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Intimate partner violence and children's health outcomes

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ABSTRACT

A growing body of literature has established that childhood health is a crucial determinant of human capital formation. Shocks experienced in utero and during early life may have far-reaching consequences that extend well into adulthood. Nevertheless, there is relatively little evidence regarding the effects of parental behaviour on child health. This paper contributes to the literature by examining the impact of intimate partner violence (IPV) on the child's health production function. Using data from the UK's Millennium Cohort Study and leveraging information on both child health and IPV, our analysis reveals that exposure to IPV is negatively associated to child's health. Children witnessing IPV in their household see their probability of being in excellent health reduced by 7 percentage points. Our results also suggest that children exposed to IPV are subject to increased morbidity, manifested in elevated risks of hearing and respiratory problems, as well as long-term health conditions and are less likely to get fully immunised.

1. Introduction

According to the 2019/2020 release of the Crime Survey for England and Wales (CSEW) 7.3% of women reported experiencing domestic abuse in the previous year and among those aged 16–59, 27.6% had experienced some form of domestic abuse since they were 16.[1] 15% of police-reported crime was related to domestic abuse.[2] The estimated costs associated with domestic abuse, including expenses borne by the criminal justice system, health services, social care, and housing, have been estimated to be about £16 billion (Walby, 2009).

It has been established that intimate partner violence (IPV) has detrimental effects on victims' employment outcomes, mental health status, and sense of self-worth and integrity (Chapman & Monk, 2015). IPV has been associated with mental health problems in parents, such as depression and anxiety (Carlson et al., 2003; Mertin & Mohr, 2001). Among women, IPV increases the likelihood of developing severe depressive, post-traumatic, and substance abuse disorders (Ehrensaft et al., 2006). Violence is frequently perceived as a means of compelling victims to conform to the perpetrator's beliefs or attitudes, becoming a source of gratification for the perpetrator or a means of extorting financial gain from the victim (Bloch & Rao, 2002; Tauchen et al., 1991).

This paper examines the potential spillover effects of intimate partner violence (IPV) on children's health. The paediatric literature suggests that children exposed to hostile environments have a cumulative disadvantage (Culross, 1999), which may have negative effects on their overall well-being, as well as their interpersonal and socio-emotional development (Ehrensaft et al., 2003; Kitzmann, 2012). Children who witness IPV are prone to engaging in antisocial behaviours, such as delinquency and running away (Dubowitz & King, 1995; Wolfe & Korsch, 1994), and may also have difficulties regulating their moods, emotions, and behaviours, displaying heightened aggression and hostile reactivity (Ehrensaft & Cohen, 2012). Research also indicates that children from households experiencing IPV may be at greater risk of developing poor self-esteem and are more likely to engage in substance

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[1] https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/articles/domesticabusevictimcharacteristicsenglandandwales/yearendingmarch2020. Domestic abuse in the CSEW survey includes: partner/ex-partner abuse (non-sexual), family abuse (non-sexual) and sexual assault or stalking carried out by a current or former partner or another family member.Domestic abuse in the CSEW survey includes: partner/ex-partner abuse (non-sexual), family abuse (non-sexual) and sexual assault or stalking carried out by a current or former partner or another family member.

[2] https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/datasets/crimeinenglandandwalesappendixtables

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abuse later in life (Holtrop et al., 2004). Furthermore, aggressive behaviour during infancy may lead to rejection by school peers (Dodge et al., 2003). A major negative consequence identified in psychiatric literature is the potential for children to internalize the use of violence as a normal means of achieving their goals, leading to future use of physical or psychological violence against their partners (Magdol et al., 1998).

Overall, it is difficult to determine a unique mechanism for how IPV may affect a child's health. There may be different biological consequences of living under stressful and fearful environments. Emerging evidence suggests that early life stress may cause changes in multiple neurochemical systems (Kaufman et al., 2000) and promote several alterations in the serotonergic system, which reduces hippocampal volume (Kaufman et al., 2004). In addition, exposure to early life stressors, such as domestic violence, has been linked to depression, subsequent poor quality of parenting, higher levels of risk-taking, and antisocial behaviours in adults (Holtrop et al., 2004).

Empirical research related to our analysis shows that IPV has a negative effect on the birth weight of children born to mothers who were assaulted while pregnant (Aizer, 2011). Specifically, pregnant women who were hospitalised due to violent assault gave birth to babies weighing an average of 163 grams less. Evidence suggests that violence during pregnancy leads to lower birth outcomes, including very low birth weight, preterm birth and lower Apgar scores (Currie et al., 2022). Similar negative effects exist when examining the impact of IPV exposure on neonatal, infant and under-five mortality in developing countries (Rawlings & Siddique, 2020). While most research has focused on the impact of IPV on birth outcomes, some evidence exists on the effects of IPV on children's development later in life (Kitzmann, 2012). Children witnessing IPV also have worsened cognitive, social and socio-emotional skills (Anderberg & Moroni, 2020). The effect of exposure to IPV goes beyond that of negatively affecting the child's own cognitive development but also exhibits negative externalities on their peers' academic performance (Carrell & Hoekstra, 2010).

Despite a growing literature on the impact of investments and shocks to child health (Almond et al., 2018; Currie, 2020), the effect of parental behaviour on children's health production function has not been widely considered, especially in the context of IPV exposure. Our paper aims to estimate the association between growing up in a violent domestic environment and child's health. We contribute to the existing literature on domestic violence by quantifying the negative spillover effect of IPV on children's health production function. To the best of our knowledge, our work is the first to examine the association between exposure to IPV and children's health in early childhood.

We employ data from the Millennium Cohort Study (MCS) to investigate the effect on a child's health of growing up in a household with IPV. In our study, we examine the impact of domestic violence on the probability of parental reporting that the child is in excellent health. We measured IPV using questions specifically designed to capture the use of force by the partner on the child's biological mother. We use regression models to examine the association between IPV and the child's health production function. To address the possibility that IPV households are not comparable to non-violent households, we estimate propensity score matching models. Additionally, we investigate the impact of IPV on a number of condition-specific measures of children's health and on the probability of receiving the recommended immunisations during their first year of life.

