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# THE CONSEQUENCES AND COSTS OF MATERNAL SUBSTANCE ABUSE IN NEW YORK CITY: A POOLED TIME-ANALYSIS, CROSS-SECTION ANALYSIS

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# THE CONSEQUENCES AND COSTS OF MATERNAL SUBSTANCE ABUSE IN NEW YORK CITY: A POOLED TIME-SERIES, CROSS-SECTION ANALYSIS

## ABSTRACT

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 1,900 to 3,800 resulting in excess neonatal admission costs of between $22 and $53 million. We conclude that illicit substance use was a major contributory factor in rapid rise of low birthweight among Blacks in New York City in the latter part of the 1980s. The impact of prenatal illicit substance use on Whites and Hispanics is less conclusive.

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## 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.

In particular low birth weight (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 5 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 percent of all singleton live births, by 1987 that figure had risen to more than 13.5 percent, a 25 percent increase in little more than 2 years. During the same decade, LBW rates among Whites also began to increase though less dramatically than among Blacks. Because LBW rates for all ethnic groups had been decreasing for 20 years up to the mid 1980's, 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, age, education level, socio-economic status, and access to prenatal care; 2) obstetrical characteristics of a particular pregnancy such as

gestational diabetes, low weight gain, infection and hypertension; and 3) 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 1980's, 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 1980's. From 1981 to 1987 the rate per thousand live births of reported maternal cocaine use during pregnancy in New York City approximately tripled from 6.7 to 20.3 coinciding with the observed upsurge in LBW rates (Habel, Lee, and Kaye 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 3 (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 that can be attributed to the rise of prenatal use of illicit drugs between 1985 and 1989 and the newborn costs of this excess morbidity. Toward this aim we use 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. Panels for Black non-Hispanics, White non-Hispanics and Hispanics are analyzed separately.

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 an indicator of prenatal illicit substance abuse for over a decade. 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 maternal substance abuse. A universal toxicology analysis of all births delivered at Harlem Hospital between 1985 and 1986 revealed that 11 percent of the newborns were exposed prenatally to cocaine or its derivative crack (Phibbs, Bateman, Schwartz 1991). Third, because of the size of New York City's population, we can analyze the experience of over 1 million births separately by race and ethnicity and across neighborhoods that vary substantially by family income, education levels, and health status.

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 and underreporting are likely to be substantial (Zuckerman et al. 1989; Chasnoff, Landress, and Barrett 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 drug use. 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 use of illicit substances, yet uncorrelated with its error.

## Analytical Framework

Economic models of infant health emphasize the distinction between the health production function and the input demand function (Rosenzweig and Schultz 1983, 1988; Corman, Joyce, and Grossman, 1987; Grossman and Joyce 1990). The production function represents the technical relationship between the birth outcome and the health inputs where the input demand function analyzes the factors which condition the use of the health inputs. To illustrate let B represent infant health, let M be medical care, and let S capture

the prenatal consumption of tobacco and cocaine.

$$B = b(M,S,\mu) \quad (1)$$

$$M = m(Y,P,\mu) \quad (2)$$

$$S = s(Y,P,\mu) \quad (3)$$

To complete the model, let Y represent the command over resources such as income, health insurance, or transfer payments and let P embody the price and availability of the health inputs. Finally, let $\mu$ represent factors associated with individual heterogeneity such as a woman's health endowment.

The empirical aim of the paper is the estimation of equation (1), the infant health production function. In particular, we seek to determine the relative contribution of illicit drugs to the rise in LBW across neighborhood and over time in New York City. Note the model also yields a reduced form production function. The substitution of equations (2) and (3) into equation (1) would show how changes in the price and availability of illicit drugs, for example, impact on infant health. Although clearly of interest, the estimation of the reduced form is limited by a lack of data on the price of illicit drugs across neighborhoods. New York City birth certificates, however, record information on the use of prenatal care, tobacco, and illicit drugs among pregnant women, and thus, make possible the estimation of a structural production function.

## Empirical Implementation

### 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.

Our measure of infant health is the race- and ethnic-specific rate of LBW births. One of the advantages of LBW in a time-series context is that infant mortality rates, unlike rates of LBW, have been greatly improved by the steady advances in neonatology (McCormick 1985). Yet, there are few empirical controls for technological change and most applications resort to trend terms.

Except for tocolytic agents however, no medical 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 1960's until the mid 1980's 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 drugs of which cocaine, and its derivative crack, appear to be the most damaging. Thus, to estimate the relative contribution of prenatal substance abuse to LBW in New York City, 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 who smoked during pregnancy, and the percentage of births to women who used illicit drugs prenatally. Illicit drugs include cocaine, heroin, methadone, and barbiturates.[1] 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. Figures 1 and 2 display the same data for LBW and illicit substance use. What is immediately apparent from Figure 1 is the

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

rapid acceleration in the rate of LBW among Blacks between 1984 and 1985, a less pronounced rise for Hispanics, and a more mild acceleration for Whites. Time-series data from New York City indicates 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.[2]

Figure 2 shows the prevalence of prenatal illicit substance use over the same 10 years. Again 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 a change, nor a change that coincides so closely with the rise in LBW as does prenatal drug use, especially among Blacks. As compelling as the visual evidence is, it should be emphasized that misclassification of prenatal drug use is likely to be severe. A widely cited 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 drugs at the first prenatal care was slightly higher among Whites (Chasnoff, Landress, and Barrett 1990). A discussion of measurement error and our remedial efforts are presented below.

The costs of LBW are limited to the initial hospitalization after delivery. The costs are from a study of neonatal intensive care units based on a 1985 stratified sample of urban hospitals (Schwartz 1989). The study estimated average neonatal cost per birth for various birthweight categories. The estimates exclude capital expenses and costs associated with medical education. There are no estimates for infants weighing less than 500 grams.

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

To obtain the average cost per LBW birth, we averaged the various birthweight categories weighted by the number of births within each group. The average neonatal cost per LBW was $9556 in 1985 dollars which is equivalent to $13,719 in 1990 based on the medical care component of the Consumer Price Index.

