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ABSTRACT

One goal of government health insurance programs is to improve health, yet little is known empirically about how important such government interventions can be in explaining health transitions. We analyze the child mortality effects of a major health insurance expansion in Costa Rica. In contrast to previous work in this area that has used aggregated ecological designs, we exploit census data to estimate individual-level models. Theoretical and empirical econometric results indicate that aggregation can introduce substantial upward biases in the insurance effects. Overall we find a statistically significant but quite small effect of health insurance on child mortality in Costa Rica.

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INTRODUCTION

As the monetary cost of health care decreases through health insurance, the demand for health care increases, potentially improving overall health outcomes. Many countries around the world are promoting health insurance coverage, in part because of these supposed health benefits. Yet, existing literature examining health improvements caused by insurance-induced increases in health care utilization is limited, particularly for developing countries.

One country whose national health insurance plan is frequently cited as a health "success" story is Costa Rica. The country's health indicators are much higher than other countries of a similar income level that do not have national health insurance, with life expectancy equivalent to that of the United States, despite a per capita income of one-fifth that of the United States. During the 1970s period when national health insurance was expanded to the vast majority of the Costa Rican population, health indicators such as child mortality dropped rapidly. Empirical studies that more closely examined the determinants of that mortality decline indicate that expanded access to primary care appeared to play a substantial role (Rosero-Bixby 1986, 1990). Of central importance, however, is what role health insurance itself played, as distinct from the many other changes in health programs and household behaviors that occurred during this period.

Recent work on Costa Rica's child mortality decline by Dow and Schmeer (in press) focused specifically on the role of health insurance and found that health insurance did not have a large causal effect. But as with most other analyses in this literature, that work was limited by its use of aggregated data with an ecological design. The present paper highlights two major implications of this type of aggregation. First, it widens confidence intervals, and as a result Dow and Schmeer (in press) could not rule out moderately sized impacts of insurance on

mortality. Second, and less recognized but potentially more pernicious, aggregation can amplify model mis-specifications such as bias from omitting important confounders.

The present study advances understanding of the mortality effects of insurance by presenting an alternative analysis of the effects of Costa Rica's national health insurance expansion in the 1970s on child mortality. We first conceptually discuss the potential limitations and biases of the common aggregated analysis approach, and explicitly link aggregation to the more precisely defined instrumental variables estimator. We then use Monte Carlo simulation to illustrate the nature of the bias from aggregated studies as compared to individual-level and instrumental variables approaches. Finally, by exploiting unique features of Costa Rica's census data we are able to estimate individual-level models, both to compare them to our aggregated estimates that parallel previous literature, and to produce more robust estimates of the causal effects of health insurance expansions on child mortality.

CONCEPTUAL FRAMEWORK

Based on a health production framework such as Schultz (1984), insurance is hypothesized to reduce child mortality by increasing the demand for medical care. Mortality of child i is a measure of health H_i, and health is a function of medical inputs M_i, non-medical health inputs S_i (such as sanitation and nutrition) chosen by the household, the health environment E_c in community c, and the unobserved health endowment θ_i:

$$(1) \qquad H_i = H(M_i, S_i, E_c, \theta_i)$$

Insurance I_i increases medical input demand, which also depends on the community health infrastructure P_c (price of quality adjusted medical care, including time dimensions

associated with geographic access), as well as socioeconomic characteristics W_i such as household wealth and schooling levels, in addition to health and unobserved preferences π_i:

(2) $M_i = M(I_i, W_i, P_c, H_i, \pi_i)$

Non-medical inputs S_i depend on similar factors as M, although the cross-price effect of insurance may be ignorable:

(3) $S_i = S(W_i, P_c, H_i, \pi_i)$

Finally, insurance may be considered as endogenous to the system, depending on factors such as socioeconomic status, health care access barriers, health status, and unobserved preferences for medical care:

(4) $I_i = I(W_i, P_c, H_i, \pi_i)$

If insurance is in fact endogenous then it does not enter the reduced form health demand function, but instead only enters the quasi-reduced form. In cases where insurance is exogenous, however, the following may instead be interpreted as the reduced form health demand function:

(5) $H_i = H(I_i, W_i, P_c, E_c, \theta_i, \pi_i)$

A complication, however, is that many of the relationships in (5) work in different directions, making it often impossible to sign net omitted variables biases in observational data with imperfect controls. For example, adverse selection would imply that less healthy individuals would have a higher demand for insurance, causing health to be negatively related to insurance, but positive selection of (healthier) higher socioeconomic status persons into positions with health insurance could instead cause a positive relationship. Thus a central implication of this model is that it is crucial for empirical research designs to take into account this potential endogeneity of insurance. We next assess previous literature in light of this endogeneity issue.

PREVIOUS RESEARCH ON HEALTH EFFECTS OF INSURANCE

A wealth of research indicates that health insurance increases health care utilization. A recent Institute of Medicine (2002) report concludes that lack of health insurance is a major barrier to health care access in the United States. The gold standard of studies on the demand effects of insurance generosity is the RAND Health Insurance Experiment conducted in the United States in the 1970s, which based on a randomized design found that demand did respond significantly to the out-of-pocket cost of care, with an elasticity of about –0.2 (Manning et al. 1987; Keeler and Rolph 1988). There are fewer studies in developing countries with strong causal designs, but literature on health care price elasticities of demand has generally found significant elasticities (Jimenez 1995).

More controversial is the extent to which health outcomes are affected by the type of health care induced by insurance-driven decreases in out-of-pocket costs. A long literature has debated the relative importance of health behaviors, public health interventions, and medical care in driving health transitions. In relation specifically to insurance, the importance of medical care is further questioned due to the fact that it is hypothesized that insurance would predominantly increase utilization of medical services that have low perceived benefits, while high benefit medical services would be more likely to be demanded regardless of insurance status. An additional literature has debated whether insurance instead affects health by creating conditions that speed technological change, but this is more relevant in fee-for-service systems at the technological frontier such as the United States than in most low-income countries such as Costa Rica.

The empirical literature on the health effects of health insurance has been plagued by poor methodological designs. In a recent survey of dozens of studies relating insurance to health in the United States, Levy and Meltzer (2001) found only a handful with designs adequate for inferring causality. Based on those they conclude that while insurance is not irrelevant to health, in general the health benefits of insurance appear quite small. Again the gold standard study is from the RAND Health Insurance Experiment, which found that the extra insurance-induced medical care on average had no effect on a wide range of health indicators, although there did appear to be some significant effects for certain vulnerable low-income groups (Brook et al. 1984). The RAND Health Insurance Experiment, however, was only designed to analyze variations in copayment levels within a relatively low catastrophic cap, thus it is not clear how generalizable the results are when considering the population that is completely uninsured. Other reliable studies identified by Levy and Meltzer (2001) included natural experiment analyses of Medicaid expansions indicating that insurance may be relevant for low-income groups (Currie and Gruber 1996a, 1996b), and a study indicating that national health insurance lowered infant mortality in Canada (Hanratty 1996).