The remainder of the paper is structured as follows. The next section introduces the MCS data and presents the variables of interest. Section III outlines the empirical strategy we use to examine the relationship between a child's health and IPV. Section IV presents the results of the impact of IPV on a child's health, additional models and several robustness checks. The final section concludes.

2. Data: The Millennium Cohort Study

The MCS survey tracks the development of approximately 19,000 children born in the United Kingdom during 2000–2001. The initial data collection was conducted when the children were nine months old, and subsequent waves were collected at various ages. Although information on IPV is available in each wave, data related to health variables for children is only available from Wave 3 onwards. Hence, for our study, we utilise Waves 3, 4 and 5 of the MCS, which were administered when the children were aged 5, 7, and 11, respectively. Our sample is comprised of all children whose biological mother served as the primary respondent, representing roughly 80% of the total sample.

2.1. Domestic violence

To determine the occurrence of domestic violence, we utilise information pertaining to incidents of Intimate Partner Violence (IPV) experienced by the mother. Specifically, the survey questionnaire contains the following query: "*People often use force in a relationship - grabbing, pushing, shaking, hitting, kicking etc. Has your husband/partner ever used force on you for any reason?*" The respondent may select one of three options: 1 (Yes), 2 (No) and 3 (Don't want to answer).[3] Although domestic abuse is experienced by males and females, the paper focuses on exposure of mothers to IPV exerted by their partner, which is the most prevalent form of IPV observed.[4] This question was asked in each wave, and we are able to exploit the variation in responses across the three waves used for the analysis. We observe respondents consistently reporting IPV in each wave but also individuals changing their responses to the IPV question between wave t and wave $t+1$.

Based on this question, we construct an indicator variable IPV1 that takes the value 1 if the biological mother answers "Yes," 0 if the answer is "No," and treat "Don't want to answer" responses as missing values. It should be noted that self-reported data has a limitation of potential under-reporting, which may result in a conservative estimate of the number of women experiencing IPV. The Crime Survey for England and Wales (CSEW) for 2013/2014 identified feeling embarrassed (22.25%) and considering the matter personal (12.92%) as the primary reasons for under-reporting of sexual assault cases. In order to mitigate the potential issue of under-reporting, we introduce a second IPV variable, IPV2, which takes a value of 1 if the biological mother responds "Yes" or "Don't want to answer" to the IPV question, and 0 if she responds "No". According to the first definition of IPV1, the prevalence of IPV in our dataset is 3.93%, 3.55%, and 3.61% for waves 3, 4, and 5, respectively. However, when using the definition of IPV2, these figures increase to 6.76%, 6.16%, and 5.33% for the same waves. These frequencies of IPV obtained using the second definition are in line with the statistics on domestic abuse prevalence reported in the CSEW in 2022.[5]

There are several considerations regarding our primary variable of interest IPV. Firstly, the information available in the MCS allows creating a definition of IPV based on the mother's exposure to violence. However, this definition does not specify severity or frequency and whether the child is simply a witness to the violence or whether they also experience direct violence in addition to their mother's exposure to IPV. Children living in a household with IPV are more likely to be abused (Skafida et al., 2022) and may be used as a tactic to exert IPV on the mother (Clements et al., 2021). It has been estimated that approximately

[3] The parental questionnaire was administered by an interviewer, although more personal or sensitive questions were self-completed by one parent at a time to lessen the misreporting effect. For further details, please refer to the user guides of each sweep.

[4] In terms of domestic abuse-related prosecutions, a study found that the large majority of defendants were recorded as male (92%), and the majority of the victims were recorded as female (77%, compared with 16% who were male). See https://www.womensaid.org.uk/information-support/what-is-domestic-abuse/domestic-abuse-is-a-gendered-crime/.

[5] https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/bulletins/domesticabuseinenglandandwales.

62% of children residing in violent households are subject to direct abuse by the parent exerting IPV (CAADA, 2014). The Domestic Abuse Act 2021,[6] which has been recently passed by the UK government, recognises children who are exposed to domestic violence as victims of domestic abuse, irrespective of whether or not they are directly abused. In this study, we adopt this definition and consider children who witness parental violence as victims of domestic abuse. Secondly, there may be indirect consequences of IPV on the child's health production function if the mother's ability to provide care is disrupted due to the abuse. Given that we cannot distinguish between direct and indirect effects, we estimate a reduced form model that captures the association of witnessing IPV on a child's health.

2.2. Child health information

From Wave 3 onward, the MCS elicits information regarding children's general health from their parents as well as whether the child suffers from specific health conditions. The parents are asked to rate their child's general health as Poor (5), Fair (4), Good (3), Very Good (2), and Excellent (1). Fig. 1 summarises the frequency distribution of parent-reported child overall health in Waves 3 to 5 for households with no IPV and households with IPV using the IPV1 definition. If we use the IPV2 definition, a similar distribution emerges. For the purpose of our study, we define a binary variable describing the child being in good health that equals 1 if the parent selects the category Excellent and 0 otherwise.

In this study, we regard the general health of a child reported by their parents as a reliable indicator of the child's health status (Kuehnle, 2014). Case et al. (2002) demonstrated a strong correlation between parental-reported general health and physician assessments of the child's objective health. It is possible that a parent's own health condition could impact their ability to accurately report on their child's health, thus introducing bias into the response. Nonetheless, there exists consistent evidence of the capacity of parents to provide accurate information about their child's health. McCormick et al. (1989) found that mothers suffering from depression could accurately distinguish between their own reported health and that of their child. Furthermore, Pulsifer et al. (1994) observed that maternal estimates of their child's developmental age align with objective measures. Research also indicates that the use of standardized surveys to systematically collect information on child health, as is done in the MCS, produces a high correlation between survey information and factual outcomes (Glascoe et al., 1991).