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 $\mathbf{d}_{kijt}$ be a vector of k dichotomous indicators of whether the woman was married, had inadequate prenatal care, had 4 or more previous live births, smoked, or used illicit drugs while pregnant; let $\beta_k$ be the coefficient vector; let $\mu_{ij}$ represent individual and district-specific heterogeneity that is time invariant; let $\gamma_t$ 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:

$$P_{ijt} = \gamma_t + \mu_{ij} + \beta_k \mathbf{d}_{kijt} + e_{ijt} \qquad (4)$$

The summation of equation (4) by individuals and division by the total number of births in a district yields the specification that is estimated in the paper. Specifically,

$$LB_{jt} = \gamma_t + \mu_j + \beta_k X_{kjt} + e_{jt} \qquad (5)$$

where $LB_{ij}$ 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 consumed drugs during pregnancy. Fixed or random effects adjust for the time- and district-specific effects ($\gamma_t$, $\mu_j$).

Equation (5) is estimated by weighted least squares.[3] 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 equation (5) is straightforward 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 drug use during pregnancy is probably sufficient incentive to deny its use. Urine toxicology analysis is an improvement over self reports, 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 drug use was similar for both groups (Chasnoff, Landress, and Barrett 1990). In sum, the percentage of infants exposed prenatally to illicit drugs 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 drug use in the $j_{th}$ district and $t_{th}$ year; let $X^*_{jt}$ be the

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[3] The weights are $n_j/(LB_j(1-LB_j))$ 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.

observed prevalence such that

$$X^*_{jt} = X_{jt} - \phi_t - \delta_j + \epsilon_{jt} \qquad (6)$$

where $\phi_t$ and $\delta_j$ represent fixed levels of underreporting that differ by year and by district respectively and $\epsilon_{jt}$ is a random term with mean zero and variance $\sigma^2_\epsilon$ that represents the random misclassification of women as users and nonusers of illicit drugs. Assume that $X_{jt}$ is the only regressor and insert equation (6) into (5).

$$LB_{jt} = (\gamma_t + \beta\phi_t) + (\mu_j + \beta\delta_j) + \beta X^*_{jt} + e_{jt} - \beta\epsilon_{jt} \qquad (7)$$

which can be rewritten as:

$$LB_{jt} = \gamma^*_t + \mu^*_j + \beta X^*_{jt} + e^*_{jt} \qquad (8)$$

Equation (8) presents the classical problem of error in variables since $cov(X^*_{jt}, e^*_{jt})$ is nonzero whereas the $cov(e^*_{jt}, \gamma^*_t)$ and $cov(e^*_{jt}, \mu^*_j)$ are zero by construction. As such, $\beta$ is biased towards the null; the effects, $\gamma^*_t$ and $\mu^*_j$, can be estimated consistently, but we cannot distinguish the underreporting ($\beta\gamma_j$ and $\beta\phi_t$) from time and district heterogeneity ($\gamma_t$, and $\mu_j$). 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, $e^*_{jt}$.[4]

An instrumental variable will allow us to obtain unbiased estimates of $\beta$, the marginal effect of observed drug use on low birthweight. The difficulty is finding a suitable instrument. In this study we use the race-specific number

[4] 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).

of deaths due to drug dependency[5] per 100,000 residents by health district and by year as an instrument for prenatal drug use.[6]

The instrument has a number of strengths. First, the rate of drug deaths as a measure of the use and availability of illicit drugs should be highly correlated with maternal substance abuse. 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 maternal drug use since they 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 drug 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.[7]

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 and the quality of obstetrical care, all potential determinants of LBW and potentially corrected 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

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

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

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

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 $\beta$'s 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 drug 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). Where appropriate, random effects estimates should provide insight as to the sensitivity of the estimates.

## Results

### Regression estimates

The regression for Blacks, Hispanics and Whites are presented in Tables 2 through 4. The tables are organized as follows: columns (1) and (2) contain the ordinary least squares (OLS) and the instrumental variable (IV) estimates respectively with no controls for time or health district heterogeneity; columns (3) and (4) display the OLS and IV estimates with a set of dummies for each year; columns (5) and (6) show OLS and IV estimates with controls for health district heterogeneity; and columns (7) and (8) control for both time and health district effects. In addition, Tables 2 through 4 use the Hausman specification test to check for error-in-variables [columns (1) and (3)] and then

to test for fixed versus random effects [columns (5) and (7)].[8] Finally, we include an estimate of rho ($\rho$), the first-order autocorrelation coefficient.

Overall, the regressions for Blacks and Hispanics work well (Tables 2 and 3). The adjusted R-squared range from .64 to .82 percent, and with two exceptions the coefficients have the expected signs and are frequently significant at conventional levels. The regressions for Whites are less satisfactory (Table 4). The goodness of fit is substantially smaller and the IV estimates lack any precision. We did not correct for first-order autocorrelation in any of the specifications since the rho's were relatively small, especially in the models with controls for unobserved heterogeneity. Prenatal drug use is an important predictor of LBW among Blacks. The coefficient on the drug measure is statistically significant in 7 of the eight specifications ($p < .01$). With respect to the magnitude of the coefficient, a one percentage point increase in the percentage of births exposed prenatally to illicit drugs, raises the rate of LBW from between .14 to .43 percentage points among Black women depending on the specification. In particular, with no controls for health district effects we reject the null hypothesis of no measurement error in the drug use variable [columns (1) and (3)]. In these instances, the IV estimator yields coefficients between 50 and almost 100 percent larger than the uncorrected estimates. When we control for health district heterogeneity, however, we no longer reject the null that residuals are correlated with reported drug use. The similarity between the OLS and the IV estimates is consistent with such a finding [columns (5) - (8)]. Nor can we reject random effects [columns (5) and (7)] a rather surprising result given the potential

[8] We use the Chi-square test for both the error in variables test and the test of fixed versus random effects. If measurement error in the substance abuse variable represented the only potential violation of orthogonality, then a t-test of the extended regression would be appropriate. The t-test is inappropriate when other regressors may be correlated with the residuals (Maddala 1988).

threats to orthogonality. Nevertheless, we present the fixed effects estimates, but we note that the random effects estimates are almost identical (results not shown). The one exception is the IV estimates with time and district controls. The IV random effects estimate of drug use differs inconsequentially from the uninstrumented version, whereas the coefficient on drug use obtained from the two-stage fixed effects estimator appears unstable [column (8)]. This latter result suggests that the two-stage, within group estimator lacks sufficient variation to be useful.