Within low-income countries there have been very few studies with reliable designs. Furthermore, the literature has relied mainly on aggregated estimators. For example, Dow and Schmeer (in press) use a difference-in-difference type design to estimate the effects of Costa Rica's 1970s insurance expansion, but because they use vital statistics data to measure mortality they are forced to aggregate to the county as the unit of observation. In contrast, the present study analyzes the same insurance expansion but draws mortality data from census mortality questions, allowing direct comparison of individual and aggregated estimators. To better

interpret this comparison, we next discuss the properties of the common aggregated approach to analyzing the insurance effects of mortality.

AGGREGATION

There is a good deal of confusion among researchers regarding the effects of aggregation on estimators. Robinson's (1950) article on ecological correlations is still cited reasonably frequently to argue that aggregated analyses produce biased results, despite the fact that as early as Goodman (1953) it was shown that Robinson's particular critique did not itself generalize to the regression context. Ever since, however, there have been on-going debates over the merits and limitations of aggregated regression estimators (see e.g. Guthrie and Sheppard 2001 for a recent debate).

To clarify the issues in the context of the insurance-mortality literature, we next present several precisely specified special cases. The first case illustrates conditions under which individual and aggregated estimators produce identical effects. The second illustrates the effect of omitted group-level confounders, in which case aggregation exacerbates the omitted variables bias. The third assumes an omitted individual-level variable, in which case aggregation may ameliorate the resulting bias. Following Moffitt (1995), we next relate the aggregated model to the instrumental variables model, which provides a well-understood framework for comparing the estimators. Monte Carlo analysis is then used to further illustrate each case.

Case I: No aggregation bias

We begin by specifying an individual-level regression model of health H_{ic} for individual i in community c as a function of the individual's insurance I_{ic}, the individual's health endowment

θ_i, community-level health infrastructure P_c, and *iid* (independently and identically distributed) error components μ_c and ε_{ic}:

$$(6) \qquad H_{ic} = \alpha_{ind} + \beta_{ind} I_{ic} + \gamma_{ind} \theta_{ic} + \delta_{ind} P_c + \mu_c + \varepsilon_{ic}$$

The insurance coefficient of interest β_{ind} is interpreted as the effect on an individual's health from changing their own insurance status. Next consider an aggregated version of this model with the community as the unit of observation, and each individual-level variable replaced by its community mean:

$$(7) \qquad \overline{H}_c = \alpha_{agg} + \beta_{agg} \bar{I}_c + \gamma_{agg} \overline{\theta}_c + \delta_{agg} P_c + \mu_c + \overline{\varepsilon}_c$$

The insurance coefficient of interest β_{agg} in this model is interpreted as the aggregate effect on community health from increasing the community health insurance rate.

There are several points to note in comparing (6) and (7). First, if (6) is indeed correctly specified (appropriate functional forms, no measurement error, error components independent of explanatory variables, etc.), then $\text{plim}(\hat{\beta}_{ind}) = \text{plim}(\hat{\beta}_{agg})$: the individual-level and the aggregate community-level insurance effects are equal. In this case it is possible to make unbiased cross-level inferences, such as using the aggregated model (7) to estimate the individual-level insurance relationship (6).

Second, the assumption that the community error component in (6) is independent of individual-level insurance implies that the community insurance rate $\bar{I}_c$ is appropriately omitted from the individual-level model (6). In the sociology literature this condition has been referred to as the absence of cross-level effects (Firebaugh 1978), while in the economics literature this is equivalent to assuming no general equilibrium or spillover effects. In our application, this could be interpreted as assuming that the health care supply increases proportionately with the community insurance rate; thus as insurance expands, previously insured individuals neither

suffer from additional facility crowding, nor do they benefit from insurance-induced technological improvements.

Third, if the community insurance rate did actually affect individual-level health independently of the individual's insurance status, such that $\xi_{ind}\bar{I}_c$ was an omitted term from (6), then β_{agg} would no longer equal β_{ind}, but would instead reflect the net combined effects of β_{ind} and ξ_{ind}. In this case cross-level inferences would be inappropriate. It would be a misnomer to refer to this generally as "aggregation bias," however, since for certain policy purposes of evaluating community insurance expansions, the net community effect β_{agg} from the aggregated regression (7) may in fact be the quantity of interest.

Finally, an important drawback of the aggregated model (7) is the fact that the aggregation process leads to loss of variation in *I*, as well as increasing multicollinearity, which both result in increased standard errors on the insurance coefficient.

Case II: Aggregation exacerbates omitted variables bias

In contrast to the above model that was assumed perfectly specified, in other cases aggregation can indeed have pernicious effects on regression coefficients. An important such case that has recently been highlighted by Hanushek, Rivkin, and Taylor (1996) occurs when community-level confounders are omitted from the model. Continuing the example of Case I, assume that (6) and (7) are now modified such that the community-level health infrastructure variable P_c is omitted from the estimation. Denote the insurance coefficients from these models with omitted variables as β_{ind}^{OVcomm} and β_{agg}^{OVcomm}. Further assume that P_c indeed belongs in (6) such that $\delta_{ind} \neq 0$, that it is correlated with the insurance variable of interest, $\text{cov}(I_{ic}, P_c) \neq 0$,

but that P_c is uncorrelated with other explanatory variables. The bias from omitting P_c can then be characterized in the individual-level equation as:

$$(8) \qquad \begin{aligned} \beta_{ind}^{OVcomm} - \beta_{ind} &= \delta_{ind} \operatorname{cov}(I_{ic}, P_c) / \operatorname{var}(I_{ic}) \\ &= \delta_{ind} \operatorname{cov}(\bar{I}_c, P_c) / \operatorname{var}(I_{ic}) \end{aligned}$$

and in the aggregated equation as:

$$(9) \qquad \beta_{agg}^{OVcomm} - \beta_{agg} = \delta_{agg} \operatorname{cov}(\bar{I}_c, P_c) / \operatorname{var}(\bar{I}_c) .$$

The key difference between (8) and (9) is that the variance of the insurance variable is smaller in the aggregated equation, implying that the omitted variables bias will be larger in the aggregated model than in the individual-level model. Thus with an unobserved community-level confounder, aggregation exacerbates omitted variables bias.

Case III: Aggregation ameliorates omitted variables bias

It is not always true though that aggregation exacerbates omitted variables bias. Consider instead an alternative extreme example in which the only mis-specification is an omitted *individual*-level variable, such as an individual's genetic health endowment θ_{ic}. Assume that $\operatorname{cov}(I_{ic}, \theta_{ic}) \neq 0$, such that the version of the individual level model that omits θ_{ic} yields a biased estimate of the insurance effect $\beta_{ind}^{OV\theta}$, due to adverse or positive selection.

