The Complexities of Family Health: Effects on Women's Employment

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The Complexities of Family Health: Effects on Women's Employment

Abstract
An extensive sociological literature links women's health, their children's health, and their disproportionate designation as unpaid caregivers to variation in women's labor supply and earnings. However, there is a dearth of research that simultaneously considers the health of multiple family members to explore how the distribution of chronic conditions within and across families may relate to women's work. Using data from the 2007 Panel Study of Income Dynamics (and its supplemental surveys, the Child Development Supplement and the Transition into Adulthood Study), this dissertation conceptualizes health as a family-level construct and explores how the distribution of chronic conditions in families relates to women's employment, hours, and earnings, with particular attention to disparities by women's educational attainment. I note substantial variation in the distribution of illness across families, and find that the relationship between health and women's employment is complex, with relationships that are diagnostically specific, vary by employment outcome, and stratified by women's characteristics, with particular impacts for women who are nonwhite, less educated, or who have more illnesses in their families. This research emphasizes the importance of multidimensional examinations of health, and the utility in considering the broader family context in which women's employment outcomes unfold.

Keywords
employment outcomes, intra-family health, labor supply, stratification, women's employment, Sociology

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THE COMPLEXITIES OF FAMILY HEALTH: EFFECTS ON WOMEN'S EMPLOYMENT OUTCOMES

BY

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DISSERTATION

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in Partial Fulfillment of the Requirements for the Degree of

Doctor of Philosophy
in Sociology

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THE COMPLEXITIES OF FAMILY HEALTH: EFFECTS ON WOMEN'S EMPLOYMENT OUTCOMES

BY

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DEDICATION

To Kevin, the best part of every day.
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ABSTRACT

THE COMPLEXITIES OF FAMILY HEALTH: EFFECTS ON WOMEN’S EMPLOYMENT

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Jessica A. Carson
University of New Hampshire, December, 2014

An extensive sociological literature links women’s health, their children’s health, and their disproportionate designation as unpaid caregivers to variation in women’s labor supply and earnings. However, there is a dearth of research that simultaneously considers the health of multiple family members to explore how the distribution of chronic conditions within and across families may relate to women’s work. Using data from the 2007 Panel Study of Income Dynamics (and its supplemental surveys, the Child Development Supplement and the Transition into Adulthood Study), this dissertation conceptualizes health as a family-level construct and explores how the distribution of chronic conditions in families relates to women’s employment, hours, and earnings, with particular attention to disparities by women’s educational attainment. I note substantial variation in the distribution of illness across families, and find that the relationship between health and women’s employment is complex, with relationships that are diagnostically specific, vary by employment outcome, and stratified by women’s characteristics, with particular impacts for women who are nonwhite, less educated, or who have more illnesses in their families. This research emphasizes the importance of multidimensional examinations of health, and the utility in considering the broader family context in which women’s employment outcomes unfold.
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INTRODUCTION

In response to the rapid growth in women’s labor force participation in the 1970s and 1980s, scholarly work exploring the links between women’s employment and health burgeoned. Early research focused heavily on the possibility that women’s participation in the workforce would exert deleterious effects on their health, a suspicion that was unsupported by an array of research on both mental and physical health (Aneshensel 1986; Baruch, Biener, and Barnet 1987; Gove and Geerken 1977; Kessler and McRae 1982; Repetti, Matthews, and Waldron 1989; Spitze 1988). Researchers soon identified a reflexive relationship between work and health, with Ross and Mirowsky (1995) noting that, “full-time employment improves health and health bolsters the odds of full-time employment” (241).

With new attention to the possibility that health might drive employment outcomes, research in the 1980s and 1990s explored how the physical health of individual family members—particularly children—impacted the characteristics of women’s labor force participation (see Roberts 1999 for an overview of this work). By the mid-1990s, explorations of the interaction between child health and maternal work were situated in a welfare reform-era context that scrutinized both low-income mothering and work (Chaudray 2004; Collins and Mayer 2010; Hays 1996). Emergent research framed poor health among welfare recipients or their children as one of many potential barriers that could prevent women from fulfilling the strict work requirements of modern welfare policy (Bloom, Loprest, and Zedlewski 2011; Burtless 1997; Danziger et al. 2002; Hershey and Pavetti 1997; Weidman, White, and Swartz 1988). This literature connected the concepts of health, work, and socioeconomic status, but

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1 Of course there is a plethora of research that also examines women’s employment as a factor in children’s health, particularly as related to breastfeeding, immunization, and obesity (e.g., Anderson, Butcher, and Levine 2003; Baker and Milligan 2008; Berger, Hill, and Waldfogel 2005; Brown et al. 2010; Ruhm 2008). This literature is useful for considering the mechanisms of health and work, but its review is beyond the scope of this paper.
in the tradition of earlier research, was largely relegated to documenting effects within the maternal-child dyad, and overlooked the role of spouses' or other family members' health, and by extension, the fuller impacts of an entire family’s health.

The importance of the family unit as a context for individuals' illnesses had not been neglected in public health and medical literature (Bomar 1990; Litman 1971; Richardson 1945; Schwenk and Hughes 1983), but little research had yet focused on health and illness at the family level. Post-welfare reform in the mid-1990s, ethnographic research began unpacking the challenges of new welfare policy; in this work, Linda M. Burton and colleagues elevated the issue of health experiences “inside” welfare recipients' families (Burton, Lein, and Kolak 2005; Burton and Whitfield 2003; Burton and Whitfield 2006). Burton and colleagues concluded that policy discourse would benefit from an improved understanding of “the role that family health plays in the economic security of low-income families” (Burton et al. 2005:494). In linking the health of entire families to their economic prospects, this work exposed the dynamic and influential nature of family health status; however, the methodological limitations of ethnographic data precluded its broader application.

Despite Burton’s call for a conceptualization of health at the family level (Burton et al. 2005), existing research has not systematically documented patterns of chronic illness within families, including the prevalence of multiple chronic conditions or of specific diagnoses within families. Such research has the potential to be both epidemiologically useful and contribute to the broader health disparities literature. In addition, the extensive documentation of individuals’ health as a barrier to employment among welfare recipients indicates that the concept of family health might be applied similarly. That is, that the patterns of health conditions within and across members of a family (patterns that might be considered “constellations of illness”) may
be underutilized as potentially explanatory of employment outcomes among women, and among low-income women in particular. As existing research tends to explore the impacts of individual-level health—among women or their children or, to a lesser degree, their spouses—on individual-level work outcomes, the current framework neglects to consider the potentially additive effects of multiple health conditions within a given family unit.

By nature of its (low-income) participants, the research linking employment and family health among welfare recipients gives rise to the possibility that constellations of illnesses may have stratified effects on employment. For higher-income families, who may have resources that can be flexibly applied to any family member or illness (e.g., health insurance coverage), the effects of multiple family health conditions on women’s employment may be buffered (or, women’s employment may be less necessary). But for lower-income families with fewer resources, these constellations of illnesses may compound the disadvantages that women already face in the labor market. As Danziger et al. (2002) note of welfare recipients:

*one or two barriers may have little effect on employment, but multiple barriers might seriously impede employment…For example, mental health and physical health problems might require frequent doctor visits, leading to absences from work. One of these problems alone might not interfere with work, but in combination with low education and few job skills, they could create obstacles on the job or in job search (17).*

Following this logic, it is possible to envision families in which multiple barriers might be health-related, creating compounded obstacles in addition to the disadvantages of low education or unstable work histories. Thus, this doctoral dissertation attempts to ameliorate these conceptual gaps, exploring how health at the family level is linked to women’s paid work across the spectrum of social class. I will first document family health status—the prevalence and distribution of illness within the families of working-age women—with attention to differences that might emerge by socioeconomic status. Second, I will explore the utility of
family health status in predicting women’s labor market supply and outcomes, particularly among women whose low resource levels might exacerbate their susceptibility to unstable patterns of work. Finally, hypothesizing that low-income women may experience both differential exposure and vulnerability to the issues of poor family health, I will attempt to identify resources that might attenuate the effects of poor family health on women’s employment, with an eye toward identifying material supports relevant in a policy context (e.g., health insurance or liquid assets).

In Chapter I, I examine the literature around the interdependencies of health status at the family-level, the possible links between health and women’s labor supply, and the implications of these relationships for women of different social classes. Chapter II describes the data, sample, measures, and analytic approach to answering these research questions. Chapter III provides an overview of the distribution of illness within and across families, including chronic condition counts and the prevalence of specific diagnoses. Chapter IV presents results related to women’s labor supply, and Chapter V, those related to women’s labor market outcomes in the form of annual earnings. Chapter VI discusses the results from the previous two chapters, contextualizing them in the larger body of research identified in Chapter I, and presents conclusions and policy implications of this work, identifies its limitations, and provides suggestions for future research.
1. REVIEW OF THE LITERATURE

Clusters of Illness within Families

In considering how constellations of illness might impact women's work outcomes, it is necessary to first review the literature around patterns of health within families. The idea of exploring health among families is not new, and research from the public health and medical fields has considered the family an important context for individual health outcomes for more than half a century (for a review of this literature, see Schwenk and Hughes 1983; see also Bauman and Grace 1974; Bomar 1990; Curry 1974; Litman 1971; Litman 1974; Marinker 1976; Richardson 1945). With some rare exceptions (e.g., Dingle, Badger, and Jordan 1964), early work around health in families centered on family as “a collection of individuals” (Schwenk and Hughes 1983:1) who provide the context for the diagnosis and treatment of a single ill member within, rather than as a discrete epidemiological unit or an organizational structure for exploring patterns of illness.

In the social sciences, there has been plentiful research on the ways that health clusters within families. A large body of work has demonstrated associations between the health statuses of spouses, which "overwhelmingly suggests evidence for concordant mental and physical health, as well as health behaviors among couples" (Meyler, Stimpson, and Peek 2007:2297). For example, Bookwala and Schulz (1996) reported that individuals’ well-being and depression scores are predictive of well-being in their spouses, a finding that has been corroborated by research both before and since (Coyne et al. 1987; Fletcher 2009; Robinson, Rodgers, and Butterworth 2008; Widmer, Cadoret, and North 1980). Wilson (2002) found strong inter-spousal correlations in chronic diseases, functional limitations, activity restrictions, and self-rated health that persist after accounting for sociodemographic determinants of health.
Finally, Monden (2007) demonstrated that individuals whose partners are in poor health are three times more likely to report poor health themselves when compared to those with healthy partners.

Alongside the literature investigating similar health status between spouses, a large body of research describes the generally-better health among married people, and how these differences arise, whether “because marriage has beneficial effects on health (marriage protection effects) and/or because healthier individuals are more likely to marry and to stay married (marriage selection effects)” (Waldron, Hughes, and Brooks 1996:113). For example, Waldron et al. (1996) found marital selection effects among women who were not employed. Joung et al. (1998) found evidence of a selection effect in terms of marital disruption, wherein married persons with poor health were more likely to become divorced. Conversely, Lillard and Panis (1996) found that among men, selection operated in both directions, where both those in poor health and those with high levels of health-encouraging behavior were more likely to marry or remarry. Whether the effects of marriage are protective or selective, inter-spousal similarities in health status may be expected.

Aside from spousal correlations, research has also documented intergenerational links in health. Cohen (1999) found a heightened incidence of depressive symptoms and psychological distress among parents and siblings of children with chronic conditions. Widmer et al. (1980) noted that the children of patients diagnosed with depression reported increases in pain and anxiety similar to those experienced by the patients themselves, only to fall back to control levels after the patient had been diagnosed and treated. Finally, Kendler, Davis, and Kessler (1997) described significant clustering of major depression, generalized anxiety disorder,
antisocial personality disorder, alcohol abuse/dependence, and drug abuse/dependence between grown children and their parents.

Alongside documentation of intra-family correlations in health, there is no shortage of research exploring the mechanisms by which the family context may influence individual health, particularly via shared environmental risks and behavioral patterns within families. The justification for this focus is well summarized by Wilson (2002), who explained,

except for those who live alone, the food we eat, the air we breathe, the recreational activities we perform, the neighborhood we live in, and the type of medical care we receive are all influenced by relationships within the household. Since all of these factors contribute to health, it makes eminent sense to model health production as occurring in a social context, in which the family is a central feature (1158).

Similarly, Ferrer et al. (2005) cited the mechanisms of a shared social and behavioral environment, common genetic risk factors, and income/asset characteristics as potential drivers of these findings.

Despite evidence that health clusters in families (see also Ross, Mirowsky, and Goldsteen 1990), prevalence assessments of illness still tend to eschew the family as a unit of analysis, focusing instead on clusters of illness in dyadic pairs (for example, between spouses, or parent and child). The scant research that does estimate constellations of illness at the family level is not representative; rather, it is based on small convenience samples of welfare recipients or families receiving certain types of health care (see Witt and DeLeire 2009), or extrapolated from broadly-focused ethnographic samples (Burton and Bromell 2010; Burton et al. 2005; Burton and Whitfield 2006; Burton and Whitfield 2003). Further, while some of this work purports to measure illness at the family level, it employs exclusionary definitions of “family,” which only consider the dyad of child and primary caregiver (ibid.).
Clusters of Illness within Families: Emergence of Specific Diagnoses

With reason to suspect that broad configurations of “illness” may congregate in families, it is worth dedicating additional attention to the co-occurring emergence of specific diagnoses in families. There is a vast, interdisciplinary literature focusing on multiple illnesses within individuals (generally termed “comorbidity” or “multimorbidity”), most of which track the prevalence and risk for various comorbid conditions among specialized populations of individuals.² This literature is relevant, for example, to diagnostic efforts and patient care in the clinical sector, to disability payments and workers’ compensation in policy research, and to labor supply in economics research (e.g., Anderson 2010; Boyd et al. 2010a, 2010b, 2010c, 2010d; Hakola et al. 2011; McAlpine and Warner 2002; Merikangas et al. 2007; Ornstein et al. 2013; Waghorn et al. 2008; Ward and Schiller 2013; Ward, Schiller, and Goodman 2014).

Assessments of co-occurring illness at the family-level are much less common, though not non-existent. For example, much of this literature orients one individual with a specific diagnosis as central, and seeks to record instances of similar diagnoses among various relatives; this research appears particularly concentrated in the areas of psychiatric and substance use disorders (e.g., Carter et al. 2005; Fendrich, Warner, and Weissman 1990; Hammen, Shih, and Brennan 2004; Kendler et al. 1997; Merikangas 1990; Weissman 1990). This literature is seemingly aimed at helping individuals better understand familial patterns of risk for certain psychiatric and substance use disorders. In other cases, the family is treated even less like a unit of analysis, and more as the context in which the consequences of a central individual’s own co-

² Throughout this paper, I use the more general term “co-occurring” illnesses, choosing this term for its flexibility compared with “comorbid” or “multimorbid.” In its classical definition, “comorbid” implies centrality of one disease (i.e., the “index disease”), with other diagnoses emerging as auxiliary. “Multimorbid” makes no assumption about disease centrality, but refers only to multiple illnesses within a single person. Here, I use co-occurring to refer to illnesses that emerge in tandem at the individual or family levels. For a more nuanced discussion of these distinctions, see Valderas et al. (2009).
occurring illnesses unfold. For example, Muslow (2007) notes that the high rates of psychiatric disorders among alcoholics can produce particularly stressful situations for families, which in turn reduces the degree of support available to the alcoholic in the treatment process.

Though few research efforts attempt to systematically assess co-occurring illnesses within families, there are several reasons to expect that chronic conditions may not be randomly distributed within and between families. Litman (1974) classifies the specific relationships between family members' health into two categories, noting that “the physical and mental health of family members may be related either directly, such as through the transmission of infectious or hereditary diseases, or indirectly, as when the physical or mental condition of one member affects some aspect of the family as an effective unit and alters the health state of the other members” (499). Applying Litman’s terminology here, I suggest that the “direct transmission” of modern chronic conditions within families is less likely characterized by the transmission of infectious illnesses or hereditary risk alone, and better described as the family’s role in the intricate interplay of genetic and environmental factors that influence the emergence of illnesses like depression, diabetes, asthma, hypertension, and cancer (Centers for Disease Control and Prevention 2014a; Cross-Disorder Group of the Psychiatric Genomics Consortium 2013; Dunevant 2008; Hofker, Fu, and Wijmenga 2014; Levinson and Nichols 2014; World Health Organization 2005). Indeed, the intricacy in the connections between health and family, which include “the socialization of health attitudes, values, knowledge and beliefs, family decision-making in health and health care, and the role of the family in health and illness behavior” (Litman 1974: 497) are arguably all “direct” influences of the family.
Further, it is possible that assessments of family-level comorbidities may vary from those known to occur at the individual level. For instance, research has consistently documented that hypertension is the most common chronic condition among U.S. adults (Anderson 2010; Elixhauser et al. 1997; Gallup-Healthways 2012; Ornstein et al. 2013; Partnership for Solutions 2004b; Ward et al. 2014). Unsurprisingly, hypertension is also a common co-occurring condition, often present in concert with other illnesses. For example, Ward and Schiller (2013) identified “arthritis and hypertension” as the most common diagnostic dyad among adults with two or more conditions; second most common was “diabetes and hypertension,” among all adult men, and all women over age 44 (“arthritis and asthma” was the second most common pairing among women aged 18-44). In fact, among those with at least two chronic illnesses, the authors found that hypertension was mentioned in four of the five most common dyads among men, and three of the five most common dyads among women (Ward and Schiller 2013).

Though hypertension has clearly been established as a condition that commonly co-occurs with others, it is unclear how hypertension might couple with other diagnoses at the family level. For example, it is unclear why hypertension and arthritis are particularly likely to appear in tandem: does this association still exist when examined not within, but across, family members? Given ongoing focus on the role that interactions between biology and environment play in understanding illness (Agrid et al. 1999; Hicks et al. 2004; Plunkett and Gordon 1960; Reich, Cloninger, and Guze 1975; Sidora-Arcoleo et al. 2012), documenting how co-occurring illnesses translate from the individual to family level of analysis is an area ripe for examination. Beyond the utility in mapping patterns of family illness, research also suggests that co-occurring conditions have serious implications for individuals’ labor supply (e.g., Hakola et al. 2011;
McAlpine and Warner 2002; Merikangas et al. 2007; Scuteri et al. 2011; Waghorn et al. 2008), discussed below.

In sum, existing research on co-occurring illnesses largely positions one family member as central, with little work focused squarely on the family itself as a unit of analysis. As a result, there is a paucity of research providing family-level estimates of specific chronic conditions and their co-occurrence across family members and still less that adopts a descriptive approach that avoids framing an individual person or a specific condition as most central. Such an approach could widen the lens with which co-occurring illnesses have been viewed by documenting the prevalence of specific illnesses or diagnostic dyads at the family level, lending detail to an increasing body of knowledge on complex health processes (e.g., the roles that genetics, environment, culture, and health practices play in shaping health outcomes), and informing a host of stakeholders on potential patterns of health risks among families.

**Clusters of Illness within Families: Implications for Low-Income Families**

Just as specific illnesses are unlikely to emerge at random within families, the distribution of across families is likely to also be patterned. In particular, the clustering of illness within families cannot be fully considered without attention to the role of social class. One of the most well documented associations in the health literature is the link between health and socioeconomic status (e.g., Link and Phelan 1995). Research shows that low-income adults are especially likely to be affected by poor health (Phelan, Link, and Tehranifar 2010), demonstrating higher rates of cardiovascular, psychiatric, infectious, and respiratory diseases, as well as cancer, diabetes, hypertension, and injury than their higher-income counterparts (Burton et al. 2005; Burton and Brommell 2010; Syme and Berkman 1976). Various mechanisms for the heightened levels of illness among low income individuals have been proposed, including the higher rates of
expression of proinflammatory phenotypes among low income groups that could account for higher incidence of respiratory and cardiovascular illnesses among these populations (Hänsel et al. 2010; Miller and Chen 2007; Miller and Chen 2010; Miller et al. 2009). Not exempt from the effects of social class, low-income children also demonstrate elevated rates of chronic illness and disability (Wise et al. 2002; Smith et al. 2002), with some estimates suggesting that one in three poor children face poor health (Currie 2008). Perhaps most troublesome, Currie (2008) notes that while poor children are equally likely to be diagnosed with asthma, they are more than three times as likely as their non-poor counterparts to experience limitations as a result of their condition.

Given this health-SES gradient, it is possible that low-income groups have a heightened prevalence of illnesses inside their families, whether manifesting as multiple members each diagnosed with a single condition, a single member with multiple illnesses, or multiple members afflicted with multiple conditions. Indeed, Bombard et al. (2012) found that 28 percent of their sample of low-income women had “three or more chronic diseases and/or risk factors” (60), compared with just 14 percent of higher-income counterparts. Monden (2007) found that 43 percent of couples in which both partners had “less than good” health were concentrated in the lowest income quartile, garnering the conclusion that "there is considerable accumulation of adverse characteristics in households and this leads to a steeper social gradient in health at the household level than at the individual level" (405).

Further, because low-income families may be limited in their access to resources for preventing and coping with illness (e.g., preventive care or health insurance coverage), such clustering may have particularly detrimental outcomes for families who are already strained by the responsibilities of making ends meet. For example, research suggests that the medical
expenses related to treating special health care needs can be burdensome, consuming 3 percent of annual income among low-income families whose child has a special need (Parish, Shattuck, and Rose 2009) and causing “financial problems” for nearly 12 percent of privately insured families with special needs children (Busch and Barry 2009). Meyers, Lukemeyer, and Smeeding (1998) note of disabilities: "when poverty and disabilities intersect, the public and private costs associated with children’s care have important policy implications" (209), but certainly a family need not be poor, nor a child necessarily disabled, for this to be true. Indeed, expanding these conceptualizations to include broader notions of family and of illness elucidates the volume of responsibilities facing those who manage their families’ health and health care needs. Whether missing work to supervise a sick child or providing emotional support for a struggling spouse, the literature has highlighted the ways in which poor family health can have impacts that are both concentrated and diffuse in their reach (e.g., Hogan 2012).

In sum, while the health literature regularly employs the family construct, existing research leaves space for several contributions. First, a timely, more general assessment of the prevalence of family illness is in order. By focusing on very specific illnesses among narrowly-defined dyads of family members, existing literature risks underestimating the dynamism of family health. With a focus on health among children or mothers or spouses, it is unclear to what degree the unequal distribution of poor health is exacerbated by clustering in families sharing a residence. Just as epidemiological knowledge around how illness is patterned among individuals aids in assessing access and targeting resources, research on clusters of illness in families can be similarly useful in identifying and ameliorating risk. In particular, clustering of poor health within families might explain variation in individual and family functioning in a variety
of contexts (e.g., labor, educational, or civic); if social class further stratifies these clusters, the explanatory power may be heightened.

It is in this context that I pose my first research question: given the vast body of literature documenting clusters of illness among family members and the well-established link between health and socioeconomic status, how are constellations of illness patterned at the family level, in general and at varying levels of socioeconomic status?

Family Health and Work: Labor Supply and the Case of Welfare Recipients

Given the above discussion, it is clear that individuals' illnesses may be associated with other family members' health. Though lacking family-level estimates of prevalence, existing research has firmly established that illness clusters in families, and good efforts have been put forth in quantifying the mechanisms of this patterning. However, considerably less research explores how this pooling of poor health within families might extend beyond reciprocal health relationships to impact family members' non-health outcomes. This section will detail the ways that individuals' health has been linked to a specific non-health outcome—employment—and pose the possibility that family-level health status could make an explanatory contribution in this area.

In order to understand how family health might impact women's work, it is necessary to first understand the modern conditions of employment with attention to critical divisions by social class. First: over the last several decades, the American labor market has undergone dramatic changes, with substantial implications for women in particular. With the large-scale entry of women into the labor force, a growing service sector, the rise of contingent and nonstandard scheduling, a shift to outsourcing, and welfare reform (Chaudray 2004; Collins and
Mayer 2010; Kalleberg 2009; Morris and Coley 2004; Price and Burgard 2006), modern labor market qualities have produced new opportunities for conflicts to arise between the demands of work and family. Though 74 percent of women aged 25-54 were employed in 2013 (Bureau of Labor Statistics 2014), research shows that women are still largely responsible for the tasks of family reproduction (e.g., Bianchi et al. 2012; Hochschild 1989) and shifts in the structure of paid work have rapidly outpaced those in unpaid work. As such, many women are faced with a substantial mismatch between the demands of finding and retaining reliable, well-paid work and ably performing their roles as wives and mothers.

The structural mismatch between work and family is perhaps most demonstrable in low-income families, where women may work inflexible, low-paying jobs, with no benefits and high turnover rates (e.g., Collins and Mayer 2010; Smith and Tessaro 2009) and conflicting responsibilities threaten to consume already-low levels of resources (Chaudray 2004; Morris and Coley 2004). With little social, economic, and cultural capital available to buffer against the unexpected, women in these families may face a choice between being responsive to immediate family needs and maintaining the employment necessary to support their families.

Perhaps because of the expectation that poor health poses a heightened ability to disrupt low-income women’s work (or perhaps due to the methodological advantage of an accessible sample), much of the family health and employment research draws upon samples of welfare recipients. Research suggests that mental and physical illness affect between 10 and 30 percent of welfare recipients (Burtless 1997; Bloom et al. 2011), a rate twice that in the general population (Danziger et al. 2002). Children of welfare recipients also suffer from chronic illness and disability at heightened rates when compared to the children of non-recipients (Meyers et al. 1998; Smith et al. 2002; Weidman et al. 2008; Wise et al. 2002). Further, welfare recipients
cite health issues as an important barrier to commencing or retaining work (Abraham 1993; Heymann and Earle 1999; Smith et al. 2002), and Hershey and Pavetti (1997) found that between 5 and 13 percent of job losses among welfare recipients can be traced to a family health problem.

Because the majority of welfare recipients are women, much of the research around barriers to employment has specifically positioned women’s employment as the outcome. Though nonrandom samples preclude generalizability in much of this literature, the emphasis on women’s work is not necessarily problematic in that women still face the historically and culturally embedded responsibility for the bulk of the informal work in the home (e.g., Bianchi et al. 2012; Geist and Cohen 2011; Hochschild 1989). In terms of health-related care specifically, Litman (1974) notes:

*Perhaps the most persistent theme running through our three generation study was the rather pervasive role played by the wife-mother in the health and health care of the family. For whatever the measure used, illnesses incurred, medical and health services used…the wife-mother remained the central agent of cure and care within the family complex (505).*

Forty years later, women still report higher incidences of missed work to care for a sick child than their male counterparts, a disparity that has remained incredibly stable over time (Álvarez 2002; Carpenter 1980; Smith and Schaefer 2012).

In conjunction with the broader factors influencing women’s attachment to the labor force (e.g., the gender wage gap, a lack of institutionalized paid family leave), it is reasonable to expect that both the effects of poor family health and the risk of resultant employment disruptions fall disproportionately on women. Thus, I intend to retain the focus on women’s

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3 The sex distribution of adult welfare recipients has remained fairly steady over time: men were 12 percent of adult AFDC recipients in fiscal year 1995 [Tables 22 and 25 in Office of Family Assistance (1995)] and 15 percent of adult TANF recipients in 2011 [the most recent data available; see Table 18 in Office of Family Assistance (2013)].
employment for this project, though the relationship between family health and men’s employment undoubtedly poses a distinctly interesting set of questions too.

**Family Health and Work: The Various Linking Mechanisms**

Research connecting family health status to women’s employment falls into several broad areas, each of which focuses on a different aspect of a large and complex causal model. First, I will review the literature linking women’s own health to their labor market outcomes, including the role that specific diagnoses (and co-occurring diagnoses) play in shaping labor supply and earnings. I then review research on the effects of children’s (and other family members’) health on women’s work. I will then draw upon a few aspects of the broader informal caregiving literature, focusing on its direct and indirect effects on employment, before identifying areas in which research could be bolstered, and noting the practical contributions that such research might make.

**Workers’ Own Health**

It is both intuitive and well established that women with health conditions face barriers in employment. Indeed, Urban and Olson (2005) and Brandon (2007) find that women’s own disabilities substantially reduce their likelihood of employment, while Blank (1989), Wolfe and Hill (1995), and Ross and Mirowsky (1995) echo these findings for women with activity limitations and poor self-rated health. Puntenney’s (1999) qualitative work supports these findings among poor women specifically, and notes that chronically ill respondents described easy access to medications and avoidance of symptom-triggering chemicals as reasons for avoiding the formal workplace. The identification of chemical exposure on the job as a barrier to work among the ill is particularly useful when considering that low-income women may be disproportionately located in jobs that require such contact (e.g., cleaning and food service).
In addition to documenting the effects of general health status on employment, there is a substantial body of research examining the effects of specific health conditions on labor supply and labor market outcomes (summarized in the Appendix as Table 17). For instance, Alexandre and French (2001) found that depression reduces the probability of being employed from an average of 43 percent to 24 percent, and that conditional on employment, depressed individuals worked an average of 8 fewer weeks annually than their non-depressed counterparts. Ettner, Frank, and Kessler (1997) found that psychiatric diagnoses reduced the probability of employment by 11 percentage points among men and women, with a reduction in conditional hours for men. Mitchell and Burkhauser (1990) note substantial differences in women’s usual hours worked by arthritis diagnosis, finding that the diagnosis accounted for about one-third of the differential in women’s hours. Finally, Mitchell (1991) found that the odds of dropping out of the labor force by age 55 are significantly higher among men with arthritis than their non-arthritic counterparts (OR = 0.88 versus 0.11, respectively).

In terms of earnings, Ng, Jacobs and Johnson (2001) found that a diabetes diagnosis was related to one-third reduction in earnings, ranging from $3,700 to $8,700 annually. Ettner et al. (1997) concur, finding that a psychiatric diagnosis has significant impacts on conditional earnings, ranging from an 18 percent reduction (about $3,500) for women and a 13 percent reduction (about $4,500) among men (see also Marcotte and Wilcox-Gök 2001 for a summary of research on mental illness and earnings). Pincus, Mitchell, and Burkhauser (1989) found a substantially larger earnings loss among arthritics, with women and men earning 30 and 63 percent as much as their non-arthritic counterparts; however, the authors also find that less than one third of the earnings losses were attributable to arthritis specifically, with education, age, and comorbidity playing much larger explanatory roles. Mitchell and Burkhauser (1990)
found within-sex variation in the impact of arthritis, as women aged 18-44 with arthritis had wages about 15 percent lower than their same-aged, arthritis-free counterparts, while women aged 45-64 had wage values less than half of their non-arthritic counterparts. However, in decomposing the differentials in wages and hours worked between women with and without arthritis, the authors found that arthritis accounts for a quarter of the wage differential for women aged 18-44, but more than a third of that for women aged 45-64, similar to the explanatory power noted by Pincus et al. (1989).

Bartel and Taubman (1979) examine the impacts of various categories of illness on earnings, stratified by recency of diagnosis. They find that a recent (past five years) diagnosis of “psychoses/neuroses” reduces log earnings by 27 percent, while even a diagnosis 20 years ago still results in a 14 percent decrease in earnings. Smaller, though still significant, effects for arthritis and respiratory ailments (bronchitis, asthma, and emphysema) also emerged. Further, while change in labor supply accounted for 22 percent of the earnings losses among people with arthritis, it accounted for nearly twice as much of the earnings effect for those with psychoses/neuroses and respiratory conditions. In short, the research on the relationship between specific conditions and employment outcomes identifies some clear associations, though it is still unclear how these diseases might function in tandem across different family members or interact with levels of resources (e.g., federal disability payments or health insurance).

A subset of this work explores the effects of conditions co-occurring at the individual-level (e.g., classic “comorbidities”) on labor supply. For example, Waghorn et al. (2008) found a negative relationship between comorbid physical health conditions and employment status. Ng et al. (2001) found that “complicating conditions” among diabetics (largely diabetes-specific, but
also including hypertension, vision problems, and heart disease) decreased the probability of labor force participation by 12 percent, and increased the predicted number of missed work days in a two-week period by 3.26.

Research also suggests that co-occurring conditions may be especially detrimental to work when conditions are of the psychiatric and physical nature. McAlpine and Warner (2002) found that those with comorbid physical and mental disorders have rates of employment about 20 percent lower than those with only physical ailments. In particular, there is a substantial degree of evidence linking co-occurring depression to poor outcomes. For example, Scuteri et al. (2011) found that hypertension was only associated with functional disability and cognitive impairment in the presence of depression. Kessler and Frank (1997) found that psychiatric disorders were related to work loss, and on average, these effects were four times larger among workers with other co-occurring psychiatric disorders. Specifically, they identify depression and anxiety, and anxiety and substance abuse as co-occurring conditions associated with particularly high mean work loss days (Kessler and Frank 1997). Hakola et al. (2011) found an elevated risk of work disability among asthma-sufferers that was exacerbated by chronic comorbidities, but especially depression, and in their review, McAlpine and Warner (2002) succinctly note, “depression exacerbates poor physical functioning” (18). In contrast, Egede (2004) explored the effects of co-occurring depression and diabetes on lost work days, finding that while a depression diagnosis increased the mean number of days lost, people with only diabetes or who had both depression and diabetes were no more likely to report work loss.

There also exists a more general discussion of the methodological importance of comorbidity in this literature. In estimating work disability, Merikangas et al. (2007) found that “associations of specific conditions with disability decreased substantially after controlling for
comorbidity, suggesting that prior studies, which generally did not control for comorbidity, overestimated disease-specific effects” (1180). This finding echoes results from Alexandre and French (2001) who examine the role of depression in labor supply and find the effects of the illness may be overestimated if not controlling for the role of co-occurring illnesses. In these more general instances, it is possible that in some cases, or for some illnesses, the effects of co-occurring conditions are less driven by the specific features of any given illnesses, and more by the mere burden of their co-occurrence. In this vein, some existing research does examine the potentially additive effects of multiple illnesses on workers, though often treated in categorical terms (e.g., “presence of three or more disorders” as in Ettner et al. (1997)). One notable exception is in Christensen and Kallestrup-Lamb (2012), who test the linear effects of workers’ number of diagnoses, and find that additional diagnoses are associated with decreased odds of retirement among a sample of Danish adults.

In concert with the earlier discussion of co-occurring illnesses in the family, the body of literature linking specific diagnoses to women’s labor outcomes coalesces to demonstrate a major gap. First, because the family has so rarely been treated as the unit of analysis in this type of research, there has been no clear estimation of specific illnesses and their co-occurrence in families. As a result, it is virtually unknown how specific intra-family diagnoses might interact to differentially influence women’s work outcomes. Because different illnesses likely impose different degrees of burden on families, there is reason to suspect that identifying the type of conditions present in a family might lend a more nuanced understanding of the ways that family illness impacts women’s work. Indeed, it is possible that examining a count of chronic conditions may indeed obscure a simpler, underlying reality: that rather than the number of conditions in a family, it may be the presence of a specific condition in a family that is most
impactful on women’s work. In other words, this literature could benefit not only from a thorough examination of the distribution and co-occurrence of specific illnesses in the family (as described in the preceding sections) but also from improved understanding of how different constellations of family illness are associated with women’s labor supply and earnings.

**Family Members’ Health and Women’s Employment**

Perhaps the best-developed area of research linking work to family health status focuses explicitly on labor supply (i.e., participation in and hours worked) among mothers whose children experience health limitations. These “limitations” have been operationalized as struggles with the activities of daily living (Earle and Heymann 2002; Loprest and Davidoff 2004), or the presence of chronic illnesses (Baydar et al. 2007; Smith et al. 2002; Wise et al. 2002), disabilities (Porterfield 2002), behavioral problems (Coley, Ribar, and Vortruba-Drzal 2011) and “poor health” (Corman, Noonan, and Reichman 2005; Kuhlthau and Perrin 2001; Powers 2001). Even amid varied operationalization of mothers’ work (e.g., hours worked or any employment), all of the above studies conclude that children’s illnesses function to reduce mothers’ engagement in the labor force.⁴

Research linking women’s work to illness among family members outside the mother-child dyad is much less common and the research that does exist tends to focus on “any family member” or other similarly unspecified household residents. Blank (1989) found that female heads of household worked fewer hours if they reported living with others who had activity limitations. Roberts (1999) noted that having a single family member with a mental illness did not impact women’s work, but that women decreased their labor force participation if the

⁴ One exception to these findings is Son et al. (2011), who found no effect for children’s physical or mental health problems on mothers’ work. I have chosen to relegate this study to a footnote, given the vague independent variable (mothers were asked if their child had ever had an illness or injury that kept her from work) and the small sample size (N=240).
family member had multiple illnesses. Finally, Álvarez (2002) found that women workers who reported having used free time to care for ill family members also had disproportionate rates of sick leave use; though not a firm conclusion, her findings suggest that working women who have sick families may draw upon their own sick leave (if available) as a sanctioned strategy for meeting care needs.

The Role of Informal Care

Another strand of research focuses explicitly on the informal or unpaid caregiving role as a mediator that links illness in one family member to a broad host of negative outcomes for other family members (see Schulz, Visintainer, and Williamson 1990 for an early meta-review on the subject). The informal caregiving literature is vast, and somewhat inappropriate for answering the driving questions of this paper (to be discussed below), therefore I selectively draw upon this work to explicate how poor family health might stunt informal caregivers’ capacity for work, and how the effects of attending to family illnesses are disproportionately heaped on women’s shoulders.