From Wave 3 onwards, the MCS also includes information on whether the child suffers from a number of health conditions. We exploit this information to extend the analysis and identify any association of IPV on measures other than general health. In particular, we use variables that capture whether the child suffers from any of the following health conditions: hearing problems, eyesight problems, respiratory problems (which include wheezing and asthma), eczema, hay fever or any long-standing illness (LSI). Some of these conditions cover a large array of illnesses whereas others, such as eczema or hay fever, are more specific.[7] Fig. 2 shows the frequency of these conditions across waves according to exposure to IPV. In addition to these health conditions, we also explore the impact that IPV could have on the probability that children are given the immunisations recommended during the first year of life. In Wave 2 of the MCS (when children were aged 3), the questionnaire included a set of questions relating to a number of vaccinations offered to infants under the age of 1 in the UK as part of the NHS vaccinations programme. We check whether IPV exposure affects the probability of having the full course of immunisations for each of the following vaccines: polio, tetanus, diphtheria, whooping, Hib and meningitis. Table A1 in the Appendix shows the proportion of children receiving immunisations.

2.3. Control variables

Our study adopts the approach of Currie (2009) by considering various health-related, environmental, and socio-economic factors, whether transmitted intergenerationally or not, that could impact a child's health. Currie (2009)'s review of the literature on the connection between child health, income, and parental education provides insights into the mechanisms through which the socio-economic environment may influence a child's health production function. In our model specification, we include covariates such as the child's age, gender and BMI. To determine obesity and overweight, we use the methodology developed by Saxena et al. (2004) to generate gender-age-specific BMI thresholds, resulting in two binary variables. Descriptive statistics for the parental-reported health variables of interest and other relevant controls are provided in Table A1 in the Appendix.

The model further adjusts for various maternal characteristics, including age, education, ethnicity, job status, marital status, and health. Education plays a vital role in determining cognitive ability and is associated with healthy behaviours (Cutler & Lleras-Muney, 2010). Educated parents tend to engage their children in more stimulating discussions and have a better network of contacts to turn to in the event of health problems (if parents do not personally know a doctor, it is more probable that someone in their social network will). We also incorporate the mother's ethnicity as it has been linked to a child's health outcomes. For instance, Dearden et al. (2006) utilise the MCS data to demonstrate that Asian and Black babies have a 5% and 6% higher likelihood, respectively, of being born with low birth weight than white babies, potentially impacting their health in the long run. Additionally, we control for paternal variables such as age, education, employment status, and health characteristics.

We include several household structure and contextual variables, namely the number of individuals residing in the household, whether the child resides in a council or housing association dwelling, and household income. Previous research has indicated a positive correlation between parental income and child health (Currie, 2009; Kuehnle, 2014; Violato et al., 2009). Therefore, we incorporate combined parental income to disentangle the impact of income from the influence of IPV on a child's health.[7]

Evidence has also established a link between neighbourhood factors and health outcomes. For example, Bilger and Carrieri (2013) demonstrated a causal effect of neighbourhoods on self-assessed health, chronic conditions, and limitations to daily activities in Italy, and Jacob et al. (2013) found that relocating to less distressed neighbourhoods had a positive effect on child mortality rates in Chicago. Given that council houses are likely to be situated in areas characterised by higher levels of deprivation, we also adjust for deprivation to account for contextual factors that may adversely affect a child's health beyond the immediate effects of parental characteristics (Atkinson & Kintrea, 2001).

In the MCS, income is defined as the combined annual income in a household from all sources after deductions, in thresholds levels. We take the midpoint of each reported interval and use the annual average consumer price index provided by the Office of National Statistics (ONS) to convert it into real income with the base year 2005 (Wave 3).

3. Empirical strategy

Our aim is to estimate the relationship between child health and IPV using a reduced form of the child's health production function. For the measure of general health (and the other health variables considered

[6] https://www.gov.uk/government/publications/domestic-abuse-bill-2020-factsheets/domestic-abuse-bill-2020-overarching-factsheet.

[7] Maternal stress during pregnancy has been linked to eczema in children. We do not explore the impact of IPV during pregnancy, however if the mother experienced IPV during pregnancy and such a stressor contributed to the development of conditions like eczema, our estimates could be picking up some of this association.

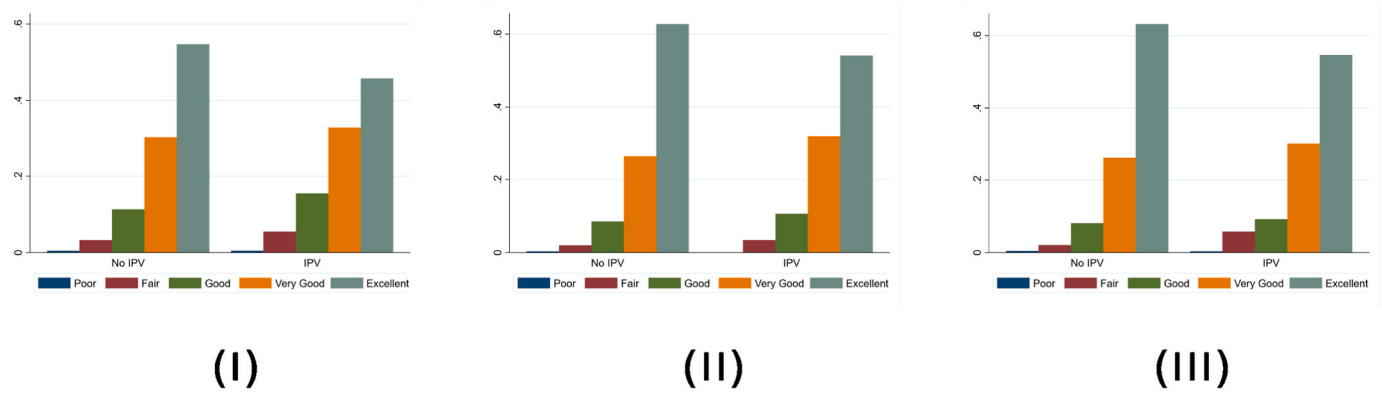

Fig. 1. Parental-reported child's health in Wave 3 (I), Wave 4 (II) and Wave 5 (III) for Households without and with IPV.