In addition, for expositional purposes assume that although θ_{ic} varies across individuals within a community, its community mean value $\bar{\theta}_c$ does not vary across communities. This is plausible for a variable such as the genetic health endowment. The result of this assumption is that there is essentially no variation in $\bar{\theta}_c$ left to be correlated with $\bar{I}_c$, implying that the aggregated estimation with this particular omitted individual-level variable still yields unbiased

estimates of the insurance effect: $\beta_{agg}^{OV\theta} = \beta_{agg} = \beta_{ind}$. Although this is an extreme example, and the result will not hold for other individual-level variables that do vary at the community level, it illustrates the important point that when omitted variables vary more at the individual-level than at the community-level, aggregation can ameliorate omitted variables bias.

Aggregation and Instrumental Variables

This third case also helps illustrate the close relationship between aggregated estimators and instrumental variables estimators. Consider an individual-level instrumental variables model in which insurance I_{ic} is treated as an endogenous variable due to correlation with the error term arising from omitted individual-level variable θ_{ic}. One potential instrument set would be a vector of community dummies $\widetilde{D}_c$, resulting in a first stage equation:

$$(10) \qquad I_{ic} = \alpha_{10} + \widetilde{\upsilon}_{c,10}\widetilde{D}_c + \delta_{10}P_c + \varepsilon_{ic,10}$$

and a second stage structural equation:

$$(11) \qquad H_{ic} = \alpha_{IV} + \beta_{IV}\hat{I}_{ic} + \delta_{IV}P_c + \mu_{c,IV} + \varepsilon_{ic,IV}$$

The central point to note about (10) is that the predicted value $\hat{I}_{ic}$ is essentially equal to the community insurance rate $\bar{I}_c$ (with an innocuous adjustment for P_c). The implication of this is that this instrumental variables estimator will be unbiased under very similar conditions as was necessary for the aggregated estimator to be unbiased in Case III: First, there must be community level variation in the insurance rate. Second, in order for the community dummies to be valid instruments, there must be no omitted community-level variables in the error term of the structural equation (11); this estimator will only help correct for certain omitted individual-level variables. One requirement of this latter condition is that in Case III, there is no community-

level variation in $\bar{\theta}_c$ (the aggregated individual variables) that affects health. Another important requirement of the latter condition is that the community mean insurance rate $\bar{I}_c$ must have no independent effect on individual health in (6) after controlling for the individual-level insurance: $\xi_{ind} = 0$. In other words, this instrumental variables estimator will only be valid if there are no general equilibrium (cross-level) effects of insurance.

One reason why it is useful to compare the aggregated estimator to the instrumental variables estimator is that it enables the use of well understood specification tests for choosing between alternative models. In particular, the Hausman endogeneity test can be used for judging whether estimates from instrumental variables models using grouped variation (similar to the aggregated models) are significantly different from OLS (individual-level) estimates.

One limitation to this testing approach is that if the Hausman endogeneity test rejects the null hypothesis that the estimates are equal, it may not be possible to distinguish between alternative causes of the rejection: (a) Rejection could be due to the presence of general equilibrium or cross-level effects. In this case the aggregated estimates could only be interpreted as reflecting the net aggregate community-level health effects of insurance expansion; furthermore, the individual-level models should be re-specified to include community insurance rates as an additional covariate. (b) Rejection could indicate omitted community-level variables, in which case both individual and aggregated estimators would be biased, but the bias would be exacerbated in the latter. (c) Rejection could indicate omitted individual-level variables, in which case both estimators are again likely to be biased, but the relative sizes of the bias is more ambiguous. If the bias is caused by variables that have a stronger correlation with insurance at the individual-level than at the community level, such as in Case III, then the aggregated estimator will have the smaller bias, but this will generally be difficult to discern.

The Hausman endogeneity test result is perhaps more useful in this context when it does not reject (assuming reasonable confidence intervals). In this case there is some small chance that individual and aggregated estimators are truly different but they are biased in such a way that they appear to coincide. However, this is a lower probability event. Instead it may be more reasonable to infer from non-rejection that there are no significant biases and that there are no general equilibrium or cross-level effects, and thus that aggregated estimators could indeed be used to make either aggregate or individual-level inferences.

Monte Carlo Illustration of Aggregation

Monte Carlo simulation is a useful tool for illustrating the previous points comparing individual-level, aggregated, and instrumental variables estimators for each of the three cases discussed above. To conduct this simulation we begin by assuming a true model reflected by equation (6), and then design the data generating process to reflect the characteristics of the actual Costa Rican data analyzed further below. In each of 100 communities we draw variables I_c and P_c from a multivariate standard normal distribution with correlation 0.5, and an *iid* standard normal community error component μ_c. We next create 100 individual observations in each community, and then draw variables I_i and θ_{ic} from a multivariate standard normal distribution with correlation 0.5, and an *iid* normal individual error component ε_{ic} with standard deviation 10. To complete the model, we generate the individual-level insurance variable $I_{ic} = I_i + I_c$, and the dependent variable $H_{ic} = I_{ic} + \theta_{ic} + P_c + \mu_c + \varepsilon_{ic}$. Thus individual-level insurance I_{ic} is correlated with both the health endowment θ_{ic} and the community

infrastructure P_c, and the true coefficients on these variables all equal one:

$$\beta_{ind} = \beta_{agg} = 1, \gamma_{ind} = \gamma_{agg} = 1, \delta_{ind} = \delta_{agg} = 1.$$

Estimates of the true models are shown in Table 1 columns [10]-[12], averaged over 500 replications. The individual-level model (6) reported in column [10] was estimated by OLS; the standard errors in all of the OLS models were adjusted for the community-level error component using Huber-type ex-post clustered corrections. The aggregated model (7) in column [11] was estimated for 100 communities after collapsing each variable to its community mean; note that the standard error of the coefficient on $\overline{\theta}_c$ is quite large, reflecting the fact that this variable was designed to have no variation when aggregated to the community-level. The instrumental variables estimator of the true model (equation 11 modified to also include θ_{ic} in both stages) in column [12] uses the vector of 100 community dummy variables as instrument in the first stage; standard errors are again corrected for community-level clustering.

The true models [10]-[12] illustrate two central results corresponding to Case I: First, when there is no mis-specification (and no general equilibrium or cross-level effects), all three estimators yield exactly the same point estimates. Aggregated estimators are unbiased in this case, and can be used for inferring either the aggregate health effects of an insurance expansion, or the equivalent individual-level effect of insurance on health. Second, the standard errors of the insurance coefficient are substantially higher in models that discard individual-level variation in insurance (either by aggregating the unit of observation to the community-level, or by using only community-level instruments).

Next, consider the results of Case II in columns [4]-[6], in which the community level health infrastructure variable P_c is omitted from the models. The key result is that while the individual-level estimator is biased up 29%, this bias is 50% in the aggregated estimator. In this

case, aggregation exacerbates the omitted variables bias. Furthermore, the instrumental variables estimator is just as biased as the aggregated estimator; this is due to the fact that the omitted community-level variable is in the error term of the structural equation, hence the community dummies are not appropriate instruments since they are not validly excluded from the structural equation.

