

The effects of birth inputs on birthweight:
evidence from quantile estimation on panel data

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ABSTRACT

Unobserved heterogeneity among childbearing women makes it difficult to isolate the causal effects of smoking and prenatal care on birth outcomes (such as birthweight). Whether or not a mother smokes, for instance, is likely to be correlated with unobserved characteristics of the mother. This paper controls for such unobserved heterogeneity by using state-level panel data on maternally linked births. A quantile-estimation approach, motivated by a correlated random-effects model, is used in order to estimate the effects of smoking and other observables (number of prenatal-care visits, years of education, etc.) on the entire birthweight distribution.

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1 Introduction

Adverse birth outcomes have been found to result in large economic costs, in the form of both direct medical costs and long-term developmental consequences. It is not surprising, then, that the public-health community has focused efforts on prenatal-care improvements (e.g., through smoking cessation, alcohol-intake reduction, and/or better nutrition) that are thought to improve birth outcomes. Birthweight has served as a leading indicator of infant health, with “low birthweight” (LBW) infants classified as those weighing less than 2500 grams at birth. Observable measures of poor prenatal care, such as smoking, have strong negative associations with birthweight. For instance, according to a report by the Surgeon General, mothers who smoke during pregnancy have babies that, on average, weigh 250 grams less (Centers for Disease Control and Prevention (2001)).

The direct medical costs of low birthweight are quite high. Based upon hospital-discharge data from New York and New Jersey, Almond et. al. (2005) report that the hospital costs for newborns peaks at around \$150,000 (in 2000 dollars) for infants that weigh 800 grams; the costs remain quite high for all “low birthweight” outcomes, with an average cost of around \$15,000 for infants that weigh 2000 grams. The infant-mortality rate also increases at lower birthweights.

Other research has examined the long-term effects of low birthweight on cognitive development, educational outcomes, and labor-market outcomes. LBW babies have developmental problems in cognition, attention, and neuromotor functioning that persist until adolescence (Hack et. al. (1995)). LBW babies are more likely to delay entry into kindergarten, repeat a grade in school, and attend special-education classes (Corman (1995); Corman and Chaikind (1998)). LBW babies are also more likely to have inferior labor-market outcomes, being more likely to be unemployed and earn lower wages (Behrman and Rosenzweig (2004); Case et. al. (2005); Currie and Hyson (1999)).

Although it has received less attention in the economics literature, high-birthweight outcomes can also represent adverse outcomes. For instance, babies weighing more than 4000 grams (classified as high birthweight (HBW)) and especially those weighing more than 4500 grams (classified as very high birthweight (VHBW)) are more likely to require cesarean-section births, have higher infant mortality rates, and develop health problems later in life.

A difficulty in evaluating initiatives aimed at improving birth outcomes is to accurately estimate the causal effects of prenatal activities on these birth outcomes. Unobserved heterogeneity among childbearing women makes it difficult to isolate causal effects of various determinants of birth outcomes. Whether or not a mother smokes, for instance, is likely to be correlated with unobserved characteristics of the mother. To deal with this difficulty, various studies have used an instrumental-variable approach to estimate the effects of smoking (Evans and Ringel (1999); Permutt and Hebel (1989)), prenatal care (Currie and Gruber (1996); Evans and Lien (2005);

Joyce (1999)), and air pollution (Chay and Greenstone (2003a, 2003b)) on birth outcomes.

Another approach has been to utilize panel data (i.e., several births for each mother) to identify these effects from changes in prenatal behavior or maternal characteristics between pregnancies (Abrevaya (2006); Currie and Moretti (2002); Rosenzweig and Wolpin (1991); Royer (2004)). One concern with the panel-data identification strategy is the presence of “feedback effects,” specifically that prenatal care and smoking in later pregnancies may be correlated with birth outcomes in earlier pregnancies. Royer (2004) provides an explicit estimation strategy to deal with such feedback effects (using data on at least three births per mother). Abrevaya (2006) shows that feedback effects are likely to cause the estimated (negative) smoking effect to be too large in magnitude.

Since the costs associated with birthweight have been found to exist primarily at the low end of the birthweight distribution (with costs increasing significantly at the very low end), most studies have estimated the effects of birth inputs on the fraction of births below various thresholds (e.g., 2500 grams for LBW and 1500 grams for “very low birthweight”). As an alternative, this paper considers a quantile-regression approach to estimating the effects of birth inputs on birthweight, so it is useful to compare the two approaches. The threshold-crossing approach fixes a common *unconditional* threshold for the entire sample, whereas the quantile-regression approach focuses upon particular *conditional* quantiles of the birthweight distribution. Denoting birthweight by bw and a birth input vector by x , a probit-based threshold-crossing model for LBW outcomes would be $\Pr(bw < 2500|x) = \Phi(x'\gamma)$. For each x , there is a conditional probability of the LBW outcome (bw below the common threshold) and estimates of γ can be used to infer the marginal effects of the birth inputs upon these conditional probabilities. For the quantile approach, a simple (linear) model for, say, the 5% conditional quantile would be $Q_{5\%}(bw|x) = x'\beta$. The value of the conditional quantile $Q_{5\%}(bw|x)$ may be below the LBW threshold of 2500 grams for some x values and above it for other x values. The estimated marginal effects (inferred from the estimates of β) would indicate how the 5% conditional quantile would be affected at all x values. These effects are not directly comparable to the probit-based effects.

For the question of economic costs, both the probit approach and quantile approach have drawbacks: (i) the probit approach is inherently discontinuous and offers only predictions of LBW vs. non-LBW outcomes, and (ii) the quantile approach combines predictions from extremely adverse x values (lower $Q_{5\%}(bw|x)$), where the costs are higher, and less adverse x values (higher $Q_{5\%}(bw|x)$), where the costs are lower. For the question of what causes LBW outcomes, the simple probit-based approach is certainly sufficient. The quantile approach, however, provides a convenient method for determining how birth inputs affect birthweight at different parts of the distribution. The closest analogy with the threshold-crossing approach would be to continuously alter the threshold value and estimate a series of probit models. Given the different aspects of the birthweight

distribution being modeled and estimated by the two approaches, our view is that these approaches should be viewed as complements to each other rather than substitutes.

A recent literature on estimation of quantile treatment effects, including Abadie, Angrist, and Imbens (2002) and Bitler, Gelbach, and Hoynes (2006), has argued that traditional estimation of average (mean) treatment effects may miss important causal impacts. Specifically, an average treatment effect inherently combines the magnitudes of causal effects upon different parts of the conditional distribution. It is quite possible, as in our birthweight application (and also in wage-distribution applications), that societal costs and benefits are more pronounced at the lower quantiles of the conditional distribution. As an example, if one estimated the average causal effect of smoking to be a reduction in birthweight of 150 grams, it could be the case that the effect of smoking on lower quantiles is substantially higher or lower than 150 grams. If a 200-gram effect were estimated at lower quantiles and a 100-gram effect at higher quantiles, this would argue for a stronger policy response than if the effects were instead stronger at the higher quantiles. Ultimately, consideration of how effects vary over the quantiles is an empirical question and one which we attempt to answer in the context of birthweight regressions in this paper.

Previous quantile-estimation approaches to estimating birth-outcome regressions have used cross-sectional data and, therefore, have suffered from an inability to control for unobserved heterogeneity. For instance, Abrevaya (2001) (see also Koenker and Hallock (2001) and Chernozhukov (2005)) uses cross-sectional federal natality data and finds that various observables have significantly stronger associations with birthweight at lower quantiles of the birthweight distribution; unfortunately, one can not interpret these “effects” as causal since the estimation has a purely reduced-form structure that does not account for unobserved heterogeneity.

The outline of the paper is as follows. Section 2 details the quantile-estimation approach, motivated by the “correlated random effects model” of Chamberlain (1982, 1984). We consider a notion of marginal effects upon conditional quantiles in which we explicitly control for unobserved heterogeneity by allowing the “mother random effect” to be related to observables. Section 3 describes the maternally-linked birth panel data for Washington and Arizona that are used in this study. Section 4 reports the main empirical results of the paper. There are some interesting differences between the panel-data and cross-sectional results. For example, the results from panel-data estimation, which controls for unobserved heterogeneity, indicate that the negative effects of smoking on birthweight are significantly lower (in magnitude) across all quantiles than indicated by the cross-sectional estimates. Section 4.2 provides a general hypothesis testing framework. Section 4.3 discusses issues related to endogeneity (e.g., feedback effects and measurement error) in the panel-data context. Section 5 discusses the theoretical panel-data model in greater detail and highlights directions for future research.

2 Quantile estimation for two-birth panel data

Despite the widespread use of both panel-data methodology and quantile-regression methodology, there has been little work at the intersection of the two methodologies. As discussed in this section, the most likely explanation is the difficulty in extending differencing methods to quantiles. The outline of this section is as follows. Section 2.1 briefly reviews the fixed effects and correlated random effects models for conditional expectations. Building upon the correlated random effects framework of Section 2.1, Section 2.2 extends the notion of marginal effects (and their estimation) to conditional quantile models. Section 2.3 discusses previous related studies.

2.1 Review of conditional expectation models with panel data

Suppose that the data source contains information on exactly two births for a large sample of mothers. A standard linear panel-data model for such a situation would be

$$y_{mb} = x'_{mb}\beta + c_m + u_{mb} \quad (b = 1, 2; m = 1, \dots, M), \quad (1)$$

where m indexes mothers, b indexes births, y denotes a birth outcome (e.g., birthweight), x denotes a vector of observables, c denotes the (unobservable) “mother effect,” and u denotes a birth-specific disturbance. To simplify notation, let $x_m \equiv (x_{m1}, x_{m2})$ denote the covariate values from both births of a given mother. From the basic model in (1), several different types of panel-data models arise from the assumptions concerning the unobservable c_m . In the “pure” random-effects version of (1), c_m is assumed to be uncorrelated with x_m . This assumption is implausible in the context of our empirical application, so attention is focused upon two models that allow for dependence between c_m and x_m : (1) the fixed-effects model and (2) the correlated random-effects model.

Fixed-effects model: The fixed-effects model allows correlation between c_m and x_m in a completely unspecified manner. The “meaning” of the parameter vector β is given by

$$\beta = \frac{\partial E(y_{mb}|x_m, c_m)}{\partial x_{mb}} \quad (2)$$

under the following assumption:

$$(A1) \quad E(u_{m1}|x_m, c_m) = E(u_{m2}|x_m, c_m) = 0 \quad \forall m. \quad (3)$$

It is well known that, under (A1), β can be consistently estimated by a first-difference regression (i.e., regressing $y_{m2} - y_{m1}$ on $x_{m2} - x_{m1}$). The reason that this strategy works for the conditional expectation hinges critically upon the fact that an expectation is a linear operator, a property that is not shared by conditional quantiles.

Correlated random-effects model: The correlated random-effects model of Chamberlain (1982, 1984) views the unobservable c_m as a linear projection onto the observables plus a disturbance:

$$c_m = \psi + x'_{m1}\lambda_1 + x'_{m2}\lambda_2 + v_m, \quad (4)$$

where ψ is a scalar and v_m is a disturbance that (by definition of linear projections) is uncorrelated with x_{m1} and x_{m2} . Combining equations (1) and (4) yields

$$y_{m1} = \psi + x'_{m1}(\beta + \lambda_1) + x'_{m2}\lambda_2 + v_m + u_{m1} \quad (5)$$

$$y_{m2} = \psi + x'_{m1}\lambda_1 + x'_{m2}(\beta + \lambda_2) + v_m + u_{m2}. \quad (6)$$

The parameters $(\psi, \beta, \lambda_1, \lambda_2)$ in (5) and (6) can be estimated by least-squares regression or other methods (see, e.g., Wooldridge (2002, Section 11.3)). The vector x_{m1} affects y_{m1} through two channels, (i) a direct effect (expressed by the $x'_{m1}\beta$ term) and (ii) an indirect effect working through the unobservable effect c_m . In contrast, the vector x_{m1} affects y_{m2} only through the unobservable effect c_m . In fact, under the additional assumption

$$(A2) \quad E(v_m|x_m) = 0, \quad (7)$$

the “meaning” of β is given by the following equation

$$\beta = \frac{\partial E(y_{m1}|x_m)}{\partial x_{m1}} - \frac{\partial E(y_{m2}|x_m)}{\partial x_{m1}} = \frac{\partial E(y_{m2}|x_m)}{\partial x_{m2}} - \frac{\partial E(y_{m1}|x_m)}{\partial x_{m2}}. \quad (8)$$

That is, β tells us how much x_{m1} affects $E(y_{m1}|x_m)$ above and beyond the effect that works through the unobservable c_m .