Research suggests that the informal caregiver role is associated with an increased likelihood of a clinical mental health diagnosis (Ennis and Bunting 2013), fewer psychosocial resources (Robinson et al. 2008; Silver et al. 1995), higher incidences of strain (Witt and DeLeire 2009), stress (George and Gwyther 1986), perceived stigma (Vickers and Parris 2005), and guilt and shame (Hill 2003). Within this literature, a subset focuses on caregiving as a source of role conflict and strain, which in turn lead to worse individual mental health outcomes (Marks 1998). Stewart (2013) indicates that role strain increases linearly with the number of care-related responsibilities reported, and others find support for a conflict specifically between informal caregiving and work roles (Morris and Coley 2004; Wang et al.
2011), a relationship that is exacerbated among female caregivers (Fredrikson 1996; Navaie-Waliser, Spriggs, and Feldman 2002). Altogether, these findings have particular implications for less advantaged groups, as the provision of care is stratified by gender (Lahaie, Earle, and Heymann 2013; Martin 2000), race (Pavalko and Artis 1997) and socioeconomic status (Tennstedt and Chang 1998). Further, other research suggests that the impacts of providing informal or unpaid care vary by social class with low income caregivers reporting heightened levels of care-related distress when compared than their higher-income counterparts (Williams et al. 2003).

A specialized portion of the informal caregiving literature specifically links caregiving to labor market outcomes, usually in terms of women’s labor activity, and thus, is more relevant here.5 Rupp and Ressler (2009) found evidence of a weak negative relationship between caregiving and employment, and Pavalko and Artis (1997) found that employed women reduce their working hours when they begin caregiving, which Van Houtven, Coe, and Skira (2013) quantified at 3-10 hours per week. However, this body of research also indicates that the relationship between caregiving and employment is not uniform, and may vary by caregivers’ characteristics. For example, Lahaie et al. (2013) observed the most dramatic decrease in labor market activity among female and less-educated caregivers. Breslau, Salkever, and Staruch (1982) found that caring for an ill child had no effect on single mothers’ work, but had an interactive effect for married mothers, wherein the negative effect was greater in non-white and low-income families. Corman et al. (2005) reveal contradictory evidence regarding poor child health and employment by marital status, wherein employment effects were significant only

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5 Cohen (1999) notes that the emphasis on outcomes for women in the caregiving literature is influenced by “historic interactions between cultural practice and research methodology: mothers tend to be the main care providers as well as the informant/participant most easily accessible to researchers” (150). One might note a parallel between this reasoning and the focus of welfare-to-work literature on women’s employment, as discussed above.
for single mothers. Finally, Henz (2006) found that low-income caregivers were more likely to leave the labor force. Other research focuses on how impacts vary by the workers’ relationship to the ill person: for instance, Arber and Ginn (1995) found that providing informal care has an increased depressive effect on employment when the care recipient is a child, a less intense effect when the recipient is a spouse, and a larger effect on married women when the recipient is a parent.

It is clear from the literature that combining paid employment and unpaid care produces certain challenges, but the focus on individuals, the highly specific definition of “caregiving,” and the inconsistent findings around family health status and labor market activity highlight a gap in the literature. First, though the informal caregiving literature considers the impacts of caring for spouses and older adults as well as children, there is no evidence that these impacts have been comprehensively assessed at the family-level (that is, among sick spouses and children, if present). Further, the informal caregiving literature generally focuses only on those who provide care over sustained periods [for example, “one month or more,” as in Marks (1998)], and usually refers to those who provide round-the-clock care for one person with a specific illness, such as cancer or dementia, or a disability that impacts the activities of daily living (Lahaie et al. 2013; Lima et al. 2008; Scharlach, Gustavson, and Dal Santo 2007; Van Houtven et al. 2013). Indeed, this type of care is critical to family functioning, but with the focus on the discrete burdens of caring for a single (often terminally ill) person, there is a noticeable absence of research relevant to managing the more mundane and routine elements of health in the family unit.⁶

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⁶ To illustrate how this exemption might be problematic, I offer a hypothetical example concerning a mother of two. The oldest child has asthma; though well controlled with medication, seasonal triggers can result in asthma attacks and leave him susceptible to respiratory illness. As a result, he is too sick for school much more frequently
Of course this type of care has implications beyond those at the level of the individual worker. Discussing a relatively broad construction of caregiving, Álvarez (2002) wrote, "difficulties in combining work and caregiving responsibilities translates [sic] to considerable financial costs to employers and governments. Hence, it is not just an issue for working families, it is an issue affecting the whole community" (2). From a societal perspective, workers' inability to meet work and family needs might result in lost productivity for employers, or in the case of job loss, increased societal costs around unemployment compensation, public health insurance, welfare, or other social safety net programs. Yet it is possible that existing estimates do not fully capture the spectrum and circumstance of those facing conflicts around work and family care. By expanding beyond traditional notions of "caregiving" to include the more routine management of family health, links between conditions inside the family and outcomes for women workers can be clarified. If women's labor market activity is measurably impacted by the health of their family members, it becomes possible to identify families for whom additional resources and supports would be beneficial in the context of meeting "welfare to work" requirements, avoiding spells of job loss, and building family stability.

Amidst the above findings, I pose a second research question: **given the relationship between family illness(es) and women's work, the psychosocial effects of caregiving,** than his peers. Her other child has an anxiety disorder that requires frequent therapeutic and pharmaceutical care, for which she must visit several out-of-town specialists. Though her children require extra supervision, medical care, transportation, and sick days, she is exempt from the “caregiver” definition in that these illnesses lack the permanency and degree of disruption associated with “true” caregiving. Still, if as a result of her children's illnesses, she is unexpectedly missing work or reducing her hours to compensate, her employment trajectory is decidedly impacted by her family's illnesses. And while providing irregular care may impose fewer consistent burdens, the inconsistency of these duties means they are also less routinized, structured, and predictable. This may have implications for low-income women in particular, in that the unpredictable nature of these care-related responsibilities may be incompatible with the inflexibility of low wage jobs (Kalleberg 2009), and women may face work-related consequences for an abrupt change of schedule or an unsanctioned sick day. Chaudray (2004) provides qualitative evidence of this effect through the lens of child care, noting that when child care sites are unable to provide specialized or emergency care to children with special needs, mothers' work is disrupted.
and the stratified vulnerability to caregiving burdens, how does family illness impact women’s labor supply and labor market outcomes, and do these effects vary by social class?

Moderators in the Family Health-Work Relationship

In considering the ways that family health might impact work, a reciprocal question also emerges; that is, if family health impacts women’s work, what might prevent or attenuate this potentially disruptive influence? That the effects of family illness may be varied, complex, and mutable aligns with an “adaptation framework,” neatly summarized by Young’s (1983) description: “even if empirically based research shows illness promoting family system disequilibrium… it is unlikely that illness produces impaired functioning for all families” (Young 1983: 396; emphasis in original). Young proposes an adaptation framework to address this divergence, which affords space to consider the impacts of family illness in a traditional Parsonian sense—that is, illness as dysfunctional to the family system, and by extension, to the broader social systems in which family is embedded—but in a way that does not discount inter-family variation. Young (1983) notes “strong evidence suggests that certain types of families, by virtue of structural or interactional patterns, can handle illness experiences without major disruption” (396). Though mentioning a role for unspecified “resources” in passing, Young’s aim is theoretical, and she does not attempt to apply this framework to identify these methods of “adaptation” herself.

In seeking to identify potential resources, I turn again to the caregiving literature, as its examination of linear relationships might provide space for testing the role of various resources. However, this work reveals little research into factors that might buffer women from the negative effects of providing informal care, and even less effort to identify factors that
could specifically prevent disruption to labor market activity. Limited existing research focuses on the role of personal (i.e., psychosocial) and social resources, and tends to exclude discussion of material resources (see Aneshensel 1999 for more on the role of different resources in ameliorating the effects of stressful events). For example, Dilworth-Anderson, Goodwin, and Williams (2004) and Gaugler et al. (2009) each find that heightened levels of psychosocial resources, like mastery, can protect caregivers from depressive outcomes. Other work shows that self-efficacy (Montoro-Rodriguez and Gallagher-Thompson 2009) and social support (Teixeira and Pereira 2013) alleviate some negative effects of the informal caregiver burden.

The body of literature exploring moderators beyond the psychosocial is small and relatively recent, largely focused on demographics and workplace policies. Corman et al. (2005) found that the link between poor child health and mothers’ work was exacerbated for unmarried, less educated, and older mothers in subgroup analyses, but stopped short of explicitly testing the degree to which education, marriage, and youth were actually protective. In examining work-family and family-work conflict, Stewart (2013) found that a supportive workplace culture reduced both types of conflict, but did not explore how reduced conflict related to workers’ likelihood of retaining their jobs. Finally, perhaps most relevant to this inquiry, Pavalko and Henderson (2006) found that employed female caregivers were more likely to remain employed and retain their regular work hours over a two-year period if flexible workplace policies (e.g., unpaid family leave, paid sick or vacation days, flexible start and stop times) were available. Examining a similar set of policies, Earle and Heymann (2012) found reduced odds that workers reported wage loss due to caregiving for family members when these policies were present, though operationalization of their outcome variable was problematic (discussed below).
With little research on the topic, it is difficult to know whether particular resources might insulate women from the potential labor market impacts of poor family health. A broad body of literature exploring barriers to stable employment emerged in the wake of welfare reform and amid samples of current or former welfare (AFDC or TANF) recipients, identified a complex web of factors that prevent women from retaining work, including low educational attainment, poor health, and having young children (e.g., Bloom et al. 2011). But while this body of work may be informative for speculating on protective factors, potential ameliorative effects have not been an explicit focus of any work reviewed here. In addition, the specialized and nonrandom samples that exclude women with higher levels of resources complicate hypothesizing about functioning of potential protective factors for families across the income spectrum.

The focus on psychosocial resources can be beneficial for understanding mechanisms by which informal caregiving impacts work, but even the most thoughtful policy would struggle to increase these resources. Earle and Heymann’s (2012) study on workplace policy is promising, but the outcome variable was operationalized as a binary response to “Has being a parent or a caregiver ever caused you to lose wages/income because of your caregiving responsibilities?” rather than as an objective measure of altered labor activity. Thus, it is possible that their results were influenced by respondents’ perceptions, wherein respondents might have been less likely to perceive having lost wages if they worked in a place they deemed supportive, and vice versa.

Indeed, the sparse literature in this area leaves space for exploring moderators in the relationship between family health status and work beyond psychosocial resources. For example, perhaps families with liquid assets (e.g., a savings account) are better equipped to
buffer the effects of family illness, able to pay for a good diagnostic test and secure treatment, or purchase childcare that can prevent missed work in the event of unexpected illness complications. Perhaps owning a vehicle matters, so that women can attend work or appointments without having to rely on strictly scheduled public transportation or favors from friends and family. Health insurance coverage, which can ensure proper preventive care before family illness worsens and necessitates intensive caregiving may be another critical moderator.

Another potential moderator in the health-employment relationship is receipt of Supplemental Security Income (SSI, or “disability”) benefits. Because SSI receipt is limited to low income families with disabilities (Social Security Administration 2012), it is unclear how SSI benefits might interact with labor supply. For instance, benefit receipt might have a substitutive effect on income, allowing women to reduce their labor supply accordingly whether because benefits are adequate for meeting needs, or for fear of losing benefits. It is also possible that SSI receipt may be a proxy for illness severity or chronicity, which in turn, may be associated with lower labor supply regardless of SSI, and thus difficult to distinguish from the former. Finally, it is possible that SSI receipt might act as a buffer, serving to provide some flexible income for addressing family illness among those who might otherwise have to reduce their labor supply to meet those needs on their own. In general, empirical tests of these and the above examples might not only provide insight into preventing job loss, but could also illustrate whether these potential buffers might protect the most vulnerable workers.

Of course, given the projections of increasing health care cost burden in an aging society (Polsky and Grande 2009), there is substantial research highlighting the necessity of exploring caregiving in a policy context (Riggs 2003), and in the context of the Affordable Care Act in particular (Ness 2011; Watts and Gaertner 2013). Though the Affordable Care Act arguably
signals the largest health care system transition in American history, there are still some gaps in coverage, available coverage may be inconsistent, and inequality in health care access is far from fully ameliorated. In addition, the modern post-welfare reform context still positions work as the solution to the malaise of “slothful” poverty, without facilitating the systemic changes and individual supports necessary for ensuring that work is possible (see, for example, Collins and Mayer 2010). Taken together, the current policy context cannot possibly provide women with airtight protection from the possibility that their families’ health issues might jeopardize the jobs on which their families depend. While a research contribution certainly cannot provide the level of security that supportive policy can, identifying resources with potential impacts at the family-level provides space for policymakers and practitioners to consider meaningful intervention prior to (or in the absence of) policy change. This approach also has the advantage of acknowledging the dynamism and adaptive capacity of families, rather than orienting families as unidimensional, passive victims of illness (Young 1983).

From this literature emerges my third and final research question: given early evidence that supportive factors might ameliorate the negative effects of caregiving, what resources moderate the hypothesized relationship between poor family health and decreased labor supply and earnings?

Review of Research Questions

In this chapter, I reviewed several key bodies of research that shape the central questions for this dissertation. First, I summarized the literature documenting connections

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7 For example: as has always been the case, when families usually eligible for Medicaid experience an increase in income—say, from a seasonal job or a fluctuation in hours worked—they may find they are inconsistently eligible for health care coverage through Medicaid. Pre-ACA, these people would have become uninsured, but under the new policy will be eligible for subsidies through health insurance exchanges. While perhaps an improvement over being uninsured, this “churning” from Medicaid to subsidized plans can result in inconsistent care and/or gaps in coverage (see Bergal 2014).
between health statuses and outcomes of various family members, generally centered on
correlations between dyads of family members (spouses, or mothers and children in particular),
and describe the established links between health and social class. I next reviewed the literature
connecting health and employment, with particular attention to the literature documenting how
illnesses within the family can lead to informal caregiving responsibilities that affect work. These
effects are particularly salient for women and populations that are traditionally disadvantaged by
social class or other factors. Finally, I explored the scant literature that describes resources
(largely psychosocial) that might act as potential buffers in the relationship between health and
women’s employment. Taken together, existing research provides space for: (1) documentation
of health that treats families as the unit of analysis, exploring the distribution of illness “inside”
families, and across families in different social class locations; (2) an empirical assessment of the
ways that a fuller examination of health at the family level might contribute explanatory power
to current understandings of women’s labor supply (specifically employment and hours worked)
and earnings; and (3) an examination of the ways in which tangible, policy-relevant resources
might act as a buffer in the proposed relationship between family health and women’s
employment. This review of the literature culminated in three specific research questions (listed
below), the first of which is explored in Chapter III, with the second and third questions
assessed in Chapter IV (as related to labor supply) and Chapter V (as related to women’s
earnings).

RQ1. Given the vast body of literature documenting clusters of illness among family members
and the well-established link between health and socioeconomic status, how are constellations of
illness patterned at the family level, in general and at varying levels of socioeconomic status?

RQ2. Given the relationship between family illness(es) and women’s work, the psychosocial
effects of caregiving, and the stratified vulnerability to caregiving burdens, how does family illness
impact women’s labor supply and labor market outcomes, and do these effects vary by social class?
RQ3. Given early evidence that supportive factors might ameliorate the negative effects of caregiving, what resources moderate the hypothesized relationship between poor family health and decreased labor market activity?
II. METHODS

Data

Overview & Structure

The data for this dissertation are drawn from the Panel Study of Income Dynamics (PSID), a nationally representative survey that began in 1968 with 18,000 individuals in 5,000 families, and includes data on employment, family, health, wealth, and more (Panel Study of Income Dynamics 2014). Administered annually from 1968 to 1997, then biennially through the present, the PSID has followed the original sample, their current spouses, and their descendants to amass data on 73,000 individuals in nearly 9,000 families by the 2011 wave. The wide array of topics, however, means that the majority of data are collected only about the “head” and “wife/wife” of a household, with fewer measures regarding other family unit members (OFUMs). To ameliorate some of these gaps, the Child Development Supplement (CDS) was launched in 1997 to capture rich data on the children aged 0-12 in PSID families, with a follow-up wave in 2002 when the children were 5-18, and another in 2007 interviewing children aged 10-18. The Transition to Adulthood (TA) supplement was created in 2005 to follow former CDS children into young adulthood, in the period between aging out of the CDS and forming their own households. In order to create health measures that include data from all possible family unit members (FUMs), I will draw upon data from the CDS, the TA, and the main PSID files, connecting health information within families by via a series of shared family identifiers.

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8 Note that publicly available data are not considered human subjects data, according to the federal definition of a human subject, and thus IRB approval is not required for this research.
9 A wife is the head's legal spouse while a “wife” is an unmarried cohabitor who has been living in the household for more than one year; before the one-year mark, a female cohabitor is termed a “first year cohabitor of the head.” In households where there is no male head (e.g., female headed households, whether unmarried or in same sex partnerships), it is possible to have a female head.
10 Unfortunately, this means that children who were born before 1983 or after 1997 are not part of the CDS sample; data collection processes treat these children as OFUMs in the main PSID only, a limitation that is discussed in greater detail below.
In selecting a data source, I also considered two other nationally representative surveys that include health measures: the Medical Expenditure Panel Survey (MEPS), and the National Health Interview Survey (NHIS) (note that these surveys are related: MEPS households are a subsample of NHIS participants; see “Medical Expenditure Panel Survey: Survey Background” 2014). Each of these surveys provides an adequate sample size, data collected at the household or family level, and sufficient detail on key demographics and employment-related outcome variables. However, in both surveys, measures of health conditions are less ideal for these purposes. First, MEPS asks respondents about a host of prevalent conditions (see “Medical Expenditure Panel Survey” 2014), but its focus on medical expenditures means that diagnosis of more specific health conditions are often only recorded if explicitly reported by the respondent, either directly or in connection with health care utilization connected to that condition (e.g., Soni 2010; 2012). Further, explicit mental health measures in the MEPS are limited to Kessler’s “K-6” psychological distress scale, which is not asked of children at all (Hedden et al. 2012). As a result, estimates of mental health status are limited to extrapolations from reports of care received (e.g., Davis 2014). This type of measure becomes problematic as existing research suggests, “financial barriers often limit the use of preventive care, which then creates higher levels of utilization at later stages” (Leclere, Jensen, and Biddlecom 1994), and person and illness characteristics have distinct influences on shaping utilization of both hospital and physician (e.g., Andersen and Newman 2005; Wright and Perry 2010; Zola 1973). As such, measures of health conditions that are extracted solely from specific instances of care utilization may not be the most suitable measures for assessing the presence of conditions in the family.
The NHIS is another potential data source, with an adequate sample size and representativeness. Regarding health conditions, all family members are asked whether they have any limitation of activity, and if yes, are asked to specify what condition causes that limitation [National Health Interview Survey (NHIS) Description 2014]. Later, one “sample” adult and child in each family are explicitly asked about an inclusive list of health conditions (ibid.). While essential for those interested specifically in the effect of limitations, the presence of a condition may matter beyond whether it limits activities; that is, a condition might be well-managed, thus imposing no limitations to daily activity; however, the efforts required to limit a condition’s effects may not be negligible. In addition, unlike in the PSID, there is no measure of severity of the limitation, which could be useful in additional future analyses. Finally, Andreski, McGonagle, and Schoeni (2009) demonstrate a near-uniform higher rate of missingness on NHIS measures when compared with PSID measures. Taken together, the MEPS and NHIS appear to be good sources for a variety of health-related illnesses—especially with the availability of health care access and cost in MEPS—though I find the PSID more suitable for these purposes.

In order to use the PSID for these analyses, it is critical to identify a common reference period among all FUMs, so that family-level health measures indicate conditions actually documented in the same period. While TA and main PSID respondents are surveyed every two years (since 2005 and 1968, respectively), the CDS was collected in 1997, 2002, and, most recently, in 2007. Thus, while the 2007 wave is not the most current PSID wave available, it is the only year for which health data from the multiple surveys can be reliably aligned.

Alongside data from the 2007 surveys, I take advantage of the PSID’s longitudinal nature to draw in measures from the 2005 and 2009 survey waves. For example, identical survey
questions asked in 2005 help to reduce missing data for statuses that are stable over time (e.g., race). Employment measures collected in 2009, referencing 2007 and 2008, have the distinct advantage of documenting employment status for the entire calendar year, rather than simply until the interview date, and provide potential to expand this analysis beyond the cross-sectional (more detail on these measures below). Thus, I exploit these 2005-2009 measures to construct rigorously matched reference periods in the data, aligning measures from various surveys so that health measures refer specifically to diagnoses by 2007, and employment measures refer explicitly to the calendar year 2008. More detail on each measure and on the implications of using recall data are provided in the “Measures” section below.

Before progressing further into a discussion of the data, a description of temporal strategy is warranted. Ideally, this dissertation would firmly establish a sense of temporal ordering via a panel approach, examining how changes in family health are associated with subsequent shifts in women’s employment characteristics. Leigh (2010) describes the difference between panel and cross-sectional approaches in his paper on elder care in Australia, relevant (albeit loosely) here, saying, “while the counterfactual in the cross-sectional approach is the behavior of non-carers, the counterfactual in the panel approach is the behavior of the same individual at a time when s/he was not carrying out caring responsibilities” (141). In other words, the panel approach can account for individual heterogeneity that might influence labor supply outside of informal care responsibilities to illustrate how within-person changes result in altered labor force outcomes. To apply this approach here, a critical component is the ability to measure family health in more than one period, so that changes in family health can be examined alongside changes in labor force outcomes. Ideally, these measures would be collected more than twice each, given known issues with a cross-lagged approach (that is,
examining associations between health\textsubscript{T1} and work\textsubscript{T2} then work\textsubscript{T1} and health\textsubscript{T2}). For instance, Heath et al. (1993) note that this approach breaks down if the two measures are highly stable over time, if there is “inconsistency between the causal lags” (Heath et al. 1993:31) (i.e., if the temporal distance between a change in health leading to a change in work is different from the distance in the opposite direction) or if measurement errors vary between the two measures (ibid). Further, the authors warn, “Reciprocal causation models can be tested only if multiple indicators are used or if measurement errors are absent or known to be equal for both traits. We have emphasized that if a single variable is used to assess each construct, then inferences about direction of causation will be sensitive to the assumption that measurement errors are uncorrelated between relatives” (Heath et al. 1993:48).

Regardless of the difficulties in stability, the development of such a model is rendered impractical here by the irregular collection of health information across FUMs over time. In the PSID, health measures are collected for women and their partners/spouses through the main survey beginning in 1999, continuing in odd-numbered years through the present. Data on young adults in the FU are collected biennially via the Transition to Adulthood surveys, beginning in 2005 and continuing into the present. However, children’s health measures are collected via the Child Development Supplement survey at only three points, spaced five years apart (1997, 2002, and 2007). Of course, this means that if family health measures are to assess conditions across the entire family simultaneously and establish a firm temporal grounding, the sole year of convergence is 2007.

I also consider the possibility of modeling employment outcomes in a 2008 and including a lagged control for employment in 2007 in order to assess how family health impacts employment outcomes net of those employment characteristics in 2007. However, preliminary
exploration of this possibility reveal that employment status in 2007 and 2008 are so strongly associated that inclusion of the lagged measure obscures effects of all other measures in the base model. It is plausible that the lag between the prospective Time 1 and Time 2 here is simply too short to produce useful indicators of temporal ordering.

A second considered alternative is to extend the outcome measures further into the future, modeling employment outcomes in 2009 or 2010. However, with the introduction of an additional survey wave, the analytic sample is reduced substantially (preliminary estimates indicate a reduction of at least 12 percent, or 541 families, prior to screening on data missingness and some secondary characteristics not yet examined). This loss is likely less problematic in terms of sample size and more problematic given the wider economic context in this period. For example, because the collection of health items is relegated to heads and wives only, respondents need not fully attrite to become ineligible for the analytic sample here, but simply exit head/wife status. As a result, those who weathered the effects of the Great Recession by “doubling up” in households with other family members would no longer be considered heads/wives of their own family units, a condition that may bias the sample away from lower income families. Further, it is unclear whether this operationalization would be an improvement over other options: using an early version of employment in 2009 as a preliminary test, I find that employment in 2007 is still strongly and significantly associated with employment in 2009, and that even with this longer lag, controlling for 2007 employment status subsumes all other effects in the model. For instance, the resulting model indicates that women are equally

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11 Because matching families across multiple surveys is an intensive process, I explore the potential inclusion of 2009 outcomes among data existing in the present data, without merging in the full set of indicators from the 2011 data. For example, most sample screening characteristics are present in my current dataset, though items like metropolitan residence (which indicates whether a family lives in a foreign country) are not included in reference to years beyond 2007. As a result, the estimate of a 12 percent sample reduction is conservative.
likely to be employed regardless of their educational attainment, a suspicious result that differs vastly from those without the inclusion of 2007 employment.

Another option is to model the effects of family health only for women who experienced change in employment characteristics (e.g., from employed to unemployed), for example, between 2007 and 2008. However, given the strong relationship between the two measures, the number of women who actually experienced this change is quite low (n=81). This sample would be insufficient even if an interaction between family health and social class were not central to this dissertation, which would splinter this group into still-smaller segments. As another metric of change, I consider modeling a change in hours worked, though this approach would obscure the substantive distinction between those who had reduced hours and those who had exited employment altogether (more on the descriptive use of these subsamples below).

After the above considerations, I settle upon employment characteristics in 2008 as the key outcome measures, with no lagged control for employment in 2007. As a result, these analyses should be largely considered cross-sectional. However, modeling labor market characteristics in 2008, rather than in 2007 affords two distinct advantages to these analyses. First, unlike for the year 2007, no sample members had missing values for hours worked or earnings in regards to 2008. Though I have imputed missing values for 2007 (more detail in the “Treatment of Incomplete and Missing Data” section, below), there is substantial reason to prioritize an outcome with fully observed values, rather than one with imputed values (for further discussion on use of an imputed measure as the dependent variable, see Young and Johnson 2010). Second, modeling employment characteristics for 2008 allows for some

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12 Missing data on hours and labor income are “assigned” by the PSID for 2008, but not 2007. These data are assigned for about three percent of all wives and use other information collected in the interview to do so.
additional descriptive analyses on the small groups of women who underwent some change in employment characteristics between 2007 and 2008 (see “Supplemental Analyses” in the Appendix).

**Data Quality**

The suitability of the PSID for answering the research questions of this dissertation is discussed in the above section, but it is also important to devote attention to the specific indicators of quality in the data used here. As the self-proclaimed “longest running longitudinal household survey in the world” (www.psidonline.isr.umich.edu), it is important to consider how the PSID compares to other surveys in terms of data quality and representativeness of the sample. In its original design, the PSID only collected data from the families of the initial sample, though two “refresher” samples of immigrant and Latino families were added in the 1990s in order to improve generalizability of the data amidst changing American demographics. Despite the rapid pace of American diversification (e.g., Johnson and Lichter 2010), there is substantial documentation that the PSID remains largely representative on many measures. Comparison of children in the PSID sample to those in the American Community Survey (ACS) sample found that the PSID child sample provides “good representation of the corresponding national population with coverage of approximately 97% of the U.S. population of children in 2007” (Duffy and Sastry 2012: 2). In particular, Duffy and Sastry (2012) used a generalized boosted regression model to compare propensity-weighted means from the PSID and ACS for age, sex, income quartiles, foreign-born parents, and Census Division of residence distributions by race. The authors found some overrepresentations in the PSID for blacks, no PSID-ACS differences for whites, and an underrepresentation of Hispanics only for residence in the New England Census Division (Duffy and Sastry 2012). When weighted, these differences demonstrate small
effect sizes, though statistically significant PSID-ACS differences remain in the share of children who are white, Hispanic, and Asian, with the worst representation for children of immigrant parents. Given recent immigration patterns and the fact that the PSID sample has not been refreshed since 1997, the authors’ finding that the PSID sampling frame is least likely to cover Asian and Hispanic children and those with a foreign-born parent is not surprising (ibid.).

In terms of income, Gouskova, Andreski, and Schoeni (2010) posited that though the PSID’s estimates of family income are slightly higher than those from the Current Population Survey (CPS), the gap between the two remained consistent between 1968 and 2007 and trends in family income at each decile tracked very closely between surveys (note, however, that family income in the PSID diverges from CPS estimates among families in the top and bottom 5 percent of the income distribution).^{13}

Regarding health measures, Andreski et al. (2009) found close alignment between estimates from the PSID and those available from the National Health Interview Survey (NHIS). Prevalence of asthma and hypertension are very similar between surveys, while the PSID demonstrates larger increases in mental illness between 2001 and 2007, and lower rates of adults in excellent health than the NHIS. In all, ongoing comparative documentation suggests that the PSID remains a good source for analyzing questions about the family unit.

**Analytic Sample**

The sample for these analyses includes women of working age (25-64 years) who live in the United States, share a household with at least one other person for whom health data are also collected (to ensure that family health can be measured; more detail on this restriction in

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^{13} Note also that Gouskova et al. (2010) do not report by how many dollars PSID-CPS estimates diverge at either end of the distribution, depicting the trend graphically with no accompanying tables. However, for families with incomes in the 5th and 95th percentiles, PSID estimates appear to exceed CPS estimates by no more than 10 percent. Of course the absolute value of a 10 percent divergence in income varies tremendously at the two extreme ends of the income spectrum.
Table 2, below), and who were present in the 2007 wave of the PSID. Because later analyses are stratified by educational attainment, I limit this sample to women aged 25 to 64, rather than the traditional 18 to 64 year old group, to avoid misstating the effects of education. That is, many women aged 18 to 24 would be precluded from inclusion in the “college graduate” group simply by being too young to have graduated, despite that college-attending women’s characteristics are likely most similar to their college graduate peers than to those who only continue education through the high school level. Additionally, because I also draw data from the 2005 and 2009 survey waves (discussed in detail in the Measures section), the sample is limited to those present in those years. Further, the sample is limited to women who are a head or wife, to ensure collection of health data, and women living in the United States, as it is unclear how this framework might apply to women in foreign labor market conditions. The largest reduction in sample size results from the age restriction on the sample, which reduces the sample by 16.2 percent. A table detailing the stepwise reductions in the sample is available in the Appendix (Table 19).

Table 1 shows various demographic and employment characteristics of this sample as compared to a similar sample from the Current Population Survey.\footnote{Specifically, I draw upon the 2007, 2008 and 2009 Annual Social and Economic Supplements. I use the 2007 sample for estimating demographic characteristics, the 2008 sample to estimate 2007 health insurance (item refers to preceding calendar year) and the 2009 sample for estimating employment detail (to reference the entire preceding calendar year, as in the PSID). To approximate the restrictions of the PSID analytic sample, CPS estimates are calculated among women aged 25-64, who are the head or spouse of the household and live in households containing at least one other person.} Characteristics of the analytic sample here align generally well with estimates from the CPS, though the PSID sample appears slightly older, whiter, wealthier, and more often married than their CPS counterparts.
Table 1. Characteristics of Analytic Sample as Compared to the Current Population Survey a

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Analytic Sample</th>
<th>Current Population Survey</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percent / Mean (SE)</td>
<td>Median</td>
</tr>
<tr>
<td>Age</td>
<td>45.11 (0.20)</td>
<td>43.00</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married/Cohabiting b</td>
<td>90.80</td>
<td>82.92</td>
</tr>
<tr>
<td>Never married</td>
<td>2.77</td>
<td>11.11</td>
</tr>
<tr>
<td>Previously married</td>
<td>6.43</td>
<td>5.97</td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td>74.37</td>
<td>69.18</td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>9.25</td>
<td>10.97</td>
</tr>
<tr>
<td>Other/Hispanic/ Multiracial</td>
<td>16.37</td>
<td>19.85</td>
</tr>
<tr>
<td>Educational Attainment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>8.86</td>
<td>10.44</td>
</tr>
<tr>
<td>High school/GED</td>
<td>34.39</td>
<td>29.02</td>
</tr>
<tr>
<td>Some college</td>
<td>25.86</td>
<td>28.91</td>
</tr>
<tr>
<td>College graduate</td>
<td>30.89</td>
<td>31.63</td>
</tr>
<tr>
<td>Region of Residence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>18.11</td>
<td>18.11</td>
</tr>
<tr>
<td>North Central</td>
<td>27.16</td>
<td>22.58</td>
</tr>
<tr>
<td>South</td>
<td>32.34</td>
<td>36.79</td>
</tr>
<tr>
<td>West</td>
<td>22.39</td>
<td>22.51</td>
</tr>
<tr>
<td>Metropolitan Residence</td>
<td>64.33</td>
<td>83.94</td>
</tr>
<tr>
<td>Number in FU</td>
<td>3.23 (0.02)</td>
<td>3.00</td>
</tr>
<tr>
<td>Number of Children in FU</td>
<td>1.25 (0.03)</td>
<td>1.00</td>
</tr>
<tr>
<td>Employment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed, 2008</td>
<td>76.49</td>
<td>74.08</td>
</tr>
<tr>
<td>Weekly Hours Worked, 2008 c</td>
<td>35.76 (0.25)</td>
<td>40.00</td>
</tr>
<tr>
<td>Labor Income, 2008 d</td>
<td>$40,247 ($1,139)</td>
<td>$31,000</td>
</tr>
<tr>
<td>Years of Work Experience</td>
<td>17.72 (0.20)</td>
<td>15.00</td>
</tr>
<tr>
<td>Resources</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anyone in FU Received SSI, 2007</td>
<td>2.65</td>
<td>N/A</td>
</tr>
<tr>
<td>Health Insurance Status, 2007 d</td>
<td>10.32</td>
<td>13.66</td>
</tr>
<tr>
<td>Partial-year coverage</td>
<td>1.86</td>
<td>86.34</td>
</tr>
<tr>
<td>Full-year coverage</td>
<td>87.82</td>
<td></td>
</tr>
<tr>
<td>Transportation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Own vehicle</td>
<td>96.86</td>
<td>N/A</td>
</tr>
<tr>
<td>Public transportation only</td>
<td>1.34</td>
<td>N/A</td>
</tr>
<tr>
<td>None</td>
<td>1.80</td>
<td>N/A</td>
</tr>
<tr>
<td>Value of Liquid Assets</td>
<td>$29,248 ($1,738)</td>
<td>$3,000</td>
</tr>
</tbody>
</table>

Note: All demographic estimates for the analytic sample are weighted with 2009 family weight (longitudinal by design), and weighted with the household weight for the CPS samples.

a Sample size for the PSID is 3,945 women (families). Sample sizes for the CPS comparison group are 44,066 in 2007, 43,551 in 2008, and 43,942 in 2009.

b For the remainder of this paper, I treat those who are married and cohabiting as one category, as presented here. However, it may be of interest to note that the 90.80 percent of women in this category are comprised of 86.07 percent of women who are legally married and 4.72 percent who live with long-term cohabiters.

c Among women employed in 2008 (analytic sample n=3,101; CPS n = 33,120).

d Note that the CPS insurance items ask whether respondents were covered by insurance at any point in the previous year, and do not inquire about months of coverage as in the PSID. Further, because the PSID measure is a family level measure, and the CPS is collected at the individual level, I recode CPS measures to the household level for improved comparability here.
In addition to the analytic sample above, I also rely on data from these women’s family unit members (FUMs) to construct a measure of health that considers all co-resident FUMs possible (see Table 2, below). As mentioned above, for inclusion in the sample, I require that women not only live with at least one family member, but that they live with at least one family member who has some health data available. This ensures that “family-level” measures of health refer to FUMs beyond just women themselves. These FUMs (n=5,564, excluding 3,945 women) are considered the “auxiliary sample” and while they will not be included in any central analyses, their health information is linked to individual women’s records to construct a family-level file with a single (female) reference person. The remaining cases—people who live in screened family units but do not themselves have health data—are not dropped from the dataset, though without health information, their presence is only considered via measures collected at the family level (e.g., number of people in the FU or vehicle ownership by anyone in the FU). Table 2 shows the distribution of family relationships in screened families, while the last two columns note the share of FUMs for whom health data have been collected.

