Welfare State and Infant Mortality[1]

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This article seeks to understand the effects of welfare-state spending on infant mortality rates. Infant mortality was chosen for its importance as a social indicator and its putative sensitivity to state action over a short time span. Country fixed-effects models are used to determine that public health spending does have a significant impact in lowering infant mortality rates, net of other factors, such as economic development, and that this effect is cumulative over a five-year time span. A net effect of health spending is also found, even when controlling for the level of spending in the year after which the outcome is measured (to account for spurious effects or reverse causation). State spending affects infant mortality both through social mechanisms and through medical ones. This article also shows that the impact of state spending may vary by the institutional structure of the welfare state. Finally, this study tests for structural breaks in the relationship between health spending and infant mortality and finds none over this time period.

The welfare state has been an important and growing area of research for political scientists, economists, and sociologists over the last several decades. Most of this research investigates the welfare state as a dependent variable utilizing multiple factors to explain or predict welfare-state structure and spending (Hicks 1999; Hicks and Kenworthy 1998; Hicks and Misra 1993; Hicks, Misra, and Ng 1995; Hicks and Swank 1984, 1992;

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Huber, Ragin, and Stephens 1993; Pampel and Williamson 1993; Skocpol and Amenta 1986; Stephens 1979). Some political sociology has extended this welfare-state research by exploring the welfare state as an independent variable. Many of these studies examine welfare-state performance in reducing income inequality (see, e.g., Headey et al. 1997; Hicks and Swank 1984; McCrate 1997; Korpi and Palme 1998). Studies using a tripartite or five-category welfare-state model have come to a general consensus that social-democratic regimes, in which the state plays the greatest role in income redistribution, lead to the most equality, whereas the liberal (or residual) welfare states produce the least equality (Headey et al. 1997; Korpi and Palme 1998).[2] This is despite the fact that residual welfare states typically pursue policies that are more targeted to helping the poor. This is often called the "paradox of redistribution strategies" (Headey et al. 1997; Korpi and Palme 1998).

One reason why the income distribution may be the dependent variable of choice for studies of the welfare state is its putative sensitivity over a short time frame to state policy decisions. If increased benefit levels go into effect or if lower marginal tax rates are adopted, they should have an almost immediate impact on relative income shares. By contrast, in determining whether educational expenditures have an impact on test scores or educational attainment, for example, what is the proper time lag for the return on government investment—one year, five years, or 20 years? The same question clouds analysis of the effects of state health investments on overall mortality rates or life expectancies.

However, while income distributions offer a potential solution to the issue of time lags and no doubt affect the mental and physical well-being of citizens, particularly the poorest members of society, the income distribution does not tell us about the overall well-being of a citizenry. In this sense, income is not a social or developmental outcome, but rather a mediating variable. In other words, two countries could have very different income distributions but have similar scores on important social indicators such as life expectancy, literacy, and so on. For example, if a country has universal, high-quality health care, how shares of national income are distributed may not matter as much to the health of the population. Rather than rely on income as the dependent variable, the current study attempts to push the research on welfare-state effects in another direction by modeling the impact of welfare-state spending on a human, developmental outcome: infant mortality. Infant mortality was picked for two reasons, the first being its putative sensitivity over a short time frame to state investments in medical care and public health. By

[2] Interestingly, the United States is an outlier with respect to its income distribution, even among the residual welfare states.

removing the time lag often involved in studying welfare spending and other health or developmental outcomes (like life expectancies), we can be more confident that a relationship is not due to confounding effects.

Second, infant mortality is deployed as the outcome measure of choice in the current study due to its reputation as a sensitive indication of social development (Judge, Mulligan, and Benzeval 1998; Nersesian 1988). That is, infant mortality is a generally accepted social indicator of a nation's health and quality of life, particularly for the poorest members of society (Anderson 1973; Morris 1979; Nersesian 1988). Infant mortality has declined steadily and consistently during the past several decades with few exceptions (WHO [World Health Organization] 1999; Van Der Gaag and Barham 1998).[3] Among countries in the Organisation for Economic Cooperation and Development (OECD), the average infant mortality rate (IMR) in 1960 was 26.5 deaths per 1,000 live births. By 1992, this figure had dropped to 6.8 deaths per 1,000 live births. As might be expected, developed nations have a much lower IMR than developing countries (Nersesian 1988; WHO 1999).

A few others have gone before us in attempting to model the impact of the welfare state on health in general and infant mortality in particular (Cochrane, Leger, and Moore 1978; Judge et al. 1998; Le Grand 1987; Pampel and Pillai 1986). These studies have come to mixed conclusions.[4] An important issue that plagues this welfare states literature and which may contribute to the mixed findings is the question of unobserved heterogeneity. That is, how do we know that it is the impact of government expenditures that matters and not some other, unmeasured variable that correlates with the outcome as well as with cross-national variation in spending levels? Since the vast majority of welfare-state research uses cross-sectional regression or pooled time series approaches, it is not clear whether the findings are the result of omitted variable bias. In other words, does Sweden have a lower IMR because of its generous welfare state, or do the same societal characteristics that give rise to the welfare state also tend to generate lower infant mortality rates? These nation-specific characteristics have previously been linked with welfare-state spending (Pampel and Pillai 1986). At the same time, these factors affect national health in general and infant mortality in particular (Judge et al. 1998; Pampel and Pillai 1986).

[3] For example Uganda, Mozambique, Zambia, and Ethiopia have experienced periodic increases in infant mortality during the past several decades due to periods of unrest in the country.

[4] For example, evidence on the association between welfare spending and health outcomes is less clear when income inequality is controlled for (Le Grand 1987; Judge et al. 1998; Pampel and Pillai 1986). These studies will be reviewed in greater detail later in the literature review.

In addition to cross-national estimation concerns, there are longitudinal issues. Specifically, one needs to be able to discern whether or not the dramatic reductions over the course of a generation reflect the triumph of the welfare state—that is, the returns to health and human services investments made by governments. Alternatively, this downward trend in infant death rates could reflect a corresponding increase in standards of living—that is, economic development. Or it could be neither—merely a result of other forces such as declines in fertility or technological advances that may not be easily captured in direct measures. Evidence can be marshaled for a variety of causal accounts. For example, in 1960, the average per capita expenditures on health by OECD governments was $163 (expressed in constant, 1985 U.S. dollars) or 6.1% of GDP. By 1992, that figure had risen to $896 (8.9% of GDP). At the same time, among the OECD countries, per capita GDP was $6,434 in 1960; by 1992, it was $14,050, indicating substantial economic development.

These simultaneous trends are shown graphically in figure 1. Infant mortality in these countries has been steadily declining over the period between 1960 and 1992. At the same time, total social spending on the part of these governments has steadily increased, as have health-related expenditures and per capita GDP. Getting the right causal story, then, is not an easy task—it requires a variety of approaches. This article investigates these trends, asking what role the rise of the welfare state has played in the decline in IMR and whether the impact of state expenditures varies by welfare-state structure. In this manner, this study attempts to use infant mortality as a heuristic to investigate broader issues concerning the welfare state. Before addressing the specific research questions we hope to answer using this heuristic, we briefly summarize the research literature on the social predictors of infant mortality, indicating what our study will add to this knowledge base.

SOCIAL PREDICTORS OF INFANT MORTALITY

Much past and present research demonstrates that infant mortality is affected by a range of distal and proximate determinants. In particular, research has focused on the relationship between infant mortality and individual-level social, behavioral, and demographic factors, including race (Cramer 1987, 1995; Hummer et al. 1999; Nersesian 1988; Singh and Yu 1995; Wise et al. 1985), socioeconomic status (Nersesian 1988), maternal education (Caldwell 1979; Flegg 1982; Pampel and Pillai 1986; Shoham-Yakubovich and Barell 1988), smoking during pregnancy (English and Eskenazi 1992), maternal age (Raymond, Cnattingius, and Kiely

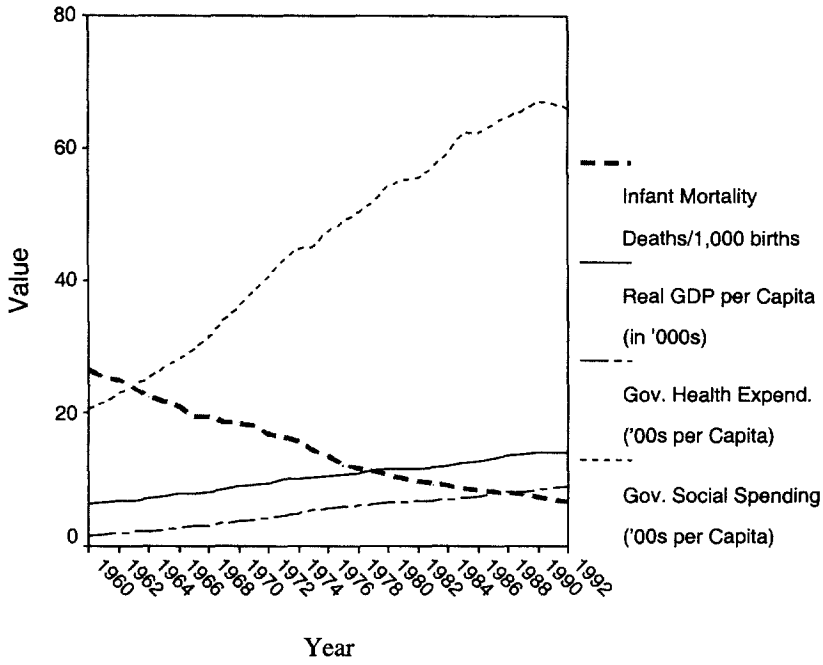

FIG. 1.—Trend in infant mortality, per capita GDP, per capita health expenditures, per capita social spending, and trade as a percentage of GDP. OECD countries, 1960–92.