2.2 Estimation of effects on conditional quantiles with panel data

For conditional quantiles, a simple differencing strategy is infeasible since quantiles are *not* linear operators — that is, in general, $Q_\tau(y_{m2} - y_{m1}|x_m) \neq Q_\tau(y_{m2}|x_m) - Q_\tau(y_{m1}|x_m)$, where $Q_\tau(\cdot|\cdot)$ denotes the τ -th conditional quantile function for $\tau \in (0, 1)$. This inherent difficulty has been recognized by others and is summarized nicely in a recent quantile-regression survey by Koenker and Hallock (2000): “Quantiles of convolutions of random variables are rather intractable objects, and preliminary differencing strategies familiar from Gaussian models have sometimes unanticipated effects.” Without being more explicit about the relationship between c_m and x_m , it is difficult to envision an appropriate strategy for dealing with conditional quantiles, although Koenker (2004) has made some progress on this front.

To consider the relevant effects of the observables on the *conditional quantiles* $Q_\tau(y_{mb}|x_m)$ (rather than $E(y_{mb}|x_m)$), we consider the analogous effects to those given in equation (8). In

particular, the effects of the observables on a given conditional quantile are given by

$$\frac{\partial Q_\tau(y_{m1}|x_m)}{\partial x_{m1}} - \frac{\partial Q_\tau(y_{m2}|x_m)}{\partial x_{m1}} \quad (9)$$

and

$$\frac{\partial Q_\tau(y_{m2}|x_m)}{\partial x_{m2}} - \frac{\partial Q_\tau(y_{m1}|x_m)}{\partial x_{m2}}. \quad (10)$$

For example, the difference in equation (9) is the effect of x_{m1} (first-birth observables) on $Q_\tau(y_{m1}|x_m)$ above and beyond the effect on the τ -th conditional quantile that works through the unobservable.

To estimate the effects given in equations (9) and (10), a model for both $Q_\tau(y_{m1}|x_m)$ and $Q_\tau(y_{m2}|x_m)$ is needed. Unfortunately, it is non-trivial to explicitly determine the conditional quantile models. Consider, for example, the simple case in which the data-generating process is given by equations (1) and (4) (which then imply equations (5) and (6)). If all of the error disturbances (u_{m1} , u_{m2} , v_m) were *independent* of x_m , then the conditional quantile functions would take a simple form (analogous to that of the conditional expectation function under assumption (A2)):

$$Q_\tau(y_{m1}|x_m) = \psi_\tau^1 + x'_{m1}(\beta + \lambda_1) + x'_{m2}\lambda_2 \quad (11)$$

$$Q_\tau(y_{m2}|x_m) = \psi_\tau^2 + x'_{m1}\lambda_1 + x'_{m2}(\beta + \lambda_2). \quad (12)$$

Under this independence assumption, the effect of the disturbances is reflected by a locational shift in the conditional quantiles (ψ_τ^1 and ψ_τ^2); the slopes do not vary across the conditional quantiles. Without the independence assumption, however, the simple linear form for the conditional quantile functions (like those in equations (11) and (12)) only arises in very special cases. In general, the conditional quantile functions involve more complicated non-linear expressions and, in fact, can not be explicitly written down without a complete parametric specification of the error disturbances.

Therefore, the conditional quantiles are viewed as somewhat general functions of x_m : say, $Q_\tau(y_{m1}|x_m) = f_\tau^1(x_m)$ and $Q_\tau(y_{m2}|x_m) = f_\tau^2(x_m)$. To estimate the effects in (9) and (10), then, *reduced-form* models for $Q_\tau(y_{m1}|x_m)$ and $Q_\tau(y_{m2}|x_m)$ are specified. These reduced-form models should be viewed as approximating the “true” conditional quantile functions $f_\tau^1(x_m)$ and $f_\tau^2(x_m)$. In this paper, a very simple form for the reduced-form models is considered, in which the conditional quantiles are expressed as linear (and separable) functions of x_{m1} and x_{m2} :

$$Q_\tau(y_{m1}|x_m) = \phi_\tau^1 + x'_{m1}\theta_\tau^1 + x'_{m2}\lambda_\tau^2 \quad (13)$$

$$Q_\tau(y_{m2}|x_m) = \phi_\tau^2 + x'_{m1}\lambda_\tau^1 + x'_{m2}\theta_\tau^2. \quad (14)$$

A more general model, as well as the appropriateness of linearity and separability, is discussed in greater detail in Section 5. Based upon (13) and (14), the effects of the observables on the conditional quantiles (see (9) and (10)) are equal to $\theta_\tau^1 - \lambda_\tau^1$ (for the first-birth outcome) and

$\theta_\tau^2 - \lambda_\tau^2$ (for the second-birth outcome). The parameters $(\phi_\tau^1, \phi_\tau^2, \theta_\tau^1, \theta_\tau^2, \lambda_\tau^1, \lambda_\tau^2)$ can be consistently estimated with linear quantile regression (Koenker and Bassett (1978)).

Although the linear approximation may at first appear to be restrictive, this strategy is the one usually employed in cross-sectional quantile regression. In the cross-sectional case, even if the data-generating process is linear in the covariates with a mean-zero error, the conditional quantiles will only be linear in the covariates in very special cases (see, e.g., Koenker and Bassett (1982)). Even in cross-sectional applications, then, the specification chosen by an empirical researcher (linear usually) should also be viewed as a reduced-form approximation to the true conditional quantile function. In fact, empirical applications of quantile regression generally start (either explicitly or implicitly) with a reduced-form approximating model of the conditional quantile function rather than the data-generating process (see, e.g., Buchinsky (1994) and Bassett and Chen (2001)). Angrist, Chernozhukov, and Fernandez-Val (2006) provide a framework for analyzing misspecification of the conditional quantile function. Although beyond the scope of this paper, it would be interesting to apply their methodology to the panel-data setting considered here.

The linear approximation approach is also an inherent feature of the correlated random-effects approach for the conditional expectation model given by (1) and (4). As Chamberlain (1982) originally pointed out, if assumption (A2) does not hold, the conditional expectation function is non-linear; in this case, equations (5) and (6) represent linear approximations (projections) and β represents the marginal effects of the covariates upon these linear approximations.

For the application in this paper, we impose the additional restriction that the effects on the conditional quantiles are the same for both birth outcomes. This restriction is similar to the implicit restriction embodied in the linear panel-data model (1), where β does not vary with b . For the conditional quantiles, let β_τ denote the (common) effect vector, so that the restriction is

$$\beta_\tau = \theta_\tau^1 - \lambda_\tau^1 = \theta_\tau^2 - \lambda_\tau^2. \quad (15)$$

Under this restriction, the conditional quantile functions in (13) and (14) can be re-written as

$$Q_\tau(y_{m1}|x_m) = \phi_\tau^1 + x'_{m1}(\beta_\tau + \lambda_\tau^1) + x'_{m2}\lambda_\tau^2 = \phi_\tau^1 + x'_{m1}\beta_\tau + x'_{m1}\lambda_\tau^1 + x'_{m2}\lambda_\tau^2 \quad (16)$$

$$Q_\tau(y_{m2}|x_m) = \phi_\tau^2 + x'_{m1}\lambda_\tau^1 + x'_{m2}(\beta_\tau + \lambda_\tau^2) = \phi_\tau^2 + x'_{m2}\beta_\tau + x'_{m1}\lambda_\tau^1 + x'_{m2}\lambda_\tau^2. \quad (17)$$

The simplest estimation strategy, based upon the second equalities in both (16) and (17), is to run a pooled linear quantile regression in which the observations corresponding to both births of a given mother are stacked together as a pair. In particular, a quantile regression (using the estimator for

the τ -th quantile) would be run using

$$\begin{bmatrix} y_{11} \\ y_{12} \\ \dots \\ y_{21} \\ y_{22} \\ \dots \\ \vdots \\ \dots \\ y_{M1} \\ y_{M2} \end{bmatrix} \quad \text{and} \quad \begin{bmatrix} 1 & 0 & x'_{11} & x'_{11} & x'_{12} \\ 1 & 1 & x'_{12} & x'_{11} & x'_{12} \\ \dots & \dots & \dots & \dots & \dots \\ 1 & 0 & x'_{21} & x'_{21} & x'_{22} \\ 1 & 1 & x'_{22} & x'_{21} & x'_{22} \\ \dots & \dots & \dots & \dots & \dots \\ & & \vdots & & \\ \dots & \dots & \dots & \dots & \dots \\ 1 & 0 & x'_{M1} & x'_{M1} & x'_{M2} \\ 1 & 1 & x'_{M2} & x'_{M1} & x'_{M2} \end{bmatrix} \quad (18)$$

as the left-hand-side and right-hand-side variables, respectively. This pooled regression directly estimates $(\phi_\tau^1, \phi_\tau^2 - \phi_\tau^1, \beta_\tau, \lambda_\tau^1, \lambda_\tau^2)$. The difference $\phi_\tau^2 - \phi_\tau^1$ represents the effect of birth parity. Birth parity can not be included explicitly in x since the associated components of β_τ , λ_τ^1 , and λ_τ^2 would not be separately identified. In a traditional panel-data context, the difference $\phi_\tau^2 - \phi_\tau^1$ would represent the “time effect.” Although the application considered here does not have any birth-invariant explanatory variables (“time-invariant” variables), such variables could be easily incorporated into (18) as additional columns in the RHS matrix; like birth parity, it would not be possible to separately identify the direct effects of these variables on y from the indirect effects (working through c) on y .

The only difficulty introduced by the pooled regression approach involves computation of the estimator’s standard errors. Since there is dependence within a mother’s pair of births, the standard asymptotic-variance formula (Koenker and Bassett (1978)) and the standard bootstrap approach, which are both based upon independent observations, can not be applied. Instead, a given bootstrap sample is created by repeatedly drawing (with replacement) a mother from the sample of M mothers and including *both* births for that mother, where the draws continue until the desired bootstrap sample size is reached. For a given bootstrap sample, the pooled quantile estimator is computed. After repeating this process for many bootstrap samples, the original estimator’s variance matrix can be estimated by the empirical variance matrix of the bootstrap estimates. Similarly, bootstrap percentile intervals for the parameters can be easily constructed.

2.3 Review of related studies

In their recent survey of quantile regression, Koenker and Hallock (2000) cite only a single panel-data application. The cited study by Chay (1995) uses quantile regression on longitudinal earnings data to estimate the effect of the 1964 Civil Rights Act on the black-white earnings differential. Chay (1995) allows the individual effect to depend on the racial indicator variable, which amounts to a shift in the conditional quantile function and is a special case of the general approach described

in Section 2.2. Interestingly, the application of Chay (1995) involves censored earnings data, so that quantile regression methods for censored data (Powell (1984, 1986)) are needed. Such censored-data quantile methods would also work with the general model of Section 2.2 but are not needed for the application considered in this paper.

A more recent application of quantile regression on panel data is Arias et. al. (2001), who estimate the returns to schooling using twins data. To deal with the unobserved “family effect,” the authors include proxy variables (father’s education and sibling’s education) in the model. This proxy-variable approach is related to the correlated random effects model in the sense that the latter specification can be viewed as using the observables x_{m1} and x_{m2} as proxies for the unobserved individual effect. One could also incorporate an external proxy (such as father’s education in the Arias et. al. (2001) case) into the correlated random effects framework.

Another panel-data study that is directly related to our empirical application is Royer (2004), who applies a correlated random effects model to maternally linked data from Texas. Royer (2004) estimates the effects of various observables (with a focus upon maternal age) on “binary” birth outcomes (such as premature birth or LBW birth). Fixed-effects estimation is also possible (in the context of the linear probability model) whereas no such alternative is available in the conditional quantile case. Royer (2004) also relaxes the strict exogeneity assumption (required for consistency of the fixed-effects estimator) in several interesting ways. Unfortunately, identification of the least restrictive models requires panel data with at least three births per mother. As a practical matter, this requirement reduces the sample size to an extent that makes the estimated effects of observables rather imprecise and introduces a possible selection bias (see the discussion in Royer (2004, pp. 39ff)). Analogous extensions to the conditional quantile models are left for future research.