Table 2. Family Roster for Screened Women and Their Family Unit Members (FUMs)

<table>
<thead>
<tr>
<th>Detailed Relationship</th>
<th>Unweighted n</th>
<th>Percent of All FUMs</th>
<th>Cumulative Percentage</th>
<th>Unweighted n with Health Data</th>
<th>Percent with Health Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Woman&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wife/&quot;Wife&quot;/ Female Head of Household</td>
<td>3,945</td>
<td>29.57</td>
<td>29.57</td>
<td>3,945</td>
<td>100.00</td>
</tr>
<tr>
<td>&quot;Spouse&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spouse/Long Term Cohabitor</td>
<td>3,376</td>
<td>25.30</td>
<td>54.87</td>
<td>3,376</td>
<td>100.00</td>
</tr>
<tr>
<td>&quot;Child&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child</td>
<td>5,535</td>
<td>41.48</td>
<td>96.35</td>
<td>2,108</td>
<td>38.08</td>
</tr>
<tr>
<td>&quot;OFUM&quot;</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grandchild</td>
<td>250</td>
<td>1.87</td>
<td>98.22</td>
<td>51</td>
<td>20.40</td>
</tr>
<tr>
<td>Parent</td>
<td>68</td>
<td>0.51</td>
<td>98.73</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>Sibling</td>
<td>41</td>
<td>0.31</td>
<td>99.04</td>
<td>7</td>
<td>17.07</td>
</tr>
<tr>
<td>Other Relative</td>
<td>93</td>
<td>0.70</td>
<td>99.74</td>
<td>22</td>
<td>23.66</td>
</tr>
<tr>
<td>Other Nonrelative</td>
<td>27</td>
<td>0.20</td>
<td>99.94</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td>First Year Cohabitor</td>
<td>8</td>
<td>0.06</td>
<td>100.00</td>
<td>0</td>
<td>0.00</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>13,343</td>
<td>100.00</td>
<td></td>
<td>9,509</td>
<td>71.27</td>
</tr>
</tbody>
</table>

Note: Relationships are classified in reference to the head and/or wife (that is, to the screened woman, her spouse, or her long term cohabitor, such that "child" encompasses a woman’s biological child, stepchild, or child of her long term cohabitor, as well as foster or adopted children).
As shown in Table 2, 96.35 percent of individuals in screened families fall in the categories of women, spouses, and children. Of course families outside of the screened sample are likely more diverse in their composition; however, because the PSID is only collected among descendants of original participants and their spouses, requiring that at least one family member has participated in a survey biases the sample toward a more traditional family structure. The last column shows more than 70 percent of FUMs in screened women’s families have at least some health data, with data collected for all women and spouses, and considerably smaller shares of other FUMs.\textsuperscript{15}

**Measures**

**Independent Variables**

Together, the PSID, TA, and CDS contained more than 115 health-related measures in 2007, documenting diagnosis of specific conditions, timing of those diagnoses, and resultant limitations (not including the more than 30 items regarding activity limitations). Given the array of health data available, there are several possibilities for operationalizing “family health.” First, with the variety of conditions available in PSID data, from allergies to schizophrenia, it is worth considering whether all conditions should be considered and aggregated into a simple condition count, or whether some exclusionary criteria should be applied (Goodman et al. 2013). Guidance in this matter is derived from existing research on co-occurring chronic conditions though there is substantial variation in measurement approaches, the vast majority of this

\textsuperscript{15} Note that 40 percent of children in these families were not born when the CDS began, and thus are ineligible for inclusion in that survey, and another 3.7 percent of children were too old when it began. The remaining children who are excluded may have lived in families with multiple children at the time of survey commencement (only two children per FU may participate), or may have joined the household later. I elect to include OFUMs who have health data in these analyses, to maximize detail on the co-resident family members’ health. The majority of these OFUMs are children living with their grandmothers (n=51). The remainder of the “other” relatives category is comprised of nieces or nephews of heads and wives (13 of 22), one cousin of the head or wife, and seven people who are classified as unspecified “other relatives” by the PSID.
research relies upon simple sums of conditions as described by Diederichs, Berger, and Bartels (2011) and Guralnik (1996). In the scant literature that does distinguish between more and less severe illnesses, some use subjective assessments of severity (e.g., Puntenney 1999), others weight items according to “impact,” via self-reported activity limitations, population mortality risk, or relation to a pre-defined threshold (e.g., blood pressure readings above a given level is classified as “severe”). The latter approach has been subject to substantial research attention, but with no real consensus in its conclusions (Diederichs et al. 2011; Diederichs et al. 2012; Fortin et al. 2005; Tooth et al. 2008; Weiss et al. 2013).

Despite the substantial discussion around this issue, complex scaling practices appear to have few distinct advantages over a simple condition count. For example, a subjective assessment of severity does not yield any intuitive improvement to a simple count, as it also lacks criteria for determining whether an illness “counts.” Stratifying illnesses based on resultant limitations raises an issue described earlier: that an illness may have a measurable impact on a family without presently imposing some kind of limitation (e.g., an illness may not be imposing a limitation because of the frequent or intensive therapies or management processes undertaken by the family). In addition, only one in five people with a chronic condition also experience some kind of activity limitation (Anderson 2010). Further, comparing illnesses to population-level measures like morbidity risk does not provide an applied, family-specific assessment that can indicate which conditions are most central in shaping the circumstances of family life and the lived experiences of illness. For example, while one might assume that illnesses known to be capable of producing intensive symptoms (e.g., bipolar disorder) may be a more intensive and

16 The effort to be systematic in inclusion of particular diseases is still somewhat unusual in assessing co-occurring chronic conditions. In their review of multimorbidity indices, Diederichs et al. (2011) found that just 41 percent of studies made any mention of why particular conditions were included; among those who gave a reason, the most popular was high prevalence in the population.
disruptive illness to manage than say, allergies, this is an assumption that cannot be tested with the PSID data.

Huntley et al. (2012) indicate that simple counts of conditions are the most common approach to measuring multimorbidity, and though inter-study comparability is complicated by variation in included measures and methods for summing related illnesses, the authors conclude that simple counts “perform almost as well as complex measures in predicting most outcomes” (134). In terms of health outcomes, research has shown that the number of chronic diseases present predicts a patient’s number of prescriptions, referrals, and hospital admissions (Condelius et al. 2008; Wolff, Starfield, and Anderson 2002), as well as levels of functional decline (Bayliss et al. 2004; Marengoni et al. 2009a; Marengoni et al. 2009b) and health-related expenditures (Friedman et al. 2006; Schneider, O’Donnell, and Dean 2009) (see also Marengoni et al. 2011 for a summary of this work). That the number of conditions present in a given person is indeed associated with intra-person health consequences suggests that this approach may most closely align with my purpose here.

As such, I rely on simple count methods to aggregate all health conditions reported by individual family members, drawing on multiple surveys to capture full reports where possible (discussed in detail in “Treatment of Missing and Incomplete Data” section, below). Health-related measures vary in number and content between surveys, perhaps due to the assumed relevance of specific conditions to each survey’s target population: for example, children in the CDS are not asked about arthritis or memory loss, though their parents are queried on these topics in the main PSID survey. In each instance, the respondent is asked whether they have ever been diagnosed with [condition] by a doctor; those who provide affirmative responses are considered to have a given condition. While the list of conditions included in any given survey is
not necessarily comprehensive, the presence of an “other chronic conditions” item in each survey suggests that respondents with ongoing health issues will likely be captured at least to some degree.\(^7\) A full list of conditions available in the three surveys is shown in Table 3, below.

Table 3. Specific Health Condition Measures Available by Survey

<table>
<thead>
<tr>
<th>Condition</th>
<th>Measure Available In:</th>
<th>Measure Available In:</th>
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<tbody>
<tr>
<td></td>
<td>CDS</td>
<td>TA</td>
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<tr>
<td>Allergies</td>
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<tr>
<td>Alcohol Problem</td>
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<tr>
<td>Anemia</td>
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<tr>
<td>Anxiety</td>
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<td>Asthma</td>
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<td>Arthritis</td>
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<td>Autism</td>
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<td>Bipolar Disorder</td>
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<tr>
<td>Cancer/Malignant Tumor</td>
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<td>•</td>
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<tr>
<td>Depression</td>
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<td>•</td>
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<tr>
<td>Developmental Delay</td>
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<tr>
<td>Diabetes</td>
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<td>•</td>
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<tr>
<td>Drug Problem</td>
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<td>•</td>
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<tr>
<td>Emotional, Nervous, or Psychiatric Problem</td>
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<td>•</td>
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<tr>
<td>Epileptic Fit</td>
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<td>•</td>
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<tr>
<td>Hearing Loss</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>Heart Attack</td>
<td>•</td>
<td>•</td>
</tr>
</tbody>
</table>

Notes: Items in blue are possible response categories to the emotional, nervous, or psychiatric problem question. PSID respondents who report an emotional, nervous, or psychiatric problem are permitted to list up to three separate diagnoses related to that problem. Similarly, TA respondents are asked whether their diagnosis is one or more of seven specific disorders. For both groups, affirmative responses on any of the follow-up mentions are aggregated into the summary measure here as separate conditions. This is also the case with conditions that may be a subset of another condition, reported in multiple categories by respondents. For example, if a condition like high lead exposure produces developmental delays, the respondent may report these as separate conditions, and there is no way of distinguishing these two conditions as related under one diagnosis.

In consultation with a family roster, I tally illnesses for specific family members, thereby creating separate measures of women’s own illnesses, spouse’s illnesses, children’s illnesses, and OFUMs’ illnesses, and link each total onto women’s record in the data. I also aggregate the individual-level measures into one item indicating the total number of conditions present in each family unit (albeit among members for whom health conditions are available). By creating individual-level measures first, I am able to also (1) test whether the

\(^7\) Of course this does not address the fact that unequal access to health care will likely yield differential rates of diagnosis across populations (see for example Fixler et al. 1993 and Liptak et al. 2008), an important issue that is acknowledged as important to the research here, despite that its quantification is beyond the scope of this paper.
impacts of women’s own illnesses vary from those of others FUMs’ illnesses, and (2) test whether specific FUMs’ illnesses vary in their impact (e.g., spouse vs. child).

In order to assess co-occurrence of specific illnesses at the family level, I retain individual-and family-level measures of several chronic conditions. Because the PSID assesses dozens of conditions it is impractical in terms of time and sample sizes to examine each separately. Thus, to identify conditions for further investigation, I draw upon the public health literature to identify conditions that are especially prevalent in the population; perhaps because of their widespread prevalence, each of these conditions are also especially likely to co-occur with other illnesses, lending an interesting dynamic to the research here. By identifying a subset of especially prevalent conditions for examination in all families, I am provided with a systematic framework for identifying patterns of illness that does not require identifying disjointed constellations of a multitude of diagnoses that emerge among individual families. Further, because these conditions are so prevalent at the population level, each diagnosis is present here in sample sizes sufficient for analysis here.\(^{18}\) In addition, conditions that arise in the public health discourse tend to be those with public health implications, providing further reason to prioritize these conditions. For example, the CDC suggests that “chronic diseases and conditions—such as heart disease, stroke, cancer, diabetes, obesity, and arthritis—are among the most common, costly, and preventable of all health problems” (CDC 2014).

Of the dozens of conditions included in the PSID, I select eight specific diagnoses for further examination: anxiety, arthritis, asthma, cancer, depression, diabetes,

\(^{18}\) Of course, that these conditions are especially prevalent population-wide may be intrinsically related to the rate at which physicians apply these diagnoses, a tautological public health statistical issue described by McKinlay (1996).
hypertension, and lung disease.19 I code each of these conditions into binary measures of “ever diagnosed” at the individual and family levels, where the family level measure indicates that any FUM has been diagnosed with the specific condition. These measures allow me to examine specific diagnoses at both the individual and family levels.

While family-level measures are useful for assessing the prevalence of specific diseases, they lack the nuance necessary for a full examination of co-occurring illness. For instance, if the above measures indicate that depression and anxiety are present in a family, the natural question becomes whether these illnesses are truly measured at the family level, or whether—especially if known to be comorbid within individuals—the diagnoses are simply individual-level comorbidities being described at the family level. To determine whether specific diagnoses co-occur across family members, I also create measures indicating cross-member diagnoses. For example, these measures allow me to identify when one family member has depression and another has anxiety, or when two people in the same family have depression diagnoses. These measures are used to better understand patterns of illness within families (Chapter III) and are also tested for inclusion in the multivariate models that follow (see Chapters IV and V).

Given the admittedly large number of health measures described above, Figure 1 summarizes the health measures used in this dissertation. In the bold font and thick-bordered box is the most general measure of family health, which represents the total number of chronic conditions in the family unit. This measure is created by aggregating the condition counts specific to women, spouses, children, and OFUMs. Tall thin-bordered boxes enclose the collection of diagnoses available for, and considered in, each specific FUMs’ condition count,

19 I also create measures of substance abuse (specifically, alcohol and unspecified drug “problems”), heart disease, heart attack, and stroke as conceptually meaningful conditions, but very low subsample sizes (n=7 families for a diagnosis of substance abuse) force their omission from all analyses here.
with women’s and spouses’ conditions from the PSID main file, and children’s/OFUMs’ conditions from the CDS/TA files. Color-coded boxes indicate specific diagnoses that are examined (singly and in co-occurring pairs, where sample sizes allow) at the family level. Finally, italicized font indicates women’s own diagnoses that are examined singly and in tandem with various other health measures in Chapters IV and V.

Figure 1. Visual Diagram of Health Measures and their Coding

<table>
<thead>
<tr>
<th>NUMBER OF CONDITIONS IN FAMILY UNIT</th>
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<tbody>
<tr>
<td><strong>Women’s Condition Count</strong></td>
</tr>
<tr>
<td>Alcohol Problem</td>
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<tr>
<td>Anxiety</td>
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<tr>
<td>Asthma</td>
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<tr>
<td>Arthritis</td>
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<td>Bipolar Disorder</td>
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<td>Cancer/Malignant Tumor</td>
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<td>Depression</td>
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<tr>
<td>Diabetes</td>
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<tr>
<td>Drug Problem</td>
</tr>
<tr>
<td>Heart Attack</td>
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<tr>
<td>Heart Disease/Condition</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Lung Disease</td>
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<tr>
<td>Memory Loss (permanent)</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
</tr>
<tr>
<td>Other Chronic Condition</td>
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<tr>
<td>Other Psychiatric Condition</td>
</tr>
<tr>
<td>Phobias</td>
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<tr>
<td>Schizophrenia</td>
</tr>
<tr>
<td>Stroke</td>
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<tr>
<td>Autism</td>
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<tr>
<td>Developmental Delay</td>
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<tr>
<td>Emotional/Nervous/Psychiatric Problem</td>
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<tr>
<td>Epileptic Fit</td>
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<tr>
<td>Hearing Loss</td>
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<tr>
<td>Heart Disease/Condition</td>
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<tr>
<td>High Cholesterol</td>
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<tr>
<td>High Lead Levels</td>
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<tr>
<td>Hyperactivity</td>
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</tbody>
</table>
Dependent Variables

In order to comprehensively assess impacts of family health on employment, I operationalize outcome measures in three ways. First, I create a binary indicator of employment based on respondents’ reports of being employed in 2008. This measure is the broadest conceptualization of women’s labor force supply, and serves to screen respondents into subsequent models predicting hours and earnings. Because 2008 is not a survey year, no direct measure of employment status exists (PSID employment status items reference current employment in survey years). Instead, 2009 heads and wives indicate their 2008 employment status via their “average” weekly hours worked in a given year (range=0-110); those reporting a value of zero are labeled by the PSID as “did not work for money in 2008.” I consider all who indicated a non-zero average to have been employed in 2008. I also treat the same measure continuously among employed women, using average number of hours worked per week in a second set of models exploring associations between family health conditions and labor supply.

The third employment measure indicates women’s annual labor market income in 2008. To create this measure, I sum three separate component measures, indicating regular labor income, income from unincorporated businesses, and farm income for all women, to ensure the full spectrum of earnings are captured, and to ensure comparability with earnings measure collected for 2007 (to be discussed below). Values for this measure range from 0 to $600,000. Women reporting hours worked but zero income were examined in close detail to determine why they might have no earnings; a very small share actually lost money on their

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20 Though differences in data availability preclude a replication of measures, this approach was inspired by Coley et al. (2011), who draw predict any employment, number of months employed of the past six, average weekly hours in the past six months, and monthly earnings from women’s main jobs.
farms or businesses that year and are still considered “employed” here. The remainder of women who reported hours worked but no earnings from wages or salaries, unincorporated businesses, or farms were recoded to “not employed” (n=10). Labor market income is not normally distributed (skewness=4.88; kurtosis=55.71); an examination of possible transformations via Stata’s ladder command reveal that normality is approximated with a natural log transformation, used in the analytic models to follow.21

**Education and Other Potential Moderators**

To determine whether the patterning and effects of family illness vary by social class, I use women’s educational attainment as a proxy for class. I choose educational attainment rather than alternate measures (e.g., family income or occupational prestige) for several reasons. First, given the PSID’s biennial structure, there are no family income measures that directly reference income in 2007. Though family income in 2006 is collected, application of this measure runs risk of overlooking year-to-year fluctuations in income, shifts that might be especially pertinent for the low income families that are of particular interest in this inquiry here. Beyond the intra-year stability that educational attainment affords, its use over occupation-based measures ensures that all sample members—whether employed or not—can be categorized. As such, all descriptive and multivariate analyses are conducted with an eye toward identifying variation in the distribution and effects of family illness by educational attainment (e.g., testing interaction effects for the latter). **Educational attainment** is collected in the PSID as “highest grade or year of school completed,” with values of 1-16 indicating actual number of years, and a value of 17 indicating “at least some postgraduate work.” As existing research suggests that educational level is more meaningfully applied as a

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21 In this paper, all executable Stata commands appear in Courier New font, per conventional StataCorp styling (StataCorp 2013b).
class proxy when measured in terms of credentials (e.g., Krieger, Williams, and Moss 1997; Oakes 2011), I recode years of education into four categories that summarize years of education: less than a high school diploma (0/11 years), high school diploma (12 years), some college (13/15 years), and college graduate or higher (16/17 years). More than 300 women were missing values on the 2007 measure, and as a relatively stable characteristic, I impute available values using the longitudinal direct substitution method, described in detail in the “Treatment of Incomplete and Missing Data” section, below. After imputation, eleven women were missing on this measure, and were excluded from the sample.

Beyond education, I have also posited that specific resources may buffer the potentially deleterious effects of poor family health on women’s employment outcomes. Using a measure indicating the number of months that each family member was covered by health insurance in 2007, I create a family-level measure that indicates full-year, partial-year, or no health insurance coverage across the entire family, missing for 3 women, who are excluded here. A second resource measure references access to transportation, created from two measures, the first indicating whether someone in the family unit owns or leases a car for personal use (e.g., private transportation), the second indicating whether the family unit has spent any money on public transportation in the past month (e.g., public transportation), with negative responses on both coded as no transportation. Including both personal vehicle ownership and access to public transportation ensures I do not bias estimates of access to transportation away from those in metropolitan areas, or those who are precluded from driving for health reasons. The few families who use both public and private transportation are coded into the latter category.

Thus far, it is unclear how the receipt of disability-related payments might intersect with the health-employment relationship. Thus, I use a measure here that indicates whether any
family unit member received **supplemental security income** in 2007. Three women were missing this measure and are excluded from the analytic sample here. A final measure indicates the value of the family’s **liquid assets** in “checking or savings accounts, money market funds, certificates of deposit, government savings bonds, or treasury bills.” As might be expected, there is tremendous variation in this measure across families, ranging from $0 to $1,500,000. Existing literature categorizes measures of wealth in various ways, including via quintiles of positive wealth (e.g., Geyer, Spreckelsen, and von dem Knesebeck 2014; Hajat et al. 2010), and in logarithmic scales when used as a dependent variable (e.g., Anastasiadis 2010). As an independent variable, there is no need to normalize the distribution via a log transformation; however, for ease of interpretation, I divide the original values by 1000 in order to create values in thousands of dollars. In addition, though the PSID collects several good measures of overall wealth, I focus on liquid assets here, as they are more easily mobilized when families face costs like prescription drug copays or unexpected childcare needs. In other words, while having a pension or owning a home may be associated with health outcomes, I suspect they are less relevant for meeting the day-to-day obligations posed by illness.

**Covariates**

A series of basic demographic measures are included in each regression model, including women’s **age** in years (including a quadratic term to test for a nonlinear function of age; a mean-centered version of age is used when the quadratic term is included). Only two people

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22 I include SSI receipt here due to its direct relationship with health. Other social safety net components (e.g., food stamps, welfare, and social security) may also play a role in predicting women’s labor force outcomes, though that exploration is tangential to the purpose here. That these income-based programs will be endogenous to labor market income (an outcome of interest here) is further reason to set aside those analyses.

23 I retain assets in its original form initially, in order to assess the possibility that the effects of assets may not be linear, and presenting the results in relation to “thousands” of dollars may abbreviate more nuanced effects at lower asset values. After fitting the models in Chapters IV and V, I determine that the relationship is indeed linear, and this transformed version of assets eases interpretability, and all results presented here use the transformed measure.
living in potential sample FUs did not report an age in 2007, but given past experience with the
PSID and known variability across reports of age and year of birth within a single individual, I
performed quality assessments of reported age values in several ways. First, so that it is possible
to later model the effects of having young children in the household, I assess quality for all
members of FUs containing a female head or wife in 2007. I calculated a year of birth using
[2007-reported year of birth] (missing for 44 potential FUMs). I then compared this calculated age
with reported age for those who had nonmissing values on both measures. Noting some
discrepancies between calculated and reported values (only 40.3 percent of women and FU
members’ values aligned perfectly), I also added age and year of birth reports from 2005 and
2009 in an attempt to improve reliability. I calculate an age from each of these years of birth
reports also, and use this set to create a final age variable that: (1) updates the reported age of
children under 2, to all of whom the PSID assigns an age value of 1; (2) compares reported
month of birth to recorded interview month, to determine whether a one-year mismatch
between reported and calculated age is due to a late birthday (e.g., person born in 2002 should
be 5 in 2007, unless their birthday occurs after the interview); (3) identifies and adjusts cases
where a given age was reported multiple survey years in a row, and (4) corrects age reports
where year of birth or age is grossly mismatched. I attempt to be systematic in this process but
not at the expense of logic; for example, in one family, a couple in their 40s has two daughters
whose ages are reported as teenagers. However, their years of birth indicate that both girls are
in their late 30s; reported age is assigned here, based on the logical ages of the remainder of
girls’ parents. In all, age values have been corrected for 109 members of screened families,
including 3 women (0.08 percent) and 106 (1.12 percent) FUMs.
**Marital status** is self-reported on the survey, coded to reflect whether a respondent is currently married/cohabiting, (coded in one category on the survey), previously married, or never married. It should be noted that throughout, I use the term “spouse” to denote both legally married and long-term cohabiters (i.e., more than one year), as is the custom in the PSID. Recent research shows few differences between those who cohabit with the intent to marry and those who cohabited before marriage (Brown, Manning, and Payne 2014; Kuperberg 2012), and for the purposes of family health assessment, it seems reasonable that residential, rather than legal, commitments would be most important. Nonetheless, I retain a flag for “female head of household” which I use to test robustness of results between wives and “wives” in later analyses (noted only where findings emerge).

Next, using the PSID’s “relationship to head” variable and the shared family identifier, I ascertain whether each woman shares the household with any biological children, legal stepchildren, or children of her partner. Because the TA survey includes young adults who have not yet formed their own households, for the purposes of this project, “**number of children in the household**” does not exclude those over the age of 18, but rather, indicates whether a woman has any of her own or partner’s offspring in the household. This decision is based on the assumption that the practical distinction between, say, those aged 19 and those aged 17 are likely minimal in terms of their impact on women’s employment decisions. Following Urban and Olson (2005), who found that increased time since the birth of one’s youngest child is a predictor of women’s employment, I create a measure that indicates whether (1) a woman has no children in the household, (2) has a child under the age of five in

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24 Some TA respondents actually do have their own households, and thus participate in the main PSID interviews; these adults are treated like any other PSID women.

25 Note that though analyses here do not exclude grown children, the vast majority of children in these households are young (mean = 11.7), and 99 percent are aged 23 or younger.
the household, or (3) only children over the age of five. Here again, I allow for a looser construction of “children” than previous research has done, suggesting that young children in the household may impact women’s labor supply whether or not the woman actually gave birth to those children.

Women’s race and ethnicity is determined through five measures, including an indicator of Hispanicity and up to four variables indicating race (respondents of one race may provide the same response on all four measures or respond to only the first variable). Women are categorized as “white, non-Hispanic,” “black, non-Hispanic,” or into the broad aggregate category of “Hispanic/other/multi-racial, non-Hispanic.” Twenty-seven women were missing values on the 2007 race/ethnicity measures; as race/ethnicity are generally stable characteristics, these cases are treated with the longitudinal direct substitution measure, described in detail in the next section; after this form of imputation, only seven women are missing race/ethnicity, and are excluded from the analytic sample.

I use a collapsed version of the Beale Rural-Urban Continuum code (“collapsed” for the public release data by aggregating the smallest types of rural areas into one “completely rural” category, to prevent identification) to determine metropolitan/nonmetropolitan status. Following the U.S. Department of Agriculture (Parker 2013), I consider places inside metropolitan areas to be “metropolitan,” and populations adjacent or not adjacent to metropolitan areas to be “nonmetropolitan.” The inclusion of this metropolitan indicator is intended to account for the possibility that the conditions of rural and urban labor markets differentially impact women’s labor supply (e.g., Gibbs, Kusmin, and Cromartie 2005; Kim et al. 2005; Slack 2014).

26 While a more nuanced racial and ethnic breakdown would be ideal, data limitations of the PSID discussed in the “Data Quality” section above preclude any finer-grained distinctions.
Finally, Wolfe and Hill (1995) found that years of work experience predicts employment among women with children in poor health. For measuring “number of years of work experience,” there is a similar indicator in the PSID, however, it is only collected at the time of sample entrance, and is never updated. There is little guidance on this topic from the PSID, which instructs users to “update these variables by recoding data for subsequent years” and users’ attempts to improve on these measures often involves a considerable amount of recalculation (see Regan and Oaxaca (2009) for a detailed summary of efforts to calculate an annual hours worked measure). The lack of detail in this measure has received attention for many years (e.g., Corcoran 1977), specifically in regards to the inconsistent availability of weeks worked measures across the lifecourse, which can be particularly important for examining women’s engagement in the labor force during childbearing years (ibid). While a “weeks worked” measure would be ideal for capturing women’s true work experience, these measures are unavailable for 1997-2003—a period in which the vast majority of my sample was of working age—making this a non-viable option.

As an alternative, I create my own adjustments to the measure collected at sample entry. To scale this measure upward, I sum the number of years that each woman indicates she was employed, beginning with the calendar year after her sample entrance, and add the result to the count reported at sample entrance. For women missing values on the original variable (6.6% of screened women), I replace only with the sum of her annual reports. Of course, this means that women who are missing the initial measure have fewer total years of work experience reported, as they had no opportunity to provide pre-PSID employment history information. However, I suggest that this measure is preferable for its accuracy for the largest share of the sample possible, and that excluding the original measure in favor of annual counts
only would more severely bias this measure. I follow suggestions for cross-checking and verifying these measures from Regan and Oaxaca (2009), though those authors had a wider range of “unrealistic” (ibid: 17) responses than in this sample, perhaps a result of the PSID’s attempts to regularly improve and re-release existing data. The resulting measure is missing data for seven women, who are excluded from the analytic sample.

For the descriptive analyses examining one-year change in employment outcomes (described in the “Supplementary Analysis” section of the Appendix), I create measures of labor market outcomes in 2007, including ever employed in 2007, hours worked, and income earned in that year. Because of the biennial data collection, employment measures for the calendar year 2007 are drawn from the 2009 survey. As with any recall data, caution is warranted when using what the PSID staff terms “t-2” data, or data that refers to the calendar year two years prior to the survey year (here, 2007). However, Andreski, Stafford, and Yeung (2008) explored the quality of such data in another technical paper, and found that these measures show “a reasonably good alignment” (2008:7) with reports with a one-year recall period, though data are more likely to be missing in t-2 reports. Employment in 2007 is indicated via an existing binary measure and average weekly hours worked ranges from 0 to 109. Peculiarly, only in 2007 are respondents allowed to report labor income over varying temporal reference periods, which I recode to reference the calendar year. For those who reported by the hour, I multiply this amount by their reported number of weekly hours, and then assume 50 weeks worked per

27 To determine to what extent these women’s inclusion might bias the coefficient on work experience downward, I estimate the final models in Tables 14, 15, and 16 with and without them in the sample. In predicting employment and income, models including these women have slightly smaller coefficients on work experience (coefficients compared with an adjusted Wald test; \( p<0.01 \)), though there is no difference in the model predicting hours. These differences do not affect conclusions around statistical significance in any case and the differences between models are largely statistical rather than substantive (e.g., in the employment model, OR= 0.104 with these women included, versus OR=0.116 without). These women are included in all models throughout the remainder of this paper.
year to calculate an annual amount. Those who reported per day were assumed to work five
days a week and 50 weeks a year; those reporting weekly or biweekly, assumed 50 weeks per
year. Those reporting monthly are assumed to work 12 months per year, and those reported
an income but no corresponding time frame were left missing. Fortunately, the vast majority of
women employed in 2007 reported on annual or monthly bases, and this practice of scaling up
from sub-monthly reports, was limited to 24 respondents.

**Treatment of Incomplete and Missing Data**

*Longitudinal Direct Substitution*

In order to minimize missing data, I draw upon data collected in multiple waves and
multiple surveys; in the PSID, populating missing data with values from another year requires
carefully tracking individual respondents’ presence and role in the FU in each survey wave. For
example, each individual PSID FU member has values on both individual and family-level files;
the latter include a series of items referring to the head and wife (if present). Of course, like
families anywhere else, those in the PSID sample are dynamic units that undergo regular
compositional changes (e.g., births, deaths, moves, marriages, divorces), meaning that the head
and wife items may not refer to the same people in consecutive waves. Though the PSID
recommends that new users interested in tracking families across waves restrict their analysis
to FUs that have experienced no compositional change, doing so can both limit and bias a
sample by excluding families experiencing various kinds of instability. With the use of family
identifiers, “sequence numbers” (indicating whether a sample member is present, or has moved,
died, or been institutionalized), and “relationship to head” reports, it is possible to clearly

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28 I do not re-scale income to account for any time off during the year if women report a monthly income, since it
is plausible that women who report this way do so because they are paid this way; thus the simplest approach for
estimating an annual sum is to multiply by 12.
identify transitions within a given family unit between survey waves. Because (generally) identical questions are asked of both the head and wife, it is also possible to draw data from the appropriate variables when women enter headship or become wives, and to ensure that the “spouse” data refer to a consistent partner between waves. To address these issues, I constructed inter-wave “status” variables for all heads and wives to serve several purposes: (1) to detect whether a 2007 wife’s 2005 data should be drawn from the head or wife variables from that year; (2) to confirm whether a 2007 spouse was the same person as in the previous wave; and (3) to limit sample loss, as tracking transitions of women whose FUs underwent some change in the 2005-2009 period allowed for retention of 404 women, or 10.2 percent of my final sample.

**Women’s Demographic Variables**

For demographic measures that have missing values and are collected at more than one time point (educational attainment, ), I first attempt a substitutive approach to imputation. Educational attainment is drawn from individual-level reports in the 2007 main PSID files, and was missing for 323 women. As a generally stable characteristic, I use a longitudinal direct substitution (LDS) approach for imputing missing cases, described as a “highly accurate form of imputation” (Heeringa and Lepowski 1986:206) for items that are stable over time, with particular strength and practicality for categorical variables (ibid.). While this method runs the risk of underestimating change (that is, those with new educational credentials would not be captured by the data imputed from 2005), Heeringa and Lepowski (1986) suggest “the LDS method of longitudinal imputation understates change, but this may be preferred to the gross overstatement of change resulting from the use of the CSHD [cross-sectional hot-deck] method” (210).
Thus, where education was missing, I draw values first from the 2005 survey and then from the 2009 survey to limit missingness here. While using educational attainment from 2009 is not ideal—in that it may assign higher educational attainment to women than was present in 2007—that the sample is restricted to those aged 25 or older suggests that most of the women have likely finished their education by the time of data collection. After this process, 11 women were still missing education values. Race and ethnicity, missing for 27 women in the 2007 panel, are imputed in a similar fashion, though drawing from a more extensive time period, given the stability of racial and ethnic identities compared with educational attainment. Using data from 2003, 2005, 2007, 2009, and 2011 to populate missing values, seven women with no race/ethnicity data remain, all of whom were omitted from the analytic sample.

Finally, additional missing values were present for three or fewer women on measures of health insurance coverage in 2007, transportation availability, and SSI receipt. Harrell (2001) suggests that where fewer than five percent of cases are missing values, complete case analysis (i.e., listwise deletion) and simple substitution of median (continuous variables) or most frequent values (categorical variables) are sound choices. In order to reduce sample loss, particularly in instances where data are not missing at random (more detail on this below), I implement a more stringent version of those guidelines, and remove cases from the analytic sample only when missing in fewer than one percent of cases. This approach results in the exclusion of 31 otherwise-screened cases from my sample, as noted in Table 1.

Heads’ and Wives’ Health Measures

First, for women and their partners/spouses, data are drawn from the 2007 panel. As with the demographic variables described above, I first employ a LDS method that substitutes values from the 2005 panel where 2007 values are missing; note that all condition measures
refer to lifetime diagnoses, and thus, should be considered “stable” characteristics between 2005 and 2007. I chose this method for its parsimony, but also under the practical consideration that with the sheer number of conditions on which data are collected (21 among heads and wives), carefully building and troubleshooting separate imputation models for each condition was not feasible, nor necessarily preferable. Thus, I include all 2007 health items among women and spouses, imputing 2005 values only where the 2007 item is missing and the 2005 item is not. Among the final sample of women and spouses, 3.4 percent of women and 1.7 percent of men had one or more conditions imputed from the 2005 survey.

Non-Head/Wife Family Members’ Health

For non-head/wife family members, imputation processes also follow a LDS approach, though somewhat more complex than that among the heads and wives. For children in the CDS, measures were first constructed from 2007 items, and substituted with values from 2002 (the next most recent wave) when 2007 measures were missing. Any CDS participants who also participated in the TA were initially excluded from CDS counts, to ensure that no family member’s conditions were summed more than once (N=528).

The most complex LDS process is among those participating in the TA, as 92 percent of TA participants were also CDS 2002 participants, a survey which inquires about several conditions not included in the TA survey. First, I create a measure of 2007 TA variables alone, substituting 2005 values where 2007 values are missing. Next, I identify TA respondents who were also in the CDS, and incorporate the measures of additional conditions not asked in the TA survey (14 conditions) into the TA respondents’ illness count. For measures that are present in the TA and the CDS (4 conditions), I substitute CDS02 measures only where TA07 and TA05 measures are missing.
**Multiple Imputation**

For variables where five to fifteen percent of cases are missing values, Harrell (2001) suggests using single imputation via predictors in the data and applying multiple imputation techniques when variables’ missingness exceeds 15 percent. However, the two measures missing the most data are labor market income and hours worked (each for 2007), two measures that I later use for descriptive analyses described in the Appendix. Given that the “change” imperative substantially narrows the sample size already, I maximize my sample size by employing multiple imputation for measures missing values at a rate of one percent or more, here, just labor market income in 2007 and hours worked in 2007. In total, 220 women were missing values on labor market income, and 38 were missing values on hours worked.

**Identifying Mechanism of Missingness**

Before beginning any process of multiple imputation, a thorough examination of the observed and missing data is key. Under Little and Rubin’s well-known framework (see White, Royston, and Wood 2010), data may be: missing completely at random (MCAR), where the probability of missingness does not depend on any other data whether observed or not; missing at random (MAR), where the probability of the data being missing may depend on observed values, but not on values that are missing; or missing not at random (MNAR), wherein the missingness of the data depends on unobserved data (see also Schafer and Olson 1998). By definition, there is no true test for ascertaining whether a variable is MAR or MNAR; since MNAR indicates that a variable’s missingness is patterned by its true values, this assumption is impossible to verify, as true values are unknown to the imputer/analyst by virtue of their missingness (He, Zaslavsky, and Landrum 2010; White et al. 2010). Further, while it is possible to test whether missingness is associated with observed values, supporting a MAR classification,
but not possible to rule out whether missingness is also associated with unobserved values (Schafer and Olson 1998). Instead, a theoretically informed consideration of possible missingness patterns and tests to verify those considerations can lend support to a MAR classification.

For both variables imputed here, I first created binary indicators of missingness, where 0 indicates nonmissing values, and 1 indicates missing. An examination of missingness patterns revealed an arbitrary pattern (i.e., that data are not missing in nested patterns). Mechanisms of missingness were first tested with Little’s MCAR test (Li 2013; Fielding, Fayers, and Ramsay 2009) for the null hypothesis that the values are jointly MCAR and Li’s (2013) test of covariate-dependent missingness (CDM); the null is not rejected on the MCAR test ($p=0.361$), and nor on Li’s CDM test ($p=0.010$), suggesting that no pattern to the missing data has been identified.\(^{29}\)

Though missingness completely at random suggests that exclusion of all cases missing data would not bias findings, I continue with the imputation process in order to maximize the sample size of usable values. Further, because MCAR data are exceedingly rare in the social sciences, I treat the result of the MCAR tests as assurance that multiple imputation results will not be biased, and conduct some additional exploration into potential patterning of missingness.

