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The Effects of Birth Inputs on Birthweight: Evidence From Quantile Estimation on Panel Data

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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 a mother smokes, for instance, is likely to be correlated with unobserved characteristics of the mother. This article 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 to estimate the effects of smoking and other observables (number of prenatal-care visits, years of education, and so on) on the entire birthweight distribution.

KEY WORDS: Birthweight; Panel data; Quantile regression.

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 2,500 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, Chay, and Lee (2005) reported 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, Klein, and Taylor 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 (Currie and Hyson 1999; Behrman and Rosenzweig 2004; Case, Fertig, and Paxon 2005).

Although it has received less attention in the economics literature, high-birthweight outcomes can also represent adverse outcomes. For instance, babies weighing more than 4,000 grams [classified as high birthweight (HBW)] and especially those weighing more than 4,500 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 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 (Permutt and Hebel 1989; Evans and Ringel 1999), prenatal care (Currie and Gruber 1996; Joyce 1999; Evans and Lien 2005), and air pollution (Chay and Greenstone 2003a,b) 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 (Rosenzweig and Wolpin 1991; Currie and Moretti 2002; Royer 2004; Abrevaya 2006). 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) provided an explicit estimation strategy to deal with such feedback effects (using data on at least three births per mother). Abrevaya (2006) showed that feedback effects are likely to cause the estimated (negative) smoking effect to be too large in magnitude.

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Because 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., 2,500 grams for LBW and 1,500 grams for "very low birthweight"). As an alternative, this article 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 2,500 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 the quantile approach have drawbacks: (1) the probit approach is inherently discontinuous and offers only predictions of LBW versus non-LBW outcomes, and (2) 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 probitbased 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 article.

Previous quantile-estimation approaches to estimating birthoutcome 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; Chernozhukov 2005) used cross-sectional
federal natality data and found that various observables have
significantly stronger associations with birthweight at lower
quantiles of the birthweight distribution; unfortunately, one
cannot interpret these "effects" as causal because the estimation has a purely reduced-form structure that does not account
for unobserved heterogeneity.

The outline of the article 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 article. There are some interesting differences between the panel-data and crosssectional 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.

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

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

2.1.1 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

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

under the following assumption:

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

2.1.2 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:

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

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

$$y_{m2} = \psi + x'_{m1}\lambda_1 + x'_{m2}(\beta + \lambda_2) + v_m + u_{m2}.$$
 (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, sec. 11.3). The vector x_{m1} affects y_{m1} through two channels: (1) a direct effect (expressed by the $x'_{m1}\beta$ term) and (2) 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

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

the "meaning" of β is given by the following equation:

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

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

Estimation of Effects on Conditional Quantiles With Panel Data

For conditional quantiles, a simple differencing strategy is infeasible because 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 (8). In particular, the effects of the observables on a given conditional quantile are given by

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

and

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

For example, the difference in (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 (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 nontrivial to explicitly determine the conditional quantile models. Consider, for example, the simple case in which the datagenerating process is given by (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)]:

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

$$Q_{\tau}(y_{m2}|x_m) = \psi_{\tau}^2 + x'_{m1}\lambda_1 + x'_{m2}(\beta + \lambda_2).$$
 (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 (11) and (12)] only arises in very special cases. In general, the conditional quantile functions involve more complicated nonlinear expressions and, in fact, cannot 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 article, 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} :

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

$$Q_{\tau}(y_{m2}|x_m) = \phi_{\tau}^2 + x'_{m1}\lambda_{\tau}^1 + x'_{m2}\theta_{\tau}^2.$$
 (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 crosssectional 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; Bassett and Chen 2001). Angrist, Chernozhukov, and Fernandez-Val (2006) provided a framework for analyzing misspecification of the conditional quantile function. Although beyond the scope of this article, it would be interesting to apply their methodology to the paneldata 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 nonlinear; in this case, (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 article, 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

$$\beta_{\tau} = \theta_{\tau}^{1} - \lambda_{\tau}^{1} = \theta_{\tau}^{2} - \lambda_{\tau}^{2}. \tag{15}$$