3 Data

Detailed “natality data” are recorded for nearly every live birth in the United States. Information on maternal characteristics (age, education, race, etc.), birth outcomes (birthweight, gestation, etc.), and prenatal care (number of prenatal visits, smoking status, etc.) is collected by each state (with federal guidelines on specific data-item requirements). Unfortunately, due to confidentiality restrictions, comprehensive natality data with personal identifiers are not available at the federal level, making it difficult to reliably construct maternally-linked panel data. However, individual states may release such personal identifiers to researchers, subject to confidentiality agreements in most cases. The data used in this study were obtained from two states, Washington and Arizona, and are described in detail below:

1. *Washington data:* The Washington State Longitudinal Birth Database (WSLBD) was provided by Washington’s Center for Health Statistics. The WSLBD is a panel dataset consisting of all births between 1992 and 2002 that could be accurately linked together as belonging to the same mother. (The original WSLBD has births dating back to 1980, but mother’s education is not available as a data item until 1992.) The matching algorithm used to construct the WSLBD used personal identifying information such as mother’s full maiden name and mother’s date of birth. For two births to be linked together, (i) an exact match on mother’s name, mother’s date of birth, mother’s race, and mother’s state of birth was required, and (ii) consistency of birth parity and the reported interval-since-last-birth was required. Only births that could be uniquely linked together were retained in the WSLBD.
2. *Arizona data:* The Arizona Department of Health Services provided the authors with data on all births occurring in the state of Arizona between 1993 and 2002. Although names were not provided, the exact dates of birth for both mother and father were provided in the data. To maternally link births together, we followed as closely as possible the algorithm used for the Washington data. For two births to be linked together, (i) an exact match on mother’s date of birth, father’s date of birth, mother’s race, and mother’s state of birth was required, and (ii) consistency of birth parity and the reported interval-since-last-birth was required. As with the Washington data, only births that could be uniquely linked together were retained. Since births could not be linked by maternal name, we decided to also require an exact match on father’s date of birth in order to minimize the chance of false matches entering the sample. (Roughly 3.5% of births that were linked on the basis of mother’s birthdate are dropped when links are also based upon father’s birthdate.) This choice turns out to have very little impact on the estimation results reported in Section 4. The decision to match upon father’s birthdate restricts the Arizona sample to mothers whose children had the same birth father, which is not a restriction of the Washington sample.

For this study, we consider only *pairs of first and second births to white mothers*. Birth outcomes (and the effects of other variables upon birth outcomes) have been found to differ across different races and at higher birth parities. The choice of subsample circumvents these issue by focusing upon a more homogeneous sample. The resulting estimates, of course, should be interpreted as being applicable to the subpopulation represented by this sample choice.

Estimation was carried out separately for the Washington data and Arizona data. The Washington data has several advantages over the Arizona data: (i) the matching of siblings for the Washington data is of higher quality due to the use of mothers’ names, (ii) the Washington data is not restricted to siblings with the same fathers, and (iii) the Washington data includes information

on the month of first prenatal visit. For these reasons, most of the detailed analysis will be reported for the Washington data. Results for Arizona will be discussed more briefly, but these results serve as a useful comparison to the Washington results.

Table 1 provides descriptive statistics for the Washington and Arizona samples, broken down by first-child and second-child births. Any mother with missing data items in either of her two births (for the variables summarized in Table 1) was dropped from the sample. The resulting samples used for estimation consist of 45,067 Washington mothers (90,134 births) and 56,201 Arizona mothers (112,402 births). Sample averages are reported for all variables, as well as standard deviations for the non-indicator variables. The “Smoke” (“Drink”) variable is equal to one if the mother reported smoking (drinking alcohol) during pregnancy. Although alcohol consumption during pregnancy is known to be severely under-reported, the “Drink” variable is included in the regressions as it may be useful a proxy for other unobservables. For Washington, the four prenatal-care categories (“No prenatal care,” “1st-trimester care,” “2nd-trimester care,” “3rd-trimester care”) were constructed on the basis of the reported month of the first prenatal-care visit. Unfortunately, the month of first prenatal-care visit is not reported in the Arizona data until 1997. As a result, only the number of prenatal visits and an indicator variable for “no prenatal care” (equal to one if there are no prenatal visits) are summarized in Table 1 and used in the empirical analysis of Section 4. The other variables are self-explanatory.

The descriptive statistics in Table 1 indicate that average birthweight increases by 88 grams at the second birth for both Washington mothers and Arizona mothers. For their second birth, women are less likely to smoke and drink and more likely to be married, have a male child, and have a first-trimester prenatal-care visit. Based on the summary statistics, the two samples of mothers are quite similar. On average, Arizona mothers are slightly less educated and have higher birthweight babies. The largest difference between the two samples appears to be the level of smoking: Washington mothers report smoking in 13.7% of pregnancies (close to the national average during this time period), whereas only 4.7% of Arizona mothers report smoking. These smoking percentages are below the overall smoking percentages for pregnant women in these two states during the periods of interest (8.9% in Arizona and 18.4% in Washington), indicating that the matching algorithms result in subsamples that over-represent non-smokers. For instance, unmarried Arizona mothers (for whom the smoking percentage is 12.7%) are far more likely to have father’s date-of-birth missing from the data (45.9% of the time, as compared to 1.1% for married mothers) and, therefore, not included in the matched sample. The reported rate of drinking during pregnancy is also lower in Arizona than Washington; these reported percentages are also lower than the overall percentages for pregnant women in the two states (2.7% in Washington, 1.4% in Arizona).

Table 1: Descriptive Statistics, Washington and Arizona Birth Panels

Variable	Washington		Arizona	
	1st Child	2nd Child	1st Child	2nd Child
Birthweight (in grams)	3442 (523)	3530 (536)	3339 (517)	3427 (505)
Male child	0.515	0.511	0.520	0.516
Mother's age	25.27 (5.25)	27.89 (5.35)	25.23 (5.26)	27.85 (5.36)
Mother's education	13.52 (2.32)	13.72 (2.21)	13.21 (2.68)	13.39 (2.61)
Married	0.751	0.853	0.780	0.886
No prenatal care	0.004	0.003	0.005	0.006
1st-trimester care	0.879	0.895	—	—
2nd-trimester care	0.107	0.093	—	—
3rd-trimester care	0.014	0.012	—	—
Smoke	0.143	0.132	0.049	0.044
Drink	0.017	0.014	0.009	0.007
# prenatal visits	12.06 (3.53)	11.63 (3.25)	11.83 (3.59)	11.73 (3.55)
Quantiles of birthweight:				
10% quantile	2807	2892	2750	2863
25% quantile	3146	3220	3061	3146
50% quantile	3458	3543	3373	3445
75% quantile	3770	3855	3685	3742
90% quantile	4060	4167	3968	4040
# of Observations	45,067	45,067	56,201	56,201

Table 1 also provides the (unconditional) 10%/25%/50%/75%/90% quantiles for first and second births in Washington and Arizona. These quantiles indicate fairly symmetric birthweight distributions, with the median quite close to the mean, the 25% and 75% quantiles roughly equidistant from the median, and the 10% and 90% quantiles roughly equidistant from the median. For both states, there is a positive shift in the entire birthweight distribution from first to second births. The shift is largest in magnitude at the 90% quantile (107 grams) for Washington births and at the 10% quantile (113 grams) for Arizona births. Finally, we note that the LBW cutoff of 2500 grams corresponds to the 3–5% quantiles of the unconditional birthweight distributions, whereas the HBW cutoff of 4000 grams corresponds to the 85–92% quantiles of the unconditional distributions.

4 Results

Regression results for the two maternally linked datasets are provided in Section 4.1, within the strict-exogeneity framework introduced in Section 2. A straightforward approach to hypothesis testing is provided in Section 4.2. Section 4.3 provides discussion related to possible violations of strict exogeneity (e.g., feedback effects or mismeasured variables).

4.1 Regression results

In the interest of space, the full set of numerical results (tables) and a detailed discussion are provided only for the Washington data (Section 4.1.1). The Arizona results are reported in a graphical format comparable to the Washington results (Section 4.1.3), but the detailed tables have been omitted and the discussion is limited to comparisons with the Washington results. (Complete tables are available upon request from the authors.)

4.1.1 Washington data

The tables report estimates for the quantiles $\tau \in \{0.10, 0.25, 0.50, 0.75, 0.90\}$ (along with least-squares estimates for comparison), although the figures presented in this section consider marginal effects at 2-percent intervals (specifically, $\tau \in \{0.04, 0.06, \dots, 0.94, 0.96\}$). Throughout this section, the dependent variable of interest is birthweight (measured in grams). In order to have a relevant comparison for the panel-data results, cross-sectional results (without incorporating the correlated random effects) are also reported. For the cross-sectional results, the panel structure of the data is only used for computing standard errors. Since each mother appears twice in the data, the pair-sampling bootstrap described at the end of Section 2.2 is used.

Tables 2 and 3 report the cross-sectional results and panel-data results, respectively. The model specification includes the variables summarized in Table 1, along with an indicator variable

Table 2: Cross-Sectional Estimation Results, Washington Data. The dependent variable is birth-weight (in grams).

	Quantile regressions					OLS
	10%	25%	50%	75%	90%	
Second child	100.65*** (7.24)	92.87*** (4.96)	93.86*** (4.40)	99.99*** (4.89)	110.37*** (6.84)	98.95*** (4.04)
Male child	87.22*** (6.25)	115.87*** (4.33)	128.34*** (3.85)	142.70*** (4.25)	160.83*** (5.65)	124.34*** (3.57)
Age	19.30*** (6.35)	13.81*** (4.02)	7.39** (3.58)	7.92** (3.93)	5.47 (5.17)	12.57*** (3.41)
Age ²	-0.385*** (0.115)	-0.258*** (0.070)	-0.131** (0.064)	-0.122* (0.070)	-0.070 (0.091)	-0.228*** (0.061)
Education	31.02** (14.32)	22.46** (8.80)	30.23*** (7.70)	28.81*** (6.71)	23.87** (10.45)	26.94*** (7.16)
Education ²	-0.744 (0.525)	-0.603* (0.324)	-0.927*** (0.285)	-0.987*** (0.250)	-0.793** (0.388)	-0.789*** (0.264)
Married	36.58*** (10.22)	27.52*** (7.61)	26.74*** (6.03)	22.41*** (6.91)	16.20* (9.19)	28.11*** (6.23)
No prenatal care	-324.75* (177.34)	-22.76 (55.29)	-36.05 (41.13)	15.54 (44.24)	176.44** (74.27)	-34.57 (47.90)
2nd-trimester care	37.34*** (11.86)	27.93*** (8.41)	24.72*** (7.00)	31.58*** (8.27)	37.96*** (10.43)	38.73*** (6.48)
3rd-trimester care	106.60*** (30.75)	59.36*** (20.43)	37.26* (19.79)	26.07 (18.81)	24.40 (23.84)	66.39*** (15.96)
Smoke	-186.05*** (11.40)	-182.29*** (7.64)	-178.57*** (6.23)	-176.05*** (7.51)	-160.02*** (9.92)	-177.97*** (6.16)
Drink	-21.80 (28.14)	-20.08 (22.67)	-7.27 (16.10)	-15.01 (20.00)	17.03 (26.55)	3.89 (16.47)
# prenatal visits	19.33*** (1.35)	16.52*** (0.89)	15.11*** (0.76)	14.82*** (0.82)	13.92*** (1.10)	18.46*** (0.85)

Bootstrapped standard errors in parentheses, using bootstrap sample size of 20,000 (10,000 pairs) and 1,000 bootstrap replications. Year dummies were included in all regressions.

‘*’: significant at 10% level (2-sided); ‘**’: 5% level; ‘***’: 1% level.

for the second child, quadratic variables for both mother’s age and education, and a full set of year-of-birth dummy variables. For the prenatal-care variables, the omitted category corresponds to first-trimester prenatal care, so the estimates for the other three prenatal-care variables (“No prenatal care,” “2nd-trimester care,” and “3rd-trimester care”) should be interpreted as differences from first-trimester prenatal care. The effect of prenatal care will therefore be captured by (i) the trimester of the first prenatal visit (if any) and (ii) the number of prenatal visits (if any).

It should be pointed out that interpreting the effect of any prenatal-care variable is a bit difficult since the *observed* prenatal care proxies for both *intended* prenatal care and pregnancy problems. For instance, if two mothers have identical intentions (at the beginning of pregnancy) with respect to prenatal-care visits, the mother that experiences problems early in her pregnancy would be more likely to have an earlier first prenatal-care visit and to have more prenatal-care visits overall. The estimated effects of the prenatal-care variables, therefore, may reflect the combined

effects of intended care and pregnancy complications. This idea has been independently investigated by Conway and Deb (2005), who (i) find that bimodal residuals result from a standard 2SLS regression of birthweight and (ii) use a two-class mixture model to explicitly allow for a difference between “normal” and “complicated” pregnancies. The estimates for the no-prenatal-care indicator variable in both Tables 2 and 3, which are significantly negative at the 10% quantile and significantly *positive* at the 90% quantile, illustrate this point. A possible explanation for the dramatic difference at the two ends of the distribution is that lack of prenatal care is more likely to proxy for lack of intended care at the lowest quantiles and more likely to proxy for a problem-free pregnancy at the highest quantiles. Alternatively, the positive effect found at higher quantiles could still be consistent with a lack of intended care since HBW outcomes have previously been associated with poor prenatal care and disadvantage mothers. (Unfortunately, the leading indicators of HBW outcomes are mother’s weight prior to pregnancy and weight gain during pregnancy. Neither of these items is available in the datasets, forcing us to focus less on the effects of birth inputs on HBW outcomes.) At the intermediate quantiles, the effect of the no-prenatal-care indicator is found to be statistically insignificant in both the cross-sectional and panel results.