Finally, consider Case III in columns [7]-[9], in which the individual-level health endowment variable θ_{ic} is omitted from the models. In this case the individual-level OLS insurance coefficient is again biased by 30%, but the aggregated estimator remains unbiased. This is because θ_{ic} was constructed to illustrate the special case of a variable that is correlated with insurance at the individual-level, but not at the community-level. Another way of viewing this example is that individual insurance choices are endogenous within communities, but that variation in insurance rates across communities is caused solely by exogenous factors, and in this case community-level dummies can serve as appropriate instruments to correct for this endogeneity.

The above has necessarily focused on stylized examples to illustrate some important but frequently poorly understood properties of aggregated estimators. One of the implications of this discussion is that a priori it is difficult to hypothesize whether or not aggregated models will perform well in any given application, but that Hausman tests provide a useful tool for assessing the relative performance of the estimators. Next, we apply the above insights to examining the mortality effect of insurance in the particular context of Costa Rica's 1970's insurance expansion.

COSTA RICAN SETTING

Infant mortality rates fell dramatically in Costa Rica over the course of the 1960s and 1970s, dropping from approximately 70 per 1,000 in 1960 to 20 by 1980. Much of this decline was in deaths from diarrhea, pneumonia, and vaccine-preventable diseases. Caldwell (1986) has argued that important factors causing this decline include high levels of female education, a strong primary care focus in the health care system, and the role of national health insurance in eliminating financial barriers to health care access. Additional factors hypothesized by other observers include water supply and sanitation interventions, advanced social development, sustained economic growth, and political stability (Mohs 1985; PAHO 1998). These factors were likely reinforced by the sharp fertility decline during the 1960s, which itself may have been caused by many of these same factors.

The factor that has received the most attention out of the above list has been Costa Rica's health care policies. For the purposes of the present paper we distinguish the national health insurance expansion during the 1970s from the other potentially important primary and secondary care interventions. While previous empirical work has documented that the policies as a whole appear to have played a substantial role in reducing infant and child mortality (Rosero-Bixby 1986), the only paper to look specifically at insurance has found its role to be modest at best (Dow and Schmeer, in press).

Mesa-Lago (1985) documents the evolution of national health insurance in Costa Rica. Prior to the 1970s health insurance was primarily held by civil servants and professionals, with very low coverage rates among agricultural workers and other laborers, despite a 1961 constitutional amendment to establish universal coverage. A key turning point was the development of the first national health plan in 1971, which reinvigorated the goal of universal

public health insurance. With political support from the government, insurance rates began to climb rapidly starting about 1973, leveling off about 1980 when the third world debt crisis began. Based on census data, health insurance coverage among children increased from 42% in 1973 to 73% by 1984. Those remaining uninsured were generally in marginalized households with only unemployed or informal sector workers; in principle the most indigent households were eligible for user fee and premium exemptions, but in practice many households are not covered by these safety net provisions. The 30% of the population who obtained insurance as a result of the 1970s expansion were generally in middle to lower socioeconomic status households, who could reasonably be expected to benefit from improved financial access to care. This change in financial access is likely to have been substantial, given that insurance granted universal access to hospitals and widespread health facilities at zero prices with no deductibles, as opposed to the uninsured who faced large out-of-pocket prices and user fees even in government-run facilities.

Concurrent with the insurance expansion were a number of other health sector changes. First, primary health care programs targeted at the uninsured were initiated in selected rural areas in 1973, and expanded to certain urban neighborhoods in 1976. Second, virtually all hospitals were nationalized in the mid-1970s and placed under the control of the government agency that administers universal insurance, the Caja Costarricense de Seguro Social. Third, the government invested in a considerable expansion of primary health care personnel and facilities. Consideration of these other health sector changes is important in our empirical analysis in part because they may be correlated at the community level with insurance rate changes, and hence should be controlled for in the regression analyses. In addition, these changes may affect the likelihood of general equilibrium or cross-level effects of insurance. For example, if the supply

side of the health care market does not expand sufficiently to meet the new demand induced by an insurance expansion, then facility crowding could result in spillovers that cause individual-level insurance effect estimates to be larger than the net community-level effects from aggregated analyses. The fact that the government did expand health care supply along with insurance lessens the likelihood of such general equilibrium effects. We next discuss in more detail our econometric methods.

METHODS

We analyze the effects of Costa Rica's insurance expansion using variants of all three of the econometric estimators discussed earlier: individual-level OLS, aggregated OLS, and individual-level instrumental variables with community dummy instruments. Equation (6) showed a stylized version of our estimating equation derived from the reduced form health demand function (5). When moving from simulations to actual data, however, we must address several omitted variables issues arising from unobservables.

First, although we can explicitly control for individuals' socioeconomic characteristics and wealth W_{ic}, and certain non-medical inputs such as water supply and sanitation S_{ic}, our data do not allow us to observe other individual characteristics such as health endowments θ_{ic}. If these unobserved health variables are in the error term then adverse or positive selection could cause insurance to be endogenous, leading to biased estimates. To attempt to test for this we will use the instrumental variables estimator; if the Hausman test fails to reject the null hypothesis of exogeneity, then we will conclude that in our application insurance is not in fact endogenous.

Second, our instrumental variables strategy allows us to test for the exogeneity of other potentially endogenous variables as well, such as wealth, and water supply and sanitation.

Although non-medical health inputs such as sanitation may not belong in the reduced form health demand equation per se, given our quasi-experimental design it is useful to include such variables in the model simply as controls for unobserved heterogeneity that may be correlated with insurance preferences. Such controls would not properly serve their purpose if they were themselves endogenous, however, hence the importance of Hausman tests for their exogeneity.

Third, we are likely to only partially observe important community-level variables such as the community health environment E_c. As discussed above, this would cause exacerbated omitted variables bias in the aggregated and instrumental variables models due to correlation between insurance and the community error component μ_c. A fix to this could be to estimate community-fixed effects versions of the model; however, with cross-sectional data this would only be possible for the individual-level OLS model. Thus our solution to this problem is to construct a synthetic panel dataset, based on repeated cross-sections of the Costa Rican national census just before (1973) and after (1984) the main insurance expansion, which then allows all three estimators to control for unobserved community-level fixed effects.