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

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

$$= \phi_{\tau}^1 + x'_{m1}\beta_{\tau} + x'_{m1}\lambda_{\tau}^1 + x'_{m2}\lambda_{\tau}^2, \qquad (16)$$

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

 $= \phi_{\tau}^2 + x'_{m2}\beta_{\tau} + x'_{m1}\lambda_{\tau}^1 + x'_{m2}\lambda_{\tau}^2.$

(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

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

$$(18)$$

as the left-side and right-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 cannot be included explicitly in x because the associated components of $\beta_{\tau}, \lambda_{\tau}^1$, 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 right-side 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. Because 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, cannot 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) cited only a single panel-data application. The cited study by Chay (1995) used quantile regression on longitudinal earnings data to estimate the effect of the 1964 Civil Rights Act on the black—white earnings differential. Chay (1995) allowed 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) involved censored earnings data, so that quantileregression 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 article.

A more recent application of quantile regression on panel data is found in Arias, Hallock, and Sosa-Escudero (2001), who estimated the returns to schooling using twins data. To deal with the unobserved "family effect," the authors included 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 that of Royer (2004), who applied a correlated random-effects model to maternally linked data from Texas. Royer (2004) estimated 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 relaxed the strict exogeneity assumption (required for consistency of the fixedeffects 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, p. 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, and so on), birth outcomes (birthweight, gestation, and so on), and prenatal care (number of prenatal visits, smoking status, and so on) 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:

 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, (1) an exact match on mother's name, mother's date of birth, mother's race, and mother's state of birth was required, and (2) 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, (1) 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 (2) consistency of birth parity and the reported interval-since-lastbirth 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 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 issues 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 the Arizona data. The Washington data have several advantages over the Arizona data: (1) the matching of siblings for the Washington data is of higher quality due to the use of mothers' names, (2) the Washington data are not restricted to siblings with the same fathers, and (3) the Washington data include 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)

Table 1. Descriptive statistics, Washington and Arizona birth panels

	Wash	ington	Arizona		
Variable	1st child	2nd child	1st child	2nd child 3,427 ₍₅₀₅₎	
Birthweight (in grams)	3,442 ₍₅₂₃₎	3,530 ₍₅₃₆₎	3,339 ₍₅₁₇₎		
Male child	.515	.511	.520	.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	.751	.853	.780	.886	
No prenatal care	.004	.003	.005	.006	
1st-trimester care	.879	.895			
2nd-trimester care	.107	.093			
3rd-trimester care	.014	.012			
Smoke	.143	.132	.049	.044	
Drink	.017	.014	.009	.007	
# prenatal visits	12.06 _(3.53)	11.63 _(3.25)	11.83 _(3.59)	11.73 _(3.55)	
Quantiles of birthweight:					
10% quantile	2,807	2,892	2,750	2,863	
25% quantile	3,146	3,220	3,061	3,146	
50% quantile	3,458	3,543	3,373	3,445	
75% quantile	3,770	3,855	3,685	3,742	
90% quantile	4,060	4,167	3,968	4,040	
# of observations	45,067	45,067	56,201 56,2		

and 56,201 Arizona mothers (112,402 births). Sample averages are reported for all variables, as well as standard deviations for the nonindicator variables. The "Smoke" ("Drink") variable is equal to 1 if the mother reported smoking (drinking alcohol) during pregnancy. Although alcohol consumption during pregnancy is known to be severely underreported, the "Drink" variable is included in the regressions as it may be useful as a proxy for other unobservables. For Washington, the four prenatalcare categories ("No prenatal care," "1st-trimester care," "2ndtrimester 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 1 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 overrepresent nonsmokers. 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 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 2,500 grams corresponds to the 3–5% quantiles of the unconditional birthweight distributions, whereas the HBW cutoff of 4,000 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 (Sec. 4.1.1). The Arizona results are reported in a graphical format comparable to the Washington results (Sec. 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 $\tau \in \{.10, .25, .50, .75, .90\}$ (along with least squares estimates for comparison), although the figures presented in this section consider marginal effects at 2% intervals (specifically, $\tau \in \{.04, .06, ..., .94, .96\}$). Throughout this section, the dependent variable of interest is birthweight (measured in grams). 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 paneldata results, respectively. The model specification includes the variables summarized in Table 1, along with an indicator variable 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 (1) the trimester of the first prenatal visit (if any) and (2) 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 prenatalcare 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 (1) found that bimodal residuals result from a standard 2SLS regression of birthweight and (2) used 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