Overall, the cross-sectional results in Table 2 are very similar to those found in previous studies using federal natality data (Abrevaya (2001); Koenker and Hallock (2001)). For the panel-data results in Table 3, unobserved heterogeneity is modeled as in Section 2.2 (see equations (16) and (17)). For the pooled quantile regressions, Table 3 reports the estimates of the marginal effects β_τ . The estimates of the parameters λ_τ^1 and λ_τ^2 are reported in the Appendix (Tables 5 and 6); these estimates measure the extent of the cross-sectional bias (through the relationship of the unobserved heterogeneity with the observables). To provide a more complete view of the variables’ effects on birthweights and to allow an easy comparison with the cross-sectional estimates, Figures 1 and 2 plot the estimated effects from both the panel and cross section. For these figures, the quantile regressions were estimated at 2% intervals, from the 4% quantile through the 96% quantile (inclusively). The panel-data estimates are represented with a solid line, and the 90% confidence intervals (bootstrap percentile intervals) for these estimates are represented with dotted lines. The cross-sectional estimates, computed at the same quantiles, are represented with a dashed line. (To avoid cluttering the figures, confidence intervals for the cross-sectional results (which can be inferred from Table 2) are not reported.) Since both age and education have quadratic terms in the model specification, the marginal-effect plots for age and education are based upon estimates evaluated at specific values for the two variables (25 years old for age and 12 years for education level).

Table 3: Panel Data Estimation Results (β_τ), Washington data. The dependent variable is birth-weight (in grams).

	Quantile regressions					OLS
	10%	25%	50%	75%	90%	
Second child	137.46*** (11.75)	113.17*** (8.30)	109.76*** (7.03)	113.56*** (7.69)	125.63*** (11.38)	122.32*** (5.99)
Male child	100.67*** (7.76)	131.64*** (5.15)	146.69*** (4.40)	160.12*** (4.82)	173.35*** (6.64)	138.75*** (3.68)
Age	-38.73*** (11.26)	-20.88*** (7.00)	-30.13*** (6.27)	-31.52*** (6.61)	-48.18*** (9.47)	-33.06*** (5.31)
Age ²	0.515*** (0.197)	0.290** (0.120)	0.467*** (0.109)	0.529*** (0.117)	0.845*** (0.161)	0.483*** (0.092)
Education	35.54* (18.25)	17.26 (11.91)	27.22*** (10.23)	17.96** (8.46)	5.97 (13.47)	19.16** (7.62)
Education ²	-1.436* (0.762)	-0.881* (0.503)	-1.029** (0.433)	-0.761** (0.388)	-0.594 (0.607)	-0.851** (0.334)
Married	39.71** (17.26)	15.49 (11.71)	27.99*** (9.21)	21.13* (11.32)	11.41 (16.05)	27.87*** (8.30)
No prenatal care	-323.39* (172.94)	-13.77 (60.05)	1.66 (51.94)	26.03 (54.87)	271.21*** (76.06)	-18.08 (49.01)
2nd-trimester care	26.80* (14.48)	6.63 (10.46)	-0.20 (8.72)	22.95** (10.18)	34.39** (13.52)	22.19*** (6.93)
3rd-trimester care	62.87* (34.96)	71.79*** (25.25)	31.75 (22.67)	30.50 (23.91)	39.22 (32.04)	55.73*** (17.32)
Smoke	-24.58 (19.47)	-60.64*** (14.23)	-81.43*** (11.45)	-57.70*** (12.09)	-56.19*** (17.58)	-56.86*** (9.10)
Drink	-44.24 (35.84)	-32.19 (25.36)	4.29 (20.00)	-1.57 (24.77)	9.25 (29.79)	2.11 (17.11)
# prenatal visits	20.01*** (1.59)	14.79*** (1.10)	12.70*** (0.88)	12.32*** (1.01)	12.65*** (1.43)	17.48*** (0.93)

Bootstrapped standard errors in parentheses, using bootstrap sample size of 20,000 (10,000 pairs) and 1000 bootstrap replications. Year dummies were included in all regressions.

‘*’: significant at 10% level (2-sided); ‘**’: 5% level; ‘***’: 1% level.

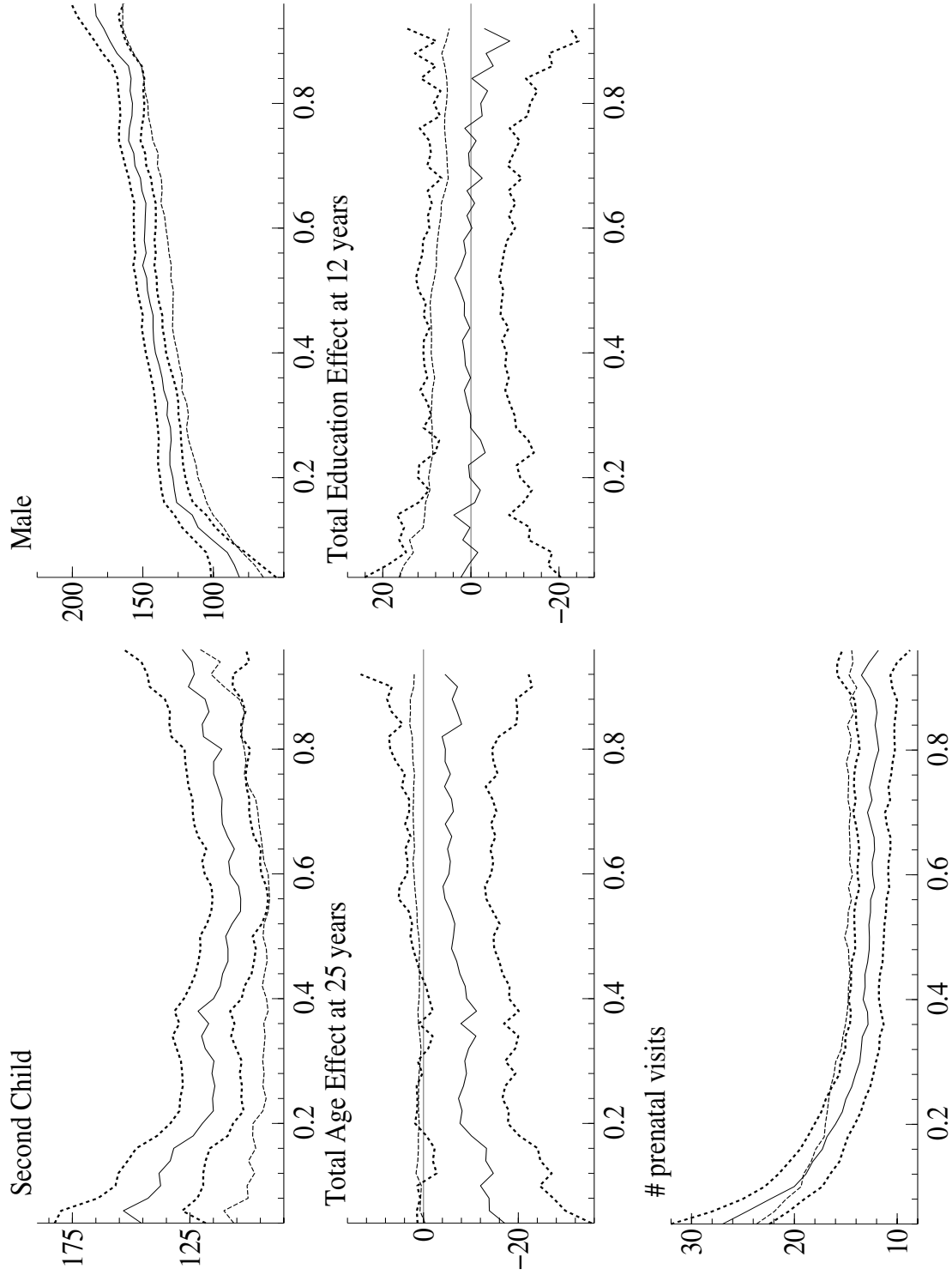


Figure 1: Part 1 of the estimated marginal effects on the conditional quantiles for Washington births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates the cross-sectional estimates.

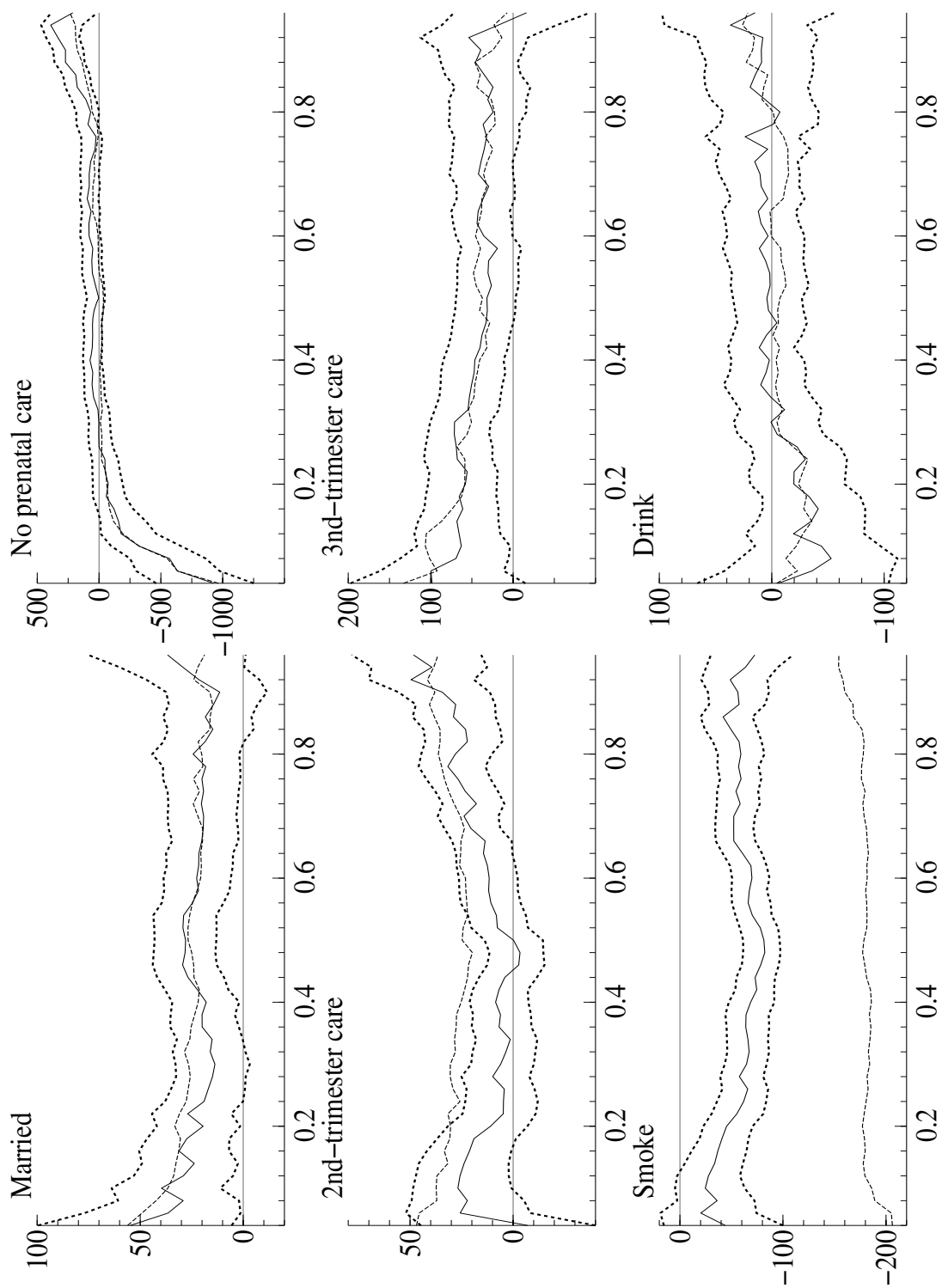


Figure 2: Part 2 of the estimated marginal effects on the conditional quantiles for Washington births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates the cross-sectional estimates.

The estimated effects of the various variables, as presented in Tables 2 and 3 and Figures 1 and 2, are discussed in more detailed below:

Second child: Birthweights are uniformly larger for second children at all quantiles, for both the cross-sectional and panel estimates. The panel estimates of the second-child effect are somewhat larger than the cross-sectional estimates, with the largest effects at the lowest quantiles (e.g., 137 grams at the 10% quantile).