In relative terms, out-of-wedlock childbearing has a much greater impact on the rate of LBW than does maternal drug use. Controlling for time and district effects [column (7)], the elasticity of unmarried births is .6 as compared to an elasticity of .05 for drug use evaluated at their respective grand means (see Table 1). Elasticities for prenatal care and smoking are .06 and .023 respectively. As we will demonstrate below, however, the relative change in illicit drug use has been so dramatic that despite its smaller elasticity, drug use is a compelling explanation for the rise in rate of LBW among Blacks in the mid 1980's.

Prenatal drug use is also important among Hispanics, but only when district effects are not controlled [columns (1) - (4), Table 3]. Nor can we reject the null of no measurement error among Hispanics. The OLS and IV estimates differ inconsequentially. As with Blacks, out-of-wedlock childbearing is the dominant risk factor in terms of elasticity. Using the coefficients in column (3) evaluated at the grand mean, a one percent increase in unmarried births raises the rate of LBW by .5 percent whereas the same increase in maternal drug use raises LBW by .09 percent. As with Blacks, however, the relative rise in prenatal drug use among Hispanics exceeds the rise in out-of-wedlock childbearing.

Important differences between the results for Blacks and Hispanics come into focus when we control for health district heterogeneity. First, we

reject random effects which is only surprising in that we could not reject the null of no measurement error in the drug use variable [columns (1) and (3)]. The fixed effects estimates for Hispanics, unlike for Blacks, indicate that prenatal drug use explains little of the within-district variation in LBW [Table 3, columns (5) - (8)]. Births to unmarried women also loses its explanatory power while prenatal care and parity are marginally significant. The results suggest that the variation in LBW that can be explained by out-of-wedlock childbearing and prenatal drug use occurs between districts. Yet, without a set of instruments for all the regressors, there is no way to distinguish unobserved heterogeneity from between-group variation.[9]

The regression estimates for Whites are unsatisfactory (Table 4). The adjusted R-squared is relatively low for aggregate data. We do show stable effects of unmarried births and maternal drug in the OLS specifications that exclude district effects, but the coefficient on drug use is zero in the other specifications [columns (5) - (8)]. The IV estimates evidence little reliability.

The results in Tables 2 through 4 underscore the basic econometric "rule of thumb" regarding instrumental variables: the estimates are as reliable as the instruments are effective. In the Appendix we present the results from the first-stage regressions. The explanatory power and the precision of the coefficient on drug deaths is apparent among Blacks except when the first-stage includes controls for time and district effects [Appendix, column (8)]. In that case the partial R-squared is greatly diminished which is reflected in the instability of second-stage results [Table 2, column (8)]. Put differently, the lack of explanatory power in the instrument jeopardizes identification (see Maddala 1988, Chapter 9). The same appears true for Whites, and to a lesser

[9] 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.

degree Hispanics.

The finding that we cannot reject the null hypothesis of no measurement error in the prenatal drug use variable when we control for district effects suggests that the within-district variation in the measurement error may be trivial.[10] We also cannot reject any difference in the random and fixed effects estimates in the Black and White specifications which implies no general correlation between the composite effects and the regressors [see equation (8)]. Among Blacks, for example, the random effects estimator yielded coefficient on maternal drug use that range from .16 to .20, which are quite close to their fixed effects counterparts (results not shown). In short, the results for Blacks appear quite robust.

To determine whether the results are sensitive to the use of a linear probability model, we re-estimated the regressions in Tables 2-4 as logits (results not shown). There were no qualitative changes of any substance. For both Blacks and Hispanics prenatal drug 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 drug use among Hispanics, but not for Blacks. The regressions for Whites were not altered appreciably by the change in functional form.

Decomposition of change in low birthweight

To demonstrate the relative contributions of the different independent variables to the actual changes in low birthweight over the period 1984-1989,

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[10] Since the inclusion of health district dummies is equivalent to a regression of deviations around the district means, the measurement error term in equation (7) can be written as $-\beta(\epsilon_{jt} - \epsilon_{j.})$ where $\epsilon_{j.}$ is the mean district-specific error. A lack of variation over time would cause this term to fall out when estimated by fixed effects.

we multiplied the coefficient for each regressor by its percentage point change over the 5 years.[11] These decompositions carry two caveats: first, the time effects are likely to be biased due to underreporting [see equation (7)]; second, such calculations should be derived from reduced form, as opposed to structural estimates. An increase in prenatal drug use, for instance, may lead to greater out-of-wedlock childbearing and less use of appropriate prenatal care. Nevertheless, the decompositions do provide insight as to the relative impact of an input holding all others constant.

Since our principal interest is to characterize the range of possible effects of substance abuse on low birthweight, we constructed decompositions for two specifications: one excluding district-specific effects which probably overestimates the independent effect of substance abuse by attributing to it some district-specific heterogeneity; and another specification that includes district-specific effects which probably underestimates the full impact of substance abuse by removing between-district variation. Because of the ineffectiveness of the substance abuse instrument in the White sample, we use non-instrumented versions of these specifications for Whites.

Table 5 demonstrates several points. To begin with, the figures establish the importance of the substance abuse variable in accounting for a large degree of the change in LBW among the Black population during this period. The high estimate attributes almost 80% of the calculated change in LBW to the effect of substance abuse while the low estimate assigns substance abuse 28% of the total change.

The contribution of out-of-wedlock childbearing to changes in LBW for all three groups is also highlighted in Table 5. In the Black sample, births

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[11] From the regression results in Tables 2-4, and the means in Table 1 the estimates in Table 5 were calculated as follows:

$$(LB_{89} - LB_{84}) = \beta(X_{89} - X_{84}) + (\gamma^*_{89} - \gamma^*_{84}).$$

to unmarried women account for between 13% and 15% of the total change in LBW. These results emphasize the importance of understanding what this variable actually measures and the mechanisms by which out-of-wedlock childbearing affects the probability of delivering a LBW infant.

