Review of Economic Studies (1997) 64, 575-603
0034-6527/97/00270575$02.00

Bounding Causal Effects Using Data from a Contaminated Natural Experiment: Analysing the Effects of Teenage Childbearing

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First version received May 1994; *final version accepted June* 1997 (*Eds.*)

In this paper, we consider what can be learned about causal effects when one uses a contaminated instrumental variable. In particular, we consider what inferences can be made about the causal effect of teenage childbearing on a teen mother's subsequent outcomes when we use the natural experiment of miscarriages to form an instrumental variable for teen births. Miscarriages might not meet all of the conditions required for an instrumental variable to identify such causal effects for all of the observations in our sample. However, it is an appropriate instrumental variable for some women, namely those pregnant women who experience a random miscarriage. Although information from typical data sources does not allow one to identify these women, we show that one can adapt results from Horowitz and Manski (1995) on identification with data from contaminated samples to construct informative bounds on the causal effect of teenage childbearing. We use these bounds to re-examine the effects of early chilbearing on the teen mother's subsequent educational and labour market attainment as considered in Hotz, McElroy and Sanders (1995*a*, 1995*b*). Consistent with their study, these bounds indicate that women who have births as teens have *higher* labour market earnings and hours worked compared to what they would have attained if their childbearing had been delayed.

1. INTRODUCTION

This paper combines two strands of the applied econometrics literature to address the identification of causal effects. It draws on the results in Horowitz and Manski (1995) (hereafter HM) on forming bounds on moments and other statistics of a random variable, Y, from a "contaminated sample". A contaminated sample of Y is one that contains realizations of Y mixed with realizations from a second ("contaminating") distribution. It also follows the recent insight of Heckman and his co-authors in characterizing the selection problem as a missing, or error-ridden, data problem.[1] Our results show how these two insights can be combined to estimate bounds on causal effects when all that is available is error-ridden data from less-than-ideal experiments. This is significant because this occurs in a wide variety of evaluation contexts. For example, Hotz and Sanders (1994, 1996) show that the techniques described below can be used to identify bounds of causal effects in controlled experiments which suffer from contamination due to the inability of experiments to eliminate various forms of non-compliance by subjects. This paper shows that the same techniques can be used when naturally occurring variation randomizes subjects of interest to "treatment" and "control" groups but in doing so also randomizes subjects of no interest to these same groups with indifference to the "needs" of researchers.

We apply our bounds to examine how giving birth as a teenager affects a woman's educational attainment and labour market performance as an adult. A controlled experiment in which age at first birth is randomly assigned would be an ideal context in which to analyse this problem. However, this experiment clearly is not an option. However, in recent work, Hotz, McElroy and Sanders (1997*a*, 1997*b*) (HMcS, hereafter) use whether a teenage woman's first pregnancy ends in a miscarriage to perform this function. If miscarriages are random with respect to other factors that influence a woman's decision to give birth, then they are an ideal instrumental variable (IV) for teen births since they exogenously delay the age at first birth.

Unfortunately, not all miscarriages occur at random. For example, epidemiological studies have found that smoking and drinking during pregnancy significantly increase the incidence of miscarriages.[2] Furthermore, such behaviours are likely to be correlated with a woman's subsequent human capital accumulation and labour market productivity. That is, miscarriages may fail the exclusion restrictions required of a proper instrumental variables estimator—that the effect of the instrument on the outcome of interest works only through its effect on the treatment variable. While not all miscarriages are random, some are. In particular, some miscarriages result from the abnormal formation of fetal chromosomes. There is strong evidence that the occurrence of such abnormalities is random.[3] Consequently, the group of women who miscarry constitute a *mixture* of women who experience random and non-random miscarriages, where it is typically unknown who experienced which type of miscarriage. As such, our data on miscarriages qualifies as a *contaminated* sample of random miscarriages in the terminology of HM. Hence, we refer to miscarriages as a *contaminated instrument* for the mother's age at birth of first child.

We show that a contaminated instrument can be used to learn a good deal about the causal effects of teenage childbearing. Since some fraction of miscarriages are random, we can draw on results from HM to derive an optimal set of non-parametric bounds for this causal effect. Using estimates of the proportion of miscarriages that are random from previous epidemiological studies and data from the National Longitudinal Survey of Youth (NLSY), we estimate the above bounds on the causal effects of early childbearing. Then, we use these bounds to address several substantive and methodological questions:

1. Do the bounds enable us to determine whether the causal effect of teenage childbearing is positive or negative for several indicators of a mother's subsequent outcomes of interest?
2. Using these bounds, can we reject *point* estimates of this causal effect derived using linear IV estimation methods? (This estimator is used by HMcS in their study of

1. See, for example, Heckman (1990) and Heckman, Ichimura, Smith and Todd (1996).
2. See Kline, Stein and Susser (1989) for a review of this evidence.
3. Kline, Stein and Susser (1989).

teenage childbearing in the U.S.) In other words, are the controversial assumptions maintained by this estimator consistent with the data?

3. Can we reject ordinary least squares (OLS) estimates of the causal effect? This estimator has been used in previous studies of the effects of teenage childbearing. However, its validity is based on assumptions which may be inconsistent with the data, as well.
4. More generally, how do the bounds based on our contaminated instrument tighten as we impose the types of additional assumptions made within traditional IV estimation strategies? Furthermore, do we learn anything about the validity of these additional restrictions from the bounds?

While applied to a specific example, the approach we develop is potentially applicable to a broader class of problems. The issue of a contaminated instrument represents a common source of concern in the application of IV estimation methods. Researchers often face the possibility that candidates for instrumental variables do not meet the standard exclusion restrictions for some sub-population of interest but meet the restriction for the remainder of the population. The approach developed in this paper provides a strategy for conducting one's analysis about causal effects when the IV conditions are met for only part of the population of interest.

The remainder of the paper is organized as follows. In Section 2, we outline the general structure of our evaluation problem, namely estimating the causal effect of early childbearing on teenagers, and how miscarriages might be used to identify these effects. In Section 3, we discuss bounding the causal effect of teenage childbearing using results from HM. In Section 4, we discuss the role that miscarriages play in reducing the uncertainty of the effect of teenage childbearing. In Section 5, we show the relationship between the IV estimator and the HM bounds. We also demonstrate how the HM bounds can be tightened by invoking some, but not all, of the controversial conditions maintained in standard IV estimation. In Section 6, we discuss the estimation of these bounds and how to test propositions about the causal effect of teenage childbearing with the sorts of data typically available. In Section 7, we deal with estimating two of the terms assumed known in the derivation of the bounds in Section 6.

In Section 8 we discuss the data. Finally, in Section 9, we present results from our analysis of the causal effects of teenage childbearing on a few outcomes of interest. We present several sets of bounds based on alternative estimates of the proportion of miscarriages that are random, the proportion of women who would have had a birth if they had not had a random miscarriage, and the nature of misreporting of pregnancies and pregnancy resolutions in our data. We also present test statistics associated with the four questions noted above. In particular, we assess the validity of conclusions obtained from using either the standard IV or OLS estimator. Finally, unlike previous applications of the non-parametric bounds considered here,[4] the bounds presented below are tight enough to determine the signs of the effects of teenage childbearing on white women's annual hours of work and earnings.

2. PREGNANCY RESOLUTION, THE CAUSAL EFFECT OF INTEREST AND THE EVALUATION PROBLEM

In this section we characterize the problem of identifying the causal effect of teenage women not delaying their childbearing until adulthood. As in HMcS, we restrict our

4. See Manski, Sandefur, McLanahan and Powers (1992) and Clements, Heckman and Smith (1994) for two applications in which the bounds are not very informative.

attention to identification of these effects for the population of women who become pregnant as adolescents. We define adolescence as any age, τ, less than 18.[5] A pregnancy can be resolved in one of four ways: it can end in a birth (B); an induced abortion (A); or one of two types of miscarriages, non-random (NR) or random ones (RM). The non-random miscarriage category includes those which are induced by such behaviours as smoking and drinking. From a choice-theoretic perspective, the first three ways of resolving pregnancies can be viewed as choices women make, either directly or indirectly. As such, these choices, and their determinants, may be correlated with the outcome variables of interest. Let D indicate the way a woman chooses to resolve her pregnancy, where $D=B$, A or NR.

In contrast to the first three methods for resolving a pregnancy, random miscarriages represent events that are exogenously imposed on pregnant women. Let Z^* indicate the occurrence of a random miscarriage, where $Z^*=RM$ or $\sim RM$. A key feature of random miscarriages is that they preclude women from choosing how their pregnancies are resolved.[6] Hence, when a random miscarriage occurs, a pregnant woman's preferences for how her pregnancy would be resolved may not be revealed. Nonetheless, it is useful to characterize what a woman's choice would be if she does not experience a random miscarriage. Let D^L indicate a woman's *latent* pregnancy resolution choice, where $D^L=B$, A or NR. A woman's latent choice status is defined to be how her pregnancy would be resolved in the absence of a random miscarriage. For now, we assume that the occurrence of a random miscarriage merely precludes choice; below, we consider the possibility that such an experience might alter a woman's preferences about subsequent childbearing or other behaviours.

Finally, let Y denote an outcome of interest (for example, annual labour market earnings) at age $t>\tau$, where we forego indexing Y by t to avoid excessive notation. Following the framework of the recent literature on the identification of treatment effects, we define Y_1 to be the outcome that would result if a woman's first birth occurs when she is a teen and Y_0 to be the outcome that would result if her childbearing is delayed.

Our interest is in the effect of a woman having a birth as a teen vs. delaying it—either to an adult age or permanently—on her subsequent outcomes for the population of women who first gave birth as teens. More precisely, we are interested in identifying:

$$\begin{aligned} \alpha(X) &\equiv E(Y_1|D=B,X)-E(Y_0|D=B,X) \\ &= E(Y_1-Y_0|D=B,X), \end{aligned} \tag{1}$$

where α may vary with X, a vector of exogenous characteristics. From this point onward, the conditioning on X is left implicit to avoid excessive notation.

The causal effect in (1) characterizes how different a teen mother's subsequent Ys would be if she postponed or forewent the birth. This causal effect is analogous to the mean effect of *treatment on the treated* in the evaluation literature. As discussed in Heckman and Robb (1985) and Heckman (1995*b*), α is a somewhat non-standard parameter from the vantage point of structural modelling in econometrics. Structural modelling is typically

5. While restricting ourselves to this population, we do not presume that a woman's decision to become pregnant is exogenous with respect to her childbearing decisions or subsequent socioeconomic attainment. Rather, our inferences are limited to a well-defined but potentially *endogenously-determined* population.

