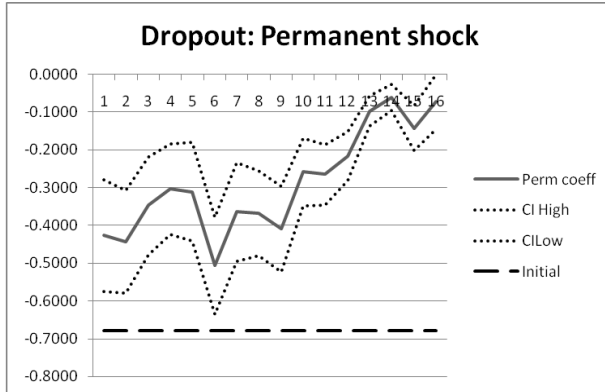


Figure 2d

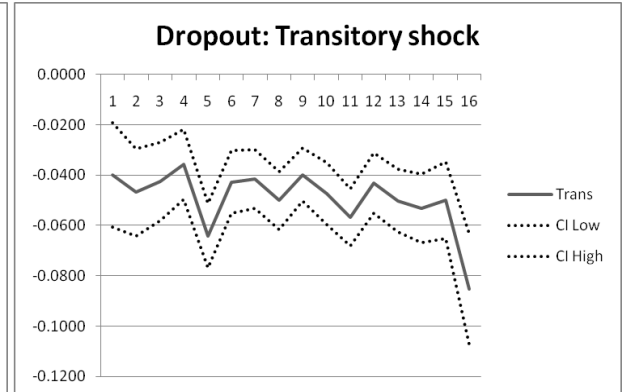


Figure 2e

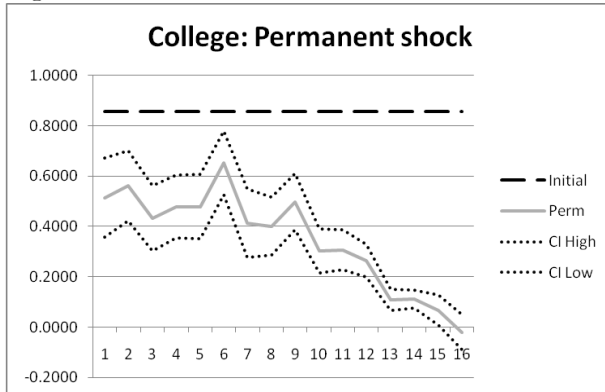
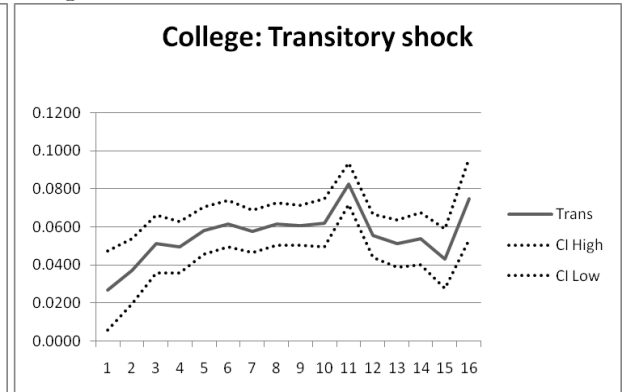


Figure 2f



Note: Estimates of the impact of permanent and transitory income shocks across child age upon child human capital. DWMD model based upon permanent income following a random walk and transitory income MA(1). Coefficients represent standard deviations in the child outcome.

Figure 2g

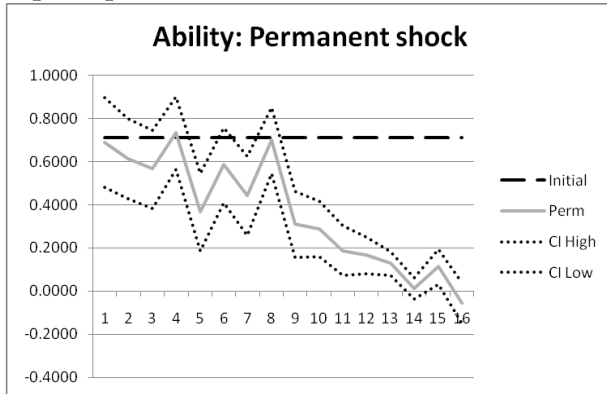


Figure 2h

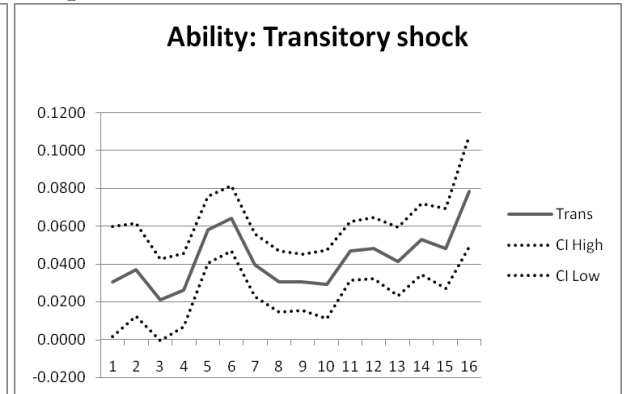


Figure 2i

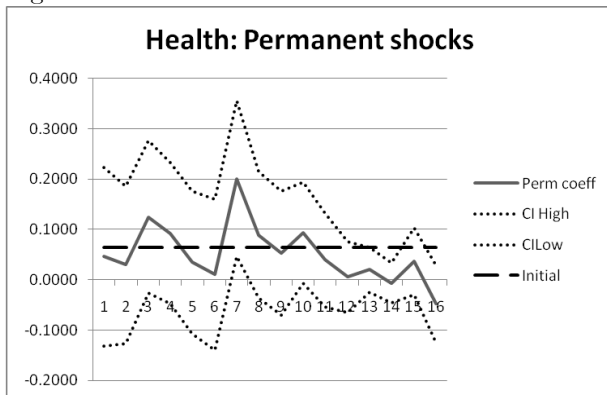
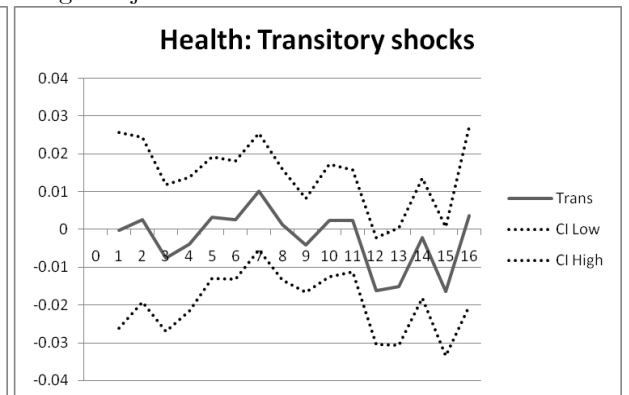


Figure 2j



Note: Estimates of the impact of permanent and transitory income shocks across child age upon child human capital. DWMD model based upon permanent income following a random walk and transitory income MA(1). Coefficients represent standard deviations in the child outcome.

Figures 3a-3j. The Effect of Paternal Permanent and Transitory Income Shocks at ages 1-16 Upon Child Human Capital Outcomes.