Male child: It is well-known that, on average, male babies weigh more at birth than female babies. The quantile estimates indicate that the positive male-child effect on birthweight is present at all quantiles of the conditional birthweight distribution. The magnitude of the effect increases when one moves from lower quantiles to higher quantiles, with the panel estimates indicating a slightly higher effect (10–20 grams) than the cross-sectional estimates.

Age and education: Figure 1 shows the estimated (one-year) effects of age and education, evaluated at 25 years of age and 12 years of education, respectively. For age, both the cross-sectional and panel estimates are very close to zero in magnitude (and statistically insignificant at a 5% level for all quantiles). For education, the cross-sectional estimates are positive across the quantiles and statistically significant (at a 5% level) except at quantiles above 80%. In contrast, the panel estimates are statistically insignificant across all quantiles. This difference could be due to two factors: (i) the amount of within-mother variation in education is quite small, with the average change in education for the sample being about 0.2 years; and, (ii) the level of education may be related to the mother-specific unobservable. For the latter factor, years of schooling is likely positively related to c_m , which would imply an upward bias in the cross-sectional estimates that is consistent with Figure 1. The issue of education being potentially mismeasured is briefly discussed in Section 4.3.2. Results for other age and education levels are reported in Abrevaya and Dahl (2006).

Marital status: The estimated positive effects of marriage on birthweight are quite similar for the cross-sectional and panel specifications, in the 20–50 gram range over the quantiles considered. One should be cautious about interpreting the cross-sectional marriage estimates as causal since marital status is an explanatory variable that *a priori* would appear to serve as a proxy for mother-specific unobservables (i.e., marital status positively correlated with c_m). The panel estimates are slightly lower than the cross-sectional estimates in the lower quantiles (until around the 40% quantile), suggesting that this might be a factor in the lower quantiles. Somewhat surprisingly, however, the panel estimates of the marriage effect remain positive throughout the range of quantiles and significantly so (at the 10% level) at nearly all the quantiles below 80%. On the whole, the estimates are consistent with a situation in which

marriage provides the birth mother with support (financial support, emotional support, etc.) that would lead to a more favorable birth outcome.

Prenatal-care visits: Lack of prenatal care is found to have a significant negative effects at lower quantiles and significant positive effects at the upper quantiles. The estimated effects are similar for both the cross-sectional and panel regressions. As discussed above, a logical explanation is that the “No prenatal care” indicator variable may proxy for poor care at lower quantiles but for problem-free pregnancies at upper quantiles. For the third-trimester-care indicator variable, the cross-sectional and panel estimates are also similar, indicating positive effects (as compared to first-trimester care) which become less statistically significant at higher quantiles. For the indicator variables, the largest difference between the cross-sectional and panel results shows up in the second-trimester-care variable; the cross-sectional estimates are statistically significant at all quantiles and range from 25 to 50 grams, whereas the panel estimates are somewhat lower (close to zero in intermediate quantiles) and only significantly positive at the highest quantiles. The effect of the number of prenatal visits is estimated to be significantly positive across all quantiles, with larger effects found at lower quantiles and the effects essentially “flattening out” (at around 14–15 grams per visit for the cross-sectional results and 12–13 grams per visit for the panel results). The estimated effects for the panel specification exhibit a sharper decline, leading to lower estimates (roughly a 2-gram per-visit differential) than the cross-sectional specification. This variable shows up significantly in the λ_{τ}^1 and λ_{τ}^2 estimates (see Tables 5 and 6), leading to the differences found and suggesting that the variable is related to the mother-specific unobservable.

Smoking: The most dramatic differences between the cross-sectional and panel results are the estimated effects of smoking. The cross-sectional results indicate that the negative effects of smoking are in the range of 150–200 grams, with larger effects at lower quantiles. The panel estimates are still significantly negative at all but the lowest quantiles, but the estimated effects are much lower in magnitude (mostly in the 50–80 gram range between the 20% and 80% quantiles). The omitted-variables explanation of this large difference would be that the smoking indicator in the cross-sectional specification is negatively related with the error disturbance in the birthweight regression equation. Consistent with this explanation, the smoking coefficients in both λ_{τ}^1 and λ_{τ}^2 are found to be significantly negative across the quantiles (Tables 5 and 6). The magnitudes of the panel estimates are also significantly lower than those found in previous work, including quasi-experimental estimates based upon cigarette-tax changes (e.g., Evans and Ringel (1999) and Lien and Evans (2005)) and experimental estimates (e.g., Permutt and Hebel (1989)). These studies have estimated causal (IV) effects of smoking on birthweight which are not statistically different from the OLS estimates; these

estimates have relatively large standard errors (due to the sources of variation exploited) and, in some cases, are even *larger* in magnitude than the OLS estimates. We believe that our panel estimates are quite credible given the compelling nature of the omitted-variables explanation in this context. We note, however, that our results do not directly contradict those found by instrumental-variables methods. First, the IV estimates are quite imprecise (large standard errors), so our estimates would also fall within reasonable confidence intervals for these previous studies. Second, the panel estimates are identified from mothers who change their smoking status for any reason whereas the IV estimates are identified from mothers who change their smoking status in response to a specific treatment (e.g., prenatal counseling or cigarette-tax increases); since these subpopulations are different, the underlying treatment effects could themselves also be different. Finally, we point out that misclassification of smoking status could explain part of the difference found here since the effect of misclassification is more severe in the panel-data case (see, for example, Freeman (1984) and Jakubson (1986)). This possibility is further discussed in Section 4.3.2.

Alcohol consumption: In contrast to the smoking results, the estimated effects of alcohol consumption are quite similar for the cross-sectional and panel specifications. Drinking is estimated to have significant negative effects at lower quantiles (below about the 20% quantile), with the magnitudes of the effects ranging between about 40 and 80 grams. Of course, very few mothers actually report alcohol consumption during pregnancy (only about 1.5% in our sample). The lack of strong statistical evidence regarding the effects of drinking could stem from the low variation in the indicator variable and the probable large rates of misclassification.

4.1.2 “Overcontrolling” and interpretation of estimates

If one is interested in the “structural” estimates related to prenatal care and smoking, it is not obvious which variables should be included in the regression specification. In particular, the estimates presented above are identified from within-mother variation (rather than variation induced by a specific policy), but we would like to be able to offer an interpretation of the estimates relevant to potential policy impacts. What would be the effect of a policy that increased the likelihood of a first-trimester prenatal visit? What would be the effect of a policy that reduced the likelihood of prenatal smoking? Related to these two questions, there are potential concerns with the specification used above, which includes variables for prenatal-care initiation, number of prenatal visits, and smoking status. If earlier prenatal-care initiation (e.g., a first-trimester visit) has the “mechanical” effect of increasing the number of prenatal-care visits, inclusion of the number of visits as a covariate effectively “overcontrols” for the effect of early prenatal-care initiation. Similarly, if prenatal care affects birthweight only through its effect on smoking initiation, inclusion of smoking

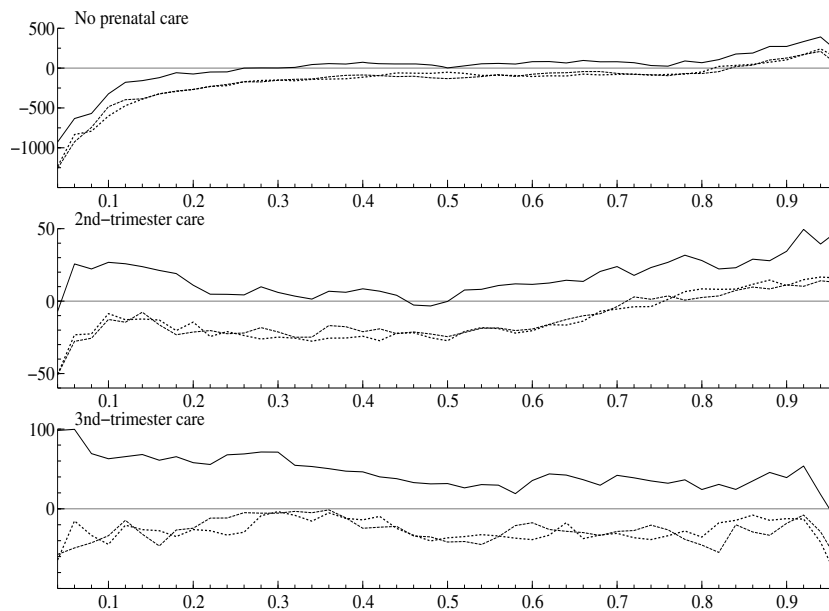


Figure 3: Results for initial-prenatal-visit variables under different specifications (Washington data). The solid line corresponds to the full specification, the dotted line to the specification in which “# of prenatal visits” is dropped, and the dashed line to the specification in which “# of prenatal visits,” “Smoke,” and “Drink” are dropped. The omitted category is first-trimester care.

status as a covariate could also be an “overcontrolling” factor.

To empirically assess the possible importance of “overcontrolling,” we re-ran the Washington regressions under two alternative specifications: (i) original specification with number of prenatal visits dropped, and (ii) original specification with number of prenatal visits, smoking status, and drinking status dropped. Figure 3 reports the estimates on the prenatal-care-initiation categorical variables for these two specifications along with the original specification. Dropping the number of prenatal visits from the specification has important consequences. For each of the three variables (which are interpreted as differences from first-trimester care), there is a significant drop in the coefficient estimates across the quantiles. The second-trimester estimate goes from being positive (and statistically insignificant) at most quantiles to being negative and statistically significant (at a 10% level) at all quantiles below 60%. The no-prenatal-care variable also becomes negative and statistically significant (at a 10% level) at all quantiles below 60%. (Note the difference in scale on the y axes for the three variables.) The third-trimester estimate goes from being significantly positive at most quantiles to being negative, but statistically insignificant, at most quantiles. Overall, if one views first-trimester prenatal care as mechanically increasing the number of prenatal-care visits, the estimates from Figure 3 indicate that the structural effect of increasing early prenatal-care initiation would be to increase birthweight at lower quantiles (with small effects

of about 20 grams for transitions from second-trimester care to first-trimester care and much larger effects for transitions from no prenatal care to first-trimester care). We also note that the estimates on the other variables (those not shown in Figure 3) remain essentially unchanged when number of visits is dropped from the specification.

When smoking status and drinking status are also dropped from the specification, there is essentially no change in the prenatal-care-initiation estimates shown in Figure 3 (the comparison between the dotted and dashed lines). This suggests that the inclusion of smoking status in the original specification (and also in the one dropping number of visits) did not have an impact on the estimated effect of the timing of prenatal-care initiation. Alternatively, in thinking of other variables as possibly “overcontrolling” for smoking status, we tried several specifications in which other covariates were dropped from specifications in which smoking status remained. For these specifications, we found estimated smoking effects that were extremely similar to those reported in Figure 1. These results make us more comfortable about interpreting the original smoking estimates (Figure 1) as structural effects upon the conditional quantile distribution.

4.1.3 Arizona data

Figures 4 and 5 plot the estimated quantile effects (4% through 96% quantiles, inclusively) for the Arizona maternally-linked sample. The same model specification discussed above was used, except that indicator variables for second-trimester and third-trimester prenatal care were not included. The figures are comparable to Figures 1 and 2 for the Washington data, with the age effect reported at 25 years and the education effect at 12 years.

Overall, there is a remarkable similarity between the results for the two samples. The common findings for the two samples include the following:

- There is a significant positive effect of the second child across all quantiles (50–125 grams in the Arizona panel estimates).
- The positive birthweight effect of a male child increases from lower to higher quantiles.
- Despite a positive estimated cross-sectional effect of education at lower quantiles, the panel estimates indicate no significant education effect.
- The effect of the number of prenatal visits is highest at lower quantiles, with the effect flattening out at higher quantiles. For both Washington and Arizona, the cross-sectional estimate of the effect is lower at lower quantiles and higher at higher quantiles.
- The magnitude of the negative smoking effect is significantly lower for the panel estimates (ranging between 40 and 80 grams for Arizona) than for the cross-sectional estimates.

Some differences between the results for the two samples are also worth noting:

- Although the cross-sectional estimates of the marriage effect are still significantly positive (p-values lower than 0.10 throughout the range of quantiles), the panel-data estimates indicate no statistically significant effect of marriage for Arizona mothers. The likely explanation of this finding is that the father's date of birth is required to match for both births of an Arizona mother (see Section 3), meaning that the father is the same even if marital status differs across the births. For the Washington sample, a change in marital status might also be related to a change in father.
- Due to the lack of indicator variables for second-trimester and third-trimester care, the estimated effects of the no-care indicator variable and the number of prenatal visits are slightly different. The magnitude of the quantile effects for number of prenatal visits is roughly 50% lower for the Arizona sample, although the shape of the quantile-effect curve is extremely similar. The shape of the no-prenatal-care effect is also very similar to that of Washington, but the estimated panel effects are not significantly different from zero at any of the quantiles.