6. This feature of random miscarriages presumes that such miscarriages occur early in a woman's pregnancy, before she could choose to abort the fetus. While not completely true, most random miscarriages do occur at very young gestational ages. Below, we discuss the implications of this feature not holding with probability one for the identification of the causal effect of early childbearing and outline strategies for dealing with this possibility in our empirical analysis.

used to identify the average effect of a potentially endogenous event (a teen birth) on Y for everyone in a population, rather than just those who are observed to chosen (in our case teen mothers) the event. We focus on the causal effect for teen mothers, α, for two reasons. First, this causal effect is more readily identified from available data than are causal effects applicable to the full population of women. Second, policies that seek to reduce the rate of teenage childbearing will likely target women who, under the *status quo*, would become teenage mothers. Knowledge of α is sufficient to assess the potential consequences of eliminating teenage childbearing for these women.[7]

The fundamental problem in identifying α is that while we observe the outcomes of teenage mothers, Y_1, we can never directly observe the counterfactual outcome, Y_0, for these women, i.e. what the outcome for these women would have been in the absence of a teen birth. At issue is what comparison group to use to obtain data on Y_0 and its expectation. Often the outcomes for women who chose not to have a teen birth are used. In general, using the outcomes for the latter group to measure the counterfactual outcomes for the former will not identify α. Rather, it will identify

$$E(Y_1|D=B)-E(Y_0|D\neq B)=\alpha+\{E(Y_0|D=B)-E(Y_0|D\neq B)\}, \tag{2}$$

where the expression in braces is the *selection-bias* term—i.e. the mean difference in outcomes that would have existed between women who had births and women who did not if both had delayed their childbearing.

In principal, a properly conducted randomized experiment could be used to eliminate the selection bias in (2) and identify α. In such a *controlled* experiment, a randomly selected sample of pregnant teenage women, who are latent birth types ($D^L=B$), would have their pregnancies terminated. Their subsequent outcomes would be compared with those for teen mothers in order to form an unbiased estimator of α. But, such an experiment clearly would be unethical, and, thus, not feasible to implement. But, as noted in the introduction, a *naturally*-occurring experiment, in which teen mothers experience random miscarriages, would seem to mimic this controlled experiment and provide ideal data for identifying α. As we shall see, using miscarriages as a natural experiment does not replicate the controlled experiment just described in several important ways. Nonetheless, to help clarify how we will use this (flawed) natural experiment to construct bounds on α, we characterize, in a precise manner, the three conditions which would need to be met for this experiment to identify α.

Condition 1. The occurrence of a random miscarriage precludes the occurrence of a birth, while the absence of a random miscarriage ensures the occurrence of a birth for latent-birth type women.

Condition 2. Random miscarriages do not affect outcomes of latent-birth type women, i.e. $E(Y_0|D^L=B, Z^*=RM)=E(Y_0|D^L=B)$.

Condition 3. Latent-birth type women and their miscarriage status are observable.

7. There are other causal effects related to teenage childbearing that might be of potential interest. For example, one may be interested in the distribution of effects rather than just the mean. The study by Clements, Heckman and Smith (1994) focuses on identifying such treatment effects at different quantiles of its distribution in the context of a training programme. For a general discussion of the distinctions between and applicability of alternative treatment effect definitions in the evaluation context, see Heckman (1992, 1995*a*) and Manski (1996).

If these Conditions are met, α is identified and could be estimated as the difference between the average outcomes of latent-birth type women who had births and who had miscarriages.

While Condition 1 is not controversial, the validity of the remaining conditions is not clear. A random miscarriage may cause behavioural responses, such as depression, if the child was wanted, or elation, if it was unwanted. This effect of the randomizing event is labelled the "Hawthorne Effect" in randomized experiments and Heckman and Smith (1995) refer to this phenomena as "randomization bias." To satisfy Condition 3 the researcher must know the identities of latent-birth type women. For women who have a child, this identification is easy. However, among those who experience a miscarriage this identification is typically not possible. The researcher is unable to distinguish between latent-birth, abortion and non-random miscarriage types in the miscarriage population. In the next three sections we explore what one can learn about α when Condition 3 is not met. We show that robust sets of bounds of α can be formed and that the natural experiment arising from the random nature of some miscarriages, can decrease the width of these bounds. We also examine how alternative assumptions can be used in place of Conditions 2 and 3 to either further tighten these bounds or point identify α.

3. BOUNDING THE CAUSAL EFFECT OF TEENAGE CHILDBEARING USING OBSERVED MISCARRIAGES AS A NATURAL EXPERIMENT

We begin by investigating what we can learn about α when only Conditions 1 and 2 hold. Under these conditions we are unable to distinguish random from non-random miscarriages, but we do observe when miscarriages occur. Let Z indicate the occurrence of a miscarriage, either random or otherwise, where $Z=M$ or $\sim M$. Although one is unable to identify the membership of the ideal comparison group for identifying α and, as such, cannot identify the distribution (or moments) of $Y_0|D^L=B, Z^*=RM$, with data from the natural experiment of women experiencing miscarriages, the distribution of $Y_0|Z=M$ is identified. This later distribution is a *mixture* of Y_0 for women of the various latent resolution types who experience random and non-random miscarriages. Thus, its mean is given by

$$\begin{aligned} E(Y_0|Z=M) = {} & [E(Y_0|D^L=B, Z^*=RM)P_B + E(Y_0|D^L=A, Z^*=RM)P_A \\ & + E(Y_0|D^L=NR, Z^*=RM)P_{NR}]\left[\frac{P_{RM}}{P_M}\right] \\ & + E(Y_0|D=NR, Z^*=\sim RM)\left[\frac{(1-P_{RM})P_{NR}}{P_M}\right], \end{aligned} \tag{3}$$

where $P_{RM}=\Pr(Z^*=RM)$ is the probability of a random miscarriage; $P_j=\Pr(D^L=j)$ is the probability of the j-th pregnancy resolution being a woman's latent choice, $j=B$, A and NR for which $P_B+P_A+P_{NR}=1$; and P_M is the probability of a miscarriage, where $P_M=P_{RM}+(1-P_{RM})P_{NR}$. Expression (3) makes clear that $E(Y_0|Z=M)$ is a *contaminated* measure of the conditional mean needed to identify α, namely, $E(Y_0|D^L=B, Z^*=RM)$.

While, in the absence of Condition 3, α is not point-identified from the natural experiment of random miscarriages, this experiment does enable us to place a bound on it. Recall from (1) that $\alpha=E(Y_1|D=B)-E(Y_0|D^L=B)$; since $E(Y_1|D=B)$ is identified from data on women who had a birth as a teen, placing a bound on α rests entirely on forming a bound for $E(Y_0|D^L=B)$. It follows from (3) that

$$E(Y_0|D^L=B, D=RM)=\left(\frac{1}{\lambda^*}\right)[E(Y_0|Z=M)-[1-\lambda^*]E(Y_0|D^L\neq B, B, Z=M)], \quad (4)$$

where λ^* $(\equiv P_B P_{RM/}P_M)$ is the proportion of miscarriages that occur randomly to latent-birth type women. While $E(Y_0|Z=M)$ is always identified (or can be consistently estimated), (4) makes clear that $E(Y_0|D^L=B)$ $[=E(Y_0|D^L=B, D=RM)]$—and, thus, α—is not identified since $E(Y_0|D^L\neq B, Z=M)$ is not identified.

But the fact that (4) holds, which follows from the natural experiment of randomly-occurring miscarriages, implies that there is a tight set of bounds on $E(Y_0|D^L=B)$. This follows from results in Horowitz and Manski (1995) on the identification of bounds for moments of random variables using data from contaminated samples so long as one knows (or can estimate) either λ^* or a *lower bound* on this proportion. Let λ denote this lower bound, i.e. $\lambda\leqq\lambda^*$. Below, we discuss how one can estimate λ from epidemiological studies of random miscarriages and vital statistics data. For now, we assume that λ is known.

The intuition for how bounds on $E(Y_0|D^L=B)$ can be formed from (4) is as follows. Suppose that half of the miscarriage population are latent-birth types and half are latent non-birth types. As such, the population of non-latent-birth types could not have a distribution of outcomes below that of the bottom half of the miscarriage population. So, the mean outcome for the bottom half of the miscarriage population is a lower bound on the average outcome for non-latent-birth types. In general, the fraction $(1-\lambda)$ of miscarriages are non-latent-birth types and the expected value of the $[1-\lambda]$-*quantile* of the distribution of outcomes among the miscarriage population is a lower bound on the average outcome for non-latent-birth types. Notationally, define $Y_{M,1-\lambda}$ to be the $[1-\lambda]$-*quantile* of the distribution of Y, i.e. $\Pr(Y\leqq Y_{M,1-\lambda}|Z=M)=1-\lambda$. Then the greatest lower bound on $E(Y_0|D^L\neq B, Z=M)$ is given by the following truncated mean

$$E(Y_0|Y\leqq Y_{M,1-\lambda}, Z=M). \quad (5)$$

By similar reasoning, the smallest upper bound on $E(Y_0|D_L\neq B, Z=M)$ is given by

$$E(Y_0|Y\geqq Y_{M,\lambda}, Z=M). \quad (6)$$

It follows from Corollary 4.1 in HM that these bounds on $E(Y_0|D^L\neq B, Z=M)$ are *sharp*, that is "they exhaust the information about the parameters that is available from the sampling process and maintained assumptions."[8]

Using (5) and (6) as bounds for $E(Y_0|D^L\neq B, Z=M)$, we define the *Horowitz–Manski* (HM) *bounds* on α as

$$\alpha\in[A_{1L}(\lambda), A_{1U}(\lambda)], \quad (7)$$

where

$$A_{1L}(\lambda)\equiv E(Y_1|D=B)-\left(\frac{1}{\lambda}\right)\{E(Y_0|Z=M)-[1-\lambda]E(Y_0|Y\leqq Y_{M,1-\lambda}, Z=M)\}, \quad (8)$$

$$A_{1U}(\lambda)\equiv E(Y_1|D=B)-\left(\frac{1}{\lambda}\right)\{E(Y_0|Z=M)-[1-\lambda]E(Y_0|Y\geqq Y_{M,\lambda}, Z=M)\}. \quad (9)$$

Notice that the HM bounds are defined even if Y does not have bounded support.

8. Horowitz and Manski (1995), pp. 281–282.

4. THE VALUE OF THE EXPERIMENT VS. THE VALUE OF THE EMPIRICAL DISTRIBUTION FOR BOUNDS

In order to construct the bounds based on equation (4), one needs only to know that there exists a sub-population for whom the experiment described by Conditions 1 and 2 hold and a lower bound on the proportion of the observed comparison group that belong to this sub-population. However, to assess the value of identifying such a sub-population, it is worth considering what we could learn in its absence.