1994),[5] and nativity/immigrant status (Hummer et al. 1999). However these sociodemographic factors are generally believed to have an indirect affect on IM mediated through individual-level variables located primarily in the biological domain (Eberstein, Nam, and Hummer 1990; Hummer et al. 1999).

Birth weight is perhaps the most recognized determinant of IM and has been shown to act as an intervening variable between most sociodemographic factors and IM (McCormick 1985; Institute of Medicine 1985).[6] Low birth weight (LBW) is defined as weighing less than 2,500 grams, and the effect of LBW can be seen by looking at some U.S. statistics relevant to time frame of the study (McCormick 1985; Shapiro et al. 1980; Wise, Wampler, and Barfield 1995). For example, Shaprio et al. (1980) found that LBW babies (less than 2,500 grams) had a neonatal mortality level of approximately 20/1,000 and very LBW babies (less than 1,500

[5] Some authors include maternal age as having a direct affect on the IMR, but there seems to be more evidence positioning it as having an indirect affect.

[6] Other direct predictors of IMR include prior fetal or infant deaths, birth order of the infant, and timing of prenatal care (see, e.g., Eberstein, Nam, and Hummer 1990).

grams) had a neonatal mortality level of almost 100/1,000, compared to the lowest level of 2/1,000 for babies weighing about 3,500 grams.

There are also factors that affect IMR but do not cause or prevent LBW. Advances in health-service technology is one such example. Although technology advances have significantly reduced IMR and are not mediated by LBW, they have served to shift the concentration of infant mortality to very LBW babies (Gortmaker and Wise 1997). Also, it is important to note that technology does not decrease unequal birth outcomes among social groups (such as by race or income level). As Gortmaker and Wise (1997) note, "the causes of the decline in infant mortality rates—intensive care technology—are different from the effects of social and economic factors that still produce disparate outcomes" (p. 155).

In addition to exploring individual-level determinants, macrolevel studies have explored links between country-level contextual variables and IMR (Boehmer and Williamson 1996; Judge et al. 1998; Pampel and Pillai 1986; Shen and Williamson 1997). These studies have found that there are important differences between factors affecting IMR in less developed countries compared to factors affecting more developed countries. In particular, some of the most significant predictors of IMR in less developed countries that do not usually cause IMR variations in more developed countries include sex of the infant (Mason 1993) and sanitation (Hertz, Herbert, and Landon 1994). Several country-level factors that influence IMR in more and less developed countries include the fertility rate (Pinnelli 1993), legal abortion rate (Puffer 1993), state health spending (Judge et al. 1998; Pampel and Pillai 1986), GDP (Flegg 1982), income inequality (Judge et al. 1998; Pampel and Pillai 1986; Rogers 1979; Flegg 1982; Waldmann 1992), and unemployment rates (Brenner 1983; Wennemo 1993).

STATE HEALTH SPENDING AND INFANT MORTALITY

Because the focus of this study is on state health spending and infant mortality rates in developed countries, we want to focus on the handful of studies that have conducted previous research on this topic. As a general statement, these studies provide mixed evidence, at best, that state health care spending in developed countries negatively influences (lowers) IMR (Babazono and Hillman 1994; Cochrane et al. 1978; Judge et al. 1998; Pampel and Pillai 1986; Woolhandler and Himmelstein 1985). When univariate correlations between IMR and health care spending are presented in these papers, they are in the expected direction (negative) and often significant. However, once multivariate methods are employed, the impact of a broad measure of government health care spending on IMR is only

significant in one study (Pampel and Pillai 1986) and in this case only for the impact of government medical expenditures on neonatal mortality rate (death from zero to 28 days after birth). The reason for insignificant and sometimes contradictory findings could be due to several factors, including (1) differing definitions of health care spending, (2) controlling for different variables, (3) varying time frames including cross section and over time analysis, (4) inclusion of different countries, and (5) a variety of methodologies, none of which included country fixed-effects models. In order to better evaluate the differences, strengths, and weaknesses of several main foundational studies on the topic, we are going to explicate each one further.

One important study was conducted by Judge, Mulligan, and Benzeval in 1998. They examined cross-national comparisons of both life expectancy and infant mortality. They included 15 countries, and data were derived from a variety of sources, including the Luxemburg Income Study (for income data), the World Bank (for health data), and the OECD (for other determinants of population health). They included an income-inequality variable that indicates the share of income available to the bottom 60% of the distribution when predicting IMR. Welfare-state spending was operationalized as social security transfers expressed as a percentage of GDP. Health expenditures were defined as the total health spending (including both public and private) as a percentage of GDP. The authors also control for the percentage of women in the total workforce.

Using OLS models, Judge et al. (1998) found that income inequality remained significant when GDP, health expenditures, and welfare-state spending were added. Income inequality only lost significance with the inclusion of the female labor force variable. Judge concludes that his data support theories that "women's social development is a determinant of reductions in levels of infant mortality" (p. 577). These results do not provide support for the theory that increased state health spending decreases IMR. There are several potential factors that may have contributed to Judge's nonsignificant welfare-state findings, including the impact of country-specific factors. Judge explains, "We also believe that a nation's health is likely to be the produce of a wide range of cultural, economic and social factors, many of which are not easily measured and most of which might interact with each other" (p. 578). This suggests that employing fixed-effects models may allow us to detect a significant effect of state health spending if it was found to be insignificant due to the spurious effects of country-specific factors.

Pampel and Pillai (1986) utilized longitudinal and cross-sectional data to explore cross-national variation in infant mortality. They examined data for the 18 countries classified by the World Bank as advanced industrial-market-economy nations. Data for each nation were collected at

five-year intervals between 1950 and 1975 and were pooled with cross-sectional data resulting in a sample size of 108 cases. Complete data on some of the medical and welfare-state variables were not available for all countries, and therefore the analyses including these indicators were conducted with a sample of 96 cases.

Pampel and Pillai utilized data from a multitude of sources and incorporated a variety of factors that they posited would affect infant mortality. These include economic development (GDP, unemployment rates, and urbanization), fertility (overall and teenage), education of women, medical and health care system (number of physicians, hospital beds, and nurses), income inequality (Gini coefficient), ethnic diversity, and welfare-state spending. Welfare-state spending was broken into three variables, all included as a percentage of the GNP: (1) government medical expenditures, (2) government public health expenditures, and (3) total government benefit expenditures for social welfare and social security. This total welfare spending includes medical and public health spending, as well as pensions, disability, unemployment, family allowances, and public assistance.

The authors used three measures of infant mortality: infant mortality rate (IMR), neonatal mortality rate (NMR), and postneonatal mortality rate (PNMR). IMR is the number of deaths to infants from birth to one year old, NMR is the number of deaths from zero to 28 days, and PNMR is the number of deaths from 29 days to one year. All of these rates have the denominator of 1,000 live births.

Through utilizing stepwise modified generalized least squares estimates, the authors found that female education and unemployment were consistently important indictors of IMR, PNMR, and NMR. GNP, number of physicians, percentage of population living in urban areas, welfare spending, and ethnic diversity were often important predictors. Public medical benefits (and less so, public health expenditures) were significantly associated with a decrease in NMR after controlling for all other variables (except general social spending, which was not included in the same model as public medical benefits). Total government spending was significantly and negatively associated with IMR, PNMR, and NMR until ethnic diversity was included—when its significant (and negative) association only remained for PNMR.

The remaining studies are not as applicable. In 1985, Woolhandler and Himmelstein conducted a study to explore the impact of military spending on infant mortality rates. They used 1972 and 1979 data for 132 countries to generate univariate correlations of IMR and %change in the variable between 1972 and 1979. They also conducted stepwise multiple linear regression for 141 countries based on the 1979 data. In addition to their independent variable of focus, they included a variety of control factors,

including GNP, government education spending, population per teacher, government health spending, population per physician, population per nurse in 1976, caloric consumption, percentage of population with access to clean water, and population density. Although they make some comparative statements in the discussion section about how militarism affected IMR differently in more developed countries versus less developed countries, the tables and results are presented for all countries together, and it is not possible to tease out how health care spending affects IMR in developed countries. Their multivariate model finds that only military expenditures, population per teacher, caloric consumption, and percentage of population with access to clean water are significant. The significance of variables most directly affecting less developed countries may indicate that the model is greatly influenced by these less developed countries and might not be indicative of what we would expect to find in developed countries.

Babazono and Hillman (1994) conducted a cross-sectional analysis of health outcomes and health care spending for 21 OECD countries using primarily 1988 data. They employed multiple linear regression to predict infant mortality and perinatal mortality using a variety of predictive variables, including total health care spending, number of in-patient beds per thousand, average length of hospital stay, physician contact, non–health care spending, public health spending, number of admissions, number of physicians per thousand people, and pharmaceutical expenditures. Babazono and Hillman (1994) found that only number of inpatient beds per thousand and non–health care spending per capita were significantly (and inversely) associated with both IMR and perinatal mortality. It is important to point out that they do not control for any economic development variables such as GDP or GNP. Finally, Cochrane, St. Leger, and Moore (1978) found that the percentage of health care provided by public funds was not a significant predictor of infant mortality or perinatal mortality using OLS regression for 1970 data from 18 developed countries.

