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NUTRITION AND INFANT HEALTH IN JAPAN

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ABSTRACT

The model presented in this paper emphasizes the importance of the mother's nutritional intake as a determinant of infant health. Using cross-sectional market averages for 1980 and 1981 in Japan, we find that the nutrient intake of the mother during pregnancy is a potential determinant of neonatal and infant mortality in Japan, with increased consumption of calcium and iron leading to improved birth outcomes. Using the results obtained from the estimation of neonatal and infant mortality production functions, we note that increases in the prices of food items, in particular milk and meat, would lead to increases in neonatal and infant mortality rates. We discover that the availability of abortion in Japan, unlike in the U.S., is positively related to mortality rates, although never significantly. Finally, we see that cigarette smoking, alcohol consumption, and poor environmental quality all have strongly adverse effects on newborn survival outcomes in Japan.

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I. INTRODUCTION

The consensus obtained from recent health economics studies is that parental behaviors - the consumption of medical care, smoking, drinking, and residence location - determine infant health through the health of the mother (Rosenzweig and Schultz 1982, 1983a, 1983b; Schultz 1984; Joyce and Grossman 1986; Corman, Joyce, and Grossman 1987; Joyce, Grossman, and Goldman forthcoming).[1]

These studies found that the use of medical care, which includes teenage family planning, prenatal care, and neonatal intensive care, as well as the use of abortion significantly reduce the risk of neonatal mortality. They also find that the delay of prenatal care and smoking by the mother have adverse effects on birth weight, gestational age, and infant mortality. Finally, air pollution, mainly sulfur dioxide, is found to induce high risks in newborn survival outcomes.

Despite the implicit assumption that the health and nutritional status of the mother is a major determinant of birth outcomes (e.g. birth weight and the gestation period) and infant health (e.g. neonatal and infant mortalities), these recent empirical studies fail to emphasize the role of the mother's nutritional status. The health and nutritional status of the

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[1] These studies emphasize that the omission of the child's health endowment, known to the parents but not to researchers, from econometric regressions leads to the misinterpretation of the estimates of the child's health production function.

mother significantly affect her productivity in both labor and non labor markets. A weak health and poor nutritional status often result in the loss of physical and mental strength, and, consequently, in frequent illness (Pitt and Rosenzweig 1985). It is reasonable, therefore, to assume that inadequate nutrient intake by the mother leads to a reduction in efficiency in the production of her child's health.[2]

Hence, we incorporate the mother's nutritional status as an input into an infant health production function. Furthermore, the omission of this variable will exacerbate the heterogeneity problem (i.e., heterogeneous health endowments across households) since the health and nutritional status of the mother appears highly correlated with the health and nutritional status of the infant. By including a variable representing the mother's health endowment which captures part of the infant's health endowment, we are able to mitigate the biases in the estimated coefficients of the other explanatory variables which are causally related to the health endowment of the infant.

Specifically, we investigate the importance of nutrient intake by the mother and other socioeconomic factors in the determination of infant health in Japan. This application of the

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[2] The importance of nutrition and health as determinants of productivity have been demonstrated theoretically as well as empirically in studies of developing countries, especially economically poor countries (Belli 1971; Selowsky and Taylor 1973; Wilford 1975; Franke and Barrett 1975; Johnston 1977; Bliss and Stern 1978a and 1978b; Ward and Sanders 1980; Wolfe and Behrman 1982 and 1983; Behrman and Wolfe 1984; and Strauss 1984 and 1986).

economic models of the family to Japanese data should be underlined for the following reasons. First, an understanding of the effect of nutrient intake by the mother on infant health is crucial for the development of policies designed to improve infant health. Secondly, despite the many empirical studies of U.S. infant health, no attempt has been made to explore theoretical and empirical studies in Japan. Finally, a quantitative evaluation of these effects rests on studies such as ours.