To determine whether data are plausibly MAR, I examined correlations between a set of theoretically-informed variables and the binary indicators of missingness (as recommended by Institute for Digital Research and Education 2014) and with a series of t-tests and logistic regressions (as recommended by Social Science Computing Cooperative 2013b). Results from these tests indicate that missingness on each of these variables is at least partially patterned by

\(^{29}\) Little’s test determines whether the means of observed data vary according to pattern of missingness; if data are not MCAR, means will vary across missingness patterns (see Fielding et al. 2009). Li’s CDM test tests a special case of MCAR: that, given covariates $X$, missingness is independent of observed ($y^o$) and unobserved ($y^m$) dependent variable vectors (see Hedeker and Gibbons 2006).
observable values (as per MAR assumptions). For example, mean 2006 labor market income is significantly lower among those missing labor market income values for 2007. While complete case analysis would bias estimates upward (i.e., by excluding the seemingly lower-income missing cases), multiple imputation should eliminate this bias. That is, although missingness of labor income does not appear to be uncorrelated with its own values, Newsom (2012) points out “missingness can even be related to the variable with missing data, as long as that relationship can be accounted for by other variables in the dataset” (1). Indeed, the presence of fully observed labor market income variables from 2006 and 2008 in the imputation model strengthen the MAR assumption (though it should be noted that these findings do not preclude the possibility that missingness of income is related to other unobservable data). That the data appear somewhat patterned according to observable measures suggests that multiple imputation is feasible for calculating reasonable values, and useful for minimizing bias that could result from listwise deletion.

**Building Imputation Models**

Given that more than one variable needs to be imputed, that the data are missing in arbitrary patterns, and that the variables to be imputed are of mixed type (i.e., continuous and categorical), I relied on multiple imputation by chained equation (MICE) as the best-suited imputation option (see StataCorp 2013b; Marchenko 2011). Where a traditional imputation model estimates the joint distribution of all included variables, chained equations populate missing values across multiple variables in an iterative process, via a sequence of univariate imputation models. For each univariate model, the regression equation has “fully conditional specification” (StataCorp 2013b) in that each prediction equation includes all variables present, except the one being imputed. This process also has tremendous flexibility around model
specification for each individual equation (e.g., regression type, variable inclusion, subsamples) (ibid.).

Stata’s mi package offers nine regression methods for imputing a variable (StataCorp 2013b); generally, the imputer should select the method that would be appropriate for building any other type of model (e.g., regress for continuous, logit for binary). Each measure to be imputed here is continuous income, and hours are all continuous variables, none are normally distributed, and each is bounded, suggesting OLS regression might not be the most appropriate choice. As such, I rely on predictive mean matching (PMM), a semi-parametric method which regresses the incomplete variable on all covariates to calculate a predicted value (see van Buuren and Groothuis-Oudshoorn 2011). The predicted value is then compared to observed values in the data, where Stata identifies observations whose observed values are closest to the predicted value, and selects one at random to become the imputed value (see StataCorp 2013b). One benefit of PMM is that “if the observed values of a variable are not normal, PMM will usually produce a distribution of imputed values that matches the distribution of the observed values more closely than regression” (Social Science Computing Cooperative 2013a). One potential caution of this method is its application in small samples, where there are few possible values from which to impute (Royston and White 2011), though this is unlikely to be problematic here.

Next, I followed Royston and White’s (2011) recommendation for selecting variables for inclusion in the imputation model, and add measures associated with the probability of missingness and those that predict the variable’s observed values. Having already identified measures associated with missingness (see above), I used a similar strategy of correlation matrices and subsequent regression models (see Bouhlila and Sallaouti 2013) to identify
measures associated with observed values of the imputed variables. Per Royston and White (2011), imputation models also include all variables to be included in the final analysis models, including covariates and outcome variables.

After selecting variables for inclusion, I used Stata’s `dryrun` option to test each imputation model for appropriate specifications prior to actual imputation (Social Science Computing Cooperative 2013a). Plotting predicted values versus residuals in a scatter plot allowed for a visual inspection of model fit, and comparison of information criterion measures (AIC and BIC) assisted in assessing fit between different versions of the model. Because MICE is an iterative method, assessing convergence of iterations is key; beginning with Stata’s default 10 iterations (Stata refers to this as the “burn in” period), I constructed a series of trace plots—graphing the means and standard deviations of each imputed variable over iteration number—to inspect for trending of lines or irregular fluctuations that might indicate difficulty in convergence. With only 10 iterations, it was difficult to discern whether trending existed or not; increasing the burn-in period to 25 iterations produced a series of graphics (available upon request) that indicated convergence was not problematic (see StataCorp 2013b and Social Science Computing 2013c).

**Accounting for Complex Sample Design**

One complication in imputing these data is the PSID’s complex sample design. Multiple imputation in complex surveys is an emerging area of research, and though the complexity

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30 In this process, I identified one observed case for which the labor market income model fit very poorly, specifically, the respondent with the highest reported labor market income value, at $1,000,000. Inspecting her income values for surrounding years ($200,000 in 2006 and $50,000 in 2008), the $1,000,000 report seems especially unlikely. In this case, I topcode her income value to value of the 99th percentile ($150,000). It should also be noted that for variables imputed under PMM methods, there is no corresponding regression command that acknowledges the non-normal distribution; I follow SSCC’s recommendation to explore model fit with traditional OLS regression techniques, despite that the assumption of normal distribution is violated in an OLS model, and not applicable under the PMM method.
introduced by these survey designs is often acknowledged, specific guidance for proceeding is seldom issued, in theory (e.g., He et al. 2010; Ye 2009; Schafer and Olson 1998; Goldstein et al. 2009), or practice (see Barrington 2010 and Brownstone 1997, where each imputes PSID data but provide no detail on addressing complex sample design). When the imputation of data with a complex sample design is acknowledged, it is often in reference to clustered data that are not also stratified (e.g., Carpenter 2011; Eddings and Marchenko 2011). Stata’s own manual notes, “In the survey context, all structural variables such as sampling weights, strata, and cluster identifiers (or at least main strata and main clusters) need to be included in the imputation model” (StataCorp 2013b: 8). As MICE equations can be survey weighted, but not adjusted for cluster and strata like a traditional complex survey equation (i.e., svyset), I employ the strategy suggested by the Social Science Computing Cooperative (2013a), specifically that: “The current recommendation is to include survey structure variables like strata and PSU in the imputation models as sets of indicator variables.”

However, with 2 clusters and 87 stratum (63 of which contain women in the analytic sample here), the PSID’s sample design is complex enough to produce thin cells which result in problems of perfect prediction. Again, the Stata manual (StataCorp 2013b) provides some guidance here, warning, “To eliminate the issue of perfect prediction during imputation, we cannot, unfortunately, drop observations and variables when estimating model parameters as is normally done during estimation using, for example, the logit command. Doing so would violate one of the main requirements of imputation modeling: all variables and cases that may be used during primary, completed-data analysis must be included in the imputation model” (StataCorp 2013b:118). Suggested solutions from StataCorp include dropping observations from the final
model or using mi impute..., augment option\textsuperscript{31}, which is not a guaranteed solution (and indeed, was insufficient for resolving this issue here).

With little existing guidance on this highly specific issue, I draw on Reiter, Raghunathan, and Kinney (2006), who write: “In some surveys the design may be so complicated that it is impractical to include dummy variables for every cluster. In these cases, imputers can simplify the model for the design variables, for example, collapsing cluster categories or including proxy variables (e.g., cluster size) that are related to the outcome of interest” (Reiter et al. 2006:148). I also find recommendations from Heeringa, West, and Berglund (2010), whose broader advice for estimating variance in complex samples includes random reassignment of PSUs to larger strata, or collapsing adjacent strata. In the PSID, strata are assigned based on sample origin (e.g., core sample, immigrant sample, and Latino sample), as well as other subpopulation factors (race, income, geography) (Rahmani 2012; Morgan and Smith 1969). To increase the cell size of these strata without losing important design detail, I create three possible design variables by: (1) dividing the strata by sample type and within each sample type, creating quartiles based on strata size, and (2) dividing the strata by sample type and within each sample type, creating quartiles based on geographic region, and (3) dividing the strata by sample type, and within each, collapsing adjacent strata to form larger groups. Exploratory analyses with the three versions indicate that the final measure correlates most strongly with the original stratum variable (Kendall’s tau-b ($\tau_B$)=0.749, versus $\tau_B=-0.216$ for strata size and $\tau_B=0.223$ for regional options) and thus, is the version used here, though results from alternate specifications are similar.

\textsuperscript{31} The augment option adds a handful of additional observations with small weights during parameter estimation in a way that prevents perfect prediction. For more details (simulation and computational), see White et al. (2010).
Number of Imputations

To identify the number of imputations necessary, Rubin (1987) provides a formula for relative efficiency\(^{32}\): \(1/(1+F/m)\) where \(F\) is the fraction of missing information for the parameter being estimated and \(m\) is the number of imputations. However, as Allison (2012b) notes, “what’s good enough for efficiency isn’t necessarily good enough for standard error estimates, confidence intervals, and \(p\)-values” (1), in that too few imputations can lead to instability in the estimation of the variance of each parameter estimate across data sets. This is addressed by more recent suggestions—via simulation evidence (Bodner 2008) and approximation of the Monte Carlo error of the \(p\)-value (White et al. 2010)—that the number of imputations should be similar to the percentage of cases that are incomplete (see also Graham, Olchowski, and Gilreath 2007). With missing information for 7.96 percent of all women who were screened on sample characteristics and employed in 2007, I settled on 10 imputations \((m=10)\). Relative efficiency was 0.9988 for weekly work hours and 0.9928 for labor income. Monte Carlo errors, indicating the “standard deviation across repeated runs of the same imputation procedure with the same data” (White et al. 2010:387) meet criteria laid out by White and colleagues for the estimated parameter (\(\hat{\beta}\)), the test statistic and the \(p\)-value under \(m\) (ibid.).

Imputation Diagnostics

To assess the quality of resulting imputations, I follow the strategy laid out by Abayomi, Gelman, and Levy (2008) to assess the fit of the imputation model (described above), examine the distribution of the imputed variables for unusual or unreasonable values, and inspect displays of the imputed values versus the observed values. Using \(m\) kernel density plots, I overlay the distribution of the observed values with the imputed and completed values, finding

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\(^{32}\) “Relative efficiency” refers to the efficiency of using \(m\) imputations versus an infinite number of imputations (Yuan 2000; Allison 2012b).
that distributions are quite similar (see Figures 14 and 15 in Appendix, depicting the distribution of hours and income where $m=1$; figures from other iterations available upon request). Of course, slight variations in distribution are acceptable under the MAR assumption here, as described by van Buuren and Groothuis-Oudshoorn (2011): “Under MCAR, univariate distributions of the observed and imputed data are expected to be identical. Under MAR, they can be different, both in location and spread, but their multivariate distribution is assumed to be identical” (13; see also Abayomi et al. 2008).

Software and Weighting

First, although the analytic strategies used in Chapters III, IV, and V are discussed in detail in the upcoming (respective) chapters, it should be noted that I use the software package Stata (SE Version 12 and IC Version 13) for all analyses here. Unless otherwise noted, all analyses are weighted with the PSID’s 2009 family-level weight (longitudinal by design) and adjusted for the PSID’s complex survey design (clustered and stratified). All single-unit strata are centered at the grand mean, using Stata’s singleunit option. Standard errors are corrected with the Taylor series linearization method; as applied in Stata, this method is actually a Huber/White/robust sandwich variance estimator (StataCorp 2013a), and thus produces standard errors robust to heteroskedasticity.
III. Results: Prevalence and Patterning of Family Illness

In this chapter, I take several approaches to examining the patterning of family illness. I begin with descriptive analyses to explore the prevalence and patterning of illnesses within families, examining the prevalence of multiple chronic conditions within families, and the distribution of multiple illnesses across all families. Next, I describe constellations of family illness by disaggregating family illness counts into a typology of patterns that considers both the number of family members who report one or more chronic conditions, and the number of conditions that afflict a given member. This typology lends nuance to the count-style measure also used in these analyses by examining the ways in which different illness patterns can emerge across and within families. I also describe distributions of illness across women and spouses, and the distribution of eight specific illnesses within families, including how those illnesses co-occur within and across family members. Throughout this chapter, I examine how the aforementioned patterns of family illness are distributed across the spectrum of women’s educational attainment.

Unless otherwise noted, I use adjusted Wald tests, Pearson’s design-based F tests (with second-order Rao and Scott correction; see Scott 2007), and k-sample equality of medians tests throughout the descriptive analyses in this chapter. One exception is the conclusion of Chapter III, where I estimate negative binomial regressions as robustness checks to describe the distribution of family illness net of a host of important demographic measures. In examining each of these dimensions of family illness, my intent is to address the paucity of research that treats health as a family-level construct, to better understand how health conditions are distributed within and across family members, and to gain an initial understanding of the ways that family health may be related to women’s labor supply and labor market outcomes. Findings
from Chapter III shape decisions for inclusion in the regression models for each of the outcome variables in Chapters IV and V.

**Presence and Prevalence of Chronic Conditions in the Family**

Perhaps the most basic point of entry for these analyses is to explore the distribution of the number of chronic conditions in families, shown in Table 4. Among all families, more than three-quarters contain at least one person with a chronic condition, and two in five families have multiple chronic conditions. The range of illnesses is substantial, from zero to 25, and the mean number of conditions is more than two across all families (significantly higher in the two lowest educational categories than the two highest). The median number of conditions does not vary much from the mean, at 2 for all but the most educated families. Figure 2 displays the estimates from Table 4 graphically, in order to ease visualization of the distribution across educational categories.

Table 4. Distribution of Chronic Conditions in Family Unit by Women's Educational Attainment

<table>
<thead>
<tr>
<th>Number of Conditions</th>
<th>All Families</th>
<th>Less than High School</th>
<th>High School</th>
<th>Some College</th>
<th>College Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>22.38</td>
<td>18.14</td>
<td>19.37</td>
<td>21.08</td>
<td>28.02</td>
</tr>
<tr>
<td>1</td>
<td>20.95</td>
<td>16.94</td>
<td>20.22</td>
<td>21.02</td>
<td>22.87</td>
</tr>
<tr>
<td>2</td>
<td>18.69</td>
<td>21.02</td>
<td>17.68</td>
<td>19.79</td>
<td>18.21</td>
</tr>
<tr>
<td>3</td>
<td>12.39</td>
<td>9.43</td>
<td>14.05</td>
<td>14.47</td>
<td>9.67</td>
</tr>
<tr>
<td>4</td>
<td>8.79</td>
<td>11.48</td>
<td>8.94</td>
<td>8.26</td>
<td>8.3</td>
</tr>
<tr>
<td>5 or more</td>
<td>16.8</td>
<td>22.99</td>
<td>19.75</td>
<td>15.38</td>
<td>12.93</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>100.00</td>
<td>100.00</td>
<td>100.01</td>
<td>100.00</td>
<td>100.00</td>
</tr>
</tbody>
</table>

Mean (SE)  

<table>
<thead>
<tr>
<th></th>
<th>2.38 (0.07)</th>
<th>3.05 (0.22)</th>
<th>2.71 (0.11)</th>
<th>2.46 (0.11)</th>
<th>2.03 (0.07)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>2.00</td>
<td>2.00</td>
<td>2.00</td>
<td>2.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Range</td>
<td>0-25</td>
<td>0-17</td>
<td>0-22</td>
<td>0-25</td>
<td>0-16</td>
</tr>
</tbody>
</table>

*a Differences in mean number of conditions are statistically significant for all but the two lowest educational groups after adjustment with the Bonferroni correction.*
The above figure reveals particularly marked disparities between those with the highest and lowest educational attainment. For instance, whereas just 18.14 percent of the least educated women’s families report no chronic conditions, this is true of 28.02 percent of college-educated women’s families. At the other end of the illness spectrum, 12.93 percent of college families have five or more illnesses, compared with nearly 23 percent of the least-educated women’s families. In examining the mean number of conditions by family, college attendance emerges as an important delineator, with mean number of conditions similar across families wherein the woman has a high school diploma or less than a high school diploma. Taken together, these descriptive findings indicate early support for the unequal distribution of poor health by social class, using educational attainment as the proxy.

To explore the robustness of the association between women’s educational attainment and the distribution of family illness, I regress several basic demographic measures on the family condition count. Given the heavily skewed distribution of the count of family conditions, and the over-dispersion of the data \([\bar{x}=2.25; \ s^2=5.97]\), I estimate a negative binomial regression.
model (Table 5). First, results from this model suggest that the findings for education are robust beyond their bivariate association, and educational attainment remains a significant predictor of number of family conditions net of various family characteristics. Specifically, the expected log count of illnesses is substantially higher among all educational categories when compared to the reference category of college graduate families. Associations with family composition also emerge, though this is very likely a result of data structure. For example, having a young child in the house is associated with fewer expected conditions, as children under 5 participate in no survey. Similarly, children over 5 are eligible for TA or CDS participation, meaning that the association with condition counts is again likely a function of the data structure. Finally, the families of Hispanic/other/multiracial women have lower expected condition counts than their white counterparts, however it is unclear here whether this is an artifact of the data (e.g., perhaps these families are less likely to be diagnosed, rather than less likely to be ill, or less likely to participate in a survey) or a truly racially-stratified effect. The effects of race/ethnicity are considered more fully in the labor supply and earnings in Chapters IV and V. As an aside, because many of the measures in this model are also present in later models predicting labor supply and earnings, a significant relationship between these measures indicates the need to closely examine for potential collinearity in models where family condition count is also treated as an independent variable.

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33 The estimated dispersion parameter (lnalpha) is significantly greater than zero, suggesting that a negative binomial model is more suitable than an alternate Poisson estimation (H0=Poisson model is equally suitable; \( p<0.0001 \)), and `linktest` reveals no issue with model specification (\( p<0.241 \) for \( \_hatsq \)). Stata’s `linktest` operates under the assumption that if the model is correctly specified, additional significant predictors will only be discovered by chance. The test generates the linear predicted value (`\_hat`) and the square of that value (`\_hatsq`) from the last fit model, and rebuilds the model with the new variables as predictors. The linear predicted value (`\_hat`) should be significant, since it is the result of that same model, but the squared values should not. A statistically significant coefficient on `\_hatsq` most often suggests an omitted variable.
Table 5. Negative Binomial Regression of Number of Chronic Conditions in Family on Demographics

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>0.030 ***</td>
<td>(0.002)</td>
</tr>
<tr>
<td><strong>Educational Attainment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than High School</td>
<td>0.336 **</td>
<td>(0.100)</td>
</tr>
<tr>
<td>High School Graduate</td>
<td>0.183 **</td>
<td>(0.054)</td>
</tr>
<tr>
<td>Some College</td>
<td>0.180 ***</td>
<td>(0.044)</td>
</tr>
<tr>
<td>College Graduate</td>
<td>Ref.</td>
<td></td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>Ref.</td>
<td></td>
</tr>
<tr>
<td>Previously Married</td>
<td>0.187</td>
<td>(0.128)</td>
</tr>
<tr>
<td>Never Married</td>
<td>0.113</td>
<td>(0.073)</td>
</tr>
<tr>
<td><strong>Number in Family Unit</strong></td>
<td>0.116 ***</td>
<td>(0.022)</td>
</tr>
<tr>
<td><strong>Age of Youngest Child</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Children</td>
<td>Ref.</td>
<td></td>
</tr>
<tr>
<td>Child(ren) Under Age 5</td>
<td>-0.281 ***</td>
<td>(0.068)</td>
</tr>
<tr>
<td>Child(ren) Over Age 5</td>
<td>0.162 *</td>
<td>(0.063)</td>
</tr>
<tr>
<td><strong>Lives in a Metropolitan Area</strong></td>
<td>-0.037</td>
<td>(0.057)</td>
</tr>
<tr>
<td><strong>Region of Residence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td>Ref.</td>
<td></td>
</tr>
<tr>
<td>North Central</td>
<td>0.028</td>
<td>(0.081)</td>
</tr>
<tr>
<td>South</td>
<td>0.065</td>
<td>(0.063)</td>
</tr>
<tr>
<td>West</td>
<td>-0.018</td>
<td>(0.095)</td>
</tr>
<tr>
<td><strong>Race/Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td>Ref.</td>
<td></td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>-0.071</td>
<td>(0.059)</td>
</tr>
<tr>
<td>Hispanic/Other/Multiracial</td>
<td>-0.244 **</td>
<td>(0.073)</td>
</tr>
<tr>
<td><strong>Family Income, 2006</strong></td>
<td>-0.001</td>
<td>(0.000)</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>0.345 **</td>
<td>(0.117)</td>
</tr>
<tr>
<td>Inalpha</td>
<td>-0.787 ***</td>
<td>(0.062)</td>
</tr>
<tr>
<td><strong>Overall F test</strong></td>
<td>30.121 ***</td>
<td></td>
</tr>
<tr>
<td><strong>N</strong></td>
<td>3,945</td>
<td></td>
</tr>
</tbody>
</table>

* p<0.05,  ** p<0.01,  *** p<0.001

In thousands of dollars.

From the results in Table 5, I also calculate predictive margins by educational attainment. These margins allow for easy estimation of predicted condition counts adjusting for characteristics in a regression model, and allow for testing between specific model predictions (such as differences in the count by women’s educational attainment). As Figure 3 shows, the
predicted number of conditions ranges from 2.98 for the least-educated families to 2.13 among the most educated. However, adjusted Wald tests reveal that differences in predicted numbers of conditions are only significant for college graduates, and all other counts are statistically similar.

Figure 3. Predicted Number of Family Conditions by Women's Educational Attainment, Net of Other Demographic Characteristics

Note: Predictive margins adjusted for characteristics shown in Table 5. Error bars indicate 95 percent confidence intervals. Differences between educational categories are only significant between college graduates and others after pairwise comparison with Bonferroni correction.

**Typology of Within-Family Illnesses**

Beyond simple counts of chronic conditions within the family, I also classify families into a typology of family health that explores categories of multiple illnesses within and across family members. Table 6 displays the results of this six-pattern classification method by educational attainment. Building upon the condition counts, the table helps to demonstrate the intra-family distribution of illnesses shown in Table 4. First, as in Table 4, this classification shows that more than half of college graduate families have no illnesses or a single person with a single illness in their family (50.89 percent; Patterns 1 and 6). In comparison, this is only true of 35.08 percent of the lowest educated families. Table 6 also demonstrates a heightened share of families with
multiple illnesses within a single FUM (Pattern 3) among high school graduates’ families, and a high share of families where multiple members have mixed numbers of diagnoses (Pattern 5) among the least-educated families. A chi-square test indicates that the association between the typology and educational attainment is statistically significant ($p<0.001$).

Table 6. Patterns of Within-Family Illness Distribution by Educational Attainment

<table>
<thead>
<tr>
<th>Pattern Number</th>
<th>Number of FUMs Diagnosed</th>
<th>Number of Conditions Each</th>
<th>All Families</th>
<th>Less than High School</th>
<th>High School</th>
<th>Some College</th>
<th>College Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>One</td>
<td>One</td>
<td>20.95</td>
<td>16.94</td>
<td>20.22</td>
<td>21.02</td>
<td>22.87</td>
</tr>
<tr>
<td>2</td>
<td>Multiple</td>
<td>One</td>
<td>11.17</td>
<td>13.61</td>
<td>9.26</td>
<td>12.72</td>
<td>11.31</td>
</tr>
<tr>
<td>3</td>
<td>One</td>
<td>Multiple</td>
<td>13.84</td>
<td>14.97</td>
<td><strong>16.56</strong></td>
<td>13.41</td>
<td>10.86</td>
</tr>
<tr>
<td>4</td>
<td>Multiple</td>
<td>Multiple</td>
<td>11.87</td>
<td>12.66</td>
<td>13.84</td>
<td>12.23</td>
<td>9.16</td>
</tr>
<tr>
<td>5</td>
<td>Multiple</td>
<td>Some one, some multiple</td>
<td>19.78</td>
<td>23.69</td>
<td>20.75</td>
<td>19.54</td>
<td>17.78</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>None</td>
<td>22.38</td>
<td><strong>18.14</strong></td>
<td>19.37</td>
<td>21.08</td>
<td>28.02</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td><strong>100.00</strong></td>
<td><strong>100.00</strong></td>
<td><strong>100.00</strong></td>
<td><strong>100.00</strong></td>
<td><strong>100.00</strong></td>
</tr>
</tbody>
</table>

Note: Cells in bold font are those that retained some association after controlling for other demographics.

To determine robustness of the associations between education and the illness typology, I employ a series of logistic models, each predicting a specific illness pattern (numbers 1-6). I find that, net of basic demographics (age, marital status, number in FU, presence of young children in the FU, metropolitan residence, region of residence, race/ethnicity, and family income), associations between educational attainment and specific patterns are not robust (not shown; available upon request). Two exceptions to this finding are in predicting families with no conditions (Pattern 6), in which a less than high school education is associated with a near

---

34 Rather than testing the effects of educational attainment in a single multinomial logit model, I use a series of logistic models here to facilitate testing the joint significance of educational attainment (entered as a factor variable) and equality of coefficients across educational categories. While a multinomial model would be necessary for yielding the full variance-covariance matrix necessary for performing tests across models (see Heeringa et al. 2010), these cross-model comparisons were not of central interest here. However, I also estimate a multinomial logit model for comparison: though coefficients are very similar and standard errors are slightly larger in the multinominal version, substantive results do not vary from those via separate logistic models.

35 In the remainder of this section, I test the robustness of bivariate associations between health measures and women’s education in by regressing health measures on demographic characteristics. Because health measures are only of interest as descriptive and independent measures in this dissertation, I do not clutter this chapter with results of each model, but rather describe whether basic demographics mediate bivariate relationships between health and women’s educational attainment.
40-percent reduction in the odds of this family pattern versus a college education (OR=0.617; \( p=0.028 \)), and in predicting a single member with multiple illnesses (Pattern 3), where a high school education nearly doubles these odds (OR=1.482; \( p=0.016 \)). Despite these few associations, it is unconvincing that this measure could substantially improve upon the simple condition count, especially since Patterns 1 and 6 (one condition and no conditions) are also estimable via the former measure. Further, these illness patterns do not have the consistent and intuitive interpretation of the family condition count; these patterns may be useful for descriptive purposes, but are likely too diffuse for practical application in later models.

**Illness among Women and Spouses**

To examine the distribution of illness among specific family unit members (FUMs), I limit my estimation to women and spouses, all of whom have health condition data. Because many children and OFUMs were not surveyed about their illnesses, resulting estimates are unlikely to be usefully applicable to all families containing children or OFUMs. Table 7 shows that among all families, 51.31 percent of women have one or more chronic conditions, and among families where a spouse is present, 53.73 percent of spouses have one or more chronic conditions. For both women and their spouses, mean number of conditions varies by women’s educational attainment; this relationship appears linear for women (i.e., higher categories of education consistently associated with lower mean conditions). For spouses, there is no difference in mean conditions in families with and without a high school diploma (Bonferroni-adjusted \( p=0.669 \)), indicating the possibility of some nonlinear relationship. This indicates a potentially nonlinear relationship between women’s education and spouses’ chronic conditions. Finally, in comparing the distribution of men and women’s conditions, it is worth noting that the share with no illnesses is much more similar among college graduate women (52.93 percent of
women and 51.03 percent of their spouses) than among the least educated women (41.23 percent of women versus 48.09 percent of spouses). Whether this is related to rates of diagnoses via classed and gendered help-seeking behaviors or some true etiology is unclear.

Table 7. Distribution of Condition Counts among Women and Spouses

<table>
<thead>
<tr>
<th>Women's Number of Conditions</th>
<th>All Families</th>
<th>Less than High School</th>
<th>High School Graduate</th>
<th>Some College</th>
<th>College Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>48.71</td>
<td>41.23</td>
<td>46.36</td>
<td>49.34</td>
<td>52.93</td>
</tr>
<tr>
<td>1</td>
<td>27.13</td>
<td>27.14</td>
<td>25.99</td>
<td>27.01</td>
<td>28.49</td>
</tr>
<tr>
<td>2</td>
<td>13.10</td>
<td>13.85</td>
<td>15.18</td>
<td>12.42</td>
<td>11.12</td>
</tr>
<tr>
<td>3</td>
<td>5.77</td>
<td>5.62</td>
<td>6.32</td>
<td>6.12</td>
<td>4.90</td>
</tr>
<tr>
<td>4</td>
<td>2.74</td>
<td>4.85</td>
<td>3.63</td>
<td>1.98</td>
<td>1.77</td>
</tr>
<tr>
<td>5 or more</td>
<td>2.57</td>
<td>7.31</td>
<td>2.52</td>
<td>3.12</td>
<td>0.78</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
</tr>
<tr>
<td>Mean (SE)</td>
<td>0.967 (0.031)</td>
<td>1.343 (0.102)</td>
<td>1.050 (0.067)</td>
<td>0.963 (0.049)</td>
<td>0.769 (0.045)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spouses' Number of Conditions</th>
<th>All Families</th>
<th>Less than High School</th>
<th>High School Graduate</th>
<th>Some College</th>
<th>College Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>46.28</td>
<td>48.09</td>
<td>42.45</td>
<td>44.78</td>
<td>51.03</td>
</tr>
<tr>
<td>1</td>
<td>28.31</td>
<td>28.39</td>
<td>27.11</td>
<td>29.74</td>
<td>28.40</td>
</tr>
<tr>
<td>3</td>
<td>6.21</td>
<td>5.45</td>
<td>7.83</td>
<td>5.82</td>
<td>5.02</td>
</tr>
<tr>
<td>4</td>
<td>2.62</td>
<td>2.38</td>
<td>3.39</td>
<td>3.10</td>
<td>1.51</td>
</tr>
<tr>
<td>5 or more</td>
<td>2.74</td>
<td>4.79</td>
<td>4.89</td>
<td>1.56</td>
<td>0.92</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
<td>100.00</td>
</tr>
<tr>
<td>Mean (SE)</td>
<td>0.917 (0.034)</td>
<td>1.055 (0.102)</td>
<td>1.209 (0.067)</td>
<td>0.984 (0.049)</td>
<td>0.813 (0.045)</td>
</tr>
</tbody>
</table>

† Of families with a spouse or partner present.

a Differences in mean number of conditions are statistically significant for all but the middle two educational categories (p<0.05).

b Differences in mean number of conditions are statistically significant for all but the first two educational categories (p<0.05).

As in the previous sections, I explore the bivariate relationships described above with a series of regression models (negative binomial here) that test women’s educational attainment as a predictor of the women’s and spouses’ condition counts (not shown; available upon request). For women, age, being unmarried, and having no high school diploma are associated with higher expected condition counts, while having young children and being Hispanic/other/multiracial is associated with fewer conditions. Though the relationship between women’s conditions and educational attainment remains net of other demographics, it is worth
noting that the difference in predicted number of conditions is small between the most and least educated; contrasting the predictive margins at each education value suggests just 0.424 conditions predicted for college graduates (1.273 versus 0.849, \( p=0.001 \)).

For spouses, women’s age and educational attainment are the only significant predictors of condition counts. However, in this case, spouses of non-high school graduates have similar expected log counts to spouses of college graduates, with significantly higher condition counts among those with high school diplomas and some college (\( p=0.005 \) for each). These associations raise questions about the gendered mechanisms of diagnosis, as well as the need to allow for potentially nonlinear associations when examining spouses’ conditions in later models.

**Distribution of Specific Conditions in the Family**

Beyond examining illness counts and patterns, I also document the prevalence of eight specific illnesses among all FUMs with health measures (see Figure 1), displayed in Table 8, below. Existing research has shown that the most common chronic condition among individuals is hypertension (Boyd et al. 2010d; Centers for Medicare and Medicaid Services 2012). Indeed this pattern emerges at the family-level as well, as 39.94 percent of families contain one or more people with this diagnosis (see Table 8). This estimate tracks relatively well with existing literature at the individual level, indicating that hypertension afflicts between 29 and 34 percent of Americans (Anderson 2010; Egan, Zhao, and Axon 2010; Gallup-Healthways 2012; Gillespie and Hurvitz 2013; Joffres et al. 2013; Roger et al. 2011) (see Table 18 in Appendix for table describing population prevalence of various illnesses). The distribution of hypertension varies by women’s educational attainment here: rates among college graduates’ families are similar to the national individual-level rates (33.59 percent), but rates in high school educated women’s families near 45 percent. Across all families, a near-two-in-five rate of hypertension is
troublesome, both because this sample explicitly excludes families headed by seniors (given the age restriction on women in this sample) and because hypertension itself can be a precursor to a host of other serious chronic illnesses (ibid.).

Table 8. Prevalence of Specific Diagnoses in Family Unit by Women’s Educational Attainment

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>All Families</th>
<th>Less than High School</th>
<th>High School Graduate</th>
<th>Some College</th>
<th>College Graduate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>39.94</td>
<td>41.27</td>
<td>44.78</td>
<td>40.62</td>
<td>33.59 ***</td>
</tr>
<tr>
<td>Asthma</td>
<td>21.34</td>
<td>28.78</td>
<td>20.90</td>
<td>20.65</td>
<td>20.27</td>
</tr>
<tr>
<td>Diabetes</td>
<td>14.71</td>
<td>24.01</td>
<td>17.85</td>
<td>14.41</td>
<td>8.70 ***</td>
</tr>
<tr>
<td>Arthritis</td>
<td>22.22</td>
<td>25.73</td>
<td>26.13</td>
<td>20.84</td>
<td>18.03 ***</td>
</tr>
<tr>
<td>Cancer</td>
<td>9.73</td>
<td>7.07</td>
<td>10.22</td>
<td>9.78</td>
<td>9.80</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>6.09</td>
<td>11.79</td>
<td>7.77</td>
<td>4.55</td>
<td>3.89 ***</td>
</tr>
<tr>
<td>Anxiety</td>
<td>5.34</td>
<td>8.00</td>
<td>6.54</td>
<td>4.68</td>
<td>3.80 *</td>
</tr>
</tbody>
</table>

Note: Asterisks indicate results of adjusted chi-square tests between the binary indicators of diagnosis at the family-level (i.e., whether or not someone in FU was diagnosed with depression) and women’s educational attainment. Cell contents refer to percent of families within an educational category in which one or more family members reports a given diagnosis (i.e., 14.04 percent of non-high school graduate women’s families have one or more members who report a depression diagnoses). * p<0.05; ** p<0.01; *** p<0.001

The next most common diagnoses in this sample—arthritis and asthma—emerge in proportions about half as high as hypertension, around 22 and 21 percent, respectively.

Population estimates of arthritis range from 22.7 percent to 10.9 percent (Barbour et al. 2013 Bolen et al. 2010; Margaretten et al. 2013; Ornstein et al. 2013), and from 11.7 percent to 8.4 percent for asthma (Anderson 2010; Gallup-Healthways 2012; Moorman et al. 2012; Ornstein et al. 2013; Xu et al. 2013). While arthritis estimates here fall at the high end of the spectrum of population estimates, asthma rates among these families are much higher than national estimates. Although asthma prevalence is much higher among children (Akinbami et al. 2012; American College of Allergy, Asthma, and Immunology 2014; Asthma and Allergy Foundation of America 2014) and it is not unusual that the prevalence rates here would exceed individual-

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36 Not only are the oldest populations excluded from heading households here, but also recall Table 2, which notes that parents or other (potentially older) relatives in the FU do not have health data.
level adult-only samples (e.g., Gallup-Healthways 2012; Ornstein et al. 2013), the family-level rate of asthma is exceptionally high here.

Like hypertension, arthritis diagnoses vary by women’s educational attainment too, ranging from about one-quarter of families with no college to 18 percent of college graduates’ families. These rates concur with educational variation in diagnoses in the population, ranging from 25.7 percent among non-high school graduates to 18.3 percent among college graduates (Barbour et al. 2013). The mechanisms by which arthritis disproportionately impacts low educated groups are unclear (e.g., Callahan et al. 2008), though research has hypothesized on the role of community resources like safe spaces for exercise and access to health care, and engagement in certain occupations as a mediators in the relationship between poverty and arthritis (Callahan et al. 2011).

Asthma rates appear remarkably similar across all families with at least a high school diploma (around 20-21 percent), elevated only among families where women have not graduated from high school (28.78 percent) though the association between women’s education and asthma diagnosis in the family just misses the cutoff for statistical significance ($p<0.055$). Heightened rates of asthma among less-educated populations would not be surprising, given that asthma can be triggered by allergens including the types of natural (e.g., pollen, dust mites) and industrial environmental factors (e.g., chemicals, paint and gasoline fumes) that might be encountered in unskilled jobs. Further, even if the presence of asthma is not significantly associated with educational attainment, the exposure to certain environmental factors may lead to stratified implications of an asthma diagnosis. For example, Moorman et al. (2012) show that among people with asthma diagnoses, persons living below the poverty line are more likely to
have had an asthma attack in the past 12 months than their non-poor counterparts (55 versus 48 percent).

Relatedly, two other diagnoses associated with educational attainment—lung disease and diabetes—may also have environmental or behavioral components that can explain their diagnostic stratification. Each diagnosis tracks very well with national estimates, but occurs among the least educated families at rates more than twice as high as among the most educated families. Existing research has demonstrated links between social class and the diagnosis of both of these illnesses, largely functioning through the mechanisms of health behavior (e.g., lung disease and cigarette smoking, and diabetes and the availability of nutritious food) (see, for example, American Lung Association 2012; Burney et al. 2013; Chaufan, Davis, and Constantino 2011; Levine 2011; Mezuk et al. 2008). Finally, anxiety diagnoses also vary by educational attainment, though the mechanisms by which this association occurs is less clear. Much of the existing research explores anxiety as a precursor to educational attainment, and uses anxiety diagnoses to predict school termination (e.g., Breslau et al. 2008; Van Ameringen, Mancini, and Farvolden 2003; Kessler et al. 1995), though it is possible that stressors associated with low educated populations also result in heightened levels of anxiety.

