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Essays in the Economics of Healthcare and Health Insurance

by

Bradley Thomas Howells

A dissertation submitted in partial satisfaction of the
requirements for the degree of
Doctor of Philosophy

in

Economics

in the

GRADUATE DIVISION
of the
UNIVERSITY OF CALIFORNIA, BERKELEY

Committee in charge:
Professor David Card, Chair
Professor Patrick Kline
Professor Steven Raphael

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Essays in the Economics of Healthcare and Health Insurance

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Bradley Thomas Howells

Abstract

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Bradley Thomas Howells
Doctor of Philosophy in Economics

University of California, Berkeley

Professor David Card, Chair

This dissertation contributes in two distinct ways to our understanding of the economics of healthcare and health insurance. Chapter 2 studies the decision process by which physicians allocate medical treatments to heart attack patients. The approach provides insight into the sources of well documented, but unexplained, disparities across demographic dimensions in health care utilization rates and health outcomes. In the model medical providers know how treatment alternatives affect patient-specific probabilities of three final health outcomes - death, readmission, and survival without readmission - and assign implicit values to each outcome that vary by patient age. The model does well in explaining the joint variation in treatments and outcomes, especially when including unobserved patient heterogeneity. Using decomposition methods, I show that a substantial fraction of gender differences in the use of intensive treatment is explained by a combination of the differences in the relative efficacy of treatment options for female patients, and the smaller implicit weight given to final outcomes of older patients. Chapter 3 explores how reforms to cash-assistance welfare programs in the United States in the mid 1990s acted as a structural shift in the health insurance and employment environment of lower income single mothers and find there may have been unintended consequences for this population's access to health insurance. With a more structured approach than is common in the literature, I estimate short and long run employment and insurance dynamics before and after the reforms. I show that reform reduced use of cash-assistance and increased the probability of employment, but created a less stable employment and health insurance environment. After the reform low income single mothers were less likely to retain the same employment and insurance status over a four month period. Although policy did not target Medicaid eligibility, individuals were less likely to retain Medicaid enrollment over the short and longer run after reform.

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Chapter 1

Introduction and Overview

The efficiency, equity, and accessibility of healthcare in the United States has been increasingly pressing on the minds of patients, physicians, insurers, and policy-makers. Over recent decades, medical innovation has rapidly expanded the set of medications, technologies, and procedures available to healthcare providers and patients. With advances in health care have come increased costs and complex reimbursement and financial contracting. Government policies have been implemented with hopes of improving the efficacy by which we delivery health care to those in need. This has brought an increased need to evaluate the efficacy of new medical treatments, payment structures, and health policies. The outputs of the healthcare system are ultimately driven by decisions made in complex and nuanced environments from which the underlying objectives, costs, and preferences are not always easily identified. Uncertainty and limited information play important roles in shaping the choices people make and in few settings is this more true than in healthcare. The provision of healthcare services emerges from an entangled web of interactions between patients, physicians, administrators, insurance companies, and policy makers, each with their own preferences, information, and constraints. In the end, the provision of healthcare is the result of complex interactions in economic markets. Understanding these interactions is necessary for physicians and patients to make optimal decision over medical care and for policy-makers to create effective policy with proper incentives and predictable results. However, healthcare markets do not always fit cleanly into traditional economic models. In the chapters that follow I explore two distinct aspects of healthcare markets and use novel economic models and techniques to study how decision-making and incentives can affect the equity, efficiency, and accessibility of healthcare in the United States.

Chapter 2 focuses on the decision-making in health care that drives the allocation and choice of medical treatments by physicians for patients. Policy-makers and health care professionals are interested in measuring the efficacy of the vast

number of treatment options now available to health care providers. Extensive research hours and resources have been devoted to the essential and challenging task of measuring the efficacy of medical treatments and surgical procedures. In practice, however, treatments are not delivered to patients in the controlled, experimental environment of a randomized clinical trial, but rather through complex interactions between health care providers and patients. Indeed, our understanding of the process linking treatment efficacy, treatment delivery, and health outcomes has arguably lagged behind innovation of new methods. In response to this, the National Institutes of Health recently committed significant resources to an initiative funding research of the "comparative-effectiveness" of health care treatments and delivery. Improving the quality of health outcomes relies not only on knowledge of the efficacy of a given medication or technology, but also on how treatments are utilized in practice. Rarely is there a treatment strategy that is known to be optimal for all patients. Rather, a decision is made over available treatment options incorporating both the information available to and the preferences of the parties involved. This decision process plays an important role in the link between health care provision and health outcomes.

I build a unique economic modeling framework for answering questions related to the equity and efficacy of health care delivery. Most notably, why do patients with similar diagnoses receive different medical treatment? In practice, how effective are medical technologies in improving health outcomes and how important are treatment allocation decisions in determining the efficacy? What explains the well documented racial and gender disparities in both utilization of new technologies and in corresponding health outcomes? Are clinical and biological explanations sufficient, or do discrimination, prejudice, and equitable access play important roles?

In Chapter 2 these questions are addressed from the perspective of a structured, outcome-driven choice model. Health care providers derive value over a small set of discrete patient health outcomes. I apply the model in the specific context of treatments for heart attack patients. Providers form an expectation over outcomes under each treatment option and allocate treatments to maximize the value of the expected outcomes net of treatment costs. Treatments influence health outcomes, and so decisions over treatments reflect the values providers place on final patient outcomes. In this way, the model uses actual treatment choices and observed patient outcomes to capture the implied weight placed on outcomes. Since providers may allocate treatments to patients using information on patient-specific treatment efficacy that I do not observe, observed treatment choices suffer from a form of selection bias. To account for this, I allow for unobserved patient heterogeneity affecting the relative efficacy of treatment options in preventing adverse outcomes. Estimation results find that the simple model does relatively well in explaining the joint variation in treatments and outcomes,

especially when patient heterogeneity is included. I use the model to address the well known disparity in use of intensive treatment for female patients and find, using decomposition methods, that a substantial fraction of gender differences are explained by a combination of the differences in the relative efficacy of treatment options for female patients, and the smaller implicit weight given to final outcomes of older patients.

Chapter 3 explores how government policies can have profound effects, often inadvertently, on access to health insurance. Just like most economic decisions, choices over health insurance coverage are driven by constraints, resources, and incentives. This includes the decision to participate in government-sponsored assistance programs, such as Medicaid. As medical costs have increased over the years, maintaining access to health care and the ability to pay for services has become paramount for lower income families. Enrollment rates in Medicaid have remained puzzlingly low over the years[1]. The choice to participate in the program balances the perceived benefits of Medicaid coverage and the health services and preventative care it provides, with the barriers to and costs, both financial and otherwise, of enrolling. The enrollment costs of Medicaid may play a larger role in Medicaid take-up rates than previous thought and the incentives driving the decision to enroll may be related in subtle ways to those driving participation in the labor force and other government programs, such as cash-assistance welfare.

In 1996 the Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA) brought significant changes to the cash-assistance welfare program in the United States. With the introduction of Temporary Assistance for Needy Families (TANF) to replace Aid to Families with Dependent Children (AFDC) new restrictions were placed on the receipt of cash-assistance and new incentives were introduced that encouraged individuals on welfare to find employment. An indirect result of this reform was a change in the link between cash-assistance and government provided health insurance, specifically Medicaid. The reforms were not intended to directly affect the accessibility of health insurance for this population; individuals who would have qualified for AFDC were to maintain their Medicaid eligibility. However, it is possible that the PRWORA acted as a structural shift in the health insurance and employment environment of lower income single mothers resulting in unintended consequences for this population's access to health insurance. Evidence suggests that the reforms increased confusion over Medicaid eligibility and the enrollment process. Chapter 3 focuses on reforms to cash-assistance welfare programs in the United States in the mid 1990s, their effect on barriers to, and costs of, enrollment in Medicaid, and the effect on the insurance status of the Medicaid-eligible population. With a more structured approach than is common in the literature, I estimate short and long run employ-

[1]See Card and Shore-Sheppard [2004]

ment and insurance dynamics before and after the reforms. I show that reform reduced use of cash-assistance and increased the probability of employment, but created a less stable employment and health insurance environment. After the reform low income single mothers were less likely to retain the same employment and insurance status over a four month period. Although policy did not target Medicaid eligibility, individuals were less likely to retain Medicaid enrollment over the short and longer run after reform. This may have been in part due to inadvertent increases in the barriers to and costs of Medicaid enrollment.

Chapter 2

Efficacy, Equity, and the Value of Outcomes: A Choice Model Approach to the Treatment of Heart Attack Patients

2.1 Introduction

Decisions in the real world are made in complex environments from which the underlying objectives, costs, and preferences are not always easily identified. Uncertainty and limited information play important roles in shaping the choices people make. In few settings is this more true than in health care. The provision of health care services emerges from an entangled web of interactions between patients, physicians, administrators, insurance companies, and policy makers, each with their own preferences, information, and constraints. It is impossible to analyze and understand the entire process simultaneously. As researchers, we narrow our focus, making use of carefully executed simplification to further our understanding of specific, targeted dimensions of the real world. The extent to which modern health care is provided efficiently and fairly has become one of the most important and studied issues in health economics. Economic models are particularly well suited for this purpose.

Over recent decades, medical innovation has rapidly expanded the set of medications, technologies, and procedures available to health care providers. Along with this expansion has come an increased need to evaluate the efficacy of new treatment options. As a result a burgeoning literature in both economics and medicine has emerged, studying issues related to medical innovation, technological change, and the efficacy and equity of medical care. How effective are new

medical technologies in improving health outcomes?[1] At what cost do we achieve these improvements?[2] How do the patterns of diffusion of new technology affect patient access to effective medical care?[3] What explains the well documented racial and gender disparities in both utilization of new technologies and in corresponding health outcomes?[4] Are clinical and biological explanations sufficient, or do discrimination, prejudice, and equitable access play important roles?[5]

The health outcomes generated by medical treatment, and hence the inherent efficiency and equity implications, are driven in practice by the decision making process that governs assignment of treatments to patients. An isolated estimate, even if precisely and accurately measured, of the efficacy of any given technology is much less informative without an understanding of how that technology will be utilized in practice. Decision making in health care settings is complicated and we know relatively little about how decisions are made. This includes who is actually making the decision, be it the physician, patient, or another third party, what are the relevant objectives, and whose preferences drive choices. Although, significant theoretical work has explored such complex principal-agent environments, empirical application of these models is often very difficult. Most existing empirical research utilizes sophisticated study designs and statistical approaches that allow for nimble side-stepping of the decision process that governs the use of medical technologies and treatments in practice[6] In this chapter, I focus directly on the process by which treatments are assigned to patients. I set the groundwork for a framework from which we learn about the connections between efficacy, equity, and outcomes in the context of treatment for heart attack patients.

I specify and estimate a simple model of treatment decisions. The approach provides insight into efficacy and equity from the perspective of an outcome driven choice model. Health care providers derive value over a small set of discrete patient health outcomes. Providers form an expectation over outcomes under each treatment option and allocate treatments to maximize the value of the expected outcomes net of treatment costs. Treatments influence health outcomes, and so decisions over treatments reflect the values providers place on final patient outcomes. In this way, the model uses actual treatment choices and observed patient outcomes to capture the implied weight placed on outcomes. Since providers may allocate treatments to patients using information on patient-specific treat-

[1]For example, McClellan et al. [1994], Hlatky et al. [1997] and Jokinen et al. [2010]

[2]See Skinner et al. [2006], Cutler and McClellan [2001], and Cutler et al. [1998]

[3]See Skinner and Staiger [2009], Chandra and Staiger [2007], and Cutler and Huckman [2003]

[4]See Anderson and Pepine [2007], Barnato et al. [2009], Guru et al. [2006], Jha et al. [2005], and Smedley et al. [2002]

[5]For example, Balsa and McGuire [2003], Chandra and Staiger [2010], and Lin et al. [2007]

[6]For example, McClellan et al. [1994] use distance to a hospital equipped with catheterization technology as an instrument for treatment intensity.

ment efficacy that I do not observe, observed treatment choices suffer from a form of selection bias. To account for this, I allow for unobserved patient heterogeneity affecting the relative efficacy of treatment options in preventing adverse outcomes.

The model does relatively well in explaining the joint variation in heart attack treatment allocation and patient outcomes. The inclusion of unobserved patient heterogeneity significantly improves the performance of the model. Estimation results suggest providers internalize expected outcomes when allocating treatments. Treatment decisions reduce death, but also place a large emphasis on preventing readmissions. Stronger consideration is given to the prevention of adverse outcomes for younger patients. The model provides insight into the well known puzzle of gender disparities in treatment and health outcomes for heart attack patients. Importantly, the model provides the framework to separately consider efficacy and equity in explaining gender differences in treatment and health outcomes of heart attack patients. Using an Oaxaca-style decomposition, I estimate the fraction of the gender gap that can be explained by differences in relative efficacy of treatment options for men versus women. This explains approximately 90% of differences in percutaneous coronary intervention, 64% in medical therapy, and 31% in readmission rates. Nevertheless, substantial unexplained gender differences remain in the predicted use of CABG and in patient death rates. An extension of the model, currently in progress, provides estimates of the fraction of the remaining, unexplained gender gaps that can be attributed to differences in the implicit weight providers place on the health outcomes of men and women.This can speak to underlying concerns over equity of care.

It may be helpful to put the framework of the model in the context of the classic educational attainment model of Willis and Rosen [1979]. In this well-known model students self-select into educational levels that provide the highest expected lifetime earnings, net of educational costs. Since many factors that influence earnings potential and educational costs are not observed by the researcher, estimating returns to education using observed education choices are plagued by bias and selection problems. The authors provide a simplified framework of the decision environment that clarifies the objectives, tradeoffs, and actions of students. Of course, their framework abstracts from reality by stripping away realistic complexities, but does so along dimensions that are not critical to the lessons and intuition of the model. Furthermore, their framework allows for a clear and intuitive exposition of the role of unobserved factors, such as “talent”, in the decision process. The authors propose a two-step procedure for adjusting for the self-selection into education levels. Many studies have since built upon this basic framework, including increasingly complex and realistic aspects of returns to, and choice of, educational attainment.

While the Willis and Rosen model can provide some useful intuition[7], the model I build differs in important substantive and structural ways. Rather than deciding on educational attainment, providers decide on a course of medical treatment. These decisions are driven by expectations over health outcomes rather than future earnings. Thus, analogous to earnings potential is treatment efficacy. Just as all information relevant for earnings potential, such as "talent", is not known to the researcher, neither is all patient-level information regarding relative efficacy of a treatment option. The structure of my model diverges in at least four significant ways. First, relevant outcomes in my model are discrete, rather than continuous. Second, in the empirical application, AMI treatment, agents choose from three options, rather than two[8]. Third, agents don't have direct control over outcomes, rather outcomes are influenced probabilistically through their choices. Finally, in place of a two-step approach for selection correction, I build a maximum likelihood model that allows for selection on unobservables in a single estimation step.

In this chapter I apply my model in the context of the treatment of heart attack patients. Heart disease is both economically and medically relevant; it is the leading cause of death in the United States. In 2006 diseases of the heart were responsible for 26.0% of total deaths in the United States (Heron [2010]). The National Heart Lung and Blood Institute estimated the costs of cardiovascular disease in the United States in 2007 were approximately $432 billion[9]. Treatment of heart attacks is expensive, and so questions over efficacy and efficiency of treatment use are relevant. There are also several advantages to focusing on a narrowly defined medical condition and patient population, some of which are unique to the treatment of heart attacks. Decision-making over the treatment of heart attacks occurs in a relatively narrowly defined environment, simplifying modeling and interpretation. On the other hand, the specific context, of course, means we must be careful when generalizing the results. I believe the structure and framework of the model presented here provides broadly appealing intuition and perspective; it can be extended to other health care environments, but this must be done with appropriate caution.

Several characteristics of heart attack treatment make it well suited for the framework of a simple choice model: a well-defined medical condition, observable time of onset, limited conflation of principal (physician) and agent (patient) preferences influencing the treatment decision, a small number of precisely defined options for treatment, and observable, ideally discrete, relevant health outcomes. First, heart attack (or acute myocardial infarction, AMI) is a well-defined condition with a standardized, broadly understood diagnostic protocol. It is relatively

[7]The reader may also derive applicable intuition from a generalized Roy model.

[8]The model easily generalizes to situations with greater than three treatment options.

[9]http://www.nhlbi.nih.gov/about/factbook-06/chapter4.htm

precisely diagnosed and well identified in available medical records.

Second, AMI is a rapid onset condition that requires time-sensitive medical attention. Patients suffering from AMI are very likely to be admitted to an inpatient facility. In this way, the data are better able to capture the full population of patients suffering AMI. Similarly, AMI patients are less likely (or able) to delay treatment[10] and are most often admitted to the nearest facility. This limits patient discretion regarding treatment timing and location. This urgency in AMI treatment also reduces the role of patient preferences in the decision process and so observed decisions can more plausibly be attributed to the health care provider. Time between the onset of the condition and the relevant treatment decision is limited which makes a one-time, static choice over treatment a more realistic modeling approach. Chronic conditions, such as diabetes or cancer may be less well suited to this modeling restriction. In such cases, a *dynamic* approach to treatment decisions in which physicians and patients are continuously making treatment decisions, including if and when to undergo elective surgical procedures, is likely more appropriate.

Third, treatments for AMI can convincingly be grouped into three distinct categories. This includes two surgical ("invasive") procedures – coronary arterial bypass grafting (CABG) and percutaneous coronary intervention (PCI) – and a third, non-surgical treatment which I will refer to as "optimal medical therapy" (OMT). There may be variation in treatments within these broad groups[11], but choices over these three treatment groups are arguably of first-order relevance. More details on AMI treatments are given in Section 2.2.

Finally, mortality and readmission rates, which can be precisely defined and are often targeted by policy-makers, are arguably the health outcomes most relevant for the treatment of AMI. This again differs from chronic conditions like diabetes for which morbidity is an important consideration for patients and health care providers[12]. Unlike morbidity, mortality and readmission can plausibly be modeled as binary outcomes, which simplifies the model.

It may be worth reemphasizing that the model I propose here is designed for, and most applicable to, a simplified health care setting in which decision over treatments occur in a relatively small and identifiable window of time. The role of patient preferences is minimized and the number of treatments available is small. The form of the model presented here is not well suited to treatment

[10]Card et al. [2009] have used the distribution of admissions by day of the week to identify "non-deferrable" medical conditions that require urgent medical attention and for which delay in treatment is less plausible. AMI is included in this group.

[11]One can imagine nesting, within PCI, a choice over bare-metal and drug-emitting stents

[12]This is not to say that morbidity is not relevant for AMI patients. Many AMI patients also suffer from heart failure, diabetes, and other debilitating medical conditions. One can think of readmission as capturing some aspect of ongoing co-morbid conditions that are unresolved through treatment of the AMI.

choices for chronic conditions which are more plausibly made in continuous time, nor for decisions over "elective" interventions such as hip replacements or CABG and PCI as a preventative measure for patients suffering stable coronary artery disease (CAD).

The model does not seek to identify an "optimal" treatment strategy or provide a prescription for AMI treatment. The model is intended to provide insight into how treatment decisions are currently being made and the extent to which they reflect values over health outcomes. The modeling framework gives a simple structure for linking observed treatment choices, treatment efficacy, preferences over outcomes, and concerns over effective and equitable allocation of medical care.

The remainder of the chapter proceeds as follows. Section 2.2 provides brief background and details on acute myocardial infarction and the available treatment options. Section 2.3 sets up the simple outcome driven choice model. Section 2.4 details the empirical approach. Section 3.5 describes the data and provides descriptive analysis. Section 3.6 gives estimation results. Section 2.7 concludes.

2.2 Background: Acute Myocardial Infarction, Treatment, and Literature

Cardiovascular disease (CVD) has been the leading cause of death for years in the United States. Perhaps the most dangerous manifestation of CVD is acute myocardial infarction, commonly known as heart attack. Treatment of AMI has drawn significant attention from researchers, both medical and economic, over the years. Indeed, the literature contains hundreds of clinical trials and research papers focused on various aspects of AMI and its treatment. Besides its obvious importance, the impetus for much of this literature is the substantial technological advancements in AMI treatment seen over the past three decades. Coinciding with large drops in mortality rates, have been the introduction and subsequent diffusion of innovative treatment technologies, including angioplasty and stenting. With apparent gains in health outcomes for AMI patients have come increased costs and subsequent concerns over whether the observed utilization patterns of these new technologies are part of an equitable and efficient provision of medical care. Despite substantial research in the area, there remains no consensus on these issues. Before proposing a simple model of heart attack treatment, I provide brief background on AMI itself as well as details on available treatment technologies.

Acute myocardial infarction (AMI) is the death or damage to heart muscle as a result of inadequate supplies of oxygen. AMI occurs when an occlusion or obstruction in one of the coronary arteries blocks blood supply to the heart. Block-

ages are typically caused by build-up of plaque on artery walls and/or a blood clot in a coronary artery. AMI is often the manifestation of slowly progressing coronary artery disease (CAD). In many people, AMI is the first symptom of CAD. Of course, treatment of CAD prior to occurrence of AMI may be desirable. As mentioned in Section 2.1, this chapter focuses on AMI patients. Treatment decisions of stable CAD patients is, for the moment, left to the existing literature, extensions of the model presented in this chapter, and future research.

Treatment of AMI aims to restore blood flow to the heart as rapidly and completely as possible to limit damage to heart tissue. As discussed in Cutler et al. [1998], the initial phase of AMI treatment ("acute phase") is critical and includes treatments and therapies administered within approximately 90 days. However, of major significance is treatment occurring within hours of AMI onset. The American Heart Association and American College of Cardiology publish guidelines for the management of patients with AMI (see Antman et al. [2004] and Anderson et al. [2007]). A similar set of guidelines is published by the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery (see Kolh and Wijns [2010]). Diagnosis and efficacy of treatment of AMI in the early stages (prior to hospital arrival) is time sensitive and subject to a relatively standardized protocol. It is recommended that patients are transported to the hospital via ambulance rather than by family or friends, providing access to defibrillation, if necessary, as well as low-cost treatments such as beta-blockers, aspirin, and intravenous nitrates. Evidence suggests that pre-hospital treatment of AMI is an important factor in the ultimate health outcome of the patient. The scope of this chapter, however, does not extend to the pre-hospital period. The model presented here focuses on the treatment decisions made for AMI patients who arrive alive at the hospital.

Upon arrival at the hospital, acute phase treatment continues. Treatment generally follows one of three treatment "branches", all three seeking to restore blood flow to the heart. The least invasive option is "medical management", which I will refer to as "optimal medical therapy" (OMT). This usually entails the use of powerful, intravenous thrombolytic drugs to break up and dissolve clots and arterial blockages. Thrombolytics are often known as "clog busting" drugs. While thought to reduce mortality when administered within hours of AMI onset, thrombolytics are not clinically appropriate for all patients. Risk of serious side effects exist, especially severe internal bleeding. Of particular concern is cranial hemorrhaging. As a result, thrombolysis is not recommended for patients suffering from a recent trauma, active internal bleeding, severe hypertension, or intracranial neoplasm (Grzybowski et al. [2003]).

In addition to medical management, more "invasive" treatment options are available. Percutaneous coronary intervention (PCI) includes angioplasty and stenting. During PCI a catheter is threaded through the femoral artery and

to the coronary artery. For angioplasty a balloon is inflated on the end of the catheter which compresses plaque and expands the blocked artery, restoring blood flow. During stenting a small mesh, metal tube resembling a scaffolding structure is placed in the blocked artery to help maintain unimpeded blood flow. Two types of stents exist: bare-metal and drug-eluting (DES). DES are coated with drugs that are meant to reduce risk of the repeat blockage, or restenosis, of the stented artery. DES first became widely available in the early 2000's following FDA approval in 2003. One concern of PCI treatment methods is that the procedure only provides temporary relief from arterial blockage and so patients often require repeat interventions.

An alternative to PCI is coronary artery bypass graft surgery (CABG). CABG is often known as "open-heart surgery" although CABG is more precisely a specific type of open-heart surgery. During CABG, blood flow is physically rerouted to avoid blocked arteries. The patient's chest is opened-up. Often, the heart is temporarily stopped and blood flow is directed through a heart-lung machine, although the surgery can be performed "off-pump". Sections of arteries are harvested from other parts of the patient's body and grafted to coronary arteries so that the blocked artery is bypassed. Needless to say, CABG is an invasive, traumatic experience. Recovery time for CABG dwarfs that for PCI and risk of perioperative mortality is thought to be higher for most patients. However, successful CABG may provide a more permanent "solution" to arterial blockages. CABG requires substantially more resources than PCI and is a more expensive procedure (Hlatky et al. [1997]).

For any given patient, all three "branches" of treatment may have advantages and disadvantages. While general guidelines exist for appropriate and effective utilization of AMI treatments, and despite the vast volumes of research, no treatment protocol exists that is appropriate for all patients; "the medical decision making surrounding PCI is complex and requires physician judgement" (Lin et al. [2007]). While this may be true, substantial quantitative data do exist on the efficacy of each treatment method. However, each piece of evidence comes with serious caveats.

The many randomized clinical trials (RCTs) studying PCI, CABG, and medical therapy[13] provide good examples. These studies provide insight into the effectiveness of a given surgical procedure or medication. However, by design these studies require a controlled, isolated environment and are often limited by concerns over small sample sizes, self-selection, and external validity. Similarly, many aspects of health care provision are simply not suitably studied through RCTs for ethical, practical, or financial reasons. In addition, for AMI treatment

[13]Examples include Hoffman et al. [2003], Serruys et al. [2001], Morrison et al. [2001], BARI [1996], CABRI [1995]. See Bakhai et al. [2005] for a detailed review of numerous published and on-going RCTs.

the rapidly changing technological landscape has quickly rendered the results of many RCTs out-dated. CABG is often said to be most appropriate for treatment of several more serious forms of CAD, including 3-vessel disease, left main disease, or vessel disease involving the left anterior descending coronary artery. However, current research even hedges on these more extreme cases, the ACC/AHA guidelines suggests that it would be "unwise to deny contemporary PCI" in many cases on "the basis of current information" (see p. 850 of Anderson et al. [2007]). In addition, most RCTs, often for logistical and enrollment purposes, focus on stable CAD patients, rather than those who are in the midst of an AMI. Decision making and the link between treatment assignment and health outcomes may be substantially different for the AMI patient population.

