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# The infant health effects of starting universal child benefits in pregnancy: Evidence from England and Wales☆

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ABSTRACT

Child benefits are typically paid from birth. This paper asks whether starting universal child benefits in pregnancy leads to improvements in infant health. Leveraging administrative birth registry and hospital microdata from England and Wales, I study the effects of the Health in Pregnancy Grant, a universal conditional cash transfer equivalent to three months of child benefit (190 GBP) as a lump sum to pregnant mothers from 2009 to 2011. I exploit quasi-experimental variation in eligibility with a regression discontinuity design in the date of birth of the baby. I find that the policy increased birth weight by 8–12 grams on average, reduced low birth weight (<2500 g) by 3-6 percent and decreased prematurity by 9–11 percent. Younger mothers, particularly those living in deprived areas, benefit the most. I present evidence that the mechanisms are unlikely to be antenatal care, nutrition or smoking, with reductions in stress remaining a possible explanation.

## 1. Introduction

Child health is increasingly recognized by parents, health systems and welfare states as a form of human capital, to which a substantial proportion of private and public expenditure is dedicated (Kelly et al., 2020; World Health Organization, 2019). Child benefits are a popular policy instrument to improve child health. Empirical evidence suggests that child benefits can improve child health by increasing parental investment in children's nutrition and wellbeing, reducing family stress, and even reducing unhealthy behaviours in the family (Milligan and Stabile, 2011; Averett and Wang, 2013).

Child benefits tend to be paid to parents from birth. There is growing evidence, however, that health inequalities start *before* birth: nutrition, stress and unhealthy behaviours before and during pregnancy are important determinants of health in infancy and beyond (Aizer et al., 2016; Averett and Wang, 2013; Cowan and Tefft, 2012). Despite this, few countries start the payment of child benefits during pregnancy.[1] One example was the UK, which in 2009 introduced a universal Health in Pregnancy Grant (HPG), a one-off lump sum equal to three months of child benefit payments (190 GBP) in the third trimester of pregnancy. The cash transfer

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[1] A notable recent example is Italy, which in 2022 introduced the Assegno Unico Universale (AUU), a new universal child allowance which starts from the seventh month of pregnancy (Vidotto and Lucangeli, 2022). Meanwhile, France and Belgium both have universal one-off "birth payments" which are either paid at birth (France) or at 28–32 weeks of pregnancy at the earliest (Belgium). Finland offers a universal maternity package with a choice between a "baby box" of clothes and toys and a one-off 170 EUR cash transfer from the twenty-second week of pregnancy. However, since the box is worth more than the transfer, most women choose the baby box over the cash, including 95 percent of first-time mothers (Koivu et al., 2020). Other related policies from low-income countries include Nepal's universal Safe Delivery Incentive Programme (SDIP) and India's targeted Janani Suraksha Yojana (JSY), which pay cash incentives for women

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was conditional on attending an antenatal appointment with a doctor or midwife. The UK government claimed that the policy would address "the serious problem of underweight babies in this country" (Public Bills Committee, 2008, c.103).

I use two administrative datasets from birth registry and hospital records in England and Wales to identify the impact of the Health in Pregnancy Grant on infant health. My identification strategy exploits an arbitrary eligibility rule for the policy, whereby mothers of all babies with an expected date of birth on or after 6 April 2009 were eligible for the cash transfer. This enables me to conduct a regression discontinuity design in the date of birth of the baby and to estimate an intention-to-treat effect.

I find that the Health in Pregnancy Grant led to a small but statistically significant increase in mean birth weight of 8–12 grams at population level. This led to a reduction in the probability of being classified as low birth weight (<2500 g) of 0.2–0.4 percentage points (3–6 percent relative to the control mean). This is likely explained by increased gestational length and a reduction in the proportion of babies who are born prematurely (before 37 weeks of gestation), of 0.6–0.8 percentage points (9–11 percent in relative terms).

Babies born to younger mothers benefit the most from these infant health effects. Among younger mothers (aged 24 or under), my preferred specification indicates that the policy led to a statistically significant increase in mean birth weight of 25 grams, thereby reducing the probability of low birth weight (<2500 g) by 9 percent and extremely low birth weight (<1500 g) by 17 percent. These effects are driven in particular by the very youngest mothers (21 and under) and those living in deprived areas.

My results are robust to two separate administrative datasets, different bandwidths, parametric and non-parametric specifications. They receive strong support from a series of sensitivity tests. In particular, I show that there are no placebo effects on 6 April in years when the HPG was not introduced. This rules out the possibility that the beginning of the financial year induced a mechanical increase in birth weight. There is also no evidence of women manipulating their expected or actual dates of birth in order to receive the grant. In sum, I find strong causal evidence that the policy boosted infant health.

There are two possible explanations for this: that the conditionality of the grant incentivized earlier engagement with antenatal healthcare, and/or that the cash itself had an impact (through nutrition, stress or behaviours). To understand these mechanisms, I use administrative hospital data on the date of the first antenatal health appointment to identify whether the grant incentivized earlier engagement. At best, the policy appears to have incentivized women to attend an appointment about a day earlier than they would have otherwise. Back-of-the-envelope calculations suggest that the magnitude of this effect is extremely unlikely to explain the size of the birth weight effect (Cygan-Rehm and Karbownik, 2022). This indicates that the mechanism for the infant health effects was not the conditionality of the policy but the cash itself.

How did the cash make a difference? There are three main potential mechanisms: improved nutrition, lower stress and reduced smoking (either via stress or negative income effects). The grant seems to have driven longer gestational length rather than faster intrauterine growth, which makes improved nutrition an unlikely explanation for the effect (Amarante et al., 2016). I also find no evidence that the grant made women less likely to smoke during pregnancy. The main remaining hypothesis is that the grant reduced stress among pregnant women, thereby reducing the risk of prematurity and boosting birth weight.

My results are consistent with the stress hypothesis in two ways. First, stress predominantly affects birth weight via gestational length rather than intrauterine growth, and I find birth weight effects to be driven by the former rather than the latter (Torche, 2011; Kramer et al., 2000). Stress during the third trimester has a particular effect on prematurity, and my findings suggest that the HPG reduced prematurity by 9–11 percent. Second, low-income, younger and older mothers are particularly at risk of prenatal stress, and these groups were the biggest winners from the grant (García-Blanco et al., 2017; Persson and Rossin-Slater, 2018).

My primary contribution is to expand economic knowledge about the gains of starting universal child benefits in pregnancy. Evidence from low- and middle-income countries indicates that conditional cash transfers during pregnancy can increase antenatal health engagement and promote infant health (Siddiqi et al., 2018; Glassman et al., 2013). Simultaneously, there is evidence in high-income contexts that universal child benefits *after* birth can improve infant health among babies born to mothers already in receipt of child benefits for existing children (Milligan and Stabile, 2011; González and Trommlerová, 2022). Since the latter literature focuses on child benefits from birth, it excludes a key demographic group: babies born to first-time mothers. Child benefits cannot have direct effects on infant health for first-borns unless they start in pregnancy. First-time and younger mothers are at an elevated risk of low birth weight and prematurity, so the gains to intervention may be higher for this group. Indeed, I show that younger mothers (particularly those aged 21 and under, most of whom will not already be receiving child benefits)[2] see the greatest infant health gains from starting child benefits in pregnancy.

Second, I demonstrate that the potential for infant health gains from windfall transfers is larger than previously thought. While the effects I identify are small in absolute and clinical terms, the one-off cash transfer in question was also small (190 GBP, or 270.66 USD at purchasing power parity in 2009) (OECD, 2021). Scaling the birth weight estimates by the size of the transfer indicates effects that are approximately seven times as large as those identified in the literature (Hoynes et al., 2015). My findings indicate that starting child benefits in pregnancy may be a particularly efficient method of narrowing inequalities in infant and child health.

Finally, I contribute to the literature on conditional cash transfers (Baird et al., 2011; Cahyadi et al., 2020; Bastagli et al., 2016) by demonstrating that the main causal mechanism for the infant health effects is unlikely to be the conditionality of attending

who give birth in hospital. These cash incentives are not paid during pregnancy but at discharge from hospital after delivery (Powell-Jackson et al., 2009; Randive et al., 2013).

[2] In 2009, 87 percent of 20–21 year old women were childless and therefore not already claiming child benefits (Office for National Statistics, 2023).

an antenatal appointment. This suggests that, particularly in contexts with universal state-funded healthcare and high antenatal engagement, the benefits of conditionality may be limited.

The structure of this paper is as follows. Section 2 describes the Health in Pregnancy Grant in detail and reviews the literature on the impact of increases in income on infant health. Section 3 describes the data and sets out my identification strategy. Section 4 presents regression discontinuity estimates of the infant health and antenatal effects of the HPG, documents treatment heterogeneity, and implements a series of robustness checks. Section 5 discusses potential interpretations of these results and how they relate to existing research. Finally, Section 6 concludes and reflects on key policy implications.

## 2. Background

### 2.1. The Health in Pregnancy Grant, 2009–2011

In April 2009, the UK government introduced the Health in Pregnancy Grant (HPG), a cash transfer equivalent to three months of child benefit (190 GBP) as a lump sum in the third trimester to all pregnant women in the United Kingdom. Mothers of all babies with an expected date of birth on or after 6 April 2009 were eligible for the HPG (Wright, 2009). In order to claim the grant, pregnant women were required to visit their midwife or doctor for an antenatal check-up from the twenty-fifth week of pregnancy, when they would be invited to fill a simple application form for the HPG. After sending the form to the UK tax authorities in a free-post envelope, recipients received a letter confirming their application. They could expect to receive their money within seven days, through a direct payment into their bank or building society account (Directgov, 2010).