Finally, though not related to educational attainment, rates of depression in this sample fall within the documented range at the population level. However, the other diagnosis not associated with education here—cancer—is present at a substantially lower rate than among population levels, at less than ten percent here, versus close to 40 percent population wide. However, this again may be related to the age of this sample, as most cancer estimates are among all age groups and are assessed as lifetime measures.
As in previous sections, I estimate a series of logistic regression models to determine whether the bivariate associations in Table 8 persist, net of demographic characteristics.\textsuperscript{37} Since the goal here is to explore the robustness of associations between educational attainment and specific diagnoses for use in later models where both education and diagnoses are independent variables, my interest here does not lie in identifying minute patterns of specific diagnostic predictors. As such, I do not detail the demographic predictors of each diagnosis here and instead, note that the bivariate associations from Table 8 remain, net of other demographics. Instead, I focus here on a finding more relevant to this purpose; that is, the multivariate relationship between educational attainment and each diagnosis can be described in one of two ways. In the first, a clear stratification between the most and least educated emerges, with high school graduates and those with some college falling somewhere in between, and similar to each other. This pattern describes the relationship between education and anxiety, arthritis, and lung disease. In the second pattern, the division is between college graduates and all others, where the predicted probability of diagnosis is similar across all categories except the most educated, and is the case for diabetes and hypertension. Figure 4 shows the predicted probability of hypertension and arthritis diagnoses—diagnoses that exemplify the patterns described above—by educational attainment.

\textsuperscript{37} I control for age, marital status, number of people in FU, presence of young children in FU, metropolitan status, region of residence, and race/ethnicity. I also test for a curvilinear impact of age and an interaction of black race/ethnicity x Southern residence, omitting each where not significant. Finally, I also include family income from 2006 in each model to determine whether income explains away the effects of educational attainment. For each diagnosis, the inclusion of income reduces the odds ratio on employment, sometimes to the point of non-significance, but does not change the overall conclusions. The sole exception to this pattern is in predicting diabetes, where including family income in the model does not produce any change in the odds ratios or p-values on educational attainment. For the results described here, family income has been omitted.
Co-occurring Diagnoses in the Family Unit

Table 9 demonstrates the rates of co-occurring diagnoses at the family level. The left-hand column identifies a particular condition, joined with other conditions in a matrix of illnesses that may also appear in a given family. For example, of families with a diagnosis of depression among one or more members, 51.1 percent also have a diagnosis of hypertension in the family, as compared with 38.31 percent of families without a depression diagnosis. These associations are at the family level, such that co-occurring diagnoses may be within a single person or multiple people; the purpose is to better understand how multiple illnesses within a family might be patterned, and whether specific illness pairs might warrant particular attention within families. Of course, this table provides a basis for exploring family-level diagnoses, but is also followed by Table 10, which documents the co-occurrence of illness across different family unit members, to help untangle inter- and intra-individual contributions to co-occurring diagnoses at the family level.
Table 9. Prevalence of Co-Occurring Illnesses within Family Units

<table>
<thead>
<tr>
<th>Present in FU</th>
<th>Depression</th>
<th>Hypertension</th>
<th>Asthma</th>
<th>Diabetes</th>
<th>Arthritis</th>
<th>Cancer</th>
<th>Lung Disease</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>-</td>
<td>38.31</td>
<td>19.28</td>
<td>13.75</td>
<td>20.63</td>
<td>9.25</td>
<td>5.60</td>
<td>3.01</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>51.10</td>
<td>35.46</td>
<td>21.34</td>
<td>33.18</td>
<td>13.06</td>
<td>9.46</td>
<td>21.35</td>
</tr>
<tr>
<td>Hypertension</td>
<td>No</td>
<td>10.35</td>
<td>-</td>
<td>19.41</td>
<td>6.89</td>
<td>14.09</td>
<td>7.40</td>
<td>4.03</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>16.26</td>
<td>24.24</td>
<td>26.48</td>
<td>34.46</td>
<td>13.23</td>
<td>9.19</td>
<td>5.42</td>
</tr>
<tr>
<td>Asthma</td>
<td>No</td>
<td>10.43</td>
<td>38.46</td>
<td>14.37</td>
<td>20.70</td>
<td>9.70</td>
<td>3.59</td>
<td>4.38</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>21.12</td>
<td>45.37</td>
<td>15.96</td>
<td>27.83</td>
<td>9.83</td>
<td>15.32</td>
<td>8.90</td>
</tr>
<tr>
<td>Diabetes</td>
<td>No</td>
<td>11.72</td>
<td>34.43</td>
<td>21.03</td>
<td>18.74</td>
<td>8.59</td>
<td>4.93</td>
<td>4.80</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>18.43</td>
<td>71.89</td>
<td>23.15</td>
<td>42.41</td>
<td>16.32</td>
<td>12.84</td>
<td>8.50</td>
</tr>
<tr>
<td>Arthritis</td>
<td>No</td>
<td>10.92</td>
<td>33.65</td>
<td>19.80</td>
<td>10.89</td>
<td>7.89</td>
<td>3.60</td>
<td>4.83</td>
</tr>
<tr>
<td>Cancer</td>
<td>No</td>
<td>12.24</td>
<td>38.39</td>
<td>21.31</td>
<td>13.64</td>
<td>20.64</td>
<td>5.78</td>
<td>5.19</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>17.05</td>
<td>54.31</td>
<td>21.56</td>
<td>24.68</td>
<td>36.94</td>
<td>9.02</td>
<td>6.76</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>No</td>
<td>12.25</td>
<td>38.62</td>
<td>19.24</td>
<td>13.65</td>
<td>20.16</td>
<td>9.43</td>
<td>5.07</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>19.74</td>
<td>60.23</td>
<td>53.66</td>
<td>31.01</td>
<td>54.06</td>
<td>14.40</td>
<td>9.58</td>
</tr>
<tr>
<td>Anxiety</td>
<td>No</td>
<td>10.56</td>
<td>39.90</td>
<td>20.54</td>
<td>14.22</td>
<td>21.81</td>
<td>9.58</td>
<td>5.82</td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td>50.78</td>
<td>40.51</td>
<td>35.55</td>
<td>23.40</td>
<td>29.65</td>
<td>12.31</td>
<td>10.93</td>
</tr>
</tbody>
</table>

Note: This table should be read as "In family units where [row condition is true], X percent also have [column condition]. Asterisks indicate results of adjusted chi-square tests between binary indicators of diagnoses at the family-level.

* p<0.05; ** p<0.01; *** p<0.001

Across all eight illnesses examined here, nearly all are associated with the presence of other diagnoses, and in all cases, the presence of one illness is associated with heightened, rather than reduced, rates of another. For instance, in families with at least one diabetic member, 71.89 percent also have at least one hypertensive member. In comparison, just 34.43 percent of families with no diabetes diagnosis have a hypertension diagnosis. In some cases, illnesses co-occur in expected patterns: for example, hypertension and diabetes are a well-known interrelated comorbid pair at the individual level, (e.g., Barnett 1994; Waeb, Feihl, and Ruilope 2001) and these family level measures are sensitive to those co-occurrences (since the table allows for co-occurring pairs to be inter- or intra-individual). Rates among these families are slightly higher than population estimates—for example, existing literature suggests that hypertension occurs in one-half to two-thirds of diabetics (Boyd et al. 2010c; Partnership for Solutions 2004a).

In this sample, depression is reliably associated with other diagnoses, with heightened rates emerging among families diagnosed with each of the seven other illnesses. Among families with an anxiety diagnosis, depression is especially prominent, present among more than half of
those families (50.78 percent). These findings are precisely in line with existing research, and Aina and Susman (2006) note “comorbidity is the rule with anxiety and depressive disorders. Anxiety and depressive disorder are often comorbid with each other…[and] are frequently found coexisting with long-standing chronic medical conditions such as cardiovascular disease and diabetes mellitus” (S9). The relationship between these illnesses and other chronic conditions appears to be complex and interactive. Aina and Susman (2006) note, “anxiety and depression may negatively influence the outcomes of medical illness and many medical problems increase the risk of suffering from depression and anxiety” (S11).

In some cases, these dual diagnoses may be related to a common underlying predictor, such as nutrition, exercise habits, community features, or genetic predisposition. In other cases, the link between illnesses may be a direct function of an illness feature, as when complications of diabetes (such as the progressive kidney disorder diabetes nephropathy) raise diabetics’ risk of hypertension (e.g., Sowers and Epstein 1995). Other potential connective mechanisms include heightened risk for one condition associated with the drug therapy for another condition (e.g., increased risk for chest-, skeletal-, and gastrointestinal-related symptoms as a function of certain rheumatoid arthritis treatments; see Gullick and Scott 2011).

Table 10 extends the descriptions in Table 9 by examining illnesses that co-occur across different family unit members. This table allows for quantification of families in which multiple members have been diagnosed with the same condition; these instances raise questions about the roles that both genetic and environmental factors play in health. Further, these estimates answer a question implicitly raised by Table 9: that is, whether illnesses co-occurring at the family level are simply a result of co-occurring illnesses within specific individuals. In other
words, where Table 9 might obscure the distinction between illnesses distributed within or across specific family members, Table 10 explicitly addresses these patterns.

Table 10. Prevalence of Co-occurring Illnesses within Different FUMs

<table>
<thead>
<tr>
<th>Condition</th>
<th>Depression</th>
<th>Hypertension</th>
<th>Asthma</th>
<th>Diabetes</th>
<th>Arthritis</th>
<th>Cancer</th>
<th>Lung Disease</th>
<th>Anxiety</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Of Those Ever</strong></td>
<td><strong>Diagnosed</strong></td>
<td><strong>With…</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>No</td>
<td>9.08</td>
<td>16.42</td>
<td>9.61</td>
<td>13.84</td>
<td>5.65</td>
<td>3.89</td>
<td>4.15</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>25.04</td>
<td>32.26</td>
<td>20.05</td>
<td>12.95</td>
<td>18.80</td>
<td>7.57</td>
<td>5.04</td>
</tr>
<tr>
<td>Hypertension</td>
<td>No</td>
<td>9.79</td>
<td>17.00</td>
<td>9.41</td>
<td>12.92</td>
<td>5.51</td>
<td>3.87</td>
<td>4.71</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>27.46</td>
<td>14.51</td>
<td>11.73</td>
<td>20.45</td>
<td>6.95</td>
<td>4.31</td>
<td>2.59</td>
</tr>
<tr>
<td>Asthma</td>
<td>No</td>
<td>9.47</td>
<td>16.01</td>
<td>9.80</td>
<td>13.60</td>
<td>5.70</td>
<td>3.78</td>
<td>4.30</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>13.68</td>
<td>24.11</td>
<td>9.29</td>
<td>19.69</td>
<td>6.13</td>
<td>5.87</td>
<td>5.55</td>
</tr>
<tr>
<td>Diabetes</td>
<td>No</td>
<td>9.72</td>
<td>16.80</td>
<td>9.66</td>
<td>13.46</td>
<td>5.54</td>
<td>3.82</td>
<td>4.40</td>
</tr>
<tr>
<td>Arthritis</td>
<td>No</td>
<td>9.82</td>
<td>16.67</td>
<td>9.30</td>
<td>12.73</td>
<td>5.49</td>
<td>3.64</td>
<td>4.50</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>17.36</td>
<td>13.91</td>
<td>11.61</td>
<td>25.73</td>
<td>9.30</td>
<td>6.19</td>
<td>4.19</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>11.09</td>
<td>13.24</td>
<td>14.76</td>
<td>21.88</td>
<td>10.52</td>
<td>5.12</td>
<td>3.68</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>No</td>
<td>9.78</td>
<td>16.54</td>
<td>9.64</td>
<td>13.73</td>
<td>5.67</td>
<td>3.91</td>
<td>4.40</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>10.50</td>
<td>20.74</td>
<td>15.39</td>
<td>30.51</td>
<td>8.53</td>
<td>5.64</td>
<td>4.27</td>
</tr>
<tr>
<td>Anxiety</td>
<td>No</td>
<td>9.58</td>
<td>16.59</td>
<td>9.72</td>
<td>14.05</td>
<td>5.73</td>
<td>3.93</td>
<td>4.26</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>21.09</td>
<td>18.69</td>
<td>11.94</td>
<td>14.93</td>
<td>6.05</td>
<td>4.34</td>
<td>11.57</td>
</tr>
</tbody>
</table>

* p<0.05; ** p<0.01; *** p<0.001

Note: This analysis is at the family-member level, rather than the family unit level and is weighted accordingly. Adjusted chi-square results refer to the association between one FUM's own diagnosis and the presence of the same diagnosis in another member of the same FU.

Table 10 also displays co-occurring diagnoses of the same condition across different family members, finding associations between diagnoses of depression, hypertension, asthma, arthritis and anxiety among different family members. One of the most striking patterns in this table might be the difference in the share of people living with someone with depression by own depression diagnosis. The table shows that 25.04 percent of people diagnosed with depression live with someone with depression, versus 9.08 percent of people who have not been diagnosed with depression themselves. For anxiety, the other non-physical condition examined here, the distribution is similarly patterned, though not as stark. These differences raise questions about shared environmental influences, genetic predispositions, and medical help-seeking. For instance, although research shows that depression has genetic components, it is also possible that the family-level clustering in this diagnosis is more related to diagnosis, in
that seeing a family member seek psychiatric diagnosis and treatment might acclimatize other individuals in the family to take action. Because people with depression are also more likely to live with a person with anxiety, and vice versa, the latter explanation might indeed extend to understanding various co-occurring diagnoses. In other cases, co-occurring illnesses at the household level may be explained away by demographic factors like age. For example, Barbour et al. (2013) report that arthritis afflicts 7.3 percent of adults aged 18-44, and 30.3 percent of adults aged 45-64. If distribution in this sample is similar, it is possible that controlling for age would reduce the odds of co-occurring arthritis in the family (described below).

Differences between Tables 9 and 10 may also demonstrate instances in which co-occurring diagnoses are confined to a single family member. For instance, Table 9 showed associations between all physical conditions and depression; that all associations (except asthma) have disappeared in Table 10 suggests that most of the associations were within-person. This interpretation is strengthened by a substantial literature indicating that physical illness leads to increased risk of depression, and vice-versa (e.g., Centers for Disease Control and Prevention 2010).

It is somewhat surprising that there are no significant associations between different family members’ diagnoses of diabetes and lung disease, especially given the role of “lifestyle factors” such as smoking and diet for these conditions in particular. That cancer is not associated with other cancer diagnoses in the family unit is unsurprising; in fact, given the broad spectrum of diagnoses that “cancer” can encompass, it is not unreasonable to wonder whether “cancer” is too broad a diagnostic category to be meaningfully patterned.

Unlike in previous sections, I do not examine each co-occurring illness pair in a multivariate framework here, given the sheer size of the co-occurrence matrix (including 64
pairs), and the fact that some co-occurring pairs affect a small share of families, and thus should only be treated descriptively. Instead, Tables 9 and 10 are intended to be informative in their own right, especially for considering how different illnesses might co-occur in ways that have particular implications for employment. Accordingly, in the following chapters, I consider how co-occurring diagnoses are related to labor supply and earnings, and where bivariate associations (and sufficient sample sizes) emerge, I employ these measures in models predicting labor supply and earnings.

In this chapter, I sought to document patterns of family illness. This process served several purposes: first, to provide a starting point for analyses in a framework that treats health as a family-level measure by testing various specification options for robustness and interpretability. Specifically, I examine the number of conditions in a family unit, the number of conditions among women and their spouses, a typology for classifying patterns of illness by number of illnesses and number of ill members, the presence of specific diagnoses at the family level, and the co-occurrence of these diagnoses within and across members of the same family unit. In this effort, nearly all of the specifications yielded interesting, interpretable findings, with the exception of the illness pattern typology (which shall be abandoned here), and yielded insight into the ways that these measures might relate to employment outcomes.

A second goal of this chapter was to determine whether the class-based stratifications in health that have been well documented at the individual level are also visible at the family level. As described in the first part of this chapter, there are indeed differences in the number of chronic conditions that families face at different points on the educational spectrum, and patterns of health disadvantage appear to operate similarly at the family level as at the individual level. I documented heightened counts of chronic conditions among the families of the least-
educated women, as well as heightened prevalence of specific diagnoses among low-educated families. These findings strengthen my hypothesizing from Chapter I in that if poor family health is not evenly distributed, the potential for unevenly distributed resources that might buffer these illnesses will be differentially strained. The following chapters undertake an exploration into the potential for illness to interact with women’s social class and resources, and attempt to detail how this uneven distribution of family illness may produce even more uneven consequences on women’s labor supply and earnings.

Finally, this examination of illnesses across family members reveals that specific diagnoses are not statically related to one family characteristic or another. Instead, the presence of a specific diagnosis in the family is disease-specific and shaped by varying contours of the family unit. In some cases, the odds of being diagnosed with an illness gradually decline at higher levels of education; for other conditions, college graduation is associated with steep reductions in risk. In both cases, these findings indicate the necessity of considering various diagnoses alongside family condition counts, exploring how specific constellations of illness take varying forms in different families.
IV. Results: Family Health and Women’s Labor Supply

In this chapter, I explore the effects of family health on women’s labor supply, by first modeling the odds of women’s employment, and then predicting women’s average number of hours worked per week. In predicting employment, I conceptualize family health in several ways, testing the predictive capacity of the total number of chronic conditions in a family, the number of conditions for a given family unit member (e.g., women’s own conditions, spouses’ conditions, children’s conditions), the presence of specific health conditions in a family, and the presence of co-occurring diagnoses in the household. Throughout, I draw on the findings from the preceding chapter to explore how women’s educational attainment interacts with the health of their families to produce class-stratified work outcomes. Finally, I explore the role of resources like health insurance coverage, access to transportation, and the value of a family’s liquid assets in buffering potential effects of poor family health on women’s labor supply. I examine the effects of family health on the number of hours women usually worked per week via a similar process, though only among working women.

Throughout this chapter, I use logistic regression models to predict women’s probability of employment and OLS regression methods to predict weekly hours worked. I test and describe various iterations of each model, examining the predictive capacity of multiple model specifications, alternately including the total number of conditions in a family, condition counts for specific members, and the presence of specific diagnoses at the family- and woman-levels. Tests on newly added parameters in building and for overall model goodness of fit help in the selection of best fit models. I also rely on summary and visual inspections of predicted values, residuals, deviance, influence, and leverage statistics. To improve the intuitive interpretation of interactive models, I present predicted probabilities and partial margin effects as figures and
make comparisons of the visual and statistical differences within. In brief, this chapter finds that the relationships between family health and women’s employment are complex, with relationships that vary according to the measures of health and labor supply examined, women’s own characteristics, and levels of family resources.

**Labor Supply: Predicting Employment**

This section explores the relationship between the patterns of family illnesses described in the preceding chapter and the odds of women working any hours in 2008; that is, the odds of being employed at all. I begin this section with a short overview of bivariate associations between the health measures detailed in the preceding chapter and women’s employment in 2008. I then proceed to the first of two tables of regression results in this section, where the first table explores the predictive capacity of different conceptualizations of family illness, and the second table explores the possible moderating effects of resources identified in the literature review (i.e., families’ health insurance coverage, value of liquid assets, availability of transportation, and receipt of SSI).

Although the preceding chapter documented associations between family health and women’s educational attainment, I reserve discussion of the health-employment associations for this section. Tables 11 and 12, below, provide brief overviews of the relationship between certain health measures and women’s labor supply. Table 11 contains the mean number of conditions at the family level, and for specific FUMs, across all families by women’s employment status.
Table 11. Mean Number of Conditions for Families and FUMs by Women’s Employment Status

<table>
<thead>
<tr>
<th></th>
<th>Employed</th>
<th>Not Employed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family Conditions</td>
<td>2.280</td>
<td>3.066</td>
</tr>
<tr>
<td>Women’s Own Conditions</td>
<td>0.819</td>
<td>1.448</td>
</tr>
<tr>
<td>Spouses’ Conditions</td>
<td>0.853</td>
<td>1.128</td>
</tr>
<tr>
<td>Children’s Conditions</td>
<td>0.593</td>
<td>0.455</td>
</tr>
<tr>
<td>OFUMs’ Conditions</td>
<td>0.015</td>
<td>0.035</td>
</tr>
</tbody>
</table>

* p<0.05; ** p<0.01; *** p<0.001

The above table shows that the bivariate relationship between women’s employment and family/FUMs is in the expected direction for the family level, and for women and spouses. The mean count of children’s conditions is actually higher among employed women than unemployed women, and OFUMs conditions are too few in number to achieve any statistically significant differences.

Table 12 shows the bivariate associations between women’s employment and specific diagnoses, measured at both the family- and woman-level. At the family level, hypertension, diabetes, arthritis, and cancer are associated with women’s employment; in each case, where the family contains one or more people with a given diagnosis, a smaller share of women are employed than in families without those diagnoses. The largest gap by diagnosis at the family level is in terms of family-level diabetes, where 61.23 percent of women in families where someone is diagnosed with diabetes are employed, versus 79.12 percent in families without such a diagnosis. At the woman-level, hypertension, diabetes, and arthritis are also associated with lower rates of employment, suggesting that the “family” effect may actually be a person-level effect wherein the woman herself is the one with the given diagnosis. However, woman-level measures of depression and asthma are also associated with lower employment rates, suggesting that there may be some variation in the relationship between specific conditions and women’s employment depending on which FUM has been diagnosed and with which disease.
Finally, while the association is not significant, it is worth noting that the distribution of employment by family-level asthma diagnoses is in an unexpected direction.

Table 12. Percent of Women Employed by Presence of Specific Diagnoses (Family- and Woman-Level)

<table>
<thead>
<tr>
<th>Health Measure</th>
<th>Family Level</th>
<th>Woman Level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No Diagnosis</td>
<td>Diagnosis</td>
</tr>
<tr>
<td>Depression</td>
<td>76.85</td>
<td>74.04</td>
</tr>
<tr>
<td>Hypertension</td>
<td>80.66</td>
<td>70.22 ***</td>
</tr>
<tr>
<td>Asthma</td>
<td>76.29</td>
<td>77.21</td>
</tr>
<tr>
<td>Diabetes</td>
<td>79.12</td>
<td>61.23 ***</td>
</tr>
<tr>
<td>Arthritis</td>
<td>79.07</td>
<td>67.47 ***</td>
</tr>
<tr>
<td>Cancer</td>
<td>77.16</td>
<td>70.26 *</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>76.95</td>
<td>69.41</td>
</tr>
<tr>
<td>Anxiety</td>
<td>76.40</td>
<td>74.58</td>
</tr>
</tbody>
</table>

* p<0.05; ** p<0.01; *** p<0.001

The relationships noted in Tables 11 and 12 suggest that the various health measures here may help to explain women's employment, and as such, are tested in a multivariate context in the models to follow. In addition, given that Chapter III shows that the distribution of condition counts and specific diagnoses vary by educational attainment, I also test for potential interactions between specific diagnoses and condition counts in the models to follow.

**Predictive Capacity of Various Illness Measures**

Table 13 shows results from the first set of logistic regressions predicting employment, with each model utilizing a different set of predictors. Model 1 regresses employment on the demographic characteristics plus number of conditions in the family unit; Model 2 tests whether women's own conditions mediate the effects of family-level conditions; and Model 3 includes conditions for women, spouses, children, and OFUMs in tandem. Model 4 incorporates specific diagnoses at the family level, and Model 5 does the same at the woman-level. Finally, Model 6
incorporates results from all previous models to establish a “best fit” version of the models.

Each of these regression models is intended to establish a basis for more complex model building later by identifying potentially important variations in several health measures’ relationships with women’s employment.
### Table 13: Logistic Regression Predicting Women’s Employment, Using Various Family Health Measures

<table>
<thead>
<tr>
<th>Condition Counts</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
<th>Model 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Conditions in FU</td>
<td>0.918 *** (0.017)</td>
<td>1.035 (0.034)</td>
<td>0.763 *** (0.028)</td>
<td>0.780 *** (0.031)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women’s Own Conditions</td>
<td>0.738 *** (0.042)</td>
<td>1.043 (0.054)</td>
<td>1.025 (0.051)</td>
<td>1.024 (0.050)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spouses’ Conditions</td>
<td>0.763 *** (0.028)</td>
<td>1.043 (0.054)</td>
<td>1.025 (0.051)</td>
<td>1.024 (0.050)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Children’s Conditions</td>
<td>0.806 *** (0.039)</td>
<td>1.038 (0.034)</td>
<td>0.989 (0.011)</td>
<td>0.989 (0.011)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OFUMs’ Conditions</td>
<td>0.780 *** (0.031)</td>
<td>1.024 (0.050)</td>
<td>1.024 (0.050)</td>
<td>1.016 (0.150)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Specific Diagnoses

- **Arthritis**: 0.702 * (0.099) 0.675 * (0.129)
- **Asthma**: 0.751 * (0.105)
- **Diabetes**: 0.599 *** (0.085) 0.499 ** (0.110) 0.650 ** (0.102)
- **Hypertension**: 1.068 (0.134) 0.909 (0.114) 1.251 (0.183)
  - [...] x White, non-Hispanic Ref. Ref. Ref.
  - [...] x Black, non-Hispanic 0.479 * (0.137) 0.680 (0.203) 0.662 (0.207)
  - [...] x Hispanic/Other/Multiracial 0.634 (0.172) 0.474 * (0.159) 0.424 * (0.145)

### Age

- Age¹: 0.909 *** (0.008) 0.908 *** (0.007) 0.907 *** (0.007) 0.910 *** (0.008) 0.910 *** (0.008) 0.908 *** (0.007)
- Age²: 0.997 *** (0.000) 0.997 *** (0.000) 0.997 *** (0.001) 0.997 *** (0.000) 0.997 *** (0.000) 0.997 *** (0.001)

### Marital Status

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Previously Married</td>
<td>1.145 (0.306)</td>
<td>1.222 (0.337)</td>
<td>1.242 (0.346)</td>
<td>0.981 (0.291)</td>
<td>1.200 (0.334)</td>
<td>1.236 (0.358)</td>
</tr>
<tr>
<td>Never Married</td>
<td>1.207 (0.221)</td>
<td>1.374 (0.253)</td>
<td>1.391 (0.285)</td>
<td>1.058 (0.200)</td>
<td>1.274 (0.243)</td>
<td>1.373 (0.281)</td>
</tr>
</tbody>
</table>

### Educational Attainment

| Less than High School | 0.515 *** (0.092) | 0.530 *** (0.095) | 0.528 *** (0.096) | 0.519 *** (0.093) | 0.521 *** (0.094) | 0.535 ** (0.100) |
| High School Graduate | 0.849 (0.111) | 0.841 (0.115) | 0.840 (0.115) | 0.860 (0.112) | 0.857 (0.115) | 0.857 (0.117) |
| Some College | 0.645 ** (0.096) | 0.637 *** (0.094) | 0.637 *** (0.094) | 0.650 ** (0.095) | 0.646 ** (0.092) | 0.651 ** (0.096) |

### Race/Ethnicity

| Black, non-Hispanic | 0.787 (0.098) | 0.809 (0.108) | 0.806 (0.108) | 1.275 (0.249) | 0.975 (0.163) | 0.936 (0.174) |
| Hispanic/Other/Multiracial | 0.801 (0.117) | 0.810 (0.119) | 0.810 (0.119) | 1.038 (0.164) | 1.029 (0.139) | 1.032 (0.134) |
| Lives in a Metropolitan Area | 1.132 (0.123) | 1.109 (0.124) | 1.108 (0.124) | 1.154 (0.124) | 1.118 (0.122) | 1.122 (0.122) |
| Years of Work Experience | 1.119 *** (0.009) | 1.119 *** (0.009) | 1.119 *** (0.009) | 1.123 *** (0.009) | 1.122 *** (0.009) | 1.121 *** (0.009) |
| Years of Work Experience² | 0.998 ** (0.001) | 0.998 ** (0.001) | 0.998 ** (0.001) | 0.998 ** (0.001) | 0.998 ** (0.001) | 0.998 ** (0.001) |
| Constant | 11.541 *** (2.114) | 12.568 *** (2.302) | 12.530 *** (2.251) | 10.800 *** (1.943) | 11.223 *** (2.052) | 11.663 *** (2.088) |
| Overall F test | 31.451 (0.281) | 31.291 *** (0.001) | 30.638 *** (0.001) | 31.733 *** (0.001) | 27.037 *** (0.001) | 27.256 *** |
| F-adjusted mean residual goodness-of-fit² | 0.469 (0.001) | 0.475 ** (0.129) | 0.475 ** (0.129) | 0.475 ** (0.129) | 0.475 ** (0.129) | 0.475 ** (0.129) |

* p<0.05, ** p<0.01, *** p<0.001

a Linear measures are centered; quadratic terms are created from centered measures.
b Colloquially known as the Archer-Lemeshow (2006) test. Recall that for the overall F test, H₀=an intercept-only model would fit as well, for the Archer-Lemeshow test H₀=there is evidence of a lack of fit. In short, good model fit is indicated by significant results for the former, non-significant results for the latter.

Note: Coefficients on diagnoses at the family level are presented in blue and those on women's own diagnoses appear in orange.

Model 1 shows that family health conditions significantly reduce women’s odds of employment, decreasing about eight percent for each additional condition in the family (OR=0.918; p<0.0001). The natural extension of this finding is to determine whether this effect...
is largely driven by women’s own conditions, and indeed, Model 2 shows that women’s number of conditions fully mediates the effect of the family condition count, with each additional condition reducing women’s odds of employment by more than one-quarter (OR=0.738; \( p=0.001 \)). In the next model (Model 3), condition counts for other family members, including spouses, children and other family unit members are included; the effect of women’s own conditions remain generally unchanged here, and no other family members’ condition counts emerge as significant predictors.\(^{38}\)

Model 4 examines the role of specific diagnoses in the family, and in building this model I include each of the eight diagnoses in Table 12 in turn, only displaying those that contributed significantly to the model. This model shows that the association between diabetes and women’s employment identified in Table 12 persists net of demographic controls, reducing the odds of women’s employment by more than 40 percent (OR=0.599; \( p=0.001 \)). In the logistic regressions testing the robustness of the relationship between specific diagnoses and demographics, summarized (but not shown) in Chapter III, it is worth mentioning one association that emerged and led to additional testing in these models: a relationship between hypertension and race/ethnicity. Recalling this association, I also enter an interaction term between race and a family hypertension diagnosis into this model, and find that divergent effects by race accounted for the lack of significance on the overall hypertension odds ratio. In particular, the effects of a family hypertension diagnosis among black women’s families are particularly strong, reducing the odds of employment by more than half (OR=0.479, \( p=0.014 \)). Calculating the predictive margins on the interaction between family-level hypertension and race/ethnicity shows that adjusting for characteristics in Model 4, black, non-Hispanic women

\(^{38}\) The results in Table 13 are among all women, though restricting estimation of Model 3 to women who live with children and spouses changes odds ratios slightly, and produces no substantively different effects.
have especially low odds of employment (see Figure 5) in the presence of a family hypertension diagnosis. Further, calculating the contrast in these margins suggest that a family hypertension diagnosis reduces black women’s predicted probability of employment by 9.5 percent \((p=0.022)\), whereas a similar diagnosis for white women or Hispanic/other/multiracial women produces no similar effect. Note that only white non-Hispanic women’s probabilities are shown for comparison, but the probability of employment among Hispanic/other/multiracial women is similar to white women’s \((p=0.1064)\).

Figure 5. Women’s Predicted Probability of Employment, by Race/Ethnicity and Family Hypertension Diagnosis

Given that the results in Models 2 and 3 indicate the particular importance of women’s own health, I next test the effects of women’s own diagnosis in Model 5. Again, I enter an interaction term between race and women’s own hypertension diagnosis into this model, but unlike in for the family-level diagnosis, the effect of own diagnosis is not significant for black women. Instead, Hispanic/other/multiracial women see reduced odds of employment similar to those experienced by black women with a family-level hypertension diagnosis in Model 4. This
suggests that the effects of hypertension on women’s employment may vary, both depending on who is hypertensive and on the characteristics of the family in which the diagnosis occurs. In addition, the inclusion of women’s own arthritis and diabetes diagnoses largely mimic the effects of those diagnoses at the family-level, reducing the odds of women’s employment between 25 and 30 percent.

Finally, in Model 6, significant predictors from the preceding models are pooled to jointly consider effects of family- and woman-level diagnoses while controlling for condition counts among different family members. Simultaneously including interactions between family- and woman-level hypertension diagnosis and race/ethnicity results in a non-significant effect of family-level hypertension and black race/ethnicity (as in Model 4), though the effects of own hypertension for Hispanic/other/multiracial women is still significant. Further, including the woman-level measure of diabetes does not mediate the relationship between a family-level diabetes diagnosis and employment (OR on family diagnosis= 0.664; p=0.028 when own diabetes is included). In contrast, including women’s own condition count in the model eliminates the effects of women’s own asthma and arthritis diagnoses, as in Model 5, as well as the family-level effects of arthritis in Model 4, suggesting that the latter was largely a woman-level effect proxied by the family-level diagnosis. Taken together, these findings further emphasize that the effects of different diagnoses have differential impacts on women’s employment, further nuanced by the family member who has been diagnosed.

Beyond health measures, there are several demographic characteristics associated with women’s employment. Across all models, the coefficient on each the linear and quadratic age terms are significant, indicating a nonlinear and concave relationship between employment and women’s age (see Figure 6, below). Each model shows consistent effects of young children in
the household, generally halving the odds of women working when compared with their childless counterparts, and a nonlinear effect of women’s work experience, where the odds of working rise steeply through nearly 20 years of work experience, and then flatten out. Finally, the relationship between educational attainment and employment was consistent across models: the odds of employment for non-high school graduates were half as high as college graduates; for those with some college, odds were about two-thirds as high as the college graduates. Formal tests for the equality of coefficients and the nonlinear hypothesis that coefficients are proportional (given the possibility of varying residual standard deviation between groups) indicate that these differences in employment by educational attainment indeed form a nonlinear relationship with women’s employment.

Figure 6. Predicted Probability of Employment by Women’s Age

Note: Predictive margins adjusted for characteristics shown in Table 13, Model 1 and are calculated adjusted for complex sample design. Error bars represent 95 percent confidence intervals.

It should be noted that identifying which of the Models 1-6 is “best” is considerably more subjective than might be true with other samples. Because these data have a complex sample design, the assumptions of likelihood-ratio tests and pseudo $R^2$ calculations—both of
which rely on log likelihoods and the corresponding assumption that cases are independent—are violated by the clustered nature of complex sample data and thus are inapplicable for examining nested models here (e.g., Sribney 2005). Instead, one can compare nested models by applying adjusted Wald tests to determine whether the coefficients on newly added variables are equal to zero (Aneshensel 2012; Heeringa et al. 2010). However, options for comparing fit between different iterations of logistic regression models with identical number of covariates (i.e., non-nested models) are limited. That is, determining whether a logistic regression model containing women’s condition count fits “better” than one containing the entire family’s condition count is difficult, as Archer and Lemeshow’s svylogitgof test can be used to examine overall model fit, though not to compare different versions of a given model. Thus, in cases where models are similar but not nested (e.g., Models 1 and 2), I present both versions here. In short, I use the criteria of parsimony, the overall F test, and the Archer-Lemeshow test to identify well-fit models. The null hypothesis of the latter was rejected in all models, suggesting that key measures may be omitted. In the next section (for Table 14), I use the findings from Table 13 to build fuller models predicting labor supply that include tests of the potentially moderating effects of family resources. In these more complete versions, I also consider misspecification effects and the potential for traditional logistic regression diagnostics to help assess model fit, described below.

**Effects of Buffering Resources**

For Table 14, I build on findings from Table 13 to further home the precision of these specifications and test the effects of potential moderating resources in the health-labor supply...
relationship. I begin by revising Table 13’s Model 1—using all family conditions as a predictor—and test for a nonlinear effect of family conditions on women’s employment \((p=0.788)\). I then interact family conditions with women’s educational attainment, to determine whether the effects of family health on employment vary by this proxy for social class. In succession, I add measures of transportation availability, health insurance coverage, value of liquid assets, and SSI receipt into the model, first testing main effects of each, then interacting each measure with the number of family conditions, and women’s educational attainment. After each addition, I use an adjusted Wald test to determine whether the coefficient on the newly added term is jointly zero, and remove those that do not contribute to the model. Using the same process, I build a series of models that include individual FUMSs’ condition counts (Model 2). Model 3 examines the effects of the specific diagnoses in the family on women’s odds of employment, and Model 4 does the same for women’s own diagnoses. Finally, Model 5 pools together Models 3 and 4 to simultaneously model effects of women’s own and family conditions.