Figure 3a

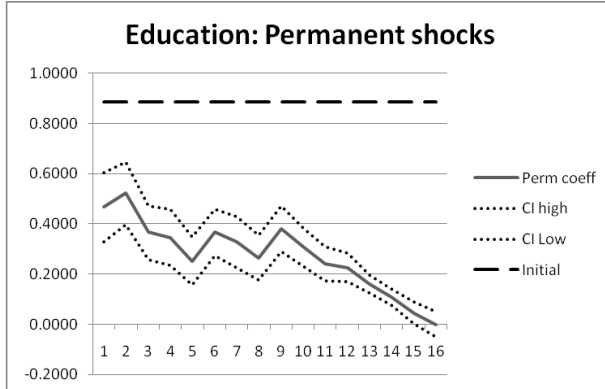


Figure 3b

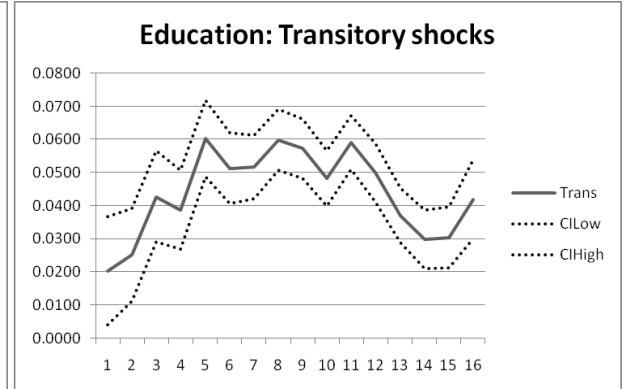


Figure 3c

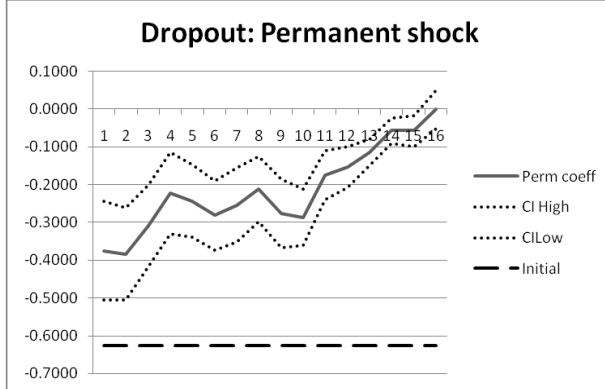


Figure 3d

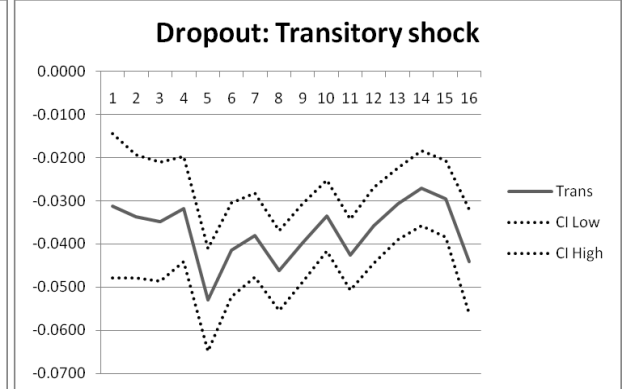


Figure 3e

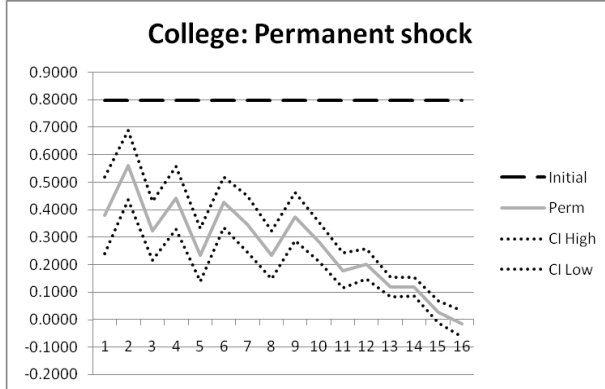
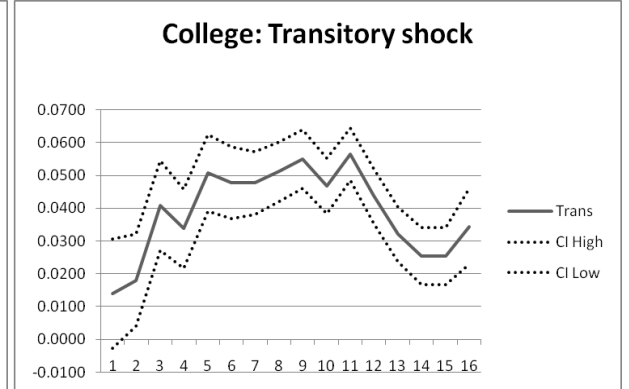


Figure 3f



Note: Estimates of the impact of transitory income shocks across child age upon child human capital using father's income. DWMD model based upon permanent income following a random walk and transitory income MA(1). Coefficients represent standard deviations in the child outcome.

Figure 3g

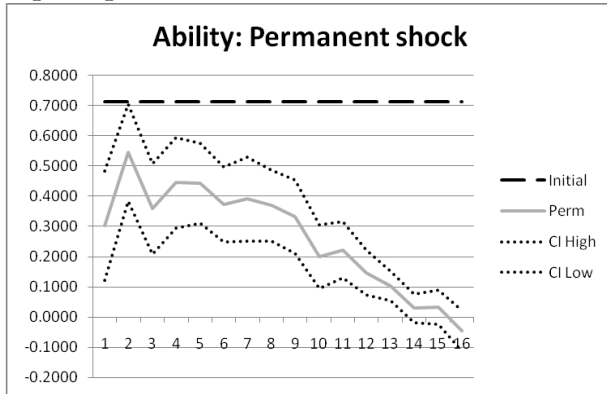


Figure 3h



Figure 3i

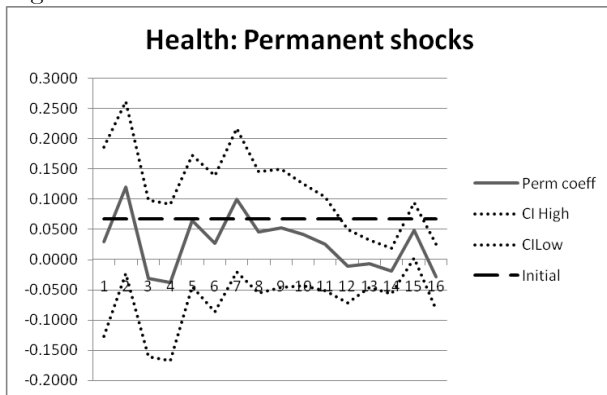
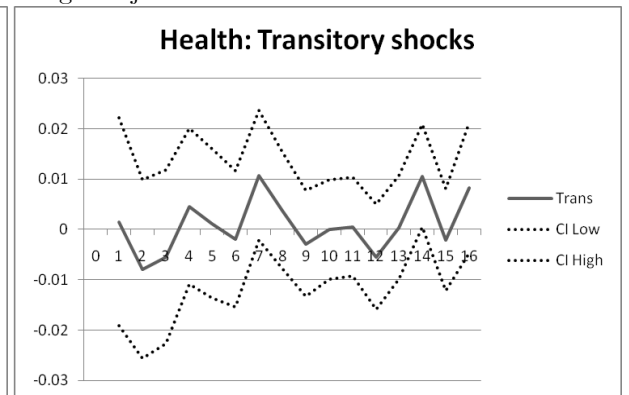


Figure 3j



Note: Estimates of the impact of transitory income shocks across child age upon child human capital using father's income. DWMD model based upon permanent income following a random walk and transitory income MA(1). Coefficients represent standard deviations in the child outcome.

Appendix 3.1. Model for Child Human Capital Accumulation

The model is a simple extension of the classic household optimisation, in which individuals choose consumption in different periods to maximise their utility, subject to a budget constraint. The extension adds the choice of the investment in their child's human capital in each period, and the additional constraint of a human capital (HC) production function, which describes the process through which parental investment is converted to child HC.

In a three period model, parents choose consumption in the first, second and third periods (c_1, c_2, c_3) as well as investment in child HC, (x_1, x_2, x_3) to maximise their expected utility, subject to a budget constraint and a human capital (h) production function. A stock of HC accumulates at the end of the three periods.

$$\max_{c_1, c_2, c_3, x_1, x_2, x_3} u(c_1) + E\beta u(c_2) + E\beta^2 u(c_3) + Eau(h)$$

The two constraints are the HC capital production function.

$$h = f(x_1, x_2, x_3)$$

and the intertemporal budget constraint

$$c_1 + x_1 + \frac{c_2 + x_2}{1+r} + \frac{c_3 + x_3}{(1+r)^2} = y_1 + \frac{y_2}{1+r} + \frac{y_3}{(1+r)^2}$$

β denotes the discount rate for consumption and a the discount rate, or parental altruism, of HC.

The particular functional form of the HC production function is important. Cunha & Heckman (2008) define a CES production function, for example

$$h = A \left[\gamma_1 x_1^\phi + \gamma_2 x_2^\phi + \gamma_3 x_3^\phi \right]^{\frac{1}{\phi}}$$

γ_1, γ_2 and γ_3 denote the productivity of investment in periods 1, 2 and 3 respectively and ϕ is a complementarity parameter and takes the value 1 if investments are perfect substitutes and $-\infty$ if investments are perfect compliments. If investments are complementary, the return to investment in period 1 (2) is increasing in the level of investment in period 2 (1). For simplicity, assume the value of $\phi = 0$, which allows for some complementarity. This is the Cobb-Douglas production function. Taking logs leads to the log linear production function, which is common in the child production function literature, for example Todd & Wolpin (2003).

Additionally, specify a logarithmic utility function, meaning that individuals are risk averse. The optimisation problem becomes

$$\max_{c_1, c_2, c_3, x_1, x_2, x_3} \ln c_1 + \beta E \ln c_2 + \beta^2 E \ln c_3 + aE \ln h$$

subject to the HC production function

$$h = x_1^\alpha x_2^\gamma x_3^\delta;$$

and the intertemporal budget constraint

$$c_1 + x_1 + \frac{c_2 + x_2}{1+r} + \frac{c_3 + x_3}{(1+r)^2} = y_1 + \frac{y_2}{1+r} + \frac{y_3}{(1+r)^2}.$$

Income uncertainty exists in the model through shocks to the income process in each period. Income is the sum of a permanent component (which follows a martingale) and a transitory component (for now MA(0), although an extension to allow serial correlation in the transitory income shocks is simple).

$$y_t = P_t + v_t$$

$$P_t = P_{t-1} + \zeta_t$$

Assume the error terms are mean zero and independent to each other; $E(\zeta_t) = E(v_t) = E(\zeta_t v_t) = 0$.