CONCEPTUAL MODEL

In order to model infant mortality and welfare-state spending, we have developed a framework that conceptualizes microlevel as well as macrolevel relationships. The model (fig. 2) shows that we are looking at how state investment in health affects (1) the individual social demographic factors mediated by LBW and (2) access to medical services that are not necessarily directly related to LBW. Because the focus of the study is on health care spending and IMR, our model explicates this relationship. However, we understand the importance of the other country-level factors

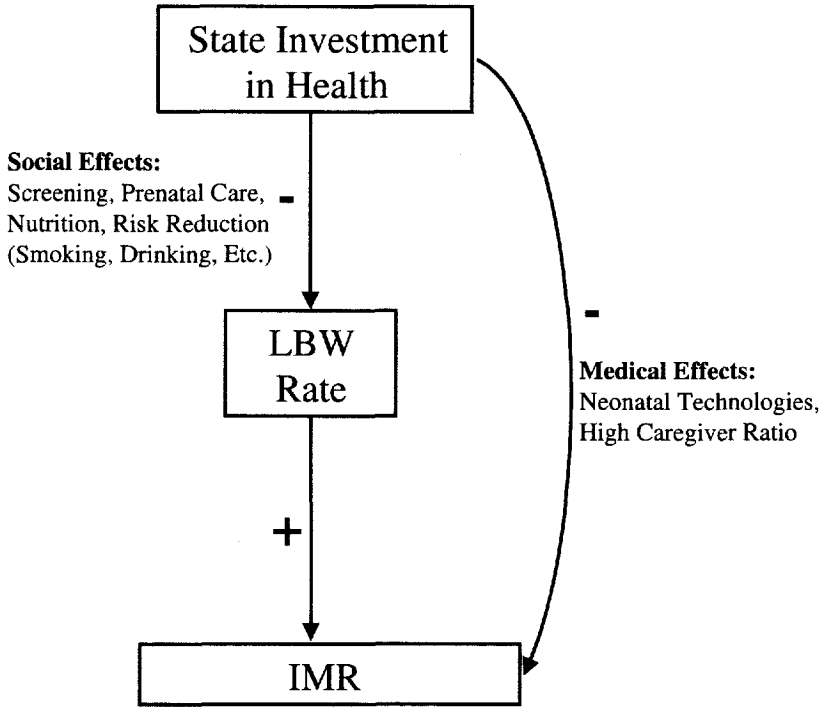

FIG. 2.—Model for the effects of state health spending on infant mortality rates

and therefore have controlled for several of them, including fertility, unemployment, and GDP. We now turn to the specific questions we hope to answer.

Do government expenditures on health affect the infant mortality rate, net of country-specific factors? If so, how—through social or medical mechanisms? Since the upward trend in spending and per capita GDP map onto each other fairly well for the entire time period under study, this becomes a critical issue to sort out in order to make claims about the differential roles of the state and of economic development (vs. other, unmeasured factors, such as technological progress that is not captured by economic-development measures). While it may appear straightforward that increased government health spending would result in better infant health outcomes, on second blush, it is not entirely obvious. After all, we are talking about countries—the United States excepted—that all had universal health coverage by the end of the study period. So, at least in theory, the entire population has access to the medical services it needs.

In such a situation, higher health expenditures may merely reflect medical costs outpacing inflation indices.

How government investments influence infant health is another important and related question. If the effect of government investments in the health care system is most evident in lowering the IMR indirectly—through lower rates of LBW—then this suggests that the impact is largely on the social front, for example, in improving prenatal nutrition and care, lowering maternal risk factors such as drinking and smoking, and so on. However, if (net of LBW rates) the government has a large impact in lowering infant mortality rates, then it implies that health spending primarily yields returns on the medical side, for example, in better access to care for infants, higher nurse-to-patient ratios in neonatal intensive care units, better neonatal technologies, and so on.

Another issue with respect to the way government investments yield returns is in the time lag involved. As mentioned earlier, this issue has perhaps prevented some comparative-welfare-state researchers in the past from examining more "human" or developmental outcomes and instead helped focus work on economic measures. By contrast, in this study, we want to tackle the time lag issue head on by asking what the proper time frame is for measuring the impact of government spending on infant health and survivorship. We will show models with a single-year measure of government spending, a three-year measure, and a five-year measure. Cumulative returns over the course of several years can be explained in two ways. First, it may be the case that many types of health investments are of a "capital" nature; building more prenatal care clinics or hospitals or investing in the latest neonatal medical technology should have benefits for more than one year. Second, some theories suggest that a mother's health long before she is pregnant is an important determinant of infant morbidity and mortality. Better health care for future mothers in one year may, then, yield better health outcomes for their as-of-yet unborn children four years later.

Does the structure of the welfare state affect the returns to government expenditures on health? We have seen that more universalistic approaches to income redistribution appear to be more effective in reducing income inequality when compared to more targeted strategies (Headey et al. 1997; Korpi and Palme 1998). Whether the same results hold true for infant mortality remains to be seen, however. In other words, does a dollar (or a euro) buy the same health benefits regardless of the structure of the welfare system? Or do the returns to public investment depend on whether benefits are a social right of citizenship (as they are in universalistic welfare regimes like Sweden) versus targeted to means-tested groups (as they are in residual welfare states such as the United States)? Targeting at-risk populations through means testing might be more effective for health

promotion than for income equalization, or this might vary to the extent that public health investments are public goods.

To the extent that investment in hospitals, health education, scientific research, and other medical or public health measures is a public good (in the specific sense that it is not divisible), whether or not health expenditures are provided within a means-tested welfare structure or not may not have a major impact on their effectiveness at reducing the IMR. For example, the largest portion of the U.S. federal government's spending on health is for Medicare and is directed at the elderly—a generally nonfertile population. However, to the extent that hospitals cross-subsidize (i.e., use money they get from Medicare to support services for poor mothers and infants, e.g., or to keep the hospital running more generally), Medicare dollars may reduce infant mortality rates. Likewise, prenatal education programs may affect not only program participants but also their neighbors who indirectly learn about health-promoting practices. In the same vein, the benefits of cleaner water and air cannot be as easily apportioned by means testing or targeted funding. To the extent that health expenditures are provided as private, divisible goods—for example, giving families vouchers for pre- or postnatal exams or providing intensive care services to specific high-risk infants—their effects may be more sensitive to either universal or means-tested approaches.

When we run models separately by state-welfare structure, we rely on the now-conventional approach of Esping-Anderson (1990). As mentioned in the introduction, Esping-Anderson uses a three-category approach: liberal (or residual), corporatist, and social-democratic (or universal) welfare regimes. (For a full discussion of these, see Esping-Anderson [1990].) For our purposes, we use only those countries that score highly on one of these three categories. Using this logic, the liberal regimes are Australia, Canada, Japan, Switzerland, and the United States. Corporatist regimes are Austria, Belgium, France, Germany, and Italy. Social-democratic ones are the Netherlands, Denmark, Norway, Finland, and Sweden. Those that were not classified into one distinct category by Esping-Anderson and are therefore dropped from this portion of the analysis include Ireland, Luxembourg, New Zealand, and the United Kingdom.

When we examine the trends of infant mortality by welfare-regime type in figure 3, we uncover a phenomenon of convergence. At the beginning of the time period, the corporatist welfare regimes had substantially higher rates of infant mortality than the liberal regimes, which in turn suffered from higher rates than the social-democratic countries. However, by the end of the period (1992), the IMRs were much more similar. This does not tell us necessarily that the structure of the welfare state does not matter; it could be the case that this convergence was achieved through different means in different country clusters. For example, infant mortality

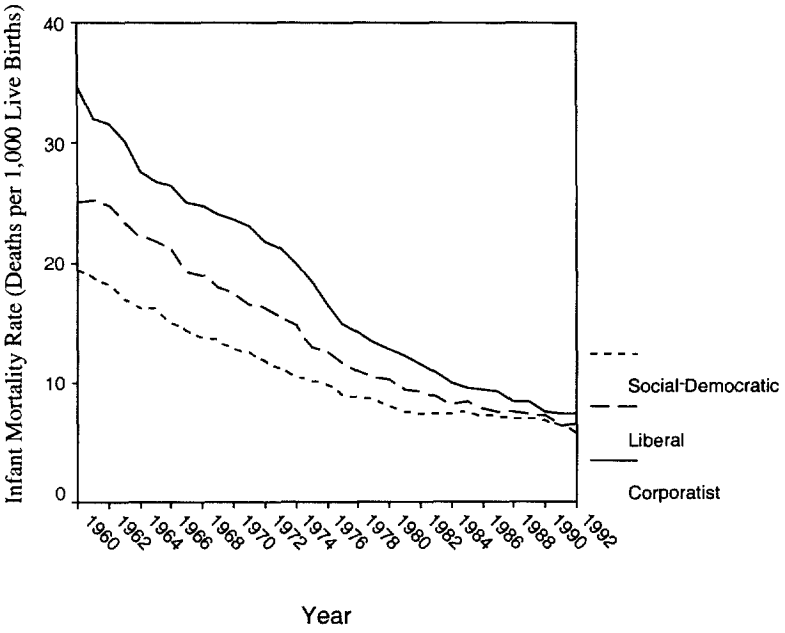

FIG. 3.—Infant mortality rates by welfare regime type, 1960–92

reductions could be the result of a stronger social safety net in social-democratic regimes versus the result of greater investment in health care technology in liberal regimes. It could also reflect changes that affect all countries without regard to regime type—for example, advances in medical techniques.