While the HPG was administered separately from child benefit, the central motivation of the policy was to start universal child benefits in pregnancy: a government minister stated in parliament that "we could call the measure "Child Benefit in Pregnancy", but we are calling it something else." (Public Bills Committee, 2008, c.105). In a government podcast to promote the HPG, it was stated that women could spend the money "on whatever you like", such as fresh fruit and vegetables, cots, nappies, pushchairs, and any other costs associated with having a healthy baby (Directgov, 2010). Supplementary Figure S1 is a screenshot of the UK government's website for the HPG. The government sent a clear message about the sorts of items that could be bought with the grant: the website includes illustrations of a birthing ball, a pushchair, a baby carrier, a cot, fruit and vegetables.

The primary aim of the HPG was to improve infant health by improving nutrition and reducing stress during pregnancy. In a parliamentary committee in 2008, a health minister argued that the HPG would "address the serious problem of underweight babies in this country" (Public Bills Committee, 2008, c.103).

The policy was controversial from the start. Opposition MPs portrayed the grant as a "gimmick" (Public Bills Committee, 2008, c.460). Childbirth charities raised concerns that the grant was poorly designed for nutritional impact, as the third trimester was too late in pregnancy for significant nutritional gains (Public Bills Committee, 2008, c.86). Political opponents also claimed that the grant would be squandered on "booze, fags, bingo or plasma screen televisions" and that its universalism was an inefficient use of resources (Public Bills Committee, 2008, cc.90-91; 103).

The HPG fell under renewed scrutiny in 2010 after a change of government and pressures for fiscal consolidation after the financial crisis. Less than two years after being rolled out across the UK, in January 2011, the HPG was abolished.

### 2.2. Background literature and mechanisms

Birth weight is known to be one of the most important health outcomes in the life course, with significant effects on subsequent child and adult health (Brooks-Gunn and Duncan, 1997). Babies who are born with a low birth weight (<2500 g) have a higher risk of infant mortality, particularly in the first 28 weeks after birth (Maruyama and Heinesen, 2020; Brooks-Gunn and Duncan, 1997). In adulthood, low birth weight can lead to "permanent health capital loss" (Maruyama and Heinesen, 2020, p.18) including higher blood pressure, Type-2 diabetes and lower offspring birth weight (Silverwood et al., 2013; Victora et al., 2008). Low birth weight is also associated with lower educational attainment and achievement, grade repetition, and lower labour market earnings (Maruyama and Heinesen, 2020; Currie and Rossin-Slater, 2015; Currie and Moretti, 2003; Behrman and Rosenzweig, 2004; Black et al., 2007). Finally, there is evidence that infant health is intergenerationally transmitted via maternal education (Currie and Moretti, 2003), thereby playing a role in the "the reproduction of inequality over generations" (Strully et al., 2010, p.535). This remains the case despite the decline in the relative importance of infant health in recent years (Maruyama and Heinesen, 2020; Goisis et al., 2017) and its dissipating importance over the life course (Maruyama and Heinesen, 2020).

The two main biomedical determinants of birth weight are gestational length (how long pregnancy lasts) and the speed of intrauterine growth (how fast the baby grows over a given period) (González and Trommlerová, 2022). Over and above its implications for birth weight, gestational length is an important infant health outcome in its own right. Premature babies (those born before 37 weeks) start their pre-school and school career weeks or even months earlier than their full-term peers. A growing literature on relative age effects has documented that children are, on average, at an advantage if they are older within their cohort (Campbell, 2014; Crawford et al., 2016). Prematurity can have long-lasting implications for academic attainment, designation with special education needs, teacher perceptions and even labour market prospects (Campbell, 2014).

Cash transfers have the potential to influence gestational length and intrauterine growth in two ways. First, cash transfers can be conditional on other behaviours (such as antenatal attendance) which improve health during pregnancy. Attending antenatal appointments at an early stage in pregnancy enables medical professionals to detect abnormalities and risk factors (including conditions like pre-eclampsia). It also facilitates access to information on how to stay healthy during pregnancy (including the

importance of folic acid supplements, and the risks of maternal smoking). Evidence from low- and middle-income countries indicates that conditional cash transfers can increase birth weight by improving antenatal care utilisation (Siddiqi et al., 2018; Glassman et al., 2013; Barber and Gertler, 2008). In a high-income context, Cygan-Rehm and Karbownik (2022) exploit a Polish reform which made receipt of an existing "baby bonus" (a universal one-off cash transfer after childbirth) conditional on attending antenatal care before 10 weeks. Since the cash transfer was already in place, the authors are able to isolate the causal effect of earlier timing of antenatal care. They find that earlier antenatal care has a modest but statistically significant effect on neonatal health, including birth weight. Exploiting an exogenous change in prenatal care usage in Pennsylvania due to a bus strike, Evans and Lien (2005) also show that prenatal care reduces the probability of smoking during pregnancy.

Second, cash transfers can improve infant health directly (González and Trommlerová, 2022; Hoynes et al., 2015; Almond et al., 2011; Strully et al., 2010). Transfers can be spent on "normal" goods and services that improve health and nutrition during pregnancy, such as fresh fruit and vegetables or wellbeing activities (González and Trommlerová, 2022; Amarante et al., 2016; Gregg et al., 2006). Cash can also improve infant health by reducing financial strain and stress. Evidence from domestic violence, family ruptures and earthquakes indicate that exposure to maternal stress has significant effects on infant health (Currie et al., 2022; Persson and Rossin-Slater, 2018; Torche, 2011). Windfall cash transfers (such as the US's Earned Income Tax Credit) have been found to reduce maternal stress and depression, with consequent implications for infant health (Boyd-Swan et al., 2016; Conway and Kennedy, 2004). Finally, there is empirical evidence that cash transfers can reduce unhealthy behaviours such as maternal smoking, alcohol and drug consumption. Some studies suggest that income has a direct and causal negative effect on smoking (Averett and Wang, 2013; Cowan and Tefft, 2012; Hoynes et al., 2015) – that it is an inferior good – though others have found this relationship to be spurious (Kenkel et al., 2014). As Hoynes et al. (2015) point out, it is also possible for cash transfers to reduce smoking if it is a normal good with a lower income elasticity than infant health, or if cash transfers lead to reductions in stress.

The HPG involved both conditionality and cash. Infant health effects could have proceeded from either, both or neither of these. As detailed in Section 2.1, the government pitched the 190 GBP grant as a lump sum that could be spent on healthy eating, wellbeing activities, or helping out with large costs in preparation for the arrival of a baby. The government also made the HPG conditional on attending antenatal healthcare: one of the main justifications for the grant being paid in the third trimester was that it would encourage women to seek antenatal health advice before 25 weeks. In fact, the only existing study of the HPG, in Scotland, found that the policy increased the odds of booking before 25 weeks by 10 percent (Leyland et al., 2017).

While Leyland et al. (2017) find significant effects of the HPG on antenatal engagement, they find no effects on birth weight, prematurity or maternal health in Scotland (Leyland et al., 2017). The study uses an interrupted time series analysis, which makes it difficult to control for contemporary events that coincided with the treatment period (April 2009 to April 2011). Given evidence that recessions impact on infant health (De Cao et al., 2022), it is possible that the global financial crisis and subsequent recession from 2008–09 may have introduced downward bias in Leyland et al. (2017)'s estimation of a treatment effect. In contrast, an RD methodology – as implemented in this paper – enables quasi-experimental conditions to be created such that potential outcomes are "as if" randomized local to the treatment cut-off (6 April 2009), and such exogenous shocks are controlled for.

## 3. Data and empirical strategy

### 3.1. Data

I use two separate administrative datasets: birth registrations microdata for England and Wales,[3] and birth episodes from Hospital Episode Statistics (HES) for England.[4] Both are high-quality administrative datasets at the individual (birth) level.[5] While the hospital data offer a rich set of variables on births, a recent linkage study found that birth registrations offer superior data quality and completeness (Ghosh et al., 2016). Birth registration is a statutory responsibility, whereas hospitals are not legally required to report certain details about the baby (including birth weight and gestational age) in hospital records.

I lead with results from the birth registry microdata. I use the hospital data to obtain data on gestational age and antenatal health, which are necessary for two purposes: to investigate effects on prematurity and antenatal engagement as outcomes; and to validate the use of actual date of birth as the running variable by comparing results with actual and expected date of birth (see Section 4.4.1). In both datasets, I drop duplicate records and stillbirths, and restrict my sample to observations between April 2006 and April 2014. Table 1 presents summary statistics for the birth registry data; Supplementary Table S1 does the same for the hospital data.

[3] This work contains statistical data from ONS which is Crown Copyright. The use of the ONS statistical data in this work does not imply the endorsement of the ONS in relation to the interpretation or analysis of the statistical data. This work uses research datasets which may not exactly reproduce National Statistics aggregates.

[4] Copyright © (2022), NHS England. Re-used with the permission of NHS England. All rights reserved.

[5] While there is an ongoing research project attempting to link these datasets (Coathup et al., 2020), unfortunately the linked data is not available for other researchers at present.

**Table 1**
Summary statistics from birth registry data, 2006–2014.

| | *N* | Mean | SD | Prop. complete |
|---|---|---|---|---|
| Birth weight (grams) | 5,604,297 | 3337.148 | 597.633 | 0.99 |
| Low birth weight | 5,604,297 | 0.071 | 0.257 | 0.99 |
| Extremely low birth weight | 5,604,297 | 0.011 | 0.105 | 0.99 |
| Multiple birth | 5,659,908 | 0.031 | 0.173 | 1.00 |
| Female baby | 5,659,908 | 0.487 | 0.500 | 1.00 |
| Maternal age (years) | 5,646,769 | 29.674 | 5.995 | 1.00 |
| Teenage mother | 5,646,769 | 0.055 | 0.228 | 1.00 |
| Index of income deprivation | 5,659,310 | 0.151 | 0.101 | 1.00 |
| Born in a NHS hospital | 5,659,908 | 0.969 | 0.174 | 1.00 |
| Lower socio-economic status (SES) | 520,635 | 0.281 | 0.450 | 0.09 |

*Note*: Data from administrative birth registrations for England and Wales. The sample is registered live births in England and Wales born between 6 April 2006 and 5 April 2014. Each column reports the number of observations, mean, standard deviation and the proportion of observations with a complete (non-missing) value respectively for each variable. Socio-economic status (SES) is only coded for a random sample of approximately 10 percent of the data, hence the lower proportion of complete observations.