The optimal level of consumption and investment are given by the equations below.

$$c_1^* = \frac{(1+r)^2 y_1 + (1+r) E[y_2] + E[y_3]}{(1+r)^2 (\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)} \quad (\text{A1})$$

$$x_1^* = \frac{a\alpha \left\{ (1+r)^2 y_1 + (1+r) E[y_2] + E[y_3] \right\}}{(1+r)^2 (\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)} \quad (\text{A2})$$

$$c_2^* = \frac{\beta(1+r)y_1}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{\beta E y_2}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{\beta(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta^2 + \beta} \quad (\text{A3})$$

$$+ \frac{\beta E y_3}{(1+r)(\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)}$$

$$x_2^* = \frac{a\alpha(1+r)y_1}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha E y_2}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta^2 + \beta} \quad (\text{A4})$$

$$+ \frac{a\alpha E y_3}{(1+r)(\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)}$$

$$c_3^* = \frac{\beta^2(1+r)^2 y_1}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{\beta^2(1+r)E(y_2)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{\beta^2(1+r)^2(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta + \beta^2} + \frac{\beta^2 E(y_3)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{\beta^2(\zeta_2 + \zeta_3 + v_3)}{a\delta + \beta^2}$$

$$x_3^* = \frac{a\alpha(1+r)^2 y_1}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(1+r)E(y_2)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(1+r)^2(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta + \beta^2} + \frac{a\alpha E(y_3)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(\zeta_2 + \zeta_3 + v_3)}{a\delta + \beta^2}$$

Substituting into the human capital production function:

$$h = [j]^\alpha [k]^\gamma [l]^\delta \quad (\text{A5})$$

$$\text{where } j = \frac{a\alpha \left\{ (1+r)^2 y_1 + (1+r)E(y_2) + E(y_3) \right\}}{(1+r)^2 (\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)}$$

$$k = \frac{a\alpha(1+r)y_1 + a\alpha E(y_2)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta^2 + \beta} + \frac{E(y_3)}{(1+r)(\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1)}$$

$$l = \frac{a\alpha(1+r)^2 y_1 + a\alpha(1+r)E(y_2) + a\alpha E(y_3)}{\beta + \beta^2 + a(\gamma + \delta + \alpha) + 1} + \frac{a\alpha(1+r)^2(\zeta_2 + v_2)}{a(\delta + \gamma) + \beta + \beta^2} + \frac{a\alpha(\zeta_2 + \zeta_3 + v_3)}{a\delta + \beta^2}$$

Parental investment in each period depends upon realised income shocks and expectations of future income. The influence of permanent income shocks is felt in each future period, whereas transitory shocks display no persistence, and are therefore relevant only in the period in which they occur.

Appendix 3.2. Estimation by DWMD

Estimation is by minimum distance. For each individual, I observe the scalar h_{iT} and define the dummy variable d_i^h to equal 1 if human capital is non-missing for this individual, and 0 otherwise.

Define observations over parental income y_i and the relevant non-missing dummy variable d_i^y as follows

$$y_i = \begin{pmatrix} y_{1,i} \\ y_{t,i} \\ \cdot \\ \cdot \\ y_{T,i} \end{pmatrix} \quad d_i^y = \begin{pmatrix} d_{1,i} \\ d_{t,i} \\ \cdot \\ \cdot \\ d_{T,i} \end{pmatrix} \quad (\text{A6})$$

I define the vector x_i and d_i by

$$x_i = \begin{pmatrix} h_i \\ y_i \end{pmatrix} \quad d_i = \begin{pmatrix} d_i^h \\ d_i^y \end{pmatrix} \quad (\text{A7})$$

The empirical moments are given by

$$\mathbf{m} = \text{vech} \left\{ \left(\sum_{i=1}^N x_i x_i' \right) \otimes \left(\sum_{i=1}^N d_i d_i' \right) \right\} \quad (\text{A8})$$

There are $\frac{T(T+3)}{2}$ unique moments. The vector of theoretical moments is given by $f(\Lambda)$ where $\Lambda = \{\sigma_{P_0}^2, \sigma_{\varepsilon_0}^2, \sigma_{\varepsilon_1}^2, \dots, \sigma_{\varepsilon_T}^2, \sigma_{\zeta_1}^2, \sigma_{\zeta_2}^2, \dots, \sigma_{\zeta_T}^2, \beta_0^P, \beta_1^P, \dots, \beta_T^P, \beta_0^T, \beta_1^T, \dots, \beta_T^T\}$.

$$f(\Lambda) = \begin{pmatrix} v * (y_1) \\ \text{cov} * (y_1, y_2) \\ \text{cov} * (y_1, y_3) \\ \cdot \\ \cdot \\ \text{cov} * (y_1, y_T) \\ \text{cov} * (y_1, h_T) \\ v * (y_2) \\ \cdot \\ \cdot \\ \text{cov} * (y_2, h_T) \\ \cdot \\ \cdot \\ \text{cov} * (y_T, h_T) \end{pmatrix} \quad (\text{A9})$$

Choose parameter values to minimise the difference between the theoretical moments, given in the identification section above, and the empirical moments contained in \mathbf{m} .

$$\min_{\Lambda} (\mathbf{m} - f(\Lambda))' - \mathbf{A} (\mathbf{m} - f(\Lambda))$$

The weighting matrix (\mathbf{A}) is the diagonal from (V^{-1}) , where V is the variance-covariance matrix of \mathbf{m} , consequently estimation is diagonally-weighted minimum distance (DWMD).

4 Maternal Smoking During Pregnancy And Early Child Outcomes

Abstract

The harm from smoking during pregnancy upon child birth outcomes is estimated, using a rich data set on a cohort of mothers and their births. Exploiting a fixed effects approach disentangles the correlation between smoking and birth weight from the causal effect. The results suggest that, despite a detailed set of controls for maternal traits, around one-third of the harm from smoking remains unexplained by observable traits of the mother. Smoking tends to reduce birth weight by 1.7%, but has no significant effect on the probability of having a low birth weight child, pre-term gestation or weeks of gestation. Exploring heterogeneity in the effect on birth weight, it is mothers who smoke for the entire nine months of gestation that suffer the harm, whereas there is an insignificant effect for mothers who chose to quit by month five. Additionally, there is evidence of potential complementarity in investment of human capital, as the impact on birth weight of smoking is much greater for low educated mothers, even controlling for the quantity of cigarettes they smoke.

4.1 Introduction

Maternal smoking during pregnancy remains prevalent in many countries, despite decades of research testifying to the harm it imposes upon the unborn children¹⁹. Since the 1998 “Smoking Kills” White Paper, maternal smoking cessation has been a target of policy in the UK. However, as one in five mothers still smoke whilst pregnant in the UK, the current practice does not seem to be having a strong impact on pregnant mothers. To better understand how policy can be targeted to pregnant mothers, the chapter paints a picture of exactly how smoking during pregnancy lowers child health at birth. Care is taken in assessing the extent to which the estimate is causal. Additionally, the heterogeneity by socio-economic status (SES) and the dynamic nature of the harm from smoking across the duration of the pregnancy is explored.

The chapter focuses on the harm from smoking upon four pregnancy outcomes: child birth weight, the incidence of having a low birth weight (LBW) child²⁰, weeks of gestation and whether the birth was pre-term²¹. As in many studies of this kind, smoking may harm these outcomes not just through the causal route, but through endogeneity inherent in the choice to smoke during pregnancy. Consequently, identification requires a technique to separate the confounding influence of the mother’s traits from the causal parameter of interest. The chapter adopts a mother fixed effects (FEF) approach, exploiting multiple births to the same mother in the data set.

There exists a large epidemiological literature examining the harm from smoking upon a range of child health outcomes, adjusting for a set of characteristics of the mother such as her education and age at birth²². A snapshot of some of the results is that smoking lowers birth weight by 5.7% (Carter *et al* 2006), that this harm accumulates throughout the months of gestation (Hebal *et al*

¹⁹See for example the US Surgeon General’s report 1977-78.

²⁰LBW infants, weighing less than 2500g, are susceptible to infant death, short- and long-term health problems and raise large hospital costs (Almond *et al* (2005)).

²¹classified as less than 38 weeks gestation.

²²See Kramer (1987) and Floyd *et al* (1993) for reviews.

1988), but that the effect on weeks of gestation is generally of much smaller magnitude than on birth weight (Kramer 1987).

The link between smoking during pregnancy and early child health has also been investigated in the economic literature, in which study designs use econometric techniques which explicitly incorporate smoking as an endogenous choice of the pregnant mother. Evans & Ringel (1999) exploit within state variation in taxes on cigarettes in the US between 1989-1992 as an instrumental variable (IV), finding that smoking during pregnancy lowers birth weight by 356-594 grams, (or 10.6-17.6% of average birth weight in their data set). The fact that the IV estimate is higher than their OLS estimates and far higher than many of the epidemiological estimates suggests that the parameter identified may be a local average treatment effect (LATE). In this case, the variation in behaviour is identified from marginal smokers enticed to change their behaviour as a result of the small tax changes across time and states. Compared to the average, these mothers will have a higher marginal benefit from smoking. This parameter is interesting to policy makers considering an increase in taxes on cigarettes, but less so for a general policy to improve child health outcomes.

Lien & Evans (2005) avoid the problem of estimating LATE by exploiting the introduction of one-off tax hikes in four US states between 1992-1994, which were sizeable enough to change the behaviour of a relatively large group of individuals. The authors estimate that smoking reduces birth weight of the child by 189 grams (or 5.6% of the average birth weight across the 4 states), which was of a similar magnitude to the OLS estimation. Unfortunately, tax changes may not be a strong instrument for measuring the dosage of cigarette smoked inhaled by the mother. Adda & Cornaglia (2006) explain that, whilst individuals may respond to the higher taxes by cutting down the number of cigarettes they consume, they can top up the level of nicotine by inhaling with a greater intensity and smoking the cigarette right down to the filter. Therefore the instrument used in the above papers is hard to interpret, if there is not a clear cut change in the dosage. This would explain, for example, why the OLS estimates in Lien and Evan's paper were of similar magnitude to IV estimates.

Almond *et al* (2005) adopt a propensity score matching approach to investigate the effect of smoking during pregnancy upon the probability of having a LBW baby and the duration of gestation. They use birth records from Pennsylvania between 1989-1991 and find that smoking during pregnancy increases the incidence of LBW by 3-4%, but has little effect on gestation. The problem with a propensity score matching technique is that it addresses selection into smoking based upon observable, not unobservable traits of the mother. Therefore, it is possible that the causal estimate of the harm from smoking is not identified in this study.