Suppose one does not impose Conditions 1 and 2 but assumes that Y has bounded support, i.e. $Y \in [Y_L, Y_U]$. Then it follows from Manski (1989, 1990) that $E(Y_0 | D^L = B) \in [Y_L, Y_U]$ and, hence,

$$\alpha \in [A_{2L}, A_{2U}], \tag{10}$$

where

$$A_{2L} = E(Y_1 | D = B) - Y_U, \tag{11}$$

$$A_{2U} = E(Y_1 | D = B) - Y_L. \tag{12}$$

We refer to A_{2L} and A_{2U} as the *non-experimental bounded outcome* (*NE-BO*) *bounds* for α. While quite general, the NE-BO bounds are not particularly tight. The width of these bounds is always $Y_U - Y_L$. Furthermore, the NE-BO bounds for α always span zero and, as such, cannot be informative as to the sign of α.

The HM bounds improve on the NE-BO bounds in two ways. They exploit the presence of an experiment and they use the shape of the empirical distribution in constructing informative bounds. It is of interest to know how important each of these factors is in obtaining tight bounds. To judge the importance of using the empirical distribution we construct the tightest set of bounds that assume Conditions 1 and 2 but that does not use the shape of the empirical distribution. Since $Y \in [Y_L, Y_U]$, it follows that $E(Y_0 | D^L \neq B, Z = M) \in [Y_L, Y_U]$. Substituting these bounds for $E(Y_0 | D^L \neq B, Z = M)$ into (4), gives

$$E(Y_0 | D^L = B) \in \left[\frac{1}{\lambda} \{E(Y_0 | Z = M) - [1 - \lambda] Y_U\}, \frac{1}{\lambda} \{E(Y_0 | Z = M) - [1 - \lambda] Y_L\} \right].$$

Using this latest set of bounds in conjunction with the earlier bounds of $E(Y_0 | D^L = B) \in [Y_L, Y_U]$, we define the *experimental bounded outcome* (*E-BO*) *bounds* for α as

$$\alpha \in [A_{3L}, A_{3U}], \tag{13}$$

where

$$A_{3L}(\lambda) = E(Y_1 | D = B) - \min \left[Y_U, \frac{1}{\lambda} \{E(Y_0 | Z = M) - [1 - \lambda] Y_L\} \right], \tag{14}$$

$$A_{3U}(\lambda) = E(Y_1 | D = B) - \max \left[Y_L, \frac{1}{\lambda} \{E(Y_0 | Z = M) - [1 - \lambda] Y_U\} \right]. \tag{15}$$

We note that these bounds are defined whenever Y is bounded from at least one side. It follows from (14) and (15), that the E-BO lower bound is greater than the NE-BO lower bound if and only if $\lambda > [E(Y_0 | Z = M) - Y_L]/[Y_U - Y_L]$ and the E-BO upper bound is less than the NE-BO upper bound if and only if $\lambda > [Y_U - E(Y_0 | Z = M)]/[Y_U - Y_L]$. The degree to which the width of the E-BO bound is smaller than the NE-BO bound gives us a measure of the importance of the experiment. The degree to which the width of the HM bound is smaller than the E-BO bound gives us a measure of the gains to using the empirical distribution with the experiment vs. using the experiment alone.

5. THE RELATIONSHIP BETWEEN THE HM BOUNDS AND IV ESTIMATION

Most previous studies of the effects of teenage childbearing have been based on non-experimental data and relied on either differencing strategies[9] or controlling for observables in a regression framework to eliminate the selection bias.[10] Others, including HMcS, use instrumental variables to identify α.[11] Each of these methods requires one to maintain various assumptions about the nature of the data and the selection process in order to identify α. Starting with Heckman and Robb (1985), a number of papers have clarified the nature of the assumptions underlying many of these methods, including those applied to experimental data. In the spirit of that work, we wish to make explicit which set of assumptions must be imposed in addition to Conditions 1 and 2 for an IV estimator to identify α.

Suppose the following conditions held:

Condition 1′. The occurrence of a random miscarriage excludes all other outcomes. In the absence of a random miscarriage,

(i) latent-birth women have a birth;
(ii) latent-abortion women have an abortion;
(iii) latent non-random miscarriage women have a non-random miscarriage.

Condition 2′. Miscarriages do not affect non-birth outcomes:

(i) $E(Y_0|D^L=B, Z^*=RM)=E(Y_0|D^L=B)$;
(ii) $E(Y_0|D^L=A, Z^*=RM)=E(Y_0|D=A)$;
(iii) $E(Y_0|D^L=NR, Z^*=RM)=E(Y_0|D=NR)=[P_B/(1-P_{NR})]E(Y_0|D^L=B)$ $+[P_A/(1-P_{NR})]E(Y_0|D=A)$;
(iv) $E(Y_0|D^L=NR, Z^*=\sim RM)=E(Y_0|D=NR)$.

Condition 3′. Miscarriages are observable events.

Condition 4′. Random miscarriages are independent of a woman's latent type, i.e. $P(D^L=j|Z^*=RM)=P(D=j|Z^*=\sim RM)=P_j$, for $j=A$, B, and NR.

Recall that conditioning on X has been suppressed. Thus, Condition 2′(iii) claims that while non-random miscarriages may be correlated with Y_0 unconditionally, they can be written as a weighted average of the latent-birth and abortion outcomes conditional

9. See Geronimus and Korenman (1992, 1993), who model differences in sister-pairs, and Rosenzweig and Wolpin (1995), who use a much more elaborate variant of differencing across family members (sisters and cousins) in modelling the effects of early childbearing on the mother's outcomes and the well-being of her children.

10. This is the approach used in many of the early studies of teenage childbearing. See Trussell (1976), Card and Wise (1978), Card (1981), Waite and Moore (1978), and Marini (1984). This corresponds to the *selection-on-observables* strategy of Barnow, Cain and Goldberger (1980).

11. Grogger and Bronars (1993) and Bronars and Grogger (1994) used another type of instrument—the random occurrence of twin births—to estimate a causal effect different from α.

on X. This is a strong assumption, but HMcS point out that if the epidemiological literature is correct, then, conditioning on smoking and drinking during pregnancy is sufficient for these conditions to hold. Given Conditions 1′–4′, it follows that[12]

$$\alpha = E(Y_1|D=B) - E(Y_0|D^L=B) = \frac{E(Y|Z\neq M) - E(Y|Z=M)}{[P_B/(1-P_{NR})]}. \tag{16}$$

The derivation of (16) is straightforward. The expectations in (16) can be written as

$$E(Y|Z\neq M) = \left[\frac{P_B}{(1-P_{NR})}\right]E(Y_1|D=B) + \left[\frac{P_A}{(1-P_{NR})}\right]E(Y_0|D=A), \tag{17}$$

and

$$\begin{aligned} E(Y|Z=M) &= \left(\frac{P_{RM}}{P_M}\right)[P_B E(Y_0|D^L=B, Z^*=RM) + P_A E(Y_0|D^L=A, Z^*=RM) \\ &\quad + P_{NR}E(Y_0|D^L=NR, Z^*=RM)] \\ &\quad + \left(\frac{(1-P_{RM})P_{NR}}{P_M}\right)E(Y_0|D=NR, Z^*\neq RM) \\ &= \left(\frac{P_{RM}}{P_M}\right)[P_B E(Y_0|D^L=B) + P_A E(Y_0|D=A) + P_{NR}E(Y_0|D=NR)] \\ &\quad + \left(\frac{(1-P_{RM})P_{NR}}{P_M}\right)E(Y_0|D=NR) \\ &= \left[\frac{P_B}{(1-P_{NR})}\right]E(Y_0|D^L=B) + \left[\frac{P_A}{(1-P_{NR})}\right]E(Y_0|D=A), \end{aligned} \tag{18}$$

where the second equality follows from Conditions 2′(i), 2′(ii), and 2′(iv) and the third from Condition 2′(iii). We note that one can form a consistent estimator of α so long as $E(Y|Z=M)$, $E(Y|Z\neq M)$, P_B and P_{NR} can be consistently estimated. Non-parametric estimators of $E(Y|Z=M)$ and $E(Y|Z\neq M)$ can be formed using standard methods, given data on miscarriages and their corresponding outcomes, Y, for a random sample of women who first became pregnant as teens. Denote these estimators as: $\hat{E}(Y|Z=M)$ and $\hat{E}(Y|Z\neq M)$. Consistent estimators of P_B and P_{NR} are not so straightforward. In general, survey data does not allow the econometrician to distinguish between random and non-random miscariages. Without this distinction, one cannot form a consistent estimator for either P_B or P_{NR}. However, assuming one could attain consistent estimates of these parameters, possibly from epidemiological studies,[13] then it follows that a consistent estimator of α is given by the sample analogue to equation (16)

$$\hat{\alpha} = \frac{\hat{E}(Y|D\neq M) - \hat{E}(Y|D=M)}{[\hat{P}_B/(1-\hat{P}_{NR})]}. \tag{19}$$

12. This identification result was first noted by Bloom (1984) in the context of controlled experiments. Also see Angrist and Imbens (1991), and Hotz and Sanders (1994, 1996) and Heckman, Smith and Taber (1994) for more on this estimator.

13. See KSS.

An alternative estimator of α replaces Conditions 1′(iii), 2′(iii), and 2′(iv), with the assumption that all miscarriages are random, i.e. $P_{NR}=0$. Then the estimator in (19), reduces to the standard IV estimator with Z as an instrument for B

$$\hat{\alpha}=\frac{\hat{E}(Y|D\neq M)-\hat{E}(Y|D=M)}{\hat{P}_B}. \tag{20}$$

Furthermore, given that P_{NR} is known, a consistent estimator of P_B is easily obtained from data on births, abortions and miscarriages for a random sample of women who first became pregnant as teens. This later estimator was used in HMcS and is the IV estimator on which we concentrate for the remainder of the paper. Note that these are just two of many possible IV estimators, each of which will be based on a different set of assumptions.

In contrast to the IV estimator, the HM bounds only require Conditions 1′(i), 2′(i), and 3′. However, the HM bounds can be improved by imposing any of the remaining conditions. In particular, it is of great interest to construct bounds that impose the more plausible assumptions but avoid imposing the most controversial ones. For example, Condition 2′(ii) states that having a random miscarriage does not effect a woman's outcome if she would have had an abortion. This condition seems no more stringent than assuming that having a random miscarriage does not have a direct effect on a women's outcome if she would have had a birth (Condition 2′(i)), a condition we must maintain to use miscarriages as an experiment.