II. ANALYTICAL FRAMEWORK

We assume that household utility depends on infant health H, a vector of commodities Z_1 which affect H, and a vector of commodities Z_2 which do not affect H (Rosenzweig and Schultz 1982 and 1983b):

$$U = U(H, Z_1, Z_2), \quad . \quad . \quad . \quad . \quad (1)$$

where U is a strictly convex, twice differentiable utility function. Let the survival probability of the infant π, representing infant health, be described by the following production function:

$$\pi = \Gamma(M, Z_1, \mu). \quad . \quad . \quad . \quad . \quad (2)$$

M represents the quantity and quality of the medical care of the mother which provides utility indirectly through infant health, Z_1 includes the mother's health, smoking and drinking by the mother, and the number of previous births, and μ represents the family specific (genetic and biological) infant health endowment, known only to household members. Maximizing this utility function subject to production and resource constraints generates demand for the infant's survival probability as a function of input prices, income, productive efficiency, environmental factors, and tastes.

The mother's health H_m is determined by her consumption of nutrients N, cigarette smoking S, alcohol drinking D, own-medical care M, environmental quality Q (e.g. air pollution), mother's age G, the health endowment of the mother μ_m, and productive

efficiency:[3]

$$H_m = h(N, S, D, M, Q, G, \mu_m). \quad . \quad . \quad . \quad . \quad (3)$$

Substituting mother's health for Z_1 in the structural production function of equation (2), we obtain a quasi-structural infant survival production function:

$$\pi = \delta(N, S, D, M, Q, G, \mu_m, \mu). \quad . \quad . \quad . \quad . \quad (4)$$

The complement of π, $1-\pi$, is defined as the probability of neonatal (or infant) mortality. Given the considerable evidence obtained in previous research that low birth weight (2,500 grams or less) is a major determinant of neonatal mortality (i.e. Eisner, et. al 1979; Harris 1982; and the aforementioned group of health economic studies), low birth weight can be included as a determinant of infant survival. Since low birth weight is an endogenous variable whose determinants are similar to those of neonatal and infant mortality, the above quasi-structural production function is obtained. The use of abortion services A and prenatal care C, included in the medical care M received by the mother, should be included in the birth weight function and, hence, in the infant survival production function.[4]

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[3] Mother's schooling is often used as a proxy variable for both productive and allocative efficiency. Productive efficiency implies that more educated individuals can produce a larger output from quantities of inputs, while allocative efficiency indicates that more educated individuals are better informed about the true effects of the inputs and, hence, choose a better input mix (Grossman and Joyce 1987). In our study, mother's schooling is treated as a determinant of the demand for health-related inputs.

[4] The recursive models of others include neonatal mortality, low birth weight, gestation, and risk of prematurity functions (Corman, Joyce, and Grossman 1987, and Joyce 1987). There are

The production function of the probability of neonatal (or infant) mortality to be estimated is:

$$1-\pi = \pi(N, S, D, A, C, X, Q, G, \mu_m, \mu). \quad . \quad . \quad . \quad . \quad (5)$$

where X is postnatal care. The health endowments of the mother and infant are unobserved although some elements are captured by the inclusion of the mother's nutrient intake. Furthermore, some of the health-related behavioral inputs are likely to be causally related to the unobserved endowments. That is, given a poor endowent, parents will seek more and better pre- and postnatal care, will be more concerned with nutrient intake, and will refrain from heavy smoking and drinking. Finally, a poor health endowment of the fetus may induce parents to use abortion services. To circumvent the endogeneity problem, we first estimate health-related behavioral input demand functions whose determinants are orthogonal to the unobserved health endowments, leading to our final, operational model of the production of neonatal (or infant) mortality:

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advantages and disadvantages to using a four equation system such as these. The major advantage is that the system is built on biological effects within a microeconomic framework, while the primary disadvantage is that the specification of each function becomes more arbitrary in order to satisfy the rank and order conditions for identification. Instead, we use a two equation system comprised of production functions for neonatal (or infant) mortality and for mother's health, implicitly assuming that mother's health both directly and indirectly effects the health of the infant. The indirect effects result from the impact of mother's health on birth weight, gestation, and the risk of prematurity. Our model of neonatal (or infant) mortality is similar that of others with the exception of our treatment of nutrient intake as an input into the production of the mother's health, and, thus, the infant's health.