Finally, differences in smoking behaviour of mothers across pregnancies have been exploited using panel data methodology, which control for mother specific heterogeneity. This is the approach I use. On a study in the US, Rosenzweig & Wolpin (1991) use the children of the 1979 National Longitudinal Survey of Youth. Mothers who had given birth by 1986 were included in the sample, restricting the age of the mother at birth to a young group, aged 21-28. The authors estimate a harm from smoking, controlling for a mother FEF of 3oz for mothers smoking less than one pack per day and 5.6oz for those smoking at least one pack on birth weight, but zero for gestation. It is worth considering the potential bias in this chapter, by the restrictions on the age of the mother at births. Mothers giving birth later in their lives tend to be of a higher SES and simultaneously are less likely to smoke during pregnancy. Therefore, by excluding the older parents, the counterfactual

group of non-smokers will be relatively less healthy than in the population of smokers, which may lead to an upwards bias in the estimate of the harm from smoking during pregnancy. This chapter avoids this problem by including maternal births up to the age of 42.

Abrevaya (2006) matches siblings with their mothers in Natality Data Sets in the United States, to identify how a change in the smoking behaviour of mothers across births translates into health outcomes for the offspring. Results show the panel data estimates to be of a smaller magnitude than the OLS estimates, finding a birth weight effect of around 144-178g and additionally, that smoking during pregnancy may increase the incidence of low birth weight children and reduce gestation. The major problem inherent in this study is in the lack of unique identifiers to match mothers. The authors take care to explore the potential bias and construct three different samples with increasingly stringent requirements for identifying a mother-child pair – for example, linking questions of marital status and father’s race if married. However, one may reasonably expect remaining difficulties in the ability to match uniquely on such characteristics. On the contrary, the analysis in this chapter uses a data set which follows the mothers from their birth in a longitudinal panel survey, to identify each birth for the mother, up to the age of 42, thereby avoiding any potential mismatch across generations.

Utilizing the UK National Child Development Study (NCDS), the chapter exploits a data set with detailed information on the mother, allowing us to control for the potential source of endogeneity through variables including grandparent smoking habits during adolescence, maternal birth weight and paternal smoking habits²³. Currie *et al* (2007) find in the UK that much of the variation in child health stems from unobserved family effects. Therefore effort is taken to eradicate any remaining unobservable elements, by adopting a FEF approach, differencing out any mother level component which is fixed across time. It may be that the maternal input is not fixed in time, therefore as a robustness check time-varying traits are controlled for to allow the mother’s behaviour to adapt to health trends. It will be of interest itself to see how different the OLS and FEF estimates are, given that the NCDS enables control for a wealth of maternal characteristics which contribute towards the maternal fixed effect.

A large contribution of the chapter is that this is the first study in the economics literature to evaluate the harm from smoking within the UK. This is important, as for example Banks *et al* (2006) find evidence that the effect of poor health accumulates differently in the USA and the UK, showing that health gradients across SES are stronger in the US, with those at the bottom of the income distribution penalized more in the US than in the UK. For this reason, this study is necessary to gain a full picture of the harm from smoking.

Another contribution is on providing evidence not just on the harm from smoking, but on heterogeneity in the harm from smoking upon early child health. The chapter examines whether the mother can undo some of the harm from smoking if she quits smoking during pregnancy. Mothers in the study are categorised into those who never smoked during pregnancy, who smoked for the first 5 months and quit and those who smoked consistently for the entire pregnancy. It is often thought that the first few months, in which the fetus develops its skeleton and organs, are the most important in terms of maternal behaviour. However, using birth weight as the outcome may lead to a different effect, as it is during the final 20 weeks that the baby gains most of its body weight. Consequently, the final months of gestation may be the most important, with respect to smoking

²³To my knowledge, no former study has controlled for paternal smoking habits.

behaviour. This is a useful contribution to the policy debate of maternal smoking, as there is often a time lag between the mother becoming pregnant and learning of the pregnancy.

The chapter additionally looks at potential complementarities in investment by considering the harm from smoking separately for low- and high- educated mothers. Complementarity in investments are present if the return to later investments are increasing in earlier investments, and *vice versa*. In this case, high educated mothers may be able to offset some of the harm from smoking.

The impact of maternal smoking during pregnancy on child outcomes is policy relevant because birth weight and gestation have been shown in the literature to drive a range of short- and long- term outcomes. An increase in birth weight lowers one year mortality rates whilst raising education and wages (Black *et al* (2007)), self-reported health and earnings (Currie & Hyson (1999)) and similarly input fetal growth²⁴ has been estimated to improve educational outcomes (Behrman & Rosenzweig (2004)). A finding that smoking during pregnancy does indeed lower pregnancy outcomes suggests that policy makers could use smoking cessation policies as tools for raising not just health, but economic prospects for individuals.

A brief summary of results is that the effect from smoking during pregnancy is surprisingly uncorrelated with the detailed set of mother level variables. In the conditional OLS regressions, smoking during pregnancy lowers child birth weight and gestation by 5.6% and 0.188 weeks respectively and increases the probability of a LBW and pre-term birth by 4.2% and 1.7% respectively. However, conditioning for remaining unobservable traits of the mother in the FEF estimate, only the birth weight effect remains significant, although it falls to 1.7%. The FEF estimate is one-third of the size of the OLS estimate. Two assumptions of the FEF estimate are explored. Firstly, the FEF estimate is identified only from parents whose behaviour changes across births. When the OLS is estimated for these “changers”, the coefficient falls to 3.3% suggesting that half of the OLS correlation is due to unobservable traits of parents. Secondly, potential failure of the strict exogeneity assumption of the FEF technique and the potential attenuation bias from measurement error means that the FEF estimate should be considered only as a lower bound. Therefore, the harm from smoking during pregnancy on child outcomes is within the range of 1.7-3.3% (43-91g) for mothers who change their behaviour during pregnancy and an upper bound of 5.6% (164g) for the total sample of mothers.

Focusing the remaining analysis on birth weight, contrary to beliefs that the greatest harm to the baby will be borne during the first trimester of pregnancy (weeks 1-12), a negligible effect on birth weight to smoking during pregnancy exists if the mother quits smoking by the fifth month of the gestation period. This result is intuitive given the chosen child outcome of birth weight, because it is during the final 20 weeks that 90% of the growth of the child occurs. This is a positive result, suggesting that government policy could be effective if aimed at mothers not just during the first trimester of pregnancy, but into the second. Finally, there is suggestive evidence of strong complementarities in investment of human capital, as it is the low educated mothers who bear the greatest burden of smoking, even conditioning on the quantity of cigarettes consumed.

The chapter is structured as follows. Section 4.2 and 4.3 presents the estimation strategy and the data set. Section 4.4 and 4.5 report and discuss the results and robustness checks. Finally, section 4.6 concludes.

²⁴Estimated as birth weight divided by gestation

4.2 Empirical Methodology

A production function for human capital h of child c , to mother m , is detailed below²⁵.

$$h_{cm} = \beta_0 + \beta_1 S_{cm} + \beta_2 X_{cm} + \varepsilon_{cm} \quad (1)$$

where outcomes $h = \{\log \text{ birth weight, low birth weight (LBW), gestation, pre-term birth}\}$.

S is a dummy variable which takes the value 1 if the mother smokes during pregnancy and 0 otherwise. β_1 is the parameter of interest in this model, the harm from smoking during pregnancy upon child human capital. Mother specific characteristics which vary across births are denoted by X . ε denotes the latent unobservable term. As panel data methods are used to identify β_1 , it is assumed that the error term can be decomposed into a maternal FEF (μ_m) and a child level idiosyncratic component (u_{cm}):

$$\varepsilon_{cm} = \mu_m + u_{cm} \quad (2)$$

Econometric issues confound the causal estimation of β_1 , namely the unobservable maternal input in the child production function. The problem of endogeneity arises from the existence of two mechanisms through which smoking during pregnancy can lower child health. The first is a causal mechanism. Tuomaa (1995) describes how child birth outcomes are driven by maternal smoking. When a mother smokes during pregnancy, nicotine causes the flow of blood between the uterus and the placenta to slow. Additionally, the intake of carbon monoxide transforms oxygen carried to the fetus into carboxyhaemoglobin. The result is that smoking during pregnancy causes fetal hypoxia – or low levels of oxygen – which slows fetal growth and reduces gestation. Note however that, as Floyd *et al* (1993) point out, “This lower birth weight results primarily from intrauterine growth retardation (IUGR), observable at all gestation ages” (pp. 381), therefore the effect on birth weight may be stronger than that for gestation.

Secondly, there may be traits of the mother which drive her to smoke during pregnancy whilst simultaneously choosing other behaviours which lower the health of the child. Smoking habits are easily predicted by SES (in terms of education and age at birth)²⁶ and family background. If the selection into smoking is also driven by unobservable traits of the mother which lower birth weight and gestation, then estimates of the harm from smoking during pregnancy will be prone to bias. Consequently, if this were the case, efforts to entice smoking mothers to quit during pregnancy would have less effect on the health of their child than conventional estimates suggest.

There are many sources of endogeneity, for example it may be the case that mothers who smoke during pregnancy have relatively high discount rates. This creates an omitted variable bias in the estimate of the harm from smoking, if this difference leads to lower child health independently of the smoking habits, for example through inducing other adverse behaviour. Simultaneously, smoking may be correlated with the health endowment of the mother. Rosenzweig & Schultz (1983) raise the issue of endogeneity of health inputs in the child health production function. They conjecture that individuals have superior information on the expected health of their children, which may cause adverse selection. For example, if the mother has a low endowment, she may increase prenatal care

²⁵ Although the specification is linear, we can allow for polynomial and interaction terms, to explore a more flexible relationship between inputs and child outcomes.