Finally, the time trend contributions reflected in the table deserve comment. If there were no underreporting of drug use, or if underreporting were time invariant, then one would expect that the contribution of time, all else constant, would be positive in the Black sample for whom LBW rates increased appreciably over the period in question and small in magnitude in the other two samples. If, however, the underreporting of substance abuse varies over time, then the composite time effects could be either positive or negative depending upon whether the degree of underreporting had increased or decreased over time. The negative signs on the time trend elements in five of the six specifications listed in the table suggest that underreporting may have decreased from 1984 to 1989 as the course of the substance abuse epidemic became more widely appreciated and greater emphasis was placed on identifying these exposures in pregnant women.

Actual versus predicted rates of LBW

To further illustrate how much of the increase in the rate of LBW in the late 1980's among Blacks, and to a lesser extent Hispanics and Whites, can be explained by illicit substance use, we used the regression estimates in Tables 2-4 to predict the annual rate of LBW from 1985 to 1989. We set the rate of prenatal drug 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 the expected, had prevalence of prenatal drug use remained at its 1984 level.

As with the decompositions in Table 5, we present two projections representing upper and lower bound estimates for each race and ethnicity (projection A and B respectively). Figure 3 makes visual the essential result

from Table 5. Projection A indicates that almost the entire rise in LBW between 1985 and 1989 can be attributed to prenatal drug use. Projection B suggests that a smaller portion of the rise can be attributed to drug use. Among Hispanics, only the projection A demonstrates any consistent impact of maternal drug use.

We used the projections in Figure 3 to estimate the cost of the excess morbidity that is directly attributable to prenatal drug 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 prenatal drug use. Multiplying the excess number of LBW births by the average cost per LBW birth reflects the costs, for the initial hospitalization only, that can be ascribed to prenatal drug use in New York City from 1985 through 1989.

The estimates are shown in Table 6. The five-year costs range from 22 to 53 million dollars based on the 1900 to 3800 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, Bateman, and Schwartz 1991; Calhoun and Watson 1991).[12] Specifically, we computed the incremental increase in the

[12] 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 1980's.

number of births exposed prenatally to illicit drugs by race and ethnicity from 1985 to 1989.[13] 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 drug use. The computations generated costs between $53 and $75 million in 1990 dollars, clearly close to our upper bound estimates.[14]

## Conclusion

We have investigated the dramatic increase in the rate of LBW which occurred in New York in the latter half of the 1980's 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 discernable 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 1900 to 3,800 resulting in excess neonatal treatment costs of between $22 and $53 million. We conclude that illicit substance use was a major contributory factor in generating the unprecedented rise in LBW among Blacks in New York City in the latter part of the 1980's.

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[13] 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). Multiplying the difference by actual number of births yielded the incremental number of births exposed prenatally.

[14] From Phibbs, Bateman, and Schwartz (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).

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 Figure 2) and the coefficient of substance abuse for the White population was marginally significant in some specifications. Thus for both 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 the illicit substance use variable. The fact that the instrument was less well correlated with illicit substance use in Whites 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, Landress, Barrett (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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## References


Calhoun BC, Watson, PT. The cost of maternal cocaine abuse:I Perinatal Cost. Obstet & Gynecol 1991; 78:731-734.

Centers for Disease Control, Atlanta, Georgia. Current trends: Statewide prevalence of illicit drug use by pregnant women--Rhode Island. Morbidity and Mortality Weekly Report 1990; 39:225-227.

Chasnoff IJ, Landress HJ, Barrett ME. The prevalence of illicit-drug or alcohol use during pregnancy and discrepancies in mandatory reporting in Pinellas county, Florida. N Engl J Med 1990; 322: 1202-06.

David RJ. Did low birthweight among US Blacks really increase? Am J Pub Health 1986; 76:380-384.

Freedman MA, et al. The 1989 revisions of the US standard certificates of live birth and death and the US standard report of fetal death. Am J Pub Health 1988; 78:168-172.

Greene W. Econometric Analysis. New York: Macmillan, 1990.

Griliches Z. Sibling models and data in economics: Beginning of a survey. J of Political Economy. 1979; 87:S37-S64.

Habel L, Lee J, Kaye K. Trends in maternal drug abuse during pregnancy in New York City 1978-1987. Presented at the 16th annual meeting of the American Public Health Association, Boston, MA. 1988.

Handler A. et al. Cocaine use during pregnancy: perinatal outcomes. Am J Epidem 1991; 133: 818-825.

Hausman J. Specification tests in econometrics. Econometrica 1978; 46:69-85.

Hausman, J and Taylor, W. "Panel data and unobservable individual effects." Econometrica 1981; 49:1377-1398.

Johnston J. Econometric Methods. New York: McGraw-Hill 1984.

Joyce T. The dramatic increase in rate of low birthweight in New York City: An Aggregate time-series analysis. Am J Pub Health 80:682-84. 1990.

Kleinman JC, Kessel SS. Racial Differences in Low Birth Weight. N Engl J Med 1983; 317:749-753.

Kleinman JC, Kopstein, A. Smoking during Pregnancy, 1967-1980. Am J Public Health 1987; 77:823-825.

Maddala, GS. The use of variance components models in pooling cross section and time series data. Econometrica 1971; 39:341-358.

Maddala GS. Limited-Dependent and Qualitative Variables in Econometrics. New York: Cambridge University Press, 1983.

Maddala GS. Introduction to Econometrics. New York: Macmillan Publishing Co. 1988.

McCalla S, Minkoff H, et al. The biological and social consequences of perinatal cocaine use in an inner city population: results of an anonymous cross-sectional study. Am J Obstet Gynecol 1991; 164: 625-30.

McCormick MC. The contribution of low birthweight to infant mortality and childhood morbidity. N Engl J Med 1985; 312:82-90.

Mundlak Y. On the pooling of time series and cross sectional data. Econometrica 1978; 46:69-86.

National Center for Health Statistics. Factors associated with low birthweight, 1976. Prepared by S. Taffel. Vital and Health Statistics, Series 21, No. 37. DHEW No. (PHS) 80-1915. Public Health Service. Washington, D.C.: U.S. Government Printing Office, April 1980.

Phibbs CS, Bateman DA, Schwartz RM. The neonatal costs of maternal cocaine use. JAMA 1991; 266: 1521-26.

Salkever D. The use of dummy variables to compute predictions, prediction

errors, and confidence intervals. Journal of Econometrics 1976; 4:393-397.

