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CONTINGENT CLAIM PRICING WITH APPLICATIONS TO FINANCIAL RISK MANAGEMENT

By

Hua Chen

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree

Of

Doctor of Philosophy

In the Robinson College of Business

Of

Georgia State University

GEORGIA STATE UNIVERSITY

ROBINSON COLLEGE OF BUSINESS

2008

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ACCEPTANCE

This dissertation was prepared under the direction of Hua Chen's Dissertation Committee. It has been approved and accepted by all members of that committee, and it has been accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Robinson College of Business of Georgia State University.

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ABSTRACT

CONTINGENT CLAIM PRICING WITH APPLICATIONS TO

FINANCIAL RISK MANAGEMENT

BY

Hua Chen

2008

Committee Chair: Samuel H. Cox and Shaun Wang

Major Academic Unit: Department of Risk Management and Insurance

This is a multi-essay dissertation designed to explore the contingent claim pricing theory with non-tradable underlying assets, with emphasis on its applications to insurance and risk management. In the first essay, I apply the real option pricing theory and dynamic programming methods to address problems in the area of operational risk management. Particularly, I develop a two-stage model to help firms determine optimal switching triggers in the event of an influenza epidemic. In the second essay, I examine mortality securitization in an incomplete market framework. I build a jump-diffusion process into the original Lee-Carter model and explore alternative model with transitory versus permanent jump effects. I discuss pricing difficulties of the Swiss Re mortality bond (2003) and use the Wang transform to account for correlations of the mortality index over time. In the third essay, I study the valuation of the non-recourse provision in reverse mortgages. I model the various risks embedded in the HECM program and apply the conditional Esscher transform to price the non-recourse provision. I further examine the premium structure of HECM loans and investigate whether insurance premiums are adequate to cover expected claims.

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1

Introduction and Overview

A contingent claim, or derivative security, is defined as a financial instrument whose future payoffs are contingent on the behaviors of some “underlying” assets. The theory of contingent claim pricing is the cornerstone of the modern theoretical and empirical research in financial economics. It is designed to measure risk and assign appropriate premiums for risk bearing. Contingent claim pricing, therefore, is inextricably linked with risk analysis and risk management.

The research on contingent claim pricing is especially important to practitioners in financial markets, because it has shed new insights into how potential financial derivatives might be priced and hedged. With the rapid development of the contingent claim pricing theory, there are more derivative securities to be designed and introduced to the market, and firms are more willing to use these securities as a tool to hedge financial risks they are facing with. The

explosive growth of financial derivative market drives the research on contingent claim pricing even further, because the goal of this research is to improve the pricing and hedging of these securities. The purpose of my dissertation is to explore the theory of contingent claim pricing with emphasis on its application to the field of financial risk management.

Contingent claim pricing theory has a long and illustrious history, which can be traced back to Bachelier (1900). He was the first to model the fluctuations of assets prices by the Brownian motion and attempt to price derivatives. Unfortunately, the impact of Bachelier's work was not recognized by economists for over fifty years until key contributions were made by Samuelson (1965) and Samuelson and Merton (1969). They model stock prices using the geometric Brownian motion, which reflects the limited liability (non-negative) property of share prices. In the early 1970s, when Black, Scholes and Merton applied the geometric Brownian motion and Ito calculus to the problem of option pricing and hedging, a completely definite and rigorous theory of option pricing was created. The option pricing theory facilitates the subsequent rapid expansion in the trading of financial derivatives, and leads directly to a general theory of contingent claim pricing related to a wide variety of financial instruments.

Contingent claim pricing theory is established on the non-arbitrage condition, that is, the absence of risk-free opportunities for making profits without any initial investment. For some derivative securities, such as forward contracts, the payoffs can be replicated by the underlying asset and a risk-free asset using a static trading strategy. Under this circumstance, the absence of arbitrage can lead to an exact pricing formula without any additional assumption. For other types of derivatives including options, the no-arbitrage restriction can only determine bounds on the option's price. An additional assumption regarding the probability distribution of the underlying asset's return is necessary for valuing such derivative contracts. Assuming the underlying's

return follows a continuous-time diffusion process and investors can trade continuously, Black, Scholes and Merton show that a portfolio can be created to dynamically replicate the payoff and fully hedge the risk of the contingent claim. In the absence of arbitrage, the hedge portfolio's return must be riskless, which results in a partial differential equation (PDE) the contingent claim's price must satisfy. Solving this PDE subject to appropriate boundary conditions determines a unique price for the contingent security.