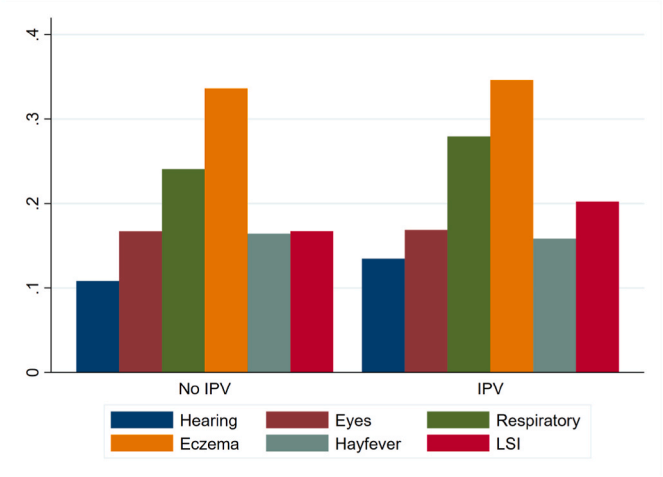

Fig. 2. Children's health conditions - Waves 3-5.

later on), we estimate the following equation:

$$H_{it} = \beta_1 IPV_{it} + X_{it}\beta_2 + \epsilon_{it}, \tag{1}$$

where H_{it} is the parental-reported health of child i in wave t, *IPV* is an indicator variable that captures child i exposure to IPV at wave t. X_{it} is a set of child characteristics, parental and household characteristics. The term ϵ_{it} is a normally distributed error term. We estimate equation (1) by means of a linear probability model.

3.1. Test of coefficient stability

The presence of unobserved factors that simultaneously affect IPV and child health could produce biased estimates. In order to assess the validity of our identification strategy, we check whether the IPV coefficient is stable to the addition of other observable factors by inspecting the direction and magnitude of the bias caused by omitted variables. The stability of coefficients to the introduction of additional controls has been used in empirical research to argue that the potential bias due to omitted variables is minimal. Altonji et al. (2005) suggested a test of coefficient stability for linear models based on the assumption that the relationship between the variable of interest and the unobservables can be recovered from the relationship between the main variable and observables. Oster (2019) formalised the test linking coefficient stability to the observed movements in the R-squared and shows that the true (bias-adjusted) coefficient of the IPV coefficient β_1^* is as follows:

$$\beta_1^* \approx \widetilde{\beta}_1 - \delta\left[\beta_1 - \widetilde{\beta}_1\right]\frac{R_{max} - \widetilde{R}}{\widetilde{R} - R} \tag{2}$$

where $\widetilde{\beta}_1$ is the IPV coefficient estimated including all the observed covariates; β_1 is the uncontrolled regression coefficient resulting from regressing the dependent variable on the IPV indicator alone; $\widetilde{R}$ is the R-squared of the controlled regression; and R is the R-squared of the uncontrolled regression. This method provides a bounding set $[\widetilde{\beta}_1, \beta_1^*]$ for the bias-adjusted value of the IPV coefficient. The bias-adjusted coefficient depends on two unknowns: δ, the proportionality coefficient reflecting the contribution of the unobservables relative to the observables in explaining IPV, and R_{max}, the maximum R^2 that could be achieved when controlling for all observables and unobservables. Oster (2019) proposes a value of $\delta = 1$ and $R_{max} = 1.3\widetilde{R}$ as reasonable upper bounds. We will quantify the bias introduced by omitting variables that affect both child's health and IPV and provide the bounding set for the true value of the main coefficient of interest β_1.

3.2. Propensity score matching

The test of coefficient stability is helpful to understand the direction and magnitude of the bias and to estimate an approximation to the bias-adjusted effect of IPV. Oster (2019) argues that it should not be used as an estimator of the treatment effect given it relies on a set of assumptions required to compute this approximation. While the results of the base case are stable to the inclusion of variables, these may not be considered unbiased estimates of IPV on the child's health. In order to further explore the validity of our basecase estimates, we use propensity score matching estimators, an approach widely used to evaluate average treatment effects on the treated (Rosebaum & Rubin, 1983). These estimators rely on matching control units to treated ones based on their conditional probability of receiving the treatment given some covariates, i.e., the distribution of covariates for treated and control groups is similar. Matching based on the conditional probability of assignment to treatment simplifies the matching process as it relies on one indicator instead of a multiplicity of them (Abadie & Imbens, 2016).

We make use of propensity score matching to estimate the average treatment effect of IPV on children's health. Thus, we match each child in the treatment group, i.e., those exposed to IPV, to a child in the control group based on the closeness of their propensity scores. To do so, we apply the nearest neighbour matching algorithm without replacement. The propensity score is estimated given the set of observables discussed in Section 2.3 (listed in Table A1 in the Appendix). These controls are assumed to be independent of treatment assignment. We check the sensitivity of the results to the choice of matching method (Caliendo & Kopeinig, 2008). Finally, we impose that the common support assumption holds, discarding those control group observations

with a propensity score is below (above) the treated's minimum (maximum) propensity.

4. Results

4.1. Child's health and IPV: Basecase results

Table 1 presents the results of the estimated coefficients obtained using a linear probability model, using the two definitions of IPV. For each definition, we first use the sample with all families and then restrict the sample to those families where both biological parents cohabit. By limiting the sample to households where both parents are present, we are able to assess whether the association varies according to family structure. Estimates are consistent across all specifications and show that IPV has a statistically significant and negative effect. Columns (1) and (3) present the estimates when using all household types, indicating that exposure to IPV reduces child health. Restricting the sample to those households where both biological parents are present renders similar estimates, as shown in Columns (2) and (4). Across all specifications, the similarity in the estimates for both samples indicates a negative association with IPV: the probability of having excellent health is 7 percentage points (pp) lower for children that witness IPV. Women exposed to IPV might be more prone to attrition and re-entry. To check for the robustness of the results to this possibility, we also estimate the effect of IPV on health using a balanced sample. Results are presented in Table A2 in the Appendix and are in line with the estimates in Table 1.

