Maternal Depression and the Production of Infant Health

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Maternal Depression and the Production of Infant Health

Abstract

Depression is most prevalent among women of childbearing age and especially among low income women, and the medical literature shows it to have adverse effects on infant health. Yet, maternal depression has been overlooked in economic studies of infant health production. This research incorporates maternal depressive symptoms into a standard infant health production model and estimates both structural and reduced form birth weight equations using samples of nonHispanic white and black women from the NMIHS. A byproduct of this research is an empirical investigation into factors associated with maternal depressive symptoms. All results show that depressive symptoms have a negative effect on birth weight and that they may operate through several channels such as smoking and prenatal care.

1. Introduction

Understanding the correlates of infant health has become an issue of great importance since we have learned that it has tremendous implications for childhood and adult health and well-being. Low birth weight is a key indicator of infant health as low birth weight babies have much higher mortality rates. In addition, caring for these low birth weight babies is substantially more costly (Currie and Gruber 1996). Perhaps even more compelling is the mounting evidence that the negative effects of being born at a low birth weight persist well into adulthood and can sometimes take many years to appear.[1] Reducing the incidence of low birth weight, possibly through improved prenatal care, is therefore a worthwhile policy goal. But what is the most effective way to do this?

The recent, dramatic expansion in the Medicaid eligibility of pregnant women has been fueled, at least in part, by the belief that it would increase prenatal care and ultimately improve infant health. However, expanded eligibility does not necessarily lead to increased participation as the take-up rate for Medicaid is far below 100 percent (Gruber 1997). Furthermore, if individuals drop their private insurance, which is presumably of superior quality, upon receipt of Medicaid, then expanding Medicaid may not improve infant health.[2] Finally, even if expanding Medicaid coverage leads to increased prenatal care, those increases may not substantially improve infant health if prenatal care is not effective.

We suggest that a potentially important element has been left out of the policy discussion -- the mother's mental health. Epidemiological research has shown that infants of depressed mothers show signs of poorer health (Abrams et al 1995; Field 1995, 1998; Dawson et al 1997a; Dawson et al 1997b; Jones et al 1997; Locke et al 1997) and are more likely to be preterm (Orr and Miller 1995; Orr, James and Prince 2002). And maternal depression is not a rare event; the lifetime risk for depression for women is estimated at 10 to 25% and peaks during their childbearing years (Wisner et al 1999; Desai and Jann 2000). Low income women are even more susceptible to depression and there is evidence that those on welfare suffer greater depression than other low income women (e.g., Lennon, Blome and English 2001). And yet, current economic research completely overlooks the role that maternal depression plays in infant health. In studies of welfare reform, there is a growing recognition of the role depression plays in preventing women from getting off welfare and in finding and keeping employment. For instance, Lennon, Blome and English (2001) provide an extensive survey of the prevalence of depression among low income women and the consequences it has for them. They also discuss and evaluate different kinds of welfare and unemployment programs that incorporate treating mental illness. Our results suggest that perhaps the Medicaid program and, more generally, the health care providers who treat pregnant women also need to recognize the important role that maternal depression plays in infant health.

We begin with a standard infant health production model in which "depression" may affect infant health directly as part of the technology of production and also as a factor that influences the choice of inputs (e.g., prenatal care). Using the National Maternal and Infant Health Survey (NMIHS), we estimate birth weight and prenatal care equations that incorporate measures of maternal depressive symptoms.[3] Because past researchers have found significant differences between whites and blacks, we stratify by race. The NMIHS does not contain the ideal measure of maternal depression -- a diagnosis of depression during the pregnancy -- and so we construct several measures to verify the validity of our results. One byproduct of constructing these measures is an investigation into factors associated with maternal depressive symptoms. Across all measures and samples we consistently find that maternal depressive symptoms have a negative direct effect on birth weight and that they may operate through other channels, such as reduced prenatal care and increased unhealthy behaviors, as well.

2. The Production of Infant Health

We extend the infant health production model of Rosenzweig and Schultz (1982, 1983) by adding 'depression' as both a factor that affects the technology of production and a taste variable. The mother's utility function is defined as

$$\max_{M} u\,(H, D, X) \quad \textit{utility function} \tag{1}$$

where *H* reflects her infant's health, *D* is her mental state or 'depression', and *X* is a composite commodity. She then maximizes this utility function by choosing prenatal care, *M*, subject to the infant health production function,

$$H = H\left(M, D, Z\right) \quad \textit{infant health production} \tag{2}$$

and her budget constraint,

$$PM + X = I \quad \textit{Budget constraint.} \tag{3}$$

Referring to equation 2, prenatal care, *M*, is expected to enhance infant health, *H*, while 'depression', *D*, is expected to adversely affect infant health. In addition, other variables (*Z*) such as the mother=s education, age and height (as suggested by Warner 1995, 1998) and past medical history, and other maternal behaviors, are expected to directly affect infant health. Equation 3 represents the budget constraint where *P* is the out-of-pocket price per unit of prenatal care, I is income and the price of the composite good is normalized to 1.0.

The woman then chooses the prenatal care, M^*, that maximizes her utility subject to equations 2 and 3, which yields the two-equation model typically estimated C the desired prenatal care demand equation,

$$M^* = M(D, Z, P, I), \tag{4}$$

and desired infant health equation,

$$H^* = H(M^*, D, Z). \tag{5}$$

This is the typical framework employed by recent studies of infant health and prenatal care (e.g., Grossman and Joyce 1990; Warner 1998; Currie and Grogger 2000). It demonstrates how variables such as age and education can have both a direct and indirect (through prenatal care) effect on infant health. We introduce maternal 'depression' as another such factor that can have dual influences – on the decision to get prenatal care as well as having a direct impact on the production of infant health. This is consistent with the epidemiological literature such as Orr and Miller (1995) that has recognized these dual avenues as well. In particular, Orr and Miller (1995, p. 169) in their review of maternal depressive symptoms and pregnancy outcomes note the "two primary mechanisms by which depressive symptoms might influence birthweight": 1) they may lead to more harmful behaviors (including delayed prenatal care), and 2) "a more direct association…" as "depressed mood has been increasingly linked to biochemical/hormonal alterations in the body."

One might argue that 'depression' may be jointly produced with infant health (health inputs affect both). However, in a recent study, Williams et al (1999, p.64) find that among family physicians, general internists and OB-GYNs, OB-GYNs stand out as having higher physician barriers to treating depression (e.g., having low confidence in or incomplete knowledge of treatment). It therefore seems unlikely that the woman is seeking prenatal care to treat her depression. We also hesitate to make the assumption implied by such an argument that maternal 'depression' is a choice variable and under the mother's control.[4] However, maternal 'depression' may be endogenous to birth weight in that the mother's unobserved health endowment may affect both. With this caveat in mind, our estimated effects of observed maternal depressive symptoms should be viewed as measuring its value as a signal of maternal health and behavioral problems that could lead to a poor birth outcome.

Despite our reservations about treating 'depression' as a choice variable, we modify this framework to allow for such a possibility in our empirical analysis. In essence, we specify a reduced form maternal 'depression' production function, which we then use to construct an instrumental variable for D to be used in estimating the structural infant health equation (5). We therefore also estimate the model using predicted values of 'depression' as a way of dealing with both the possible endogeneity of maternal 'depression' and the limitations of our 'depression' measure. One byproduct of this research, then, is an empirical investigation into factors associated with maternal depressive symptoms, which has been overlooked in health economics research.[5]

This theoretical framework clarifies the identifying restrictions used in the typical infant health model C income (I) and factors affecting the price of prenatal care (P) such as insurance status and community level variables identify prenatal care.[6] Warner (1998) discusses in detail his difficulty in finding satisfactory restrictions and notes that the structural birth weight equation may be weakly identified as a result. Such weak identification has been blamed in part for the lack of effectiveness of prenatal care found by many researchers (see, for example, Currie and Grogger 2000, who use policy changes in Medicaid and welfare as identifiers).

We wish to isolate the effect of adding depression to the standard model of infant health and so we employ typical identifying restrictions. In particular, we use a number of aggregate variables -- health care price index aggregated to the state level, the ratio of Medicaid fees to private fee levels paid to OB-GYNs in the state (as in Gray 2001), population density, number of OB-GYNs per 1000 state population, number of general practitioners per 1000, and number of hospitals and HMO's per 1000. We also include some individual-level ones -- whether the mother's prenatal care was paid for by Medicaid or private insurance, her income and whether she lives in an urban area. As discussed in more detail in the results section, we subject the instrument sets to the usual tests, which they pass for every specification reported.

The variables common to both specifications are mother=s height, age, and age squared, mother's education and education squared, number of own children living with her, whether she is living with the father, and her medical history (parity, number of prior fetal deaths). We also include the gender of the infant in both equations for consistency, although it is probably not known at the time that prenatal care is sought. This is our structural model.

As another test of the effects of depression on birth weight, we also estimate a reduced form (or quasi-structural form, in the case of models in which depressive symptoms is viewed as endogenous) birth weight equation in which prenatal care is eliminated and birth weight is regressed on all of the variables in the system. In this way, the combined direct and indirect effects of depressive symptoms on birth weight are captured without the need to identify prenatal care. Currie and Grogger (2000) employ a similar exercise in determining the effects of welfare and Medicaid on infant health.