These modifications result in the following estimation version of the individual-level equation (6), where *t* indexes year Y (1973 or 1984):

$$(12) \qquad H_{ict} = \alpha_{12} + \beta_{12} I_{ict} + \lambda_{12} W_{ict} + \delta_{12} P_{ct} + \eta_{12} Y_t + \tilde{\upsilon}_{c,12} \tilde{D}_c + \varepsilon_{ict,12}$$

Our estimating equation for the aggregated model is the same as (12), but with individual-level variables being replaced by the year-specific community mean:

$$(13) \qquad \overline{H}_{ct} = \alpha_{13} + \beta_{13} \bar{I}_{ct} + \lambda_{13} \overline{W}_{ct} + \delta_{13} P_{ct} + \eta_{13} Y_t + \tilde{\upsilon}_{c,13} \tilde{D}_c + \varepsilon_{ct,13}$$

It is worth noting that this aggregated estimator (13) is analogous to the state-year fixed effects "difference-in-difference" models used by a number of United States studies which Levy and Meltzer (2001) have referred to as producing some of the more valid estimates of insurance

effects on health. This is also the model used by Dow and Schmeer (in press) in the only other work analyzing the effects of the Costa Rican insurance expansion.

For the instrumental variables model, with communities observed in multiple years the instrument set is expanded to consist of the year-specific community dummies. Thus after including community fixed effects in the main equation (12), the model is identified by the community × year interaction terms. This produces instrumental variables estimates that again correspond quite closely to the difference-in-differences aggregated model (13). The first stage equation is:

$$(14) \qquad I_{ict} = \alpha_{14} + \lambda_{14} W_{ict} + \delta_{14} P_{ct} + \eta_{14} Y_t + \tilde{\upsilon}_{c,14} \tilde{D}_c + \tilde{\varsigma}_{c,14} Y_t \times \tilde{D}_c + \varepsilon_{ict,14}$$

and the second stage structural equation is the same as (12) after substituting the predicted value $\hat{I}_{ict}$ from (14) for I_{ict}.

An additional econometric detail is that the aggregated estimator is implemented via OLS, weighted by the community population size. Finally, Huber-type community-clustered robust standard errors are reported in the tables.

DATA

Data linking individual-level insurance and mortality outcomes are rare, which is one reason why many studies resort to aggregated analyses. In this study we are able to analyze 100% microsamples of the 1973 and 1984 Costa Rican censuses, exploiting their unique feature of asking each woman not only about the survivorship of her children, but also her health insurance status. Although ideally we would prefer to observe the mortality experience of each child, we only actually observe child mortality based on the mothers' reports of total children ever born and surviving, thus we follow previous literature such as Trussell and Preston (1982)

and estimate the determinants of infant and child mortality using mothers as the units of observation.

The main insurance expansion occurred between 1973 and 1980. Thus although we do not have historical insurance information on each woman, in general insurance rates for women were stable over the several years prior to each of these censuses. To focus on the mortality only of recently born children, for whom current insurance measured in the census is plausibly an indicator of insurance at the time of birth, we restrict our analysis sample to women ages 18 to 25. In addition, because of high non-response to the "children ever born" question among single women, we restrict the sample to only ever married women. Finally, the sample is further restricted to only those women who reported at least one live birth, resulting in an analysis sample of 134,036 women (53,196 from the 1973 census and 80,840 from the 1984 census).

Dependent Variable

Our child mortality dependent variable H is constructed from two standard census questions regarding the number of live births a woman has ever had (B), and how many of those children are still alive (L): $H = (B - L)/B$. This dependent variable raises a number of analysis issues. First, in order to make results generalizable to the population of children, rather than the population of mothers, regressions are modified to incorporate weights reflecting each woman's number of children ever born.

Second, there is likely to be some degree of under-reporting of births and deaths, particularly for live-born children who died in the immediate postpartum period. A study of national child national estimates from these census data found that the 1973 census results in unbiased estimates, but the 1984 census slightly overestimates child mortality (Behm and

Robles-Soto 1990). Furthermore, it is possible that such under-reporting could differ for home-births and hospital births, which could introduce bias given that the insurance expansion is associated with a decrease in home births. Unfortunately the patterns of such under-reporting are not well understood either in general or in our specific data, thus we simply raise this as a potential caveat (a caveat that also applies to alternative data sources including vital statistics that imperfectly observe in-home mortality).

Third, mortality will increase with age because of the greater average number of child years of risk exposure. Trussell and Preston (1982) have developed methods to adjust the mortality dependent variable for exposure time; their methods are particularly useful for making inferences about formal mortality rates in samples with wide age ranges. For our purposes of regression estimation among a narrow age group, however, we prefer the less parametric alternative method of simply correcting for exposure by including women's age as a right-hand side explanatory variable.

Fourth, it is not self-evident to which time period and child's age this mortality ratio refers. However, early studies by William Brass and other demographers suggest that child mortality ratios of mothers aged 18 to 24 stand approximately for the probability of dying in the first two years of life in a time point two years before the date of the survey or census (United Nations 1983).

Explanatory Variables

The main explanatory variable of interest is insurance status. Crucially, both the 1973 and 1984 Costa Rican censuses collect data on social health insurance status for each household member. Because children generally have the same insurance status as their mother, the

mother's insurance status is a good indicator of both whether a woman had access to insured health care during pregnancy and childbirth, and whether the child had access to insured health care after birth.

The census also provides a rich array of individual and household-level control variables corresponding to the constructs in the reduced form health demand equation (5). The specific variables included in the analysis are further defined in Table 2. The mothers' demographic and socioeconomic characteristics W_i controlled for in the analysis include age, educational attainment, migration status, and wealth. The final set of household-level variables, included in the model as controls for unobserved heterogeneity correlated with insurance, are three water supply and sanitation measures.

Most of these variables are self-explanatory, with the exception of wealth. Wealth is represented in the estimation as the first two principal components from a vector of eleven housing quality and asset indicators. The first wealth principal component can be interpreted as a wealth measure; the factor loadings on the second component make it more difficult to interpret, but we include it in the model as an additional control variable. These indicators are reduced to their principal components because of the degrees of freedom and multicollinearity challenges arising in the aggregated models; the individual-level models, however, are not substantively changed when instead including the eleven underlying variables directly.

The last set of variables in the model are those that measure community-level health infrastructure. These are not directly available from the censuses in Costa Rica, hence they have been constructed from a variety of different survey and administrative data sources. These include travel time to the capital city San Jose, the community proportion of individuals covered by the post-1973 primary health care programs targeted at the uninsured, the community

proportion of the population whose nearest health clinic was inaugurated between 1973 and 1984, and finally the proportion of deaths not medically certified is used as another proxy variable for community access to care.

An additional key variable required for the analysis is the definition of communities. Extensive previous effort has been applied to this problem by Rosero-Bixby (1991), resulting in the country being broken into a set of 99 mutually exclusive "counties" that are both geographically and politically meaningful. This county grouping is applied in the present paper as the definition of the community, and the community-level health infrastructure control variables have all been constructed at this county level.

