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The consequences and costs of maternal substance abuse in New York City

A pooled time-series, cross-section analysis*

Theodore Joyce

City University of New York, New York, NY, USA and NBER, New York, NY, USA

Andrew D. Racine

Columbia University, New York, NY, USA and NBER, New York, NY, USA

Naci Mocan

University of Colorado at Denver, Denver, CO, USA and NBER, New York, NY, USA

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We use a pooled time-series cross-section of live births in New York City between 1980 and 1989 to investigate the dramatic rise in low birthweight, especially among Blacks, that occurred in the mid 1980s. After controlling for other risk factors, we estimate that the number of excess low birthweight births attributable to illicit substance abuse over this period ranged from approximately 1,482 to 3,359. The increase represents between 3.2 and 7.3% of all LBW over the period resulting in excess neonatal admission costs of between $18 and $41 million.

1. Introduction

Of all the predictors of neonatal and infant morbidity and mortality, none is more powerful than birthweight. Reflecting characteristics of the newborn's genetic endowment, gestational age, and intrauterine environment, birthweight explains more of the variation in health status over the first 12 months of life than any other single factor.

Correspondence to: Theodore Joyce, Ph.D., National Bureau of Economic Research, 269 Mercer Street, 8th Floor, New York, NY 10003, USA.

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In particular low birthweight (LBW, <2,500 grams) is known to be an important herald of adverse neonatal and infant health outcomes including need for neonatal intensive care, prolonged initial hospital stay and likelihood of rehospitalization within the first year of life. Because of its importance to health status and health care costs, much effort has been devoted to analyzing the causes of low birth weight in individuals, and to studying the factors which affect the rate of low birth weight across different populations and over time.

In New York City, an alarming increase in the rate of LBW over the past five years, particularly among Blacks, has been documented recently [Joyce (1990)]. Whereas up until 1984 the LBW rate for non-Hispanic Blacks had averaged around 11% of all singleton live births, by 1988 that figure had risen to more than 13.5%, a 25% increase in little more than two years. During the same decade, LBW rates among Whites also increased though less dramatically than among Blacks. Because LBW rates for all ethnic groups had been decreasing for 20 years by the mid 1980s, the increase in LBW births represents the most serious deterioration in the health of New York City's infant population in a generation, with the possible exception of pediatric HIV disease.

Cross-sectional epidemiologic studies have identified a series of factors associated with the risk of delivering LBW infants. These may be divided into: (1) demographic characteristics of the mother including race, marital status, and age; (2) socio-economic factors such as schooling, income and insurance coverage; (3) obstetrical characteristics of a particular pregnancy such as gestational diabetes, low weight gain, infection and hypertension; and (4) ongoing maternal exposures to a variety of substances including tobacco, alcohol and illicit drugs. To understand the changes that have taken place in LBW in New York City during the 1980s, it is important to determine how such correlates have varied over this period of time.

Of particular interest in this regard has been the explosion in the use of cocaine during the 1980s. From 1985 to 1988 the rate of *reported* maternal cocaine use during pregnancy in New York City increased almost 300% from 5.3 to 21.1 per thousand live births, coinciding with the observed upsurge in LBW rates [Habel et al. (1988)]. Furthermore, in-utero exposure to cocaine has been repeatedly documented to be associated with an increase in the risk of LBW with relative risks calculated to be on the order of three [Handler et al. (1991)]. Finally, mirroring changes in LBW rates, a Black–White differential appears to exist in the pattern of cocaine usage in the city. The reported prevalence of cocaine use among pregnant women in the Black community is twice what it is in the White community though differential reporting bias between these groups, as has been observed elsewhere, may be partially responsible for this finding.

The purpose of the present study is to examine, on a population wide

basis, the link between changes in LBW rates and changes in patterns of substance abuse in New York City over the past decade. In particular, we estimate the excess number of LBW births and the newborn costs of this excess morbidity that can be attributed to the rise in the prenatal use of illicit substances between 1985 and 1989. Toward this aim we estimate a structural health production function with a pooled time-series cross-section of all New York City singleton live births, aggregated by health district, for the ten-year period 1980–1989. With data from New York City birth certificates we analyze separate panels for Black non-Hispanics, White non-Hispanics and Hispanics.

There are several advantages of New York City vital statistics for such an analysis. First, the New York City birth certificate is the only one in the country that has had, for over a decade, a specific indicator of prenatal illicit substance abuse. Other states and vital registration areas did not include such an indicator until the 1988–1989 revision [Freedman et al. (1988)]. Second, New York City was one of the earliest metropolitan areas to identify a dramatic rise in prenatal maternal substance abuse. A universal toxicology analysis of all births delivered at Harlem Hospital between 1985 and 1986 revealed that 11% of the newborns were exposed prenatally to cocaine or its derivative, crack [Phibbs et al. (1991)]. Third, because of the size of New York City's population, we can analyze the experience of over one million births separately by race and ethnicity and across neighborhoods that vary substantially in family income and social stability.

The disadvantage of vital statistics concerns the measurement of the prenatal substance abuse variable. Based on a combination of self-reports to physicians and positive toxicology screens applied selectively at delivery, misclassification is likely to be substantial [Zuckerman et al. (1989); Chasnoff et al. (1990)]. To correct for the likely measurement error in the prenatal substance abuse variable, we employ the rate of drug deaths in each district as an instrument for prenatal substance abuse. Because drug deaths are based on coroners' reports, the incentives for underreporting are less obvious. We argue that the rate of drug deaths should be correlated with the prenatal use of illicit substances, yet uncorrelated with its error.