The structure of the health care system is another important welfare-state consideration. In order to examine this issue, we pursued two tracks. First, we ran all countries except the United States (the only one without universal health care by the end of the time period). Results with the United States excluded were the same (see the appendix, below). Running the United States alone showed similar health spending effects as those in the other countries using a reduced form model (which excluded LBW).[7]

The second approach we use is to examine the case of Canada, where the change to a publicly based health care system occurred during the period covered by this study. This provides a unique opportunity to assess

[7] However, when we run analysis of LBW or of infant mortality using LBW, the effects for the United States are not significant (though the coefficients have the same signs and magnitude). This is due to the fact that LBW information was available only between 1975 and 1992 for the United States, and thus the N is effectively reduced to 18.

how the structure of the health care system itself affects the relationship between expenditures and health outcomes.[8] While the process was a gradual one, it was not until 1972 that all the provincial and territorial plans had been extended to include physicians' services. This year, then, marks the beginning of a system of universal coverage through national health insurance for both hospital and medical care in all of Canada, leaving the United States as the lone OECD country without universal coverage.[9] To assess the impact of this change, we estimate models for a structural break in the impact of health care and welfare spending on entire pooled time series and on the Canadian data in particular. This is done by estimating the base model with the addition of a dummy variable that receives a score of "1" if the data point falls on that year or afterward and a "0" if it precedes that year. This variable picks up a mean shift in the IMR net of measured characteristics. In addition to this indicator variable, we add to the model interaction terms, which consist of this dummy variable multiplied by other variables of interest—in our case, health care spending levels. The year in question is then allowed to vary across the entire time series, and the resultant series of models are plotted, examined for unique troughs, and evaluated using any of a number of possible comparative approaches (such as BIC statistics, Chow test, or χ^2 tests from the base model). (For examples of this approach elsewhere, see Andrews [1993]; Western and Healy [1998]; or Piehl et al. [1999].) We contrast the search for a structural break in Canada with the possibility of a structural break in the entire set of countries.

Finally, the "welfare state" is a lot more than just social insurance and health care. It is constituted of a rich array of laws and policies. Here we test for the importance of one particular change in the state: the legali-

[8] Most other countries established universal health care before the start of our time series. The exceptions—besides Canada and the United States—include Japan (1961), Sweden (1962), Luxembourg (1964), Italy (1980), and Australia (1984). However, there are data limitations for these countries in that they have limited or no data prior to their respective dates listed above.

[9] The Canadian government summarizes the history of its health care system up to 1972: "The trend to universal, publicly financed health insurance began in 1947 when the province of Saskatchewan introduced a public insurance plan for hospital services. In 1956, the federal government, seeking to encourage the development of hospital insurance programs in all provinces, offered to cost-share hospital and diagnostic services on a roughly fifty-fifty basis. By 1961, all ten provinces and the two territories had signed agreements establishing public insurance plans that provided universal coverage for at least in-patient hospital care that qualified for federal cost-sharing. Public medical care insurance also began in the province of Saskatchewan, providing coverage for visits to, and services provided by, physicians outside hospitals. The federal government enacted medical care legislation in 1968 to cost-share, again on a roughly fifty-fifty basis, the costs of provincial medical care services." (Health Canada 2000, on-line documentation: http://www.hc-sc.gc.ca/datapcb/datahesa/E_sys.htm).

zation of abortion in 1973 in the entire United States. Though some states had legalized abortion prior to this date (Donohue and Levitt 1999), *Roe v. Wade* represented a legal watershed that changed abortion laws in the majority of U.S. states. The relationship between health spending, abortion, and infant morbidity and mortality may be a complex one. However, there is ample reason to suspect that there may have been some change in the way that state spending influenced infant risks at or around this time. Therefore, we test for a structural break in the U.S. time series using the same approach we employed for Canada and the OECD-wide data.

RESEARCH STRATEGY

In modeling the impact of welfare spending on life outcomes such as infant mortality, there are several presenting problems. Each of these raises important methodological and theoretical issues. We have already alluded to the most important issue of unobserved-variable bias. In order to address this concern, we deploy a country fixed-effects approach to address issues of country-specific unobserved factors that may be driving previously reported results. Country-fixed models represent an effective way of factoring out unobserved differences between nations that may be generating biased effects in OLS or pooled time series GLS regression models. By contrasting the infant mortality rates from different years of the same country, we eliminate most potentially biasing factors. These include the distribution of income and power, ethnic diversity, diet, other culturally based health practices, and the overall health endowment of the populous more generally (to the extent that they are stable over time). In short, this approach factors out anything that is fairly constant within countries over the time period in question. So the effect of GDP per capita, for instance, is only the effect of the transient portion of GDP per capita, the difference between years within a country. As such, fixed-effects coefficients may represent lower-bound estimates of the "true" effect. However, any effect that is presented as significant in this framework has passed a strict test, immune to most concerns of selection bias, unobserved variable bias, and so on, to the extent that these result from between-country factors. (For a fuller discussion of the merits and drawbacks of this methodology, see Griliches [1979].)

This fixed-effects approach has become increasingly popular in sociology as of late as a way to address the problem of unobserved heterogeneity. Even though several researchers have used this approach, it differs significantly enough from OLS approaches to merit some elaboration here (for other examples, see Firebaugh and Beck 1994; Currie and Tho-

mas 1995). The traditional OLS formulation is shown in equation (1) below:

$$\mathbf{Y} = \alpha + \mathbf{X}\beta + \epsilon, \tag{1}$$

where **X** represents the matrix of variables and observations specified in the model, and β, its associated vector of coefficients. However, in the case of social spending and infant mortality, for example, we can be fairly certain there are lurking variables; that is, there is another vector of unobserved characteristics that is biasing our estimates of β, such as the ethnic, cultural, dietary, health, and even genetic endowments of each country. Equation (2) makes this set of variables explicit as the matrix of variables and observations, **NAT**, along with its corresponding vector of coefficients, δ.

$$\mathbf{Y} = \alpha + \mathbf{X}\beta_1 + \mathbf{NAT}\delta + \epsilon'. \tag{2}$$

When we have multiyear data, we can solve this problem, as shown in equation (3), below, by taking difference scores between our **Y** variable at times t_1 and t_2 (say the infant mortality rate) and regressing that against the difference in **X** variables at times t_1 and t_2 (say health care spending). In this equation, the unobserved country-level characteristics that are assumed to remain constant drop out in modeling the difference:

$$\Delta\mathbf{Y}_{t_1,t_2} = \alpha + \Delta\mathbf{X}_{t_1,t_2}\beta_1 + \Delta\mathbf{NAT}_{t_1,t_2}\delta + \Delta\epsilon'_{t_1,t_2}. \tag{3}$$

However, we have not eliminated unobserved characteristics that are unique to the individual country-years or those that vary nonlinearly over time. For example, we cannot eliminate bias in our models that may be due to the fact that there was a viral epidemic one year, a fiscal crisis, and so on. These are year/country-specific, unobserved differences. To the extent that these differences are correlated with our **X** variables, β will be biased. However, the extent to which our **X** variables are causally prior to these unobserved, country/year characteristics, their omission is not as troubling but merely suggests that part of the effects we report may work indirectly through such mechanisms. We attempt to control for two important within-country factors that might otherwise generate bias in our estimates. They are the demographic structure of the country's population and the current state of the economy. There are other variables that may vary within countries over time that we would have liked to control for but were not able to given data limitations. Most notably these include the abortion rate, immigration rate, and the average level of formal schooling for women (the female labor force participation rate was tested as a proxy, was not significant, and was dropped from the final models).

Another related concern in performing this type of research is the issue of aggregation bias (also known as ecological fallacy). When using ag-

gregated data—such as the IMR for a country in a given year—it may be the case that the observed relationship between this outcome and one or more of the independent variables in the model is a statistical artifact of particular dynamics at the individual level. For instance, with respect to the relationship between income inequality and health, there is currently a debate raging about the use of aggregate data to estimate this relationship. (For the view that income inequality is bad for your health, see, e.g., Wilkinson [1996, 1997]; Kennedy, Kawachi, and Prothrow-Stith [1996]; Kennedy et al. [1998].) Critics of the claim that there is a relationship between income inequality and health suggest that such a relationship may be an artifact of a nonlinear relationship between income and health at the individual level and that contextual effects are spurious. (For this view, see, e.g., Mellor and Milyo [1999]; Miller [1999].)