RESULTS

Summary statistics for the insured and uninsured women in each of our two data years are presented in Table 3. Insurance coverage rates among our sample women increased dramatically between 1973 and 1984, from 46% to 74%. During the same period our child mortality ratio fell even more markedly, from 53 to 18 deaths per 1000 live births. The association between the insurance expansion and the national mortality drop has been noted by many observers (e.g., Mesa-Lago 1985), but of course many other health determinants improved over this time period as well. For example, the proportion of women ages 18-25 with any secondary education doubled from 20% to 41%, the proportion of women living in households without internal bathrooms dropped in half from 42% to 19%, and the proportion of women living in households with a refrigerator doubled from 19% to 41% (Table 3).

While the above results refer to the aggregate national level relationship, we see that at the county level the relationship between the size of the insurance change and the size of the

mortality change over time is also large, before controlling for potential confounders. This can be seen from the OLS aggregated regression in Table 4 column [2] that includes year and county fixed effects but no other control variables (this regression is equivalent to a "difference-in-differences" design). The coefficient from this regression indicates that the 29 percentage point insurance expansion explains (before adding controls) 40% of the observed child mortality decline between the 1973 and 1984 censuses ($-.0485 \times .29/(.053 - .018) = .4$).

This county-level aggregated effect is almost four times higher, however, than the unconditional *individual*-level relationship shown in Table 4 column [1]. The standard error of the insurance variable in the aggregated model is also much higher than in the individual model, by a factor of 10. In order to further compare the different results between these models, Table 4 column [3] reports the individual-level instrumental variables regression that uses the 98 county$\times$year interaction dummies for identification. The test of the joint explanatory power of these instruments in the first stage is highly significant (as is the joint significance test for the base county fixed effects dummies), implying that the instruments are strong enough to avoid weak-instrument bias. As was illustrated in the Monte Carlo results from Table 1, when using our actual data the estimated insurance coefficient from the instrumental variables model turns out to be quite similar to the estimate from the aggregated model, in both size and precision. Furthermore, the Hausman test rejects the null hypothesis that the instrumental variable estimate (and by implication the aggregated estimate) is statistically equal to the individual-level estimate.

As discussed earlier, there are multiple potential reasons why the Hausman test could reject the equality of the individual and the aggregated estimates. This could be due to the presence of general equilibrium or cross-level effects that cause the estimators to estimate different underlying constructs, it could be due to omitted community-level variables which

result in exacerbated omitted variables bias in the aggregated estimator, or it could be due to omitted individual-level variables that may cause insurance to be endogenous in the individual-level model.

Given that we do in fact observe a rich array of individual and community-level control variables, we can further explore the relative inferences from individual versus aggregated models by next considering estimates that include this complete set of controls. Table 4 column [4] presents this individual-level OLS model, indicating that the insurance effect drops by half after including controls, but is still statistically significantly different from zero. Control covariates that also significantly affect child mortality in this model include mother's education, water supply and sanitation, and household wealth. Community health care infrastructure indicators are less significantly related to child mortality, though not irrelevant.

When next considering the aggregated results with the full set of controls (Table 4 column [5]), the key finding is that the insurance coefficient drops to virtually zero. Based on the above theoretical discussion of omitted variables bias in aggregated models, this suggests that the correlation between insurance and these controls may have operated mostly at the community level, hence their omission led to a much larger omitted variables bias in the aggregated model than in the individual-level model.

In terms of standard errors, Table 4 indicates that with controls the aggregated model still yields standard errors on the insurance coefficient that are an order of magnitude larger than the individual-level model. Furthermore, virtually all of the control variables are statistically insignificant in the aggregated model, arising from some combination of reduced variation and multicollinearity. Based on the fact that the insurance standard errors in the aggregated model

changed little after adding the control variables, however, it does not appear that the insurance coefficient itself was influenced by multicollinearity.

Again the instrumental variables model can assist in formally testing the difference between aggregated and individual-level results. Comparing models with controls, the instrumental variables model (Table 4 column [6], which treats only insurance as endogenous) is again quite similar to the aggregated model in terms of both the insurance coefficient and its standard error, and hence has much larger standard errors than the individual-level model. The exogenous controls in the instrumental variables model, however, have virtually the same coefficients and precision as the individual-level model, given that these exploit the same level of variation (this result was also found in the Monte Carlo simulation of Table 1). Formally comparing these models with controls, the Hausman test now fails to reject the null hypothesis that the instrumental variables and individual-level OLS models are equivalent. This is true whether including all controls in this Hausman test, or running the Hausman test on the insurance variable alone.

As one final test, we explored a second instrumental variables model that additionally treated the wealth and water supply and sanitation variables as endogenous (using the same set of county×year instrumental variables as used to instrument insurance alone). One interpretation of this model is that these potentially endogenous control variables now rely only on their community-level variation to control for confounding. The results of this model (Table 4 column [7]) are virtually identical to the previous instrumental variables model that treated only insurance as endogenous. The Hausman tests again cannot reject the null hypothesis that these variables are jointly exogenous, or that the insurance variable alone is unaffected by their endogeneity. This suggests that these variables appear mainly to be controlling for community-

level confounding, which again would explain why the aggregated model appeared to be much more biased by their omission than did the individual-level model.

DISCUSSION

The result of the above model comparisons and tests is that the OLS individual-level specification with controls (and community fixed effects) is our preferred model (Table 4 column [4]). Furthermore, unless there happen to be countervailing remaining biases in the various models, the results suggest that insurance is not endogenous in this preferred model after including controls, and that there were no general equilibrium or cross-level effects of the insurance expansion. This implies that the aggregated model with controls (Table 4 column [5]) would also yield unbiased effects that could be interpreted at either the aggregated or individual levels.

However, this aggregated model with controls is substantially inferior due to its larger confidence intervals. When comparing the upper bound (in absolute value) of the 95% confidence intervals on the insurance effect, the models yield somewhat different inferences. The point estimate of insurance in the individual model implies that the 1970s insurance expansion explained only 4% of Costa Rica's marked child mortality drop between 1973 and 1984, and the 95% confidence interval can bound this effect to be under 6%. By contrast, the 95% confidence interval from the aggregated model can only bound this effect to be under 16%, which would not be an insubstantial intervention.[1] Translating these effects into more generalizable magnitudes, the individual-level point estimates imply an insurance-mortality

[1] This estimate compares quite closely to Dow and Schmeer (in press). Based on vital statistics data on all births in 1973 and 1984, the aggregated estimates in that paper bounded the insurance effect to explain at most 20% of Costa Rica's infant mortality drop over this period. This suggests that sample limitations in the present paper to estimate mortality for all children of only ever married women 18-25 did not lead to substantial biases.

elasticity of -0.10 with an upper bound of -0.14, as compared to the aggregated upper bound elasticity of -0.37. Although not radically different in this application after controlling for a rich set of observed confounders, the difference is indeed substantive.