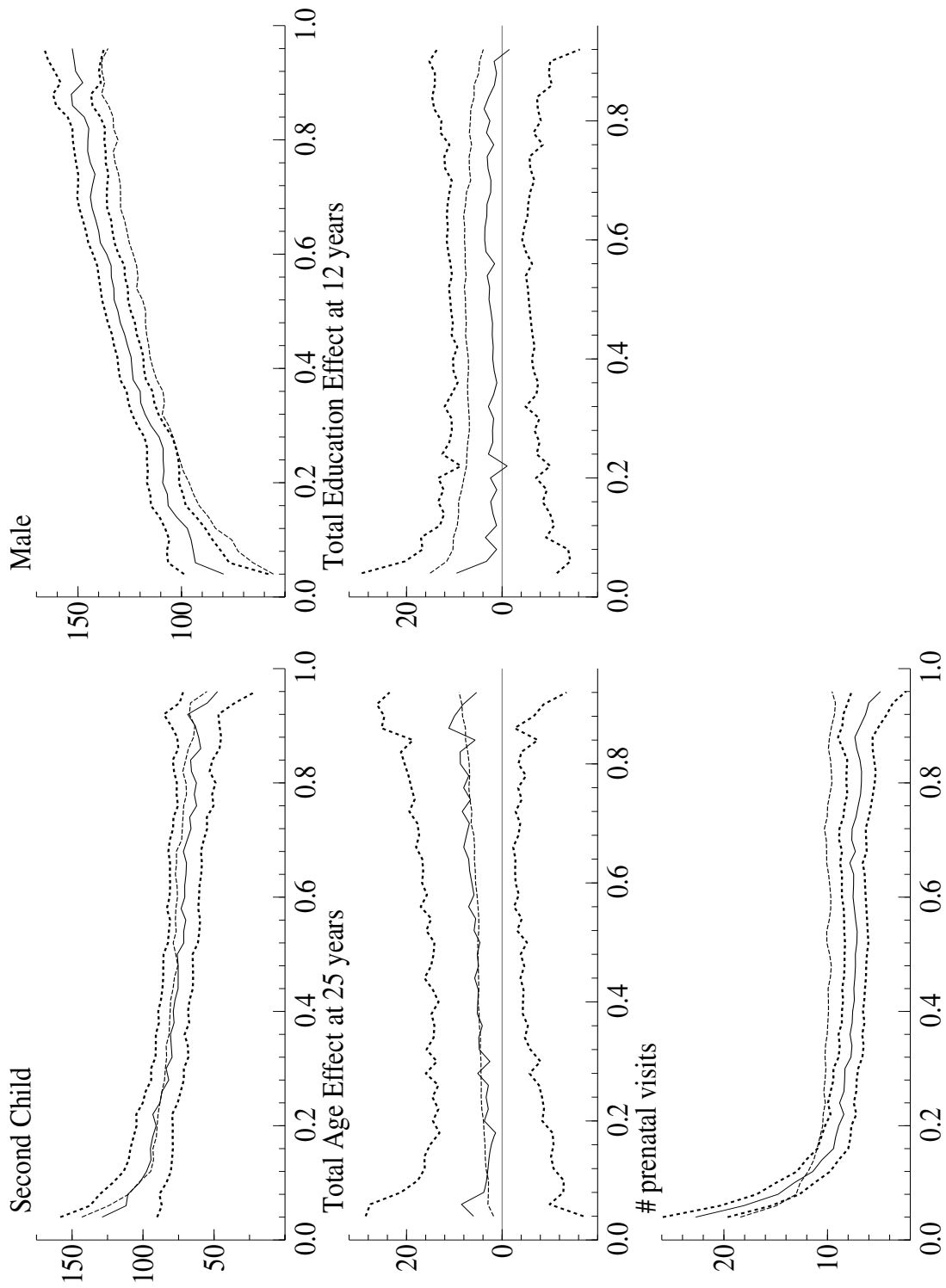


Figure 4: Part 1 of the estimated marginal effects on the conditional quantiles for Arizona births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates the cross-sectional estimates.

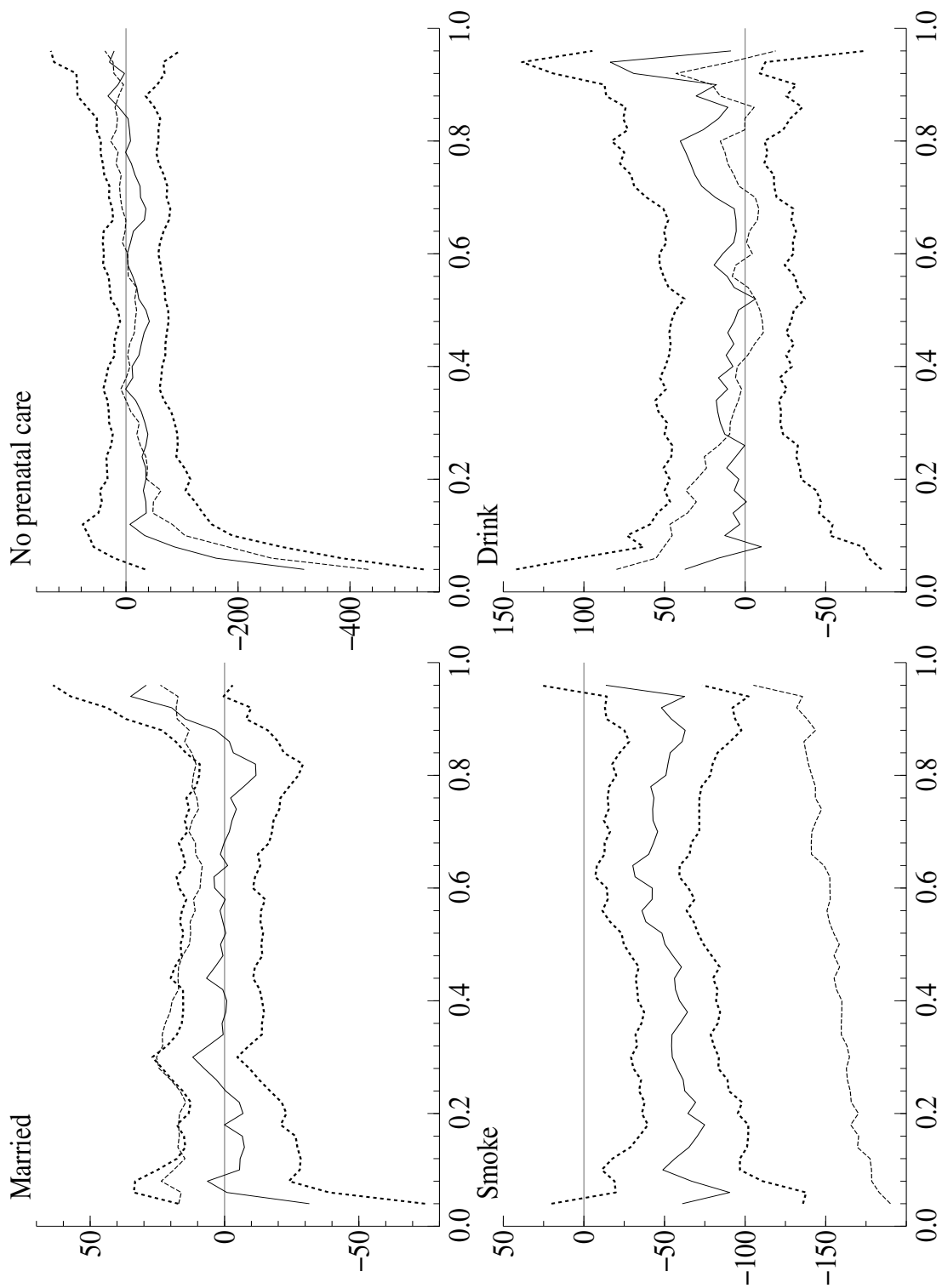


Figure 5: Part 2 of the estimated marginal effects on the conditional quantiles for Arizona births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates the cross-sectional estimates.

4.2 Hypothesis testing

In this section, we discuss the results of several hypothesis tests that were used in order to test the model specification and/or the significance of differences across the estimates at different quantiles. The minimum-distance (MD) framework of Buchinsky (1998) is used (and extended to the panel-data case) to test various (linear) restrictions placed on the parameters in the estimated models.

4.2.1 Minimum-distance testing framework

Let p denote the number of different quantiles at which the model is estimated, with τ_1, \dots, τ_p denoting the quantiles. For a given quantile τ , individual elements of the parameter vectors β , λ_τ^1 , and λ_τ^2 (recall the model in (16) and (17)) are referenced by subscripts as follows:

$$\beta_\tau = (\beta_{\tau 1}, \dots, \beta_{\tau L})', \quad \lambda_\tau^1 = (\lambda_{\tau 1}^1, \dots, \lambda_{\tau K}^1)', \quad \lambda_\tau^2 = (\lambda_{\tau 1}^2, \dots, \lambda_{\tau K}^2)',$$

where K is the number of variables in x_{m1} and x_{m2} . For generality, β_τ has $L \geq K$ elements, to allow for additional variables (e.g., time-invariant regressors) that may not appear in the λ estimates. Then, for a given quantile τ , the full parameter vector is denoted

$$\gamma_\tau \equiv \left(\phi_\tau^1, \beta_{\tau 0}, \beta_\tau', \lambda_\tau^{1'}, \lambda_\tau^{2'} \right)', \quad (19)$$

where $\beta_{\tau 0} \equiv \phi_\tau^2 - \phi_\tau^1$. The (stacked) parameter vector for all of the estimated quantiles is denoted

$$\gamma \equiv (\gamma_{\tau_1}', \gamma_{\tau_2}', \dots, \gamma_{\tau_p}')' \quad (20)$$

and has dimension $p(2K + L + 2) \times 1$. Further, let $\hat{\gamma}$ denote the estimator of γ , and define \hat{A} to be the estimated variance-covariance matrix (obtained via the bootstrap) of $\hat{\gamma}$.

In the MD framework, the “restricted” parameter estimator is defined as

$$\hat{\gamma}^R = \arg \min_{\gamma^R \in \Theta} (\hat{\gamma} - R\gamma^R)' \hat{A}^{-1} (\hat{\gamma} - R\gamma^R), \quad (21)$$

where R is a restriction matrix that will depend on the type of restrictions imposed. Since only linear restrictions are considered, $\hat{\gamma}^R$ can be written explicitly as

$$\hat{\gamma}^R = \left(R' \hat{A}^{-1} R \right)^{-1} \left(R' \hat{A}^{-1} \hat{\gamma} \right). \quad (22)$$

The asymptotic variance of $\hat{\gamma}^R$ is given by

$$\text{var}(\hat{\gamma}^R) = \left(R' \hat{A}^{-1} R \right)^{-1}. \quad (23)$$

For the purposes of hypothesis testing, note that under the null hypothesis that the restrictions are true (i.e., $H_0 : \gamma = R\gamma^R$), the following MD test statistic has a limiting chi-square distribution:

$$(\hat{\gamma} - R\hat{\gamma}^R)' \hat{A}^{-1} (\hat{\gamma} - R\hat{\gamma}^R) \xrightarrow[H_0]{d} \chi_M^2, \quad (24)$$

where M is the number of restrictions (i.e., $M = \text{rows}(R) - \text{columns}(R)$). The Appendix provides specific details on the appropriate choice of R and M for each of the tests described below.

4.2.2 Test results

Using the MD testing framework, the following hypothesis tests were conducted:

Test of correlated random effects: To determine whether a “pure” random effects specification (in which c_m is uncorrelated with x_m) would be rejected for a given quantile τ , the null hypothesis $H_0 : \lambda_\tau^1 = \lambda_\tau^2 = 0$ is tested. For the quantiles $\tau \in \{0.10, 0.25, 0.50, 0.75, 0.90\}$, the null hypothesis is overwhelmingly rejected with p-values extremely close to zero.

Test of the equality of the “effect vector” across quantiles: This test considers whether there are *any* statistically significant differences in the β_τ estimates across two different quantiles. For the panel specifications, we conducted this test for each pairwise combination of quantiles from the set $\{0.10, 0.25, 0.50, 0.75, 0.90\}$. For Washington, the p-values (all below 2%) indicate very significant differences across the quantiles. For Arizona, there are significant differences between the lowest quantiles (10% and 25%) and other quantiles; however, the p-values for the 50%/90% and the 75%/90% comparisons do not indicate a statistically significant difference in the β_τ estimates.