Contingent claim's price can also be derived using the martingale pricing approach, which was developed by Cox and Ross (1976), Harrison and Kreps (1979), and Harrison and Pliska (1981). They argue that in a complete market, the non-arbitrage condition ensures the existence of a unique risk-neutral measure (equivalent martingale measure) such that the discounted underlying asset's price is a martingale under this measure. The contingent claim's price can be obtained by calculating the expected payoff under the risk-neutral measure and discounting it back to time zero using risk-free rates. Of course, under these conditions the stochastic discount factor (pricing kernel) also exists uniquely. We can get the contingent claim's price by calculating the expected payoff under the physical probability distribution and discounting by the stochastic discount factors.

A direct application of the optional pricing theory is the regime switching problem, which is pioneered by Mossin (1968) and generalized by Brennan and Schwarz (1985) and Dixit (1989). They defined the concepts of option to enter and option to abandon as part of the firm's value, and solve the entry and exit trigger prices as indicators for firms' decision policies. Their work was extended in many ways and finalized by Brekke and Oksendal (1994). In my first essay, "An Option-Based Operational Risk Management on Pandemics", I apply the real option pricing theory to the area of operational risk management. Particularly, I propose a two-stage model to

help firms determine the optimal switching triggers in the event of an influenza pandemic. This research is motivated by current concerns about the possible outbreak of avian influenza pandemics and the Center of Disease Control and Prevention (CDC)'s instructions to "set up authorities, triggers, and procedures for activating and terminating the company's response plan, altering business operations (e.g. shutting down operations in affected areas)". [1] In the first stage, I propose a regime-dependent epidemic model to simulate the spread of the virus, depending on whether the firm is active or inactive. In the second stage, I view the reactivation decision as a call option and the suspension decision as a put option, and use the dynamic programming approach to determine the optimal switching thresholds. Numerical examples and sensitivity analysis are provided to illustrate the effects of a flu attack.

Influenza pandemics not only bring substantial financial losses to businesses because of the interruption of regular operations, but also have a severe impact on life insurers or reinsurers. According to Weisbart (2006), a moderate influenza pandemic similar to the 1957 and 1968 outbreaks could cost U.S. life insurers $31 billion in additional death claims, and a severe one similar to 1918 pandemic could cost up to $133 billion. How to hedge the mortality risk caused by catastrophic events, such as influenza pandemics, becomes a key issue for the life insurance industry. Traditionally, these risks are shared by insurers or reinsures. More recently, mortality securitization creates a new way of transferring mortality risks to capital markets. The markets for mortality bonds are far from complete, since the underlying is a public mortality index based on a certain population, which is not traded in the market. In a complete market, there exists a self-financing trading strategy which can replicate all contingent claims at any time. As a result, there exists one and only one equivalent martingale measure that we can use to price securities.

[1] See CDC. 2005. Business Pandemic Influenza Planning Checklist. Available at http://www.pandemicflu.gov/plan/business/businesschecklist.html

In an incomplete market, however, there are non-attainable contingent claims. In other words, there are cash flows which cannot be replicated by self-financing trading strategies. How to value the mortality-linked securities and hedge mortality risks in an incomplete market attracts my attention.

There are mainly two approaches for security valuation in an incomplete market. The first method extends the risk-neutral pricing to an incomplete market (see Milevsky and Promislow, 2001; Dahl, 2004; Dahl and Møller, 2005; Miltersen and Persson, 2005). The financial economic theory tells us the arbitrage-free condition ensures the existence of risk-neutral measures, even in an incomplete market. However, the risk-neutral measure is not unique in this case. The problems are which risk-neutral measure should we pick for valuation purposes, and how can we construct hedging strategies for non-attainable contingent claims to "minimize the risk"? Cairns, Blake and Dowd have a detailed discussion on this issue and take the EIB longevity bond as an example (see Cairn, Blake and Dowd, 2006a, 2006b).

With the convergence of financial and insurance markets, there has been a major increase in the trading and securitization of insurance risks. Techniques used in insurance pricing are being considered for pricing derivative contracts in financial markets. The second approach uses a distortion operator to create a risk-adjusted distribution, and obtain the fair value of securities under this risk-adjusted measure. There are a few distortion operators we can choose from, such as the Wang transform (Wang, 2000, 2002, 2003) and the Esscher transform (Esscher, 1932). Examples of using the Wang transform include Lin and Cox (2005), Dowd, Blake, Cairns and Dawson (2006), Denuit, Devolder and Goderniaux (2007), and the example of employing the Esscher transform can be seen in Siu, Tong and Yang (2004), Li, Boyle, Hardy and Tan (2007). Both of the distortion operators have attractive features and are widely used in pricing insurance

and financial products. I will focus on the distortion approach here.