Table 2. Cross-sectional estimation results, Washington data

	Quantile regressions					
	10%	25%	50%	75%	90%	OLS
Second child	100.65***	92.87***	93.86***	99.99***	110.37***	98.95***
	(7.24)	(4.96)	(4.40)	(4.89)	(6.84)	(4.04)
Male child	87.22***	115.87***	128.34***	142.70***	160.83***	124.34***
	(6.25)	(4.33)	(3.85)	(4.25)	(5.65)	(3.57)
Age	19.30***	13.81***	7.39**	7.92**	5.47	12.57***
-	(6.35)	(4.02)	(3.58)	(3.93)	(5.17)	(3.41)
Age^2	385***	258***	131**	122*	070	228***
	(.115)	(.070)	(.064)	(.070)	(.091)	(.061)
Education	31.02**	22.46**	30.23***	28.81***	23.87**	26.94***
	(14.32)	(8.80)	(7.70)	(6.71)	(10.45)	(7.16)
Education ²	744	603*	927***	987***	793**	789***
	(.525)	(.324)	(.285)	(.250)	(.388)	(.264)
Married	36.58***	27.52***	26.74***	22.41***	16.20*	28.11***
	(10.22)	(7.61)	(6.03)	(6.91)	(9.19)	(6.23)
No prenatal care	-324.75*	-22.76	-36.05	15.54	176.44**	-34.57
-	(177.34)	(55.29)	(41.13)	(44.24)	(74.27)	(47.90)
2nd-trimester care	37.34***	27.93***	24.72***	31.58***	37.96***	38.73***
	(11.86)	(8.41)	(7.00)	(8.27)	(10.43)	(6.48)
3rd-trimester care	106.60***	59.36***	37.26*	26.07	24.40	66.39***
	(30.75)	(20.43)	(19.79)	(18.81)	(23.84)	(15.96)
Smoke	-186.05***	-182.29***	-178.57***	-176.05***	-160.02***	-177.97***
	(11.40)	(7.64)	(6.23)	(7.51)	(9.92)	(6.16)
Drink	-21.80	-20.08	-7.27	-15.01	17.03	3.89
	(28.14)	(22.67)	(16.10)	(20.00)	(26.55)	(16.47)
# prenatal visits	19.33***	16.52***	15.11***	14.82***	13.92***	18.46***
	(1.35)	(.89)	(.76)	(.82)	(1.10)	(.85)

NOTE: The dependent variable is birthweight (in grams). 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.

Table 3. Panel-data estimation results (β_{τ}) , Washington data

	Quantile regressions					
	10%	25%	50%	75%	90%	OLS
Second child	137.46***	113.17***	109.76***	113.56***	125.63***	122.32***
	(11.75)	(8.30)	(7.03)	(7.69)	(11.38)	(5.99)
Male child	100.67***	131.64***	146.69***	160.12***	173.35***	138.75***
	(7.76)	(5.15)	(4.40)	(4.82)	(6.64)	(3.68)
Age	-38.73***	-20.88***	-30.13***	-31.52***	-48.18***	-33.06***
	(11.26)	(7.00)	(6.27)	(6.61)	(9.47)	(5.31)
Age^2	.515***	.290**	.467***	.529***	.845***	.483***
C	(.197)	(.120)	(.109)	(.117)	(.161)	(.092)
Education	35.54*	17.26	27.22***	17.96**	5.97	19.16**
	(18.25)	(11.91)	(10.23)	(8.46)	(13.47)	(7.62)
Education ²	-1.436*	881*	-1.029**	761**	594	851**
	(.762)	(.503)	(.433)	(.388)	(.607)	(.334)
Married	39.71**	15.49	27.99***	21.13*	11.41	27.87***
	(17.26)	(11.71)	(9.21)	(11.32)	(16.05)	(8.30)
No prenatal care	-323.39*	-13.77	1.66	26.03	271.21***	-18.08
•	(172.94)	(60.05)	(51.94)	(54.87)	(76.06)	(49.01)
2nd-trimester care	26.80*	6.63	20	22.95**	34.39**	22.19***
	(14.48)	(10.46)	(8.72)	(10.18)	(13.52)	(6.93)
3nd-trimester care	62.87*	71.79***	31.75	30.50	39.22	55.73***
	(34.96)	(25.25)	(22.67)	(23.91)	(32.04)	(17.32)
Smoke	-24.58	-60.64***	-81.43***	-57.70***	-56.19***	-56.86***
	(19.47)	(14.23)	(11.45)	(12.09)	(17.58)	(9.10)
Drink	-44.24	-32.19	4.29	-1.57	9.25	2.11
	(35.84)	(25.36)	(20.00)	(24.77)	(29.79)	(17.11)
# prenatal visits	20.01***	14.79***	12.70***	12.32***	12.65***	17.48***
-	(1.59)	(1.10)	(.88)	(1.01)	(1.43)	(.93)