#### *3.1.1. Birth registry microdata, 2006–2014*

Birth registry data provide the gold standard administrative data on births in England in terms of completeness and quality (Ghosh et al., 2016). Parents have a statutory responsibility to register a child being born within 42 days of the birth. The data are thereby exhaustive, covering all births that take place in England and Wales. Registration is completed by the mother and/or father at the local registry office. These details are digitized and processed by the Office for National Statistics (ONS). The ONS links the birth registry with a birth notification, which they receive from the doctor or midwife who was present at the baby's birth. The birth notification includes the birth weight of the baby, as measured in grams by the doctor or midwife at the time of birth.

The final birth registry microdata include the actual date of birth of the baby, birth weight (grams), stillbirth status, multiple birth status, sex of the baby, maternal and paternal age, and the postcode of the mother's residence. These variables are close to 100 percent complete. My main outcome variable – birth weight – is a discrete variable measured immediately after birth in grams.[6] I construct two dummy variables to capture the medical definitions of low birth weight (<2500 g) and extremely low birth weight (<1500 g).

The data do not have variables on income or earnings. While the ONS does code a random 10 percent sample of the data with the National Statistics Socio-economic Classification (NS-SEC) occupation category of the mother and the father, the sample size is too small for RD estimation with small bandwidths. Instead, I use the postcode of the mother's address to match in the index of income deprivation at Lower Super Output Area (LSOA) level (a small neighbourhood of approximately 1500 people). The index of income deprivation is a score capturing the proportion of the population in the area who are in receipt of means-tested benefits and likely on a low-income.[7]

#### *3.1.2. Hospital Episode Statistics microdata, 2006–2014*

My second source of data is administrative hospital records (Hospital Episode Statistics) from the National Health Service (NHS) in England. The NHS is the main provider of healthcare in the UK and offers universal healthcare which is free at the point of use, funded by general taxation. English NHS hospitals collect these data so that hospital activity can be reimbursed by the government through payment-by-results.[8] The vast majority (97 percent) of registered births in England take place in NHS hospitals (see Table 1). The data exclude the remaining 3 percent of births that take place in private hospitals, at home or elsewhere.

The hospital microdata include the baby's birth weight (grams), gestational age at birth (weeks),[9] gestational age at the first antenatal appointment (weeks), the number of babies in the pregnancy, maternal age at birth (years) and ethnicity. The data extract does not include daily date of birth; instead I have access to a bespoke variable for the actual week of birth relative to the 6 April

[6] I code outliers with a recorded birth weight less than 265 grams or larger than 5650 grams as missing. This includes missing values coded as '9999' or '9998'. Supplementary Figure S2 shows a histogram of the final birth weight distribution.

[7] I match index of income deprivation scores for England and Wales from the nearest available data point to the introduction of the HPG (2015) to the births data at LSOA level by the mother's postcode of residence.

[8] While the NHS is UK-wide, healthcare is devolved to the four nations of the UK (England, Wales, Scotland and Northern Ireland), so Hospital Episode Statistics only cover England.

[9] I drop implausible gestational ages based on the birth weight data in line with the method described in Herbert et al. (2017).

2009 cut-off.[10] I calculate the expected week of birth ($e_{wob}$) using gestational age at birth in weeks and the actual week of birth relative to the cut-off of 6 April 2009 ($wob$).[11]

### 3.2. Empirical strategy

I identify the infant health effects of the Health in Pregnancy Grant by exploiting plausibly exogenous variation in the date of birth of the baby with a regression discontinuity design. The policy design of the HPG makes it an ideal candidate for a regression discontinuity, since it was simultaneously universal and administered on an arbitrary basis by expected date of birth: all mothers of babies with an expected date of birth on or after 6 April 2009 were eligible for the grant. To the best of my knowledge, no other relevant policy affecting pregnant women was introduced discontinuously on 6 April 2009.

While arbitrary cut-offs were used both in the introduction and the abolition of the HPG (Howard, 2016), I focus exclusively on the introduction cut-off. The abolition of the HPG in January 2011 (effective for expected dates of birth from April 2011) unfortunately coincided with the restriction of the Sure Start Maternity Grant to the first child and the abolition of the baby tax credit, which may confound identification of the effects of the HPG's abolition.

Since claiming the HPG was dependent on attending an antenatal check-up, it exhibited imperfect compliance. Estimates from tax authority expenditure data suggest high take-up rates of 92.9 percent.[12] This high but imperfect compliance introduces "fuzziness" in the discontinuity, as the probability of being treated increases sharply from 6 April 2009 but falls short of reaching unity. Since receipt of the HPG is not measured in the data, I do not estimate a treatment on the treated (TOT) or local average treatment effect (LATE) through a fuzzy RD approach. Instead, I estimate an intention-to-treat (ITT) effect with a traditional sharp RD, with actual date of birth as the running variable.

My main empirical specification is the following:

$$Y_i = \beta_0 + \beta_1 \mathbb{1}[dob_i \geq 0] + \beta_2 dob_i + \beta_3 dob_i \times \mathbb{1}[dob_i \geq 0] + \epsilon_i \tag{1}$$

$Y_i$ is the outcome of choice (e.g., birth weight in grams), $dob_i$ is the baby's actual date of birth in days relative to the introduction cut-off $\bar{x}$ (6 April 2009), $\epsilon_i$ is the error term, and $i$ indexes individual births. $\beta_1$ is the coefficient of interest and represents the ITT effect of the HPG.

My preferred specification is a non-parametric local linear RD: this allows the slope of the prediction to change either side of the cut-off, thereby letting the data speak for itself local to the discontinuity (Hahn et al., 2001; Cattaneo et al., 2019). As recommended by Calonico et al. (2021), I select my preferred bandwidth by optimizing the coverage error rate (CER) and use robust bias-corrected standard errors (Cattaneo et al., 2019; Hyytinen et al., 2018). I test versions of this specification by changing the bandwidth size and smoothing kernel (triangular or uniform), and by adding a vector of controls (maternal age (years), multiple birth status, income deprivation score, and the sex of the baby). Additionally, I test for robustness to parametric specifications with linear, quadratic and cubic trends in the running variable, controls and day-of-the-week fixed effects.

While the HPG was implemented on the basis of expected date of birth ($e_{dob}$), this is not recorded in birth registry data. I therefore use actual date of birth ($dob$) as a proxy for expected date of birth ($e_{dob}$). This can be expected to introduce some measurement error in the running variable. One concern is that this measurement error could be non-random. In Section 4.4.1, I use the hospital data to validate the use of actual date of birth against expected date of birth and show that my results are robust to measurement error of this kind.

### 3.3. Identification checks

The identifying assumption of RD designs is that individuals lack precise control over the running variable in question. This could be violated in two ways within this context. First, women could postpone conception to push their expected or actual date of birth until after the 6 April 2009 cut-off, thereby making them eligible for the HPG. Second, women could mislead healthcare workers about the date of their last menstrual period in order to manipulate their expected date of birth. They could alternatively persuade healthcare workers to record an inaccurate expected date of birth at their ultrasound dating scan.

The first general discussion of eligibility requirements that mentions the approximate cut-off of April 2009, according to newspaper reports, appears to have been September 2007 (BBC News, 2007). However, no specific cut-off date was given, and

[10] The secure data application process for Hospital Episode Statistics is lengthy and approval from the Confidentiality Advisory Group is required for access to daily date of birth. Since expected date of birth is not directly recorded in birth episodes, the only way of calculating the expected due date of the baby is to combine data on the gestational age at birth (in weeks) and the actual week of birth of the baby. It is therefore not necessary to know the daily date of birth for these purposes. Given this, I requested a bespoke actual week of birth variable relative to the treatment cut-off of 6 April 2009 to be created by NHS England. Actual dates of birth from 31 March–5 April are coded with an actual week of birth of −1; 6–13 April 2009 are coded as 0; 14–21 April 2009 are coded as 1; and so on.

[11] I calculate the expected week of birth ($e_{wob}$) as the following: $e_{wob} = wob + (40 - gestage)$, where $wob$ is the actual week of birth relative to 6 April 2009 and $gestage$ is gestational age at birth in weeks. Since missingness of gestational age is not random, I replace expected week of birth with the actual week of birth if the former is missing to avoid sample selection. Results are virtually unchanged when doing this.

[12] For the 2009–2010 financial year, reported expenditure on HPG grants – excluding administrative costs – totalled 137.8 million GBP in the UK as a whole (Her Majesty's Revenues and Customs, 2010, p. 55). Given that the grant was a flat-rate lump sum of 190 GBP for all women, I estimate that approximately 725,260 women received the grant in 2009–2010. This implies an extremely high take-up rate of the 781,000 maternities that were recorded in the UK in 2009 (Office of National Statistics, 2021; National Records of Scotland, 2021; Northern Ireland Statistics and Research Agency, 2020), of approximately 92.9 percent.

it was not stated what point of gestation would have to been reached by April 2009 to be eligible. The possibility that prospective mothers could have precisely timed their conception in order to get the grant is, therefore, remote. In addition, the hypothesis that women could persuade healthcare workers to misrepresent their expected date of birth seems implausible. Survey evidence indicates that in the year of the HPG's introduction, 90 percent of pregnant women attended an ultrasound dating scan, which is the main way of assessing gestational age and expected date of birth (Redshaw and Heikkila, 2010). Gestational age is objectively estimated from the crown–rump length (CRL) in the ultrasound scan by a specialist sonographer, who is subject to regular retrospective quality assurance (Royal College of Radiologists, 2014; Lodge et al., 2014; Salomon et al., 2013). Additionally, misrepresenting expected dates of birth would carry clinical risks. The accurate recording of gestational age is an important piece of clinical information for most obstetric and neonatal decisions and procedures, both before and after birth. Medical professionals need to know an accurate gestational age in order to assess small-for-gestational-age (SGA) status, intrauterine growth retardation (IUGR), and the optimal timing of induction (Trust, 2020; Salomon et al., 2013).