Input Demand Functions:

$$N, S, D, A, C, X = f(P, Y, Q, G, K), \quad . \quad . \quad . \quad . \quad (6)$$

and

Health Production Function:

$$1-\pi = g(N^*, S^*, D^*, A^*, C^*, X^*, Q, G), \quad . \quad . \quad . \quad . \quad (7)$$

where P is a vector of input prices, Y is household income, Q and G are as above, K is a vector of exogenous socioeconomic variables, and the variables with an asterisk are endogenous, health-related, behavioral inputs. The treatment of the behavioral inputs as endogenous allows us to circumvent the problem of the heterogeneity of the health endowment.

III. EMPIRICAL IMPLEMENTATION

A. Regression Variables

The variables used in our model are cross-sectional market averages in 1980 and 1981.[5] Table 1 contains the definitions, means, standard deviations, and sources of the variables used in this study. The probability of neonatal mortality (death of an infant in the first 27 days of life) is defined by the ratio of the observed number of neonatal deaths to the total number of live births, while the probability of infant mortality (death of an infant in the first 364 days of life) is defined by the ratio of the observed number of infant deaths to the total number of live births. Recently, in Japan, neonatal mortality accounts for approximately two-thirds of all infant mortality, suggesting that any attempt to explain the recent behavior of infant mortality must focus on neonatal mortality and that infant health can be improved by the identification of important determinants of neonatal mortality.

Sufficient nutrient intake is indispensable for both fetal and maternal well-being. Fetal growth will be retarded and the risk of mortality or morbidity increased by deficiencies in the

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[5] Japan is administratively divided into forty-seven prefectures of which forty-three carry the suffix ken and the remaining four are known as to, do, or fu. Each prefecture is subdivided into shi (cities) and gun or shicho (rural country). We employ data for urban areas, referred to as shi due to the availability of data on household food consumption which allows us to calculate nutrient intake, whose effects on infant health are our primary concern.

Table 1
Definitions, Means, Standard Deviations, and Sources of Variables

Variable	Definition
Neonatal Mortality Rate	Deaths of infants less than 28 days old per 1,000 live births, μ=4.692, σ=0.708, source: Vital Statistics (VS)
Infant Mortality Rate	Deaths of infants less than 365 days old per 1,000 live births, μ=7.105, σ=0.844 source: VS
Cigarettes*	Number of cigarettes sold per person ages 20 and over per month, μ=210.740, σ=41.990, source: Min-Ryoku (MR)
Liquor*	Liters of whiskey and brandy sold per person ages 20 and over per month, μ=0.387, σ=0.170, source: MR
Protein	Annual consumption, in grams, of protein per person in a typical urban household, μ=18099, σ=4.965E+4, source: Annual Report on the Family Income and Expenditures Survey (ARFIES), and Kagawa (1987)
Calcium	Annual consumption, in milligrams, of calcium per person in a typical urban household, μ=180154, σ=7.592E+5, source: ARFIES, Kagawa
Iron	Annual consumption, in milligrams, of iron per person in a typical urban household, μ=2827, σ=6.558E+3, source: ARFIES, Kagawa
Vitamin A	Annual consumption, in microgram, of Vitamin A per person in a typical urban household, μ=251711, σ=7.700E+5, source: ARFIES, Kagawa
Vitamin C	Annual consumption, in milligrams, of Vitamin C per person in a typical urban household, μ=250783, σ=1.573E+6, source: ARFIES, Kagawa
Midwife	Proportion of live births attended by a midwife, μ=0.040, σ=0.020, source: VS
Abortion	Number of induced abortions per 1,000 live births, μ=20.398, σ=8.797, source: VS
Hospital Beds*	Number of general beds in hospitals and clinics per 1,000 population, μ=10.377, σ=5.477, source: Japan Statistical Yearbook
Mother's Age*	Proportion of live births by mothers ages 19 years or less, μ=0.010, σ=0.003, source: VS
Sulfur Dioxide*	Sulfur dioxide level in parts per million per cubic meter, μ=39.640, σ=10.945, source: MR
Particulates*	Suspended particulates level in milligrams per cubic meter, μ=61.802, σ=15.674, source: MR

* The symbols μ and σ represent the weighted sample mean and standard deviation, respectively. Variables with an asterisk (*) are prefecture-(ken in Japanese) specific.