A final complication in the model are maternal behaviors such as smoking and drinking alcohol, which are strongly associated with poor birth outcomes, yet are likely a maternal choice. Including these variables and treating them as endogenous further strains an already weakly identified birth weight equation. Most studies therefore either omit these behaviors (e.g., Liu 1998; Currie and Grogger 2000) or treat them as exogenous, sometimes using information on behaviors before the pregnancy, (e.g., Grossman and Joyce 1990; Warner 1998; Kaestner 1999). Because we suspect that such maternal behaviors are also strongly related to maternal 'depression,' we estimate the model both with and without these behaviors. When they are included, we use the behavior *before* the pregnancy to avoid endogeneity bias. In this way, we can investigate whether maternal smoking and drinking is an important avenue for maternal 'depression' to affect birth outcomes.

3. Maternal 'Depression' – Cause, Effect and Data Issues

Maternal 'depression' is a term that is sometimes used to describe two distinct phenomena, "a major depressive disorder and high levels of depressive symptoms" (Orr and Miller 1995, p. 166). "A clinical diagnosis of depression is generally made on the basis of the presence, for at least 2 weeks, of a depressed mood and/or loss of interest or pleasure in activities that are usually a source of enjoyment, along with other symptoms such as appetite and sleep disturbance; fatique and loss of energy; feelings of worthlessness; and difficulty in concentrating. These symptoms represent a change in functioning from that which is normal for the individual." (Orr and Miller 1995, p. 166). In contrast, many screening tools, such as the CES-D measure used in our analysis and described shortly, are designed to estimate the prevalence of high levels of depressive symptoms.[7] Therefore, our empirical study is actually studying the impact of *depressive symptoms* on infant health, not depression *per se*.

Ample evidence exists in the medical literature that maternal 'depression' is associated with poor infant and child health and development. However, a persistent question is the direction of causality C do depressed mothers lead to unhealthy infants or do unhealthy infants lead to depressed mothers? As summarized in Field (1995), it appears to be a combination of both. One strong piece of evidence that maternal depression causes poor infant health is provided by Abrams et al (1995) who look at the behavior of *newborns* born to depressed mothers versus those born to nondepressed but otherwise comparable mothers. They find that A... infants of depressed mothers are already showing nonoptimal behaviors at birth..,@ and that they are Areminiscent of newborns who are small for date,@ (p. 238). Even more relevant for our study is the evidence provided by Orr, James and Prince (2002), who find that mothers with higher depressive symptoms (measured by the CES-D) are more likely to have a preterm birth.

In one respect, the issue of causality is irrelevant for us because we are looking at birth weight. Interactions that occur after the birth should have no impact. However, the *timing* of our measures of depressive symptoms raises again the issue of causality. The 1988 NMIHS surveyed the mother on average 17 months after the birth of her child and the depressive symptoms measure refers to the past week.[8] Therefore, our measure of depressive symptoms is taken after the delivery of the infant.

This poses two problems for us. First of all, our measure may be only weakly correlated with depressive symptoms during the pregnancy and it may also capture post-partum depression. However, Najman et al (2000), a study that follows women up to 5 years after the birth, lessens this concern considerably by finding that postpartum depression is usually short-lived and mild, and that many cases of depression that occur after the birth are recurrences of a previous episode. Moreover, research on postpartum depression in general consistently finds that 'depression' during pregnancy is one of the strongest predictors of postpartum depression (e.g., Gotlib et al 1991; Seguin et al 1999; Beck 2001; Josefsson et al 2001). Thus, to the extent that our measure is capturing postpartum depression, there is strong evidence that it is reasonably correlated with prenatal depression.

We can also gain some reassurance from the broader 'depression' literature. In Kessler et al 1997, for instance, over seventy percent of individuals with either 'minor' depression (2-4 symptoms) or 'major' depression (5 or more symptoms) suffers recurrent episodes of depression and have had more than 8 episodes on average. Again, this suggests that our measure of depressive symptoms may be reasonably correlated with prenatal depressive symptoms. And, to the extent that our measure is not a good proxy for prenatal depression, any estimated effects should be biased towards zero, making our estimates conservative.

The second problem, however, could lead to a falsely stronger effect via reverse causality. As noted above and in Field (1995) having a low birthweight or otherwise unhealthy infant may cause a woman to become 'depressed.' However, Singer et al (1999) provides two insights. First, it is not the birth weight of the infant that matters, but whether they are high or low risk. Second, the effects of having a sick infant vary as the child grows older. We use these insights in developing our instrument for depressive symptoms.

Another possible problem for our measurement of depressive symptoms occurs because the NMIHS contains no information about treatment. This could be a serious problem if the treatment itself has an effect on birth weight. A woman who is successfully treated for 'depression' would exhibit few or no depressive symptoms and yet may have a poor birth outcome precisely because of the treatment. Another possible problem is if the *choice* by the mother to receive treatment is in some way correlated to the birth outcome (if, for example, women who more heavily discount the health of their infants opt for pharmacological treatment). We believe, however, that it is unlikely that this bias is widespread in our data and to the extent it exists should bias our results against finding an effect of depressive symptoms on birth weight.

Existing evidence regarding the safety of pharmacological treatment of depression is far from definitive although some suggest that exposure may lead to increased risk of premature birth and lower birth weight (e.g., Grush and Cohen 1998; Wisner et al 1999).[9] However, this uncertainty about safety during pregnancy likely leads to low treatment rates among pregnant women, which is corroborated by existing evidence.[10] Thus, given the apparent low rate of pharmacologic treatment among pregnant women and the uncertain effects on birth weight of such treatment, we are doubtful that this is having a serious impact on our results. Furthermore, it appears that whatever impact there is would tend to bias our results towards zero as women with low apparent depressive symptoms have adverse birth outcomes due to the treatment they are receiving. And, to the extent that it is a choice the woman makes, one would expect this choice to be made by women who either expect greater costs of going untreated or more heavily discount the costs to their infants. Both cases should lead to a downward bias (towards zero) of the effect of *observed* depressive symptoms on birth weight.

Given the possible problems with our measure, we conduct sensitivity checks by constructing three additional measures and then using them to check the robustness of our results. The first new measure uses the depression measure taken at the 1991 NMIHS follow-up to construct an average of depressive symptoms at two points in time (i.e., (CES-D in 1988 NMIHS + CES-D in 1991 NMIHS)/2). Given the recurrent nature of 'depression,' using the level of depressive symptoms at two points in time may be a superior predictor of a general susceptibility to depressive symptoms. Even in our data there is evidence of a recurrent quality as these two measures are fairly highly correlated. However, this measure still suffers from the possibility of reverse causality (i.e., sick infants cause depressed mothers) and that the effects of choosing treatment are ignored.

Our other two measures use the survey measures along with other information about the mother to predict her depressive symptoms during pregnancy. This approach is like using the two-sample instrumental variables approach of Angrist and Krueger (1992, 1995) in which the mothers, at a later date, are providing instruments for themselves during the pregnancy. Specifically, we estimate the 1988 NMIHS measure of depression as a function of characteristics of both the mother and infant, as well as how much time has lapsed since the birth. We then predict her depression during the pregnancy 1) using variable values during the pregnancy instead of after (where possible), and 2) setting infant characteristics and the length of time since the birth to the sample averages. In this way, we hope to capture any changes in the mother's environment between the pregnancy and the time of the survey and also control for any depressive symptoms that are induced by having a sick infant. Using a predicted measure of depressive symptoms also addresses the issue of its possible endogeneity in that we are essentially using an instrumental variable.

The specific depressive symptoms equation we estimate is

> *1988 NMIHS CES-D = f (receives AFDC*^{s}*, receives medicaid*^{s}*, has private insurance*^{s}*, income in the 12 months before pregnancy, cohabits with dad*^{s}*, number of kids cohabiting*^{s}*, mother's education and education*^{2}*, mother's age and age*^{2}*, age at first marriage, number of marriages, never married, number of previous fetal deaths, cigarettes smoked*^{s}*, alcoholic drinks before pregnancy, marijuana or cocaine before pregnancy, exercised before pregnancy, wanted pregnancy, urban, state mental health agency expenditures per capita, psychiatrists per 1000, psychologists per 1000, social workers per 1000, number of multiservice mental health organizations per 1000, HMOs per 1000, pregnant now*^{s}*, length of time since birth, length*^{2}*, length*number of sick baby visits per month, length*^{2}** number of sick visits)*

The superscript *s* denotes that the variable's value refers to the time of the survey; these variables are all replaced with their values during the pregnancy (with the exception of smoking, which is replaced with its value *before* the pregnancy). For example, receipt of AFDC has been found to be highly correlated with depressive symptoms (Lanzi et al 1999; Lennon, Blome and English 2001) and so the CES-D measure is estimated as a function of AFDC receipt at the time of the survey. We then use whether the mother received AFDC during the pregnancy to predict her depressive symptoms during the pregnancy. Other variables did not change during the period (e.g., education, age at first marriage) or are only available at one point in time (e.g., family income) and so the same values are used for estimation and prediction.