NOTE: The dependent variable is birthweight (in grams). 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.

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 disadvantaged 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 the 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 paneldata results in Table 3, unobserved heterogeneity is modeled as in Section 2.2 [see (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 Appendix B (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 the 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).

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

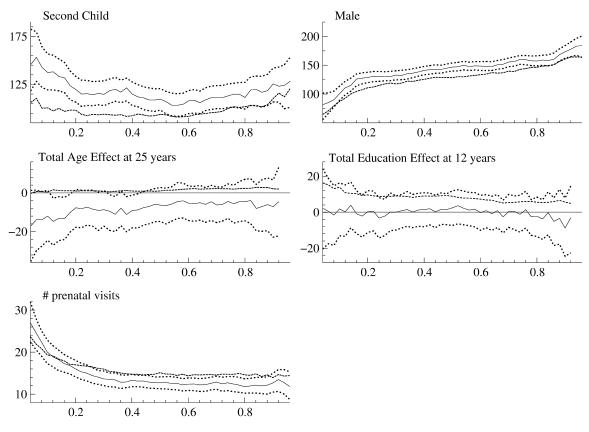


Figure 1. Part 1 of the estimated marginal effects on the conditional quantiles for Washington births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates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

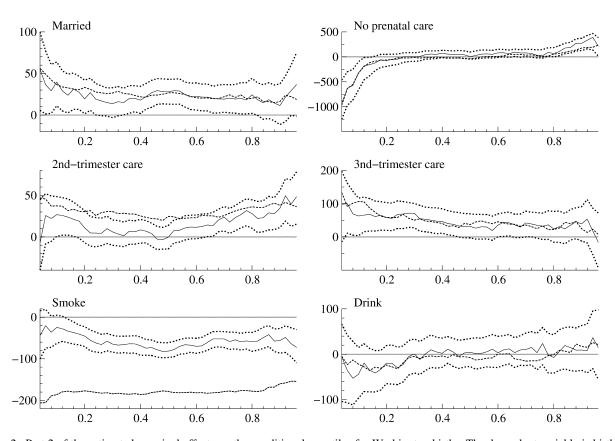


Figure 2. Part 2 of the estimated marginal effects on the conditional quantiles for Washington births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates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

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: (1) the amount of within-mother variation in education is quite small, with the average change in education for the sample being about .2 years, and (2) 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 because 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, and so on) that would lead to a more favorable birth outcome.

Prenatal-care visits: Lack of prenatal care is found to have significant negative effects at the 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 previously, 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 secondtrimester-care variable; the cross-sectional estimates are statistically significant at all quantiles and range from 25 grams 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 for 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 crosssectional 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; Lien and Evans 2005) and experimental estimates (e.g., Permutt and Hebel 1989). These studies have estimated causal (IV) effects of smoking on birthweight that 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); because 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 because the effect of misclassification is more severe in the paneldata case (see, e.g., Freeman 1984; 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.