Several studies in the economic literature augment the findings of RCTs. These studies usually avoid focusing on narrow clinical settings and rather use broader measures of variation in utilization rates of treatment technology to estimate the benefits (and costs) associated with AMI treatment. McClellan et al. [1994] uses patient distance to nearest catheterization-equipped hospital to sidestep the treatment decision via instrumental variables and provide estimates of the efficacy of PCI for marginal patients. They estimate significantly smaller effects of PCI on mortality than commonly reported in contemporary RCTs. A series of papers[14] focus on variation over time and across hospitals in treatment utilization rates driven by the diffusion of treatment technology, especially that of angioplasty in the late 1980s and early 1990s. They tend to find increased productivity from intensive AMI treatment, but show that patterns of technology diffusion are critical. They show results for the extensive margin (decision of facilities to adopt new treatment technology) to be important and easier to interpret. Results for the intensive margin, the decision within a facility as to how and when to utilize a treatment technology, are more limited. Instruments can help alleviate concerns over unobservables and selection, but this approach limits the interpretation to marginal patients and abstracts away from the decision making process that may drive observed utilization rates for infra-marginal cases. Cutler et al. [1998] examine the costs and benefits of intensive treatment for cardiovascular disease, while Cutler and McClellan [2001] look more generally at whether benefits from technological advances in medical treatments outweigh the costs.

Complementing this literature are studies showing that treatment utilization varies with patient characteristics. Racial and gender differences in utilization rates of intensive AMI treatments are well established and have drawn the attention of policy-makers nationwide[15]. These studies generally find that minorities

[14]See, for example Cutler and McClellan [1996], Cutler and Huckman [2003], Chandra and Staiger [2007], and Skinner and Staiger [2009]

[15]See, the Institute of Medicine (2002) report *Unequal Treatment: Confronting Racial and*

and women have lower utilization rates, even after controlling for patient and hospital characteristics. Kressin and Petersen [2001] provides a review of this literature. Schneider et al. [2001] explore whether racial differences can be explained by inappropriate "overuse" of intensive treatment in certain patient groups. They find that overuse is greater among white males, but that this cannot fully explain observed racial differences. Epstein et al. [2003] provide some evidence that at least some of the racial disparity is explained by a higher rate among blacks to not receive revascularization when it is indicated. Whittle et al. [1993] focus on patients discharged from Veteran's Affair hospitals where the role of financial incentives is minimized. They still find significant racial differences in the use of invasive cardiovascular procedures and suggest that these differences are likely due to social or clinical factors. There is a belief, however, that clinical factors do not fully explain the gender and racial disparities. Prejudice, clinical uncertainty and stereotyping may play a role (see Balsa and McGuire [2003]). In recent work Chandra and Staiger [2010], propose a test of prejudice and statistical discrimination and apply it to the case of AMI treatment. They do not find evidence of prejudice-based explanations and suggest that women and blacks benefit less from treatment. The authors offer several channels for the latter observation, including clinical, social, and financial factors.

This project contributes to these literatures. Unlike most previous studies, my modeling approach directly ties patient characteristics to treatment choices through their influence on expected health outcomes. This provides insight into the mechanism producing the observed relationship between patient characteristics and treatments. While such an approach does not provide an "optimal" prescription for AMI treatment, it may shed some light on how providers currently value outcomes, the role of information in the treatment assignment process, and whether treatment assignment is significantly driven by factors that do not directly influence patient outcomes. In the next section I outline the simple, outcome driven choice model for doing this.

2.3 A Model of Utility Maximizing Treatment Choice

This section describes the simple outcome-driven model of treatment decisions that will be the focus of the remainder of the chapter. In the model, there is a single action, the choice of treatment plan. This action is taken by the health care provider. The modeling environment begins upon an AMI patient's admission to an inpatient facility. At this point, the provider assesses the health status of the

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patient and considers treatment options, choosing the treatment that maximizes their own expected utility. Utility in the model takes a very simple form; providers value health outcomes and incur costs from implementing treatments.

There are three possible patient outcomes: in-hospital death, readmission, and survival without readmission (or "healthy"). Each outcome (y) is assigned a utility value (u^y). Each treatment plan (s) has a "cost", (c_s) associated with it. Realized utility, $U(s)$, can be expressed as

$$U(s) = \begin{cases} u^{death} - c_s & \text{if In-Hospital Death} \\ u^{readmit} - c_s & \text{if Readmission} \\ u^{healthy} - c_s & \text{if Healthy} \end{cases}$$

The only control the provider has over health outcomes in the model is through the choice of treatment plan. Once this decision is made patient outcomes are generated according to stochastic processes and provider utility is realized. The provider chooses a treatment after forming an expectation over the probability of each outcome. These probabilities may differ across treatment options and patients. The provider is assumed to behave rationally and know how treatments affect the probabilities of outcomes for each patient. The value of health outcomes are assumed to be not path dependent. That is, the value of an outcome is the same regardless of the treatment plan used to achieve it.[16]Figure 2.1 provides an illustration of the sequential framework and payout structure of the simple model. Note that $s = 0$ corresponds to OMT, $s = 1$ to CABG, and $s = 2$ to PCI.

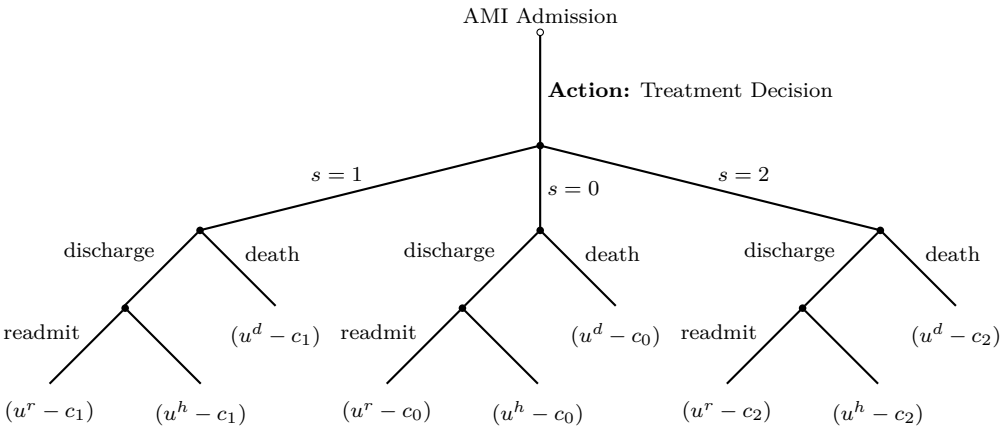


Figure 2.1: Diagram of Simple Model of AMI Treatment

[16]This is a strong assumption. For example, invasive procedures may have comorbidities associated with them that may significantly alter the quality of life, which should be considered along with effects on mortality (Jokinen et al. [2010]).

Since the values of outcomes are constant across treatments, to determine the utility maximizing choice the provider considers two pieces of information: relative efficacy and relative cost. Relative efficacy refers to the difference in the probability of health outcomes between two treatments. For example, probability of death may be higher for CABG but if the patient survives and is discharged alive, the probability of readmission due to complications may be lower. That is, CABG could offer advantages in long-term stability at the "price" of short term mortality risk. This could be true for one patient, but not for the next. Relative costs, as defined in this model, take a very general interpretation. The most obvious component of "costs" are reimbursement payments. Additionally, costs could include the labor and capital required to perform a given treatment. Costs could take on a "softer" interpretation. For example, Lin et al. [2007] surveyed a sample of cardiologists and found that non-clinical factors such as fear of regret, avoidance of legal responsibility, and desire to ease patient anxiety may induce use of PCI in patients with stable coronary artery disease[17]. Costs, as defined in the model, capture factors that influence the value of a treatment option that cannot be attributed to the efficacy of the treatment as it relates to the set of discrete health outcomes.

The three patient outcomes are mutually exclusive. The provider wants to maximize realized utility and so chooses the treatment option, s^*, that maximizes expected utility. The provider's expected utility for treatment s can be written

$$\mathbb{E}\left[U\left(s\right)\right] = Pr(d|s)u^d + \left(1 - Pr(d|s)\right) Pr(r|s)u^r + \left(1 - Pr(d|s)\right)\left(1 - Pr(r|s)\right) u^h - c(s)$$

where notation has been simplified so that $death = d$, $readmit = r$, and $healthy = h$. The provider chooses s^* such that $\mathbb{E}\left[U\left(s^*\right)\right] > \mathbb{E}\left[U\left(s\right)\right] \; \forall s$.

Note that "death outside the hospital" is not included in the set of outcomes. The assumption, in the context of the model, is that patients are readmitted to inpatient facilities before they die. For most cases, this assumption may be acceptable. However, some sudden causes of death, especially in the elderly population, such as stroke, may violate this assumption. The implications of this assumption for my empirical application are discussed in later sections. For modeling purposes, it is sufficient to think of the value of the "readmission" outcome as including the likelihood of future death conditional on readmission. The reader will note that the model generalizes easily to include a fourth outcome, "out of hospital death". For exposition and (current) data considerations, I limit the model to the three outcomes described above.

[17] Treatment patterns for stable CAD may differ significantly from those for emergency AMI, but nonetheless, we should be aware of the possibility of such non-clinical factors when interpreting results.

2.3.1 Role of Information Asymmetries and Unobserved Heterogeneity

In making a treatment choice, the provider forms an expectation over the probability of each outcome. In doing so, he incorporates all available, relevant observable information in evaluating the efficacy of a treatment for a given patient. It is likely that the provider uses information in the treatment decision that I, as the researcher, do not observe. The result is a wedge between the provider's beliefs about the relative efficacy of a treatment option for a given patient and my estimation of the relative efficacy.

For example, it may appear to me, using observable information, that a patient would enjoy significant reductions in risk of readmission if they were to receive CABG. However, the provider observes something about that patient that I do not, which makes them very unlikely to survive CABG. In this case, I may see the patient assigned to PCI or OMT and it will appear that the provider does not strongly consider readmission when making treatment choices.

In this way, information asymmetries and unobserved heterogeneity can affect conclusions from observational studies of both the values placed on health outcomes by providers and the efficacy of each treatment option. The empirical specification allows for information asymmetries of this kind. How this is done is made clear in the next section, which builds the empirical version of the simple model described above.

2.4 Empirical Framework

The previous section described a simple framework for modeling the assignment of treatments to patients. In the model, health care providers make a choice of treatment so as to maximize their expected utility. Providers derive utility over a set of discrete health outcomes and incur treatment-specific costs. The full model allows for heterogeneity in the efficacy of a treatment for a given patient. This heterogeneity is observed by the provider but not by the researcher.

The rest of this section builds the empirical model that is used to estimate the model on data of AMI patients in California.

2.4.1 Specification of Outcomes and Utility

In order to implement the model described above I must specify stochastic processes for both in-hospital death and readmission, as well as the expected utility function. I begin with the stochastic processes for the outcomes, but since these process may vary with the treatment choice the patient undergoes, it is helpful to note that a treatment choice will be noted as $s \in \{0, 1, 2\}$, where $s = 1$

indicates CABG, $s = 2$ indicates PCI, and $s = 0$ indicates medical treatment with no surgical intervention (OMT), along with an indicator y_s that is equal to 1 if a patient has undergone treatment s.

Outcome Equations

There are two outcomes of interest: in-hospital death and readmission. I adopt a very simple, intuitive specification for each outcome. I allow the probability of an outcome to depend on characteristics of the patient. These characteristics are observable to the provider, but may be partially unobservable to the researcher. In addition, I allow the effect of characteristics, both observed and unobserved, to vary with treatment choice, s. That is, for example, patients with heart failure are allowed to have different outcome probabilities for CABG, PCI, and OMT. This flexibility is consistent with both the medical literature and the data and provides minimal computational and modeling drawbacks.

Both observed outcomes of the model are discrete, binary events. An important aspect of the model is that these outcomes are sequential and mutually exclusive. Readmission is never observed for patients who die in the hospital. As such, the probability of readmission defined here should be interpreted as the probability, at the time of the initial admission, of readmission, conditional on the patient being discharged alive. The *ex ante*, unconditional probability of readmission is easily constructed using the probability of death and this conditional readmission probability.

Define indicators, y_d and y_r, that are equal to one if death and readmission are observed, respectively. Assume each outcome is generated by a latent, random variable that is normally distributed, conditional on patient characteristics[18]. Define these by y_d^* and y_r^* for death and readmission, respectively. Death is observed when $y_d^* > 0$ and readmission when $y_r^* > 0$. The probability of an outcome is a function of the treatment choice, s, as well as characteristics of the patient observed by both the provider and the researcher, X. In addition, the provider may have more information than the researcher about the efficacy of a treatment for a given patient. This unobserved (to the researcher) heterogeneity is modeled with patient-specific random effects[19], $a_{i,s}$, that enter the in-hospital death and readmission equations. These assumptions generate very simple forms for the

[18]This latent process can be thought of as the evolving health status of the patient, when health deteriorates sufficiently an event is triggered: death if the patient is in the hospital and readmission if the patient has been discharged.

[19]There are several approaches to modeling unobserved heterogeneity. I adopt a simple specification here, although this could quite easily be extended in several ways in future work. For example, interacting the random effect with observable characteristics would allow for variation in the severity of co-morbidities in a way I cannot infer from the data.

probability of each outcome for patient i for treatment s.

In-hospital Death:

$$\begin{aligned} y_d &= 1 \text{ if } y_d^* > 0 \\ y_d^* &= X\beta_s^d + a_{i,s}^d + \epsilon_d \\ \epsilon_d &\sim N(0, \sigma_d^2) \\ p_{i,s}^d &\equiv Pr(y_d = 1 | s_i, X, a_{i,s}^d) = \Phi\left(\frac{X\beta_s^d + a_{i,s}^d}{\sigma_d}\right) \end{aligned}$$

Readmission, conditional on discharge:

$$\begin{aligned} y_r &= 1 \text{ if } y_r^* > 0 \\ y_r^* &= X\beta_s^r + a_{i,s}^r + \epsilon_r \\ \epsilon_r &\sim N(0, \sigma_r^2) \\ p_{i,s}^r &\equiv Pr(y_r = 1 | s_i, X, a_{i,s}^r, y_d = 0) = \Phi\left(\frac{X\beta_s^r + a_{i,s}^r}{\sigma_r}\right) \end{aligned}$$

where $\Phi(z)$ is the Gaussian distribution function evaluated at z, X are patient characteristics observed by the researcher and the provider, and $a_{i,s}^d$ and $a_{i,s}^r$ are the patient-specific random effects for death and readmission, respectively. As written above, the unobserved heterogeneity is independent across the six outcome equations. In my estimations I impose structure on correlation of the unobserved heterogeneity across treatments and outcomes[20]. In particular, I assume there is a single dimension of unobserved heterogeneity, α, which enters each of the six outcome equations. A vector of loading factors, $\psi = [\psi_1 \ \psi_2]$, then scales the unobserved factor in each equation. The resulting functional forms for the a_s^d and a_s^r are shown in Table 2.1

Table 2.1: Functional Form for Unobserved Heterogeneity

Treatment	Death	Readmission
$s = 0$ (OMT)	$a_0^d = \alpha$	$a_0^r = \alpha$
$s = 1$ (CABG)	$a_1^d = \psi_1\alpha$	$a_1^r = \psi_1\alpha$
$s = 2$ (PCI)	$a_2^d = \psi_2\alpha$	$a_2^r = \psi_2\alpha$

This form is fairly restrictive, but has some appealing properties. In particu-

[20]Note the assumption that ϵ_d and ϵ_r are independent. This requires that death and readmission are not correlated in ways unobserved to the provider. This assumption can be relaxed.

lar, two features are worth emphasizing. First, ψ_1 and ψ_2 allow for unrestricted correlation between the effect of the unobserved heterogeneity on the efficacy of CABG and PCI. *A priori* it is not obvious what should be the sign of this correlation. It may be that a given patient is a "better" fit for CABG and a "worse" fit for PCI than would otherwise be predicted from observables, or vice versa. It allows for either loading to be zero if unobserved heterogeneity is not significantly related to outcomes for that particular treatment. Second, the restriction on the correlation between death and readmission is substantive. Within a treatment, if the unobserved factor increases the probability of death, it will also increase the probability of readmission post-discharge, and vice versa. Although restrictive, if we think of α as a single index of unobserved health or "frailty", then this restriction requires that frailty similarly increase risk of death and readmission, which is at least intuitively palatable.[21]

For each patient, the value of unobserved heterogeneity is observed by the provider, who can incorporate this information into the treatment decision. Since I do not observe the heterogeneity, I must specify a marginal density for α_i, call it $f(\alpha)$. I assume that α has a point-mass distribution with a relatively small number of points of support, M. For each point of support, I estimate a location, α_m and a probability, π_m. Under this specification, incorporating unobserved heterogeneity adds to the model $2M$ parameters for the point-mass locations and probabilities and two parameters for the loading factors.

Choice Equation: Expected Utility over Outcomes

Since health outcomes are mutually exclusive the realized utility from treatment choice s is given by

$$U(s) = y_d \cdot u^d + y_r \cdot u^r + (1 - y_d) \cdot (1 - y_r) \cdot u^h - c(s)$$

where u^d and u^r are the values placed on in-hospital death and readmission, and u^h is the value of a discharged patient who does not require readmission. The costs of the treatment plan s are given by c_s. Importantly, death and readmission are mutually exclusive events; only one of u^d, u^r, and u^h is realized for any given patient. Prior to making a treatment choice, the provider forms an expectation of the probability of each outcome event and chooses the treatment that maximizes expected utility. Expected utility is the probability-weighted sum of the value obtained under each outcome minus expected costs. Outcome probabilities

[21] There are many ways in which the functional form of the unobserved heterogeneity could be relaxed. Adding a loading factor for readmission that is constrained to be positive is one potential extension. This would simply involve multiplying the a^r by $\exp(\lambda)$ and estimating λ along with ψ_1 and ψ_2. A second would be to add separate loading factors for CABG and PCI readmission, ψ_3 and ψ_4.

depend on patient characteristics and choice of treatment and are defined as in Section 2.4.1. In making a treatment choice, the provider considers the following expression for the expected utility of treatment s:

$$\begin{aligned} V\left(s|X, a_i\right) &= \mathbb{E}\left[U\left(s|X, a_i\right)\right] + \xi_s \\ &= p_{i,s}^d u^d + \left(1 - p_{i,s}^d\right) p_{i,s}^r u^r + \left(1 - p_{i,s}^d\right)\left(1 - p_{i,s}^r\right) u^h - c_s + \xi_s. \end{aligned}$$

where ξ_s are factors related the value of treatment s that are observed by the provider, but not by the researcher and not captured in $U\left(s\right)$. In the estimations, I normalize u^h to 0 and so u^d and u^r should be interpreted as the utility of death and readmission *relative* to the utility obtained from treating a patient who is discharged alive and not readmitted ("healthy"). This normalization reduces the provider's expected utility to

$$V\left(s|X, a_i\right) = p_{i,s}^d u^d + \left(1 - p_{i,s}^d\right) p_{i,s}^r u^r - c_s + \xi_s$$

Under the assumption that health care providers are von Neumann-Morgenstern expected utility maximizers, the optimal treatment, s^*, is defined as

$$s^* = \arg \max_{s \in \{0,\, 1,\, 2\}} V\left(s|X, a_i\right) \text{ so that } V\left(s^*|X, a_i\right) > V\left(s|X, a_i\right) \ \forall\ s \in \{0,\, 1,\, 2\}.$$

Under the assumption that ξ_s is distributed extreme value 1, the probability of observing choice of treatment s for patient i is given by the familiar and convenient expression,

$$p_{i,s} \equiv Pr(s_i^* = s_i|X, a_i) = \frac{exp(\delta_s)}{\sum_{k=0}^{2} exp(\delta_k)}, \text{ where } \delta_s = \mathbb{E}\left[U\left(s|X, a_i\right)\right].$$

The model, as described above, assumes that the values of outcomes are constant with respect to patient characteristics and treatment choice. This assumption can be relaxed, allowing for a more flexible functional form for u^d and u^r to allow for the values of outcomes to vary along important policy dimensions such as gender, race, insurance status, or hospital type. This flexibility is an important strength of the model framework. This can be easily implemented by interacting relevant characteristics Z with u^d and u^r so that $V\left(s|X, Z\right) = p_s^d u^d Z + \left(1 - p_s^d\right) p_s^r u^r Z - c_s + \xi_s$. Such extensions are particularly relevant in the context of racial and gender disparities in treatment utilization and health outcomes. The mortality and readmission functions capture differences in treatment assignment that are related to treatment efficacy. The extent to which

demographic components of Z are significant contributors to the treatment assignment function suggests that providers value patient outcomes differently for different groups of patients. The preferred model for this chapter includes a single dimension of flexibility for outcome values; u^d and u^r are functions of age.

$$\begin{aligned} u^d(Z) &= u_0^d + u_1^d \cdot (age - 65) \\ u^r(Z) &= u_0^r + u_1^r \cdot (age - 65) \end{aligned}$$

Justification for this additional flexibility to the functional form for utility is drawn from the quality of life literature. The idea being a surgical procedure is an investment in a patient's future health and the return on this investment, if successful, is diminished if the patient has fewer years to benefit from it.

An additional extension, and perhaps more precise formulation of this idea, would allow u^d and u^r to depend on the patient's overall health status. Successful treatment of AMI may be less valued for patients who are more likely to die from causes unrelated to AMI in the near future. In addition, successful treatment of AMI may be more valued for patients who, in absence of AMI, are overall healthier. This could be implemented by including the average probability for patient i of in-hospital death across treatments in Z so that

$$\begin{aligned} u^d(Z) &= u_0^d + u_p^d \cdot \overline{p^d} \\ u^r(Z) &= u_0^r + u_p^r \cdot \overline{p^d}. \end{aligned}$$

where $\overline{p^d} = \sum_{s=0}^{2} \frac{p_s^d}{3}$.

2.4.2 Constructing the Likelihood

The parameters of the model are estimated via maximum likelihood, including those for the choice equation ($\{u^d, u^r, c_s\}$), the outcome equations ($\{\beta_s^d, \beta_s^r\}$), and the distribution of patient heterogeneity ($\psi_1, \psi_2, \alpha_m, \pi_m\}$). For each patient, we observe one of three possible treatment choices. For each treatment choice, we observe one of three subsequent outcomes; the patient dies in-hospital (Death), is discharged and readmitted (Readmission), or is discharged and not readmitted (Healthy). This generates nine, observable, mutually exclusive sequences of treatment and outcomes. Call each treatment-outcome sequence y^j and let $p(y^j|X, Z, \alpha; \theta)$, be the probability of observing sequence y^j given X, Z, α, and the parameters of the model θ, where $\theta = \{\beta_s^d, \beta_s^r, u^d, u^r, c_s, \psi_1, \psi_2, \alpha_m, \pi_m\}$. Let $p(y^j)$ be shorthand for $p(y^j|X, Z, \alpha; \theta)$. Table 2.2 illustrates the nine simple forms that comprise the likelihood.

Table 2.2: Likelihood for 9 Outcomes

y^j	Observed Outcome	$p(y^j)$	Likelihood
y^1	OMT $\rightarrow$ Death	$p(y^1)$	$p_0 \cdot p_0^d$
y^2	OMT$\rightarrow$Readmission	$p(y^2)$	$p_0 \cdot (1-p_0^d) \cdot p_0^r$
y^3	OMT$\rightarrow$ Healthy	$p(y^3)$	$p_0 \cdot (1-p_0^d) \cdot (1-p_0^r)$
y^4	CABG$\rightarrow$ Death	$p(y^4)$	$p_1 \cdot p_1^d$
y^5	CABG$\rightarrow$ Readmission	$p(y^5)$	$p_1 \cdot (1-p_1^d) \cdot p_1^r$
y^6	CABG$\rightarrow$ Healthy	$p(y^6)$	$p_1 \cdot (1-p_1^d) \cdot (1-p_1^r)$
y^7	PCI$\rightarrow$ Death	$p(y^7)$	$p_2 \cdot p_2^d$
y^8	PCI$\rightarrow$ Readmission	$p(y^8)$	$p_2 \cdot (1-p_2^d) \cdot p_2^r$
y^9	PCI$\rightarrow$ Healthy	$p(y^9)$	$p_2 \cdot (1-p_2^d) \cdot (1-p_2^r)$

From these expressions, for each patient I construct:

$$P_i(Y_i|X,Z,\alpha;\theta) = \prod_{j=1}^{9} (p(y^j))^{d_i^j}$$

where Y_i is a vector of the nine outcome indicators y^j and d_i^j is an indicator which is equal to one if treatment-outcome sequence y^j is observed for patient i. Since $P_i(Y_i|X,Z,\alpha;\theta)$ is conditional on α, which is unobserved to the researcher, the likelihood of the observed treatments and outcomes is given by a probability-weighted average of the likelihood under each possible value of support for the point-mass distribution[22]. Formally,

$$P_i(Y_i|X,Z;\theta) = \sum_{m=1}^{M} \pi_m P_i(Y_i|X,Z,\alpha_m;\theta)$$

The log-likelihood then takes the familiar form:

$$\ell\ell(Y|X,Z;\theta) = \sum_{i=1}^{N} \ln(P_i).$$

To estimate the full model with unobserved heterogeneity, maximize log-likelihood with respect to the vector of model parameters θ.

[22]If instead I assume a normally distributed random effect so that $f(\alpha) = \phi(\frac{\alpha}{\sigma_\alpha})$ then integrating over this density would give the likelihood, $P_i(Y_i|X,Z;\theta) = \int_{-\infty}^{\infty} \frac{1}{\sigma_\alpha} P_i(Y_i|X,Z,\alpha_i;\theta)\phi(\frac{\alpha}{\sigma_\alpha})d\alpha$.