We base our IPV variable on the question that asks whether the "husband/partner ever used force". Thus, answers to this question could reflect a legacy of exposure, e.g., past abuse experienced by the mother and not necessarily witnessed by the child. Although we do not have information on the exact timing of the abuse, we can exploit differences in IPV reporting across waves in order to differentiate effects between those that report being continuously exposed to IPV and those that transitioned into IPV exposure in one of our study waves. Table A3 in the Appendix shows the results when excluding from the sample individuals consistently exposed to IPV across all waves (i.e., those that respond yes in all waves). This leaves in the sample those that transition into the IPV variable within the study period, allowing us to estimate the effect on the newly exposed to IPV. The estimates are similar to those in Table 1, only slightly smaller in magnitude. This suggests that there is an unambiguous negative effect of IPV on child's health irrespective of the onset of IPV. If mothers report IPV across all waves, the negative effect on child's health remains stable.

4.2. Coefficient stability

In this section, we present the results of using the method proposed by Oster (2019) to estimate the direction of the bias and quantify its magnitude. We define the bounding set for different values of the parameter δ and using the R_{max} suggested by Oster (2019), $R_{max} = \min[1.3\widetilde{R}, 1]$. Table 2 below shows the bounding sets for each of the IPV definitions and samples used, as in Table 1. The top panel in Table 2 shows the coefficients and R-squared for the regressions without controls, β_1 and R, and for the regressions with controls, $\widetilde{\beta}_1$ and $\widetilde{R}$. Panel B presents the bounding set $[\widetilde{\beta}_1, \beta_1^*]$ for varying values of δ. The bias-adjusted estimate of IPV is negative in all bounding sets presented, therefore indicating that the potential presence of omitted variable bias does not change the sign of the effect. The values of the lower bound $\widetilde{\beta}_1$ and upper bound β_1^* are relatively close. This is especially the case at lower values of δ, suggesting that unobservables are not significantly biasing the IPV parameter. To further assess the relevance of the unobservables, we also compute the value of δ required to give an effect for IPV equal to zero. The bottom of this panel shows that across all specifications, the values of are very large. This points to conclude that the selection on unobservables would have to be considerably larger with respect to observables, which Oster (2019) argues is unlikely to be the case in empirical applications.

4.3. Propensity score matching

Our base case estimates indicate children witnessing IPV are 7pp less likely to report excellent health. Following concerns this estimate may be biased due to omitted variables, we use the (Oster, 2019) test of coefficient stability to quantify the bias introduced by unobservables. The contribution of unobservables seems to be small relative to the contribution of the observables. In order to check the robustness of our empirical specification, we use propensity score matching methods. We

Table 1
Impact of IPV on Children's health.

Sample	IPV1		IPV2	
	(1)	(2)	(3)	(4)
	All	Both Parents	All	Both Parents
Excellent	−0.068***	−0.071***	−0.071***	−0.070***
	(0.017)	(0.019)	(0.014)	(0.015)
N	27213	23832	27864	24386
R2	0.050	0.049	0.052	0.051
Time Effects	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Parental controls	Yes	Yes	Yes	Yes
Household controls	Yes	Yes	Yes	Yes

Notes: The dependent variable takes value 1 if the parent responds excellent to the question on child's overall health. Standard errors in parentheses are robust to heteroskedasticity and clustered at child level. Models are estimated using the unbalanced sample. Specifications in columns (1) and (3) use the sample of children in all household types. Specifications in all other columns consider only households in which both biological parents cohabit. *Controls* include the set of variables for the child-related variables (age, sex), parental controls (age, educational level - degree or higher, race, marital status for both the main respondent and the partner) and household-related variables (number of people in the household and whether the family lives in a council house or housing association). Reference category for maternal ethnicity is white and for marital status is Other. Wave fixed effects are included. Significance Levels: $^{+}\ p < 0.10$, $^{**}\ p < 0.05$, $^{***}\ p < 0.01$.

Table 2
Test of coefficient stability.

Sample	IPV1		IPV2	
	(1)	(2)	(3)	(4)
	All	Both parents	All	Both parents
Panel A				
β_1	−0.083	−0.093	−0.103	−0.11
R	0.008	0.008	0.009	0.009
$\widetilde{\beta}_1$	−0.068	−0.071	−0.071	−0.07
$\widetilde{R}$	0.05	0.049	0.052	0.051
Panel B				
$\delta = 1$	[-0.068, −0.0626]	[-0.071, −0.0631]	[-0.071, −0.0594]	[-0.07, −0.0554]
$\delta = 0.5$	[-0.068, −0.0653]	[-0.071, −0.0671]	[-0.071, −0.0652]	[-0.07, −0.0627]
$\delta = 0.2$	[-0.068, −0.0665]	[-0.071, −0.0694]	[-0.071, −0.0687]	[-0.07, −0.0671]
$\delta = 0.1$	[-0.068, −0.0675]	[-0.071, −0.0702]	[-0.071, −0.0698]	[-0.07, −0.0685]
Panel C				
δ	9.774	8.741	6.134	5.319

Notes: $R_{max} = 1.3\widetilde{R}$, with $\widetilde{R} = 0.05$, the $R_{max} = 0.065$. The lower bound of the bounding set is $\widetilde{\beta}_1$ and the upper bound is the bias-adjusted treatment effect β_1^*.

match children that witness IPV with the control group of children not exposed to IPV. By using the propensity score method and matching exposed and non-exposed children based on the observables, any difference in children's health will arise through exposure to IPV. Table 3 shows the results of the propensity score matching using different algorithms. Panel A shows the average treatment effect on the treated (ATT) obtained using the nearest neighbouring matching algorithm without replacement. This method matches the treated child with that in the control group with the closest propensity score. The estimates in column (1) show that exposure to IPV reduces a child's health by 6.1pp, whereas the ATT is 7pp when using the sample where both parents cohabit, i.e., the effect is more pronounced when the perpetrator lives in the household. Panel B presents the ATT when using the nearest neighbouring matching method using the four closest neighbours to our treated unit to construct the counterfactual. The ATTs range between 6.2 and 7.6pp, depending on the sample and definition used, and are more in line with the basecase estimates. Panel C shows the ATTs when using radius matching, obtained by imposing a threshold of 0.001 as the maximum distance in propensity score between the IPV-exposed child and the control unit. The estimated ATTs are pretty aligned in magnitude across specifications at around 7pp. The last method presented in Panel D uses Kernel matching, which matches the IPV-exposed unit to a counterfactual constructed as the weighted average of all children in the control group. The ATTs show a similar pattern: the ATTs obtained using the other matching methods with estimated effects between 4.2 and 7.3pp reduction in health for IPV-exposed children. All estimated ATTs in Table 3 were obtained by imposing common support. Not imposing common support does not change the results, as the number of observations excluded is extremely low, with less than a handful of observations eliminated.