For the current study, this concern is less salient. First, we are not making claims about the social context resulting from the aggregation of individual data. That is, our independent variables are not a function of individual-level measures (as is the income distribution). Our independent variables of interest are, by definition, nation-state–level variables of government expenditures and national economic activity. While the number of individuals affects them (since they are expressed as per capita measures), they are not captive of individual-level relationships through aggregation. Still, there remains the possibility that the reductions in infant mortality that result from economic growth or state investment are not occurring among the portions of the population that are receiving the benefits of such growth or spending.[10] For example, the government could be investing in prenatal health care for the poor, but an observed reduction in infant mortality occurs predominantly among the middle class.[11] The fixed-effects approach deployed here curtails this possibility for the most part (and is the method that critics use to debunk the income inequality/health relationship) since we are talking about within-country *changes* in spending or standard of living and within-country *changes* in the IMR. A cross-sectional approach would be much more vulnerable to aggregation bias. More serious for the current study is the concern of reverse causality, to which we now turn.

[10] By way of a solution, some researchers might argue that the ideal approach would be to have individual-level data with social spending variables appended to them for a multilevel analysis, but then we are back to the problem of unobserved variable bias. That is, since we cannot generate infant-specific fixed-effects models, we cannot know whether there is positive or negative selection bias at work. Another drawback to the use of individual data to detect effects of welfare spending is the issue of public goods discussed in the welfare state structure section, above.

[11] Again, as suggested earlier, this could imply that state investment (or economic growth) might not be a private, divisible good.

Particularly when we use aggregated data, we cannot be entirely sure that welfare spending is affecting health (or other) outcomes, and not the reverse. It is an equally plausible story that higher rates of at-risk infants, for instance, drive up public health and social spending by generating more costs and that the causal story that previous researchers have been telling is actually backward. Also, the infant mortality rate might be acting as a proxy for health care problems in the entire population, which would drive up governmental health expenditures. Finally, it may be the case that higher benefit expenditures affect fertility decisions and, ultimately, infant mortality. Specifically, if more generous state benefit levels (health, income support, or otherwise) positively affect fertility rates disproportionately among the highest risk groups of potential mothers (e.g., the young and poor), we should expect a positive, counterintuitive association between social spending and infant mortality rates (Plotnick 1990).[12] We attempt to address these issues by explicitly accounting for the possibility of reverse causation or spurious effects. Specifically, we include the welfare and health spending variables the year after the IMR is measured in addition to the year of the IMR measure. The logic is that if the IMR is acting as a proxy for greater health "needs," those higher costs should exist the subsequent year, in addition to the contemporaneous year. Also, if there is a spurious portion to the effects of government expenditures, it will be captured in the joint variance of the two sets of variables, leaving the spending variables measured during the contemporaneous year to reflect the unique or "true" effect. This approach to ruling out spurious association has been used elsewhere (see Mayer 1997).

DATA

In order to model the IMR among rich countries over time, we use a data set constructed by Huber et al. (1997) called the Comparative Welfare States Data Set. This data set itself is a compilation of demographic, social spending, political, and economic variables compiled and made comparable from a variety of sources. To these data, we appended rates of LBW from the OECD Health Dataset, and to a subset of the data, we also added a measure of income inequality (Gini coefficient) from the Deininger and Squire data set. Finally, we included total fertility rate (TFR) and %urban population from the World Tables of Economic and Social In-

[12] It is important to note that several studies provide evidence refuting the idea that welfare serves as an incentive for women to have children (Blank 1995; Moore, Morrison, and Glei 1995).

dicators, 1950–92.[13] The data show autocorrelation, so we estimated feasible generalized least squares with an autoregression coefficient that is common to all countries along with panel-corrected standard errors, as recommended by Beck and Katz (1996).[14] Since the panels were unbalanced, it was particularly important to perform sensitivity analysis. We ran extreme bounds and jackknife analysis (described in the appendix and presented in table A1) and found that results were rebust to changes in the model specification and to the exclusion of potentially influential cases. Below, we describe the variables from this data set that we will be using in our analysis.

Year.—Given the trends toward declining infant mortality rates, in combination with growing welfare-state budgets and rising health care costs over the same time period, it is important to include time as a covariate. We also tested a year fixed-effects model; that is, we included dummy variables for each year of the study (save one suppressed category) in addition to the country dummies. This did not change our results for the other variables, so we stuck to the simpler model with the overall trend indicator, "year."

Total fertility rate (TFR).—This variable captures age-specific fertility rates for women. Specifically, the TFR is the total number of children 1,000 women would bear in their lifetime if at each age their childbearing conformed to the current rate for their age group. It is calculated from age-specific fertility rates—the number of children born to 1,000 women in each age group in a specified year. Combining the age-specific fertility rates yields the TFR, and dividing it by 1,000 applies it to the individual woman. Since higher fertility rates may put a strain on state resources, it is important to control for this. This variable comes from World Tables of Economic and Social Indicators, 1950–92. (We also tested models using the crude birth rate per 1,000 people in the country's population of a given year, and results were unchanged.) Ideally, we would like birth rates for specific groups who may have different risk profiles—very young or very old mothers, for instance, or by income level. However, data on subpopulations were not available.

[13] For a fuller description of the construction of the Comparative Welfare States Data Set (or for the data themselves), please see the documentation provided on-line at http://www.lis.ceps.lu/index.htm. For the OECD Health Dataset, see http://www.oecd.org/els/health/software/; for the Deininger and Squire data set, see http://www.worldbank.org/research/growth/dddeisqu.htm; and for the World Tables of Economic and Social Indicators, 1950–92, see ICPSR #6159 at http://www.icpsr.umich.edu:8080/ABSTRACTS/06159.xml?format=ICPSR.

[14] Results were not different in models using a generalized linear model approach, as discussed by Kmenta (1986), where the autoregression coefficient is allowed to vary by country.

Percentage urban.—The percentage of a population residing in an urban area is important to control for because it is a potential proximate determinate of infant mortality. For example, it is possible that living in an urban area may be associated with decreased infant mortality due to an increased access to health care resulting from a higher concentration of services in urban areas (Pampel and Pillai 1986). Or, it could be related specifically to access of specialized services such as tertiary hospitals with neonatal intensive care units (Phibbs et al. 1996). However, other research also points to possible negative associations of urban residence and infant mortality. For example, urban areas may have a higher density of people in poverty, and community poverty has been associated with low birth weight and infant mortality (Matteson, Burr, and Marshall 1998; Roberts 1997). This variable comes from World Tables of Economic and Social Indicators, 1950–92.

Proportion elderly.—We include a variable that represents the percentage of the population that is over 65 years of age. A higher proportion of elderly puts a strain on state resources and may represent a greater generational transfer from young to old, so it is important to control for this demographic indicator. We have also tested models with the dependency ratio—that is, the proportion of the population that is either over 65 or under 15 years of age (results not shown). Models were not sensitive to which formulation was used. These variables are constructed from Labour Force Statistics (OECD).

Unemployment rate.—This variable is included as a measure of the current state of the economy of the particular country in question. Higher unemployment rates may lead to worse rates of infant mortality due to resource strain at the family or state level. Some research has shown that unemployment is linked to higher rates of LBW (a mediating variable for infant mortality) on the county level in the United States (Catalano and Sexner 1992).[15]

Real GDP per capita.—We also control for the per capita share of the total output of the domestic economy expressed in constant dollars (using the Chain index based on Purchasing Power Parities [PPPs] in 1985 international prices). This formulation of GDP is recommended (over, e.g., the Laspeyres Index) for intertemporal analysis, "as it brings changing relative prices into the analysis explicitly through a chain index" (Huber, Ragin, and Stephens 1997, p. 9). Controlling for this variable is meant to factor out the overall level of economic development of the country for

[15] A number of other economic measures were not significant and are not presented in the final tables: %change in the consumer price index (the inflation rate), growth rate (%change in real gross national or domestic product), and the female labor force participation rate (used as a proxy for female education level, which was not available).

a given year. The source for this variable is the Penn World Tables. (For more information, see, e.g., Summers and Heston [1991]). The effect is nonlinear, and we include this variable logged to the base *e*. This allows for a percentage interpretation of the impact of economic development and growth. (When we use a quadratic function, results are similar.)

Public health spending.—This is the total public expenditure on health also expressed in per capita 1985 dollars using the same conversion approach described above (logged to the base *e*). This variable comes from the OECD, *Health Care Systems in Transition* (1990) and for the years thereafter from table 2 of Eco-Santé Health Data (OECD 1995). We test a single-year measure, one that averages over three years, and one that takes a five-year running average. (We tested measures that averaged more than five years and found that they did not perform as well as the five-year one, so we stopped there.)

Income inequality.—For a subset of the country-years, we are able to append the Gini coefficients of income inequality from the Deininger and Squire data set.[16] We used only those data that Deininger and Squire labeled "accept," meaning of reliable quality and which were available on an annual basis (for at least a few years) in order that they could be used in time-series analysis. These results are presented below. We also tested models with Gini indices of income inequality constructed from the Luxembourg Income Study data.[17] However, we discarded these from the final analysis since they are only available for select years (about two–three years per country on average, 49 country-years in total) and therefore are not conducive to fixed-effects or pooled time-series analysis.

Low birth weight.—Rates of LBW (< 2,500 grams) as a percentage of all live births is both a dependent and independent variable in the analysis. Birth weight is central to further reductions in the IMR among developed countries since death rates for the neonatal period (first month of life) are quite sensitive to birth weight (Luke et al. 1993). For example, among babies born in the United States in 1991, the IMR for those who weighed over 2,500 grams (five pounds, eight ounces) at birth was 0.348%; it was 2.178% for babies born between 1,500 and 2,500 grams; meanwhile, a staggering 29.648% of very low birth weight infants (VLBW, less than 1,500 grams, or three pounds, five ounces) died within the first year of life.[18] In fact, in 1991, medical complications associated with LBW and

[16] The seven countries with Gini data include Canada, Finland, Italy, Japan, Sweden, the United Kingdom, and the United States.