2.5 Data, Sample Construction, and Descriptive Analysis

The primary data source used is a non-public version of the Healthcare Cost and Utilization Project's (HCUP) State Inpatient Database (SID) for California from the years 2003 to 2007. The HCUP databases are sponsored by the Agency for Healthcare Research and Quality. The SID is the full universe of inpatient discharges in the state of California. These data include the hospital discharge records for all inpatient admissions in the state of California and contain data elements providing patient clinical diagnostic and procedural information, patient demographics, hospital identification, length of admission stay, whether the admission was scheduled at least 24 hours prior, quarter and year of admission and discharge, and primary insurance type. Diagnostic information includes a vector of up to 25 ICD-9 diagnosis codes, including specification of the primary diagnosis. A sequence of up to 21 procedure ICD-9 procedure codes indicates which, if any, medical procedures were performed during the patient's stay. Each record also contains information on the source of the patient's admission, be it through the emergency department (ED), transfer from another hospital or healthcare facility, or "routine" channels (such as physician referral or an outpatient clinic). Likewise, the disposition of the patient upon discharge is observed. This provides information as to whether the patient was discharged home, transferred to another facility, or died while in the hospital.

I supplement the SID with HCUP's Revist Database files. These files provide a patient identification number with which I can link a patient's records over time. In addition, with the Revisit Databases I know the number of days that have elapsed between each inpatient admission. After linking each patient's records over time I construct my sample of AMI patients. I identify all patients, age 50 through 85 that were admitted with a primary diagnosis code of AMI during 2003 to 2007[23]. Ideally, my sample would be limited to patients suffering their first AMI, since treatment protocol and efficacy may be different for subsequent heart attacks. Unfortunately, this information is not directly available in SID. Instead, I limit my sample to the first occurrence of AMI in my time period and for codes indicating an "initial episode of care". Additionally, I only use patients for whom their first AMI in my sample occurred between 2005 and the second quarter of 2007[24]. This restriction ensures that each patient in the sample has not

[23]ICD-9 codes used to identify AMI: 410.00, 410.01, 410.10, 410.11, 410.20, 410.21, 410.30, 410.31, 410.40, 410.41, 410.50, 410.51, 410.60, 410.61, 410.70, 410.71, 410.80, 410.81, 410.90, 410.91

[24]Patients first observed with AMI in the third and fourth quarter of 2007 do not have sufficiently long post-AMI data to track readmission and future health outcomes.

suffered an AMI in at least the previous two years[25]. For each AMI admission, I identify if the patient underwent CABG or PCI[26]. An AMI patient who does not undergo CABG or PCI is assigned to the "optimal medical treatment" (OMT) treatment group. To qualify the surgical procedure must occur during the initial AMI admission[27].

2.5.1 Important Sample Restrictions: Facilities and Transfers

It is worth addressing two substantive aspects of my sample construction. First, I have limited my analysis to hospitals that have the technology to perform all three treatment options. Since I do not directly observe the technological capacity of each facility in my data, I restrict my analysis to patients admitted to facilities that perform CABG, PCI, and OMT for at least some fraction of their AMI patients. Specifically, a facility is included in my sample if I observe at least 20 PCI and 20 CABG procedures performed on AMI patients between 2005 and 2007. This definition is similar to that used in McClellan et al. [1994]. As discussed in Section 2.2, both the adoption and utilization margins are important when considering the overall effect of new treatment technologies. However, in this chapter, I am focusing on utilization decisions rather than technology adoption. Thus, I restrict my sample to facilities that have already invested in the relevant technologies. During the 1980s and 1990s during the large expansion of angioplasty technology, we might have been concerned that this restriction would eliminate the important margin in AMI treatment. However, this is less of a concern between 2005 and 2007. Over this time period, we can feel more comfortable in the stability of treatment technology diffusion[28]

Second, I do not consider AMI patients who are transferred between hospitals. Specifically, I drop from the sample AMI patients who are transferred between

[25]This procedure leaves open the possibility that patients in my sample for 2007 are, on average, healthier than those suffering AMI in 2005, since they are guaranteed a slightly longer "AMI-free" pre-period.

[26]ICD Codes for CABG: 36.10, 36.11, 36.12, 36.13, 36.14, 36.15, 36.16, 36.17, 36.18, 36.19; and for PCI: 00.66, 36.01, 36.02, 36.05, 36.06, 36.07

[27]I allowed one exception to this rule. I considered a patient to have undergone a procedure if that procedure occurs during a subsequent admission provided the admission is scheduled in advance and occurs within 7 days of the initial AMI *admission* date. This is motivated by medical literature suggesting that the optimal time for CABG may be up to seven days post-AMI. However, after placing other restrictions on the sample construction, this allowance did not contribute any observations to the sample.

[28]It is worth noting, that in the early 2000's the FDA began to approve drug-emitting stents (DES) for use in PCI. Bare-metal stents (BMS) had been in use for several years. While DES may have impacted the efficacy of PCI, substantial technological investment was not required for facilities to replace BMS with DES.

facilities during their initial admission for AMI. A similar restriction is made by Chandra and Staiger [2010][29]. Transferred patients may have received treatment at a previous facility, which would change the decision environment. In addition, patients are not transferred randomly. The decision to transfer is in itself a decision over treatment options. While a very important and interesting component of the complete picture of AMI treatment decisions, I do not address this stage of treatment decision making in this chapter.

Although I do not consider transfers in this project, the model is actually well suited to studying transfers and their effect on patient outcomes. An extension of the model could remove both restrictions on facilities and on transfers and expand the choice set of treatment options to include transfer as an explicit "treatment", with corresponding outcome functions that represent the expected mortality and readmission rates for patients that undergo transfer. Alternatively, the choice set could remain confined to the three AMI treatments, yet outcome functions for CABG and PCI for hospitals without the necessary technology would include adjustments for the reality that transferred patients may suffer reduced efficacy of both CABG and PCI compared to an similar, but untransferred patient. Transfers, while opening up treatment options for patients admitted to smaller, less equipped facilities, take time. Facilities must consider the reduced efficacy of CABG and PCI post-transfer relative to immediate OMT when deciding to transfer a patient. Indeed, preliminary analysis of the data indicates that transferred patients are much more likely to receive CABG and PCI than the average patient admitted to a technology-equipped facility. These patients could be those for which the transferring hospital expects higher than average improvement in outcomes from intensive treatment. However, it could be also related to non-clinical considerations such as insurance payments or the interaction between patient demographics and the value of outcomes. The extended model, including transfers, can be used to estimate the implicit costs and benefits of transfers. Ultimately, a simulation exercise could provide counter-factual treatment assignment and outcome estimates for patients who were initially admitted to facilities lacking intensive treatment technology. The literature does not currently have a fully integrated model of transfers and the implications for patient outcomes and health care costs.

[29]Studies in the hospital quality literature typically assign outcome measures for transferred patients to either the hospital of initial admission or final treatment, although some drop transferred patients altogether (Jensen et al. [2009] test the sensitivity of their quality measures to these assumptions).

2.5.2 Summary of Data

After constructing the sample as described above, I am left with a sample of 47,438 AMI patient observations. Table 2.3 provides means for observable characteristics of the patients in the sample. The bottom section of the table indicates that 12.2% of the sample undergoes CABG, 56% PCI, and 31.8% OMT. Looking across columns, there are clear differences in observable traits of patients who receive CABG, PCI, and OMT. Of course, without further analysis we do not know the cause of these differences. Patient demographics include age, race, gender, and primary insurance type. Patients who do not undergo intensive treatment (CABG or PCI) are, on average, older, more likely to be female or black and more likely to have Medicare as their primary insurance payer. Intensive treatment patients are also much more likely to be covered by private insurance than patients undergoing OMT. There is an indicator for the quartile of the median income of the patient's zip code. It appears that CABG patients may come from slightly poorer zip codes than PCI patients. The "UIC" variables are "urban influence codes" that put counties into four broad categories: large metro areas ($>$ 1 million residents), small metro areas ($<$ 1 million residents), micropolitan areas (non-metro counties with an urban "cluster" of at least 10,000 residents), and rural counties (not part of a metro or micropolitan area). Counties are included in a given metro or micropolitan area if at least 25% of commuting is to the metro or micropolitan area[30]. To give these numbers some context, consider that in 2003 77% of CA population resided in a county coded as a "large metro area" and 20.6% were from a "small metro area". If anything, it appears that CABG patients are slightly more likely to be from smaller metro areas.

The second section of the table provides summary information on the health status of patients *upon admission* for AMI. The SID data contain codes that indicate whether a medical condition was present at the time the patient was admitted. I utilize this information to reduce the endogeneity that would plague *ex post* diagnosis information. I include health indicators that are generally considered relevant risk factors for both onset of AMI as well as for complications during revascular procedures. It is this type of conditions that a physician might consider when determining expected efficacy of CABG and PCI[31]. Almost 30% of the sample suffers from congestive heart failure, although only 19.2% of PCI patients. Over half of the sample suffers from hypertension, which is relatively similar across treatment groups, with OMT having the lowest incidence. Can-

[30] See http://www.ers.usda.gov/Briefing/Rurality/UrbanInf for more information

[31] Unfortunately, my data lack the detailed medical chart data that is often used in other studies of AMI treatment. For example, I do not have information on blood pressure, body weight, injection fraction, or results of lab tests. Inclusion of this information may improve the predictive power of the outcome functions. The inclusion of unobserved heterogeneity will capture some of the relevant contribution of omitted health information.

Table 2.3: Sample Means for Patient Characteristics

Demographics	**All**	**CABG**	**PCI**	**OMT**
Age (years)	68.204	67.753	66.195	71.912
black	0.050	0.035	0.039	0.076
female	0.359	0.276	0.305	0.484
Medicare	0.516	0.503	0.441	0.654
Medicare - DI	0.044	0.038	0.041	0.051
Medicaid	0.071	0.081	0.063	0.080
Private Insurance, <65 yrs.	0.237	0.231	0.310	0.113
Private Insurance, >65 yrs.	0.063	0.068	0.063	0.061
Other Payment Type	0.069	0.078	0.082	0.042
1stQrtile Zip Med. Inc.	0.238	0.257	0.221	0.260
2ndQrtile Zip Med. Inc.	0.253	0.277	0.239	0.268
3rdQrtile Zip Med. Inc.	0.252	0.244	0.259	0.244
4thQrtile Zip Med. Inc.	0.234	0.196	0.258	0.207
Missing Zip Med. Inc.	0.022	0.026	0.023	0.020
UIC: Large Metro Area	0.724	0.680	0.719	0.748
UIC: Small Metro Area	0.241	0.274	0.243	0.224
UIC: Micropolitan Area	0.019	0.026	0.021	0.014
UIC: Rural	0.011	0.015	0.012	0.007
UIC: Missing	0.005	0.006	0.005	0.006
Health Status	**All**	**CABG**	**PCI**	**OMT**
Heart Failure	0.295	0.381	0.192	0.445
Hypertension	0.551	0.597	0.566	0.509
STEMI	0.468	0.360	0.591	0.295
Diabetes	0.355	0.449	0.300	0.416
Peri.Vasc. Disease	0.060	0.072	0.040	0.090
Coronary Atherosclerosis	0.827	0.961	0.893	0.658
Other Atherosclerosis	0.034	0.063	0.024	0.041
CardioShock	0.052	0.067	0.052	0.045
Hyperlipidemia	0.407	0.453	0.435	0.341
EndStageRenal	0.022	0.022	0.014	0.037
Cancer	0.043	0.031	0.029	0.074
Anemia	0.218	0.391	0.140	0.288
Number of Diagnoses (POA)	8.603	9.312	7.286	10.647
Length of Stay (days)	5.847	12.682	4.453	5.674
Diagnostic - Heart	0.755	0.876	0.896	0.462
Diagnostic - Vessel	0.799	0.897	0.960	0.476
Hospital Info	**All**	**CABG**	**PCI**	**OMT**
Teaching	0.134	0.120	0.132	0.143
Public	0.078	0.055	0.083	0.079
Private For-Profit	0.155	0.155	0.157	0.152
Private Not-for-Profit	0.742	0.763	0.733	0.750
Hospital Type Missing	0.025	0.028	0.027	0.019
Avg. Avl. Beds	349.7	343.4	347.1	356.8
N	47439	5796	26551	15092
Treatment				
CABG	0.122			
PCI	0.560			
OMT	0.318			

cer and end-stage renal disease are more prevalent among OMT patients[32]. PCI patients are less likely to be diabetic than either CABG or OMT patients. The data also contains information on the type of infarction: ST elevation infarction (STEMI) or non-ST elevation infarction (NSTEMI). Relative efficacy of treatments likely differ across the two types of infarction. Note that although the incidence of STEMI differs across treatment options, both types are represented in all three treatment groups. "Number of diagnoses" is a simple count of the number of diagnosis codes listed on the patient's discharge records (maximum is 25). I only count codes that are flagged as present on admission. This count can be thought of as a rough proxy for the patient's overall health[33]. On average, PCI patients have the fewest number of diagnoses and OMT the most. The final three rows of this section are measures that are endogenous to the treatment obtained during the hospital stay. Patients stay in the hospital for an average of 5.85 days. CABG patients have a significantly longer average stay in the hospital. This is unsurprising given the invasive nature of CABG and the longer expected recovery time relative to PCI.

The third section of the table provides information on the ownership type of the patient's admitting hospital and whether the hospital is designated as a teaching hospital. Teaching hospitals and public facilities seem to perform fewer CABGs. OMT patients are in slightly larger hospitals, on average, as measured by number of beds.

Overall, these summary statistics indicate that there are significant differences in the patient populations that receive each type of treatment for AMI. This is not unexpected. However, what is not clear from Table 2.3 is how the treatment assignment process is related to expectations over patient outcomes under each treatment option. Are patients assigned to CABG because they are relatively low risk for death or readmission or because without some intervention they are very likely to suffer negative outcomes? Similarly, if one treatment option offers reduced risk of in-hospital death, but greater risk of readmission in the near future, how does this tradeoff enter the treatment decision process? The model described above puts a structure on treatment assignment so that we can answer some of these questions. However, first I need to identify the health outcomes for each patient: which patients die in the hospital, which are discharged, and of these, which are readmitted.

[32]The relationship between the treatment of heart disease and cancer is discussed in Lleras-Muney and Honoré [2006]. The authors make the compelling case that improvement during recent decades in treatment of heart disease has masked improvements in cancer treatment. Patients who previously would have died from heart disease now live longer and eventually die from cancer.

[33]The Charlson index could provide an alternative measure.

2.5.3 Health Outcomes

In order to estimate the model of treatment choices, I must define, and identify in the data, a set of health outcomes. For the model as described above the health outcomes of interest are death and readmission. Deaths occurring during the inpatient stay are easily identified in the SID by a variable describing the disposition of the patient upon discharge. As Cutler and Huckman [2003] point out, death during admission is a relatively rare event for both CABG and PCI and so one might prefer mortality rates over a longer time horizon. Total AMI related deaths may differ from in-hospital deaths for two reasons, as mentioned in Cutler et al. [1998]. First, AMI patients may die before hospitalization. My data does not include patients who are not admitted to the hospital for at least a full day. Second, death may occur post-discharge. The SID, even in combination with the Revisit Database does not allow observation of deaths that occur outside of an inpatient facility or emergency department.However, by using the Revisit Database, I can verify that a patient is alive if they ever appear again in SID (or Emergency Department or Ambulatory Surgery databases). Of course, I can observe post-discharge death that occurs during a subsequent readmission. Patients who are discharged alive and who are not observed in a subsequent readmission record will necessarily be coded in the data as "healthy". To the extent that this group contains patients who have died outside the hospital, estimates may be biased. This is especially troubling if death outside the hospital is differentially likely to occur for a subset of treatment options. However, the effect of this bias should be to *increase* the value of a readmission. Thus, the large negative value I estimate for readmissions can be seen as a sort of an upper bound. Despite this, in-hospital death fits naturally into the sequential structure of the model as a relevant outcome for the choice environment. Were data available inclusion of post-discharge deaths may be best added as an additional "branch" in the sequence of potential patient outcomes, with a separate value weight in the utility function. Within the modeling framework, post-discharge death is only captured to the extent that the value of readmission incorporates expected death during readmission and a conditional expectation over future death.

Defining and identifying readmission in the data is a slightly more complex task. For each patient in my sample who is discharged from the initial AMI admission alive, I use the Revisit Database to track subsequent inpatient admissions. In the context of the model, relevant readmissions are those that are related to the treatment choice made during the initial AMI admission. Thus, I define a readmission using criteria based on evidence and research in the medical literature (see Curtis et al. [2009] and Hannan et al. [2003]). To be included as a readmission, the admission must be an unscheduled inpatient admission

occurring within 90 days of discharge from the initial AMI admission[34]. In addition, the primary diagnosis code must be for ischemic heart disease, stroke, post-surgical infection, or chest pain or the admission must include a revascularization procedure (CABG or PCI) or a diagnosis code for AMI (need not be primary diagnosis)[35]. I also include emergency department visits that result in death, but not emergency department visits that do not result in either death or admission to an inpatient facility. While there may be other plausible definitions of relevant readmissions, I have attempted to limit my definition to only include events that are both serious in nature and plausibly related to the treatment undertaken during the initial AMI admission. See Table 2.4 for the distribution of readmissions.

Table 2.4: Distribution of Reasons for Readmission, by Treatment During Initial AMI Admission

			Initial Treatment					
	Total		**CABG**		**PCI**		**OMT**	
Readmission Reason	N	%	N	%	N	%	N	%
Heart Failure	3,078	24.2	432	34.53	1,222	17.44	1,424	31.91
Ischemic Heart Disease	2,767	21.75	152	12.15	2,086	29.77	529	11.85
Stroke	111	0.87	15	1.2	41	0.59	55	1.23
Post-Surgical Infection	243	1.91	149	11.91	61	0.87	33	0.74
Chest Pain	1,251	9.83	122	9.75	807	11.52	322	7.21
CABG (and none of the above)	116	0.91	2	0.16	87	1.24	27	0.6
PCI (and none of the above)	936	7.36	62	4.96	787	11.23	87	1.95
AMI (and none of the above)	4,219	33.17	317	25.34	1,916	27.34	1,986	44.5
Total	12,721	100	1,251	100	7,007	100	4,463	100
Procedure during Readmit?	N	%	N	%	N	%	N	%
CABG	299	2.35	5	0.36	138	1.97	63	1.41
PCI	2,159	16.97	232	16.85	1,881	26.84	144	3.23

Note: The top panel reports the distribution of primary cause for readmission within 90 days of initial discharge for AMI-patients in the sample. Each column is the treatment administered during the initial AMI admission. Each row gives the primary reason for readmission. The bottom panel provide the percentage of readmitted patients who receive CABG and PCI during readmission, regardless of the primary reason for readmission.

Table 2.5 gives the fraction of patients that die in-hospital and are readmitted within 90 days of discharge for the entire sample and by treatment type. For the entire sample, 5.8% of AMI patients die while in the hospital and 16.4% are readmitted for an "AMI-related" reason. On average, OMT patients are the

[34]I use a readmission window of 90 days post-discharge. Most studies use either a 30 or 90 day window. The results do not substantively change when using a different readmission window.

[35]ICD codes: Heart Failure 428.x, 402.x1, 404.x1, 404.x3; Ischemic Heart Disease 411, 411.0, 411.1, 411.8, 411.81, 411.89, 414.0, 414.0x, 414.1x, 414.2, 414.3, 414.8, 414.9; Stroke 434, 434.0, 434.00, 434.01, 434.1, 434.10, 434.11, 434.9, 434.90, 434.91; Post-Surgical Infection 998.5, 998.51, 998.59; Chest Pain 786.5, 786.51, 786.52, 786.59, 413.0, 413.1, 413.9; Acute Myocardial Infarction 410.x, 412

most likely to die in the hospital (11.6%) and most likely to be readmitted post-discharge (18.5%). PCI patients are the least likely to die, while CABG patients enjoy the lowest readmission rates. These numbers confirm the findings from medical literature that on average, when compared with PCI, CABG may pose a higher immediate mortality risk, but that it provides a more stable, permanent solution to heart disease. That OMT patients die and are readmitted at the highest rate is not surprising, given the summary statistics in Table 2.3. OMT patients are on average older and sicker. However, it is not clear whether health outcomes for OMT patients would've been even *worse* had they been assigned to intensive treatment procedures.

Table 2.6 provides descriptive evidence of the substantial gender disparities in utilization and health outcomes discussed earlier. Note that both CABG and PCI are more heavily used for male patients and that females experience worse health outcomes, as measured by both in-hospital mortality and readmission rates. I use the model to explore the gender gaps in Section 2.6.4.

Table 2.5: Summary of Health Outcomes, by Initial Treatment

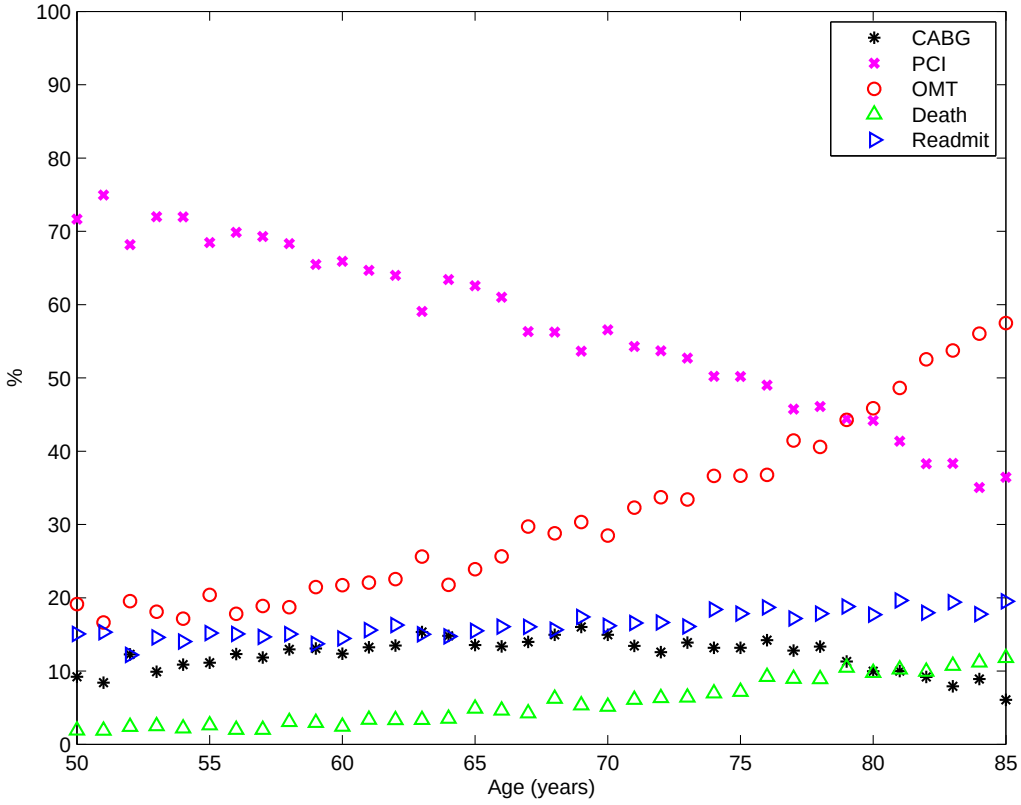
	All		**CABG**		**PCI**		**OMT**	
Outcome	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
In-hospital Death	2771	0.058	259	0.045	768	0.029	1744	0.116
90 Day Readmission	7773	0.164	808	0.139	4167	0.157	2798	0.185

Table 2.6: Summary of Treatments and Health Outcomes, by Gender

Gender	**CABG**	**PCI**	**OMT**	**Death**	**Readmission**
Males	0.1379	0.6061	0.256	0.0524	0.1495
Females	0.094	0.4767	0.4293	0.0691	0.1895

Treatments and outcomes vary by age in the data. Figure 2.2 provides the age profile for treatments and outcomes. The rate of PCI declines with patient age. It starts at just over 70% for patients 50 years old and drops to under 40% for patients 85 years old. OMT rate, on the other hand, steadily increases with age, from about 20% at age 50 to just under 60% at age 85. CABG rates follow a slight "inverse-U" shaped age profile, starting at about 9% at age 50, peaking at age 69 over 15% and then declining to just over 6% at age 85. In-hospital death increases fairly steadily as patients age, from about 2% to 11%. Readmission rates also increase with age ranging from 12% to almost 20%. Are age-related patterns of treatment choice reflective of changes in treatment efficacy as patients age? Or are they driven by factors unrelated to patient outcomes? The choice model can help answer these questions.

Figure 2.2: Age Profile: Treatments and Outcomes



2.6 Results

The estimation results are presented in four sections. The first section presents the estimation results for the full model, focusing on the estimates for the value of health outcomes (u^d and u^r) and treatment costs (c_s). In the second section, I provide the parameter estimates for and characterize the unobserved heterogeneity. The third section reports estimates of the efficacy of the three treatment options. Finally, the fourth section gives the results of an Oaxaca-style decomposition of the difference in treatment intensity and outcomes for female versus male patients.

2.6.1 Estimation Results for the Full Model

In this section I present and discuss the maximum likelihood estimates for the full choice model described in Section 2.4. I estimated several versions of the model with different number of mass points of support for the unobserved heterogeneity. The preferred specification includes four mass points and I focus on results from this model. The addition of unobserved heterogeneity significantly improved the fit of the model[36], although including more than four points of support did not significantly increase the log-likelihood.

In this section I will focus on the estimates of the parameter vector θ from the four mass-point model. In particular, I will focus on the parameters of the treatment choice equation, which include the values of outcomes u^d and u^r and the cost of treatment c_s. The estimates of the unobserved heterogeneity, $\{\psi_1, \psi_2, \alpha_m, \pi_m\}$, are discussed in detail in the next section. Recall that $\theta = \{\beta_s^d, \beta_s^r, u^d, u^r, c_s\}$. The vectors β_s^d and β_s^r are the coefficients on the patient observable characteristics that affect the probability of in-hospital death and post-discharge readmission, respectively, for treatment option s. These estimates are generally consistent with previous medical research. The interested reader can find the estimates in Appendix **??**. However, it is worth mentioning here the coefficients on female in the mortality equation. Females are associated with significantly higher risk of death under both CABG and PCI, but significantly *lower* risk of death under OMT. This puzzling result is consistent with evidence in the literature of significant differences in observed health outcomes for female AMI patients. Section 2.6.4 addresses the gender differences in more detail.