2. Empirical implemention

2.1. Data

The data include all singleton live births to Black non-Hispanics, White non-Hispanics, and Hispanic residents of New York City between 1980 and 1989 inclusive. Individual birth certificates have been aggregated to the health-center district by year, race, and ethnicity. New York City is divided into 30 health center districts, each with approximately 250,000 residents.

Hence the aggregated data set contains 300 observations (10 years by 30 districts) for each of the three groups.

We estimate a structural health production function with the race- and ethnic-specific rate of LBW as the outcome of interest. One of the advantages of LBW in a time-series context is that infant mortality rates, unlike rates of LBW, have been greatly affected by the steady advances in neonatology [McCormick (1985)]. Yet, there are few empiricial controls for technological change and most applications resort to trend terms. Except for toxolytic agents however, no technological advances have had any appreciable impact on the rate of LBW.

The slow, but steady decline in the rate of LBW experienced in the United States and New York City from the late 1960s until the mid 1980s has been attributed to the increased utilization of appropriate prenatal care, better nutrition, and a declining proportion of births to adolescents [Kleinman and Kessel (1983); National Center for Health Statistics (1980)]. Offsetting these favorable trends has been the rise in out-of-wedlock births and, most recently, the prenatal consumption of illicit substances particularly cocaine and its derivative, crack. Thus, to estimate the relative contribution of prenatal illicit substance use to LBW within the framework of a structural production function, we include by year and by health district the percentage of births to women with no or unknown prenatal care, the percentage of births out-of-wedlock, the percentage of births to women with four or more previous live births, the percentage of births to women who smoked during pregnancy, and the percentage of births to women who used illicit substances prenatally. Illicit substances include cocaine, heroin, methadone, and barbiturates.[1]

Following the recent literature, we limit the inputs to medical, behavioral and obstetrical factors associated with adverse birth outcomes [Rosenzweig and Schultz (1983, 1991); Corman et al (1987); Grossman and Joyce (1990)]. We experimented with specifications including maternal age as a covariate and found little effect for this variable. Previous work has indicated that at the extremes of the age spectrum, women in their forties and adolescents less than 15, some influence on the probability of delivering LBW infants is to be expected. The association between adolescent childbearing and adverse birth outcomes, however, has been related to primarily social and behavioral factors [Institute of Medicine (1985)]. We anticipate that in our regressions much of this effect would be captured at the low end of the age spectrum by the variable representing out-of-wedlock births.[2]

We also exclude socio-economic factors such as education, income, and insurance status since these are fundamentally enabling variables that

[1]We exclude marijuana because it was added specifically to the birth certificate in 1988. Although methadone is not an 'illicit' drug, individuals may use it illicitly.

[2]Details of these regressions are available upon request to the authors.

condition the demand for more proximate inputs such as prenatal care, illicit substances, and other exposures including tobacco.[3] Thus, in a reduced form demand for infant health, LBW would be regressed on income, education, insurance coverage, AFDC benefits as well as the price and availability of medical care, cigarettes, and illicit substances [Rosenzweig and Schultz (1983); Corman and Grossman (1987)].

The annual means across all the health districts for the rate of LBW and each of the five determinants are shown in table 1 by race and ethnicity from 1980 to 1989. Figs. 1 and 2 display the same data for LBW and illicit substance use. What is immediately apparent from fig. 1 is the rapid acceleration in the rate of LBW among Blacks between 1984 and 1988, a less pronounced rise for Hispanics, and a more mild acceleration for Whites. Time-series data from New York City indicate that the rate of LBW among Blacks and Whites has trended slowly downwards since 1968 [Joyce (1990)]. In short, the upturn in the rate of LBW is unprecedented in over a generation and probably longer.[4]

Fig. 2 shows the prevalence of prenatal illicit substance use over the same ten years. The rapid increase among Blacks beginning in 1985 is unmistakable. Hispanics show a much smaller rise and Whites display an actual decline. None of the other regressors evidence as dramatic change, nor a change that coincides so closely with the rise in LBW as does prenatal illicit substance use, especially among Blacks. As compelling as the visual evidence appears, it should be emphasized that misclassification of prenatal illicit substance use is likely to be severe. A recent prevalence study in Florida showed that under a mandatory reporting system Blacks were more likely than Whites to be reported to health authorities after delivery, even though the prevalence of alcohol and illicit substances detected at the first prenatal care visit was slightly higher among Whites [Chasnoff et al. (1990)]. A discussion of measurement error and our remedial efforts are presented below.

The costs of LBW that we consider are limited to the initial hospitaliza-

[3]Mother's education is often included in the birthweight production function as a proxy for parental efficiency in household production [Grossman (1972)]. Others have argued that education has only an indirect effect on birthweight by conditioning the parent's choice of health inputs [Rosenzweig and Schultz (1982, 1983, 1991)]. We experimented with education, but found the results were insignificant and/or perverse depending upon how the measure was specified. Moreover, in the full-fixed effects specification, the impact of education on both LBW and the other coefficients was minor. These results are consistent with other work based on a single cross-section of individual births in New York City in which mother's education had relatively little impact on birthweight, but was a powerful determinant of prenatal care [Grossman and Joyce (1990)].

[4]Computerized birth certificates records are available from 1963. Data on birthweight prior to the 1960s is more questionable, especially among blacks, since home births were much more common [David (1986)].