Furthermore, based on the fact that the individual and aggregated estimates were radically different before controlling for confounders, our results do suggest the importance of careful investigation of the effects of aggregation in any given application. As emphasized in our theoretical consideration of the effects of aggregation, however, it would be a mistake to blindly assume that aggregated estimators always yield more biased estimates than individual-level estimators. One reason for interpreting aggregation as equivalent to an instrumental variables estimator is to further emphasize the point that aggregation is not in and of itself uniformly dangerous. As with instrumental variables techniques, aggregated estimators have their place, with the same inherent possibilities for use and abuse. Every estimator has its strengths and weaknesses, and applications that carefully compare across different levels of aggregation may often prove the most convincing.

Based on our analysis, we conclude that although insurance does have a statistically significant effect on child mortality, this effect is quite small. If all else had been held constant from 1973 to 1984 except for the insurance intervention, instead of the actual child mortality drop from 53 to 18 deaths per 1000 live births, Costa Rica's mortality would have dropped to only 51.6. As a health intervention, expanding insurance coverage from 46% to 74% of our population did not have much effect on child mortality.

There are a number of potential reasons why the insurance expansion might have been ineffective. For example, it could be that the treated group who became newly insured in fact already had complete access to care even before the expansion, hence the insurance did not affect

utilization but operated only as a financing intervention. However, we find this explanation unconvincing a priori, given that in settings throughout the world health care user fees have been found to lower utilization (Jimenez 1995). We believe it is plausible that a substantial portion of the large increase in national health care expenditures over this period could have been induced by the price-effects of insurance.

A second potential explanation of the lack of insurance effects is that the particular types of medical care induced by insurance had very little effect on child mortality outcomes. Economic insurance theory predicts that uninsured individuals will be willing to pay out-of-pocket for the most cost-effective types of health care, hence insurance will increase utilization primarily for less effective health care interventions that are not worth their full cost. For example, if insurance primarily induces increases in expensive hospitalization, and if the available hospital quality and technology yields relatively low health benefits, then although insurance might have effects on selected health outcome measures, the insurance expansions might not have major effects on aggregate health indicators such as mortality. Further research on the exact types of health care demand induced by insurance would be particularly useful for further exploring these issues.

Also important will be to conduct similar analyses on the health effects of insurance in a wider range of settings. Generalizability of individual studies can always be questioned due to specific institutional features. For example, the fact that Costa Rica made available a wide range of primary health care interventions regardless of insurance status may have contributed to a smaller estimated effect of insurance than would be found in another setting that expanded insurance prior to investing in expanded primary care programs. This of course begs the question of whether such primary care programs would be more cost-effective at achieving

health gains, if costly insurance programs appear to have little mortality benefit beyond these primary care programs. Clearly additional research is needed to better explore these and related questions, such as the value of insurance in reducing the risk of catastrophic expenditures as compared with the opportunity cost of insurance expansions crowding out alternative potentially more effective health interventions.

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Table 1: Monte Carlo Simulations of Effects of Omitting Individual and Community Level Controls

Variable Name	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]	[10]	[11]	[12]
	OLS Indiv	OLS Aggreg	IV Indiv	OLS Indiv	OLS Aggreg	IV Indiv	OLS Indiv	OLS Aggreg	IV Indiv	OLS Indiv	OLS Aggreg	IV Indiv
Insurance	1.504	1.499	1.499	1.286	1.496	1.496	1.294	1.005	1.005	1.005	0.999	0.999
	(.096)	(.168)	(.164)	(.107)	(.169)	(.164)	(.092)	(.166)	(.161)	(.0994)	(.166)	(.161)
Individual Health				0.862	0.718	0.757				1.003	0.996	1.005
				(.114)	(1.690)	(.131)				(.112)	(1.45)	(.129)
Community Infrastructure							0.859	1.002	1.002	1.003	1.006	1.005
							(.148)	(.166)	(.160)	(.147)	(.166)	(.160)

Notes:

1. "OLS Indiv" are individual-level models estimated by OLS using 10,000 observations (100 individuals in each of 100 communities).
2. "OLS Aggreg" are community-level models estimated by OLS using 100 observations on community mean values.
3. "IV Indiv" are individual-level instrumental variables models using 10,000 observations, with 99 community dummies as instruments.
4. Individual-level OLS and IV models report Huber community-clustered standard errors.
5. Monte Carlo results are based on 500 replications, with true coefficients all equal to one.

Table 2: Variable Definitions and Summary Statistics for Ever Married Women Ages 18 to 25 with Live Births

Variable Name	Description	Mean	Standard Deviation
Dependent variable			
Child mortality	Number of children died / children ever born	0.032	0.126
Kids ever born	Number of children ever born	2.025	1.213
Kids currently alive	Number of children currently alive	1.927	1.111
Independent variables			
Individual characteristics			
Insurance	Dummy = 1 if woman has health insurance coverage	0.631	0.483
Age	Age of woman in years	22.289	2.100
Educational Attainment			
None (omitted)	Dummy = 1 if no education	0.044	0.205
Primary education	Dummy = 1 if primary education only	0.625	0.484
Secondary education	Dummy = 1 if secondary or higher education	0.331	0.470
Migration status	Dummy = 1 if woman has migrated in the past 5 years	0.247	0.431
Household sanitation characteristics			
No sanitation	Dummy = 1 if household does not have own sanitation	0.186	0.389
No bath	Dummy = 1 if household does not have own bathroom	0.281	0.449
No water	Dummy = 1 if household does not have own piped water	0.336	0.472
Wealth indicators			
Cement roof	Dummy = 1 if household has cement roof	0.039	0.194
Wood or tile floor	Dummy = 1 if household has wood or tile floor	0.675	0.468
Cement or brick walls	Dummy = 1 if household has cement or brick walls	0.233	0.422
Electric light	Dummy = 1 if household has electric lighting	0.722	0.448
No light	Dummy = 1 if household does not have any lighting	0.029	0.168
Electric or gas cooking fuel	Dummy = 1 if household has electric or gas cooking fuel	0.499	0.500
Stove	Dummy = 1 if household has a stove	0.600	0.490
Refrigerator	Dummy = 1 if household has a refrigerator	0.322	0.467
Radio and television	Dummy = 1 if household has radio and/or televesion	0.822	0.383
Telephone	Dummy = 1 if household has a telephone	0.099	0.299

Household density	Number of persons per room in household	1.424	1.006
Wealth1	First principle component of wealth indicators	0.306	1.830
Wealth2	Second principle component of wealth indicators	-0.037	1.149
Community characteristics			
Travel time to San Jose	Travel time to San Jose (capital city), hours	3.230	3.021
Primary health care programs	Proportion of population covered by post-1973 primary health care programs	0.357	0.343
Nearest clinic opened after 1973	Proportion of population with nearest clinic inaugurated since 1973	0.225	0.349
Deaths not certified	Proportion of deaths not medically certified	0.164	0.202
Number of observations		134036	

Notes:
1. All data except community characteristics are drawn from the 1973 and 1984 Costa Rican censuses.
2. Wealth1 and Wealth2 are the first two principle components of cement roof, floor, cement or brick walls, electric light, no light, electric or gas cooking fuel, no stove, no refrigerator, no radio or tv, no telephone and household density*(-1).