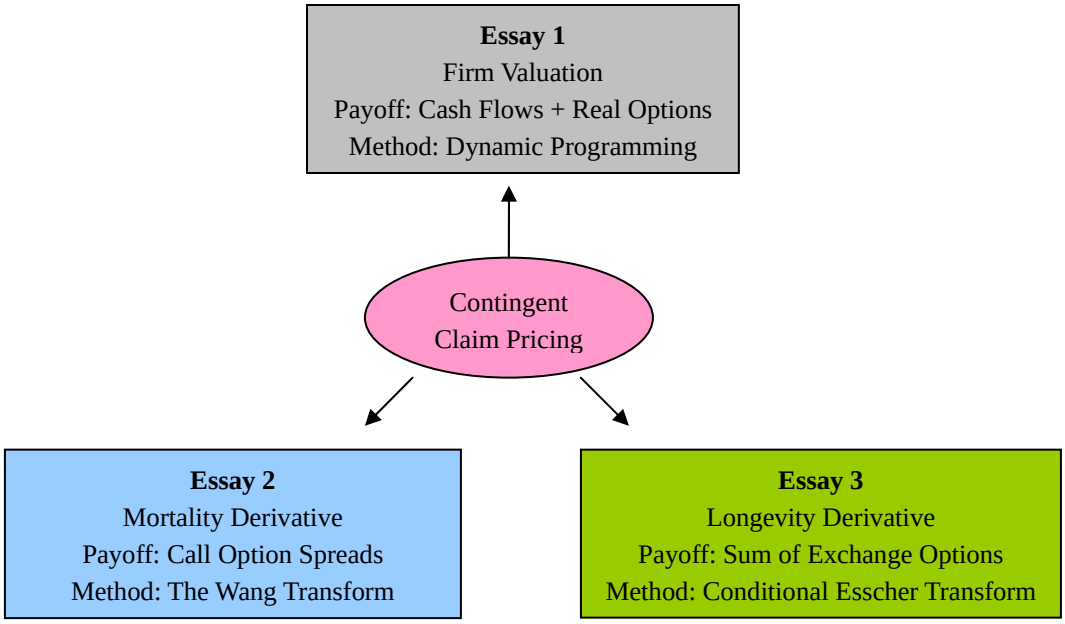
My second essay studies the design and valuation of mortality securitization in an incomplete market framework. In this essay, I propose a generalized Lee-Carter model with transitory jump effects, in order to capture the mortality deterioration caused by catastrophic events. I also examine alternative models with permanent jump effects, and find that modeling mortality via permanent jump effects underestimates the market price of mortality risk. I use the Swiss Re mortality bond in 2003 as an example to show how to apply my mortality model and the Wang transform to value mortality-linked securities. Pricing the Swiss Re mortality bond is difficult because the mortality index is correlated across countries and over time. Cox, Lin and Wang (2006) employ normalized multivariate exponential tilting to take into account correlations across countries. I show in this essay how to account for correlations of the mortality index over time by simulating the mortality index and changing the measure on paths.

We have to admit that mortality deterioration caused by catastrophic events is perhaps a one-in-100 years' incidence. However, one can observe a clear trend of mortality improvement over the past hundreds of years. It has also been evident that mortality is improving in a stochastic way. Longevity risk is referred to as the uncertainty in the aggregate morality improvement. The mortality improvement place strains not only on fiscal sustainability of social security systems, but also on individuals' capacity to accumulate enough private savings to finance their retirement. The reverse mortgage, as one financial innovation to provide elderly homeowners cash flows until their deaths, has attracted public attention. My third essay examines the mechanism of the reverse mortgage, and analyzes various risks embedded in its non-recourse provision. Particularly, I extend the generalized Lee-Carter model with permanent jump effects to capture the longevity risk, and model the house price index in the GARCH

framework. I employ the conditional Esscher transform to price the non-recourse provision, which can be written as a series of exchange options with different times to maturity. I further examine the premium structure of reverse mortgage loans and investigate whether insurance premiums are adequate to cover expected claims. I find that the Federal Housing Administration (FHA) collects higher premiums than normally needed, on average, and the Home Equity Conversion Mortgage (HECM) program is sustainable.