Test of the equality of individual variables’ effects across quantiles: For a given variable (for example, marital status), this test checks whether the estimated effects at different quantiles are significantly different. The set of different quantiles considered is the same as that used in Tables 2–3. For the marriage indicator, for instance, the null hypothesis would be $H_0 : \beta_{\tau=0.10}^{\text{married}} = \beta_{\tau=0.25}^{\text{married}} = \beta_{\tau=0.50}^{\text{married}} = \beta_{\tau=0.75}^{\text{married}} = \beta_{\tau=0.90}^{\text{married}}$. Since both age and education enter into the model specification in two terms (a linear term and a quadratic term), the appropriate tests for these two variables are joint tests of equality. The test results (p-values) for all of the variables, in both the cross-sectional and panel specifications, are reported in Table 4 for Washington and Arizona. The results are very much in line with the quantile-estimate graphs in Figures 1–2 and Figures 4–5. Two variables (male-child indicator and number of prenatal visits) vary significantly across the quantiles for both the cross-sectional and panel specifications. The effect of the no-prenatal-care indicator also varies significantly (p-value of 0.010 in the cross section and 0.004 in the panel) for the Washington sample. On the other hand, there is no statistical evidence that the effects of marital status or drinking vary over quantiles in either specification. The cross-sectional estimated effects of both age and education vary significantly across quantiles, whereas the panel estimated effects do not. For the smoking-indicator variable, the p-value for the Washington cross-sectional results is

Table 4: Tests of Marginal-Effect Equality Across Quantiles. For each covariate, p-values based upon cross-sectional and panel-data estimates are reported for the null hypothesis of equality of marginal effects for the five quantiles 0.10, 0.25, 0.50, 0.75, and 0.90. Results are based upon 1,000 bootstrap replications.

	Washington		Arizona	
	Cross Section	Panel Data	Cross Section	Panel Data
Second child	0.135	0.113	0.000	0.049
Male child	0.000	0.000	0.000	0.000
Age, age ² jointly	0.006	0.088	0.000	0.944
Education, education ² jointly	0.014	0.336	0.002	0.927
Married	0.574	0.457	0.565	0.739
No prenatal care	0.010	0.004	0.442	0.874
2nd-trimester care	0.620	0.059	—	—
3rd-trimester care	0.154	0.638	—	—
Smoke	0.257	0.063	0.236	0.834
Drink	0.578	0.602	0.346	0.905
# prenatal visits	0.005	0.000	0.005	0.000

quite high (0.396), whereas the p-value (0.063) for the panel specification suggests a more significant difference in the estimated effects across quantiles. Finally, it should be noted that the choice of the quantile set $\{0.10, 0.25, 0.50, 0.75, 0.90\}$ is admittedly arbitrary, following what has become the convention for quantile regression.

4.3 Endogeneity Issues

In this section, we consider the sensitivity of the estimation results to possible sources of endogeneity. Note that the estimation method introduced in Section 2 (and discussed further in Section 5) is based upon the assumption of strict exogeneity and, in general, will be inconsistent when this assumption is violated. The two most important sources of endogeneity in the current application are: (1) a “feedback effect” by which the first-birth outcome (birthweight) influences second-birth explanatory variables (e.g., a low-birthweight first-birth outcome causing a mother to quit smoking for the second birth) and (2) mismeasured explanatory variables.

4.3.1 Feedback or dynamic effects

The issue of feedback effects is discussed at length in Abrevaya (2006) in the context of a conditional-expectation model, where instrumental-variables methods (using lagged birthweights as instruments) can be utilized. Unfortunately, there is no obvious analogue to instrumental variables in

the conditional-quantile context. Instead, to see if allowing for dynamic effects alters the panel-data estimates in an important way, we consider an augmented model specification in which lagged birthweight is included as an explanatory variable. Specifically, since data on two births per mother are available, y_{m1} (first-birth birthweight) is included as a right-hand-side variable for the second-birth equation. (Considering matched panel data with three births per mother reduces the sample size to an extent which makes all of the estimates imprecise.) Only a single coefficient for y_{m1} in the second-birth equation can be estimated; there is no way to separately identify coefficients within both β_τ and λ_τ^2 .

Overall, the inclusion of lagged birthweight in the second-birth equation does not have a large effect on the estimated effects of the other observable variables. Lagged birthweight is found to be a significant predictor of second-birth birthweight, with coefficient estimates around 0.45 at the lower quantiles and gradually decreasing to around 0.40 at the higher quantiles. Despite the significant effects of lagged birthweight, the estimated effects of smoking do not change much. The new estimates are mostly flat at around -75 grams, just slightly below the original panel estimates. The interested reader is referred to Abrevaya and Dahl (2006) for more details.

Another source of endogeneity related to “feedback effects” is that the decision to have a second child may itself be affected by the first-birth outcome. For instance, one might expect mothers with a particularly adverse (good) first-birth outcome to be less (more) likely to have a second child. This source of endogeneity is difficult to handle without more a more explicit model of the second-birth selection/attrition process, and the present data do not provide obvious candidates for convincing exclusion restrictions in such a model.

4.3.2 Measurement error

To examine the issue of measurement error, we focus on two explanatory variables: education and smoking. Since the panel-data estimated effects of education are insignificant, it is worthwhile to examine whether measurement error in education might be causing an attenuation bias in the estimated effects. For smoking, the panel-data approach yields vastly different estimates (lower in magnitude) than the cross-sectional approach. As discussed in Section 4.1.1, one possible cause of these lower magnitudes is misclassification of smoking status (specifically, smokers being misclassified as non-smokers). Freeman (1984) and Jakubson (1986) have shown that the attenuation bias from misclassification of an explanatory variable can be more severe in the panel-data case than the cross-sectional case. Unfortunately, cross-validation data are not available for the education and smoking variables.

For education, there are cross-sectional units for which the education variable must be mismeasured due to inconsistencies in reporting from the first-birth data to the second-birth data.

Using the Washington data, we dropped all observations having (i) more additional education than possible given the change in age ($\Delta Education > \Delta Age + 1$) or (ii) a drop in years of education ($\Delta Education < 0$). There were 5,455 mothers (12% of the sample) with such inconsistencies in reported education, with 346 mothers in group (i), 5,117 in group (ii), and 8 in both. Re-running the panel-data regressions on the less error-ridden subsample yielded extremely similar results to the original regressions, with the education effects remaining insignificant and the other estimated effects being nearly identical. More details are provided in Abrevaya and Dahl (2006). Of course, there may be other forms of reporting errors in education (see, for example, Kane, Rouse, and Staiger (1999)), but cross-validation data would be required to examine these.

To gauge the sensitivity of the smoking-effect estimates to possible misclassification of smoking status, we conducted simulations where we artificially introduced misclassification into the estimation sample. In each simulation, we misclassified a fraction q (for $q = 0.1$ and $q = 0.2$) of observed smokers as non-smokers and then compute the panel-data estimates (at $\tau \in \{0.1, 0.2, \dots, 0.8, 0.9\}$). (We assume no misclassification of non-smokers as smokers.) Abrevaya and Dahl (2006) report details of the simulation results, which we briefly summarize here. As expected, the artificial misclassification induces an attenuation bias in the estimated smoking effect. The simulations suggest biases on the order of 20–80% for the misclassification rates considered, biases which are far too small to explain the difference between the cross-sectional and panel estimates. For 20% misclassification, the simulations suggest that misclassification bias accounts for nearly half of the difference between the panel and cross-sectional estimates; however, at quantiles away from the median (especially so at the 10% quantile), the misclassification bias accounts for less of the difference from the cross-sectional estimates. Finally, we note that the “model” of misclassification used to create the artificial datasets is too simplistic since it does not allow for (i) a mother’s reporting errors to be correlated over time or (ii) reporting errors to be correlated with other observables. The first issue is considered by Abrevaya (2006), where it is shown that positive correlation in misreporting (i.e., a mother who misreports during her first pregnancy is more likely to misreport during her second pregnancy) reduces the extent of the misclassification bias. The second issue is considerably more difficult to analyze without some type of cross-validation data.

5 Discussion of the theoretical model

In this section, we provide a more formal discussion of the panel-data framework introduced in Section 2. In doing so, we highlight the pros and cons of the proposed approach and suggest directions for future research. To be consistent with the standard panel-data notation, this section will use i (rather than m) to denote cross-sectional units and t (rather than b) to denote time. The number of

time periods $T \geq 2$ for each cross-sectional unit is assumed to be fixed, whereas the number of cross-sectional units $n \rightarrow \infty$. The random variables $\{(x_{i1}, \dots, x_{iT}, u_{i1}, \dots, u_{iT}, c_i)\}_{i=1}^n$ are assumed to be i.i.d. draws from their underlying distributions. The observed data are $\{(y_{i1}, \dots, y_{iT}, x_{i1}, \dots, x_{iT})\}_{i=1}^n$, where each y_{it} is generated according to the model

$$y_{it} = x'_{it}\beta + c_i + u_{it}. \quad (25)$$

The relationship between c_i and $x_i \equiv (x_{i1}, \dots, x_{iT})$ is described by

$$c_i = \phi(x_i) + v_i, \text{ where } E(v_i|x_i) = 0. \quad (26)$$

Equation (26) is not restrictive, in the sense that the conditional-mean ‘‘assumption’’ ($E(v_i|x_i) = 0$) is merely a normalization that fixes location.

For any $\tau \in (0, 1)$, the conditional τ -th quantile of y_{it} is

$$Q_\tau(y_{it}|x_i) = x'_{it}\beta + \phi(x_i) + Q_\tau(v_i + u_{it}|x_i). \quad (27)$$

The assumptions related to the last term in (27), $Q_\tau(v_i + u_{it}|x_i)$, dictate how the marginal effects of x_{it} upon $Q_\tau(y_{it}|x_i)$ can be identified. Consider the following two simplifying assumptions:

$$(B1) \ v_i \text{ is independent of } x_i \quad (28)$$

$$(B2) \ Q_\tau(u_{it}|x_i, v_i) = Q_\tau(u_{it}|x_{it}). \quad (29)$$

Assumption (B1) is commonly used in estimation of non-linear panel-data models with correlated random effects. The data-generating process allows x_i to affect the level of the y_{it} variables (through the $\phi(x_i)$ function), but Assumption (B1) restricts the quantiles of the fixed effect to not depend upon x_i . Assumption (B2), which says that the quantiles of u_{it} depend upon x_i only through x_{it} , allows for arbitrary forms of x_{it} -related heteroskedasticity. Note that Assumption (B2) also imposes a form of strict exogeneity since the presence of feedback effects (relationships between past u 's and future x 's) or lagged dependent variables would violate this assumption.

Taken together, Assumptions (B1) and (B2) imply that

$$Q_\tau(v_i + u_{it}|x_i) = Q_\tau(v_i + u_{it}|x_{it}) \equiv f_{\tau,t}(x_{it}). \quad (30)$$

The t subscript in $f_{\tau,t}(x_{it})$ allows the relationship between the u_{it} distribution and x_{it} to change with t . (If this relationship is assumed to be the same over time, the t subscript could be omitted, leaving $f_\tau(x_{it})$.) Plugging equation (30) into equation (27) yields

$$Q_\tau(y_{it}|x_i) = x'_{it}\beta + f_{\tau,t}(x_{it}) + \phi(x_i). \quad (31)$$

In general, both $f_{\tau,t}$ and $\phi(x_i)$ will be non-linear functions. Consider the following simple example, which illustrates the inherent non-linearity of $f_{\tau,t}$:

Example: Linear-scale heteroskedasticity

The following distributional assumptions for c_i and u_{it} are made:

$$c_i|x_i \sim N(\psi + x'_{i1}\lambda_1 + \dots + x'_{iT}\lambda_T, \sigma_c^2)$$

$$u_{it}|c_i, x_i \sim N(0, (x'_{it}\gamma_t)^2)$$

These assumptions generalize the linear-scale model considered by Koenker and Bassett (1982) to a panel-data setting and also impose normality. The variance of $(c_i + u_{it})|x_i$ is $\sigma_c^2 + (x'_{it}\gamma_t)^2$, which implies $f_{\tau,t}(x_{it}) = z_\tau \sqrt{\sigma_c^2 + (x'_{it}\gamma_t)^2}$, where z_τ denotes the τ -th quantile of the standard normal distribution. In this example, linearity of $f_{\tau,t}$ with respect to x_{it} would only arise when $\sigma_c^2 = 0$.

In the context of the general model described above (with Assumptions (B1) and (B2)), the estimation approach proposed in Section 2 uses linear approximations for the two components ($x'_{it}\beta + f_{\tau,t}(x_{it})$ and $\phi(x_i)$) of the conditional quantile in equation (31). Specifically, generalizing the notation used in Section 2 to $T \geq 2$, $x'_{it}\beta_\tau$ is used to approximate $x'_{it}\beta + f_{\tau,t}(x_{it})$, and $\psi_\tau^t + x'_{i1}\lambda_\tau^1 + \dots + x'_{iT}\lambda_\tau^T$ is used to approximate $\phi(x_i)$. The conditional-quantile function in equation (31) is additively separable in the functions of x_{it} and x_i , with $\phi(x_i)$ entering the conditional-quantile function in the same way in each time period. This separability makes it possible to directly estimate the marginal effects of interest (i.e., the effects of x_{it} upon $x'_{it}\beta + f_{\tau,t}(x_{it})$) by the proposed approach.