"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 status as a covariate could also be an "overcontrolling" factor.

To empirically assess the possible importance of "overcontrolling," we reran the Washington regressions under two alternative specifications: (1) original specification with number of prenatal visits dropped, and (2) original specification with number of prenatal visits, smoking status, and drinking status dropped. Figure 3 reports the estimates on the prenatalcare-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 firsttrimester 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

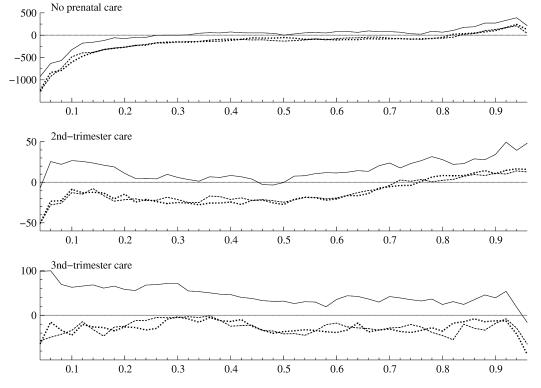


Figure 3. Results for initial-prenatal-visit variables under different specifications (Washington data). The solid line corresponds to the full specification, the dotted line to the specification in which "# of prenatal visits" is dropped, and the dashed line to the specification in which "# of prenatal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

that the estimates on the other variables (those not shown in Fig. 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 (Fig. 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 previously 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 crosssectional 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 .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

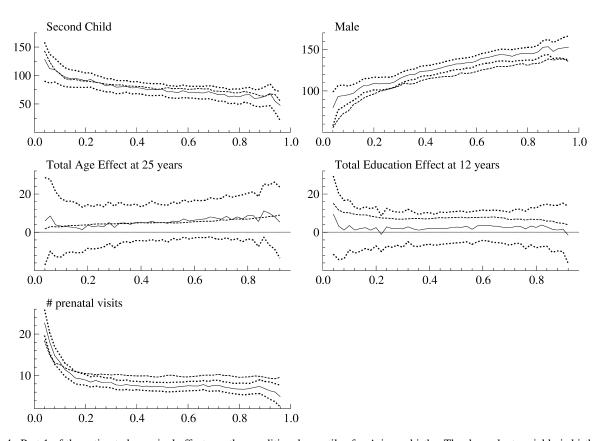


Figure 4. Part 1 of the estimated marginal effects on the conditional quantiles for Arizona births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates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

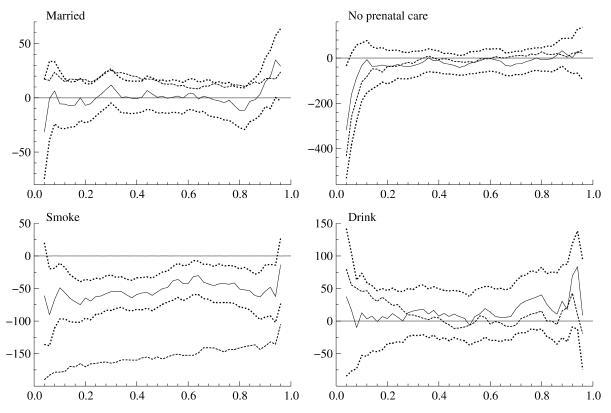


Figure 5. Part 2 of the estimated marginal effects on the conditional quantiles for Arizona births. The dependent variable is birthweight (in grams). The solid line indicates the panel-data estimates, the dotted lines are 90% confidence bands for the panel-data estimates, and the dashed line indicates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

finding is that the father's date of birth is required to match for both births of an Arizona mother (see Sec. 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.

• Because of 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.