Schmidt, P and Sickles, R. "Production frontiers and panel data." Journal of Business & Economic Statistics 1984; 2:367-374.

Schutzman DL, Frankenfield-Chernicoff M, Clatterbaugh HE, Singer J. Incidence of Intrauterine Cocaine Exposure in a Suburban Setting. Pediatrics 1991; 88: 825-827.

Schwartz, R. What Price Prematurity? Fam Plan Perspect 1989; 21:170-174.

Zuckerman B, Frank DA, Hingson R, et al. Effect of maternal marijuana and cocaine use on fetal growth. N Engl J Med 1989; 320: 762-68.

FIGURE 1

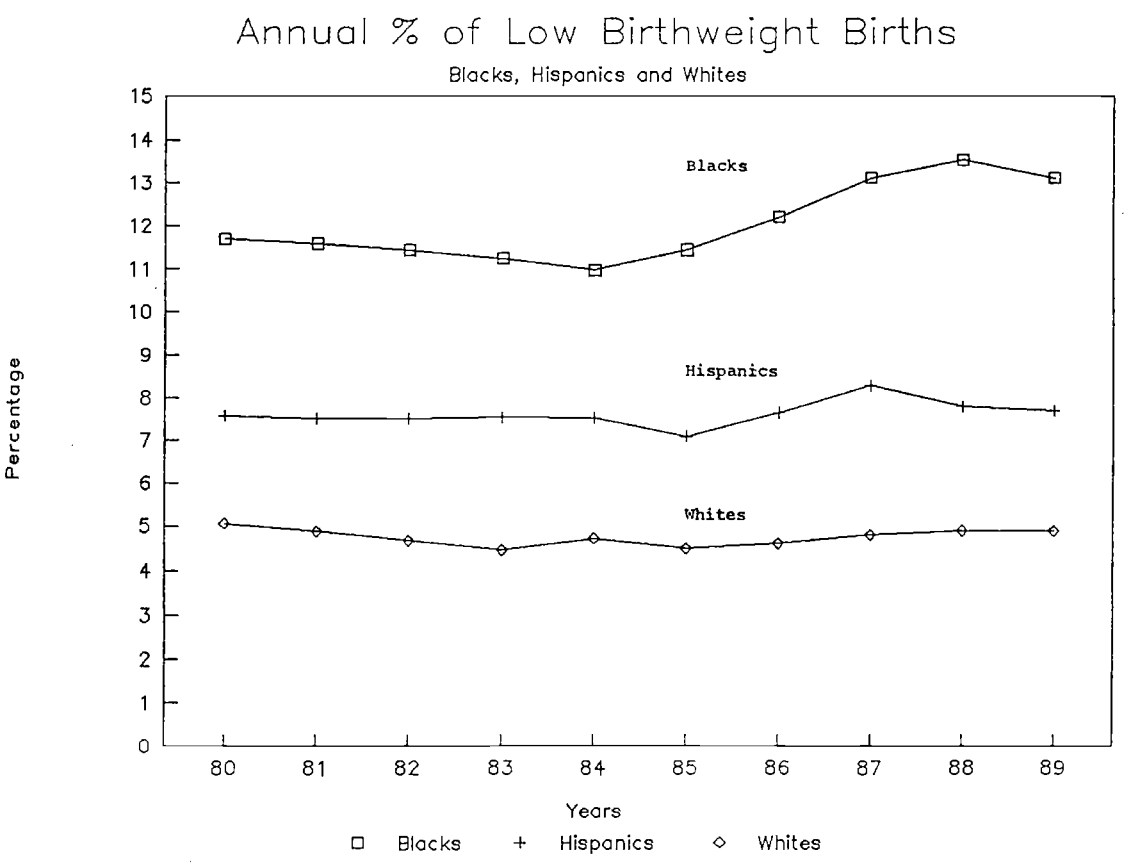
Annual % of Low Birthweight Births
Blacks, Hispanics and Whites
Percentage
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
0
Blacks
Hispanics
Whites
80
81
82
83
84
85
86
87
88
89
Years
Blacks
Hispanics
Whites

FIGURE 2

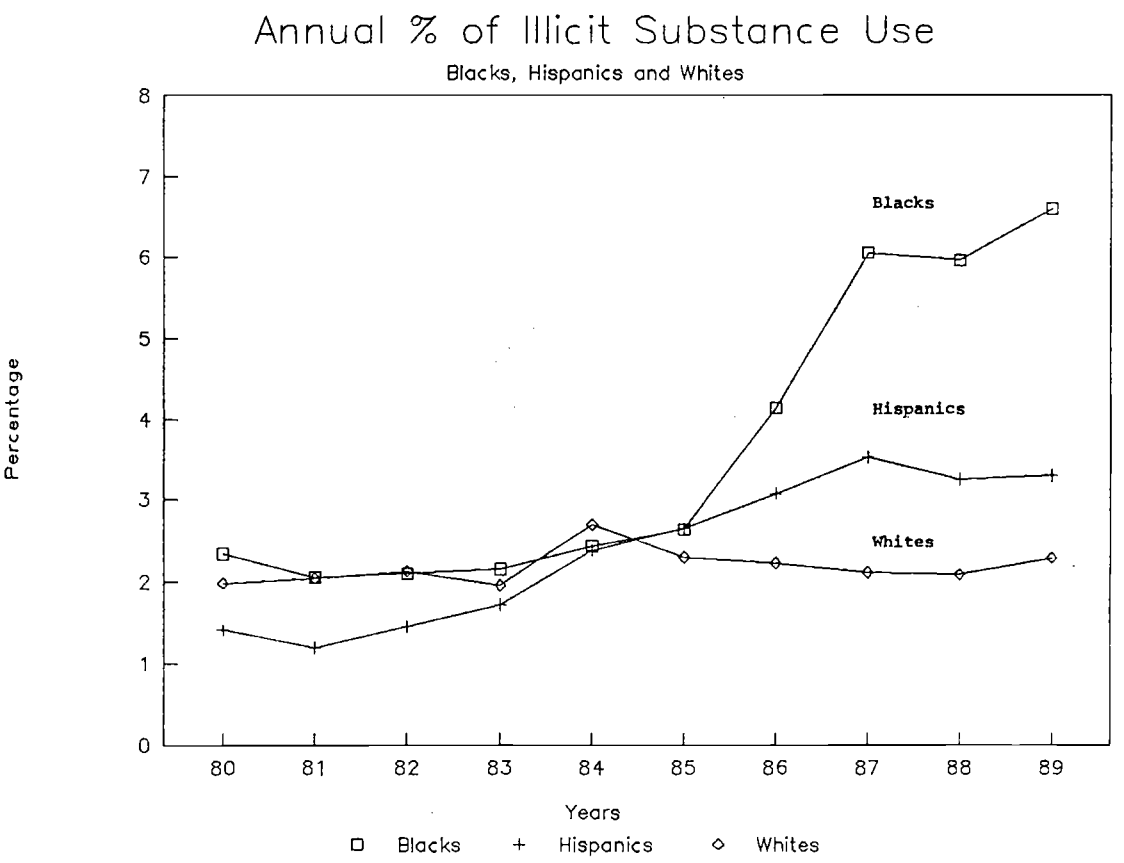
Annual % of Illicit Substance Use
Blacks, Hispanics and Whites
Percentage
0
1
2
3
4
5
6
7
8
80
81
82
83
84
85
86
87
88
89
Years
Blacks
Hispanics
Whites
Blacks
Hispanics
Whites