Several maternal behaviors are only available before and during the pregnancy -- exercise, drinking alcohol and using drugs. For these we used the measures taken before the pregnancy to estimate depression because we believe they more closely approximate the mother's behavior after the birth.[11] We also use smoking, drinking, drug and exercise behaviors *before* the pregnancy to predict depression to avoid their possible endogeneity with birth weight, which as we note in section 2 is a common practice in infant health models. However, because many infant health studies omit these behaviors, we estimate an alternative specification that omits them from the depression equations; due to possible endogeneity we also omit whether the pregnancy is wanted.

We include several state-level variables that attempt to capture the availability of treatment for depression. These include the number of treatment facilities and HMO's, state expenditures on mental health agencies, and practitioners who might treat depression. Due to the insights from Singer et al (1999) and Najman et al (2000), we allow the length of time since the birth to enter in nonlinearly and affect the impact of having a sick infant, the latter of which is measured by the average number of sick visits per month for the infant. To purge our predicted measure of the effects of having a sick child and the passage of time, we set the number of sick visits and the length of time between the interview and the birth to the sample means.[12]

The variables in our depressive symptoms equation that provide identifying variation are therefore the mothers' receipt of AFDC, her marital history, variables measuring the availability of mental health treatment and, when included, behaviors and whether the mother wanted the pregnancy. In sum, we use four different measures of depression C 1) the 1988 NMIHS survey measure, 2) the average of the 1988 and 1991 NMIHS survey measures, 3) a predicted measure using the above equation and appropriate values of the variables, and 4) a predicted measure excluding behaviors (smoking, drinking, drugs and exercise) and whether the mother wanted the pregnancy. Admittedly, none of these measures is ›ideal;= however, finding similar results across the measures adds credence to their validity.

4. Data Description

Our primary data comes from the National Maternal and Infant Health Survey, 1988 (NMIHS), which is publicly accessible data published by the National Center for Health Statistics (NCHS). NMIHS contains information on women who were pregnant in 1988, although the actual interview took place on average 17 months after the delivery (and thus after 1988). Following Kaestner (1999) and Warner (1998), only data from the live birth sample are used. Grossman and Joyce (1990) point out that this selection causes bias because women who experience a live birth may be healthier or may have desired their child more. However, both Kaestner (1999) and Warner (1998) omit this correction, and Gray (2001) explores its impact by also including the fetal death sample and finds no substantial difference.[13] Therefore for simplicity and to conform to past studies using the NMIHS, we exclude it from our model.

Another consideration is that the NMIHS is not a simple random sample. It oversamples fetal deaths and infant deaths. Within the live birth sample, it oversamples blacks and, for both blacks and nonblacks, low and very low birth weight babies. Both because of this oversampling and because previous research finds important racial differences in birth weight (e.g., Warner 1995, 1998; Liu 1998) we stratify our sample into blacks and whites. This permits us to explore whether differences in maternal mental health can help explain some of the racial differences in birth outcomes that others have found. To control for the oversampling of low and very low birth weight babies, we follow Warner (1998), Kaestner (1999) and Gray (2001) and use the sample weights provided by the NMIHS.

As most studies do, we eliminate multiple births because such babies tend to be born at shorter gestation and lower birth weights, and because it leads to multiple birth observations that share one prenatal care observation. To construct the final samples, we begin with the 9146 live, singleton births. We then eliminate, in the following order, the small number of women who had no prenatal care (N=284)[14], those missing information for number of prenatal care visits (N=10), and those missing information for parity (N=79). We follow the typical practice of omitting teenagers (< 19 years old) and much older mothers (> 50 years), which eliminates 1168 observations. Following Warner (1998), gestations less than 20 weeks or greater than 45 weeks (N=48) and birth weights below 400 grams or above 6000 grams (N=35) were deleted. We also eliminate the 30 observations from Hawaii and the 28 observations from Alaska. After all of these exclusions, there are (9146-284-10-79-1168-48-35-30-28 =) 7464 observations, of which 3342 are nonHispanic blacks and 3232 are nonHispanic whites.

A large number of observations do not have the 1991 follow-up information such that the depression measure used in the second measure is unavailable. When the 1991 depression variable is used, we therefore use smaller subsamples of 2605 nonHispanic blacks and 2766 nonHispanic whites in estimation. Likewise, many of the observations had missing values for number of sick baby visits and length of time between the 1988 interview and the birth. Recall that these variables are only necessary to obtain estimates for equation (6) and are then set to sample means to predict depressive symptoms. We therefore use still different sample sizes to estimate the depressive symptoms. We use these different samples both to bolster sample size where possible and to minimize any bias that might be introduced by excluding observations with missing data.[15, 16]

The descriptions, means and standard deviations of the variables used in our analysis are reported in Appendix A1. The key variables in our analysis are prenatal care, birth weight and maternal depressive symptoms. Although there are a number of possible measures of prenatal care, the most widely used and least criticized measure is the *onset* (or delay) of prenatal care since it is widely believed that early prenatal care is important to a healthy pregnancy and it does not share the problem with number of visits that difficult pregnancies lead to more care.

Likewise, birth weight is the most commonly used measure of infant health in studies that use individual-level data and especially in those employing structural models. Some also estimate the incidence of low birth weight (e.g., Joyce 1999; Kaestner 1999; Gray 2001) as it is well established that low birth weight is associated with greater health risks and costs. While birth weight (continuous) is our primary measure of infant health, we include the incidence of low birth weight (discrete) as a measure when we estimate the reduced form equation.

The last key variable in the model is the mother=s mental health, which is measured in the NMIHS with the CES-D scale. This scale was developed in 1969 as part of the Community Mental Health Assessment (CMHA) program to identify the presence and severity of depressive symptomatology in the general population. However, it was not intended to discriminate among different types of depression nor to distinguish primary depressive disorders from secondary depression (Radloff and Locke, 1986). The CES-D scale is a self-reported survey consisting of 20 questions concerning depressed mood, feelings of guilt, worthlessness, helplessness, and hopelessness, psychomotor retardation, loss of appetite and sleep disturbance. Each response is scored from 0 to 3 based on the frequency of the occurrence in the past week, yielding a range of 0 to 60. Higher scores reflect more depressive symptoms.

The literature (e.g., Roberts and Vernon 1983; Radloff and Locke 1986; Zimmerman and

Coryell 1994; Locke et al. 1997; Lennon, Blome and English 2001) has consistently used a score of 16 or greater to indicate depression. Given this agreed-upon threshold, we also estimate our model using a depression dummy variable which equals one if the CES-D exceeds 15. The CMHA found that these scores are correlated with the presence of clinical depression diagnosed by a psychologist (Radloff and Locke 1986). Our variable is not, therefore, a clinical diagnosis but rather a measure of depressive *symptoms.* Although there are many symptom checklists, the CES-D is the most commonly used scale in studies of depressive symptoms in welfare populations (Lennon, Blome and English 2001).

Figure 1 reports the relative frequency of the CES-D in the 1988 survey for both samples. Two results are immediately apparent. First, the majority of women do *not* show depressive symptoms, as classified by the CES-D. Second, black women have more depressive symptoms than white women. Whereas only 18.7% of white women have CES-D scores greater than 15, 35.5% of black women do (see Appendix A.1).[17] The incidence of elevated depressive symptoms is therefore almost twice as great among black women as it is for white women. Furthermore, Figure 1 reveals that this tendency continues throughout the distribution of CES-D scores; white women have higher relative frequencies than blacks at scores of 9 or less and black women have higher frequencies than whites for scores above 9. This validates the recent concern that 'depression' is widespread among low income women because, as Table 1 and Appendix A.1 reveal, our black sample has much lower incomes than our white sample. It also suggests that maternal 'depression' could help explain at least part of the differences in birth outcomes between whites and blacks, as well as low income women more generally.

Table 1 reports the means of our key variables for 'nondepressed' versus 'depressed'

(1988 CESD score > 15) mothers, by sample. It is immediately apparent that the 'depressed' mothers have worse birth outcomes. Whereas the average birth weight is only about 100 grams less for 'depressed' mothers, the incidence of low birth weight (<2500 grams) and very low birth weight (<1500 grams) is much higher for 'depressed' mothers. Likewise, prenatal care begins later for 'depressed' mothers and they report facing much greater barriers to obtaining prenatal care.[18] 'Depressed' mothers appear disadvantaged in other ways as well; they are more likely to participate in Medicaid and AFDC and less likely to have private insurance, have lower family incomes and are more likely to be never married. Consistent with Saffer and Dave (2002), they also consume higher levels of tobacco and alcohol. Finally, we also see the persistence of depressive symptoms across the two surveys. The follow-up CES-D scores are quite a bit higher for those who were 'depressed' in the initial survey, and these 'depressed' mothers are about three times more likely to be classified as 'depressed' in the follow-up survey than the 'nondepressed'.

Table 1 suggests that maternal 'depression' may be potentially important in determining birth outcomes and may operate through several avenues, such as prenatal care and maternal behaviors. Our econometric results, presented in the next section, allow us to see if these results stand up to different 'depression' measures and to controlling for confounding factors such as age, education, economic status and other socioeconomic variables.