Table 1

Means and standard deviations by year, race/ethnicity.[a]

	80	81	82	83	84	85	86	87	88	89	80–89
Blacks											
% LBW	11.7	11.6	11.4	11.2	11.0	11.4	12.2	12.8	13.6	13.1	12.1
	(1.8)	(2.5)	(2.1)	(2.0)	(2.1)	(2.2)	(2.7)	(2.9)	(2.9)	(2.6)	(2.5)
% No care	9.1	9.0	10.6	10.8	13.2	17.1	19.9	17.8	15.2	16.3	14.2
	(3.9)	(3.8)	(3.9)	(3.8)	(3.7)	(6.5)	(6.7)	(6.6)	(5.5)	(7.2)	(6.5
% Drugs	2.3	2.0	2.1	2.1	2.4	2.6	4.1	5.3	6.0	6.6	3.7
	(1.2)	(1.3)	(1.4)	(1.1)	(1.6)	(1.8)	(2.5)	(2.8)	(3.2)	(3.5)	(2.8)
% Unmarried	62.1	62.3	63.5	64.1	63.6	64.6	65.5	65.6	65.8	66.9	64.5
	(11.2)	(1.2)	(10.9)	(11.6)	(11.2)	(11.1)	(11.1)	(11.0)	(9.9)	(10.0)	(10.8)
% Smoke	8.5	8.9	8.5	7.9	6.2	6.2	6.7	6.7	10.7	10.3	8.1
	(7.8)	(9.7)	(8.6)	(7.8)	(6.0)	(6.0)	(6.1)	(5.5)	(6.0)	(5.4)	(7.0)
% Parity > 3	14.0	14.2	14.8	15.8	11.0	14.3	14.0	13.6	16.9	16.0	14.5
	(2.5)	(2.5)	(2.5)	(4.6)	(2.0)	(4.4)	(4.3)	(2.5)	(4.3)	(5.9)	(4.1)
Hispanics											
% LBW	7.6	7.5	7.5	7.5	7.5	7.1	7.7	7.8	7.8	7.7	7.6
	(1.6)	(1.6)	(1.7)	(1.4)	(1.7)	(1.5)	(1.8)	(1.7)	(1.8)	(1.8)	(1.7)
% No care	10.1	9.7	9.7	10.4	9.9	11.7	13.8	13.7	13.2	14.8	11.9
	(7.7)	(6.9)	(5.4)	(5.1)	(4.8)	(4.8)	(6.1)	(6.3)	(4.5)	(7.0)	(6.2)
% Drugs	1.4	1.2	1.4	1.7	2.4	2.6	3.1	3.1	3.3	3.3	2.4
	(0.7)	(0.8)	(1.0)	(1.1)	(1.8)	(1.7)	(1.9)	(1.8)	(1.7)	(1.9)	(1.7)
% Unmarried	44.8	46.5	47.3	48.5	49.5	50.4	52.5	52.2	51.9	54.3	50.1
	(13.6)	(12.8)	(12.9)	(12.7)	(12.3)	(12.0)	(12.1)	(11.2)	(10.5)	(10.2)	(12.1)
% Smoke	5.0	6.9	5.4	5.1	4.5	4.5	5.2	4.1	5.7	5.4	5.2
	(3.8)	(7.0)	(4.7)	(4.7)	(2.7)	(3.0)	(3.0)	(2.2)	(2.7)	(2.4)	(3.8)
% Parity > 3	9.9	10.1	10.0	10.3	7.8	8.4	8.7	8.1	13.6	13.1	10.1
	(2.4)	(2.3)	(2.5)	(2.4)	(1.7)	(2.4)	(2.5)	(1.8)	(6.9)	(8.2)	(4.6)
Whites											
% LBW	5.1	4.9	4.7	4.5	4.7	4.5	4.6	4.8	4.9	4.9	4.8
	(1.2)	(1.0)	(1.3)	(0.9)	(0.7)	(0.8)	(1.0)	(1.0)	(1.3)	(1.5)	(1.1)
% No care	2.9	2.7	3.2	3.7	3.5	4.6	5.2	4.7	6.9	7.1	4.5
	(1.8)	(1.2)	(1.7)	(2.3)	(1.6)	(2.6)	(2.8)	(2.4)	(2.4)	(3.5)	(2.7)
% Drugs	2.0	2.0	2.1	2.0	2.7	2.3	2.2	2.1	2.1	2.3	2.2
	(2.3)	(2.3)	(2.4)	(1.9)	(2.2)	(1.6)	(1.5)	(1.3)	(1.2)	(1.4)	(1.8)
% Unmarried	9.1	9.6	10.0	9.7	10.5	11.1	12.1	12.4	13.2	15.4	11.3
	(6.7)	(6.1)	(6.1)	(6.4)	(6.7)	(6.3)	(6.8)	(6.2)	(7.9)	(8.1)	(6.9)
% Smoke	2.2	2.1	2.0	1.9	2.2	2.4	3.0	2.9	6.4	7.0	3.2
	(1.9)	(2.0)	(1.7)	(1.8)	(1.8)	(2.1)	(2.2)	(2.0)	(3.5)	(3.7)	(3.0)
% Parity > 3	10.2	11.0	11.7	12.1	9.7	13.0	12.9	12.7	15.7	14.8	12.4
	(5.4)	(5.4)	(5.5)	(6.4)	(6.7)	(7.4)	(7.2)	(6.8)	(8.2)	(8.3)	(7.0)

[a] Means and standard deviations are weighted by the number of births in the Health District.