²⁶ For example, in the 2000 UK Millennium Cohort Study, smoking mothers were on average 2.6 years younger and had 1.29 fewer years of education than non-smoking mothers.

in anticipation of her child's poor endowment. Additionally, to the extent that parents assortatively mate by health behaviour, paternal smoking habits will be an important control in the child health production function, to identify the causal effect of maternal smoking. If smoking mothers tend to live with smoking fathers, then the harm from the maternal smoking will be overestimated, picking up the harm from passive smoking.

When estimating the harm from maternal smoking upon child human capital, two approaches are employed to take into account the endogenous decision of the mother to smoke during pregnancy.

Firstly, using an OLS framework, proxies for the potential sources of bias control for a wealth of information about the mother spanning her lifetime. Using the NCDS, variables are included which aim to capture the discount rate, or measures of the endowment of maternal human capital, which are often unobserved in other studies within this literature. These are the birth weight of the mother and her height at age 16. As mentioned above, information exists additionally on the smoking behaviour of the child's father. To the extent that parents assortatively mate by smoking habits, this acts as an additional control for the endogeneity.

Using this methodology, the estimate β_1 will be unbiased if $E(\mu_m + u_{cm} | S_{cm}, X_{cm}) = 0$. It is impossible to guarantee that endogenous smoking behaviour of the mother is controlled for with the above methodology. Therefore secondly, the chapter exploits information on sibling births for all mothers with more than one child, estimating a FEF model. The within-group estimator will eliminate μ_m which is common across different births to the same mother. The regression below shows the difference equation which is estimated for each child in the FEF approach.

$$h_{cm} - \bar{h}_m = \delta_1 (S_{cm} - \bar{S}_m) + \delta_2 (X_{cm} - \bar{X}_m) + (u_{cm} - \bar{u}_m) \quad (3)$$

where \bar{h}_m , \bar{S}_m , \bar{X}_m and \bar{u}_m denote the human capital, smoking status, covariates and the error term respectively, averaged across all births for each mother. For simplicity, this can be written as $\Delta h_{cm} = \delta_1 \Delta S_{cm} + \delta_2 \Delta X_{cm} + \Delta u_{cm}$ where Δ denotes a child level deviation from the maternal mean.

The FEF estimate is employed to overcome endogeneity in the human capital production function. To be assured that the FEF estimate is causal requires exploration of the assumptions of the method itself. Strict exogeneity, measurement error and identification in the FEF estimate solely from individuals changing smoking behaviour are now explored in turn.

Strict exogeneity requires $E(u_{cm} | \{X_{cm}\}_{c=1}^T, \{S_{cm}\}_{c=1}^T) = 0$ where T is the total number of children for a mother. There are cases when this may be invalid within this particular framework, which are detailed, in order to understand the direction of any potential bias in the estimate.

The potential failure of strict exogeneity is that mothers may respond to the endowment of the birth of a previous child when choosing smoking behaviour during a current pregnancy. As child endowment is unobservable to econometricians, the consequence is that present smoking behaviour is driven by past realisations of the error term, violating strict exogeneity. Abrevaya (2006) describes that a response of parents along this dimension would induce a negative bias in the estimate of the harm from smoking. Unfortunately, it is not possible to explore the existence of this bias any further than taking note that the estimated coefficient will potentially be biased towards zero and therefore considered as a lower bound estimate.

A second failure of the strict exogeneity assumption is that changes in health habits may con-

found the estimates. A decision to quit smoking is endogenous and, any reduction in the smoking behaviour of the mother may be accompanied by additional health improvements. This is controlled for by including a set of time varying measures of healthiness or discount rates, which are whether the mother drinks over the recommended level of alcohol and whether she participates in sporting activities. Controlling for time varying traits is to move away from the FEF assumption of a mother input which is time constant, adding an additional mother level term which picks up varying habits over and above the FEF. As the estimates to the harm from maternal smoking during pregnancy are not significantly changed by this inclusion, I conclude that failure of the strict exogeneity does not impose a large bias on the estimates.

Griliches & Hausman (1985) describe the attenuation bias from measurement error that is larger in a model where the explanatory variable is in changes not levels. Without a secondary source of information on smoking habits, or a valid instrument, it is not possible to control for this bias and the estimates can therefore be seen as a lower bound to the harm from smoking. One possibility that can be controlled for, however, is that the misreporting of smoking habits are correlated with parental characteristics. If, for example, high SES mothers are more aware from the harm from smoking, they may be more likely to misreport their smoking habits. I exploit the fact that across time, smoking has become more stigmatized during pregnancy, meaning that the correlation between misreporting and SES would have increased across time, to understand the extent of this bias of the correlation between smoking habits and maternal SES. Section 4.5.2 documents the intuition and reports no bias from potential correlation between the mismeasurement of smoking habits and maternal background.

The final issue regarding the causality of the FEF estimate is that in this estimation strategy, identification stems from mothers whose smoking behaviour changes across pregnancies. This means that outcomes for mothers who smoke during pregnancy for all births or for no births, do not create the variation which generates the estimate. It is possible that these group of mothers (called ‘non-changers’ for the remainder of the chapter) will look different to the “changers”. To understand that extent to which this is true, the OLS is estimated additionally for the group of changers, which is compared to the FEF estimate to understand the true extent of endogeneity in the smoking parameter

4.3 Data

The NCDS is a longitudinal panel data set, whose participants are the cohort of children born in the UK in one week in 1958. The most recent period of observation at the time of writing was in 2001, which gives over 40 years of information on the cohort members. It is the information on children of the cohort members which is exploited for this study, using the in-depth pregnancy information on siblings. The children in the sample are born between 1973-2000.

The sample includes 3368 female cohort member mothers and 6860 children²⁷. 2799 of these mothers (and 6291 children) had more than one child, which is necessary for the FEF approach, and therefore will be the chosen sample.

An advantage of the panel data nature of the NCDS over other data sets with pregnancy information, is that it allows us to observe in-depth smoking habits of pregnant mothers. The incidence of

²⁷I restrict the sample to families with fewer than six children, to eliminate outliers.

smoking during pregnancy is observed and whether the mother stopped smoking by the fifth month of gestation.

The birth outcomes of the children of the cohort members are very detailed. The measures of child human capital are log birth weight, whether the child was low birth weight (LBW), weeks of gestation and whether the birth was a pre-term birth (<38 weeks). Note that gestation is believed to be measured with lower accuracy than birth weight, due to errors in identifying the exact date of conception. Observed additionally is child gender, which will be an important control as boys tend to be heavier than girls at birth.

Given that the cohort members have been tracked in the NCDS since birth, the chapter incorporates a wealth of information on the mothers which will drive their smoking behaviour and form important inputs into the child human capital production function. To proxy for the mother's inherent discount rates, or attitudes towards health, controls are included for the grandfather's social class at the date of birth of the mother, as well as her parents' smoking habits when she was aged 16. As explained in the model, the endowment of the child is partly driven by maternal health endowments. The endowment is proxied using information on the mother's birth weight observed directly in the first wave of the NCDS and additionally the mother's height at age 16. To my knowledge, no other study looking at the impact of maternal smoking upon child birth outcomes has controlled directly for such health inputs of the mother. However, it is a very important control in the child production function for three reasons. Firstly, Conley & Bennet (2000) report the impact of maternal birth weight upon the probability of having a LBW child. Using PSID sibling data between 1968-92, the authors find LBW of the mother increases the probability of the child being LBW by a factor of four. Secondly, it is important to control for endowments in child human capital as for example, low birth weight may not be a negative outcome, rather a genetic trait. Finally, if mothers adapt their behaviour in response to their own health endowments, the result of insufficient control along this dimension will produce a bias in the estimates of the birth weight effect of maternal smoking. Of course, this endowment will be differenced out in the FEF estimates. Of interest itself is whether these extensive controls for the mother's endowment and environment will produce an OLS estimate in the range of the FEF estimate, or whether unobserved heterogeneity still remains.

It is taken into account whether the mother lives with a smoker. Living with a smoker may drive child birth weight, through a direct and an indirect channel. Firstly, through the indirect channels, to the extent that partners do assortatively mate by "healthy behaviour", controlling for the mother living with a smoker will in part absorb the endogeneity inherent in the mother's smoking status during pregnancy. Further, the event of living with a smoker may drive motivation for the mother to quit smoking, therefore the control will allow a more accurate description of the mechanisms through which mothers smoke during pregnancy. The direct channel is through passive smoking. It is possible to partly disentangle these two effects as the FEF should eradicate the indirect mechanism, leaving only the passive smoking effect.

Black *et al* (2005a) find that birth order is an important determinant of child outcomes, thus this will be a control in the data. It is additionally important however given the identification as, if for example mothers decide to smoke for the first child but not for the second, then the change in health will be perceived to change smoking behaviour when really, it is the birth order effect that is being picked up. Additional controls are marital status, ethnicity education of the mother and the age of mother at birth. The descriptive statistics for this data set are reported in the section below.

4.4 Results

4.4.1 Descriptive Statistics

Summary statistics are reported in Table 1. The total number of children born to female cohort members in the sample is 6291. Of these, 68% were born to non-smoking mothers, 6% born to mothers who smoked for the start of the pregnancy but quit by month five and 26% of the mothers smoked consistently for the duration of the pregnancy. Child birth weight tends to fall with the duration of the smoking habit during pregnancy. The mean birth weight for non-smoking births is 239g heavier than the mean for the consistently smoking sample and the probability of having a LBW child is 0.6 percentage points higher. There is no large difference in the mean gestation period, however pre-term births are more likely for the smoking mothers. Smoking during pregnancy is not random across observable mother characteristics, as smoking mothers give birth at an earlier age, have more children, leave school at a younger age, are three times more likely to live with a smoker and have parents who smoked when the mothers were aged 16. All in all, these statistics signify that mothers smoking during pregnancy tend to have less advantageous outcomes.