The most important topic for future research is to consider the situation in which Assumption (B1) is violated. If the quantiles of v_i depend upon x_i , the conditional quantiles of y_{it} will not have the additive-separability property seen above. Instead, the conditional quantile function would be a nonseparable function of x_{it} and x_i , say $Q_\tau(y_{it}|x_i) = f_{\tau,t}(x_{it}, x_i)$. The effect of interest is the derivative of $f_{\tau,t}$ with respect to its first argument, where integration over the distribution of x_i could provide some type of average effect. A nonparametric approach to this problem, along the lines of Altonji and Matzkin (2005), may be feasible. Alternatively, a linear-index approach (to simplify how x_{it} and/or x_i enter into $f_{\tau,t}$) may prove useful. Another important topic for future research is relaxation of strict exogeneity. Violations of strict exogeneity have been considered extensively in the conditional-expectation panel-data models. For linear panel-data models, instrumental variables estimation is the usual solution to violations of the strict exogeneity assumption, but such an approach is not likely to carry over to conditional-quantile models.

Acknowledgments

An earlier version of this paper circulated under the title, “The effects of smoking and prenatal care on birth outcomes: evidence from quantile estimation on panel data.” Bill O’Brien, Patricia Starzyk, and Dr. Beth Mueller offered invaluable assistance in providing access to the Washington State Longitudinal Birth Database. The authors are also grateful to Christopher Mrela of the Arizona Department of Health Services for providing birth data. The terms of the data-sharing agreements with these two states do not allow release of this data. The authors would also like to thank Roger Koenker and Daniel Morillo, who wrote the RQ 1.0 package for Ox that was used as the starting point for the quantile estimation in this paper. An Associate Editor and two referees provided extremely useful comments that vastly improved this paper. The first author acknowledges financial support from a Kinley Trust Grant (Purdue University), the Robert Wood Johnson Foundation, and the National Science Foundation (SES-0451660). The second author acknowledges financial support from the Danish National Research Foundation and the Danish Social Sciences Research Council (grant 275-06-0105).

Appendix A: Details on hypothesis testing

This section of the Appendix provides details on the hypothesis tests conducted in Section 4.2.2.

- (*Test of correlated random effects*) Test of $H_0 : \lambda_{\tau_i}^1 = 0 \wedge \lambda_{\tau_i}^2 = 0$ simultaneously $\forall i \in \{1, \dots, K\}$ and $\forall \tau \in \{\tau_1, \tau_2, \dots, \tau_p\}$. Define

$${}_{p(L+1) \times p(2K+L+2)} R' \equiv \left[\begin{array}{cc} I_{p \times p} \otimes \left[\begin{array}{cc} 1 & O_{1 \times (2K+L+1)} \end{array} \right] \\ I_{p \times p} \otimes \left[\begin{array}{cc} O_{(L+1) \times 1} & \begin{array}{cc} I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{array} \end{array} \right] \end{array} \right],$$

and use $M = 2pK$.

- (*Test of the equality of the “effect vector”*) Test of $H_0 : \beta_{\tau_1 i} = \beta_{\tau_2 i} = \dots = \beta_{\tau_p i}$ simultaneously for $\forall i \in \{0, 1, 2, \dots, L\}$. Let i_p be a $(p, 1)$ vector of ones. To perform this test, define

$$R' \equiv \left[\begin{array}{cc} I_{p \times p} \otimes \left[\begin{array}{cc} 1 & O_{1 \times ((L+1)+2K)} \end{array} \right] \\ I_{p \times p} \otimes \left[\begin{array}{cc} O_{2K \times (L+2)} & I_{2K \times 2K} \end{array} \right] \\ i'_p \otimes \left[\begin{array}{cc} O_{(L+1) \times 1} & \begin{array}{cc} I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{array} \end{array} \right] \end{array} \right]$$

and use $M = (p-1)(L+1)$.

- (*Test of the equality of individual variables’ effects (single parameter)*) Test of $H_0 : \beta_{\tau_1 i} = \beta_{\tau_2 i} = \dots = \beta_{\tau_p i}$ for a single $i \in \{0, 1, 2, \dots, L\}$. Let

$$\begin{aligned} E_1 &\equiv I_{p \times p} \otimes \left[\begin{array}{cc} 1 & O_{1 \times ((L+1)+2K)} \end{array} \right], \\ E_2 &\equiv I_{p \times p} \otimes \left[\begin{array}{cc} O_{2K \times (L+2)} & I_{2K \times 2K} \end{array} \right], \\ E_3 &\equiv i'_{p-1} \otimes \left[\begin{array}{cc} O_{(L+1) \times 1} & \begin{array}{cc} D_{ii, (L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{array} \end{array} \right], \end{aligned}$$

and

$$E_4 \equiv \begin{bmatrix} O_{(L+1) \times 1} & I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} & E_3 \\ O_{(p-1)L \times 1} & O_{(p-1)L \times (L+1)} & O_{(p-1)L \times 2K} & I_{(p-1) \times (p-1)} \otimes S_{-i} \end{bmatrix},$$

where

$$S \equiv \begin{bmatrix} O_{(L+1) \times 1} & I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{bmatrix},$$

and S_{-i} is equal to S without the i 'th row. $D_{ii, (L+1) \times (L+1)}$ is a matrix of zeros except for the entry (i, i) which equals unity. Then, the test of H_0 can be performed by defining $R \equiv (E'_1, E'_2, E'_4)'$, with $M = p - 1$.

- (*Test of the equality of individual variables' effects (joint test of two parameters)*) Test of $H_0 : \beta_{\tau_1 i} = \beta_{\tau_2 i} = \dots = \beta_{\tau_p i} \wedge \beta_{\tau_1 j} = \beta_{\tau_2 j} = \dots = \beta_{\tau_p j}$ for $i, j \in \{0, 1, 2, \dots, L\}$ and $i \neq j$. Let

$$\begin{aligned} E_1 &\equiv I_{p \times p} \otimes \begin{bmatrix} 1 & O_{1 \times ((L+1)+2K)} \end{bmatrix}, \\ E_2 &\equiv I_{p \times p} \otimes \begin{bmatrix} O_{2K \times (L+2)} & I_{2K \times 2K} \end{bmatrix}, \\ E_3 &\equiv i'_{p-1} \otimes \begin{bmatrix} O_{(L+1) \times 1} & D_{(ii, jj) (L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{bmatrix}, \end{aligned}$$

and

$$E_4 \equiv \begin{bmatrix} O_{(L+1) \times 1} & I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} & E_3 \\ O_{(p-1)(L-1) \times 1} & O_{(p-1)(L-1) \times (L+1)} & O_{(p-1)(L-1) \times 2K} & I_{(p-1) \times (p-1)} \otimes S_{-ij} \end{bmatrix},$$

where

$$S \equiv \begin{bmatrix} O_{(L+1) \times 1} & I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K} \end{bmatrix},$$

and S_{-ij} is equal to S without rows i and j . $D_{(ii, jj) (L+1) \times (L+1)}$ is a matrix of zeros except for the entries (i, i) and (j, j) which both equal unity. To test H_0 , define $R \equiv (E'_1, E'_2, E'_4)'$ and use $M = 2(p - 1)$.

Appendix B: Additional results

This section of the Appendix contains the Washington results for the estimates of λ_τ^1 and λ_τ^2 (for $\tau \in \{0.10, 0.25, 0.50, 0.75, 0.90\}$) in Tables 5 and 6, respectively.

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Table 5: Panel-Data Estimation Results for λ_{τ}^1 , Washington Data. The dependent variable is birthweight (in grams). The coefficients represent the relationship between the covariates and the first-birth component of the correlated random effect.

	Quantile regressions					OLS
	10%	25%	50%	75%	90%	
Male child	-30.70*** (7.66)	-28.68*** (5.47)	-25.51*** (4.88)	-24.21*** (5.61)	-18.24** (7.30)	-23.95*** (4.50)
Age	-4.50 (14.77)	-15.19 (9.42)	-4.64 (8.98)	-1.27 (9.23)	6.72 (12.06)	-8.29 (8.37)
Age ²	0.222 (0.277)	0.341* (0.180)	0.151 (0.174)	0.017 (0.180)	-0.153 (0.231)	0.224 (0.160)
Education	-13.98 (15.75)	11.77 (11.43)	8.73 (11.39)	14.02* (8.51)	26.44** (11.75)	11.72 (8.58)
Education ²	0.613 (0.672)	-0.290 (0.467)	-0.321 (0.460)	-0.464 (0.380)	-0.811 (0.534)	-0.360 (0.363)
Married	-7.62 (15.06)	4.09 (9.89)	-7.19 (8.65)	-3.28 (10.02)	-2.29 (13.90)	-6.32 (8.27)
No prenatal care	103.15 (86.87)	97.91** (46.97)	40.78 (45.18)	52.16 (47.98)	-71.85 (54.24)	34.70 (39.72)
2nd-trimester care	23.91 (14.63)	25.88** (10.54)	30.29*** (8.66)	12.18 (9.91)	15.69 (12.98)	20.56** (8.43)
3rd-trimester care	69.72** (29.96)	20.88 (23.28)	20.51 (22.15)	24.55 (25.24)	-0.73 (33.56)	31.31 (19.93)
Smoke	-101.93*** (16.34)	-61.35*** (12.88)	-52.67*** (10.23)	-74.32*** (11.43)	-69.53*** (14.73)	-70.28*** (9.62)
Drink	0.07 (30.75)	-3.04 (21.46)	-26.93 (21.41)	-15.49 (25.15)	-16.20 (28.79)	-15.45 (18.92)
# prenatal visits	6.26*** (1.47)	7.18*** (1.10)	6.88*** (0.86)	6.99*** (1.09)	6.50*** (1.29)	5.58*** (0.96)

Bootstrapped standard errors in parentheses, using bootstrap sample size of 20,000 (10,000 pairs) and 1,000 bootstrap replications.

*: significant at 10% level (2-sided); **: 5% level; ***: 1% level.

Table 6: Panel-Data Estimation Results for λ_{τ}^2 , Washington Data. The dependent variable is birthweight (in grams). The coefficients represent the relationship between the covariates and the second-birth component of the correlated random effect.

	Quantile regressions					OLS
	10%	25%	50%	75%	90%	
Male child	5.77 (7.83)	-2.60 (5.48)	-9.28* (5.05)	-9.04* (5.48)	-7.66 (7.06)	-5.66 (4.53)
Age	78.18*** (14.10)	57.53*** (10.06)	54.65*** (9.25)	51.34*** (9.24)	61.12*** (12.79)	65.83*** (8.38)
Age ²	-1.328*** (0.246)	-0.973*** (0.177)	-0.921*** (0.164)	-0.820*** (0.166)	-0.984*** (0.227)	-1.088*** (0.149)
Education	0.04 (19.97)	-2.70 (12.80)	-10.04 (10.41)	1.40 (9.49)	2.31 (12.28)	1.80 (9.23)
Education ²	0.375 (0.782)	0.441 (0.508)	0.544 (0.429)	0.035 (0.401)	0.182 (0.534)	0.193 (0.378)
Married	17.64 (14.46)	10.59 (12.10)	7.17 (9.12)	3.28 (10.54)	10.50 (14.46)	7.76 (8.78)
No prenatal care	-84.39 (103.30)	-101.96* (53.07)	-100.54* (54.15)	-81.13 (58.07)	-135.76 (98.83)	-66.57 (55.43)
2nd-trimester care	3.87 (14.09)	12.69 (10.37)	14.43 (9.56)	2.53 (10.25)	-5.51 (13.06)	11.53 (8.18)
3nd-trimester care	4.32 (38.55)	-39.33 (27.14)	7.65 (25.40)	-17.88 (23.27)	-30.06 (31.01)	-8.17 (20.40)
Smoke	-86.33*** (16.56)	-83.49*** (12.97)	-69.97*** (10.95)	-74.34*** (10.51)	-61.31*** (15.58)	-78.52*** (9.30)
Drink	38.86 (38.29)	11.79 (28.74)	9.67 (19.71)	-11.79 (23.26)	31.91 (32.37)	23.65 (20.25)
# prenatal visits	-7.19*** (1.56)	-4.21*** (1.12)	-3.36*** (0.85)	-3.34*** (0.92)	-3.22** (1.33)	-4.64*** (0.95)

Bootstrapped standard errors in parentheses, using bootstrap sample size of 20,000 (10,000 pairs) and 1,000 bootstrap replications.

*: significant at 10% level (2-sided); **: 5% level; ***: 1% level.

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