

WELFARE REFORM AND INFANT HEALTH: THE IMPACT OF
MANDATORY MATERNAL EMPLOYMENT

By

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Dissertation

Submitted to the Faculty of the
Graduate School of Vanderbilt University
in partial fulfillment of the requirements for

the degree of

DOCTOR OF PHILOSOPHY

in

Leadership and Policy Studies

May, 2010

Nashville, Tennessee

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CHAPTER I

INTRODUCTION

Nearly 30 million American children--almost 40% of all Americans under 18--live in or near poverty (National Center for Children in Poverty [NCCP], 2006a; U.S. Census Bureau [U.S. Census], 2006a). Due to their poverty, these children face an increased risk of disease and death both in childhood (Aber, Bennett, Conley, & Li, 1997; Brooks-Gunn & Duncan, 1997; Children's Defense Fund [CDF], 1994; Geltman, Meyers, Greenberg, & Zuckerman, 1996; L. V. Klerman, 1991; Singh & Yu, 1996) and later in life (Claussen, Davey-Smith, & Thelle, 2003; Lawlor, Ebrahim, & Davey Smith, 2002). Poor children are twice as likely to be in bad health as are non-poor children (Brooks-Gunn & Duncan, 1997; Montgomery, Kiely, & Pappas, 1996), suffering a greater *number* and greater *severity* of health problems than their more affluent counterparts (Brekke, Hjortdahl, Thelle, & Kvien, 1999; Geltman et al., 1996; CDF, 1994; L. V. Klerman, 1991). It is vital, therefore, to the health of children and the future of our nation that we develop public policies that effectively address child poverty and mitigate its adverse effects on health.

On August 22, 1996, Congress passed the Personal Responsibility and Work Opportunity Reconciliation Act of 1996 (PRWORA) or "Welfare Reform" (Pub. L. 104-193), "the biggest national policy change in our history affecting poor families with children" (Edelman, 1999, p. 1493). With this sweeping legislation, Congress abolished the Aid to Families with Dependent Children (AFDC) program that had guaranteed

income support to poor families for over 60 years (Bane & Weissbourd, 1998; Cashin, 1999; Quigley, 1998; Wright, 1998), replacing it with the block-grant Temporary Assistance for Needy Families (TANF) program (PRWORA §§ 601-606, 1996). In addition, PRWORA explicitly repealed the federal entitlement to assistance (PRWORA § 601, 1996), set time limits for receipt of benefits (PRWORA § 608, 1996), and effectively shifted authority for “welfare” to the states, giving them “unprecedented discretion in choosing which families to assist, what services to provide, what requirements to impose,” and what penalties to apply (Larner, Terman, & Behrman, 1997, p. 6).

The hallmark of Welfare Reform, however, was the implementation of “workfare,” an explicit requirement that adult recipients--predominantly single mothers--work to receive benefits (PRWORA § 607, 1996). While AFDC had previously exempted mothers of children under 3 years of age from work requirements, TANF provides no such guarantee, giving states the *option* to exempt single parents of infants (less than 12 months of age) (PRWORA § 607(b)(5), 1996). Twenty-five states provide work exemptions to those caring for infants up to the age of 12 months and four states extend the exemption beyond 12 months (Urban Institute [Urban], 2005a). Twelve states, however, limit the exemption to those caring for infants under 3 months of age, four states between 4 and 6 months of age, and six states provide no exemption for those caring for children (i.e., mothers are required to work immediately after delivery or adoption to continue TANF benefits) (Urban, 2005a).

In the wake of Welfare Reform’s new work requirements, an estimated 1 million U.S workers--most notably single mothers--entered the paid labor market (1996-2002)

(Blank, 2002). This rise in labor force participation (LFP) was higher among mothers of infants than among U.S. mothers in general (Blank, 2002), and was particularly significant among female TANF recipients for whom there was a 135% increase in LFP in the 10 years following PRWORA's passage (calculated from data available from the Administration for Children & Families [ACF], *Characteristics and Financial Circumstances of TANF Recipients*, n.d.). This increase in maternal employment as a result of PRWORA's work requirements has the potential to significantly affect children—particularly infants. Despite the potential impact on infants, few studies have evaluated how infants have fared under the “welfare-to-work” waiver programs undertaken by states in the lead-up to Welfare Reform or the work requirements mandated by the PRWORA legislation. Given this gap in the existing literature, ***this research examines the impact of Welfare Reform's mandatory maternal employment provisions on infant health.***

Background

PRWORA explicitly requires that TANF recipients engage in “allowable work activity” to receive benefits (PRWORA § 607, 1996). States have discretion in defining allowable work activity, in setting the required number of work hours, and in establishing categories of exemptions--if any--from work requirements (e.g., single parents with young children). Under Welfare Reform, states may penalize recipients who do not comply with work requirements, with great latitude in defining their sanction policies. State sanctions for work noncompliance vary considerably, ranging from a partial reduction in benefits to immediate--often permanent--case closure (Urban, 2005a). With

more than 70% of TANF recipients being children and 90% of *adult* TANF recipients being women (calculated from data available from ACF, *Characteristics*, n.d.)--predominantly single mothers--PRWORA generally means either maternal employment or state-imposed sanctions.

Maternal employment cannot be categorically defined as either positive or negative for family and children. While maternal employment can increase family income and access to material resources, job-related factors such as stress and limited flexibility can adversely affect the quality of parenting offered (Parcel & Menaghan, 1997; Zaslow & Emig, 1997). Although a great number of factors influence the effect of maternal employment on children's well-being, the most significant factors impacting young children and infants appear to be: the number of hours worked, the quality of child care offered, and the child's age and stage of development (Brooks-Gunn, Han, & Waldfogel, 2002; Clark-Kauffman, Duncan, & Morris, 2003; Han, Waldfogel, & Brooks-Gunn, 2001; Shields & Behrman, 2002).

First, the more time a child spends in child care, the greater the chance of a negative impact--particularly when child care exceeds 30 hours per week (Brooks-Gunn et al., 2002; Han, 2005). Second, low-income mothers are more likely to utilize lower quality informal child care arrangements than higher quality center-based care (Shields & Behrman, 2002). And third, maternal employment, at least full-time employment, generally has a deleterious cognitive effect during the first year of life (Hill, Waldfogel, Brooks-Gunn, & Han, 2005; Ruhm, 2004), although this may apply more to White rather than Black or Hispanic children (Waldfogel, Han, & Brooks-Gunn, 2002; Han et al.,

2001) and may not apply to low-income children (Desai, Chase-Lansdale, & Michael, 1989; Vandell & Ramanan, 1992).

In addition to the adverse cognitive effect of full-time maternal employment during the first year of life, mothers' early return to work has also been shown to have adverse *health* consequences for infants. One recent study found that mothers' return to work within 12 weeks of delivery was associated with decreased rates of breastfeeding, immunizations, and well-child care follow-up (all health-promoting behaviors, American Academy of Pediatrics [AAP], 1997, 2007) for their infants (Berger, Hill, & Waldfogel, 2005). Additionally, studies of parental leave policies in European countries have found that extending job-protected *paid* leave after delivery reduces infant mortality (primarily during the postneonatal period, days 28-364 of life) and early childhood mortality--with an estimated 2.5-3.4% reduction in infant mortality for a 10-week extension of paid leave (Ruhm, 2000; Tanaka, 2005; Winegarden & Bracy, 1995). Given this body of research, how has Welfare Reform's mandatory transition of low-income mothers into the low-wage labor market affected their young children?

Volumes of Welfare Reform evaluations were undertaken in the years following its enactment, assessing caseload levels, work participation, child well-being, health insurance coverage, economic self-sufficiency, family structure, and child support enforcement, among others. These evaluations have often proved challenging given the "evolving" nature of the 51 different TANF programs, the changing economic picture in the U.S. across time, the interconnectedness of TANF with other means-tested programs that were themselves changing, and uncertainty as to how best to "parameterize" or

“bundle” the TANF policies (Blank, 2002; Grogger, Karoly, & Klerman, 2002; National Research Council [NRC], 2001).

Much of the evaluation of Welfare Reform has focused on its effect on the “welfare rolls,” or TANF caseloads. In the first 10 years following enactment of PRWORA, TANF enrollment declined by a remarkable 67.7% (calculated from data available from ACF, *Characteristics*, n.d.). This decline was *not* generally associated with higher incomes, as the rise in income from mandatory employment programs that did not include financial incentives was offset by the losses in welfare benefits (Blank, 2002)--particularly for families with less-educated parents (Bennett, Lu, & Song, 2004). The significant decrease in TANF enrollment *was*, however, associated with a small decline in Medicaid enrollment and a decrease in health care utilization; specifically, the decrease in TANF enrollment was associated with a decrease in prenatal care and a small increase in the incidence of low birthweight among infants born to mothers with lower educational attainment (Bitler & Hoynes, 2006; Kaestner & Lee, 2005).

Few studies have assessed the impact of TANF’s mandatory maternal work requirements on children. This research has generally demonstrated that “programs that increase both [the] parents’ employment and their income produce positive effects for preschool and early school-age children, even as they negatively affect adolescents” (Clark-Kaufman et al., 2003, pp. 299-300; see also, Blank, 2002; Grogger et al., 2002). The impact of these mandatory maternal employment provisions on infants, however, has not been well-studied. The only study noted of the impact of Welfare Reform’s mandatory employment on infants has demonstrated that “stringent” welfare work requirements (greater than 18 hours work required, enforced by sanctions) present in 28

states resulted in a decline in breastfeeding rates (Haider, Jacknowitz, & Schoeni, 2003)--breastfeeding being associated with many health benefits for infants (AAP, 1997).

Research

With limited studies available on maternal employment and infants in the TANF context, this research examines the relationship between Welfare Reform's mandatory maternal employment and infant health through cross-sectional analyses of the 5-year period following implementation of PRWORA, using the state as the unit of analysis. The research assesses the impact on infant health of three TANF work requirements for mothers of infants:

1. the timing of mothers' return to work after delivery or adoption,
2. the number of work hours required for mothers of infants, and
3. the sanctions imposed for noncompliance with work requirements.

A fourth TANF work-related policy, "earnings disregards," is also considered: Earnings disregards set the income level TANF recipients may earn before TANF benefits are reduced and have been shown to affect the impact of TANF employment programs on school-aged children.

As its measure of infant health, the research utilizes infant mortality, "the most sensitive index we possess of social welfare and sanitary administration" (Yankauer, 1990, p. 653, citing Newsholme, 1910), known to be sensitive to socioeconomic status (SES) and maternal employment. A population's infant mortality rate (IMR), the number of deaths of infants under 1-year of age per 1,000 live births, is the sum of neonatal mortality (deaths between 0 and 27 days) and postneonatal mortality (28 days to 364

days). Neonatal mortality is generally considered “related to maternal health problems prior to or during pregnancy or in the labor and delivery period,” and accounts for two-thirds of overall infant mortality in the U.S., as in most industrialized countries (L. V. Klerman, 1991, p. 138). Postneonatal mortality is generally considered more “related to the environments that infants experience after returning home,” and accounts for one-third of infant mortality in the U.S. (L. V. Klerman, 1991, p. 138). Although the “2/3-1/3” neonatal-postneonatal mortality split represents the infant mortality pattern for the U.S. general population, the infant mortality pattern for the low SES population differs: infant mortality is more evenly distributed between the two periods in the low SES population, with a roughly 55%-45% split (calculated from data available from the Centers for Disease Control and Prevention [CDC], n.d.).

With no identifiable dataset linking infant health indicators--specifically infant mortality--to maternal TANF status, the TANF population is approximated for this research. In order to approximate the TANF population within the CDC’s “Linked Birth and Infant Death Data Set” [Linked Birth], maternal education and marital status are key: the rate of low educational attainment (less than high school education) among the TANF population is more than twice that of the general population (48% versus 20%), and the marital rate is well under that of the general population, even when “married and separated” are combined (23% versus 57%) (calculated from data available from the ACF, *Characteristics*, n.d., and the U.S. Department of Labor, *Current Population Survey*, n.d.). Based on the TANF adult population demographics, unmarried women with less than a high school education best approximate TANF recipients within the limits of the Linked Birth dataset.

Controls are introduced for baseline infant mortality, using the 1995 state infant mortality indicator, and macroeconomic conditions/employment opportunities, using the state unemployment rate for the target year. A control is also included for race given the well-established “Black-White” racial disparity in infant mortality, using the percentage of births to Black mothers in the study population for the target year. Because 15-17 year-olds are included in the study population data but are not subject to TANF work requirements, a fourth control is included for teenage births, using the state teen birth rate for 15-17 year-olds.

Organization

This paper is organized as follows:

Chapter II explores the background literature surrounding two significant issues in relationship to children’s health. The first is the relationship between poverty and children’s health. This section addresses the broader issues of the impact of socioeconomic status on health as well as child poverty in the United States, before narrowing the discussion to infants and their health. The second relationship underlying this research is the relationship between public policy and children’s health. This section discusses health policy, highlighting the unintended health consequences of public policy, before providing an overview of the history of and approaches to public policy addressing child poverty and welfare. The section finishes with a discussion of the lead-up to Welfare Reform efforts.

Chapter III builds on the discussion of public policy with a focused discussion of Welfare Reform. This chapter first details the 1996 PRWORA legislation, highlighting

the provisions with the greatest impact on women and children, then provides an overview of the existing literature surrounding Welfare Reform evaluation.

Chapter IV presents the research and analysis. The chapter begins by defining the conceptual framework of the research and detailing the research methodology. The chapter culminates in a presentation of the research findings with a discussion in which policy implications of the findings are highlighted. Chapter V is the conclusion, summarizing the findings and policy implications, and offering further areas of research.

Note: Because of the different health outcomes experienced by infants of African-born Black mothers as compared to U.S.-born Black mothers, the term “African-American” is inexact and confusing. This paper will, therefore, reference mothers’ country of origin when relevant, and will use the following racial/ethnic classifications: “Black” to refer to Non-Hispanic Black Americans of African or Caribbean descent, “White” to refer to Non-Hispanic White Americans of European descent, “Hispanic” to refer to Americans of Mexican, Puerto Rican, Cuban, or Central/South American descent, “Asian/Pacific Islander” to refer to Americans of East Asian, Southeast Asian, or Pacific Islands descent, and “American Indian/Alaska Native” to refer to Americans descended from U.S. indigenous groups.

CHAPTER II

BACKGROUND LITERATURE

Poverty and Children's Health

The adverse effects of poverty on children's health are well-documented and represent part of a larger phenomenon: the SES-health relationship, in which groups with lower SES face a greater risk of disease and death than do groups with higher SES in that society (T. Evans, Whitehead, Diderichsen, Bhuiya, & Wirth, 2001; Sorlie, Backlund, & Keller, 1995). The strong inverse relationship between SES and risk of disease extends to a wide range of diseases, across all stages of life, for most measures of socioeconomic status (Kitagawa & Hauser, 1973). This relationship is particularly burdensome for children--children are both more likely to be poor *and* more vulnerable to the negative effects of poverty (CDF, 1994; Geltman et al., 1996).

The Socioeconomic Status--Health Relationship

The strong *inverse* relationship between SES and risk of disease, or alternatively, the *direct* relationship between SES and health status, has been one of the most enduring relationships in health literature (Diderichsen, Evans, & Whitehead, 2001; Kaplan, Haan, Syme, Minkler, & Winkleby, 1987; Kaplan, Pamuk, Lynch, Cohen, & Balfour, 1996; J. Lynch & Kaplan, 2000; Macintyre, 1997; Syme & Berkman, 1976). It has been labeled "socioeconomic inequalities in health" (Adler, Boyce, Chesney, Folkman, & Syme, 1993), "social deprivation and health" (Eames, Ben-Shlomo, & Marmot, 1993), "health

inequities” (T. Evans et al., 2001; Hofrichter, 2003), and “health inequalities” (Davey Smith, 2003). The relationship, however, may best be understood as an “SES-health gradient” (Goodman, 1999; M. G. Marmot, Shipley, & Rose, 1984); that is, “systematically poorer health and a shorter life span . . . with each successively lower position in any given system of social stratification” (Diderichsen et al., 2001, p. 13).

An Overview of the Socioeconomic Status--Health Relationship

Research clearly establishes that, as compared to those with higher SES in their society, people with lower SES face a greater risk of health problems ranging from cardiovascular disease (Kaplan & Keil, 1993) and infectious diseases (CDF, 1994; L. V. Klerman, 1991) to neural tube defects (Wasserman, Shaw, Selvin, Gould, & Syme, 1998) and mental illness (Goodman, 1999). In addition to a greater risk of acquiring disease, individuals with lower SES are also diagnosed at later stages of disease (Farley & Flannery, 1989; J. Mandelblatt, Andrews, Kao, Wallace, & Kerner, 1996), experience a greater severity of disease (Brekke et al., 1999), worse prognosis following illness (Kovingas, Marmot, Fox, & Goldblatt, 1991; Schrijvers, Mackenbach, Lutz, Quinn, & Coleman, 1995; R. D. Williams et al., 1992), more significant functional limitations during their lifetimes (Guralnik, Land, Blazer, Fillenbaum, & Branch, 1993; Liao, McGee, Kaufman, Cao, & Cooper, 1999; J. W. Lynch, Kaplan, & Shema, 1997), and a greater risk of death (Eames et al., 1993; M.G. Marmot & Shipley, 1996; McDonough, Duncan, Williams, & House, 1997; Sorlie et al., 1995). This relationship exists even in the presence of universal access to health care (Bauman, Silver, & Stein, 2006; Pincus, Esther, DeWalt, & Callahan, 1998; Seguin et al., 2005), regardless of how SES is

measured (i.e., income, educational attainment, or occupation) (Davey Smith, Hart, Hole, et al., 1998; Lantz et al., 1998; Pincus & Callahan, 1994).

The SES-health relationship is pervasive, consistent across both time and place. As early as the Middle Ages, a relationship between SES and health was recognized when Paracelsus noted disproportionately high rates of disease among miners in Europe in the mid-16th century (J. Lynch & Kaplan, 2000). By the 19th century, data clearly documented a poverty-health relationship (Berkman & Kawachi, 2000; Chapin, 1924; Davey Smith, Carroll, Rankin, & Rowan, 1992; J. Lynch & Kaplan, 2000). In the 1840s, Villerme (cited in J. Lynch & Kaplan, 2000) associated area rent levels with mortality rates in Paris, while Farr (cited in J. Lynch & Kaplan, 2000) linked mortality rates to occupation in England. More modern researchers have also examined the historical existence of the SES-health relationship. In 1924, Providence, Rhode Island's Superintendent of Health examined local Census Bureau data from 1865, finding mortality rates were more than double among nontaxpayers (tax-exempt lower income individuals) than among taxpayers (higher income individuals) (Chapin, 1924). More recently, Davey Smith et al. (1992) and colleagues correlated the height of Glasgow graveyard obelisks (a surrogate for wealth) dating between 1800 and 1920 with the individual's age at death: those with taller grave markers (presumably wealthier families) were older at death (Davey Smith et al., 1992). Clearly, the relationship between SES and health has existed through recorded history, indicating that, while risk factors for disease have changed across time, they have consistently clustered among those in lower SES groups (T. Evans et al., 2001; J. W. Lynch, Kaplan, & Salonen, 1997).

In addition to constancy across time, the SES-health relationship has also been consistent across place, affecting all countries regardless of stage of development--from “developed” affluent countries to “developing” countries with marked material deprivation (Diderichsen et al., 2001; T. Evans et al., 2001; Mackenbach et al., 2005). The existence of an SES-health relationship *within* all countries, however, does not assert an SES-health relationship *between* countries: the SES-health relationship is a relationship between SES and health among people *within* a society, **not** *between* societies (see M. Marmot, 2001). Consider that “poor people in the United States are rich by world standards, but they have worse health than the average in some poor countries” (M. Marmot, 2001, p. 135). In 1990, for example, Black men in Harlem, with an average annual family income of \$24,174, were half as likely to survive to 65 years of age as their Black male counterparts in Queens-Bronx where the average annual family income was \$51,606 (Geronimus, Bound, Waidmann, Hillemeier, & Burns, 1996)--consistent with the known SES-health relationship; but, Black men from Harlem were *also* less likely to survive to age 65 than men in Bangladesh (McCord & Freeman, 1990) where the 1990 average annual family income was only \$200 (“Bangladesh,” 2007). Evidence such as this has raised the question of whether risk of disease and death is associated with *absolute income* (an individual’s material circumstances) or *relative income* (income distribution within a society), or *both*.

The Distinction Between Absolute Income and Relative Income

While the adverse health effects of low absolute income--that is, low individual income--and its subsequent deprivation in material living standards have been extensively documented (see, e.g., Aber et al., 1997; Betson & Michael, 1997; CDF, 1994; Geltman

et al., 1996; Hillemeier, Lynch, Harper, Raghunathan, & Kaplan, 2003; L. V. Klerman, 1991; Seguin et al., 2005;), some evidence suggests that disparities or gaps in income distribution, or “income inequality,” also adversely affect health (see, e.g., De Vogli, Mistry, Gnesotto, & Cornia, 2005; Kaplan et al., 1996; Kennedy, Kawachi, & Prothrow-Stith, 1996; Kawachi, 2000; J. W. Lynch et al., 1998; Ronzio, Pamuk, & Squires, 2004; Wilkinson, 1997a).

Absolute income. “[Absolute] income affects health because it influences individuals’ consumption of commodities that affect health” (Gravelle, 1998, p. 383). In other words, low absolute individual income, or poverty, adversely affects an individual’s ability to purchase health-influencing “commodities,” such as housing, nutrition, sanitation, and medical care, which in turn adversely affects health. That material deprivation is associated with poverty is generally known, and its adverse relationship to health is both intuitive and well-documented (see, e.g., Fiscella & Franks, 1997; Hillemeier et al., 2003; Schrijvers, Stronks, van de Mheen, & Mackenback, 1999; Sorlie et al., 1995).

Relative income. Ecological studies in the 1970s began to explore the role of income distribution, or relative income, on health. This research asserted that unequal income distribution--that is, income inequality--is correlated with higher mortality rates and lower life expectancy (Le Grand, 1987; Rodgers, 1979). Subsequent research seemingly confirmed the association between greater income inequality and increased mortality rates (Eames et al., 1993; Kaplan et al., 1996; J. W. Lynch et al., 1998; Wilkinson, 1986, 1990)--particularly homicide rates (Kennedy et al., 1996)--as well as a correlation of income inequality with: lower life expectancy (Wilkinson, 1992), poorer

self-rated health (Kennedy, Kawachi, Glass, & Prothrow-Stith, 1998), and worse birth outcomes (Kaplan et al., 1996). These findings suggested that income inequality adversely affects the health of the entire population of less egalitarian societies, not just those with lower incomes in those societies (Mackenbach, 2002).

While there is a body of research seemingly supporting the relative income hypothesis, there is, at present, no uniform statement regarding the relationship between income inequality and health. Some assert that relative income affects mortality more than does absolute income in developed countries (Wilkinson, 1997a). But research finding that relative income “contributes an additional effect on mortality over the effect of deprivation alone” (Ben-Shlomo, White, & Marmot, 1996, p. 1014; see also, Wolfson, Kaplan, Lynch, Ross, & Backlund, 1999), or that there is a “contextual effect of state income inequality on individual mortality risk” (Lochner, Pamuk, Makuc, Kennedy, & Kawachi, 2001, p. 389), suggests the role of relative income is additive rather than primary over absolute income. In addition, some research has found the relationship exists only for specific subpopulations; for example, that income inequality impacts mortality only among nonelderly, middle-class adults (Daly, Duncan, Kaplan, & Lynch, 1998), and that the relationship of income inequality with self-reported health exists only among young and middle-aged White adults in the United States (LeClere & Soobader, 2000). Still other research has raised the possibility that the relationship between income inequality and health may exist only in the United States, and not in other developed countries (see Subramanian & Kawachi, 2004), such as Canada (Ross et al., 2000), Denmark (Osler et al., 2002), Japan (Shibuya, Hashimoto, & Yano, 2002) or New Zealand (Blakely, O’Dea, & Atkinson, 2003).

Some have questioned whether relative income plays a role in health or mortality at all--that is, whether relative income has an independent relationship with mortality or whether it is instead a statistical artifact stemming from the well-established relationship between absolute income and health (Gravelle, 1998; Judge, 1995). This assertion has been supported by research demonstrating that only individual income, not income inequality, is associated with mortality (Fiscella & Franks, 1997). Research exploring the roles of relative and absolute income among children arrived at a similar conclusion, finding that infant and child mortality are more closely linked to absolute income--that is, a family's ability to meet basic needs--than to relative income (Hillemeier et al., 2003). Others have questioned whether income inequality may actually "reflect the effects of other socioeconomic variables that are also related to mortality" such as education--as in research finding that education is a better predictor of mortality than is income inequality (Muller, 2002, p. 23). These findings were echoed by research finding that chronic medical conditions were associated with educational attainment and family income, but not with income inequality (Sturm & Gresenz, 2002).

Adding to the debate are questions over which unit of analysis (i.e., ecologic or individual) and what level of geographic aggregation (i.e., metropolitan area, county, state, country, cross-country) are appropriate to determine the income inequality, as different effects have been noted for different units of analysis and at varying levels of aggregation (Soobader & LeClere, 1999). Finally, one attempt to reanalyze earlier research supporting the relative income hypothesis did not find a relationship between income inequality and population health when using first difference modeling of data

across several decades rather than the cross-sectional analysis of single-year data performed by other researchers (Mellor & Milyo, 2001).

The debate regarding the roles of absolute and relative income in health is far from over, and has prompted some researchers to suggest that there are actually “two types of problems to understand and address . . . the relation between inequality and ill health . . . [and] the relation between poverty and ill health” (M. Marmot, 1996, p. 48). If there are indeed two problems--or two aspects of a single problem--the question remains: how do they affect health? By what mechanisms do *social* factors such as SES affect an individual’s *physiology* or *biology*, and ultimately health status?

Mechanisms of the Socioeconomic Status--Health Relationship

The mechanisms of the SES-health relationship are not well understood. Given the complexities of dealing with the overlay of social and biological processes, some have promoted a multilevel approach to understanding the SES-health relationship or “social determinants of health” that encompass both individual attributes *and* community level attributes (M. Marmot, 2000). An alternative way of understanding the mechanisms or pathways of the SES-health relationship is exploring *how* SES is measured. SES is a complex, multidimensional phenomenon that is influenced by countless variables, and can be measured by a variety of indicators “each reflect[ing] somewhat different individual and societal forces associated with health and disease” (Winkleby, Jatulis, Frank, & Fortmann, 1992, p. 816).

A multilevel approach to the SES-health relationship. Exploring the mechanisms of the SES-health relationship through a multilevel approach “entails an understanding of how society operates, an appreciation of the major causes of diseases under study, [and]

an understanding of psychological processes and how they may interact with relevant biological mechanisms” (M. Marmot, 2000, p. 365). Such an approach requires a conceptual framework that recognizes the health effects of both individual attributes *and* community-level attributes. These health-influencing attributes, or determinants of health, can be viewed as a series of concentric circles, moving outward from *individual attributes* (biological, behavioral, and psychosocial factors), to *social relationships* (family structure, support networks), to *physical environment* (living and working conditions, community characteristics), to *institutions* (educational systems, medical care/public health systems) and, finally, to *macrosocial factors* (cultural norms, social stratification, public policies). Factors within each of these levels impact health to varying degrees. While a full discussion of these factors and levels is beyond the scope of this paper, what follows is a brief overview of this framework for the SES-health relationship.

1. *Individual attributes.* Individual attributes related to health status can generally be categorized as biological, behavioral, and psychosocial factors (J. W. Lynch, Kaplan, Cohen, Tuomilehto, & Salonen, 1996).

Biological factors encompass such factors as genetics and specific biological processes linked to adverse health occurrences. Research demonstrates that some biological processes linked to adverse health outcomes occur more frequently in lower SES groups, which places these individuals at an increased risk of specific diseases. Biological processes that are disproportionately high among lower-income individuals include stress-related responses and risk factors for coronary heart disease, such as: elevated plasma fibrinogen (Steptoe, Kunz-Ebrecht, Owen, Feldman, Rumley, et al.

2003), elevated systolic blood pressure, and elevated cortisol levels in both adults (Steptoe, Kunz-Ebrecht, Owen, Feldman, Willemsen, et al., 2003) and in children (Lupien, King, Meaney, & McEwen, 2000).

Behavioral factors, or more accurately behavioral *risk* factors, such as diet, physical activity, and smoking status, are well known for their relationship to health status. Extensive research has demonstrated that behaviors known to increase the risk of disease (e.g., obesity, sedentary lifestyle, alcohol consumption, and cigarette smoking) are more common among individuals in lower SES groups (Davey Smith, Hart, Hole, et al., 1998; Lantz et al., 1998; J. W. Lynch, Kaplan, Cohen, et al., 1996). Although health risk behaviors are associated with adverse health effects and occur disproportionately among lower SES individuals, research suggests the influence of these behaviors “explains only a modest proportion”--12-13% in one study--of the relationship between SES and adverse health outcomes (Lantz et al., 1998, p. 1707).

Psychosocial factors such as depression, hostility, and social isolation are more pervasive among individuals in lower SES groups. These factors have been increasingly linked to adverse health outcomes, such as all-cause mortality (Berkman, 1995), with researchers theorizing such potential pathways as neuroendocrine regulation and immune response (Berkman, 1995; T. Evans et al., 2001). These factors overlap with social relationships discussed more completely later under *Community-level attributes*.

Clearly, various risk factors in these three categories of individual-level attributes adversely affect health *and* occur disproportionately among lower SES groups. Identifying these factors, however, does not explain *why* these factors or “constellation of characteristics” (J. W. Lynch et al., 1996) occur more among poorer people. Nor do

these identified factors appear to fully explain the relationship between SES and health. Other factors, conditions, or circumstances must also play a role in the SES-health relationship.

2. *Community-level attributes.* In addition to the above-noted individual attributes, factors external to the individual also affect health, and, therefore, play a role in the SES-health relationship. These external, or community-level attributes, can be viewed as layers moving outward from the individual, from those closest to the individual or having the most immediate effect on the individual, to those further away with a more indirect effect. These concentric circles, or layers, of community attributes include *social relationships, physical environment, institutions, and macrosocial factors.*

Social relationships and the role of the social environment on health are incorporated in much of the above-referenced research on individual-level psychosocial factors. The presence of social relationships, in the form of social cohesion (Kawachi & Kennedy, 1997; Wilkinson, 1997b), social support and networks (Berkman, 1995; Berkman & Syme, 1979; Seeman, Kaplan, Knudsen, Cohen, & Guralnik, 1987), and social capital (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Pearce & Davey Smith, 2003), have all been found to positively affect health: that is, being socially integrated has a positive effect on health. Beginning with Durkheim's seminal 1897 work linking elevated suicide rates with the lack of social integration, research has demonstrated that lack of social integration--that is, social isolation--is linked to adverse health occurrences ranging from cardiovascular disease and stroke to HIV/AIDS and premature death (Berkman & Glass, 2000; Kawachi & Berkman, 2000). Potential pathways for the effect of social environment on health include behavioral pathways

(such as influence on health behaviors) (Berkman & Glass, 2000; Berkman & Syme, 1979) and biological processes altering host susceptibility (such as neuroendocrine regulation and immune responses) (Berkman & Glass, 2000; Berkman & Syme, 1979; T. Evans et al., 2001).

The *physical environment*--the physical conditions in which individuals live and work--affects health both directly and through its effect on the social environment.

Physical environment--living conditions. Inadequate or substandard housing has long been associated with ill health (Srinivasan, O'Fallon, & Dearry, 2003) including asthma, lead poisoning, neurological conditions, and behavioral dysfunction, among others (Bashir, 2002; Kim, Staley, Curtis, & Buchanan, 2002). These conditions fall disproportionately on children of color and those in lower SES groups (Bashir, 2002)--further evidence of the SES-health relationship. In addition to the design and quality of buildings, the "built environment" and land use--including the availability and distribution of sidewalks, parks, community facilities, food stores, transportation, traffic patterns (Frumkin, 2003)--are linked to health through physical activity levels (Handy, Boarnet, Ewing, & Killingsworth, 2002), healthy food choices (Morland, Wing, Diez Roux, & Poole, 2002), motor vehicle injuries (Srinivasan et al., 2003), social cohesion, and other factors. Other community characteristics such as proximity to nature (with its positive health effect) (Frumkin, 2003) or to industry and toxic emissions (with their adverse effect on air and water quality) (Srinivasan et al., 2003) also play a role in the SES-health relationship. High levels of air pollution, for example, have been associated with adverse respiratory health effects among young children (Peled et al., 2004) and low birth weight among infants (Lin, Li, & Mao, 2004).

Physical environment--working conditions. In addition to living conditions, the physical environment also incorporates working conditions which also affect health. The link between the hazardous exposures and hardships of manual laborers and subsequent illness was recognized as early as Ancient Egypt and by such 1st century physicians as Hippocrates and Pliny (Abrams, 2001). Documentation of the impact of working conditions on worker health began with 15th century European literature that detailed various occupational diseases suffered by miners, smelter workers, and other manual laborers (Abrams, 2001). It was not until 1713, however, that the “first comprehensive presentation of occupational diseases” was undertaken by Italian physician, Ramazzini (Abrams, 2001, p. 39). In his seminal work, Ramazzini chronicled the link between working conditions and their adverse health effects, and supported the concepts of “guilds and corporations of workers” and “laws . . . to secure good conditions for the workers” (Abrams, 2001, p. 40)--reforms that would not begin to take root for nearly a century. Some 50 years after Ramazzini’s treatise, the Industrial Revolution, with its technologic developments, changed both the quality and the quantity of occupational hazards. Work in the factories, mills, and mines of the era, with their long hours, physical exertion, poor air quality, and hazardous exposures, increased workers’ risk of injuries (e.g., from mechanized equipment or fire), respiratory illnesses due to toxic inhalants (e.g., pneumoconiosis [“Black Lung”] among coal miners or byssinosis [“Brown Lung”] among textile workers), systemic illnesses as a result of toxic exposures (e.g., lead or mercury poisoning), malignancies (e.g., testicular cancer among chimney sweeps or bone cancer due to radium among watch dial painters), and ultimately death (Abrams, 2001). These work-related injuries and illnesses were chronicled by

physicians/epidemiologists in the mid-19th century--Virchow, Engels, Thackrah, and Chadwick--laying the groundwork for subsequent reform and policy change (Abrams, 2001). These efforts highlight the interconnectedness of the next two layers of health-influencing community attributes: *institutions* and *macrosocial factors*.

The primary *institutions* that affect health and, therefore, play a role in the SES-health relationship, are the educational, health care, and public health systems.

Institutions--education. Education has long been recognized as directly linked to health: those with higher levels of education have higher levels of health (J. Lynch & Kaplan, 2000). This link likely stems from a variety of factors associated with education--its association with higher income, the direct knowledge education provides, and the broader cognitive processes offered by education that affect health care utilization, among others (Davey Smith, Hart, Hole, et al., 1998; Diderichsen et al., 2001; J. Lynch & Kaplan, 2000;). Of particular interest for children's health is the role of maternal education:

Maternal literacy and schooling is known to be associated with a more efficient management of limited household resources, greater utilization of available health service, better health-care practices, lower fertility, and more child-centered caring behavior. . . . In general, therefore, women's education has an enormous effect on nutrition and health. (Mehrotra, 1997, p. 40)

Institutions--health care and public health systems. The other significant institutions, the *health care and public health systems*, both address health, but each with a different focus and set of responsibilities. The health care system is focused on "the care and treatment of the individual, while public health's central focus is on populations," and health care's charge is traditionally viewed as "cure" or treatment of disease while that of public health is prevention (Brandt & Gardner, 2000, p.708). Both

systems have enjoyed tremendous advances over the past decades to centuries: health care from its rudimentary medical techniques to its modern diagnostic procedures, pharmacologic agents, surgical and other treatment options, and biomedical research; and public health's expansion beyond its historical roles of sanitation reform, quarantine, disease tracking, and immunization campaigns, to its current activities in health surveillance, risk factor identification, monitoring of regulation compliance, and policy advocacy. Given the prominent role these two institutions play in the SES-health relationship, and in children's health specifically, these two institutions are explored more fully. In so doing, it may be instructive to view the often complementary roles of the health care and public health systems by considering one specific disease process: cervical cancer.

Institutions--health care and public health systems. The case of cervical cancer: As early as the mid-19th century, physicians tracking cervical cancer noted the cancer occurred more frequently among married women and prostitutes and only infrequently among young, unmarried women and nuns--the cancer, therefore, presumably involved sexual behavior (Martin, 1967; Rigoni-Stern, 1987). Early detection of cervical cancer was not possible, however, until the introduction of the "Pap smear" in 1943 (named for Dr. George Papanicolaou's technique of examining cervical cell smears for the presence of cervical cancer) (National Cancer Institute [NCI], 2004). The Pap smear and, to a lesser degree, subsequent improvements in medical treatment (cone biopsy, radiation, chemotherapy) have been credited with the dramatic decline in the cervical cancer death rate in the United States: from 18 per 100,000 women in 1950 to 11 in 1970 and to 4.6 in 2000 (Bernard, 2005). This noted decline, however, masks the fact that the onset of the

decline actually *preceded* the introduction of the Pap smear--a 36% decline between 1930 (when cervical cancer represented the most common cause of cancer death in U.S. women at 28 per 100,000) and 1950--with widespread use of the Pap smear not occurring until the late 1950s (Bernard, 2005; Gardner & Lyon, 1977; Janieck & Averette, 2001; B. Williams, 1978). Public health advocates use this data to support the instrumental role of public health and health education in the “pre-Pap” cervical cancer decline.

Nineteenth-century epidemiologic suggestions of an association between cervical cancer and sexual behavior have been confirmed in the last decade: medical researchers have found that the necessary causative agent is the sexually transmitted human papillomavirus (HPV) (Khan et al., 2005; NCI, 2004). A vaccine effective in preventing HPV infections was approved in 2006 and is now in use--a traditional public health function using a product of advanced medical research. The strides realized in the fight against cervical cancer, however, presuppose the existence of adequate health care and public health infrastructure. While the United States and other industrialized nations have experienced a dramatic decline in cervical cancer incidence and death rates over the past 5 decades, cervical cancer remains the second most common cancer (and third most frequent cause of cancer death) among women globally--with 80% of cases occurring in developing countries (J. S. Mandelblatt et al., 2002; NCI, 2004).

Institutions--health care and public health: SES issues. Despite the advances noted in the health care and public health systems, both systems are factors in the SES-health relationship through inequalities in: access to health care (e.g., higher rates of uninsured and underinsured among low-SES individuals); the availability of health care (e.g., more nonfinancial barriers to care for lower SES individuals utilizing public sector

services, such as longer wait times for appointments and longer waiting room times); presence of a usual source of care (e.g., more provider shortages and turnover in the public sector health care); and, the quality of health care provided (e.g., lower SES individuals experience more difficulty being referred to specialists and having satisfying communication with providers) (Guendelman & Pearl, 2001; Simpson et al., 2005).

Continuing with the cervical cancer example: there are disparities in both the detection of and survival of cervical cancer based on SES, source of insurance (i.e., public vs. private), and race (Bradley, Given, & Roberts, 2004), with the majority of cervical cancer occurring among those without recent Pap screening--often due to lack of access to care--and disproportionately affecting women living in “high poverty or low education” areas (Leyden et al., 2005).

Macrosocial factors. The final layer of community-level attributes affecting health, *macrosocial factors* are often distinguished from the other layers because they represent more “distal” social factors than the other layers’ more “proximal” social factors (Amick, Levine, Tarlov, & Walsh, 1995). Distal, yet pervasive, factors such as cultural norms, social stratification, gender/racial/ethnic status and relations, political philosophy, economic systems, and public policy provide a context in which we all live--a context that ultimately impacts our health. In other words, “the social and physical environment” in which we live and work

are influenced by cultural systems (including values, beliefs and rituals, and their meanings in a particular community), the political system that drives health policy and community organization, and economic systems and prosperity (which influence income distribution, housing, employment and other important determinants of health). (Patrick & Wickizer, 1995, p 68)

These largely intangible factors are often expressed via public policy. Which issues are chosen to be addressed and how they are addressed shape our social and physical environments and influence our health. Because of the significant role of public policy in the SES-health relationship, and because of this paper's focus on the role of public policy in health, several examples of public policies that address previously mentioned public health/SES-health issues follow: hazardous work environment, cervical cancer prevention, and inequalities in access to health care.

Macrosocial factors--public policy and work environment. As noted above, documentation of hazardous work environments and work-related injuries and illnesses began in the early 18th century, and accelerated in the 19th century as a result of the changes ushered in by the Industrial Revolution. This documentation helped inform the public as to the status of industrialized labor and lay the groundwork for later occupational safety reform and policies. With growing public awareness of the unchecked occupational hazards, reform efforts began in the 1830s targeting first the issue of child labor (Abrams, 2001). These efforts were state-level, with only some states enacting laws that regulated the minimum age and/or maximum hours worked by children (Abrams, 2001). Subsequent reform efforts remained at the state level and were incremental in their approach, addressing workers compensation, factory inspection, maximum work hours, and child labor (Abrams, 2001).

When the federal government began investigating industrial diseases at the turn of the 20th century through its Bureau of Labor, however, its findings heightened the public's calls for reform during this Progressive Era (MacLaury, 1981). But such efforts remained limited or industry-specific until Depression-era legislation established the

Bureau of Labor Standards “to promote safety and health for the entire work force” and brought sweeping changes that banned child labor, set a minimum wage (Federal Labor Standards Act of 1938), and allowed the Department of Labor to “ban contract work under hazardous conditions” (Walsh-Healey Public Contracts Act of 1936) (MacLaury, 1981, n.p.). The role of the federal government in promoting occupational health and safety continued to expand throughout the mid-20th century with a patchwork of protective legislation and culminated in the passage of the comprehensive Occupational Safety and Health Act of 1970 to “assure . . . safe and healthful working conditions” (MacLaury, 1981, n.p.). The story of occupational safety reforms demonstrates how changing public perceptions and values influence political will and action (or inaction) which, in turn, shapes our “social and physical environment” and ultimately our health.

Macrosocial factors--public policy and cervical cancer. The current controversy surrounding cervical cancer prevention also demonstrates the significant role public sentiment can play in policymaking, but raises issues of gender, religious ideology, and social stratification as well. Cervical cancer prevention, like many health problems, includes three aspects of prevention: *tertiary prevention* (medical care to minimize the effects of the disease), *secondary prevention* (screening to detect early disease in asymptomatic individuals), and *primary prevention* (measures to prevent the onset of the disease). Policies regarding tertiary prevention generally lie within the biomedical/health care system (such as treatment or rehabilitation protocols). Secondary prevention efforts are often shared by both health care and public health systems (such as defining recommendations for the frequency of Pap smears and HPV screening). Primary prevention efforts generally fall to the public health system (such as vaccine schedules

and protocols). It is cervical cancer's primary prevention measures that have raised the current controversy.

The HPV vaccine to prevent or reduce the risk of cervical cancer was approved for use by the FDA in 2006 for girls and women between the ages of 9-26 (Kaiser Family Foundation [Kaiser], 2008). In 2006, the Advisory Committee on Immunization Practices (ACIP) recommended the HPV vaccine be "routinely given to girls when they are 11-12 years old," recognizing that the vaccine is most effective when administered prior to initiation of sexual activity, and that vaccines, in general, are most effective when given to the population-at-large (creating "herd immunity") (CDC, 2006, n.p.). It was then, as immunization policies always are, up to the states to set their specific vaccine policy: whether it would be "mandatory" or "recommended"; what exemptions would apply if the vaccine is mandatory; the age it should be given; and, whether only girls--or girls and boys--should be vaccinated.

At the heart of the controversy that followed lies the fact that HPV is a sexually transmitted disease: some groups objected to making the vaccine mandatory, suggesting that HPV vaccination would increase sexual activity among teens. If, however, vaccines are not mandatory, many "non-objecting" adolescents may remain unvaccinated in part because insurance companies often do not cover the cost of optional vaccinations (an estimated cost of \$360 for the HPV vaccine)--thereby creating an enormous financial barrier for low-income individuals (Kaiser, 2008). Compounding this is the fact that Medicaid, the primary health care insurance for low-income women (ages 19 and over)--considers vaccines an "optional benefit," and coverage is, therefore, at the discretion of

states whether to cover. At present, half the states have elected to make the HPV vaccine mandatory (Kaiser, 2008), and the controversy continues throughout all states alike.

Macrosocial factors--public policy and health insurance coverage. Another health issue significantly impacted by public policy is that of health insurance coverage--particularly for children. Voluntary health insurance programs in the U.S. have evolved from the accident insurance programs of the Civil War era, to the earliest employment-based insurance plans of the 1910s, and on to the elaborate system of public and private health insurance programs we have today. Calls for government involvement in health insurance began in the early 20th century, when national health insurance represented a major component of former President Theodore Roosevelt's platform in his failed 1912 bid for re-election. Later efforts to establish a role for the federal government in health insurance were stalled for years: although President Franklin Delano Roosevelt's call for government protection from the "cradle to the grave" was to include health insurance for the aged, no such plan was included in the "New Deal"; and when President Harry S Truman became the first sitting U.S. President to publicly advocate national health insurance, a series of bills were introduced but did not pass Congress. It was not until 1960 that the federal government assumed a role in the provision of health insurance under the Kerr-Mills Act in which the federal government provided payments to states for medical programs for the aged who were not otherwise on public assistance--this was the precursor program to Medicare and, consequently, Medicaid (Trattner, 1999).

Medicare and Medicaid were established in 1965 under the Social Security Act of 1965 as joint federal-state entitlement programs providing health care coverage to vulnerable populations. While Medicare focused on health care for Americans aged 65

and older, Medicaid sought to “provide health care services to low-income children deprived of parental support, their caretaker relatives, the elderly, the blind, and individuals with disabilities” (Centers for Medicare and Medicaid Services [CMS], *Medicaid Milestones*, p.1). Medicaid was initially limited to recipients of the AFDC program--commonly referred to as welfare--but underwent expansions in eligibility criteria as well as services throughout the 1980s (Hakim, Boben, & Bonney, 2000).

After a failed 1977 attempt to expand Medicaid eligibility to all poor children under 6--regardless of AFDC status, “concern about high infant mortality and morbidity in the United States and the erosion of private insurance coverage sparked major expansions in the Medicaid program” during the 1980s (Dubay & Kenney, 1997, p.185). With these expansions, Medicaid eligibility was no longer limited to AFDC recipients, first extending eligibility to low-income pregnant women and later to both low-income women and children with increasingly higher levels of income (Hakim et al., 2000). It should be noted, however, that while poor and low-income children, and later women, were the focus of Medicaid and its expansions, the program is not limited to these populations, and that program spending is less for women and children than other enrolled populations (i.e., the elderly and the disabled). In 2005, children represented 48% of the 52 million Medicaid enrollees and adults represented 27%, but they only accounted for 19% and 12% (respectively) of the \$252 billion spending (Kaiser, n.d.). The other primary enrollment groups, the elderly and the disabled, instead represented only 9% and 16% of Medicaid enrollment, respectively, but accounted for 24% and 43% of the spending--in other words, these two groups represented roughly one-quarter of the enrollees but more than two-thirds of the spending (Kaiser, n.d.).

Medicaid was explicitly “de-linked” from AFDC in 1996 with the passage of PRWORA--Welfare Reform. Concern ensued, however, as Medicaid enrollment declined and the number of uninsured children in “working poor” families rose (Hakim et al., 2000). In 1997, policymakers responded to this concern by establishing the State Children’s Health Insurance Program (SCHIP) which provides health insurance for uninsured children from low-income working families whose incomes are too high to qualify for Medicaid (Urban Institute, 2005b). Since the implementation of SCHIP, the percentage of uninsured low-income children (< 18 years) has dropped from 23% in 1997 to 14 % in 2005 (Kaiser, 2007) resulting in improved access to health care services and health care utilization (particularly preventive services) for these low-income children (Urban Institute, 2005b). As of 2005, Medicaid provided health insurance for 28 million poor- and low-income children and SCHIP for 6 million low-income children (Kaiser, 2007). Despite this success, an estimated 9 million low-income children remain uninsured--a key issue during debates surrounding the 2007 re-authorization of SCHIP (Kaiser, 2007). These debates highlighted some of the macrosocial forces at issue in public policy: cultural views on responsibility for children, political ideology regarding the role of government, and, priority-setting in the face of an economic downturn.

Pathways for community attributes. As for how these community-level attributes affect health, several “pathways from the social context to health outcomes” have been proposed (Diderichsen et al., 2001, p. 15). Some researchers have theorized a pattern of differential health-damaging exposures between social strata or income groups (Diderichsen et al., 2001). These “differential exposures” range from greater occupational exposure to hazardous conditions among manual/lower income workers to

increased exposure to infectious agents among those living in impoverished settings (Diderichsen et al., 2001). Other researchers theorize a “differential vulnerability,” suggesting that “even when a given risk factor is distributed evenly across social groups, its impact on health may be unevenly distributed due to underlying differences between social groups in their vulnerability or susceptibility to that factor” (Diderichsen et al., 2001, p. 17). Some have expanded this theory to a “hypothesis of generalized susceptibility,” suggesting that the higher rates of seemingly unrelated diseases among lower “social classes” may result from a common underlying cause--such as “various forms of life change and life stress” (Syme & Berkman, 1976, p. 5). These theories are not mutually exclusive, and may both play roles in the SES-health relationship.

Using SES measures to understand the SES-health relationship. Although the mechanisms of the SES-health relationship are not entirely understood, a discussion of the mechanisms or pathways of the SES-health relationship is intertwined with *how* SES is measured. SES is a complex, multidimensional phenomenon influenced by countless variables, and measured by a variety of indicators. While the SES-health relationship exists “regardless of how SES is measured (i.e., income, educational attainment, or occupation)” (Davey Smith, Hart, Hole, et al., 1998; Lantz et al., 1998; Pincus & Callahan, 1994), that is not to say that all measures of SES measure the same concepts, or that they reflect the same pathways in their relationship to health (Liberatos, Link, & Kelsey, 1988; Winkleby et al., 1992; see, e.g., Singh-Manoux, Clarke, & Marmot, 2002). While these three measures of SES--income, occupation, and education--represent various dimensions of SES and are mutually associated (Davey Smith, Hart, Hole, et al., 1998),

each reflects somewhat different individual and societal forces associated with health and disease . . . income reflects spending power, housing, diet, and medical care; occupation measures prestige, responsibility, physical activity, and work exposures; and education indicates skills requisite for acquiring positive social, psychological, and economic resources. (Winkleby et al., 1992, p. 816)

Although different approaches have been developed to address the multidimensionality of SES--such as, a composite index of several indicators or a weighting of multiple measures--*how* SES is measured “is likely to affect the association obtained with the disease or risk factor under study” for some conditions and may actually vary the SES-health relationship (Liberatos et al., 1988, p. 107). Some research, for example, has found that the strength of association between SES and various health behaviors in pregnant women varies according to which SES measure is used (Gazmararian, Adams, & Pamuk, 1996). Other research has found mortality to be a function of income at lower SES levels, but a function of education at higher SES levels (Backlund, Sorlie, & Johnson, 1999). What follows is a brief discussion of each of the three primary SES measures (education, occupation, and income) and their relevance to understanding the pathways of the SES-health relationship as well as the challenges of measuring SES in children.

1. *Education.* Although a single indicator of SES cannot offer the reliability or accuracy of composite or multiple measures, education has been shown to be the “most judicious SES measure” when using a single SES indicator (Winkleby et al., 1992). Education has been deemed to be highly reliable and valid, and has the added advantages of easy reporting, applicability regardless of employment status, and, in general, stability after early adulthood (Winkleby et al., 1992). In addition to these aspects of education as an SES indicator, education also appears to be relatively comparable internationally, and

is, therefore, considered a more useful indicator in cross-country comparisons than occupation (Kunst & Mackenbach, 1994; Valkonen, 1993).