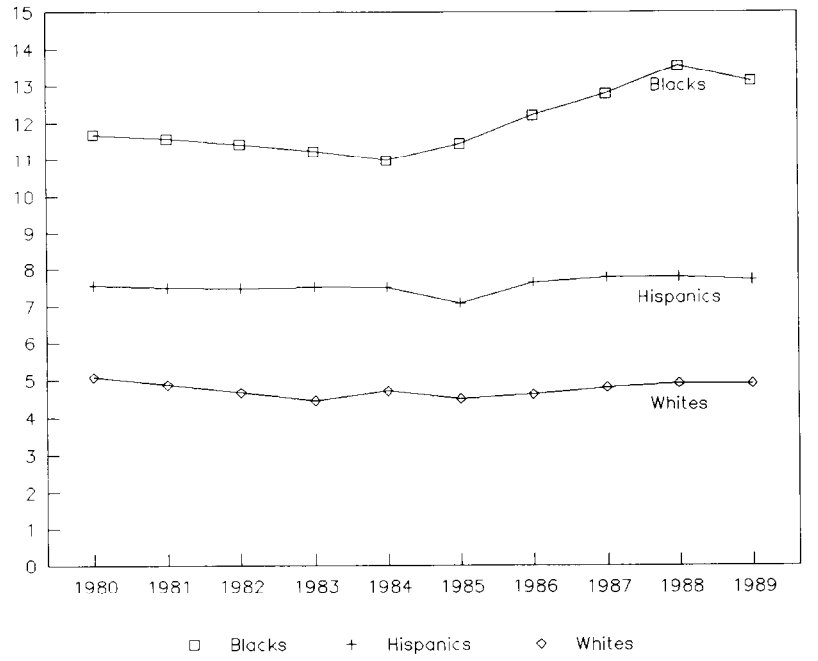

Fig. 1. Annual percentage of low birthweight births.

tion after delivery.[5] They originate from a study of LBW costs (net of capital expenses and medical education) based on a 1985 stratified sample of urban hospitals that included costs of all levels of newborn hospital care from routine to tertiary NICU care [Schwartz (1989)]. The study estimated the average neonatal cost per birth for various birthweight categories including survivors and non-survivors but exclusive of infants born less than 500 grams. To obtain the average cost per LBW birth we averaged the costs per birthweight category weighted by the number of births in each group. The average neonatal cost per LBW was $9,556 in 1985 dollars which is equivalent to $12,096 in 1991 when inflated by the consumer price index.

In using these cost data we are making two implicit assumptions: (1) that the sample of urban hospitals in Schwartz's study do not differ substantially

[5]The total opportunity costs of a low birthweight birth include the sum of: (1) the cost of the initial hospitalization; (2) the costs of any rehospitalization due to the effects of low birthweight; (3) the costs of physical, educational and vocational rehabiliation and/or long term disability; (4) the foregone income of caretakers responsible for securing adequate care for the infant; (5) the foregone future income of the child associated with the potential sequelae of low birthweight; and (6) the psychic or emotional costs to caretakes and children of low birthweight and its consequences. We chose to address only the first of these because we had no direct data on rehospitalization or disability rates among the sample we examined nor with the available data could we have reasonably estimated foregone parental or patient future income.

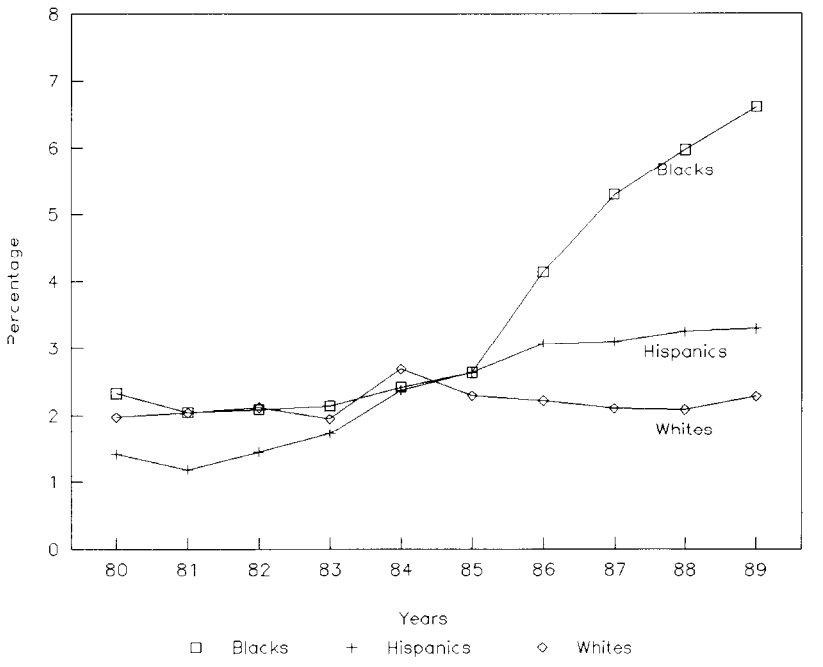

Fig. 2. Annual percentage of reported prenatal illicit substance use.

in their cost schedules from the New York City hospitals caring for the children in our study; and (2) that the clinical course of the LBW births in Schwartz's sample do not differ appreciably from the clinical course of the population we have studied. To the extent that the costs of hospital care in New York City exceed national averages, our figures will underestimate the true costs of excess LBW births. The same will be true if the etiology of LBW in our population results in substantial complications and delayed discharge relative to Schwartz's sample. If, on the other hand New York City hospital costs are systematically less than a national urban average and/or if our population of LBW infants either die prematurely or are discharged early relative to Schwartz's sample then our figures will overestimate the true costs of excess New York City LBW births. We attempt to validate our cost estimates by using data from two other studies as a comparison (see below).