Table 2.7 reports the estimates for the values of outcomes, u^d and u^r. The negative estimates for u_0^d and u_0^r indicate that treatments are allocated, at least in part, to prevent death and readmission. The age-slope coefficients are positive (u_1^d

[36]The log-likelihood for the model with one point of support was -64,923.9. This improved to -64,874.3 for the four point model, an increase of 49.6 which required the estimation of eight additional parameters.

and u_1^r), so the value of preventing adverse outcomes is larger for younger patients. Combining the main and age effect gives a range for the value of outcomes. The estimate for the value of death ranges from -12.14 for a 50 year-old patient, to -8.92 for a 65 year-old patient, to -4.63 for an 85 year-old patient. The value of readmission ranges from -36.90 for a 50 year-old, to -34.93 for a 65 year-old, and -32.30 for an 85 year-old. The values of readmission are quite a bit larger in magnitude than those for in-hospital death, and decline less quickly with age.

Table 2.7: Estimation Results: Outcome Weights

	u^y	S.E.
u_0^d	-8.923	1.050
u_1^d	0.215	0.038
u_0^r	-34.926	4.330
u_1^r	0.131	0.0582

Outcome Weights by Age

	50 years	65 years	85 years
u^d	-12.14	-8.92	-4.63
u^r	-36.90	-34.93	-32.30

N= 47,439

Note: Estimation results are for the full model and include unobserved patient heterogeneity.

Table 2.8 reports the estimates for treatment costs, c_s. Treatment costs are modeled using indicators for the primary insurance type listed on the patient's hospital record. Insurance types are Medicare, Medicaid, private insurance, and "other payer", which includes self-pay, as well as various other government insurance plans, county indigent programs, and charity care. The omitted category is private insurance. These parameters do not directly estimate financial costs, rather they serve to capture financial costs, along with anything affecting the expected utility of a treatment choice that does not operate through the value of outcomes and the relative efficacy of treatments (these aspects are captured by the value weights and the outcomes equations, respectively). Despite this loose definition, I will refer to these as estimates of "costs". The cost coefficients in Table 2.8 are interpreted as the costs of CABG and PCI *relative* to costs of OMT. The first column of estimates are for CABG, the second are for PCI. The large positive coefficient on the constant for CABG (1.5178) reflects the procedure's high relative cost. There are several possible sources for this high cost. First, less generous reimbursement rates may make CABG less profitable. In addition, CABG requires more capital, labor, and time to perform. This is costly in itself,

but also makes it more likely that a facility will be constrained in how many CABGs they are able to perform in a given period of time. Interestingly, the cost coefficient on PCI is negative (-1.972). When there is no advantage in relative efficacy of either PCI or OMT, PCI is preferred. There are several potential explanations for this. The reimbursement rates for PCI may be generous so that the most profitable AMI treatment is PCI. In addition, Lin et al. [2007] provide survey evidence that in some cases physicians may perform PCI out of fear of regret for "not doing everything they could" were the patient to die. They also cite concerns over litigation as motivating PCI usage in some cases. This result suggests that providers may be "over-using" PCI, at least relative to its affect on patient outcomes. Rows two through four give the coefficients on the insurance indicators. These parameters pick-up any differences in uncovered charges or other types of costs that differ across insurance types. Positive coefficients on both Medicare and Medicaid suggest that CABG and PCI are relatively less attractive to providers for patients covered by these government plans than for patients covered by private plans. Without more detailed data on costs, I am unable to determine the underlying factors driving this difference.

Table 2.8: Estimation Results: Treatment "Costs" (c_s)

	(1) (CABG - OMT)		(2) (PCI - OMT)	
	Est	SE	Est	SE
Constant	1.5178	0.2194	-1.972	0.2063
Medicare	0.4256	0.0507	0.4323	0.0431
Medicaid	0.3633	0.0717	0.6302	0.062
Other Pay	-0.0964	0.0773	0.1766	0.0648
N= 47,439				

Note: Estimation results are for the full model and include unobserved patient heterogeneity. Omitted category is private insurance.

To see how well the model does in describing the observed treatment choices and health outcomes, I revisit the age-profile seen in Figure 2.2. Age enters the model in two ways. First, a flexible cubic polynomial is included in the outcome functions to allow the efficacy of each treatment to depend on the age of the patient. Given this, we would expect the model to track the age-profile for the outcomes fairly well, which is what I find. However, how well can the model capture the age variation in treatment choices? Age enters the model in a second way – through the utility weights for health outcomes. Specifically, the value of health outcomes is a linear function of age. As discussed above, the value of preventing death and readmission is greater for younger patients.

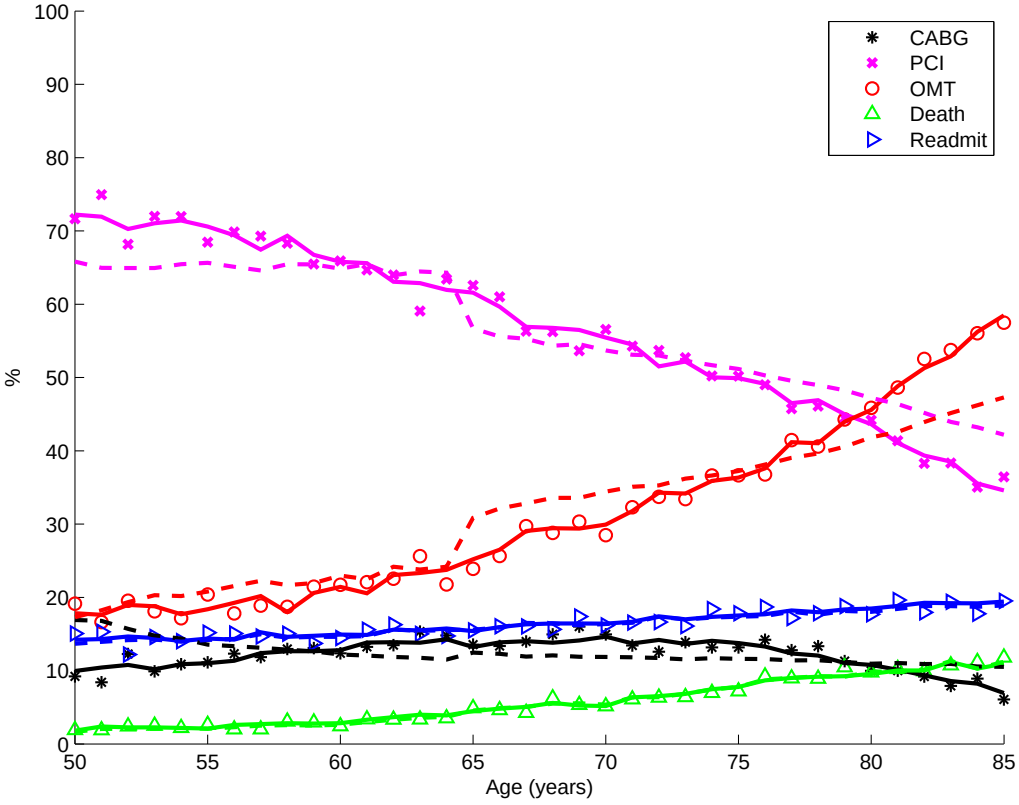
The linear specification gives limited flexibility for the model to capture the decline in PCI, increase in OMT, and inverse-U shape in CABG as patients get older. The model does a relatively good job in capturing the age-profile of treatment choices. Including unobserved heterogeneity significantly improves the model's ability in this regard. Figure 2.3 demonstrates this result. The markers represent the observed percent of patients of a given age receiving each treatment and experiencing each health outcome. This is identical to what is in Figure 2.2. The solid lines are simulated treatment choices and health outcomes from the estimated parameters of the full model. The dashed lines are simulated treatments and outcomes using parameter estimates from a two-step estimation approach that does not allow for unobserved patient heterogeneity to affect the relative efficacy of treatments options in preventing death and readmission[37] The full model, which includes unobserved heterogeneity, improves the model's ability to describe the joint variation in age, treatments, and outcomes. For example, the two-step model does not capture the inverse-U shape in CABG rates, predicting a monotonic decline in CABG rates as patients age. The full model does much better in tracking the age-profile of CABG rates.

2.6.2 Unobserved Heterogeneity: Estimates and Characterization

In this section I provide the estimates for the distribution of unobserved heterogeneity. As discussed in the previous section, the preferred specification for the heterogeneity is a point-mass distribution with four points of support. The distribution is characterized by the four location parameters, α_m, the probabilities associated with each mass point, π_m, and the loading factors, ψ_1 and ψ_2 that capture differences in how the heterogeneity affects the efficacy of CABG and PCI relative to OMT. The estimates are provided in Table 2.9. These estimates are somewhat difficult to interpret on their own, so I provide an intuitive characterization. First, there are a few important observations. The loading factors, ψ_1 and ψ_2 enter the outcome equations with opposite signs. This suggests that unobserved factors generally makes one of CABG or PCI more efficacious in preventing death and readmission than would be predicted from patient observables and makes the other less effective. Whether for a given patient it is CABG or PCI that is improved depends on the sign of α. That ψ_2 is positive indicates that the heterogeneity influences the efficacy of PCI and OMT in the same direction. Intuitively, if a patient is a better fit for PCI than expected from observables,

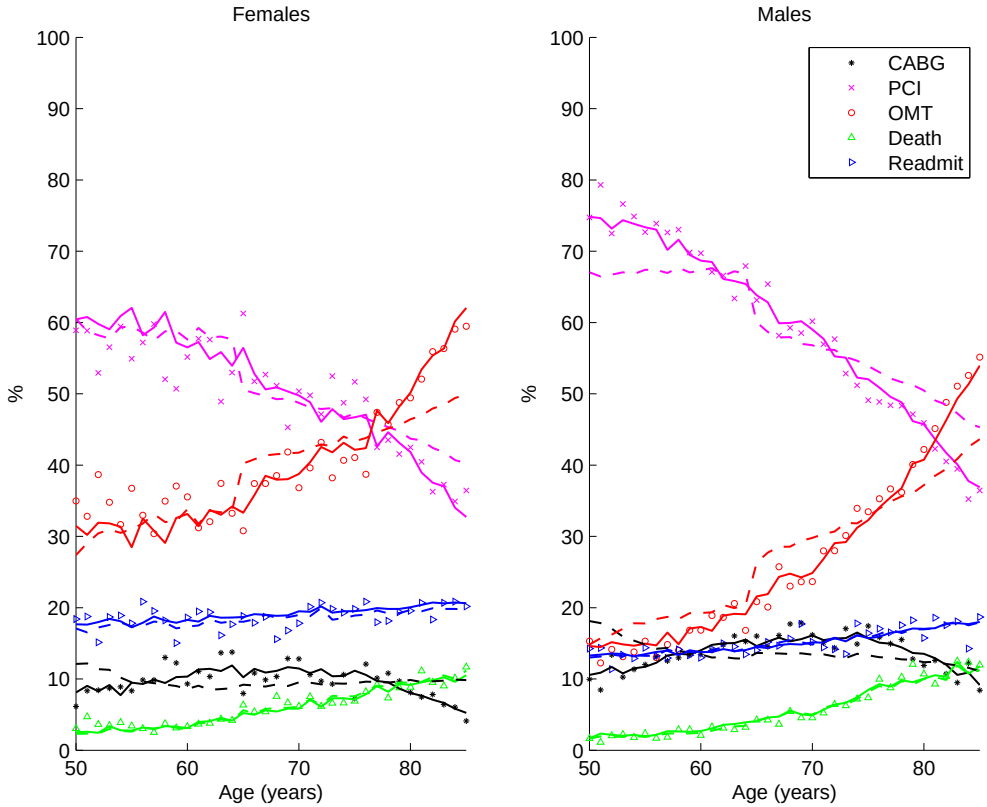
[37]The two-step approach is performed as follows. First, estimate the six outcome equations independently to get estimates of β_s^d and β_s^r. Using these estimates, generate predictions for each patient of p_s^d and p_s^r $\forall s$. Second, insert these predictions in the expected utility equation and estimate $\hat{u}^d$, $\hat{u}^r$, and $\hat{c}_s$.

Figure 2.3: Age Profile, Observed and Simulated: Treatments and Outcomes



Note: Observed treatment and health outcome rates are represented by the markers. Solid lines are simulated treatment and outcomes for the full sample generated using the parameter estimates of the full model, including patient heterogeneity. The dashed lines are simulated treatment and outcomes for the full sample generated from parameter estimates using a two step estimation procedure which does not include heterogeneity. The procedure first estimates outcome probabilities and then inserts predicted outcome probabilities before estimating the treatment choice equation.

Figure 2.4: Age Profile, Observed and Simulated, by gender: Treatments and Outcomes



Note: Observed treatment and health outcome rates are represented by the markers. Solid lines are simulated treatment and outcomes for the full sample generated using the parameter estimates of the full model, including patient heterogeneity. The dashed lines are simulated treatment and outcomes for the full sample generated from parameter estimates using a two step estimation procedure which does not include heterogeneity. The procedure first estimates outcome probabilities and then inserts predicted outcome probabilities before estimating the treatment choice equation.

that patient is also a better fit for OMT. An alternative intuition is that the unobserved factors that make CABG more or less effective are different than those for PCI or OMT. Given the invasive, surgical nature of CABG, this result is not surprising.

Table 2.9: Full Model: Heterogeneity Estimates

Mass Points	EST	SE	π_m
α_1	0	-	0.6561
α_2	-0.1046	0.0411	0.1753
α_3	-0.2504	0.109	0.0532
α_4	-0.0357	0.0227	0.1154
Loading Factors			
ψ_1	-3.652	2.4999	
ψ_2	3.641	0.9403	
N= 47,439			

Once I have obtained estimates of the heterogeneity parameters, I use them to characterize the role of information asymmetries in the treatment assignment process. Each mass point can be thought of, roughly, as a separate unobserved factor, or "type", that affects the efficacy of treatments. The location of the mass-point, α_m, and the loading factors, ψ_1 and ψ_2 describe how the outcomes for that "type" differ from that predicted from observables. The estimates of π_m provide the population weights for each of these "types". Table 2.10 provides an intuitive description for each mass point, or "type". Type_1 has the highest probability weight, 0.656 and is the best fit for CABG and worst fit for PCI (in terms of predicted health outcomes). Type_2 is associated with reduced efficacy of CABG and so patients are a better fit for PCI and OMT. Type_3 is the most extreme, but also associated with the lowest probability weight. Type_3 is associated with extremely effective PCI. A hypothetical patient who was entirely characterized by Type_3 unobservables would almost always receive PCI. Finally, Type_4 can be seen as a middle ground, with CABG efficacy between that of Type_1 and Type_2.

Each patient in the sample can be thought of as having a weighted average of the four unobserved types. The estimates for π_m give the population weights for the types. However, for each patient, we know more about their mix of unobserved types. I use information from the likelihood function and a simple application of Bayes' rule to estimate the posterior distribution of the mass-points. Versions of this procedure have been used to estimate the posterior distribution of random effects in the context of measuring hospital-level adjusted mortality rates (Thomas et al. [1994]) and physician-level quality indices (Johnson [2009]).

Table 2.10: Full Model: "Types" of Heterogeneity

$Type_m$	**Description**	π_m
$Type_1$	HigherCABG/LowPCI	0.6561
$Type_2$	PCI or OMT	0.1753
$Type_3$	Excellent PCI	0.0532
$Type_4$	Some of Each	0.1154

Card and Hyslop [2005] use a similar approach in their paper studying subsidies and welfare-leavers. TenHave and Localio [2004] describes an empirical Bayes' approach for general mixed effects logistic models.

I back out the posterior distribution of the mass points for each patient, given the marginal distribution of the heterogeneity and the likelihood evaluated at each point of support. The calculation to obtain the posterior distribution is straight forward:

$$\pi_{m,i} = \frac{\pi_m P_i(Y_i|\alpha_m)}{\sum_{n=1}^{M} \pi_n P_i(Y_i|\alpha_n)}.$$

The $\pi_{m,i}$ are patient-specific weights for each unobserved type. I next use these weights to explore how treatment assignment and health outcomes are related to unobserved heterogeneity. To do this, I use the posterior distribution of the mass points to construct weighted averages of outcomes by patient type. Specifically, for an outcome y I construct $\bar{y}_m$ in the following way

$$\bar{y}_m = \frac{\sum_{i=1}^{N} \pi_{im} y_i}{\sum_{i=1}^{N} \pi_{im}}$$

The resulting $\bar{y}_m$ give a sense of the extent to which the observed treatment assignments and health outcomes are associated with each "type" of heterogeneity.

The top panel of Table 2.11 gives the percent of each type that is associated with each treatment and outcome. In other words, if a patient was 100 percent Type_1 he would receive CABG 17.9% of the time and would have an *ex ante* probability of death of 6.83%. On the other hand, a patient entirely Type_3 would receive PCI 98.1% of the time and would neither die or need readmission 92.58% of the time. Comparing each column to the last column gives a sense for how different the outcomes are for a given type as compared with the sample averages.

Table 2.11: Outcomes Weighted by Heterogeneity "Type"

	% of Type				
	Type1	**Type2**	**Type3**	**Type4**	**Total**
CABG	17.9	0.1	0.0	3.9	12.2
PCI	43.2	85.9	98.1	63.7	56.0
OMT	38.9	14.0	1.9	32.4	31.8
Death	6.83	3.57	1.11	5.86	5.84
Readmission	17.69	13.88	6.32	17.42	16.39
Neither	75.48	82.55	92.58	76.72	77.77

	% of Treatment/Outcome				
	Type1	**Type2**	**Type3**	**Type4**	**Total**
CABG	96.2	0.2	0.0	3.6	100.0
PCI	50.6	26.9	9.3	13.1	100.0
OMT	80.2	7.7	0.3	11.8	100.0
Death	76.68	10.72	1.01	11.58	100.0
Readmission	70.82	14.85	2.05	12.27	100.0
Neither	63.66	18.62	6.33	11.39	100.0
π_m	65.60	17.54	5.32	11.55	100.0

Note: The top panel gives the percent of each "type" associated with each treatment and outcome. The bottom panel gives the percent of each treatment and outcome that is associated with each "type". Values are weighted by the posterior distribution of the unobserved heterogeneity. The probability weights, $\pi_{m,i}$, are computed using an application of Bayes' rule, the marginal distribution of the unobserved heterogeneity, and the likelihood evaluated at each point of support. Specifically, $\pi_{m,i} = \pi_m P_i(Y_i|\alpha_m) / \sum_{n=1}^{M} \pi_n P_i(Y_i|\alpha_n)$.

The bottom panel of Table 2.11 gives the percentage of each outcome that is associated with each type. Here the relevant comparison is with the bottom row, which gives the sample weight for that type. Type_1 is associated with 96.2% of CABG procedures, rather than the sample weight of 65.60%. Type_1 are also associated with relatively more deaths and readmissions (76.68% and 70.82%, respectively). Type_3 patients would almost always receive PCI, and so accounts for 9.3% of the total PCI procedures, which is almost twice what would be predicted from its sample weight of 5.32%. The message from Table 2.11 is that the estimated unobserved factors are strongly associated with treatment choices and patient outcomes. This is consistent with the visual evidence of Figure 2.3.

I also use the posterior distribution to examine whether unobserved heterogeneity is related to patient observables that were not included in the model. For example, the patient's median zip code income quartile was not included in the model. We might wonder whether unobserved heterogeneity estimates are picking up factors related to the income status of a patient that affect the efficacy of treatment. To explore this, I again construct "type-weighted" averages, but this time for patient characteristics that were not included in the death and readmission equations. Differences between the type-weighted average and the sample average gives a sense if the heterogeneity is picking up factors related to the characteristics that are unobserved and important in treatment efficacy and allocation. Results are presented in Table 2.12. Differences across types are very small for almost all patient characteristics, suggesting that heterogeneity is not strongly related to income, rural/urban status, insurance type, and hospital ownership type.

2.6.3 Treatment Effects

Implicit in the model are estimates of relative treatment effects. For each patient, the outcome equations provide probabilities of death and readmission for each treatment options. These probabilities are used by providers when making treatment allocations and are estimates of patient-specific treatment efficacy. The ability to generate counterfactual treatment effects is a distinct advantage of the model. Table 2.13 gives the mean estimated probability of death and readmission for each treatment option, grouped by observed treatment. That is, column (1) gives the treatment that was actually assigned to the patient, columns (2)-(4) give the average predicted probability of death if the treatment indicated in the column header had been chosen, columns (5)-(7) give the average predicted probability of readmission if the treatment indicated in the column header had been chosen.

The differences in columns (2)-(4) and (5)-(7) give the relative efficacy of treatments on death and readmission, respectively. For example, patients who received PCI (row 2) had a mortality rate of 2.9%. If these patients had instead

Table 2.12: Non-model Characteristics Weighted by Heterogeneity "Type"

	All	**Type1**	**Type2**	**Type3**	**Type4**
Med.Zip Income: 1st qtr	0.238	0.241	0.231	0.228	0.235
Med.Zip Income: 2nd qtr	0.253	0.256	0.246	0.242	0.251
Med.Zip Income: 3rd qtr	0.252	0.251	0.255	0.257	0.253
Med.Zip Income: 4th qtr	0.234	0.229	0.247	0.251	0.238
Missing Income	0.023	0.023	0.022	0.022	0.023
Large Metro	0.724	0.721	0.729	0.727	0.728
Small Metro	0.241	0.243	0.237	0.239	0.238
Non-Metro	0.019	0.020	0.018	0.019	0.018
Rural	0.011	0.011	0.011	0.012	0.011
Missing Urban/Rural	0.005	0.005	0.005	0.004	0.005
Medicare	0.560	0.560	0.561	0.560	0.560
Medicaid	0.071	0.071	0.070	0.068	0.071
Other Insurance	0.069	0.069	0.069	0.070	0.069
Avg. Avail. Beds	349.744	349.461	350.285	350.776	350.053
Teaching Hospital	0.134	0.132	0.138	0.140	0.135
Government	0.078	0.076	0.082	0.081	0.080
For-Profit	0.155	0.154	0.156	0.157	0.155
Not For-Profit	0.742	0.745	0.737	0.735	0.741
Missing Hospital Type	0.025	0.025	0.026	0.026	0.025

Note: Each column reports patient characteristics weighted by the posterior distribution of the unobserved heterogeneity. These probability weights, $\pi_{m,i}$, are computed using an application of Bayes' rule, the marginal distribution of the unobserved heterogeneity, and the likelihood evaluated at each point of support. Specifically, $\pi_{m,i} = \pi_m P_i(Y_i|\alpha_m) / \sum_{n=1}^{M} \pi_n P_i(Y_i|\alpha_n)$.

Note: The reader may note that this same exercise can be performed for the patient characteristics included in the estimation of the death and readmission functions. As one would expect, these variables are orthogonal to the heterogeneity.

Table 2.13: Predicted Treatment Efficacy, by Observed Treatment

	Counterfactual Probabilities					
	In-hospital Death			Readmission		
(1)	(2)	(3)	(4)	(5)	(6)	(7)
Observed Treatment	CABG	PCI	OMT	CABG	PCI	OMT
CABG	0.053	0.051	0.097	0.154	0.249	0.188
PCI	0.085	0.029	0.093	0.177	0.159	0.148
OMT	0.109	0.066	0.113	0.191	0.264	0.181
Full Sample	0.089	0.044	0.100	0.179	0.203	0.163

been given CABG or OMT the model predicts a mortality rate of 8.5% and 9.3%, respectively. Thus, these patients enjoyed a reduction of 5.6% or 6.4% in probability of death as a result of the treatment allocation decision. Note the trade-off between death and readmission. The model suggests that patients who were given CABG would have had similar in-hospital death risk if given PCI (5.3% versus 5.1%), but a dramatically increased risk of readmission. The results presented in Table 2.13 can be used in this way to examine how observed treatment allocations are related to trade-offs between mortality and readmission.

2.6.4 The Gender Gap: Efficacy or Equity?

One of the most important advantages of the model proposed in this project is the natural framework it provides for studying differences in treatment utilization and health outcomes across different patient demographic groups. As seen earlier in the chapter, there are significant differences between men and women in treatment rates and health outcomes. Women receive far fewer CABG and PCI and suffer worse health outcomes. What are the underlying causes of these differences?

Using an Oaxaca-style decompositoin I estimate the fraction of the gender disparities that are explained by differences in observable characteristics between men and women which are related to the efficacy of AMI treatments. I construct weights in the style of DiNardo et al. [1996] using the predicted probabilities from a logit regression of all observable patient characteristics that are used in the choice model on whether the patient is female[38]. For all the men in the sample, I generate ten "replicants" and then simulate treatment choices and health outcomes using the parameter values from the full model, including the distribution of unobserved heterogeneity. I then apply the weights to the simulated outcomes. The simulated, weighted treatment and health outcome rates provide the approximate treatment utilization and health outcomes that would occur for men if they were treated like men, but had the distribution of observable characteristics of women. To the extent that the distribution of simulated treatments and outcomes match that observed for women, gender disparities are driven by treatment efficacy.

The results of this decomposition are reported in Table 2.14. A substantial fraction of the gender differences are explained by differences in the relative efficacy of treatments for female patients: 90% for PCI, 63.5% for OMT, and 65.3% for avoiding both death and readmission. The explained fraction is smaller for readmission rates (31.1%), although non-trivial. The decomposition does not help to explain gender differences in CABG rates and in-hospital mortality. The

[38]Specifically, the weights are $\frac{\hat{p}_i}{1-\hat{p}_i}$ where $\hat{p}_i$ is the predicted value from the logit regression on whether patient i is female

results suggest that a substantial portion of the gender disparities documented in the literature are related to differences between men and women in the efficacy of AMI treatments. Note that one limitation of this method is that I do not capture differences between men and women in the relative effect on treatment efficacy of unobserved factors. For example, body weight (unobserved in my data) could negatively affect the efficacy of surgical treatments more for women than for men. The effect of body weight differences on the gender gap would errantly be "unexplained" by the decomposition.