While occupation and income are generally considered measures of adult SES (Davey Smith, Hart, Hole, et al., 1998), educational attainment typically provides some measure of SES levels earlier in life--that is, educational attainment often reflects parental SES levels and, therefore, access to educational opportunities (J. Lynch & Kaplan, 2000). An individual's educational achievement affects future occupation and income, which in turn affect working conditions, quality of living conditions and neighborhood, and the ability to purchase other health influencing commodities (J. Lynch & Kaplan, 2000; Davey Smith, Hart, Hole, et al., 1998). Despite the relationship between educational attainment and future occupation and income, it should be noted that education represents *potential* for occupation and income--a potential that is not always realized (Liberatos et al., 1988; Sorlie et al., 1995).

In addition to the indirect health influences (via occupation and income) mentioned above, education may also provide less tangible "goods" that in turn affect health--such as direct knowledge regarding health-related behaviors, broader cognitive resources affecting knowledge acquisition and utilization of health services, and psychological processes of self-esteem and self-efficacy (Diderichsen et al., 2001; Davey Smith, Hart, Hole, et al., 1998; J. Lynch & Kaplan, 2000; Winkleby et al., 1992). Education affects the knowledge of and practice of health-related behaviors: research demonstrates higher rates of behavioral risk factors (such as smoking, low physical activity, poor diet, and alcohol use) among individuals with little education (see, e.g., Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989). Some research, however, has

brought into question the relative contribution of this concentration of behavioral risk factors in the relationship between lower educational attainment and higher mortality rates. This research, while confirming the inverse relationship between education and mortality, suggests that behavioral factors account for some of this relationship, but that material factors account for a larger part of the association between education and mortality (Lantz et al., 1998; Schrijvers et al., 1999).

Despite its usefulness as an SES indicator, concern has been raised over the validity of education as an SES indicator in cross-cohort comparisons due to the variation of educational attainment between age cohorts given that education has become increasingly more accessible over time (Liberatos et al., 1988). Another cautionary note on the use of education as a measure of SES comes from research that has suggested the relationship between health and education (and income) declines after age 65 (House et al., 1994; Sorlie et al., 1995), although this finding is not universal (see, e.g., Feldman, Makuc, Kleinman, & Cornoni-Huntley, 1988). An additional issue regarding the use of education as an SES indicator relates to the question of how education is best measured--that is, whether the most appropriate measure is the continuous variable of number of years of schooling (e.g., < 12 years, > 16 years) or a discrete classification (e.g., degree or certification) that confers occupational opportunities (Liberatos et al., 1988).

2. *Occupation.* Research on the relationship between employment/occupation and health extends back to at least the Industrial Revolution when the poor working conditions of mines, mills, and factories were found to have negative health consequences (J. Lynch & Kaplan, 2000). Over time, however, research has transitioned from a focus on the adverse health effects of the physical workplace characteristics--such

as physical exertion and hazardous exposures--to a greater focus on the role of the organizational and psychosocial aspects of work--such as “work stress” (J. Lynch & Kaplan, 2000; Sorlie et al., 1995; Theorell, 2000). Occupational sources of stress--such as lack of control or autonomy and high workplace demand with low economic return--have been linked to adverse health outcomes, including cardiac disease and mortality (J. Lynch & Kaplan, 2000; J. Lynch, Krause, Kaplan, Tuomilehto, & Salonen, 1997; M. G. Marmot & Shipley, 1996).

Employment grade, or level of occupational classification, has been linked to differential health outcomes, with lower grades (e.g., manual laborers) having higher mortality rates and health-risk behaviors (e.g., smoking) than do higher grades (Davey Smith, Hart, Hole, et al., 1998; M. G. Marmot & Shipley, 1996). The varying health behaviors found among the different employment grades (Blane, Hart, Davey Smith, Gillis, Hole, & Hawthorne, 1996) has led some to suggest that, beyond the health effects of workplace characteristics for an occupation, there exists a “social environment” associated with occupation or employment grade that also affects health behaviors (Davey Smith, Hart, Hole, et al., 1998). Although occupation as an SES measure has been used extensively in British research (Davey Smith, Hart, Hole, et al., 1998; M. G. Marmot & Shipley, 1996; Sorlie et al., 1995; see e.g., Schrijvers et al., 1999; Sturm & Gresenz, 2002), some have questioned the validity of various classifications of occupations--pointing to the potential for subjectivity in determining classifications and the failure to account for changes in educational requirements for and income generation by occupations over time (Liberatos et al., 1988).

In addition to the adverse health effects of lower employment grades, changes in work status or work environment also appear to adversely affect health (Kasl & Jones, 2000). Loss of employment--unemployment, lay-offs, and early retirement--is more common among unskilled and semi-skilled (lower wage) workers ("blue collar") than among managerial and clerical workers ("white collar") (Bartley & Owen 1996). In general, loss of employment has been linked to adverse health effects, such as: increased mortality rates from both cancer and cardiovascular disease (even when the loss of employment was unrelated to illness) (Morris, Cook, & Shaper, 1994); suicide (Lewis & Sloggett, 1998); higher health-risk behaviors; and, mental health problems (Kasl & Jones, 2000; Wadsworth, Montgomery, & Bartley, 1999)--all adverse health effects that appear to persist for years (Wadsworth et al., 1999). Even the anticipation of or threat of job loss--that is, job insecurity--has been linked to adverse health outcomes, such as a decline in self-reported health (Ferrie, Shipley, Marmot, Stansfeld, & Davey Smith, 1995).

3. *Income.* Whereas occupation often flows from educational attainment, income level usually results from both education and occupation. Income--that is absolute or individual income--directly affects "material conditions" which in turn affect health (J. Lynch & Kaplan, 2000). "Income affects health because it influences individuals' consumption of commodities that affect health" (Gravelle, 1998, p. 383). These health-influencing commodities range from housing and clothing to nutrition and sanitation to child care and medical care, among others (J. Lynch & Kaplan, 2000). The concept of a relationship between adequate material conditions and health stems from a long-standing understanding or framework that recognizes the need for adequate living conditions and food as well as appropriate sanitation for safe water and waste removal to maintain health

(J. Lynch & Kaplan, 2000). Although there is some debate as to whether income versus wealth is more relevant to health, or whether there is an income level at which SES-health effects plateau, in general, higher income means increased purchasing power for health-influencing goods and services and, therefore, better health (J. Lynch & Kaplan, 2000).

As discussed above, some research suggests that the effect of income extends beyond material conditions and individual income (absolute income) to the relative position of income within society (relative income) and the degree of income inequality within a society. Two primary--perhaps mutually reinforcing--theories have been offered as to mechanisms underlying a relationship between levels of income inequality and health. The first is the so-called “*neo-material*” *explanation* (J. W. Lynch, Davey Smith, Kaplan, & House, 2000), describing an underinvestment in social spending and health-promoting infrastructure such as education, housing, and medical care (Davey Smith, 1996; Kaplan et al., 1996; J. W. Lynch et al., 1998). The underinvestment, or disinvestment, in these neo-material factors, the theory asserts, leads to adverse health consequences for lower income individuals (Davey Smith, 1996; Kaplan et al., 1996; J. W. Lynch, Davey Smith, et al., 2000, 1998; Shi et al., 2004). In other words, while the level of economic resources is related to health status, “the way in which [societies] *allocate* these resources also matters” (Carrin & Politi, 1997, p. 4). The second primary theory involves *psychosocial processes* of inequality, such as increased frustration and perception of deprivation (Wilkinson, 1994), erosion of social capital--“the features of social organization . . . which facilitate cooperation for mutual benefit” (Kawachi et al., 1997, p. 1491), and lack of social cohesion or social integration (Berkman & Glass, 2000;

Kawachi & Berkman, 2000). This theory asserts that less egalitarian societies are less socially cohesive--adversely affecting trust levels, cooperation, and psychosocial stress, and ultimately compromising health (Shi et al., 2004; Berkman & Glass, 2000; Kawachi & Berkman, 2000; Kawachi et al., 1997).

Concerns about the use of income as an SES measure extend beyond the debate regarding the appropriate roles of absolute income versus relative income in the SES-health relationship. Critics of income as a single measure of SES point to the potential unreliability of data due to its sensitivity, the age-dependence of income, and the variations of income both *within* occupations (same job but different incomes) and *between* occupations with widely varying educational requirements (different jobs with income levels that are unrelated to educational level) (Liberatos et al., 1988). Other critics view the use of income as measuring a single-point in adult life, rather than capturing “the health effects of sustained exposure to low income” (J. Lynch & Kaplan, 2000, p. 26). Such concerns are encompassed in the “lifecourse perspective” accounting for the accumulation of risk throughout life, discussed later in the paper.

4. *SES measures for children.* Measuring SES among children presents not only the challenges noted for each individual SES measure discussed above, but includes issues beyond these described limitations or criticisms. First, when considering a dependent child, any SES measure generally refers to family or parental characteristics rather than the child’s individual SES measure. A child typically has little or no control over these family SES measures or the distribution or allocation of family resources. And consider that both parental education and occupation measures are susceptible to

potential discrepancies between parent and child status, such as a college-bound teen from a “working class” family (Liberatos et al., 1988).

Additionally, as noted above, different SES measures likely reflect different concepts and pathways in their relationship to health--with the additional complexity that the relationship is with a child’s health by way of a parent’s resources (Winkleby et al., 1992). An example of the varying pathways of different SES measures can be seen in the case of parental SES and a child’s later cognitive functioning and risk of dementia. One study has found that, while both maternal education and paternal occupation are both related to a child’s subsequent cognitive functioning and risk of dementia, it is through different pathways: maternal education is directly related to adult cognitive functioning through its influence on the child’s cognitive development, while paternal occupation is indirectly related to later cognitive functioning through its influence on the child’s subsequent educational attainment (Kaplan et al., 2001).

While some researchers consider the most appropriate SES measure for children to be the highest household educational attainment (maternal or paternal), and others use maternal occupation rather than education (Liberatos et al., 1988), a very large body of literature identifies maternal education as the most significant SES measure for child health--particularly in developing countries (Bicego & Boerma, 1993; Cleland & van Ginneken, 1988). Many scholars assert that maternal education has the greatest impact on child health as compared to all other measures of child SES (Liberatos et al., 1988). Maternal education--sometimes referred to as maternal schooling--is positively associated with a wide range of improved child health outcomes: higher child nutritional status (S.H. Cochrane, Leslie, & O’Hara, 1982); better health services utilization patterns (Bicego &

Boerman, 1993) including higher rates of immunizations (Racine & Joyce, 2007); better child health status as measured by height-for-age (Barrera, 1990); and, lower infant and child mortality rates, with an estimated decrease in child mortality of between 2-5% (S. Cochrane, O'Hara, & Leslie, 1982) and 6-9% (Cleland & van Ginneken, 2008) for each additional year of maternal education attained. There is a debate, however, as to the mechanism of maternal education's protective pathways. While some researchers assert that maternal education is a proxy for higher income with its increased access to material resources (Desai & Alva, 1988), others point to more intangible processes to account for the health-promoting behaviors surrounding sanitation, nutrition, reproduction, and health care utilization associated with maternal education. These researchers attribute maternal education's health-promoting effects to direct knowledge of disease etiology and health-related behaviors, broader cognitive resources affecting knowledge acquisition important to child survival, and psychological processes such as autonomy and empowerment (Basu & Stephenson, 2005).

Maternal education is not just related to child health and survival in the developing world context. In the United States, for example, maternal education is closely associated with IMR: in the U.S., the IMR for infants born to mothers with less than 12 years of education is twice that of infants born to mothers with 16 or more years of education (Singh & Kogan, 2007). The role of maternal education in infant mortality is discussed more fully in a later section.

Despite the documented protective effect of maternal education and its widespread acceptance and use as a measure of childhood SES, it does have limitations: the health protective effects offered by maternal education vary depending upon

“contextual variables” such as community resources (Dargent-Molina, James, Strogatz, & Savitz, 1994) or family income and resources (Reed, Habicht, & Niameogo, 1996; Ruel, Habicht, Pinstrup-Andersen, & Groh, 1992). Several studies have demonstrated that, while maternal education offers positive child health effects to middle- and higher-income populations, it offers *no* protective effects to the more “economically and socially” disadvantaged populations (Bairagi, 1980; Dargent-Molina et al., 1994; Ruel et al., 1992). These studies suggest that maternal education alone is not sufficient to overcome a lack of physical resources available to a woman and her family. Other studies have had similar findings--that is, the protective effects of maternal education were blunted or absent among poor (low income) populations--but with the additional finding that maternal education had no positive effect--or even had a *negative* effect--on child health among the relatively wealthiest women (Reed et al., 1996). Although no data accounts for this finding, it has been suggested that higher levels of education have allowed the relatively wealthier women to participate in market activities outside of the home, with less time spent on child caregiving in the home and an uncertain quality of caregiving offered (Reed et al., 1996).

While this body of literature stems from research done in developing countries, research from industrialized countries--including the United States--also demonstrates the adverse health effect of low family income on children, with some studies finding an independent relationship between family income and child health even when controlling for parental education (Aber et al., 1997; Seguin et al., 2005). Various studies have found low family income to be associated with higher rates of a number of medical conditions in children--unintentional injuries, high blood lead levels, ear infections and

hearing loss, developmental delays, hospital admissions--as well as elevated mortality rates during infancy and childhood (Aber et al., 1997; Mare, 1982; Rodwin & Neuberg, 2005; Seguin et al., 2005; Seguin, Xu, Potvin, Zunzunegui, & Frohlick, 2003). In sum, although several SES measures--particularly maternal education--appear to be associated with children's health, the most relevant factor for children and their health appears to be absolute income: that is, a family's ability to meet basic needs (Backlund et al., 1999; Hillemeier et al., 2003; see, e.g., Seguin et al., 2005; Seguin et al., 2003). Given the crucial--arguably pre-eminent--role that absolute income plays in children's health, this paper addresses the effect of low absolute income--poverty--on children and their health.

Child Poverty in the United States

“The concept of poverty is simple and familiar: a child living in poverty lacks goods and services considered essential to human well-being” (Betson & Michael, 1997, p. 26). When financial resources are limited, access to health-influencing “goods and services” is limited as well. For children living in poverty, these limited resources include: adequate nutrition, safe and decent housing, quality child care, positive schools and learning environments, safe and supportive neighborhoods, health care and medical supplies, healthy recreational facilities, transportation and communication, and community resources such as stores and employment opportunities (CDF, 1994). In addition, poverty appears to increase family stress and conflict, which in turn compromises the well-being of the children (CDF, 1994). Although each of the identified factors can contribute to the adverse effects of poverty on children

independently, the risk factors appear to interact, multiplying the adverse effect on child outcomes (Brooks-Gunn & Duncan, 1997; CDF, 1994; Huston, 1991).

What follows is a discussion of the measurement of poverty, the types and causes of poverty, the extent of poverty among U.S. children, and poverty's impact on the physical health of infants. It is noted, however, that child poverty is not unique to the United States. Over 1 billion children worldwide suffer some form of material deprivation, with the greatest proportion being in developing countries (UNICEF, 2005). As compared to poverty in developed countries, both the scale and the character of poverty is different in developing countries where unclean water, lack of sanitation, and hunger contribute to the high childhood death rate--fully 99% of the 10.9 million global childhood deaths (under 5 years of age) occurred in developing countries in 2000 (World Health Organization [WHO], 2002). This paper, however, will limit its discussion to child poverty in the United States, where nearly 30 million American children live in or near poverty.

Measuring Poverty

Poverty in the United States generally refers to annual family incomes that fall below the federal poverty line (FPL) set annually by the federal government. Although there has been controversy about the accuracy and adequacy of the FPL's measure of poverty in the U.S., it remains the most widely used measure for poverty (CDF, 1994; Huston, 1991).

Official poverty thresholds were originally developed by Mollie Orshansky, an economist with the Social Security Administration, in the early 1960s as "a measure to assess the relative risks of low economic status . . . among different demographic groups

of families with children” rather than as a general measure of poverty (Fisher, 1992, n.p.). Her calculation of poverty thresholds--or poverty lines--developed from 1955 data indicating that food represented one-third of the after-tax income for families of three or more in the general population; the calculation did not, however, target consumption patterns among low-income individuals (Fisher, 1992; Huston, 1991; NCCP, 2006b). Based on her findings, federal poverty thresholds are calculated by multiplying the estimated cost of an “economy food budget” by three and adjusting for family size, age of the head of household, and the number of minor children (Huston, 1991). Poverty thresholds are issued annually by the census bureau based solely on income, and is adjusted annually for cost of living increases: the level for 2006 was roughly \$16,000 for a family of three, and \$20,500 for a family of four (U.S. Census, 2006b). Poverty *guidelines* are simplified versions of poverty *thresholds* issued by the Department of Health and Human Services for administrative purposes, such as determining program eligibility (not for statistical purposes) (Department of Health & Human Services [DHHS], 2007).

Poverty thresholds, or the FPL, have been criticized for simultaneously *overestimating* poverty by failing to include in-kind transfers received (such as Food Stamps, public housing subsidies, Medicaid, and child care assistance) or assets, and *underestimating* poverty by failing to incorporate earnings taxes paid by low-income families, work-related costs (such as child care and transportation), or the disproportionate rate of medical spending among low-income families (Betson & Michael, 1997; CDF, 1994; Huston, 1991; National Academy of Sciences [NAS], 1996; NCCP, 2007). In addition, current consumption patterns have changed since the original

1955 dataset, with costs of housing, health care, child care, and transportation accounting for more of an average family's budget than previously, and food accounting for less (NCCP, 2007). Also, the measure is limited by its failure to address geographic or regional differences in the cost of living (Betson & Michael, 1997; CDF, 1994; NAS, 1996; NCCP, 2007). The cost of housing, for example, is 162% higher in New York City than in rural Mississippi (Betson & Michael, 1997). Finally, the FPL fails to reflect the dynamics of poverty: it does not address the trends in and duration of poverty among these families (Corcoran & Chaudry, 1991) or any of the variations in poverty below the level, such as the severity of the poverty experienced (Huston, 1991) (e.g., those living in "extreme" or "severe" poverty--with an income of less than half of the FPL) (U.S. Census, 2005).

In the early 1990s, Congress called upon the NAS to provide a new poverty measure (NCCP, 2007). The NAS expert panel's recommendations for a revised poverty measure were published in 1995, and included the following: (a) create a measure that more accurately reflects current family expenses and consumption patterns; (b) adjust the measure to account for different family types; (c) vary the measure by geographic area to account for differences in the cost of housing and update the index between decennial censuses; and, (d) calculate family resources using disposable income (post-tax income less out-of-pocket medical care expenses, child care and child support expenses, and a work-related transportation allowance) as well as "near-money income" (non-medical in-kind benefits such as Food Stamps and subsidized housing) (NAS 1996; NCCP, 2007). While such recommendations for a revised measure of poverty as well as other alternative measures, such as a level relative to the median income rather than an absolute

measure (Huston, 1991), have been suggested, the FPL remains the official index of poverty in the United States. Although an alternative poverty measure would likely lead to more families being officially categorized as “poor,” many suggest that even these measures would underestimate the cost of supporting a family (NCCP, 2007) as “on average, families need an income equal to two times the federal poverty level to meet their most basic needs” (NCCP, 2006a). Despite its inadequacies, the FPL likely provides a “reliable measure of changes in child and family poverty over short spans of time, such as decades” (CDF, 1994, p. 7).

While poor families subsist on incomes below the FPL, “low income” families include those with incomes between 100-200% of FPL (NCCP, 2006a). Low income families, therefore, include families of four with a 2006 income of roughly \$20,500 and \$41,000 (i.e., between FPL and twice FPL), while poor families of four had a 2006 income of less than \$20,500 (i.e., below FPL) (U.S. Census, 2006b). Because of the material deprivation that exists in this low-income range, these individuals are often included in statistics describing child poverty. In this paper, “poverty” *rates and statistics* will refer specifically to those living below the FPL, while statistics relating to low-income individuals will indicate low-income or “poor and low-income.” The *broader concepts* of “child poverty,” “poor children,” and “lower SES,” however, will be used to indicate material deprivation among poor and low-income children.

Understanding the Types and Causes of Child Poverty

One important aspect of child poverty is the dynamics--the timing, variation, directionality, and duration--of poverty among children (Corcoran & Chaudry, 1997; Huston, 1991). Although the timing, variation, severity, and directionality of poverty all

contribute to the diverse experiences of children living in poverty, it is the duration of poverty—long term versus short term--that appears to impact child outcomes most significantly (Brooks-Gunn & Duncan, 1997; Corcoran & Chaudry, 1997). Stated simply, the longer a child lives in poverty, the more significant the negative consequences of poverty are likely to be (Brooks-Gunn & Duncan, 1997).

Most children that experience child poverty experience short-term, or transitory, poverty, typically as a result of unemployment among men, or divorce among women (Corcoran & Chaudry, 1997; Huston, 1991). One study by the Panel Survey of Income Dynamics (PSID) tracing the experiences of a group of children over a 15-year period found that one in three children experienced at least 1 year of poverty during the 15 years (Corcoran & Chaudry, 1997, citing Duncan & Rodgers, 1988; Lewit, Terman, & Behrman, 1997). Of those children that experienced poverty, two-thirds spent fewer than 5 years in poverty, while only 15% spent 10 years or more in poverty (Corcoran & Chaudry, 1997, citing Duncan & Rodgers, 1988; Lewit et al., 1997).

The most significant risk factors identified by the PSID study for long-term, or persistent, poverty were: race, family structure, and parental disability (Corcoran & Chaudry, 1997; Duncan, 1991; Lewit et al., 1997).

Race. A disproportionate number of Black children live in long-term poverty. Although White children accounted for approximately 60% of the poor children, Black children represented nearly 90% of the long-term poor (Lewit et al., 1997).

Family structure. Several family structure factors affected the duration of child poverty. Children born to never-married mothers were much more likely to experience long-term poverty than were those born to married mothers (Duncan, 1991).

Interestingly, however, this was the only factor identified that had a worse impact on White children than on Black children, albeit it a small difference (Duncan, 1991). The other (related) family structure factor that places children at risk for long-term poverty was single-parenting, particularly single-mother families (Duncan, 1991; Huston, 1991).

Parental disability. Another major risk factor for experiencing long-term child poverty was having a disabled head of household--this being the most significant factor contributing to duration of poverty among Black children (Duncan, 1991).

Of note, several factors contributed to the duration of poverty among Black children--particularly when occurring in combination--that had little to no effect on poverty among White children: living in the South, living in a rural area, and low-educational attainment among heads of household (Duncan, 1991). It should be noted, however, that the summary of findings presented was based on data originally presented in 1988. It is unclear how these findings might have changed in the ensuing years.

Why so many children live in poverty--either short term or long term--is a complex issue. Any discussion of the etiology of child poverty necessarily explores the causes of adult poverty given that "children are poor because they live with adults who are poor" (Betson & Michael, 1997, p. 27). The determinants for a rise in adult poverty can be categorized as: "1) macroeconomic and demographic forces which affect the overall income distribution and 2) factors that affect an individual's earning capacity, such as education, age, and race" (Betson & Michael, 1997, p. 29).

Economic and demographic forces leading to greater poverty include the declining wages of less-educated workers, which resulted in an increase in income inequality during the 1970s and 1980s (Betson & Michael, 1997; Huston, 1991). While

this increase in inequality would not necessarily have led to greater poverty by itself, this increase combined with the failure of hourly wages to adjust for inflation, has resulted in greater poverty (Betson & Michael, 1997; CDF, 1994). In addition to low hourly wages, the rise of single-parent families--particularly single mother families--has also contributed to a rise in poverty (Betson & Michael, 1997; CDF, 1994; McLanahan, Atone, & Marks, 1991). Single-mother families are more likely to live in poverty, specifically long-term poverty, than are two-parent families (McLanahan et al., 1991) due to the lack of a second income, the “historically low earning power of women,” and the inadequacy of child support enforcement (CDF, 1994, p. 5).

The major factors affecting the other primary determinant of adult poverty, *individual earning capacity*, are education, race, and age (Betson & Michael, 1997). “Education level is an indicator of market skills that yield higher earnings; age proxies for job skills acquired through experience; and race, because of discrimination, affects both job market opportunities and payoffs” (Betson & Michael, 1997, p. 29). Using the economic index of adult self-sufficiency--that is “having sufficient resources to maintain the adults in a family above the official poverty level”--to explore these factors, studies have shown that high school graduates are 2.5 times more likely to be self-sufficient, and college graduates to be 7.3 times more likely to be self-sufficient than those without a high school education (Betson & Michael, 1997, p. 30). Additionally, younger persons are less likely to be self-sufficient than their older counterparts: nearly 30% of those under 25 years of age were not self-sufficient--a rate 3.3 times that of individuals in their 30s (Betson & Michael, 1997). Race also bears heavily on poverty: “As compared with whites, black adults have lower earnings on average and are more likely to be

unemployed or out of the labor force” (Betson & Michael, 1997, p. 29). In fact, Black adults are three times as likely as White adults to not be self-sufficient (Betson & Michael, 1997). Each of these factors plays a significant role in the cause of poverty when viewed independently. When viewed together, however, the net effect is staggering. A Black adult, for example, who is both young and a high school drop-out, has only a 15% chance of being self-sufficient (Betson & Michael, 1997).

The Extent of Child Poverty in the United States

In the United States, poverty falls disproportionately on children. Child poverty rates, measured as the percentage of children < 18 years living below the federal poverty line (FPL), have exceeded adult poverty rates since record-keeping began in the late 1950s. Although poverty rates decreased during the 1970s and early 1980s, the gap between child and adult poverty rates increased significantly (Betson & Michael, 1997; Corcoran & Chaudry, 1997)--a gap that persists today: the 2005 child poverty rate (< 18 years) of 17.6% is nearly 60% higher than the adult poverty rate (18-64 years) of 11.1, and is 75% higher than the poverty rate among seniors (> 65 years) of 10.1 (U.S. Census, 2006a).

The United States has the highest rate of child poverty among industrialized countries (UNICEF, 2005). Of the 73 million children (< 18 years) living in the United States, 28.4 million of them live in poor (below FPL) or low-income (between FPL and twice- FPL) families; that is, 39% of all U.S. children are either poor (17%) or low-income (21%) (NCCP, 2006a). Poverty does not affect children uniformly, however. There is a greater concentration of poverty and low-income status among certain subpopulations: 42% of young children (< 6-years-old) are poor or low-income; 61% of

Latino and Black children; 57% of children of immigrant parents; 49% of children in urban areas and 47% in rural areas; and, 42% of children in the South and 40% in the West are poor or low income (NCCP, 2006a). Additionally, contrary to public perceptions regarding poor families, 49% of all poor and low-income children live with married parents, and 81% live in families in which at least one parent is employed (55% full-time, 26% part-time) (NCCP, 2006a).

The Impact of Poverty on Infant Health

“In general, illness, disability, and premature death are more frequent among infants, children, and adolescents who either live in poor families or live in poverty without family support” (L. V. Klerman, 1991, p. 136). In fact, poor children are twice as likely to be in bad health than are non-poor children (Brooks-Gunn & Duncan, 1997). The “bad health” that occurs among poor children encompasses a wide variety of conditions and disabilities, and includes a greater risk of death (Brooks-Gunn & Duncan, 1997; CDF, 1994; L. V. Klerman, 1991). Poor children are “at higher risk of exposure to conditions that produce adverse health effects, [and] are more susceptible to poor outcomes from these exposures” (Geltman et al., 1996, p. 385), suffering a greater *number* and greater *severity* of health problems than their more affluent counterparts (Brekke et al., 1999; CDF, 1994; Geltman et al., 1996; L. V. Klerman, 1991). Although the adverse effects of poverty extend to children’s mental health, cognitive ability, school achievement, behavior and conduct, as well as other areas of well-being across all stages of childhood, this discussion focuses on the impact of poverty on the physical health of

those children who are both more likely to live in poverty and more likely to be adversely affected by poverty--young children, particularly infants.

Early Childhood in the SES--Health Relationship

Infancy and early childhood are important periods--perhaps the most important--for subsequent development (Stein, Schettle, Wallinga, & Valenti, 2002), so the impact of poverty on health is particularly pronounced in these early stages of life (Black, 1993). Because the early years of life set the stage for later physical, emotional, and intellectual development (Eisenberg, 1999), poor health in early childhood can produce significant impairments in health for years to come (Black, 1993).

Biological programming and the *lifecourse perspective* represent two avenues of research exploring the significance of poverty in early life on later health. Research on *biological programming* asserts the existence of critical periods in early development (*in utero* and infancy) during which “insults” related to lower SES (e.g., inadequate nutrition, toxic environmental exposures) produce lasting impairments related to later chronic diseases (Barker, 1992). Low birth weight, for example, is associated with the development of cardiovascular disease in adulthood (Barker, 1995; Barker, Osmond, Winter, Margetts, & Simmonds, 1989) and small size at birth and infancy followed by accelerated weight gain during childhood is associated with later coronary heart disease, type 2 diabetes, and hypertension (Barker, Eriksson, Forsen, & Osmond, 2002).

The *lifecourse perspective* emphasizes the cumulative effect of risks associated lower SES (e.g., environmental, illnesses) that occur in early life: that is, the accumulation of risks experienced in early life resulting in the onset of disease later in life (Davey Smith, Hart, Blane, & Hole, 1998; Kuh & Ben-Shlomo, 2004; J. Lynch et al.,

1997). “Cumulative socioeconomic disadvantage,” for example, has been associated with cardiovascular disease (morbidity, mortality, and risk factors), as well as lower self-rated health, impaired physical and cognitive functioning, and higher all-cause mortality (Davey Smith & Hart, 2002, p.1296). Interestingly, however, one study found the atherosclerotic process in adulthood to be affected by childhood SES independent of adult SES or known risk factors only among women--not men (Rosvall et al., 2002).

Early childhood experiences as influenced by SES are, therefore, important factors in child health and subsequent adult health. As discussed above, however, the mechanisms of the relationship between low SES and health are not well-understood. Various theories and pathways for the adverse impact of low SES on health, presented above, have been put forward and are applicable to children as well. Specific to children, however, some have pointed to the role of material factors, the quality and quantity of parent/child interaction, and unspecified processes linked to maternal education as particularly relevant to the relationship between low childhood SES and adverse health outcomes (Kaplan et al., 2001). The relationship between low SES and the most vulnerable phase of early childhood--infancy--is explored next.

Measuring Infant Health

Measuring children’s health employs various indicators of children’s health and well-being. These indicators can be categorized in a variety of ways. One method of categorizing health indicators is by grouping categories of outcomes: physical health, emotional and mental well-being, cognitive ability, school achievement, behavior and conduct, as well as other outcomes. Alternatively, indicators can be categorized by age group: prenatal to 2 years, early childhood (3 to 6 years), late childhood (7 to 10 years),

early adolescence (11 to 15 years), and late adolescence (16 to 19 years) (Brooks-Gunn & Duncan, 1997). These categories can be combined, such as physical health outcomes grouped by age (e.g., infant mortality rates).

Some indicators of well-being are relevant for all age groups (e.g., general health status and mortality rates), while others are unique to specific age groups (e.g., child care and family reading for young children) or a set of age groups (e.g., childhood immunizations for young children through early adolescence, or cigarette smoking for early to late adolescence). Although the mechanism of action of poverty likely differs by age group, “children living in poverty have poorer health than their more affluent peers” (Geltman et al., 1996, p. 384) across all age groups regardless of how health is measured.

Categories of physical health indicators. In general, indicators of physical health can be categorized as mortality (death), morbidity (sickness), and disability (handicapping conditions), and are stratified by age.

Mortality occurs disproportionately among children in lower SES groups--a fact considered well-established for quite some time: “No fact is better established than that the death rate, and especially the death rate among children, is high in inverse proportion to the social status of the population” (Antonovsky & Bernstein, 1977, p. 453, citing Newsholme, 1910). The current leading causes of death in children ages 1-4 are accidents, birth defects, cancer, homicide, heart disease, and pneumonia and influenza (National Center for Health Statistics [NCHS], 2006). The leading causes of death among children 5-14 years of age are strikingly similar: accidents, cancer, birth defects, homicide, suicide, and heart disease (NCHS, 2006). All of these causes of death occur disproportionately among low-income and poor children: “Low-income children are

more likely than others to die during childhood, in every age group and across every cause of death” (CDF, 1994, pp. 65-66). One study, for example, has found that low-income children are 8.4 times more likely to be a victim of homicide than their more affluent counterparts, 5.4 times more likely to die from congenital anomalies, 5.4 times more likely to die from infectious/parasitic diseases, and 4.3 times more likely to die from fire (CDF, 1994).

Morbidity, or sickness, can be roughly divided into infectious (communicable) diseases, chronic (long-standing) conditions, and injuries. All forms of morbidity are more prevalent among poor children: infectious diseases, such as vaccine-preventable diseases (e.g., measles) (L. V. Klerman, 1991), AIDS (Singh & Yu, 1996), and tuberculosis (Barr, Diez-Roux, Knirsch, & Pablos-Mendez, 2002; Drucker, Alcabas, Bosworth, & Sckell, 1994); chronic health problems, such as asthma (Newacheck & Halfon, 2000), and lead poisoning (Bornschein et al., 1984; Brody et al., 1994; Lanphear, & Roghmann, 1997); and, injuries--both unintentional (e.g., motor vehicle accidents, drownings, and firearms (Shenassa, Stubbendick, & Brown, 2004)) and intentional (such as those resulting from child maltreatment (L. V. Klerman, 1991; Vondra, 1993)).

The final set of indicators in the three-part physical health categorization is *disabilities*, which includes both physical impairments (such as functional limitations, sensory loss, and developmental disabilities) and mental health issues. Disabilities across the spectrum are more prevalent among poor children than among more affluent children (Newacheck & Halfon, 1998) and can themselves lead to further financial burdens and psychosocial adjustment difficulties (Witt, Riley, & Coiro, 2003).

Overview of infant health indicators. Indicators of infant health and well-being include measures of health status, nutritional status, health care utilization, and childcare, among others. Indicators of infant health status do overlap with measures of birth outcomes, as in the incidence of low birthweight (LBW) and very low birthweight (VLBW), because birth outcomes are so intertwined with infant health and later health consequences. Some indicators of infant health relate to growth and development such as length (e.g., growth retardation) and weight (e.g., over- and under-weight). Other infant health measures are indicators used across all age groups that are limited to infancy, such as IMR and the incidence of specific diseases and disabilities during infancy (e.g., anemia and lead poisoning). Nutritional status among infants includes breastfeeding rates, as well as the measures of food insecurity used in other age groups. Health care utilization is assessed by measures of well-child care coverage and immunization rates, and can include prenatal care because of the close relationship of prenatal care with birth outcomes and infant health (consider, for example, that infant deaths are 37% higher among infants of mothers who delayed or had no prenatal care as compared with infants of mothers who began prenatal care in the first trimester) (CDC, 2007). Childcare measures include the type of childcare utilized and the time spent in childcare.

Each of the listed indicators or measures does reflect or impact infant health, as do other factors such as family structure, socioeconomic status, housing/living conditions, and neighborhood characteristics, among others. This paper, however, focuses specifically on infant health and the impact of Welfare Reform. Therefore, what follows is a discussion of two specific indicators of infant health that represent indices of overall

infant health status that are each potentially influenced both by Welfare Reform provisions and by poverty: infant mortality and the incidence of low birthweight.

Infant mortality. A population's IMR, the number of deaths of infants under 1 year of age per 1,000 live births, is generally considered to be the best single indicator of population health: "Infant mortality is one of the most important indicators of the health of a nation, as it is associated with a variety of factors such as maternal health, quality and access to medical care, socioeconomic conditions, and public health practices" (MacDorman & Mathews, 2008, p.1). Long considered "the most sensitive index we possess of social welfare and sanitary administration" (Yankauer, 1990, p. 653, citing Newsholme, 1910), the IMR is the sum of neonatal mortality (deaths between 0 and 27 days) and postneonatal mortality (28 days to 364 days). In 2004, the overall IMR was 6.78 for the U.S., the sum of the neonatal mortality rate (4.52) and the postneonatal mortality rate (2.26), with the top five causes of death--accounting for more than half of the deaths--being: congenital malformations (20%), disorders related to short gestation and low birth weight (16%), Sudden Infant Death Syndrome (SIDS) (8%), maternal complications (6%), and accidents (4%) (CDC, 2007). Deaths occurring during the neonatal period "are generally related to maternal health problems prior to or during pregnancy or in the labor and delivery period" while those occurring during the postneonatal period "are generally believed to be related to the environments that infants experience after returning home" (L. V. Klerman, 1991, p. 138).

1. *Historical and international context of IMR.* During the 20th century the IMR in the United States decreased from approximately 100 in the year 1900 to 6.9 in 2000--a greater than 90% decline in the 20th century (CDC, 1999, 2002a). Justifiably heralded as

a significant public health achievement, public health officials have credited improved sanitary and living conditions, rising living standards, improved nutrition, decreases in fertility rates, as well as advances in neonatal medical care, for the dramatic decline in IMR (CDC, 1999).

Despite this significant accomplishment, the U.S. has one of the highest IMRs among industrialized nations: in 2004, “the United States ranked 29th in the world in infant mortality, tied with Poland and Slovakia”--a fall from its international ranking as 12th in 1960 (MacDorman & Mathews, 2008, p. 2). Of the 30 industrialized nations designated “advanced economies” by the International Monetary Fund (International Monetary Fund [IMF], 2007), only two had a 2006 IMR higher than the United States’ rate of 6.43: Israel (6.89) and Cyprus (7.04), with the U.S. lagging behind such countries or city-states as Singapore (2.29), Slovenia (4.40), and Taiwan (6.29) (U.S. Census, 2007). While most of the nations (40%) had IMRs between 4.0-5.0, fully 10% of these industrialized nations had IMRs between 2.0-3.0--rates approaching one-third that of the United States (U.S. Census, 2007).

Asynchronous declines in neonatal mortality and postneonatal mortality have marked the significant decline in the overall IMR during the 20th century. The focus on public health and social welfare during the early- to mid-part of the century, with policies and programs addressing sanitation and living conditions, improved an infant’s environment and contributed to a significant decline in postneonatal mortality rate--a decline that was accelerated with medical advances in antimicrobials and fluid replacement therapy during the 1930s and 1940s: between 1930 and 1949 the overall IMR declined 52%, with a 40% decline in neonatal mortality and a 66% decline in

postneonatal mortality (CDC, 1999). During the latter part of the century, however, technological advances in neonatal medicine contributed to a marked decline in neonatal mortality--a 41% decline between 1970 and 1979, for example, as compared to a 14% decline in postneonatal mortality (CDC, 1999). Some researchers, however, have raised the issue of whether some of the improvement in neonatal mortality has actually resulted in “the postponement of some deaths from the neonatal to the postneonatal period” (Piper, 1991, p. 1046) rather than a prevention of infant mortality altogether.

2. *Elevated IMR in vulnerable subpopulations.* The remarkable decline in IMR for the U.S. masks the disproportionately high IMR for specific vulnerable subpopulations. IMRs, for example, are higher for mothers who begin prenatal care late or have none at all, are teenagers, have less than a high school education, are unmarried, or smoke during pregnancy--all factors related to lower socioeconomic status--as well as for mothers living in the South as compared to the West or Northeast (CDC, 2002b). In addition to these “risk factors,” there exists a significant and persistent racial disparity in IMR. The IMR for Black infants, while declining, has remained substantially higher than that of their White counterparts: at 13.25, it was nearly twice the national rate in 2004 and nearly two-and-a-half times the 5.66 rate for Whites (CDC, 2007).

The disparity between Black and White IMRs is not the only racial disparity in IMR within the United States: IMRs vary between all racial/ethnic groups in the U.S. The variation between racial/ethnic groups, however, is not always to the disadvantage of the minority group: while the national IMR for the U.S. was 6.78 in 2004, Asian/Pacific Islanders had the lowest IMR of all racial/ethnic groups in the U.S. (4.67), followed by Hispanics (5.55), and then non-Hispanic Whites (5.66) (CDC, 2007). The racial disparity

does, however, serve to disadvantage the American Indian/Alaska Natives (8.45) and, quite strikingly, non-Hispanic Blacks (13.25) (CDC, 2007).

“The mortality disadvantage of Black Americans is observed across all of the major categories of infant death” but is most significant for “disorders related to short gestation and low birth weight”--nearly four times higher for Black infants than for White (David & Collins, 2007, p. 1192). “Most of the Black-White gap in first-year mortality is attributable to the higher rate of Black infants born at very low birth-weight” (VLBW or less than 1500 g)--a rate three times higher among Black infants than White (David & Collins, 2007, p.1193). Fully two-thirds of the Black-White IMR gap is attributable to the difference in the incidence of VLBW infants (CDC, 2002c). Of note, the Black-White racial disparity in IMR does not appear to extend to infants born to foreign-born Black mothers: the birthweight patterns for infants of African- or Caribbean-born Black mothers approximate those of U.S.-born White mothers (David & Collins, 2007; Kleinman, Fingerhut, & Prager, 1991). However, the birthweight patterns for infants later born to the first-generation daughters of these foreign-born mothers more closely approximate that of the U.S.-born Black population (David & Collins, 2007).

Research remains unclear as to the cause or causes of the excess IMR among infants born to Black mothers. Some studies, for example, have found that income or poverty status does not have a statistically significant effect on neonatal mortality rates for infants born to Black mothers (Brooks-Gunn & Duncan, 1997), leading to an assessment that the Black-White “racial disparity in IMRs has not been explained fully by differences in socioeconomic status” (CDC, 2002c, p. 330). This evidence and that of the different birthweight patterns based on nativity suggest that “racial differences in

health reflect more than differences in economic resources alone” (Geronimus, Hicken, Keene, & Bound, 2006, p. 5). Some have theorized that “neighborhood violence [and] other unsatisfactory aspects of their residential environment, and stressful life events” contribute to disproportionately high rates of low birthweight (LBW) and IMR among infants born to U.S.-born Black mothers (David & Collins, 2007, p. 1194), while others suggest “the stress of living in a race-conscious society” plays a role (Geronimus et al., 2006, p. 5). Still others point to inequities in health care provision as the root of the racial disparity in neonatal mortality, but to “environmental, social, and economic factors” for the declining, but persistent, racial disparity in postneonatal mortality (Din-Dzietham & Hertz-Picciotto, 1997, p. 787). Assertions regarding inequities in health care provisions appear bolstered by at least one study finding that the use of tertiary care settings (referral hospitals with subspecialty care, such as neonatal intensive care) appears to reduce the racial disparity among high-risk infants (Collins, 1992).

3. *SES disparity in IMR.* In addition to the persistent racial disparity in IMR noted above, there is a long-standing SES disparity in IMR: “Low-income children . . . [are] more likely than others to die before their first birthday”--even when potentially confounding factors (including race) are controlled for (CDF, 1994, p. 64). Specifically, the IMR and each of its components--the neonatal mortality rate and the postneonatal rate--are all higher among poor infants than among non-poor infants (L. V. Klerman, 1991; Singh & Kogan, 2007).

Poverty has been estimated to increase the risk of death among infants by almost 50% in both the neonatal and postneonatal periods (Gortmaker, 1979) through its adverse

impact on prepregnancy maternal health, prenatal health, and prenatal care utilization, as well as the newborn's environment and material conditions.

Though poverty's negative effect on all of these areas is well-known, the relative impact is not known. Some assert that poverty's greatest impact on infant health is via the newborn's postneonatal environment rather than on the prenatal environment (Gortmaker, 1979), while others suggest a greater role via its impact on birthweight distribution (Collins, 1992). Infant survival--particularly during the neonatal period--is closely related to birthweight, with higher birthweight conferring greater survival advantage (Paneth, 1995). Birthweight patterns, in turn, are directly related to SES: higher SES groups have higher (more protective) mean birthweights (Paneth, 1995). The impact of birthweight on the health of infants (and beyond) will be discussed more fully below.

Whether measured as parental income, educational attainment, or occupation, the SES disparity in IMR exists and has for some time (Antonovsky & Bernstein, 1977; Gortmaker, 1979; Paneth, Wallenstin, Kiely, & Susser, 1982). Marx' epidemiologic work in mid-19th century Britain found higher IMRs in more industrialized areas where working class populations predominated (Gortmaker, 1979). Such historical findings of an elevated IMR due to low parental income have led to varied attempts to mitigate this relationship, such as the work of the U.S. Children's Bureau to improve U.S. children's health in the early 1920s (Gortmaker, 1979). Despite such efforts, however, the historic disparity does persist, with relative SES disparities in IMR actually *increasing* since 1985 (Singh & Kogan, 2007).

There are, however, several caveats to the general statement regarding SES disparities in IMR. First, despite higher rates of poverty and lower maternal education attainment, infants born to Hispanic mothers generally have higher birth weights and lower rates of infant mortality, approximating those of White mothers (CDC, 2002c). Possible contributing factors include “cultural practices, family support, selective migration, diet, and genetic heritage” (CDC, 2002c, p. 330). Second, although the disparities in IMR by SES exist among both Black and White populations in the United States, the SES disparity in IMR is greater among Whites than among Blacks, Hispanics, or Asians (Singh & Kogan, 2007). This may be, in part, a factor of the third caveat: higher maternal educational attainment appears to confer more protection for infants born to White mothers than other racial/ethnic groups (Singh & Kogan, 2007).

4. *Maternal education and IMR.* Considered by some to be a more sensitive indicator of infant mortality risk than occupation or income, maternal education as a measure of SES for infant mortality risk is robust but has limitations. As previously noted, having less than a high school education is considered a risk factor for infant mortality (CDC, 2002b). The 2004 IMR, for example, was nearly twice as high for infants born to women with less than 12 years of education than infants born to women with 16 years of education or more (CDC, 2007). This disparity may be a result of differences in “social, behavioral, and health care factors known to be associated with infant mortality” such as “smoking during pregnancy, delayed or no prenatal care, and lack of health care coverage” that “varies substantially by county deprivation levels and maternal education” (Singh & Kogan, 2007, p. 937).

Some researchers assert that the strong correlation between maternal education and infant health is not causal, that education acts merely as a proxy for SES and relative levels of material conditions for mothers and infants (Desai & Alva, 1998). Others suggest that maternal education appears to offer a protective effect for their infants, primarily during the postneonatal period (Din-Dzietham & Hertz-Picciotto, 1997), through knowledge regarding health care utilization (pre- and postnatal care) and health behaviors (improved nutrition, avoidance of risk factors) (Desai & Alva, 1998).

If present, the protection of increased maternal education does not appear to confer the same benefit to Black mothers as it does to White mothers in the United States (Din-Dzietham & Hertz-Picciotto, 1997). While the White-Black racial gap in IMR previously discussed is present across all maternal education levels, it actually *widens* with higher educational attainment (Din-Dzietham & Hertz-Picciotto, 1997; Singh & Yu, 1995) and is actually *highest* between Black and White college graduates (Din-Dzietham & Hertz-Picciotto, 1998; Scott-Wright, Wrona, & Flanagan, 1998). In one study, Black college graduates had IMRs *three times* that of their White counterparts, with an IMR just under that of White women with only 9 years of education (Maryland Department of Health and Mental Hygiene, 1998). An explanation for the relative protection of education based on race is not clear. Theories include income differentials between Black and White college graduates, possible differences in utilization of and/or quality of prenatal care, differential environmental exposures, and stress associated with racial discrimination and lower SES neighborhoods (Din-Dzietham & Hertz-Picciotto, 1998). But while this may explain the Black-White IMR disparity, it does not appear to address why the disparity increases with rising educational attainment.

One course of research yet to be fully explored is the different patterns in IMRs based on maternal age for different racial/ethnic groups. The IMR patterns based on maternal age is quite different for Black mothers than other racial/ethnic groups: while White, Hispanic, and Asian mothers between 30-34 years of age have the lowest IMRs within their respective racial/ethnic group, followed by ages 25-29, 35-39, and then 20-24, the lowest IMRs for Black mothers is between 20-24 years of age, and rises for each subsequent age group--ages 25-29, 30-34, and then 35-39 (CDC, 2007). Given the likelihood of later childbearing among college-graduates, the higher IMRs for Black college-graduates may, in part, reflect postponing childbearing beyond the maternal ages with lower IMRs. This does not, however, explain why the IMR pattern is different among infants born to Black mothers (lowest in younger mothers, 20-24, and rising with each progressive age group) than those born to mothers of others racial/ethnic groups. In fact, one study found the lowest IMR (both neonatal and postneonatal) among infants born to Black mothers to be in Black primiparous (first pregnancy) teens, 18-19 years old (Kleinman et al., 1991). While some have suggested a “weathering” phenomenon--an accumulation of stress likely associated with racial discrimination (Geronimus et al., 2006)--accounts for the progressively higher IMR as the age of the Black mother increases, the answer is unclear at present.

Low birthweight.

1. *Low birthweight, preterm birth, and intrauterine growth restriction.*

Birthweight and period of gestation are the two most important predictors of an infant’s subsequent health and survival. “Infants born too small and/or too soon have a much greater risk of death and both short-term and long-term disability” (CDC, 2007, p. 5).

Normal birthweight is considered to be 2500 g (approximately 5.5 pounds) or greater (CDC, 2007). Birthweights of less than 2500 g are considered LBW, and VLBW for those birthweights less than 1500 g (approximately 3.3 pounds) (CDC, 2007).

“An infant’s chances of survival increase rapidly with increasing birthweight,” with the lowest IMRs being for birthweights between 3000--4999 g (approximately 6.6--11 pounds) (CDC, 2007, p. 7). The 2004 IMR among normal birthweight infants (2500g or greater) was 2.26, while the IMR among LBW infants was 57.64, and among VLBW infants specifically was 244.5 (CDC, 2007). LBW occurs as a result of either preterm delivery and/or intrauterine growth restriction (IUGR) (also called intrauterine growth retardation or small-for-gestational-age birth (SGA)) (L. V. Klerman, 1991; Reichman, 2005). Stated simply, infants are LBW either because they were born too soon, did not grow enough in utero, or both (Paneth, 1995). Although preterm delivery and IUGR are both causes of LBW, “it is preterm delivery that underlies most low birth weight in the United States” and other industrialized nations (Paneth, 1995, p. 22).

A full-term pregnancy is considered 37-41 weeks in duration; delivery prior to this timeframe is considered “preterm” and is categorized more narrowly by the number of weeks of gestation (CDC, 2007). In general, the longer the period of gestation, the more health benefits conferred to the infant: while full-term infants (37-41 weeks’ gestation) had an IMR of 2.39 in 2004, infants born between 34-36 weeks of gestation had an IMR of 7.32, between 32-33 weeks an IMR of 16.06, and before 32 weeks an IMR of 182.45--this group representing 2% of infants born but accounting for 55% of the infant deaths in 2004 (CDC, 2007). Even within the “full-term” category there are variations in IMR demonstrating the health benefits conferred by longer periods of

gestation: in 2004, the IMR for full-term infants born between 37-39 weeks' gestation was 2.61, while the IMR for full-term infants born between 40-41 weeks was 2.0 (CDC, 2007).

Unlike preterm birth which refers specifically to the length of gestation (pregnancy)--that is, deliveries that occur earlier than 37 weeks, IUGR refers to the inadequacy of fetal growth during the pregnancy--specifically, an estimated fetal weight below the 10th percentile for gestational age (Mari & Hanif, 2007). In other words, IUGR refers to a fetus whose estimated weight is less than 90% of fetuses at that point in a pregnancy. The 10th percentile is used because of the increased perinatal mortality and morbidity associated with birthweights below this point (Mari & Hanif, 2007). The “failure to attain optimal intrauterine growth” associated with IUGR can be the result of pathological conditions, such as pregnancy-induced hypertension/preeclampsia, placental insufficiency, or placental abruption (Ananth, Demissie, Kramer, & Vintzileos, 2003; Mari & Hanif, 2007, p. 497). Because of the compromised in utero growth in these “pathological” cases, IUGR is generally an indication for induction of labor, often before 32 weeks; such a birth is considered a “preterm SGA birth” (Ananth et al., 2003; Mari & Hanif, 2007). In cases where the IUGR is not considered pathological, such as fetuses considered “constitutionally small,” early induction is not generally indicated and the birth is considered a “term SGA birth” (Ananth et al., 2003; Mari & Hanif, 2007).