Most of the other variables are also from the NMIHS and are self-explanatory, with the exception of our aggregate level variables. Since only a woman=s state of residence (not city) is identified in the NMIHS, we must aggregate all data into state-level data. The health care price index is created by the American Chamber of Commerce Researchers Association (ACCRA)

Cost of Living Index, which includes price data from 256 US cities. Population density is taken from the Statistical Abstracts of the United States. The ratio of Medicaid to private fees for OB-GYNs is from Loprest and Gates, *State Level Data Book on Health Care Access and Financing* (1990, p. 103). Per capita state mental health agency per capita expenditures is from NASMHPD Research Institute, *Funding Sources and Expenditures of State Mental Health Agencies, Fiscal Year 1997.* The number of multiservice mental health organizations is from CMHS, *Mental Health, United States 1994.* The number of hospitals, HMO's, OB-GYNs, general practitioners, psychiatrists, psychologists and social workers are obtained from the *Area Resource File.* All such variables are converted to per 1000 state population.

5. Main Empirical Results

Table 2 reports the key results from the structural and reduced form birth weight equations using four alternative observed measures of depression -- the CES-D score in the 1988 survey and the average of the 1991 and 1988 surveys are each used as both a continuous measure and a discrete measure of depressive symptoms. For comparison, a 'typical' or baseline birth weight structural model is also estimated and reported at the top of Table 2. (The full set of results for these models is available upon request.) We estimate each variation of the model first excluding and then including maternal smoking and drinking (*before* pregnancy) to investigate the relationship between maternal behaviors, depressive symptoms and birth outcomes.

For every specification reported, we perform two tests on the quality of the instrument set: 1) whether the variables are jointly significant in the prenatal care equation, and 2) whether the overidentifying restrictions are supported by the data. We therefore check that the instruments have explanatory power in the prenatal care equation and, at the same time, do not have explanatory power in the infant health equation. All specifications satisfy both tests. We also perform a joint test of the significance of our aggregate-level identifiers and find that they are jointly statistically significant as well.

Recall that the structural model allows depression to have both direct and indirect (through *Onset*) effects on birth weight. Because an increase in the *Onset* of prenatal care means *less* prenatal care, we therefore expect depressive symptoms to increase *Onset*.[19] (Likewise, we expect *Onset* to decrease birth weight.) We can explore the direct effect of depressive symptoms by examining its effect on birth weight when prenatal care is controlled for. In contrast, our reduced form equations yield only an estimate of the total effect of depression.

Looking first at our baseline estimates of the structural model, it may be disconcerting to find that prenatal care has no effect on the birth weights of white infants; however, finding prenatal care to have a highly variable and often insignificant effect on birth weight is entirely consistent with past research. As noted by Currie and Grogger (2000), Warner (1998) and others, the often modest and widely variable effects of prenatal care may be due in part to difficulties in identifying the equation.[20] Our results suggest that the onset of prenatal care has no statistically significant effect for whites. For blacks, however, delaying prenatal care by one week appears to decrease birth weight anywhere from 65 to 83 grams and is statistically significant. These results are at the high end of the ranges of estimates reported by Warner (1998), who uses the same data and stratifies by race. The effects of maternal behaviors and the other (unreported) explanatory variables, such as health history, age and education, are as typically found in the literature.

Adding depression to the model does not substantively affect the estimated impact of prenatal care (or maternal behaviors) on birth weight, which perhaps suggests that our depression measures are bringing information to the model that is new and relatively uncorrelated with the typical regressors included. For both whites and blacks there is strong evidence that depression lowers birth weight, although the direct and indirect effects of depression appear to be very different between the two groups. For black women, depressive symptoms significantly delay the onset of prenatal care, which in turn has a significantly negative effect on birth weight. The direct effect of depressive symptoms on birth weight, however, although negative is usually statistically insignificant. Therefore, maternal depressive symptoms appear to affect the birth outcomes of black mothers primarily through delaying prenatal care.

The opposite is true for white mothers. ‘Depression’ has no impact on the onset of prenatal care and prenatal care, in turn, has no significant effect on birth weight. However, the direct effect of depressive symptoms on birth weight is consistently negative and often statistically significant. Our results therefore suggest that observed depressive symptoms are associated with lower birth weights for both blacks and whites, but that it operates through delayed receipt of prenatal care for black mothers.

Including maternal smoking and drinking tends to reduce the estimated effects of depressive symptoms, but the results are otherwise similar. The impact on white mothers, in particular, is affected by their inclusion; this suggests that one avenue by which maternal depressive symptoms ‘directly’ affect birth weight is through these behaviors. Black mothers, in contrast, are less affected, which makes sense because these behaviors are not significantly associated with prenatal care.

To avoid the problem of identifying prenatal care and to obtain estimates of the *total* effect of observed depression on birth weight, we next estimate a reduced form birth equation. Perhaps even more important is whether treating maternal depression will improve poor birth outcomes – i.e., reduce the incidence of low birth weight. It is not entirely clear that increasing birth weight will improve infant health in the general population, but there is little doubt that reducing the incidence of low birth weight will. To answer this question, we also estimate a reduced form probit in which a low birth weight dummy (a birth weight of less than 2500 grams) is the dependent variable.[21]

These reduced form equations include all of the identifying variables from the prenatal care equation but excludes prenatal care itself; the results are reported in the second panel of Table 2. Note that income is now included in the birth weight equation. A fair criticism of our structural model is that income is a questionable identifier (even though it passed our tests) and that depressive symptoms may be capturing its effects in the birth weight equation. The reduced form estimates show that the effects of 'depression' are operating through some other mechanism besides reduced income. The same reasoning applies for Medicaid and private insurance participation. However, some might argue that these latter two variables are endogenous. To address this, we also estimate 'policy reduced form' equations in which these two variables are replaced with the Medicaid eligibility thresholds, which vary by household size and state. The results are nearly identical and so are not reported for brevity.

The *total* effects of observed depressive symptoms on birth weight are significantly negative and consistent across samples and measures. A one point increase in the 60-point CES-D scale translates into a 2-5 gram decrease in birth weight, for instance suggesting that a woman with severely elevated symptoms (CES-D=50) would have an infant that is 100-250 grams lighter than one with no symptoms (CES-D=0). Although not a tremendous difference in weight, it roughly corresponds with the observed difference in birth weights between blacks and whites in our sample (Appendix A.1). The discrete effect of being 'depressed' (CES-D > 15) is to decrease birth weight by 29-73 grams. It is also reassuring to find that the total effect of maternal 'depression' obtained from the reduced form model corresponds very well to the sum of the direct and indirect effects obtained from the structural models.[22] Finally and perhaps more important is that both the discrete and continuous measures of 'depression' statistically significantly increase the probability of having a low birth weight birth for both samples.

6. Results with Predicted Depressive Symptoms

As described in section 3, we construct instruments for our observed measure of depressive symptoms taken from the 1988 survey, with the goal of purging our measures of any endogeneity and adjusting for the fact that our observed measure is taken after the birth. The empirical results from these regressions are included in Appendix A.2, both with and without maternal smoking, drinking and drug use and whether the mother wanted the pregnancy as regressors. For the most part, the results are sensible and are fairly stable across the samples, although our equation fits white mothers' depressive symptoms better than black mothers', perhaps in part because the mother's age and education are only important factors for white mothers. In general, being economically disadvantaged, poor maternal behaviors, not wanting the pregnancy and being pregnant now are all associated with higher symptoms. Having a sick baby also increased depressive symptoms, so there is some weak evidence of possible reverse causality. In terms of our aggregate variables, the availability of psychiatrists is associated with lower levels of depressive symptoms for both blacks and whites. While many of the aggregated variables are not individually significant, they are jointly statistically significant at at least the 5% level in all specifications, thereby helping to add meaningful variation to our depressive symptoms instrument.

Using these coefficient estimates, we predict the mother's depressive symptoms during the pregnancy, assigning all observations a 1.0 for the pregnant now dummy variable, and the sample averages for average number of sick visits and length of time since the birth. When available, we substitute the values of the variables *during* the pregnancy for the ones at *the time of the survey*. Table 3 repeats the exercises reported in Table 2 using the two predicted 'depression' measures (plus their dummy variable counterparts). Because the estimated behavior coefficients are similar, we only report the *Onset* and depression coefficients. In addition, for simplicity and to be logically consistent we do not report models that include maternal behaviors in the infant health model but not in the CES-D regressions.

Given the rather poor explanatory power of the depressive symptoms regressions, the main message from this exercise is surprisingly similar. Depressive symptoms decrease the birth weights of both whites and blacks, but for black mothers appear to operate only through delaying prenatal care. The total effects of the reduced form models again correspond well to the sum of the direct and indirect effects of the structural form models. The importance of maternal behaviors is even more pronounced here, which is expected because they are important predictors of depressive symptoms. Either including maternal behaviors in the infant health model or excluding them as predictors of depressive symptoms reduces the impact of depressive symptoms, especially for black mothers. This could be due to the poor fit of the depression regressions for black mothers, especially when the behavior and 'wanted' variables are omitted.