I test for manipulation around the cut-off empirically with a McCrary test, which examines whether there is discontinuous bunching in the density of births around the cut-off. If women manipulated their dates of birth, there would be a statistically significant positive McCrary estimate and graphical evidence of bunching to the right of the cut-off. Supplementary Figure S3 and Supplementary Figure S4 show McCrary manipulation plots for the running variable in the birth registry data (actual date of birth) and the hospital data (actual week of birth and expected week of birth). McCrary RD estimates and p-values are included on each graph. They are all negative and statistically insignificant: the null hypothesis of no bunching cannot be rejected. This indicates that there was no manipulation of expected or actual dates of birth by women or healthcare workers.

I test the identifying assumption that births below the cut-off are a valid counterfactual for those above the cut-off further by implementing covariate balance tests. Supplementary Table S2 and Supplementary Table S3 display summary statistics separately for the treatment and control group (as defined by being above or below the 6 April 2009 cut-off) with the birth registry and hospital data respectively. Treatment and control groups are broadly balanced by key covariates. Supplementary Table S4 presents a full set of formal covariate balance results. Multiple births, female babies, local deprivation rates, teenage mothers, births in NHS hospitals, and parents in low socio-economic status (SES) occupations are all clearly balanced around the cut-off. Two models indicate statistically significant results for maternal age, but these are sensitive to the choice of bandwidth and smoothing kernel, and are null when the CER-optimal bandwidth from my main results is utilized (see Column 1 of Supplementary Table S4, which corresponds with Column 1 of Table 2 below). Additionally, Supplementary Figure S5 shows that there is no compelling evidence of discontinuities around 6 April 2009 for key covariates. This lends support to the assumption that births are "as if" randomized either side of the cut-off, thereby supporting the validity of a regression discontinuity design.

## 4. Results

I present my findings in four parts. First, I estimate the intention-to-treat effect of the HPG on infant health, and investigate the extent to which this is driven by intrauterine growth or longer gestational length. Second, I leverage data on antenatal health booking to test whether the infant health effects are driven by conditionality or cash. Third, I document heterogeneity in the effects of the grant. Finally, I present two major robustness checks to check for non-random measurement error in the running variable and for effects at placebo cut-offs.

### *4.1. Effects on infant health*

#### *4.1.1. Birth weight*

Table 2 presents RD estimates of the birth weight effects of the Health in Pregnancy Grant using the birth registry data. Column 1 is my preferred model and is a non-parametric local linear RD with a triangular kernel for the bandwidth which optimizes the coverage error rate (CER), as recommended by Calonico et al. (2021). This model indicates a 11.8 gram increase in mean birth weight (significant at the 5 percent level) and a 0.3 percentage point (4 percent relative to the control mean) fall in the probability of low birth weight (significant at the 10 percent level). The estimated effect on extremely low birth weight (<1500 g) is marginally insignificant, at a magnitude of 0.1 percentage points (9 percent relative to the control mean).

Column 2 repeats the same specification as Column 1 for the bandwidth which minimizes the mean-squared error (MSE). This produces a larger bandwidth than Column 1 and suggests similar birth weight effects: an 8.3-gram increase in mean birth weight and a 0.2 percentage-point decline in low birth weight, both of which retain significance at the 5 percent level. The estimated effect on extremely low birth weight (a reduction of 0.1 percentage points) becomes statistically significant at the 5 percent level.

Column 3 tests for robustness to the choice of smoothing kernel by implementing a uniform kernel for the CER-optimal bandwidth. Effects are almost identical to those from Column 1. Finally, Column 4 tests for sensitivity to the addition of controls: maternal age (years), multiple birth status, income deprivation score, and the sex of the baby. Effects on mean birth weight are virtually unchanged when controls are added. The low birth weight effect loses significance when controls are added, but remains identical in magnitude across models, as does the coefficient for extremely low birth weight.

As Supplementary Table S5 shows, these results are stable across bandwidths. Effects are also very similar under parametric specifications with a linear, quadratic or cubic functional form, controls and day-of-the-week fixed effects, as shown in Supplementary Table S6 and Table S7.

Back-of-the-envelope calculations indicate that the significant effects on mean birth weight and the low birth weight dummy from Table 2 are consistent: prior to the reform, 0.6–1.8 percent of low birth weight babies were within 8.3–11.8 grams (the lower

**Table 2**
Effect of the Health in Pregnancy Grant on birth weight.

| | Regression discontinuity | | | | Control mean |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| Birth weight (grams) | 11.798** | 8.251** | 11.593* | 10.903** | 3336.136 |
| | (5.485) | (3.920) | (6.398) | (4.751) | |
| Bandwidth (days) | 61.20 | 133.11 | 37.74 | 73.03 | |
| *N* | 232,489 | 506,032 | 141,456 | 277,610 | 4,202,919 |
| Low birth weight | −0.003* | −0.002* | −0.004* | −0.003 | 0.071 |
| | (0.002) | (0.001) | (0.002) | (0.002) | |
| Bandwidth (days) | 102.04 | 221.92 | 83.23 | 117.04 | |
| *N* | 386,988 | 849,363 | 315,626 | 445,099 | 4,202,919 |
| Extremely low birth weight | −0.001 | −0.001** | −0.001 | −0.001 | 0.011 |
| | (0.001) | (0.001) | (0.001) | (0.001) | |
| Bandwidth (days) | 146.13 | 317.81 | 99.48 | 157.22 | |
| *N* | 555,549 | 1,221,205 | 376,211 | 597,308 | 4,202,919 |
| CER-optimal | X | | X | X | |
| MSE-optimal | | X | | | |
| Controls | | | | X | |
| Kernel | Triangular | Triangular | Uniform | Triangular | |

*Note*: The table reports the coefficient $\beta_1$ from Eq. (1) for different outcome variables. Column 1 is a local linear RD with a triangular kernel and a CER-optimal bandwidth. Column 2 is a local linear RD with a triangular kernel and an MSE-optimal bandwidth. Column 3 is a local linear RD with a uniform kernel and a CER-optimal bandwidth. Column 4 is a local linear RD with a triangular kernel, a vector of controls (maternal age (years), multiple birth status, income deprivation score, and the sex of the baby) and a CER-optimal bandwidth. All standard errors are robust bias-corrected and are listed in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Data from administrative birth registrations for England and Wales.

and upper bound on the treatment effect of Table 2) of the 2500 g threshold. If birth weight gains were constant across the birth weight distribution, this would imply an expected reduction in low birth weight in the order of 0.04–0.13 percentage points. As shown in Supplementary Figure S6, quantile regression suggests that the greatest birth weight effects are at the very bottom of the birth weight distribution. Given this, the expected effect on low birth weight can be expected to be slightly higher than 0.04–0.13 percentage points. My observed treatment effect on low birth weight from Table 2, of 0.2–0.4 percentage points, is consistent with this.

Fig. 1 shows the birth weight effects from Model 1 graphically, with RD estimates and robust bias-corrected standard errors labelled on the graph. There is a clear positive discontinuity in birth weight at the introduction of the HPG. The figure indicates that the birth weight data is linear around the cut-off, making a local linear RD appropriate. As I show in Supplementary Figure S7 and Supplementary Figure S8, these increases in the raw birth weight data also lead to small negative discontinuities in low and extremely low birth weight.

#### *4.1.2. Gestational length and prematurity*

Next, I use the hospital data on gestational age to investigate effects on gestational length and prematurity. The hospital data do not include daily date of birth, so here I use week of birth (relative to 6 April 2009) as the running variable. Models 1–4 in Table 3 are defined in the same way as those from Table 2. All models indicate a small positive effect on gestational age at birth which is statistically significant at the 1 percent level. This effect is robust to the use of an MSE-optimal bandwidth (Column 2), a uniform smoothing kernel (Column 3) and the addition of controls (Column 4). The magnitude of the effect is small – ranging from 0.07–0.08 weeks (less than a day).

The HPG does, however, appear to have made a sizeable difference to babies at risk of being born prematurely. My preferred model (Column 1 of Table 3) indicates a decline in the probability of being born prematurely (before 37 weeks) of 0.7 percentage points (10 percent relative to the control mean). This effect is statistically significant at the 1 percent level and is stable in magnitude and significance across models, ranging between 0.6–0.8 percentage points. Fig. 2 illustrates the effect on prematurity with RD estimates from Model 1: again, there is a visible discontinuity at the introduction of the HPG.

While gestational length and prematurity are infant health outcomes in their own right, they are also relevant because more time *in utero* leads to higher birth weights. I investigate whether the observed birth weight effects from Section 4.1.1 can be explained by the above increases in gestational length in two ways. First, in Supplementary Table S8 I follow González and Trommlerová (2022) in adding fixed effects for gestational length (in weeks) to my birth weight RD model. This helps to test whether the increases in birth weight are attributable to longer gestational length or to faster intrauterine growth. The birth weight effects shrink and lose statistical significance when gestation fixed effects are added: conditional on gestation, the null hypothesis that babies are no heavier due to the grant cannot be rejected. This suggests that the birth weight gains are explained by longer gestation and the reduction of prematurity, rather than faster intrauterine growth.