Since the mid-1980s, the percentage of preterm and LBW births in the United States has increased as a result of multiple births (in part due to an increase in the use of assisted reproductive technology) as well as changes in the medical management of pregnancy (such as increased utilization of induction and Cesarean section) (CDC, 2007).

It should be noted, however, that the rate of LBW among singleton births has remained stable, further highlighting the role of the rise in multiple births in the overall LBW increase (Reichman, 2005). During this same timeframe, the percentage of *term* SGA births has decreased, but the percentage of *preterm* SGA births has increased (Ananth et al., 2003). This change may be partially accounted for by a change in the medical management of IUGR (induction and Cesarean section) and the timing of these interventions--that is, a shift toward earlier stages of pregnancy for induction of IUGR (Ananth et al., 2003).

As with IMR, the United States has among the highest LBW rates of any industrialized nation: with a 2002 rate of 8%, the U.S. LBW rate is higher than most industrialized countries--double that of 4 of the 30 industrialized nations--and is even double that of such developing countries as Serbia and Montenegro, Estonia, and Lithuania (UNICEF, 2004). The U.S. also has one of the highest rates of preterm birth of any industrialized nation at 12.3% in 2001 (Russell et al., 2007).

2. *Consequences of LBW and preterm birth.* Low birthweight is used globally as an indicator of maternal health (prepregnancy and during pregnancy), prenatal care utilization, and infant health (UNICEF, 2004). The use of a single LBW threshold to identify infants in need of specialized care or at risk during the neonatal period, however, has been criticized by some as a “crude indicator” (Paneth, 1995). Because different populations--and often subpopulations--have different birthweight patterns and means, some have advocated defining the normal birthweight for a population by risk for adverse outcomes--that is, by incorporating the risk of infant mortality and pathological conditions associated with specific birthweights for each population (Paneth, 1995). For

example, infants of Japanese women and Black American women have roughly identical mean birthweights, but the IMR is significantly higher (roughly three times higher) for Black Americans (Paneth, 1995). Therefore, the mean birthweight represents a normal, healthy birthweight among Japanese infants, but a pathological condition--or LBW--for Black American infants (Paneth, 1995). For now, however, LBW remains defined as a birthweight of less than 2500 g (approximately 5.5 pounds) globally.

The concern over LBW stems from its association with a variety of adverse health outcomes and neurodevelopmental disorders (Hack, Klein, & Taylor, 1995; L. V. Klerman, 1991; Paneth, 1995). The most significant adverse outcome associated with LBW is infant mortality: “One of the most important processes that may determine an infant’s chance of survival is birth-weight” (Finch, 2003, p. 1833). In industrialized countries, LBW--particularly VLBW--and preterm birth are directly (and closely) related to neonatal mortality (Paneth, 1995; Russell et al., 2007). Although the IMR for LBW and preterm infants has decreased since the 1960s--particularly among VLBW infants--owing to advances in neonatal care, it remains that these infants have higher rates of mortality and morbidity than do normal birthweight infants both in infancy and later in life (CDC, 2007; Hack et al., 1995). As referenced above, the IMR for LBW infants is over 25-fold that of normal birthweight infants, and is nearly 110-fold higher for VLBW infants (CDC, 2007). As for preterm infants (< 37 weeks’ gestation), the IMR is 15-fold higher preterm infants than for term infants, and 75-fold higher for very preterm infants (< 32 weeks’ gestation) (Russell et al., 2007).

In addition to a greater risk of infant mortality (particularly neonatal mortality), LBW and preterm birth are also associated with a broad spectrum of adverse outcomes

ranging from short-term neonatal morbidity to longer-term developmental abnormalities (Hack et al., 1995). Although some LBW and preterm infants have few or no adverse outcomes, LBW and/or preterm birth in general are associated with a number of adverse health and neurodevelopmental outcomes: cerebral palsy, mental retardation/subnormal intelligence, blindness and deafness, respiratory distress syndrome and bronchopulmonary dysplasia associated with respiratory infections, feeding difficulties, growth delays, and asthma (Hack et al., 1995; Reichman, 2005). The likelihood of an adverse outcome increases as birthweight decreases; that is, smaller, earlier infants have higher risks of adverse outcomes (Hack et al., 1995). Although this is particularly true for significantly smaller, earlier infants, one study of “near-term infants” (34--36 ½ weeks’ gestation) found that even these relatively mature infants were more likely to have neonatal morbidity, such as respiratory distress and sepsis (infection), with a decrease in neonatal hospital costs found for each successive week of gestation (Wang, Dorer, Fleming, & Catlin, 2004). Of note, as survival rates for the smallest infants--extremely LBW infants (500--999 g)--have improved over the past decade or two, neonatal *morbidity* (primarily respiratory complications) has increased (Stoelhorst et al., 2005) and the risk for “significant neurodevelopmental impairment” has increased, likely due to the higher rates of neonatal morbidity (Wilson-Costello, Friedman, Minich, Fanaroff, & Hack, 2005).

The adverse health and developmental outcomes associated with LBW and/or preterm birth appear to extend beyond infancy, into childhood, adolescence, and adulthood (Hack et al., 1995). Some studies, for example, have demonstrated lower IQs, higher rates of neurosensory impairments, and lower academic achievement scores

among 20 year-olds born with VLBW (Hack et al., 2002). Also, as noted in a previous section, some research has correlated LBW with the development of cardiovascular disease in adulthood (Barker et al., 1989) and correlated small size at birth and infancy followed by accelerated weight gain during childhood with later coronary heart disease, type 2 diabetes, and hypertension (Barker et al., 2002). Not all research, however, has demonstrated that the adverse outcomes associated with LBW extend into adolescence and adulthood. Some studies have found that social experiences, such as home environment, race/ethnicity, family structure, and other factors, disproportionately affect the long-term developmental outcomes among LBW infants; that is, the social environment plays a larger role in eventual academic achievement than does birthweight (Boardman, Hummer, Padilla, & Powers, 2005). It should be noted, however, that the research demonstrating a larger role for social factors on adverse developmental outcomes included *all* LBW infants, while the research demonstrating long-term adverse developmental effects of birthweight narrowed its population to VLBW--those most at risk for adverse outcomes based on birthweight.

An additional concern over LBW and/or preterm birth is the increased health care costs associated with the hospitalization of and pediatric care for these infants.

Technological advances in the treatment of the newborn over the past two decades have increased the viability of even the earliest preterm infants (McCormick & Richardson, 2002; Russell et al., 2007; Wilson-Costello et al., 2005). Despite these advances, however, “the incidence of severe acute complications of very preterm/LBW infants, accompanied by chronic medical conditions, have not markedly diminished since the mid-1990s” (Horbar et al., 2002; Russell et al., 2007, p. e2). As a result, LBW and

preterm birth represent a disproportionate share of hospital costs (Gilbert, Nesbitt, & Danielsen, 2003; Russell et al., 2007). In general, “hospital costs decrease with increasing birth weight and gestational age, with the smallest and earliest infants having the highest costs and longest length of stay” (Gilbert et al., 2003; Russell et al., 2007, p. e2). As noted above, one study has found that, even among near-term infants (34--36 ½ weeks’ gestation), hospital costs decrease for each successive week of gestation (Wang et al., 2004).

In a recent study of the 4.6 million infant hospitalizations in the United States in 2001, preterm birth/LBW accounted for 8% (384,200) of all infant hospital stays, but 47% (\$5.8 billion) of all infant costs, and represented 6% of all pediatric (< 18 years) admissions for the year, but 27% of all pediatric hospital costs (Russell et al., 2007). Of the preterm/LBW hospital stays, the earliest and smallest infants (< 28 weeks’ gestation, < 1000 g) represented the largest proportion of hospital costs, accounting for 8% of all preterm/LBW stays but one-third of the hospital costs for preterm/LBW infants (Russell et al., 2007). It should be noted that “hospital costs” do not include physician fees or costs for rehabilitation, outpatient costs, or home health care for these infants, or maternal costs such as outpatient treatment or lost productivity (Russell et al., 2007). A final note regarding payers should also be included: while the average cost did not vary between payers (Medicaid versus private payer/insurance), Medicaid covered 42% of all preterm/LBW infants, and only 37.5% of uncomplicated newborns, suggesting either that the demographics of the Medicaid-population have a higher risk set for LBW/preterm birth or that the incurred costs create Medicaid eligibility (Russell et al., 2007).

3. *Causes of and risk factors for LBW.* Due to the increased risks of adverse health and developmental outcomes--as well as the increased costs--associated with LBW, IUGR, and preterm birth, efforts have been made to identify and reduce their causes. Because LBW is itself a risk factor for infant mortality, risk factors for LBW overlap with those for infant mortality: maternal age under 17 or over 34, low SES, unmarried status, low maternal education, Black race, and inadequate or no prenatal care (CDC, 1994). LBW-specific risk factors include: low pre-pregnancy weight-for-height, inadequate weight gain during pregnancy, smoking and substance use, as well as multiple medical conditions occurring before and/or during pregnancy (CDC, 1994). Considering LBW risk factors, IUGR and preterm delivery have several risk factors in common, but each condition also has a distinct set of risk factors, some of which are amenable to change and others that are not within “immediate control” (Chomitz, Cheung, & Lieberman, 1995). Many, though not all, of the risk factors for IUGR are amenable to change, while risk factors for preterm birth are not generally within immediate control.

Risk factors for IUGR. The major risk factors for IUGR are maternal smoking, low weight gain during pregnancy, and low prepregnancy weight, with lesser risk factors including: alcohol or drug (illicit and some prescriptions) use, excessive stress or anxiety, heavy physical labor or exertion, chronic maternal illness, domestic violence, primiparous status (first pregnancy), gestational hypertension, fetal anomaly, and a history of prior IUGR deliveries (CDC, 1994; Chomitz et al., 1995; Meyer, Jonas, & Tonascia, 1976; Nathanielsz, 1995; Paneth, 1995). The two most significant IUGR risk factors, maternal smoking and low maternal weight gain, are related to maternal SES and are, therefore, part of the SES-health relationship. They are explored below. In addition,

given the potential of these risk factors to change, the role of prenatal care in reducing LBW are explored as well.

Of the risk factors noted, the single most significant risk factor for LBW/IUGR is maternal smoking (Chomitz et al., 1995; Goldenberg & Rouse, 1998; Meyer et al., 1976; Nathanielsz, 1995; Paneth, 1995). Maternal smoking has been identified as a risk factor for LBW--and infant mortality--since the 1950s (Meyer et al., 1976). Some early studies, at a time when maternal smoking in the U.S. was estimated between 34%-54%, found a 53% increase in LBW risk among “light smokers” (< 1 pack per day) as compared to nonsmokers, and an increase in LBW risk of 130% among “heavy smokers” (1+ pack per day) (Meyer et al., 1976). Maternal smoking rates have declined significantly since then, with a self-reported rate of 11.4% in 2002 (CDC, 2004); however, the risk of LBW for infants of pregnant smokers has not diminished.

Several studies have attempted to further quantify the *risk* of maternal smoking for LBW. One such study found a 26% increase in the odds of LBW for every five additional cigarettes smoked per day (Kleinman & Madans, 1985). Other studies have attempted to quantify the *effect* of maternal smoking. These studies have generally found a dose-response effect; that is, a progressive decline in birth weight for each increase in number of cigarettes smoked (Kleinman, Pierre, Madans, Land & Schramm, 1988). Even a decrease in the amount smoked, therefore, improves birthweight outcomes--when the goal of total smoking cessation cannot be achieved (Sexton & Hebel, 1984). Another issue beyond that of a dose-response of maternal smoking is that of the timing of maternal smoking. One recent study, although confirming the increased incidence of LBW among pregnant smokers, found that it was smoking status during the *third*

trimester of pregnancy that was the strongest predictor of birthweight (Bernstein et al., 2005). Specifically, this study found that there was a 12g--13g (approximately 0.45 ounces) loss of birthweight for each day a pregnant woman smoked between 2-10 cigarettes during the third trimester (Bernstein et al., 2005). The study also confirmed the dose-response as well, finding that for each cigarette smoked per day during the third trimester, there was a loss of 27g (nearly 1 ounce) of birthweight (for example, 6 cigarettes per day representing a 27g greater loss in birthweight than 5 cigarettes) (Bernstein et al., 2005).

The pathways for the impact of maternal smoking on birthweight are not entirely clear. Various theories have been offered, ranging from biological to behavioral to psychosocial pathways. Evidence supports several biological pathways including reduced oxygen to the fetus (Misra & Nguyen, 1999) and nutritional deficiencies (Haslam & Lawrence, 2004). Suggested behavioral pathways include poor dietary intake, increased alcohol consumption (Wu, Buck, & Mendola, 1998), and lower rates of prenatal vitamin use (Haslam & Lawrence, 2004). Proposed psychosocial pathways include less social support and less wantedness of pregnancy (Wu et al., 1998). It should be noted in reviewing these pathways that maternal smokers are at once more likely to have nutritional deficiencies due to smoking, more likely to have inadequate dietary intake, and less likely to take vitamin supplements.

The other significant risk factor for LBW/IUGR involves maternal nutritional status; that is, low weight gain during pregnancy and/or low prepregnancy weight-for-height. Maternal nutrition, both before and during pregnancy, is a “major factor affecting fetal growth” (Nathanielsz, 1995, p. 59). Evidence indicates a “nearly linear association

between maternal weight gain during pregnancy and birth weight, and an inverse relationship to the rate of low birth weight” (Chomitz et al., 1995, p.123). Maternal weight gain during pregnancy is a function of several factors, including dietary intake and prepregnancy weight, which is itself a function of past nutritional status, genetics, and environmental factors (Chomitz et al., 1995).

Because so many of the risk factors for IUGR are amenable to change, the role of prenatal care in reducing LBW is often raised, but is somewhat controversial. Many researchers have grappled with the challenge of identifying “adequacy” of prenatal care (whether by when initiated, the number and spacing of visits, the content of the visit, the type and location of provider, etc.) as well as accounting for the variability of prenatal care (Alexander & Korenbrot, 1995; Lu, Tache, Alexander, Kotelchuck, & Halfon, 2003). Some have raised concerns over the great number of “non-randomized” studies, and question the role of self-selection in these studies (that is, more “health-conscious” women being more likely to seek prenatal care) (Alexander & Korenbrot, 1995).

Some randomized trials have demonstrated positive effects of prenatal care on the incidence of LBW, but often the effects are for specific types of prenatal care on specific populations. One such randomized study found prenatal care performed by a multidisciplinary group reduced LBW among first-time low-income mothers, but not among low-income mothers who had been pregnant previously, and found no effect of “standard” prenatal care on any of the low-income mothers (McLaughlin et al., 1992). One randomly selected matched-cohort study found group prenatal care (that is, sessions involving groups of pregnant women) to reduce LBW while individual prenatal care did not (Ickovics et al., 2003).

These findings, and the otherwise equivocal results found for prenatal care in LBW, have prompted some to address the ineffectiveness of prenatal care “in its present form” by calling for a “reconceptualization of prenatal care” (Lu et al., 2003, p.362). It should be restated, however, that the use of prenatal care is associated with lower IMRs: the overall IMR for infants of women with no prenatal care is 5 ½ -fold that for infants of women initiating prenatal care in the first trimester (CDC, 2007). It is unclear, however, what role the “self-selection” of those seeking prenatal care plays in this disparity.

Risk factors for preterm birth. Unlike the risk factors for IUGR, many of which are amenable to change, most risk factors specific to preterm birth are not generally within “immediate control”: maternal (intrauterine) infection, structural abnormality of the cervix, maternal stress or anxiety, a history of preterm delivery, and, to a lesser extent, compromised maternal nutritional status (e.g., low pre-pregnancy weight, low weight gain during pregnancy, anemia, low zinc levels) (Berkowitz, 1981; Goldenberg & Rouse, 1998; Nathanielsz, 1995)--with some researchers questioning the role of maternal weight gain in preterm birth, viewing it as much more closely associated with intrauterine growth (Paneth, 1995).

Of all the risk factors for preterm birth, the single most significant risk factor is maternal (intrauterine) infections, accounting for up to 80% of *early* preterm births (Goldenberg & Rouse, 1998) and between one-third to one-half of *all* preterm births (Nathanielsz, 1995). These intrauterine infections can have their basis in a urinary tract infection (symptomatic or asymptomatic), sexually transmitted disease, or bacterial vaginosis, among other infections (Goldenberg & Rouse, 1998). It should be noted, however, that research suggests that it is *prepregnancy* asymptomatic intrauterine

infection that is tied to preterm birth, with risk factors including: an increased number of lifetime sexual partners, a history of pelvic inflammatory disease (PID), and a history of intrauterine contraceptive device (IUD) use (Toth, Witkin, & Ledger, 1988; Yost et al., 2006).

Various treatment protocols to prevent preterm labor have been utilized with variable results. There is little evidence that preterm births are prevented by: patient education and counseling, at home monitoring of uterine activity, bed rest, nutritional supplementation (except for addressing specific mineral deficiencies), or prenatal care (Goldenberg & Rouse, 1998; Lu et al., 2003)--although some researchers (and practitioners) refute the assertion that prenatal care does not prevent or delay preterm birth (Ickovics et al., 2003). The following treatments have demonstrated some success in delaying a preterm delivery: cervical cerclage (to address structural abnormalities of the cervix), hormone therapy (for those with a history of preterm labor), tocolytics (medications to stop contractions, appear to delay labor by 48 hours), and screening for and antibiotic treatment for urinary tract infection and bacterial vaginosis (Goldenberg & Rouse, 1998). Despite the modest successes of these treatments in delaying preterm delivery, gains in effectively addressing preterm birth have been limited.

In addition to the previously noted risk factors, one significant factor predisposing toward preterm birth (and IUGR to a lesser extent) warrants discussion: race/ethnicity. As with IMR, there is a significant racial disparity in preterm birth. And, as with IMR, the racial disparity serves to disadvantage Black infants, but not Hispanic or Asian/Pacific Island infants. The incidence of *LBW* among American/Alaska Native (7.5%), Hispanic (6.8%), and Asian/Pacific Islander (7.9%) infants closely approximate

the incidence among White infants (7.1%) (CDC, 2007). Black infants, however, have a strikingly higher incidence of LBW (13.7%)--nearly double that of White infants (CDC, 2007). The incidence of *preterm birth* has a similar pattern, with the incidence among Hispanic (12.0%) and Asian/Pacific Island (10.5%) infants closely approximating that of White infants (11.6%) (CDC, 2007). The incidence of preterm birth among Native American/Alaska Native infants (13.7%), however, is 18% higher than White infants, and among Black infants (17.9%) is 54% higher (CDC, 2007). Looking more narrowly at extremely preterm birth (< 28 weeks' gestation) reveals an even greater disparity--a 4-fold higher incidence among Black infants than among White infants (Schempf, Branum, Lukacs, & Schoendorf, 2007).

These racial disparities in preterm birth have persisted for decades, and account for the majority--roughly *two-thirds* by some estimates--of the racial disparity in IMR (David & Collins, 2007). Other estimates suggest these disparities may account for an even higher proportion of the disparity in IMR: one study found that the Black-White disparity in preterm birth accounts for fully 80% of the racial disparity in IMR, and *all* of the disparity in neonatal mortality (Schempf et al., 2007). It should be noted, however, that among extremely (< 28 weeks' gestation) and very (28--31 weeks' gestation) preterm births, Black infants actually have a survival advantage over White infants; that is, these Black infants have lower IMR than do comparably-aged White infants (Schempf et al., 2007).

At least one study, however, has found that the racial disparity is nearly entirely explained by the presence of "specific medical and socioeconomic characteristics" (Lieberman, Ryan, Monson, & Schoenbaum, 1987, p. 743). When these researchers

controlled for maternal age of less than 20 years, single marital status, welfare receipt, and low maternal education (less than high school), race was no longer a “significant” risk factor for preterm birth and virtually disappeared as a risk factor when maternal hematocrit level was also taken into account (Lieberman, et al., 1987). SES, therefore, plays a significant role in LBW--perhaps a greater role than does race.

4. *SES and LBW.* Low SES adversely affects birth outcomes through its impact on prepregnancy health and nutritional status, prenatal health and health behaviors (smoking, diet, substance use, prenatal vitamin use), psychosocial factors (social support, stress), and prenatal care utilization--all of which impact birthweight (Chomitz et al., 1995; Gortmaker, 1979; Haslam & Lawrence, 2004; Paneth, 1995; Wu et al., 1998). In fact, the relationship between low SES and adverse birth outcomes appears to be mediated primarily through the adverse effect of SES on birthweight patterns: “Low birth weight is the central biological mediator of the relationship of social class and economic conditions to infant mortality in industrialized countries” although the precise mechanisms are unclear (Paneth, 1995, p.21).

Despite the generally recognized relationship between SES and LBW, it is unclear what the appropriate measure for SES is in its relationship to LBW. Parental occupation, educational achievement, income, and marital status are all associated with birthweight patterns (Paneth, 1995), but each appears to have a different relationship with--or, different mediating causes of--birthweight (Finch, 2003). Further complicating a complete understanding of the relationship between SES and LBW is the correlation among various SES measures: “Educational attainment, marital status, maternal age, and income are interrelated and are often used to approximate socioeconomic status, but no

single factor truly measures its underlying influence” on LBW (Chomitz et al., 1995, p.122).

As with infant mortality, maternal education is a key factor in risk of LBW: low educational attainment is associated with higher rates of low birthweight (Chomitz et al., 1995). One study has found parental education levels (combined maternal and paternal) to be the best overall predictor of LBW (Parker, Schoendorf, & Kiely, 1994). Most research, however, focuses exclusively on low maternal education because of its close relationship to the prenatal health behaviors associated with LBW: higher rates of smoking (Kleinman & Madams, 1985), lower maternal weight gain (Chomitz et al., 1995), and lower rates of prenatal care (Gortmaker, 1979). There are other factors associated with low maternal education that also influence birthweight, including “nonfinancial characteristics of the family” such as maternal age, marital status, number of children (Chomitz et al., 1995; Gortmaker, 1979).

In addition to parental education, income is also relevant to the risk of LBW: “Poverty is strongly and consistently associated with low birth weight” (Paneth, 1995, p.19). In one study evaluating the impact on LBW rates of SES measures of income, occupation, and state income inequality, only income had an independent net effect on birthweight, suggesting it is “absolute material conditions” that are most significant in the probability of LBW (Finch, 2003). Other studies have confirmed the pre-eminent role of income in the likelihood of LBW (Gortmaker, 1979; Starfield et al., 1991), while others have found that income had no effect on birthweight after controlling for parental birthweight (Conley & Bennett, 2000) or on infant hospitalization rates (associated with LBW) (Strobino, Ensminger, Nanda, & Kim, 1992).

Complicating an understanding of the appropriate measure of SES and LBW is the “complex joint effect patterns” of various SES measures with race--that is, the differential impact of SES measures based on race (Savitz et al., 2004). Income, for example, does not appear to have the same impact on LBW rates among Black and White women. At least one study has found that poverty increases the risk of LBW among White women, but NOT among Black women: the LBW rates were “equally high” among poor White women and poor Black women--in other words, the racial disparity in LBW “is greatly reduced among the poor” (Starfield et al., 1991, p. 1172). Another study added a further level of complexity, finding that poverty was associated with LBW only among Black women with > 12 years of education, and only among White women with low educational levels (Savitz et al., 2004).

A final complicating issue in understanding the role of SES in LBW is that of “neighborhood effects.” Stated simply, individual-level risk factors (such as maternal education, age, prenatal care utilization, and health insurance status) “behave differently depending upon the characteristics of the neighborhood of residence” (O’Campo, Xue, Wang, & Caughy, 1997, p. 1113). Specifically, one study found that census-tract variables, such as rate of homeownership, unemployment rate, crime rate, and per capita income, “modify the associations between individual-level factors and the risk of low birthweight” (O’Campo et al., 1997, p. 1115). Another study of LBW among Hispanics also found that place of residence influences birthweight (Collins & Shay, 1994). That study demonstrated that, although LBW rates are “paradoxically” low for Hispanics (that is, similar to rates among Whites despite being “a poorly educated and medically underserved minority”), there are variations in LBW rates based on nativity and residence

of mother (Collins & Shay, 1994, p. 184). This study found that, while LBW rates increase as census tract median family income decrease, “urban poverty is negatively associated with Hispanic birth weight only when the mother is Puerto Rican or a U.S. born member of another subgroup” (such as Mexican-American) (Collins & Shay, 1994, p. 184).

Such findings have prompted some to call for less emphasis on individual-level attributes when considering LBW (and IMR) and a greater focus on community-level or structural factors such those census-tract variables noted above, as well as other community variables such as racial composition of neighborhood and state, urban concentration of population, and the percent of population living below poverty (Bird, 1995). The link between these variables and birth outcomes (LBW and IMR) highlights the role of poverty in infant health, and the need to explore the role of public policy and its impact--intended or unintended--on poverty and health.

Public Policy and Children’s Health

The Relationship Between Public Policy and Health

Defining Public Policy

Prior to any discussion of the relationship between public policy and health, a clear understanding of what “public policy” is must exist. In a practical sense, public policy includes the laws and regulations passed at the local, state, and federal levels, and court decisions at the state and federal levels. Restricting a definition of public policy to existing laws or “recorded decisions” (Howlett & Ramesh, 2003), however, fails to

incorporate: (a) important aspects of the policymaking process such as problem identification and agenda setting, analysis of potential choices or alternatives, the decision-making process, and the participants/actors and their influence (Howlett & Ramesh, 2003; Koven, Shelley, & Swanson, 1998); (b) a recognition that not only does governmental *action* set policy, but governmental *inaction* as well (Dye, 1972); (c) the “inter-relatedness” of decisions made by multiple actors or groups of actors that address a single problem (Jenkins, 1978); (d) any potential discrepancy between the intention of and the implementation of recorded decisions (Hanks, Herz, & Nemerson, 1994); or, importantly, (e) the political forces, competing interests, and constraints involved (Howlett & Ramesh, 2003).

While there is little consensus about a single definition of public policy, a definition using common elements between definitions can be derived: Public policy is the sum of actions and inactions that are undertaken by the government or its agents through a decision-making process choosing among alternatives, made in the “public’s” name to “achieve societal goals” (Cochran & Malone, 2005), whether implemented as intended or not, and including any unintended consequences that might ensue (Birkland, 1997; Cochran & Malone, 2005; Dye, 1972; Howlett & Ramesh, 2003; Jenkins, 1978; Kingdon, 2003; Koven et al., 1998; Peters, 1984). Public policy includes a variety of international and domestic policy areas: economics, environment, education, agriculture, crime, family, welfare, and health (Koven et al., 1998). While these areas represent diverse sets of policies, policymaking within each policy area impacts the health of a population to varying degrees.

Understanding “Health Policy”

Health policy is generally considered the area of public policy that encompasses issues related to *medical care* (i.e., individual medical services) or *public health* (i.e., population-based health promotion and disease prevention measures). The term, however, is somewhat of a misnomer as it is not the only area of public policy that influences health--all areas of public policy encompass policies that can potentially impact health.

Health policy and medical care. Health policies targeting *medical care* include policies that:

1. facilitate access to health care, such as policies establishing public health insurance programs like the State Children's Health Insurance Program (SCHIP) (established under Title XXI of the Social Security Act by the Balanced Budget Act of 1997), a joint federal/state program providing health insurance for low-income children not financially eligible for Medicaid, and laws establishing public hospitals or health agencies;
2. regulate health insurance coverage, such as the Mental Health Parity Act of 1996, requiring most group health plans that offer mental health benefits to provide these benefits at a level equivalent to medical/surgical benefits;
3. regulate specific medical procedures or services, such as the Mammography Quality Standards Act of 1992, establishing requirements designed to improve the quality of mammograms;
4. require the provision of specific health care services, such as the Emergency Medical Treatment and Active Labor Act of 1986 (EMTALA) (enacted as part of the Consolidated Omnibus Budget Reconciliation Act of 1985), requiring that emergency

rooms in Medicare-provider hospitals medically screen and stabilize all patients prior to transfer or discharge regardless of insurance status or ability to pay;

5. promote the quality of medical care provided, such as

--the case law surrounding the “medical informed consent doctrine,” requiring sufficient information be provided to the patient by the physician/appropriate representative for an informed decision regarding a treatment/procedure, stemming from such watershed cases as *Schloendorff v. Society of New York Hospitals* (1914) (articulating the premise that an individual’s freely given consent is required for each procedure), and *Canterbury v. Spence* (1972) (establishing a physician’s “duty to disclose” the risks posed by treatment);

--the case law surrounding “generally accepted standards of medical practice,” the standard of care for physicians in the performance of their duty, deviation from which can result in medical malpractice; and

--the accreditation, certification, and credentialing requirements established for individual health care providers and health care organizations/hospitals;

6. attempt to prevent fraud and abuse in medical charges, such as the Stark Act which limits physician referral of Medicare/Medicaid patients to entities in which the physician has a financial interest;

7. promote patient privacy, such as the privacy regulations established by the Health Insurance Portability and Accountability Act of 1996 (HIPAA) regarding “identifiable health information”; and

8. address ethical issues in the health care arena, such as the case law regarding “end of life” decisions through “advance directives” (e.g., living wills), legal instruments

by which patients indicate the health care they wish provided should they become unable to make medical decisions.

Health policy and public health. Health policies addressing the *public health* system include policies:

1. intended to prevent the spread of infectious or communicable health diseases and conditions, such as

--policies requiring vaccination as a condition of school enrollment, rooted in such cases as *Jacobson v. Commonwealth of Massachusetts* (1905) (upholding the states' authority to require compulsory vaccination) and *Zucht v. King* (1922) (upholding the city's authority to ban from school all those without vaccination certification); and

--policies that enable federal, state, and local governments to examine or test individuals suspected of carrying a communicable disease or individuals in relevant occupations (e.g., health care workers, food handlers) and to quarantine or isolate individuals with or thought to be infected by specific diseases;

2. directed toward the prevention or reduction of noninfectious diseases, such as reducing tobacco-related health conditions by banning cigarette advertising on radio and television, beginning with *Capitol Broadcasting Co. v. Mitchell* in 1971 (restricting the advertisement of cigarettes in broadcast media) or by limiting the sales of cigarettes to minors, and policies that establish occupational safety standards;

3. aimed at preventing injuries, such as mandatory seat-belt and motorcycle helmet use; and

4. that promote the general health and welfare of the public, such as consumer safety laws, regulations for and inspections of food, drugs, and medical devices, as well

as inspection and licensure of restaurants, housing, and the workplace. Despite such examples of policies promoting public health and welfare, it should be noted that the U.S. Supreme Court has held that the state does not have an affirmative duty to (i.e., is not required to) protect its citizens (*DeShaney v. Winnebago County Department of Social Services*, 1989).

Quite clearly, medical care and public health measures, and the regulation thereof, are *essential* to individual and population health. Medical care and the equitable access to it are, however, “necessary but not sufficient” for health (see, Bauman et al., 2006; De Vogli et al., 2005; McKeown, 1979; Pincus et al., 1998; Seguin et al., 2005): “Health is a product of many social, political, and economic forces and institutions outside of health that produce risks for health and illness” (Hofrichter, 2003, p. 33). Health status is influenced not only by policies that specifically address medical care and the public health system, but also (and arguably more significantly) by policies that create, regulate, or modify social circumstances and conditions--the “social determinants of health” (M. Marmot & Wilkinson, 1999). Although *medical* advances have contributed to an improvement in health, significant improvements in health status in the U.S. during the 19th and 20th centuries can also be linked to major *social* reforms (McKeown, 1979)--strides in sanitation, the provision of safe food and housing, improvements in living and working conditions, the abolition of child labor, Social Security, and advances in civil rights among others (see, e.g., Gostin, 2000; Hofrichter, 2003).

The Intersection of Public Policy and Public Health

While the impact of health policies on health status is obvious and direct, policies that affect social circumstances also have a bearing on health status, directly or indirectly,

as an intended or unintended consequence. The following are seven examples of public policies--not typically considered health policy--that have impacted health.

Tax policies and birthweight. As discussed earlier, there is a well-documented link between maternal smoking and the incidence of low birthweight. Various researchers have attempted to estimate one “external cost of smoking” by considering the “long-term costs of low birth weight”--calculated as \$4.80 per pack by one 1991 estimate (W. N. Evans & Ringel, 1999, p. 136). One 1999 analysis using state-level birthweight data and state cigarette excise tax rates estimated the existence of “causal relationships between cigarette taxes, maternal smoking behavior, and birth outcomes” such that “increases in cigarette tax rates have a beneficial impact on mean birth weight” (W. N. Evans & Ringel, 1999, p. 152). In other words, as cigarette excise taxes increase, maternal smoking during pregnancy decreases, and average birth weight increases. A 2003 study confirmed this finding, expanding it to include a decrease in “relapse smoking” (less resumption of smoking) with increases in cigarette taxes (Colman, Grossman, & Joyce, 2003).

Employment policies and spread of infection. Paid time off (including paid sick leave), one of several non-wage employee benefits offered by many employers, is not required by U.S. federal law (Lovell, 2004). Nearly half of all private-sector U.S. workers have no paid sick leave (59 million) and even fewer have paid time off for care of sick children--and recent trends suggest employers are reducing their existing sick leave programs (Lovell, 2004). Without paid sick leave, employees are--quite simply--more likely to come to work while sick. Concerns have been raised about worker productivity and quality among sick workers, but it is the increase in the spread of

infection among other workers (Skatun, 2003) and among clients (including patients in the health care setting) (Li, Birkhead, Strogatz, & Coles, 1996) that is most troubling. The inclusion of paid time off as an employee benefit decreases sick workers coming to work, thereby decreasing the spread of infection within a population.

Civil rights legislation and infant health. In the years immediately following World War II, the U.S. experienced a decrease in infant mortality--much of it related to the decline in mortality due to antibiotic-responsive conditions (e.g., pneumonia and diarrhea) (Almond, Chay, & Greenstone, 2003). This improvement in infant health was enjoyed disproportionately by White infants over Black infants, particularly for the postneonatal period (Almond et al., 2003). This disparity between White-Black infant mortality continues, but narrowed significantly in the mid-1960s: “Relative to pre-existing trends, the black infant mortality rate declined sharply from 1965-1975,” particularly among Black infants in the rural South (Almond et al., 2003, p. 10). While the Black IMR had increased between 1955 and 1965, the rate was nearly *halved* between 1965 and 1971 (Almond et al., 2003).

This noted decline in Black IMR “was driven by a remarkable decrease in causes of postneonatal death considered preventable by medical treatment, such as diarrhea and pneumonia” (Almond et al., 2003, p. 2). Research has demonstrated that access to hospital care among Black mothers and infants increased--especially in the rural South (a radical departure from its pre-Civil Rights racial segregation)--following the passage of Title VI of the 1964 Civil Rights Act which “prohibited discrimination in all institutions receiving federal funding, including public hospitals” (Almond et al., 2003, p. 7). The landmark Civil Rights legislation, therefore, prohibited racial discrimination in public

hospitals, thereby increasing access to health care among Black mothers and infants, and ultimately decreased infant mortality and improved the health of Black infants in the rural South (Almond et al., 2003).

Traffic pattern policies and child injuries. Pedestrian injuries due to automobile collisions are a leading cause of death among children aged 5-14 (Tester, Rutherford, Wald, & Rutherford, 2004). These accidents are more likely to occur near a child's home, and are more likely to involve head injuries and/or be fatal (Tester et al., 2004). Various policies have been implemented to modify traffic patterns to increase child safety in these residential areas: speed bumps, street closures, median barriers, traffic circles, restrictions on street parking, and speed limits (Agran, Winn, Anderson, Tran, & Del Valle, 1996; Tester et al., 2004). Of these safety measures, research has shown that it is the presence of speed bumps, restrictions on street parking (to increase visibility), and speed limits (when enforced) that reduce the risk of child pedestrian injuries (Agran et al., 1996; Tester et al., 2004).

Environmental policies and infant mortality. In 1970, Congress passed the Clean Air Act (CAA), creating a role for the federal government in what had previously been under the purview of state governments--reducing air pollution (Chay & Greenstone, 2003a). With this legislation, Congress established the Environmental Protection Agency (EPA) whose tasks included setting the maximum allowable concentration for total suspended particulates (TSPs) (a significant part of air pollution) (Chay & Greenstone, 2003a). Following the passage of the CAA, the U.S. experienced an immediate improvement in air quality--AND an immediate reduction in infant mortality, particularly during the neonatal phase (Chay & Greenstone, 2003a). This decline in neonatal infant

mortality has been attributed to the reduction in exposure to TSPs both during infancy and during the prenatal period (Chay & Greenstone, 2003a), with subsequent research documenting a 1% decline in TSPs resulting in a 0.35% decline in infant mortality at the county level (Chay & Greenstone, 2003b).

Land use policies and adolescent obesity. Childhood and adolescent obesity rates in developed countries have risen to epidemic proportions in recent years (Reilly, 2006). Among the many factors contributing to this significant rise in obesity rates has been the rise in consumption of fast-food (Austin et al., 2005). In the past three decades, “fast-food retail sales in the United States have soared 900%, from \$16.1 billion in 1975 to a projected \$153.1 billion in 2004” (Austin et al., 2005, p. 1575). With the increase in childhood and adolescent obesity and the known link between obesity and fast-food consumption, research has increasingly focused on the role of fast-food consumption among children and adolescents. Recent research shows that not only are fast-food restaurants concentrated within short distances to schools (Austin et al., 2005), but that this proximity is directly correlated with youth obesity rates (Davis & Carpenter, 2009). The absence of land use policies and zoning ordinances regulating the location of fast-food restaurants--particularly their proximity to schools--is, therefore, a factor in the current childhood and adolescent obesity epidemic.

Home mortgage policies and vector-borne diseases. Home mortgage policies, including adjustable rate mortgages, followed by a downturn in the housing market and a sluggish economy have all contributed to the recent increase in home foreclosures (Reisen, Takahashi, Carroll, & Quiring, 2008). The rise in home foreclosures has been particularly notable in western states, such as California and Nevada, where outdoor

swimming pools are more common. Home foreclosures often result in abandoned properties with neglected pools, creating new habitats in these unmaintained pools for *Culex pipiens* mosquitoes, the vectors for West Nile Virus (Reisen et al., 2008). Although mortgage-holding banks are officially responsible for upkeep of these properties, in the absence of banks assuming the upkeep, mosquito control activities often fall to local vector control agencies (Reisen et al., 2008). Vector control activities by nonowner agencies, however, are often hampered by locked protective fencing surrounding pools (Reisen et al., 2008). In one study, the 300% increase in community foreclosures was “associated with a 276% increase in the number of human West Nile virus cases during the summer of 2007” (Reisen et al., 2008), p. 1747).

These are some examples of “non-health” public policies that affect health--particularly among children. When considering public policy and child health, however, one of the most significant areas of public policy is that of child welfare--policies that combat child poverty and promote child welfare and well-being.

Public Policy Addressing Child Poverty and Social Welfare

A History of Public Policy Addressing Child Poverty and Social Welfare

Early poor relief and social welfare efforts. As with many social customs and institutions, early colonists in America modeled their policies for the poor on existing British common law and customs (Axinn & Stern, 2001; Handler, 1995; Katz, 1996; Trattner, 1999). Arguably “the first legislation on welfare” was the Statute of Laborers, enacted in 1349 in England (Handler, 1995, p. 10). This statute, enacted during a period of labor shortage following the devastation of the “Black Death,” attempted “to force

beggars to seek work by preventing the giving of alms” thereby seeking to “restrict laborers’ movement, require them to work, and fix their wages” (Handler, 1995, pp. 10-11).

The next significant piece of social welfare legislation was the English Poor Law of 1601. This law mandated work for the able-bodied under penalty of whipping, branding, or even death, and created a legal duty for family members to care for each other if within their means (parents for children and grandchildren, children for needy parents and grandparents) (Trattner, 1999). This “kin responsibility” denied public aid for anyone who had family that could take them in, and allowed the state to remove children from the home to indenture or apprentice them (Axinn & Stern, 2001; Katz, 1996). The law did, however, establish that poor relief was a public responsibility and that “the state had a responsibility to supplement ordinary efforts to relieve want and suffering and to insure the maintenance of life” (Trattner, 1999, p. 11). The law recognized “there were helpless or needy people who not only deserve such assistance but who had a legal right to it” and made poor relief a local responsibility, undertaken by the smallest governing unit (such as towns) (Trattner, 1999, p. 11). This emphasis on local responsibility created a disincentive for localities to accept the poor in their area (they were “potential recipients of relief”) and allowed for the return of the poor to the place of their legal residence (Axinn & Stern, 2001, p. 16).

Poor relief in Colonial America was accomplished either through “outdoor” relief (in the home) or through “indoor” relief (institutional) (Axinn & Stern, 2001; Katz, 1996; Trattner, 1999). Outdoor relief, money given to poor persons that were allowed to remain in their homes or in the homes of community members (at public expense), was

generally reserved for widows, children, the elderly, and the sick or disabled (Axinn & Stern, 2001; Katz, 1996). Indoor relief, on the other hand, removed the poor from private homes and placed them in public institutions--almshouses or poorhouses (Axinn & Stern, 2001; Katz, 1996). Although arguably more poor were helped through outdoor relief, Colonial-era poor relief stressed the use of indoor relief, and over time specialized institutions developed (such as workhouses, institutions for the mentally ill, the blind, and the deaf, orphanages, and penitentiaries or houses of correction) (Axinn & Stern, 2001; Katz, 1996).

The underlying principle of public responsibility for the poor did not, however, go unchallenged (Axinn & Stern, 2001). Opponents of poor relief questioned both the effectiveness of the system and whether most recipients actually needed relief (Katz, 1996; Trattner, 1999). These critics asserted, as with recent debates over welfare, that public assistance “destroyed character,” created a disincentive to work, and led to overpopulation among the poor (Katz, 1996, p. 18; see also, Trattner, 1999). In short, the argument went, the poor laws system “was not only morally wrong . . . but it was also economically unsound” (Trattner, 1999, p. 50). Some called for the abolition of poor laws while others claimed such relief was best accomplished by private charity (Axinn & Stern, 2001; Katz, 1996). Through these public dialogues a distinction developed that persists today--a distinction between the “impotent poor” (deserving poor) and the “able poor” (undeserving poor) (Katz, 1996, p. 19). Over time, policies would diverge for those deemed “deserving poor” (such as widows, children, and veterans) and those deemed “undeserving poor” (such as able-bodied persons and unmarried mothers) (Katz, 1996; Trattner, 1999).

The years between the Revolutionary War and the Civil War were “years of major political, economic, and social changes” (Axinn & Stern, 2001, pp. 37-38). Population changes, resulting from higher life expectancy, increased immigration, and geographic expansion, as well as economic changes, due in large measure to industrialization, led to an expanded market economy and increased urbanization (Axinn & Stern, 2001). People making their way to work in industries often crowded into cities and increasingly relied on money wages (rather than the self-sufficiency of farming), making them more susceptible to economic downturns and unemployment spikes (Axinn & Stern, 2001). Overcrowding in the cities, the insufficiency of poor relief (particularly in the face of economic depressions), and the “deplorable” conditions of public institutions (poorhouses and other specialized institutions) were significant factors in the trend of America’s social welfare system toward reform efforts (Axinn & Stern, 2001; Trattner, 1999). Reform movements focused their efforts on labor reform and the protection of vulnerable groups, focusing on improved sanitation, universal suffrage, humane treatment of the insane, rehabilitation efforts for criminals, the abolition of slavery, better poor relief, and “child-saving” (Axinn & Stern, 2001; Katz, 1996; Trattner, 1999). Child-saving would become a major focus of social welfare in the years following the Civil War (Katz, 1996).

In the aftermath of the Civil War, America again experienced “major political, economic, and social changes,” with population and economic growth, heightened urbanization and industrialization, and intensification of poverty (Axinn & Stern, 2001, pp. 37-38). The principle of public responsibility for poor relief was increasingly challenged and states reduced—abolished—their outdoor relief efforts, focusing instead on public institutions (Axinn & Stern, 2001; Trattner, 1999). With the decline in state

and federal social welfare efforts and the increased need for assistance in the wake of the economic depressions of that era, private charitable organizations proliferated to fill the void in poor relief (Axinn & Stern, 2001; Trattner, 1999). This growth--or “excess”--in private relief-giving agencies brought claims of inefficiencies and fraud, and raised concerns over the potential of relief to encourage “indolence” (Trattner, 1999, p. 92).

It was in this environment that the Charity Organization Movement developed to increase the efficiency of charity work and provide a rational or “scientific” approach to charity work (Axinn & Stern, 2001; Trattner, 1999). The resulting charity organization societies sprouted up in cities across the country, referring “worthy” relief applicants to appropriate agencies and maintaining registries of these applicants and any assistance given (Trattner, 1999). Criticism of these charity organization societies was common, however, for distributing relief based on the “worthiness” of applicants and focusing on the “moral lapse” of the poor as the problem rather than their environment (Axinn & Stern, 2001; Trattner, 1999, p. 99). These societies later broadened their view in light of data (often their own product) that demonstrated the predominant role of social conditions (rather than personal weaknesses) in poverty--unemployment, sickness, work-related injuries--and set the stage, with other efforts such as the Settlement House Movement, for subsequent reform efforts (Trattner, 1999).

Child-saving and beyond. Many of the significant reform efforts of the post-Civil War era centered on child welfare. The focus on child welfare was broadly accepted given: the compelling appeal of children in need; the great numbers of children in need (tens of thousands were orphaned as a result of the Civil War); a greater awareness of the

process of child development; a recognition that children were the key to the nation's future; and, the lack of responsibility of children for their plight (Trattner, 1999).

Reform efforts directed toward child welfare, or child-saving, took a variety of forms. Children were increasingly removed from institutions and placed in private homes (foster care) due to a growing perception that institutions were not positive environments for children (Katz, 1996; Trattner, 1999). Such foster care included the "orphan trains" in which orphans were removed from Eastern cities and placed with agrarian families of the West (Trattner, 1999). Children not placed in foster homes were often apprenticed out or removed from the poorhouses and placed in institutions for dependent children--as a result, the number of these institutions rose dramatically between 1870 and 1910 (Katz, 1996). A significant advance in promoting child welfare occurred in 1875 when New York became the first state to create a Society for Prevention of Cruelty to Children (SPCC) (Myers, 2006). Fashioned on the already established society for protection of animals, the SPCC was created following a dramatic rescue--and the ensuing legal action--of a young girl, Mary Ellen, from an abusive home (Myers, 2006). Other societies were created across other states, as were other nongovernmental agencies directed toward child protection (Myers, 2006).

Other child-saving efforts included compulsory school attendance laws, the establishment of juvenile courts and reform schools, and campaigns to end child labor with laws that limited or regulated child labor (Katz, 1996; Trattner, 1999). The growing belief in family preservation limited government efforts to remove children from families due to poverty, and facilitated the implementation of state Widows'/Mothers' Pensions laws that provided a small stipend to widows with dependent children and ultimately laid

the framework for our current welfare system (Katz, 1996). Another aspect of child-saving was encompassed in the advent of the Public Health Movement. At a time when the mortality rates for children under 5 years of age were as high as 135 per 1000 live births, true child-saving would need to address this tragedy (Trattner, 1999, p. 140). With a focus on improved sanitation, nutrition, and living standards and with a new awareness of “germ theory,” public health efforts--such as public education, mandatory reporting of communicable diseases, segregation and treatment of disease carriers (e.g., Tuberculosis sanitariums), ordinances requiring health-promoting activities (e.g., pasteurization of milk), and compulsory vaccination--resulted in a decrease in infectious diseases and a decline in infant and child mortality (Katz, 1996; Trattner, 1999).

Efforts directed toward child welfare continued into the Progressive Era of the early 20th century. A 1909 White House Conference on Dependent Children did much to promote standards for child care and foster care and led to the establishment of adoption agencies, smaller-scale boarding of those not “suitable” for adoption, and later the U.S. Children’s Bureau, established in 1912 (Trattner, 1999). Charged with investigating and reporting on “all matters pertaining to the welfare of children and child life,” the children’s bureau, under the able direction of Julia Lathrop, “soon became the central, and in some cases the sole, source of authoritative information about the welfare of children and their families throughout the United States” (Trattner, 1999, p. 218). Choosing to first target infant mortality, the children’s bureau found not only “shockingly high infant mortality [largely caused by] deficient family income as well as by a lack of adequate medical facilities and hence prenatal care--but they discovered an exceedingly

high death rate for mothers as well”; more women died from childbirth in the United States each year than from any other cause except tuberculosis (Trattner, 1999, p. 219).

Such tragic findings were unacceptable and prompted Julia Lathrop to push for improved maternal and child health facilities, efforts later embodied in the Sheppard-Towner Act of 1921 (Trattner, 1999). Passed amidst cries of “socialized medicine” and concerns over female-run public clinics, this Act provided grants to states to establish maternal and child health centers (with care provided regardless of the mother’s economic or work status) and provide targeted health-promoting services such as public health nursing, and strengthened the state health department system (Stetson, 1997; Trattner, 1999). Between 1921 and 1929, 45 states opened nearly 3000 public maternal and child health centers (predominantly in rural areas) and the nation saw a significant drop in infant and maternal mortality rates (Trattner, 1999). Despite the documented successes of the Act, with the weakened economy and the political environment of 1929 Congress responded to the cries of socialized medicine and criticisms by the (male) medical profession and allowed the Act to expire without renewing it (Katz, 1996; Trattner, 1999). The Act did, however, bring the “federal government into the field of child welfare through the area of health” and laid the foundation for many of the subsequent joint federalism programs (“cooperative federal-state programs”) established by the Social Security Act of 1935 (Trattner, 1999, p. 221).

In addition to the establishment of the Children’s Bureau--with its subsequent promotion of the Sheppard-Towner Act--the 1909 White House Conference on Dependent Children was also instrumental in facilitating the Widows’/Mothers’ Pensions Movement (Trattner, 1999). With many relief agencies refusing to assist women capable

of working, widows were often faced with the choice of working long hours for low wages, or giving their children up for placement in institutions or adoption (Trattner, 1999). With the very real possibility of having children unsupervised (with its potential for delinquency) or removed from the home (either voluntarily or as a result of mother's ill health and subsequent institutionalization), public sentiment generally favored keeping children in their own home when possible (Trattner, 1999). States, therefore, created pension statutes that provided small stipends to allow widows to stay in the home and care for their children (Katz, 1996; Trattner, 1999). These statutes established "suitable homes" requirements and provided stipends to widows with dependent children but not divorced mothers, often involving administrative hurdles as well as clear discrimination based on race, natality, and other factors (Katz, 1996; Trattner, 1999).

As with the Sheppard-Towner Act, the Widows'/Mothers' Pension Movement laid the groundwork for subsequent social welfare programs (Axinn & Stern, 2001; Trattner, 1999). Under the Social Security Act of 1935, this system would become the social welfare program entitled Aid to Dependent Children (ADC), later the Aid to Families with Dependent Children (AFDC), and still later the Temporary Assistance for Needy Families (TANF) under Welfare Reform. This transition is discussed further later in this chapter (see "The Era of Welfare Reform").