To see the effect that maintaining Condition 2′(ii) has on the bounds for α, note that (4) can be written as

$$E(Y_0|D^L=B)$$
$$=\frac{1}{\lambda}[E(Y_0|Z=M)-\theta E(Y_0|D=A)-[1-\lambda-\theta]E(Y_0|D^L=RM, Z=M)], \tag{21}$$

where θ ($\equiv P_A P_{RM}/P_M$) is the proportion of miscarriages that occurred randomly to latent-abortion types. Using bounds analogous to (5) and (6) for $E(Y_0|D^L=RM, Z=M)$, it follows that the *modified HM bounds* for α are given by[14]

$$\alpha\in[A_{4L}(\lambda,\theta), A_{4U}(\lambda,\theta)], \tag{22}$$

where

$$\begin{aligned} A_{4L}(\lambda,\theta)\equiv{}&E(Y_1|D=B)\\ &-\frac{1}{\lambda}\{E(Y_0|Z=M)-\theta E(Y_0|D=A) \qquad (23)\\ &\qquad -[1-\lambda-\theta]E(Y_0|Y\leqq Y_{M,1-\lambda-\theta}, Z=M)\},\\ A_{4U}(\lambda,\theta)\equiv{}&E(Y_1|D=B)\\ &-\frac{1}{\lambda}\{E(Y_0|Z=M)-\theta E(Y_0|D=A) \qquad (24)\\ &\qquad -[1-\lambda-\theta]E(Y_0|Y\geqq Y_{M,\lambda-\theta}, Z=M)\}. \end{aligned}$$

14. If one strengthens Condition 2′(ii) to the distribution of Y given one is a latent-abortion type, f_A, is invariant with respect to a miscarriage, then one can compute the distribution of the miscarriage group purged of the latent-abortion types, i.e. $g=(f_M-P_Af_A)/(1-P_A)$. Then one can compute the bounds on α described in (9) and (10) using g, instead of f_M. These bounds are at least as tight as the bounds in (23) and (24).

As long as $E(Y_0|D=A)>E(Y_0|Y_{M,1-\lambda}\leqq Y\leqq Y_{M,1-\lambda-\theta}, Z=M)$ then $A_{4L}(\lambda,\theta)>A_{1L}(\lambda,\theta)$. Similarly as long as $E(Y_0|D=A)<E(Y_0|Y_{M,\lambda-\theta}\leqq Y\leqq Y_{M,\lambda}, Z=M)$ then $A_{4U}(\lambda,\theta)<A_{1U}(\lambda,\theta)$. Thus, imposing Condition 2′(ii) has the potential of tightening the bounds around α without imposing the remaining IV assumptions necessary for point identification.

6. ESTIMATING THE BOUNDS FOR α AND USING BOUNDS TO TEST VARIOUS HYPOTHESES

Obtaining consistent estimates of the four sets of non-parametric bounds on α is relatively straightforward. In particular, one needs to replace the various population conditional expectation functions used to form the bounds with their sample analogues. For example, given a random sample of size N, the truncated means used to form $[A_{3L}, A_{3U}]$ can be consistently estimated with the following sample statistics

$$\hat{E}(Y_0|Y\leqq Y_{M,1-\lambda}, Z=M)=\frac{\sum_{i=1}^{N} Y_i\cdot 1(Z_i=M)1(Y_i<\hat{Y}_{M,1-\lambda})}{\sum_{i=1}^{N} 1(Z_i=M)1(Y_i<\hat{Y}_{M,1-\lambda})}, \tag{25}$$

and

$$\hat{E}(Y_0|Y\geqq Y_{M,\lambda}, Z=M)=\frac{\sum_{i=1}^{N} Y_i\cdot 1(Z_i=M)1(Y_i>\hat{Y}_{M,\lambda})}{\sum_{i=1}^{N} 1(Z_i=M)1(Y_i>\hat{Y}_{M,\lambda})}, \tag{26}$$

where $\hat{Y}_{M,1-\lambda}$ and $\hat{Y}_{M,\lambda}$ denote the $[1-\lambda]$- and λ-quantiles of the empirical distribution of Y for the miscarriage sample ($Z=M$). In addition, one must have knowledge of λ. Non-parametric kernel estimation methods can be used to form all of these estimates.[15]

Horowitz and Manski (1996) show that estimates of the HM bounds formed with sample analogues have a normal asymptotic distribution under the assumption that λ is known. Furthermore, this result can be readily extended to situations in which λ is consistently estimated from external data sources.[16] An immediate implication of their results is that our third and fourth sets of bounds on α will be distributed asymptotically normal, since these bounds are a linear combination of HM bounds and sample means. Given the difficulty in computing the asymptotic covariance matrices for these estimators, we employ bootstrapping methods to estimate them. In the bootstrap, all computations were conditional on the regressors and, hence, the covariance's are conditional on the distribution of the regressors in our sample.

Knowledge of the asymptotic distributions for the estimators of the bounds on α can be exploited to construct classical hypothesis tests about α. In all of the tests that follow, let s run from 1 to 4 and index the four sets of bounds. We focus on three sets of tests. First, tests on the sign of α. To test whether we can reject that α is *negative*, we evaluate the following hypotheses:

$$H_0: A_{sL}\leqq 0 \text{ vs. } H_A: A_{sL}>0,$$

15. See Härdle (1990) for a description of kernel estimation methods.

16. In the empirical analysis presented below, we do consider bounds that are based on estimates of λ and θ derived from the same sample as used to estimate the other conditional expectation functions comprising the bounds. While not directly covered by the Horowitz and Manski (1996) results, their results can be readily extended to this case.

and to test whether we can reject that α is *positive*, we evaluate:

$$H_0: A_{sU} \geqq 0 \text{ vs. } H_A: A_{sU} < 0.$$

Given the asymptotic normality estimates, the conduct of these tests is straightforward.

Second, we assess the appropriateness of estimators which point-identify α but are based on assumptions beyond those needed for the bounds. The existing empirical literature of the effects of teenage childbearing contains a number of such point estimators whose validity is predicated on maintaining alternative sets of assumptions. As we have noted, one of the virtues of using the non-parametric bounds on α developed in Section 4 is they contain the true α under relatively weak assumptions. We focus on the validity of both the linear IV estimator used in HMcS and an ordinary least squares (OLS) estimator, where we include a set of exogenous variables in an attempt to control for selection bias.[17]

To illustrate the structure of the test, consider testing the validity of the IV estimator discussed in Section 3. If the IV estimator is valid then Conditions 1′–4′ hold, and it follows that

$$\alpha \in [A_{sL}, A_{sU}]. \tag{27}$$

Letting ϕ denote the vector $(A_{sL}, A_{sU}, \alpha)'$, condition (27) is equivalent to the joint hypothesis that

$$\alpha - A_{sL} \geqq 0 \quad \text{and} \quad A_{sU} - \alpha \geqq 0, \tag{28}$$

or that the vector $\mu \equiv R\phi \geqq 0$, where

$$R = \begin{pmatrix} -1 & 0 & 1 \\ 0 & 1 & -1 \end{pmatrix}. \tag{29}$$

In this context, the null hypothesis is $\mu \geqq 0$ vs. the alternative $\mu \in R^2$. The latter formulation makes clear that this is a test of whether parameters of interest satisfy a set of inequality constraints. For a discussion of the distributional properties of tests of joint inequality constraints see Perlman (1969) and Wolak (1989). In our application, because it is known that at most one of the inequality constraints in (28) can be violated in any sample, the test statistic T (defined below) has the following distribution

$$T = (R\hat{\phi} - R\phi)' \Sigma_\phi^{-1} (R\hat{\phi} - R\phi) \sim \tfrac{1}{2}\chi_1^2,$$

where Σ_ϕ is the variance–covariance matrix of ϕ. Again, we employ bootstrapping methods to estimate Σ_ϕ.

The third and final test assesses the validity of the exclusion restriction for latent-abortion types (Condition 2′(ii)) which is imposed to attain the fourth set of bounds. If this assumption is true, then the bounds imposing these restrictions should lie inside bounds that do not. Given that all of the bounds have a normal asymptotic distribution, it is straightforward to test the following null hypotheses

$$H_0: A_{4L} \geqq A_{sL} \quad \text{and} \quad H_0: A_{4U} \leqq A_{sU}.$$

7. ESTIMATING λ AND θ

In constructing the bounds in Section 4 and the hypothesis tests in Section 5, it was assumed that λ and θ, the proportion of miscarriages that occur randomly to latent-birth

17. See Barnow, Cain and Goldberger (1980) and Heckman and Robb (1985) for a discussion of the conditions under which the OLS estimator will be a consistent estimator of α.

and latent-abortion types, respectively, were known. Clearly, they are not known and must be estimated. It is important to note that as long as estimates of these two parameters do not overstate their prevalence in the miscarriage population, then the bounds presented in Section 4 will still contain the true parameter. Therefore, we estimate lower bounds on these parameters, $\tilde{\lambda}$ and $\tilde{\theta}$.

Let $Q \equiv P_{RM}/P_M$, then $\lambda = QP_B$ and $\theta = QP_A$. Let $\tilde{Q}$, $\tilde{P}_B$, and $\tilde{P}_A$ denote corresponding lower bounds on Q, P_B, and P_A, respectively. Then, lower bounds on λ and θ are given by $\tilde{\lambda} = \tilde{Q}\tilde{P}_B$ and $\tilde{\theta} = \tilde{Q}\tilde{P}_A$. In the empirical analysis presented below, we construct four alternative estimates of $\tilde{\lambda}$ and $\tilde{\theta}$ based on two estimates of $\tilde{Q}$ and two sets of estimates of $\tilde{P}_B$, and $\tilde{P}_A$.

Both methods for estimating $\tilde{Q}$ maintain that non-random miscarriage types are the least likely to report their miscarriages. That is:

Assumption 1. The rate of underreporting by non-random miscarriage types is at least as great as underreporting in the entire population of miscarriages.

Assumption 1 implies that the proportion of miscarriages that are random is greater in our sample than in the population as a whole, $Q \leqq \tilde{Q}$.[18] One estimate of $\tilde{Q}$ is derived from epidemiological studies of the causes of random miscarriages in the whole population. The largest and most comprehensive study was conducted by Kline and Stein (1987) (KS), who studied the chromosomal composition of 1922 fetuses miscarried in the New York City area. Kline, Stein and Susser (1989) (KSS), using the data collected by KS, karotype the miscarriages and find lethal chromosomal abnormalities in 38% of them.[19,20] Furthermore, KSS suggest that many times the karyotyping of fetal tissue fails to detect a chromosomal abnormality when it exists. Also many chromosomally normal miscarriages may occur for reasons uncorrelated with outcomes of interest. Therefore, our first estimate for $\tilde{Q}$ of 0·38 is extremely conservative in that it only attributes randomness to this one type of miscarriage. We label bounds using these estimates as the "$\tilde{Q} = 0{\cdot}38$" bound.