4.4. Extensions: alternative measures of health

Our analysis has focused mainly on measures of a child's general health as reported by the child's mother. We next explore the impact that IPV has on a range of health conditions: hearing problems, eyesight problems, respiratory problems (which include wheezing and asthma), eczema, hay fever or LSI as one can argue that the stress likely to be triggered by IPV exposure could trigger or worsen each of these conditions. Table 4 shows the results of the impact of IPV on these health-specific conditions. The coefficients are precisely estimated for the regressions on hearing and respiratory problems. The estimates show that IPV is associated with an increase between 2 and 2.7pp in the probability of suffering from hearing problems, whereas for the case of respiratory problems, the association indicates IPV increases the likelihood of suffering from these conditions by 3pp. Although we present results for a reduced number of condition-specific outcomes (those available in the MCS questionnaires), we also include in the table the results for the regression that examines the impact of IPV on the probability of having a long-standing illness (LSI). This is a broader definition of illness for which results in Table 4 show a consistent negative association between IPV and child's health. The results indicate that children living in a household with IPV are 3pp more likely to suffer from a long-term condition.

Overall, these results suggest that IPV is associated with an increased morbidity. However, only the coefficients of conditions that might be more susceptible to be triggered by exposure to IPV, such as hearing and respiratory problems, are significant compared to the coefficients associated to hay fever, for instance, which arises as an allergic reaction to external causes.

In addition to the health conditions examined above, we also explore the impact that IPV could have on the probability that children receive the immunisations recommended during the first year of life: polio,

Table 3
Propensity score matching.

	IPV1		IPV2	
	(1)	(2)	(3)	(4)
Sample	All	Both Parents	All	Both Parents
Matching algorithm				
Panel A: NN(1)				
ATT	−0.0612***	−0.0686***	−0.0745***	−0.0456***
Panel B: NN(4)				
ATT	−0.0765***	−0.0666***	−0.0652***	−0.0619***
Panel C: Radius Matching				
ATT	−0.0658***	−0.0692***	−0.0690***	−0.0683***
Panel D: Kernel Matching				
ATT	−0.0543***	−0.0721***	−0.0661***	−0.0428***
N	27,213	23,832	27,864	24,386

Notes: Panel A shows the nearest matching (NN) algorithm for the nearest neighbour with no re-placement. Panel B shows the results of using NN matching four control observation per each treated observation. Panel C shows radius matching based on maximum distance of 0.001 in the propensity score between treated and untreated observations. Panel D use the Kernel matching method using the normal. Results are provided for matched observations with common support. Matching variables use include child, mother and household characteristics, plus an indicator for survey wave in all specifications. Specification for the subsample of both parents cohabiting also include paternal controls. See Notes in Table 1 for the list of matching variables included. Significance levels: $^{+}p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 4
Impact of IPV on Children's health.

	IPV1		IPV2	
	(1)	(2)	(3)	(4)
	All	Both Parents	All	Both Parents
Hearing	0.027**	0.024*	0.019*	0.015
	(0.013)	(0.013)	(0.010)	(0.010)
N	27071	23694	27719	24245
R2	0.009	0.009	0.008	0.008
Eye	−0.002	−0.002	0.005	0.006
	(0.014)	(0.015)	(0.011)	(0.012)
N	27082	23706	27730	24257
R2	0.022	0.021	0.022	0.021
Respiratory	0.029*	0.031*	0.031**	0.030**
	(0.016)	(0.017)	(0.013)	(0.014)
N	27214	23832	27865	24386
R2	0.033	0.034	0.034	0.034
Eczema	0.017	0.016	0.009	0.010
	(0.018)	(0.020)	(0.014)	(0.015)
N	27203	23821	27854	24375
R2	0.011	0.012	0.011	0.012
Hayfever	−0.001	0.004	−0.003	−0.002
	(0.013)	(0.014)	(0.010)	(0.011)
N	27132	23757	27780	24309
R2	0.037	0.037	0.037	0.038
LSI	0.029**	0.028*	0.032***	0.033***
	(0.014)	(0.016)	(0.012)	(0.013)
N	27205	23826	27856	24380
R2	0.017	0.016	0.017	0.016
Time Effects	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Parental controls	Yes	Yes	Yes	Yes
Household controls	Yes	Yes	Yes	Yes

Notes: Models are estimated using the unbalanced sample. Data on health conditions are available in Waves 3 to 5. Please see notes in Table 1 for controls included. Standard errors in parentheses are robust to heteroskedasticity and clustered at child level. Significance levels: $^{+}p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

tetanus, diphtheria, whooping, Hib and meningitis. These are variables available in Wave 2 of the MCS (when children were aged 3). Immunisations prevent children from catching potentially life-threatening infectious diseases. Information on immunisations also reflect indirectly use of health care services by the child. An incomplete course of vaccines could lead to adverse health outcomes, and thus can be used as a proxy of maternal behavioural changes detrimental for the child.

We estimate the corresponding linear probability model for each of the immunisation variables on the contemporaneous exposure of IPV (i.e., also reported in Wave 2). The results are presented in Table 5. All estimates for both the IPV1 or IPV2 definitions yield negative and statistically significant coefficients. Most of the coefficients presented in the table are precisely estimated and these suggest that exposure to IPV reduces the probability of receiving immunisations, with the magnitude of the effect varying between 1.4pp and 2.5pp. This is a small effect, but considering the threat to a child's health of not having the full set of vaccines, it becomes apparent that IPV could potentially lead to serious health consequences in the long-term. Thus, overall, assuming that the parent mostly in charge of the immunisations is the mother, these results are supportive of the existence indirect effects of IPV.[8] The abuse may limit her ability to follow-up on medical appointments and thus, her contribution to the child's health production function.

Table 5
Immunisations during first year of life.