One limitation of this analysis is that, if gestational age is a mechanism for birth weight effects via the HPG, it could be a "bad control". Given this, I sense-check this analysis with a back-of-the-envelope calculation where I take the average birth weight difference between a baby born one week earlier (185 grams) and multiply this by the treatment effect on gestational age in weeks from Table 3. This implies that the expected birth weight effect from increased gestational length is 13 grams, which is very similar to my main birth weight estimate. It is therefore plausible that the observed birth weight effects are explained by gestational age.

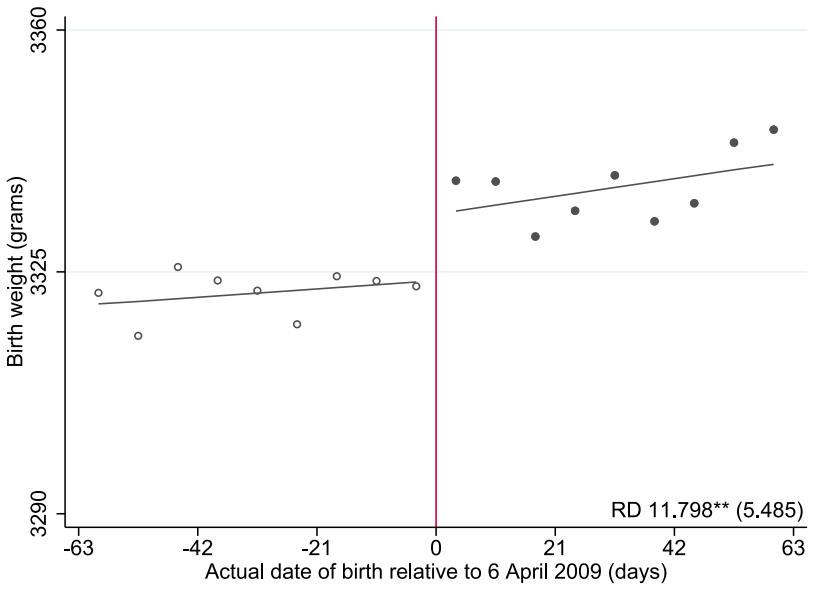


**Fig. 1.** Effect of the Health in Pregnancy Grant on birth weight.
*Note*: The figure plots mean birth weight by actual week of birth bin for the CER-optimal bandwidth from Table 2 (61.2 days). A linear spline is fitted to the raw data either side of the cut-off. The red vertical line indicates the first date of birth (6 April 2009) for which pregnant women were eligible for the Health in Pregnancy Grant. The running variable is actual date of birth. The figure also reports the coefficient $\beta_1$ from Eq. (1), with associated robust-bias-corrected standard errors in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Data from administrative birth registrations for England and Wales.

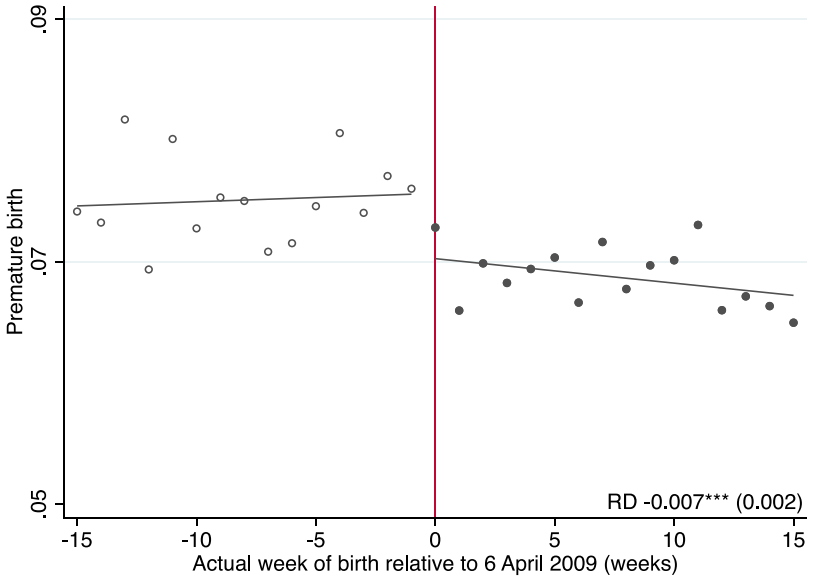


**Fig. 2.** Effect of the Health in Pregnancy Grant on the probability of being born premature (<37 weeks).
*Note*: The figure plots the proportion of babies who are born before 37 weeks of gestation by actual week of birth bin for the CER-optimal bandwidth from Table 3 (15.04 weeks). A linear spline is fitted to the raw data either side of the cut-off. The red vertical line indicates the first date of birth (6 April 2009) for which pregnant women were eligible for the Health in Pregnancy Grant. The running variable is actual week of birth because the hospital data do not include daily date of birth. The figure also reports the coefficient $\beta_1$ from Eq. (1), with associated robust-bias-corrected standard errors in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Data from administrative Hospital Episode Statistics for England.

**Table 3**
Effect of the Health in Pregnancy Grant on gestational length, prematurity and antenatal engagement.

| | Regression discontinuity | | | | Control mean |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| Gestational age at birth (weeks) | 0.070*** | 0.084*** | 0.072*** | 0.067*** | 39.198 |
| | (0.017) | (0.013) | (0.019) | (0.019) | |
| Bandwidth (weeks) | 17.03 | 36.23 | 12.45 | 20.59 | |
| *N* | 307,416 | 652,059 | 218,404 | 231,154 | 2,531,746 |
| Premature birth | −0.007*** | −0.006*** | −0.007*** | −0.008*** | 0.070 |
| | (0.002) | (0.002) | (0.002) | (0.003) | |
| Bandwidth (weeks) | 15.04 | 31.99 | 11.08 | 17.20 | |
| *N* | 270,966 | 561,840 | 200,929 | 197,905 | 2,531,746 |
| Gestational age at first antenatal assessment | 0.042 | −0.169** | 0.057 | 0.007 | 13.259 |
| | (0.103) | (0.077) | (0.119) | (0.129) | |
| Bandwidth (weeks) | 8.42 | 17.81 | 5.63 | 9.09 | |
| *N* | 131,451 | 269,370 | 85,876 | 92,537 | 2,260,436 |
| CER-optimal | X | | X | X | |
| MSE-optimal | | X | | | |
| Controls | | | | X | |
| Kernel | Triangular | Triangular | Uniform | Triangular | |

*Note*: The table reports the coefficient $\beta_1$ from Eq. (1) for different outcome variables. Dependent variables are defined as in Supplementary Table S1. Column 1 is a local linear RD with a triangular kernel and a CER-optimal bandwidth. Column 2 is a local linear RD with a triangular kernel and an MSE-optimal bandwidth. Column 3 is a local linear RD with a uniform kernel and a CER-optimal bandwidth. Column 4 is a local linear RD with a triangular kernel, a vector of controls (maternal age (years), multiple birth status, and whether the baby's recorded ethnicity is White British). The running variable is actual week of birth because the hospital data do not include daily date of birth. All standard errors are robust bias-corrected and are listed in parentheses. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Data from administrative Hospital Episode Statistics for England.

### 4.2. Effects on antenatal health engagement

The HPG was available to pregnant women from the twenty-fifth week of pregnancy, and was conditional on having attended an antenatal appointment. Since changes in antenatal usage could contribute to infant health effects, I leverage the hospital data to test whether the grant incentivized earlier attendance at the first antenatal appointment. Unfortunately, the hospital data do not include the timing of all antenatal appointments, just the first one. As shown in Supplementary Table S1, the mean gestational age at the first appointment is 13 weeks – much earlier than receipt of the HPG. However, the timing of the first antenatal appointment is a relevant margin of response for three reasons. First, Supplementary Table S3 shows that, prior to the HPG, 9 percent of mothers had not attended their first antenatal appointment by the twenty-fifth week. These mothers may have been incentivized to attend when they became eligible for the HPG in the twenty-fifth week. Second, HPG eligibility was based on expected date of birth as validated by a doctor or midwife and, for most mothers, this is determined by a "dating scan" at the first antenatal appointment. If mothers were keen to establish their eligibility for the grant, they may have been more likely to book their appointment early. Third, evidence indicates that the infant health gains to antenatal attendance are stronger in the early stages of pregnancy, particularly the first trimester (Cygan-Rehm and Karbownik, 2022). Infant health effects from the HPG via antenatal attendance are arguably more likely to occur, therefore, from the first appointment than subsequent ones.

RD estimates on gestational age at the first antenatal appointment in Table 3 are variable in sign and significance. Models 1, 3 and 4 suggest positive and non-significant effects of 0.007–0.06 weeks. Model 2 suggests a negative effect of 0.17 weeks (about a day), which is significant at the 5 percent level. However, placebo cut-off tests for this variable in Supplementary Table S9 indicate that the treatment effects are very similar to placebo effects in other years. This suggests that the significant effect on antenatal attendance in Column 2 of Table 3 is spurious and unlikely to be causally attributable to the HPG.

In Supplementary Table S10 I investigate whether there are valid antenatal health effects concentrated at specific stages of pregnancy by listing results for four dummy variables which capture attendance during the first, second, or third trimester, and before 25 weeks. The point estimates are again variable. Similarly, these dummy variables do not perform well in placebo cut-off tests.[13] Taken together, these results indicate that the null hypothesis of no treatment effect of the HPG on antenatal care cannot be decisively rejected.

### 4.3. Heterogeneous treatment effects

While the HPG was universal, effects are unlikely to have been uniform across the population: diminishing returns imply that 190 GBP should make a greater difference to low-income and disadvantaged women. I test for such treatment heterogeneity by running separate RD analyses by maternal age and the index of income deprivation, a geographical index of socio-economic status.