2.2. *Methods*

We use minimum chi-squared methods, since the dependent variable is the direct aggregation of a dichotomous outcome [Maddala (1983)]. In particular, let P_{ijt} equal one, if individual i residing in health district j, has a LBW

birth in year t and zero otherwise; let d_{kijt} be a vector of k dichotomous indicators of whether the woman was married, had inadequate prenatal care, had four or more previous live births, smoked, or was exposed to illicit substances while pregnant; let β_k be the coefficient vector; let μ_{ij} represent individual and district-specific heterogeneity that is time invariant; let γ_t be the time effects that impact city-wide in any particular year and let e_{ijt} be the residuals. The model at the individual level can be specified as follows.

$$P_{ijt}=\gamma_t+\mu_{ij}+\beta_k d_{kijt}+e_{ijt}. \tag{1}$$

The summation of eq. (1) by individuals and division by the total number of births in a district yields the specification that is estimated in the paper. Specifically,

$$\mathrm{LB}_{jt}=\gamma_t+\mu_j+\beta_k X_{kjt}+e_{jt}, \tag{2}$$

where LB_{jt} is the proportion of LBW births and X_{kjt} is a vector of the five health inputs: The proportion of births to women out-of-wedlock, with inadequate care, with four or more previous live births, to women who smoked and who used illicit substances during pregnancy. Fixed or random effects adjust for the time- and district-specific effects (γ_t, μ_j).

Eq. (2) is estimated by weighted least squares.[6] Provided the number of births in each district is sufficiently large to insure that there are no cells with a zero probability of LBW, the linear probability function is less vulnerable to the shortcomings associated with the linear probability model at the individual level. Its primary advantage is the straightforward interpretation of the coefficients. Nevertheless, to insure that the results are not sensitive to the linear specification, we estimate a logit model as well.

The estimation of eq. (2) is unambiguous if the regressors are uncorrelated with the errors. In our analysis the orthogonality assumption is likely to be violated for several reasons. First, measurement error in the substance abuse variable appears likely. Until recently, women who tested positive at delivery for illicit substances in New York City were often separated from their newborns. Even in the absence of such sanctions, the social stigma attached to illicit substances use during pregnancy is probably sufficient incentive to deny its use. Urine-toxicology analysis is an improvement over self reports,

[6]The weights are $n_j/(\mathrm{LB}_j(1-\mathrm{LB}_j))$ where n_j is the number of births in district j and LB_j is the district specific rate of low birthweight. A district had to have at least 100 race- or ethnic-specific births to be included in the regressions.

but is unlikely to overcome the problem of underreporting since urinalyses are applied selectively. A recent study showed that Black women were more likely to be characterized as users relative to White women, even though the prevalence of illicit substance use was similar for both groups [Chasnoff et al. (1990)]. In sum, the percentage of infants exposed prenatally to illicit substances as reported on birth certificates, a mixture of self reports and urinalyses, undoubtedly underreports the true prevalence.

To illustrate the empirical implications let X_{jt}, be the true prevalence rate of prenatal illicit substance use in the jth district and tth year; let X_{jt}^* be the observed prevalence such that

$$X_{jt}^* = X_{jt} - \phi_t - \delta_j + \varepsilon_{jt}, \tag{3}$$

where ϕ_t and δ_j represent fixed levels of underreporting that differ by year and by district respectively and ε_{jt} is a random term with mean zero and variance σ_ε^2 that represents the random misclassification of women as users and non-users of illicit substances. Assume that X_{jt} is the only regressor and insert eq. (3) into (2).

$$\mathrm{LB}_{jt} = (\gamma_t + \beta\phi_t) + (\mu_j + \beta\delta_j) + \beta X_{jt}^* + e_{jt} - \beta\varepsilon_{jt}, \tag{4}$$

which can be rewritten as

$$\mathrm{LB}_{jt} = \gamma_t^* + \mu_j^* + \beta X_{jt}^* + e_{jt}^*. \tag{5}$$

Eq. (5) presents the classical problem of error in variables since $\mathrm{cov}(X_{jt}^*, e_{jt}^*)$ is non-zero whereas the $\mathrm{cov}(e_{jt}^*, \gamma_t^*)$ and $\mathrm{cov}(e_{jt}^*, \mu_j^*)$ are zero by construction. As such, β is biased towards the null; the effects, γ_t^* and μ_j^*, can be estimated consistently, but we cannot distinguish the underreporting ($\beta\phi_t$ and $\beta\delta_j$) from time and district heterogeneity (γ_t and μ_j). The situation becomes much more complicated if the underreporting is not fixed, but is assumed to have some asymmetrical distribution and is added to the error term, e_{jt}^*.[7]

An instrumental variable will allow us to obtain unbiased estimates of β, the marginal effect of observed illicit substance use on low birthweight. The difficulty is finding a suitable instrument. In this study we use the race-specific number of deaths due to drug dependency[8] per 100,000 residents by

[7]In this case the underreporting would be analogous to the problems associated with inefficiency in the frontier production function literature. Since the degree of underreporting is an interesting, but not central issue of the study, we do not try to disentangle the effects from the underreporting with arguably heroic assumptions about the distribution of the time- and district-specific underreporting [Schmidt and Sickles (1984)].