FIGURE 3

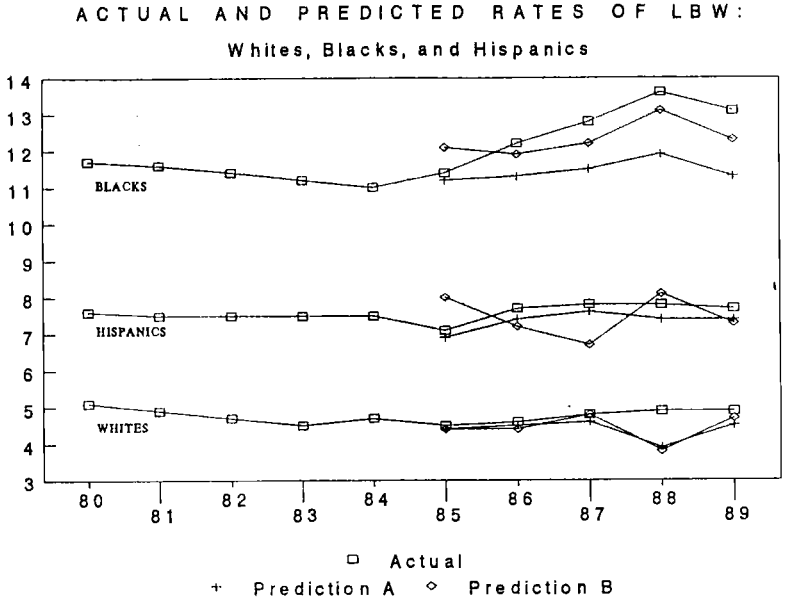
ACTUAL AND PREDICTED RATES OF LBW:
Whites, Blacks, and Hispanics
14
13
12
11
10
9
8
7
6
5
4
3
BLACKS
HISPANICS
WHITES
80
81
82
83
84
85
86
87
88
89
Actual
Prediction A
Prediction B

**TABLE 1**

**Means and Standard Deviations by Year, Race/Ethnicity**

**Blacks**

| | '80 | '81 | '82 | '83 | '84 | '85 | '86 | '87 | '88 | '89 | '80-89 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| % LBW | 11.7 (1.8) | 11.6 (2.5) | 11.4 (2.1) | 11.2 (2.0) | 11.0 (2.1) | 11.4 (2.2) | 12.2 (2.7) | 12.8 (2.9) | 13.6 (2.9) | 13.1 (2.6) | 12.1 (2.5) |
| % No Care | 9.1 (3.9) | 9.0 (3.8) | 10.6 (3.9) | 10.8 (3.8) | 13.2 (3.7) | 17.1 (6.5) | 19.9 (6.7) | 17.8 (6.6) | 15.2 (5.5) | 16.3 (7.2) | 14.2 (6.5) |
| % Drugs | 2.3 (1.2) | 2.0 (1.3) | 2.1 (1.4) | 2.1 (1.1) | 2.4 (1.6) | 2.6 (1.8) | 4.1 (2.5) | 5.3 (2.8) | 6.0 (3.2) | 6.6 (3.5) | 3.7 (2.8) |
| % Unmarried | 62.1 (11.2) | 62.3 (1.2) | 63.5 (10.9) | 64.1 (11.6) | 63.6 (11.2) | 64.6 (11.1) | 65.5 (11.1) | 65.6 (11.0) | 65.8 (9.9) | 66.9 (10.0) | 64.5 (10.8) |
| % Smoke | 8.5 (7.8) | 8.9 (9.7) | 8.5 (8.6) | 7.9 (7.8) | 6.2 (6.0) | 6.2 (6.0) | 6.7 (6.1) | 6.7 (5.5) | 10.7 (6.0) | 10.3 (5.4) | 8.1 (7.0) |
| % Parity>3 | 14.0 (2.5) | 14.2 (2.5) | 14.8 (2.5) | 15.8 (4.6) | 11.0 (2.0) | 14.3 (4.4) | 14.0 (4.3) | 13.6 (2.5) | 16.9 (4.3) | 16.0 (5.9) | 14.5 (4.1) |