mother's diet (King and Butterfield 1986). A number of nutrients must be considered as inputs into the production of maternal and infant health. The inclusion of all nutrients is, however, impractical, forcing us to choose a tractable subset which includes protein, calcium, iron, vitamin A, and vitamin C.[6]

Protein, obtained from dairy products, meats, and grains, stimulates fetal and maternal tissue growth and increases maternal circulating blood.[7] A protein deficiency during pregnancy decreases birth weight and causes toxemia. Excessive protein consumption may induce a premature birth and excessive neonatal mortality. Calcium, present in dairy products and leafy vegetables, is important for fetal skeletal growth and maternal calcium metabolism. A calcium deficiency would lower the bone densities of the mother and infant. Iron, found in meats, grains, and leafy vegetables, is needed for the manufacturing of hemoglobin, which carries oxygen through the bloodstream, in both fetal and maternal red blood cells. Anemia, the principal consequence of an iron deficiency, is associated with increased incidences of spontaneous abortion, premature delivery, low birth weight births, stillbirth, and perinatal death. Vitamins A and C are essential for tissue growth. Vitamin A, obtained from green

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[6] The number of calories consumed is often included in studies of nutrition demand in developing countries. We choose not to include calories due to their high correlation with protein consumption which we do include.

[7] The following arguments on the effects of nutrients on the human body are taken from Worthington-Roberts and Vermeersch, 1985, and Williams, 1985.

and yellow vegatables, is also important in bone growth, while vitamin C, found primarily in citrus and other fruits and vegatables, stimulates iron absorption. Impaired vision and premature birth are two of the consequences of a vitamin A deficiency, while renal anomalies and multiple malformations result from excessive intake. Massive intake of vitamin C adversely influences fetal metabolism, but a deficiency has not be shown to affect the course or outcome of pregnancy.

To quantify the above five nutrients we subgroup average household food consumption into fourteen categories: non-glutinous rice, bread, noodles, cereal and other flours, fish and shellfish, dried fish, meat, vegetables, fruits, pickles, eggs, milk, powdered milk, and cheese. The annual per capita quantity consumed of each food is obtained by dividing the average household consumption by the average number of persons per household. The annual per capita nutrient intakes are then defined as weighted sums of per capita food consumption, where the weights are the quantity of each nutrient in 100 grams of each food.[8]

Cigarette and alcohol consumption, whose adverse affects on birth outcomes have been well documented, are also included as health related behavioral inputs. Placental abnormalities are

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[8] Formally, let F_i be the annual per capita quantity of food i consumed (in 100's of grams), and a_{ni} be the quantity of nutrient n in 100 grams of food i. Then annual per capita consumption of nutrient n is defined as:

$$N_n = \Sigma_i \, a_{ni} F_i, \quad i=1,2,\ldots,14.$$

The weights, a_{ni}, are taken from Kagawa (1987).

determined, in part, by both the number of years the mother has smoked and her current smoking habits. Women who smoke during pregnancy give birth to smaller babies than nonsmokers (Worthington-Roberts and Vermeersch 1985), while the consumption of more than two alcoholic drinks per day increases the risk of delivering a baby with physical and developmental abnormalities (Brown 1983).

We use the the levels of sulfur dioxide and particulates in the air to control for environmental quality, allowing us to compare the effects of these pollutants in Japan to those found in the U.S. There is some evidence that air pollutants increase infant mortality rates by raising the percentage of low birth weight births (Joyce, Grossman, and Goldman, forthcoming).

As proxies for pre- and postnatal care, direct measures of which are unavailable, we include the availability of general hospital beds, abortion, and the delivery of an infant by a midwife. We expect a negative relationship between hospital bed availability and neonatal and infant mortality arising from the inverse relationship between the availability of pre- and postnatal care (assumed highly correlated with the availability of beds) and their prices. The effect of abortion on mortality is ambiguous. If, through their use of amniocentisis or prenatal checkups, more mothers decide to terminate their pregnancies, there will be fewer high risk births, lowering mortality. Alternatively, if abortion is used in place of contraception or to remedy contraceptive failure (frequent in Japan, as suggested

by Preston, 1986) or illegitimacy, a weak positive relationship between abortion use and mortality would result, given the high correlation observed between fertility and infant mortality.