[8]The determination of death due to drug dependency is based on the International Classification of Disease, Ninth Edition, number 304.

health district and by year as an instrument for prenatal illicit substance use.[9]

The instrument has a number of strengths. First, the rate of drug deaths as a measure of the use and availability of illicit substances should be highly correlated with maternal illicit substance use. Second, the rate of drug deaths has no direct effect on birth outcomes and thus, is exogenous to the model. Third, drug deaths should be unrelated to the error in reported material illicit substance use since the former are not based on self-reports, but are determined by a medical examiner with no obvious incentive to under or overreport. Thus, we will regress prenatal illicit substance use on the rate of drug deaths and include the predicted value from this first-stage in the LBW regression. Controls for time and district effects in the first-stage will be included when they are present in the second stage as well.[10]

Unmeasured differences across health districts represent a second reason why the orthogonality assumption is unlikely to hold even if underreporting were not an issue. We have no data, for example, on nutrition, stress, sexually transmitted diseases or the quantity and quality of obstetrical care, all potential determinants of LBW and potentially correlated with the included regressors.

Finally, orthogonality may be compromised because life-cycle models of fertility treat the number of births, the care received, and the health of each child at birth as endogenous. The simultaneity of childbearing decisions are most relevant with longitudinal data on individual women or families. Since our data is more accurately characterized as an aggregation of repeated cross-sections the simultaneous equations bias should be muted. More practically, we lack the data to effectively instrument all the inputs or to distinguish true simultaneity from omitted variables bias.

A fixed effects estimator is preferred in the presence of measurement error, underreporting, and unobserved heterogeneity. The estimator makes no assumptions about the distribution of the effects, and the βs in a multivariate specification are consistent even if the effects and/or the underreporting are correlated with the regressors [Mundlak (1978)]. We lose, however, cross-district variation with the fixed effects (or within group) estimator. We suspect this may be an important source of variation given the disparity in prenatal illicit substance use across neighborhoods. Thus, we will use the Hausman test to ascertain whether a random effects estimator is feasible [Hausman (1978); Hausman and Taylor (1981)].

[9]Drug deaths are available for White and Non-Whites only aggregated for both genders. For Hispanics, we took a weighted average of the two rates assuming that 85% of all Hispanics are White, an approximation based on the 1980 census for New York City.

[10]We will not include the other inputs in the first stage because as discussed below, their exogeneity is questionable.

Table 2

Low birthweight rate regressions corrected and uncorrected for measurement error – Blacks, Hispanics, and Whites.[a]

	Blacks		Hispanics		Whites	
Variables	A	B	A	B	A	B
% Drugs	0.432[b]	0.144	0.308[b]	0.019	0.054	0.016
	(5.75)	(2.64)	(3.01)	(0.29)	(2.25)	(0.43)
% Smoke	0.064	0.014	0.014	0.027	0.037	0.047
	(3.37)	(1.59)	(0.71)	(1.20)	(1.99)	(1.58)
% No care	0.040	0.046	0.011	0.034	0.064	0.073
	(1.86)	(2.40)	(0.75)	(1.65)	(2.39)	(1.92)
% Unmarried	0.087	0.099	0.089	0.008	0.066	0.085
	(6.56)	(3.01)	(11.66)	(0.39)	(6.15)	(2.96)
% Parity > 3	0.003	0.020	0.050	0.033	−0.025	0.001
	(0.13)	(1.01)	(3.15)	(2.12)	(3.70)	(0.01)
Constant	4.260	4.88	2.390	5.236	4.217	3.88
	(5.77)	(2.46)	(7.98)	(7.39)	(25.63)	(15.90)
Time effects	yes	yes	yes	yes	yes	yes
Health district effects	no	yes	no	yes	no	yes
F	42.52	31.43	46.27	23.82	10.88	5.26
χ^{2c}	14.34	0.62	0.09	0.11	0.31	0.11
χ^{2d}	–	3.91	–	22.00	–	5.15
Adj R^2	–	0.82	–	0.77	0.35	0.39
Rho[e]	0.20	−0.15	0.19	−0.11	0.03	0.06
N	280	280	290	290	260	260

[a] The absolute value of t-statistics are in parentheses.
[b] Corrected for measurement error by instrumental variables.
[c] Hausman test of errors in variables. $\chi^2(1)$ of 0.05 level = 7.89.
[d] Hausman (1978) test of random vs. fixed effects. Critical $\chi^2(5)$ at 0.05 level = 16.75.
[e] First-order autocorrelation coefficient [see Greene (1990)].

3. Results

3.1. Regression estimates

The main regressions are presented in table 2.[11] Columns (1), (3) and (5) contain the estimates for Blacks, Hispanics and Whites, respectively, controlling for time, but not district effects. These specifications probably overstate the impact of illicit substance use on LBW by attributing to it district-specific heterogeneity. Columns (2), (4) and (6) control for both time and district effects. By contrast, these specifications yield lower bound estimates of illicit substance use since all between-district variation has been removed.

Overall, the regressions for Blacks and Hispanics work well. The adjusted R-squared range from 0.77–0.82, and the coefficients have the expected signs and are frequently significant at conventional levels. The regressions for Whites are less satisfactory for the goodness of fit is substantially smaller. We

[11] A more detailed discussion of results with additional specifications is presented in a longer version of this paper [Joyce et al. (1991)].

did not correct for first-order autocorrelation in any of the specifications since the rho's were relatively small, especially in the models which control for unobserved heterogeneity.