Table 2.14: Decomposition of Gender Gap: Relative Efficacy

	Raw Data			**Decomposition**	
	(1)	(2)	(3)	(4)	(5)
	Women	Men	Gap	Men_w	% Explained
CABG	0.094	0.138	-0.044	0.143	-12.0
PCI	0.477	0.606	-0.129	0.491	89.3
OMT	0.429	0.256	0.173	0.366	63.5
Death	0.069	0.052	0.017	0.077	146.1
Readmission	0.190	0.150	0.040	0.162	31.1
Neither	0.741	0.798	-0.057	0.761	65.3

Note: This table reports a decomposition of the differences between women and men in treatment utilization and health outcomes. Columns (1) and (2) give the observed proportion of women and men, respectively, receiving each treatment and experiencing each outcome. Column (3) is the difference in columns (1) and (2), the gender gap. Column (4) gives treatments and outcomes re-weighted to balance observable characteristics across genders. Column (5) reports the percent of the gender gap that is explained by the decomposition.

The decomposition provided evidence that some, but not all, of the gender disparities in treatment utilization and health outcomes in AMI patients can be attributed to differences in the relative efficacy of treatments for men and women. I can use my model to explore equity avenues for explaining the remainder of the gender gap. To do this, allow the utility associated with health outcomes, u^d and u^r, to be functions of demographics, in this case gender. The value of outcomes would take the following form

$$\begin{aligned} u^d(Z) &= u_0^d + u_1^d \cdot (age - 65) + u_{female}^d * female \\ u^r(Z) &= u_0^r + u_1^r \cdot (age - 65) + u_{female}^r * female. \end{aligned}$$

This added flexibility in the utility function specifically targets issues of equity. If u_{female}^d is significant, it suggests that treatment decisions are, at least in part, driven by variation in the values over outcomes across gender, not just by treatment efficacy. A large difference across genders in the values placed on health outcomes by providers may imply inequitable provision of medical care.

2.7 Conclusion

In this chapter I specified a simple model of treatment decisions in which health care providers derive value from patient outcomes and allocate treatments in order to maximize utility over expected patient outcomes, net of treatment costs. Since treatments directly influence outcomes, decisions over treatments reflect the values providers place on different outcomes. In this way, the model estimated the implicit weight providers place on outcomes that rationalize actual treatment allocations and observed patient outcomes. The structure of the model provides distinct roles for factors which influence treatment assignment through relative treatment efficacy and for those which influence treatment assignment through other channels, such as equity in care and costs of treatments.

The model was applied to the specific context of the treatment of heart attack patients in the state of California from 2005-2007. The model did relatively well in describing the joint variation in observed treatment allocations and patient outcomes. The inclusion of unobserved patient heterogeneity in relative treatment efficacy, which is modeled using a point-mass distribution, significantly improved the performance of the model by allowing for selection into treatments based on information known to the health care provider, but not to the researcher. I suggested a simple method for characterizing the unobserved heterogeneity using an application of Bayes' rule and provided informal evidence that unobserved information relevant to treatment assignment is related to treatment efficacy, and hence patient outcomes, but is not driven by patient demographics not directly related to treatment efficacy such as income, insurance status, and urban/rural status.

Estimation results showed that treatment decisions reflect significant negative weight on adverse patient outcomes, such as in-hospital mortality and unscheduled readmission. The emphasis on preventing adverse outcomes is greater for younger patients. I used the model to address the well known disparity in use of intensive treatments for female versus male heart attack patients. An Oaxaca-style decomposition suggested that a substantial fraction of the gender disparities can be explained by differences in relative treatment efficacy for female patients. This method explained approximately 90% of differences in rates of percutaneous coronary intervention, 64% in medical therapy, and 31% in readmission. A substantial gender gap remained unexplained for the predicted use of CABG and in patient death rates. I described how the model will be used to estimate the fraction of the remaining gender gaps attributable to equity-related factors, specifically to differences in the weights providers place on the health outcomes of men and women. Estimation and results for this method are currently in progress. These methods can be applied to other patient demographics, such as race or insurance status.

Chapter 3

Medicaid Enrollment and Welfare Reform: An Examination of Health Insurance Dynamics

3.1 Introduction

In 1996 the Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA) brought significant changes to the cash-assistance welfare program in the United States. With the introduction of Temporary Assistance for Needy Families (TANF) to replace Aid to Families with Dependent Children (AFDC) new restrictions were placed on the receipt of cash-assistance and new incentives were introduced that encouraged individuals on welfare to find employment. An indirect result of this reform was a change in the link between cash-assistance and government provided health insurance, specifically Medicaid. While the direct administrative link between cash welfare and Medicaid eligibility had been gradually reduced during the late 1980's and early 1990s, it was still the case that many families receiving cash-assistance were also eligible for and enrolled in Medicaid.

In this chapter, I use welfare reform under the PRWORA as a structural shift in employment and health insurance environments. I study how welfare reform may have inadvertently changed the barriers to and costs of enrollment in Medicaid and attempt to disaggregate the effect of reform on health insurance from that on employment and welfare participation using a more structured approach to estimation than currently exists in the literature. I use a simple framework to estimate the effects of welfare reform and changes to enrollment costs on the health insurance status of the affected subpopulation. Because insurance and employment status are intimately related, the structure of the model allows for a disentanglement of insurance and employment dynamics. Estimates suggest

that implementation of TANF significantly altered both short run (four months) and longer run (up to 10 years) insurance and employment dynamics for single mothers. At least a portion of the changes can be attributed to shifts in the costs and barriers faced when enrolling in Medicaid.

The reforms were not intended to directly affect the accessibility of health insurance for this population; individuals who would have qualified for AFDC were to maintain their Medicaid eligibility. It is possible, however, that the reform increased confusion about Medicaid eligibility and the enrollment process. While receiving welfare checks, individuals had a direct financial incentive (or a need) to visit a social worker either to pick up the check or to take care of paperwork to maintain welfare eligibility. This frequent encounter with a social worker may have served to reduce barriers to enrollment in Medicaid. Reduced financial incentive to see social workers after TANF along with potential increase in confusion among the social workers regarding Medicaid eligibility may have reduced Medicaid enrollment post-TANF. In part, this chapter focuses on these costs to enrollment in Medicaid and how they affect the insurance status of the Medicaid-eligible population.

To help understand the importance of enrollment costs we would like to identify a scenario in which the enrollment cost of Medicaid changes while the (perceived) health benefits do not. The implementation of TANF provided such an environment. It is important to be clear about what is meant by enrollment costs in this project. For private insurance plans, especially those provided by employers, it is not unreasonable to think enrollment costs are small, if not negligible. Employers and private insurance companies provide documentation and support services for potential enrollees that reduce the burden of enrolling and maintaining insurance coverage in a private plan. Employers have Human Resource departments devoted entirely to helping employers with benefits and insurance decisions. In addition, most private plans automatically re-enroll an individual each year with minimal to no effort required. However, the same may not be true of Medicaid enrollment. While the monetary cost of enrollment in Medicaid is minimal, there may be many other barriers faced by individuals who may wish to enroll in Medicaid. Within the Medicaid-eligible population there may exist language barriers, documentation requirements, and informational asymmetries that make enrollment an arduous process. This may include straightforward costs such as time requirements and travel costs, but also includes more abstract notions stemming from a lack of information about the enrollment process or the availability of Medicaid services. In addition, in order to remain enrolled from period to period, there may be some administrative costs (paperwork, etc). Many individuals who are eligible for Medicaid may not even be aware of their eligibility. While such barriers may at first seem trivial when compared with the benefits of health insurance, there is evidence in the literature, discussed in detail below,

that efforts to reduce language and informational barriers increase enrollment in Medicaid (Aizer [2003b] and Aizer [2003a]).

Individuals who are eligible for Medicaid and are uninsured are able to enroll in Medicaid at the time of a hospitalization or trip to the emergency room. As such, the enrollment decision we are most concerned with for Medicaid eligible individuals is whether to enroll in Medicaid *before* the onset of a health episode. This *ex ante* enrollment increases access to preventative care and information about health services that would otherwise be unknown to the individual. In addition, it may also increase the speed at which care can be provided in time-sensitive health episodes.

There is no clear consensus in the literature as to the preventative health benefits of enrollment in Medicaid. It may be that enrollment encourages use of services that are expensive with minimal long term health benefits. On the other hand, enrollment in Medicaid may provide easier access to effective preventative care as well as providing individuals with more information on the availability of preventative services. In this chapter I take an agnostic stance as to the health value of enrollment in Medicaid. Rather, I focus on the role of administrative barriers and enrollment costs that drive enrollment in Medicaid, choices to remain uninsured, and decisions to purchase private insurance. This is not to say that as researchers we shouldn't care about the health benefits of enrollment. From a policy perspective it is important to understand whether the low take-up rates[1] observed in the literature are a result of low actual (or perceived) health benefits of enrollment, or a result of high barriers to enrollment. If in fact enrollment costs are largely responsible for low take-up rate, then policy makers can act accordingly to better encourage enrollment. If, on the other hand, the health benefits of enrollment are low, then we need not pursue (potentially expensive) policies aiming to reduce enrollment costs.

The project contributes to and bridges two lines of existing literature, explained briefly here and in more detail in Section 3.3. The first addresses Medicaid enrollment, coverage rates, and responses to changes in eligibility requirements. Studies that focus on changes in eligibility document the low take-up rate for newly eligible children and pregnant women.[2] The issue of low take-up rates is supplemented with studies that suggest that a portion of the low enrollment in Medicaid (and other social assistance programs) may be a result of high barriers to enrollment. These enrollment costs may include language barriers, lack of information, overly complex application processes, or negative social stigma associated with participation.[3]

[1] See Card and Shore-Sheppard [2004] for discussion of low take-up rates for Medicaid expansion.

[2] See Cawley et al. [2006] and Currie and Gruber [1996], respectively

[3] For example, a series of studies by Anna Azier Aizer [2003a] and Aizer [2003b] use results

The second line of literature documents the link between cash-assistance welfare programs and Medicaid. Most closely related are studies focusing on the changes in health insurance coverage rates in response to the welfare reforms occurring under the Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA) of 1996. A series of studies provide evidence that welfare reform affected the insurance status of many of the women and children most directly affected by the reforms.[4] This project builds on this evidence and further proposes that welfare reform may have shifted the enrollment barriers individuals faced when making insurance (private insurance, no insurance, or Medicaid) and employment decisions. This shift provides leverage to learn more about how enrollment costs and administrative barriers drive take-up rates and participation rates in socially provided assistance programs.

The rest of the chapter is organized as follows. Section 3.2 provides a very brief background on welfare reform and Medicaid. Section 3.3 reviews in more detail the related literature. Section 3.4 proposes a simple modeling framework for examining health insurance and employment transition dynamics. Section 3.5 describes the data. Section 3.6 provides estimation results including four-month transition probabilities and longer run dynamics. Finally, Section 3.7 concludes.

3.2 Background: Medicaid and Welfare Reform

In order to understand how welfare reform may have changed the health insurance environment of the Medicaid eligible population, it is important to detail the close link between welfare programs and Medicaid and how the nature of this connection was altered by reform to welfare programs in the mid-1990s.

Throughout their histories, Medicaid and cash welfare programs have been intimately related. For a period of time eligibility for Medicaid and cash assistance, or AFDC, were linked directly. Qualification for AFDC resulted in access to Medicaid coverage. However, during the late 1980s and early 1990s the automatic link between the two programs was gradually weakened. Perhaps the most dramatic decoupling of cash assistance and Medicaid occurred in 1996 when AFDC was replaced by Temporary Assistance for Needy Families, or TANF. TANF implemented lifetime constraints on the duration of eligibility for cash assistance and introduced incentives for welfare recipients to leave cash-assistance and enter the workforce. In addition, the federal regulation gave state governments more autonomy over cash assistance programs which resulted in variation in timing of

from a California initiative to suggest that language barriers, lack of information, and other impediments to enrollment play a significant role in Medicaid enrollment rates.

[4]See, for example, Kaestner and Kaushal [2003], Cawley et al. [2006], and Ham and Shore-Sheppard [2005].

reforms as well as in eligibility requirements and administrative protocols.

During the early 1990s, many families still received Medicaid coverage through their enrollment in the AFDC program. Although Medicaid eligibility did not require AFDC participation, many families who qualified for one program qualified for the other. TANF reform threatened to further decouple the link between the two programs, by changing (often reducing) eligibility for cash-assistance. Wary of the close ties between Medicaid and AFDC, policy-makers made some efforts to avoid any disruption by PWRORA and TANF to health insurance coverage of the poor. Language in PWRORA H.R.3734 [1996] make clear that under new law states may tighten their criteria for cash welfare benefits, but that for the purposes of health insurance, individuals who would have qualified for AFDC under the requirements in effect as of July 16, 1996 would remain eligible for Medicaid. In addition, to ease transition from welfare, states were required to provide up to 12 months of transitional Medicaid coverage for individuals who became ineligible due to increased earnings or increased child support.[5] Thus, TANF reforms actively attempted to avoid reductions in access to health insurance, and in particular access to Medicaid, while reducing participation in cash assistance welfare programs. In practice, however, enforceability of the provision of Medicaid was questionable for individuals qualifying under the 1996 AFDC criteria. Likewise, and perhaps even more importantly, welfare reform may have altered the accessibility of Medicaid through indirect channels.

Garrett and Holahan [2000b] provide a clear description of some potential unintended side effects of the PWRORA and TANF reforms:

> "First, at the same time that PRWORA preserved Medicaid eligibility for welfare leavers, it also made Medicaid eligibility rules much more complex. This situation makes it more difficult to understand who is eligible, not only for the former welfare recipients themselves but also for their welfare caseworkers, who are probably their main source of information on Medicaid. Second, state administrative requirements for Medicaid coverage (such as the completion of complicated application forms and in-person interviews during working hours) place difficult burdens on newly working parents. Third, for former welfare recipients who originally acquired Medicaid coverage as part of the application process for cash assistance, having to apply individually for Medicaid may simply be too difficult. Finally, a certain stigma may be attached to Medicaid receipt that could deter some former welfare recipients from reapplying." (Garrett and Holahan [2000b], p. 4)

To motivate the idea that Medicaid enrollment may have remained linked to welfare after PWRORA, consider Figure 3.1 which plots the coverage rates for

[5] One of the goals of PWRORA was to crackdown on fathers deliquent on child support payments. It is conceivable that incomes of single mothers could rise above assistance thresholds with the addition of child support payments.

Figure 3.1: Coverage Rates: Welfare and Health Insurance

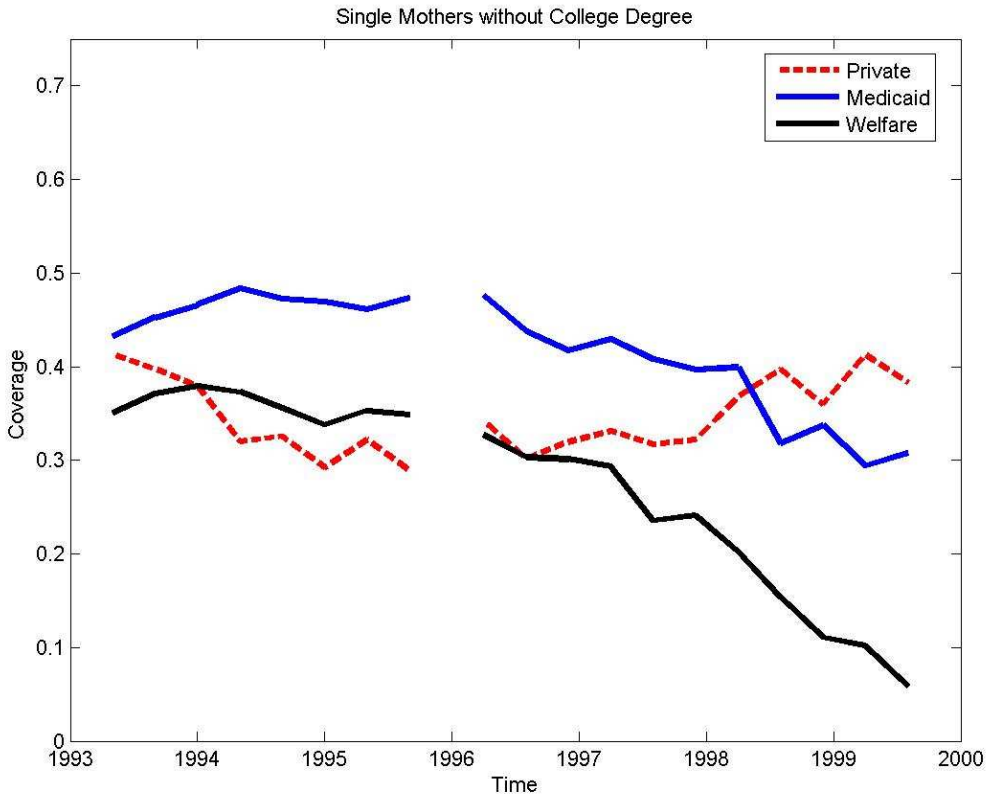
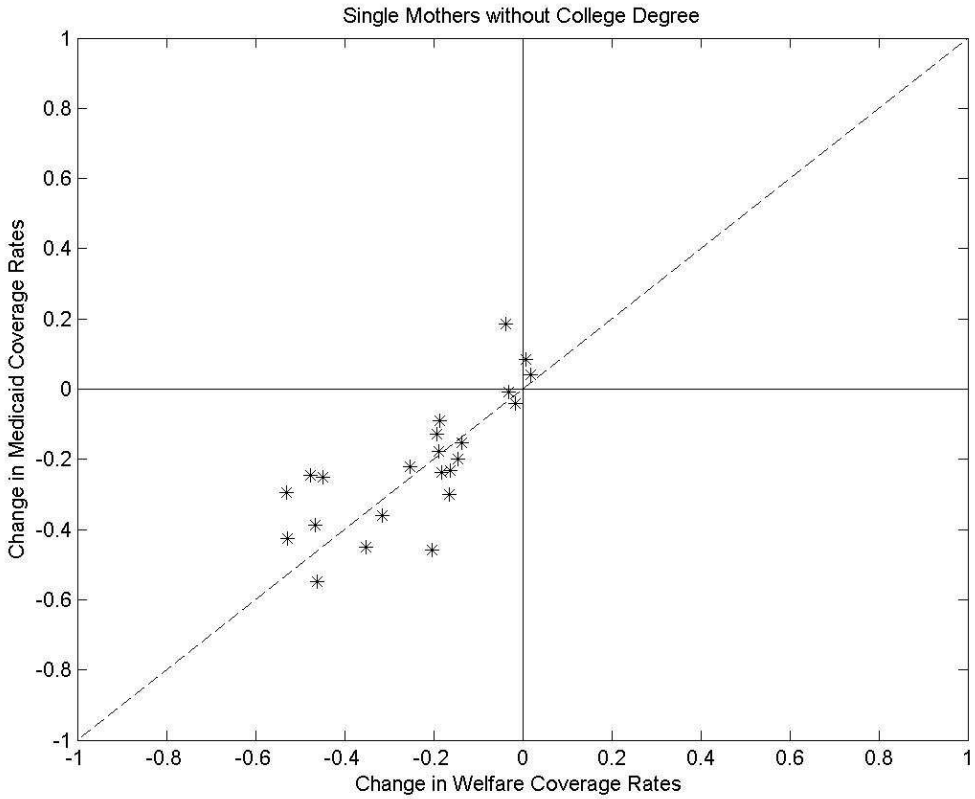


Figure 3.2: State Level Changes in Welfare and Medicaid Coverage Rates: '95 to '98



welfare, Medicaid, and private insurance from 1993 through 1999. The data are from the 1993 and 1996 Survey of Income and Program Participation (SIPP) panels (as described in Section 3.5). The sample includes single mothers without a college degree ages 18 through 44 years. There are obvious declines in both Medicaid and welfare coverage during the post-1996 period. Reductions in Medicaid are not as large as those for welfare. We see some increase in private insurance rates during the second half of the 1990s. The gap in the data between mid-1995 and early 1996 is a result of the lack of overlap between the 1993 and 1996 SIPP panels. This is discussed more in Section 3.5.

The contemporaneous reduction in welfare and Medicaid is even more clear in Figure 3.2, which plots the the state-level changes in welfare and Medicaid coverage between 1995 and 1998 for single mothers without a college degree between

the ages of 18 and 44 years. The data are again from the 1993 and 1996 SIPP panels (as described in Section 3.5). The reported changes are in the coverage rates for welfare and Medicaid for the sample population. Both welfare and Medicaid saw significant reduction over the three year period in almost all states. In addition, changes cluster near the 45 degree line, suggesting close to one-for-one reductions in welfare and Medicaid. The bulk of this project revolves around the link between welfare and Medicaid and the (unintended) effect of welfare reform on the insurance status of single mothers.

3.3 Existing Literature

3.3.1 Medicaid Expansions and the Low Take-up Rate

In their widely cited paper, Currie and Gruber [1996] evaluate the cost and benefits of the Medicaid expansions in the early 1990s. Specifically, they focus on pregnant women and birth outcomes. The authors exploit state-level variation in the magnitude and timing of Medicaid eligibility expansions. They estimate a model using simulated state-level Medicaid policies to show that expanding eligibility of pregnant women resulted in a significant (approximately 8.5%) reduction in infant mortality. They also provide evidence that some increases in Medicaid coverage resulting from eligibility expansions was due to crowding-out of private insurance.

Card and Shore-Sheppard [2004] examine the Omnibus Reconciliation Act (OBRA) of 1989 and OBRA 1990 expansions of Medicaid eligibility and provide insight into relative importance of low marginal take-up rate for Medicaid and the crowdout of private insurance. The authors focus on the discontinuities in Medicaid eligibility for young children that was introduced by the OBRA expansions. Using regression-adjusted difference in difference estimates the authors show that the 1989 expansions resulted in only an 8 percentage point increase in Medicaid coverage and that 1990 expansions had a minimal impact on coverage. In addition, they show that the increase in Medicaid coverage is not explained by crowdout of other types of health insurance. They conclude that the modest impact on overall rates of health insurance coverage of the Medicaid expansions is largely a result of low take-up rate for Medicaid by newly eligible individuals, rather than of crowdout. The paper's scope does not extend to a study of the factors driving the low take-up rate. However, this issue has garnered some attention in the literature.

In a series of two papers, Aizer [2003a] and Aizer [2003b], Anna Aizer examines the role that processing and informational costs may play in the enrollment and take-up rates of Medicaid. Both papers focus on an outreach program implemented in California in mid-1998. Both papers show that language barriers

may play an important role in preventing enrollment in Medicaid by non-English speaking individuals.

3.3.2 The Link Between Medicaid and Welfare

A significant line of literature exists documenting various aspects of the link between cash welfare programs and Medicaid. Research in this area has focused on two main research questions. First, what was the impact on insurance rates of the mid-1990s welfare reform? Second, did welfare reform have significant effects on health care utilization and health status? Most papers studying welfare reform focus on either insurance coverage or on health care utilization and health outcomes.

In an early study of welfare reform and health insurance, Garrett and Holahan [2000a] report descriptive details on the health insurance status of those women who left welfare between 1995 and 1997. The authors use a cross-section of 100,000 US households provided in the 1997 National Survey of America's Families to identify welfare leavers. Of the leavers, 36% were covered under Medicaid, 23% were covered under employer/private insurance while 41% were uninsured.

Kaestner and Kaushal [2003] utilize a more rigorous research design to study the effect of welfare policy on the insurance status of single, low-education mothers. Using data from the Current Population Survey (CPS) from 1992 - 1999 they employ a difference-in-difference (DD) methodology to look at the effect of changes in welfare caseload on insurance status. They are most interested in disentangling the effect on welfare caseload caused by welfare policy from that caused by other factors. Their results suggest that only 30% of the change in insurance status due to decrease in welfare caseload due to welfare policy.

Cawley et al. [2006] employ a similar approach to Kaestner and Kaushal [2003], but do not focus specifically on changes in welfare caseload. Instead, they look at welfare reform more generally and its impact on the health insurance status of a defined target population. They do this for both single mothers and children. Using panel data from Survey of Income Program Participation (SIPP) they identify off of individuals who change insurance status in states that enacted either AFDC waivers or TANF during the sample period. They run separate regressions for various types of health insurance coverage: Medicaid, employer provided private insurance, any private insurance, and any insurance. They find that TANF reduced probability of any coverage for welfare-eligible women by 8.1% and of Medicaid coverage by 6.7%. Some increase (3.4%) in employer provided insurance is reported. While their approach does not examine in detail the factors driving these changes in insurance probabilities, their results are roughly consistent with my initial results concerning changes in insurance dynamics post-welfare reform.

In their paper, Ham and Shore-Sheppard [2005] address the link between Medicaid and Welfare from a different perspective. Rather than looking at effects of welfare reform on Medicaid enrollment, they look at expansions in Medicaid eligibility and how they affect welfare participation and labor market outcomes. The authors use data from the CPS for the years 1988-1996 to show that expansions to Medicaid eligibility had no detectable effect on welfare participation rates. This result will be relevant when we examine in greater detail the effect of the 1996 welfare reform on Medicaid enrollment.

In summary, there is some evidence in the literature that welfare reform decreased enrollment in Medicaid and that some, but not all, of this decrease was offset by increases in the rate of private insurance coverage. Evidence suggesting that welfare reform may have impacted the insurance status of single women and children motivates an important line of literature which seeks to identify impacts of welfare reform on health care utilization and health outcomes. Some evidence exists suggesting that enrollment increases use of preventative services Currie and Gruber [1996]. However, the ultimate health benefits of these preventative services are less understood. Indeed, most work focuses on benefits of Medicaid services for pregnant women. Little is known about the impact on health outcomes of Medicaid for other demographic groups.