Approaches of Public Policy Addressing Child Poverty and Social Welfare

Efforts to address child poverty can be divided into three general approaches: (a) preventing poverty by increasing individual earning capacity through employment-related initiatives; (b) reducing the level of poverty by supplementing income with other sources of cash; and (c) mitigating the adverse effects of poverty by providing non-cash

assistance, such as food, health care, housing, and other forms of assistance (Levitan, Mangum, Mangum, & Sum, 2003; Plotnick, 1997; see also, Devaney, Ellwood, & Love, 1997).

Preventing poverty through employment-related initiatives. Efforts to increase earning capacity attempt to “eliminate the causes of poverty rather than merely mitigate its symptoms” through various “employment-related initiatives” (Levitan et al., 2003, p. 33). Such initiatives can be broadly categorized into four groups:

1) education, occupational training, job search assistance, and subsidized private-sector jobs; 2) attempts to restructure or supplement the normal workings of the labor market through minimum wages, public employment, and antidiscrimination laws; . . . 3) economic development programs to rejuvenate depressed urban and rural areas. (Levitan et al., 2003, pp. 33, 35)

and, (4) efforts to facilitate employment among low-wage workers (Plotnick, 1997).

1. *Employment initiatives.* Because of the integral link between educational attainment and individual earnings, the federal government has often focused on education initiatives in its antipoverty efforts: attempts to improve the education of historically disadvantaged groups (e.g., Head Start); providing federal funds (e.g., funds to address educational needs of children with disabilities); establishing performance standards and accountability (e.g., “No Child Left Behind” program); and promoting vocational and technical education (e.g., Carl D. Perkins Vocational and Applied Technology Act of 1990) (Levitan et al., 2003). Other education-related initiatives include training and development programs that offer a “second chance at employment preparation and job placement services” (e.g., Workforce Investment Act of 1998) (Levitan et al., 2003, p. 213).

Job preparedness, however, is not always sufficient to obtain a job, and, even if obtained, full-time employment is not always a guarantee of rising out of poverty. Other antipoverty employment-related efforts, therefore, focus on: expanding job opportunities, particularly in rural and urban areas (e.g., economic development programs); improving access to available jobs (e.g., public employment services); decreasing non-merit barriers to jobs (e.g., enforcement of antidiscrimination labor policies); and, increasing the “rewards” offered by work (e.g., establishment of a federal minimum wage) (Levitan et al., 2003; Plotnick, 1997).

2. *Programs facilitating employment.* The final aspect of employment-related antipoverty initiatives includes efforts directed at facilitating employment. These efforts focus on issues of child care, health care coverage, transportation, relocation of subsidized/low-income housing near job opportunities, among others (Plotnick, 1997). With the rising number of working mothers in the U.S.—31% of mothers of infants were employed in 1976 as compared to 59% in 1998 (Levitan et al., 2003)—and the mandatory employment provisions under the Welfare Reform legislation, access to child care is arguably the most significant of these issues facilitating employment.

“Child care is essential if low-income families—especially single-parent families—are to pursue self-support” (Levitan et al., 2003, p. 150). Low-income families pay a higher percentage of their family income for child care than do higher-income families (Levitan et al., 2003). Therefore, the federal government has assumed a role in supporting and funding child care services—although recent efforts have targeted middle- and upper-income families rather than low-income families (Levitan et al., 2003). One such federal child care program is the “child and dependent care tax credit” which allows

for “work-related child care expenses [to be taken as] a credit against federal income tax liability for a portion of actual child care expenditures” (Levitan et al., 2003, p.150).

Because it does not assist with any up-front costs of child care and does not apply to families whose incomes are too low to pay taxes, this program is of limited use to low-income families (Levitan et al., 2003).

Until the passage of Welfare Reform, the Social Services Block Grant served as the “primary source of child care assistance to the poor” from its introduction in 1981 (Levitan et al., 2003, p.150). Although this block grant from the federal government to the states gave states discretion over their utilization of funds, child care represented the largest single allocation of funds (Levitan et al., 2003). Under the Welfare Reform legislation, however, all federal sources of child care assistance were combined under the Child Care and Development Fund (discussed later) (Levitan et al., 2003, p.150). As with other aspects of Welfare Reform, the 1996 legislation gave states unprecedented discretion over utilization of child care funds--including the option of not funding child care at all (Levitan et al., 2003). State discretion extends to defining eligibility criteria (e.g., states may set family income limits at or *below* the federally defined “85 percent of the state’s median family income”), allocating additional state funds (e.g., states have the option of transferring up to 30% of TANF funds to their CCDF), and establishing health and safety standards for child care providers (e.g., licensing requirements) (Levitan et al., 2003, p. 152). Despite such funding, a report by the Department of Health and Human Services in 2000 estimated that “of [the] nearly 12 million children who are eligible for child care assistance, only 12 percent were receiving any help” (Levitan et al., 2003, p. 150).

Reducing poverty through cash supports. “The most direct assistance to the poor, though not necessarily the most effective or the most popular, is simply to give them money” (Levitan et al., 2003, p. 43). Policies that provide supplemental income--many of which are rooted in the Social Security Act of 1935--can be broadly categorized as either “social insurance programs” or as “public assistance programs.”

1. *Social insurance programs.* The two primary social insurance programs are Old Age, Survivors, and Disability Insurance (OASDI)--commonly referred to a “Social Security”--and Unemployment Insurance (UI). In contrast to public assistance programs “which provide income support on the basis of need alone,” social insurance programs are funded through payroll tax contributions and are distributed based on prior earnings (Levitan et al., 2003, p. 43). “The Social Security system taxes employed persons to support those who are retired, disabled, and survivors of deceased workers” (Levitan et al., 2003, p. 46). Social Security retirement benefits, though not intended to be “the sole source of retirement income,” represented the total retirement income for 18% of those aged 65 and over in 1998, and 90% of the retirement income for 30% of this population (Levitan et al., 2003, p. 46). Though not an antipoverty program, OASDI “is the primary reason that Americans over age 65 have the lowest poverty rates of any age group” (Levitan et al., 2003, p. 43). As a testament to both its antipoverty effect and the relationship between poverty and mortality, consider that, while the decline in the mortality rate for those aged 45-64 was 2.6-times greater in the 20 years following passage of OASDI than the decline in the 20 years prior to the Act, the decline in mortality rate for those aged 65 and older (the recipients of OASDI’s cash support) was

133-times greater in the 20 years after OASDI's passage than before (calculated from data available through the National Vital Statistics System [NCHS], *Historical Data*).

The second primary social insurance program is Unemployment Insurance (UI) which provides a temporary partial wage for unemployed persons. "Because eligibility and benefits are based on past earnings and work experience, rather than on need, the poor are often excluded or receive meager benefits" (Levitan et al., 2003, p. 54).

Additional restrictions on eligibility instituted in recent years, such as "minimum earnings" (that is, individual salary earnings must be higher than this state-established minimum in order to be eligible for UI benefits), have often served to exclude the poor from UI benefits: in 1975, fully 81% of all unemployed persons received UI benefits in 1975, while only 38% did in 1999 (Levitan et al., 2003).

2. *Public assistance programs.* In contrast to social insurance programs which are based on prior earnings and are available to all "covered persons" regardless of income, public assistance programs are "means-tested" and are provided on the basis of need alone (Levitan et al., 2003). The public assistance programs of the Social Security Act of 1935 included the Aid to Dependent Children (ADC) program (later the Aid to Families with Dependent Children (AFDC), and still later the Temporary Assistance to Needy Families (TANF) program), as well as the Old Age Assistance (OAA) program and the Aid to the Blind and Totally Disabled (ABTD) program (later combined as the Supplemental Security Income (SSI) program) (Levitan et al., 2003; Plotnick, 1997).

"These means-tested programs were financed, in part, by the federal government through formula matching grants but were administered by the states, which were given

considerable discretion in determining eligibility standard and benefit levels” (Joseph, 1999, p. 1).

Perceived by many as an extension of the Mothers’ Pensions programs, ADC was intended to help children who had been “deprived of parental support or care by reason of the death, continued absence from the home, or physical or mental incapacity of a parent” (Joseph, 1999, p. 2). Federal funding for ADC was initially less than that of both OAA and ABTD programs, with a designated federal match (or contribution) of one-third as compared to the one-half match provided in OAA and ABTD programs (Joseph, 1999). Although the federal matching rates were subsequently equalized in 1939, the number of people covered by the ADC program lagged behind the other public assistance programs throughout the 1940s and early 1950s--ADC enrollment, for example, was less than half that of OAA in 1945 (Joseph, 1999). In 1950, ADC coverage was extended to “needy adult caretakers (usually mothers) of dependent children” and was later further expanded to “some two-parent families in which the principal wage earner was able-bodied but unemployed” (at the discretion of the state), prompting a change in the program name to Aid to Families with Dependent Children (AFDC) in 1962 (Joseph, 1999, p. 3). The noted expansions in eligibility, the “growing numbers of ADC mothers [who] were divorced or never married,” as well as the rapid growth in AFDC caseloads would later lead to calls for--and ultimately enactment of --“welfare reform” (Joseph, 1999, p. 3).

Another public assistance programs with its beginnings in the Social Security Act of 1935 is the Supplemental Security Income (SSI) program. Formed in 1972 when two Social Security Act programs (OAA and ABTD) were combined, SSI “pays benefits to disabled adults [including the aged] and children who have limited income and

resources” (Social Security Administration [SSA], 2008). Over the years since the program’s inception, the profile of SSI recipients has changed. The “aged” progressively account for a smaller proportion of recipients (17% in 2006), while the proportion of “disabled” increases (83% in 2006) (Levitan et al., 2003; SSA, 2006). Although the category disabled encompasses a range of physical and mental disabilities, the most common disability is “mental disorder”--mental retardation and mental illness--accounting for half of all working-age recipients and nearly two-thirds of all childhood cases (Levitan et al., 2003). Among the disabled recipients, children (under 18 years) have represented “the greatest percentage growth since 1974--tripling between 1990 and 1996 alone,” in contrast to the decline in the proportion of aged from 58% of recipients in 1974, to 31% in 1999, to 17% in 2006 (Levitan et al., 2003, p. 74; SSA, 2006). Despite this percentage growth in recipients under 18 years, children accounted for less than 15% in 2006, with adults 18-64 years old representing 57% of the recipients, and those 65 years or older representing 28% (SSA, 2006). Due to concerns about this increase, however, restrictions on SSI eligibility were included in the 1996 Welfare Reform legislation in an effort to stem the tide of increasing enrollment (Levitan et al., 2003).

Not associated with the Social Security Act of 1935, the Earned Income Tax Credit (EITC) was enacted in 1975 due to the “anomaly of a situation in which the poor were paying taxes, while federal policy sought to raise their income” (Levitan et al., 2003, p. 82). The federal EITC reduces tax liability for low-wage earners--particularly those with children--through tax credits which vary according the number of dependent children (Levitan et al., 2003). If the tax credit exceeds the tax liability of low-wage earners, the family receives a refund, as compared to tax exemptions which reduce

taxable income (Levitan et al., 2003). Although the EITC is only available to employed individuals (that is, those with “earned income”), the program is viewed as a “powerful antipoverty tool” and is credited with “lift[ing] 2.6 million children out of poverty in 1999” (Levitan et al., 2003, p. 83). The EITC program underwent a series of expansions between the mid-1980s and the early 1990s, providing nearly \$31 billion in 2000 to almost 16 million families with children and 3 million wage-earners without children (Levitan et al., 2003).

While most of the cash support programs target either families with children or the aged, there are some programs available for other categories of individuals: veterans’ benefits, workers’ compensation, and general assistance. The first two are categorical with specific eligibility requirements. General assistance, however, is state-funded cash support for the needy who are not otherwise eligible for other cash support programs (typically non-aged, non-disabled males), or who are eligible--and in the process of enrolling--for other programs (Levitan et al., 2003).

Mitigating the effects of poverty through “non-cash” assistance. Programs that seek to mitigate the effects of poverty through non-cash assistance--that is, the provision of goods and services--occupy an increasingly large portion of the federal government’s antipoverty expenditures: in-kind assistance represented 15% of government “outlays in aid of the poor” in 1960, 50% in 1968, and fully 80% in 2000 (Levitan et al., 2003, p. 89). Most of these in-kind assistance programs focus on three basic needs--“medical care, food, and shelter”--with social services representing a fourth and final category (Levitan et al., 2003, p. 89).

1. *Medical care.* Because “health services are now considered to be an essential ingredient of even a minimum standard of living . . . the federal government assumed major responsibility for funding health care for the aged and the poor” with its enactment of two significant health insurance programs in 1965: Medicare and Medicaid (Levitan et al., 2003, p. 90). Medicare and Medicaid were enacted as Title XVIII and Title XIX of the Social Security Act in 1965, extending health insurance to those aged 65 and older and to “low-income children deprived of parental support, their caretaker relatives, the elderly, the blind, and individuals with disabilities” respectively (CMS, *Key Milestones in CMS Programs*, p. 1). While Medicare does have some antipoverty effect through its coverage of the elderly, it is Medicaid that focuses more directly on the poor.

Although states administer the Medicaid program through joint federal-state funding and determine eligibility criteria, the federal government specifies basic health services that must be offered, including the Early and Periodic Screening, Diagnostic, and Treatment (EPSDT) services for children. EPSDT services--medical, dental, vision, and hearing--are intended to “assess [children’s] health needs through initial and periodic examinations and evaluations, and also to assure that the health problems found are diagnosed and treated early” (CMS, *Medicaid Early & Periodic Screening & Diagnostic Treatment Benefit*, p.1). As discussed previously, Medicaid underwent a series of expansions in eligibility and services throughout the 1980s, but was explicitly de-linked from AFDC under the 1996 PRWORA legislation. Concerns over how children would fare with this change among other factors, led to the creation of the State Children’s Health Insurance Program (SCHIP), discussed earlier, under Title XXI of the Social Security Act.

In addition to these health insurance programs, the federal government also funds community health centers to provide “health care services to medically underserved communities and vulnerable populations” (Health Resources and Services Administration [HRSA], *The Health Center Program*, p. 1). These community-based centers “serve populations with limited access to health care . . . includ[ing] low income population, the uninsured, those with limited English proficiency, migrant and seasonal farmworkers, individuals and families experiencing homelessness, and those living in public housing” (HRSA, *The Health Center Program*, p. 1).

During the 1970s, the federal government expanded Title V (Social Security Act) health care programs targeting maternal and child health among low-income families (HRSA, *Maternal and Child Health*; Levitan et al., 2003). In 1981, these programs were converted to a Block Grant Program (HRSA, *Maternal and Child Health*), with reduced funding and a subsequent reduction in prenatal and delivery services in most states (Levitan et al., 2003). The Maternal and Child Health Services Block Grant Program continues to provide prenatal and postnatal care for women as well as preventive care for children (HRSA, *Maternal and Child Health*). Other federal programs address special concerns in maternal and child health, such as Healthy Start which targets high-risk women and infants, and the Universal Newborn Hearing Screening which detects hearing impairments in infants (HRSA, *Maternal and Child Health*).

2. *Food.* The federal government’s role in providing food to the poor began during the Great Depression with a New Deal food program that redistributed farm surpluses to needy persons and schoolchildren (Devaney et al., 1997; Levitan et al., 2003). Although the program was discontinued in 1943, a recognition of the ongoing

need for a food program in 1962 led to the creation of a pilot food stamp program which was later codified under the Food Stamp Act of 1964 (Devaney et al., 1997). The Food Stamp Program (FSP) was expanded during the 1970s and became “the cornerstone of the public assistance safety net for low-income individuals” with the federal government funding the benefit, and the federal government and states sharing the administrative costs (Devaney et al., 1997, p. 92). The FSP, renamed the Supplemental Nutrition Assistance Program (SNAP) in October 2008, provides low-income households with debit cards to be used in grocery stores; the amount of the benefit is determined by the number in the household and demonstrated need, and the distribution is monthly (J. A. Klerman & Danielson, 2009; Wiseman, 2009).

The FSP was cut, however, under PRWORA: “The act reduced benefits, eliminated benefits for most groups of legal immigrants, and tightened work requirements for able-bodied adults without dependents” (U.S. Department of Agriculture [USDA], Economic Research Service, 2007, p. 32). Specifically, PRWORA gave states the option of withholding part—or all—of a family’s Food Stamps benefit for failure to comply with work requirements. The FSP caseloads had begun to decline in the early 1990s largely as a result of favorable economic conditions, but experienced a sharp drop following Welfare Reform given the new restrictions on eligibility and in part due to administrative barriers among other factors (J. A. Klerman & Danielson, 2009). It should be noted that, while PRWORA eliminated FSP benefits for most legal immigrants, the 2002 Farm Bill restored eligibility of major groups of legal immigrants.

In addition to the FSP, the federal government also funds nutrition programs that target vulnerable populations: pregnant/postpartum women and children. The Special

Supplemental Food Program for Women, Infants, and Children (WIC) “was established in 1972 to counteract the negative effects of poverty on prenatal and pediatric health” (USDA, Food and Nutrition Service, 2001, p. 13). Because pregnancy and infancy are such critical periods for subsequent health and development, WIC provides “food supplements, nutrition education, and access to health care” for pregnant and postpartum women, infants, and children between 1 and 4 years of age (USDA, Food and Nutrition Service, 2001, p. 13). Significant--though not universal--evidence demonstrates positive health effects of WIC participation ranging from improved birth outcomes, improved nutritional status among infants and children, and higher immunization rates (Devaney et al., 1997; USDA, Food and Nutrition Service, 2001). Like the restrictions and reductions PRWORA placed on the FSP, WIC was also impacted by PRWORA: the legislation eliminated the requirement of outreach for the WIC program, and denied eligibility to women in prison or juvenile detention (USDA, Food and Nutrition Service, 2001). In addition, PRWORA gave states the option to exclude immigrants from the WIC program, though no states have done so.

Federal nutrition programs that target school-aged children are collectively referred to as School Nutrition Programs: the National School Lunch Program (NSLP), the School Breakfast Program (SBP), and the Special Milk Program (SMP) (Devaney et al., 1997). Created in 1946, the NSLP now provides subsidized or free lunches to more than 30 million low-income children (USDA, Food and Nutrition Service, n.d.). The SBP program was initiated in 1966 in response to the large number of children arriving at school without breakfast (Devaney et al., 1997). Like the NSLP, the SBP provides a free or reduced-price meal (breakfast) to low-income children (Levitan et al., 2003). The

SMP, providing free or low-cost milk to low-income children, is available only in schools that do not participate in the other school nutrition programs; as the NSLP and the SBP programs have expanded, the SMP has decreased in scope over time (Levitan et al., 2003).

3. *Shelter*. Although food represents an “absolute biological necessity . . . shelter now outweighs food in the budgets of most poor families” (Levitan et al., 2003, p. 102). The federal government, therefore, seeks to provide a “decent home and suitable living environment for every American family” through housing assistance for low-income families (Devaney et al., 1997, p. 17). These housing subsidies can be categorized as either household-based subsidies or project-based subsidies (Devaney et al., 1997). “Household subsidies provide rent subsidies to families to use in existing privately owned apartments as long as the rent is below the fair market rent” (Devaney et al., 1997, p. 17). Project-based subsidies, on the other hand, “include low-rent public housing and Section 8 construction or rehabilitation, through which the federal government subsidizes the rents of apartments or housing units built by private developers” (Devaney et al., 1997, p. 7).

“Section 8 housing” refers to housing assistance subsequent to Section 8 of the Housing and Community Development Act of 1974 (Levitan et al., 2003). Section 8 “subsidizes construction and operation of housing project and provides vouchers with which eligible renters can obtain access to nonproject rental units in the community” (Levitan et al., 2003, p. 107). While the majority of housing assistance is in the form of project-based subsidies, recent trends have been toward increased authorization of household-based subsidies (Devaney et al., 1997). The Public Housing and Reform Act

of 1998 was one such effort, attempting to “reduce concentrations of poverty in public housing projects while protecting access to housing assistance for the poorest families” with a shift away from Section 8 housing and toward rent vouchers (Levitan et al., 2003, p. 106).

4. *Social services.* In addition to the “basic essentials” of medical care, food, and shelter, programs that seek to mitigate the effects of poverty include those that provide “social services.” Such social services programs range from enrichment opportunities with Head Start to legislation providing for child protection and placement.

Established in 1965, “Head Start programs promote school readiness by enhancing the social and cognitive development of children through the provision of educational, health, nutritional, social and other services” to low income children (Administration for Children & Families (ACF), *About the Office of Head Start*, p. 1). Head Start provides “comprehensive child development services” to nearly one million children each year through settings with low teacher-student ratio and parental involvement--including employment (ACF, *About the Office of Head Start*). In 1995, an Early Head Start program was added to serve the needs of pregnant women and children under age 3 (Levitan et al., 2003). Various studies of Head Start’s outcomes have shown positive cognitive, achievement, and behavioral effects (Devaney et al., 1997; Levitan et al., 2003). Enrollment is open to children (birth to 5 years of age) whose family income is below the poverty line, whose family receives TANF or SSI, and to children in foster care, with programs having the option of enrolling up to 10% of children who do not meet these criteria (ACF, *About the Office of Head Start*). Despite at least 90% of

enrolled children being from these at-risk groups, the program reaches less than half of all eligible children (Levitan et al., 2003).

Though not strictly focused on low-income children, child welfare and protection programs and legislative efforts are addressed here because “poor children face more than their share of . . . child neglect, abuse, and emotional disturbances” (Levitan et al., 2003, p. 141). As discussed earlier, many of the earliest social welfare programs in the U.S. were directed toward child welfare and protection. Although state child protection societies were established as early as 1875, the federal government’s official role in child welfare began in 1909 with the “White House Conference on the Care of Dependent Children” and the subsequent establishment of the Children’s Bureau in 1912. As do many other social welfare and antipoverty efforts, federal child welfare programs have their roots in the Social Security Act of 1935.

Federal child welfare legislation typically addresses child protection issues (e.g., preventing child abuse and neglect), out-of-home care (e.g., foster care), and permanent child placement (e.g., adoption) (see, ACF, *Characteristics and Financial Circumstances*). Title IV-B of the Social Security Act established the Child Welfare Services Program which provided grants to states to support child protective services (Levitan et al., 2003). A 1961 amendment to that act, Title IV-E, established the Foster Care Program in which funds were provided to states to support safe out-of-home placement through foster care payments (Levitan et al., 2003). In 1974, federal legislation, Child Abuse Prevention and Treatment Act (CAPTA), provided funds to states to address child abuse and neglect and mandated that states establish child abuse reporting laws--an effort states had begun during the 1960s (Levitan et al., 2003). Since

1974, Congress has enacted a series of bills amending the aforementioned legislation to better protect children, and expanding the scope to permanent placement and adoption (ACF, *Characteristics and Financial Circumstances*).

Although there is debate as to the relative advantages of the three different antipoverty approaches--prevention through employment-related efforts, reduction through cash support, and mitigation of its effects through non-cash assistance--it is clear that the most effective strategy would use all three complementary strategies. Welfare Reform--with its mandatory work requirements, its creation of the TANF cash assistance program, and its restrictions on FSP eligibility--draws from all three antipoverty approaches.

The Era of Welfare Reform

From Welfare to "Welfare-to-Work"

The federal government's role in providing income support for low-income families began in 1935 with the Aid to Dependent Children (ADC) program (Blank & Blum, 1997; Larner et al., 1997; Stetson, 1997). This program, enacted under the Social Security Act of 1935, "provided a subsidy to families with fathers who were deceased, absent, or unable to work" (Blank & Blum, 1997, p. 29). Based on the premise that children are "innocent of their parents' misfortunes and mistakes," and assumptions that men provided the family's income (Larner et al., 1997, p. 5) while women were caregivers (Stetson, 1997), the program sought to protect children from poverty by providing cash assistance (Larner et al., 1997) that offered "a reasonable subsistence compatible with decency and health" (Blank & Blum, 1997, p. 30).

While the federal government established the ADC program guidelines, states were granted much discretion in defining eligibility and benefit levels (Page & Larner, 1997). Some states used this discretion to exclude “undesirable” families from the program--targeting “children of never-married mothers or African-American mothers” (Blank & Blum, 1997, p. 30). ADC often supplemented the state mothers’ pension programs (Blank & Blum, 1997) which themselves excluded those--such as unmarried mothers--who had “demonstrated immorality” (Stetson, 1997, p. 340). The net result, therefore, was that most ADC recipients were children of White widows (Stetson, 1997).

Early criticism about ADC focused on the program’s possible encouragement of unwed motherhood. These concerns were initially allayed by the “perception of ADC [as] a program for families headed by widows” (Blank & Blum, 1997, p. 30). The concerns, however, were again raised as aid for widows shifted from the ADC program to the newly-created Survivors Benefits under the Social Security program--a shift that left the “less deserving” single mothers on ADC rolls (Blank & Blum, 1997, p. 30). During the 1940s, the increase in divorce, separation, and never-married mothers compounded concern over funding the less deserving and prompted 19 states to exclude children from the program based on birth status (e.g., children born to unwed mothers) (Blank & Blum, 1997).

Despite the growing criticism over ADC, several 1950 amendments to the ADC legislation extended the program beyond assistance for children to include mothers as well as the children of fatherless homes (Stetson, 1997). With this expanded scope, the program--renamed Aid to Families with Dependent Children (AFDC)--now reached more Black and Hispanic women, as well as divorced and unmarried mothers (Stetson, 1997).

During the 1960s, changing public perceptions about “the merits of helping poor mothers stay home with their children” (Blank & Blum, 1997, p. 30) was fueled by the rising employment rates among women as well as the ever-increasing caseloads (increasing 44% between 1960-1965 and nearly doubling between 1965-1970 [Joseph, 1999]) and intensified public criticism over AFDC (Blank & Blum, 1997). It was during this period of rapid growth in enrollment that a series of U.S. Supreme Court decisions “established new legal protection for AFDC recipients” (Joseph, 1999, p. 3), holding “that welfare was an ‘entitlement’ that could not be denied without due process” in the landmark case *Goldberg v. Kelly* (1970) (Tanner, 2003, p. 29).

In response to public concerns about program discouragement of marriage and growing caseloads, Congress instituted a series of fundamental changes to the growing program. Among these changes were requirements that states attempt to establish paternity (with later amendments strengthening child support enforcement), the inclusion of unemployed male parents with a work history in 1962, and legislation that allowed state waivers of specific federal program requirements--waivers that would allow for pilot or demonstration projects by the states (Blank & Blum, 1997; Joseph, 1999; Stetson, 1997).

More significantly, the 1960s saw a shift away from support for mothers to stay home and a new focus on “economic self-sufficiency” with the 1967 implementation of the Work Incentive Program (WIN) (Blank & Blum, 1997; Larner et al., 1997).

Although participation in these programs was initially voluntary, participation was later mandatory for those welfare recipients with “no special responsibilities at home and no preschool children” (Blank & Blum, 1997, p. 31). The WIN program provided job

training to some welfare recipients, offering them work assignments (Larner et al., 1997) ostensibly to “foster a sense of dignity, self-worth and confidence” (Stetson, 1997, p. 344). Welfare-rights advocates argued that the WIN programs put “women only in low-paid dead-end jobs, with pay barely above the welfare level itself” (Stetson, 1997, p. 344). Funding for WIN was inadequate from the outset, and its goals were never fully realized (Blank & Blum, 1997).

The Omnibus Budget Reconciliation Act (OBRA) of 1981 reshaped WIN, defining work experience to include unpaid work in public agencies and allowing states to subsidize on-the-job training (Blank & Blum, 1997; Stetson, 1997). Studies of state welfare-to-work programs in 1986 indicated that “although the gains were not dramatic,” these programs could be cost-effective while increasing employment (Blank & Blum, 1997, p. 32). Further research, however, demonstrated that, while there was a small benefit in both employment rates and annual earnings, there was no significant reduction in welfare caseloads (Larner et al., 1997).

As the federal government increased the work requirements for welfare recipients between 1967 and 1986, some federal policymakers alternately “attempted to alleviate family poverty by supplementing income through less restrictive methods than the AFDC programs” (Blank & Blum, 1997, p. 32). During these years, initiatives such as the Earned Income Tax Credit (EITC), the negative income tax, child support assurance, and the Food Stamp Program were proposed (Blank & Blum, 1997). The resistance to these programs reflected the nation’s articulated focus on self-reliance, as these proposals were often struck down (Blank & Blum, 1997).

President Reagan's 1986 call for a "study of how the welfare system could be changed" ultimately led to the enactment of the Family Support Act of 1988 (Blank & Blum, 1997; Stetson, 1997). This legislation again focused on mandatory education and training services for welfare recipients, this time providing funds for necessary services such as child care (Blank & Blum, 1997). The Act, however, did not provide sufficient resources to cover all eligible individuals and was criticized for failing to adequately address "the nation's problems of poverty and dependency" (Blank & Blum, 1997, p. 34). Such criticism proved well placed as Congress yet again undertook the issue of Welfare Reform in 1995. In this debate, however, Welfare Reform would no longer mean adjusting the system; Welfare Reform would soon lead to a total repeal of AFDC.

A Transition from AFDC to TANF

As a federal "entitlement" program, AFDC guaranteed benefits to low-income families that met defined eligibility criteria without time limits (Larner et al., 1997). Eligible families included those families with children in which the "father was absent or unemployed" whose income was below a threshold defined by each state (Larner et al., 1997, p. 5). As a joint federal-state program, the federal government promulgated guidelines for the AFDC program, while the states actually determined eligibility and benefit levels (Larner et al., 1997). States were, therefore, free to set the income eligibility below the federal poverty level, and in 1992, "only 63% of children living in poor families received AFDC benefits" (Larner et al., 1997, p. 5). In addition to the joint federal-state effort in defining AFDC criteria, the program was also jointly funded by federal and state governments (Blumstein & Sloan, 2000).

From its inception, AFDC provided recipients with monthly cash assistance. Grant levels varied considerably from state to state: consider that while the median state grant was \$366 per month for a family of three in 1994, the range extended from \$120 in Mississippi to \$923 in Alaska (Page & Larner, 1997). Many recipients also received other benefits “such as Medicaid, child care assistance, food stamps, and subsidized housing through a variety of related programs” (Page & Larner, 1997, p. 22). Despite receiving the combined value of these programs, many families were often left below the federal poverty level (Larner et al., 1997).

Public concern over the rising number of never-married adult welfare recipients (48% in 1993) (Larner et al., 1997) and a public perception of growing welfare expenditures (despite the fact that the population served by AFDC and the proportion of federal budget both remained consistent at 5%) throughout the 1980s and 1990s were reflected in the program’s growing lack of popularity during these decades (Page & Larner, 1997). The federal government responded by increasingly emphasizing employment for welfare recipients, expanding the state’s role in work programs (Larner et al., 1997), and allowing the states to request waivers of federal program requirements from the Department of Health and Human Services (DHHS) under Section 1115 of the Social Security Act (Lurie, 2001; Page & Larner, 1997).

Section 1115 waivers exempted states from specific federal AFDC requirements permitting states to redesign their AFDC programs by adopting new policies related to “the financial and non-financial rules governing eligibility for benefits and the way earnings are treated in calculating benefits” (Lurie, 2001, p. 379). State waivers were instituted between 1992-1996 and encompassed six policy areas:

1. time limits (restricting the length of time for receipt of benefits),
2. work requirements (defining the onset of work requirements following AFDC enrollment),
3. young child work exemption (setting the age of the youngest child for which the parent is exempted from work requirements),
4. expanded earnings disregards (allowing recipients to earn more before benefits are reduced),
5. family cap (restricting or excluding benefits for additional children born to AFDC recipients), and
6. increased severity of sanctions (establishing penalties for noncompliance with work requirements) (Hofferth, Stanhope, & Harris, 2002).

With these waivers, the balance of shared federal-state authority over the AFDC program began to shift from the federal government to the states. This shift was nearly complete after the passage of the Personal Responsibility and Work Opportunity Reconciliation Act of 1996 (PRWORA).

CHAPTER III

WELFARE REFORM

Welfare Reform Legislation

Overview of PRWORA

On August 22, 1996, Congress passed the Personal Responsibility and Work Opportunity Reconciliation Act of 1996 (PRWORA) or Welfare Reform, “the biggest national policy change in our history affecting poor families with children” (Edelman, 1999, p. 1493). With this sweeping legislation, Congress abolished the Aid to Families with Dependent Children (AFDC) program that had guaranteed income support to poor families for over 60 years (Bane & Weissbourd, 1998; Cashin, 1999; Quigley, 1998; Wright, 1998), replacing it with the block-grant Temporary Assistance for Needy Families (TANF) program (PRWORA §§ 601-606, 1996). In addition, PRWORA repealed the federal entitlement to assistance (PRWORA § 601, 1996), set time limits for receipt of benefits (PRWORA § 608, 1996), and effectively shifted authority for welfare to the states, giving them “unprecedented discretion in choosing which families to assist, what services to provide, what requirements to impose, and how to respond to families who cannot find work to support their households within the allotted time” (Larner et al., 1997, p. 6). The hallmark of Welfare Reform, however, was the implementation of “workfare,” an explicit requirement that adult recipients--predominantly single mothers--work to receive benefits (PRWORA § 607, 1996).

PRWORA Goals

PRWORA's preamble declares an intent to address the "crisis in our Nation" brought on by rising out-of-wedlock childbearing (Lurie, 2001), attributing much of the "crisis" to the incentives encompassed in the AFDC welfare system (Orr, 2001). With PRWORA, Congress sought to "promote work over welfare and self-reliance over dependency" through job preparation, work, and marriage (Wright, 1998, p. 558).

Specifically, the legislation was to

- (1) provide assistance to needy families so that children may be cared for in their own homes,
- (2) end the dependence of needy parents on government benefits,
- (3) prevent and reduce the incidence of out-of-wedlock pregnancies . . . , and
- (4) encourage the formation and maintenance of two-parent families. (PRWORA § 601(a), 1996)

It should be noted that PRWORA's stated goals of promoting self-sufficiency, discouraging out-of-wedlock childbearing, and encouraging marriage did not address child poverty--though more than 9 million poor children (nearly 70% of AFDC recipients) relied on AFDC cash benefits in the lead-up to PRWORA (calculated from data at ACF, *Overview of the Child Care and Development Fund*).

PRWORA's Federal-State Governance

Although PRWORA establishes statutory goals, it gives states unprecedented discretion in developing their TANF programs to accomplish these goals. Under AFDC, federal laws and guidelines provided detailed minimum requirements governing state welfare programs with the option of states exceeding these minimum requirements--the shared governance scheme of "joint federalism." PRWORA, however, "sets only a few restrictions and mandates for the states, as well as several financial incentives and penalties" (Lurie, 2001, p. 380): PRWORA no longer sets forth the *minimum* services

required as was done for AFDC, but rather sets the *maximum* services allowable for TANF without a state exemption or use of state funds.

TANF Block Grant

In replacing AFDC with TANF, PRWORA replaced AFDC's matching federal-state funding scheme with TANF's federal block grants to states. Under AFDC's funding arrangement, the federal government matched state AFDC expenditures, "with poorer states' expenditures matched at a higher rate than wealthier states" (Weaver, 2002, p. 1). Each state's expenditures were unique, based on the size of the caseload and the benefit levels: as the number of AFDC recipients and/or benefit levels increased, state expenditures increased--and so too did the federal contribution. Under TANF's revised funding scheme, federal block grants are distributed to states as *fixed* annual sums--irrespective of state spending, changes in caseload, economic cycles, or inflation (Weaver, 2002). The federal block grant amount for each state is based on federal AFDC spending from 1992-1994 (Page & Larner, 1997; Wright, 1998), and was fixed at \$16.5 billion total annual spending for 1997-2002 (Weaver, 2002). In addition to establishing TANF's block grant funding scheme, PRWORA included a "maintenance of effort" provision, requiring states to maintain spending at 75% of their pre-PRWORA levels, or 80% of that level if a state's work participation targets are not met (Lurie, 2001; Weaver, 2002).

No Individual Entitlement

Although the U.S. Supreme Court held that welfare was an entitlement that could not be denied without due process in the 1970 landmark case, *Goldberg v. Kelly*, they deemed welfare "a matter of statutory entitlement for persons qualified to receive them"--

not a constitutional entitlement (1970, p. 262). Congress was, therefore, able to end the statutory entitlement under PRWORA by stating: “This part shall not be interpreted to entitle any individual or family to assistance under any State program funded under this part” (PRWORA § 601(b), 1996). In addition to ending the entitlement to welfare, PRWORA also dismantled several procedural safeguards including requirements that stated: “give anyone the opportunity to apply for aid, provide aid with reasonable promptness, and correct over- and under-payment promptly” (Lurie, 2001, p. 380). *“Earned Welfare”*

One seminal change under Welfare Reform was “the use of welfare laws to attempt to modify behavior” (L. A. Williams, 1992, p. 721). Under this “New Paternalism” or Earned Welfare (Sawhill, 1995), the federal government conditions eligibility and benefits on recipients modifying certain behaviors. Unlike AFDC’s entitlement to assistance, TANF eligibility is not guaranteed based on income levels set by the federal government (Wright, 1998). PRWORA instead explicitly mandates that participants engage in “work activities,” and gives states discretion to condition receipt of benefits on behaviors such as “ensur[ing] that minor dependent children attend school” (so-called “Learnfare”) (PRWORA § 604, 1996), immunizing minor children, and paying bills on time, with potential sanctions again being levied against the family (Sullivan, 1997; L. A. Williams, 1992). One of the more controversial behavior modification programs states now have the option of implementing is the “Family Cap” program, in which benefits are reduced or eliminated for “additional children conceived after a mother begins receiving [TANF]” (L. A. Williams, 1992, p. 736). Penalties for failure to

comply with any of these TANF requirements could include reduction of benefits or termination of assistance for the recipient--or the entire family (PRWORA § 607, 1996).

Work Requirements

TANF departed from AFDC with its explicit requirement that adult recipients work to receive benefits. PRWORA requires that TANF recipients engage in “allowable work activity” to receive benefits (PRWORA § 607, 1996). States have discretion in defining “allowable work activity,” in setting the required number of work hours, and in establishing categories of exemptions--if any--from work requirements (e.g., single parents with young children). Under PRWORA, states may penalize recipients who do not comply with work requirements, with great latitude in defining their sanction policies. State sanctions for work noncompliance vary considerably, ranging from a partial reduction in benefits to immediate--often permanent--case closure (Urban Institute, 2005a). PRWORA’s employment provisions are discussed more completely later.

Time Limits

Unlike the open-ended eligibility of AFDC, PRWORA sets a federally-mandated lifetime maximum of 5 years for receipt of TANF benefits by adults (PRWORA § 608, 1996). Although PRWORA sets this “60 months (whether or not consecutive)” maximum time limit, it permits states to set a shorter lifetime limit (PRWORA § 608, 1996). A number of states have chosen this option, imposing shorter time limits than the federally-defined 60 month maximum, including Connecticut at 21 months and seven other states at 24 months (Urban Institute, 2005a). While states may use state funds to continue TANF assistance after the lifetime limit has been reached, or may provide specific exemptions from the lifetime time limit, a state may exempt only up to 20% of

its caseload (PRWORA § 608, 1996). In addition to the 5-year lifetime maximum, PRWORA also sets a 2-year limit for the receipt of benefits for any one period of time (PRWORA § 608, 1996). Again, states have the option of setting a maximum limit of less than 24 months (PRWORA § 608, 1996).

Relationship of TANF to Other Federal Programs

The relationship between TANF and other federal benefit programs is often complex. The 30-year link between AFDC and Medicaid was severed under PRWORA (Page & Lerner, 1997). Historically, AFDC recipients were categorically eligible for--and automatically enrolled in--Medicaid; PRWORA, however, explicitly decoupled Medicaid from TANF. The intent behind this “decoupling” was to allow former welfare recipients to potentially receive Medicaid even if they were no longer eligible for TANF benefits. To accomplish this, PRWORA created a new category of Medicaid eligibility through Section 1931 (Ellwood, 1999). Under Section 1931, the so-called Medicaid savings clause, PRWORA mandated Medicaid eligibility for individuals whose incomes were below the 1996 AFDC income limit (Ellwood, 1999). In addition to “saving” Medicaid eligibility for this newly-defined category, Section 1931 also allowed states to expand their Medicaid eligibility through the use of income disregards and other mechanisms (Ellwood, 1999). In contrast to the attempt to preserve Medicaid eligibility through Section 1931, PRWORA paradoxically gave states the option of denying Medicaid coverage to non-pregnant adult TANF recipients who otherwise met Medicaid eligibility but failed to comply with TANF work--and other--requirements (Cherlin, Bogen, Quane, & Burton, 2002).

Medicaid coverage for children has remained somewhat protected under TANF: children remain eligible for Medicaid even if their parents' Medicaid coverage is terminated (Cherlin et al., 2002). In addition, the State Children's Health Insurance Program (SCHIP) was passed a year after enactment of PRWORA in an attempt to expand health insurance coverage to low-income children not eligible for Medicaid, discussed earlier. As with Medicaid, SCHIP is not linked to TANF. Despite these efforts to maintain and expand health insurance coverage among low-income children as well as the legislated expansions in Medicaid eligibility during the 1980s, approximately 9 million low-income children have remained uninsured in the U.S. (Kaiser, 2007).

Federal nutrition programs are also intertwined with TANF. The Food Stamp Program (FSP), renamed the Supplemental Nutrition Assistance Program (SNAP) in 2008, was cut under PRWORA: "The act reduced benefits, eliminated benefits for most groups of legal immigrants, and tightened work requirements for able-bodied adults without dependents" (USDA, Economic Research Service, 2007, p. 32). PRWORA gave states the option of withholding part--or all--of a family's Food Stamps benefit for failure to comply with work requirements. The slow decline in FSP caseloads during the early 1990s became a steep decline following PRWORA given the new restrictions on eligibility, administrative barriers, and other factors (J. A. Klerman & Danielson, 2009). It should be noted that, while PRWORA eliminated FSP benefits for most legal immigrants, the 2002 Farm Bill restored eligibility of major groups of legal immigrants. Like the restrictions and reductions PRWORA placed on the FSP, the Special Supplemental Food Program for Women, Infants, and Children (WIC) was also impacted by PRWORA: the legislation eliminated the requirement of outreach for the WIC

program, and denied eligibility to women in prison or juvenile detention (USDA, Food and Nutrition Service, 2001). In addition, PRWORA gave states the option to exclude immigrants from the WIC program, though no states have done so.

PRWORA Provisions that Affect Children

Welfare Reform affects women and children disproportionately, both because they represent a greater percent of the welfare rolls and because many of the provisions directly target them. PRWORA's stated goals of discouraging out-of-wedlock childbearing and encouraging marriage have resulted in provisions that directly affect women and children: family cap policies, restrictions on benefits for teen mothers, "bonuses" for states conducting abstinence-only education or demonstrate a "decrease in illegitimacy" or a reduction in teenage pregnancies, the exclusion of illegal (and some legal) immigrants from federal public benefits including nutritional services and Medicaid, strengthening of child support enforcement, and restriction of access to Supplemental Security Income (SSI). In addition, Welfare Reform's overarching goal of promoting self-sufficiency through mandatory employment also disproportionately affects women and children: More than 70% of TANF recipients are children and 90% of *adult* TANF recipients are women--predominantly single mothers (calculated from data available at ACF, *Overview*).

Family Cap Programs

PRWORA attempts to discourage childbearing among welfare recipients by giving states the authority to adopt a "child exclusion" or family cap policy--policies that exclude or reduce benefits for additional children born to TANF recipients (PRWORA, §

608, 1996). Twenty-one states have family cap policies in place, most having been implemented under Section 1115 waivers prior to PRWORA (Urban Institute, 2008). There have, however, been legal challenges to family cap policies. New Jersey's family cap program was challenged on equal protection and due process grounds in *C.K. v. Shalala* (1995). The state's policy that eliminated the standard increase in benefits for new children born to those receiving AFDC was upheld on grounds that the program did not "constrain the welfare mother's right to bear as many children as she chooses, but simply requires her to find a way to pay for her progeny's care" (*C.K. v. Shalala*, 1995, p. 1015).

In the early 1990s, Arkansas and New Jersey were the first states to impose a family cap under a Section 1115 welfare waiver (Cashin, 1999; Wise, Chavkin, & Romero, 1999). Arkansas reported no effect on the birth rate among AFDC recipients, largely due to nearly half of the "female case heads" no longer being fertile (due to sterilization or menopause) (Wise et al., 1999). In New Jersey, however, there was a reported decrease of 452 "babies conceived by New Jersey mothers already on welfare" over the first 3 months of the cap (as compared to the previous year's data) (Cashin, 1999, p. 363). This decrease in births among AFDC recipients in New Jersey was associated with an increase in abortions and the use of family planning services (Wise et al., 1999), although the decreased birth rate and increased abortion rate may have applied only to Black women and not to White or Hispanic women (Jagannathan & Camasso, 2003). The noted increase in abortions among women on AFDC stood in contrast to the decreased abortion rates and increased use of contraceptives and female sterilization among non-AFDC recipients during that same timeframe (Wise et al., 1999).

More recent research, however, suggests that the economic incentives and disincentives of PRWORA have not had a significant impact on women's reproductive behaviors (Kelly & Grant, 2007). Specifically, research demonstrates that family cap policies have neither reduced non-marital births (Dyer & Fairlie, 2004; Kearney, 2004) nor impacted abortion rates (Joyce, Kaestner, Korenman, & Hanshaw, 2004).

Teen Mothers

PRWORA's stated intent to address the "crisis in our Nation" brought on by rising out-of-wedlock childbearing (Lurie, 2001) led to a legislative focus on teen pregnancy. PRWORA's section addressing teen pregnancy delineated many of the adverse outcomes for mother and child associated with teen births, such as an increased risk of low birthweight babies, but inaccurately cited the "rising rates of nonmarital teen births . . . [that] will continue to climb" as the impetus for its focus: the rate of teen births had actually begun to decline in 1991 (Kelly & Grant, 2007, p. 879)--a full 12% decline between 1991-1996 (Kost & Munger, 1996)--largely due to increased contraceptive use (Boonstra 2002).

In accordance with its goal of reducing non-marital teen pregnancy, PRWORA prohibits states from providing TANF benefits to unmarried parents under 18 years of age who have not completed high school or its equivalent, unless they live at home (or in another adult-supervised setting) and attend high school (or an alternative educational program directed at obtaining a high school diploma or its equivalent) as soon as the youngest child is 3-months-old (PRWORA §§ 607 & 608, 1996). In short, for a minor parent (under 18 years of age and not married) to receive TANF benefits, she (or he)

must live with a parent or guardian--who is working--or in a state-approved setting and must attend school (Kalil & Danziger, 2000; Urban Institute, 2008).

Despite PRWORA's stated goals, research suggests that "economic-based incentives have only a weak, and inconsistent, impact on the reproductive behaviors of women [with] similar patterns for teenagers and for adult women" and that PRWORA has seemingly not impacted teen pregnancy rates (Kelly & Grant, 2007, p. 897).

Although most studies have indeed found that PRWORA has not significantly impacted teen pregnancy rates, one study found a greater decline in teen pregnancy rates among younger teens as compared to older teens following implementation of PRWORA (Lopoo & DeLeire, 2006). Another study, however, has actually found that PRWORA's minor parent provisions were "associated with a 10% *increase* in nonmarital childbearing among teens," with the researchers theorizing that "for some teens, receiving their parents' support in caring for their child may add a measure of security" (Boonstra, 2000, p. 9). It should be noted that these studies evaluated the effect of PRWORA on teen pregnancy independent of the overall teen pregnancy rates which steadily declined between 1991-2005, but increased significantly among 15-19 year-olds between 2005-2006 (CDC, 2009).

In addition to the co-residence and educational requirements imposed on teen parents, PRWORA also gives states the option of denying TANF assistance to teen parents altogether (PRWORA §§ 607 & 608, 1996). At present, nine states disallow minor parents as head of a TANF "assistance unit" (that is, the minor parent cannot receive TANF benefits in his or her own name), and at least one state prohibits cash

assistance for the children born to teen parents unless the teen is married (Urban Institute, 2008).

Concerns regarding the co-residence requirement were raised during the passage of PRWORA, given the potential for some teen mothers to be forced to live in abusive homes in order to receive benefits (Cashin, 1999). Subsequent research has found that “[t]een coresidence with their mothers does not appear to buffer against the experience of child care problems, depressive symptoms, or domestic violence” (Kalil & Danziger, 2000, p. 775). Additionally, this research has found that, while compliance with requirements and satisfaction with living arrangements appears to be high among teen mothers receiving TANF, there has been an adverse affect on various aspects of psychological well-being as well as on life stress among these mothers (Kalil & Danziger, 2000).

Abstinence-Only Education and Bonuses for Decreased Teen Pregnancy

“In a contradictory fashion, [PRWORA] seeks simultaneously to reduce nonmarital births (especially among teenagers) without increasing abortions, while restricting at least some women’s access to contraception” (Kelly & Grant, 2007, p. 879). PRWORA provided \$50 million each year for “states to provide abstinence-only education programs” between 1998 and 2002 (Institute for Women’s Policy Research [IWPR], 1997b). The funds, provided to all 50 states, were to be used to provide programs that “promote abstinence and not contraception” (IWPR, 1997b). PRWORA required that abstinence programs have, as their “exclusive purpose, teaching the social, psychological, and health gains to be realized by abstaining from sexual activity,” and must include teaching

that a mutually faithful monogamous relationship in the context of marriage is the expected standard of human sexual activity; . . .that sexual activity outside of the context of marriage is likely to have harmful psychological and physical effects; . . . [and] that bearing children out-of-wedlock is likely to have harmful consequences for the child, the child's parents, and society. (PRWORA § 701, 1996)

The intended audience for the abstinence-only programs is teenagers, despite the declining teen pregnancy rates preceding PWRORA's passage (Kost & Munger, 1996). While the attempt to decrease teen pregnancy may be well-placed, abstinence-only education has not been shown to be an effective vehicle in accomplishing this goal. Research has consistently found that abstinence-only education programs do not delay initiation of sexual intercourse among teenagers (Kanto, Santelli, Teitler, & Balmer, 2008) or impact their rates of unprotected sex (Trenholm et al., 2008), while most comprehensive sexual education programs--teaching about abstinence, contraception, and disease-prevention--are associated with a delay in the initiation of sexual activity as well as increased condom and contraceptive use (Kirby, 2008). PRWORA's focus on abstinence-only education has resulted in a documented decline in knowledge of birth control methods among teenagers, from 81% in 1995 to 66% in 2002 among males, and from 87% to 70% females (Lindberg, Santelli, & Singh, 2006). This lack of knowledge among teenagers of birth control methods, and failure to decrease sexual activity among teenagers, appears to stem from abstinence-only programs' lack of exposure to contraceptive information as well as the documented misinformation disseminated within the programs. A 2004 Congressional Report found that over two-thirds of the abstinence-only education programs that were evaluated provided inaccurate information regarding contraceptives, abortion, and basic scientific information such as the route of HIV transmission (U.S. House of Representatives, 2004).

Exclusion of Immigrants from Federal Benefits

PRWORA represented a change in “policy toward immigrants” when it “drew a distinction between the eligibility of citizens and non-citizens” (Haider, Schoener, Bao, & Danielson, 2004). While the legislation placed new requirements and restrictions on receipt of TANF benefits for citizens (i.e., work requirements, time limits), it severely restricted eligibility for noncitizens (i.e., immigrants) for receipt of “federal public benefits” including: SSI, Food Stamps, non-emergency Medicaid, family planning services under Title X of the Public Health Service Act of 1970, as well as Women, Infants and Children (WIC) nutritional services (PRWORA § 401, 1996).