Finally, mother's age is included as a regressor due to the high risk of light birth weight and neonataly mortality found among teenage mothers (Geronimus 1987, and Corman, Joyce, and Grossman 1987).

B. Estimation

We assume that the probability of a neonatal death P is given by the logistic function:

$$P = \frac{1}{1 + \exp-(\alpha + \Sigma_i \beta_i X_i)} \quad , \quad i=1,\ldots, m, \qquad (8)$$

where X is a vector of right-hand-side variables, and α and β are parameters. Solving for the logarithm of the ratio of the probability of neonatal mortality to the probability of survival, the following linear equation is obtained:

$$\ln [P/(1-P)] = \alpha + \Sigma_i \beta_i X_i \quad . \quad . \quad . \quad . \quad . \quad (9)$$

The marginal effect of a one-unit change in X_i on the probability of neonatal mortality is given by:

$$\delta P/\delta X_i = \beta_i P(1-P) . \quad . \quad . \quad . \quad . \quad (10)$$

Weighted regressions are fitted separately for neonatal and infant mortality due to our expectations of differential impacts of the health related behavioral inputs on mortality.[9] For each mortality function, we estimate a basic ordinary least squares

[9] The weight is defined as $[BxPx(1-P)]^{1/2}$, where B is the total number of live births.

(OLS) model which includes cigarette smoking, alcohol consumption, protein, calcium, vitamin A, hospital beds, sulfur dioxide, and particulates. We also estimate an extended OLS model which includes, in addition to the variables in the basic model, iron, vitamin C, midwife deliveries, abortion services, and mother's age. We then reestimate the extended model using two-stage least squares methods based on the assumption that an omitted variable, the infant's health endowment, is correlated with the health related behavioral inputs. Using Wu's T_2 statistic (Wu 1973, 1974; Nakamura and Nakamura 1981) we then test the hypothesis of a zero correlation between the OLS residuals and the health-related inputs. If a correlation exists between them, i.e. the heterogeneity problem exists, then the OLS estimates will be biased and inconsistent.

One interest is to estimate the net effect of food prices on infant health through their effect on the consumption of food nutrients, requiring information on the own- and cross-price elasticities of demand for the foods. The elasticity of infant health (neonatal mortality, for example) with respect to the price of food j is obtained as:

$$\sigma_{neo,Pj} = (1-P)\ \Sigma_i\ \Phi_{neo,Nn}\ E_{Nn,Pj}\ ,\quad n=1,\ldots,5 \qquad (11)$$

where the n are the five nutrients in the infant health production functions and $\Phi_{neo,Nn}$ is the elasticity of infant health with respect to nutrient n. $E_{Nn,Pj}$ is a weighted average of the price elasticities of demand for the foods which contain nutrient n, and is defined as follows (Pitt 1983):

$$E_{Nn,Pj} = \frac{\Sigma_i \, a_{ni} E_{ij} F_i}{\Sigma_i \, a_{ni} F_i} \quad , \quad . \quad . \quad . \quad . \quad (12)$$

where i=1,...,h are the h foods supplying nutrient n, and E_{ij} is the price elasticity of demand for food i (F_i) with respect to the price of food j.[10] This estimation of the effects of various food prices on infant health is important in designing public policy to improve infant health. Of note here are the restrictive trade policies in place in Japan (particularly those on food imports such as dairy products and meats) which lead to higher prices for many commodities than would exist in a competitive market. The more competitive market system which may result from less restrictive trade policy in Japan may also improve the well-being of other individuals in Japan.

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[10] These food price elasticities are uncompensated. To estimate own- and cross-price elasticities of demand we used seemingly unrelated equations procedure. For computational feasibility, we selected seven food categories: bread, fish and shellfish, meat, milk, eggs, vegatables, and fruits. Each demand equation contains the seven food prices and various household characteristics (including income, head of household's age, size of household, women's education, and the number of children). In this procedure, identical regressors should not be used in each of the equations, leading us to keep only those household characteristics which were statistically significant in each equation, while the seven prices are always kept. The results of this procedure are available on request.