What are we to conclude then? For whites, the evidence is strong; the direct effect of depressive symptoms on birth weight is consistently negative, whether we use an observed measure or an instrument, a continuous or discrete measure, or a structural or reduced form model . Of the 42 depression coefficients estimated in a birth weight equation or low birth weight probit, all suggest a negative impact on birthweight and 29 are statistically significant. The magnitude varies quite a bit across the different measures. When the observed measures are used, the estimated effect is quite modest – approximately 2-3 grams per point on the CES-D scale or a 29-62 gram decrease for being 'depressed'. In contrast, when the instruments are used the effect is much more substantial and ranges from a 10 to 44 gram decrease per point on the CES-D scale or a 90 – 176 gram decrease for being 'depressed'.

For blacks, the evidence is more mixed. Three out of four measures (either continuous or discrete) suggest strongly that depression delays prenatal care and leads to lower birth weights. For the observed measures the magnitudes are again modest – about 4 grams per point on the CES-D scale or 58-73 gram decrease for being 'depressed'. When the instruments are used, the results become volatile, perhaps because our CES-D regressions have a poorer fit for this group.

7. Concluding Remarks

Studies of depression show that its economic costs, in terms of treatment costs and lost earnings due to mortality and morbidity, are quite large (Greenberg et al 1993a, b). More recently, there has been a growing recognition that treating depression may play an important role in helping the low income and unemployed population, especially those on welfare. Our research suggests that addressing the problem of depression within the low income population has benefits that extend well beyond employment into the production of infant health. Furthermore, it also offers some cost effective ways to improve infant health through the Medicaid program -- by augmenting the recent Medicaid expansions with greater attention to screening for and treatment of maternal depression. Our results also have a lesson for health care providers. A recent study by Williams et al (1999, p. 64) find that among family physicians, general internists and OB-GYNs, the OB-GYNs stand out as having higher physician barriers to caring for depression (e.g., having low confidence in or incomplete knowledge of treatment). Our research provides evidence of the potential costs to the infant of failing to detect and treat depression during pregnancy and suggests that greater effort should be directed at improving physician recognition and treatment. Our results provide compelling evidence that maternal depressive symptoms are associated with lower birth weight and highlights some of the ways they may affect birth outcomes. For black mothers, it appears to operate through delaying prenatal care whereas for white mothers it has more of a direct effect. For both groups, there is evidence that it may also operate through increased maternal smoking and drinking.

There remain, however, some unanswered questions. The results for black women are empirically fragile when we instrument maternal 'depression'. This is disappointing because this is the group that suffers more depressive symptoms in our sample and has a higher incidence of low birth weight and very low birth weight births. For both samples, the magnitudes range from quite modest to substantial depending upon the measure used. It is possible that depressive symptoms are acting as a signal for the mother's health endowment and behaviors. We therefore may not have isolated the independent effect on depressive symptoms on birth outcomes;

however, we can conclude that it is a meaningful signal of a potentially troubled pregnancy. This is useful especially given the relative ease of using the CES-D as a diagnostic tool.

And yet there are also reasons to believe that our estimates may be conservative. First, our measures of ‘depression’ are not ideal and may be only weakly correlated with depression during the pregnancy. Second, we have eliminated the poorest birth outcomes by focusing only on live births (in order to remain comparable with other studies of birth weight). Finally and perhaps most importantly we have ignored many possible avenues for depression to have an effect on infant health. For example, maternal smoking has consistently been found to be one of the most important determinants of infant health, and our depression regressions confirm the casual observation that smoking is associated with maternal depression. Indeed, as Saffer and Dave (2002) note, mental illness leads individuals to consume higher amounts of addictive goods and may also affect their price-responsiveness. It is therefore possible that treating depression may be a more effective way of reducing maternal smoking than tax increases or other policies. We explore this idea by re-estimating our model both including and excluding maternal smoking (and drinking) as factors affecting birthweight and as predictors of ‘depression’. But there are other avenues as well. As noted by Lennon, Blome and English 2001 and others, depression can make it more difficult to obtain and retain quality employment. As a result, depression may affect income, insurance status and even family structure, all of which can possibly have both direct and indirect effects on birth weight. Taken together, our research suggests that treating maternal depression is a promising new approach to improving infant health and one that warrants further investigation.

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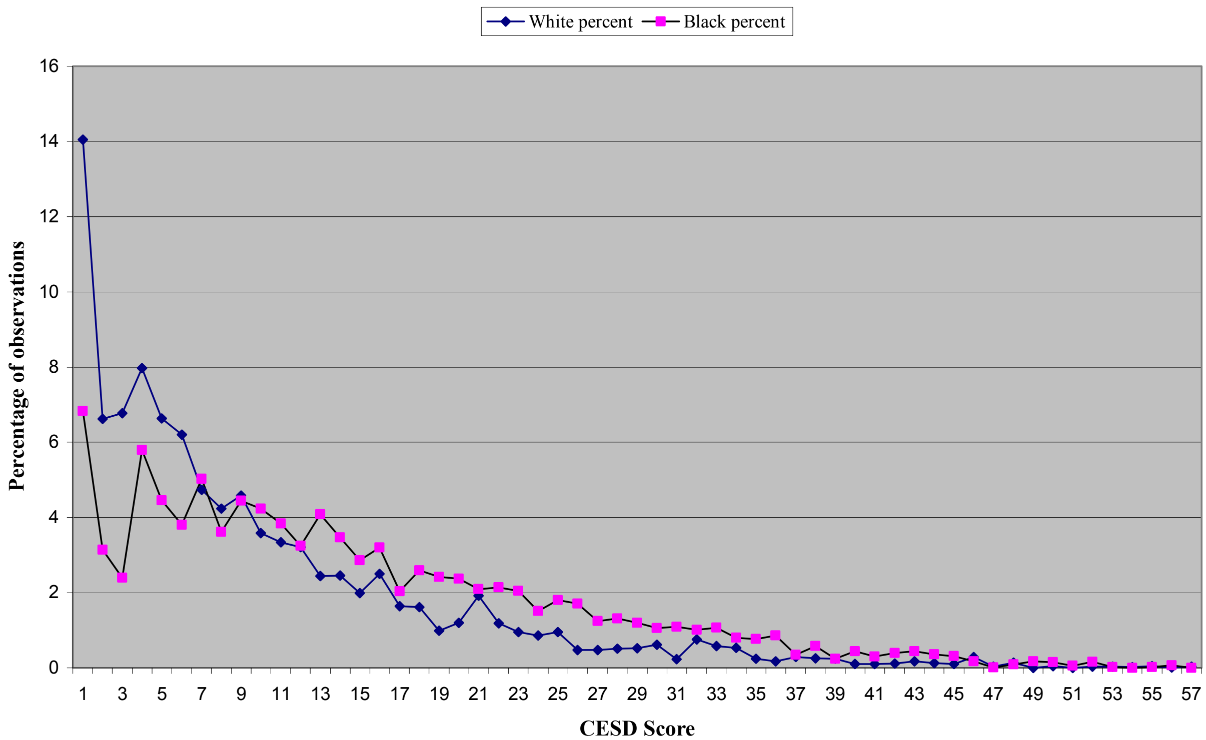
Figure 1. Relative Frequencies of CESD Scores, by Sample



<u>Table 1:</u> Key Characteristics of ‘Depressed’ (CES-D in 1988 > 15) and ‘Nondepressed’ Mothers, by Group and Sample-weighted

	Black		**White**	
	‘Nondepressed’	**‘Depressed’**	**‘Nondepressed’**	**‘Depressed’**
CESD in 1991[a]	9.09	15.4	7.02	13.99
‘Depressed’ in 1991[a]	15.6%	41.6%	8.3%	34.6%
Birthweight (in grams)	3197.96	3096.95	3458.87	3376.80
Percent low birthweight (<2500g)	10.1%	13.3%	4%	6.7%
Percent very low birthweight (<1500g)	2%	2.5%	0.6%	0.9%
Week prenatal care (PNC) began	9.4	10.7	8.4	9.2
Percent with barriers to PNC	9.7%	18.9%	7.8%	24.8%
Income	$19,130.85	$13,687.76	$34,647.74	$24,436.34
Percent Medicaid paid for PNC	39.9%	55%	8.8%	20.4%
Percent private insurance paid for PNC	39.1%	24.5%	74.2%	56.6%
Percent on AFDC during pregnancy	32%	43.9%	6%	12.9%
Percent never married	46%	56.9%	5.7%	13.7%
Cigarettes smoked per day during pregnancy	1.73	2.60	2.85	5.04
Alcoholic drinks per week during pregnancy	.119	.432	.172	.264
N =	**2125 [1681]**	**1217 [924]**	**2580 [2244]**	**652 [522]**

[a]The sample size for this variable is reported in brackets at the bottom of the table.