Our second estimate of $\tilde{Q}$ is based on an alternative view of the current state of epidemiological knowledge on risk factors of miscarriages. To date, there is substantial evidence that smoking or drinking during pregnancy, or having an intrauterine device in place at the time of conception, raises the rate of chromosomally normal miscarriages. but, according to KSS, the medical evidence on an association between other behavioural factors and miscarriages remains less conclusive.[21] Therefore, we presume that a miscarriage is random if it occurs to a woman who never smoked cigarettes nor drank alcohol during her pregnancy.[22] For women who smoked cigarettes, we assume that they smoked

18. This holds under two conditions: (1) Reporting of a miscarriage is independent of whether it occurred randomly. Since women typically do not know the reason for their miscarriage this assumption seems reasonable; and (2) Groups at risk for non-random miscarriage, such as woman who smoke or drink, are less likely to report their miscarriages than the population as a whole.

19. Similar rates were found in the following studies: Ohama *et al.* (1986) found 0·48; Hassold *et al.* (1980) found 0·49; Creasy *et al.* (1976) found 0·31.

20. KSS note that existing bio-medical knowledge and evidence provides more than reasonable support for assuming that chomosomal aberrant miscarriages are random, i.e. beyond the control of the women.

21. KSS note that other factors (nutrition, cocaine use, etc.) affect gestational age, the weight of babies at birth, and ultimately infant mortality, but there is no evidence that they affect the incidence of miscarriage.

22. We only have data on smoking and drinking during the year of a woman's pregnancy, and, hence, assume that if she smoked or drank in the year of her pregnancy, then she smoked or drank during her pregnancy. Also, we ignore the effect of using an IUD for two reasons: (1) It is uncommon among teen women; and (2) Our data has no information on contraceptives used while women were teens.

15 or more cigarettes a day.[23] KSS finds that these women are 60% more likely to experience a miscarriage. Similarly, for women who drank alcohol, we assume they drank 1 to 2 drinks a day during pregnancy. Harlap, Shiono and Ramcharan (1980) find that consumption at such levels leads to no more than a 100% increase in miscarriages at gestational ages where miscarriages typically occur. These risk factors lead to the following percentages of miscarriages being random: 62·5% for woman who smoked, 50% for women who drank, and 39·5% for women who both smoked and drank.[24] Given these rates and utilizing the data in the NLSY on respondent's smoking and drinking behaviour, we estimate that 84% of the miscarriages in our sample were random and label bounds using these estimates as the "$\tilde{Q}=0{\cdot}84$" bound.[25]

In estimating $\tilde{P}_B$ and $\tilde{P}_A$, we make use of two facts. First, we are implicitly estimating $\tilde{P}_{NR}$, the proportion of latent-non-random miscarriages in the random miscarriage group. If an upper bound on $\tilde{P}_{NR}$ exists, then the remainder of the random miscarriages are latent-birth and latent-abortion types and the problem reduces to decomposing this remainder between these two types. We use Assumption 1 to bound $\tilde{P}_{NR}$. Under this assumption, the non-random miscarriage types compose no more than 3% of the random miscarriage group.[26] Hence, we know that $\tilde{P}_B+\tilde{P}_A \geqq 0{\cdot}97$. To be conservative, we impose the restriction that $\tilde{P}_B+\tilde{P}_A=0{\cdot}97$, guaranteeing that non-random miscarriage types are not under-represented.

Second, note that the bounds on α are continuous in $\tilde{\lambda}$ and $\tilde{\theta}$, and $\tilde{\lambda}$ and $\tilde{\theta}$ are linear functions of $\tilde{P}_B$ and $\tilde{P}_A$, respectively. Thus, the bounds are continuous in $\tilde{P}_B$ and $\tilde{P}_A$. These two facts suggest the following strategy: Generate two sets of estimates of $\tilde{P}_B$ and $\tilde{P}_A$ subject to the restriction $\tilde{P}_B+\tilde{P}_A=0{\cdot}97$, over-representing latent-birth types in one estimate and under-representing them in the other. By continuity, we know that any decomposition of the random miscarriage group subject to the above restriction and falling between these two sets of estimates, which includes the true decomposition, will produce bounds that lie between those resulting from using these estimates. Hence, if the bounds under the two sets of estimates are qualitatively similar, then we have confidence in their robustness. Following this strategy, we computed two sets of estimates of $\tilde{P}_B$ and $\tilde{P}_A$.

To justify this first set of estimates for $\tilde{P}_B$ and $\tilde{P}_A$, we assume that underreporting is equal across the three latent types. Note that this assumption does not impose the restriction that under-reporting is independent of Y. That is:

Assumption 2. While we allow underreporting of pregnancies, underreporting is at a constant rate across latent-types.

When Assumption 2 holds, $\tilde{P}_B$ and $\tilde{P}_A$ can be estimated from the NLSY sample. The sample proportions of births and abortions among non-miscarried pregnancies, adjusted for the lower risk exposure to a miscarriage of latent-abortion types, can serve as an

23. This was done for two reasons: (1) This was the largest dose response reported by KSS; and (2) Our data has no reliable information on the quantity of cigarettes or alcohol consumed. So, we assume that all women who smoked or drank did so in large quantities, guaranteeing a lower bound on the fraction of miscarriages that are random.

24. We obtained the 39·5% figure by assuming that the effects of smoking and drinking are independent.

25. Since smoking and drinking is slightly larger among black women in our sample, 82% of miscarriages are treated as random.

26. Under the 84% random miscarriage assumptions, 16% of miscarriages are non-random. If one assumes that 14% of pregnancies end in a miscarriage, then 2·25% of pregnancies end in a non-random miscarriage. From vital statistics 45% of pregnancies end in births and 41% end in abortions. Adjusting for risk exposure of both abortions (1/3) and non-random miscarriages (1/2), the percentage of non-random miscarriage is 1·8.

estimate of $\tilde{P}_B$ and $\tilde{P}_A$.[27] Using data on the number of births and abortions in our sample, we estimate that latent-birth types account for 92% of miscarriages for blacks and 85% for non-blacks. Since Assumption 2 implies that latent-birth types report all of their miscarriages, these estimates probably over-represent latent-birth types. We label these estimates of $\tilde{P}_B$ and $\tilde{P}_A$ as "Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY Data," since no adjustment to the NLSY data on pregnancy resolutions is needed if Assumption 2 holds.

We construct a second set of estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on the number of births and abortions in the population of teenage women as recorded in data from U.S. Vital Statistics and the Alan Guttmacher Institute. From this data, we estimate that latent-birth types account for 79% of miscarriages for blacks and 74% for non-blacks.[28] If latent-birth types report their miscarriages more often than the population as a whole, then these estimates under-represent the percentage of latent-birth types in our sample. We label the resulting estimates as "Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on Auxiliary Data." Although these two sets of estimates should be viewed as alternatives, when the exclusion restriction for latent-abortion types is not imposed the later estimates are more conservative since they produce a smaller estimate of the proportion of latent-birth types.

8. DATA

We use the National Longitudinal Survey of Youths (NLSY). The NLSY is an annual survey originating in 1979 of a nationally representative sample of youths who were 14 to 21 years old in 1979. In 1983 a retrospective pregnancy history was administered and thereafter a pregnancy history was administered approximately every 2 years. In addition, a self-administered questionnaire was administered in 1984 which attempted to record all abortions not recorded in the NLSY open interviews prior to that date.

We use for our analysis the 980 women in the NLSY who reported a pregnancy prior to their 18th birthday, including those women in the oversamples of blacks and Hispanics. Of those pregnancies 727 resulted in births, 185 terminated in an abortion and 68 ended in a miscarriage. These numbers imply that 74% of non-miscarried pregnancies are brought to term in our sample. However, the corresponding number for the population as a whole is only 52%.[29] Hence, abortions are almost certainly underreported. Miscarriages may also be underreported, but it is difficult to determine the degree of this problem since there are no data sources, comparable to the AGI data, with which to verify their accuracy.[30] This underreporting can potentially bias all of our estimators. Additionally, our data contains very limited information on any behaviours that the epidemiological literature suggest effects miscarriage rates.

To get a feel for the data, Table 1 presents the sample means for the four outcomes of interest by pregnancy resolution and race. These measures are the following: attainment

27. If the distribution of gestational ages of miscarriages and abortions were identical, then exactly half of all latent-abortion types who would eventually have a miscarriage preempt the miscarriage with an abortion. Since the abortion distribution is skewed toward earlier months than the miscarriage distribution, latent-abortion types are exposed to approximately one-third of the risk of experiencing a miscarriage compared to latent-birth types.

28. From U.S. Vital Statistics data, we observe the number of births to women under age 18 and from AGI data we observe the number of abortions in this age group.

29. All sample averages are adjusted by the NLSY weights to reflect the population of women 14 to 21 in 1979. In 1984, Vital Statistics data recorded 300,207 births to women under age 18 and AGI reported 273,068 abortions to women under age 18.

30. Jones and Forrest (1992) compare the responses on pregnancy in the self-administered questionnaire (SAQ) to the responses in the open survey and find that it is very rare that a pregnancy was reported as resolving in a miscarriage in the open survey and then reported resolving in an abortion in the SAQ.

of a high school diploma, attainment of a general equivalency diploma (GED), accumulation of labour market experience as measured by hours worked and the value of human capital as measured by annual earnings. Also recorded at the bottom of this table are OLS and IV estimates of the effect of teenage childbearing which do not control for any Xs. For non-blacks, the IV and OLS estimates for hours of work and earnings are quite different and, in some cases, statistically so. In particular, the IV estimates for these two outcomes imply that teenage childbearing raises hours worked and earnings, while the OLS estimates indicate that teenage childbearing lowers these outcomes.[31] We note that the IV results are novel and quite different from those found in previous studies. Precisely because they are at odds with the literature, it is important to establish whether having used a contaminated instrument has led to these results.

9. RESULTS

In this section, we present our empirical estimates of alternative bounds on the causal effect of teenage childbearing for the outcomes discussed in Section 8. We start with an analysis of the exclusion restriction on latent-abortion types which is imposed in both the IV estimator and the fourth set of bounds presented in Section 5. Then, we use these bounds to examine each of the outcomes noted above and to test the validity of the IV and OLS estimators for these outcomes. From this point onward, the OLS and IV estimates of α control for the following background and demographic characteristics: the women's Armed Forces Qualifying Test (AFQT) score, her family's income in 1978, her mother's and father's education, whether she lived in an intact family at age 14, whether she lived in a female headed household at age 14, and whether her family was on welfare in 1978.