4.2 Hypothesis Testing

In this section, we discuss the results of several hypothesis tests that were used 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, \ldots, τ_p denoting the quantiles. For a given quantile τ , individual elements of the parameter vectors $\beta_{\tau}, \lambda_{\tau}^{1}$,

and λ_{τ}^2 [recall the model in (16) and (17)] are referenced by subscripts as follows:

$$\begin{split} \beta_{\tau} &= (\beta_{\tau 1}, \dots, \beta_{\tau L})', \\ \lambda_{\tau}^1 &= (\lambda_{\tau 1}^1, \dots, \lambda_{\tau K}^1)', \qquad \lambda_{\tau}^2 = (\lambda_{\tau 1}^2, \dots, \lambda_{\tau K}^2)', \end{split}$$

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

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

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

$$\gamma \equiv \left(\gamma_{\tau_1}', \gamma_{\tau_2}', \dots, \gamma_{\tau_n}'\right)' \tag{20}$$

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

In the MD framework, the "restricted" parameter estimator is defined as

$$\widehat{\gamma}^{R} = \underset{\gamma^{R} \in \Theta}{\operatorname{arg\,min}} (\widehat{\gamma} - R\gamma^{R})' \widehat{A}^{-1} (\widehat{\gamma} - R\gamma^{R}), \tag{21}$$

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

$$\widehat{\gamma}^R = (R'\widehat{A}^{-1}R)^{-1}(R'\widehat{A}^{-1}\widehat{\gamma}). \tag{22}$$

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

$$\operatorname{var}(\widehat{\gamma}^R) = (R'\widehat{A}^{-1}R)^{-1}.$$
 (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:

$$(\widehat{\gamma} - R\widehat{\gamma}^R)'\widehat{A}^{-1}(\widehat{\gamma} - R\widehat{\gamma}^R) \xrightarrow{d} \chi_M^2, \tag{24}$$

where M is the number of restrictions [i.e., M = rows(R) - columns(R)]. Appendix A 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 $\tau \in \{.10, .25, .50, .75, .90\}$, 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 $\{.10, .25, .50, .75, .90\}$. 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 (e.g., 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 and 3. For the marriage indicator, for instance, the null hypothesis would be $H_0: \beta_{\tau=.10}^{married} = \beta_{\tau=.25}^{married} =$ $\beta_{\tau=.50}^{married} = \beta_{\tau=.75}^{married} = \beta_{\tau=.90}^{married}$. 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 the 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 and 2 and Figures 4 and 5. Two variables (malechild indicator and number of prenatal visits) vary significantly across the quantiles for both the cross-sectional and panel specifications. The effect of the no-prenatalcare indicator also varies significantly (p-value of .010 in the cross section and .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

Table 4. Tests of marginal-effect equality across quantiles

	Washington		Arizona	
	Cross section	Panel data	Cross section	Panel data
Second child	.135	.113	.000	.049
Male child	.000	.000	.000	.000
Age, age ² jointly	.006	.088	.000	.944
Education, education ² jointly	.014	.336	.002	.927
Married	.574	.457	.565	.739
No prenatal care	.010	.004	.442	.874
2nd-trimester care	.620	.059		
3nd-trimester care	.154	.638		
Smoke	.257	.063	.236	.834
Drink	.578	.602	.346	.905
# prenatal visits	.005	.000	.005	.000

NOTE: 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 .10, .25, .50, .75, and .90. Results are based upon 1,000 bootstrap replications.

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 quite high (.396), whereas the *p*-value (.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 {.10, .25, .50, .75, .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 Sec. 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, because data on two births per mother are available, y_{m1} (first-birth birthweight) is included as a right-side variable for the second-birth equation. (Considering matched panel data with three births per mother reduces the sample size to an extent that 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 .45 at the lower quantiles and gradually decreasing to around .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 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. Because 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 paneldata 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 nonsmokers). 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 (1) more additional education than possible given the change in age ($\Delta Education > \Delta Age + 1$), or (2) 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 (1), 5,117 in group (2), and 8 in both. Rerunning the panel-data regressions on the less error-ridden subsample yielded results extremely similar 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, e.g., 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 = .1 and q = .2) of observed smokers as nonsmokers and then computed the panel-data estimates (at $\tau \in \{.1, .2, ..., .8, .9\}$. (We assume no misclassification of nonsmokers as smokers.) Abrevaya and Dahl (2006) reported 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 that 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 because it does not allow for (1) a mother's reporting errors to be correlated over time, or (2) reporting errors to be correlated with other observables. The first issue is considered by Abrevaya (2006), who showed 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.