Despite limited data availability, some evidence on health outcomes exists in the literature. Bitler et al. [2005] use data from the Behavioral Risk Factor Surveillance System (BRFSS) for 1990 through 2000 to estimate the impact of welfare reform on health care utilization and health outcomes. They employ a difference-in-difference approach and exploit state-level variation in the adoption of AFDC waivers and TANF. They provide evidence that welfare reforms resulted in a reduction in health care utilization and a decrease in single women's access to health care. However, it is possible that welfare reform reduced utilization for those individuals covered by Medicaid, but may have increased the accessibility and utilization for those individuals acquiring private insurance. The authors do not address these potentially offsetting effects.

In another study, Currie and Grogger [2002] study the effects of changing welfare caseloads on prenatal care, infant mortality, and infant birth weight. Using data from all birth certificates for the period 1990-1996 they can identify utilization of prenatal care by the mother. Their results suggest that increases in Medicaid eligibility increased use of prenatal care and that decreasing welfare caseloads decreased use of care. The authors claim that there is evidence that as of 1996 reforms to Medicaid policy had not completely broken the link between enrollment in welfare and Medicaid.

In their paper, Leonard and Mas [2008] examine individuals who were forced off of cash welfare as a result of new welfare participation policies implemented by TANF. They show that "timing-off" of welfare has a negative impact on infant

mortality.

Although they do not focus on welfare reform, Aizer et al. [2007] show that health services provided by Medicaid are important to health outcomes. They provided evidence that policies implemented in California that forced women to switch from traditional Medicaid to a Medicaid Managed Care plan negatively impacted birth outcomes.

3.4 A Simple Model of Insurance and Employment Transitions

This section describes a simple modeling framework for insurance and employment states. In the model the individual faces a menu of up to three insurance choices: private insurance, public insurance (Medicaid), and no insurance. Each insurance option provides health benefits (either immediate or in the future), but may be costly to obtain and maintain (enrollment/administrative costs). In addition, the individual is a member of one of three employment states: employed, on welfare, neither employed nor on welfare. Each of the three states provide a level of monetary income. In each period the individual evaluates the health benefits, income, and costs imposed by each insurance option and employment state and chooses the combination which maximizes utility. In practice, the individual either retains the same type of coverage as the previous period or switches to a different coverage type. Likewise with employment state.

The individual's utility ($U^*_{i,j,m}$) is a function of health status, insurance costs, and income such that utility for individual i choosing insurance option j and employment state m is given by:

$$\begin{aligned} U^*_{i,j,m} &= u(Income_j, Health_m, Costs_{j,m}) \\ Income_j &= g\left(Y_j\right) \\ Health_m &= h(Q_m) \\ Costs_{j,m} &= c(P_m, E_j, E_m) \end{aligned}$$

The income or monetary value of employment state j is given by Y_j. The individual's health is a function of the quality of the insurance plan (Q_m). The costs faced by the individual are a function of the insurance plan's premiums and co-payments for that period (P_m) and any costs associated with obtaining or maintaining insurance option m (E_m) and employment status j (E_j). These will be referred to "enrollment costs". The quality of an insurance plan includes access to services, especially preventative services, as well as the quality of services pro-

vided. Enrollment costs for an insurance plan can include the costs of initiating enrollment if the individual is new to the plan, or costs of retaining coverage if the individual was enrolled in choice j in the previous period and may depend on which insurance state the individual is in to start the period (I_i^t). It may also include any social stigma attached to the type of plan (mostly for Medicaid).

To implement the basic setup, we condition on membership to a specific insurance plan m and employment status j at the beginning of period t. Thus, for individuals with employment/welfare status j and insurance status m in period t ($s_i^t = \{j, m\}$) we can write the utility obtained from employment/insurance option $\{k, n\}$ for period $t+1$ ($s_i^{t+1} = \{k, n\}$) as:

$$\begin{aligned} U^*_{i,k,n}(X_i, s_i^t = \{j, m\}) = \quad & (\gamma_k + \gamma_n) + \gamma_1 Y_k + \gamma_2 E_k + \gamma_3 Q_n + \gamma_4 P_n + ... \\ & + \gamma_5 E_n + X_i \beta_{k,n} + \xi_{i,k,n} \end{aligned}$$

where Y_k is the income or monetary value of employment/welfare status k, Q_n is the quality of the insurance option n, P_n is the cost of insurance option n including premiums and copayments, E_k and E_n are the enrollment costs associated with obtaining or maintaining employment/welfare status k and insurance option n, [6] X_i are individual characteristics that may affect the value placed on each option by the individual and $\xi_{i,k,n}$ are unobserved components affecting utility for employment/insurance state $\{k, n\}$.

In the next period, the individual moves to the employment/insurance state that provides the highest utility. That is,

$$s_i^{t+1} = \{k, n\} \text{ iff } U^*_{i,k,n} \geq U^*_{i,j,m} \ \forall \{j, m\} \neq \{k, n\}.$$

Since we cannot identify utility *levels*, we normalize by the value of remaining in the same state. We have that for an individual initially in state $\{j, m\}$, the value of moving to state $\{k, n\}$ *relative* to remaining in $\{j, m\}$ is given by:

$$\begin{aligned} U_{i,k,n}(X_i, s_i^t = \{j, m\}) = \quad & U^*_{i,k,n} - U^*_{i,j,m} = \\ & (\gamma_k - \gamma_j) + (\gamma_n - \gamma_n) + \gamma_1 (Y_k - Y_j) + ... \\ & + \gamma_2 (Q_n - Q_m) + \gamma_3 (P_n - P_m) + X_i (\beta_{k,n} - \beta_{j,m}) + ... \\ & + \gamma_4 (E_k - E_j) + \gamma_5 (E_n - E_m) + (\xi_{k,n} - \xi_{j,m}) \end{aligned}$$

[6] This may also include the level of employment opportunities, the transparency of the enrollment process and eligibility criteria for social programs, the accessibility of social workers to provide information about assistance programs, the difficulty/complexity of any application process, etc.

or using simplifying notation of the form $A_{b,c} = A_b - A_c$ and suppressing the arguments of $U_{i,k,n}(\cdot)$, we write:

$$\begin{aligned} U_{i,k,n} = \quad & U^*_{i,k,n} - U^*_{i,j,m} = \\ & \gamma_{k,j} + \gamma_{n,m} + \gamma_1 Y_{k,j} + \gamma_2 Q_{n,m} + \gamma_3 P_{n,m} + X_i(\beta_{k,n} - \beta_{j,m}) + ... \\ & + \gamma_4 E_{k,j} + \gamma_5 E_{n,m} + (\xi_{k,n} - \xi_{j,m}) \end{aligned}$$

I then make the following three assumptions:

- (A1) characteristics of employment and insurance states are constant within a regime.
- (A2.1) welfare reform operates through changes in Y_j, E_j, and E_m
- (A2.2) welfare reform does not affect Q_m or P_m

Assumption A1 requires that all aspects of insurance plans and employment/welfare states do not change, except for what changes are induced by welfare reform. For example, the quality of Medicaid does not change over time *within* the pre-reform regime.

Assumptions A2.1 and A2.2 state that the changes induced by welfare reform only operate through changes to the monetary value of employment states, Y_j),[7] and the enrollment costs of employment, welfare, and insurance plans (E_j and E_m). This allows for Q, and P to affect the relative values of employment/insurance states, but is does *not* allow for them to affect *changes* in the relative values as a result of welfare reform.

Under these three assumptions and writing post-reform variables with a superscripted w we have the following relationships:

$$\begin{aligned} Q^w_m &= Q_m \\ P^w_m &= P_m \\ Y^w_j &= Y_j + y_j \\ E^w_j &= E_j + e_j \\ E^w_m &= E_m + e_m, \end{aligned}$$

where the signs of the e_j and e_m depend on whether welfare reform increases or decreases the enrollment costs of that state. Likewise, the sign of y_j depends on if reform increased or decreased the value of that employment/welfare state.[8] Al-

[7] Perhaps through increased work incentives and decreased welfare benefits.

[8] Given the goals of PRWORA it may be reasonable to assume $Y^w > Y$ for employment and $Y^w < Y$ for welfare.

though not necessary, welfare reform operate through the additive shifts imposed by e_j and e_m.

Observing separately Q_m, P_m, Y_j, E_j, and E_m may not be possible. However, we can combine these terms and rewrite $U_{i,k,n}(X_i, s_i^t = \{j, m\})$ as:

$$U_{i,k,n} = U^*_{i,k,n} - U^*_{i,j,m} = NV_{k,n} + X_i(\beta_{k,n} - \beta_{j,m}) + (\xi_{i,k,n} - \xi_{i,j,m}),$$
$$\text{where } NV_{k,n} = \gamma_{k,j} + \gamma_{n,m} + \gamma_1 Y_{k,j} + \gamma_2 Q_{n,m} + \gamma_3 P_{n,m} + \gamma_4 E_{k,j} + \gamma_5 E_{n,m}$$

where $NV_{k,n}$ represents the value of the employment/insurance option, net of price and enrollment costs. Under assumption A2.2 price and quality are not affected by welfare reform (conditional on observables) so that the only difference in $NV_{k,n}$ across periods is through changes in monetary values of employment/welfare options (Y_j) and in enrollment costs ($E^w_{k,j}$ and $E^w_{n,m}$). More formally, the expected difference in utility pre and post-reform is

$$E[U^w_{i,k,n} - U_{i,k,n}] = NV^w_{k,n} - NV_{k,n} = \gamma_1 y_{k,j} + \gamma_4 e_{k,j} + \gamma_5 e_{n,m}.$$

Knowing this, we can easily combine the pre and post welfare reform utilities into a single equation, using a dummy variable to indicate post-reform:

$$U_{i,k,n} = NV_{k,n} + \mathbf{1}^w[\gamma_1 y_{k,j} + \gamma_4 e_{k,j}] + \mathbf{1}^w \gamma_5 e_{n,m} + X_i(\beta_{k,n} - \beta_{j,m}) + (\xi_{i,k,n} - \xi_{i,j,m})$$

where $\mathbf{1}^w$ is an indicator equal to 1 in the post welfare reform period, γ_1 and γ_4 capture the jump in relative value of employment option k, such as work incentives or reduced welfare benefits, and γ_5 the jump in relative value of insurance option n induced by reforms. Ideally γ_1, γ_4, and γ_5 would be separately identified. It is γ_5 which captures the shifts in enrollment costs to Medicaid which are most relevant to policy initiatives focused on Medicaid enrollment. In the model presented in this chapter, γ_1, γ_4, and γ_5 are not separately identified and thus changes in enrollment costs of insurance options and changes in monetary values and enrollment costs of employment and welfare are confounded.

Finally, we write utility as:

$$\begin{aligned} U_{i,k,n} &= \delta_{k,n} + (\xi_{i,k,n} - \xi_{i,j,m}) \\ \delta_{k,n} &= NV_{k,n} + \mathbf{1}^w[\gamma_1 y_{k,j} + \gamma_4 e_{k,j}] + \mathbf{1}^w \gamma_5 e_{n,m} + X_i(\beta_{k,n} - \beta_{j,m}) \end{aligned}$$

Note that $\delta_{i,k,n}$ captures the mean value of option $\{k, n\}$ *relative* to the mean value of option $\{j, m\}$ and that $\delta_{i,j,m} = 0$.

With the additional standard assumption that the $\xi_{i,j,m}$ are distributed EV1, we can write the probabilities of each choice in the familiar conditional logit form. We have that the probability of membership for individual i in employ-

ment/insurance state $\{k, n\}$ in the next period conditional on beginning the period in state $\{j, m\}$ is given by:

$$Pr(s_i^{t+1} = \{k, n\} | X_i, s_i^t = \{j, m\}) = \frac{\exp(\delta_{i,k,n})}{\sum_{j=1}^{3} \sum_{m=1}^{3} \exp(\delta_{i,j,m})}$$

In practice, an individual may not have access to all three insurance options in each period. In order to enroll in Medicaid, an individual must satisfy the restrictive conditions for eligibility. Indeed, most individuals in the US do not qualify for Medicaid, and as such, will only face a choice between purchasing private insurance and remaining uninsured. Since this project is concerned with insurance choices revolving around Medicaid, we mainly consider a subpopulation that is much more likely to qualify for Medicaid. Furthermore, and especially for this subpopulation, it may be not be realistic to think that every individual can choose private insurance. Private insurance can be very expensive unless it is subsidized by an employer. This is especially true for the subpopulation considered in this project. In reality, the price of private insurance is much lower if the individual is employed and if her employer offers a private insurance plan to employees. This variation in the cost of private insurance can be captured in P_p. As a result, some individuals without access to employer-sponsored private insurance may find P_p so large that it overwhelms the perceived health benefits ($U_p < 0$). Such an individual will then choose between Medicaid (if eligible) and being uninsured.

In addition, shocks to enrollment costs are not the only channel through which welfare reform may have reduced Medicaid enrollment. An individual may be less likely to overcome enrollment costs of Medicaid in the current period if they feel they will be likely to obtain private insurance in the next period. Since welfare reform provided work incentives, it is possible that an increase in the expectation of private insurance in the future may have driven down enrollment in Medicaid, as individuals choose to wait for private insurance rather than "pay" the enrollment costs of Medicaid now. It also worth mentioning that although welfare reform may have increased the likelihood of employment in the next period, this does not necessarily imply that private insurance became more affordable to this population. Indeed, many of the jobs former welfare recipients obtained were low-skilled, low-wage jobs for which employer-sponsored health insurance was not necessarily included in benefit packages. As such, many former Medicaid enrollees may have found jobs without private insurance, but lost their Medicaid eligibility or simply thought they no longer qualified for Medicaid. This would result in an increase in the probability of individuals switching from Medicaid to no insurance coverage.

3.5 Data

I use individual-level data obtained from the 1993 and 1996 panels of the Survey of Income Program Participation (SIPP). The 1993 panel covers calendar months February 1993 through January 1996. The 1996 panel covers April 1996 through March 2000. For both panels, respondents are interviewed in four month intervals. During each interview, the respondent is asked to provide information pertaining to the current month, as well as the previous three months. Because information not specific to the current month must be recalled from the past it may be subject to bias and reporting errors. I focus only on data pertaining to the month of the interview to reduce reporting error in my sample. After dropping non-interview months, I have one observation every four months for each survey respondent. Ignoring sample attrition, respondents in the 1993 panel have up to 9 observations while 1996 respondents have up to 12 observations.

Because welfare reform mainly occurred in early and mid 1996, the 1996 SIPP panel does not provide sufficient pre-reform coverage. Likewise, the 1993 Panel ends in January of 1996, before states had enacted major reforms. As a result, I must construct a panel of individuals for the pre-reform period from the 1993 SIPP and a panel of individuals for the post-reform period using the 1996 SIPP. Using these two panels, I analyze the changes in insurance dynamics induced by welfare reform.

Table 3.1: Summary Statistics

	pre-PRWORA 1993-1995		**post-PRWORA** 1997-1999	
	Mean	StdDev	Mean	StdDev
Age	27.37	6.14	29.03	6.80
Nonwhite	0.50	0.25	0.47	0.25
Hispanic	0.20	0.16	0.18	0.15
HS Diploma	0.79	0.16	0.80	0.16
Some College	0.38	0.24	0.41	0.24
MnthlyFamInc ($1000s)	2.36	2.63	2.08	2.80
Number of Kids	1.81	1.10	1.88	1.16
Employed	0.48	0.25	0.65	0.23
Welfare	0.37	0.23	0.18	0.15
Number 4 month Obs	3126		5567	

The sample is restricted to single mothers ages 18-44 years old. This sample definition is common in the literature and is the subpopulation for whom we would expect Medicaid and welfare policy to have the greatest impact. The sample is also restricted to women without a college degree. Controls for high school diplomas and at least some college education are available. There is one

observation per individual per four month interval. Because marriage status may affect insurance and employment options, a women must be single at the beginning and the end of the four month period for that four month observation to be included in the sample. The final sample includes 1255 women and 8693 four month individual observations.[9]

Table 3.1 provides summary statistics for basic demographics. The demographic composition of the sample between the pre- and post-reform periods are quite similar. As expected the two periods differ in the likelihood that single mothers are employed (48% pre-PRWORA and 65% post-PRWORA) and receiving cash welfare (37% pre-PRWORA and 18% post-PRWORA). The signs of these differences are not surprising. Reforms were intended to reduce the reliance on welfare and encourage employment.

This project is focused on the effect of welfare reform on the insurance status of single mothers. However, as described in the model in Section 3.4, insurance status and employment status are intimately related and are best modeled jointly. Doing so allows for a more complete picture of the effects of the reform and the experience of single mothers in the mid-1990s. Each observation in the SIPP sample is assigned an employment/insurance state (s_i). This state is determined by the employment (or welfare) and insurance status of the individual at the time of the SIPP survey interview. From the SIPP we observe whether the individual is employed at the time of the survey and whether the individual is currently receiving welfare benefits. From this the individual is assigned one of four employment "states": Welfare, Work, No Work/No Welfare, or Work $ Welfare. Similarly, the SIPP identifies the insurance status of each individual at the time of the survey. Specifically, I assign each individual one of three insurance states: No Coverage (Unins.), Medicaid, or Private. There are 12 possible pairings of employment and insurance states, however, since all individuals who are receiving welfare are also enrolled in Medicaid, only 8 states are relevant in this context. This restriction has both intuitive and empirical appeal. In the pre-reform period, welfare was directly linked to Medicaid. Individuals who were eligible for and enrolled in welfare were almost universally eligible and enrolled in Medicaid. Post-reform this is no longer true, indeed this decoupling is the motivation for this project. However, post-reform individuals who were still receiving welfare would still be eligible for, and very likely enrolled in, Medicaid. The increased enrollment costs and confusion over eligibility operated in the reverse direction so that people who were no longer receiving welfare may still have been eligible for Medicaid, but either did not realize it, or were not able or willing to overcome the increased enrollment burden.

Table 3.2 gives the observed distribution for the eight employment/insurance

[9]Broken down by period: pre-reform, 468 individuals and 3126 observations; post-reform, 787 individuals and 5562 observations.

Table 3.2: Employment-Insurance States

	States (s_i)		% of Sample		Number of Obs	
	Work/Welfare	**Insurance**	**'93-'95**	**'97-'99**	**'93-'95**	**'97-'99**
1	No Work, No Welfare	No Coverage	7.73	8.25	242	459
2	No Work, No Welfare	Medicaid	6.39	7.68	200	427
3	No Work, No Welfare	Private	4.03	4.74	126	264
4	Welfare	Medicaid	33.66	14.01	1052	779
5	Work	No Coverage	12.09	17.70	378	984
6	Work	Medicaid	6.20	8.63	194	480
7	Work	Private	26.95	34.67	842	1929
8	Work & Welfare	Medicaid	2.95	4.32	92	240
		Totals	100	100	3126	5562

states. Prior to PRWORA it was most common for single mothers in this sample to both receive welfare and be enrolled in Medicaid (33.66%). However, note that this drops to 14.01% after post-PRWORA. At the same time we see increases in the four "employed" states, perhaps most notably an increase from 26.95% to 34.67% in single mothers who are both employed and covered by private health insurance. Overall, it appears there is a substantial reduction in single mothers on welfare in the post-reform period. This is not surprising, since the policy targeted a reduction in welfare numbers. Table 3.2 is, however, a static glimpse of the effect of welfare reform on both employment and health insurance and does not give a full picture of the experience of the single mothers affected by the reform. Although its important to know the change in distribution of employment and insurance status after the reform, to fully understand the impact of the policy we need to look more carefully at the dynamics of employment and insurance status. For example, from Table 3.2 there appears to be a reduction in single mothers on welfare and enrolled in Medicaid. What was the experience of these women? If they are not enrolled in welfare any longer, are they employed or unemployed? Did they retain their Medicaid enrollment? Questions such as these are difficult, if not impossible to answer using the type of static summary statistics seen in Table 3.2. One of the advantages of the SIPP is that it is a panel and so individuals can be tracked over time. As such, we can observe the employment and insurance status of individuals every four months. For example, we might be interested in the experience of the sample of women who are receiving welfare benefits and are enrolled in Medicaid. It is this population that was presumably most directly affected by the reforms to welfare. Were single mothers less likely to retain welfare and Medicaid status after four months in the post-reform world? Of the women leaving welfare, do they find employment? Do they lose their Medicaid and if so, is it replaced by private health insurance? Do the answers to these questions differ

in the post-reform period? Tables 3.3 and 3.4 help to answer these questions.

Table 3.3: Welfare/Medicaid Sample: Employment/Insurance Status after 4 Months (%)

	No Work, No Welfare			Welfare		Work		Work & Welfare
Years	Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
1993-1995	1.10	1.73	0.00	90.89	0.99	1.62	0.63	3.05
1997-1999	3.13	11.91	0.00	71.23	1.84	4.36	1.02	6.51
Difference	2.03	10.19	0.00	-19.66	0.85	2.74	0.39	3.46

Table 3.3 gives the employment/insurace status of the Welfare/Medicaid sample four months later. Prior to PRWORA, almost 91% of single mothers on welfare and Medicaid would retain this status after four months. Of the 9.11% that transition to a different employment/insurance status after four months, 3.05% retained welfare and Medicaid status, but had also found employment, 1.10% were unemployed, without welfare, and uninsured, only 0.63% had found employment and were covered by private health insurance, and 1.73% retained Medicaid enrollment but were unemployed and without welfare, and 1.62% were still enrolled in Medicaid but employed. The second row of Table 3.3 provides similar transition information but for the post-PRWORA period (1997-1999). Perhaps most striking is that in this period only 71.23% of the Welfare/Medicaid sample retained that status after four months. Almost 12% of the "welfare/medicaid leavers" in the post-reform period retained their Medicaid enrollment after four months, but were unemployed at the time, compared to 1.73% in the pre-form period. There is also a 2.03% and 0.85% increase in the percentage of welfare/medicaid leavers that are not covered by health insurance after four months (unemployed and employed, respectively). Table 3.4 summarizes slightly differently the information in Table 3.3. This table focuses only on the individuals who leave Welfare/Medicaid after four months and provides the distribution for their destinations. We see that over 41% of "leavers" retain Medicaid, but are unemployed after four months in the post-reform period, compared to 18.9% in the pre-reform period. In addition, although in Table 3.3 it may have appeared that women leaving Welfare/Medicaid were more likely to be uninsured after four months, Table 3.4 shows that this is not true. Conditional on leaving Welfare/Medicaid women are less likely in the post-reform period to find themselves in one of the two states without health insurance.

Table 3.4: Welfare/Medicaid Leavers: Distribution of Destinations after 4 Months (%)

	No Work, No Welfare			Work			Work & Welfare
	Unins.	Medicaid	Private	Unins.	Medicaid	Private	Medicaid
1993-1995	12.05	18.94	0.00	10.88	17.79	6.91	33.42
1997-1999	10.87	41.41	0.00	6.41	15.15	3.55	22.61
Difference	-1.18	22.47	0.00	-4.47	-2.65	-3.36	-10.81

Taken at first glance, the observations from Tables 3.3 and 3.4 seem to suggest that PRWORA did not drastically or negatively affect the health insurance status of single mothers who were on welfare and Medicaid. However, these tables focus only on single mothers in the Welfare/Medicaid state and thus do not provide the full picture of the effect of the reform on employment and insurance dynamics for the affected subpopulation. Tables 3.5 and 3.6 expand to the full SIPP sample of single mothers and provide transition percentages for all eight of the employment/insurance states. The left column gives the employment/insurance state of the individual at the beginning of the four month period. The other columns give the percentage of each initial state that ends up in each destination state after four months. The main diagonal, thus, is the percent that retain the same employment/insurance status after four months. Table 3.7 gives the difference in the pre- and post-reform transition matrix. That the main diagonal is mostly negative indicates that employment and insurance dynamics became less stable following welfare reform. Also note that all entries for the destination state Welfare/Medicaid are negative. This indicates that individuals are less likely to move into welfare in the post-reform period. In addition, most values under the destination state Work/Private insurance (arguably the most desirable state from a policy perspective) are positive. That is, post-reform there individuals are more likely transition to employment with private insurance. However, note that individuals who are working, but uninsured, are actually less likely post-reform to transition into employment with private insurance (-1.88%) and more likely to remain employed and without insurance (2.99%). This suggests that although reform may have encouraged employment, individuals who did find employment are less likely to find jobs that ultimately provide them with health insurance benefits.

Table 3.5: Observed 4 month Transition Matrix, Before PRWORA (1993-1995)

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	61.22	7.59	3.80	4.95	17.37	0.00	5.06	0.00
No Work, No Welfare	Medicaid	13.15	56.32	2.34	14.54	1.82	10.84	0.99	0.00
	Private	11.01	1.51	63.32	0.00	3.83	0.00	20.33	0.00
Welfare	Medicaid	1.10	1.73	0.00	90.89	0.99	1.62	0.63	3.05
	Unins.	9.43	0.00	0.52	0.00	67.61	5.76	16.67	0.00
Work	Medicaid	3.99	9.10	0.00	4.94	13.58	57.82	5.95	4.62
	Private	2.05	0.00	3.68	0.00	7.67	0.00	86.60	0.00
Work & Welfare	Medicaid	0.00	2.52	0.00	21.05	4.27	20.64	3.12	48.41

Table 3.6: Observed 4 month Transition Matrix, After PRWORA (1997-1999)

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	57.92	10.03	3.64	4.13	19.38	0.00	4.91	0.00
No Work, No Welfare	Medicaid	10.64	60.16	1.00	10.93	4.14	11.70	1.43	0.00
	Private	6.54	1.96	54.63	0.00	3.73	0.00	33.14	0.00
Welfare	Medicaid	3.13	11.91	0.00	71.23	1.84	4.36	1.02	6.51
	Unins.	8.48	0.00	0.80	0.00	70.61	5.32	14.79	0.00
Work	Medicaid	2.70	10.35	0.00	2.92	16.06	53.62	11.12	3.23
	Private	1.75	0.00	3.75	0.00	4.95	0.00	89.55	0.00
Work & Welfare	Medicaid	0.00	3.21	0.00	9.24	5.54	21.28	4.45	56.28

Transition matrices of the form seen in Tables 3.5 and 3.6 serve as the main tool of analysis in the project. The matrices themselves and how transition probabilities changed after reform provide a much more complete picture of the interaction between employment, insurance and reform than is possible from the static, more reduced form approach more commonly seen in the literature. In this section, I have reported the empirical transition probabilities observed in my SIPP sample. However, as discussed previously, the SIPP panels do not span the time of the reform. Therefore, although from the data I can track individuals in four month intervals, I am unable to observe the same individuals both pre- and post-reform. As a result, its possible that differences in transition probabilities observed in this section are driven by differences in the composition of the two SIPP samples rather than by changes induced by reform. In the next section I take a more rigorous approach to generating transition probabilities in an attempt to address this concern.