[17] For further information on the Luxembourg Income Study, see http://www.lis.ceps.lu/access.htm

[18] See tables from the linked birth/infant death file at the National Center for Health Statistics' (1998) Web address: http://www.cdc.gov.nchswww/datawh/statab/unpubd/mortabs.htm

preterm delivery were the primary cause of death among black infants and the third leading cause of death for white infants. Further bolstering the importance of studying LBW is research that demonstrates that both the neonatal and postneonatal infant mortality rates are highly sensitive to the rate of low birth weight. Specifically, when the percentage of LBW births is reduced, a greater corresponding reduction in the percentage of infant deaths occurs (Johnson and Zaki 1988). In other words, the mortality "elasticity" for LBW is greater than one.

Infant mortality.—This is the ultimate dependent variable in the analysis and is measured as the number of deaths from birth to one year of age per thousand live births. This is compiled by Huber, Ragin, and Stephens (1997) from a variety of sources. The World Health Organization has defined a live birth as a product of conception that shows signs of life irrespective of its gestational age. Most U.S. states have adopted this definition, and a majority of states require that all fetuses over 20 weeks of gestation be reported regardless of whether the infant was alive at birth (Sepkowitz 1995). Conversely, many industrialized nations do not require reporting of birth and subsequent death until the infant reaches 28 weeks of gestation (Cartlidge and Patrick 1995). France does not register a birth until 48 hours—many deaths are called stillbirths (Sachs et al. 1995). In England, a woman in labor prior to 28 weeks is admitted to a gynecology ward (Sachs et al. 1995). Japan does not report as life those who die shortly after birth, if born prior to 22 weeks or with congenital abnormalities (Sachs et al. 1995). These reporting differences further emphasize the need for within-country controls such as those being employed here (Howell and Blondel 1994).

Table 1 provides the mean values and standard deviations (total and within-country) for the variables used in the analysis. As shown in the table, the mean IMR for our countries over the time period 1960–92 is approximately 11.05, which is within the range of other studies (see, e.g., Pampel and Pillai 1986; WHO 1999). Other variables also fall within the expected ranges based on previous studies.

FINDINGS

Model 1 of table 2 shows a basic model of the impact of demographic, economic, and welfare spending variables on IMR for the OECD countries. Over and above the effects of country averages and the overall trend (year), we see that a number of demographic and economic factors matter in predicting the IMR. Infant mortality rates are higher when countries are more urbanized, but they decline with economic growth (as reflected by the effect of GDP). The total fertility rate and the proportion of the

TABLE 1
MEANS, TOTAL STANDARD DEVIATIONS, AND WITHIN-GROUP STANDARD DEVIATIONS

Variable	Mean	Total SD	Within-Group SD	*N*
Infant mortality (per 1,000 births)	11.848	5.502	4.644	349
LBW rate (%)	5.576	.966	.433	349
Total fertility	1.918	.430	.358	349
Proportion over 65	12.598	2.506	1.298	349
Proportion of population urban	73.777	11.981	2.930	349
Real GDP per capita (in thousands)	11.293	2.769	2.040	349
Unemployment rate	.049	.037	.021	349
Health care spending per capita	620.030	239.316	193.372	349
Gini coefficient	31.748	4.131	1.841	158
Year	1979	8.481	7.511	349

NOTE.—$N = 349$; countries = 19.

population over age 65 are not significantly related to the IMR in this model, while the unemployment rate demonstrates a counter-intuitive result of being negatively associated with the IMR. That is, the higher the unemployment rate, the lower the IMR. As quizzical as this finding is, it does hold up in all the models for infant mortality and many for low birth weight rates (as we shall see below). Turning to the variable of primary interest, model 1 shows that health care spending on the part of the government is significantly associated with a reduction in IMR in that country. Each 1% increase in per capita health spending is associated with a decline of 0.184% in the IMR. Models 2 and 3 test the hypothesis that state investments in health care are cumulative, that is, whether state health investments from several years prior contribute to a lower rate of LBW. This could be a result of, for example, the increasing effectiveness of a public awareness campaign, investments in capital such as better hospitals, and so on. Results from models 3 (a three-year measure) and 4 (a five-year measure) show that as the number of years that health spending is measured increases (as a moving average), the impact of the variable increases. (After five years, the impact declines again.) Each 1% increase in the per capita public health budget over a three-year period is associated with a decline of 0.232% in the IMR (model 2). Likewise, each 1% increase in the per capita health budget over a five-year period is associated with a 0.348% decrease in the IMR (model 3). The reader should note that when we control for health spending over these longer time frames, the effect of GDP per capita becomes insignificant, as does the effect of ur-

banity. On the other hand, the proportion elderly becomes significant and positively associated with infant mortality.

Models 4, 5, and 6 of table 2 test for the possibility of reverse causality or spurious effects in the welfare-state variables by including the spending measures for the year after which infant mortality is measured (for the rationale behind this approach, see the methods section, above).[19] When we do this, we find that the $T + 1$ year variable for health care spending is not significant. Even after controlling for this possibly spurious component of the health care spending variable, the main effect of health care spending (over a one-year period) is still significant (and is even stronger, though not statistically different from the coefficient in model 1). We can be more confident in claiming that the effect of health care spending is not spurious. Models 5 and 6 of table 2 perform the same exercise for the three- and five-year health spending variables, yielding similar results. Finally, model 7 tests whether net of the rate of LBW there is still a direct effect of health spending on infant mortality. When we control for the LBW rate (which itself is highly significant), we find that the effect of health spending is reduced in magnitude but remains significant. Each 1% increase in the rate of LBW is associated with a 0.363% increase in the IMR. Net of LBW rates, each 1% increase in health spending is associated with a 0.128% lower IMR. This suggests that part of the effect of health expenditures on the IMR works through birth weight and part is independent of that factor (refer to fig. 2).

Given that the IMR is so sensitive to the rate of LBW, table 3 models LBW as a dependent variable using the same set of models as was presented for the IMR in table 2. Here we find a trend toward increasing rates of LBW in some models (and an insignificant trend result in others). We also find that in some models, but not others, the rate of LBW is reduced with economic development. However, we also find a couple of quizzical coefficients here. In some models the unemployment rate is negatively associated with the rate of LBW. That is, the higher the unemployment rate, the lower the rate of LBW. This does not accord with other research (see, e.g., Catalano and Sexner 1992). However, the reader should note that this effect is only significant when health spending is measured over a single year. When we extend the horizon of health spending, we find that the effect of unemployment disappears; therefore, this coefficient probably reflects something about the correlation of overall health spending with the business cycle and not something particular about unemployment lowering rates of LBW. Percentage of the population

[19] This causes us to lose cases, since the year 1992 is lost for those countries that had data for it. When 1992 is excluded from the original models, no significant changes are detected in the coefficients, indicating that this is not likely to be generating bias.

TABLE 2
MODELS FOR INFANT MORTALITY RATES

	(1)	Three-Year Measure (2)	Five-Year Measure (3)	Reverse Causality/ Spurious Effects (4)	Three-Year Measure and Reverse Causality (5)	Five-Year Measure and Reverse Causality (6)	With LBW (7)
Year	−.022***	−.033***	−.030***	−.023***	−.032***	−.029***	−.024***
	(.003)	(.004)	(.005)	(.004)	(.004)	(.005)	(.003)
Log LBW							−.363***
							(.069)
Total fertility rate	.011	−.012	−.008	.011	−.034	−.027	.007
	(.029)	(.036)	(.041)	(.030)	(.036)	(.040)	(.029)
Proportion over 65	−.002	.029+	.036*	.003	.035*	.042**	−.003
	(.014)	(.015)	(.016)	(.014)	(.015)	(.015)	(.014)
%urban	.013**	.008	.009	.012**	.008	.008	.012**
	(.005)	(.005)	(.006)	(.004)	(.005)	(.006)	(.005)
Log GDP per capita	−524***	−.162	−.115	−.510***	−.187	−.156	−.488***
	(.117)	(.159)	(.163)	(.119)	(.158)	(.162)	(.115)
Unemployment rate	−1.991***	−1.258**	−1.332**	−1.893***	−1.39**	−1.427**	−1.568**
	(.385)	(.452)	(.468)	(.399)	(.450)	(.459)	(.384)
Log per capita health care spending (in thousands)	−.184**	−.232***	−.348***	−.211**	−.208*	−.349***	−.128*
	(.059)	(.065)	(.075)	(.068)	(.083)	(.093)	(.059)

Log per capita health care spending, year$_{t+1}$				.044	−.074	−.034	
				(.059)	(.085)	(.089)	
Constant	47.463***	69.513***	63.660***	50.495***	67.632***	61.928***	51.682***
	(6.614)	(8.409)	(9.080)	(6.892)	(8.322)	(8.866)	(6.646)
Common autoregressive term (1)	.556	.537	.514	.549	.534	.507	601
Wald χ^2	3,247.86	2,787.62	2,516.30	3,391.85	2,916.02	2,684.49	3,084.96
df	25	25	24	26	26	25	26
N	349	311	273	348	310	272	349