Table 2: Key Estimated Coefficients when Observed CES-D Measures Used (t-statistic in parentheses)				
	Blacks		**White**	
I. Structural Models	**Observed 1988**	**Average 1988 & 1991**	**Observed 1988**	**Average 1988 & 1991**
A. Baseline, no depression				
1. Birthweight: Onset	-81.69**	-70.55**	-11.76	-1.842
	(-2.95)	(-2.53)	(-.86)	(-.14)
2. Birthweight with behaviors: Onset	-76.86**	-63.79**	3.79	13.68
	(-2.85)	(-2.35)	(.25)	(.94)
Smoke	-8.36**	-7.56**	-9.32**	-9.64**
	(-3.75)	(-3.36)	(-11.10)	(-10.31)
Drink	-5.76	-3.60	-6.29**	-5.99*
	(-.81)	(-.44)	(-2.13)	(1.78)
B. Continuous 'depression'				
1. Onset: CES-D Score	.039**	.023*	-.0033	-.0076
	(4.34)	(1.93)	(-.41)	(-.61)
2. Birthweight: Onset	-82.49**	-64.71**	-8.25	1.375
	(-2.70)	(-2.34)	(-.62)	(.10)
CES-D Score	-.552	-3.212**	-2.890**	-2.849**
	(-.34)	(-2.13)	(-3.15)	(-1.96)
C. Continuous 'depression' with behaviors				
1. Onset: CES-D Score	.038**	.021*	-.003	-.007
	(4.43)	(1.80)	(-.44)	(-.59)
Smoke	-.008	-.021	-.013	-.011
	(-.46)	(-1.18)	(-1.20)	(-.97)
Drink	.044	.085	.092**	.09**
	(.88)	(1.54)	(2.97)	(2.68)
2. Birthweight: Onset	-79.47**	-59.27**	-5.77	15.18
	(2.68)	(-2.22)	(.39)	(1.04)
CES-D Score	-.136	-2.76**	-1.85*	-1.46
	(-.09)	(-1.99)	(-1.90)	(-.95)
Smoke	-8.37**	-7.32**	-9.127**	-9.517**
	(-3.66)	(-3.27)	(-10.61)	(-9.97)
Drink	-5.52	-2.83	-6.28**	-6.105*
	(-.79)	(-.36)	(-2.12)	(-1.78)

*Denotes statistical significance at 90% level. **Denotes statistical significance at 95% level.
All estimated standard errors are robust and adjusted for clustering at the state level.

Table 2 Continued: Key Estimated Coefficients when Observed CES-D Measures Used (t-statistic in parentheses)					
		Blacks		**White**	
I. Structural Models – con't		**Observed 1988**	**Average 1988 & 1991**	**Observed 1988**	**Average 1988 & 1991**
D. 'Depression' dummy					
1. Onset:	CES-D>15	.796**	.296	.171	.061
		(4.04)	(1.04)	(.89)	(.21)
2. Birthweight:	Onset	-82.574**	-66.973**	-10.277	.945
		(-2.72)	(-2.40)	(-.75)	(.07)
	CES-D>15	-5.51	-44.079	-45.189*	-61.583**
		(-.17)	(-1.64)	(-1.92)	(-2.26)
E. 'Depression' dummy with behaviors					
1. Onset	CES-D >15	.777**	.274	.173	.068
		(4.02)	(.99)	(.94)	(.24)
	Smoke	-.007	-.02	-.014	-.012
		(-.40)	(-1.15)	(-1.25)	(-1.02)
	Drink	.051	.09*	.091**	.090**
		(1.02)	(1.66)	(2.93)	(2.67)
2. Birthweight:	Onset	-78.474**	-60.789**	4.65	15.63
		(-2.66)	(-2.24)	(.31)	(1.06)
	CES-D >15	-1.46	-39.34	-30.38	-47.11
		(-.05)	(-1.65)	(-1.19)	(-1.57)
	Smoke	-8.37**	-7.42**	-9.22**	-9.51**
		(-3.72)	(-3.31)	(-10.79)	(-9.52)
	Drink	-5.63	-3.34	-6.29**	-6.13*
		(-.79)	(-.42)	(-2.13)	(-1.79)
II. Reduced Form Models					
A. Continuous 'depression'					
1. Birthweight:	CES-D Score	-3.91**	-4.84**	-2.892**	-2.90**
		(4.40)	(-4.04)	(-3.21)	(-2.01)
2. Low Birthweight Probit Derivative:					
	CES-D Score	.0013**	.0013**	.00082**	.00063**
		(3.56)	(2.87)	(5.53)	(2.38)

*Denotes statistical significance at 90% level. **Denotes statistical significance at 95% level.
All estimated standard errors are robust and adjusted for clustering at the state level.

<u>Table 2 Continued:</u> Key Estimated Coefficients when Observed CES-D Measures Used (t-statistic in parentheses)				
I. Reduced Form Models – con't	**Blacks**		**White**	
	Observed 1988	**Average 1988 & 1991**	**Observed 1988**	**Average 1988 & 1991**
B. <u>Continuous 'depression' with behaviors</u>				
1. Birthweight CES-D Score	-3.336**	-4.187**	-1.906**	-1.62
	(-3.92)	(-3.32)	(-1.99)	(-1.07)
Smoke	-7.72**	-5.99**	-9.18**	-9.65**
	(-4.21)	(-2.75)	(-10.31)	(-10.05)
Drink	-8.24*	-6.84	-5.806**	-4.71
	(-1.74)	(-1.18)	(-2.26)	(-1.50)
2. Low Birthweight:				
CES-D Score	.001**	.001**	.0006**	.0003
	(3.01)	(2.15)	(4.37)	(1.48)
Smoke	.0026**	.002**	.0013**	.0014**
	(4.39)	(2.82)	(6.59)	(6.58)
Drink	.005**	.0049**	.0004	.0004
	(3.14)	(2.18)	(0.49)	(0.48)
<u>C. 'Depression' dummy</u>				
1. Birthweight: CES-D >15	-72.848**	-65.936**	-46.721**	-61.985**
	(-3.60)	(-3.54)	(-1.96)	(-2.23)
2. Low Birthweight				
Probit Derivative: CES-D >15	.0229**	.014*	.0178**	.0055
	(2.10)	(1.70)	(3.56)	(1.05)
<u>D. 'Depression' dummy with behaviors</u>				
1. Birthweight: CES-D >15	-64.46**	-58.39**	-29.483	-46.50
	(-3.44)	(-3.08)	(-1.16)	(-1.56)
Smoke	-7.82**	-6.09**	-9.27**	-9.66**
	(-4.25)	(-2.79)	(-10.45)	(-10.06)
Drink	-8.96*	-7.86	-5.93**	-4.70
	(-1.90)	(-1.35)	(-2.33)	(-1.50)
2.Low Birthweight Probit Derivative:				
CES-D >15	.018*	.001	.014**	.002
	(1.84)	(1.20)	(2.91)	(0.41)
Smoke	.0027**	.002**	.0013**	.0014**
	(4.37)	(2.84)	(6.73)	(6.68)
Drink	.005**	.005**	.0005	.0005
	(3.32)	(2.29)	(0.55)	(0.50)
N =	**3342**	**2605**	**3232**	**2766**

*Denotes statistical significance at 90% level. **Denotes statistical significance at 95% level.
All estimated standard errors are robust and adjusted for clustering at the state level.

Table 3: Key Estimated Coefficients when Predicted CES-D Used (t-statistics in parenthesis)					
I. Structural Models		**Blacks**		**Whites**	
		Predicted with Behaviors	**Predicted without Behaviors**	**Predicted with Behaviors**	**Predicted without Behaviors**
A.Continuous 'depression'					
1. Onset:	CES-D	.249** (4.81)	.072 (.66)	.243** (3.05)	-.395** (2.08)
2.Birthweight:	Onset	-91.143* (-1.81)	-96.967** (-2.20)	29.492 (1.19)	18.55 (1.19)
	CES-D	1.19 (.08)	8.86 (.65)	-36.15** (4.07)	-20.86** (-2.03)
B.Continuous 'depression' with behaviors					
1. Onset:	CES-D	.437** (5.59)		.295** (3.49)	
2.Birthweight:	Onset	-82.82* (-1.85)		9.38 (.41)	
	CES-D	23.64 (1.21)		-7.51 (-.84)	
C. 'Depression' dummy					
1. Onset:	CES-D>15	.422 (1.35)	-.010 (-.23)	1.09** (2.34)	.594 (1.11)
2.Birthweight:	Onset	-82.293** (-2.44)	-82.562** (-2.68)	13.483 (.80)	2.66 (.19)
	CES-D>15	-5.84 (-.13)	4.53 (.12)	-182.17** (-4.64)	-118.91** (-2.22)
D. 'Depression' dummy with behaviors					
1. Onset:	CES-D	.385 (1.33)		.043** (2.42)	
2.Birthweight:	Onset	-86.96** (-2.58)		17.12 (0.97)	
	CES-D>15	42.48 (.97)		-103.98** (-2.68)	
II. Reduced Form Models					
A.Continuous 'depression'					
1. Birthweight:	CES-D	-23.16** (-3.88)	6.782 (.57)	-36.38** (-7.44)	-44.88** (-2.36)
2. Low Birthweight Probit derivative:	CES-D	.010** (5.0)	-.002 (-.67)	.0062** (4.61)	.0109** (3.31)

*Denotes statistical significance at 90% level. ** Denotes statistical significance at 95% level. All estimated standard errors are robust and adjusted for clustering at the state level.