Illicit substance use is an important predictor of LBW among Blacks [columns (1) and (2)]. The coefficient ranges from 0.14 to 0.43 and is statistically significant in both specifications ($p<0.01$). Thus, a one percentage point increase in the percentage of births exposed prenatally to illicit substances raises the rate of LBW from between 0.14 to 0.43 percentage points.

For Blacks, instrumenting the illicit substance use measure in the OLS specification without district effects appears important for two reasons. First, the Hausman test applied to the non-instrumented specification rejects the null hypothesis of no measurement error ($\chi^2(1)=14.34$) in the variables; second, the IV coefficient is almost twice as large as the one obtained by OLS (not shown). Once we control for district-specific effects, however, we cannot reject the null hypothesis of no measurement error, and the coefficient on illicit substance use is only one-third as large as in the specification without district effects [column (2)]. Moreover, we cannot reject random effects ($\chi^2(5)=3.91$), a surprising result given the potential threats to orthogonality. The coefficient on illicit substance use obtained by random effects differed inconsequentially from the fixed effects estimate in column (2). This suggests that the effects are linearly related to the right-hand-side regressors [Mundlak (1978)].

The results for Hispanics are similar to Blacks but only in the specification without district-specific controls [column (3)]. The coefficient on illicit substance use is not quite as large, but remains statistically significant ($p<0.01$). As with Blacks, marital status is an important risk factor, but smoking is not. Contrary to the Black specifications, there is no evidence of measurement error in the illicit substance use variable, and although we present the IV estimate, it differed trivially from the OLS estimate (not shown).

Controls for district-specific effects substantially alter the results for Hispanics (column (4)]. The coefficient for illicit substance use is small and statistically insignificant. Similarly, marital status loses its explanatory power whereas prenatal care and parity are marginally significant. Moreover, we reject random in favor of fixed effects ($\chi^2(5)=22.00$). The results indicate that the variation in LBW that can be explained by illicit substance use and out-of-wedlock childbearing occurs between districts. Yet, without a set of instruments for all the regressors, there is no way to distinguish unobserved heterogeneity from between-district variation.[12]

[12]In actuality, the problem would be more difficult since district and time specific underreporting may be related differently to the included regressors, especially reported drug use, than the more general unobserved heterogeneity.

The regression estimates for Whites are unsatisfactory [columns (5) and (6)]. The adjusted R-squared is relatively low for aggregate data even though the coefficients on out-of-wedlock childbearing, illicit substance use, smoking and prenatal care are statistically significant in the OLS specification that excludes district effects. As with Hispanics, the inclusion of district-specific controls eliminates the explanatory power of illicit substances and out-of-wedlock childbearing. The IV estimates evidence little reliability (not shown).

To determine whether the results are sensitive to the use of a linear probability model, we re-estimated the regressions in table 2 as logits (results not shown). The transformation produced no qualitative changes. For both Blacks and Hispanics prenatal illicit substance use is a statistically significant risk factor for LBW when controls for unobserved heterogeneity are excluded and the elasticities computed at the mean of the right-hand-side variables are very similar. As with the linear specification, controlling for district specific effects eliminates the impact of illicit substance use among Hispanics, but not for Blacks. The regressions for Whites were not altered appreciably by the change in functional form.

3.2. Actual versus predicted rates of LBW

To illustrate how much of the increase in the rate of LBW in the late 1980s among Blacks, and to a lesser extent Hispanics and Whites, can be explained by illicit substance use, we used the regression estimates in table 2 to predict the annual rate of LBW from 1985 to 1989. We set the rate of prenatal illicit substance use at its 1984 level, while substituting annual means for each of the other regressors (see table 1). Thus, the projections reflect the rate of LBW that would have been expected, had the prevalence of prenatal illicit substance use remained at its 1984 level.

We present two projections based on the two specifications in table 2 which represent upper- and lower-bound estimates for each race and ethnicity (projection A and B respectively). Projection A indicates that, within the Black population, a large proportion of the rise in LBW between 1985 and 1989 can be attributed to prenatal illicit substance use, while Projection B suggests a more modest impact for this factor. Among Hispanics, only projection A demonstrates any consistent impact of maternal illicit substance use.

We used the projections in fig. 3 to estimate the neonatal costs of the excess morbidity that are directly attributable to prenatal illicit substance use. For each year beginning in 1985 we multiplied the actual rate of LBW and the two projected rates by the total number of race- and ethnic-specific births to obtain the actual and projected number of LBW births annually. The difference between the actual and projected number of LBW births summed over the five years represents the excess morbidity attributable to

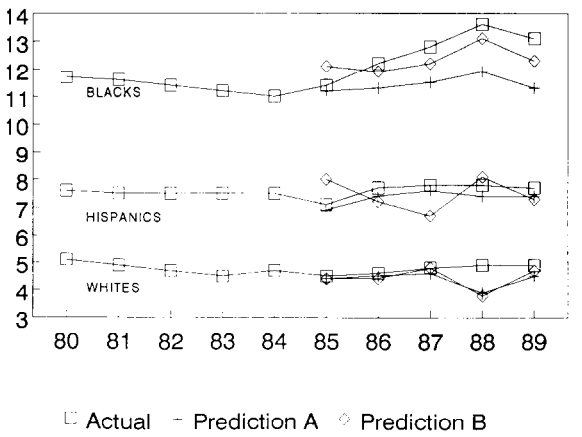

Fig. 3. Actual and predicted rates of LBW.