After estimating the models described above, I examine a series of diagnostic measures to determine whether the model might be improved for some \(j\) combinations of \(x\) values. It should be noted that making comparisons between different models (as described above) is unfortunately not the only area in which the nascence of logistic regression modeling of complex survey data is revealed. A host of diagnostic procedures for influence statistics, available in Stata for traditional logistic models, are not yet available for complex survey data (Heeringa et al. 2010; Hosmer, Lemeshow, and Sturdivant 2013). As such, I follow recommendations from Heeringa et al. (2010) for evaluation of fit, who suggest that in lieu of forgoing these assessments altogether, analysts should use any tests available (at present, solely the Archer-Lemeshow \textit{svylogitgof} test), then re-estimate models in a standard logistic
regression model. The authors note, “serious lack of fit should be quantifiable even though the standard program tools do not correctly reflect the variances and covariances of the parameter estimates given the complex sample design” (Heeringa et al. 2010: 244). An examination of misspecification effects—that is, a comparison of the design-based variance and variance from the same model fitted without accounting for complex sample design—reveals that the survey design contributes substantially to the model’s variance, suggesting that the unweighted diagnostics should be cautiously interpreted as suggestive, seen only as tools for improving model specification. That is, at most, these statistics help to identify cases for which the models may be particularly poorly fit and examination in conjunction with other descriptive measures can reveal potentially omitted measures.

For each of Models 1 through 5, I calculate a variety of change in Pearson chi-squared $(\Delta \chi^2_p(j))$, influence $(\Delta B_j)$, and change in deviance $(\Delta \chi^2_D(j))$ statistics, and inspect the fit of each model graphically. Plotting $\Delta \chi^2_D$ and $\Delta \chi^2_p$ values by the linear prediction ($\hat{y}$) and weighting by $\Delta B$ and actual probability weights can provide some sense of overall influence in the final models. I also compare the distribution of influence statistics with general “rules of thumb” proposed by Hamilton (1992). Only around 6 percent of cases could be considered to have high $\Delta \chi^2_p$ values, while no cases have $\Delta B$ or $\Delta \chi^2_D$ values that approach the Hamilton’s proposed cutoff for high values. By examining the highest values of $\Delta \chi^2_p$, I find that the models are better fit for employed women than unemployed. By examining the 99th percentile of $\Delta \chi^2_p$ values in conjunction with corresponding women’s characteristics, I note an uneven distribution of these cases by region of residence. Specifically, these models appear especially poorly fit for women who live in the South, and for black, non-Hispanic Southern women in particular. As such, I revise models to include both a regional indicator and an interactive measure of black race/ethnicity and
Southern residence; doing so reduces the values of $\Delta \chi^2_p$ at the 25$^{\text{th}}$, 50$^{\text{th}}$, and 75$^{\text{th}}$ percentiles by 1-3 percent. Table 14 presents the results of these models.
Table 14. Logistic Regression Predicting Women’s Employment, With Potential Moderators Included

<table>
<thead>
<tr>
<th>Condition Counts</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR SE</td>
<td>OR SE</td>
<td>OR SE</td>
<td>OR SE</td>
<td>OR SE</td>
</tr>
<tr>
<td>Number of Conditions in Family</td>
<td>0.927 (0.016)</td>
<td>0.966 (0.025)</td>
<td>1.097 * (0.045)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women’s Own Conditions</td>
<td>0.778 *** (0.028)</td>
<td>0.767 *** (0.038)</td>
<td>0.716 *** (0.043)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[...] x SSI</td>
<td>0.707 * (0.097)</td>
<td>0.723 * (0.101)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spouses’ Conditions</td>
<td>1.060 (0.058)</td>
<td>1.063 (0.058)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Children’s Conditions</td>
<td>1.055 (0.055)</td>
<td>1.063 (0.057)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OPLMN Conditions</td>
<td>1.100 (0.174)</td>
<td>1.128 (0.180)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Specific Diagnoses

- **Diabetes**: 0.654 * (0.105)
- **Hypertension**: 1.164 (0.157)
- [...] x White, non-Hispanic
- [...] x Black, non-Hispanic: 0.399 ** (0.109)
- [...] x Hispanic/Other/Multiracial: 0.632 (0.169)
- **Lung Disease**: 4.381 ** (2.031)
- [...] x Family Condition Count: 0.827 ** (0.056)
- [...] x Women’s Condition Count: 0.624 * (0.121)

Resources

- **Value of Liquid Assets ($1000s)**: 0.998 *** (0.000)
- Family Received SSI: 0.361 *** (0.106)
- **Access to Transportation**
  - Own Vehicle: 1.045 (0.354)
  - Public Transportation: 1.185 (0.541)
  - No Transportation: 0.293 *** (0.106)
- **Age**
  - Age: 0.909 ** (0.008)
  - Age 2: 0.997 *** (0.000)
- **Marital Status**
  - Married: 1.262 (0.337)
  - Never Married: 1.468 (0.282)
- **Race/Ethnicity**
  - White, non-Hispanic: 1.650 (0.439)
  - Black, non-Hispanic: 1.650 (0.439)
  - Hispanic/Other/Multiracial: 0.852 (0.112)
- **Location**
  - Lives in a Metropolitan Area: 1.082 (0.122)

<table>
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<th>Model 3</th>
<th>Model 4</th>
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<td>OPLMN Conditions</td>
<td>1.100 (0.174)</td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p<0.05, ** p<0.01, *** p<0.001

a Linear measures are centered and quadratic terms are created from centered measures.
b Colloquially known as the Archer-Lemeshow (2006) test. Recall that for the overall F test, H0= an intercept-only model would fit as well, for the Archer-Lemeshow test H0=there is evidence of a lack of fit. In short, good model fit is indicated by significant results for the former, non-significant results for the latter.

Note: Coefficients on diagnoses at the family level are presented in blue and those on women’s own diagnoses appear in orange.
In Model 1, the number of conditions in the family reduced women’s odds of employment by about 7 percent for each additional condition, though the effects did not diverge by educational attainment (interactions between health measures and women’s educational attainment are tested in each model in Table 14, and not shown where non-significant). In addition to family conditions, this model also tests the effects of resources, including liquid assets, SSI receipt, access to transportation and health insurance coverage (excluded where not significant). Here, receipt of SSI significantly reduces the odds of employment by nearly two-thirds and having no transportation reducing the odds by more than 70 percent. Further, the value of a family’s liquid assets was associated with a small decline in the odds of employment (OR=0.998; p=0.001).

Model 2 includes distinct condition counts for women and their family members, and results show that women’s conditions, rather than condition counts among other family members, are the only significant health predictor. Again, both having no transportation and value of liquid assets have similar effects to those shown in Model 1. This model also shows a significant interaction between SSI receipt and women’s conditions, as shown in Figure 7, below. This figure suggests that receipt of SSI is associated with different outcomes for women’s labor supply, depending on the number of conditions with which women have been diagnosed, specifically the declines in women’s predicted probability of employment at higher numbers of conditions are exacerbated among women whose families received SSI.

---

40 In each model, I test the effects of health insurance as a continuous measure of months covered as well as the categorical measure indicating no coverage, partial-year coverage, and full-year coverage, presenting only version that are significant in a given model. All potentially moderating factors are entered on their own, and also interacted with educational attainment and health measure in the model to determine whether the availability of key resources has stratified impacts on women’s employment. In all cases, non-significant interactions are discarded from the models shown in Table 14.
Figure 7. Predicted Probability of Women’s Employment in 2008 by Family Condition Count and Family’s SSI Receipt

Note: Predicted probabilities adjusted for characteristics displayed in Table 14, Model 2 and adjusted for complex sampling design. Number of conditions truncated at 7 for display only. Differences in predicted probabilities by SSI receipt are significant at two conditions or more (Bonferroni-adjusted p<0.01). Error bars indicating 95 percent confidence intervals are omitted for readability, but available upon request.

Given that these data are unable to assess causality, it is difficult to identify the precise mechanism of this relationship, though several possibilities present themselves. For example, it is possible that women with higher numbers of conditions are less likely to be employed, and SSI provides substitutive income that accelerates women’s exit from the labor force. It is equally possible that SSI itself is unrelated to women’s employment, and instead is a proxy for illness severity; that is, women who receive SSI are those with the most limiting or burdensome illnesses, and that illness severity (unavailable as a measure here) would mediate this relationship. Finally, because SSI receipt is a family-level measure, it is also possible that someone else in the household has a severe disability, and that the association women’s own health and SSI receipt is a result of complex interactions in intra-family health.

Model 3 assesses the effects of specific illnesses within the family, while controlling for family condition counts. In this model, the family condition count is no longer significant (as in
Model 1), instead, with family-level diagnoses mediating this relationship. That the effects of the family condition count are attenuated by the inclusion of specific diseases suggests that the number of conditions in the family is less important for predicting women’s employment than are the type of conditions in the family. Here, there are significant main effects for diabetes, reducing the odds of employment by one third (OR=0.654; p=0.010). Given earlier findings indicating a specific relationship between hypertension and race/ethnicity, I include that interaction here and find that, as in Table 13, a family-level diagnosis of hypertension reduces the odds of employment more intensely among black women when compared to a similar diagnosis in white women’s families. Finally, lung disease is also associated with women’s employment, with varying effect by the number of conditions in the family unit. Figure 8 shows the interactive effects of this relationship, wherein a family diagnosis of lung disease decreases the predicted probability of women’s employment at higher numbers of conditions in the family. Further, women in families with a lung disease diagnosis show initially higher odds of employment that women in families without such a diagnosis, and when interacted with family condition counts, the result is that the predicted probability of employment converges between women in families with and without the diagnosis (differences between diagnostic categories are only statistically significant through three conditions). In this model, resources (liquid assets, SSI receipt, and transportation) matter in nearly identical ways as those described for Model 1.
Model 4 adds women’s own diagnoses to the results from Model 2, but unlike the similar models at the family level (Models 1 and 3), the effect of women’s own condition count does not dissipate when accounting for specific illnesses, suggesting that both the number and type of women’s own illnesses is associated with odds of employment. Unlike in the family-level models, women’s own diagnosis of diabetes is not associated with employment ($p=0.137$ when included in Model 4, excluded here). As in Table 13, the divergent interaction between family- and woman-level hypertension and race/ethnicity emerges, where women’s own diagnoses matter for Hispanic/other/multiracial women (compared with the family-level effects for black women in Model 3). Finally, like in the family-level model, the interaction between women’s own lung disease and condition counts is significant here, shown in Figure 9. As in Figure 8, a lung disease diagnosis is initially associated with a higher probability of employment, which declines as women’s condition count rises. Unlike in Figure 8, calculated where the main effects of family condition counts were non-significant, Figure 9 shows the declining probability of
employment for all women at higher number of own conditions, a relationship that is exacerbated when one of those conditions is lung disease. Because lung disease is relatively rare, the standard errors at the woman-level produce the below effects, though differences in the two lines are only significant through two conditions, and the probability of employment is similar across lung disease diagnoses after that point.

Figure 9. Predicted Probability of Women’s Employment by Own Lung Disease Diagnosis and Number of Own Conditions

Note: Predictive margins are adjusted for characteristics appearing in Table 14, Model 4. Differences between diagnostic categories are statistically significant through two conditions after Bonferroni correction ($p<0.05$).

Finally, Model 5 includes health measures at both the woman and family level by incorporating predictors from earlier models. First, women’s condition count still has a significant effect on the odds of employment, though the interaction with SSI receipt disappears when controlling for family-level lung disease diagnosis. This finding supports the conjecture that SSI receipt may be a proxy for more severe illness in the family; that is, that the earlier association between the two (Figure 7) may be a result of women’s own health (condition counts) interacting with the health of other family members who receive SSI. Again, diabetes at the family level remains a significant predictor not explained by women’s own diabetes.
diagnoses (p=0.813 when included in Model 5, omitted from results here). For hypertension, the inclusion of family- and woman-level interactions with race/ethnicity produces non-significant findings for each (I elect to include only the interaction with the larger F statistic on the joint interaction), and again hypertension in black women’s families reduces the odds of employment. Finally, I test the interaction between lung disease and condition counts at the family and woman level, finding that the latter is non-significant when controlling for the former, though this effect seems more likely to be a function of the standard error on women’s own diagnoses rather than a meaningful mediating effect. Again, as in all previous models, the effects of assets and transportation are significant predictors of employment, with effects that are consistent across educational attainment and family health.41

Perhaps the most interesting result from Table 14 is that even when controlling for women’s own condition counts and women’s own diagnoses, health at the family level still matters. In Models 2, 4, and 5, women’s own condition counts have a linear relationship with employment, with each additional condition reducing the odds of employment. Even the interactions with own lung disease and family SSI receipt still show a negative slope on women’s condition counts. However, family health does not appear to operate in the same way. The effect of family condition counts on employment is not uniformly linear, and its effect dissipates when controlling for specific illnesses within the family (Model 3). In other words, specific diagnoses in the family function in varying ways within and across families, even when women’s own diagnoses are accounted for, and the dynamics of family health matter in ways that are not simply additive.

41 Note that I interact each resource with family- and woman-level condition counts, and also with specific diagnoses. However, because many illnesses are relatively rare, the interaction with categorical measures produces very thin cells; though some results are statistically significant (e.g., having no transportation and a cancer diagnosis in the family dramatically reduced the odds of employment), the small cell sizes suggest that these estimates are unstable. As a result, I omit all interactive measures where one or more cells contain fewer than 10 cases.
In terms of demographic characteristics, as in Table 13, women’s age has a strong and consistent curvilinear effect on the odds of being employed across all models. Again, having young children in the family reduces women’s odds of working by more than half across all models, and in models including spouses’ condition counts, never married women are more likely to be employed. Again, educational attainment is associated with employment as in Table 13: for women without a diploma the odds of working are half as high as their college-educated counterparts, about two-thirds as high for women with some college, and about equally high for high school graduates. Years of work experience increased women’s odds of working in a nonlinear way, with the odds of working increasing to a certain threshold of work experience, before flattening out. Finally, the interaction between black race/ethnicity and Southern residence is significant in all models, reducing the odds of employment by more than half. These very low odds of employment among Southern black women might be explained in several ways: first, research suggests that black unemployment rates exceed those of other race/ethnicities, and that black workers are concentrated in sectors that were hard-hit by the lead up to the recession (Department of Labor 2012). Further, Southern women, and black Southern women in particular, have a long history of involvement in unpaid and informal work (Walker, Dunn, and Dunn 2003), which is likely not reflected in formal measures of employment. Finally, the historical legacies of the South for blacks likely intersect with employment opportunities in ways that are immeasurable here.

Beyond the measures included in Table 14, I also consider the possibility that specific diagnoses may have differential impact on women’s work depending on the health status of others in the household. In Chapter III, I documented patterns of co-occurring illnesses within the family; here, I test whether any of these pairs has particular impacts on women’s odds of
employment. Because these condition pairs are so numerous (see Table 10), and some conditions are relatively rare, I allow sample size to dictate which co-occurring conditions are tested, using only pairs that affect (an arbitrary) sample size of 100 families or more. Using specifications from Model 5, I include binary indicators of specific illness pairs in successive iterations, and find no effects of any co-occurring diagnoses tested. I also explore interactions between diagnoses at the family level and women’s condition counts by interacting women’s condition counts with specific diagnoses in Model 5, and find no significant results here either.

In finalizing the models presented in Table 14, I re-calculate a set of influence statistics, as with the models in Table 13, and find similar results: just over 6 percent of cases have $\Delta \chi^2_p$ values over 4.0, while no cases’ $\Delta B$ values and no cases’ $\Delta \chi^2$ values approach Hamilton’s (1992) proposed cutoff for high values (1.0 and 4.0, respectively). To ensure that this final model does not suffer from multicollinearity, I also calculate the tolerance of each included measure in succession (omitting interactive measures, per Allison (2012a)), finding the lowest tolerance on age (1 - $R^2_k$ = 0.366)—largely resulting from the inclusion of years of work experience—though the tolerance here is still adequately distant from the cutoff proposed by Hamilton (1992) at which point models are estimable, but less stable (0.20). Results from the Archer-Lemeshow test suggest a lack of fit for Models 1, 2, and 3; that these models that do not account for health at both the woman- and family-levels indicates the key role that each plays in predicting employment.

42 These tests specifically use the version of co-occurring conditions presented in Table 10, where one person in the family has one condition and a separate person has the other. This does not preclude the inclusion of those with multiple similar diagnoses (e.g., a family in which a woman has hypertension and arthritis while her spouse has hypertension is included in the hypertension/arthritis pair) but does not include families in which the two diagnoses are present in a single person (e.g., if the spouse in the earlier example had no conditions, the woman would not be included in the hypertension/arthritis pair). The purpose of this strategy is to explicitly consider the role of cross-member illness interactions in the family unit. Co-occurring conditions tested in these models include depression/hypertension, depression/asthma, hypertension/hypertension, hypertension/asthma, hypertension/diabetes, hypertension/arthritis, asthma/arthritis, diabetes/arthritis, and arthritis/arthritis.
In this section, I examined the predictive capacity of family health on women’s employment. Findings thus far suggest that the relationship between family health and women’s employment is complex; broadly speaking, both the number and type of conditions present in a family have some associations with women’s employment. However, as shown in Table 14, the effects of condition counts and specific diagnoses interact with other family characteristics and resources in complex ways. For instance, women’s own number of conditions accounts for much of the impact of overall family condition count, though specific diagnoses at the family level have effects that extend beyond simply women’s own diagnoses. Further, the effects of diagnoses at the family level cannot be fully explained by women’s own diagnoses, suggesting that both women’s own health and the health of their families have some distinct impacts on labor supply. In the next section, I explore another component of labor supply—women’s average number of hours worked per week—and use the findings here to shape those analyses and examine the dynamics of both family health and labor supply.

**Labor Supply: Number of Hours per Week**

This section estimates the effects of family illness on women’s labor supply by examining the relationship between family health and working women’s average number of hours worked per week. As in the previous section, I estimate models employing the various measures of family health, testing each for improvement in model fit. I also examine the effects of potential resources (i.e., health insurance, SSI receipt, transportation availability, and liquid assets) in and test whether their effects diverge by women’s educational attainment, and health measures at the woman- and family-levels. I begin this section with a description of my efforts to identify the existence of a selection bias in estimating women’s weekly hours worked (as women do not randomly select into the labor force) before transitioning into a description of results. A
summary of the associations between family health and women’s labor supply will follow at the end of this chapter.

**Heckman Selection Model**

Before describing this section’s results, it is worthwhile to address the preparatory steps for these analyses, namely, addressing the selection bias inherent in the fact that women do not randomly select into employment (see Golder 2011 for a practical discussion of this issue, and the inspiration for the below example). In this section, I provide a brief overview of James Heckman’s “sample selection bias as a specification error” (Heckman 1979), describe the results of the selection models here, and the calculation of the inverse Mills ratio, which can be used to correct for selection bias in all models estimating hours worked and wages earned (e.g., Lechmann and Schnabel 2012).

To estimate the effects of education on women’s wages, let us use the following equation:

\[ y_i = \beta x_i + \epsilon_i \]  

[Equation 1]

where \( y_i \) is the predicted wage, \( x_i \) is education, and \( \epsilon_i \) is the error term. Because women in the labor force may vary from other women in unmeasured ways (for example, one unmeasured characteristic might be personal motivation: women who are highly motivated are more likely to enter the labor force), it is necessary to account for the bias associated with this non-random entry. If women do not enter the labor force at random, the equation “selecting” them into the labor market is:

\[ U_i = w_i \gamma + u_i, \]  

[Equation 2]

where \( U_i \) is women’s likelihood of entering the labor market, \( w_i \) is the known vector of characteristics that impact the decision to work (e.g., education), and \( u_i \) represents any
unmeasured characteristics (here, personal motivation), which are assumed to be normally
distributed with the error term from the equation predicting wages ($\epsilon_i$). However, some
women who are not highly educated ($w_i$) choose to enter the labor force—perhaps because
they are highly motivated (assumed to be captured in $u_i$). If this is the case, then highly
educated women have a normal range of errors, while women with low education (and high
motivation) have much larger error terms, since such an important characteristic is
unmeasured. The result is that whether or not education is correlated with motivation in the
population, it is correlated with motivation in the sample. If indeed motivation leads to higher
wages, the effect of education is dampened in the wage equation (because the sampled women
with low education are highly motivated, education emerges as a less-strong predictor). A
plethora of existing research has demonstrated the degree of bias that may result from not
addressing these selection issues, as well as the importance of carefully specifying the equations
for doing so (for a thoughtful discussion of appropriate and erroneous applications of this
approach in criminological literature, see Bushway, Johnson, and Slocum 2007).

According to Heckman’s general theory, the selection equation should be more general
than the analytic equation, including exclusion restrictions that predict participation (here, in
the labor market), but are not correlated with the error term in the outcome equation (here, hours worked) (Bar, Kim, and Leukhina 2013). For example, one popular exclusion criteria are
spousal earnings (ibid.), as a high level of spousal income may preclude women from needing to
enter the workforce; further, given tendencies toward assortative matching, spousal income
may account for other unobserved characteristics among women (e.g., Eika, Mogstad, and Zafar
2014; Groothuis and Gabriel 2010). Because my sample includes both married and unmarried
women, I create here a more generalized version of “other income” that allows for spousal
earnings for married women and receipt of food stamps, welfare, and/or child support for all women.

To determine whether these selection variables are appropriate, I first regress hours worked demographics and the income variables described above. Adhering to the requirements of a Heckman model, none of these measures predict the actual outcome measure (hours worked). However, when similarly regressed on employment (the participation measure), none of these measures actually predict employment in this sample, suggesting that the instruments are weak. Repeated attempts to adjust these measures and/or replace with more suitable selection measures were unsuccessful, and without a good selection measure, a selection model is not estimable. As a result, the regressions models in this chapter are estimated with OLS techniques, though it should be acknowledged that the presence of a selection bias is a possibility throughout.

Results

Drawing on findings from the previous section, I take a systematic approach to modeling hours worked by first testing the effects of the number of conditions in the family, then condition counts for individual FUMs. The next two models incorporate diagnoses at the family- and woman-level, respectively, and the final model draws on findings from previous models to present the “best” fit model (“best” again being a somewhat subjective delineation, due to the

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As an exploratory measure, I use Stata’s svy heckman command to estimate the selection equation with the above income measures (in various iterations) as the exclusion variables in the employment equation. With complex survey data, no likelihood ratio test is reported; instead, I calculate an adjusted Wald test for the coefficient on the transformation of athrho (a transformation of rho, the correlation between the error terms of the participation and outcome equations), and fail to reject the null hypothesis that athrho=0 (p<0.302), meaning that as instrumented by these measures, no selection bias was detected. Further, upon inspection of the two models (selection and outcome), I note that the coefficients vary tremendously, suggesting that the Heckman selection equation may not be an improvement on traditional OLS estimates in the absence of improved measures.
limited regression diagnostic techniques applicable to complex survey data, discussed in detail below) and tests for potential moderating effects of family resources.

Table 15 shows models predicting average number of hours worked per week among women who worked any hours in 2008. Model 1 shows that the number of conditions in the family unit is not a significant predictor of hours worked. A visual inspection of a scatterplot of work hours and family condition count overlaid with a LOWESS (locally weighted scatterplot smoother) curve indicates essentially no relationship between the two, with the fitted line appearing neatly perpendicular to the x-axis.
Table 15. OLS Regression Models Predicting Average Weekly Hours Worked, Among Employed Women Only

<table>
<thead>
<tr>
<th>Model</th>
<th>Condition Counts</th>
<th>B</th>
<th>SE</th>
<th>B</th>
<th>SE</th>
<th>B</th>
<th>SE</th>
<th>B</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Conditions in Family</td>
<td>-0.041</td>
<td>0.116</td>
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<td></td>
<td>Women's Own Conditions</td>
<td>-0.253</td>
<td>0.222</td>
<td>-0.192</td>
<td>0.254</td>
<td>-0.424</td>
<td>0.291</td>
<td>-0.437</td>
<td>0.289</td>
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<td></td>
<td>Spouses' Conditions</td>
<td>0.308</td>
<td>0.233</td>
<td>0.311</td>
<td>0.237</td>
<td>0.390</td>
<td>0.239</td>
<td>0.386</td>
<td>0.240</td>
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<td></td>
<td>Children's Conditions</td>
<td>-0.215</td>
<td>0.198</td>
<td>-0.220</td>
<td>0.200</td>
<td>-0.182</td>
<td>0.201</td>
<td>-0.148</td>
<td>0.202</td>
</tr>
<tr>
<td></td>
<td>OFUMs' Conditions</td>
<td>2.318</td>
<td>1.241</td>
<td>2.354</td>
<td>1.210</td>
<td>2.264</td>
<td>1.173</td>
<td>2.517</td>
<td>0.945</td>
</tr>
<tr>
<td>Specific Diagnoses</td>
<td>Depression</td>
<td>0.121</td>
<td>1.613</td>
<td>-1.625</td>
<td>1.940</td>
<td>0.196</td>
<td>1.615</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>[...]x Less than High School</td>
<td>7.199</td>
<td>3.148</td>
<td>9.032</td>
<td>3.196</td>
<td>7.283</td>
<td>2.928</td>
<td></td>
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<tr>
<td></td>
<td>[...]x High School</td>
<td>-1.025</td>
<td>1.963</td>
<td>0.475</td>
<td>2.093</td>
<td>-0.794</td>
<td>1.911</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>[...]x Some College</td>
<td>-2.316</td>
<td>1.943</td>
<td>0.111</td>
<td>2.374</td>
<td>-2.362</td>
<td>1.972</td>
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<td></td>
<td>[...]x College</td>
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<td></td>
<td>Lung Disease</td>
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<td>[...]x Less than High School</td>
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<td>[...]x High School</td>
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<td>[...]x Some College</td>
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<td>Health Insurance Status</td>
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</tr>
<tr>
<td></td>
<td>Part-Year Coverage</td>
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<td></td>
<td>Full-Year Coverage</td>
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<tr>
<td>Family Received SSI</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>偏低</td>
<td>Age</td>
<td>-0.326</td>
<td>***</td>
<td>0.044</td>
<td>0.046</td>
<td>-0.339</td>
<td>***</td>
<td>0.046</td>
<td>0.044</td>
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<tr>
<td></td>
<td>Age2</td>
<td>-0.007</td>
<td>*</td>
<td>0.003</td>
<td>0.003</td>
<td>-0.008</td>
<td>*</td>
<td>0.003</td>
<td>0.003</td>
</tr>
<tr>
<td>Marital Status</td>
<td>Married</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Previously Married</td>
<td>1.753</td>
<td>1.214</td>
<td>2.083</td>
<td>1.244</td>
<td>2.296</td>
<td>1.233</td>
<td>2.495</td>
<td>1.226</td>
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<tr>
<td></td>
<td>Never Married</td>
<td>2.212</td>
<td>0.812</td>
<td>2.139</td>
<td>0.850</td>
<td>2.235</td>
<td>0.857</td>
<td>2.173</td>
<td>0.852</td>
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<td>Age of Youngest Child</td>
<td>No Children</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Child(ren) Under Age 5</td>
<td>-4.458</td>
<td>***</td>
<td>0.654</td>
<td>0.663</td>
<td>-4.823</td>
<td>***</td>
<td>0.671</td>
<td>0.675</td>
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<td></td>
<td>Child(ren) Over Age 5</td>
<td>-1.645</td>
<td>**</td>
<td>0.498</td>
<td>0.523</td>
<td>-1.539</td>
<td>**</td>
<td>0.523</td>
<td>0.516</td>
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<td>Three Generation Household</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Less than High School</td>
<td>1.350</td>
<td>1.100</td>
<td>0.903</td>
<td>1.120</td>
<td>0.236</td>
<td>1.111</td>
<td>0.022</td>
<td>1.133</td>
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<tr>
<td></td>
<td>High School Graduate</td>
<td>0.224</td>
<td>0.568</td>
<td>0.102</td>
<td>0.500</td>
<td>0.233</td>
<td>0.544</td>
<td>-0.021</td>
<td>0.541</td>
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<tr>
<td></td>
<td>Some College</td>
<td>-0.484</td>
<td>0.638</td>
<td>-0.545</td>
<td>0.643</td>
<td>-0.242</td>
<td>0.651</td>
<td>-0.711</td>
<td>0.700</td>
</tr>
<tr>
<td></td>
<td>College Graduate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td>White, non-Hispanic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hispanic, Other/Multiracial</td>
<td>2.451</td>
<td>***</td>
<td>0.767</td>
<td>0.768</td>
<td>2.145</td>
<td>***</td>
<td>0.764</td>
<td>0.771</td>
</tr>
<tr>
<td></td>
<td>Lives in a Metropolitan Area</td>
<td>1.734</td>
<td>*</td>
<td>0.732</td>
<td>0.728</td>
<td>1.675</td>
<td>*</td>
<td>0.736</td>
<td>0.736</td>
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<tr>
<td></td>
<td>Years of Work Experience</td>
<td>0.318</td>
<td>***</td>
<td>0.052</td>
<td>0.053</td>
<td>0.323</td>
<td>***</td>
<td>0.053</td>
<td>0.051</td>
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<tr>
<td></td>
<td>Years of Work Experience2</td>
<td>-0.008</td>
<td>*</td>
<td>0.004</td>
<td>0.004</td>
<td>-0.008</td>
<td>*</td>
<td>0.004</td>
<td>0.004</td>
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<tr>
<td></td>
<td>Constant</td>
<td>37.242</td>
<td>***</td>
<td>0.765</td>
<td>0.782</td>
<td>37.147</td>
<td>***</td>
<td>0.771</td>
<td>0.786</td>
</tr>
<tr>
<td></td>
<td>Overall F test</td>
<td>12.332</td>
<td>***</td>
<td>0.064</td>
<td>0.069</td>
<td>9.053</td>
<td>***</td>
<td>0.073</td>
<td>0.079</td>
</tr>
<tr>
<td></td>
<td>R-Squared</td>
<td>0.064</td>
<td>0.069</td>
<td>0.073</td>
<td>0.079</td>
<td>0.079</td>
<td>0.085</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p<0.05, ** p<0.01, *** p<0.001
a Linear measures are centered and quadratic terms are created from centered measures.
Note: Coefficients on diagnoses at the family level are presented in blue and those on women's own diagnoses appear in orange.

Model 2 produces largely similar results with no relationship between any FUMs’ condition counts and predicted weekly hours, including women’s own conditions. However, in the initial version of Model 2 (not shown), OFUMs’ condition count was associated with an increase in predicted hours (B=2.652; p=0.029). Because so few women have information on OFUMs’ conditions (recall Table 2), a significant effect prompts additional exploration.
that the majority of OFUMs with health data are grandchildren (64 percent), I consider whether the effect of OFUMs’ conditions is actually a proxy for the effects of multigenerational families instead. I include an indicator of a three-generation family (where a woman, her child, and grandchild are all present in the FU) in the model, and find that predicted weekly hours increase by 3.041 in these households, and the effects of OFUMs’ conditions are reduced to non-significance (as presented in Model 2). While it is not possible to determine precisely why multigenerational families are associated with an increase in work hours, several possibilities arise. For example, women may increase their labor supply to support additional family members, or the presence of an extra parent in the household may relieve childcare-related constraints on women’s work hours compared to other types of families.  In all, findings from Models 1 and 2 suggest an obvious divergence between predictors of different measures of women’s labor supply (employment versus weekly hours), specifically in terms of the role of women’s own condition count, which was a significant predictor of employment throughout Tables 13 and 14.

Model 3 estimates the effects of specific illnesses in the family on women’s work, testing the main effect of each diagnosis as well as the interactive effects of those diagnoses with women’s condition counts and women’s educational attainment, again with the goal of determining whether certain diagnoses have class- or health-stratified effects. For each of the eight diagnoses examined here (anxiety, arthritis, asthma, cancer, depression, diabetes, hypertension, and lung disease), no main effects or interactive effects with women’s condition counts emerge. When interacted with educational attainment and entered into the model in

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44 I retain this measure in subsequent models, as the coefficient is significant and its inclusion increases the R^2 value. It should also be noted that an interaction term between OFUMs’ condition count and the three-generation family indicator is not significant (p =0.140).
sequence, however, both depression and arthritis have effects on women’s employment. In each case, the effects of the family diagnosis were significant only for the least-educated women, and served to increase predicted hours worked ($B=4.534; p=0.024$, not shown). When simultaneously included in Model 3, however, neither effect emerged, suggesting that the model might suffer from overfitting. As a result, I select the interaction with the higher (joint) $F$ statistic—depression—for inclusion in the final model.\footnote{Note that a test on the co-occurrence of these diagnoses in the family was not significant, either as a main effect ($p=0.311$) or as an interactive measure with education ($p=0.204$).} Figure 10 presents the results of that interaction.

Figure 10. Predicted Weekly Hours Worked by Depression Diagnosis in Family and Women’s Educational Attainment

![Graph showing predicted weekly hours worked by depression diagnosis and educational attainment.](image-url)

Note: Predictive margins adjusted for characteristics shown in Table 15, Model 3. Differences in hours worked by depression diagnoses are statistically significant for less than high school only. Error bars represent 95 percent confidence intervals.

The effects of depression as shown in Figure 10 are not explained away by the inclusion of women’s own depression in the model, either as a main or interactive effect (not shown), and thus may be interpreted in a variety of ways. First, it may be important to consider that women with no high school diploma may be more likely to be working in hourly-paid jobs, meaning that hours worked are directly related to earnings. In this case, it is possible that
women are compensating for reductions in labor supply related to spousal depression. Another possibility arises when considering that depression co-occurs with other conditions at high rates (see Table 10): it is possible that women with depression in their family increase their labor supply in response to additional health-related expenses in the family. Finally, it is also possible that women’s long work hours may result in heightened levels of depression in their families; for instance, the substantial body of work linking women’s work to child outcomes was described in Chapter 1.

The effect of arthritis by educational attainment, described above but omitted from the Table, also functioned similarly to that shown in Figure 10. However, that women’s own arthritis diagnosis did not mediate this effect has particular implications for the interpretation of this finding, as only women and spouses were surveyed about arthritis (see Table 3 and Figure 1). In other words, if women’s own diagnosis does not account for the effect (as I find here), then the effect of a family-level arthritis diagnosis is a spousal one. This effect is considerably more intuitive than the finding on depression: that weekly hours worked increase when a spouse has arthritis—and only among the lowest educated women—suggests that additional hours worked may be a response to spouses’ own employment. Specifically, this effect may hinge upon two specific factors: (1) the tendency toward assortative mating, that would indicate low-educated women are likely married to low-educated men, and (2) the concentration of low-educated men in low-skilled jobs, including manual labor. Taken together, it is plausible then that an arthritis diagnosis might disrupt a low-educated man’s working trajectory, causing his wife to recalibrate her weekly hours worked to compensate for the lost income.

Model 4 demonstrates that the effects of women’s own diagnoses are associated with hours worked. First, net of all other characteristics in the model, women with asthma are
predicted to work more than two-and-a-half additional hours weekly. It is unclear why
women’s own asthma diagnoses might increase labor supply (or why working additional hours
might increase asthma diagnoses) other than to suggest that asthma might be a mediator
between specific types of jobs and hours worked; for example, women working in food service
or industry settings might have higher risks for asthma as well as longer required work hours.
Model 4 also shows that the effects of women’s own lung disease and depression diagnoses are
also educationally stratified; though the women’s own depression (and its interaction with
education) did not mediate the relationship between family-level depression diagnoses and
work for low-educated women shown in Model 3, a woman-level effect emerges in Table 4,
with an effect similar to that shown in Figure 9. In the case of own depression diagnoses, the
most intuitive interpretation is not as causally modeled here. That is, long work hours may
result in heightened rates of depression among women, or that depression is symptomatic of
conditions that lead women to increase labor supply, such as stressors associated with financial
strain in the family.

In contrast, the relationship between women’s lung disease diagnoses and hours
worked is in the expected direction, though this relationship is only made clear when
calculating the predictive margins on this interaction, as shown in Figure 11. Whereas previous
interactions between educational attainment and diagnoses were most relevant for the least
educated women, the effects of lung disease are only evident among the most educated women,
for whom a diagnosis is associated with a substantial decline in predicted hours worked.
Contrasting these predicted margins reveals this effect as a decrease of 7.82 hours worked for
college graduates with a lung disease diagnosis (Bonferroni-adjusted p=0.009). It is possible that
a diagnosis of lung disease results in reduced work hours only among those for whom such a reduction is financially feasible.

Figure 11. Predicted Weekly Hours Worked by Women’s Own Lung Disease Diagnoses and Women’s Educational Attainment

![Graph showing predicted weekly hours worked by women's own lung disease diagnoses and women's educational attainment.](image)

Note: Predictive margins adjusted for characteristics shown in Table 15, Model 4. Differences by diagnosis status are statistically significant for college graduates (Bonferroni-adjusted p<0.05). Error bars represent 95% confidence intervals.