To sum up, my three essays study the contingent claim pricing theory and its applications to financial risk management. The structure of my thesis can be viewed clearly in Figure 1.1.

Figure 1.1: The Structure of My Thesis



I contribute to the existing literature in several aspects. In my first essay, I make the first attempt to apply real option pricing to help a firm make the operational decisions when an influenza pandemic severely depletes its work force. I illustrate how firms' values might be affected by the embedded real options, and offer an applicable method for large businesses to implement suspension-reactivation strategy. An unexpected influenza pandemic may also impose

great challenges on life insurance industries. In my second essay, I propose a generalized Lee-Carter model with jumps to capture extreme mortality events and apply the proposed model in pricing mortality bonds via the Wang Transform. In addition, I present a pricing strategy that accounts for the correlation of the mortality index over time. Mortality improvement is on the other side against mortality deterioration. In my third essay, I show that my proposed mortality model can also be used to capture the rare longevity events, but it should be adapted with permanent jump effects. I analyze the risks underlying reverse mortgage products and price the non-recourse provision using the conditional Esscher transform.

2

An Option-based Operational Risk Management on Pandemics

In a financial market, a call option gives the buyer the right but not the obligation to buy a stock (or other underlying assets) at a specified price (strike price) on a specified date (maturity date). The holder determines whether or not to exercise the option on the maturity date when the stock price is observed. The option is more valuable when there is more uncertainty, i.e., when the volatility term of the underlying asset is greater. We live in an uncertain world. Business managers need to make strategic investment decisions with an unforeseen future. A firm with an opportunity to invest is holding an option analogous to a financial call option—it has the right but not the obligation to buy an asset at some future time of its choosing. Business managers are making contingent decisions—decisions to invest or disinvest that depend on unfolding events. To address the analogy with options on financial assets, the opportunities to acquire real assets

are sometimes called “real options”. In light of real options that seem to be everywhere, nowadays theorists and practitioners unanimously believe that we should consider these real options when analyzing corporate decisions. The celebrated Black-Scholes option pricing theory can be adapted for applications to strategic investments in the real world. In this chapter, I employ the real option approach to study firms’ optimal strategic decisions in the event of an influenza pandemic.

2.1. Introduction

There were a total of 31 pandemics occurring in the past 500 years and 3 in the past century, of which the 1918-1919 “Spanish flu”, the most severe one, killed up to 50 million people worldwide and 500,000 in the United States (Rasmussen, 2005). Historic data have shown that influenza pandemics happen with frightening regularity and occur every 30 to 50 years (Knapp, 2006). Given this pattern, the possibility of another pandemic attack is not considered remote. Ever since the isolated outbreaks of avian influenza in 2003, scientists have been particularly worrying about the influenza A (H5N1) virus.

The CDC predicts 2 to 7 million deaths and medical treatment for tens of millions people even in a moderately severe scenario (Jia and Tsui, 2005). Although pandemics attack the insurance industry severely, their impacts on any other business organizations are startling as well. Businesses would probably be shut down for the purpose of quarantine; people would be reluctant to expose themselves to crowds; corporate earning would plunge which would further trigger defaults on corporate debts; besides, there is a likely crash in consumer confidence. The World Bank claims if the impact of a moderately severe pandemic were to last for a year, the economic loss would be $100 to $200 billion for the U.S., and around $800 billion globally

(WBEAPR, 2005). The Congressional Budget Office (CBO, 2005) estimates that a severe influenza pandemic would result in a loss of 5 percent of GDP compared with what it would otherwise have been.[2]

Dynamics of human epidemics is an important topic in epidemiology and mathematical biology. An enormous literature has been developed in this field, the history of which can be traced back to pioneers such as Kermack and McKendrick (1927). Ever since the publication of Bailey (1957), mathematical epidemiology has grown rigorously. A wide variety of epidemic models have been mathematically formulated, analyzed and empirically fitted (see reviews in Dietz, 1967; Wickwire, 1977; Becker, 1978; Dietz and Schenzle, 1985; Hethcote, 1994). Basically, they can be classified into two main streams: deterministic models and stochastic models. In essence, the spread of a disease through a given population is a stochastic process. Nevertheless, deterministic models may often be used to obtain acceptable approximations for relatively large populations. Whereas these deterministic models are relatively easy to work with using numerical methods, I prefer stochastic models since they usually provide more information about the intrinsic variability of the system.