It is important to understand mothers' changing smoking behaviour across births, as the identification exploits variation within mothers, across siblings. Table 2 shows the transition matrix for three child families. Within each cell, the top and bottom values represent the column and total percentages respectively. For each child, the mother is classified as smoking during pregnancy (=1) or not (=0). Looking at behaviour across the first and second births smoking habits are very persistent. Of the mothers smoking (not smoking) for the first birth, 78% (89%) smoke (do not smoke) for the second. Similarly, of the mothers refraining from smoking for the first, only one in ten choose to smoke for the second birth. The numbers are similar for comparisons in the remaining cells. Despite this persistence in habits, there is variation in behaviour. When the smoking status of mothers changes across births, mothers are more likely on average to quit smoking than to start smoking.

4.4.2 Regression Results

Table 3 reports the estimated coefficients and standard errors for the incidence of smoking during pregnancy for the four pregnancy outcomes. All regressions control for child year of birth dummies, to absorb health trends in smoking habits and the analysis clusters at the level of the mother²⁸. Moving from column 1 to 5, the controls are sequentially included. The raw harm from smoking during pregnancy is a reduction in child birth weight by 5.8%, an increase in the probability of having a LBW child by 4.5%, a reduction in gestation by 0.224 weeks and an increase in the probability of having a pre-term birth by 2.3%. All of these estimates are significantly different to zero.

The inclusion of additional controls will reduce the magnitude of the estimate, if generally less healthy mothers smoke and give birth to lighter children. In column 2, controls are added firstly for a set of standard child health production function inputs. The coefficients either increase or have no effect on the coefficients, indicative that on average, omitting these controls led to an upward bias, contrary to the expectations. This result is driven mainly by the control for birth order, which appears positively correlated with child birth weight and also with maternal smoking habits. That

²⁸The full set of results can be seen in Appendix 5.1.

is, conditional upon traits such as the mother's age and education, mothers tend to smoke more for later births.

Moving across the table, as further controls are added up to column 5, the estimates for the harm from smoking fall in magnitude, as was expected. However, each set of controls reduces the effect only slightly. For example, including the set of health and endowment controls, which may be important in driving smoking behaviour and child health, reduces the effect on birth weight by 0.2 percentage points. Information on the partner's human capital and smoking behaviour are important controls for the estimate of maternal smoking. Smoking during pregnancy now reduces log birth weight and weeks of gestation by 5.6% (or 164g) and 0.188 weeks respectively, and raises the probability of giving birth to a LBW child and pre-term births by 4.2% and 1.8% respectively. The birth weight estimate is in line with that of Lien & Evans (2005), Rosenzweig & Wolpin (1991) and at the upper bound of Abrevaya (2006) and the estimate of the harm on the probability of low birth weight is very similar to that found in Almond *et al* (2005). Controlling for a detailed set of background traits of the mother, including whether her parents smoked during her adolescence, has no significant impact upon the harm from smoking during pregnancy²⁹.

Although the first 5 columns of Table 3 control for a particularly detailed set of traits of the mothers in the data set, it is not possible to be certain that the endogenous decision to smoke during pregnancy has been fully accounted for. Therefore, in column 6, a mother FEF model is estimated to difference out any inherent healthiness or endowment of the mother which may have been creating an omitted variable bias in the previous estimates.

The estimated coefficients for the dependent variable log birth weight are displayed. Row 1 shows the harm on log birth weight has fallen dramatically, from 5.6% to 1.7% (or 43g), a difference which is statistically significant. This finding that the FEF estimate is lower than the OLS replicates those of Rosenzweig & Wolpin (1991) and Abrevaya (2006). The results suggest that two thirds of the harm from smoking upon child birth weight is driven by unobservable traits of the mother. This is somewhat surprising, given the wealth of controls incorporated into the final OLS estimate – from the choices of the mother regarding her education and age at birth, measures of her health endowment and also behaviour of her family spanning her lifetime. Therefore, the chapter now seeks to understand exactly what the FEF estimate is capturing.

The FEF estimate will exploit variation in the smoking behaviour of each mother across her multiple births. That is, unless the mother changes habits (called a changer) from one birth to another, she will not influence the estimate of the harm from smoking. Within a heterogeneous returns model, the harm from smoking for changers may differ than that for mothers whose habits persist (non-changers). The FEF estimate, rather than estimating the harm from smoking controlling for unobservables may instead estimate this effect for changers. In column 7 therefore, the OLS is estimated (linear probability model) for log birth weight and gestation (LBW and pre-term births) for a sample of changers, with the full set of time varying controls. The number of observations has fallen accordingly, to 734.

As expected, the harm from smoking, excluding those mothers who smoke consistently across births, is lower than the previous estimate in column 5, owing to the selection into the two groups of changers and non-changers. Comparing the harm from smoking upon log birth weight, in the

²⁹Note that we ran these regressions including additionally families with only one child. It may be that one child families are particular, hence excluding them will estimate the effect for only a subgroup of the population of households. None of the estimated coefficients were statistically different to Table 3.

FEF model and the OLS for changers, looking at the appropriate comparison group, one third of the harm stems from unobservable traits. The harm is now 3.3%, or an effect of 91g. However the OLS bias is still large and the FEF estimate is significantly different to the OLS estimate for the changers. Comparing the estimate of the harm from smoking for changers, to the literature, the harm for these groups of mothers is lower than for most studies, which again is indicative that the group of mothers who change their behaviour during pregnancy are a select group. This is the estimate to compare to the FEF estimate however and possibly the most policy relevant estimate, as the non-changers do not demonstrate marginal behaviour so may not be induced to change their habits through smoking cessation policies.

Column 6 shows that the FEF regressions are insignificantly different to zero for all three remaining outcomes. Comparing these coefficients to column 7, there is small and only just significant impact upon the probability of pre-term gestation, for the group of changers, increasing the probability by 3%, which compares to 1.8% for the full sample and 1.4% in the FEF regression.

For the remaining analysis, attention is restricted to the outcome log birth weight, as FEF estimates and regressions for the changers produce small or insignificant results for the other three outcomes. It is interesting to briefly consult the estimates in the birth weight equation, of other inputs into the child production function, in columns 1-3 of Appendix 5.1. Boys are heavier than girls, that the mother's education, age and marital status at birth are all insignificant in these conditional regressions. What is interesting is the difference between the coefficient for living with a smoker, in the OLS and the FEF regression. Section 4.3 outlined that living with a smoker may have an effect on child birth weight firstly through passive smoking and secondly by changing the behaviour of the mother. Additionally, controlling for a variable for whether the mother lived with a smoker may condition for endogeneity of the mother's choice to smoke, if there exists assortative mating by health behaviour. Whilst in the OLS regression, living with a smoker lowers child birth weight by 2.1%, there is no significant effect once the mother FEF is controlled for, suggesting that passive smoking plays only a small role in the transmission of poor health to the fetus. Indeed, this finding is supported by scientific evidence. Jarvis *et al* (2001) found that the cotinine³⁰ concentration for passive smokers is only 0.6-0.7% that of smokers.

4.4.3 Heterogeneity

The next stage of the analysis is to explore heterogeneity in the harm from smoking during pregnancy. Results are reported in Table 4. The first column estimates an OLS, conditional upon the full set of controls, then in column 2 OLS for the group of changers is estimated and finally column 3 reports estimates from a FEF model, for subsets of the sample of mothers.

Duration In the first regression of Table 4, possible non-linearity in the duration of pregnancy the mother smoked for is examined, as scientific studies suggest that if mothers give up smoking, the month they quit may be important for the health of their child. The detailed information in the NCDS allows us to change the explanatory variable to dummies for the mother not smoking, smoking during the first 5 months and for the entire 9 months of pregnancy.

In all three specifications, a negligible birth weight effect is estimated of smoking during the first

³⁰Nicotine converts to cotinine which remains for longer in the blood than nicotine so is therefore a more accurate measure of smoking than nicotine

five months, relative to non-smokers during pregnancy. This result is replicated in scientific research where, for example, Hebal *et al* (1988) find that women quitting smoking during pregnancy bear no harm of the smoking on birth weight.

This result could be due to two factors. Considering the stages of growth and development of the fetus during the pregnancy, it is in the first trimester of pregnancy (weeks 1-12) that the baby develops facial features, limbs, heart and organs. During the second trimester (weeks 13-28) the fetus strengthens and grows. 90% of weight growth occurs from week 20 onwards. Consequently, in order to get a fuller picture of the harm from smoking, further investigation is needed into the harm from smoking for early months upon other health outcomes of the children. However, data limitations mean that this is outside the scope of this study. Another explanation is that the group of quitters is possibly an endogenous one, with unobserved heterogeneity leading to smoking cessation during pregnancy and simultaneously other precautions taken to ensure the fetus is healthy. None of the controls for health behaviour vary within a pregnancy therefore is not possible to adequately capture the endogeneity. For this reason, a cautious interpretation is given to the result and conclude that mothers are able to undo the harm from smoking during pregnancy if they change their behaviour during the first two trimesters, which may include quitting smoking.

A large impact of smoking consistently for the full 9 months of pregnancy is estimated, relative to not smoking. In column 1, the OLS effect on the full sample, shows that mothers smoking for the entire 9 months of pregnancy will give birth to babies 7.2% (or 212g) lighter than non-smoking mothers. This falls to 5% (150g) when excluding mothers who smoke continuously and estimate the effect for changers only. Finally, column 3 shows that differencing out the mother FEF reduces the estimate by more than half, compared to column 2 – indicating that for the full gestation smoker mothers, at least 50% of the harm from smoking is due to unobservable factors omitted from OLS regressions. Again, this suggests that along with quitting smoking, it is important for mothers to adapt other behaviour to improve the health of their child.