**Hispanics**

| | '80 | '81 | '82 | '83 | '84 | '85 | '86 | '87 | '88 | '89 | '80-89 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| % LBW | 7.6 (1.6) | 7.5 (1.6) | 7.5 (1.7) | 7.5 (1.4) | 7.5 (1.7) | 7.1 (1.5) | 7.7 (1.8) | 7.8 (1.7) | 7.8 (1.8) | 7.7 (1.8) | 7.6 (1.7) |
| % No Care | 10.1 (7.7) | 9.7 (6.9) | 9.7 (5.4) | 10.4 (5.4) | 9.9 (5.1) | 11.7 (4.8) | 13.8 (6.1) | 13.7 (6.3) | 13.2 (4.5) | 14.8 (7.0) | 11.9 (6.2) |
| % Drugs | 1.4 (0.7) | 1.2 (0.8) | 1.4 (1.0) | 1.7 (1.1) | 2.4 (1.8) | 2.6 (1.7) | 3.1 (1.9) | 3.1 (1.8) | 3.3 (1.7) | 3.3 (1.9) | 2.4 (1.7) |
| % Unmarried | 44.8 (13.6) | 46.5 (12.8) | 47.3 (12.9) | 48.5 (12.7) | 49.5 (12.3) | 50.4 (12.0) | 52.5 (12.1) | 52.2 (11.2) | 51.9 (10.5) | 54.3 (10.2) | 50.1 (12.1) |
| % Smoke | 5.0 (3.8) | 6.9 (7.0) | 5.4 (4.7) | 5.1 (4.7) | 4.5 (2.7) | 4.5 (3.0) | 5.2 (3.0) | 4.1 (2.2) | 5.7 (2.7) | 5.4 (2.4) | 5.2 (3.8) |
| % Parity>3 | 9.9 (2.4) | 10.1 (2.3) | 10.0 (2.5) | 10.3 (2.4) | 7.8 (1.7) | 8.4 (2.4) | 8.7 (2.5) | 8.1 (1.8) | 13.6 (6.9) | 13.1 (8.2) | 10.1 (4.6) |

TABLE 1 (Continued)

Whites

| | '80 | '81 | '82 | '83 | '84 | '85 | '86 | '87 | '88 | '89 | '80-89 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| % LBW | 5.1 (1.2) | 4.9 (1.0) | 4.7 (1.3) | 4.5 (0.9) | 4.7 (0.7) | 4.5 (0.8) | 4.6 (1.0) | 4.8 (1.0) | 4.9 (1.3) | 4.9 (1.5) | 4.8 (1.1) |
| % No Care | 2.9 (1.8) | 2.7 (1.2) | 3.2 (1.7) | 3.7 (2.3) | 3.5 (1.6) | 4.6 (2.6) | 5.2 (2.8) | 4.7 (2.4) | 6.9 (2.4) | 7.1 (3.5) | 4.5 (2.7) |
| % Drugs | 2.0 (2.3) | 2.0 (2.3) | 2.1 (2.4) | 2.0 (1.9) | 2.7 (2.2) | 2.3 (1.6) | 2.2 (1.5) | 2.1 (1.3) | 2.1 (1.2) | 2.3 (1.4) | 2.2 (1.8) |
| % Unmarried | 9.1 (6.7) | 9.6 (6.1) | 10.0 (6.1) | 9.7 (5.4) | 10.5 (6.7) | 11.1 (6.3) | 12.1 (6.8) | 12.4 (6.2) | 13.2 (7.9) | 15.4 (8.1) | 11.3 (6.9) |
| % Smoke | 2.2 (1.9) | 2.1 (2.0) | 2.0 (1.7) | 1.9 (1.8) | 2.2 (1.8) | 2.4 (2.1) | 3.0 (2.2) | 2.9 (2.0) | 6.4 (3.5) | 7.0 (3.7) | 3.2 (3.0) |
| % Parity>3 | 10.2 (5.4) | 11.0 (5.4) | 11.7 (5.5) | 12.1 (6.4) | 9.7 (6.7) | 13.0 (7.4) | 12.9 (7.2) | 12.7 (6.8) | 15.7 (8.2) | 14.8 (8.3) | 12.4 (7.0) |

* Means and standard deviations are weighted by the number of births in the Health District

TABLE 2

Low Birthweight Rate Regressions Corrected and Uncorrected for Measurement Error -- Blacks[a]

| Model | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Variable | OLS | IV | OLS | IV | OLS | IV | OLS | IV |
| % Drugs | .269 | .416[b] | .223 | .432[b] | .281 | .243[b] | .144 | -.054[b] |
| | (6.68) | (7.41) | (4.38) | (5.75) | (7.04) | (3.98) | (2.64) | (.21) |
| % Smoke | .083 | .077 | .074 | .064 | .056 | .067 | .014 | .011 |
| | (4.82) | (4.27) | (4.07) | (3.37) | (2.47) | (2.93) | (1.59) | (.45) |
| % No care | .030 | .026 | .045 | .040 | .028 | .037 | .046 | .049 |
| | (1.79) | (1.53) | (2.20) | (1.86) | (1.79) | (2.31) | (2.40) | (2.47) |
| % Unmarried | .084 | .081 | .089 | .087 | .061 | .127 | .099 | .117 |
| | (6.85) | (6.45) | (6.90) | (6.56) | (1.90) | (4.14) | (3.01) | (3.56) |
| % Parity >3 | .022 | .025 | .002 | .003 | .045 | .049 | .020 | .017 |
| | (1.09) | (1.18) | (.09) | (0.13) | (2.51) | (2.64) | (1.01) | (1.85) |
| Constant | 4.117 | 3.827 | 4.481 | 4.260 | 6.00 | 1.96 | 4.88 | 4.17 |
| | (6.30) | (5.73) | (6.19) | (5.77) | (3.10) | (1.06) | (2.46) | (2.04) |
| Time effects | no | no | yes | yes | no | no | yes | yes |
| Health district effects | no | no | no | no | yes | yes | yes | yes |
| F | 113.94 | | 42.52 | | 35.89 | | 31.43 | |
| $\chi^{2,c}$ | 14.09 | | 14.34 | | .07 | | .62 | |
| $\chi^{2,d}$ | --- | | --- | | 2.12 | | 3.91 | |
| Adj $R^2$ | .67 | | .68 | | .81 | | .82 | |
| Rho[e] | .40 | .34 | .21 | .35 | -.03 | -.03 | -.11 | -.06 |
| N | 280 | 280 | 280 | 280 | 280 | 280 | 280 | 280 |

[a] The absolute value of t-statistics and asymptotic 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)