Recently, new interest arises in research using epidemic modeling as a decision aid for optimal control policies such as immunization, worker furloughs, and quarantines. Based on a deterministic epidemic model, Finkelstein, Smart, Gralla and d'Oliveira (1981) construct a decision system under which alternative public immunization strategies can be compared. They find that vaccinating the population at large is sometimes favored over targeting at the highest-risk groups. Meltzer, Cox and Fukuda (1999) employ Monte Carlo mathematical simulations and reach the same conclusion. Jia and Tsui (2005) use SARS as a case study to quantify the impact

[2] Congressional Budget Office. 2005. *A Potential Influenza Pandemic: Possible Macroeconomic Effects and Policy Issues*.

of various control policies. These models, however, only evaluate the effectiveness of control measures on pandemics based on national needs, and conduct cost/benefit analysis from the macroeconomic perspective. They provide neither operating instructions for large businesses to prepare for pandemic risks, nor any insights as to the triggers for implementing optimal control strategies.

Operational risk is defined as the risk of loss resulting from inadequate or failed internal processes, people and systems, or from external events (Basel Committee, 2004).[3] Influenza pandemic is one of the examples. In the event of influenza pandemics, businesses play an especially important role in protecting employees' health as well as minimizing the economic losses to the whole society. Business continuity planning has become a key component of operational risk management. It emphasizes the maintenance of critical operations and services during a crisis or a timely recovery of business after a disruption. Companies that provide infrastructure services, such as power and telecommunications, should devote significant resources to ensure continued operations during a crisis. Firms in financial sectors, like banks or insurance companies, also have a special responsibility to plan for business continuity and maintain the stability of the financial system. In order to assist businesses to plan for the outbreak of a pandemic, the HHS (Department of Health and Human Services) and the CDC has developed a checklist, which identifies necessary activities for large businesses to prepare for the impact and establish policies for implementation during a pandemic.[4] In particular, it requires businesses to "set up authorities, triggers, and procedures for activating and terminating the company's response plan, altering business operations (e.g. shutting down operations in affected

[3] Basel Committee on Banking Supervision. 2004. International Convergence of Capital Measurement and Capital Standards. Bank for International Settlements.

[4] CDC. 2005. Business Pandemic Influenza Planning Checklist. Available at http://www.pandemicflu.gov/plan/business/businesschecklist.html

areas)."

This essay is motivated by current concerns about the possible outbreak of avian flus and the CDC's instructions for large businesses. Basically, these are the questions I want to address:

- In the event of an infectious disease such as an influenza epidemic, should a profit maximizing firm continue to operate with the loss of productivity of its employees, or suspend the business (or parts of its business) temporarily in order to avoid contagion?
- Does a firm's intention to maximize its value contradict with the purpose of controlling the disease?
- And most important of all, what are the optimal triggers for firms to implement the suspension-reactivation strategy?

I propose a two-stage model in order to answer these questions. The intuition is straightforward. In the first stage of the model, I adapt a simple susceptible-infective (SI) model to describe the dynamics of an epidemic that spreads in a given company (or parts of its businesses). I accommodate external contagion and possible deaths in this model and modify it to a stochastic process. The productivity of an employee is reduced once he/she gets infected. The disease will spread and the fraction of the infective is increasing over time, which diminishes the revenue of the company due to the decrease in average productivity. When the fraction goes above a certain high threshold (mothballing threshold thereafter), the manager may want to temporarily suspend its business (or parts of its business in the most affected areas) and send employees, whether infected or non-infected, back home. The separation of the employees may help to control the disease. When the fraction of the infective drops to a certain low threshold (reactivation threshold thereafter), the manager may want to call the employees back to work and continue the business. Therefore, a regime-switching model is employed in the second

stage to determine these optimal switching thresholds. The regime switching model is based on the theory of real option valuation and can be solved by dynamic programming methods. I will discuss this in detail in the methodology part.

My results suggest that given the parameter values in this study, it is optimal for the firm to lay up the business (or parts of its business) when the fraction of infected employees is higher than 27%, and to reactivate the operation when the fraction drops to 5%. Considering uncertainty in the future, firms are more conservative about the decisions of suspension and reactivation. Upon the condition that firms incur lump sum costs when switching between regimes, sensitivity analysis shows that the mothballing threshold increases with the switching costs. On the contrary, the reactivation threshold decreases with the costs. By implementing disease control measures, firms can increase their values in both regimes, and thereby control contagions at the same time as maximizing their profits.