Socio-Economic Status It is of interest whether the impact of maternal smoking during pregnancy displays heterogeneity across the distribution of education. This would be the case for two main reasons. Firstly, if there are complementarities in investment of human capital, then high educated mothers are able to extract a higher return to their investment, or a lower harm from smoking. Additionally, high education may shift the budget constraint faced by a pregnant mother. This means that high educated mothers may be able to reduce the harm, by for example buying a higher quality of prenatal care than low educated mothers.

Mothers are defined as having a low level of education if they leave school by the compulsory age, which is 16 for the NCDS cohort, and high education otherwise. In Table 4, comparing across regressions 2 and 3, in column 1 the OLS harm from smoking is nearly double for low educated mothers than high educated mothers. The harm for low educated mothers classified as changers, shown in the second column of regression 2, is 1.6 percentage points higher than for the total sample of smokers, at 5% (150g). For the high educated changers, there is no significant effect of smoking. In the final column over half of the harm from smoking for low educated mothers is differenced out in the FEF model, as compared to the previous column, and the harm from smoking falls to 1.9% (53g). For the high educated mothers, there is no significant effect of smoking during pregnancy in a mother FEF model. This suggests that the harm from smoking is borne by low educated mothers.

This result may be due to the fact that the quantity of cigarettes consumed is inversely related to education. Indeed, low educated mothers on average smoke 3 cigarettes a day more than high educated mothers, hence the above result may be due to a dosage effect, rather than due to complementarities or difference in the budget constraint. Therefore, in regressions 4 and 5, the explanatory variable of interest is changed, to the quantity of cigarettes consumed during pregnancy³¹. The results show that the harm from smoking each cigarette is relatively higher for low educated mothers, at 0.2% per cigarette in the FEF regression, compared to a result insignificantly different to zero for other mothers. The significance of this result is very relevant for policy targeting. As it is the low educated mothers who are more likely to smoke, who smoke a greater quantity during pregnancy and whose child receives the greatest harm for each cigarette smoked during pregnancy, it seems necessary that the low educated, or more generally low SES mothers, be the focus of targeted policies to change behaviour during pregnancy.

4.5 Robustness Checks

4.5.1 Health Habits

The decision of a mother to quit smoking is endogeneous. This will create a problem for the identification strategy, if the driving force for the mother to quit also influences other investment behaviour, which raises child health. This is overcome by controlling for health traits of the mother which are time-varying as, across births the mother may change her behaviour according to health trends or experience. The controls incorporated are whether the mothers exercise regularly and drinks over the recommended allowance of alcohol³², at the period of observation most recent to the birth of the child. In the sample, 66% of mothers report regular sporting activity and 26% drink over the level of alcohol recommended by the government. These are good proxies for discount rates, or risky behaviour of individuals. Controlling for time-varying traits moves away from the FEF assumption of a mother input which is time constant, however inclusion of these two proxies do not change the coefficient on maternal smoking at all³³. This suggests that the method of assuming a maternal FEF in the production function of child health is indeed robust.

4.5.2 Misreporting of Smoking Status

Abrevaya (2006) details the measurement error likely in the fixed effect estimate of the harm from maternal smoking during pregnancy upon child health. In this study, the consequences are that the FEF estimates are reported as a lower bound, as the possible sources of the measurement error are not possible to examine closely with the current data.

However, one other aspect of measurement error which can be explored is that the misreporting of smoking habits, which may be correlated with maternal SES. High SES mothers may be more likely to misreport their smoking status during pregnancy, if they are relatively more responsive to the stigmatization of smoking during pregnancy. This could be because, with a higher level of education, they are more aware of, or responsive to improved knowledge regarding the harm

³¹The cohort members were not directly questioned about the number of cigarettes they smoked during pregnancy, but rather quantity smoked at each wave of observation. Therefore, assigning a value of the quantity of cigarettes smoked requires restricting the sample to mothers who reported not changing their habits during pregnancy.

³²This recommended level of alcohol is 14 units per week for women and 21 units per week for men.

³³The FEF coefficient on maternal smoking is still 1.7% (standard error 0.0099)

from smoking. Health trends have developed such that smoking during pregnancy has become more stigmatized over time, meaning that if misreporting by SES is a problem, it is one that will have increased across time. It is possible therefore to examine the bias from a correlation between misreporting of smoking status and maternal SES, by interacting the education of the mother with the child's year of birth.

The FEF model of Table 3, column 6 is estimated including an additional term for the interaction between the education of the mother (where low and high education are defined as above) with the child's year of birth. The result is that the coefficient on smoking changes by a very small and insignificant amount, from 1.72% to 1.71%, suggesting that the bias is insignificantly different to zero. Again, the FEF estimates seem particularly robust estimates.

4.5.3 Other Effects of Smoking

This chapter has analysed the harm from smoking upon child birth weight and gestation. It can be argued however that smoking during pregnancy may drive other outcomes of the child, which drive child health independently to the smoking. For example, Tuormaa (1995) lists some of the other effects from maternal smoking as placenta previa and placental abruption³⁴. These two conditions may lead to pre-term births, which will affect birth weight. The estimate has identified the total effect of smoking on birth weight, which is arguably the most interesting. However, a robustness test is conducted, by controlling for gestation in the birth weight equation and also for a dummy variable which equals 1 if there were complications during pregnancy and 0 otherwise. The problems cited by Tuormaa were mentioned by the mothers as complications during the birth, as well as others such as heart problems.

Table 5 shows the estimate of smoking upon log child birth weight under the three specifications, with a full set of controls including complications during pregnancy and gestation. Compared to Table 3, the estimated "direct" impact of smoking during pregnancy is lower, as expected, once these two pregnancy outcomes are factored out. The OLS for the total sample and for changers respectively, has fallen to 4.6% and 3.1% respectively, and FEF estimate is 0.3 percentage points lower, at 1.4%. Complications during pregnancy reduce child birth weight by 4.9%-6.2% and an increase of gestation by one week will raise the child birth weight by between 4.7%-5.5%. This table suggests that the channels through which smoking does lower early child human capital are partly explained by gestation and the incidence of complications during pregnancy.

4.6 Conclusion

The chapter used a data set very rich in information about mothers and children, in order to estimate the harm on child human capital from smoking during pregnancy. Estimated OLS conditional effect on birth weight was 5.6% for the total sample of mothers, 3.3% for the sample of changers and the FEF 1.7%. The FEF estimate is a lower bound, hence the harm from smoking is reported as 1.7-3.3% (43-91g). These estimates are relatively low compared to most existing studies, which is due to the fact that the FEF estimates exploit variation in the group of mothers who exhibit a changing behaviour across births. That is, mothers who smoke consistently across all births, who are arguably

³⁴See Appendix 5.2 for precise details of other outcomes

a select group of mothers, do not contribute to the FEF estimate. However, the marginal mothers seem the most policy relevant group to target, in order to change smoking habits.

The fact that the FEF estimate is considerably lower than the OLS estimate suggests that unobserved traits of the mother play a large role in the development of child human capital over and above her smoking habits. Therefore policies targeting child human capital should aim to educate pregnant mothers on health behaviour generally during pregnancy, rather than just smoking behaviour specifically. There needs to be more understood about exactly what the unobservable traits are, to get inside this black box of child health outcomes.

A negligible effect was found of maternal smoking during the pregnancy, if the mother quits by month five, which suggests that there is a cumulative effect of smoking. This is reassuring, as cessation or education programs do not have to just target pregnant mothers early in the gestation, but can have some effect later in the pregnancy. The harm from smoking was found to be non-linear and decreasing across the education of the mothers, results which are robust to controls for the quantity of cigarettes smoked during pregnancy.

Smoking cessation policies are not enough to address inequalities in child health. Further, not only is it the low SES mothers who choose to smoke, but they are also the mothers bearing the greatest burden from the smoking. Therefore, any potential solution must offer help to these mothers, to target those with the worst habits and poorest records of child health.

Table 1: NCDS Sample Statistics.

Smoking during pregnancy	Never	At the start	Consistently	Total Sample
Number of obs	4266	378	1647	6291
% of sample	67.8	6.0	26.2	100
Birth weight (grams)	3412.17 (546.29)	3411.32 (571.03)	3173.49 (588.40)	3349.63 (568.77)
LBW	0.05 (0.22)	0.05 (0.22)	0.11 (0.31)	0.06 (0.25)
Gestation (weeks)	39.73 (2.05)	39.76 (2.41)	39.53 (2.44)	39.68 (2.18)
Pre-term Gestation	0.10 (0.30)	0.08 (0.28)	0.13 (0.34)	0.10 (0.31)
Mother age	28.95 (5.36)	25.88 (4.08)	25.36 (4.92)	27.82 (5.43)
Age left school	17.40 (2.24)	16.70 (1.50)	16.32 (1.18)	17.08 (2.03)
Mother Height (inches)	63.94 (2.70)	64.00 (2.53)	63.39 (2.47)	63.80 (2.64)
Live with a smoker	0.23 (0.42)	0.49 (0.50)	0.62 (0.49)	0.34 (0.48)
Grandmother smoked, mum age 16	0.32 (0.47)	0.39 (0.49)	0.46 (0.50)	0.36 (0.48)
Grandfather smoked, mum age 16	0.39 (0.49)	0.45 (0.50)	0.50 (0.50)	0.43 (0.49)

Standard deviation is shown in parentheses.

Note: sample of mothers with at least 2 children and fewer than 6.

Table 2: Transition matrix of smoking habits, for 3 child families.