Table 3: Summary Statistics by Insurance Status for Ever Married Women Ages 18 to 25 with Live Births

Variable Name	1973			1984		
Dependent variable	Insured	Uninsured	All	Insured	Uninsured	All
Child mortality	0.039	0.064	0.053	0.016	0.022	0.018
Kids ever born	1.987	2.426	2.224	1.829	2.078	1.893
Kids currently alive	1.869	2.215	2.056	1.785	2.011	1.842
Independent variables						
Individual characteristics						
Insurance	1.000	0.000	0.459	1.000	0.000	0.744
Age	22.349	22.047	22.186	22.432	22.141	22.357
Educational Attainment						
None (omitted)	0.030	0.101	0.069	0.018	0.055	0.028
Primary education	0.651	0.793	0.728	0.520	0.669	0.558
Secondary education	0.319	0.105	0.203	0.462	0.276	0.414
Migration status	0.264	0.278	0.271	0.221	0.259	0.231
Household sanitation characteristics						
No sanitation	0.161	0.325	0.250	0.115	0.226	0.144
No bath	0.263	0.549	0.418	0.147	0.318	0.191
No water	0.241	0.498	0.380	0.267	0.420	0.306
Wealth indicators						
Cement roof	0.047	0.017	0.031	0.050	0.029	0.044
Wood or tile floor	0.780	0.639	0.704	0.672	0.612	0.657
Cement or brick walls	0.199	0.085	0.137	0.331	0.190	0.295
Electric light	0.807	0.457	0.618	0.843	0.641	0.791
No light	0.018	0.030	0.024	0.024	0.054	0.032
Electric or gas cooking fuel	0.552	0.257	0.392	0.632	0.387	0.569
Stove	0.768	0.465	0.604	0.648	0.451	0.598
Refrigerator	0.277	0.116	0.190	0.465	0.246	0.409
Radio or television	0.844	0.720	0.777	0.891	0.738	0.852
Telephone	0.072	0.031	0.050	0.147	0.086	0.131
Household density	1.381	1.780	1.597	1.208	1.606	1.310

Wealth1	0.522	-0.765	-0.174	0.914	-0.225	0.623
Wealth2	-0.279	-0.094	-0.179	0.048	0.083	0.057
Community characteristics						
Travel time to San Jose	2.521	5.044	3.886	2.698	3.092	2.799
Primary health care programs	0.000	0.000	0.000	0.587	0.608	0.592
Nearest clinic opened after 1973	0.000	0.000	0.000	0.362	0.405	0.373
Deaths not certified	0.171	0.352	0.269	0.088	0.114	0.095
Number of observations	24411	28785	53196	60143	20697	80840

Notes:

1. The difference between 1973 and 1984 observations are statistically significant for all variables except no stove with a p-value <0.0001.
2. The difference between insured and uninsured women in 1973 and 1984 is statistically significant for all variables.

Table 4: Regression Results of Insurance Effects on Child Mortality for Ever Married Women Ages 18 to 25 with Live Births

Variable Name							
Independent variables	[1]	[2]	[3]	[4]	[5]	[6]	[7]
	OLS Indiv	OLS Aggreg	IV Indiv	OLS Indiv	OLS Aggreg	IV Indiv	IV Indiv
Individual characteristics							
Insurance	-0.0131 **	-0.0485 **	-0.0535 **	-0.0049 **	0.0050	0.0004†	0.0102†
	(.0010)	(.0096)	(.0117)	(.0011)	(.0120)	(.0124)	(.0122)
Age				0.0021 **	0.0070	0.0021 **	0.0023 **
				(.0003)	(.0096)	(.0003)	(.0004)
Primary education				-0.0231 **	0.0076	-0.0235 **	-0.0161 **
				(.0033)	(.0649)	(.0037)	(.0052)
Secondary education				-0.0351 **	0.0180	-0.0358 **	-0.0246 *
				(.0033)	(.0776)	(.0042)	(.0106)
Migration status				0.0025 *	0.0166	0.0027	0.0024
				(.0014)	(.0108)	(.0015)	(.0016)
Household characteristics							
No sanitation				0.0054 **	0.0092	0.0055 **	0.0128†
				(.0019)	(.0243)	(.0020)	(.0263)
No bath				0.0066 **	0.0242	0.0069 **	0.0296†
				(.0017)	(.0183)	(.0017)	(.0199)
No water				0.0062 **	0.0172	0.0063 **	0.0285†
				(.0013)	(.0191)	(.0014)	(.0219)
Wealth1				-0.0016 **	-0.0015	-0.0019 *	-0.0001†
				(.0005)	(.0048)	(.0007)	(.0050)
Wealth2				0.0019 **	0.0037	0.0020 **	0.0080†
				(.0005)	(.0059)	(.0005)	(.0060)
Community characteristics							
Travel time to San Jose				0.0063 *	0.0046 *	0.0065 *	0.0049 *
				(.0026)	(.0021)	(.0026)	(.0024)
Primary health care programs				0.0024	-0.0014	0.0016	0.0019
				(.0074)	(.0060)	(.0074)	(.0068)
Nearest clinic opened after 1973				-0.0033	-0.0009	-0.0036	-0.0036
				(.0039)	(.0036)	(.0038)	(.0038)
Deaths not certified				0.0496 **	0.0340	0.0517 **	0.0378
				(.0168)	(.0175)	(.0184)	(.0197)

Year 1984	-0.0441	**	-0.0219	**	-0.0323	**	-0.0240	**	-0.0200	*	-0.0240	**	-0.0270 **
	(.0027)		(.0036)		(.0042)		(.0037)		(.0090)		(.0037)		(.0061)
Constant	0.0644	**	0.0762	**	0.0846	**	0.0234		-0.1546		0.0214	**	-0.0118
	(.0016)		(.0044)		(.0060)		(.0066)		(.2227)		(.0073)		(.0138)
Joint significance of county fixed effects	p<0.0001		p<0.0001		p<0.0001		p<0.0001		p<0.0001		p<0.0001		p<0.0001
First stage instrument strength					p<0.0001						p<0.0001		p<0.0001
Hausman endogeneity test: all coefficients					p=0.001						p=0.662		p=0.0750
Hausman endogeneity test: insurance coefficient only					p=0.0006						p=0.6626		p=0.2118
N	134036		198		134036		134036		198		134036		134036

Notes:

1. *significant at 5%; **significant at 1% level (robust clustered standard errors in parentheses)
2. "Indiv" indicates individual-level unit of analysis; "aggreg" indicates county-year aggregated unit of analysis.
3. [†]Regressions 3, 6 and 7 treat insurance as endogenous; Regression 7 also treats no sanitation, no bath, no water, wealth1 and wealth2 as endogenous. County-year interaction dummies are used as the identifying instruments for all endogenous variables.