Before we begin, it is useful to discuss a few findings that will greatly facilitate the exposition of the results. First, the results for black women are not as informative as those for non-blacks. In particular, we are rarely able to reject any of the hypothesis considered in Section 6. As such, we will concentrate on non-blacks and note when the results for blacks are significant. Second, the estimates of the HM bounds under the two sets of assumption used to estimate $\tilde{P}_B$ and $\tilde{P}_A$ are qualitatively similar for all of the outcomes considered. This result can be seen by examining Tables 2 through 5. These tables contain the estimates of the various bounds for α that were described in Sections 4 and 5, as well as P-Values for the various tests that were discussed in Section 5 for the four measures of maternal attainment mentioned above. In the discussion that follows, we shall focus our attention on the estimates of bounds for non-blacks based on the estimates of $\tilde{P}_B$ and $\tilde{P}_A$ from auxiliary data and $\tilde{Q}=0{\cdot}84$.

9.1. *The exclusion restriction on latent-abortion types*

Imposing Condition 2′(ii), the exclusion restriction on latent-abortion types, greatly tightens the bounds, at times by as much as 70%. Given the amount of information gained by invoking this assumption, it is clear that we would like to base our analysis on the bounds that use it. However, the validity of this assumption must be addressed first.

To examine the validity of the exclusion restriction, we conducted tests of whether the upper (lower) bound on α when the restriction is imposed is lower (higher) than the

31. We note that the finding in Table 1—that the IV estimates of the effects of teenage childbearing on hours worked and earnings for non-black women are positive—is not a particularly fragile one. To see why, note that the mean of the miscarriage group is below the mean of the birth group.

TABLE 1

Mean outcomes by race and pregnancy resolution status and estimates of α[a]

Pregnancy resolution status	Black women: Has high school diploma at age 25	Black women: Has GED at age 25	Black women: Annual hours of work at age 27	Black women: Annual labour market earnings at age 27	Non-black women: Has high school diploma at age 25	Non-black women: Has GED at age 25	Non-black women: Annual hours of work at age 27	Non-black women: Annual labour market earnings at age 27
Miscarriage	0·626	0·072	999·98	6053·30	0·515	0·088	660·85	3857·00
	(0·348)	(0·186)	(738·80)	(5129·10)	(0·322)	(0·183)	(542·34)	(3919·10)
Not miscarriage:	0·499	0·152	934·25	5881·10	0·469	0·204	1097·30	8193·20
	(0·036)	(0·026)	(70·19)	(544·03)	(0·039)	(0·032)	(76·38)	(733·62)
Birth	0·461	0·157	890·89	5288·30	0·382	0·217	977·80	6345·00
	(0·027)	(0·020)	(51·47)	(381·84)	(0·032)	(0·027)	(60·87)	(508·08)
Abortion	0·732	0·122	1197·60	9464·30	0·658	0·175	1355·50	12092·00
	(0·162)	(0·120)	(386·24)	(3325·00)	(0·084)	(0·067)	(179·05)	(1932·80)
Not birth	0·698	0·106	1133·90	8336·10	0·632	0·159	1225·30	10584·00
	(0·167)	(0·112)	(381·36)	(3127·60)	(0·106)	(0·080)	(233·38)	(2353·60)
OLS estimate of α	−0·236	0·051	−243·06	−3047·80	−0·250	0·058	−247·54	−4238·60
	(0·169)	(0·114)	(384·82)	(3150·80)	(0·111)	(0·085)	(231·53)	(2407·80)
IV estimate of α	−0·147	0·093	−76·55	−200·71	−0·068	0·169	638·51	6391·80
	(0·406)	(0·218)	(863·31)	(6002·30)	(0·473)	(0·269)	(797·08)	(5823·40)

[a] Standard errors in parentheses.

upper (lower) bound obtained when it is not.[32] Note that these tests assess the validity of the joint hypothesis that the exclusion restrict is true and that $\tilde{\theta}$ is a lower bound on θ. We present the P-Values associated with these tests in Table 6 for the four outcomes considered in this paper. While the hypothesis cannot be rejected for most of the outcomes, we do find evidence against its validity when estimating the effects on attaining a high school diploma at age 25 for both demographic groups and for labour market earnings at age 27 among non-black women. For the latter outcome, the test of the null hypothesis that the upper bound on α for earnings, when the assumption is invoked, lies below the corresponding upper bound when it is not imposed is decisively rejected with a P-Value of 0·001. Note that this rejection occurs when $\tilde{Q}=0{\cdot}38$; when $\tilde{Q}=0{\cdot}84$ we fail to reject.

To explore the robustness of the rejection of the exclusion restriction, we tested its validity at other ages. While not reported here, we tested the exclusion restriction for all possible combinations of the ages between 18 and 30, our four outcomes, black and non-black women and the alternative estimates of $\tilde{\lambda}$ and $\tilde{\theta}$. This resulted in a total of 832 tests.[33] Across all of these tests, the assumption was rejected in 21 instances when the level of significance was 0·05 and in 38 instances when it was 0·10. Note that this corresponds to rejecting the null in 2·5% and 4·6% of the tests at 95% and 90% confidence levels, respectively. Furthermore, examination of the results revealed that 21 of the 38 rejections occurred when estimating the effect of teenage childbearing on the likelihood of attaining a high school diploma. The remaining 17 rejections showed no discernible pattern with respect to outcomes, ages or demographic groups. We conclude from these tests that there is strong evidence that imposing the exclusion restriction on latent-abortion types is inappropriate in the context of a high school diploma, but that there is not strong evidence that it is violated when estimating the effects of teenage childbearing for the other outcomes. Note that while we rejected this assumption when estimating α for earnings among non-black women at age 27, we fail to reject this assumption for earnings at any other age for black or non-black women. Therefore, in the remainder of the paper, we concentrate on bounds and tests of propositions using bounds on α that impose the exclusion restriction on latent-abortion types, except when analysing the attainment of a high school degree.

9.2. *Attainment of a High School Degree*

Table 2 presents the estimated bounds on the effect of teenage childbearing for whether a woman had obtained a high school diploma by age 25. In columns 1 and 2, we present estimates of the bounds for α which impose none of the standard IV assumptions. Note that since having attained a high school diploma is a dichotomous outcome, we do not present separate estimates for the E-BO and HM bounds. It can be shown that the E-BO and HM bounds are equal if the outcome is dichotomous and no additional restrictions are imposed. (Note that this equivalence of the E-BO and HM bounds is also true in the case of the GED outcome.)

The NE-BO bounds for non-black women are $-0{\cdot}626$ and $0{\cdot}374$. These are relatively uninformative, but can be substantially improved by exploiting the natural experiment resulting from random miscarriages. Using the natural experiment, the largest HM lower

32. Obviously, testing these two propositions could be conducted *jointly*. The form of such a test would be the same as the tests of inequality constraints discussed in Section 6. While more appropriate that the two marginal ones we perform, implementing the joint test is computationally burdensome. In particular, calculation of the weights $w(K, k, \Sigma)$ for the test statistics in (28) do not have closed form solutions for values of k (and K) greater than 3. See Wolak (1989) for a discussion of the issue.

33. These tests are not independent.

TABLE 2

Non-parametric bounds on and tests of effects of teenage births on probability of woman receiving a high school diploma

	Non-parametric bounds:				P-values for tests of "Signs" of teen childbearing effects:						P-values for tests of whether parametric estimator falls within bounds:			
	Imposing no restrictions		Imposing assumption 6C		Imposing no restrictions		Imposing assumption 6C		Point estimates by estimation method:		Imposing no restrictions		Imposing assumption 6C	
Type of bound	A_{sL} (1)	A_{sU} (2)	A_{sL} (3)	A_{sU} (4)	$H_0: A_{sL} \leqq 0$ (5)	$H_0: A_{sU} \geqq 0$ (6)	$H_0: A_{sL} \leqq 0$ (7)	$H_0: A_{sU} \geqq 0$ (8)	OLS[a] (9)	IV[a] (10)	OLS (11)	IV (12)	OLS (13)	IV (14)
Black women:														
NE-BO bounds	−0·545	0·455	NA	NA	1·000	1·000	NA	NA	−0·157 (0·059)	−0·098 (0·102)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	=HM	=HM	−0·545	0·442	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−0·545	0·172	−0·545	0·168	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	=HM	=HM	−0·317	−0·047	=HM	=HM	1·000	0·350			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}82$	−0·278	−0·032	−0·238	−0·047	1·000	0·398	1·000	0·350			1·000	1·000	1·000	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds: $\tilde{Q}=0{\cdot}38$	=HM	=HM	−0·545	0·445	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−0·545	0·149	−0·545	0·149	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	=HM	=HM	−0·327	−0·013	=HM	=HM	1·000	0·465			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}82$	−0·345	0·053	−0·234	−0·013	1·000	1·000	1·000	0·464			1·000	1·000	1·000	1·000
Non-black women:														
NE-BO bounds	−0·626	0·374	NA	NA	1·000	1·000	NA	NA	−0·178 (0·057)	−0·127 (0·100)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	=HM	=HM	−0·626	0·359	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−0·578	0·281	−0·492	0·280	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	=HM	=HM	−0·198	0·061	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}84$	−0·251	0·113	−0·165	0·061	1·000	1·000	1·000	1·000			1·000	1·000	0·873	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	=HM	=HM	−0·626	0·361	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−0·568	0·285	−0·520	0·285	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	=HM	=HM	−0·187	0·109	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}84$	−0·298	0·120	−0·151	0·107	1·000	1·000	1·000	1·000			1·000	1·000	0·774	1·000

[a] Standard errors in parentheses.

bound is $-0{\cdot}298$, which is 48% larger than the NE-BO lower bound for this group, and the smallest HM upper bound is $0{\cdot}120$, which is 68% smaller than the NE-BO upper bound. While exploiting the existence of random miscarriages tighten the bounds, it is clear that the HM bounds do not enable us to determine whether the effect is positive or negative. Recall that to reject that α is negative amounts to rejecting the null hypothesis that $A_{sL} \leqq 0$ and to reject that α is positive amounts to rejecting the null that $A_{sU} \geqq 0$. As is clear from the P-Values in columns 5 and 6, we are not able to reject either of these hypotheses.