[13] Placebo cut-off tests for these variables are available on request.

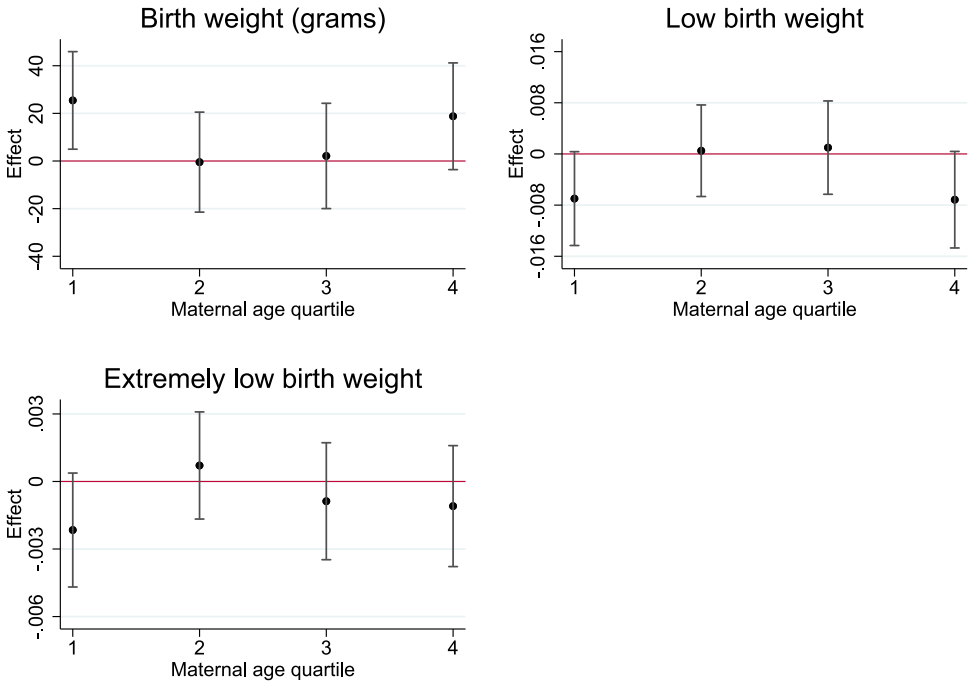


**Fig. 3.** Birth weight effects by maternal age quartile.
*Note*: Each panel plots the coefficient $\beta_1$ from Eq. (1), estimated separately for each maternal age quartile. The dependent variables are birth weight (grams), low birth weight (<2500 g) and extremely low birth weight (<1500 g) respectively. To ensure consistency across quartiles, the same CER-optimal bandwidth for the relevant variable from Table 2 is used for each quartile. The ranges of the maternal age quartiles are as follows: 24 and under (1st quartile); 24–29 years; 29–34 years; and 34 and over (4th quartile). Error bars represent 95 percent confidence intervals. Data from administrative birth registrations for England and Wales.

Interestingly, Fig. 3 indicates that birth weight effects are u-shaped by maternal age quartile, with younger (24 and under) and older (34 and older) mothers benefiting the most. Younger mothers (aged 24 and under) experienced a 25-gram increase in mean birth weight (statistically significant at the 5 percent level); older mothers saw a marginally insignificant increase of 19 grams. For more granular estimates by maternal age, Supplementary Figure S9 plots RD estimates by maternal age decile. Effects are more erratic and less precise when deciles are used due to the smaller sample size, but it is telling that the only group to have had significant birth weight effects are the very youngest mothers (aged 21 and under). This group saw a significant and sizeable 35-gram increase on average – almost threefold the effect on the population as a whole.

Fig. 3 also indicates that effects on low birth weight are also u-shaped across the maternal age distribution. Younger mothers (aged 24 and under) and older mothers (aged 34 and over) both experienced a reduction in the probability of being low birth weight of 0.7 percentage points (9 percent relative to the control mean for each group), statistically significant at the 10 percent level. Additionally, younger mothers benefited from a reduction in extremely low birth weight of 0.2 percentage points (17 percent), which is significant at the 10 percent level.[14]

To probe these results by maternal age further, in Supplementary Table S11, I split younger and older mothers into those living in areas with above- or below-median levels of deprivation. This reveals that the effects were driven predominantly by low-income young mothers. Babies born to mothers aged 24 or under in deprived areas were the only group to see significant birth weight effects: a 29-gram increase in mean birth weight, and one-percentage-point reduction in low birth weight (12 percent).

### *4.4. Robustness checks*

As documented above, my results are robust to different bandwidths (Supplementary Table S5), choice of smoothing kernel (Table 2) and parametric and non-parametric approaches (Table 2, Supplementary Table S6 and Supplementary Table S7). In this section I present results from two more major robustness checks. First, I test whether my results are an artefact of systematic measurement error in the running variable. Second, I conduct four placebo cut-off tests at 6 April in 2007, 2008, 2010 and 2012.

[14] Unfortunately, I am not able to investigate heterogeneous effects on prematurity or antenatal health engagement with the hospital data. First, just 67 percent of babies' records have data on maternal age. When combined with the completeness of the birth weight, gestational age, and antenatal appointment data, this means that approximately half of the sample cannot be used in maternal age heterogeneity analyses. Since the remaining sample are likely to be systematically different from the missing observations, such results would be biased. Second, the hospital data only include postcode of residence for a handful of observations, so the index of income deprivation cannot be used.

#### 4.4.1. Measurement error in the running variable

One potential problem with my empirical strategy could be that using actual date of birth ($dob$) as a proxy for expected date of birth ($e_{dob}$) leads to systematic measurement error in the running variable. Some late babies ($dob > e_{dob}$) will be categorized as treated when they were not, while some premature babies ($e_{dob} > dob$) will be categorized as untreated when they were. Both of these effects would introduce upward bias in my estimated treatment effect, since late babies are heavier and premature babies lighter on average. I test for this possibility in two ways.

First, I exploit the hospital microdata to validate the use of actual date of birth against expected date of birth. In the hospital data, I do not have access to daily date of birth, and gestational age is provided in discrete weeks rather than days. I therefore compare results with actual and expected week of birth as the running variable.[15] As shown in Supplementary Table S13, RD estimates of the effect on mean birth weight are consistently positive when using expected week of birth, with all models indicating significant effects in the region of 17–30 grams, apart from Model 4, which indicates a non-significant effect of 10 grams. Comparing full results for expected week of birth in Supplementary Table S13 with those for actual week of birth in Supplementary Table S14, the estimated effect with expected week of birth as the running variable is statistically indistinguishable from the effect with actual week of birth in all models. The direction of the measurement error varies across models, ranging from estimates that are 53 percent lower with expected week of birth in Model 1 to estimates that are 55 percent higher in Model 3. This is at least suggestive of a random component to the measurement error. To be conservative, however, I apply the upper bound of this attenuation rate to my main estimates from Table 2. This implies a birth weight increase of at least 5.5 grams for the population as a whole, and 16.4 grams for my highest-impact sample (mothers aged 21 or under).

Supplementary Figure S10 graphically compares the RD with actual and expected week of birth as the running variable. A clear discontinuity persists in the data when using expected week of birth. Supplementary Figures S11, S12 and S13 repeat the same validation for low birth weight, extremely low birth weight and prematurity: again, estimates are statistically indistinguishable across running variables but this time effects are identical or, indeed, larger when using expected week of birth.

Second, I use a "donut" RD to exclude small bands of observations around the cut-off, as recommended by Barreca et al. (2011) in the case of measurement error in the running variable. If systematic measurement error is a problem, it should be more of an issue for observations that are close to the cut-off: these are the observations which will be "miscategorized" in the control or treatment group and thereby create upward bias in the treatment effect (Barreca et al., 2011). Results for a series of donut sizes are listed in Supplementary Table S15. The magnitude of the RD estimate in fact increases incrementally when donuts of radius 1–3 days are applied, before decreasing slightly to 7.4 grams with a 7-day radius, and finally increasing again to 9.5 grams with a 21-day radius. These results indicate that the observed birth weight effects are robust to adjusting for measurement error in the running variable local to the cut-off, and that this bias turns out to be minor: an increase of 7–15 grams withstands the donut test. This aligns with the result of the above expected date of birth test and suggests that the observed treatment effects are not an artefact of measurement error. Taken together, these checks indicate that systematic measurement error in the running variable is minor, and does not present a threat to identification.

#### 4.4.2. Placebo cut-off tests

Next, I test the validity of the RD by conducting placebo cut-off tests at the treatment date (6 April) in four different years, both before and after the introduction of the policy.[16] Birth weights vary by season, and there is a natural incline in April (Currie and Schwandt, 2013). Additionally, 6 April represents the start of the financial year in the UK. It is important to check whether the observed infant health effects are simply the reflection of discontinuous variation around this date. Full placebo cut-off results for birth weight are given in Table 4.[17] There are no significant birth weight effects, in either direction, at any of the placebo cut-offs. Fig. 4 illustrates this by plotting the binned birth weight data around each placebo cut-off: no discontinuities are visible.[18] Supplementary Figures S14 and S15 also show that there are no negative placebo effects for low or extremely low birth weight dummy variables respectively.[19]

Placebo cut-off results for prematurity with the hospital data are listed in Supplementary Table S17. These are slightly less clean than the birth weight placebos, but the sign of the coefficient varies. There are significant effects in 2010, but these are positive (the opposite direction of the treatment effect) and only one model in one year (2007) suggests a statistically significant negative effect. As Supplementary Figure S16 shows, the graphical evidence for my preferred model (Column 1 of Supplementary Table S17) does not indicate negative discontinuities in prematurity in placebo years. This suggests that the observed negative treatment effect on prematurity, which is by contrast very robust across models, is likely to be attributable to the policy.