The PRWORA legislation distinguished between “unqualified” immigrants (illegal or unauthorized immigrants) and “qualified” immigrants (legal immigrants) (PRWORA, §§ 401 & 402, 1996), and further distinguished between “pre-enactment” qualified immigrants (legal immigrants residing in the U.S. prior to August 22, 1996) and “postenactment” qualified immigrants (legal immigrants arriving after that date) (Haider et al., 2004). Under this legislation, unqualified immigrants are barred from receiving TANF, Medicaid, or any other “federal public benefits” (PRWORA, § 402, 1996). This ban on benefits for illegal immigrants extends to state benefits programs as well.

PRWORA precludes states from distributing state-financed cash benefits to illegal immigrants, “unless the state passes legislation explicitly authorizing the distribution” (Cashin, 1999, p. 361). PRWORA also barred states from providing welfare benefits or Food Stamps to postenactment qualified immigrants (i.e., those who arrived in the U.S. legally after August 22, 1996) during their first 5 years in country (PRWORA, § 403, 1996). Under PRWORA, states also have the option of denying federal public benefits to

qualified immigrants *after* the 5-year period, and may also deny benefits to pre-enactment qualified immigrants (those residing in the country legally prior to August 22, 1996) (PRWORA, § 401, 1996).

Under Welfare Reform, therefore, states must deny federal public benefits for all unqualified immigrants, must deny most benefits to postenactment qualified immigrants for their first 5 years in country, may choose to deny benefits to postenactment qualified immigrants after the 5-year period, and may choose to deny benefits to pre-enactment qualified immigrants. Despite the significant restrictions on immigrant eligibility imposed by PRWORA, “nearly all states chose to cover immigrants under federally funded programs” when given the option (Haider et al., 2004, p. 748). Fewer states chose to provide state-funded programs for those immigrants deemed ineligible for federal benefits (i.e., postenactment qualified immigrants) (Haider et al., 2004).

Subsequent federal legislation restored eligibility to some categories of immigrants. The Balanced Budget Act of 1997 (BBA) restored the eligibility for all “pre-enactment needy and disabled immigrants” and “mandated the continuation of SSI and Medicaid to all legal immigrants who were receiving SSI on August 22, 1996” (Haider et al., 2004, p. 749). The Agriculture Research, Extension, and Education Reform Act of 1998 (AREERA) restored Food Stamp eligibility to pre-enactment immigrants who were minors (18 years or under) or elderly (65 years or older) on August 22, 1996, or who were disabled (Haider et al., 2004). The most recent piece of legislation, the Farm Security and Rural Investment Act of 2002 (“Farm Bill”), restored eligibility to legal (i.e., qualified) immigrants who were disabled regardless of date of entry to the U.S., who had been in the U.S. for at least 5 years, or were minors (18 years

of age and under) regardless of date of entry to the U.S. (Capps, Koralek, Lotspeich, Fix, Holcomb, & Reardon-Anderson, 2004).

Stronger Child Support Enforcement

PRWORA strengthens child support enforcement by requiring states to create a central case registry for all child support orders and mandates other changes in state laws and procedures surrounding child support enforcement (Legler, 1996). These mandates include establishing procedures for wage withholding orders, adoption of the Uniform Interstate Family Support Act (UIFSA) to allow state agencies to “send income withholding orders directly across state lines to employers,” expansion of access to information from state and local government agencies as well as private entities, and the automation of databases for the accessed information (Legler, 1996, p. 541). The cornerstone of PRWORA’s child support enforcement, however, is the paternity establishment process. Under PRWORA, states must establish procedures that compel alleged fathers in a contested paternity proceeding to submit to genetic testing without an order from a judicial or administrative tribunal (Legler, 1996). Mothers receiving TANF benefits must cooperate with paternity establishment procedures under threat of reduction in or elimination of benefits (Legler, 1996).

Initial evaluation shows some gains in child support enforcement following implementation of PRWORA. Child support receipt increased among low- and middle-income children of never-married mothers as a result of paternity establishment requirements as well as new hire directories--state-maintained directories of newly hired employees which are matched against child support orders and followed by wage-withholding orders where applicable (Urban Institute, 2002). It should be noted that

research has found that the increased child support enforcement efforts preceding PRWORA were associated with increased welfare exits and decreased re-entries among young mothers (Huang, Kunz, & Garfinkel, 2002) as well as decreased nonmarital childbearing, particularly among younger, never-married, Black and Hispanic women (Plotnick, Garfinkel, McLanahan, & Ku, 2006).

Restricted Access to Supplemental Security Income (SSI)

Prior to the enactment of PRWORA, children could qualify in one of two ways for Supplemental Security Income (SSI), the primary federal-state program for support of disabled persons (Keepnews, 1998). SSI eligibility was granted either through having a disability on an established list of conditions, or by having an “individual functional assessment” conclude that “a child, even though his or her condition was not on the government list, was sufficiently impaired to qualify for SSI” (Keepnews, 1998, p. 55).

The new law eliminated the use of individual assessments (Keepnews, 1998) and limited the definition of disability (Quigley, 1998). By creating a more limited definition of disability, PRWORA made it more difficult for disabled children to qualify for, and receive, benefits (Quigley, 1998). Although subsequent amendments sought to lessen PRWORA’s restriction on SSI eligibility (Keepnews, 1998), nearly 100,000 children had lost SSI benefits by 1998 (Bass & Mosley, 2001) with an estimated overall 22% reduction in childhood SSI cases by 2001 (Schmidt, 2004).

Maternal Employment

PRWORA explicitly requires that TANF recipients engage in “allowable work activity” to receive benefits (PRWORA § 607, 1996). States have discretion in defining allowable work activity, in setting the timing and required number of work hours, and in

establishing categories of exemptions--if any--from work requirements (e.g., single parents with young children). In addition, states may penalize recipients who do not comply with work requirements, with great latitude in defining their sanction policies. With 90% of *adult* TANF recipients being women--predominantly single mothers--PRWORA generally means either maternal employment or state-imposed sanctions.

Activities allowed under the first 20 hours of allowable work activity include: unsubsidized employment, subsidized private sector or public sector employment, on-the-job training, community services programs, child care services, and vocational training, among others (PRWORA § 607, 1996). Those activities qualifying after the first 20 hours include: job skills training directly related to employment, education directly related to employment, attendance at secondary school leading to a GED, and others (PRWORA § 607, 1996). Unlike AFDC, TANF does not consider postsecondary education to be “work” for the purposes of the statute, allowing states to choose whether or not to define it as allowable work activity, and, if so, the length of time it may be considered a work activity. In 1998, 25 states had disallowed postsecondary education as work, with 15 states limiting college time to 2 years or less (Cox & Spriggs, 2002). Within 2 years of PRWORA’s passage, there was a 20% decline nationally in college enrollment among welfare recipients as compared to poor women who were not TANF recipients (Cox & Spriggs, 2002). One study has estimated that state policies accounted for 13% of this overall decline, with TANF employment provisions accounting for the remainder of the decline (Cox & Spriggs, 2002).

One of the obvious consequences of decreasing postsecondary education rates among welfare recipients, particularly women, is their subsequent lack of access to both

higher earnings and greater employment rates. In 2000, the median income for women with a bachelor's degree was more than triple that of women with no high school diploma, and double that of women with a high school diploma or GED (Cox & Spriggs, 2002). In addition, those with college degrees had higher employment rates, and more stable employment histories (Cox & Spriggs, 2002). Although some have suggested that "work-first" policies lead to higher wages due to greater work experience, evidence suggests that a college degree is worth \$3.65 per hour in wages for working mothers (in 1997 dollars, relative to high school graduate wages), while 1 year of work experience is worth only \$0.10 per hour (IWPR, 1998).

PRWORA requires that recipients begin work within 24 months of TANF enrollment, but, as with other provisions, gives states the option to set shorter timeframes (PRWORA § 607, 1996). Only five states set the 24-month time period, with 31 states requiring work immediately after enrollment, and the remaining states with time periods up to 6 months after enrollment (Urban Institute, 1999). PRWORA also set the minimum number of work hours required. Although the 24-month timing requirement was a set parameter, PRWORA increased the required number of work hours over time: 20 hours per week in 1997, increasing to 30 hours per week in 2000 for one-parent families and 35 hours per week for two-parent families (PRWORA § 607, 1996). Just over half of the states set their required hours policies as defined by PRWORA, but 7 states set higher requirements and 14 set lower requirements or allowed for individual determinations (Urban Institute, 1999). PRWORA does, however, establish a separate requirement for single parents with a child under 6 years of age at 20 hours per week (PRWORA § 607,

1996). Only 14 states have adopted this lower requirement for parents of young children (Urban Institute, 1999).

Like other employment provisions, categories of exemptions from work requirements are largely at the discretion of the state. While AFDC had previously exempted mothers of children under 3 years of age from work requirements, PRWORA gives states the option of exempting parents of infants under 1 year of age, but provides no such guarantee (PRWORA § 607, 1996). Twenty-five states provide work exemptions to those caring for infants up to the age of 12 months and four states extend the exemption beyond 12 months (Urban Institute, 2005a). Twelve states, however, limit the exemption to those caring for infants under 3 months of age, four states between 4 and 6 months of age, and six states provide no exemption for those caring for children (i.e., mothers are required to return to work immediately after delivery or adoption in order to continue TANF benefits) (Urban Institute, 2005a).

PRWORA allows states to penalize recipients who do not comply with the work requirement, giving states the option to reduce benefits or even “terminate such assistance” (PRWORA § 607, 1996). States have subsequently adopted sanction policies that range from a written warning to a temporary reduction in TANF benefits to a permanent termination of all TANF benefits to the entire family (Urban Institute, 2005a). Although PRWORA prohibits states from sanctioning single parents of preschool children who cannot find child care, it does allow states to count this time toward the benefit time limits (PRWORA § 607, 1996). States may, however, choose to exempt single parents from the work requirement altogether, defining the children’s ages and other conditions, but may not exempt more than 20% of their caseloads (Wright, 1998).

Child Care

“Workfare means day care: Child care is the essential ingredient in welfare reform and mothers’ employment” (Scarr, 1998, p. 100). Under PRWORA, three child care programs previously-established under the Social Security Act (AFDC/JOBS Child Care, Transitional Child Care, and At-Risk Child Care) were consolidated under the Child Care and Development Block Grant (CCDBG) (ACF, *Laws & Policies*; Joseph, 1999) through which states receive discretionary, mandatory, and matching funds (General Accounting Office [GAO], 2001). States may transfer up to 30% of their federal TANF funds to the CCDBG, but must maintain their own state funding at a minimum of 75% of “historic” state spending (Joseph, 1999)--the state’s maintenance of effort--in order to receive federal matching funds (GAO, 2001). The CCDBG and subsequent child care subsidy funding under the Social Security Act were combined and re-named the Child Care and Development Fund (CCDF) (ACF, *Laws & Policies*).

Through CCDF, child care subsidies are available to parents receiving TANF who either work or participate in educational activities, to parents who have recently left welfare rolls, and to working parents whose income puts them at risk of needing TANF, and may also be used for children in need of protective services (GAO, 2001). States must, however, spend at least 4% of their CCDF funds to improve the quality and availability of care and offer additional related services to parents (GAO, 2001). Within these broad guidelines,

states have discretion in deciding how these funds will support child care, including who will be eligible, the payment mechanism to be used to pay providers, and the portion of TANF funds to be used for child care versus other eligible support services. (GAO, 2001, p. 7)

Under CCDF's child care programs, states may determine income eligibility and family prioritization criteria. Federal guidelines allow states to provide child care subsidies to families with incomes up to 85% of the state median income, but give states the option to set a lower threshold (GAO, 2001). States also have discretion in defining prioritization criteria, generally prioritizing on either TANF status or income level (GAO, 2001). Most states have chosen to prioritize families based on TANF or TANF-transition status rather than on income level (GAO, 2001).

In addition to defining eligibility criteria, states also determine the amount of their subsidies by setting both the payment rates they provide to child care providers as well as the co-payment required of subsidized families (Abt Associates, 2000). States also choose their payment mechanism for child care subsidies, with the option of subsidizing either through vouchers provided to families--certificates ensuring state payment of a portion of the child care fee that can be used with any child care provider--or through contracts directly with child care providers to provide care for subsidized families (GAO, 2001). Most states have chosen to subsidize child care through vouchers rather than contracts, with most families choosing center-based care over group home or family child care (GAO, 2001).

Overall state child care spending has increased in the wake of CCDF, with significant increases in state spending per federally eligible child (Abt Associates, 2000). Despite these spending increases, however, "only a fraction of the children potentially eligible [for child care] under federal law" are served by state child care programs (Abt Associates, 2000, p. 18) with many states experiencing waiting lists (GAO, 2001).

Maternal Employment and Children

The Impact of Maternal Employment on Children

Maternal employment cannot be categorically defined as either positive or negative for family and children. In fact, maternal employment appears to affect children in conflicting, and at times mutually tempering, ways (Parcel & Menaghan, 1997). While maternal employment can have positive effects for children through its increase in family income and access to material resources (Harvey, 1999), job-related factors such as stress and limited flexibility can adversely affect the quality of parenting offered (Parcel & Menaghan, 1997; Zaslow & Emig, 1997).

The impact of maternal employment on family and children appears to be a function of many factors: family income, wage levels, job-specific factors, mother's attitude toward work, demographics of mother and children, stage of development for the child, and the availability of quality child care (Bane & Weissbourd, 1998; Moore & Driscoll, 1997; Parcel & Menaghan, 1997; Youngblut et al., 2001; Zaslow & Emig, 1997). Although all of these factors influence the effect of maternal employment on children's well-being, the most significant factors impacting *young* children appear to be: the number of hours worked, the child's age and stage of development, and the quality of child care offered (Brooks-Gunn et al., 2002; Clark-Kauffman et al., 2003; Han et al., 2001; Shields & Behrman, 2002).

First, the more time a child spends in child care, the greater the chance of a negative impact--particularly when child care exceeds 30 hours per week (Brooks-Gunn et al., 2002; Han, 2005). Second, maternal employment, at least full-time employment,

generally has a deleterious effect during the first year of life (Hill et al., 2005; Ruhm, 2004), although this may apply more to White rather than Black or Hispanic children (Han et al., 2001; Waldfogel et al., 2002) and may not apply to low income children (Desai et al., 1989; Vandell & Ramanan, 1992). And third, low-income mothers are more likely to utilize lower quality informal child care arrangements than higher quality center-based care (Shields & Behrman, 2002).

Much of the available research on the impact of maternal employment on children centers on middle- to high-income families with two parents. Although some research isolates low-income families, very limited research is available on maternal employment among single mothers or when employment is not voluntary--that is, the characteristics of TANF recipients. The following, however, highlights research that is relevant to the TANF population: the impact of low-wage employment, employment among single mothers, and non-voluntary employment. It should be noted that, although several studies use child health outcomes, most research on the effect of maternal employment on children utilizes cognitive functioning and behavioral outcomes as indicators of child well-being. Also, because the context of maternal employment is quite complex, methodological challenges for research exist that have, at times, led to different conclusions regarding the impact of maternal employment on children--even when the same dataset is used. The different findings are presented where relevant.

Mothers working in low-wage, repetitive jobs tend to have lower quality home environments than do mothers working in well-paid, interesting jobs (Moore & Driscoll, 1997; Parcel & Menaghan, 1997; Zaslow & Emig, 1997). Specifically, a mother's attitude toward her work contributes to family life: mothers with a "discrepancy between

actual and desired employment” or who find their work unstimulating tend to exhibit parental role strain, while those who find their work gratifying and challenging demonstrate more positive parenting (Youngblut et al., 2001, p. 347; see also Moore & Driscoll, 1997; Parcel & Menaghan, 1997; Zaslow & Emig, 1997). In particular, job-related factors such as job stability, higher occupational complexity (offering self-direction and variety), and flexibility of hours tend to be supportive of positive parenting (Moore & Driscoll, 1997; Parcel & Menaghan, 1997; Youngblut et al., 2001; Zaslow & Emig, 1997). In addition to these job-related factors, stable employment patterns, rather than intermittent employment, have been associated with positive behavior and cognitive effects on children (Greenstein, 1993; Moorehouse, 1991), although not all studies have found that the stability of employment affects children at all (Desai et al., 1989).

The question of the relative impact of maternal employment on children in low-income families is unsettled. Some research suggests the negative effects of maternal employment are more pronounced in children from low-income families (Han et al., 2001), while other research has demonstrated positive effects of maternal employment among low-income families (Harvey, 1999; Vandell & Ramanan, 1992), and still other research has found no effect at all (Desai et al., 1989).

Research evaluating the impact of maternal employment on children in single-parent families has shown mixed results. Some research has demonstrated positive effects of maternal employment during the first one (Ruhm, 2000) to three years of life (Harvey, 1999) for children in single-parent families as compared to married families. Another study, however, found positive effects for Black--but not White--children of single-parent families (Milne, Myers, Rosenthal, & Ginsburg, 1986), while another found

no effect of maternal employment on children in single-families at all (Hall, Gurley, Sachs, & Kryscio, 1991).

As for the impact of nonvoluntary maternal employment on children, at least one study has found positive motor development outcomes associated with greater choice for mothers regarding whether to be employed (Youngblut, Loveland-Cherry, & Horan, 1993). Another study found no impact of nonvoluntary maternal employment on children, but did find the presence of signs of parental strain for “nonemployed mothers wishing to work” (Youngblut et al., 2001, p. 348).

In addition to the factors of wages, family structure, and choice in employment, it appears that the gender and race of children are also relevant in evaluating the impact of maternal employment. Several studies have demonstrated positive effects of maternal employment on daughters, while showing some negative effects on sons (Alessandri, 1992; Brooks-Gunn et al., 2002; Zaslow & Emig, 1997) particularly for maternal employment during the first year of life (Harvey, 1999), although this may apply only to children in higher income families (Desai et al., 1989) and has not been found universally (Waldfogel et al., 2002). Another line of research has explored the role of race in evaluating the impact of maternal employment on children. In addition to the study noted earlier that found a *positive* effect of maternal employment for Black--but not White--children in single-parent families (Milne et al., 1986), several studies have similarly found *negative* effects of maternal employment during the first year of life for White children, but not for Black or Hispanic children (Han et al., 2001; Waldfogel et al., 2002). Another study also found a negative effect of maternal employment among White but not Black children--but only for subsequent verbal/language arts outcomes; the *reverse* was

true for math skills--that is, the negative effect of maternal employment on subsequent math skills was greater among Black children than White children (Ruhm, 2004).

Two aspects of maternal employment that are particularly relevant to infants are the timing of a mother's return to work and the "intensity" of the mother's work or number of hours worked. Mothers' early return to work during the first year of life has been shown to have adverse health consequences for infants. First, mothers who return to work early are less likely to engage in infant health-promoting behavior. A recent study found that mothers' return to work within 12 weeks of delivery was associated with decreased rates of breastfeeding, immunizations, and well-child care follow-up (all health-promoting behaviors (AAP, 1997, 2007) for their infants (Berger et al., 2005). This study builds on the larger body of literature demonstrating lower initiation rates of breastfeeding (Hawkins, Griffiths, Dezateux, & Law, 2007) and higher cessation rates of breastfeeding among mothers who return to work during a child's first year (Lindberg, 1996; Roe, Whittington, Beck-Fein, & Teisl, 1999)--particularly during the first 12-weeks postpartum (Guendelman et al., 2009). Additionally, several studies of parental leave policies in European countries have found that extending job-protected *paid* leave after delivery reduces infant mortality (primarily during the postneonatal period, days 28-364 of life) as well as early childhood mortality--with an estimated 2.5-3.4% reduction in infant mortality for a 10-week extension of paid leave (Ruhm, 2000; Tanaka, 2005; Winegarden & Bracy, 1995).

In addition to the timing of mothers' return to work, the intensity of that work also appears to a significant factor for infants. Full-time maternal employment during the first year of life (Hill et al., 2005; Ruhm, 2004) and possibly the second year of life (Baydar &

Brooks-Gunn, 1991; Belsky & Eggebeen, 1991; Sherlock, Synnes, & Koehoorn, 2008; Youngblut et al., 2001; Zaslow & Emig, 1997) has generally been associated with adverse cognitive effects, although this may apply more to White rather than Black or Hispanic children (Han et al., 2001; Waldfogel et al., 2002) and to boys more than girls (Alessandri, 1992; Harvey, 1999), and may have no effect (Desai et al., 1989) or even a positive effect on children in low-income families (Vandell & Ramanan, 1992, finding positive effects on children's subsequent math performance, but not language arts).

Issues of Child Care

For low-income mothers, the presence of affordable, available, and quality child care services is directly linked to the likelihood of finding and keeping employment (IWPR, 1997a; Kisker & Ross, 1997) and to the ability of parents to manage the stresses of both work and family life (Scarr, 1998). Quality of child care is of particular concern for mothers, and can affect their decisions about jobs, hours worked, or even whether or not to work (Kisker & Ross, 1997). This concern over the quality of child care is borne out by the research: although not all research has found the quality of child care to be a determinant in the impact of child care on children (Blau, 1999), most research has found that higher quality child care is associated with positive socioemotional outcomes in children, with some studies finding enhanced language, reading, and math skills associated with higher quality care (Votruba-Drzal, Coley, & Chase-Lansdale, 2004). “Child care quality may be particularly salient for subgroups of children from low-income families,” such as children with low cognitive scores, those from less stimulating home environments, those in care for higher numbers of hours, and boys more than girls

(Vortuba-Drzal et al., 2004, p. 296). In other words, those children at greatest risk appear to be most impacted by the quality of child care (Vortuba-Drzal et al., 2004).

The trade-off of cost and quality in child care presents a major dilemma for families, particularly needy families (Scarr, 1998). Affordability of services, or “cost in relation to family income” (Scarr, 1998, p. 100), restricts the range of child care options available to low-income families “although they pay a higher percentage of their income for child care (23% versus 6% in high-income families)” (Scarr, 1998). Given the relatively high cost of center-based child care--generally associated with higher quality (Vortuba-Drzal et al., 2004), low-income mothers are more likely to seek out informal child care arrangements than are their higher income counterparts (Hofferth, 1999; Kisker & Ross, 1997). These informal arrangements are typically less reliable than center-based care, and can ultimately disrupt maternal employment (Kisker & Ross, 1997).

Evaluating Welfare Reform

An Overview of Welfare Reform Evaluation

With its implementation of Welfare Reform, “the United States embarked on a major social experiment with its social welfare and safety net programs for the poor” (NRC, 2001, p. 1). Given the far-reaching changes set forth in the PRWORA legislation as well as the statutorily-defined 2002 expiration of many its provisions, a great number of evaluations of Welfare Reform were undertaken by public, private, and academic organizations, projects, and individuals during the late 1990s and early 2000s. Researchers focused on various aspects of Welfare Reform, such as caseload levels, work

participation, family structure, child support enforcement, economic self-sufficiency, and child outcomes, for local, state, multi-cities or states, and national populations of TANF recipients, “leavers” (i.e., those who have recently left the TANF program), and special populations within these groups (e.g., children with disabilities, mothers with barriers to employment). These Welfare Reform evaluations have often proved challenging given the “evolving” nature of the 51 different TANF programs, the changing economic picture over time in the U.S., the complexities of TANF with its inter-connectedness with other means-tested programs that were themselves changing, and the often relative inadequacy of data (Blank, 2002; NRC, 2001).

In addition to this body of research on Welfare Reform, an overlapping field of research was also undertaken during this same timeframe focused on Welfare Demonstration Programs--welfare projects or programs undertaken by 43 states (or, rarely, counties or cities) through federal Section 1115 waivers prior to the implementation of PRWORA. These waivers allowed states to create programs intended to achieve overarching welfare goals without the need to adhere to the federally-established welfare program guidelines. Many aspects of the state waiver programs (e.g., mandatory work requirements, family caps, time limits, and sanctions) were later codified in PRWORA. Because of the similar goals and the statutory overlap of Welfare Demonstration Projects with PRWORA, evaluations of these projects are often incorporated into the Welfare Reform research literature.

Given the sheer volume Welfare Reform research, a targeted synopsis of Welfare Reform evaluation is offered. What follows is an overview statement of Welfare Reform

research findings related to overall program functioning, maternal employment, and children's health and well-being.

A Synopsis of Welfare Reform Evaluation Findings

Caseloads

Much of the evaluation of Welfare Reform has focused on its dramatic effect on the welfare rolls, or TANF caseloads. In the first 5 years following enactment of PRWORA, TANF enrollment declined sharply to *half* of its preimplementation levels (Urban Institute, 2006). After this rapid 5-year decline, caseloads continued to drop but more slowly, with a remarkable 67.7% decrease in the first 10 years of implementation (Calculated from data available from ACF, *Characteristics*). Declines in caseloads were noted in every state, and resulted from both decreases in families entering the program, and increases in families leaving the program (Blank, 2002; Golden, 2005). Given such significant decreases in caseloads, it is not surprising that the program participation rate (i.e., the proportion of eligible individuals enrolled in TANF) dropped as well, with only 48% of eligible families being enrolled in 2002 as compared to 80% in 1996 (Golden, 2005; Urban Institute, 2006).

Labor-Force Participation

“Participation in work and work-related activities by welfare recipients increased considerably” following implementation of PRWORA (Urban Institute, 2006). Labor force participation (LFP) among TANF recipients increased from 31% in 1997 to 44% in 1999, before decreasing to 39% in 2002 (Urban Institute, 2006). Many TANF recipients have multiple barriers to employment, such as limited education, mental or physical

disabilities, a child with a disability, or language difficulties (Golden, 2005). Despite the presence of such barriers, employment rates quadrupled among these recipients, increasing from 5% in 1997 to 20% in 1999, before dropping to 14% in 2002 (Golden, 2005). These patterns and the similar employment pattern among the larger population of single mothers with less than a high school education (a population heavily represented among TANF recipients), as well as the available research on employment among current and former TANF recipients, all suggest that LFP among TANF recipients is influenced by a combination of TANF work policy, the economy, and other work incentive programs, such as the Earned Income Tax Credit (EITC) (Blank, 2004; Fang & Keane, 2004).

Income/Poverty

With increased employment among TANF recipients, earned income among recipients rose “substantially” between 1997 and 2002, with an increase in the median family income from \$7,196 in 1997 to \$11,820 in 2002 (Urban Institute, 2006). Despite this increase in earned income, poverty remained high among recipients (with 69% living in poverty in 2002) as “increases in earnings appear [to have been] entirely offset by losses in public assistance income” (Blank, 2002, p. 1147; Urban Institute, 2006). Such “offsets” occur with TANF policies that decrease TANF benefits as earned income increases, leaving overall income unchanged. TANF’s mandatory employment programs alone, therefore, have little antipoverty effect, with some resulting in actual losses in overall income (Blank, 2002). Given this, many states instituted “earnings disregards,” financial incentives which do not decrease TANF benefits until a recipient’s earned income reaches a specific level set by the state (Blank, 2002). When employment

programs were combined with “generous” earnings disregards, employment and earned income increased and poverty rates decreased (Blank, 2002).

Health Insurance/Health Care

“An unintended consequence of welfare reform was to adversely impact the health insurance coverage of economically vulnerable women and children” (Cawley, Schroeder, & Simon, 2006, p. 502). Although Congress specifically de-linked Medicaid from TANF in order to preserve access to health insurance for those who leave welfare, Medicaid enrollment plummeted in the wake of Welfare Reform (Golden, 2005). The decrease in Medicaid enrollment has been attributed to confusion among would-be recipients over eligibility as well as to administrative barriers--that is, separate application processes for both TANF and Medicaid (Cawley et al., 2006; Golden, 2005). The drop in Medicaid enrollment does not appear to have been offset by increases in private insurance, despite the increase in employment (Bitler, Gelbach, & Hoynes, 2005). This decrease in health insurance coverage among low-income women and children was associated with decreases in health care utilization--including preventive care (check-up, breast exam, Pap smear) (Bitler et al., 2005) and prenatal care (Currie & Grogger, 2002; Kaestner & Lee, 2005). Health insurance coverage among children later increased following the implementation of the SCHIP, but with little to no impact on uninsured adults (Golden, 2005).

Child Well-Being

Welfare Reform’s impact on children is generally mediated through its effects on maternal employment and income. As with maternal employment in general, the impact of Welfare Reform on children varies by developmental stage (Clark-Kauffman et al.,

2003). Much of the research surrounding child well-being has focused on cognitive development, school achievement, and child behavior, because these indicators appear to be quite sensitive to income changes (Morris, Gennetian, & Duncan, 2005). Mandatory employment programs have been associated with small improvements in school-achievement and behavior among preschool and young school-aged children--but *only* when combined with generous earnings supplements (such as the earnings disregards discussed earlier) (Blank, 2002; Clark-Kauffman et al., 2003) and *only* as long as these supplements continue (Morris et al., 2005). This small positive impact appears to result more from an increased utilization of center-based child-care and after-school programs than from a change in the home environment (Blank, 2002; Morris et al., 2005).

In contrast to younger children, adolescents appear to have been adversely affected by Welfare Reform. Mandatory employment programs were associated with decreased school-achievement and increased behavior problems among adolescents (Blank, 2002), particularly among those with younger siblings, even in the presence of earnings supplements (Morris et al., 2005). In sum, therefore, “programs that increase both [the] parents’ employment and their income produce positive effects for preschool and early school-age children, even as they negatively affect adolescents” (Clark-Kaufman et al., 2003, pp. 299-300). Other research has focused more on the characteristics of employment, finding that while the intensity of maternal employment (number of hours worked) had little impact on the behavior of school-aged children, employment instability (transitions between working and not working) were associated with anxiety and depressed behavior among these children (Kalil & Dunifon, 2007).

But what of infants, for whom there are no such measures of achievement or behavior problems? Little research exists evaluating the impact of Welfare Reform on infants, specifically infant health. Haider et al. (2003) evaluated the effect of welfare work requirements on breastfeeding rates by clustering states into eight categories based on the stringency of three aspects of maternal employment: the timing of mothers' return to work, the number of hours required per week, and the sanctions policies. They found that "stringent" TANF work policies (requirements that mothers return to work within the first 6 months of having a child and work more than 18 hours per week under threat of full-family sanctions) were associated with decreased rates of breastfeeding (Haider et al., 2003). Despite the importance of this study, breastfeeding rates measure potential benefits to infant health rather than infant health itself. Currie and Grogger (2002) and Kaestner and Lee (2005) evaluated the impact of changes in welfare caseloads on prenatal care utilization. Both studies found that decreases in welfare caseloads were associated with decreases in prenatal care utilization, Currie and Grogger (2002) finding this relationship mediated through decreases in Medicaid enrollment. Kaestner and Lee (2005) also examined the impact of welfare caseloads on birthweight, finding a small increase in low birth weight associated with declines in welfare caseloads. Although these are very important findings given the association of early and adequate prenatal care utilization with positive birth outcomes and the strong correlation of birthweight with subsequent infant health and survival, both indicators are *predictors* of infant health rather than measures of infant health directly. It remains, therefore, to assess the impact of Welfare Reform's mandatory maternal employment on infant health.

CHAPTER IV

WELFARE REFORM AND INFANT HEALTH: THE IMPACT OF MANDATORY MATERNAL EMPLOYMENT

This research explores the relationship between Welfare Reform's mandatory maternal employment and infant health. The section begins by defining the conceptual framework informing the research followed by a discussion of the research methodology. The research findings are then presented with a subsequent discussion of the results.

Conceptual Framework

Maternal Characteristics and Infant Health

There is a well-established relationship between a variety of maternal characteristics and infant health:

1. *Underlying health/nutritional status*: higher levels of maternal health and nutritional status confer health benefits to infants;
2. *Age*: the youngest (< 20 years) and the oldest (> 40 years) maternal ages are associated with higher rates of adverse infant health outcomes, such as infant mortality;
3. *Race/ethnicity*: a significant and persistent Black-White racial disparity exists in infant mortality--the IMR for Black infants is nearly two-and-a-half times the rate for White infants; this disparity does not extend to Hispanic ethnic groups for whom the IMR approximates that of the White population;

4. *Educational attainment*: higher levels of maternal education confer health benefits to infants--maternal education of less than a high school education (< HS) is considered a risk factor for infant mortality, with the IMR being nearly twice as high for infants born to women with < HS than infants born to women with 16 years of education or more;

5. *SES/income*: there is a strong inverse relationship between SES and risk of disease--low SES is associated with a greater risk of both disease/health problems (higher frequency and more severe) and death; the IMR and each of its components--the neonatal mortality rate and the postneonatal rate--are all higher among poor infants than among non-poor infants; and

6. *Marital status*: unmarried maternal status is associated with higher rates of adverse infant health outcomes--the IMR for infants born to unmarried mothers is twice that infants born to married mothers.

These maternal characteristics directly affect infant health, but also indirectly influence infant health through:

1. *Prenatal health behaviors*: maternal smoking, inadequate prenatal care, poor nutritional intake/weight gain among mothers, and maternal alcohol/substance use have all been associated with lower birthweight and directly with adverse health outcomes among infants;

2. *Birthweight*: higher birthweight (approximately 6 ½-11 pounds) confers health benefits to infants; risk factors for low birthweight include maternal age under 15 or over 35, low SES, unmarried status, low maternal education, Black race, and inadequate or no prenatal care, as well as the prenatal health behaviors noted above; and

3. *Infant health-related behaviors*: breastfeeding, immunizations, and well-child care are all infant health-promoting behaviors related to maternal characteristics; rates of breastfeeding initiation and duration are lower among younger mothers, Black mothers, and mothers with lower education levels, are unmarried, received no or inadequate prenatal care, or are smokers (see Figure 1).

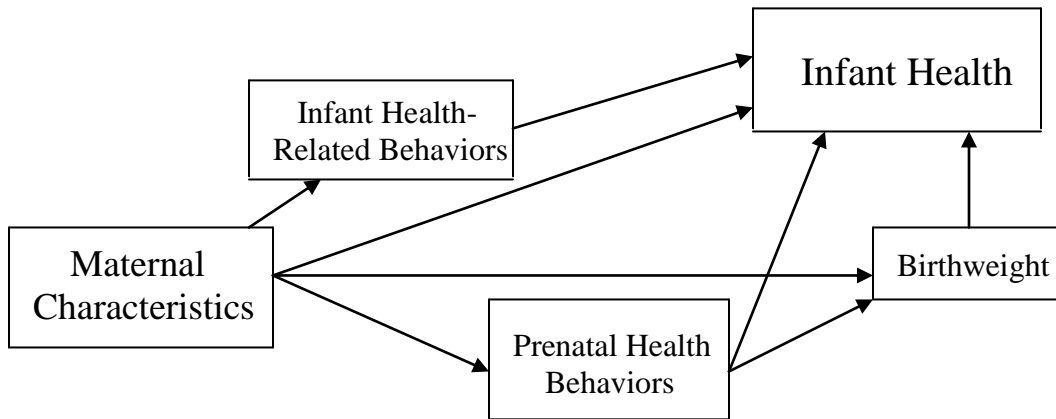


Figure 1. Maternal Characteristics Affect Infant Health

Welfare Reform and Mothers

Welfare Reform resulted in changes in maternal:

1. *Time endowment*: participation in work and work-related activities increased significantly among TANF recipients following the implementation of PRWORA, changing the amount--and perhaps quality--of family time;

2. *Economic resources*: as labor-force participation increased among TANF recipients, their earned income also increased; however, increases in earnings were largely offset by subsequent losses in public assistance income unless Earnings Disregards policies were in place;

3. *Child care utilization:* as work activity has increased among TANF recipients, so too has their utilization of child-care and after-school programs;
4. *Stress levels:* financial strain and employment instability, common among the TANF population, have been associated with maternal stress and mental health problems;
5. *Medicaid coverage:* Medicaid enrollment decreased among low-income women and children in the wake of PWRORA, with no apparent offset by increases in private insurance; and
6. *Health care utilization:* the decrease in health insurance following Welfare Reform was associated with decreases in health care utilization--including preventive care (check-up, breast exam, Pap smear) and prenatal care (see Figure 2).

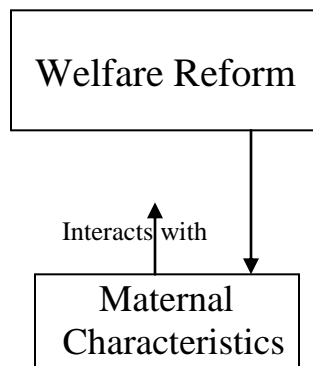


Figure 2. Interaction of Welfare Reform and Maternal Characteristics

Welfare Reform and Infants

Welfare Reform also impacted infants, as mediated through maternal factors, with changes in:

1. *Infant health-related behaviors*: “stringent” TANF work policies have been associated with decreased breastfeeding rates,

2. *Prenatal health behaviors*: decreases in welfare caseloads have been associated with decreases in prenatal care utilization, perhaps due to decreases in Medicaid, and

3. *Birthweight*: the declines in welfare caseloads were associated with a small increase in low birthweight (see Figure 3).

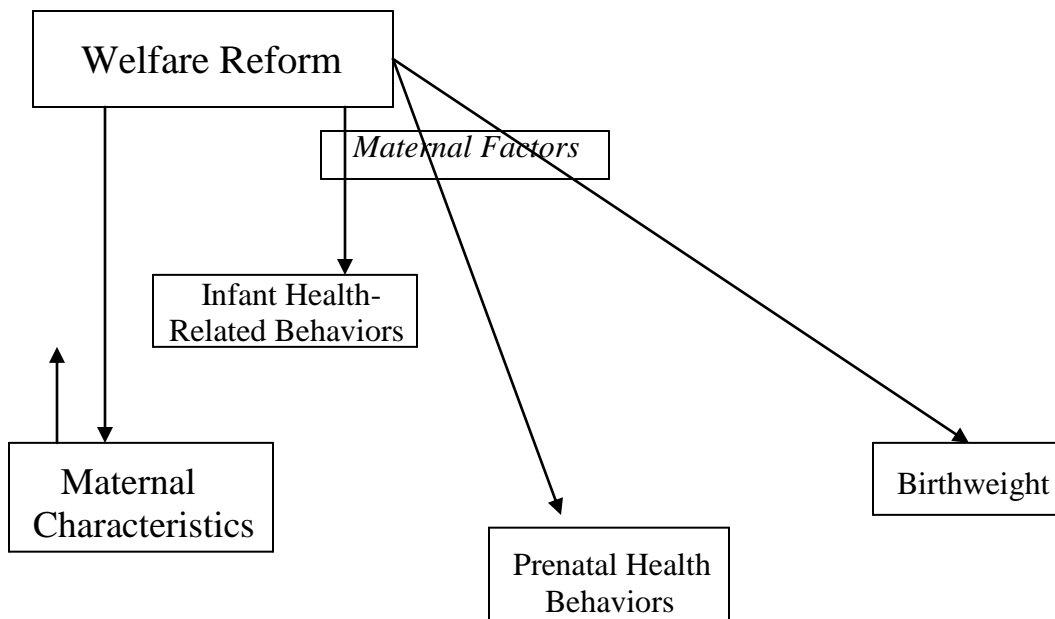


Figure 3. Welfare Reform Adversely Affects Infant Health-Related Pathways

Combining all of the components reveals a comprehensive conceptual framework (see Figure 4). The unknown relationship between Welfare Reform and infant health forms the research question for this project: *How have Welfare Reform’s mandatory maternal employment provisions impacted infant health?*

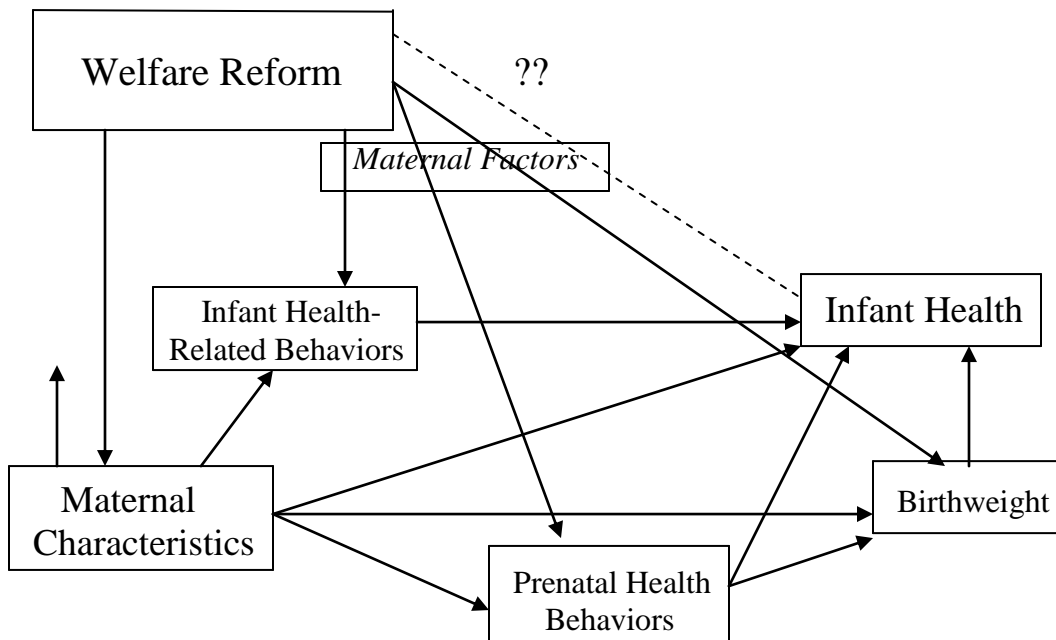


Figure 4. Conceptual Framework

Research Methodology

The goal of this research is to describe the relationship between Welfare Reform’s mandatory employment provisions and infant health by performing cross-sectional analysis of TANF work provisions and infant mortality while controlling for macroeconomic conditions, race, baseline infant mortality, and teen pregnancy for the 5 years following PRWORA’s implementation with the state as the unit of analysis using SPSS statistical analysis software linear regression modeling.

Unit of Analysis

With the discretion given to states by PRWORA to define their welfare programs, 51 different welfare programs (50 states plus the District of Columbia) now exist, each

with its own unique set of work requirements and penalties. The state is the unit of analysis for this research.

Independent Variables¹

Review of the literature surrounding the impact of maternal employment on infant well-being--both within and beyond the welfare context--consistently underscores the importance of the timing of a mother's return to work and the intensity, or number of hours, of her work. Specifically, early return to work (particularly within a child's first 3-4 months of life) and higher intensity of work (particularly exceeding 30 hours per week) are associated with adverse health and development outcomes for infants.

Research evaluating maternal employment in the TANF context suggests that sanctions for work noncompliance also play a role in the impact of maternal employment on children. Sanctioned families are more likely to experience material hardship (Kalil, Seefeldt, & Wang, 2002), which could adversely affect children--particularly infants. In addition, Lohman, Pittman, Coley, and Chase-Lansdale (2004) found that preschool children of sanctioned families had higher rates of "problematic developmental outcomes," although it is unclear whether this is causal given that TANF recipients with lower educational levels are more likely to be sanctioned (Kalil et al., 2002). Haider et al. (2003) evaluated the effect of TANF work requirements on breastfeeding rates by clustering states into categories based on the stringency of the timing of mothers' return to work, the number of hours required per week, and the Sanctions policies, and found that stringent TANF work policies were associated with decreased breastfeeding rates.

¹ State policies are drawn from the *Welfare Rules Database* at the Urban Institute.

1. *Work exemption.* The number of months mothers are exempted from TANF work requirements after delivery or adoption: 1 - (> 12 months), 2 - (12 months), 3 - (3-6 months), 4 - (no exemption);

2. *Hours of work.* The number of hours mothers of infants are required to work per week: 1 - (Case-by-Case), 2 - (< 25 hours), 3 - (25–29 hours), 4 - (30+ hours); and

3. *Sanctions.* The penalty or benefit reduction imposed for noncompliance with work requirements: 1 - (Lenient), 2 - (Intermediate), 3 - (Restrictive), 4 - (Stringent).

Categories are presented as increasing levels of stringency. Those for “Work Exemption” and “Hours of Work” were based on findings in the literature and review of the patterns of state policies. “Sanctions,” however, include wide variations across the states in the timing, intensity, and type of penalties applied to different programs (e.g., loss of entire TANF benefit for 1 month versus partial loss of both TANF benefit and food stamps for 3 months) and, therefore, required characterization.

An algorithm by which to categorize sanction policies was developed (Appendix A) based on: (a) what the penalty was (e.g., entire versus partial benefit, TANF versus TANF and Food Stamps); (b) who was impacted (e.g., adult alone versus entire family); (c) its duration (including whether penalty ceased once recipient began working); and, (d) when it was imposed (e.g., first versus subsequent offense). It is, in the end, challenging to compare such disparate sets of penalties. So, certain principles guided the development of the algorithm: (a) a greater loss of benefits is more stringent than a smaller loss, (b) penalties applied to children or the entire family are more stringent than those applied to adults only, (c) the immediate effect has higher priority than delayed or longer-term effects, (d) combining a reduction in TANF benefits with a loss of food

stamps is more stringent than a loss of TANF benefits alone, (e) penalties that apply for a specific time regardless of whether a recipient returns to work are more stringent than those that allow the recipient to “cure” the penalty, and (f) immediate and permanent loss of entire family benefits is the most severe penalty imposed. Once the algorithm was established and states were categorized into four categories, the results were compared with the sanction characterizations done by four other sets of researchers² to ensure a good fit.

Additional Variables

Two additional variables are included in some regression analyses. “Earnings Disregards,” or financial incentives which allow recipients to continue to receive their TANF benefits until their earned income reaches a state-set level, may influence the relationship between TANF’s maternal employment provisions and child well-being. Although Earnings Disregards are actually work incentives rather than work requirements, several studies have found that mandatory employment programs are associated with small improvements in school-achievement and behavior among preschool and young school-aged children--but *only* when combined with *generous* earnings supplements (Blank, 2002; Clark-Kauffman et al., 2003) and *only* as long as these supplements continue (Morris et al., 2005). This small positive impact appears to be a result of increased utilization of center-based child-care as well after-school programs rather than from a change in the home environment (Blank, 2002; Morris et al., 2005). With these findings, Earnings Disregards are also considered in the analysis to

² See Pavetti & Bloom (2001); General Accounting Office (GAO) (2000); Council of Economic Advisers (CEA) (1999); and Burke & Gish (1998), though all use a 3-category characterization.

evaluate its role, if any, in the relationship between TANF's mandatory maternal employment provisions.

States have defined their Earnings Disregards policies as one of five policy types: fixed deductions, percentage deductions, both fixed and percentage deductions, flat grant amounts, or, none. In order to categorize the earned disregards, the actual dollar amount of the Earnings Disregards were calculated using a 35-hour work week at \$6.35 per hours, or \$965 monthly earned income, based on the literature surrounding the wages and work hours of TANF recipients and low-income workers (see Hanson & Andrews, 2009; Loprest, 2001; and Lerman, Duke, & Valente, 1999).

1. *Earnings Disregards*. The amount of money a family can earn prior to TANF benefit reduction: 1 - (\geq \$550), 2 - (\$400 - \$549), 3 - (\$200 - \$399), 4 - (\$0 - \$199).

The second additional variable is "Sanctions Waivers." In the lead-up to PRWORA, states were able to individuate their welfare programs through Section 1115 waivers of welfare requirements. Many of these waivers were later codified in the PRWORA legislation: Sanctions was one of these policy areas. Eight states had Sanctions waivers in place in 1995, prior to the passage of PRWORA.

2. *Sanctions Waivers*. Whether a state had a sanction waiver in place in 1995: 0 - No, 1 - Yes.

Dependent Variables

Although a number of indicators of infant health and well-being exist,

infant mortality is one of the most important indicators of the health of a nation, as it is associated with a variety of factors such as maternal health, quality and access to medical care, socioeconomic conditions, and public health practices. (MacDorman & Mathews, 2008, p.1)

Long considered “the most sensitive index we possess of social welfare and sanitary administration” (Yankauer, 1990, p. 653, citing Newsholme, 1910), the Infant Mortality Rate (IMR) is the sum of neonatal mortality (deaths between 0 and 27 days) and postneonatal mortality (28 days to 364 days).

In the U.S., as in most industrialized countries, neonatal mortality, which is “related to maternal health problems prior to or during pregnancy or in the labor and delivery period,” accounts for two-thirds of overall infant mortality, while postneonatal mortality, more “related to the environments that infants experience after returning home,” accounts for one-third (L. V. Klerman, 1991, p. 138). The “2/3–1/3” neonatal-postneonatal mortality split observed in the general U.S. population does not, however, represent the infant mortality pattern of the lower SES population for whom infant mortality is more evenly distributed between the two periods with a roughly 55%-45% split (calculated from data available from the CDC, *CDC Wonder System*).

Although there are changes in health status short of mortality that are not captured in mortality rates, IMR has been shown to be sensitive to changes in SES and maternal employment (among other factors).

1. *Infant Mortality Rate (IMR)*. The number of deaths of infants under 1 year of age per 1000 live births, which is the sum of:

2. *Neonatal Mortality Rate (NMR)*. The number of deaths of infants (1-27 days) per 1000 live births, and

3. *Postneonatal Mortality Rate (PMR)*. The number of deaths of infants (28-364 days) per 1000 live births.

For this research, the mortality rates are limited to single births (approximately 98% of births in the U.S.) because of the very different risks and outcomes associated with multiple births.

Data

The CDC's National Center for Health Statistics (NCHS), in partnership with the states, collects data on births and deaths (among other data) in the U.S. through its National Vital Statistics System (NVSS). The NVSS includes the "Linked Birth and Infant Death Data Set" (Linked Birth), state-level data of infant births and deaths linked to a number of infant and maternal characteristics that are available online through the CDC (*CDC Wonder System*). Data presented by maternal age are available in 5-year clusters (e.g., 15-19 year-olds) with no option for disaggregation.

It should be noted that in 1999, in an effort to increase precision and reliability, the NCHS began to "suppress" infant mortality rates when there are "fewer than 20 in the numerator;" it continues to provide the raw data in these cases. For this research, when rates have been suppressed due to insufficient numbers, an average rate is calculated using the raw data for the target year and the subsequent year--an average to increase the reliability, and the subsequent year because of the overlap of pregnancies in the target year with births in the subsequent year.

Study Population

With no identifiable dataset linking infant health indicators--specifically infant mortality--to maternal welfare status, the TANF population must be approximated for this

research. The Linked Birth data include maternal age, education, marital status, and race/ethnicity.

In order to approximate the TANF population among the Linked Birth data, comparisons of the demographic data for the TANF adult population versus the U.S. general population are relevant:

1. *TANF Adult Population*³

- 90% are women,
- 48% have less than a high school education,
- 77% are “unmarried” (single, divorced, widowed), and
- 23% are married or separated.

2. *U.S. General Adult Population*⁴

- 20% have less than a high school education,
- 43% are “unmarried” (single, divorced, widowed), and
- 57% are married or separated.

Based on the TANF adult population demographics, unmarried women with less than a high school education best approximate TANF recipients given the limits of the Linked Birth dataset.

One study evaluating Welfare Reform and infant health (see Kaestner & Lee (2005) identified the study population as infants of unmarried women between the ages of 20-49 years with low education. While the low education and unmarried status do approximate the TANF population, it removes the infants of older teenagers (18-19 years

³ Calculated from data available from the ACF, *Characteristics* (1998-2002).

⁴ Calculated from data available from the ACF, *Characteristics* (1998-2002) and the U.S. Census Bureau, *Population Finder*.

old) who are subject to employment requirements for TANF eligibility, as well as a small minority of younger teens who could be subject to TANF work requirements (if emancipated minor status is achieved through marriage and/or educational attainment). When considering infants of welfare recipients, teenagers are a significant consideration: although teenagers (< 20 yrs) represent only 7-8% of the “adult” TANF population, these teenagers must, by definition, be a mother, who in all likelihood has a young child (given that she remains a teenager). Given this, infants of teens 18-19 years old should be included; the CDC Linked Birth dataset, however, clusters maternal age by 5-year periods (i.e., 15-19 year-olds together) with no option of disaggregating data for individual year of maternal age.