Now consider the OLS and IV estimates of α which are presented in columns 9 and 10, respectively. The OLS estimate is $-0{\cdot}178$ for non-black women and significantly different from zero, indicating that the effect of teenage motherhood on the chances of obtaining a high school diploma are large and negative. In contrast, the corresponding IV estimate for this effect is $-0{\cdot}127$ and not significantly different from zero. Columns 11 through 14 of Table 2 present P-Values for the tests that the OLS and IV estimates fall inside each set of bounds. We *cannot* reject the hypothesis that the OLS estimator falls within our bounds. However, it does appear that the inference one would draw from it, namely, that the effect of teenage childbearing is large and negative, is corroborated by neither the IV estimate nor the non-parametric bounds estimates.

9.3. *Attainment of a GED*

While our bounds on the effect of teenage childbearing make little headway in resolving the direction or magnitude of the effect on high school completion, they are more informative concerning the impact on the other measures of human capital accumulation. Table 3 presents the results on the effect of early motherhood on the woman's likelihood of attaining a GED. The bounds of α for this outcome indicate that teenage childbearing *raises* the proportion of non-black teen mothers who receive a GED between $0{\cdot}112$ and $0{\cdot}213$. The P-Value associated with the test that α is negative (i.e. testing H_0: $A_{sL} \leqq 0$) is $0{\cdot}200$ for non-black women ($0{\cdot}134$ for black women). While this value may be too large to reject that teenage childbearing lowers the probability of completing a GED, one can clearly reject that early childbearing has a large and negative effect on the likelihood of a teen mother attaining a GED. Furthermore, the OLS and IV estimates tend to corroborate the conclusion that teen mothers have a higher probability of attaining a GED than they would have if they had delayed their childbearing.

9.4. *Hours worked*

Table 4 presents estimates and tests for the effects of teenage childbearing on a mother's annual hours worked at age 27. Comparing the NE-BO bounds with those which make use of miscarriages, we again find that exploiting this natural experiment can substantially tighten the bounds on α. The HM bounds show that the effect of teenage childbearing for non-blacks is to raise hours worked between 420 and 932 hours, on average (see columns 3 and 4). The P-Value of the test that the lower bound is negative is $0{\cdot}044$ (in column 7). Hence, we can reject that the effect is negative with a relatively high level of statistical confidence. While the IV estimate lies within these bounds, the OLS estimate lies well outside of them. Furthermore, the P-Value of the test of the hypothesis that the OLS estimate lies within the bounds is $0{\cdot}001$ (see column 13), a strong rejection. Clearly, even though the OLS estimate conditions on observables that are highly correlated with hours worked (particularly AFQT), simply controlling for these variables does not appear

TABLE 3

Non-parametric bounds on and tests of effects of teenage births on probability of woman receiving a general equivalency diploma

Type of bound	Non-parametric bounds:				P-values for tests of "Signs" of teen childbearing effects:				Point estimates by estimation method:		P-values for tests of whether parametric estimator falls within bounds:			
	Imposing no restrictions		Imposing assumption 6C		Imposing no restrictions		Imposing assumption 6C				Imposing no restrictions		Imposing assumption 6C	
	A_{sL} (1)	A_{sU} (2)	A_{sL} (3)	A_{sU} (4)	$H_0: A_{sL} \leqq 0$ (5)	$H_0: A_{sL} \geqq 0$ (6)	$H_0: A_{sL} \leqq 0$ (7)	$H_0: A_{sU} \geqq 0$ (8)	OLS[a] (9)	IV[a] (10)	OLS (11)	IV (12)	OLS (13)	IV (14)
Black women:														
NE-BO bounds	−0·845	0·155	NA	NA	1·000	1·000	NA	NA	0·057 (0·040)	0·071 (0·059)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0·38$	=HM	=HM	−0·052	0·155	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0·38$	−0·059	0·155	−0·052	0·155	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0·82$	=HM	=HM	0·063	0·155	=HM	=HM	0·186	1·000			=HM	=HM	0·882	1·000
HM bounds; $\tilde{Q}=0·82$	0·056	0·155	0·063	0·155	0·214	1·000	0·186	1·000			1·000	1·000	0·882	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0·38$	=HM	=HM	−0·064	0·155	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0·38$	−0·094	0·155	−0·064	0·155	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0·82$	=HM	=HM	0·069	0·155	=HM	=HM	0·200	1·000			=HM	=HM	0·812	1·000
HM bounds; $\tilde{Q}=0·82$	0·040	0·155	0·069	0·155	0·313	1·000	0·200	1·000			1·000	1·000	0·812	1·000
Non-black women:														
NE-BO bounds	−0·787	0·213	NA	NA	1·000	1·000	NA	NA	0·091 (0·048)	0·140 (0·084)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0·38$	=HM	=HM	−0·062	0·213	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0·38$	−0·087	0·213	−0·062	0·213	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds: $\tilde{Q}=0·84$	=HM	=HM	0·102	0·213	=HM	=HM	0·123	1·000			=HM	=HM	0·848	1·000
HM bounds; $\tilde{Q}=0·84$	0·077	0·213	0·102	1·213	0·188	1·000	0·123	1·000			1·000	1·000	0·848	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0·38$	=HM	=HM	−0·077	0·213	=HM	=HM	1·000	1·000			=HM	=HM	1·000	1·000
HM bounds; $\tilde{Q}=0·38$	−0·132	0·213	−0·077	0·213	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0·84$	=HM	=HM	0·112	0·213	=HM	=HM	0·134	1·000			=HM	=HM	0·761	1·000
HM bounds; $\tilde{Q}=0·84$	0·057	0·213	0·112	0·213	0·282	1·000	0·134	1·000			1·000	1·000	0·761	1·000

[a] Standard errors in parenthesis.

to eliminate the selection bias associated with estimating the causal effect of teenage childbearing on a teen mother's subsequent labour supply.

The results in Table 4 are only for age 27. In Figure 1, we graph the estimates of the HM bounds as well as the OLS and IV estimates for α associated with annual hours worked for each age from 18 to 30 for non-black women. What Figure 1 vividly shows is that conclusions reached about α, the bounds and the point estimates based on the data for age 27 are not exceptions. At all ages but 29 and 30, the OLS estimate is less than the estimate of the HM lower bound, while the IV estimate lies within the bounds at all ages. Furthermore, the estimate of the HM lower bound lies above zero at almost all ages beyond 19. Thus, the conclusion of HMcS that teen mothers worked more hours during their early adulthood compared to the hours they would have worked if they had delayed their childbearing appears to be a robust finding, even though their instrument may be contaminated.

9.5. *Earnings*

Table 5 presents estimates and tests for the effects of teenage childbearing on a teen mother's annual labour market earnings at age 27. Many of the conclusions drawn for annual hours of work also hold for labour market earnings. The estimates of the HM bounds in columns 3 and 4 indicate that teenage childbearing *raises* earnings from \$4565 to \$6043, on average, and we can reject that teenage childbearing causes a decrease in earnings at age 27 (P-Value = 0·004). While the OLS estimate indicates that teenage childbearing lower a mother's annual earnings by \$3016, this estimator can be rejected at any conventional level of significance even when the exclusion restriction on latent-abortion types is not invoked (see columns 11 and 13). The IV estimate of α for labour market earnings of \$4147 falls below the estimate of the HM lower bound. However, we cannot reject the null hypothesis that the true HM bounds contain the IV estimate (P-Value = 0·345). Thus, the evidence derived with these non-parametric bounds is that the effect of teenage childbearing on a woman's annual earnings is positive, consistent with the results found by HMcS.

In Figure 2, we graph the estimates of the HM bounds as well as the OLS and IV estimates for the effect of teenage childbearing on earnings at each age from 18 to 30 for non-black women. Again, the graph indicates that the conclusions drawn about α at age 27 hold at most ages.

10. CONCLUSION

We conclude this paper by discussing the substantive implications of what we have been able to learn about the effects of teenage motherhood on several measures of a teen mother's subsequent human capital acquisition by exploiting the natural experiment which miscarriages provides. First, the bounds indicate we cannot unambiguously determine whether teenage childbearing decreases the propensity to receive a high school diploma. However, the bounds imply that this effect is much smaller than reported elsewhere in the literature. Second, we find that relaxing many (but not all) of the traditional IV restrictions does not qualitatively change the findings of the HMcS study with respect to annual hours of work or labour market earnings. In particular, their conclusions based on a standard IV estimator are quite robust, even after accounting for the (potentially) contaminated nature of their instrument. Third, we confirm that the use of standard OLS methods is simply not effective in estimating the causal effects of teenage childbearing. Inferences

TABLE 4

Non-parametric bounds on and tests of effects of teenage births on woman's annual hours of work

	Non-parametric bounds:				P-values for tests of "Signs" of teen childbearing effects:						P-values for tests of whether parametric estimator falls within bounds:			
	Imposing no restrictions		Imposing assumption 6C		Imposing no restrictions		Imposing assumption 6C		Point estimates by estimation method:		Imposing no restrictions		Imposing assumption 6C	
	A_{sL}	A_{sU}	A_{sL}	A_{sU}	$H_0: A_{sL} \leqq 0$	$H_0: A_{sU} \geqq 0$	$H_0: A_{sL} \leqq 0$	$H_0: A_{sU} \geqq 0$	OLS[a]	IV[a]	OLS	IV	OLS	IV
Type of bound	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Black women:														
NE-BO bounds	−3,120	880	NA	NA	1·000	1·000	1·000	1·000	−143·50 (125·26)	−46·55 (214·91)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−1,984	880	−1·921	880	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−1,455	880	−1,407	880	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	−447	855	−384	701	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}82$	−443	322	−379	267	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−2,454	880	−2,188	880	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−1,731	880	−1,590	880	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	−666	880	−399	863	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}82$	−622	539	−393	360	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
Non-black women:														
NE-BO bounds	−3,026	974	NA	NA	1·000	1·000	1·000	1·000	−141·00 (107·08)	506·66 (200·46)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−879	974	−694	974	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−785	974	−598	974	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	136	974	321	973	0·267	1·000	0·070	1·000			0·043	1·000	0·001	1·000
HM bounds; $\tilde{Q}=0{\cdot}84$	136	813	321	800	0·264	1·000	0·069	1·000			0·041	1·000	0·001	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−1,155	974	−746	974	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−954	933	−637	933	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	11	974	420	974	0·482	1·000	0·045	1·000			0·332	1·000	0·001	1·000
HM bounds; $\tilde{Q}=0{\cdot}84$	11	937	420	932	0·481	1·000	0·044	1·000			0·316	1·000	0·001	1·000

[a] Standard errors in parentheses.