		1 st child		2 nd child	
		0	1	0	1
2 nd child	0	0.89	0.22		
		0.549	0.083		
3 rd child	1	0.11	0.78		
		0.069	0.300		
3 rd child	0	0.92	0.33	0.95	0.19
		0.576	0.122	0.623	0.067
3 rd child	1	0.08	0.67	0.05	0.81
		0.050	0.252	0.030	0.280

Key: Row 1: Column percentage, Row 2: Total percentage

Table 3: Regression of dummy variable for mother smoking during pregnancy, upon four pregnancy outcomes.

Regression	1	2	3	4	5	6	7
	Raw	+ standard controls	+ health, endowment	+ partner information	+ real background	FEF	OLS / LP (changers)
1	Log birth weight	-0.058 (0.007)***	-0.064 (0.007)***	-0.062 (0.007)***	-0.056 (0.008)***	-0.017 (0.009)*	-0.033 (0.012)***
2	LBW	0.045 (0.009)***	0.047 (0.010)***	0.046 (0.010)***	0.042 (0.010)***	0.008 (0.012)	0.021 (0.013)
3	Gestation (weeks)	-0.224 (0.075)***	-0.212 (0.077)***	-0.206 (0.078)***	-0.188 (0.081)**	-0.080 (0.118)	-0.035 (0.123)
4	Pre-term gestation	0.023 (0.010)**	0.023 (0.010)**	0.022 (0.010)**	0.018 (0.011)	0.014 (0.017)	0.030 (0.018)*
N	6291	6291	6291	6291	6291	6291	734

Standard errors in parentheses. All regressions control for child year of birth dummies. Standard controls: age mother at birth, age mother

squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height. Father

information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class

at birth, grandmother and grandfather smoked when mother 16. LP denotes the linear probability model. Key: *, **, *** denotes significantly

different from zero at the 10%, 5%, 1% level respectively.

Table 4: Heterogeneity in the harm from smoking during pregnancy.

Dependent variable is log child birth weight.

Regression	Sample	1 OLS (all)	2 OLS (changers)	3 FEF
1	Quit by month 5	0.002 (0.011)	-0.022 (0.015)	-0.009 (0.012)
	Smoke 9 months	-0.072 (0.008)*** N=6291	-0.050 (0.014)*** N=734	-0.024 (0.012)** N=6291
2	Low education	-0.062 (0.009)*** N=3980	-0.049 (0.014)*** N=509	-0.019 (0.011)* N=3980
3	High education	-0.035 (0.014)** N=2311	0.016 (0.023) N=225	-0.017 (0.018) N=2311
4	Low education, quantity	-0.004 (0.001)*** N=3320	-0.003 (0.002)* N=145	-0.002 (0.001)* N=3320
5	High education, quantity	-0.002 (0.001) N=2049	0.001 (0.003) N=54	-0.001 (0.002) N=2049

Standard errors in parentheses. All regressions control for child year of birth dummies.

Standard controls: age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height. Father information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class at birth, grandmother and grandfather smoked when mother 16.

Key: *, **, *** denotes significantly different from zero at the 10%, 5%, 1% level respectively.

Table 5: Additional controls for birth outcomes.

	(1) OLS (all)	(3) OLS (changers)	(2) FEF
did CM smoke during pregnancy	-0.046 (0.005)***	-0.031 (0.010)***	-0.014 (0.008)*
Complications during birth	-0.049 (0.008)***	-0.062 (0.030)**	-0.051 (0.009)***
Child gestation	0.054 (0.002)***	0.055 (0.011)***	0.047 (0.003)***
Obs	6291	734	6291
R-squared	0.45	0.46	0.42

Standard errors in parentheses. All regressions control for child year of birth dummies.

Standard controls: age mother at birth, age mother squared, education mother, married at birth, child sex, birth order, ethnicity. Health endowment: maternal birth weight and height. Father information: age left school, live with smoker. Real background information: region where mother born, region at 11, grandfather's social class at birth, grandmother and grandfather smoked when mother 16.

Key: *, **, *** denotes significantly different from zero at the 10%, 5%, 1% level respectively.

Appendix 4.1. Full Regression Results

	OLS (all)	OLS (changers)	FEF
Did CM smoke during pregnancy	-0.056 (0.008)***	-0.033 (0.012)***	-0.017 (0.009)*
Child sex	0.031 (0.005)***	0.001 (0.014)	0.031 (0.005)***
Age parent left school	0.000 (0.002)	-0.004 (0.006)	
Age when mother had child	0.005 (0.057)	-0.126 (0.182)	0.057 (0.055)
Mother age child squared	-0.000 (0.001)	0.003 (0.004)	-0.001 (0.001)
Married when had baby	0.000 (0.007)	-0.030 (0.024)	0.009 (0.017)
Birth order	0.021 (0.003)***	0.019 (0.008)**	0.024 (0.006)***
Black	-0.106 (0.040)***	-0.103 (0.042)**	
India/Pakistan	-0.095 (0.027)***	0.000 (0.000)	
Other Asian	0.000 (0.000)	0.000 (0.000)	
Mixed Race	-0.090 (0.199)	0.000 (0.000)	
Mother's height aged 16 inches	0.008 (0.001)***	0.002 (0.004)	
Log birth weight parent	0.147 (0.020)***	0.143 (0.071)**	
Partner's age left school	-0.001 (0.002)	-0.003 (0.008)	-0.008 (0.008)
Live with smoker	-0.021 (0.008)***	0.007 (0.023)	-0.015 (0.018)
Region mum's birth	-0.001 (0.002)	-0.002 (0.008)	
Region mum aged 11	0.000 (0.001)	0.004 (0.008)	
Grandfather's social class, mum's birth	-0.005 (0.009)	0.026 (0.043)	
SES dad when cm born	-0.001 (0.001)	-0.001 (0.004)	
Father smoked, mum 16	0.009 (0.006)	0.004 (0.021)	
Mother smoked, mum 16	-0.004 (0.007)	-0.016 (0.020)	
Observations	6291	734	6291

Appendix 4.2. Smoking during pregnancy: other effects

Smoking during pregnancy may reduce child birth weight. Additionally, there may be other health problems. Tuormaa (1995) lists the other effects as:

reduced gestation and prematurity, increase miscarriages, placenta previa, placental abruption, premature rupture of membranes, sudden infant death syndrome.

6 Conclusion

This thesis has explored two aspects of the life cycle of skill formation. Firstly, it has provided evidence on whether the timing of income, over and above the level of family income, causes child human capital to accumulate differently. This was done in two ways. In chapter 2, using a fully flexible nonparametric functional form, income conditional upon permanent income, was estimated at ages 0-5, 6-11 and 12-17 upon a range of subsequent child outcomes. Placing no restrictions on the functional form of the human capital production function meant that not just the productivity of income, but also the complementarity in the return to income in each period was estimated. Whilst the theoretical literature provides arguments for parental investment in early years having a larger effect than later years, I found that early income was approximately as productive as income received aged 6-11, and less productive than income received age 12-17. Complementarities were found, for a group of poor parents likely to face credit constraints. In chapter 3, shocks to a stochastic income process were decomposed into permanent and transitory components, and subsequently the effect of these shocks upon child outcomes was estimated across the full range of childhood, from age 1-16. Permanent income shocks shift household wealth and subsequently investment in child human capital. An earlier shock was found to have a larger effect than a later shock, which was expected as the change to parental investment would last for more periods if the shock was realised early on. Transitory income shocks had a much smaller coefficient, which was in general constant across child age. The findings of these two papers are somewhat surprising, given the arguments in the theoretical literature which expect the return to parental investment to be higher early in the lifetime of a child. Indeed, whilst it was not possible to formally identify whether the mechanisms behind the findings were due to a higher return to later investment, they definitely indicate an important, non-negligible return to investment during adolescence. This has strong policy implications, suggesting that it is not just early intervention that will raise adolescent outcomes, but later investments will also yield a return. My plan for future research is to estimate a fully structural model of the role of the timing of income shocks upon child outcomes. The research will benefit from a model in which there are two types of investment goods - time and financial investment and additionally income shocks will be endogenised. This methodology would allow me to say something stronger about the mechanisms driving the differential return to income shocks.

The second aspect of skill formation was to evaluate the correlation between early child outcomes and parental inputs. Chapter 4 considered neo-natal smoking behaviour, and follow Todd & Wolpin (2007) in allowing for a history of inputs observed prior to the smoking behaviour through a maternal fixed effect. This chapter showed that the parameter estimated is very sensitive to the methodology adapted, as the coefficient on smoking during pregnancy fell by around two thirds once a maternal fixed effect was controlled for. However, what is often ignored by papers employing a fixed effect is that the estimate uses variation in inputs and outcomes only for individuals who change behaviour across births. Once the OLS was estimated on the group providing variation in the fixed effect, the coefficient was much closer to the fixed effect estimate. However, there was still a difference, which shows that even controlling for a wide range of maternal traits, much of the correlation between maternal smoking during pregnancy and child outcomes is caused by unmeasurable traits of the mother. Finally, chapter 5 considered whether the early determinants of test score outcomes at age 6 are similar across countries. The findings are that mother's education, family size and child gender are correlated with child test score outcomes to the same extent in the UK and the US. Additionally,

they show similar correlates in around 50% of the countries considered in the PIRLS dataset. This is a new finding. There is a plan to extend the PIRLS analysis, to create a measure for how similar or different countries are to each other in terms of their institutional settings, for example in the proportion of mothers working in the labour market or in the distribution of income, to take a step towards understanding how it is that countries with apparently different institutional settings have correlates between familial inputs and child outcomes which are similar in magnitude.

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