Select CDC natality documents provide data on birth rates by single year of maternal age, and include the same maternal characteristics included in the Linked Birth dataset--but do not combine marital status and educational status (i.e., data is provided based on marital status *or* education status, but *not* for a married status at an educational level). Based on analysis of the singleton birth data for each maternal characteristic (i.e., marital status, educational status), births to 15-17 year-olds account for up to 25% of the singleton births to unmarried women with less than a high school education, with births to 18-19 year-olds representing a near-identical proportion. While some 15-17 year-old TANF recipients could be subject to TANF work requirements, the proportion would be very small; so infants born to this age group should not be included in the study population. Without the option of disaggregating Linked Birth data based on individual year of maternal age, however, excluding 15-17 year-olds would also mean excluding 18-19 year-olds--up to 25% of the study population (calculated from data available in CDC,

National Vital Statistics Reports (2002b, 2007). So, the entire age cluster (15-19 year-olds) were included in the study population, with births to 15-17 year-olds being controlled for by using the state teen birth rate for this age group. Therefore, the study population for this research is defined as ***singleton infants born to unmarried women of all ages (15+ years-old) with less than a high school education.***

Controls

Four controls are included in this research/regression analysis.

1. *Macroeconomic conditions:* With the key role of macroeconomic conditions in Welfare Reform's mandatory employment focus, employment opportunities are controlled for using the ***state unemployment rate for the target year;***

2. *Race:* Given the well-established "Black-White" racial disparity in IMR, race is controlled for using the ***percentage of births to Black mothers in the study population for the target year;***

3. *Baseline infant mortality:* The baseline infant mortality is controlled for using the ***1995 state infant mortality indicator (IMR, NMR, PMR)***--the year prior to the passage of PRWORA;

4. *Teen births:* As indicated earlier, births to 15-17 year-olds are controlled for by using the ***state teen birth rate for 15-17 year-olds;***

Time Series

PRWORA was enacted in August 1996, with implementation occurring across the states at various points between September 20, 1996 and January 1, 1998. In order to

capture the relationship between maternal employment and infant health during the economic growth of the 1990s as well as the economic downturn following the year 2000, the time series encompasses the 5 years immediately following PRWORA’s implementation: 1998, 1999, 2000, 2001, and 2002. Analysis beyond 2002 is not possible: the “U.S. Standard Certificate of Birth” was revised in 2003 but was inconsistently adopted by states between 2003-2005. Maternal education and prenatal care data have been excluded for multiple states for each year between 2003-2005.

Study Design

Cross-sectional regression analyses was conducted for 1998, 1999, 2000, 2001, and 2002, utilizing SPSS statistical analysis software linear regression modeling (see Table 1).

Table 1

Cross-Sectional Regression Analyses

$$Y_{it} = \alpha + \beta_1 EXEMPT_{it} + \beta_2 HOURS_{it} + \beta_3 SANCTIONS_{it} + \beta_4 UNEMPLOY_{it} + \beta_5 RACE_{it} + \beta_6 BASELINE95_i + \beta_7 TEEN_{it}$$

Where:

- Y_{it} = infant health indicator (IMR, NMR, PMR) for singleton infants born to unmarried mothers (15+ years of age) with <HS in state i for year t
 - $\beta_1 EXEMPT_{it}$ = dummy variable for TANF work exemption policy for mothers of infants in state i for year t (12 months+ vs. < 12 months)
 - $\beta_2 HOURS_{it}$ = dummy variable for TANF policy on hours of work required for mothers of infants in state i for year t (< 25 hrs or “case-by-case” determinations vs. 25+ hrs)
 - $\beta_3 SANCTIONS_{it}$ = dummy variable for the characterization of the TANF sanction policies in state i for year t (Lenient–Intermediate vs. Restrictive–Stringent)
 - $\beta_4 UNEMPLOY_{it}$ = unemployment rate in state i for year t
 - $\beta_5 RACE_{it}$ = % births to Black mothers among singleton births to unmarried mothers (15+ years of age) with <HS in state i for year t
 - $\beta_6 BASELINE95_i$ = 1995 infant health indicator (IMR, NMR, PMR) for singleton infants born to unmarried mothers (15+ years of age) with <HS in state i
 - $\beta_7 TEEN_{it}$ = teen birth rate for 15-17 year-olds in state i for year t
-

Limitations

While this research offers an introductory overview of the previously unexplored relationship between Welfare Reform's mandatory maternal employment and infant health, several aspects of the research methodology and design limit the interpretation and generalizability of the research findings. First, the need to approximate the TANF population limits the interpretation of the findings. Next, there are a great many changes in infant health status short of infant mortality that are missed by this research (e.g., changes in health care utilization among infants would not be captured by infant mortality as a dependent variable). Also, it should be recognized that, as an ecological study--one in which a population rather than an individual is the unit of analysis--the inferences that can be made are limited. Population or aggregate data can mask variations within the population; assuming the population rate or average reflects the characteristics of individual members of the population has been termed the "ecological fallacy." The final limitation is related to the cross-sectional design. As a cross-sectional study, this research is descriptive and does not assign causality.

Findings

Descriptive Findings

TANF Recipients

Caseloads. TANF caseloads declined a remarkable 70% in the 10 years following PRWORA's 1996 passage, from a total of 13,749,927 recipients in 1995 to 4,103,529 in 2006. The proportion of recipients, however, remained constant across time

with children as 70% of recipients, women as 90% of adults, and men as 10% of adults (see Figure 5).

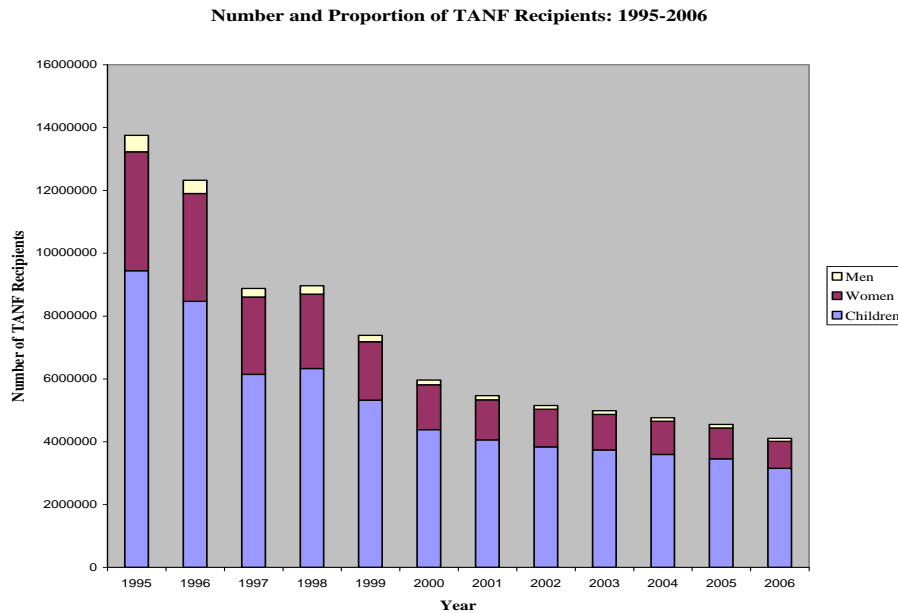


Figure 5. Number and Proportion of TANF Recipients: 1995-2006

*The proportion of Women to Men for 1997-1999 and 2006 are estimated from historical data.

Source: Calculated from data available at *Characteristics and Financial Circumstances of TANF Recipient*, by Administration for Children & Families, n.d., retrieved from <<http://www.acf.hhs.gov/programs/ofa/character/index.html>>

Employment. Employment rates among women receiving TANF increased 135% in the decade following PRWORA's enactment, with the percentage of employed women on TANF rising from 9.0% in 1995 to a high of 27.8% in 1999 before declining to 23.7% in 2006 (see Figure 6).

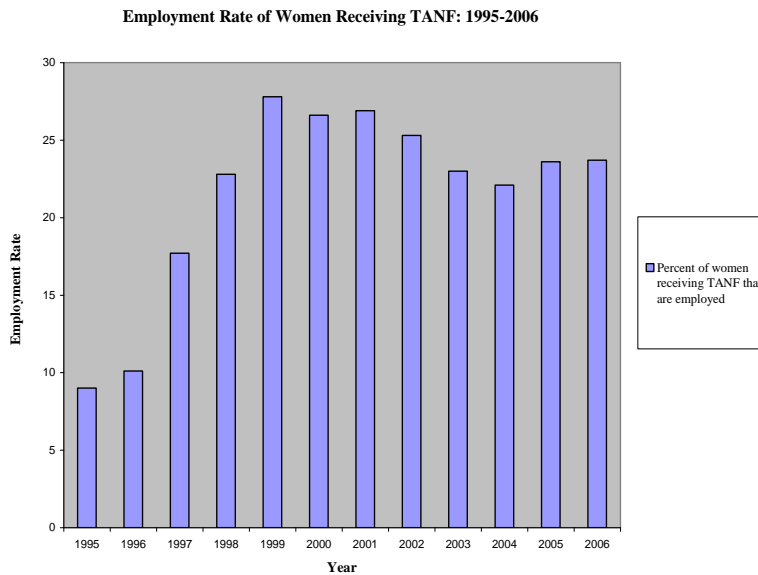


Figure 6. Employment Rate of Women Receiving TANF: 1995-2006

*The employment rates for 1997-1999 were estimated from the overall employment rate among TANF recipients and the employment rates among women in the U.S. available at: <www.aspe.hhs.gov/hsp/indicators06/ch3.pdf> and www.bls.gov/cps/wlf-databook2006.htm.

Source: Calculated from data available at *Characteristics and Financial Circumstances of TANF Recipient*, by Administration for Children & Families, n.d., retrieved from <<http://www.acf.hhs.gov/programs/ofa/character/index.html>>

Study Population

*In relation to overall U.S. births.*⁵ Singleton births to unmarried women (15+ years) with less than a high school education--the study population--averaged 487,831 per year between 1995-2002, representing an average of 12.73% of the roughly 3.83 million annual singleton U.S. births to women 15 years of age and older.

As a proportion of all U.S. births, the study population average was roughly equivalent to that of the unmarried HS group (12.86%), and accounted for a smaller proportion of U.S. births than married women in the three other maternal education groups: HS (19.28%), some college (16.27%), and 16+ years (21.65%). The study

⁵ All "U.S. births" discussed in this section are singleton births to women 15 years of age or older.

population represented a larger proportion of U.S. births than the married < HS group (8.92%), the unmarried group with some college (5.43%), and the smallest group, the unmarried group with 16+ years education (1.43%) (see Figure 7).

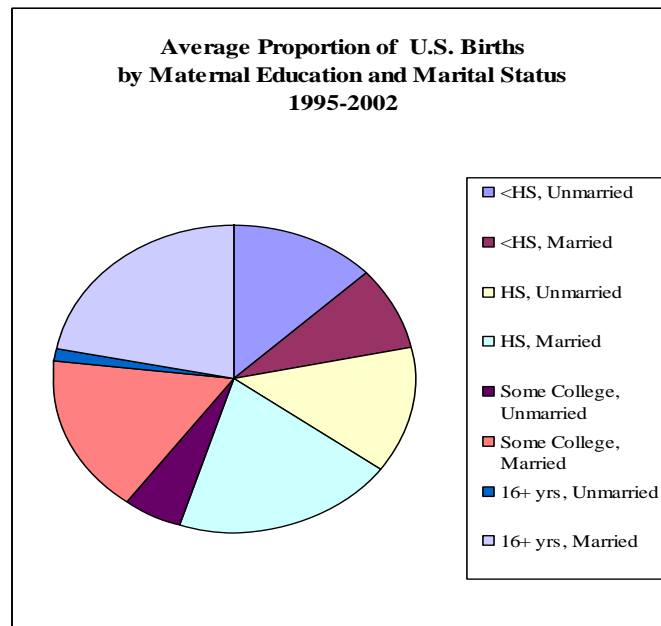


Figure 7. Average Proportion of U.S. Births by Maternal Education and Marital Status: 1995-2002

Source: Calculated from data available at *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The number of births in the study population (< HS, unmarried) increased 1.87% between 1995-2002, from 483,851 in 1995 to 492,877 in 2002; but given the overall 2.4% increase in U.S singleton births to women 15+ years of age, births in the study population accounted for a slightly smaller proportion of U.S. births in 2002 (12.67%) than in 1995 (12.74%). The relative stability of the study population as a proportion of U.S. births during the 1995-2002 time period is in part attributable to two competing

trends: a decrease in births to mothers with < HS education, and a rise in births to unmarried mothers.

Maternal education. U.S births grouped by maternal education are slightly more concentrated among women with a high school (12 years) education as compared to other educational groups: births to women with a high school education represent an average of 32.14% of U.S. births; those with less than a high school education 21.65%; some college (13-15 years) 21.61%; and, 16+ years of education 23.08% (see Figure 8).

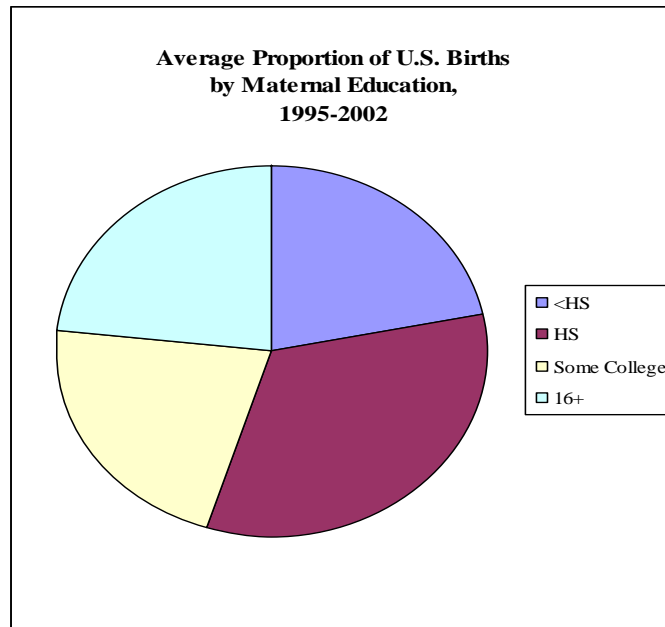


Figure 8. Average Proportion of U.S. Births by Maternal Education: 1995-2002

Source: Calculated from data available at *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The largest change in U.S. births between 1995-2002 among maternal education groups occurred in women with 16+ years of education with a 23.57% increase in the number of births, rising from comprising 20.86% of births in 1995 to 25.17% in 2002.

The number of U.S. births to women with a high school education decreased nearly 6% between 1995-2002, while those to women with less than a high school education decreased by 1.17%. There was very little change in the number of births to women with some college, with only a 0.15% increase (see Figure 9).

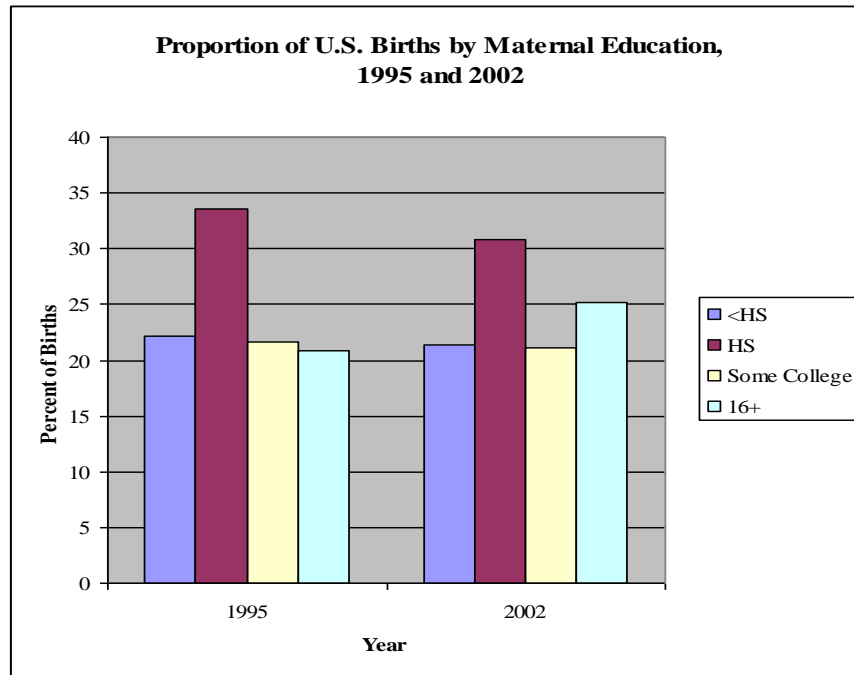


Figure 9. Proportion of U.S. Births by Maternal Education: 1995 and 2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The decrease in births to mothers with < HS education from 22.17% of U.S. births in 1995 to 21.37% in 2002 was followed by a more dramatic decline in the subsequent years. Between 2002 and 2005, the proportion of U.S. births to mothers with < HS education decreased by roughly one-third, from 21.37% in 2002 to only 14.24% of U.S. births in 2005 (see Figure 10).

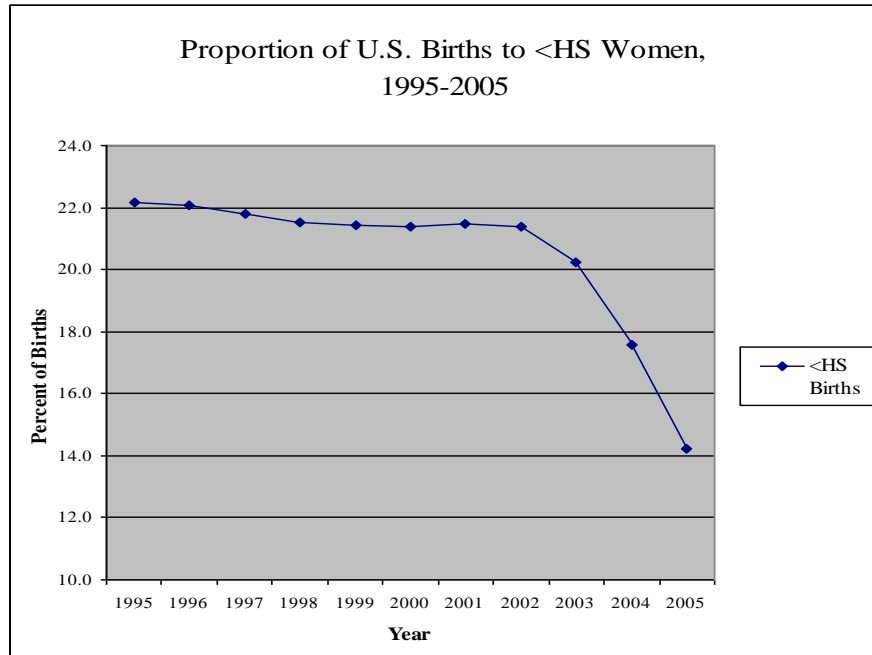


Figure 10. Proportion of U.S. Births to < HS Women, 1995-2005

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

Maternal marital status. U.S. births grouped by maternal marital status break down into a “1/3-2/3” split: births to unmarried women account for approximately one-third of U.S. births, while those to married women account for two-thirds. Between 1995-2002, however, the number of births to married women decreased by 3.0%, while births to unmarried women increased by 6.44%, rising from 32.09% of U.S. births in 1995 to 34.13% in 2002. This increase was accelerated after 2002, rising by nearly 9% to 37.1% of all U.S. births by 2005 (see Figure 11).

Maternal marital status and education. The increase in the number of births to unmarried women occurred in all four maternal education groups, progressively higher as educational attainment increases (1.87% < HS, 11.67% HS, 19.57% some college, 3.89% 16+ years). Note that despite the increase in the number of births to unmarried < HS

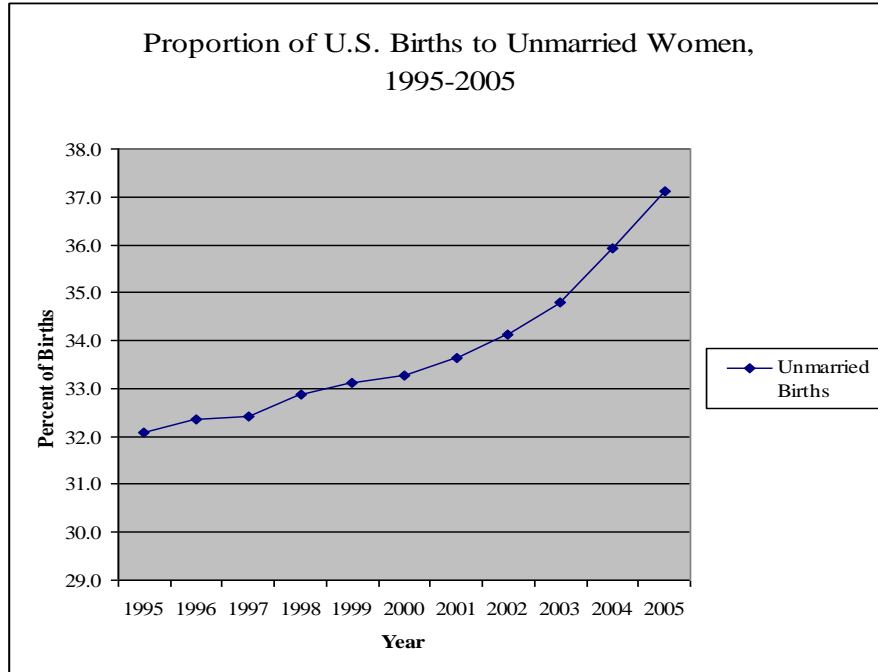


Figure 11. Proportion of U.S. Births to Unmarried Women: 1995-2005

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

women, its proportion of U.S. births decreased slightly between 1995-2002. The number of births to married women decreased in three of the education groups (-5.29% < HS, -16.25% HS, -5.53% Some College), increasing only among women with 16+ years of education (23.54%) (see Figure 12).

Maternal marital status and age. The number and proportion of U.S. births to unmarried women increased between 1995-2002 in all age groups except teens (15-19 years). The number of births to teens (15-19 years) decreased nearly 10% between 1995-2002, decreasing the proportion of U.S. births to unmarried teens from 9.78% to 8.62%. All other age groups experienced an increase in the number and proportion of births to unmarried women, with an increase of: 21.89% among 20-24 year-olds, 17.06% among

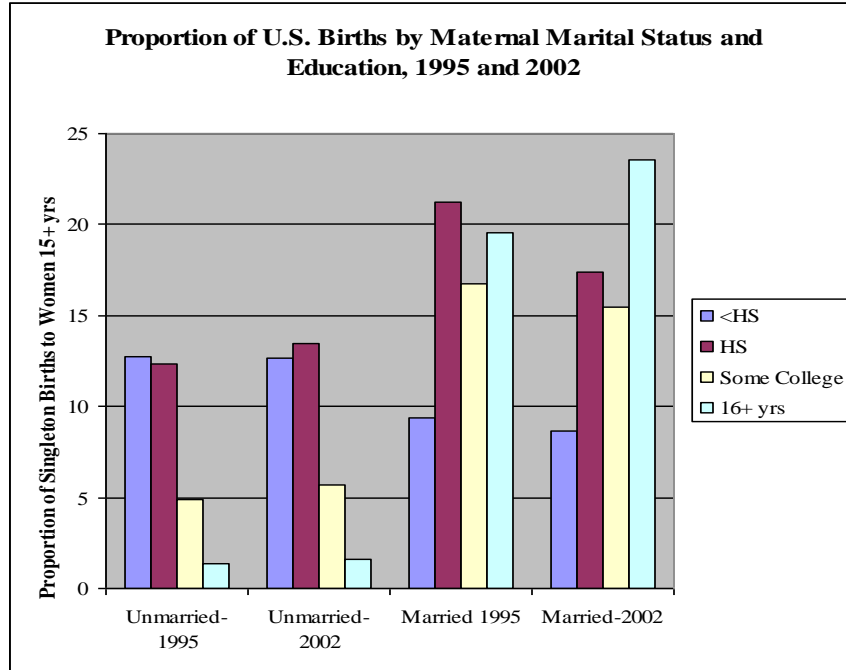


Figure 12. Proportion of U.S. Births by Maternal Marital Status and Education: 1995 and 2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

25-29 year-olds, 4.09% among 30-34 year-olds, 9.39% among 35-39 year-olds, 35.53% among 40-44 year-olds, and, 61.07% among 45+ years (see Figure 13).

Maternal age. Teenagers (15-19 years) represent the largest age group on average in the study population (46.73%), with the proportion decreasing with each successive age group: 20-24 years-29.80%, 25-29 years-13.43%, 30-34-6.5%, 35-39 years-2.88%, 40-44 years-0.64%, 45+ years-0.03%. The age distribution pattern of the study population stands in contrast to the older, more bell-shaped age distribution pattern for overall U.S. births: 15-19 years-12.13%, 20-24 years-25.08%, 25-29 years-27.15%, 30-34-22.89%, 35-39 years-10.62%, 40-44 years-2.05%, 45+ years-0.09% (see Figure 14).

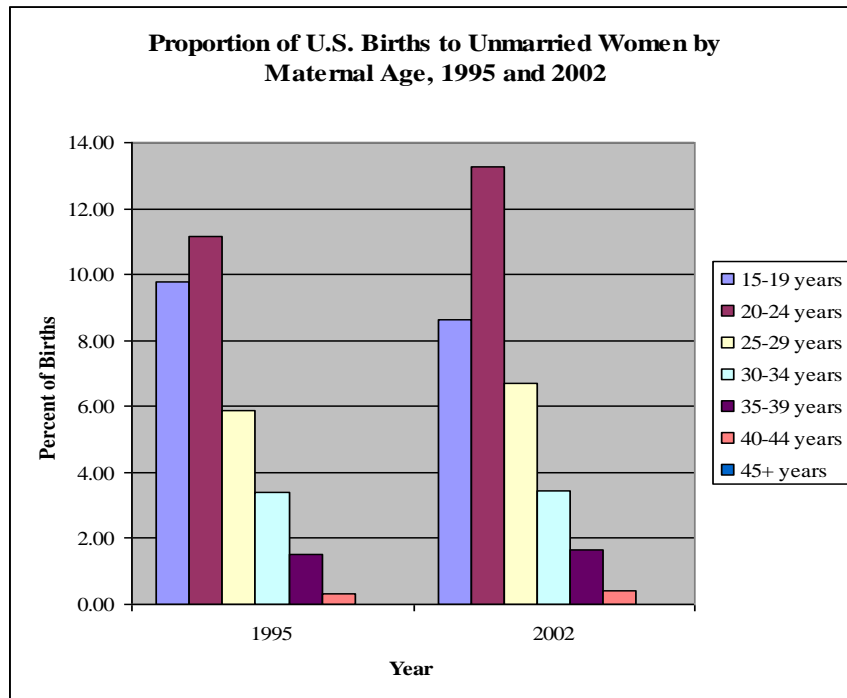


Figure 13. Proportion of U.S. Births to Unmarried Women by Maternal Age, 1995 and 2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, [http:// wonder.cdc.gov](http://wonder.cdc.gov)

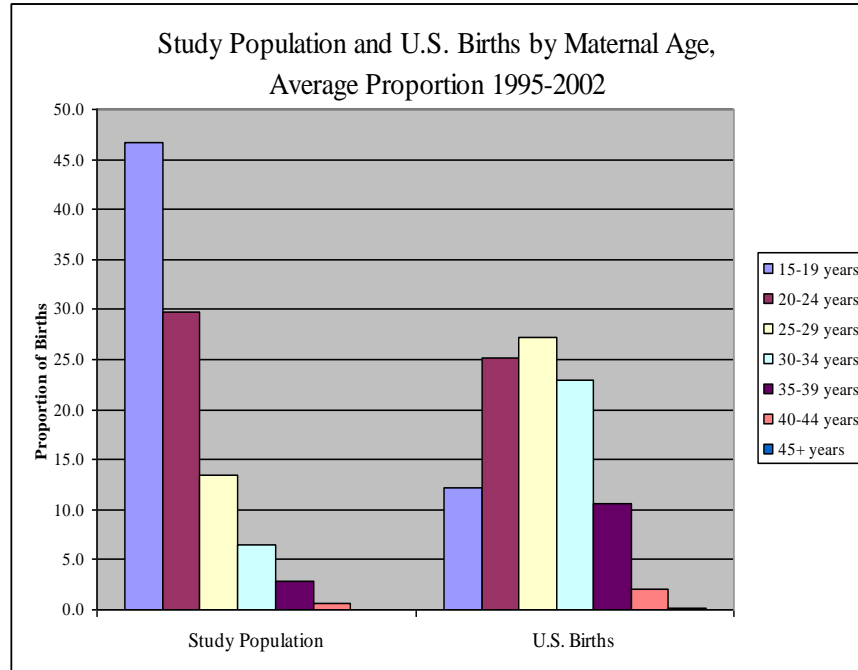


Figure 14. Study Population and U.S. Births by Maternal Age, Average Proportion 1995-2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

Although teens represent the largest proportion of births of any age group in the study population, it was the only age group in which the number--and the proportion--of births decreased between 1995-2002. The number of births to teens in the study group decreased by nearly 16%, from 241,311 in 1995 to 203,119 in 2002--a decrease from 49.46% of births in the study population in 1995 to 41.63% in 2002. The number of births in the study population increased for all other age groups between 1995-2002: 20-24 years-24.28%, 25-29 years-17.39%, 30-34-4.83%, 35-39 years-14.31%, 40-44 years-35.90%, 45+ years-30.53%. Additionally, each age group represented a larger proportion of births in the study population over time, particularly the 20-24 year age group which increased by 24.28%, rising from 26.93% of the study population in 1995 to 33.47% in 2002 (see Figure 15).

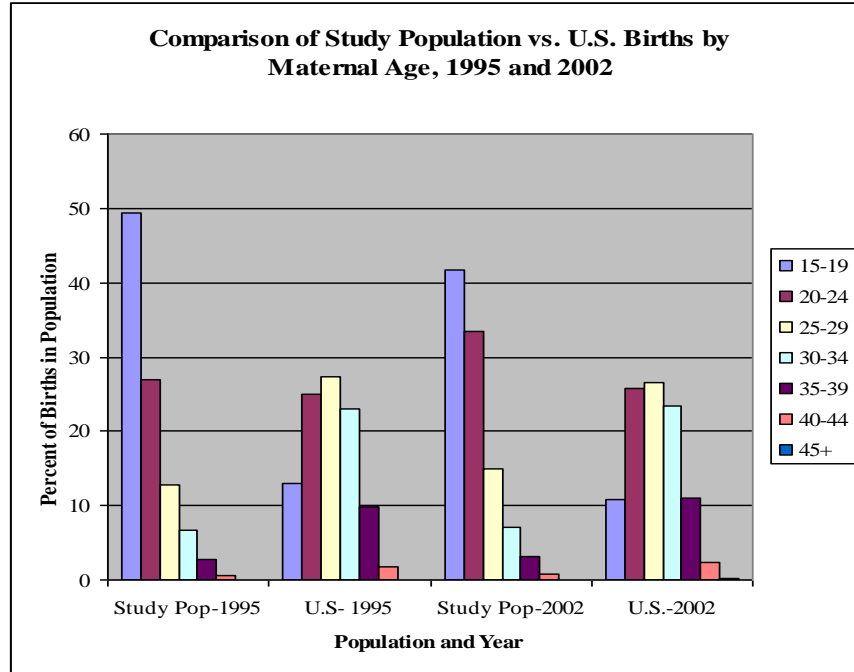


Figure 15. Comparison of Study Population vs. U.S. Births by Maternal Age, 1995 and 2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The decrease in number and proportion of births to teens in the study population reflects a larger trend of decreasing teen births in the U.S. beginning in 1992. Teen births in the U.S. decreased by 15% between 1995-2002 from 492,655 in 1995 accounting for 13.01% of U.S. births, to 418,666 in 2002 accounting for 10.78% in 2002 (see Figure 16).

Some research has indicated that the decrease in the teen birth rate that began in 1992 was largely a result of increased contraceptive use, but the role of abortion in this decline is not clear. The current research controls for teen births among 15-17 year-olds, but does not specifically address the issue of teen pregnancy.

Racial/ethnic composition. Of the births in the study population between 1995-2002, the average racial/ethnic composition was: Black - 27.25%, White/Non-Hispanic - 33.30%, Hispanic - 33.71%, Asian - 1.39%, and Native - 2.03% (see Figure 17).

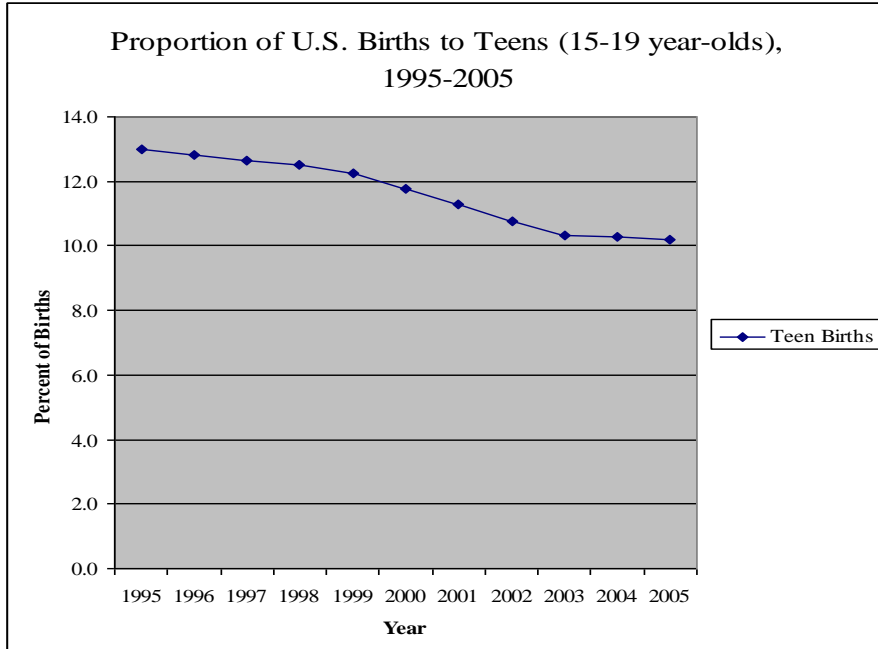


Figure 16. Proportion of U.S. Births to Teens (15-19 Year-Olds), 1995-2005

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

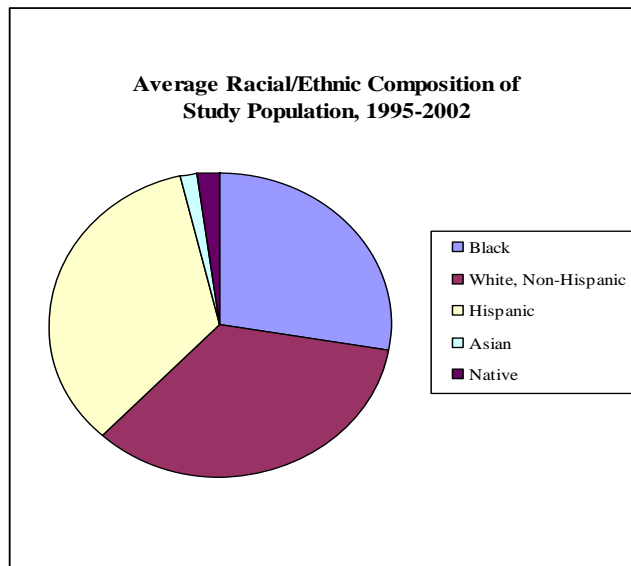


Figure 17. Average Racial/Ethnic Composition of Study Population: 1995-2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The relatively even racial/ethnic distribution among Black, White/Non-Hispanic, and Hispanic births in the study population stands in marked contrast to that of U.S. births in during those years: Black - 14.99%, White/Non-Hispanic - 59.01%, and Hispanic - 20.87% (see Figure 18).

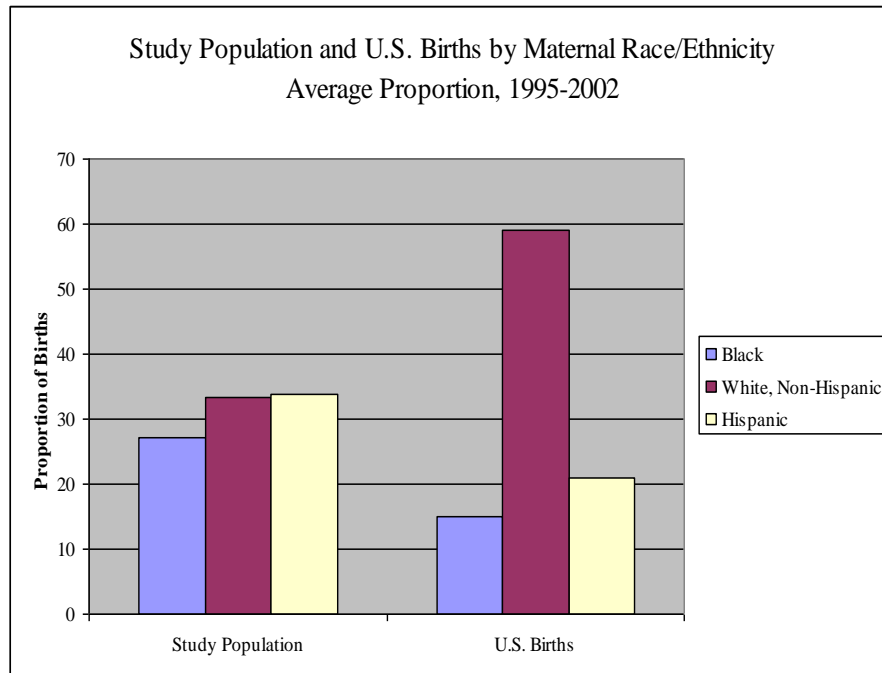


Figure 18. Study Population and U.S. Births by Maternal Race/Ethnicity Average Proportion: 1995-2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The average racial/ethnic composition of the study population, however, fails to reflect the trends over time. While births in the study population to Black mothers declined 16.5% from 143,319 in 1995 to 119,652 in 2002, and those to White, Non-Hispanic mothers declined 7.5% from 166,834 in 1995 to 154,335 in 2002, births to Hispanic mothers increased 31.7% from 145,385 in 1995 to 191,432 in 2002 (Figure 19).

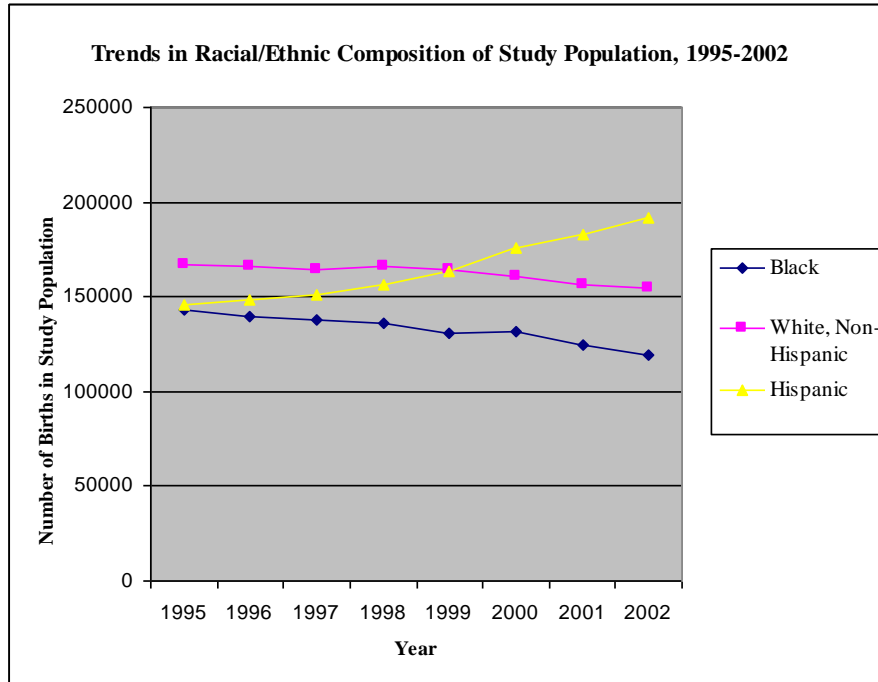


Figure 19. Trends in Racial/Ethnic Composition of Study Population: 1995-2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

Regional composition. There are regional variations in the composition of the study population. On average, 14.73% of the births in the study population were in the Northeast, 20.35% in the Midwest, 39.17% in the South, and 25.76% in the West. The five most populous states, California, Florida, Illinois, New York, and Texas, accounted for 43.03% of the study population (see Figure 20).

TANF Work Policies

For policies governing Work Exemptions and Hours of Work, more states adopted the two mid-range categories (categories 2 and 3) rather than the most lenient or the most stringent policy category. States adopted Sanctions policies more evenly across

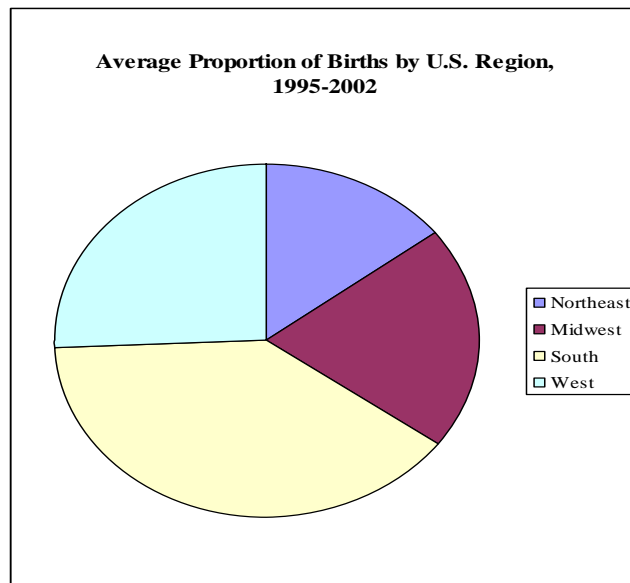


Figure 20. Average Proportion of Births by U.S. Region: 1995-2002

Source: Calculated from data available *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

the four categories, with some tendency toward the most stringent policies. The number of states adopting an Earnings Disregards policy increased as the stringency increased.

States are categorized below by their 1999 TANF work policies, as determined from information available at the Urban Institute's *Welfare Rules Database* (see Table 2). Although there are no identifiable patterns among state policy adoption, there is some regional concentration for state Sanction policies (see Figure 21).

Infant Mortality

During the 20th century the IMR in the United States decreased from nearly 100 in the year 1900 to 6.9 in 2000--a greater than 90% decline over the century (CDC, 1999a, 2002a). The public health programs of the early- to mid-part of the century, focused on

Table 2

States Categorized by 1999 TANF Work Policies

<u>Work Exemption</u>			
1	2	3	4
<u>>12 mo</u>	<u>12 mo</u>	<u>3-6 mo</u>	<u>None</u>
AL	AK	AR	AZ
MA	CA	DE	ID
NH	CO	FL	MT
TX	CT	HI	UT
VT	DC	IN	
VA	GA	IA	
	IL	MI	
	KS	NE	
	KY	NJ	
	LA	NY	
	ME	ND	
	MD	OK	
	MN	OR	
	MS	SD	
	MO	TN	
	NV	WA	
	NM	WI	
	NC	WY	
	OH		
	PA		
	RI		
	SC		
	WV		
<hr/>			
6	23	18	4

<u>Hours of Work</u>			
1	2	3	4
<u>Case</u>	<u><25 hrs</u>	<u>25-29 hrs</u>	<u>30+ hrs</u>
AK	CO	AK	CA
AZ	DE	AR	NE
CT	HI	DC	NJ
MD	IA	FL	NC
MA	KY	GA	TN
ND	LA	ID	TX
OR	MI	IL	VA
UT	MN	IN	WI
VT	MS	KS	
	NH	ME	
	NM	MO	
	OH	MT	
	PA	NV	
	RI	NY	
	SC	OK	
	SD		
	WA		
	WV		
	WY		
<hr/>			
9	19	15	8

<u>Sanctions</u>			
1	2	3	4
<u>Lenient</u>	<u>Intermediate</u>	<u>Restrictive</u>	<u>Stringent</u>
AR	AK	IA	AL
CA	AZ	MD	DE
DC	CO	MA	FL
HI	CT	MI	GA
ME	IL	MT	ID
MN	IN	NJ	KS
MO	KY	NM	LA
NH	NC	ND	MS
NY	OR	PA	NE
RI	TX	SD	NV
VT	WV	TN	OH
WA		UT	OK
			SC
			VA
			WI
			WY
<hr/>			
12	11	12	16

<u>Earnings Disregards</u>			
1	2	3	4
<u>Lenient</u>	<u>Intermediate</u>	<u>Restrictive</u>	<u>Stringent</u>
CA	DC	AK	AL
CT	KS	AZ	AR
FL	ME	ID	CO
HI	MA	LA	DE
IL	NH	MD	GA
IA	NJ	MI	IN
NM	NY	MN	KY
OH	OK	MT	MS
RI	OR	NV	MO
	PA	ND	NE
	UT	SD	NC
	WA	VT	SC
		WV	TN
		WY	TX
			VA
			WI
<hr/>			
9	12	14	16

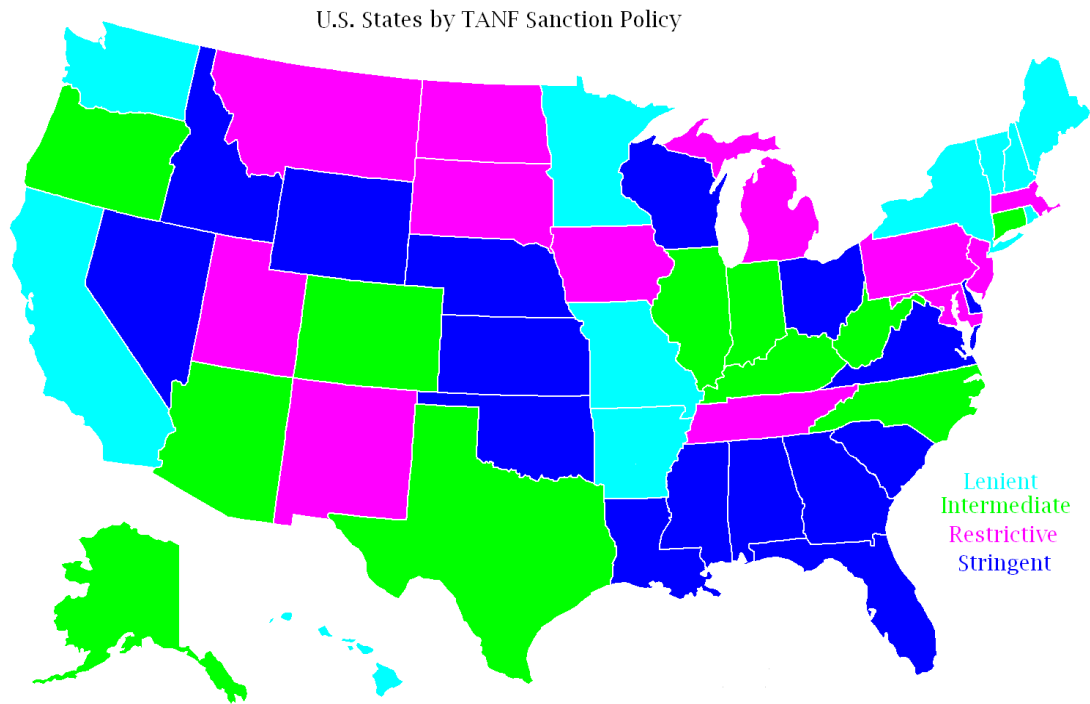


Figure 21. Regional Concentration for State Sanction Policies

sanitation and living conditions, combined with medical advances in antimicrobials and fluid replacement therapy during the 1930s and 1940s, improved an infant’s environment and contributed to a significant decline in postneonatal mortality rate in the first half of the century (CDC, 1999a). During the latter part of the century, however, technological advances in neonatal medicine contributed to a more significant decline in neonatal mortality (CDC, 1999a) (see Figure 22).

Infant mortality data (single births) for the research time period (1995-2002) demonstrate not only the continued decline in infant mortality over time, but the adverse effect of low maternal education and unmarried status as well (see Table 3).

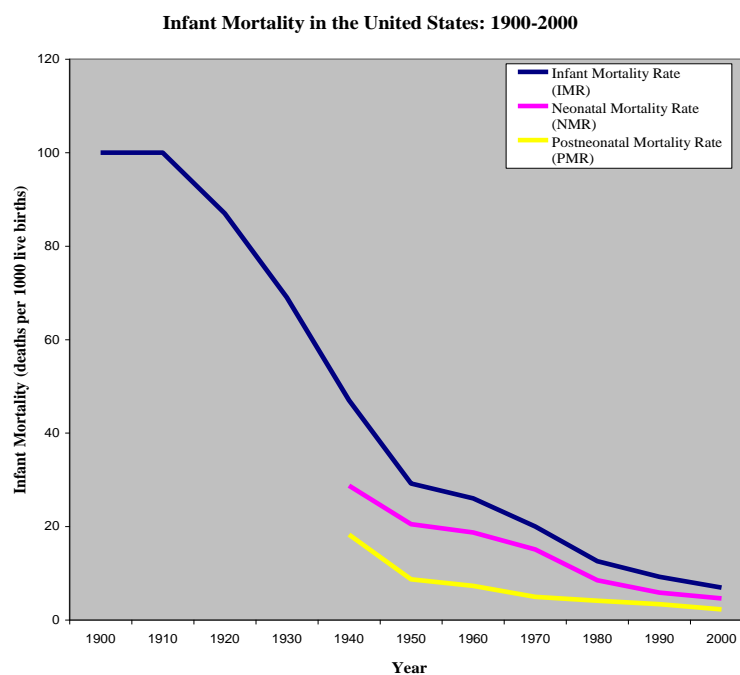


Figure 22. Infant Mortality in the United States: 1900-2000

*The IMR for 1910, 1920, and 1930 are approximated from documented percent-changes in IMR.

Source: From “Achievements in public health, 1900-1999: Healthier mothers and babies,” by Centers for Disease Control and Prevention, 1999a, *MMWR*, 48(38), pp. 849-858 and “Infant mortality statistics from the 2004 period linked birth/infant death dataset,” by Centers for Disease Control and Prevention, 2007, *National Vital Statistics Report*, 55(14), pp. 1-33.

Table 3

Infant Mortality Data: 1995-2002

	IMR			NMR			PMR		
	Overall U.S.	Unmarried <HS	Married 16+	Overall U.S.	Unmarried <HS	Married 16+	Overall U.S.	Unmarried <HS	Married 16+
1995	6.82	10.36	3.70	4.26	5.56	2.50	2.55	4.80	1.20
1996	6.56	10.09	3.51	4.12	5.39	2.43	2.45	4.70	1.08
1997	6.43	9.74	3.49	4.08	5.30	2.44	2.34	4.44	1.05
1998	6.36	9.43	3.48	4.07	5.17	2.45	2.29	4.26	1.03
1999	6.22	9.34	3.26	4.01	5.19	2.31	2.21	4.15	0.96
2000	6.11	9.20	3.30	3.94	5.11	2.35	2.17	4.10	0.95
2001	5.99	8.85	3.10	3.80	5.00	2.21	2.19	4.05	0.88
2002	6.09	9.15	3.09	3.91	5.00	2.27	2.18	4.16	0.83
Average	6.32	9.52	3.37	4.02	5.22	2.37	2.30	4.33	1.00

Source: *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

The unweighted average IMR (1995-2002) for the study population (“Unmarried < HS”)--singleton infants of unmarried women (15+ years-old) with less than a high school education--of 9.52 is one-and-a-half times that of the “Overall U.S.” rate of 6.32, and *three times* the average rate of 3.37 for singleton births to married women with 16+ years of education (“Married 16+”). This disparity is particularly prominent for postneonatal mortality rates (PMR)--so closely tied to an infant’s home environment--in which the average PMR for Unmarried < HS of 4.33 is nearly twice that of the Overall U.S. average PMR of 2.30 and nearly *four-and-a-half times* the average of 1.00 for Married 16+. The neonatal mortality rates (NMR), while closer than PMR, continues the pattern of disparity: the average NMR for Unmarried < HS of 5.22 is nearly one-and-a-half times that of the Overall U.S. average NMR of 4.02 and just over twice the average of 2.37 for Married 16+ (see Figure 23).