DISCUSSION OF THE THEORETICAL MODEL

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

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

$$c_i = \phi(x_i) + v_i$$
, where $E(v_i|x_i) = 0$. (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

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

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)
$$v_i$$
 is independent of x_i , (28)

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

Assumption (B1) is commonly used in estimation of nonlinear panel-data models with correlated random effects. The datagenerating 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 heteroscedasticity. Note that assumption (B2) also imposes a form of strict exogeneity because 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

$$Q_{\tau}(v_i + u_{it}|x_i) = Q_{\tau}(v_i + u_{it}|x_{it}) \equiv f_{\tau,t}(x_{it}).$$
 (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 (30) into (27) yields

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

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

Example (Linear-scale heteroscedasticity). 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 (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 $\psi^t_{\tau} + x'_{i1}\lambda^t_{\tau} + \cdots + x'_{iT}\lambda^T_{\tau}$ is used to approximate $\phi(x_i)$. The conditional-quantile function in (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.

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APPENDIX A: DETAILS ON HYPOTHESIS TESTING

This 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 $\forall i \in \{1, ..., K\}$ and $\forall \tau \in \{\tau_1, \tau_2, ..., \tau_p\}$. Define

$$\begin{split} R'_{p(L+1)\times p(2K+L+2)} \\ &\equiv \begin{bmatrix} I_{p\times p} \otimes \begin{bmatrix} 1 & O_{1\times(2K+L+1)} \end{bmatrix} \\ I_{p\times p} \otimes \begin{bmatrix} O_{(L+1)\times 1} & I_{(L+1)\times(L+1)} & O_{(L+1)\times 2K} \end{bmatrix} \end{bmatrix}, \\ \text{and use } M = 2pK. \end{split}$$

• (Test of the equality of the "effect vector".) Test of $H_0: \beta_{\tau_1 i} = \beta_{\tau_2 i} = \cdots = \beta_{\tau_p i}$ simultaneously $\forall i \in \{0, 1, 2, \ldots, L\}$. Let i_p be a (p, 1) vector of 1's. To perform this test, define

$$R' \equiv \begin{bmatrix} I_{p \times p} \otimes [1 & O_{1 \times ((L+1) + 2K)}] \\ I_{p \times p} \otimes [O_{2K \times (L+2)} & I_{2K \times 2K}] \\ i'_p \otimes [O_{(L+1) \times 1} & I_{(L+1) \times (L+1)} & O_{(L+1) \times 2K}] \end{bmatrix},$$
 and use $M = (p-1)(L+1)$.

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

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

and

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

where

$$S \equiv [O_{(L+1)\times 1} \quad I_{(L+1)\times (L+1)} \quad O_{(L+1)\times 2K}],$$

and S_{-i} is equal to S without the ith 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 $H_0: \beta_{\tau_1 i} = \beta_{\tau_2 i} = \cdots = \beta_{\tau_p i} \wedge \beta_{\tau_1 j} = \beta_{\tau_2 j} = \cdots = \beta_{\tau_p j}$ for $i, j \in \{0, 1, 2, \dots, L\}$ and $i \neq i$. Let

$$E_1 \equiv I_{p \times p} \otimes [1 \quad O_{1 \times ((L+1)+2K)}],$$

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

and

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

where

$$S \equiv [O_{(L+1)\times 1} \quad I_{(L+1)\times (L+1)} \quad O_{(L+1)\times 2K}],$$

and S_{-ij} is equal to S without rows i and j. Here $D_{(ii,jj)(L+1)\times(L+1)}$ 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 appendix contains the Washington results for the estimates of λ_{τ}^1 and λ_{τ}^2 (for $\tau \in \{.10, .25, .50, .75, .90\}$) in Tables 5 and 6, respectively.