TABLE 5

Non-parametric bounds on and tests of effects of teenage births on woman's annual labour market earnings

	Non-parametric bounds:				P-values for tests of "Signs" of teen childbearing effects:				Point estimates by estimation method:		*P*-values for tests of whether parametric estimator falls within bounds:			
	Imposing no restrictions		Imposing assumption 6C		Imposing no restrictions		Imposing assumption 6C				Imposing no restrictions		Imposing assumption 6C	
Type of bound	A_{sL} (1)	A_{sU} (2)	A_{sL} (3)	A_{sU} (4)	H_0: $A_{sL} \leqq 0$ (5)	H_0: $A_{sU} \geqq 0$ (6)	H_0: $A_{sL} \leqq 0$ (7)	H_0: $A_{sU} \geqq 0$ (8)	OLS[a] (9)	IV[a] (10)	OLS (11)	IV (12)	OLS (13)	IV (14)
Black women:														
NE-BO bounds	−60,120	5,130	NA	NA	1·000	1·000	1·000	1·000	−1838·0 (1030·8)	54·0 (1622·6)	1·000	1·000	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−12,210	5,130	−11,696	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−9,343	5,130	−8,912	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	−2,906	5,130	−2,392	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM Bounds; $\tilde{Q}=0{\cdot}82$	−2,906	2,078	−2,392	2,025	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−15,063	5,130	−12,908	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−11,203	5,130	−9,666	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}82$	−4,228	5,130	−2,073	5,130	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}82$	−4,165	3,205	−2,073	3,072	1·000	1·000	1·000	1·000			1·000	1·000	1·000	1·000
Non-black women:														
NE-BO bounds	−58,939	6,312	NA	NA	1·000	1·000	1·000	1·000	−3016·0 (1000·9)	4147·1 (1416·7)	1·000	0·607	NA	NA
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−3,966	6,312	−2,334	6,312	1·000	1·000	1·000	1·000			1·000	1·000	0·756	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−3,645	6,198	−2,125	6,198	1·000	1·000	1·000	1·000			1·000	1·000	0·636	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	1,662	6,312	3,294	6,312	0·134	1·000	0·015	1·000			0·000	1·000	0·000	1·000
HM bounds; $\tilde{Q}=0{\cdot}84$	1,662	5,830	3,294	5,830	0·132	1·000	0·015	1·000			0·000	1·000	0·000	1·000
Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data:														
E-BO bounds; $\tilde{Q}=0{\cdot}38$	−5,493	6,312	−1,900	6,312	1·000	1·000	1·000	1·000			1·000	1·000	0·662	1·000
HM bounds; $\tilde{Q}=0{\cdot}38$	−5,102	5,443	−1,660	5,443	1·000	1·000	1·000	1·000			1·000	1·000	0·535	1·000
E-BO bounds; $\tilde{Q}=0{\cdot}84$	971	6,312	4,565	6,312	0·284	1·000	0·004	1·000			0·001	1·000	0·000	0·344
HM bounds; $\tilde{Q}=0{\cdot}84$	971	6,043	4,565	6,043	0·280	1·000	0·004	1·000			0·001	1·000	0·000	0·345

[a] Standard errors in parentheses.

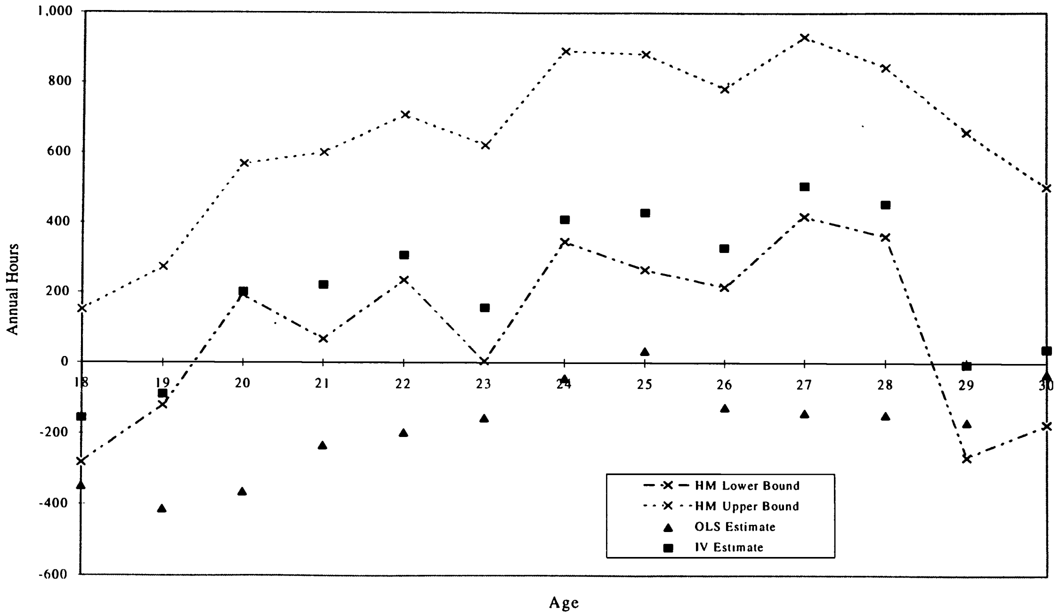

FIGURE 1

HM bounds, OLS and IV estimates of the effects of teenage childbearing on women's annual hours of work for non-black women (condition 2′(ii) imposed and proportion of random miscarriages = 0·84)

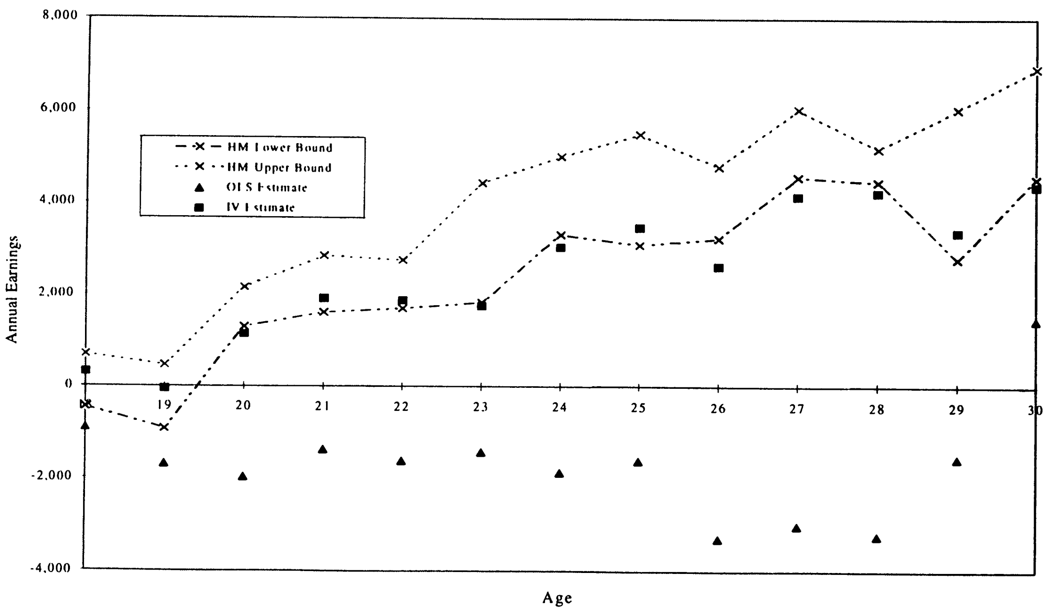

FIGURE 2

HM bounds, OLS and IV estimates of the effects of teenage childbearing on women's annual labour market earnings for non-black women (condition 2′(ii) imposed and proportion of random miscarriages = 0·84)

TABLE 6

P-values for the tests of condition 2′.(ii)

	Black women				Non-black women			
	Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data P-values for tests of:		Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data P-values for Tests of:		Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on NLSY data P-values for tests of:		Estimates of $\tilde{P}_B$ and $\tilde{P}_A$ based on auxiliary data P-values for tests of:	
Outcome[a]	H_0: $A_{4L} \geqq A_{3L}$	H_0: $A_{4U} \leqq A_{3U}$	H_0: $A_{4L} \geqq A_{3L}$	H_0: $A_{4U} \leqq A_{3U}$	H_0: $A_{4L} \geqq A_{3L}$	H_0: $A_{4U} \leqq A_{3U}$	H_0: $A_{4L} \geqq A_{3L}$	H_0: $A_{4U} \leqq A_{3U}$
Tests when $\tilde{Q}=0{\cdot}38$								
High School Diploma	1·000	1·000	1·000	0·103	1·000	0·255	1·000	0·106
GED	1·000	1·000	1·000	1·000	1·000	1·000	1·000	1·000
Annual Hours Worked	1·000	1·000	1·000	1·000	1·000	1·000	1·000	0·142
Annual Earnings	1·000	1·000	1·000	1·000	1·000	0·422	1·000	0·001
Tests when $\tilde{Q}=0{\cdot}82$ *or* 0·84								
High School Diploma	1·000	1·000	1·000	1·000	1·000	1·000	1·000	1·000
GED	1·000	1·000	1·000	1·000	1·000	1·000	1·000	1·000
Annual Hours Worked	1·000	1·000	1·000	1·000	1·000	1·000	1·000	0·484
Earnings	1·000	1·000	1·000	1·000	1·000	0·209	1·000	0·285

[a] Percent of random miscarriages in parentheses.

about the causal effects for the outcomes we have analysed based on OLS estimators are quite misleading. While the evidence on this is much weaker for black teenage mothers, it is quite strong and consistent for non-black women.

This study demonstrates that using the information available in natural experiments is capable of resolving conflicts about causal effects, even when the natural experiment may not be rich enough to meet the requirements of a standard IV estimator, i.e. when one's instrument is contaminated. To be sure, the value of such experiments need not always be conclusive, as evidenced by our findings for the effects of teenage childbearing on a mother's likelihood of obtaining a high school diploma. Nonetheless, the bounds we set out in this paper provide a very promising avenue for exploiting the scope for identification which such experiments can provide while not clouding this promise by invoking extraneous, and often inappropriate, assumptions.

Acknowledgements. We wish to thank the editor, two anonymous referees, Alex Cavallo, James Heckman, Charles Manski, Tomas Philipson, Jacob Klerman, Nick Ronan, and other participants in seminars at CEMFI, UCLA, Carnegie-Mellon, Columbia, Queens and Harvard Universities and the Universities of Virginia and Chicago for helpful comments on an earlier version of this paper. We also wish to thank Joel Horowitz and Hidehiko Ichimura for useful discussions concerning the estimation and testing methods undertaken in the current paper. This research was supported by a grant from the National Institute of Child Health and Human Development.

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