IV. RESULTS

Ordinary least squares (OLS) and two-stage least squares (TSLS) estimates of the neonatal and infant mortality production functions are reported in Table 2. Selected nutrient price elasticities and health price elasticities are contained in Table 3. Equations (1) and (4) in Table 2 are the OLS estimates of the basic neonatal and infant mortality production functions, respectively, and include the variables on cigarette smoking, alcohol consumption, nutrient consumption (protein, calcium, and vitamin A), hospital beds, and air pollutant levels. Equations (2) and (5) are the respective extended OLS models which add two additional nutrients (iron and vitamin C), medical care measures (midwife deliveries and abortion services), and mother's age to the basic models. The estimates of these extended equations obtained using two-stage least squares are reported in equations (3) and (6) of Table 2.

Cigarette and alcohol consumption are found to have strong adverse effects on infant health as measured by the neonatal and infant mortality rates. Similarly, higher levels of air pollutants are found to result in higher neonatal and infant mortality rates. With respect to the mother's nutrient intake, calcium, iron, and vitamin C have the expected negative signs, while the signs on protein and vitamin A are positive. Among the medical inputs, the signs of the estimated coefficients on midwife deliveries and hospital beds are as expected.

Table 2
Estimates of Logit Neonatal and Infant Mortality Production Functions[a]

Explanatory Variable	Neonatal Mortality OLS (1)	Neonatal Mortality OLS (2)	Neonatal Mortality TSLS (3)	Infant Mortality OLS (4)	Infant Mortality OLS (5)	Infant Mortality TSLS (6)
Constant	-17.427	-17.106	-29.100	-17663	-17.301	-32.912
	(-6.51)	(-3.57)	(-2.91)	(-6.51)	(-3.49)	(-3.16)
Ln Cigarettes[b]	0.746	0.609	1.017	0.696	0.575	0.974
	(2.39)	(1.87)	(1.90)	(2.20)	(1.75)	(1.78)
Ln Liquor[b]	0.879	0.917	1.059	0.882	0.903	0.982
	(7.66)	(6.39)	(4.02)	(7.57)	(6.16)	(3.58)
Ln Protein[b]	5.348	5.660	10.003	5.490	5.686	9.835
	(6.04)	(3.44)	(3.36)	6.08)	(3.39)	(3.22)
Ln Calcium[b]	-7.918	-6.544	-7.143	-8.158	-6.743	-6.507
	(-6.04)	(-4.08)	(-2.20)	(-6.10)	(-4.11)	(-1.94)
Ln Iron[b]	----	-1.458	-6.215	----	-1.351	-6.397
	----	(-0.87)	(-1.91)	----	(-0.79)	(-1.90)
Ln Vitamin A[b]	4.652	4.305	5.470	4.811	4.478	5.932
	(6.09)	(4.91)	(3.16)	(6.18)	(4.98)	(3.32)
Ln Vitamin C[b]	----	-0.209	-0.384	----	-0.240	-0.931
	----	(-0.36)	(-0.28)	----	(-0.40)	(-0.65)
Ln Midwife[b]	----	0.131	0.258	----	0.152	0.333
	----	(1.96)	(2.18)	----	(2.24)	(2.69)
Ln Abortion[b]	----	0.151	0.404	----	0.118	0.252
	----	(1.24)	(1.54)	----	(0.96)	(0.95)
Ln Hospital Beds	-0.578	-0.674	-0.659	-0.511	-0.589	-0.498
	(-3.90)	(-4.09)	(-3.10)	(-3.43)	(-3.59)	(-2.34)
Ln Mother's Age	----	0.010	-0.321	----	0.008	-0.395
	----	(0.07)	(-1.37)	----	(0.05)	(-1.58)
Ln Sulfur Dioxide	0.463	0.384	0.022	0.432	0.358	0.032
	(2.97)	(2.34)	(0.09)	(2.75)	(2.17)	(0.13)
Ln Particulates	0.678	0.674	0.555	0.765	0.746	0.623
	(3.49)	(3.29)	(2.11)	(3.84)	(3.54)	(2.30)
R^2	0.857	0.873	0.832	0.852	0.869	0.827
F	63.56	42.22	30.49	61.03	41.00	29.35
Wu Test F			2.723			3.444

[a] Ln stands for natural logarithm. All estimates are based on a sample of 94 prefectures. Asymptotic t-ratios are shown in parentheses. The critical asymptotic t-ratios are: 1.28 for a one-tailed test and 1.64 for a two-tailed test at the 10 percent level; 1.64 for a one-tailed test and 1.96 for a two-tailed test at the 5 percent level; and 2.33 for a one-tailed test and 2.58 for a two-tailed test at the 1 percent level. The F statistic for each equation is significant at the 1 percent level.