Model 5 includes the significant predictors from Models 3 and 4 and incorporates the resources of SSI receipt, transportation, health insurance coverage, and liquid assets. Women’s own asthma diagnosis is still associated with higher weekly work hours in Model 5, and the interactive effects of depression/lung disease by educational attainment remain similar across models. The one notable change in effects between measures included in Models 3/4 and 5 is that OFUMs’ condition count emerges as significant in Model 5, associated with a 2.5-hour increase in predicted hours worked. This association remains significant, despite the inclusion of a three-generation household indicator that reduced this association to non-significance in Models 2-4. A closer examination of this association suggests some confounding with SSI receipt (mean number of OFUMs conditions is significantly higher among families who received...
SSI), though the inclusion of this measure does not substantially change the coefficient on OFUMS’ condition counts.\textsuperscript{46}

As in Table 14, SSI receipt is associated with reduced labor supply, by nearly five fewer hours worked per week (B=-4.693; p=0.026), though again, this relationship may be a substitutive income effect (where SSI receipt offsets the need for labor income), or a proxy for the severity of illnesses within the home. Unlike in Table 14, Model 5 shows a significant association between health insurance coverage and hours worked. Specifically, in families with only partial year coverage, women are predicted to work an additional 4.198 hours weekly when compared to women whose families had no insurance during the year. Again, the directionality of this relationship is unclear: it is possible that women who work more hours are more likely to be eligible for health insurance coverage, though the lack of no association between hours worked and full-year coverage sheds some doubt on this interpretation. It is also possible that the quality of women’s employment might explain both hours worked and part-year health insurance coverage; for example, if women transition between jobs that require long hours and seldom provide benefits, they may experience fluctuating eligibility for public or private insurance. Though these analyses are treated as cross-sectional, it is also worth noting the possibility that women may have increased their labor supply in 2008 in an attempt to avoid the instabilities in health insurance coverage experienced in the previous year (2007, when health insurance coverage is measured). This interpretation also allows for the possibility that part-year health insurance coverage is unrelated to women’s work altogether—

\textsuperscript{46}To test whether the coefficient on OFUMs’ condition count varies substantially between models, I estimate the model with and without SSI receipt as a predictor, then use Stata’s \texttt{suest} (seemingly unrelated estimates) command to combine estimates and (co)variance matrices into one vector of parameters and a combined robust-sandwich type covariance matrix. Calculating an adjusted Wald test after this model allows for formal cross-model tests of coefficients; differences in the coefficient on OFUMs’ condition counts were not significant here (F=0.92; p=0.3399) and I include both predictors in the model presented here.
the measure here does not specify that insurance is employer-based—and that additional hours worked are associated with women working full time in response to the loss of health insurance provided by another family member, a possibility that is particularly salient considering these measurements’ co-occurrence with the beginning of the Great Recession.

In terms of demographic characteristics associated with work hours, the nonlinear effect of age identified in the models predicting employment (Tables 13 and 14) persists across all models here. In addition, predicted work hours are higher among those who are unmarried, nonwhite, and who have more years of work experience, and lower among women with children (particularly those younger than age 5), results that are coherent with existing literature. Still, in each model shown in Table 15, the $R^2$ values indicate very low explanatory power, suggesting that the vast majority of variance in hours worked is still unexplained after accounting for the included measures.47

In this section, I examined the effects of family health on women’s weekly hours worked. Unlike in predicting employment, Table 15 shows that counts of chronic conditions are generally poor predictors of average weekly work hours among employed women; indeed, a relationship between family condition count and hours worked—evident in the bivariate stage—was reduced to non-significance simply by introducing women’s age into the model. Similarly, women’s own condition counts were not significantly associated with hours worked in any of the models here, nor were counts among spouses or children. Instead, specific conditions were much more closely associated with weekly hours worked, and effects were

---

47 In building the final model in this table, I also recall the effects of region of residence in earlier analyses and test for main effects and the interaction between black race/ethnicity and Southern residence, finding no significant results for either ($p=0.9310; p=0.644$) (not shown). I also examine the main effects of access to transportation and value of liquid assets, as well as each interacted with the family condition count, finding no significant effects of any on hours worked.
intensely stratified by educational attainment (e.g., depression, arthritis, and lung disease). Further, this section makes clear that while specific diagnoses are fairly useful predictors of labor supply, again the degree to which a specific diagnosis matters varies drastically. Where hypertension and diabetes were important predictors of employment (Table 14), neither predicted weekly work hours in any of the model, with effects of depression emerging instead. Perhaps the most important aggregate lesson thus far is that the dynamics of family health impact women’s labor supply in varying ways, and effects vary tremendously depending on which component of labor supply is modeled: employment or weekly hours worked.

**Summary of Results**

In this chapter, I assessed the ways in which family health is related to women’s labor supply. Using condition counts at the family and FUM levels, and measures of specific diagnoses at the family- and woman-levels, I find substantial variability in the predictive capacity of different illness measures both within and across measures of women’s labor supply. For predicting employment, women’s own condition counts were reliably associated with lower odds of employment, with each additional condition reducing the odds of employment by about one-quarter. In no cases did general counts among specific family members predict employment, though the presence of specific diagnoses at the family level—namely diabetes, hypertension, and lung disease—reduced women’s employment. In addition, lung disease interacted with number of conditions at the family- and woman-levels, and was associated with accelerated declines in employment at higher numbers of conditions.

For predicting hours worked, condition counts were irrelevant at both the family- and woman-levels, with diagnoses of asthma, depression, and lung disease emerging as most salient.
Again, the effects of specific diagnoses were not uniform, with depression and lung disease diagnoses producing effects that were stratified by women’s educational attainment.

For both measures of labor supply examined here, the role of resources also varied, with the value of liquid assets and the availability of transportation associated with the probability of employment, and health insurance coverage associated with predicted weekly hours worked. In no cases were the effects of these resources different by women’s educational attainment, or by measures of family health. In contrast, SSI receipt emerged as a significant predictor for each outcome, though its interaction with women’s condition counts was not salient in predicting hours worked. This suggests that SSI receipt may indeed be a proxy for the severity of women’s illnesses, and that women who have multiple conditions and receive SSI are excluded from the labor force, and thus the estimation of weekly hours worked.

In the next chapter, I conclude my analyses of family health on women’s labor market outcomes by examining the effects of health on women’s earnings. These analyses are structured to build upon findings from Chapters III and IV by assessing how various health measures impact women’s earnings, and the potential that effects may be stratified by social class (women’s educational attainment), the broader context of health within the family (family- and woman-level condition counts), and the availability of resources like health insurance, liquid assets, SSI receipt, or the availability of transportation.
V. Results: Family Health and Women’s Earnings

Chapter V concludes the analyses of family health on women’s labor market characteristics: whereas the previous chapter explored the relationship between family health and two measures of labor supply, this brief chapter focuses on women’s labor market income, and assesses how various configurations of own and family health impact that relationship. As in the models predicting weekly hours worked, I employ OLS regression throughout, testing various measures of family health and their interactions with characteristics of women and their families.\(^{48}\) As in the previous sections, I continue to assess the role of moderating resources and the potentially stratified effects of health, or moderators, by women’s educational attainment.

The findings in this chapter are considerably less intuitive than those in the preceding sections. First, unlike in Chapter IV, specific diagnoses are less regularly predictive of women’s earnings than are the linear count of women’s own conditions. Further, whereas most of the relationships between health and women’s labor supply were in expected directions, the results here are often surprising, and specific to subsets of working women. It appears that the dynamic family health framework that applied well to modeling labor supply may be less influential in predicting earnings.

As in the preceding sections, I begin by assessing the role of condition counts at the family- and woman-levels in the first two models, before incorporating specific diagnoses at the family- and woman-levels in the third and fourth models. The final model in this section again pools results from the preceding versions to create a best-fit model that also considers the role

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\(^{48}\) At the outset of these analyses, I first estimate a selection equation via Stata’s `svy heckman` command. I begin with the same income measures described above (spousal income, child support receipt, welfare receipt, and food stamps receipt) as possible factors in women’s selection into the labor force, and estimate the selection equation accordingly. As with the hours equation above, a test of the coefficient on `athrho` cannot reject the null hypothesis that `athrho=0` \((p<0.856)\), and thus that a selection bias exists.
of resources. It is worth noting that in each of these models, my central interest is in predicting income, without the confounding effects of hours worked, controlled in each of the below models. Table 16, below, presents these results.
Table 16. OLS Regression Models Predicting (Logged) Labor Income, Among Employed Women Only

<table>
<thead>
<tr>
<th>Condition Counts</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>SE</td>
<td>B</td>
<td>SE</td>
<td>B</td>
</tr>
<tr>
<td>Number of Conditions in Family</td>
<td>-0.048 *** (0.012)</td>
<td></td>
<td>-0.123 *** (0.025)</td>
<td>-0.136 *** (0.026)</td>
<td>-0.098 ** (0.030)</td>
</tr>
<tr>
<td>Women's Own Conditions</td>
<td></td>
<td></td>
<td>-0.033 ** (0.010)</td>
<td>-0.034 ** (0.011)</td>
<td>-0.032 ** (0.010)</td>
</tr>
<tr>
<td>... x Months of Health Insurance</td>
<td></td>
<td></td>
<td>0.012 (0.022)</td>
<td>0.005 (0.026)</td>
<td>0.012 (0.021)</td>
</tr>
<tr>
<td>Spouses' Conditions</td>
<td></td>
<td></td>
<td>0.118 (0.086)</td>
<td>0.119 (0.095)</td>
<td>0.117 (0.091)</td>
</tr>
<tr>
<td>Children's Conditions</td>
<td></td>
<td></td>
<td>0.118 (0.086)</td>
<td>0.119 (0.095)</td>
<td>0.117 (0.091)</td>
</tr>
<tr>
<td>OFM's Conditions</td>
<td></td>
<td></td>
<td>0.118 (0.086)</td>
<td>0.119 (0.095)</td>
<td>0.117 (0.091)</td>
</tr>
<tr>
<td>Specific Diagnoses</td>
<td></td>
<td></td>
<td>0.417 ** (0.155)</td>
<td>0.381 * (0.148)</td>
<td></td>
</tr>
<tr>
<td>Arthritis</td>
<td></td>
<td></td>
<td>-0.171 * (0.071)</td>
<td>-0.156 * (0.063)</td>
<td></td>
</tr>
<tr>
<td>... x Women's Condition Count</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer</td>
<td></td>
<td></td>
<td>0.162 * (0.079)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td>0.034 (0.081)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>... x Less than High School</td>
<td></td>
<td></td>
<td>0.034 (0.081)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>... x High School</td>
<td></td>
<td></td>
<td>-0.065 (0.096)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>... x Some College</td>
<td></td>
<td></td>
<td>0.001 (0.092)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>... x College</td>
<td></td>
<td></td>
<td>0.001 (0.092)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months of Health Insurance Coverage</td>
<td>0.027 *** (0.007)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age2</td>
<td>-0.011 ** (0.003)</td>
<td></td>
<td>-0.013 *** (0.004)</td>
<td>-0.013 *** (0.004)</td>
<td>-0.012 *** (0.003)</td>
</tr>
<tr>
<td>Age2 x</td>
<td>-0.001 *** (0.000)</td>
<td></td>
<td>-0.001 *** (0.000)</td>
<td>-0.001 *** (0.000)</td>
<td>-0.001 *** (0.000)</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previously Married</td>
<td>-0.066 (0.087)</td>
<td></td>
<td>-0.007 (0.094)</td>
<td>-0.020 (0.095)</td>
<td>-0.015 (0.101)</td>
</tr>
<tr>
<td>Never Married</td>
<td>-0.063 (0.081)</td>
<td></td>
<td>-0.005 (0.077)</td>
<td>-0.011 (0.079)</td>
<td>-0.011 (0.079)</td>
</tr>
<tr>
<td>Age of Youngest Child</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Children</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child(ren) Under Age 5</td>
<td>-0.017 (0.065)</td>
<td></td>
<td>-0.031 (0.063)</td>
<td>-0.035 (0.061)</td>
<td>-0.024 (0.062)</td>
</tr>
<tr>
<td>Child(ren) Over Age 5</td>
<td>0.034 (0.051)</td>
<td></td>
<td>0.013 (0.049)</td>
<td>0.009 (0.047)</td>
<td>0.014 (0.047)</td>
</tr>
<tr>
<td>Educational Attainment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than High School</td>
<td>-0.893 *** (0.091)</td>
<td></td>
<td>-0.897 *** (0.088)</td>
<td>-1.005 *** (0.097)</td>
<td>-0.999 *** (0.091)</td>
</tr>
<tr>
<td>High School Graduate</td>
<td>-0.484 *** (0.049)</td>
<td></td>
<td>-0.495 *** (0.048)</td>
<td>-0.462 *** (0.061)</td>
<td>-0.479 *** (0.052)</td>
</tr>
<tr>
<td>Some College</td>
<td>-0.332 *** (0.041)</td>
<td></td>
<td>-0.334 *** (0.041)</td>
<td>-0.332 *** (0.046)</td>
<td>-0.331 *** (0.041)</td>
</tr>
<tr>
<td>College Graduate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>-0.204 *** (0.054)</td>
<td></td>
<td>-0.199 *** (0.053)</td>
<td>-0.206 *** (0.051)</td>
<td>-0.207 *** (0.051)</td>
</tr>
<tr>
<td>Hispanic/Other/Multiracial</td>
<td>-0.120 (0.061)</td>
<td></td>
<td>-0.111 (0.059)</td>
<td>-0.094 (0.059)</td>
<td>-0.100 (0.062)</td>
</tr>
<tr>
<td>Lives in a Metropolitan Area</td>
<td>0.233 *** (0.051)</td>
<td></td>
<td>0.227 *** (0.050)</td>
<td>0.227 *** (0.050)</td>
<td>0.224 *** (0.048)</td>
</tr>
<tr>
<td>Years of Work Experience</td>
<td>0.024 *** (0.003)</td>
<td></td>
<td>0.024 *** (0.004)</td>
<td>0.024 *** (0.003)</td>
<td>0.024 *** (0.003)</td>
</tr>
<tr>
<td>Average Weekly Hours Worked</td>
<td>0.056 *** (0.002)</td>
<td></td>
<td>0.056 *** (0.002)</td>
<td>0.056 *** (0.002)</td>
<td>0.055 *** (0.002)</td>
</tr>
<tr>
<td>Constant</td>
<td>8.495 *** (0.125)</td>
<td></td>
<td>8.526 *** (0.125)</td>
<td>8.517 *** (0.131)</td>
<td>8.510 *** (0.124)</td>
</tr>
<tr>
<td>Overall F test</td>
<td>119.633 ***</td>
<td></td>
<td>99.042 ***</td>
<td>74.859 ***</td>
<td>70.701 ***</td>
</tr>
<tr>
<td>R-Squared</td>
<td>0.438</td>
<td></td>
<td>0.445</td>
<td>0.449</td>
<td>0.452</td>
</tr>
<tr>
<td>N</td>
<td>3,098</td>
<td></td>
<td>3,098</td>
<td>3,098</td>
<td>3,098</td>
</tr>
</tbody>
</table>

*p<0.05, **p<0.01, ***p<0.001

Note: Each model also tested for effects of liquid assets; no main or interactive effects emerged, and thus none are included in the models presented here. Coefficients in blue represent health measures at the family-level, while coefficients in orange represent those at the woman-level.
In Model 1, the coefficient on family conditions indicates that each additional condition in the family is associated with a decline in annual log earnings of about five percent. Model 2 incorporates condition counts for individual FUMs; here, condition counts for both women and their children are associated with reductions in log earnings, with each additional condition reducing log earnings 12 and 3 percent, respectively. Unlike in the preceding chapter, OFUMs’ condition counts have no impact on women’s log earnings.

Model 3 tests the effects of the eight specific diagnoses described in the previous sections (anxiety, arthritis, asthma, cancer, depression, diabetes, hypertension, and lung disease), testing each condition as a main effect and as interacted with women’s educational attainment and condition counts at the woman- and family-levels. The only diagnoses relevant at the family level are cancer and hypertension, the effects of which diverge by educational attainment. First, cancer has a distinctly positive effect on women’s log earnings, with such a diagnosis associated with log earnings that are 16 percent higher than among families without cancer. Including women’s own cancer diagnoses in the model does not mediate this relationship ($p=0.009$ when own cancer is included). While the direction of this relationship is unanticipated, existing research has suggested informal caregivers in higher-educated groups may increase earnings in response to family illness, in order to hire paid caretakers (Lilly et al. 2007). That higher log earnings are associated with cancer diagnoses are at the family level and not explained away at the individual level, and that cancer can be an especially burdensome diagnosis lends credibility to this potential mechanism; however, effects of a family cancer diagnoses did not vary significantly by educational attainment in this sample (perhaps due to the relatively low reports of cancer here). For hypertension, there is a significant effect of family-level diagnosis by women’s educational attainment, with substantially higher log earnings.
predicted among low-educated women in families with this diagnosis (B=0.417; p=0.028) as compared with low-educated women without such a diagnosis. It is difficult to identify a causal pathway that might lead low-educated women with hypertensive families to have increased earnings; however, it is plausible that women without high school diplomas may have families who are more likely to access health care and receive such a diagnosis if they among the higher-earnings non-graduates.

As with Model 3 at the family level, Model 4 includes woman-level diagnoses; here, there is no significant effect of cancer, though the relationship between hypertension and educational attainment persists at the individual level. In addition, the interactive effects of arthritis and women's condition count are significant, as shown in Figure 12, below. At lower numbers of conditions, women with arthritis have higher predicted earnings than women without arthritis, and as conditions increase, the predicted earnings of arthritic women dip below those without arthritis, with predictive margins significantly lower among arthritic women by 6 conditions.
Figure 12. Predictive Margins on Log Earnings for Women’s Own Arthritis Diagnoses by Women’s Condition Counts

Note: Predictive margins adjusted for all characteristics listed in Table 16, Model 4. Margins are calculated in terms of log earnings, which I transform back into dollars for presentation purposes here. Differences by arthritis diagnosis are significant at 0 and 1 condition, then again at 6 and 7 conditions ($p<0.05$). Note that “zero” conditions is not a feasible value for women diagnosed with arthritis, but is calculated and graphed for comparison purposes only.

Model 5 pools results from the preceding models and incorporates resources into the model by testing main effects of each resource (health insurance coverage, access to transportation, SSI receipt, and value of liquid assets) and the effects of each interacted with women’s educational attainment and family- and woman-level condition counts. When entered simultaneously into the model, the interactions between hypertension diagnoses at the family/woman-levels and educational attainment are not significant; I retain the interaction with women’s own diagnosis, which yields a higher $R^2$ value and has a larger $F$ statistic. The interaction between women’s arthritis diagnosis and condition count remains significant, though the effects of cancer at the family level dissipate in this model. Of all resources tested, only the interaction between months of health insurance coverage and women’s condition count is significant. Unlike in Table 15, a continuous measure indicating months of health insurance coverage was a better fit than the categorical indicator of no coverage, partial-year coverage,
and full-year coverage. Figure 13 shows the predictive margins associated with this interaction, indicating higher income among those with full-year insurance coverage, and the lowest among those with no insurance; further, declines in predicted earnings at higher numbers of conditions are more drastic for those with no insurance coverage.

Figure 13. Predictive Margins on Log Earnings for Months of Health Insurance Coverage by Women’s Own Condition Count

Note: Predictive margins adjusted for all characteristics listed in Table 16, Model 5. Margins are calculated in terms of log earnings, which I transform back into dollars for presentation purposes here. Margins by months of insurance coverage are calculated for all month values, but shown only for 0, 6, and 12 for readability.

In terms of demographics, as with all previous models, the curvilinear effect of age remains intact, as do the positive impacts of years of women’s work experience. Unlike in the previous sections, for labor income, there is a consistent positive effect of metropolitan residence in all models, so that net of all other covariates, metropolitan residence is associated with roughly a 20 percent increase in annual log earnings. Also unlike in previous models, the presence of young children in the household does not impact log income, with the effects fully mediated by controlling for average weekly hours worked. The effects of educational attainment are robust across all model specifications here, with higher values of predicted log income at higher levels
of educational attainment. There was no nonlinear effect of work experience in these models, and the quadratic term is omitted here.

In terms of model fit for this section, the $R^2$ value on all models is relatively large, with nearly 47 percent of the variance in women’s log earnings explained in Model 5. However, much of the models’ explanatory power is a result of controlling for hours worked; prior to its inclusion, the models’ $R^2$ value was closer to 15 percent (though the effects of various health indicators were not substantially different in those models). By excluding the highest earners, Examining the residuals from Model 5 alongside observed earnings indicates that the model is better fit for higher earning women than for those at the lower end of the wage spectrum. Identifying additional measures that improved the model fit for low-earning women was largely unsuccessful.⁴⁹

In this chapter, I assessed the relationship between family health and women’s log income, documenting the ways in which condition counts and specific diagnoses at the woman- and family-levels are related to income. As in the models predicting employment (Tables 13 and 14), women’s own condition count was a relevant predictor of income, associated with decreased earnings in each model. Unlike in earlier sections, however, children’s condition counts also matter here, associated with a 3 percent decline in women’s earnings across all models. As in earlier chapters, specific diagnoses are useful predictors, and again, demonstrate effects that are

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⁴⁹ To determine the effects of outliers on these models, I identify cases with log income values in the highest and lowest one percent of values (n=32 and 30, respectively), and re-run Model 5 while excluding each group in turn. I then calculate predicted values and residuals, and use an adjusted Wald test to compare the mean value of residuals from the full and reduced sample model; when excluding high earners, test results indicate a large difference ($F=896.90$, $p=0.0001$) values, although the absolute values are quite small ($e=-0.0111$ in the full model and $e=0.003$ in the reduced). I use `suest` to generate a simultaneous parameter vector and covariance matrix from the full and reduced models, and test for equality of the overall model ($F=3.50; p=0.002$) and on specific coefficients of interest (e.g., arthritis $\times$ women’s conditions: $F=0.94; p=0.398$). Despite statistically significant differences in models, there are few substantively meaningful differences between models, and I am hesitant to exclude cases with otherwise reasonable outlying values for model convenience. Results excluding low earners are similar to those regarding high earners and are available upon request.
stratified by the demographic and other health characteristics of women’s families. In this section, women’s arthritis diagnoses interacted with their condition counts to predict lower earnings, just as lung disease interacted with women’s condition counts to predict lower weekly hours worked in Chapter IV. Also similar to the preceding chapter assessing labor supply, the effects of health were not uniformly associated with decreased income. For example, a cancer diagnosis in the family was associated with increased earnings, as was the presence of hypertension among the least-educated women. In the case of income specifically, it is especially difficult to speculate about the causal ordering of these relationships, since income is likely key in connecting families with health care and elevating their probabilities of being diagnosed with particular conditions. Of course, it is also possible that the presence of certain diagnoses do indeed precede heightened levels of income—for example, women might seek higher-paying jobs for reasons related to family or own health, such as purchasing caregiving services, securing health insurance, or meeting medical expenses. However, it is not possible to assess this relationship more deeply here, given the limitations in these data (discussed in greater detail in the following chapter).

In addition to health effects, there are also some common findings regarding resources between this chapter and the preceding one. For example, women’s own condition counts interact again with resource measures (months of health insurance coverage here, and SSI receipt in Chapter IV). However, the results from this section regarding resources are again considerably more difficult to speculate upon than those in the preceding chapter, largely because of the strong associations between women’s income and measures like health insurance coverage and liquid assets value. The lack of clarity in these findings suggest that estimating the effects of family and individual health on earnings may be better-suited to
estimation over a longer period, for example in a longitudinal model that can assess aggregate
effects of health across the lifecourse and untangle causal ordering. Regardless, the systematic
assessment of these effects is valuable for shaping the direction of future research in this area,
and for considering potential improvements to the modeling of women’s income.
VI. Discussion and Conclusions

The central purpose guiding this dissertation has been threefold: that is, to determine (1) how multiple chronic conditions are distributed within and across families, (2) whether family health is associated with women’s employment and related outcomes, and (3) if family health does predict women’s employment characteristics, to determine whether this relationship operates uniformly across social class. I also explore the possibility that certain resources might “buffer” families from the potential effects of poor family health, and that those resources might function differently based on the health and demographic characteristics of women’s families. In each stage of the analytic process, I attempted to systematically explore these issues within a framework that situates family health as dynamic, where different measures of family health reveal wide variation in their effects on women’s employment. Equally important is a discussion of the implications around these findings that can situate this research back into the broader framework of health and employment as delineated in Chapter I. In this chapter, I will review some of the major findings from the preceding pages, describe some of the limitations of this work, and discuss the implications of these findings in frameworks geared toward informing policy and future research.

Summary of Findings

In Chapter III, I sought to document patterns of family health by exploring the distribution of chronic conditions within and across families. My intent was to apply a framework that treated intra-family health as a conceptually meaningful construct—an approach absent from the majority of the existing literature—and to examine the landscape of intra-family health, assessing how the features of family health might vary by social class. I began that chapter by examining the distribution of multiple chronic conditions in families, with particular
attention to variation by women’s educational attainment, here the proxy for social class. The findings demonstrated that families of the lowest educated women were indeed disproportionately likely to report additional chronic conditions, with higher mean condition counts and a lower proportion of families reporting to be illness-free than among the families of college-educated women. Implicit in these findings are questions associated with risks for illness, including health-related behaviors and access to health care, as well as the implications of specific illnesses (discussed below).

Chapter III also explored the distribution of eight specific diagnoses (anxiety, asthma, arthritis, cancer, diabetes, depression, hypertension, and lung disease) across families, identified as especially prevalent and meaningful in a public health context. The finding that certain conditions—namely hypertension, diabetes, arthritis, lung disease, and arthritis—are associated with women’s educational attainment raises the question whether the effects of differentially distributed illness might also be divergent by class. Beyond the presence of given diagnoses at the family level, this chapter also documented how these eight specific diagnoses were distributed within families, examining the prevalence of certain diagnostic pairs in families (as in Table 9), and within distinct individuals within families (as in Table 10). Of particular interest are the elevated rates of certain diagnoses that occur in families, which highlights the unique role of the family as a site in which genetic, environmental, cultural, and behavioral components intersect. For these co-occurring illness pairs—for example, more than one quarter of family unit members here who have a depression diagnosis live with at least one other person with depression—both the roots and implications are unknown. That is, it is possible that a diagnosis in one family member may actually lead to diagnosis in another, by improving recognition of symptoms or acknowledgement of shared family risk characteristics. In the case of depression,
the presence of one person who has already been diagnosed may increase awareness of mental
health in the family unit more broadly, and facilitate diagnosis for others afflicted. Engagement
with treatment options might be improved as knowledge on coping, therapy, and
pharmaceuticals are shared within the family. However, it is equally possible (and perhaps
simultaneously possible) that co-occurring diagnoses in the family can complicate overall family
well-being, as multiple family members struggle to cope with illnesses that may be better
understood within the family, but not necessarily more easily managed just because that
knowledge exists.

In short, the patterning of illness within the family suggests that the family unit is more
than simply a context in which illness unfolds. Rather, it is particularly important that the health
conditions of one family member may be related to those in another: Litman (1974) stresses
the importance of the connections between health and family, describing “the socialization of
health attitudes, values, knowledge and beliefs, family decision-making in health and health care,
and the role of the family in health and illness behavior” (1974: 497). Indeed, for illnesses like
hypertension and lung disease, health behaviors that structure risk, like high sodium intakes and
tobacco use, are well known. In terms of a “culture of health,” research demonstrates that
orientations toward certain health practices are transmissible: for example, Quadrel and Lau
(1990) note that adolescents’ and young adults’ attitudes toward physician use are associated
with their parents’ attitudes toward the same. In short, Chapter III demonstrates that health is
not only patterned within families in terms of condition counts, or general burdens of poor
health, but also in the concrete appearance of specific illnesses. There is a substantial patterning
to the distribution of specific diagnoses within family units, which only emphasizes the
importance of conceptualizing family health in multiple ways.
For Chapter IV, the goal was to assess the ways that patterns of family health documented in Chapter III might impact women’s labor supply, and to determine whether tangible resources like savings accounts or vehicle ownership might temper the effects of poor family health on women’s labor supply. I attempted to answer the first part of the question by examining the number of conditions in the family and among specific family members, including women themselves, and by assessing the role of specific illnesses at both the family- and woman-levels on the odds of women’s employment and their predicted weekly hours worked. By utilizing multiple health measures, I attempted to capture some of the nuanced features of family health that emerged in Chapter III.

I began each of the regression models by testing the role of the most general measure—number of conditions in the family unit—and progress through a systematic effort of testing increasingly specific health measures. While initial models in Chapter IV demonstrated a linear relationship between family health and women’s employment, as the analyses progressed, it became clear that the relationship between family health and women’s employment is complex, with effects that vary by demographic and health characteristics of women’s families, and that emerge in different constellations depending on the outcome measure. For example, several diagnoses were associated with women’s employment, but are not predictive of weekly hours worked (e.g., hypertension and diabetes). Other diagnoses, like asthma and depression, were associated with work hours, but not the odds of employment. In several cases, the effects of specific diagnoses were captured only through interactive measures, associated with lower labor supply only for some groups. In each case, these diagnoses were associated with labor supply among women whose characteristics are traditionally associated with a disadvantaged social location: women with no high school diploma, women of color, and women who have
multiple illnesses, either themselves or in their families. That specific diagnoses may be
important predictors of women's employment outcomes is not a new idea (e.g., Bartley et al.
1992), however, the systematic assessment of different diagnoses at the family- and woman-
levels and their stratified inter-family effects is, to my knowledge, a new addition to this
literature.

Regarding the second relevant question for this chapter, I also examined the potentially
moderating role of resources, and identify few consistent effects. By and large, SSI receipt,
health insurance coverage, the availability of transportation, and the value of liquid assets did
not “buffer” women from the effects of poor health. Instead, these resources demonstrated
complex relationships with the outcome measures, and the role of each in women’s broader
landscape of employment-related decision-making is unclear, requiring further research.
However, in all, the complexity of the interactive relationships discovered in Chapter IV
directly links to findings from Chapter III by underscoring the dynamism of family health, and
the importance of multi-dimensional approaches that include multiple measures of health among
workers and their families.

In Chapter V, I assessed the impacts of family health on women’s log income and again
identified a series of complex and varied relationships. As with Chapter IV, there appeared to
be an initial linear impact of family health, which is then mediated by women’s own conditions.
As specific diagnoses at the family- and woman-level were incorporated into the models, it was
again evident that the presence of specific illnesses does not have straightforward impacts
across all families. Yet again, a different constellation of illnesses emerged as the most salient
predictors of income, including cancer, arthritis, and hypertension, and, for the first time,
children’s condition counts. As in Chapter IV, Chapter V found that most of the relationships
between health and women’s employment were concentrated among particular subgroups of women, including women with multiple illnesses and women without high school diplomas. Admittedly, these subgroup effects on earnings are not as intuitively interpretable as the effects on labor supply; however, that both woman- and family-level measures are important here again underscores the importance of considering diagnoses among workers and their families. The following section turns to a more explicit discussion of the class-related stratification in the effects of certain health measures on women’s employment outcomes and examines a few of the most prevalent effects in greater detail.

**Interactions between Social Class, Family Health, and Family Characteristics**

Findings from Chapters IV and V indicate that certain conditions matter more than others, and that often, conditions are only predictive along certain segments of the population. For instance, I found that a family-level hypertension diagnosis was strongly related to black women’s probability of employment, while a similar diagnosis mattered much less for non-black women. Some illnesses also had disproportionately strong relationships with employment for the lowest-educated women, with arthritis and depression related to higher predicted work hours among women without a high school diploma. These results suggest that not only are lower-educated women’s families disproportionately burdened with chronic conditions, as shown in Chapter III, but also that the effects of these illnesses may be disproportionately patterned too, as shown in Chapter IV.

One illustration of the disproportionate influence of health is the stronger relationship between family hypertension and employment for black women compared to the relationship with this diagnosis for white women, as shown in Chapter IV. In particular, the relationship between hypertension and race suggests some specific function of high blood pressure for black
families in particular. It should be noted that black adults experience hypertension at rates about twice those of white adults (Williams 2002), and a substantial volume of research attempts to explain this relationship via the mechanisms of racism and discrimination (e.g., Brondolo et al. 2011; Cozier et al. 2006; Krieger 1990), exposure to environmental factors (e.g., Ford, Kim, and Dancy 2009), chronic stress (Hicken et al. 2014), and heightened rates of obesity (Flegal et al. 2010). Other research focuses on the health-related consequences of high rates of hypertension, noting that especially high rates of hypertension-related morbidity and mortality for blacks (e.g., Gillum 1996). However, there is a real dearth of research that attempts to describe how the nexus of hypertension, its risk factors, and/or its related effects might shape outcomes beyond the arena of health (e.g., economic outcomes). Clearly a more explicit examination of the effects of hypertension on employment among black families is one potential avenue for extending this literature.

At this point, it is unclear by what mechanism hypertension might affect the probability of employment specifically for black women’s families. It is possible that a hypertension diagnosis is a proxy for a more intensive set of symptoms not captured in the health measures here (for example, the link between renal issues and hypertension is exacerbated for black women; see Williams 2002). In this case, improving measurement of associated symptoms and co-occurring conditions related to hypertension could improve the capacity for identifying whether these employment effects truly are related to hypertension in particular, or whether they are better described by a mutual relation with some other factor. For instance, some research finds a link between hypertension and cognitive decline, which could explain why a seemingly benign diagnosis like hypertension might actually produce more substantial effects (see Elias et al. 2012; Kuo et al. 2005; Knopman et al. 2009). Finally, it is also possible that this
relationship runs in the opposite causal direction, and that elevated rates of hypertension are more prevalent among families in which women do not work, perhaps linked by heightened levels of stress (e.g., Hicken et al. 2014).

In the models on weekly hours worked, family-level depression and arthritis were each associated with labor supply among the lowest educated women. In each case, a family-level diagnosis is dramatically different for women without high school diplomas than for other women: while arthritis and depression have no effect on hours worked for women of any other educational group, the diagnoses are associated with large increases in weekly hours worked for the least-educated women. That controlling for women's own diagnosis did not attenuate this relationship suggests that specific diagnoses in the family have a distinct relationship with women’s work, independent from women’s own diagnoses. In another example, women’s own lung disease diagnoses were associated with hours worked for some educational groups, specifically in that only the most educated women have a reduction in hours associated with a lung disease diagnosis. This coheres with existing research that suggests that the relationship between women’s own health and employment varies by social class, particularly in that lower income women have fewer opportunities to “choose” to reduce their hours or exit the labor force (e.g., Bartley et al. 1992).

Taken together, the interactive findings reviewed here certainly indicate that the relationship between health at the family- and woman-levels and women’s employment is complex. It appears that while women’s own conditions have some linear effects, the effects of family illness operate differently. In Chapter I, I propose that the established links between workers’ own health, children’s health, informal caregiving, and employment raise the possibility that informally managing routine family health care could become burdensome for women, and
attenuate their participation in the labor force. However, the findings of this dissertation indicate that women’s role in managing family health may be associated less with some quantitatively intensive burden, like a high number of chronic conditions, and more centrally associated with addressing the dynamics of varying diagnoses among family members. Further, the finding that a spousal arthritis diagnosis is associated with increased work hours for low-educated women reveals a different potential linking mechanism between women and their families’ health. That is, women may be responsible for helping family members manage the burdens of illness (e.g., addressing symptoms, administering medication, or keeping contact with health care providers), the coordination of which might lead to a necessary reduction in labor supply. In this framing, family illness results in health-related tasks and care that become women’s responsibility. However, these findings indicate the equally plausible result that women may also find themselves directly responsible for addressing the implications of their families’ illnesses that are unrelated to care and health management, as when an ill spouse must reduce his labor supply or when a child enters a costly form of therapy. In these cases, family illness would result in obligations that are not within the sphere of health management, reframing the way that women understand the responsibilities that shape their working lives. In short, it is evident that poor family health does not uniformly attenuate women’s labor supply (e.g., Lilly et al. 2007), and instead operates in complex, family- and diagnosis-specific ways.

**Policy Implications**

In light of the findings described in the preceding sections, it becomes especially important to consider the implications of this research in a policy framework. First, the high rates of chronic condition(s) and co-occurring illnesses among American adults is not a new finding, though the estimates from this research accord well with existing estimates of
individual-level disease prevalence. However, one of Chapters Ill’s most central contributions to this literature is the documentation of high rates of co-occurring illnesses across different members of the same family unit. That certain diagnoses occur at high rates across family members suggests that social policy research would not be amiss in conceptualizing the family as a site for the production of health. If risk factors for poor health are shared within a family—whether those risks are environmental, behavioral, cultural, or genetic—there could be real utility in addressing shared risk factors at the family level, and drawing on the natural supports of a family unit to help cultivate safer, smarter environments. At the same time, the role of social class in these associations is a key consideration. As with research on social class and health at the family level, the stratified distribution of illness raises issues of individual responsibility for health, individual and family rights in the transmission of health-related habits, beliefs, and practices, and the implications of not addressing these issues for broader issues of inequality. As complex as these emergent questions are, existing research does provide some guidance on practical efforts that can reduce health inequalities. For example, one body of literature suggests that the relationship between social class and poor health can be traced to childhood, where early pathways into disease (especially those related to early immune system factors) are related to lifelong trajectories of health (Ziol-Guest et al. 2012). In these cases, infusions of income in these early periods might help assuage some of the health disparities that emerge by adulthood. One practical example of such an effort would be the Earned Income Tax Credit, one of the most highly regarded social safety net programs for its effects on a whole host of outcomes, from employment to health to scholastic achievements (see Marr, Huang, and Sherman 2014 for an overview of this literature).
In this paper, health insurance coverage had few consistent effects on the relationship between family health and labor market outcomes, and those that did emerge were difficult to assess in any exogenous context. Of course, this does not indicate that health insurance is not critical to families in a host of other important ways. For example, having health insurance coverage might not influence the dynamics between family health and work outcomes, but it certainly could influence the chances of a family becoming chronically ill in the first place.