NOTE.—Panel-corrected SEs are in parentheses.
$^{+}$ $P < .10$.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

TABLE 3
MODELS FOR LOW BIRTH WEIGHT RATES

	(1)	Three-Year Measure (2)	Five-Year Measure (3)	Reverse Causality/ Spurious Effects (4)	Three-Year Measure and Reverse Causality (5)	Five-Year Measure and Reverse Causality (6)
Year	.010**	.002	.003	.009**	.003	.004
	(.003)	(.003)	(.004)	(.003)	(.003)	(.004)
Total fertility rate	.033	−.032	−.039	.032	−.044^{+}	−.051^{+}
	(.024)	(.025)	(.030)	(.024)	(.025)	(.030)
Proportion of population over 65	−.014	.010	.012	−.010	.014	.016
	(.012)	(.010)	(.012)	(.012)	(.010)	(.012)
%urban	.005	−.011**	−.010*	.004	−.011**	−.010*
	(.004)	(.004)	(.005)	(.004)	(.004)	(.005)
Log GDP per capita	−.160^{+}	.075	.074	−.156^{+}	0.55	.045
	(.092)	(.107)	(.119)	(.094)	(.107)	(.120)
Unemployment rate	−1.123***	−.182	−.174	−1.068**	−.239	−.211
	(.300)	(.300)	(.336)	(.310)	(.301)	(.334)
Log per capita health care spending (in thousands)	−.129**	−.151**	−.182**	−.137**	−.145**	−.189**
	(.047)	(.044)	(.057)	(.051)	(.054)	(.068)

Log per capita health care spending, year$_{t+1}$			.021	−.032	−.014	
				(.043)	(.054)	(.060)
Constant	−16.344**	−1.14	−3.36	−14.409*	−2.161	−4.462
	(5.392)	(5.716)	(6.842)	(5.638)	(5.744)	(6.843)
Common autoregressive term (1)	.679	.607	.641	.674	.620	.655
Wald χ^2	520.18	946.55	705.71	543.41	933.51	692.94
df	25	25	24	26	26	25
N	349	311	273	348	310	272

NOTE.—Panel-corrected SEs are in parentheses.
$^{+}$ $P < .10$.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

living in cities (urbanity) also demonstrates a negative association with LBW—when the health spending variable is measured over multiple years. Finally, when we move to the multiple-year measure, the trend effect goes away. The one effect that remains significant across all models is the effect of health spending. Again here, the effects appear to be cumulative. The coefficient for the three-year measure is larger than that for the one-year measure. Likewise, the coefficient for the five-year measure is yet larger (though the differences are not statistically discernable). For each 1% increase in health spending in a given year, the associated incidence of LBW is 0.129% lower. When we move to a five-year average, each additional percentage of per capita government health spending is associated with a rate of LBW that is 0.182% lower.

Table 4 represents an attempt to determine how income inequality may act as a confounding factor for the effects of health care spending. We find that in the reduced sample ($N = 155$) for which Gini coefficients are available, the Gini coefficient is marginally positive for model 1, indicating the possibility that increases in income inequality result in a higher incidence of LBW—controlling for stable, country-specific factors. In this model, health care spending is significantly negative in predicting the LBW rate, as it was in the previous table. For the IMR model 2, we find that the Gini coefficient is not significant. However, the effect of health spending is robust to its inclusion. So, it appears that income inequality variation within a country has effects on birth outcomes via the social pathway outlined in figure 1.

Next, we examine the issue of whether the returns to state spending vary by welfare regime type. To address this question, we test our basic models on groups of countries classified by welfare regime type.[20] In this analysis (see table 5), we use—as a matter of convention—Esping-Anderson's tripartite classification schema (see discussion in the methods section, above). This analysis includes only the subset of countries that are clearly classified into one of the three categories by Esping-Anderson. When we break the sample out like this for the LBW analysis, we find that the health-spending variable loses its significance for all groups except for the corporatist countries, for which it is still significant. The same pattern of results is evident with respect to predicting infant mortality. In social-democratic and liberal regimes, within-country changes in per capita health care spending do not appear to be significantly related to the IMR. Effects are strong, however, for the corporatist regimes.

[20] We are running separate models for each regime type because it is not possible to include regime type as a variable in our fixed models presented in tables 2–4. Fixed-effects models include a dummy for each country, and therefore inclusion of an additional dummy for regime type would not have any variance.

TABLE 4
GINI COEFFICIENT MODELS FOR LOW BIRTH WEIGHT AND INFANT MORTALITY RATES

	LBW (1)	IMR (2)
Year	.004	−.023***
	(.004)	(.004)
Total fertility rate	−.017	−.060
	(.030)	(.368)
Proportion of population over 65	.026+	.027
	(.015)	(.018)
%urban	−.002	.004
	(.004)	(.005)
Log GDP per capita	−.107	−.543**
	(.136)	(.168)
Unemployment rate	−.923**	−2.679**
	(.327)	(.418)
Log per capita health care spending (in thousands)	−.155**	−.198*
	(.059)	(.077)
Gini coefficient	.006*	−.004
	(.002)	(.003)
Constant	−4.699	49.992***
	(7.273)	(9.142)
Common autoregressive term (1)	.525	.273
Wald χ^2	674.49	3,190.72
df	21	21

NOTE.—Panel-corrected SEs are in parentheses. $N = 155$.
+ $P < .10$.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

Finally, we tested for a structural break in the impact of health spending. The results of this effort are shown in figure 4. Models were compared using a BIC statistic: $\text{BIC} = L_k^2 - df_k^* \log N$, where L_k^2 is the likelihood ratio test statistic under model k; df_k is the degrees of freedom for model k; and N is the sample size (see Raftery 1986). The model with the most negative statistic is preferred. (In other words, we are looking for a trough, not a peak). A difference of 10 points or more between a given model and the other models is very strong evidence for a structural break; a difference greater than six is considered a strong indication; and any difference under six is considered moderate to weak evidence. Though there appears to be a trough in 1966, it does not appear to be a significant improvement over all other potential models; for example, the difference in BIC statistics between this model and the model that specifies a break in 1984 is just over two points. Rather, the pattern appears to be one of random fluctuation. This does not mean that there were not significant structural shifts within individual countries—such as the legalization of

TABLE 5
WELFARE STATE COEFFICIENTS FOR LOW BIRTH WEIGHT AND INFANT MORTALITY RATES BY REGIME TYPE

	LBW			IMR		
	Liberal	Corporatist	Social-Democratic	Liberal	Corporatist	Social-Democratic
Log per capita health care spending (in thousands) 3-year measure	−.83	−.187***	−.067	−.112	−.520***	−.061
	(.076)	(.050)	(.095)	(.104)	(.082)	(.087)
Common auto-regressive term (1)	.727	.186	.644	.167	−.068	.434
Wald χ^2	238.02	269.21	143	5,620.59	7,249.51	1,250.53
df	11	11	11	11	11	11
N	85	61	108	85	61	108

NOTE.—IMR analyses do not include LBW as a control. This methodology enables us to explore the total (direct and indirect) effect of health care spending on IMR. Panel-corrected SEs are in parentheses.
$^{+}$ $P < .10$.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

abortion within all states of the United States in 1973 or the introduction of universal health care in Canada in the late 1960s and early 1970s; however, the estimation of country-specific structural breaks for the entire OECD group would have entailed the inclusion of 36 additional parameters (a main term and an interaction term for each country)—something that is not feasible given the number of country-years available. However, we were able to test the presence of a structural break in Canada and the United States, two countries for which there are adequate reasons to suspect one. In the case of Canada, we find that there is a trough in 1967 but that it is less than two points lower than the next lowest BIC statistic, that for 1987 (fig. 5). Likewise, in the case of the United States (fig. 6), we find no such structural break around the time that abortion was legalized nationwide through the 1973 *Roe v. Wade* decision.

DISCUSSION

This study has shown that even among rich countries, increased investment in public health yields benefits in the form of a lower incidence of LBW and a lower IMR (even net of the LBW rate). These findings are

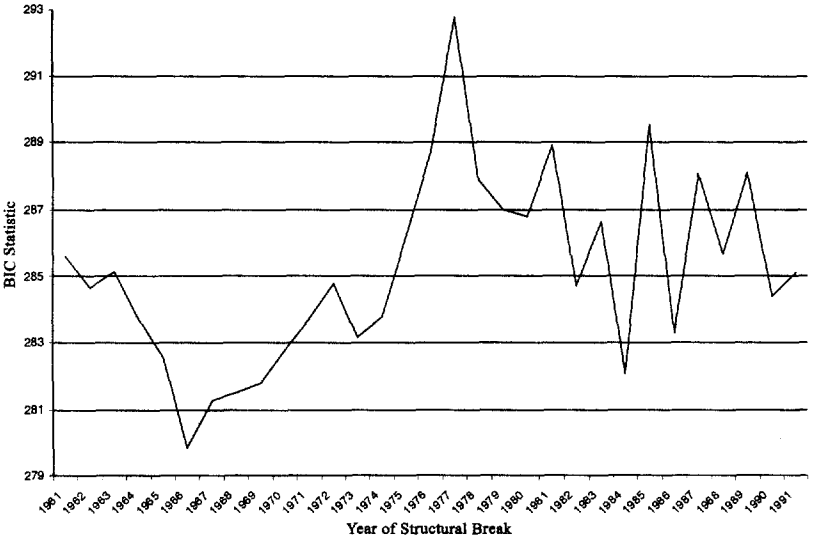

FIG. 4.—Test for structural break in the effect of health spending on infant mortality rates, entire sample, BIC statistic.

robust to all the models presented here and pass several very rigorous methodological tests. First, they hold true, even accounting for serial correlation in the time-series data, for trend effects and for unobserved, between-country differences. Second, even when we attempt to account for reverse causality and spurious portions of the effect, by including health spending the year after infant mortality is measured, we find that health spending is robust.