[15] To verify that it does not matter whether daily actual date of birth or actual week of birth is used, in Supplementary Table S12 I show results from the birth registry data with actual week of birth as the running variable. Results are very similar to those with actual date of birth in Table 2. When comparing results with actual week of birth across the two datasets, RD estimates are larger and more variable in the hospital data (11–28 grams) than the births data (8–11 grams) when using actual week of birth as the running variable. This is likely to be due to the superior data quality of the birth registry data: the births data contain complete birth weight data on all births that took place in England and Wales, whereas the hospital data only record birth weight for 75 percent of births (see Supplementary Table S1 for completeness of all variables).

[16] I do not use 2011 as a placebo cut-off because the Health in Pregnancy Grant was abolished for all women with an expected date of birth on or after 16 April 2011 (alongside other changes to financial support affecting families with children and pregnant women), so 2011 is not plausibly a "placebo".

[17] As recommended by Cattaneo et al. (2019), to avoid contamination by "real" treatment effects, for years in which the policy was not in place (2007, 2008 and 2012) I use only control observations, and for years in which the policy was already in place (2010), I use only treatment observations.

[18] Supplementary Table S16 verifies there are no significant positive placebo effects when expected date of birth is used as the running variable in the hospital data.

[19] Low birth weight displays a significant positive effect in 2010, but if anything this lends further support to the assumption that 6 April does not led to spurious negative effects on low birth weight.

**Table 4**
Placebo cut-off tests.

| | Regression discontinuity | | | | | Control mean |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| April 2007 | −6.835 | −0.375 | −8.791 | −6.941 | −2.762 | 3323.678 |
| | (5.713) | (4.047) | (6.655) | (4.961) | (7.359) | |
| Bandwidth (days) | 61.20 | 133.11 | 37.74 | 73.03 | 38.52 | |
| *N* | 222,739 | 488,344 | 133,977 | 266,623 | 137,760 | 674,820 |
| April 2008 | 3.101 | −4.443 | −0.587 | 2.203 | 1.248 | 3329.084 |
| | (5.548) | (3.947) | (6.456) | (4.831) | (6.501) | |
| Bandwidth (days) | 61.20 | 133.11 | 37.74 | 73.03 | 46.19 | |
| *N* | 234,931 | 510,818 | 142,975 | 280,238 | 178,122 | 1,374,884 |
| April 2010 | −2.988 | −5.217 | 2.151 | 0.799 | 5.663 | 3338.664 |
| | (5.509) | (3.908) | (6.426) | (4.771) | (7.292) | |
| Bandwidth (days) | 61.20 | 133.11 | 37.74 | 73.03 | 37.38 | |
| *N* | 234,455 | 514,759 | 142,377 | 280,846 | 142,377 | 711,524 |
| April 2012 | 3.364 | 2.114 | 4.022 | 2.471 | 4.208 | 3333.814 |
| | (5.383) | (3.840) | (6.277) | (4.666) | (6.182) | |
| Bandwidth (days) | 61.20 | 133.11 | 37.74 | 73.03 | 48.76 | |
| *N* | 240,088 | 525,013 | 145,766 | 287,814 | 189,163 | 2,786,407 |
| CER-optimal | | | | | X | |
| Controls | | | | X | | |
| Kernel | Triangular | Triangular | Uniform | Triangular | Triangular | |

*Note*: The table reports the coefficient $\beta_1$ from Eq. (1) for a series of placebo cut-offs when the HPG was not introduced: 6 April 2007, 6 April 2008, 6 April 2010 and 6 April 2012. Birth weight (grams) is the dependent variable. Column 1 is a local linear RD with a triangular kernel and the CER-optimal bandwidth from Column 1 of Table 2. Column 2 is a local linear RD with a triangular kernel and the MSE-optimal bandwidth from Column 2 of Table 2. Column 3 is a local linear RD with a uniform kernel and the CER-optimal bandwidth from Column 3 of Table 2. Column 4 is a local linear RD with a triangular kernel, a vector of controls (maternal age (years), multiple birth status, income deprivation score, and the sex of the baby) and the CER-optimal bandwidth from Column 4 of Table 2. Column 5 is a local linear RD with a triangular kernel for the bandwidth which is CER-optimal for the specific placebo cut-off. All standard errors are robust bias-corrected and are listed in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Data from administrative birth registrations for England and Wales.

## 5. Discussion

This paper exploits a quasi-experiment from England and Wales to show that paying universal child benefits from pregnancy improves infant health. I find that the Health in Pregnancy Grant (2009–2011) led to statistically significant increases in birth weight and reductions in prematurity. These effects are robust across a variety of specifications. This contrasts sharply with regression discontinuity results for placebo cut-offs and baseline covariates, which are almost exclusively null. Both institutional context and a formal McCrary test suggest that manipulation of the running variable was unlikely.

My preferred specification indicates that the HPG increased birth weight by 12 grams on average. This represents 2 percent of a standard deviation, which would typically be considered an extremely small effect size. However, when taking into account the small size of the grant, the scaled effects of the HPG on birth weight are surprisingly large when benchmarked against estimates from the existing literature. The Hoynes et al. (2015) estimates are at the upper end of those from the literature.[20] Using quasi-experimental variation from the Earned Income Tax Credit (EITC) in the US, Hoynes et al. (2015) find that an increase in EITC windfall income of 1000 USD (2009 prices) led to an increase in mean birth weight of 18 grams for their highest-impact sample (black, single, low-educated mothers). The highest-impact sample in my study (younger mothers aged 21 or under)[21] saw an increase in mean birth weight of 35 grams according to my preferred specification. Scaling these birth weight effects by the size of the cash transfer, my results imply birth weight gains from windfall transfers that are approximately seven times as large as those of Hoynes et al. (2015).[22]

Another way to interpret the size of these effects is to convert them into a money metric by considering potential effects on earnings. Black et al. (2007) leverage exogenous variation from twins with administrative data from Norway to show that a 10

[20] Chung et al. (2016) find that birth weight increases by 17.7 grams from a cash transfer of 2256 USD (in 2009 prices), which implies lower birth weight effects than Hoynes et al. (2015). Estimates from Mocan et al. (2015) – that mean birth weight increases by 100 grams if mothers' earnings double – are also lower. González and Trommlerová (2022) meanwhile find that a 2500 EUR universal child benefit paid to mothers from birth reduced very low birth weight (<1500 g) among subsequent children by 0.7 percentage points (83 percent), but find no statistically significant effects on mean birth weight or low birth weight (<2500 g).

[21] This group is the bottom decile of the maternal age distribution; effects are shown in Supplementary Figure S9.

[22] The HPG was a lump-sum transfer of 190 GBP (2009 prices), which is equivalent to 270.66 USD at purchasing power parity (OECD, 2021). Estimates from Hoynes et al. (2015) are for a 1000 USD increase in 2009 prices. Scaling the HPG estimate for my highest-impact sample therefore implies that a 1000 USD windfall would increase mean birth weight by 130 grams, which is approximately seven times the size of Hoynes et al. (2015)'s highest estimate. Even when adjusting for minor measurement error in the running variable, my results suggest an effect of 16 grams for mothers aged 21 and under, which when scaled is approximately three times the size of Hoynes et al. (2015)'s estimates.

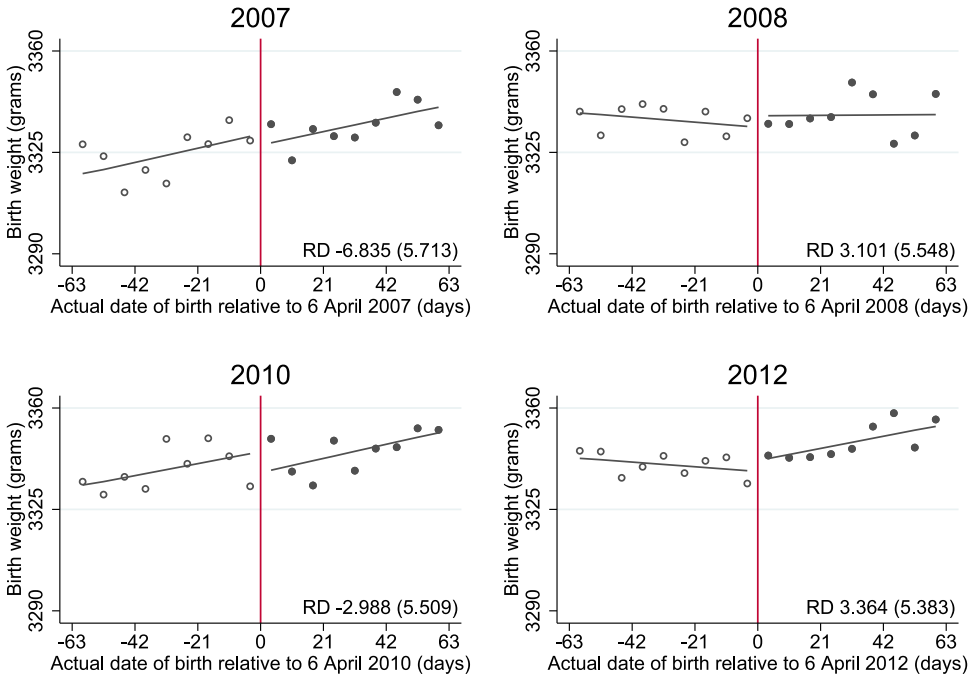


**Fig. 4.** Placebo cut-off tests.

*Note*: The figure shows results from placebo cut-off tests on 6 April in four years when the policy was not introduced: 2007, 2008, 2010 and 2012. Each panel plots mean birth weight by actual week of birth bin for the CER-optimal bandwidth from Table 2 (61.2 days). A linear spline is fitted to the raw data either side of the cut-off. The red vertical line indicates the placebo cut-off on 6 April for each year, with day 0 denoting the first day of placebo eligibility. The running variable is actual date of birth. Each panel also reports the coefficient $\beta_1$ from Eq. (1), with associated robust-bias-corrected standard errors in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Data from administrative birth registrations for England and Wales.

percent increase in mean birth weight leads to a one percent increase in annual earnings in adulthood. I find that the HPG increased birth weight by 0.35 percent on average, which according to the elasticity from Black et al. (2007) would imply an increase in annual earnings of 0.035 percent. Assuming average earnings growth and a social discounting rate equal to the average UK interest rate over the period 2009–2022, this suggests an increase of median lifetime annual adult earnings of approximately 545 GBP per adult due to the HPG. While this is small within a lifetime context, it is nearly three times as large as the per-capita cost of the policy.[23]

A further contribution of this paper is to offer evidence on possible causal mechanisms, notably the conditionality and the cash itself. The policy implications of these two mechanisms are very different. At one extreme, if the infant health effects were wholly explained by an increase in antenatal attendance due to the conditionality of the grant, then it could be cheaper and more efficient to mandate antenatal attendance or encourage it through other means. At the other extreme, if there were no changes in antenatal attendance then it would suggest that only the cash matters. Unconditional cash transfers could then be just as effective, and administratively simpler to implement.