Of note is the increase in IMR between 2001-2002, a 1.7% increase in the Overall U.S. IMR from 5.99 to 6.09--the first increase in infant mortality in over 40 years. This increase was particularly significant among the Unmarried < HS population, with a 3.4% increase from 8.85 to 9.15. There was no increase among the Married 16+ population. Initial evaluation has determined the increase in IMR between 2001-2002 resulted from an increase in very-low-birthweight (VLBW) births, but it is unclear what factors contributed to this increase in VLBW (MacDorman, Martin, Mathews, Hoyert, & Ventura, 2005).

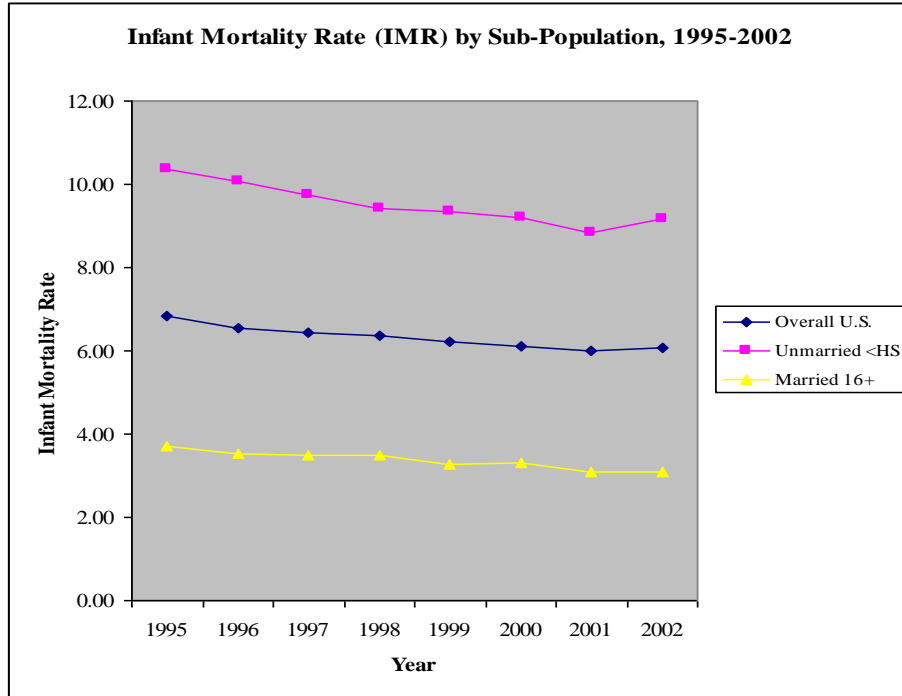


Figure 23. Infant Mortality Rate (IMR) by Subpopulation: 1995-2002

Source: *CDC Wonder System: Infant Deaths* by Centers for Disease Control and Prevention, n.d., available from CDC website, <http://wonder.cdc.gov>

Control Variables

Macroeconomic conditions. This research controls for macroeconomic conditions using the state unemployment rate for the target year. The U.S. seasonally adjusted unemployment rate decreased between 1996-2000, but began to rise in 2001 (see Figure 24).

Race. Given the persistent Black-White disparity in infant mortality, this research controls for race using the percentage of births to Black mothers in the study population. Although births to Black mothers accounted for an average of 15.23% of the total births in the U.S. between 1995-2002, they accounted for an average of 27.27% of the births in the study population during this time period. There was, however, a general

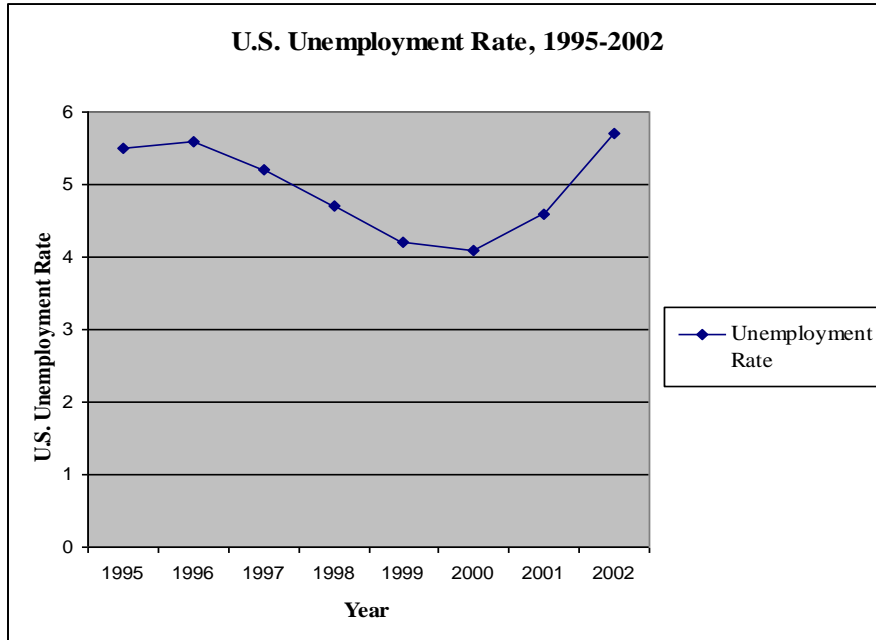


Figure 24. U.S. Unemployment Rate: 1995-2002

Source: U.S. Dept of Labor, Bureau of Labor Statistics-Regional and State Employment and Unemployment, www.bls.gov/schedule/archives/laus_nr.htm

decline in the percentage of births to Black mothers in the study population (see earlier discussion regarding the racial composition of the study population).

Teen births. Because the Linked Birth dataset includes 15-17 year-olds who are not likely subject to TANF work provisions, the teen birth rate for 15-17 year-olds (the number of live births per 1000 females in the 15-17 year-old population) is included to control for this.

The teen birth rate (15-19 year-olds) began a steady decline in 1992, decreasing 12.40% from the 1991 high of 62.1 to the 56.8 rate in 1996, the year of PROWRA's passage. During the research time period of 1995-2002, there was a 24.30% decline in the 15-19 year teen birth rate, more so among 15-17 year-olds for whom the birth rate decreased 32.50% from the 1992 rate of 36.0 to the 2002 rate of 24.3, rather than the 18-19 year-olds with a 22.88% decrease from 89.1 in 1995 to 72.8 in 2002 (see Figure 25).

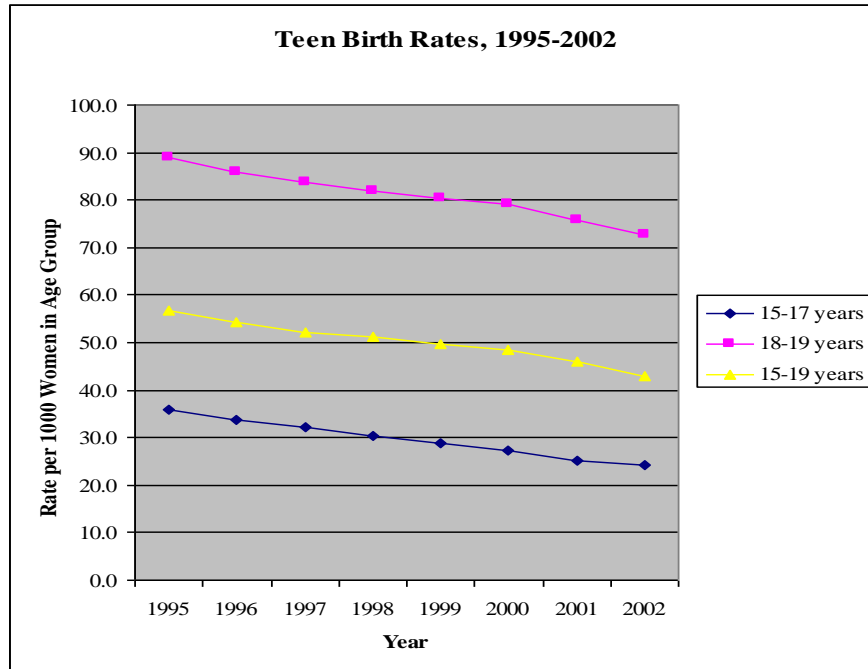


Figure 25. Teen Birth Rates: 1995-2002

Source: “Declines in teenage birth rates, 1991-1998: Update of national and state trends,” by Centers for Disease Control and Prevention, 199b, *National Vital Statistics Report*, 47(26), 1-10; “Births: Final data for 1999,” by Centers for Disease Control and Prevention, 2001, *National Vital Statistics Report*, 49(1), 1-100; “Births: Final data for 2000,” by Centers for Disease Control and Prevention, 2002d, *National Vital Statistics Report*, 50(5), 1-101; “Births: Final data for 2001,” by Centers for Disease Control and Prevention, 2002e; and “Births: Final data for 2002,” by Centers for Disease Control and Prevention, 2003, *National Vital Statistics Report*, 52(10), 1-113.

The teen birth rate differs from the teen pregnancy rate. While the birth rate is calculated based on the number of live births per population, the pregnancy rate represents the number of pregnancies in the population. As noted earlier, some research has demonstrated that the decrease in the teen birth rate that began in 1992 was largely a result of increased contraceptive use, but the role of abortion in this decline is not clear. This research, therefore, does not specifically address the issue of teen pregnancy. The teen birth rate also differs from the proportion of teen births--the percentage of births to teens in a population (e.g., study population)--discussed earlier.

Correlations

TANF Work Policies

Correlations were performed to assess: (a) the relationship between the four TANF work policies (Work Exemptions, Hours of Work, Sanctions, Earnings Disregards); (b) changes in the TANF work policies over time; (c) whether TANF work policies were enforced; and (d) any relationship between the work policies and pre-existing state conditions.⁶

1. Is the stringency of the four TANF work policies correlated with each other?

There is no statistically significant correlation between the degree of stringency of one TANF work policy with the degree of stringency of any other policy (see Table 4).

2. Did the TANF work policy choices change over time? While there was some

change in the policies governing the number of work hours over time (as required by PRWORA), there was virtually no change in the other three work policies: Exemption, Sanctions, or Earnings (see Table 5).

Table 4

Correlation Table: Relationship between TANF Work Policies

1999	Exemption	Hours	Sanctions	Earnings
Exemption	1.00 <i>p</i> =.000			
Hours	.035 <i>p</i> =.805	1.00 <i>p</i> =.000		
Sanctions	.138 <i>p</i> =.334	- .057 <i>p</i> =.692	1.00 <i>p</i> =.000	
Earnings	-.057 <i>p</i> =.691	.209 <i>p</i> =.142	.231 <i>p</i> =.102	1.00 <i>p</i> =.000

⁶ All correlations noted or discussed are statistically significant.

Table 5

Correlation Table: Change in TANF Work Policies over Time

	Exemption 1999	Hours 1999	Sanctions 1999	Earnings 1999
Exemption 2002	.970 <i>p</i> = .000*			
Hours 2002		.693 <i>p</i> = .000*		
Sanctions 2002			.940 <i>p</i> = .000*	
Earnings 2002				.950 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

3. *Were the TANF work policies enforced (i.e., is the TANF policy choice an accurate measure of policy impact)?* Enforcement measures for Exemption, Hours, and Sanctions policies suggest there was some level of policy enforcement by states or behavioral change as a result of policy. More stringent Exemption and Sanction policies were correlated with higher case closures due to sanctions; the stringency of Exemption policy was also directly correlated with work participation among TANF recipients; and, the stringency of the policies setting the required number of work hours was correlated with the higher average number of hours TANF recipients worked (see Table 6).

4. *Is there any relationship between state TANF work policies and pre-existing state macroeconomic conditions, demographics, access to health care, teen birth rate?*

Macroeconomic conditions: The stringency of Sanctions and Earnings Disregards policies are indirectly correlated with pre-existing (1995) unemployment and percentage of state population on AFDC (i.e., the higher the state unemployment rate or percentage of population on AFDC prior to PRWORA, the more lenient the subsequent state Sanctions and Earnings Disregards policies) (see Table 7).

Table 6

Correlation Table: TANF Work Policies and Measures of Enforcement

	% Cases Closed Due to Sanctions – 1999	TANF Work Participation Rate - 1999	Average # Hours Worked by TANF Recipients – 1999
Exemption 1999	.298 <i>p</i> = .034*	.339 <i>p</i> = .016*	
Hours 1999			.380 <i>p</i> = .006**
Sanctions 1999	.300 <i>p</i> = .033*		

*Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Table 7

Correlation Table: Work Policies and 1995 State Macroeconomic Conditions

	Unemployment Rate 1995	% State Pop on AFDC 1995	% State Pop under FPL 1995	% State Pop on FSP⁷ 1996
Exemption 1999	-.197 <i>p</i> = .165	-.182 <i>p</i> = .201	-.065 <i>p</i> = .650	-.207 <i>p</i> = .146
Hours 1999	.101 <i>p</i> = .482	.125 <i>p</i> = .381	.077 <i>p</i> = .592	.045 <i>p</i> = .752
Sanctions 1999	-.291 <i>p</i> = .038*	-.390 <i>p</i> = .005**	-.006 <i>p</i> = .967	-.122 <i>p</i> = .392
Earnings 1999	-.279 <i>p</i> = .047*	-.351 <i>p</i> = .011*	.101 <i>p</i> = .483	.062 <i>p</i> = .663

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Demographics: The stringency of Sanctions and Earnings Disregards policies is directly correlated with the percentage of the 1995 AFDC population that was Black (i.e., states with higher Black AFDC population in 1995 had more stringent Sanctions and Earnings Disregards policies); while Earnings Disregards policies are indirectly correlated with the Hispanic proportion of the 1995 AFDC population (i.e., states with

⁷ The 1996 Food Stamps enrollment data is used in this research as the 1995 data was not available.

higher Hispanic AFDC population in 1995 had more lenient Earnings Disregards policies). The state demographics, specifically the percentage of the state population that was Black in 1995, was indirectly correlated with subsequent Exemption policies (i.e., states with higher Black populations in 1995 had more lenient Exemption policies) (see Table 8).

Table 8

Correlation Table: TANF Work Policies and 1995 State Demographics

	% State Pop - Black 1995	% AFDC Pop - White 1995	% AFDC Pop - Black 1995	% AFDC Pop - Hispanic 1995
Exemption 1999	-.278 <i>p</i> = .048*	.146 <i>p</i> = .307	-.273 <i>p</i> = .053	-.014 <i>p</i> = .923
Hours 1999	.137 <i>p</i> = .336	-.189 <i>p</i> = .183	.229 <i>p</i> = .106	.010 <i>p</i> = .943
Sanctions 1999	.121 <i>p</i> = .397	-.161 <i>p</i> = .260	.306 <i>p</i> = .029*	-.143 <i>p</i> = .316
Earnings 1999	.257 <i>p</i> = .069	-.051 <i>p</i> = .721	.329 <i>p</i> = .018*	-.326 <i>p</i> = .020*

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Access to health care and teen births: There was no correlation between TANF work policies and either measure of health care access (the percentage of state population that was either on Medicaid or was uninsured in 1995), or the state teen birth rate of 1996 (see Table 9).

Control Variables

Correlations were performed to assess: (a) the relationship between three of the control variables (*Race*--percentage of births in study population to Black women in a

Table 9

Correlation Table: TANF Work Policies and 1995 Access to Health Care, Teen Births

	% State Pop – Medicaid 1995	% State Pop - Uninsured 1995	Teen Birth Rate 1996
Exemption 1999	.022 <i>p</i> = .877	- .102 <i>p</i> = .475	- .101 <i>p</i> = .481
Hours 1999	.048 <i>p</i> = .737	.193 <i>p</i> = .175	.175 <i>p</i> = .220
Sanctions 1999	- .112 <i>p</i> = .434	.092 <i>p</i> = .520	.079 <i>p</i> = .579
Earnings 1999	- .097 <i>p</i> = .497	.074 <i>p</i> = .607	.192 <i>p</i> = .178

state), *Teen Births*⁸ (state teen birth rate for 15-17 year-olds), *Unemployment* (state unemployment rate), and (b) the degree of consistency of these control variables over time. The fourth control variable, Baseline Infant Mortality (1995), is addressed in the next section on “Infant Mortality.”

1. *Are the control variables related to each other?* The percentage of births to Black women in the study population (Race) in the state was consistently directly correlated with the state teen birth rate for 15-17 year-olds (1995, 1999, and 2002) (i.e., states with higher representation of Black women in the study population had higher teen birth rates), and the state teen birth rate was consistently directly correlated with the state unemployment rate (1995, 1999, and 2002) (i.e., states with higher teen birth rates had higher unemployment rates) (see tables 10-12).

⁸ The 1996 Teen Birth rate (15-17 year-olds) was used in this research as the 1995 rate was not available.

Table 10

Correlation Table: Control Variables - 1995

1995	Race	Teen (1996)	Unemploy
Race	1.00 <i>p</i> = .000*		
Teen (1996)	.603 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
Unemploy	.233 <i>p</i> = .100	.461 <i>p</i> = .001*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Table 11

Correlation Table: Control Variables - 1999

1999	Race	Teen	Unemploy
Race	1.00 <i>p</i> = .000*		
Teen	.562 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
Unemploy	.026 <i>p</i> = .857	.419 <i>p</i> = .002*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Table 12

Correlation Table: Control Variables - 2002

2002	Race	Teen	Unemploy
Race	1.00 <i>p</i> = .000*		
Teen	.489 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
Unemploy	.207 <i>p</i> = .146	.394 <i>p</i> = .004*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

2. *Are the control variables consistent over time?*

Race: The percentage of births to Black women in the study population of each state remained very consistent across time (see Table 13).

Table 13

Correlation Table: Race, 1995-2002

Race	1995	1999	2002
1995	1.00 <i>p</i> = .000*		
1999	.997 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
2002	.994 <i>p</i> = .000*	.997 <i>p</i> = .000*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Teen births: The state teen birth rates (15-17 year-olds) remained rather consistent over time, although the general decline in teen births (discussed above) attenuated the relationship with 2002 rates (see Table 14).

Table 14

Correlation Table: Teen, 1996-2002

Teen	1996	1999	2002
1996	1.00 <i>p</i> = .000*		
1999	.991 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
2002	.515 <i>p</i> = .000*	.970 <i>p</i> = .000*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Unemployment: There was a definite secular trend in state Unemployment rates, although the rising unemployment after 2000 (discussed above) attenuated the results somewhat (see Table 15).

Table 15

Correlation Table: Unemployment, 1995-2002

Unemployment	1995	1999	2002
1995	1.00 <i>p</i> = .000*		
1999	.753 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
2002	.513 <i>p</i> = .000*	.548 <i>p</i> = .000*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Infant Mortality

Correlations were performed to assess: (a) whether there is a relationship between infant mortality and measures of *access to health care* in the state (percentage of state population on Medicaid or uninsured), state *macroeconomic conditions* (percentage of state population on AFDC, under the Federal Poverty Line, or on Food Stamps), or state *demographics* (percentage of state population that is Black); (b) the relationship of infant mortality with control variables; and (c) the trend in infant mortality over time.

1. *What is the relationship between infant mortality and access to health care, state macroeconomic conditions, or state demographics?* The statistically significant relationship is the correlation between infant mortality--specifically neonatal mortality--with the percentage of state population that is Black reflecting the disproportionately high infant mortality among Black infants (see Table 16).

Table 16

Correlation Table: Infant Mortality and State Variables, 1995

1995	IMR	NMR	PMR
Medicaid	.135 <i>p</i> = .341	.022 <i>p</i> = .880	.183 <i>p</i> = .198
Uninsured	-.122 <i>p</i> = .122	-.112 <i>p</i> = .112	-.085 <i>p</i> = .552
AFDC	-.027 <i>p</i> = .851	.031 <i>p</i> = .828	-.075 <i>p</i> = .599
< FPL	.099 <i>p</i> = .491	-.007 <i>p</i> = .961	.160 <i>p</i> = .263
FSP (1996)	.068 <i>p</i> = .634	.051 <i>p</i> = .724	.049 <i>p</i> = .732
Black Pop	.392 <i>p</i> = .004*	.399 <i>p</i> = .004*	.159 <i>p</i> = .266

*Correlation is significant at the 0.01 level (2-tailed).

2. *How closely is infant mortality related to the control variables (Race, Teen Births, Unemployment)?* Infant mortality is consistently correlated with race, again demonstrating the link between infant mortality Black race. The relationship is particularly an issue during the neonatal period, although race was also correlated with postneonatal mortality in 1999 and 2002. Infant mortality was correlated with the teen birth rate in 1999, during the postneonatal phase. And, infant mortality was *indirectly* correlated with unemployment in 1995 during the neonatal phase (see tables 17-19).

3. *Are infant mortality trends consistent across time?* Infant mortality trends stay relatively consistent across time, with occasional variation (e.g., neonatal mortality between 1999-2000, and postneonatal mortality for 1999) (see tables 20-22).

Table 17

Correlation Table: Control Variables and Infant Mortality, 1995

1995	IMR	NMR	PMR
Race	.419 <i>p</i> =.002**	.424 <i>p</i> =.002**	.173 <i>p</i> = .226
Teen (1996)	.145 <i>p</i> = .311	.136 <i>p</i> = .342	.072 <i>p</i> = .616
Unemploy	-.297 <i>p</i> = .034*	-.291 <i>p</i> = .038*	-.132 <i>p</i> = .357

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Table 18

Correlation Table: Control Variables and Infant Mortality, 1999

1999	IMR	NMR	PMR
Race	.605 <i>p</i> =.000**	.540 <i>p</i> =.000**	.473 <i>p</i> =.000**
Teen	.313 <i>p</i> = .025*	.224 <i>p</i> = .114	.297 <i>p</i> = .034*
Unemploy	-.057 <i>p</i> = .694	-.139 <i>p</i> = .330	.050 <i>p</i> = .730

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Table 19

Correlation Table: Control Variables and Infant Mortality, 2002

2002	IMR	NMR	PMR
Race	.441 <i>p</i> =.001**	.384 <i>p</i> =.005**	.297 <i>p</i> = .034*
Teen	.193 <i>p</i> = .175	.063 <i>p</i> = .660	.245 <i>p</i> = .084
Unemploy	-.045 <i>p</i> = .752	-.183 <i>p</i> = .198	.123 <i>p</i> = .388

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Table 20

Correlation Table: IMR, 1995-2002

IMR	1995	1999	2002
1995	1.00 <i>p</i> = .000*		
1999	.602 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
2002	.522 <i>p</i> = .000*	.458 <i>p</i> = .001*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Table 21

Correlation Table: NMR, 1995-2002

NMR	1995	1999	2002
1995	1.00 <i>p</i> = .000*		
1999	.407 <i>p</i> = .003*	1.00 <i>p</i> = .000*	
2002	.233 <i>p</i> = .100	.348 <i>p</i> = .012*	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

Table 22

Correlation Table: PMR, 1995-2002

PMR	1995	1999	2002
1995	1.00 <i>p</i> = .000*		
1999	.622 <i>p</i> = .000*	1.00 <i>p</i> = .000*	
2002	.351 <i>p</i> = .012*	.218 <i>p</i> = .124	1.00 <i>p</i> = .000*

*Correlation is significant at the 0.01 level (2-tailed).

TANF Work Policies and Infant Mortality

Correlations were performed to assess: (a) the relationship of each year's (1998-2002) TANF work policies (Exemption, Hours,⁹ Sanctions,¹⁰ Earnings) with infant mortality for that year; and (b) any relationship between baseline infant mortality (1995) and subsequent TANF work policies.

1. *Were TANF work policies correlated with infant mortality, 1998-2002?* State Sanctions and Earnings Disregards policies were correlated with state IMR in 1998, 1999, and 2001, and approached statistical significance in 2000. This appears to be largely during the neonatal phase for Sanctions, as Sanctions policy and NMR were correlated in 1998, 1999, 2000, and 2001, while Sanctions were only correlated with PMR in 1999. Earnings policies were correlated with NMR in 1999 and 2001, and with PMR in 1998 and 1999 (see tables 23-25).

2. *Was baseline infant mortality correlated with subsequent TANF work policies?*¹¹ Although correlations between (a) Sanction policies and IMR and PMR, (b) Earnings Disregards policies and IMR and PMR, and (c) Exemption policies and PMR began to approach statistical significance (with *p*-values ranging from .065 to .086), there were no statistically significant correlations between baseline infant mortality measures and subsequent TANF policies (see Table 26).

⁹No information was available regarding 1998 state "Hours" policies; given that the number of hours mandated by PRWORA was the same for 1998 and 1999 as well as the limited change exhibited over the subsequent years, the 1999 "Hours" policies were utilized.

¹⁰While information regarding the most severe sanctions was available for all years, the 1998 initial sanctions were not available. The 1998 "Sanctions" policies were approximated based on the subsequent policy pattern as well as any change in the most severe sanctions between 1998-1999.

¹¹ The 1999 TANF work policies are used for these correlations, being the first year in which complete information was available for all policies.

Table 23

Correlation Table: TANF Work Policies and IMR, 1998-2002

IMR	Exemption	Hours	Sanctions	Earnings
1998	-.019 <i>p</i> = .897	-.098 <i>p</i> = .494	.316 <i>p</i> = .024*	.389 <i>p</i> = .005**
1999	.107 <i>p</i> = .454	.022 <i>p</i> = .877	.470 <i>p</i> = .001**	.411 <i>p</i> = .003**
2000	.104 <i>p</i> = .467	-.038 <i>p</i> = .792	.267 <i>p</i> = .059 ^	.273 <i>p</i> = .052 ^
2001	.116 <i>p</i> = .417	-.063 <i>p</i> = .659	.326 <i>p</i> = .020*	.359 <i>p</i> = .010**
2002	-.036 <i>p</i> = .800	-.033 <i>p</i> = .817	.261 <i>p</i> = .064	.223 <i>p</i> = .116

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

^Correlation approaches statistical significance.

Table 24

Correlation Table: TANF Work Policies and NMR, 1998-2002

NMR	Exemption	Hours	Sanctions	Earnings
1998	.025 <i>p</i> = .862	-.051 <i>p</i> = .724	.330 <i>p</i> = .018*	.245 <i>p</i> = .083
1999	.150 <i>p</i> = .294	-.027 <i>p</i> = .851	.351 <i>p</i> = .012*	.393 <i>p</i> = .004**
2000	.064 <i>p</i> = .658	-.044 <i>p</i> = .758	.314 <i>p</i> = .025*	.256 <i>p</i> = .070
2001	.060 <i>p</i> = .677	-.205 <i>p</i> = .150	.346 <i>p</i> = .013*	.390 <i>p</i> = .005**
2002	-.100 <i>p</i> = .484	-.203 <i>p</i> = .153	.252 <i>p</i> = .075	.168 <i>p</i> = .238

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Table 25

Correlation Table: TANF Work Policies and PMR, 1998-2002

PMR	Exemption	Hours	Sanctions	Earnings
1998	-.060 <i>p</i> = .677	-.105 <i>p</i> = .463	.162 <i>p</i> = .257	.364 <i>p</i> = .009**
1999	-.002 <i>p</i> = .990	.053 <i>p</i> = .712	.461 <i>p</i> = .001**	.277 <i>p</i> = .049*
2000	.117 <i>p</i> = .412	-.016 <i>p</i> = .914	.161 <i>p</i> = .259	.234 <i>p</i> = .098
2001	.163 <i>p</i> = .254	.092 <i>p</i> = .522	.216 <i>p</i> = .128	.243 <i>p</i> = .085
2002	.053 <i>p</i> = .713	.170 <i>p</i> = .232	.149 <i>p</i> = .295	.178 <i>p</i> = .211

*Correlation is significant at the 0.05 level (2-tailed).

**Correlation is significant at the 0.01 level (2-tailed).

Table 26

Correlation Table: Baseline Infant Mortality and 1999 TANF Work Policies

	Exemption	Hours	Sanctions	Earnings
Base IMR	.082 <i>p</i> = .569	.037 <i>p</i> = .797	.261 <i>p</i> = .065	.251 <i>p</i> = .076
Base NMR	-.115 <i>p</i> = .423	.077 <i>p</i> = .591	.142 <i>p</i> = .321	.121 <i>p</i> = .398
Base PMR	.252 <i>p</i> = .074	-.028 <i>p</i> = .845	.243 <i>p</i> = .086	.250 <i>p</i> = .077

Regression Analysis

Linear Regression Modeling

Cross-sectional linear regression modeling was conducted for 1998-2002 to explore the relationship of TANF work policies (Exemption, Hours, and Sanctions) and additional policies (Earnings Disregards, Sanctions Waivers) with infant mortality once control variables had been introduced. There are several assumptions and limitations inherent in linear regression modeling:

- The relationship between variables is assumed to be linear;
- The residuals (the difference between the predicted and observed values) are assumed to be distributed normally;
- The regression results are descriptive, and do not determine underlying causal mechanisms; and
- The number of variables is limited by the number of observations: while there should ideally be 10-20 observations per variable, there must be a minimum of 5 observations per variable. With 51 observations, this research is limited to 10 or fewer total variables.

Multiple Regression Analysis: TANF Work Policies and Infant Mortality

Because of the secular trends in infant mortality, the baseline mortality rate is statistically significant, particularly in the postneonatal phase (in 1998-2001) rather than in the neonatal phase (only in 1998).

The results reinforce the role of race in infant mortality, particularly during the neonatal phase. Race was statistically significant for IMR (in 1999-2001), and in all years for NMR (in 1998-2002), but not for PMR in any year.

Of the three TANF work policies initially included, the only statistically significant TANF work policy is the Sanction policy with PMR in 1999: *States with more stringent Sanction policies had higher PMRs in the study population in 1999* (see tables 27-29).

Table 27

Regression Model Coefficients: TANF Work Policies and IMR, 1998-2002

IMR	1998	1999	2000	2001	2002
Exemption	.227 <i>p</i> = .603	.314 <i>p</i> = .604	.602 <i>p</i> = .419	1.085 <i>p</i> = .118	-.242 <i>p</i> = .748
Hours	-.342 <i>p</i> = .545	-.066 <i>p</i> = .903	-.042 <i>p</i> = .951	-.405 <i>p</i> = .518	-.347 <i>p</i> = .612
Sanctions	.282 <i>p</i> = .644	1.032 <i>p</i> = .081	.319 <i>p</i> = .661	.898 <i>p</i> = .197	.580 <i>p</i> = .434
Unemployment	-.128 <i>p</i> = .645	-.043 <i>p</i> = .878	.564 <i>p</i> = .151	.373 <i>p</i> = .363	.048 <i>p</i> = .898
Race	.026 <i>p</i> = .141	.044 <i>p</i> = .017*	.065 <i>p</i> = .005**	.045 <i>p</i> = .039*	.027 <i>p</i> = .257
Baseline Rate	.393 <i>p</i> = .002**	.366 <i>p</i> = .004**	.361 <i>p</i> = .021*	.371 <i>p</i> = .009**	.421 <i>p</i> = .009**
Teen Births	-.003 <i>p</i> = .932	.020 <i>p</i> = .574	-.011 <i>p</i> = .797	-.032 <i>p</i> = .471	.010 <i>p</i> = .8398
(Constant)	5.831 <i>p</i> = .002**	3.797 <i>p</i> = .033*	1.937 <i>p</i> = .389	2.709 <i>p</i> = .233	3.960 <i>p</i> = .161
R²	.397	.560	.500	.470	.349
Mean Value a, b	10.22	9.88	9.74	9.43	9.74

^aDependent Value is number of deaths per 1000 live births < 1 year of age.

^bMean is an unweighted average.

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 28

Regression Model Coefficients: TANF Work Policies and NMR, 1998-2002

NMR	1998	1999	2000	2001	2002
Exemption	.544 <i>p</i> = .159	.627 <i>p</i> = .162	.359 <i>p</i> = .396	.826 <i>p</i> = .053 ^	.119 <i>p</i> = .812
Hours	-.266 <i>p</i> = .461	-.228 <i>p</i> = .580	-.048 <i>p</i> = .906	-.628 <i>p</i> = .119	-.741 <i>p</i> = .121
Sanctions	.178 <i>p</i> = .647	.263 <i>p</i> = .551	.450 <i>p</i> = .299	.515 <i>p</i> = .242	.821 <i>p</i> = .113
Unemployment	-.194 <i>p</i> = .288	-.151 <i>p</i> = .494	.230 <i>p</i> = .345	-.363 <i>p</i> = .180	-.343 <i>p</i> = .209
Race	.024 <i>p</i> = .037*	.038 <i>p</i> = .006**	.050 <i>p</i> = .001**	.052 <i>p</i> = .000**	.035 <i>p</i> = .035*
Baseline Rate	.238 <i>p</i> = .035*	.123 <i>p</i> = .335	.089 <i>p</i> = .484	-.151 <i>p</i> = .214	-.070 <i>p</i> = .639
Teen Births	-.017 <i>p</i> = .472	.002 <i>p</i> = .929	-.016 <i>p</i> = .530	-.018 <i>p</i> = .521	-.008 <i>p</i> = .811
(Constant)	4.896 <i>p</i> = .000**	4.295 <i>p</i> = .002**	2.833 <i>p</i> = .039*	6.503 <i>p</i> = .000**	6.838 <i>p</i> = .001**
R²	.401	.388	.471	.412	.302
Mean Value a, b	5.71	5.65	5.34	5.17	5.34

^aDependent Value is number of deaths (1-27 days) per 1000 live births.

^bMean is an unweighted average.

^Approaches statistical significance.

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 29

Regression Model Coefficients: TANF Work Policies and PMR, 1998-2002

PMR	1998	1999	2000	2001	2002
Exemption	-.244 <i>p</i> = .534	-.274 <i>p</i> = .381	.294 <i>p</i> = .472	.512 <i>p</i> = .136	-.004 <i>p</i> = .994
Hours	-.137 <i>p</i> = .712	.036 <i>p</i> = .900	-.082 <i>p</i> = .828	.056 <i>p</i> = .857	.371 <i>p</i> = .424
Sanctions	-.015 <i>p</i> = .971	.702 <i>p</i> = .027*	-.331 <i>p</i> = .424	.370 <i>p</i> = .284	-.258 <i>p</i> = .607
Unemployment	-.019 <i>p</i> = .918	-.049 <i>p</i> = .738	.098 <i>p</i> = .654	.349 <i>p</i> = .089	.004 <i>p</i> = .986
Race	.008 <i>p</i> = .444	.014 <i>p</i> = .107	.027 <i>p</i> = .019	.018 <i>p</i> = .065	.019 <i>p</i> = .194
Baseline Rate	.459 <i>p</i> = .000**	.445 <i>p</i> = .000**	.485 <i>p</i> = .000**	.442 <i>p</i> = .000**	.292 <i>p</i> = .266
Teen Births	.013 <i>p</i> = .589	.017 <i>p</i> = .354	.006 <i>p</i> = .821	-.011 <i>p</i> = .628	.020 <i>p</i> = .557
(Constant)	1.833 <i>p</i> = .049*	1.097 <i>p</i> = .118	.840 <i>p</i> = .370	-.071 <i>p</i> = .937	2.033 <i>p</i> = .161
R²	.351	.585	.477	.550	.209
Mean Value a, b	4.51	4.25	4.39	4.28	4.40

^aDependent Value is number of deaths (28-364 days) per 1000 live births.^bMean is an unweighted average.

^Approaches statistical significance.

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Multiple Regression Analysis: Expanding to Additional Policies

When both the Earnings Disregards policy and the pre-existing Sanctions waiver policy (whether a sanction waiver policy was in place prior to PRWORA's 1996 passage) were factored in, the baseline infant mortality rate and race remained statistically significant, following the same pattern as regression results for the three TANF work policies, with the exception of race now being statistically significant for PMR in 2000.

With the addition of the Earnings policy and the Sanction waiver status, the Sanction policy was more statistically significant for PMR in 1999, and was now statistically significant for overall IMR in 1999. *States with more stringent Sanction policies had higher overall infant mortality in the study population in 1999, specifically during the postneonatal period.*

With the inclusion of Earnings Disregards and Sanction waiver status, the Exemption policy was statistically significant during the neonatal phase in 2001: *States with more stringent Exemption policies had higher NMR in the study population in 2001.*

Earnings Disregards policy was statistically significant for IMR in 1998, 1999, and 2001, specifically during the neonatal period in 1999 and 2001: *States with more stringent Earnings Disregards policies had higher overall infant mortality in the study population in 1998, 1999, and 2001, specifically during the neonatal period in 1999 and 2001.*

Sanction waiver policy (a dichotomous variable, 0-No, 1-Yes) was also statistically significant for overall infant mortality in 1999 and 2000, during the neonatal period for both 1999 and 2000, and during the postneonatal phase in 1999. *For these*

time periods, states that did not have a sanction waiver in place had higher infant mortality in the study population (see tables 30-32).

Table 30

Regression Model Coefficients: Full TANF Policies and IMR, 1998-2002

IMR	1998	1999	2000	2001	2002
Exemption	.292 <i>p</i> =.624	.622 <i>p</i> =.228	.922 <i>p</i> =.203	1.186 <i>p</i> =.079	-.366 <i>p</i> =.637
Hours	-.389 <i>p</i> =.479	-.320 <i>p</i> =.487	-.351 <i>p</i> =.596	-.466 <i>p</i> =.441	-.285 <i>p</i> =.681
Sanctions	.385 <i>p</i> =.518	1.304 <i>p</i> =.012*	.628 <i>p</i> =.377	1.022 <i>p</i> =.136	.443 <i>p</i> =.565
Earnings	1.411 <i>p</i> =.018*	1.331 <i>p</i> =.006**	.616 <i>p</i> =.340	1.338 <i>p</i> =.029*	.242 <i>p</i> =.442
Waiver	-.253 <i>p</i> =.753	- 2.317 <i>p</i> =.001**	- 2.277 <i>p</i> =.021*	-.749 <i>p</i> =.391	.736 <i>p</i> =.461
Unemployment	-.041 <i>p</i> =.877	-.004 <i>p</i> =.985	.403 <i>p</i> =.287	.399 <i>p</i> =.314	.098 <i>p</i> =.798
Race	.027 <i>p</i> =.112	.041 <i>p</i> =.008**	.061 <i>p</i> =.007**	.043 <i>p</i> =.040*	.028 <i>p</i> =.245
Baseline Rate	.331 <i>p</i> =.009**	.364 <i>p</i> =.001**	.372 <i>p</i> =.015*	.336 <i>p</i> =.015*	.397 <i>p</i> =.015*
Teen Births	-.027 <i>p</i> =.475	-.002 <i>p</i> =.942	-.020 <i>p</i> =.639	-.049 <i>p</i> =.258	.006 <i>p</i> =.905
(Constant)	5.877 <i>p</i> =.004**	3.734 <i>p</i> =.013*	2.586 <i>p</i> =.232	2.673 <i>p</i> =.222	3.347 <i>p</i> =.254
R²	.476	.707	.569	.535	.366
Mean Value ^{a, b}	10.22	9.88	9.74	9.43	9.74

a. Dependent Value is number of deaths (28-364 days) per 1000 live births.

b. Mean is an unweighted average.

^ . Approaches statistical significance.

*. Significant at the 0.05 level (2-tailed).

** . Significant at the 0.01 level (2-tailed).

Table 31

Regression Model Coefficients: Full TANF Policies and NMR, 1998-2002

NMR	1998	1999	2000	2001	2002
Exemption	.477 <i>p</i> =.227	.717 <i>p</i> =.087	.578 <i>p</i> =.144	.787 <i>p</i> =.049*	.028 <i>p</i> =.956
Hours	-.234 <i>p</i> =.521	-.320 <i>p</i> =.400	-.252 <i>p</i> =.499	-.648 <i>p</i> =.082	-.703 <i>p</i> =.149
Sanctions	.163 <i>p</i> =.684	.397 <i>p</i> =.340	.709 <i>p</i> =.084	.535 <i>p</i> =.197	.739 <i>p</i> =.171
Earnings	.503 <i>p</i> =.185	.977 <i>p</i> =.012*	.383 <i>p</i> =.287	1.118 <i>p</i> =.003**	.119 <i>p</i> =.588
Waiver	.229 <i>p</i> =.671	- 1.151 <i>p</i> =.043*	- 1.691 <i>p</i> =.003**	-.165 <i>p</i> =.755	.452 <i>p</i> =.514
Unemployment	-.152 <i>p</i> =.410	-.068 <i>p</i> =.737	.141 <i>p</i> =.532	-.283 <i>p</i> =.258	-.309 <i>p</i> =.275
Race	.023 <i>p</i> =.049*	.033 <i>p</i> =.009**	.044 <i>p</i> =.001**	.047 <i>p</i> =.001**	.035 <i>p</i> =.041*
Baseline Rate	.243 <i>p</i> =.035*	.198 <i>p</i> =.099	.139 <i>p</i> =.235	-.117 <i>p</i> =.304	-.073 <i>p</i> =.636
Teen Births	-.023 <i>p</i> =.353	-.012 <i>p</i> =.637	-.022 <i>p</i> =.355	-.029 <i>p</i> =.267	-.010 <i>p</i> =.785
(Constant)	4.550 <i>p</i> =.000*	3.534 <i>p</i> =.006**	3.043 <i>p</i> =.018*	5.715 <i>p</i> =.000**	6.375 <i>p</i> =.003**
R²	.430	.514	.581	.526	.314
Mean Value ^{a, b}	5.71	5.65	5.34	5.17	5.34

a. Dependent Value is number of deaths (28-364 days) per 1000 live births.

b. Mean is an unweighted average.

^ . Approaches statistical significance.

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 32

Regression Model Coefficients: Full TANF Policies and PMR, 1998-2002

PMR	1998	1999	2000	2001	2002
Exemption	-.157 <i>p</i> =.690	-.106 <i>p</i> =.723	.371 <i>p</i> =.384	.554 <i>p</i> =.120	-.086 <i>p</i> =.869
Hours	-.184 <i>p</i> =.617	-.080 <i>p</i> =.767	-.146 <i>p</i> =.711	.044 <i>p</i> =.889	.415 <i>p</i> =.381
Sanctions	.140 <i>p</i> =.733	.869 <i>p</i> =.005**	-.258 <i>p</i> =.547	.425 <i>p</i> =.239	-.365 <i>p</i> =.486
Earnings	.820 <i>p</i> =.052 ^	.170 <i>p</i> =.549	.086 <i>p</i> =.829	-.003 <i>p</i> =.993	.081 <i>p</i> =.712
Waiver	-.410 <i>p</i> =.448	- 1.050 <i>p</i> =.010**	-.484 <i>p</i> =.396	-.328 <i>p</i> =.474	.582 <i>p</i> =.387
Unemployment	.056 <i>p</i> =.760	-.057 <i>p</i> =.689	.064 <i>p</i> =.781	.334 <i>p</i> =.113	.039 <i>p</i> =.875
Race	.008 <i>p</i> =.453	.014 <i>p</i> =.101	.027 <i>p</i> =.024*	.018 <i>p</i> =.075	.019 <i>p</i> =.186
Baseline Rate	.366 <i>p</i> =.003**	.427 <i>p</i> =.000**	.480 <i>p</i> =.000**	.445 <i>p</i> =.000**	.275 <i>p</i> =.061 ^
Teen Births	-.002 <i>p</i> =.924	.011 <i>p</i> =.534	.004 <i>p</i> =.874	-.013 <i>p</i> =.583	.020 <i>p</i> =.560
(Constant)	1.885 <i>p</i> =.041*	1.357 <i>p</i> =.046*	1.037 <i>p</i> =.292	.039 <i>p</i> =.967	1.668 <i>p</i> =.283
R²	.415	.650	.487	.556	.226
Mean Value ^{a, b}	4.51	4.25	4.39	4.28	4.40

a. Dependent Value is number of deaths (28-364 days) per 1000 live births.

b. Mean is an unweighted average.

^ . Approaches statistical significance.

* . Significant at the 0.05 level (2-tailed).

** . Significant at the 0.01 level (2-tailed).

To assess the contribution of Earnings Disregards and Sanctions Waivers individually to the fit of the regression equation, additional regression analyses were performed for 1999, the year in which most policy findings were concentrated. These analyses demonstrate that *each additional policy--Earnings Disregards and Sanctions Waivers--contributes to a better fit of the regression equation, with Earnings Disregards playing more of a role in NMR and Sanctions Waivers more of a role in PMR* (see tables 33-35).

Table 33

Regression Model Coefficients: Additional TANF Policies and IMR, 1999

IMR - 1999	TANF Work Policies	Work Policies plus Earnings	Work Policies plus Waiver	Work Policies plus Both
Exemption	.314 <i>p</i> = .604	.342 <i>p</i> = .548	.590 <i>p</i> = .291	.622 <i>p</i> = .228
Hours	-.066 <i>p</i> = .903	-.085 <i>p</i> = .868	-.298 <i>p</i> = .550	-.320 <i>p</i> = .487
Sanctions	1.032 <i>p</i> = .081	.937 <i>p</i> = .092	1.397 <i>p</i> = .013*	1.304 <i>p</i> = .012*
Earnings		1.313 <i>p</i> = .014*		1.331 <i>p</i> = .006**
Waiver			- 2.295 <i>p</i> = .003**	- 2.317 <i>p</i> = .001**
Unemployment	-.043 <i>p</i> = .878	.093 <i>p</i> = .882	-.087 <i>p</i> = .731	-.004 <i>p</i> = .985
Race	.044 <i>p</i> = .017*	.046 <i>p</i> = .009**	.039 <i>p</i> = .018**	.041 <i>p</i> = .008**
Baseline Rate	.366 <i>p</i> = .004**	.320 <i>p</i> = .007**	.410 <i>p</i> = .001**	.364 <i>p</i> = .001**
Teen Births	.020 <i>p</i> = .574	-.007 <i>p</i> = .835	.011 <i>p</i> = .733	-.002 <i>p</i> = .942
(Constant)	3.797 <i>p</i> = .033*	3.546 <i>p</i> = .034*	3.987 <i>p</i> = .014*	3.734 <i>p</i> = .013*
R²	.560	.619	.646	.707

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 34

Regression Model Coefficients: Additional TANF Policies and NMR, 1999

NMR - 1999	TANF Work Policies	Work Policies plus Earnings	Work Policies plus Waiver	Work Policies plus Both
Exemption	.627 <i>p</i> = .162	.576 <i>p</i> = .176	.760 <i>p</i> = .088	.717 <i>p</i> = .087
Hours	-.228 <i>p</i> = .580	-.213 <i>p</i> = .586	-.329 <i>p</i> = .418	-.320 <i>p</i> = .400
Sanctions	.263 <i>p</i> = .551	.198 <i>p</i> = .636	.452 <i>p</i> = .309	.397 <i>p</i> = .340
Earnings		.937 <i>p</i> = .020*		.977 <i>p</i> = .012*
Waiver			- 1.076 <i>p</i> = .074	- 1.151 <i>p</i> = .043*
Unemployment	-.151 <i>p</i> = .494	-.069 <i>p</i> = .746	-.154 <i>p</i> = .473	-.068 <i>p</i> = .737
Race	.038 <i>p</i> = .006**	.036 <i>p</i> = .006**	.036 <i>p</i> = .008**	.033 <i>p</i> = .009**
Baseline Rate	.123 <i>p</i> = .335	.153 <i>p</i> = .210	.164 <i>p</i> = .195	.198 <i>p</i> = .099
Teen Births	.002 <i>p</i> = .929	-.007 <i>p</i> = .800	-.002 <i>p</i> = .935	-.012 <i>p</i> = .637
(Constant)	4.295 <i>p</i> = .002**	3.559 <i>p</i> = .007**	4.301 <i>p</i> = .001**	3.534 <i>p</i> = .006**
R²	.388	.463	.433	.514

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 35

Regression Model Coefficients: Additional TANF Policies and PMR, 1999

PMR - 1999	TANF Work Policies	Work Policies plus Earnings	Work Policies plus Waiver	Work Policies plus Both
Exemption	-.274 <i>p</i> = .381	-.266 <i>p</i> = .399	-.117 <i>p</i> = .694	-.106 <i>p</i> = .723
Hours	.036 <i>p</i> = .900	.036 <i>p</i> = .901	-.079 <i>p</i> = .768	-.080 <i>p</i> = .767
Sanctions	.702 <i>p</i> = .027*	.698 <i>p</i> = .029*	.872 <i>p</i> = .005**	.869 <i>p</i> = .005**
Earnings		.144 <i>p</i> = .636		.170 <i>p</i> = .549
Waiver			- 1.042 <i>p</i> = .010**	- 1.050 <i>p</i> = .010**
Unemployment	-.049 <i>p</i> = .738	-.035 <i>p</i> = .819	-.074 <i>p</i> = .593	-.057 <i>p</i> = .689
Race	.014 <i>p</i> = .107	.014 <i>p</i> = .107	.013 <i>p</i> = .102	.014 <i>p</i> = .101
Baseline Rate	.445 <i>p</i> = .000**	.430 <i>p</i> = .000**	.444 <i>p</i> = .000**	.427 <i>p</i> = .000**
Teen Births	.017 <i>p</i> = .354	.016 <i>p</i> = .410	.013 <i>p</i> = .102	.011 <i>p</i> = .534
(Constant)	1.097 <i>p</i> = .118	3.559 <i>p</i> = .135	1.394 <i>p</i> = .038*	1.357 <i>p</i> = .046*
R²	.585	.587	.646	.650

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Discussion

Overview of Regression Findings

In analyzing the relationship between TANF Work Policies (Exemption, Hours, and Sanctions) and infant mortality, this linear regression modeling indicates that, after controlling for state macroeconomic conditions (unemployment), race (Black), baseline infant mortality (1995), and teen birth rates (15-17 years-old), **states with more stringent Sanctions policies had higher postneonatal mortality (PMR) in the study population** ($\beta = .702$, p -value = .027) in 1999, but for no other year or measure of infant mortality. Neither Exemption nor Hours policies were statistically significant in any year for any measure of infant mortality.

When the Earnings Disregards policy was included and the Sanction Waiver status was factored into the linear regression model, **the statistical significance of the Sanction policy in the 1999 PMR increased** ($\beta = .869$, p -value = .005), and the Exemption policy became statistically significant for NMR in 2001 ($\beta = .787$, p -value = .049) such that **states with more stringent Exemption policies had higher neonatal mortality (NMR) in the study population in 2001**.

The Earnings Disregards policy was statistically significant for IMR in 1998, 1999, and 2001, specifically during the neonatal period in 1999 and 2001: **States with more stringent Earnings Disregards policies had higher overall infant mortality in the study population** in 1998 ($\beta = 1.411$, p -value = .018), 1999 ($\beta = 1.331$, p -value = .006), and 2001 ($\beta = 1.338$, p -value = .029), **specifically during the neonatal period** in 1999 ($\beta = .977$, p -value = .012) and 2001 ($\beta = 1.118$, p -value = .003).