	IPV1		IPV2	
	(1)	(2)	(3)	(4)
Sample	All	Both Parents	All	Both Parents
Polio	−0.017*	−0.019*	−0.010	−0.011
	(0.010)	(0.011)	(0.007)	(0.007)
Ns	9067	8512	9325	8741
R2	0.009	0.014	0.009	0.013
Tetanus	−0.017*	−0.023**	−0.013*	−0.017**
	(0.010)	(0.011)	(0.007)	(0.008)
Ns	9067	8512	9325	8741
R2	0.009	0.013	0.009	0.013
Diphtheria	−0.019*	−0.025**	−0.011	−0.014*
	(0.010)	(0.011)	(0.007)	(0.008)
Ns	9067	8512	9325	8741
R2	0.011	0.016	0.011	0.015
Whooping	−0.020*	−0.022*	−0.012	−0.014*
	(0.011)	(0.012)	(0.008)	(0.008)
Ns	9067	8512	9325	8741
R2	0.010	0.013	0.009	0.012
Hib	−0.017	−0.025**	−0.010	−0.015*
	(0.011)	(0.012)	(0.008)	(0.009)
Ns	9067	8512	9325	8741
R2	0.010	0.014	0.010	0.013
Meningitis	−0.017	−0.022*	−0.013	−0.016*
	(0.012)	(0.013)	(0.009)	(0.009)
Ns	9067	8512	9325	8741
R2	0.009	0.011	0.009	0.011
Time Effects	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Parental controls	Yes	Yes	Yes	Yes
Household controls	Yes	Yes	Yes	Yes

Notes: Models are estimated using the unbalanced sample. Data on vaccination is available in Wave 2. Please see notes in Table 1 for controls included. Standard errors are clustered at the child level. Significance levels: $^{+}p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5. Concluding remarks

This paper looks at the relationship between IPV and child health, using data from the MCS, a large longitudinal, nationally representative cohort sample of children born in the UK between September 2000 and January 2002. Our primary focus is on whether child's exposure to domestic abuse, reported by the mother's own experience of physical abuse at the hands of her partner, has on child's health. We first estimate a linear probability model, and our base case estimates indicate that the children exposed to IPV are 7pp less likely to be reported as in excellent health. These results are robust to the use of different IPV definitions and across sub-samples. The presence of omitted variables may introduce bias into these findings. Utilizing the Oster (2019) methodology to assess the stability of the coefficients suggests that the role of unobservable factors is relatively less significant than that of observable ones for estimating the impact of IPV on a child's health.

We further use propensity score matching methods to estimate the effect of IPV exposure, testing the results to the use of different empirical strategies. Overall, results are in line with the basecase. Our analysis provides compelling evidence that the estimated effects reliably quantify the influence of IPV on a child's health, indicating that IPV exposure exerts a significant adverse effect. Our analysis expands to quantify the impact of IPV on various health conditions, encompassing the presence of specific health conditions, and the likelihood of receiving recommended immunisations within the first year of life. All estimates point towards the deleterious effects of IPV on morbidity and the likelihood of receiving the full complement of recommended immunisations.

It is plausible that child exposure to IPV may produce both direct and indirect effects, stemming from the direct violence inflicted upon the child, and indirect effects that arise when the mother's ability to care for the child is affected as a consequence of the abuse. While we are unable to differentiate the direct from the indirect effects on our measure of general health, the negative impact of IPV on immunisations underscores the possibility that mothers in abusive relationships may see their ability to provide care and thus their contribution to their child's physical well-being disrupted.

The data on IPV from the MCS has some limitations. First, the question we use to proxy for IPV asks whether the husband/partner has ever used force on them. The MCS has no information on the severity of the attack(s) and their frequency. These are aspects of exposure to IPV that are likely to aggravate the child's health. Secondly, we only observe in the data whether the respondent has experienced physical violence, but no other types of violence such as sexual or emotional violence. Rawlings and Siddique (2020) estimate the effects of physical and sexual violence on neonatal, infant and under-5s mortality and find very similar detrimental effects of exposure to any of these types of violence on mortality. Anderberg and Moroni (2020), use data where is possible to distinguish between physical and emotional abuse. They observe that most of physical abuse is linked to emotional abuse and combine these for their analysis. Based on this, we could argue that our question on physical abuse could be partly capturing psychological abuse. Thirdly,

[8] The MCS does not specify which parent accompanies the child for immunisations. However, the 'Childhood Vaccines: Parental Attitudes Survey 2022' findings suggest it is typically the mother. This survey, conducted by the UK Health Security Agency (UKHSA) in collaboration with Bounty, aimed to understand the views of parents with children aged 2 months to 5 years on vaccination. The survey results indicate that in 98% of cases, the mother completed the questionnaire, which suggests it is the mother who is responsible.

we consider children exposure to IPV are also victims of IPV, as recognised in the Domestic Abuse Act 2021, but acknowledge that children that witness and are directly abused may experience a larger reduction in health.

This paper contributes to the extant literature on the relationship between IPV and child outcomes, as it examines the association between IPV and child's health. Our findings offer compelling evidence of the negative spillover effects of IPV, corroborating previous research that links IPV during pregnancy to compromised infant health (Aizer, 2011; Currie et al., 2020; 2022), while also highlighting the far-reaching repercussions of IPV not only for neo-natal health but well into childhood. Given the vital role that child health plays in human capital formation and its cumulative impact on subsequent stages of life, policies intended to address the health needs of children who bear witness to IPV demand careful consideration.

CRediT authorship contribution statement

Mireia Jofre-Bonet: Conceptualization, Methodology, Supervision, Writing – original draft, Writing – review & editing. **Melcior Rossello-Roig:** Conceptualization, Data curation, Methodology, Writing – original draft, Writing – review & editing. **Victoria Serra-Sastre:** Conceptualization, Formal analysis, Methodology, Writing – original draft, Writing – review & editing, Supervision.

Data availability

The authors do not have permission to share data.