Table 3 Continued: Key Estimated Coefficients when Predicted Depression Used (t-statistics in parenthesis)					
II. Reduced Form Models – con't		**Blacks**		**Whites**	
		Predicted with Behaviors	**Predicted without Behaviors**	**Predicted with Behaviors**	**Predicted without Behaviors**
B. Continuous 'depression' with behaviors					
1. Birthweight:	CES-D	-3.41 (-.47)		-9.61 (-1.46)	
2. Low Birthweight Probit derivative:	CES-D	.0002 (.11)		.0022 (1.40)	
C. 'Depression' dummy					
1. Birthweight:	CES-D>15	-47.42* (-1.67)	9.27 (.31)	-172.64** (-5.53)	-122.95** (-2.44)
2. Low Birthweight Probit derivative:	CES-D>15	.013 (1.07)	-.018 (-1.36)	.030** (3.96)	.012 (.99)
D. 'Depression' dummy with behaviors					
1. Birthweight:	CES-D>15	1.59 (.60)		-89.685** (-2.90)	
2. Low Birthweight Probit derivative:	CES-D>15	-.011 (-.85)		.013* (1.67)	

*Denotes statistical significance at 90% level. ** Denotes statistical significance at 95% level.
All estimated standard errors are robust and adjusted for clustering at the state level.

Appendix A.1. Variable Definitions, Sample-weighted Means and Standard Deviations			
Name	**Variable Definition**	**Blacks**	**White**
1. Variables in Structural Birthweight equation			
Birth weight	Infant's birth weight in grams	3162.09 (619.95)	3443.53 (552.61)
Low birthweight	Dummy variable for birth weight less than 2500 grams	0.1125 (0.316)	.045 (.21)
Onset	Week of the first prenatal care visit	9.886 (5.84)	8.52 (4.46)
CES-D in 1988	CESD measure taken in 1988 survey (ranges from 0-60)	13.62 (10.84)	8.91 (9.38)
CES-D > 15	dummy variable if CESD88 greater than 15	0.355 (0.479)	.187 (.39)
Mother's age	Mother's age at delivery	25.646 (5.05)	27.36 (5.02)
Education	Years of education	12.49 (1.95)	13.28 (2.18)
Income	Total family income in 12 months prior to delivery, in thousands	17.197 (16.896)	32.739 (20.94)
Male	dummy variable if infant is male	.514 (.50)	.523 (.50)
Parity	Dummy variable if any prior pregnancies	.705 (.456)	.671 (.47)
Number of fetal deaths	Number of prior fetal deaths	.408 (.854)	.336 (.71)
# of kids cohabiting	Number of own children living with mother during most of pregnancy	1.21 (1.3)	.90 (1.03)
Dad cohabits	Dummy variable for if baby's father lived with mother during the pregnancy	.515 (.50)	.908 (.29)
Never married	Dummy variable for never married	.499 (.50)	.072 (.26)
Urban	Dummy variable if live in urban county	.795 (.40)	.743 (.44)
Mother's height	Mother's height in inches	64.67 (2.85)	64.88 (2.61)
2. Identifying Variables in Prenatal Care Equation			
ACCRA	ACCRA price index for health care items, state level	99.180 (11.3)	101.76 (12.1)
Population Density	Population density for the state	315.48 (1043.95)	217.41 (362.50)
Medicaid	Dummy variable for if Medicaid paid for prenatal care	.453 (.50)	.110 (.32)
Private insurance	Dummy variable for if private insurance paid for prenatal care	.339 (.473)	.709 (.45)

Appendix A.1 Continued. Variable Definitions, Sample-weighted Means and Standard Deviations			
Name	**Variable Definition**	**Blacks**	**White**
2. Identifying Variables Continued			
OB Medicaid fee ratio	Ratio of maximum Medicaid fees to private fee levels for OB-GYNs	.587 (.18)	.589 (.17)
OB-GYN per 1000	Number of OB-GYNs per 1000 state population	.134 (.04)	.125 (.03)
General Practice per 1000	Number of MD's general practice per 1000 state population	.026 (.01)	.030 (.03)
Hospitals per 1000	Total hospital per 1000 state population	.0029 (.002)	.0033 (.004)
HMOs per 1000	Total HMO's per 1000 state population	.0002 (.0002)	.0003 (.0004)
	Largest sample size used	3342	3232
3. 1988 and 1991 Depression Average			
AvgCESD	Average of the 1988 and 1991 CESD measures	12.388 (8.4)	8.52 (7.2)
Depressed avg.	Dummy variable if average is 16 or greater	.305 (.46)	.158 (.36)
	Larges sample size used	2605	2766
4. Variables Used only in the Depression Regressions			
Receives AFDC now	Dummy variable for receiving AFDC at time of 1988 survey ('now')	.395 (.49)	.075 (.26)
Receives Medicaid now	Dummy variable if baby's health care is paid for by Medicaid 'now'	.424 (.49)	.091 (.29)
Has private insurance now	Dummy variable if baby's health care is paid for my private insurance 'now'	.297 (.46)	.607 (.49)
Smokes now	How many cigarettes smoked per day 'now'	3.08 (6.54)	4.95 (9.22)
Drinks before pregnancy	Number of alcoholic drinks per week before pregnancy	.685 (2.52)	1.15 (2.72)
Drugs before pregnancy	Dummy variable if reported smoking or using cocaine/crack in the three months before pregnant	.057 (.23)	.063 (.24)
Exercise before pregnancy	Dummy variable if exercised/played at least 3 times per week before	.494 (.50)	.464 (.50)
Dad cohabits now	Dummy variable if baby's father is with mother 'now'	.453 (.50)	.876 (.33)
# of kids cohabit now	Number of own children living with mother 'now'	2.10 (1.49)	1.66 (1.21)
Age at 1st marriage	Mother's age at first marriage (only for those who have married)	21.88 (3.9)	21.64 (3.6)
Number of marriages	Number of marriages, =1 if one =2 if two or more	.545 (.58)	1.06 (.45)

<u>Appendix A.1 Continued.</u> Variable Definitions, Sample-weighted Means and Standard Deviations			
Name	**Variable Definition**	**Blacks**	**White**
Wanted pregnancy	Dummy variable if wanted pregnancy at an earlier time and at that time, or wanted pregnancy at that time	.376 (.48)	.648 (.48)
Pregnancy now	Dummy variable if pregnant 'now'	.084 (.28)	.083 (.28)
Length of time since delivery	Number of months between interview and delivery	17.29 (5.05)	16.35 (4.89)
Length* sick visits	Number of months* average number sick baby visits per month	2.46 (3.53)	2.86 (3.55)
Per capita MH expenditures	State mental health agency per capita expenditures	49.80 (33.45)	47.66 (24.78)
Psychiatrists per 1000	Number of psychiatrists per 1000 state population	.0006 (.0008)	.0006 (.0006)
Psychologists per 1000	Number of psychologists per 1000 state population	.015 (.01)	.016 (.01)
Social workers per 1000	Number of social workers per 1000 state population	.093 (.04)	.096 (.03)
MH services organizations per 1000	Number of multiservice mental health organizations per 1000 state population	.005 (.002)	.006 (.002)
	Largest sample size used	2654	2635

Appendix A.2: CES-D Score OLS Regression Coefficient Estimates (t-statistics in parenthesis)

Variable	Blacks		Whites	
Intercept	26.18** (2.54)	23.99** (2.35)	47.94** (6.16)	50.62** (6.70)
Receives AFDC now	.468 (.78)	.642 (1.16)	1.73 (1.15)	1.70 (1.12)
Receives Medicaid now	.645 (1.03)	.771 (1.19)	1.28 (0.94)	1.62 (1.14)
Has private insurance now	-.97** (-1.98)	-1.22** (-2.28)	-.432 (-1.21)	-.484 (-1.29)
Income	-.022 (-1.42)	-.027 (-1.64)	-.031** (-3.17)	-.037** (-3.37)
Smokes now	.084** (2.26)		.039* (1.83)	
Drinks before pregnancy	.429** (4.17)		.131 (1.51)	
Drugs before pregnancy	.99 (1.35)		2.65** (2.67)	
Exercise before pregnancy	.610 (1.42)		-.439 (-1.05)	
Dad cohabits now	-1.55** (-2.38)	-2.04** (-3.47)	-2.00** (-2.37)	-2.79** (-3.19)
# of kids cohabit now	-.178 (-1.59)	-.07 (-0.67)	-.06 (-.36)	.025 (.14)
Years of education	-1.03 (-.90)	-.936 (-.81)	-2.51** (-.3.41)	-2.54** (-3.70)
Education squared	.020 (0.46)	.015 (.34)	.082** (3.21)	.082** (3.46)
Mother's age	.017 (.03)	.184 (.36)	-1.00** (-2.27)	-1.210** (-2.75)
Mother's age squared	-.003 (-.31)	-.005 (-.59)	.016** (2.15)	.019** (2.58)
Age at 1st marriage	.063 (1.21)	.039 (.73)	-.062 (-1.35)	-.054 (-1.05)
Number of marriages	-.503 (-.69)	-.543 (-.69)	.136 (.20)	.452 (.63)
Never married	-.072 (-.04)	-.421 (-.23)	-2.44* (-1.69)	-1.49 (-.97)