Table 3.7: Change in 4 month Observed Transition Matrix

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	-3.30	2.44	-0.17	-0.82	2.00	0.00	-0.15	0.00
No Work, No Welfare	Medicaid	-2.51	3.84	-1.34	-3.61	2.32	0.86	0.44	0.00
	Private	-4.47	0.45	-8.70	0.00	-0.10	0.00	12.82	0.00
Welfare	Medicaid	2.03	10.19	0.00	-19.66	0.85	2.74	0.39	3.46
	Unins.	-0.95	0.00	0.28	0.00	2.99	-0.44	-1.88	0.00
Work	Medicaid	-1.29	1.25	0.00	-2.02	2.47	-4.20	5.17	-1.39
	Private	-0.30	0.00	0.07	0.00	-2.72	0.00	2.95	0.00
Work & Welfare	Medicaid	0.00	0.70	0.00	-11.82	1.27	0.64	1.34	7.87

3.6 Results: Welfare, Employment, and Insurance

In this section I construct covariate-adjusted transition matrices similar to those seen in the previous section. Because my data is comprised of two disjoint samples of single mothers, one for the pre-reform period and one post-reform, the covariate adjustments help to address concerns that observed changes to employment and insurance dynamics are driven by the composition of the samples rather than induced by welfare reform. To construct these transition matrices, I use equation 3.4 to estimate an independent multinomial logit for each of the eight possible initial employment/insurance states in the model ($s_i^t = \{j, m\}\ \forall\{j, m\}$). Individual level controls are included, including age, race, educational level, family income, and the number of children. These covariates help to control for differences in the composition of the two SIPP panels. In addition, an indicator for the post-reform period is included, which will capture the structural shift in employment and insurance dynamics. Using the resulting coefficient estimates from these estimations I construct covariate-adjusted transition matrices for both the pre- and post- welfare reform periods. Each estimation provides a vector of coefficients specific to each outcome state and the initial state ($\beta_{k,j}$). The elements of $(\beta_{k,n} - \beta_{j,m})$ give the effect of the independent variables on the probability an individual will transition to state $\{k, n\}$ in the next period *relative* to remaining in the initial state $\{j, m\}$.

3.6.1 Estimates for the sample of Single Mothers on Welfare and Medicaid

I first report results for the initial employment/insurance state of Welfare/Medicaid ($s_i^t = 1$) These results are given in Table 3.8. Each column gives the vector of coefficient estimates for transitions into state s^{t+1} given the individual begins the period on welfare/Medicaid. It is worth noting that results are only reported for six of eight possible outcomes. The Welfare/Medicaid state is not reported because it is used to normalize; all reported coefficients are effects on transitions relative to remaining in welfare/Medicaid. Also, the no work, no welfare, private insurance state is not reported since I have restricted the probability of transition to this state from Welfare/Medicaid to be zero. This restriction has intuitive appeal; it seems highly unlikely that an unmarried individual, in a 4 month period, would go from receiving welfare and covered by Medicaid to not receiving welfare, not working, and covered by a private insurance plan. While possible, transitions of this type are extremely rare in the data.

The predicted probability of an average single mother moving from initial

Table 3.8: Multinomial Logit Estimates for Employment/Insurance Model

	Destination State (s^{t+1})					
	(a)	(b)	(c)	(d)	(e)	(f)
	No Welfare/No Work			Work		Work & Welfare
	Unins.	Medicaid	Unins.	Medicaid	Private	Medicaid
post-PRWORA	1.287	2.177	0.803	1.223	0.477	0.988
	(0.376)	(0.251)	(0.463)	(0.301)	(0.624)	(0.250)
Constant	3.099	-4.874	4.160	-27.815	-4.133	2.877
	(4.292)	(2.295)	(4.004)	(6.495)	(8.114)	(3.702)
Age	-0.451	0.031	-0.543	1.815	0.028	-0.337
	(0.275)	(0.151)	(0.278)	(0.477)	(0.609)	(0.259)
Age^2	0.007	0.000	0.009	-0.034	-0.002	0.005
	(0.004)	(0.002)	(0.005)	(0.009)	(0.011)	(0.004)
Nonwhite	-0.269	-0.349	-0.449	0.428	-0.018	-0.647
	(0.455)	(0.249)	(0.445)	(0.389)	(0.655)	(0.307)
Hispanic	-0.250	0.004	-0.163	-0.544	-0.517	-0.375
	(0.436)	(0.274)	(0.532)	(0.532)	(1.074)	(0.351)
Some College	0.055	-0.606	0.765	1.071	1.498	0.681
	(0.544)	(0.318)	(0.643)	(0.347)	(0.654)	(0.311)
FamInc.($1000s)	-0.062	0.107	-0.122	-0.073	0.185	-0.122
	(0.114)	(0.078)	(0.256)	(0.116)	(0.073)	(0.109)
Number of Kids	-0.059	0.046	-0.255	-0.195	-0.487	-0.140
	(0.187)	(0.082)	(0.186)	(0.137)	(0.349)	(0.127)
R2	0.0931					
N	1963					

Note: Sample consists of single mothers without a college degree who began the 4 month period on welfare and enrolled in Medicaid.

state $\{j, m\}$ to outcome state $\{k, n\}$ over a four month time horizon is given by:

$$\widehat{Pr}(s_i^{t+1} = \{k, n\} | \bar{X}_i, s_i^t = \{j, m\}) = \frac{\exp(\widehat{\delta_{i,k,n}})}{\sum_{j=1}^{3} \sum_{m=1}^{3} \exp(\widehat{\delta_{i,j,m}})} \tag{3.1}$$

$$\widehat{\delta_{i,k,n}} = \widehat{NV_{k,n}} + \mathbf{1}_w[\widehat{\gamma_1 y_{k,j}} + \widehat{\gamma_4 e_{k,j}} + \widehat{\gamma_5 e_{n,m}}] + \bar{X}_i(\widehat{\beta_{k,n}} - \widehat{\beta_{j,m}}) \tag{3.2}$$

Using the coefficient estimates in Table 3.8, Table 3.9 reports the predicted percentage of single mothers on welfare and Medicaid that transition to each of the other 7 states after a four month interval. Table 3.10 reports the predicted distribution of destinations for the sample of single mothers who leave Welfare/Medicaid status after four months. These are the covariate-adjusted corollaries to Tables 3.3 and 3.4. Although slightly quantitatively different, the qualitative story is not changed by the covariate adjustment. This is reassuring that the observations made in the previous section were not driven solely by sample composition.

3.6.2 Estimates of Transition Matrices

I estimate similar specifications for each of the other 7 possible initial states, producing seven sets of coefficient estimates similar in nature to those reported in

Table 3.9: Welfare/Medicaid Sample: Estimated 4 month work/insurance status (%)

	No Work, No Welfare			Welfare		Work		Work & Welfare
Years	Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
1993-1995	0.82	2.05	0.00	92.08	0.56	2.08	0.42	2.01
1997-1999	2.32	14.16	0.00	72.25	0.98	5.53	0.53	4.23
Difference	1.50	12.11	0.00	-19.83	0.42	3.45	0.11	2.22

Table 3.10: Welfare/Medicaid Leavers: Estimated distribution of 4 month destination (%)

	No Work, No Welfare			Work			Work & Welfare
	Unins.	Medicaid	Private	Unins.	Medicaid	Private	Medicaid
1993-1995	0.10	0.26	0.00	0.07	0.26	0.05	0.25
1997-1999	0.08	0.51	0.00	0.04	0.20	0.02	0.15
Difference	-0.02	0.25	0.00	-0.04	-0.06	-0.03	-0.10

Table 3.8. Using these coefficient estimates I compute predicted probabilities for transition into each state for each initial condition (initial employment/insurance state). For each initial condition I do this for an "average" individual using the average value of each independent variable for that initial condition. I compute a predicted probability for each of the 8 potential outcome states for each of the 8 initial conditions.

After computing predicted transition probabilities for all eight initial conditions and all eight outcome states, the resulting 64 predicted probabilities can be organized into an 8x8 transition matrix of the same form as seen in Table 3.5. Each row corresponds to the employment/insurance state of the individual at the beginning of the 4 month period t. Likewise, each column is the employment/insurance state at the beginning of the next period $(t+1)$. I do this for both the pre and post-reform periods to generate two estimated, covariate-adjusted transition matrices, reported in Tables 3.11 and 3.12, respectively.

Table 3.11: Estimated 4 month Transition Matrix, Before PRWORA (1993-1995)

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	62.15	6.39	1.18	3.88	19.41	0.00	6.98	0.00
No Work, No Welfare	Medicaid	8.11	59.66	0.51	19.45	2.51	8.48	1.28	0.00
	Private	26.65	3.47	57.12	0.00	4.40	0.00	8.36	0.00
Welfare	Medicaid	0.82	2.05	0.00	92.08	0.56	2.08	0.42	2.01
	Unins.	8.64	0.00	0.08	0.00	75.03	4.11	12.14	0.00
Work	Medicaid	5.92	7.88	0.00	6.75	9.54	60.87	3.80	5.24
	Private	2.49	0.00	0.95	0.00	9.81	0.00	86.76	0.00
Work & Welfare	Medicaid	0.00	1.80	0.00	17.49	3.59	24.40	0.01	52.71

Table 3.12: Estimated 4 month Transition Matrix, After PRWORA (1997-1999)

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	61.29	7.09	1.39	2.11	21.47	0.00	6.65	0.00
No Work, No Welfare	Medicaid	6.56	63.72	0.14	14.17	5.51	8.18	1.71	0.00
	Private	16.57	3.09	59.25	0.00	4.33	0.00	16.76	0.00
Welfare	Medicaid	2.32	14.16	0.00	72.25	0.98	5.53	0.53	4.23
	Unins.	7.90	0.00	0.18	0.00	75.77	3.86	12.29	0.00
Work	Medicaid	4.34	10.83	0.00	3.72	12.71	55.46	9.25	3.70
	Private	1.99	0.00	1.36	0.00	6.81	0.00	89.83	0.00
Work & Welfare	Medicaid	0.00	1.79	0.00	5.49	4.74	25.58	0.00	62.40

In order to easier observe the changes in transition probabilities across periods, I subtract the pre-reform matrix (Table 3.11) from the post-reform matrix (Table 3.12). The difference in 4 month transition probabilities are reported in Table 3.13. The largest difference is in the retention rate for the welfare/Medicaid state. The probability that an average single mother on welfare and Medicaid will remain on welfare and Medicaid to start the following period is 19.83 percentage points lower in the post-reform period. In addition, such an individual is 12.11 percentage points more likely to remain on Medicaid but no longer receive welfare and 3.45 percentage points more likely to remain covered by Medicaid but to be working rather than receiving welfare. Taken together, these numbers suggest that welfare reform reduced likelihood of remaining on welfare, but that the effect on Medicaid may be more complex. In Table 3.13 we also see that most elements in the northwest quadrant are negative and most in the northeast are positive, indicating that individuals who were not working and not receiving welfare were more likely to begin working after reform. However, we also see an increase in the probability of moving from no work/no welfare to working with no insurance coverage.

Table 3.13: Change in 4 month Estimated Transition Matrix

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	-0.86	0.70	0.21	-1.77	2.06	0.00	-0.33	0.00
No Work, No Welfare	Medicaid	-1.54	4.06	-0.37	-5.28	3.00	-0.30	0.43	0.00
	Private	-10.08	-0.38	2.12	0.00	-0.07	0.00	8.40	0.00
Welfare	Medicaid	1.50	12.11	0.00	-19.83	0.42	3.45	0.11	2.22
	Unins.	-0.74	0.00	0.10	0.00	0.74	-0.25	0.15	0.00
Work	Medicaid	-1.58	2.95	0.00	-3.03	3.17	-5.41	5.44	-1.54
	Private	-0.50	0.00	0.41	0.00	-3.00	0.00	3.07	0.00
Work & Welfare	Medicaid	0.00	-0.01	0.00	-12.00	1.15	1.18	0.00	9.68

3.6.3 Simulation of Longer Horizons

The transition probabilities reported in Tables 3.11 and 3.12 provide insight into the employment and insurance dynamics of the welfare/medicaid subpopulation and how these dynamics changed after welfare reform. Although more informative than a simple static estimate of changes in Medicaid enrollment and welfare caseloads, the four month transition window is still a relatively limited scope for fully understanding the impact of the reform. It would be useful to observe transitions over longer time horizons. The simplest way to do this would be to track individuals for more than four months and compute the transition rates over the longer horizon. There are several problems with this approach. Over longer time periods there are an increasing umber of confounding changes to the economic environment that may be incorrectly attributed to welfare reform. In an ideal scenario, we would observe the employment and insurance status of individuals immediately before reform was enacted and then be able to track changes to this status over longer time horizons in a world *without* welfare reform. Because the actual implementation of reform encroaches on our ability to observe this counterfactual. [10] In response to this, I propose a more structured framework for summarizing the information contained in the transition probabilities over longer time horizons. Tables 3.11, 3.12, and 3.13 only provide a snapshot of the changes in dynamics induced by welfare reform. They represent the 4 month transition dynamics of single mothers. We would like to know how these short term dynamics may have altered the insurance status of single mothers over longer time horizons. To this we need a fairly restrictive assumption that conditional on observables the transition dynamics within a reform environment are constant over time.[11] With this assumption and the estimated 4 month transition matrices I compute a transition matrix (T_τ), in 4 month intervals, for any time horizon. We have that

$$T_\tau = \prod_{1}^{\tau} T_1$$

where τ is the number of 4 month intervals, T_1 is the baseline four month estimated transition matrix reported in Tables 3.11 and 3.12 and T_τ is the transition matrix for τ 4 month periods ($\tau/3$ years). Each row of T_τ gives the probability of transition, given the initial state, to each state after τ four month periods. For example, in Table 3.14, I report the resulting one year transition matrix for the pre-reform period (T_3^{pre}). As expected, state-dependence decreases over time.

[10] Note that the relatively limited variation in the timing of reform across states does not provide sufficient "control" groups to implement a standard difference in difference estimation

[11] That is, four month transition dynamics *within* a reform environment are a first-order Markov process

Table 3.14: Estimated 1 Year Transition Matrix, Before PRWORA (1993-1995)

		Destination State							
		No Work, No Welfare			Welfare	Work			Work & Welfare
Initial State		Unins.	Medicaid	Private	Medicaid	Unins.	Medicaid	Private	Medicaid
	Unins.	29.60	7.68	1.55	9.90	30.41	2.84	17.77	0.26
No Work, No Welfare	Medicaid	11.47	24.40	0.76	36.51	9.14	10.77	5.33	1.61
	Private	31.00	6.74	19.43	3.99	18.34	1.30	19.14	0.06
Welfare	Medicaid	2.36	4.22	0.06	80.42	2.54	5.21	1.68	3.51
	Unins.	13.87	1.79	0.58	1.63	49.68	5.98	26.05	0.42
Work	Medicaid	10.31	10.10	0.33	18.01	17.96	27.28	10.47	5.54
	Private	6.73	0.50	1.60	0.34	20.86	0.94	69.01	0.02
Work & Welfare	Medicaid	4.17	6.02	0.07	33.13	9.87	25.67	3.53	17.54

I construct similar transition matrices for time horizons in four month intervals up to 10 years [12]. I do this for both the pre and post reform periods, producing 30 transition matrices for each period. The element in row m and column n provides the probability that an individual initially in employment insurance state m will have transitioned into state n at the end of the time horizon specific to that transition matrix. For example, using Table 3.14 we can see that 1.68% of single mothers initially on welfare and covered by Medicaid (row 4) will be working and covered by private insurance after one year.

By looking at the fourth row of these transition matrices for each time horizon, we see how the probability of membership in each of the 8 employment/insurance states changes over time for single mothers initially on welfare and covered by Medicaid. Furthermore, since the focus of this study is on health insurance coverage, we can combine employment/insurance states that correspond to the same type of insurance coverage in order to see the total probability of coverage by each insurance type. For example, again focus on row 4 of Table 3.14. A single mother initially on welfare and covered by Medicaid will be uninsured after one year with probability $2.36\% + 2.54\% = 4.90\%$ (column 1 + column 5). A similar exercise reveals that such a single mother will be covered by Medicaid after 1 year with probability 93.36% and covered by private insurance with probability 1.74%. This can be done for each time horizon and for both the pre and post-reform periods. The resulting insurance transition probabilities are reported in Figures 3.3 and 3.4 for the pre- and post-reform periods respectively. Recall, these probabilities are conditional on initial membership in the Welfare/Medicaid state.

To see the effects of welfare reform on the insurance experience of single welfare mothers, I subtract the pre-reform probabilities from the post-reform. These differences are reported in Figure 3.5 and can be interpreted as the effect of welfare reform on the health insurance status of single mothers initially on welfare. We see a sharp reduction the probability of Medicaid coverage over the first 3 years. We also observe a steady increase in the relative likelihood of private insurance coverage over the first few years. Finally, welfare reform initially causes an increase in the likelihood of no coverage for these single mothers, but this effect peaks after 2 years and then steadily declines. [13]

[12] Using a four month transition matrix to construct a 10 year transition horizon requires strong assumptions about the stability of the transition structure. However, my goal is to use these longer horizon matrices only to provide some suggestive insight into the implied long run dynamics of welfare reform. For the purposes of estimating policy effects, I restrict my attention to time horizons of 3 years or less.

[13] Longer time horizons should be interpreted with care. They are not predictions for the actual experience of US welfare mothers in the ten years following welfare reform. Over longer horizons other factors would have changed the dynamics of employment and insurance transitions, including other new policies and changes in economic conditions. In addition, the model in this project does not incorporate the effects of time limits on welfare benefits introduced by

Figure 3.3: Pre-reform Probability of Insurance Coverage - Single Mothers on Welfare/Medicaid

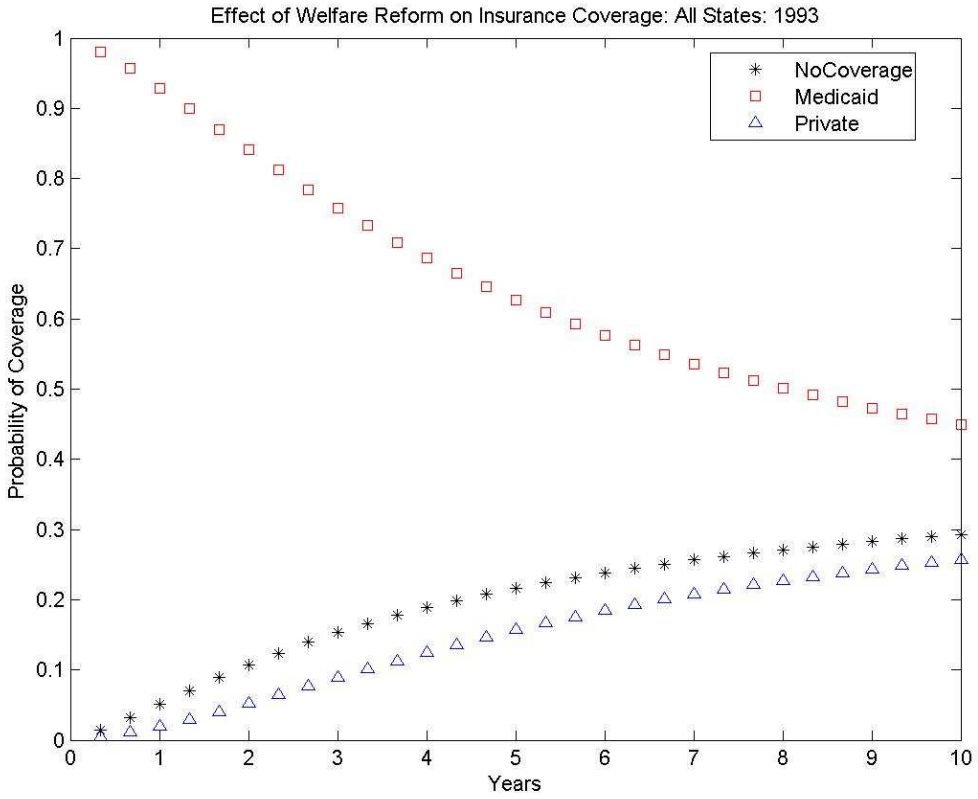


Table 3.15: Effect of Welfare Reform on Insurance Coverage: Single Mothers on Welfare

	Insurance Type		
Horizon	Uninsured	Medicaid	Private
4 Months	1.92	-2.03	0.11
1 Year	8.06	-10.41	2.35
10 Year	7.88	-30.19	22.31
Maximum Effect	16.30	-35.09	22.87
Long Run	2.82	-20.38	17.56

Note: Table reports the percent change single mothers covered by each insurance type over the time horizon given in the left column. Sample consists of single mothers receiving welfare benefits at the beginning of the simulated period.

TANF. As Leonard and Mas [2008] show, these time limits may have real effects on the health and well-being of single mothers and their children. Since time limits were approximately five years in most states and my sample covers only 1997-1999, it is unlikely that timing out of welfare has confounded my transition estimates. Although forward-looking single mothers may have altered behavior which could have affected the estimated 4 month transitions out of welfare.

Figure 3.4: Post-reform Probability of Insurance Coverage - Single Mothers on Welfare/Medicaid

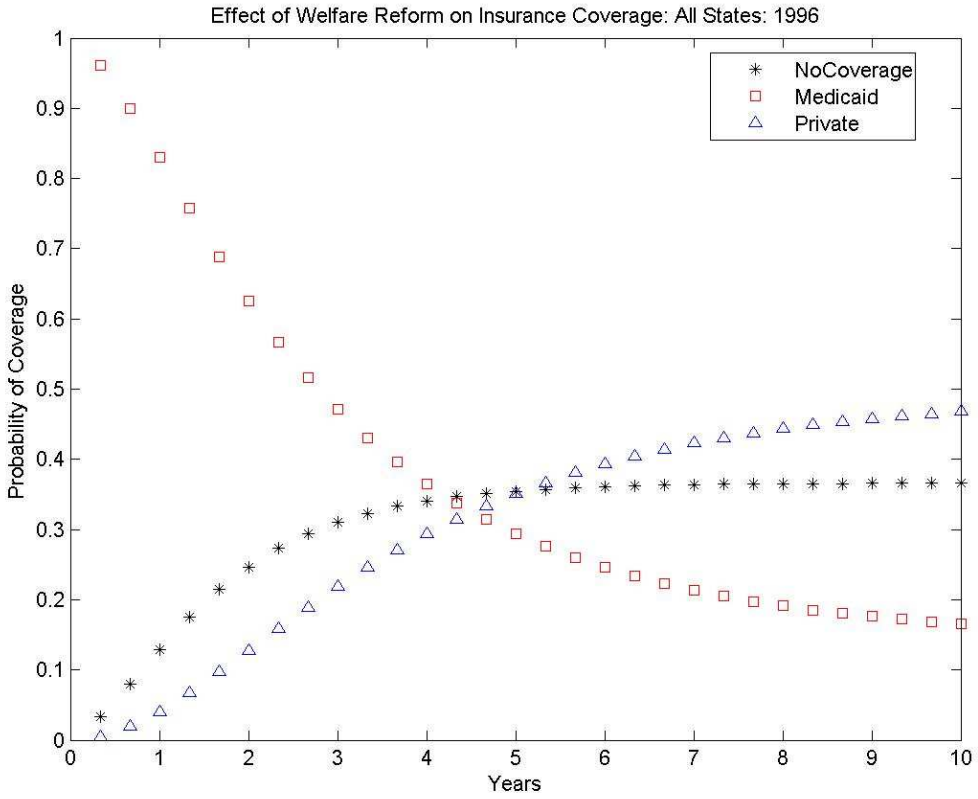
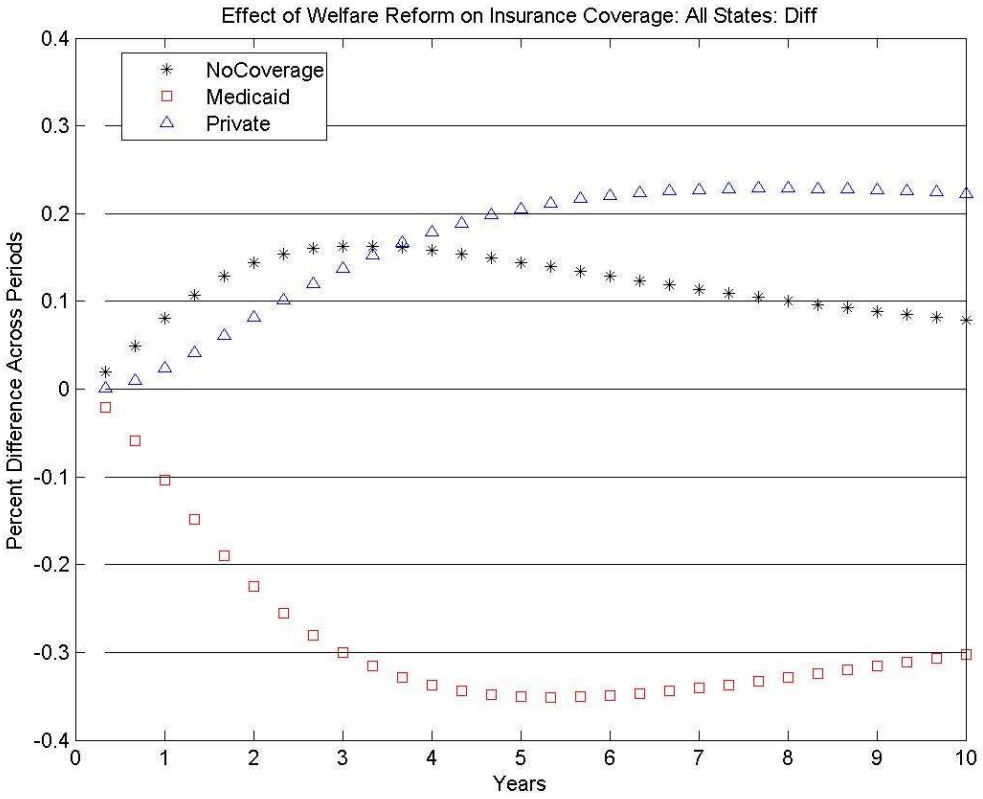


Table 3.15 provides a static summary of the information contained in Figure 3.5. Each row reports the effect of welfare on health insurance coverage for different time horizons. Row 1 gives the short-run, four month, effect. Under such a short horizon, there is little effect of reform on private insurance coverage. There is a quick reduction in likelihood of Medicaid coverage and most of these individuals losing Medicaid coverage appear to become uninsured. Row 2 is the one year effect. After one year, probability of Medicaid coverage is reduced by 10.41 percentage points. We also see a large increase, 8.06 percentage points, in individuals without insurance coverage, but now there is a 2.35 percentage point increase in the probability of private coverage. Thus, even after a single year, we begin to see that PRWORA's work incentives may be having an effect. Row 3 gives the effect after 10 years, row 4 is the maximum effect observed for each insurance type. Finally, row 5 is the long-run, steady-state effect. While this last

Figure 3.5: Effect of Welfare Reform on Insurance Coverage - Single Mothers on Welfare/Medicaid



row must be interpreted with caution, it does suggest that welfare reform shifted the dynamics in a manner that implies a slight long-run increase in uninsured single mothers, a large reduction in Medicaid coverage, and an almost as large increase in private insurance coverage.