The absence of a Sanction Waiver in place before PRWORA's passage was also statistically significant for overall infant mortality in 1999 ($\beta = -2.317$, p -value = .001) and 2000 ($\beta = -2.277$, p -value = .021), during the neonatal period for both 1999 ($\beta = -1.151$, p -value = .043) and 2000 ($\beta = -1.691$, p -value = .003), and during the postneonatal phase in 1999. ***For these time periods, states that did not have a Sanction Waiver in place prior to PRWORA had higher infant mortality in the study population.***

As for the role of control variables, given the secular trends in infant mortality, the baseline mortality rate is statistically significant in this regression model, particularly in the postneonatal phase (in 1998-2001) rather than in the neonatal phase (only in 1998). The regression results also reinforce the role of race--specifically Black race--in infant mortality, particularly during the neonatal phase. Black race was statistically significant for IMR (in 1999-2001), and in all years for NMR (in 1998-2001), but not for PMR in any year. The relationship between Black race and neonatal mortality is not surprising, as most of the Black-White disparity in infant mortality is due to the significantly higher incidence of very low birthweight (VLBW) among Black infants--three times higher than among White infants--placing these infants at higher risk of NMR. Given that most of the policy effects on infant mortality occurred in 1999, what follows is an in-depth analysis and discussion of the statistical findings for that year.

*Discussion of Regression Findings for 1999*¹²

Regression analysis has demonstrated a statistically significant relationship between Sanction policy and PMR, and Earnings Disregards policy and NMR in the study population in 1999. Statistically significant correlations provide some context for the relationship of these policies to infant mortality. In order to better understand the

¹² Correlation coefficients (r) and significance levels are provided for those not previously provided.

often complex interaction of the demographic, macroeconomic, health care, and policy variables at issue, a graphical representation of the statistically significant relationships found through correlation or regression analysis, and their direction (“+” = direct relationship; “-” = indirect relationship) is presented in Figure 26.

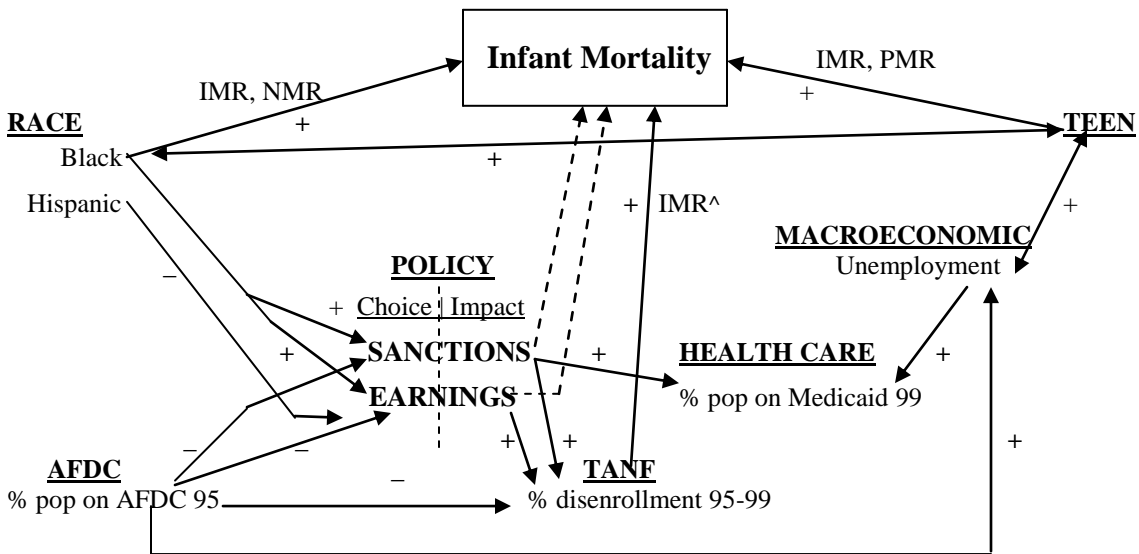


Figure 26. Statistically Significant Relationships Surrounding TANF Work Policies and Infant Mortality, 1999

^Approaches statistical significance, p -value = .051.

Beginning with the two TANF policies, Sanctions and Earnings, the left side represents inputs related to policy choice. States with higher proportions of Black AFDC recipients in 1995 subsequently chose more stringent Sanctions and Earnings policies. Countering this, however, was the indirect relationship of AFDC enrollment and Sanctions and Earnings policies: states with a higher proportion of its population on

AFDC in 1995 subsequently chose more lenient Sanction and Earnings policies. The proportion of Hispanic AFDC recipients was also indirectly related to subsequent Earnings policies. If TANF policy choice were affected by perceptions of or data related to the AFDC program and/or its recipients, it should be noted that the 1996 teen birth rate (15-17 year-olds)--an explicit focus of PRWORA--was *not* related to any subsequent policy choice.

Moving now to the well-established relationships between race and infant mortality, and teen births and infant mortality, the direct impact on infant mortality of race and teen motherhood was reinforced in these findings: there was a direct relationship between the proportion of the study population that was Black and infant mortality (specifically during the neonatal period) as well as a direct relationship between the teen birth rate and infant mortality (specifically during the postneonatal period). As noted earlier, the increased NMR among Black infants results from the disproportionately high VLBW, while the higher PMR among teens is a greater reflection of possible adverse “home environments.” Black race and teen births were also directly related with each other: states with higher Black proportions of their study population had higher teen birth rates; or stated conversely, states with higher teen birth rates had higher Black proportions of their study population.

Teen birth rate was also directly related to unemployment: states with higher teen birth rates had higher unemployment; or conversely, states with higher unemployment had higher teen birth rates. Unemployment in 1999 was also directly related to the AFDC enrollment in 1995 ($r = .516$, $p\text{-value} = .000$), as well as a number of other state macroeconomic and health care access measures for 1995 and 1999 (Medicaid

enrollment⁹⁹ ($r = .467, p\text{-value} = .001$), Uninsured rate⁹⁹ ($r = .543, p\text{-value} = .000$), Poverty rate⁹⁵ ($r = .610, p\text{-value} = .000$), Food Stamps utilization⁹⁵ ($r = .644, p\text{-value} = .000$) given the secular trends in unemployment. Additionally, unemployment in 1999 was directly related to Medicaid disenrollment between 1995-1999 ($r = .412, p\text{-value} = .003$), but *not* TANF disenrollment during those years.

While review of the literature includes research that suggests an unintended disenrollment in Medicaid as a result of disenrollment in TANF, there was no relationship found between Medicaid loss (1995-1999) and TANF loss (1995-1999) in this research ($r = .170, p\text{-value} = .233$). In addition, the scale of the two programs' disenrollment was vastly different: while there was roughly a 46% disenrollment in TANF between 1995-1999, there was only a 2% disenrollment in Medicaid during that time period. Additionally, some research in the literature has found an adverse effect of Medicaid disenrollment on infant health measures: no relationship was found in this research between Medicaid disenrollment and infant mortality in the study population. There *was*, however, a direct relationship between TANF disenrollment and overall infant mortality in the study population (IMR) that approached statistical significance ($r = .275, p\text{-value} = .051$), with results suggesting the relationship was more significant for NMR ($r = .252, p\text{-value} = .074$) than for PMR ($r = .212, p\text{-value} = .135$). Finally, TANF disenrollment was also indirectly related to 1995 AFDC enrollment: states with higher proportions of their population enrolled in AFDC in 1995 had *lower* rates of disenrollment from TANF between 1995-1999 (measured as percent reduction from 1995 AFDC enrollment) ($r = -.379, p\text{-value} = .006$).

Medicaid and TANF disenrollment are relationships on the right side--or impact side--of the two TANF policies, Sanctions and Earnings. TANF disenrollment between 1995-1999 was directly related to both Sanctions ($r = .490$, p -value = .000) and Earnings policies ($r = .348$, p -value = .012): states with more stringent Sanctions or Earnings policies had higher TANF disenrollment rates between 1995-1999. Sanctions were also indirectly related to the 1999 state Medicaid enrollment (measured as a percentage of the state population) ($r = -.296$, p -value = .035), but *not* Medicaid disenrollment between 1995-1999 ($r = .191$, p -value = .179): states with more stringent Sanction policies had lower Medicaid enrollment in 1999.

Though not correlated with each other, Sanctions and Earnings policies are both directly related to infant mortality as determined by correlations and regression analysis. The question is whether the relationship between each policy and infant mortality is an independent relationship, or whether it is mediated through known relationships with Black race, TANF disenrollment, Medicaid coverage, or other unmeasured variable. A correlation table is provided for the relevant relationships (see Table 36).

Prior to analysis of underlying mechanisms, the relationship between the two TANF policies and infant mortality is explored. States were grouped by TANF policy category, and the unweighted average determined for each category. The proportion of states in each category that exceeded the infant mortality for the study population as a whole (all policy categories together) were calculated. The first data are for IMR by Sanction category (see Table 37 and Figure 27). That state infant mortality rises in the study population with increasing stringency of Sanction policy can clearly be seen. Continuing with neonatal mortality (NMR) by Sanction category (see Table 38 and

Table 36

Correlation Table: Sanctions and Earnings Disregards Policies in Relationship to Selected Variables, 1999

1999	Sanctions	Earnings
Race of 1995 AFDC Recip	.306 <i>p</i> = .029*	.329 <i>p</i> = .018*
Race of 1999 Study Pop	.282 <i>p</i> = .045*	.292 <i>p</i> = .038*
TANF Loss (95-99)	.490 <i>p</i> = .000**	.348 <i>p</i> = .012*
Medicaid Enroll (1999)	-.296 <i>p</i> = .035*	-.025 <i>p</i> = .839
IMR	.470 <i>p</i> = .001**	.411 <i>p</i> = .003**
NMR	.351 <i>p</i> = .012*	.393 <i>p</i> = .004**
PMR	.461 <i>p</i> = .001**	.277 <i>p</i> = .049*

* Significant at the 0.05 level (2-tailed).
 ** Significant at the 0.01 level (2-tailed).

Table 37

IMR by Sanctions Category, 1999

LENIENT			INTERMEDIATE		
State	1999 IMR	> Avg	State	1999 IMR	> Avg
AR	9.90	*	AK	7.03	
CA	5.96		AZ	7.46	
DC	12.77	*	CO	7.94	
HI	7.75		CT	8.46	
ME	6.16		IL	12.19	*
MN	9.53	*	IN	11.60	*
MO	12.77	*	KY	11.31	*
NH	5.92		NC	12.37	*
NY	8.24		OR	8.20	
RI	6.29		TX	6.77	
VT	6.29		WV	9.47	*
WA	6.99				
<i>Unwt Avg</i>	8.21	33%	<i>Unwt Avg</i>	8.57	45%

RESTRICTIVE			STRINGENT		
State	1999 IMR	> Avg	State	1999 IMR	> Avg
IA	10.14	*	AL	13.79	*
MD	12.49	*	DE	15.40	*
MA	6.76		FL	10.28	*
MI	11.92	*	GA	11.51	*
MT	11.72	*	ID	11.21	*
NJ	7.26		KS	9.30	
NM	8.48		LA	12.86	*
ND	15.95	*	MS	13.50	*
PA	10.78	*	NE	10.62	*
SD	8.19		NV	8.32	
TN	10.35	*	OH	11.33	*
UT	6.34		OK	11.80	*
			SC	11.60	*
			VA	10.33	*
			WI	11.91	*
			WY	8.60	
<i>Unwt Avg</i>	10.03	58%	<i>Unwt Avg</i>	11.5	81%

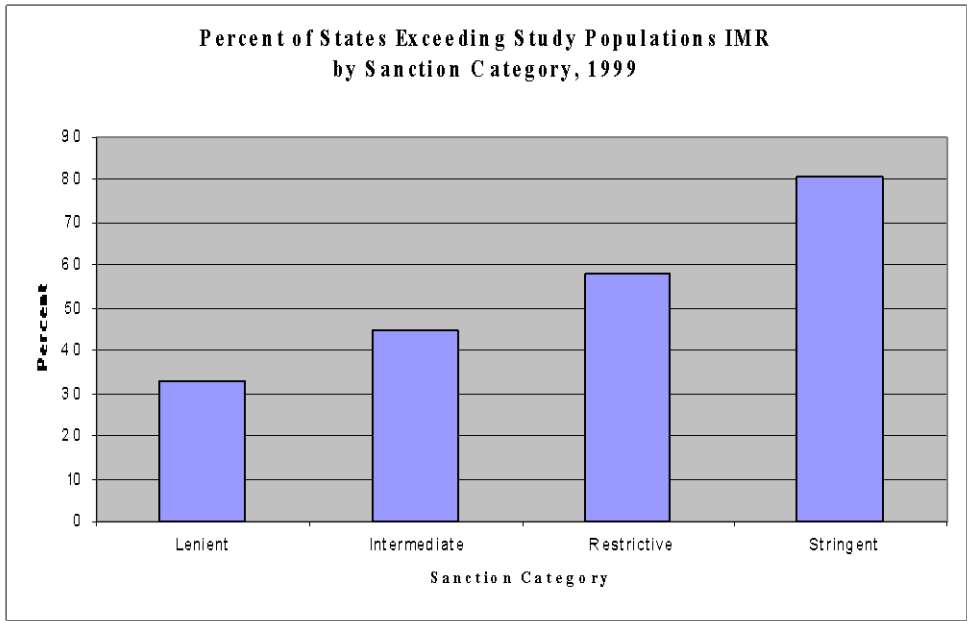
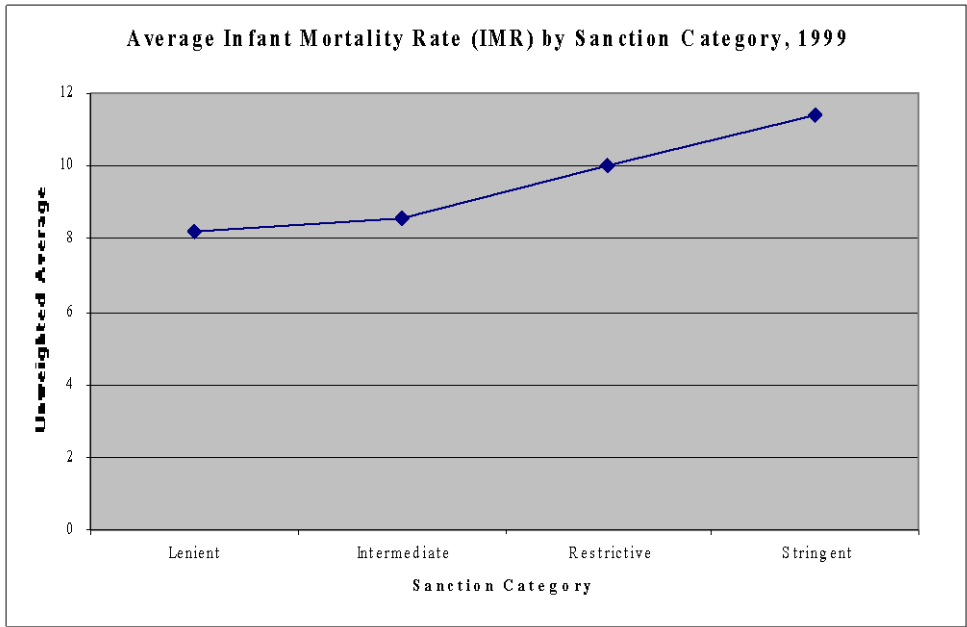


Figure 27. IMR by Sanctions Category

Table 38

NMR by Sanctions Category, 1999

LENIENT			INTERMEDIATE		
State	1999 NMR	> Avg	State	1999 NMR	> Avg
<i>Unwt Avg</i>	4.91	33%	<i>Unwt Avg</i>	5.37	55%

RESTRICTIVE			STRINGENT		
State	1999 NMR	> Avg	State	1999 NMR	> Avg
<i>Unwt Avg</i>	5.76	67%	<i>Unwt Avg</i>	6.45	81%

Figure 28). Finally, the postneonatal mortality (PMR) by Sanction category (see Table 39 and Figure 29). All measures of infant mortality demonstrate the same pattern of higher state infant mortality in the study population associated with greater stringency of Sanction policy.

Similar processes were conducted for Earnings Disregards policy. The first data are for IMR by Earnings Disregards category (see Table 40 and Figure 30). Continuing with neonatal mortality (NMR) by Earnings Disregards category (see Table 41 and Figure 31). Finally, the postneonatal mortality (PMR) by Earnings Disregards category (see Table 42 and Figure 32).

As can be seen from the graphics, the Earnings Disregards categories, though similarly correlated with infant mortality measures as Sanctions policy, do not follow the same progressive, linear pattern as do Sanctions categories. When, however, the Lenient or Intermediate category is excluded, or the two categories were combined, the unweighted averages would follow a more progressive, linear pattern.

Clearly a relationship exists between the stringency of Sanction and Earnings policies and infant mortality in 1999. This is confirmed with correlations and with

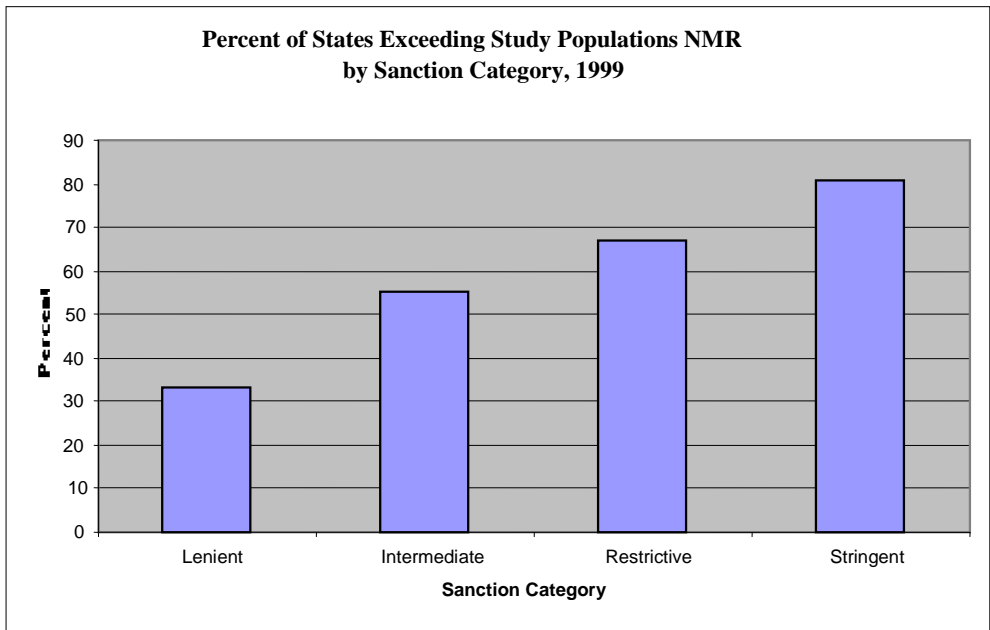
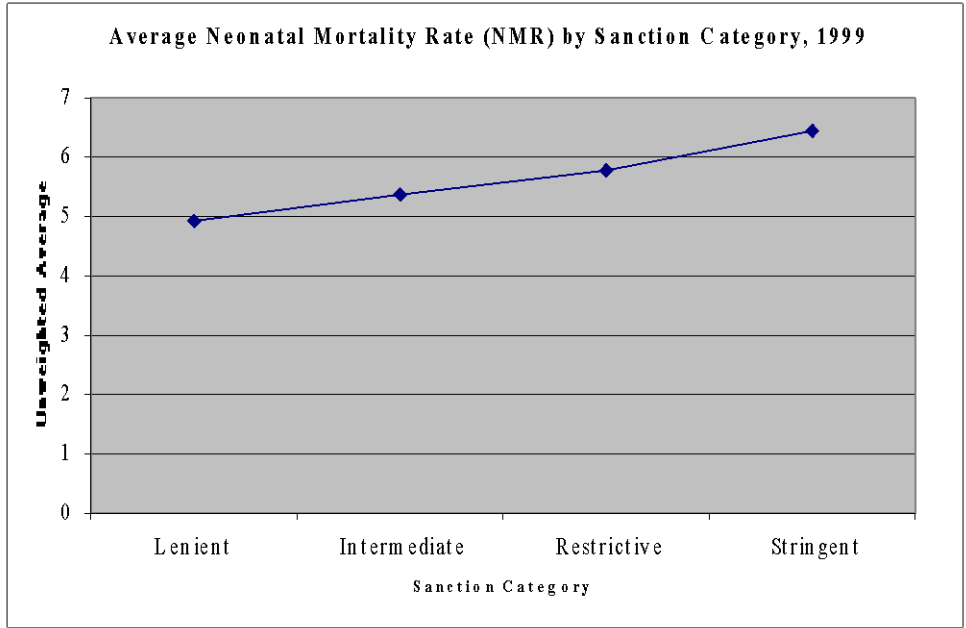


Figure 28. NMR by Sanctions

Table 39

PMR by Sanctions Category, 1999

LENIENT		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	3.28	25%

INTERMEDIATE		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	3.98	45%

RESTRICTIVE		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	4.54	67%

STRINGENT		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	4.94	88%

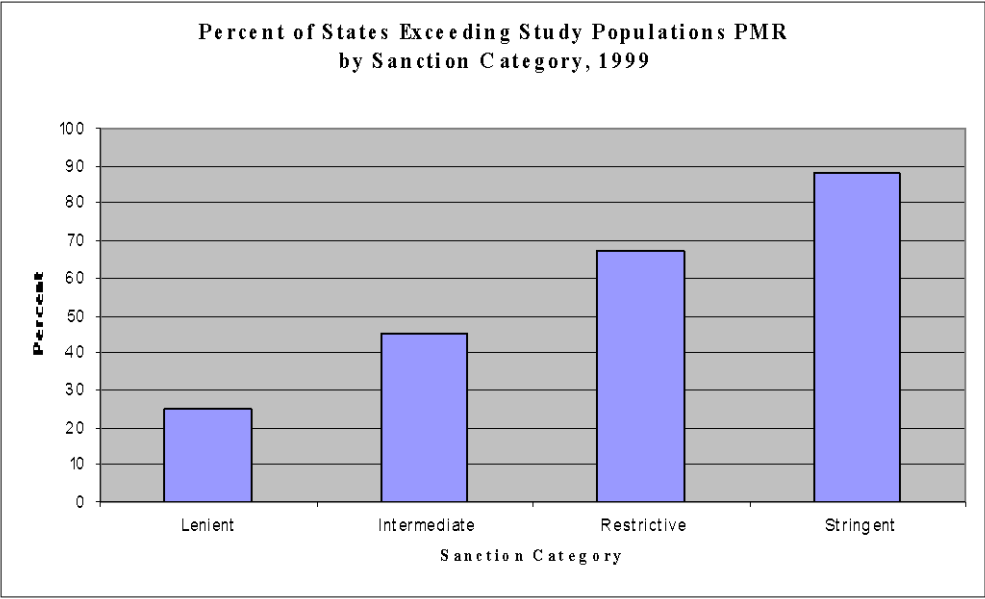
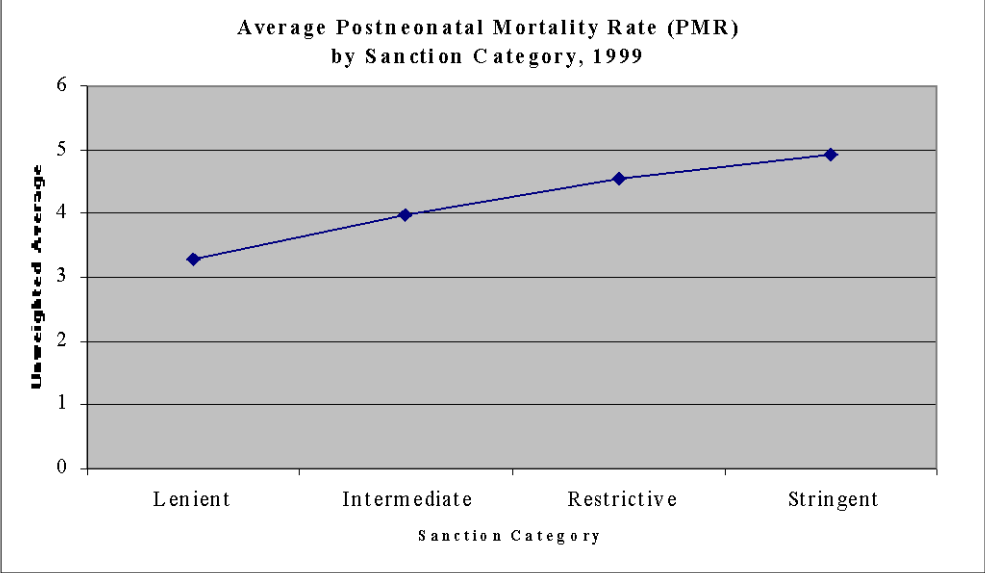


Figure 29. PMR by Sanctions

Table 40

IMR by Earnings Disregards Category, 1999

LENIENT		
State	1999 IMR	> Avg
CA	5.96	
CT	8.46	
FL	10.28	*
HI	7.75	
IL	12.19	*
IA	10.14	*
NM	8.48	
OH	11.33	*
RI	6.29	
<i>Unwt Avg</i>	8.99	44%

INTERMEDIATE		
State	1999 IMR	> Avg
DC	12.77	
KS	9.30	
ME	6.16	
MA	6.76	
NH	5.92	*
NJ	7.26	*
NY	8.24	*
OK	11.80	*
OR	8.20	
PA	10.78	
UT	6.34	
WA	6.99	
<i>Unwt Avg</i>	8.38	36%

RESTRICTIVE		
State	1999 IMR	> Avg
AK	7.03	
AZ	7.46	
ID	11.21	*
LA	12.86	*
MD	12.49	*
MI	11.92	*
MN	9.53	*
MT	11.72	*
NV	8.32	
ND	15.95	*
SD	8.19	
VT	6.29	
WV	9.47	*
WY	8.60	
<i>Unwt Avg</i>	10.07	57%

STRINGENT		
State	1999 IMR	> Avg
AL	13.79	*
AR	9.90	*
CO	7.94	
DE	15.40	*
GA	11.51	*
IN	11.60	*
KY	11.31	*
MS	13.50	*
MO	12.77	*
NE	10.62	*
NC	12.37	*
SC	11.60	*
TN	10.35	*
TX	6.77	
VA	10.33	*
WI	11.91	*
<i>Unwt Avg</i>	11.35	88%

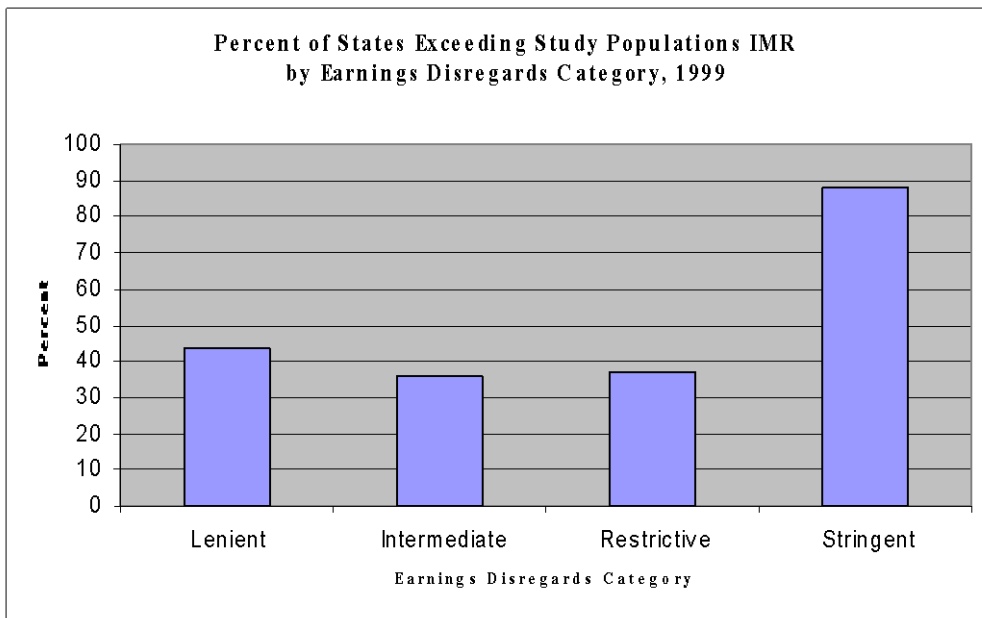
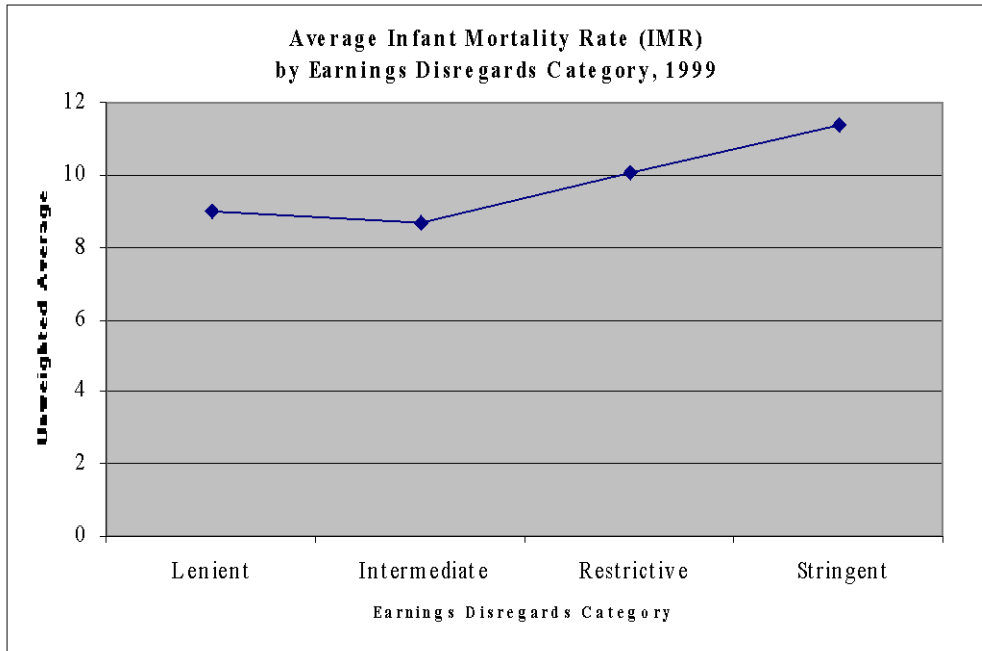


Figure 30. IMR by Earnings Disregards Category

Table 41

NMR by Earnings Disregards Category, 1999

LENIENT		
State	1999 NMR	> Avg
<i>Unwt Avg</i>	5.03	67%

INTERMEDIATE		
State	1999 NMR	> Avg
<i>Unwt Avg</i>	4.86	50%

RESTRICTIVE		
State	1999 NMR	> Avg
<i>Unwt Avg</i>	5.65	57%

STRINGENT		
State	1999 NMR	> Avg
<i>Unwt Avg</i>	6.58	88%

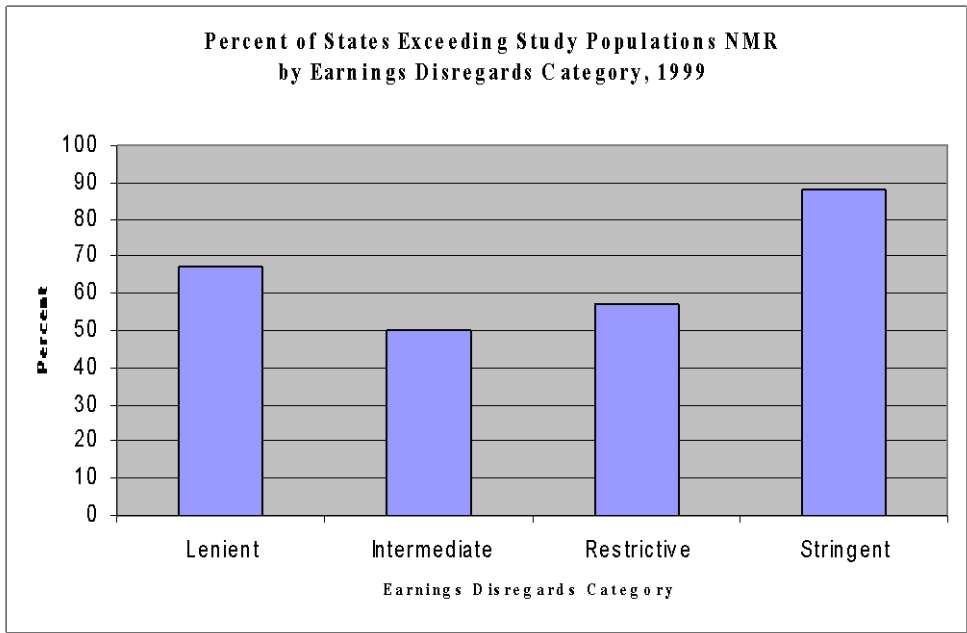
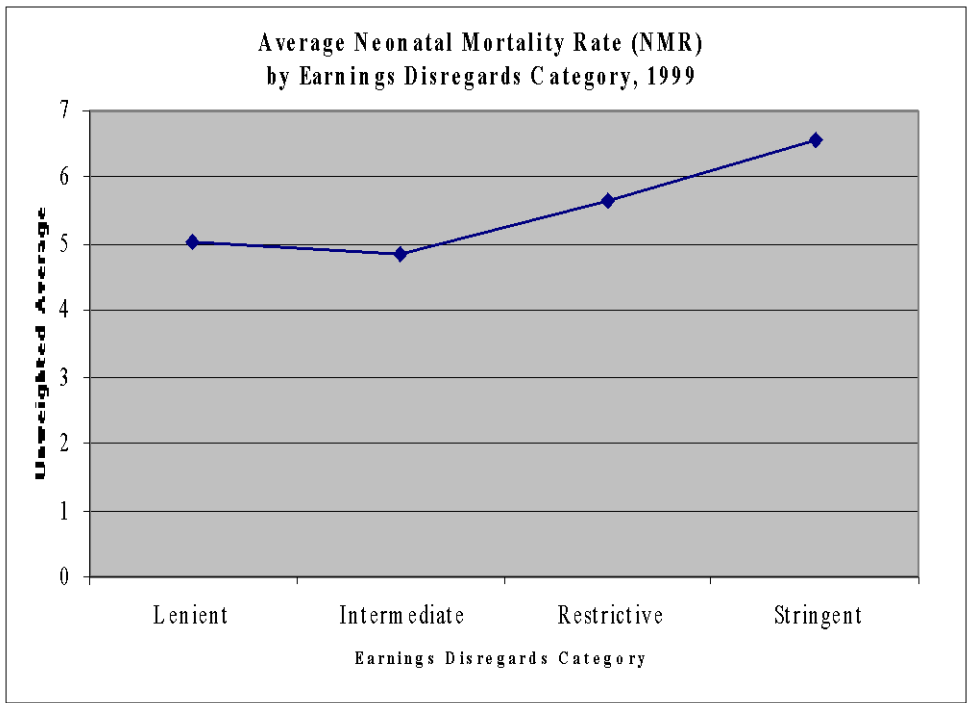


Figure 31. NMR by Earnings Disregard Category

Table 42

PMR by Earnings Disregards Category, 1999

LENIENT		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	3.97	17%

INTERMEDIATE		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	3.51	33%

RESTRICTIVE		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	4.42	50%

STRINGENT		
State	1999 PMR	> Avg
<i>Unwt Avg</i>	4.75	81%

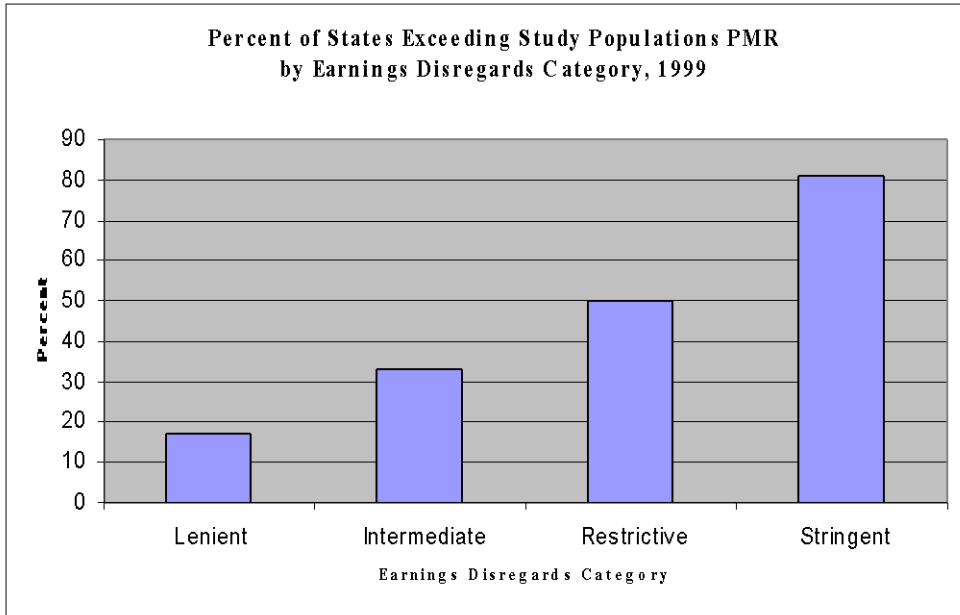
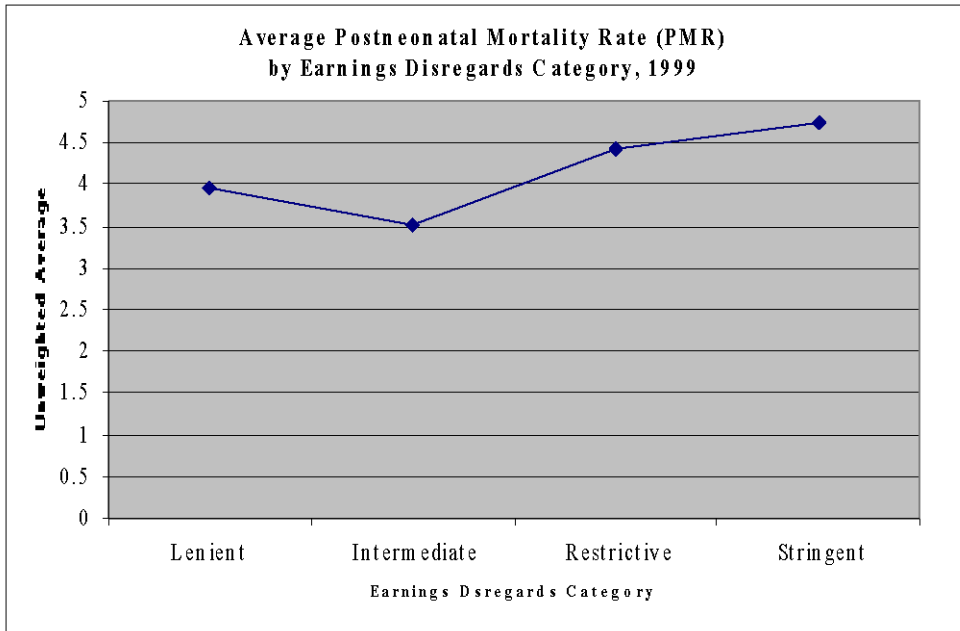


Figure 32. PMR by Earning Disregards Category

regression analysis: states with more stringent Sanctions policies had higher postneonatal mortality (PMR) ($\beta = .702$, p -value = .027) in the study population in 1999, when Earnings Disregards and Sanctions waiver status were not considered in the analysis. When the Earnings Disregards policy was included and the Sanction waiver status was factored into the linear regression model, the statistical significance of the Sanctions policy in the 1999 PMR increased ($\beta = .869$, p -value = .005) and the policy was now statistically significant for IMR ($\beta = 1.304$, p -value = .012). The Earnings Disregards policy was also statistically significant for IMR in the study population in 1999 ($\beta = 1.331$, p -value = .006), specifically during the neonatal period ($\beta = .977$, p -value = .012). It remains unclear, however, whether the relationship between each policy and infant mortality is an independent relationship, or whether it is mediated through any of the known relationships with race, TANF disenrollment, Medicaid coverage, or other unmeasured variable.

Cross-sectional regression analysis was again conducted for 1999. The initial TANF work policies (Exemption, Hours, Sanctions) were included, as were Earnings Disregards policy and Sanction Waiver status. First, regression analyses were performed controlling for race in one of two ways: (a) with race as originally defined--the percentage of births to Black mothers in the study population in a state; then, (b) with race defined as the proportion of AFDC recipients in a state that were Black in 1995. Next, the same regression analysis was performed with *no* control for race. Then, regression analysis was performed with the original race control variable, but factoring in the percentage of TANF disenrollment that occurred in a state between 1995-1999, and

then including state Medicaid coverage (as a percentage of state population) for 1999 instead of TANF disenrollment. The results are presented in tables 43-45.

With the exception of the analysis conducted without a control for race, there was very little difference in the regression coefficients (β) or coefficient of determinations (R^2) in the various analyses. When no control was included for race, the R^2 decreased for IMR and NMR, and to a lesser extent PMR, meaning less variance had been explained. Race, therefore, clearly plays a role in the relationship between TANF work policies and infant mortality. In what manner and to what extent race is involved cannot be determined in this research. TANF disenrollment, though correlated with infant mortality and both Sanctions and Earnings Disregards policies, did not account for any additional variance; nor did the inclusion of state Medicaid enrollment as a variable.

Table 43

Regression Model Coefficients: Sanctions and Earnings Disregards Policies with IMR, 1999

1999	IMR		
	Sanctions	Earnings Disregards	R-squared
Race of 1999 Study Pop (SP)	1.304 <i>p</i> = .012*	1.331 <i>p</i> = .006**	.707
Race of 1995 AFDC Recip	1.288 <i>p</i> = .012*	1.280 <i>p</i> = .007**	.713
No Race	1.695 <i>p</i> = .002**	1.283 <i>p</i> = .012*	.652
Race (SP) + TANF Loss	1.398 <i>p</i> = .010**	1.438 <i>p</i> = .005**	.710
Race (SP) + Medicaid Pop	1.313 <i>p</i> = .015*	1.332 <i>p</i> = .006**	.707

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 44

Regression Model Coefficients: Sanctions and Earnings Disregards Policies with NMR, 1999

1999	NMR		
	Sanctions	Earnings Disregards	R-squared
Race of 1999 Study Pop (SP)	.397 <i>p</i> = .340	.977 <i>p</i> = .012*	.514
Race of 1995 AFDC Recip	.407 <i>p</i> = .328	.942 <i>p</i> = .016*	.513
No Race	.776 <i>p</i> = .069	1.055 <i>p</i> = .012*	.425
Race (SP) + TANF Loss	.371 <i>p</i> = .398	.946 <i>p</i> = .024*	.515
Race (SP) + Medicaid Pop	.388 <i>p</i> = .369	.977 <i>p</i> = .014*	.514

*Significant at the 0.05 level (2-tailed).

**Significant at the 0.01 level (2-tailed).

Table 45

Regression Model Coefficients: Sanctions and Earnings Disregards Policies with PMR, 1999

1999	PMR		
	Sanctions	Earnings Disregards	R-squared
Race of 1999 Study Pop (SP)	.869 <i>p</i> = .005*	.170 <i>p</i> = .549	.650
Race of 1995 AFDC Recip	.855 <i>p</i> = .006*	.154 <i>p</i> = .585	.655
No Race	1.008 <i>p</i> = .001*	.155 <i>p</i> = .594	.625
Race (SP) + TANF Loss	.986 <i>p</i> = .002*	.298 <i>p</i> = .311	.668
Race (SP) + Medicaid Pop	.938 <i>p</i> = .003*	.162 <i>p</i> = .567	.659

*Significant at the 0.01 level (2-tailed).

CHAPTER V

CONCLUSION

Regression analysis analyzing the relationship between TANF work policies (Exemption, Hours, and Sanctions) and infant mortality, while controlling for state macroeconomic conditions, race, baseline infant mortality, and teen birth, demonstrated a statistically significant relationship between Sanction policy and PMR in 1999: States with more stringent Sanctions policies had higher postneonatal mortality (PMR) in the study population in 1999, but for no other year or measure of infant mortality. Neither Exemption nor Hours policies were statistically significant in any year for any measure of infant mortality.

When Earnings Disregards policy and Sanction waiver status were factored into the linear regression model, Sanction policy increased in its significance with 1999 PMR and was now statistically significant for IMR. In addition, Exemption policy became statistically significant for NMR in 2001, such that states with more stringent Exemption policies had higher neonatal mortality (NMR) in the study population in 2001. The Earnings Disregards policy was statistically significant for IMR in 1998, 1999, and 2001, specifically during the neonatal period in 1999 and 2001: States with more stringent Earnings Disregards policies had higher overall infant mortality in the study population in 1998, 1999, and 2001, specifically during the neonatal period in 1999 and 2001.

The 1999 Sanction and Earnings Disregards policies have statistically significant relationships with Black race, TANF disenrollment, and Medicaid coverage, but further

regression analysis indicated only Black race appeared to account for additional variance in the regression model. It remains unclear, therefore, whether the relationship between these policies and infant mortality are independent relationships, or whether--and to what extent--they are mediated through any of the known relationships (with race, TANF disenrollment, Medicaid coverage) or some other as yet unmeasured variable. Given the array of statistically significant relationships, and the complexities and inter-relationships surrounding both TANF policy and infant mortality, it seems likely that multiple factors are involved. Statistically significant correlations between the Sanctions and Earnings Disregards policies and a variety of contemporaneous variables, as well as significant correlations between the stringency of subsequent policy adoption with pre-existing state demographics and macroeconomic conditions, suggest the possibility of state “policy environments” and conditions that are more or less supportive of maternal and infant health, at least among this low SES population. Further analysis regarding causal mechanisms and intervening variables in these relationships is necessary for a better understanding of the role of TANF in infant health, but such analysis is beyond the scope of this research.

In addition to questions about causal mechanisms underlying the relationship between both Sanction and Earnings Disregards policies and infant mortality in the study population in 1999, is the question of why the policy effects were concentrated in 1999, but were limited or nonexistent in the other years. Because TANF programs were implemented in states between September 30, 1996 and January 1, 1998, the year 1999 represents the first year in which all states had had their TANF programs in place for at least 1 year. With the newness of the programmatic changes, the most dramatic program

effects occurred soon after TANF implementation. Consider the case of TANF disenrollment: of the 59% reduction in U.S. welfare caseloads between 1993 and 2000, nearly 40% of the decline occurred between January 1998 and June 2000. It stands to reason that any potential adverse effects of TANF policies would more likely occur during times of greatest program effect. Additionally, there are some anecdotal reports of states being “over-enthusiastic” in their early enforcement efforts of TANF’s new policies. High sanction rates in some states--including Tennessee--were challenged by advocacy groups, ultimately resulting in the establishment of procedural safeguards. These safeguards, however, were not in place immediately after TANF implementation.

While this research provides a comprehensive review of PRWORA and an important description of state infant mortality among infants of unmarried women with less than a high school education in the years immediately following its implementation, there are limitations in the interpretation of these research findings. These results are susceptible to both over-interpretation and under-interpretation. Concerns regarding over-interpretation are rooted first in the need to approximate the TANF population as well as the cross-sectional design employed. First, the study population was the *most like* the TANF population that could be defined within the limits of the Linked Birth data--but it was not the TANF population; caution must be taken to not attribute these results to the TANF population. These findings apply to infants of unmarried women with less than a high school education, which is not necessarily the same as the TANF population. Second, the cross-sectional design allows for describing relationships at a point in time, but does *not* assign causality. This analysis, therefore, describes a situation in which

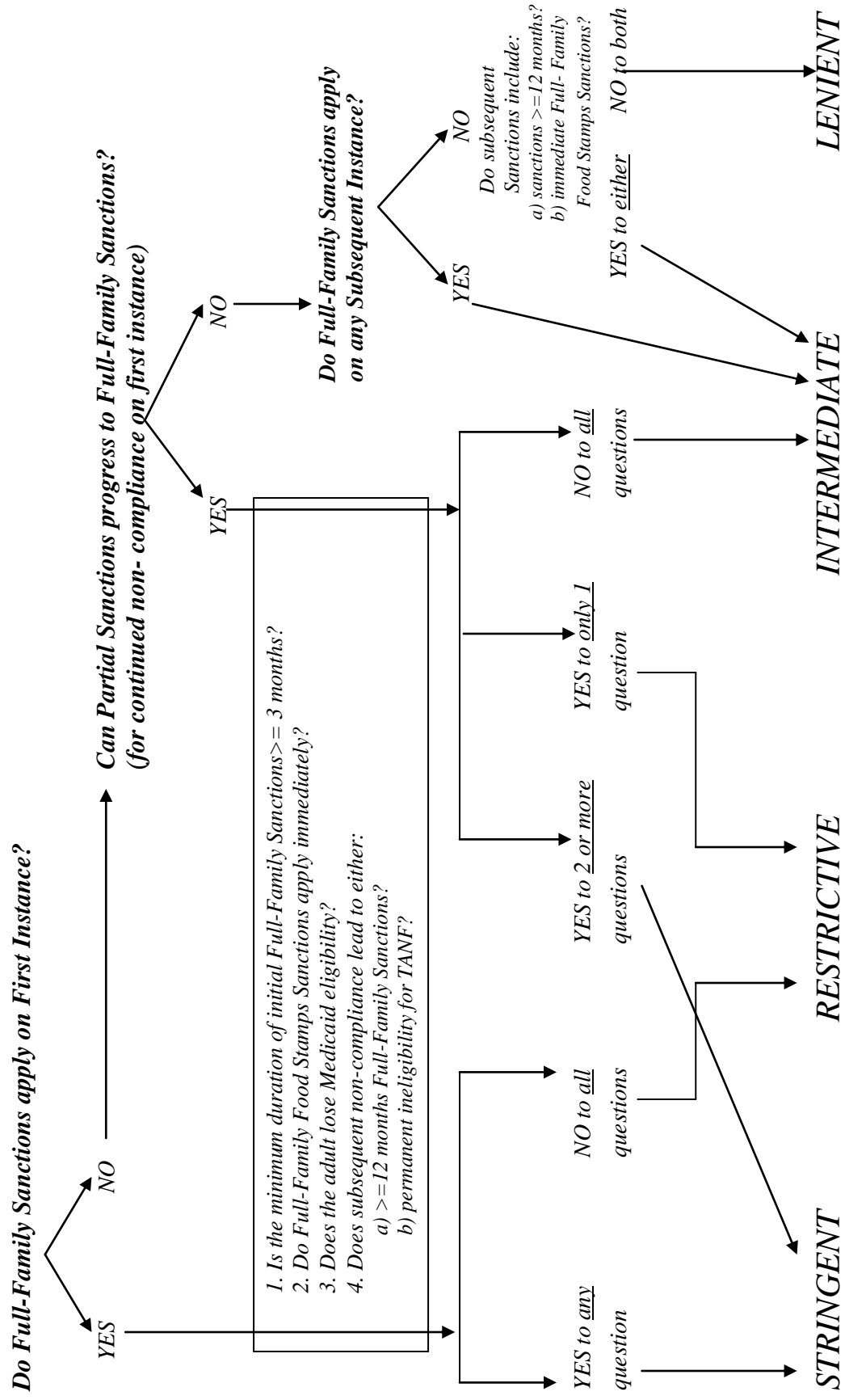
states with more stringent TANF policies have higher infant mortality among the study population, but does not assert that TANF caused the increase in infant mortality.

These findings are also subject to under-interpretation both because of the utilization of infant mortality as a dependent variable, and because of its ecological design. First, there are many adverse health consequences that could be experienced by infants that are not captured by infant mortality rates. Any increase in sickness or health care utilization, or decrease in health-promoting behaviors--all measures short of infant mortality--would not be captured by this dependent variable. Therefore, there could be adverse health effects for infants that were not found with this analysis. Also, as an ecological study, variations in the population are not accounted for. Increased infant mortality, for example, that is concentrated in one demographic group or geographic area would not be found.

Despite these limitations in interpretation, this analysis has found statistically significant increases in infant mortality in the study population associated with the stringency of Sanction and Earnings Disregards policies. It provides a foundation on which subsequent research can build. Further research that looks more deeply at causation, or isolates the TANF population, or utilizes an infant health measure short of mortality, could flow from this research.

APPENDIX A

DETERMINING SANCTION CATEGORY



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