Access to preventive health care—an important component of health insurance in general, but also of the health care reforms under the Patient Protection and Affordable Care Act more specifically—may help families retain health-positive practices and avoid escalation of early symptoms, key for several of the sometimes-preventable diseases measured here (e.g., hypertension). Insurance coverage can also protect families from poor financial outcomes, such as full responsibility for large medical costs around expensive and unexpected health problems.

Further, this dissertation cannot account for health insurance quality, likely an important component of how well insurance performs as a buffer. Future research should explore how outcomes might vary by type of insurance (e.g., public versus private coverage), insurance source (e.g., employer-based health insurance versus directly purchased), and affordability (e.g., high deductible versus subsidized via the marketplace).

Another key consideration is how the effects of insurance on family health and employment-related outcomes might have changed since the implementation of the Affordable Care Act, which the data here precede. Since its implementation in 2010, the Affordable Care Act has led to some substantial changes in access to health care. More research is needed to consider how the interactions of health care costs, mandates on employers to meet certain
coverage requirements, and the role of employees (and in particular, their hours worked) might interact in the context of family health.

As shown in Chapter IV, health problems—whether among women workers or their family members—are associated with decreases in labor supply. These findings accord with research on concepts related to family health reviewed in Chapter I (e.g., informal caregiving) in that the bulk of existing research finds that poor health leads to declines in labor supply. However, in modeling the effects of health on women’s hours worked in Chapter IV and on earnings in Chapter V, I find that health cannot be adequately conceptualized as a uniform suppressor of labor supply and earnings. Instead, there are some subgroups of women for whom poor health in the family is associated with an increase in hours and earnings. Identifying these differential impacts of family health is critical when considering relevant policy implications. For instance, if poor family health precludes women from participating in the labor force, a set of policies around improving the health of workers and their families via affordable health care, providing formal or informal respite options for women who face serious informal care burdens, and helping women to access health related resources could be important. On the other hand, if women are increasing their hours in response to certain diagnoses in the family—for example, as with low-educated women whose spouses have arthritis—the policy burden may be less central to the health system, and more related to the labor market. For example, ensuring that low-skilled workers have options for pensions, retirement, or disability-related coverage might ensure that women are not forced to compensate in hours for a change in their spouses’ labor supply. Other work-relevant policies more broadly relevant to low income populations might also be effective here, including an increased minimum wage and ensuring qualified workers take advantage of refundable tax credits (e.g., the Earned Income Tax
Credit) that can provide important emergency funds or savings buffers for other points in the year.

One important approach for ensuring workers are protected from health-related circumstances is to increase the availability of sick leave, and paid sick leave more specifically. For women whose own health conditions are associated with reduced labor supply (e.g., the reduced odds of employment associated with hypertension among black women), this type of policy could prevent women from having to make the choice between looking after their own health and losing a job. Especially in workplaces with irregular or contingent scheduling, an unexpected illness can be incredibly disruptive. Without the protection of a regular schedule, it is easy for managers to revise women’s schedules week-to-week, reducing hours as repercussions for workers seen as unreliable. The protection of even a handful of paid sick days might be effective for these groups in particular.

Limitations & Directions for Future Research

Perhaps the most significant limitation of this work is its cross-sectional nature. As a result, a causal relationship between family health and women’s employment could not be estimated. Since a plethora of literature has established that the relationship between employment and health is bidirectional (e.g., Cai 2010; Ross and Mirowsky 1995), there is little reason to expect that this relationship consistently operates in a single direction, especially without longitudinal data. To remedy this, a dataset that includes additional data points, tracking changes in family health and illness over time would allow for an estimation of a multilevel model that could, for example, account for individual heterogeneity in propensity to work and examine how the impacts of family health and employment unfolds over time. Beyond the generally superior ability for longitudinal data to document causal relationships, such a data
structure could be particularly important when examining the role of family illness specifically. For example, if a child is diagnosed with a severe or limiting chronic condition at birth, women may adjust their labor supply immediately in response. In this case, measurement of labor supply that precedes the child’s birth could improve understanding of these processes, though such research is currently prohibited by the lack of detailed health measures in other surveys (e.g., NLSY; NHIS; MEPS) and the lack of longitudinal information on the family in the PSID.

Despite that the data here preclude identification of causal relationships between family health and employment, I suggest that regardless of the directionality of this relationship, the “end result” is largely the same. For instance, much of the existing research related to “family health” and employment is framed around women as caregivers (e.g., Carmichael et al. 2009; Leigh 2010) and poses the possibilities that (1) caregiving burdens lead to employment outcomes, or (2) women’s pre-existing employment statuses result in a “self-selection” into informal caregiving. As applied here, the more appropriate questions are whether the burdens of poor family health lead to employment outcomes, or whether employment outcomes lead to poor family health. For this research, the notion of “self-selection” is less about women making a (constrained) choice to have their families be unhealthy, and more about whether the effects of low employment results in poor family health. Even if the bulk of the relationship between family health and women’s employment is in the latter direction—that is, that this relationship is best described as low levels of employment leading to disproportionate levels of family illness—the burdens of navigating family illness in a context of unstable employment are likely still complex and difficult to manage. In other words, whether family illness disrupts women’s employment or is a product of the chronic stressors and financial burdens associated with non-employment, the psychological burdens of care, the physiological burdens of illness, and the
economic burdens of non-employment are all present, and may be mutually reinforcing. Nonetheless, working to identify the degree to which this relationship is bidirectional is still important, and is absolutely a key direction for future research.

In relation to the class-stratified findings of this dissertation, I pose the possibility that the relationship between family health and women’s employment may fall somewhere in the midst of the two extremes described above. That is, poor family health may not necessarily annihilate women’s employment prospects, nor be a characteristic concentrated only among those with limited labor force participation. Instead, it is possible that family illness, whether high condition counts or the presence of specific illnesses, merely exacerbates the “looseness” of some women’s attachment to the labor force, with effects concentrated among the least-stably-employed women (see Heitmueller 2007). If this is the case, the results here are still meaningful, despite the inability to establish a causal relationship, because whether poor health leads to poor employment prospects or poor employment prospects lead to poor health, there is likely a long-term cost to women and their families regardless. Navigating health issues while facing stretches of time out of the labor force (without earnings and while not accruing work experience) likely jeopardizes families’ financial stability in the long run, regardless of the direction of the association. Of course, this is not to suggest that determination of a causal relationship is not important; indeed, doing so is key for propelling this area of research forward. However, given the strong emphasis on the importance of causality in much of sociological research, it is important to recall that documenting associations amid likely bidirectional relationships is still informative for assessing the implications of a given nexus of factors like health and employment.
Beyond the issue of untangling causality, another limitation to be acknowledged is the inconsistent availability of health measures across the family unit. Though it is rare to find a data source that includes data on more than one adult and child in the same family as in the PSID (see discussion of data sources in Chapter II), the PSID’s measures are still far from perfect. In particular, because the main PSID’s health items specifically query heads and wives of family units (FUs), there is an underestimation of health characteristics among FUMs who are not wives, mothers, husbands, or fathers of the primary family unit. This underrepresentation of other family unit members (OFUMs) becomes particularly problematic when considering the context of an aging population and the shift to informal elder care. As such, the so-called “sandwich generation” of (generally) women who care for aging parents while raising children of their own are likely not fully represented here. The consequences of this underrepresentation likely translate to an undercounting of illnesses, especially co-occurring or advanced illnesses that afflict aging populations at higher rates. However, it is difficult to estimate how the conclusions here might be affected if data on all family members were available, since there are no known sources of existing research that might provide reasonable comparison points of family health. Of course, practicality plays a role in the availability of such data, as surveying an entire family is an expensive and laborious undertaking, and many surveys are simply not structured to do so (e.g., the NHIS surveys one sample adult and child, as described in Chapter II).

In addition to the limitations on survey participants, the PSID data are also limited in the scope of their health condition measures, particularly in terms of quantifying the severity of illnesses. These analyses would undoubtedly be improved by the inclusion of more detailed health descriptions. However, where other data often inquire about the severity and resultant
limitations associated with specific conditions, examining the impact of family health on women’s work might be substantially improved by also understanding how women’s time is allocated to family health care. For instance, a person might report few limitations associated with an illness, but that result might be a result of their consistent efforts to manage that condition. If women are responsible for some or all of this management activity—for example, coordinating medical care, purchasing or administering medication, assisting in therapies, or transporting sick FUMs to medical appointments—then it is reasonable to expect that the demands on their finite time may adversely impact labor supply or labor market outcomes.

Beyond the brevity of detail in these measures, it is also unclear to what degree reported diagnoses are reflective of true prevalence, versus unevenly distributed rates of formal diagnoses. While help-seeking patterns have long known to vary by gender (Addis and Mahalik 2003; Good et al. 1989), it is unclear how diagnostic propensities might interact with factors of access to health care (e.g., health insurance coverage) or other family characteristics. Additionally, the degree to which reported diagnoses are associated with actual diagnoses should be considered here. Accuracy in reporting likely varies by diagnosis, though at least some research finds good performance of self-report diagnoses (e.g., as with arthritis, per Sacks et al. 2005). While additional detail on these health measures would improve this research, the collection of detailed, corroborating health information across entire families is likely to be prohibitively expensive and thus unrealistic.

Finally, additional moderating variables, including indicators of the availability of paid sick leave or vacation time, and a quantification of child care efforts contributed by others (both in and outside the household) may have improved this research. For instance, the willing support of a live-in grandparent, a spouse with access to flexible leave time, or access to a childcare center
that does not remove children who are ill (e.g., Chaudray 2004) might further stratify the effects of family health on women’s employment.

Although this research began to document some of the relationships between family illness and employment, more questions remain. Future research should seek additional data sources that can expand on measures of illness within and across family members, and can examine these effects in a longitudinal framework. Another potential direction for future research might include qualitative efforts to better understand the components of women’s commitment to, and responsibility for, managing family health, and identifying how those responsibilities intersect with responsibilities of providing financial stability in a family where other members may not be able to work. Researchers might also employ a time use framework for assessing the health-related activities that women undertake within the family. In one example, research reviewed in Chapter I found that women are more likely to miss work due to a child’s illness than are men. However it is still unknown in what other health-related activities women regularly engage, how time consuming these activities might be, and how decision making around decisions to miss work for one’s own illness or another family member’s illness unfold in the context of women’s lives.

In short, this dissertation has identified the prevalence of multiple illnesses within family units, the disproportionate patterning of family illnesses by women’s educational attainment, and the complex relationships between different measures of health and women’s employment. In nearly every case, these findings suggest that family illness does not have a simple additive effect on women’s employment; that is, it is not possible to say that family members who are sick require greater levels of care that is regularly sought from women, as might be true in the caregiving literature. Instead, I find important variations within the category of family health;
that certain family members diagnosed with specific illnesses, in the context of families with particular characteristics and resources, bear different influences on women’s employment. As a result, perhaps the most overarching conclusion in this dissertation might be in its answer to the question is family health related to women’s employment outcomes, to which I might answer—in the tradition of countless other sociological writings—yes, but it’s complicated.
REFERENCES


Ann Arbor, MI: Survey Research Center, Institute for Social Research, University of Michigan.


Bayliss, Elizabeth A., Martha S. Bayliss, John E. Ware, John F. Steiner. 2004. “Predicting Declines in Physical Function in Persons with Multiple Chronic Medical Conditions: What Can We Learn from the Medical Problem List?” Health and Quality of Life Outcomes 2:47-55.


Friedman, Bernard, H. Joanna Jiang, Anne Elixhauser, and Andrew Segal. 2006. “Hospital Inpatient Costs for Adults with Multiple Chronic Conditions.” Medical Care Research and Review 63(3):327-346.


StataCorp. 2013a. “SVY Glossary.” College Station, TX: Stata Press.
StataCorp. 2013b. “Stata Multiple-Imputation Reference Manual: Release 13.” College Station, TX: Stata Press.


## Table 17. Summary of Literature on Employment and Specific Diagnoses

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Employment Outcome</th>
<th>Effect</th>
<th>Citation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthritis</td>
<td>Hours Worked</td>
<td>Reduced by 32-38 percent</td>
<td>Mitchell and Burkhauser (1990)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Exit labor force before age 55</td>
<td>Odds increased seven-fold</td>
<td>Mitchell (1991)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Withdrawal from the labor market</td>
<td>Increased by 50 percent</td>
<td>Christiansen and Kallestrup-Lamb (2012)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Arthritis</td>
<td>Risk increased by 1.8 compared to those with no asthma</td>
<td>Hakola et al. (2011)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Arthritis + Depression</td>
<td>Risk increased by 3.6 compared to those with no asthma</td>
<td>Hakola et al. (2011)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Arthritis + One other comorbid condition</td>
<td>Risk increased by 2.2 compared to those with no asthma</td>
<td>Hakola et al. (2011)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Arthritis + two other comorbid conditions</td>
<td>Risk increased by 4.5 compared to those with no asthma</td>
<td>Hakola et al. (2011)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Cancer (malignant)</td>
<td>Withdrawal from the labor market</td>
<td>Decreased by 29 percent</td>
</tr>
<tr>
<td>Depression</td>
<td>Depression</td>
<td>Reduced by 24-43 percent</td>
<td>Alexandre and French (2001)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Diabetes</td>
<td>Odds increased by 50 percent</td>
<td>Egede (2004)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Diabetes with “complicating” condition</td>
<td>Probability of labor force participation</td>
<td>Reduced by 12 percent</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Diabetes with “complicating” condition</td>
<td>Missed work days (2-week period)</td>
<td>Increased by 3.26</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Diabetes + depression</td>
<td>Extended work loss (&gt;= 7 days)</td>
<td>Odds increased by 3.15</td>
</tr>
<tr>
<td>Hypertension + depression</td>
<td>Hypertension + depression</td>
<td>Reduced by 11 percentage points</td>
<td>Scuteri et al. (2011)</td>
</tr>
<tr>
<td>Mental and behavioral disorders</td>
<td>Mental and behavioral disorders</td>
<td>Reduced by 20 percent (compared to those with physical disorders only)</td>
<td>McAlpine and Warner (2002)</td>
</tr>
<tr>
<td>Multiple Psychiatric disorders</td>
<td>Multiple Psychiatric disorders</td>
<td>Work loss days (per month, per 100 workers)</td>
<td>49 days versus 11 among those with a single disorder</td>
</tr>
<tr>
<td>Multiple Psychiatric disorders</td>
<td>Multiple Psychiatric disorders</td>
<td>Work cutback days (per month, per 100 workers)</td>
<td>346 days versus 66 among those with a single disorder</td>
</tr>
<tr>
<td>Psychiatric Diagnoses</td>
<td>Psychiatric Diagnoses</td>
<td>Probability of employment</td>
<td>Reduced by 11 percentage points</td>
</tr>
</tbody>
</table>

### Employment Outcome

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Effect</th>
<th>Citation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthritis</td>
<td>Earnings</td>
<td>Reduced by 21-38 percent</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Earnings</td>
<td>Reduced by 23 percent</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Earnings</td>
<td>Reduced by 30-35 percent</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Earnings</td>
<td>Reduced by 32 percent</td>
</tr>
<tr>
<td>Psychiatric Diagnoses</td>
<td>Earnings</td>
<td>Reduced by 13-18 percent</td>
</tr>
<tr>
<td>Psychiatric Diagnoses</td>
<td>Log earnings</td>
<td>Reduced by 18 percent</td>
</tr>
</tbody>
</table>
Table 18. Summary of Literature on the Prevalence of Selected Conditions among U.S. Adults

<table>
<thead>
<tr>
<th>Condition</th>
<th>Documented Range</th>
<th>Citation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>8.4 - 11.7</td>
<td>Anderson 2010; Gallup-Healthways 2012; Ornstein et al. 2013; Moorman et al. 2012; Xu et al. 2013</td>
</tr>
<tr>
<td>Cancer</td>
<td>40.4 - 41.5</td>
<td>American Cancer Society 2014; National Cancer Institute 2014; Siegel et al. 2013</td>
</tr>
<tr>
<td>Depression</td>
<td>8.3 - 29.9</td>
<td>Bromet et al. 2011; Gallup-Healthways 2012; Kessler et al. 2012; Ornstein et al. 2013; Shim et al. 2011</td>
</tr>
</tbody>
</table>

Note: Estimates from Anderson (2010) include children, and Roger et al. (2011) and Beckles and Chou (2013) exclude 18 and 19 year olds.

a Includes both lifetime and 12-month diagnoses, though Kessler et al.’s (2012) and Michael et al.’s (2007) lifetime prevalence estimates are at opposite ends of this range (10.1 percent versus 28.7 percent), likely due to variation in measures used.

b American Cancer Society (2014) and Siegel et al. (2013) only provide probabilities calculated separately by sex, which I average here.

c Definitions vary between studies, from lifetime diagnosis of major depressive episode (e.g., Kessler et al. 2012) to presence of symptoms (including mild) in past two weeks (e.g., Shim et al. 2011).

d Estimate from Agency for Healthcare Research and Quality (2000) is among hospital patients admitted for a different condition (10.9 percent).
Table 19. Construction of the Analytic Sample

<table>
<thead>
<tr>
<th>Operationization</th>
<th>Purpose</th>
<th>Unweighted N</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wives</strong> Relation to head = wife</td>
<td>Select legally married women</td>
<td>4,110</td>
</tr>
<tr>
<td>&quot;Wives&quot; Relation to head = &quot;wife&quot;</td>
<td>Select long-term cohabiting women</td>
<td>564</td>
</tr>
<tr>
<td>Female Heads of Household Relation to head = head, sex = female</td>
<td>Select unmarried female heads</td>
<td>2,574</td>
</tr>
<tr>
<td><strong>SUBTOTAL (All women in own family unit)</strong></td>
<td></td>
<td>7,248</td>
</tr>
<tr>
<td>Actually in FU in 2007 FU status = in family at time of interview</td>
<td>Ensure Head/Wife data refer to woman still in household, rather than someone who moved out or died.</td>
<td>7,076</td>
</tr>
<tr>
<td>Actually in FU in 2005 FU status = in family at time of interview</td>
<td>Ensure Head/Wife data refer to woman from 2007, rather than an head/wife who moved or died by 2007</td>
<td>6,690</td>
</tr>
<tr>
<td>Actually in FU in 2009 FU status = in family at time of interview</td>
<td>Ensure Head/Wife data refer to woman from 2007, rather than a replacement head/wife</td>
<td>6,291</td>
</tr>
<tr>
<td>Not just in FU, but in as a head or wife Relation to head in 2005/2009 = wife, &quot;wife,&quot; or head</td>
<td>Data only collected for head/wife</td>
<td>5,974</td>
</tr>
<tr>
<td><strong>SUBTOTAL (Screened on data quality and presence in key samples)</strong></td>
<td></td>
<td>5,954</td>
</tr>
<tr>
<td>Aged 25-64 Age after LDS imputation and related efforts (see text)</td>
<td>Limit to working-aged women</td>
<td>4,988</td>
</tr>
<tr>
<td>Lives with Others Number in FU&gt;1</td>
<td>Limit to women who live with one or more family</td>
<td>4,479</td>
</tr>
<tr>
<td>Lives with Others Who Have Some Health Data Number of FUMs (besides woman) in PSID main file, TA, or CDS &gt;=1</td>
<td>Limit to women who live with one or more family member(s) who were also in a survey</td>
<td>3,992</td>
</tr>
<tr>
<td>Does not live in a foreign country Indicated by value on Beale rural-urban continuum measure</td>
<td>Conceptual framework may be inapplicable outside of U.S.</td>
<td>3,976</td>
</tr>
<tr>
<td><strong>SUBTOTAL (Screened on key demographics)</strong></td>
<td></td>
<td>3,976</td>
</tr>
<tr>
<td>Cases Missing on Selected Variables Missing on variables with low overall missing rates (less than 0.35 percent of cases)</td>
<td>Prevent listwise deletion in regression models (multiple imputation unlikely to change results)</td>
<td>3,945</td>
</tr>
<tr>
<td><strong>FINAL SAMPLE (Women)</strong></td>
<td></td>
<td>3,945</td>
</tr>
</tbody>
</table>

a Necessary since the PSID still assigns relation to head status for sample members who have been lost between waves (e.g., death, moved out), presence in the FU is indicated by a second variable.

b The "year new head/"wife" measures are used to indicate the last year in which certain demographic details were updated. For example, the measure indicating "years of work experience since age 18" is not updated at each survey wave. Instead, it is collected when a new head or wife is established, and PSID users are advised to use this measure in conjunction with the "year new head/"wife" measure to determine by how many years the work experience measure should be scaled upward. It was in updating the work experience measure that I identified an unusual code on the "year new head/"wife" measure, indicating cases for which "Collection of background data omitted in error for Head [WIFE]. Data for this case may refer to some former Head [WIFE]."
Figure 14. Kernel Density Plot of Observed, Imputed, and Completed Data on Average Weekly Hours Worked in 2007 for $m=1$

Note: Data are imputed for 38 women. See Chapter II for a description of multiple imputation methodology.

Figure 15. Kernel Density Plot of Observed, Imputed, and Completed Data on Labor Income in 2007 for $m=1$

Note: Data are imputed for 220 women. See Chapter II for a description of multiple imputation methodology.
Supplemental Analyses: Addressing Endogeneity and Causal Ordering

Throughout this dissertation, I am unable to establish a sense of causal ordering, due to a lack of sufficient data on health across the family unit, as described in Chapter II. This is perhaps the most significant limitation to this work, as the findings here are descriptive of associations, rather than causal relationships, in the data. Further, it should be noted that measures of family health might be endogenous to employment, a violation of the assumptions of regression analyses. Given these limitation, this section attempts to identify factors that might preclude the directionality of the relationship implied by the models in Chapters IV and V. Below I briefly review why health and labor supply may be at particular risk for endogeneity, and highlight some exploratory strategies for assessing endogeneity and temporal features in a subsample of the data. It should be noted that my intent in this section is certainly not to establish causality, which is beyond the capacity of the data at hand, but rather to determine whether the limited analyses around endogeneity and change over a short period of time provide evidence that the directionality implied in the preceding chapters is implausible for any reason.

First, there has been substantial research done on the causal direction of the relationship between informal or unpaid caregiving and employment outcomes. For instance, Carmichael, Charles and Hulme (2009) demonstrate that unemployed people are indeed more likely to become caretakers than their employed counterparts. In discussing the effects of informal caregiving specifically on labor supply, Leigh (2010) highlights the methodological consequences of obfuscating this relationship, writing, “people may choose to take on caring responsibilities precisely because they are not in paid work. In this case, estimates derived from cross-sectional studies may exaggerate the impact of caring on labor force participation” (141).
Finally, as summarized by Lilly, Laporte, and Coyte (2007), it becomes important to identify whether women “self-selected into the unpaid caregiving role because they were already outside, or had looser attachment to, the labor force when faced with initial caregiving decisions” (658). There is also a substantial literature on the issue of health as potentially endogenous to labor market behavior, suggesting indirect (e.g., capacity to invest in one’s own health) and direct links (e.g., stress or poor working conditions) between the two [see Cai (2010) for a succinct review of this specific topic, and Ross and Mirowsky (1995) for the bidirectional relationship between health and employment more broadly].

If family health might predict women’s employment, and women’s employment may predict family health, family health can be termed *endogenous* to employment. To determine whether family health is indeed endogenous, let the following (simplified) regression equation represent the models in Chapter IV:

\[ y_i = \beta_0 + \beta_1 x_i + \beta_2 a_i + e_i \]  

[Equation A]

where \( y \) is women’s employment, \( x \) is a vector of exogenous covariates, \( a \) is family health, and \( e \) is the disturbance term. If we consider family health to be potentially endogenous, we assume that the following equation is also true:

\[ a_i = \beta_0 + \beta_1 x_i + \beta_2 y_i + u_i \]  

[Equation B]

If this is indeed the case, when used in Equation A, \( a \) will correlate with the model’s error term, producing inconsistent estimates. To correct for the inconsistencies produced by this endogeneity, I rely on an instrumental variable approach (Antonakis et al. 2014; Wooldridge 2012). This method reduces this bias by identifying exogenous measures from the first equation (\( x \)) plus additional “instrumental” measures (\( z \)) with no relationship with the outcome measure (\( y \)), aside from their relationship with the potentially endogenous measure. These exogenous
measures are used to predict values of the endogenous variable (a), which are then used to fit the full equation [Equation 3] (Adkins 2009). As a result of this correction, the resulting estimates are consistent, having removed overlapping variance between a and e (Antonakis et al. 2014). One popular strategy is a two stage least squares approach, in which parameters for each equation are estimated separately; here, adhering to requirements of the complex sample I use the maximum likelihood estimator, which estimates these equations jointly.

Here, in suspecting that family health may be endogenous to employment, I construct a series of measures that might predict the number of chronic conditions in a family unit, but not employment status. To determine whether I have identified appropriate instruments, I regress family condition count on all (exogenous) covariates plus five potential instrumental variables: number of people in the household, whether the woman in the household has a BMI that meets criteria for obesity, whether she smokes cigarettes, and two proxy measures of SES stratification: binary measures of food stamps receipt and home ownership. A negative binomial regression indicates that each of these measures is indeed predictive of family health conditions ($p<0.0001$ for each). An examination of $F$ statistics shows that the value on each measure exceeds the “rule of thumb” proposed by Stock, Wright, and Yogo (2002): that $F$ statistics must generally exceed 10 for inferences to be reliable. Here, those statistics range from 14.79 for number of people in the family unit to 228.42 for women’s obesity indicator.

The next step to this process is ensuring that the instrumental variables not only predict family health conditions, but that these measures are uncorrelated with the outcome measure (employment), net of other covariates. I first predict family health conditions using all available exogenous measures (from main equation plus instrumental variables) and find that the test statistics on the proposed instrumental variables reveals no remaining relationship with labor
supply. Calculating residuals from this first stage model, I then re-fit the main model of interest using the residual from the first stage model as a generated regressor (i.e., as an independent variable). This measure can be considered the more-efficient, Durbin “flavor” of the Hausman statistic (Baum, Schaffer, and Stillman 2003), and the t-test here can be interpreted as the regression-based Durbin-Wu-Hausman test (see Baum et al. 2003 for a discussion of various Hausman statistics, and Cong 1999 for a discussion of the regression-based method). While this suggests that OLS estimates may not be inconsistent, there is evidence that this type of Hausman test is unreliable under some circumstances (e.g., Doko and Dufour 2012). Erring on the side of caution, I consider the Hausman test and continue exploration of the possible endogeneity.

I next estimate an instrumental variable probit model (ivprobit) using the exogenous variables described above as instruments (see Table 20). The instrumented measure of family health as shown in Model 2 produces less compelling effects than the traditional regression model in Model 1, though the coefficient is similar between models. From this model, a Wald test for exogeneity suggests that family conditions are indeed endogenous to employment (Bonanno and Li 2012), and the standard error is substantially larger in the instrumental variable equation.
Table 20. Comparison of Results from Traditional and Instrumental Variable Approaches: Predicting Women’s Employment in 2008 Using Number of Conditions in Family Unit

<table>
<thead>
<tr>
<th></th>
<th>Model 1: Standard Logistic Regression</th>
<th>Model 2. IV Probit Estimation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>SE</td>
</tr>
<tr>
<td>Number of Conditions in FU</td>
<td>-0.074</td>
<td>0.018</td>
</tr>
<tr>
<td>Age</td>
<td>-0.093</td>
<td>0.009</td>
</tr>
<tr>
<td>Age²</td>
<td>-0.003</td>
<td>0.001</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married/Cohabiting</td>
<td>REF</td>
<td></td>
</tr>
<tr>
<td>Previously married</td>
<td>0.080</td>
<td>0.297</td>
</tr>
<tr>
<td>Never married</td>
<td>0.344</td>
<td>0.189</td>
</tr>
<tr>
<td>Age of Youngest Child</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Youngest Child Under 5</td>
<td>-0.805</td>
<td>0.213</td>
</tr>
<tr>
<td>Youngest Child 5 or Older</td>
<td>0.050</td>
<td>0.117</td>
</tr>
<tr>
<td>Educational Attainment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than High School</td>
<td>-0.622</td>
<td>0.185</td>
</tr>
<tr>
<td>High School</td>
<td>-0.194</td>
<td>0.146</td>
</tr>
<tr>
<td>Some College</td>
<td>-0.426</td>
<td>0.163</td>
</tr>
<tr>
<td>College Grad</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race/Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black, non-Hispanic</td>
<td>0.807</td>
<td>0.282</td>
</tr>
<tr>
<td>Hispanic/Other/Multiracial</td>
<td>-0.183</td>
<td>0.137</td>
</tr>
<tr>
<td>Region of Residence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northeast</td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Central</td>
<td></td>
<td></td>
</tr>
<tr>
<td>South</td>
<td>-0.219</td>
<td>0.150</td>
</tr>
<tr>
<td>[...] × Black, non-Hispanic</td>
<td>-1.099</td>
<td>0.293</td>
</tr>
<tr>
<td>West</td>
<td>-0.225</td>
<td>0.168</td>
</tr>
<tr>
<td>Lives in Metropolitan Area</td>
<td>0.072</td>
<td>0.118</td>
</tr>
<tr>
<td>Years of Work Experience</td>
<td>0.101</td>
<td>0.008</td>
</tr>
<tr>
<td>Liquid Assets</td>
<td>-0.002</td>
<td>0.000</td>
</tr>
<tr>
<td>SSI Receipt</td>
<td>-0.992</td>
<td>0.285</td>
</tr>
<tr>
<td>Transportation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Own Vehicle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Public Transportation Only</td>
<td>0.110</td>
<td>0.457</td>
</tr>
<tr>
<td>No Vehicle</td>
<td>-1.230</td>
<td>0.363</td>
</tr>
<tr>
<td>s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insigna</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>0.922</td>
<td>0.269</td>
</tr>
<tr>
<td>n</td>
<td>3.771</td>
<td>3.771</td>
</tr>
<tr>
<td>Overall F test</td>
<td>F(22,42)=20.63  ***</td>
<td>F(22,42)=26.29  ***</td>
</tr>
<tr>
<td>Wald chi-square test of exogeneity</td>
<td>23.177</td>
<td>***</td>
</tr>
<tr>
<td>Weak instrument tests</td>
<td>Statistic</td>
<td>p-value</td>
</tr>
<tr>
<td>Anderson-Rubin</td>
<td>5.25</td>
<td>0.385</td>
</tr>
<tr>
<td>Conditional-likelihood ratio</td>
<td>5.02</td>
<td>0.078</td>
</tr>
<tr>
<td>Lagrange multiplier (LM)</td>
<td>4.98</td>
<td>0.075</td>
</tr>
<tr>
<td>Overidentification (J test)</td>
<td>1.24</td>
<td>0.718</td>
</tr>
</tbody>
</table>

Note: Because the instrumental variables were not screened in building the earlier sample, these measures have some missing values. Thus the sample for these tables does not correspond with the sample from the earlier models.
In instrumental regressions, weak instruments can lead to poor performance of Wald tests and biased estimates (Finlay and Magnusson 2009). To test for this possibility, I use the post-estimation command `rivtest`, examining the resulting Anderson-Rubin statistic (AR), Kleibergen-Moreira Lagrange multiplier (LM), overidentification ($J$) test, the combined LM-$J$ test, and the conditional-likelihood ratio (CLR) test (ibid), and include these results in Table 20. In short, the null hypothesis on the $J$ test is that the instrumental variables are uncorrelated with the error term and thus correctly excluded from the model; the null for the LM test is that the value of the structural parameter is zero (assuming non-significance of the $J$ test). The AR test is the equivalent of substituting the instruments for the endogenous variable, and testing their joint significance. The CLR and LM-$J$ tests are also joint tests of LM and $J$, though more efficient than the ER test (Finlay and Magnusson 2009). `Rivtest` then draws on these statistics to compute weak-instrument-robust confidence intervals around the endogenous variable’s coefficient, which “guarantees that our confidence intervals have the correct coverage probability despite the instruments’ strength or weakness” (Finlay and Magnusson 2009:399). Here, the overidentification test suggests that the instruments are indeed correctly excluded ($p=0.718$). The AR test suggests no effect of the instrumented version ($p=0.385$), though results from the more efficient CLR and LM-$J$ tests are substantially closer to achieving statistical significance at the 0.05 level.

Figure 16 plots the predicted probabilities from the two models, using the corrected confidence intervals for the instrumental variable regressions, described by Finlay and Magnusson (2009) above. In this figure, the widening of standard errors is evident in the confidence intervals, though the direction and magnitude of family health’s effects is similar between models.
The findings from these analyses suggest that family health may indeed be endogenous to women’s employment, but that the effect of an instrumented version is generally similar. Of course successful estimation of an instrumental variable regression model does not provide evidence of a causal relationship between family health and employment (or indeed of any relationship between family health and employment). Rather, these findings indicate evidence of some statistical relationship between these measures and do not preclude the possibility that the relationship might be causal.

To consider the potential for causality from another vantage point, I also include some descriptive analyses of the very small share of women in this sample who experienced a change in employment characteristics between 2007—the reference period for the health data here—and 2008, the point of measurement for employment outcomes used throughout this dissertation. I first identify women who were employed in 2007 but not in 2008, and examine the bivariate distribution of condition counts and specific illnesses in this subpopulation (n=67).
For this group, the mean number of (family and own) conditions appears higher among women who lost jobs, but the differences are not large enough to achieve statistical significance ($p=0.324$ and $p=0.259$, respectively). In terms of specific illnesses, none of the diagnoses at the family level are associated with one-year job loss; however, several of women’s own diagnoses are present at significantly higher levels among women who lost jobs in that period. Figure 17 represents the gaps in reported diagnoses by condition and change in employment status, below.

Figure 17. Percent of Women with Specified Diagnosis by Change in Employment Status 2007 to 2008

<table>
<thead>
<tr>
<th>Condition</th>
<th>Still employed</th>
<th>No longer employed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Percentages calculated only among women employed in 2007 and adjusted for complex sample design. Differences between employed and no longer employed women are statistically significant ($p<0.05$) for hypertension and asthma, and significant at the $p<0.10$ level for diabetes (0.086).

I also examine the small group of women who experienced a shift from full time employment (defined here as average hours per week reported at 35 hours or more) to part time employment (i.e., between one and 34 average weekly hours worked). I run a series of small, subsample logistic regression models regressing this change in hours on a series of demographic measures and enter specific diagnoses one at a time. I find that the women’s own
conditions are not statistically significant predictors of changes in employment status, but that having a diabetes diagnoses at the family level doubles the odds of women’s job loss during this period (OR=2.053; p=0.015), net of a host of other characteristics.\textsuperscript{50} Finally, I repeat these analyses among those who were employed in 2007 and 2008, predicting the probability of experiencing a 10 percent or higher reduction in income over the year; in this model, I find that women’s condition count is a significant predictor of this income loss (OR=1.216; p=0.001).

Unlike the models in Chapters IV and V that model a single year’s employment outcome, modeling change ensures that women’s employment characteristic is known at both time points (i.e., that the lower income did not precede the measurement of condition counts). While this is not enough to establish a causal relationship, that these findings concur very well with results from Chapters IV and V raise no red flag for constructing the cross-sectional models as they were done in those chapters.

In a final attempt to temporally situate the relationship between health and employment, I draw upon a small set of measures indicating at what age women were diagnosed each of \textsuperscript{51}11 specific conditions to form a measure of “timing of earliest diagnosis” among women diagnosed with one or more conditions.\textsuperscript{51} In this way, it is possible to discern whether early-life diagnoses are associated with reduced odds of employment by 2008. From this measure, I code two additional categorical measures indicating “first diagnosis was before age 18” and “first diagnosis was before age 25.” Using these new measures in turn, I use Model 2 from Table 13 as my base model, and predict employment in 2008 among women with one or more diagnoses. In each

\textsuperscript{50} Covariates in this simple model include women’s own condition counts, counts for spouses and children, age, age-squared, marital status, race/ethnicity, presence of child(ren) over/under age five, metropolitan status, and years of work experience. Results available upon request.

\textsuperscript{51} Women who reported being diagnosed with stroke, heart attack, heart disease, hypertension, asthma, lung disease, diabetes, arthritis, memory loss, cancer, or other chronic conditions were asked at what age they were diagnosed with this condition. I create the “timing at earliest diagnosis” measure by simply identifying the lowest reported age across all diagnoses.
case, timing of women’s diagnoses does not achieve statistical significance. Taken together, the results of these supplementary analyses were not terribly informative in their own right, but raised few red flags about the feasibility of modeling the relationship between health and employment cross-sectionally in these data.