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Table 5. Panel-data estimation results for λ_{τ}^{1} , Washington data

	Quantile regressions					
	10%	25%	50%	75%	90%	OLS
Male child	-30.70***	-28.68***	-25.51***	-24.21***	-18.24**	-23.95***
	(7.66)	(5.47)	(4.88)	(5.61)	(7.30)	(4.50)
Age	-4.50	-15.19	-4.64	-1.27	6.72	-8.29
	(14.77)	(9.42)	(8.98)	(9.23)	(12.06)	(8.37)
Age^2	.222	.341*	.151	.017	153	.224
-	(.277)	(.180)	(.174)	(.180)	(.231)	(.160)
Education	-13.98	11.77	8.73	14.02*	26.44**	11.72
	(15.75)	(11.43)	(11.39)	(8.51)	(11.75)	(8.58)
Education ²	.613	290	321	464	811	360
	(.672)	(.467)	(.460)	(.380)	(.534)	(.363)
Married	-7.62	4.09	-7.19	-3.28	-2.29	-6.32
	(15.06)	(9.89)	(8.65)	(10.02)	(13.90)	(8.27)
No prenatal care	103.15	97.91**	40.78	52.16	-71.85	34.70
•	(86.87)	(46.97)	(45.18)	(47.98)	(54.24)	(39.72)
2nd-trimester care	23.91	25.88**	30.29***	12.18	15.69	20.56**
	(14.63)	(10.54)	(8.66)	(9.91)	(12.98)	(8.43)
3nd-trimester care	69.72**	20.88	20.51	24.55	73	31.31
	(29.96)	(23.28)	(22.15)	(25.24)	(33.56)	(19.93)
Smoke	-101.93***	-61.35***	-52.67***	-74.32***	-69.53***	-70.28***
	(16.34)	(12.88)	(10.23)	(11.43)	(14.73)	(9.62)
Drink	.07	-3.04	-26.93	-15.49	-16.20	-15.45
	(30.75)	(21.46)	(21.41)	(25.15)	(28.79)	(18.92)
# prenatal visits	6.26***	7.18***	6.88***	6.99***	6.50***	5.58***
_	(1.47)	(1.10)	(.86)	(1.09)	(1.29)	(.96)

NOTE: 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. 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

	Quantile regressions					
	10%	25%	50%	75%	90%	OLS
Male child	5.77	-2.60	-9.28*	-9.04*	-7.66	-5.66
	(7.83)	(5.48)	(5.05)	(5.48)	(7.06)	(4.53)
Age	78.18***	57.53***	54.65***	51.34***	61.12***	65.83***
	(14.10)	(10.06)	(9.25)	(9.24)	(12.79)	(8.38)
Age^2	-1.328***	973***	921***	820***	984***	-1.088***
	(.246)	(.177)	(.164)	(.166)	(.227)	(.149)
Education	.04	-2.70	-10.04	1.40	2.31	1.80
	(19.97)	(12.80)	(10.41)	(9.49)	(12.28)	(9.23)
Education ²	.375	.441	.544	.035	.182	.193
	(.782)	(.508)	(.429)	(.401)	(.534)	(.378)
Married	17.64	10.59	7.17	3.28	10.50	7.76
	(14.46)	(12.10)	(9.12)	(10.54)	(14.46)	(8.78)
No prenatal care	-84.39	-101.96*	-100.54*	-81.13	-135.76	-66.57
	(103.30)	(53.07)	(54.15)	(58.07)	(98.83)	(55.43)
2nd-trimester care	3.87	12.69	14.43	2.53	-5.51	11.53
	(14.09)	(10.37)	(9.56)	(10.25)	(13.06)	(8.18)
3nd-trimester care	4.32	-39.33	7.65	-17.88	-30.06	-8.17
	(38.55)	(27.14)	(25.40)	(23.27)	(31.01)	(20.40)
Smoke	-86.33***	-83.49***	-69.97***	-74.34***	-61.31***	-78.52***
	(16.56)	(12.97)	(10.95)	(10.51)	(15.58)	(9.30)
Drink	38.86	11.79	9.67	-11.79	31.91	23.65
	(38.29)	(28.74)	(19.71)	(23.26)	(32.37)	(20.25)
# prenatal visits	-7.19***	-4.21***	-3.36***	-3.34***	-3.22**	-4.64***
	(1.56)	(1.12)	(.85)	(.92)	(1.33)	(.95)

NOTE: 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. 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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