Table 3

High and low estimates of excess LBW births and excess costs associated with illicit drug use among pregnant women in New York City: 1985–1989.[a]

	Excess LBW births[b]		Excess costs (000's)	
	High	Low	High	Low
Blacks	2,276 (9.4%)	577 (2.4%)	$27,530	$6,979
Hispanics	477 (3.5%)	365 (2.7%)	$5,770	$4,415
White	606 (7.7%)	540 (6.9%)	$7,330	$6,532
Total	3,359 (7.3%)	1,482 (3.2%)	$40,630	$17,926

[a] The average cost of a LBW birth is $12,096 in 1991 dollars [Schwartz (1989)].
[b] The figures in parentheses represent the excess LBW births as a percentage of total LBW births 1985–1989.

prenatal illicit substance use. We also express these raw numbers as percentages of total LBW births over the same period. Multiplying the excess number of LBW births by the average costs per LBW birth provides the costs, *for the initial hospitalization only*, that can be ascribed to prenatal illicit substance use in New York City in the period 1985–1989.

The estimates are shown in table 3. The five-year costs range from 18 to 41 million dollars based on the 1,500–3,400 excess LBW births. As we have stated throughout, the most robust results are for Blacks. Although the high estimates for Hispanics and Whites reflect statistically significant effects, the low estimates do not. Thus, a more conservative lower bound estimate would count only the costs pertaining to Blacks.

To check the reasonableness of these estimates, we applied the neonatal

cost of maternal cocaine use from two recent studies to the New York City birth certificate data (Phibbs et al. (1991); Calhoun and Watson (1991)].[13] Specifically, we computed the incremental increase in the number of births exposed prenatally to illicit substances by race and ethnicity from 1985 to 1989.[14] The product of these births and the estimated cost of exposure as reported in the above studies represents an alternative estimate of the neonatal costs of prenatal illicit substance use. The computations generated costs between $53 and $76 million in 1991 dollars, close to our upper bound estimates.[15] If one considers that both these studies include capital and education expenses in their estimates and that the latter study is based on charges as opposed to costs, one would conclude that, with adjustments, these estimates would more closely approximate our own findings.

4. Conclusion

We have investigated the dramatic increase in the rate of LBW that occurred in New York in the latter half of the 1980s with separate panels of live births among Blacks, Whites and Hispanics across health districts from 1980 to 1989. We found that the independent effect of illicit substance use varied substantially by race with little effect demonstrable among Whites, a potentially important but not robust effect discernible among Hispanics, and a statistically significant effect detectable in the Black population. Depending on whether the model controls for unobserved heterogeneity or not, we calculate that the number of excess LBW births attributable to illicit substance use over this period range from approximately 1,500–3,400 resulting in excess neonatal treatment costs of between $18 and $41 million. We conclude that illicit substance used was a major contributory factor in generating the unprecedented rise in LBW among Blacks in New York City in the latter part of the 1980s.

We were unable to accurately document the excess LBW births or costs attributable to illicit substance use in the White and Hispanic populations. This is not to say that no relationship exists in these populations. Illicit substance use among Hispanics was clearly on the rise over this period (see table 1 and fig. 2) and the coefficient of substance abuse for the White population was marginally significant in some specifications. Thus for both

[13]To the best of our knowledge these are the only two studies to have addressed the cost of maternal substance abuse since the introduction of crack cocaine in the mid 1980s.

[14]In particular, we subtracted the proportion of births exposed prenatally to drugs in 1984 from the proportion exposed in each of the subsequent years (see table 1). We then multiplied the excess proportion exposed each year by the actual number of births that year and summed over the entire period to derive the total excess number of births exposed prenatally.

[15]From Phibbs et al. (1991) we used the cost to medical discharge for the cocaine and polydrug users ($8,450). Calhoun and Watson (1991) reported the incremental charge per discharge ($12,000).

these groups there is reason to believe that illicit substance use may constitute an important risk factor explaining some of the observed variation in LBW, but our results do not permit any strong conclusions.

The lack of robust results for Hispanics and Whites may be due partly to differential reporting of illicit substance use in these groups relative to Blacks. One suggestive piece of evidence in this regard relates to the instrumental variable we used for illicit substance use. The fact that the instrument was less well correlated with illicit substance use in White and Hispanics relative to Blacks could conceivably arise from three possibilities: (1) drug related deaths were reported differently for Blacks relative to the other two groups; (2) the link between drug related deaths and prenatal illicit substance use was weaker in the latter groups; or (3) prenatal illicit substance use was reported differently in Blacks relative to Whites and Hispanics. Considering the evidence presented by Chasnoff et al. (1990) in describing prenatal illicit substance use reports in another venue, we believe the third of these possibilities to be the most likely.

Despite these limitations, this study remains the first population-based, multivariate longitudinal investigation which has attempted to quantify the independent contribution of prenatal illicit substance use to the epidemic rise in LBW in an entire urban population. The findings, while more robust for the Black than for the Hispanic or White populations, demonstrate how important a risk factor illicit substance use has been in recent years. We believe that future studies using more controlled data collection methods will be able to extend the quantitative findings to non-Black groups. Though the implications of these observations for New York City and similar urban settings are disturbing, we believe that the identification of a risk factor in the face of an ongoing epidemic is a critical first step in devising primary prevention strategies to ameliorate the condition.

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