TABLE 3

Low Birthweight Rate Regressions Corrected and Uncorrected for Measurement Error -- Hispanics[a]

| Model | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Variable | OLS | IV | OLS | IV | OLS | IV | OLS | IV |
| % Drugs | .173 | .214[b] | .281 | .308[b] | .017 | .076[b] | .019 | .121[b] |
| | (3.87) | (2.35) | (6.12) | (3.01) | (0.31) | (0.69) | (0.29) | (0.39) |
| % Smoke | .042 | .029 | .021 | .014 | .023 | .025 | .027 | .028 |
| | (2.17) | (1.52) | (1.10) | (0.71) | (1.09) | (1.16) | (1.20) | (1.24) |
| % No care | .008 | -.001 | .030 | .011 | .038 | .037 | .034 | .035 |
| | (0.54) | (-0.007) | (2.01) | (0.75) | (2.01) | (1.98) | (1.65) | (1.65) |
| % Unmarried | .078 | .087 | .075 | .089 | .005 | .006 | .008 | .009 |
| | (9.33) | (11.02) | (9.40) | (11.66) | (0.30) | (0.43) | (0.39) | (0.45) |
| % Parity >3 | .040 | .045 | .030 | .050 | .031 | .028 | .033 | .032 |
| | (2.64) | (3.00) | (1.91) | (3.15) | (2.34) | (2.03) | (2.12) | (2.04) |
| Constant | 2.416 | 1.984 | 3.022 | 2.390 | 5.235 | 5.152 | 5.236 | |
| | (8.64) | (7.33) | (9.69) | (7.98) | (8.75) | (8.72) | (7.39) | (6.25) |
| Time effects | no | no | yes | yes | no | no | yes | yes |
| Health district effects | no | no | no | no | yes | yes | yes | yes |
| F | 104.09 | | 46.27 | | 29.53 | | 23.82 | |
| $\chi^{2,c}$ | .3 | | .09 | | .37 | | .11 | |
| $\chi^{2,d}$ | --- | | --- | | 46.45 | | 22.00 | |
| Adj $R^2$ | .64 | | .68 | | .77 | | .77 | |
| Rho* | .26 | .22 | .08 | .16 | -.11 | -.12 | -.11 | -.13 |
| N | 290 | 290 | 290 | 290 | 290 | 290 | 290 | 290 |

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

TABLE 4

Low Birthweight Rate Regressions Corrected and Uncorrected for Measurement Error -- Whites[a]

| Model | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Variable | OLS | IV | OLS | IV | OLS | IV | OLS | IV |
| % Drugs | .051 | .181[b] | .054 | .127[b] | .023 | -3.618 | .016 | 2.099 |
| | (2.04) | (1.10) | (2.25) | (0.95) | (0.61) | (0.15) | (0.43) | (0.33) |
| % Smoke | .012 | .009 | .037 | .033 | .023 | .024 | .047 | .053 |
| | (0.77) | (0.51) | (1.99) | (1.76) | (0.97) | (0.15) | (1.58) | (0.46) |
| % No care | .021 | .015 | .064 | .056 | .012 | .007 | .073 | .069 |
| | (0.81) | (0.54) | (2.39) | (2.06) | (0.35) | (0.03) | (1.92) | (0.45) |
| % Unmarried | .065 | .067 | .066 | .070 | .031 | .028 | .085 | .086 |
| | (5.87) | (5.33) | (6.15) | (5.96) | (1.25) | (0.17) | (2.96) | (0.73) |
| % Parity >3 | -.027 | -.029 | -.025 | -.026 | -.023 | -.024 | .001 | .001 |
| | (3.82) | (3.54) | (3.70) | (3.46) | (1.40) | (0.22) | (0.01) | (0.02) |
| Constant | 3.991 | 3.764 | 4.217 | 4.089 | 3.977 | 7.075 | 3.88 | 2.479 |
| | (32.30) | (13.71) | (25.63) | (16.88) | (19.00) | (0.35) | (15.90) | (0.56) |
| Time effects | no | no | yes | yes | no | no | yes | yes |
| Health district effects | no | no | no | no | yes | yes | yes | yes |
| F | 21.87 | | 10.88 | | 5.38 | | 5.26 | |
| $\chi^{2,c}$ | .64 | | .31 | | .02 | | .11 | |
| $\chi^{2,d}$ | -- | | -- | | 3.41 | | 5.15 | |
| Adj $R^2$ | .29 | | .35 | | .34 | | .39 | |
| Rho* | .15 | .17 | .03 | .13 | .15 | .58 | .06 | .50 |
| N | 260 | 260 | 260 | 260 | 260 | 260 | 260 | 260 |

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

TABLE 5

Decomposition of change in low birthweight rates 1984-1989
high and low estimates

| % Point Change in LBW attributable to.... | Blacks | | Hispanics | | Whites | |
|---|---|---|---|---|---|---|
| | High[a] | Low[b] | High[a] | Low[b] | High[a] | Low[b] |
| Drugs | 1.81 | 0.60 | 0.28 | 0.02 | -0.02 | -0.01 |
| Smoke | 0.26 | 0.06 | 0.01 | 0.02 | 0.18 | 0.23 |
| No care | 0.12 | 0.14 | 0.05 | 0.17 | 0.23 | 0.26 |
| Unmarried | 0.29 | 0.33 | 0.43 | 0.04 | 0.32 | 0.42 |
| Parity3 | 0.02 | 0.10 | 0.27 | 0.17 | -0.13 | 0.01 |
| Time | -0.23 | 0.91 | -0.78 | -0.11 | -0.49 | -0.79 |
| Total | 2.27 | 2.14 | 0.25 | 0.31 | 0.09 | 0.12 |
| Actual Percentage Point Change in LBW | 2.1 | | 0.2 | | 0.2 | |

[a] Based on coefficients from model using instrumented OLS with time dummies for blacks and hispanics (column 4, Tables 2 and 3) and non-instrumented OLS with time dummies for whites (column 3 Table 4).

[b] Based on coefficients from model using non-instrumented OLS with time and district dummies (column 7, Tables 2, 3 and 4).

**TABLE 6**

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 | | Excess costs | |
|---|---|---|---|---|
| | High | Low | High | Low |
| Blacks | 2760 | 1075 | $37,864 | $14,748 |
| Hispanics | 477 | 365 | $ 6,544 | $ 5,007 |
| Whites | 606 | 540 | $ 8,314 | $ 7,408 |
| Total | 3843 | 1980 | $52,722 | $22,161 |

[a] The average cost of a LBW birth is $13,719 in 1990 dollars (Schwartz 1989).