Appendix

Table A1
Descriptive Statistics

	NO IPV		IPV1		IPV2	
	Mean	Std.Dev	Mean	Std.Dev	Mean	Std.Dev
Child Vars.						
Excellent	0.599	0.490	0.509	0.500	0.489	0.500
Hearing	0.108	0.311	0.135	0.342	0.127	0.333
Eye	0.167	0.373	0.169	0.375	0.175	0.380
Respiratory	0.241	0.428	0.279	0.449	0.287	0.453
Eczema	0.336	0.473	0.346	0.476	0.335	0.472
Hayfever	0.164	0.371	0.159	0.365	0.154	0.362
LSI	0.167	0.373	0.202	0.402	0.208	0.406
Polio	0.984	0.127	0.963	0.189	0.970	0.171
Tetanus	0.982	0.134	0.960	0.196	0.965	0.183
Diphteria	0.983	0.129	0.960	0.196	0.969	0.175
Whooping	0.977	0.150	0.952	0.214	0.961	0.195
Hib	0.975	0.156	0.952	0.214	0.961	0.195
Meningitis	0.969	0.173	0.947	0.224	0.953	0.212
Age	7.249	2.448	7.125	2.435	6.998	2.378
Gender	0.506	0.500	0.498	0.500	0.518	0.500
Obese	0.029	0.168	0.031	0.172	0.027	0.162
Overweight	0.066	0.248	0.069	0.254	0.061	0.239
Maternal Vars.						
Mother Age	37.016	6.056	36.421	6.464	35.895	6.532
Mother Education	0.417	0.493	0.369	0.483	0.340	0.474
Mother In Work	0.687	0.464	0.638	0.481	0.590	0.492
Widowed/Other	0.006	0.080	0.008	0.089	0.010	0.098
Divorced Separated	0.046	0.208	0.067	0.250	0.059	0.235
Married	0.733	0.442	0.636	0.481	0.638	0.481
Remarried	0.078	0.268	0.096	0.294	0.088	0.283
Single	0.137	0.344	0.193	0.395	0.206	0.404
White	0.902	0.298	0.880	0.326	0.839	0.368
Mixed	0.005	0.072	0.011	0.104	0.013	0.112
Indian	0.023	0.151	0.021	0.143	0.029	0.167
Pakistani	0.039	0.194	0.049	0.217	0.076	0.266
Black	0.018	0.133	0.028	0.164	0.026	0.159
Other Race	0.013	0.111	0.012	0.108	0.017	0.131
Paternal Vars.						
Father Age	39.415	6.729	39.225	7.572	38.762	7.489
Father Education	0.437	0.496	0.394	0.489	0.381	0.486
Father In Work	0.914	0.280	0.860	0.347	0.843	0.363
Household Vars.						
People in HH	4.892	1.207	5.020	1.267	5.057	1.355
Council House	0.141	0.348	0.224	0.417	0.240	0.427
HH Income (log)	10.208	0.630	10.095	0.645	10.008	0.666
N	26200		1013		1664	

Note: This table shows the descriptive statistics for Waves 3 to 5 for the subsample of children with no IPV, and children who live in households with IPV according to both definitions IPV1 and IPV2. IPV is based on the question *"People often use force in a relationship - grabbing, pushing, shaking, hitting, kicking etc. Has your husband/partner ever used force on you for any reason?"*. IPV1 equals 1 if the mother answers "Yes", equals 0 if the answer is "No" and consider "Don't want to answer" as a missing value. IPV2 takes value 1 if the mother responded "Yes" or "Don't want to answer" and 0 if they answered "No". LSI stands for Longs-standing illness. Descriptive statistics for immunisations (polio, tetanus, diphtheria, whooping cough, hib and meningitis) are from wave 2, with a sample size for No IPV of 8690 observations, IPV1 of 377 and IPV2 of 635 observations.

Table A2
Impact of IPV on Children's Health - Balanced sample

Sample	IPV1		IPV2	
	(1)	(2)	(3)	(4)
	All	Both Parents	All	Both Parents
Excellent	−0.056***	−0.062***	−0.069***	−0.073***
	(0.020)	(0.022)	(0.017)	(0.018)
N	20515	18317	20925	18678
R2	0.046	0.044	0.048	0.046
Time Effects	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Parental controls	Yes	Yes	Yes	Yes
Household controls	Yes	Yes	Yes	Yes

Notes: The dependent variable takes value 1 if the parent responds excellent to the question on child's overall health. Standard errors in parentheses are robust to heteroskedasticity and clustered at child level. Models are estimated using the balanced sample. Specifications in columns (1) and (3) use the sample of children in all household types. Specifications in all other columns consider only households in which both biological parents cohabit. *Controls* include the set of variables for the child-related variables (age, sex), parental controls (age, educational level - degree or higher, race, marital status for both the main respondent and the partner) and household-related variables (number of people in the household and whether the family lives in a council house or housing association). Reference category for maternal ethnicity is white and for marital status is Other. Wave fixed effects are included. Significance Levels: $^{+}p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A3
Impact of IPV on Children's Health - Restricted sample

Sample	IPV1		IPV2	
	(1)	(2)	(3)	(4)
	All	Both Parents	All	Both Parents
Excellent	−0.059***	−0.061***	−0.065***	−0.067***
	(0.018)	(0.019)	(0.014)	(0.016)
N	27120	23751	27662	24219
R2	0.050	0.049	0.052	0.051
Time Effects	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Parental controls	Yes	Yes	Yes	Yes
Household controls	Yes	Yes	Yes	Yes

Notes: The sample excludes those respondents that report IPV in each wave. The dependent variable takes value 1 if the parent responds excellent to the question on child's overall health. Standard errors in parentheses are robust to heteroskedasticity and clustered at child level. Models are estimated using the unbalanced sample. Specifications in columns (1) and (3) use the sample of children in all household types. Specifications in all other columns consider only households in which both biological parents cohabit. *Controls* include the set of variables for the child-related variables (age, sex), parental controls (age, educational level - degree or higher, race, marital status for both the main respondent and the partner) and household-related variables (number of people in the household and whether the family lives in a council house or housing association). Reference category for maternal ethnicity is white and for marital status is Other. Wave fixed effects are included. Significance levels: $^{+}p < 0.10$ ** $p < 0.05$, *** $p < 0.01$.

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