[b] Endogenous in the Two-Stage Least Squares equations.

Comparing the estimates obtained using two-stage least squares with those obtained using ordinary least squares, one can see that the coefficients obtained using TSLS are often substantially larger than the corresponding OLS estimates. Also, the Wu test rejects the null hypothesis of zero correlation between the endogenous health inputs and the residuals at the one percent significance level, suggesting, as Rosenzweig and Schultz (1983a, page 42) conclude, that "parents do respond to differences in their genetic and/or environmental health endowments."[11] In particular, women with poor unobserved health endowments are more aware of hazardous health inputs (such as cigarette smoking and alcohol consumption) during pregnancy than those with better endowments, leading them to consume less of these inputs and, hence, to reduce the risk of a poor birth outcome. In elasticity terms, the effects of cigarette smoking on neonatal and infant mortality range from 0.61 to 1.01 and from 0.57 to 0.97 respectively.[12] The comparable elasticities for liquor consumption

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[11] This result is consistent with the results of studies using U.S. data, including those of Rosenzweig and Schultz (1982, 1983a, and 1983b); Corman, Joyce, and Grossman (1987); Joyce (1987); Joyce, Grossman, and Goldman (forthcoming).

[12] The estimated elasticities are obtained by multiplying (1-P) by the estimated coefficients of the logit neonatal and infant mortality production functions. (1-P) is equal to 0.9953 for the neonatal elasticities and is equal to 0.9929 for the infant elasticities.

Our estimated elasticities using Japanese data are somewhat larger than the estimates using U.S. data. For example, the cigarettes elasticity of neonatal mortality for whites in the U.S. is estimated to be in the range 0.41 to 0.51 (Corman, Joyce, and Grossman 1987; Joyce 1987; and Joyce, Grossman, and Goldman forthcoming).

fall in the range 0.87 to 1.05 for neonatal mortality and in the range 0.88 to 0.98 for infant mortality.

All of the coefficients on the nutrients, with the exception of those on vitamin C, are statistically significant in the TSLS regressions. An increase in the mother's consumption of calcium and iron during pregnancy reduces the probabilities of neonatal and infant mortality. However, increases in the mother's consumption of protein and vitamin A during pregnancy lead to adverse effects on infant health.[13]

Of particular interest in this research are the effects of changes in food prices on neonatal and infant mortality, specifically those foods which are the main sources of the nutrients we consider. The price elasticities of selected nutrients and health price elasticites for neonatal and infant mortality are reported in Table 3.[14]

A negative nutrient price elasticity implies than an uncompensated increase in the price of that food will result in a decrease in the consumption of a particular nutrient. A row of negative nutrient price elasticities suggests that an increase in the price of that food will result in decreased consumption of

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[13] While these results are somewhat surprising, there is some evidence of the adverse effects of excessive consumption of these nutrients on infant health (Worthington-Roberts and Vermeersch 1985, pages 91-95 and 105).

[14] The nutrient price elasticities are calculated using equation (12) and the results obtained from the seemingly unrelated equations estimation procedure. The health price elasticities are obtained by applying equation (11) using the estimates obtained from TSLS.

Table 3
Nutrient Price Elasticities and Health Price Elasticities

Price of Variable	Nutrients					Neonatal Mortality	Infant Mortality
	Protein	Calcium	Iron	Vitamin A	Vitamin C		
Milk	-0.326	-0.863	-0.433	-0.721	-0.999	2.024	1.819
Meat	0.162	-0.010	0.139	-0.081	-0.020	0.390	0.305

each of the nutrients. For example, an increase in the price of milk leads to a reduction in the consumption of all nutrients and, consequently, increases the probability of neonatal and infant mortality. The values of the health price elasticity of neonatal and infant mortality with respect to the price of milk are 2.024 and 1.819, respectively. That is, a one percent rise in the price of milk induces about a two percent rise in the probabilities of neonatal and infant mortality. Alternatively, an increase in the price of meat does not decrease the consumption of all nutrients due to substitution effects among the various foods. However, an increase in the price of meat also increase the probabilities of neonatal and infant mortality, as seen by their estimated health elasticities of 0.390 and 0.305, respectively. These results imply that a weakening of the current strong restrictions on the importing of dairy products and meat would result in an improvement in infant health.