Appendix A.2 Continued: CES-D Score OLS Regression Coefficient Estimates (t-statistics in parenthesis)				
Variable	**Blacks**		**Whites**	
Number of prior fetal deaths	-.196 (-.81)	-.164 (-.71)	.134 (.51)	.276 (1.01)
Wanted pregnancy	-2.20** (-5.10)		-1.994** (-5.10)	
Pregnant now	1.782** (2.68)	1.798** (2.66)	1.90** (2.30)	1.72** (2.09)
Length of time since delivery	-.121 (-.26)	-.174 (-.37)	-.130 (-.58)	-.098 (-.43)
Length of time squared	.004 (0.38)	.006 (.50)	.0049 (.73)	.004 (.53)
Length* sick visits	.788** (3.18)	.816** (3.30)	.425** (2.21)	.427** (2.20)
Length squared* sick visits	-.026* (-1.89)	-.027* (-1.86)	-.0069 (-.58)	-.007 (-.58)
Urban	-.552 (-.86)	-.476 (-.76)	.190 (.43)	.40 (.93)
Per capita MH expenditures	.052** (3.02)	.044** (2.60)	.018 (1.57)	.015 (1.26)
Psychiatrists per 1000	-1671.24** (-2.35)	-1573.34** (-2.10)	-643.37* (-1.88)	-507.70 (-1.43)
Psychologists per 1000	8.09 (0.20)	15.53 (0.37)	15.35 (.65)	11.53 (0.44)
Social workers per 1000	-26.23 (-1.41)	-23.53 (-1.20)	-6.34 (-.59)	-7.89 (-.68)
MH services organizations per 1000	-92.70 (-.77)	-64.39 (-0.47)	-143.44* (-1.91)	-120.60 (-1.58)
HMOs per 1000	241.07 (1.08)	258.39 (1.09)	-63.43 (-.36)	-63.64 (-.35)
R-Squared	.1214	.094	.1502	.130
F-Statistic	144.25	56.55	77.03	80.92
N=	2654	2654	2635	2635

*Denotes statistical significance at 90% level.
** Denotes statistical significance at 95% level.
All estimated standard errors are robust and adjusted for clustering at the state level.

Endnotes

[1]See for example Currie and Hyson (1999) who find that negative health (and other) effects of being born at a low birth weight persist well into adulthood, even after controlling for socioeconomic and other background characteristics. The popular press has also discovered this link; the cover story of the 9/27/99 issue of *Newsweek* reports that low birth weight is associated with a greater risk of heart disease and obesity.

[2]Dropping private insurance to participate in Medicaid is referred to as Acrowd out.@ For further discussion and estimates of Acrowd out,@ see Cutler and Gruber (1996) and Dubay and Kenney (1997).

[3]As we discuss shortly, there is a clear distinction between clinically diagnosed depression and measures of depressive symptoms or mood. Our empirical study is investigating the latter, which we refer to as 'depressive symptoms.' In our more general discussions which may apply to either or both, such as in our theoretical framework, we use the term 'depression' for simplicity.

[4]Saffer and Dave (2002) also make this assumption in their theoretical model that looks at the effects of mental illness on the consumption of addictive goods. However, to the extent that women choose whether and how to be treated, especially when pregnant and medications may pose risk to the fetus, one might be able to argue that it is a 'choice'. Another consideration for us is the argument that some types of depression may be the direct result of the infant health production process – postpartum depression or depression that results from having a sick infant. As discussed further in section 3, this may be relevant for us because of the way in which 'depression' is measured.

[5]To our knowledge, Chatterji and Markowitz (2004) is the only other economic study to estimate factors associated with maternal depression, and their focus is on the effects of maternity leave and returning to work on maternal health *after* the birth. They also use the NMIHS.

[6]For example, Grossman and Joyce (1990) use insurance status, the availability of WIC centers and prenatal clinics in the area and the percentage in poverty to identify prenatal care.

[7]However, recent research by Kessler et al 1997 suggests that the demarcation between "major" depression (with 5 or more symptoms) and 'minor' depression (2-4 symptoms) may not be as clear.

[8]To our knowledge, the only other publicly available dataset with all of the necessary information to conduct this study is the PRAMS, which has a state-developed question asking the respondent whether she ever felt ›depressed= during her pregnancy. Not only do these measures require the woman to define ›depressed= (unlike the CESD measure used in the NMIHS, which we discuss shortly), but it is only available for one state, Georgia. Although imperfect, the NMIHS appears to be the best dataset to address this issue.

[9]For a discussion of alternative treatments and their risks during pregnancy see also Lamberg (1999) and Gold (2003). All report few risks associated with most treatments, but caution that more research still needs to be done. For a discussion of the different kinds of depression and alternative treatments, as well as a study that reveals how frequently it goes undiagnosed (and inappropriately treated when diagnosed), see Wells et al (1996).

[10] For instance, Nonacs and Cohen (2003, p. 548) state "the wish to avoid pharmacologic treatment during pregnancy is common," and "…women commonly choose or are counseled to discontinue antidepressant treatment during pregnancy." Another example is Marcus et al (2003) who use the CES-D to detect depressive symptoms in pregnant women and find that only 13.8% of women exceeding the cutoff score for 'depression' reported receiving *any* formal treatment for depression. Of those who reported 'depression' in the last six months, only 24.6% reported currently receiving treatment. Recognizing that 'any treatment' includes many others besides pharmacologic, especially during pregnancy, suggests the rate of such treatment is low. In addition, Kessler et al 1997 find the use of medications to be low among the general depressed population, with only 10-20% reporting that 'they ever took medications more than once for their depression' (Table 5, p. 24).

[11]For example, a pregnant woman may change her exercise routine or limit her drinking while pregnant and then return to her old ways after the birth. Smoking is available at the time of the survey and so we use that variable to predict depressive symptoms. However, treating it like the other behaviors by instead using smoking *before* pregnancy did not substantively affect the results.

[12]In preliminary work we instead used birthweight or the number of sick visits in the past month (each interacted with the length of time quadratic), which produced similar results but had slightly less explanatory power. Note that it does not matter what values we choose for these variables as long as they are uniformly chosen for all observations, as it is the variation in the depression measure across observations that identifies the depression coefficient in the birthweight and prenatal care equations.

[13]The NMIHS actually consists of three different samples – live births, infant deaths and fetal deaths-- and it oversamples fetal and infant deaths. Gray (2001) estimates his model both including and excluding the fetal deaths sample and finds it makes no material difference.

[14]Although this could be another potential source of self-selection, the small number of observations made controlling for it infeasible.

[15]For instance, women who attrite or who do not know how many sick visits their child has had may differ in systematic ways from those who do. The sizes of the black and white samples used to estimate equation 6 are 2654 and 2635, respectively.

[16]Maternal birth weight has been found to be an important predictor of the infant's birth weight (Warner 1998). However, once again a large number of observations have missing data for this variable, especially for the black sample. Estimating the model both with and without maternal birthweight lead to similar results and so we report only those estimates from the larger sample that exclude it.

[17]These percentages are somewhat higher than the usually reported incidence and are constructed using the sample weights. For instance, Wisner et al 1999 reports a *lifetime* risk of 10-25% for women and Lennon, Blome and English 2001 reports a range of 12 to 36% for prevalence of a major depressive disorder (during a 12 month period) among welfare populations. However, the CES-D is a measure of depressive *symptoms* and as such likely has a higher prevalence. In studies that use the CES-D, Lennon, Blome and English 2001 report a range of estimates of 24.9 to 56.7% among welfare populations, which corresponds very well to the estimated prevalence in our black sample.

[18]This variable is based on questions in the NMIHS that asks the woman what barriers

(e.g.,transportation, economic, psychological), if any, she faced in obtaining prenatal care. In preliminary work, we used this variable as another identifier and the results were quite similar. Concerned it might be endogenous, we dropped it from the final model.

[19]Joyce et al (1983), in a case study of women who received no prenatal care, suggest that "internal" barriers such as depression, fear and denial, are more to blame than "external" ones, such as financial, transportation and child care problems. This is also evident in our Table 1 in which 'depressed' mothers sought prenatal care later and reported more barriers to care than nondepressed mothers.

[20]For example, Grossman and Joyce (1990) finds a decrease in birth weight of 12 to 37 grams per **month** delay in prenatal care for blacks and 4 to 23 grams for whites. Liu (1998) reports a range of 6 to 197 grams per month and Warner reports a range of -18 to 50 per **week**. Recent work by Conway and Deb (2003) offers an alternative explanation for the weak effects found for prenatal care, that researchers have failed to adequately account for the different kinds of pregnancies (i.e., 'complicated' versus 'normal').

[21]Recall that the NMIHS sample weights are used to adjust for the oversampling of poor birth outcomes. We also considered estimating the incidence of very low birth weight (< 1500 grams) but the weighted incidence is so low (the highest is 2.1 percent for the black sample) that we concluded such estimation is implausible.

[22]For example, using the average CES-D measure for blacks (column 2) in Model B – a model with statistically significant indirect and direct effects -- yields an indirect effect of 1.49 grams and a direct effect of 3.212 grams per CES-D point, for a total of 4.702 grams. Model C for the same specification yields a total effect of 4.0 points (1.24 + 2.76). The results from the other models, measures and samples yield similar results that

are very much in line with the reduced form total effects. This verification lends additional support to the structural IV model results.