3.7 Conclusion

In the mid-1990s, the United States implemented significant reform to its cash-assistance welfare programs. The Personal Responsibility and Work Opportunity Reconciliation Act of 1996 (PRWORA) replaced Aid to Families with Dependent Children (AFDC) with Temporary Assistance for Needy Families (TANF) as the country's main cash-assistance welfare program. With TANF came a reduction in eligibility for cash assistance and an increased focus on employment incentives.

The reform was not intended to directly affect the health insurance status of individuals who were enrolled in Medicaid. However, the reform indirectly decoupled cash-assistance and Medicaid, potentially imposing unintended costs and barriers to Medicaid enrollment. In addition, while TANF encouraged employment it was not clear how a shift from cash-assistance to labor force participation would affect the ability of single mothers to obtain health insurance of any kind, either Medicaid or private insurance.

In this chapter, I study the effects of welfare reform in the mid-1990s on single mothers in the United States. The simple modeling framework used is more structured than commonly found in the literature. I estimate short and long run employment and insurance dynamics before and after the implementation of PRWORA. I show that reform reduced use of cash-assistance and increased the probability of employment for the affected subpopulation, but in doing so also created a less stable employment and health insurance environment. After the reform single mothers were less likely to retain the same employment and insurance status over a four month period. In addition, although policy was not intended to affect Medicaid eligibility, we see individuals less likely to retain Medicaid enrollment over both the short and longer run. Although some of these individuals transition from Medicaid into private insurance there is a 1.92% increase in the uninsured over a four month period which increases to 8.06% after one year.

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Chapter 4

Appendix to Chapter 2

This appendix provides the coefficient estimates of the in-hospital death and readmission equations ($\{\hat{\beta}_s^d, \hat{\beta}_s^r\}$). The specification includes the mass-point distribution of unobserved heterogeneity with four points of support.

Table 4.1: Estimation Results, Full Model: In-Hospital Mortality

	CABG		**PCI**		**OMT**	
	$\hat{\beta}_1^d$	S.E.	$\hat{\beta}_2^d$	S.E.	$\hat{\beta}_0^d$	S.E.
Black	0.3431	0.2428	-0.0551	0.1505	-0.0677	0.0998
Female	0.2061	0.1132	0.2418	0.053	-0.2588	0.0445
Heart Failure	0.3785	0.0813	0.4944	0.0465	0.1689	0.0391
Hypertension	-0.2122	0.0578	-0.1323	0.0304	-0.2024	0.0266
STEMI	0.2983	0.063	0.3142	0.0353	0.5194	0.0319
Diabetes	-0.0563	0.0691	0.1166	0.0421	0.0313	0.0361
Peri. Vasc. Disease	0.1263	0.0812	0.201	0.0516	0.1203	0.0441
Coronary Atherosclerosis	-0.6008	0.0902	-0.2705	0.0392	-0.3389	0.0313
Other Atherosclerosis	0.3798	0.083	0.2588	0.066	0.0758	0.0595
CardioShock	1.1256	0.0638	1.1863	0.0382	1.1663	0.0479
Hyperlipidemia	-0.2831	0.061	-0.2529	0.0335	-0.1893	0.0288
EndStageRenal	0.3233	0.1168	0.3287	0.0742	0.1468	0.0647
Cancer	0.2982	0.1074	0.242	0.0588	0.1742	0.0462
Anemia	-0.1097	0.0722	0.3463	0.0489	0.0762	0.0408
(age-49)	0.0036	0.0382	-0.0052	0.0204	0.0089	0.0173
$(\text{age-49})^2/100$	0.0884	0.2086	0.1786	0.1142	0.0924	0.0964
$(\text{age-49})^3/10{,}000$	-0.0932	0.0341	-0.0308	0.0188	-0.0212	0.0159
FemaleXDiabetes	0.0309	0.1090	-0.0369	0.0609	0.0901	0.0535
FemaleXHeartFail	-0.066	0.1191	-0.0519	0.0658	0.1443	0.0561
FemaleXAnemia	-0.2346	0.1122	-0.3158	0.0659	-0.1813	0.058
BlackXDiabetes	-0.6665	0.3369	0.0145	0.1536	-0.0267	0.1116
BlackXHeartFail	-0.0708	0.2929	-0.0735	0.167	-0.0394	0.1144
BlackXAnemia	0.0503	0.2872	0.1046	0.1581	-0.0521	0.1162
Constant	-1.7404	0.2533	-2.4504	0.1177	-1.5936	0.0952
N= 47,439						

Note: Table gives estimation coefficients for equation determining in-hospital mortality for AMI patients. Results are from the full model, including unobserved patient heterogeneity.

Table 4.2: Estimation Results, Full Model: Readmission 90 days Post-Discharge

	CABG		**PCI**		**OMT**	
	$\hat{\beta}_1^r$	S.E.	$\hat{\beta}_2^r$	S.E.	$\hat{\beta}_0^r$	S.E.
Black	0.1983	0.0542	0.1358	0.0419	0.0845	0.0485
Female	0.2177	0.0267	0.1445	0.0195	0.0853	0.024
Heart Failure	0.2501	0.0256	0.3101	0.0216	0.2045	0.0238
Hypertension	-0.0457	0.0165	-0.0153	0.0125	0.0006	0.0147
STEMI	0.0544	0.0192	-0.0651	0.0154	0.0853	0.0179
Diabetes	0.0901	0.0219	0.1028	0.0166	0.0637	0.0195
Peri.Vasc. Disease	0.1589	0.0306	0.1692	0.0258	0.1155	0.0288
Coronary Atherosclerosis	-0.2157	0.0374	-0.1376	0.0266	0.088	0.0246
Other Atherosclerosis	0.1117	0.0442	0.1773	0.0332	0.1394	0.0372
CardioShock	0.1693	0.0384	0.2314	0.0324	0.276	0.0376
Hyperlipidemia	-0.0696	0.0162	-0.0712	0.0127	-0.0377	0.0147
EndStageRenal	0.2614	0.0494	0.2192	0.0426	0.1964	0.0468
Cancer	0.2121	0.0366	0.1637	0.0294	0.1014	0.0341
Anemia	0.1629	0.0289	0.3165	0.0243	0.2527	0.0232
(age-49)	-0.3954	0.8688	-0.1012	0.6666	-0.4702	0.766
$(\text{age-49})^2/100$	-0.0014	0.0527	0.0110	0.0404	0.0276	0.0467
$(\text{age-49})^3/10{,}000$	0.0072	0.0093	0.0021	0.00712	-0.0029	0.0082
FemaleXDiabetes	0.0309	0.0336	0.0457	0.0267	0.0778	0.031
FemaleXHeartFail	0.0153	0.0357	0.0037	0.0285	0.0453	0.0328
FemaleXAnemia	-0.1118	0.0375	-0.1842	0.0314	-0.1201	0.0337
BlackXDiabetes	0.019	0.0722	0.0258	0.0578	0.0396	0.0652
BlackXHeartFail	0.0724	0.0722	0.1007	0.061	0.1029	0.0672
BlackXAnemia	-0.1286	0.0764	-0.1083	0.0636	-0.1557	0.071
Constant	-1.0676	0.0646	-0.8678	0.0491	-1.1952	0.044
N= 47,439						

Note: Table gives estimation coefficients for equation determining readmission, post-discharge for AMI patients. Results are from the full model, including unobserved patient heterogeneity.

Chapter 5

Appendix for Chapter 3

5.1 Average Effect of Welfare Reform on Insurance Coverage

In Section 3.4 I presented estimated transition probabilities and insurance status results for an average single mother on welfare. In this section, rather than focusing on the effect for the average individual, I propose a method for estimating the average effect across the distribution of single mothers in the SIPP sample. I do this for women begin the four month period on welfare and Medicaid, but it can be done for any initial condition.

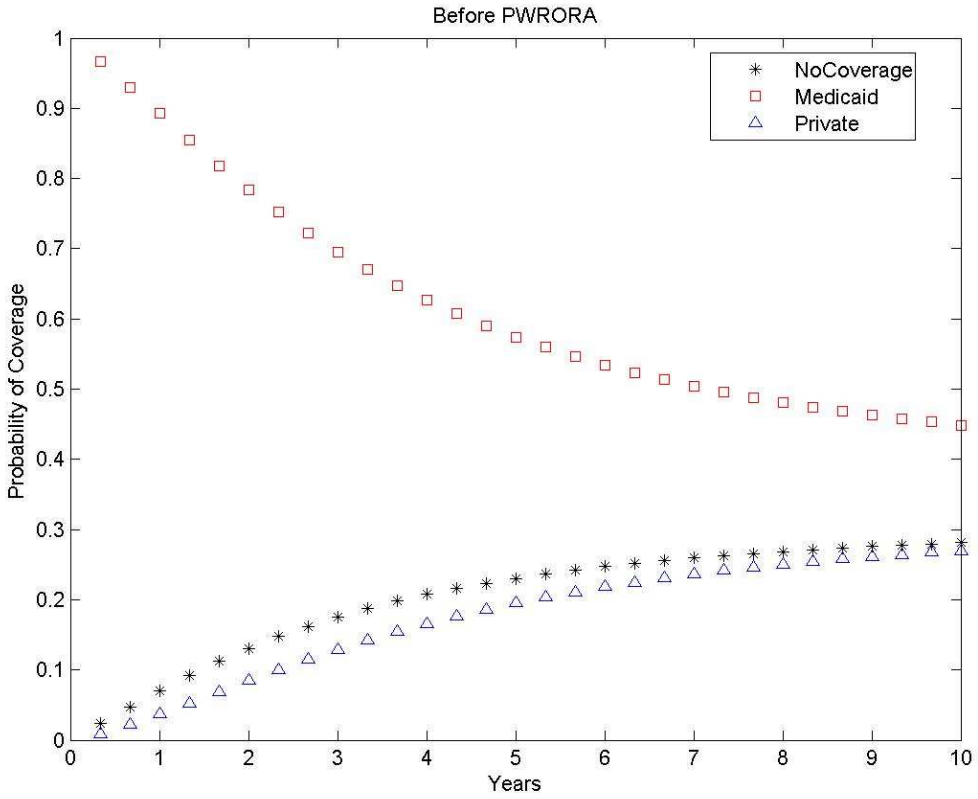
I take the first observation for each women age 18 to 44 years in the 1996 SIPP panel. I restrict the sample again to single, low education mothers. I additionally restrict to only women who report begin on welfare and covered by Medicaid in the first interview of the 1996 SIPP. The result is a sample of 194 women covering March, April, May, and June of 1996 [1].

For each of these women I use the results from the multinomial logit specifications described in Section 3.4 to generate predicted transition probabilities for all eight initial conditions. This allows me to construct two estimated transition matrices for each of the women in the sample, one for the pre-reform and one for the post-reform period, just as I did for the "average" individual in Section 3.4.

Once I have estimated transition matrices for each individual, I follow the same procedure as above. I roll forward the four month matrix to generate a set of transition matrices for longer time horizons. From these I pull the total probability of coverage by each insurance type by summing across the appropriate columns along the row corresponding to the sample's initial state (in this case, on welfare and covered by Medicaid). This results in three probabilities for each individual in the sample, one for each of no coverage, Medicaid, and private

[1] One month for each wave of the '96 panel.

Figure 5.1: Pre-reform Probability of Insurance Coverage - Single Mothers on Welfare/Medicaid



insurance. Finally, using the SIPP panel weights, I construct the weighted average probability of coverage by each of the insurance types across all individuals in the sample. This is done for both the pre and post reform period. Finally, by subtracting the results for the pre-reform period from those for the post-reform, I am left with the weighted average effect of welfare reform on the population of single, low education mothers who were receiving welfare checks and were covered by Medicaid during the first part of 1996.

Figures 5.1 and 5.2 plot the weighted average probability of coverage by each insurance type over various time horizons for the 1996 welfare/Medicaid sample. Figure 5.3 plots the weighted average effect of welfare reform on this population. Table 5.1 provides the effect for time horizons of four months, one year, and three years. Table 5.2 provides estimates of the number of women affected by welfare reform, assuming that 7 million single mothers were enrolled in both welfare and

Figure 5.2: Post-reform Probability of Insurance Coverage - Single Mothers on Welfare/Medicaid

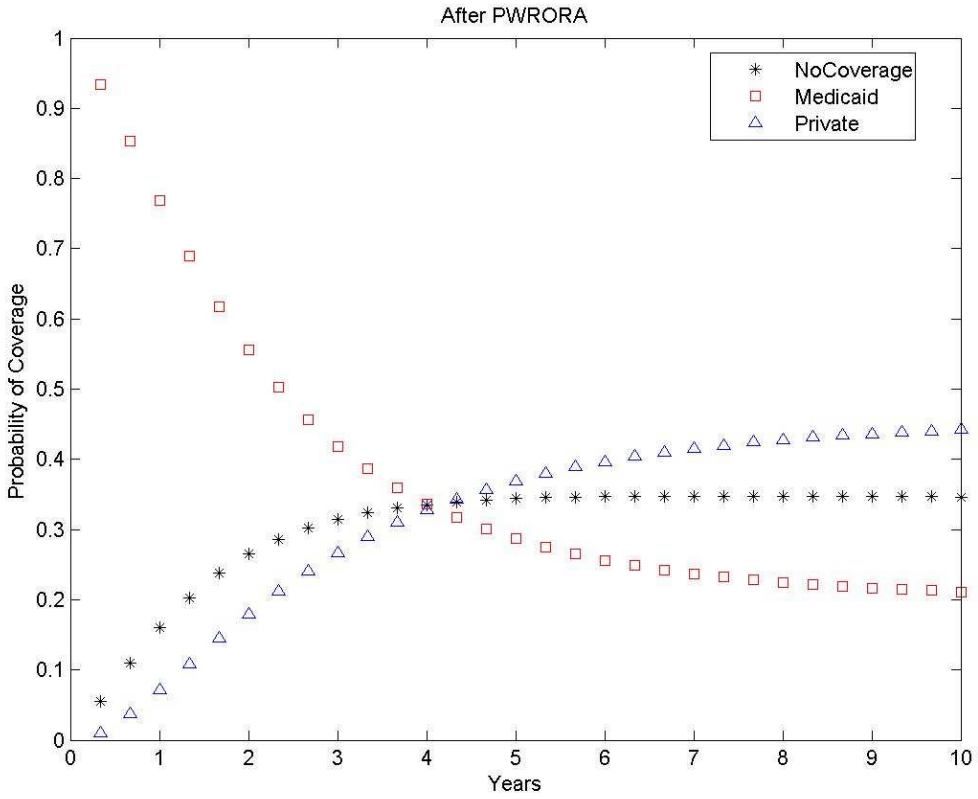


Table 5.1: Average Effect of Welfare Reform (% change) on Insurance Type - Single Mothers on Welfare

	Initial Insurance Type		
	Uninsured	Medicaid	Private
4 Months	3.14	-3.27	0.13
1 Year	8.94	-12.33	3.39
3 Years	13.90	-27.63	13.73

Note: Table reports the percent change single mothers covered by each insurance type over the time horizon given in the left column. Sample consists of single mothers receiving welfare benefits at the beginning of the simulated period.

Medicaid in early 1996.

Figure 5.3: Effect of Welfare Reform on Insurance Coverage - Single Mothers on Welfare/Medicaid

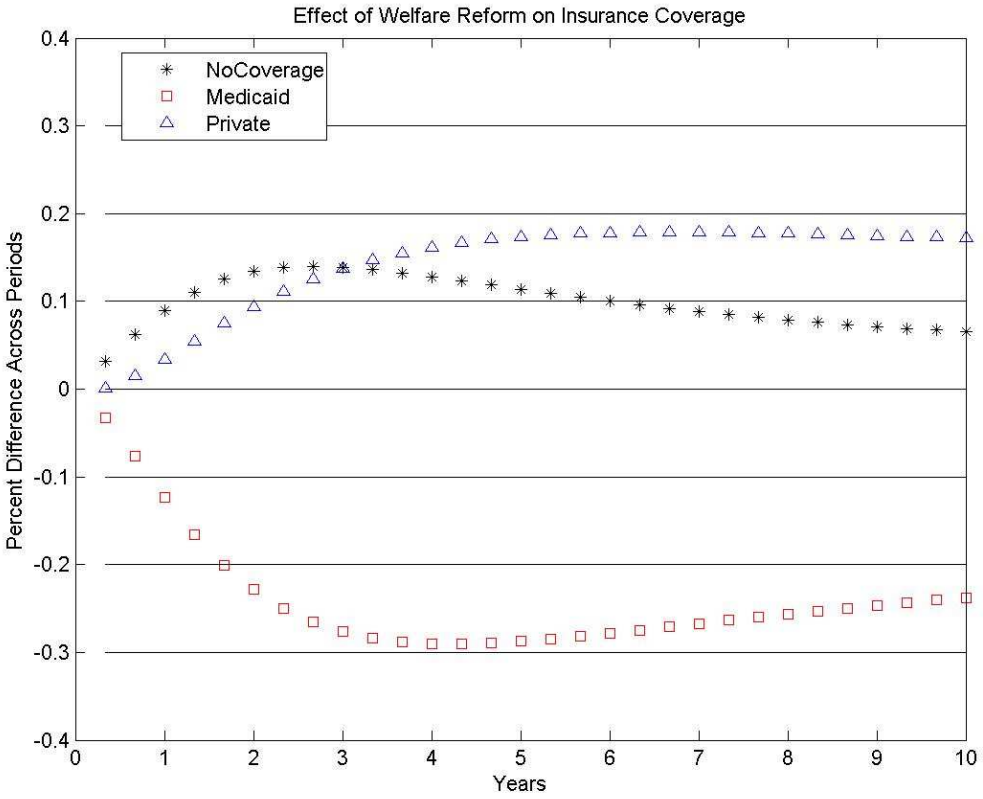


Table 5.2: Number of Single Mothers Affected by Welfare Reform (1000s) assuming 7M on Welfare in 1996

	Initial Insurance Type		
	Uninsured	Medicaid	Private
4 Months	220	-229	9
1 Year	625	-863	238
3 Years	973	-1934	961

Note: Table reports the estimated change in number of single mothers covered by each insurance type over the time horizon given in the left column. Estimates based on 7 million single mothers receiving welfare in 1996.

5.2 Disaggregation of Welfare Effect

In this section I disaggregate the results reported in Figure 3.5. Results for each insurance type are generated by summing across employment/insurance

states with the same insurance coverage. For example, the time series of the effect of welfare reform on "no coverage" is the combination of the effect on two employment/insurance states: no working/no welfare/no coverage and working/no coverage. In this section I report these effects separately.

Figure 5.4: Decomposition of the Effect of Welfare Reform: No Coverage

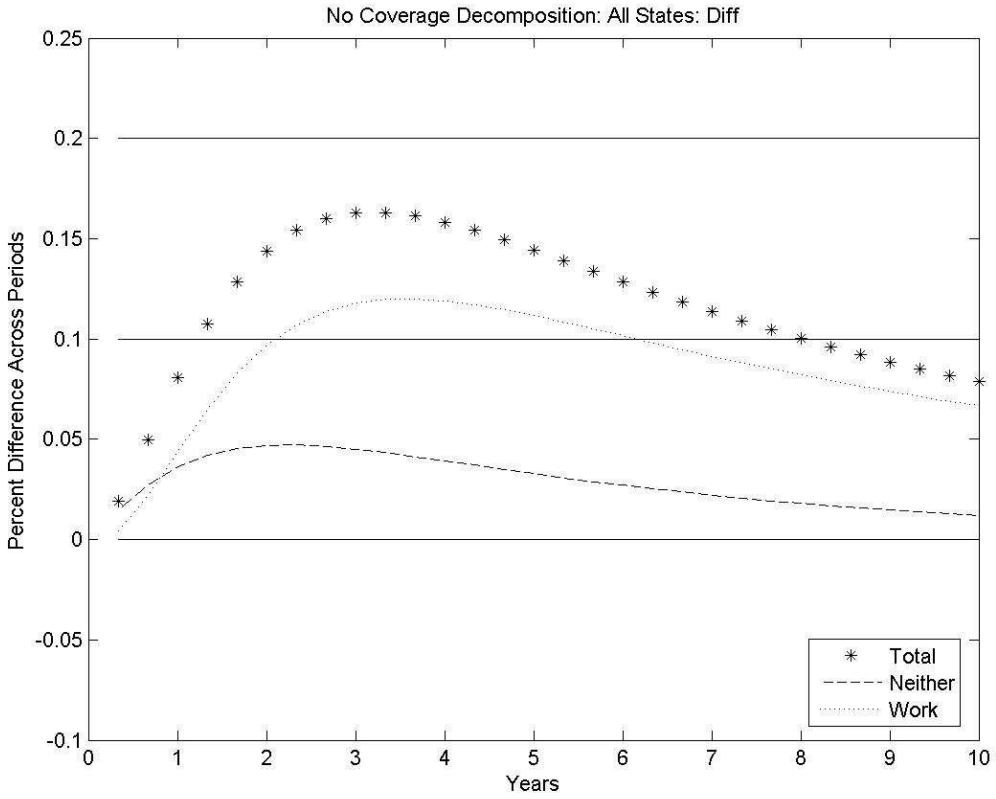


Figure 5.5: Decomposition of the Effect of Welfare Reform: Medicaid

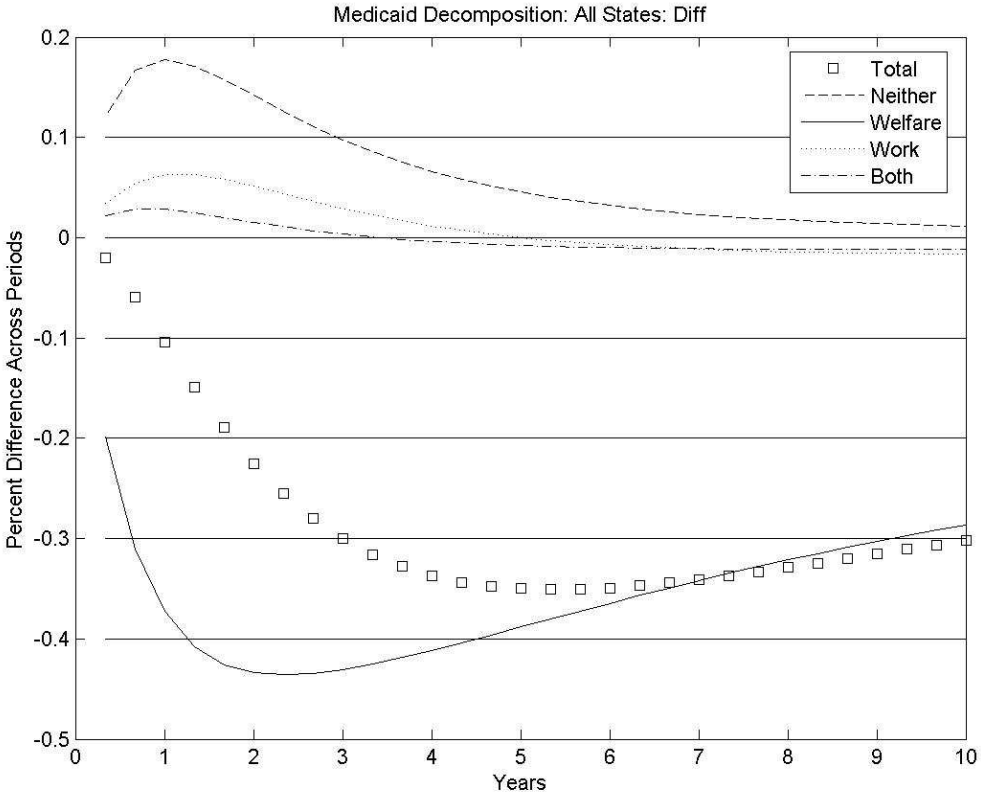


Figure 5.6: Decomposition of the Effect of Welfare Reform: Private Insurance