While I find some suggestive minor effects on earlier antenatal health engagement, these are very similar to placebo effects in other years. Even taking the highest estimates at face value, a back-of-the-envelope calculation indicates that they are too small to explain the magnitude of the birth weight effect. In a similar policy setup involving a cash incentive for antenatal care, Cygan-Rehm and Karbownik (2022) find that a 10 percentage point increase in the antenatal care during the first trimester increases birth weight by 0.14 percent. At best, my results indicate a one percentage-point increase in antenatal care usage during the first trimester (see Supplementary Table S10). Applying the effect sizes from Cygan-Rehm and Karbownik (2022), my observed increase in antenatal usage would imply an increase in birth weight of 0.014 percent. Instead, I observe an increase in birth weight of 0.35 percent. It seems reasonable to conclude that the primary mechanism for the infant health effects was the cash itself, rather than the conditionality.

[23] These calculations are based on median gross annual earnings in 2009 in the UK (21,310 GBP) (Office for National Statistics, 2022). I assume that an adult works from the age of 18 to retirement (66 years in the UK). I inflate median annual earnings using the formula $Earnings_t = 21,310 * (1+i)^t$ where $i$ is the mean annual gross earnings growth from 2009 to 2022 (2.1 percent) and $t$ is the age of the individual. I discount the future by calculating the present value of earnings as equal to $Earnings_t/(1+r)^t$, where $r$ is the mean Bank of England interest rate from 2009 to 2022 (1.1 percent) (Bank of England, 2022) and $t$ is the age of the individual. Assuming a more conservative discount rate of 3.5 percent (the highest interest rate over the period 2009–2022, which was in December 2022), the HPG would be expected to increase lifetime median earnings by 208 GBP; assuming a more relaxed discounting rate of 0.1 percent (the lowest interest rate over the period 2009–2022, which was in March 2020), one would expect an increase of 861 GBP.

How could the cash itself have had such an effect? There are three main possibilities: subsidizing better nutrition; reducing the likelihood of smoking; and reducing prenatal stress. The medical literature generally indicates that nutrition affects intrauterine growth but does not affect gestational length (Amarante et al., 2016). In Section 4.1.2, I find that birth weight effects are driven by longer gestation, rather than faster intrauterine growth. It is therefore unlikely that the effects are explained by improved nutrition during pregnancy.

In Supplementary Figure S17, I examine published government data on the proportion of women who report to be smoking at the time of delivery by quarter of birth. If the HPG had reduced smoking, one would expect to see a sharper decline in population-level smoking rates among pregnant women during the policy's implementation (between 2009 Q2 and 2011 Q1 inclusive) because the policy was universal. Supplementary Figure S17 shows no evidence of such a decline. This makes it extremely unlikely that the infant health effects were due to reductions in smoking.

By a process of elimination, the final mechanism is stress. My analysis of this as a mechanism remains speculative, since I do not have data to verify stress levels. However, the stress hypothesis is consistent with my results in two respects.

First, research suggests that stress predominantly affects birth weight via gestational length and prematurity, rather than intrauterine growth (Torche, 2011; Kramer et al., 2000). In Section 4.1.2, I show that the birth weight effects of the HPG are explained by gestational length and not by intrauterine growth. I also document a reduction in prematurity of 9–11 percent. Maternal stress during the third trimester contributes to prematurity via the release of maternal glucocorticoids, which stimulate excess production of the corticotrophin releasing hormone (CRH) and can cause early delivery (Foureaux Koppensteiner and Manacorda, 2016; Torche, 2011). Since the HPG was paid during the third trimester, it is possible that it mitigated against stress-induced prematurity of this kind.

Second, if stress is the main mechanism then one would expect the effects of the grant to be larger for groups at greater risk of prenatal stress. This is precisely what I observe: babies born to younger mothers (24 and under) in deprived areas were the biggest winners from the HPG. Poverty and financial instability are associated with "an accumulation of multiple chronic stressors" (Kramer et al., 2000, p.204) and higher cortisol levels, which are likely to affect women living in deprived areas and younger women (Aizer et al., 2016; Cohen et al., 2006). The u-shaped effects by maternal age are also consistent with the stress hypothesis in that older mothers face an elevated risk of prenatal stress, anxiety and depression (García-Blanco et al., 2017).[24]

The arrival of a baby is expensive, particularly for first-time mothers. It typically involves large fixed costs during the third trimester, such as buying a pushchair, a cot or other baby equipment. One possibility is that the lump sum of the HPG subsidized these costs and alleviated financial stress among low-income younger mothers.[25] While anecdotal and subject to selection bias, some users on the public online forum *Mumsnet* reported using the HPG to reduce stress, help with financial difficulties and promote wellbeing. One woman reported using the 190 GBP to buy a pushchair; another said it would reduce her overdraft; another reported investing in a 10-week course of aqua-natal swimming and yoga classes (Mumsnet, 2009). A final possibility, which remains speculative due to data limitations, is that the HPG enabled women to take earlier unpaid maternity leave prior to the birth of the baby, thereby enabling them to get more rest during the period when babies gain weight very rapidly.[26]

## 6. Conclusion

Universal child benefits from birth are a popular policy tool to improve child health. The importance of infant health to child health, adult health and socio-economic outcomes is clear. Yet little research has considered what the potential infant health gains are from starting universal child benefits in pregnancy. This paper provides new causal evidence that starting child benefits in pregnancy leads to infant health benefits.

I do so by exploiting a quasi-experiment in the UK from 2009, when all pregnant mothers were eligible for a Health in Pregnancy Grant (HPG), a lump sum transfer equivalent to three months of child benefit (190 GBP) during the third trimester of pregnancy. Using administrative birth registry and hospital data, I take advantage of an arbitrary eligibility rule for the HPG based on expected date of birth. I implement a regression discontinuity design in the date of birth of the baby, which enables me to overcome selection bias and to identify the causal effects of the policy.

I find that starting universal child benefits in pregnancy increases birth weight and reduces prematurity. Younger mothers (aged 24 and under), particularly the very youngest and the most deprived, benefit the most. While the size of the effect is small in absolute terms, when scaling these effects by the size of the transfer, this implies birth weight gains from windfall income of seven times the magnitude of those found elsewhere.

[24] Advanced maternal age involves higher risks of adverse health outcomes during conception, pregnancy and birth, including reduced fertility, miscarriage, stillbirth and chromosomal abnormalities (García-Blanco et al., 2017). Older women are also more likely to have additional caring responsibilities and greater professional commitments, which increase stress during pregnancy (García-Blanco et al., 2017).

[25] The heterogeneous treatment effects are unlikely to be due to differential take-up rates of the grant: take-up overall was extremely high, at 93 percent, and existing evidence suggests that there were no significant differences in antenatal incentivisation. Leyland et al. (2017) find that deprived mothers attended antenatal care 0.5 weeks earlier and non-deprived mothers attended 0.2 weeks earlier, these differences are not statistically significant. They also find no differences by single parent status. An alternative possibility is that the heterogeneity reflects the elevated risk of lower birth weight among these groups.

[26] Taking time off through unpaid maternity leave has been associated with increases in birth weight, but less so for poor, single and low-educated women due to the associated reduction in income (Rossin, 2011). The HPG may have enabled poorer women to circumvent such financial repercussions, thereby reducing stress. Unfortunately, there is no available data on maternity leave to test this hypothesis, so it remains speculative.

These effects do not appear to be explained by increases in antenatal care via the conditionality of the grant. Instead the cash itself seems to have been important. Suggestive evidence indicates that nutrition and smoking are unlikely channels: stress reduction, mediated through longer gestation, is a more plausible candidate.

These results have striking policy implications. First, they demonstrate that there are infant health gains to be made from starting universal child benefits in pregnancy. At the time of writing, this is policy-relevant given the recent arrival of a new universal child benefit from pregnancy in Italy (Vidotto and Lucangeli, 2022), and recent calls to extend the US Child Tax Credit to pregnant mothers (Campaign, 2022). Second, since conditionality played, at best, a minor role in the effects of the grant, it seems reasonable to conclude that infant health effects could be found for unconditional cash transfers. Finally, my findings suggest that family policies to improve child health should place greater attention on younger and first-time mothers, most of whom are excluded from existing child-related benefits because they only start from birth.

Many developed countries pay universal child benefits from birth as part of a wider "cradle to grave" package of financial support that supports health across the life course. Fewer have experimented with starting this financial support *in utero*. This paper demonstrates that doing so may provide hitherto unrealized infant health benefits.

## CRediT authorship contribution statement

**Mary Reader:** Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Roles/Writing – original draft, Writing – review & editing.

## Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.jhealeco.2023.102751.

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