The magnitudes of these effects are not trivial. Each additional 1% in per capita health care spending—in the base models—is associated with a reduction in the LBW rate by 0.129% and the IMR by 0.184% (directly and indirectly through lower rates LBW). When we move to a cumulative-effects-of-state-spending model over a three- or five-year period, we find that the results are even stronger. Over a five-year period, an increase of 1% in per capita of health spending (per year on average) may yield a decrease in the LBW rate of 0.182%. The cumulative effects of state spending on infant mortality are even more impressive. The same increase in spending is associated with a 0.348% reduction in the infant death rate. The effects of health spending, therefore, may work both through the social and medical pathways illustrated in figure 1—that is, directly on IMR, net of LBW and through LBW as a mediating mechanism. This effect cannot be attributed to constant, between-country, unobserved differences such as in culture, health practices, or even genetic makeup.

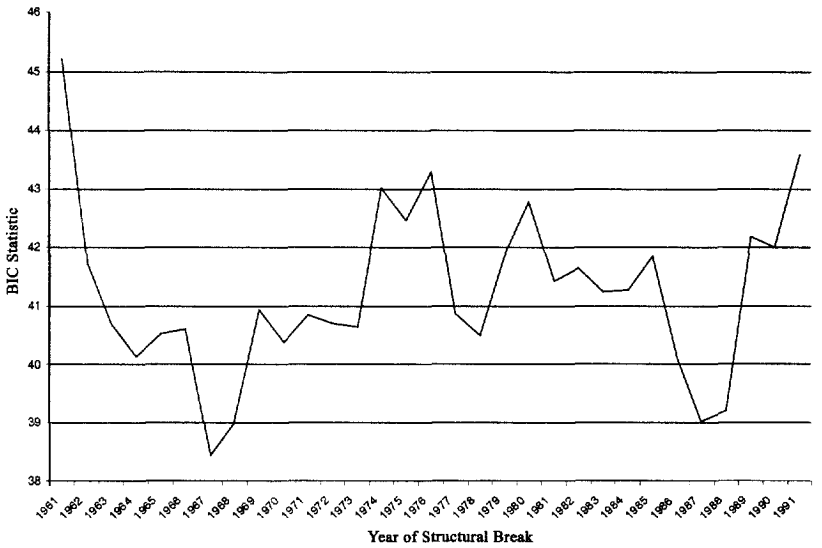

FIG. 5.—Test for structural break in the effect of health spending on infant mortality rates in Canada, BIC statistic.

Likewise, they cannot be attributed to levels of income inequality since they are robust to the inclusion of Gini coefficients. They also withstand the inclusion of health spending the year after IMR and LBW are measured as a test of spuriousness.

It is interesting to note that when we break out the analysis by welfare regime type, only among corporatist welfare-state regimes do we observe significant effects of health spending on both IMR and LBW rates. Given sample size limitations when the analysis is broken out in this fashion, it would not be fair to say that health spending does not have any effect in social-democratic and liberal regimes, but it might be reasonable to conclude that the effects are stronger in the corporatist states. The reader should note that in figure 3, we saw that it was corporatist countries that began this study period with the highest infant mortality levels of the three groups and that demonstrated the steepest decline in the subsequent period, though the rates of health spending (and their slopes) are not dissimilar among the three groups. These findings with respect to welfare-state structure represent a first cut and deserve further attention. For example, we adopted Esping-Anderson's three-category approach out of convention; however, recent research has shown that these categories may not be as applicable to the realm of health care provision as they are to income support (Gran 1999). This is particularly troubling since a number of the OECD countries were left out of this portion of the analysis for

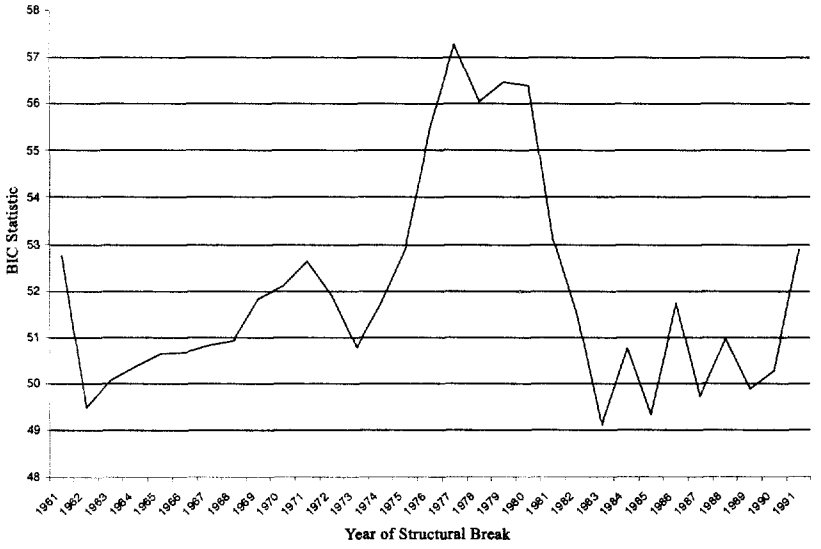

FIG. 6.—Test for structural break in the effect of health spending on infant mortality rates in the United States, BIC statistic.

failing to fall into a distinct group. However, the more nuanced the categories become (such as Korpi and Palme's [1998] five-group typology), the fewer countries become included in each, making model estimation all the more difficult. What may be needed is analysis that regroups the countries meaningfully but restricts the number of categories to two or three.

While the structure of the welfare state may matter with respect to the returns on health investment, we find no convincing evidence that the institutional structure of the health care delivery system matters in determining the "bang for the buck"—that is, the impact of state health expenditures. Having changed to a universal health care system in the middle of this time series, Canada provides a unique case to examine the impact of universal coverage. However, there does not appear to be a significant structural break in the impact of health spending during this period. (Neither does there appear to be a break in the effect for the entire sample of OECD countries.) Nor do we find evidence of a structural break around 1973 in the United States, when abortion was made legal nationwide. It appears, then, that these conditions do not significantly affect the impact that state health expenditures have on IMR.

Finally, while persuasive in demonstrating associations between government health spending and positive birth and infant health outcomes, this analysis—like most observational social science—is far from deter-

mining causality. Other researchers who want to push further in the attempt to document a causal relationship may want to investigate with more precision the mechanisms behind the findings presented here. For instance, in addition to examining the rate of LBW and infant mortality separately, as is done in this article, it might be useful to break out infant mortality by cause of death and timing (i.e., neonatal mortality versus postneonatal mortality). It also might be useful to examine the returns to different types of health spending, such as capital expenditures, preventative care, ambulatory care, and so on. Also, data that linked microlevel outcomes to macrolevel policy changes would be preferable to data wholly measured at the macrolevel, as is used here. Of course, experimental designs would be the best of all. We hope that future researchers push the agenda in this direction since the topic of infant mortality is important to social science, medical practice, and public policy.

APPENDIX

Sensitivity Analyses

Given the unbalanced nature of the panel data and the uncertainty as to the correct model specification, we ran two types of sensitivity analyses: jackknife and extreme bounds analysis. (For an explanation of these analyses, see Leamer discussion in Western and Healy [1998].) In the jackknife analysis, each country is, in turn, taken out of the pool, and the remainder of the cases are run using the model being tested to determine whether the results are driven by influential cases. The extreme-bounds analysis tests the coefficients' sensitivity to model specification by excluding each variable (one at a time) and running the resulting reduced model. Table A1, below, reports the lower- and upper-bounded coefficient estimates for each of these approaches for the models of LBW and IMR using panel-corrected standard errors, a common autoregressive term, and country fixed effects (model 1 of tables 2 and 3, respectively). As can be seen in the table, the main effect of health care spending is robust to the exclusion of any other predictor or the exclusion of each country. The effects of other variables are also consistent with those reported in tables 2 and 3.

TABLE A1
SENSITIVITY ESTIMATES FOR MODELS OF LBW AND IMR

	LBW		IMR	
	Jackknife	Extreme Bounds	Jackknife	Extreme Bounds
Year	.004, .014	.005, .009	−.030, −.012	−.029, −.022
LBW rate (ln) ...	. . .	. . .	.316, .448	.278, .0387
Total fertility rate	.009, .046	.025, .065	−.014, .067	.008, .62
Age over 65	−.032, .012	−.017, .010	−.040, .016	−.54, .010
%urban	−.005, .013	.002, .006	.008, .020	.008, .016
Log GDP per capita	−.325, −.094	−.304, −.032	−.604, −.193	−.861, −.304
Unemployment rate	−1.407, −.919	−1.227, −.645	−2.475, −1.408	−3.188, −1.290
Log per capita health care spending (in hundreds)	−.171, −.090	−.172, −.105	−.225, −.137	−.317, −.106

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