Among the medical care inputs, we find that a positive and significant relationship exists between midwife deliveries and both mortality rates, with the coefficients obtained in the TSLS regressions about twice those obtained in the OLS regressions.

These results indicate that women with poor unobserved health endowments are likely to seek the care of medical doctors rather than midwives and, consequently, they reduce their risk of neonatal and infant mortality.

Contrary to U.S. findings (for example, Corman and Grossman, 1985; Corman, Joyce, and Grossman, 1987; and Joyce, 1987), the coefficient on the abortion variable is positive, albeit never significant. The persistent positive sign may indicate that abortion is used in Japan in place of contraception or to remedy contraceptive failure rather than to prevent high risk births, as suggested by Preston (1986).

The coefficient on the hospital bed variable, our proxy for the availability of pre- and postnatal care, is consistently and significantly negative in all equations, as expected. Greater availability of these services reduces market prices for this care, inducing women to initiate care earlier in their pregnancy as well as after birth. The hospital bed elasticity is in the range from -0.58 to -0.67 for neonatal mortality, and varies from -0.50 to -0.58 for infant mortality.

Finally, we find a strong positive relationship between air pollutant levels and both neonatal and infant mortality. Although sulfur dioxide has the largest impact on birth outcomes in the U.S., we find that particulates are more important in Japan. The particulates elasticity of neonatal mortality ranges from 0.55 o 0.67, with the corresponding elasticity of infant mortality in the range 0.62 to0.76. The sulfur dioxide elas-

ticities of neonatal and infant mortality lie in the ranges 0.38 to 0.46 and 0.36 to 0.43, respectively.[15] Thus, the impact of particulates on infant health is about twice as large as that of sulfur dioxide.

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[15] These elasticities are much greater than the corresponding elasticities found in the U.S. For example, Joyce, Grossman, and Goldman (forthcoming) find a sulfur dioxide elasticity of neonatal mortality for whites in the range from 0.02 to 0.08. Moreover, they never find a significant relationship between particulates and both the neonatal and infant mortality rates, possibly due to collinearity between the five pollutant measures they include in their neonatal mortality production functions.

V. CONCLUSIONS

The recent empirical health economic studies of U.S. infant and neonatal mortality rates, as well as those of low birth weight, have overlooked the importance of the mother's nutritional status as a determinant of infant health. These studies emphasize medical care, smoking, drinking and residence location as determinants of infant health. While using a theoretical and methodological approach similar to these studies, our model emphasizes the role of the mother's nutritional status in determining neonatal and infant mortality rates.

Using cross-sectional market averages for 1980 and 1981 in Japan, we find that cigarette smoking, alcohol consumption, and environmental quality all have strongly adverse effects on newborn survival outcomes in Japan, as in the U.S. Additionally, we see that women who initiate care earlier in their pregnancy as well as in the postnatal period reduce their risk of neonatal and infant mortality. Unlike in the U.S., we discover that the availability of abortion increases, although never significantly, neonatal and infant mortality rates, possibly due to its use in place of contraception. Finally, we note that the nutrient intake of the mother during pregnancy is a potential determinant of neonatal and infant mortality in Japan, with increased consumption of calcium and iron leading to improved birth outcomes. Somewhat surprisingly, we find that increased consumption of protein and vitamin A lead to higher neonatal and infant

mortality rates. Using the results obtained from the estimation of neonatal and infant mortality production functions (in addition to those obtained from the estimation of a system of demand equations), we conclude that increases in the prices of food items, in particular milk and meat, would lead to increases in neonatal and infant mortality rates. This finding indicates that less restrictive import measures in Japan, particularly on dairy products and meats, would reduce neonatal and infant mortality rates below their already relatively low levels.

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