The rest of this chapter is organized as follows. In section 2.2, I modify the *SI* model to describe the spread of the virus in a large bushiness. In section 2.3, I discuss the real option pricing and the regime switching model. Dynamic programming is presented as a methodology in section 2.4. I provide numerical results based on the modified *SI* model and extend the analysis to the modified *SIR* model in section 2.5 and 2.6. Conclusions and discussions are given in section 2.7.

2.2. Stage I: The Regime-Dependent Stochastic Epidemic Model

In this section, I adapt a simple *SI* model to simulate the spread of the virus, which may depend on the regime that the firm is currently in. It is noteworthy that although I consider the case of a manufacturing firm here, the same model can be applied to firms providing critical

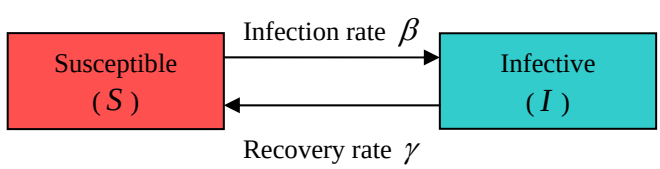
services, such as infrastructure services and financial services. Actually, it is more reasonable to regard the firm as a division or parts of a large business that is in the affected areas.

2.2.1. The Susceptible-Infective (SI) Model

Almost all epidemic models share the common feature, that is, dividing the modeled population into different groups (susceptible, infective, and recovered, etc), and studying the disease transmission between different groups. The simplest epidemic models only consider two categories: susceptible (S) and infective (I). The class S denotes the group of individuals who are healthy but susceptible to a certain disease. In an environment exposed to the virus, some people may be infected with the disease (with or without symptoms) and are capable of transmitting the disease to others. These people are considered in the class I. I refer readers to Bailey (1957) and Bartholomew (1973) for more details.

The SI model considers a mild, short-lived epidemic (e.g. influenza) in a closed population. That is, there is no entry into or departure from the population. Given the time scale of influenza epidemics, demographic turnover (birth or death) is not considered. Moreover, the *SI* model assumes that the disease does not confer immunity. In other words, the recovered people may have access to the virus and are likely to get infected again. The transition dynamics of the *SI* model is illustrated in Figure 2.1.

Figure 2.1: The Simple *SI* Model



Let S_t and I_t denote the fractions of population that reside in the state S and I,

respectively, as a function of time, the dynamics of the disease evolves as follows:

$$\begin{cases} dI_t = (\beta I_t S_t - \gamma I_t)dt \\ S_t = 1 - I_t \end{cases}, \tag{2.1}$$

or,

$$dI_t = [\beta I_t (1 - I_t) - \gamma I_t]dt . \tag{2.2}$$

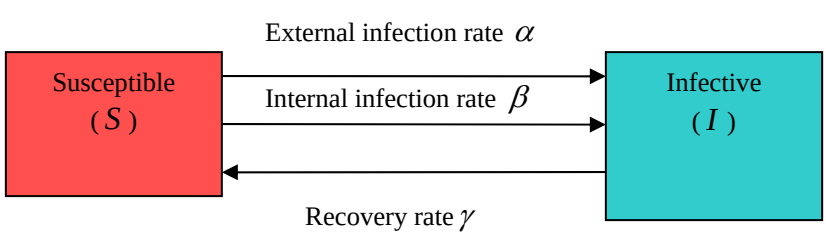
The transition rate β between the state S and the state I is referred to as the infection rate within the population, which is simply the rate at which susceptible individuals become infected by an infectious disease. The transition rate γ is called the recovery rate, which is the inverse of the average duration of the infective period.

2.2.2. The Modified SI Model Allowing for External Contagions

Note that I am considering a large business organization instead of the whole population. The spread of the disease within the business is not only affected by the interaction between the susceptible and the infective employees in the firm, but also influenced by some external sources. The susceptible employees are inevitably getting in touch with the outside world, and thereby are subjected to infection. I use α denote the infection rate from external sources. Within a small time interval of length Δt, the additional increase of the infective from the external infection should be $\alpha S_t \Delta t$. The dynamics of the disease within the firm can be expressed as follows:

$$dI_t = [\beta I_t (1 - I_t) - \gamma I_t + \alpha(1 - I_t)]dt . \tag{2.3}$$

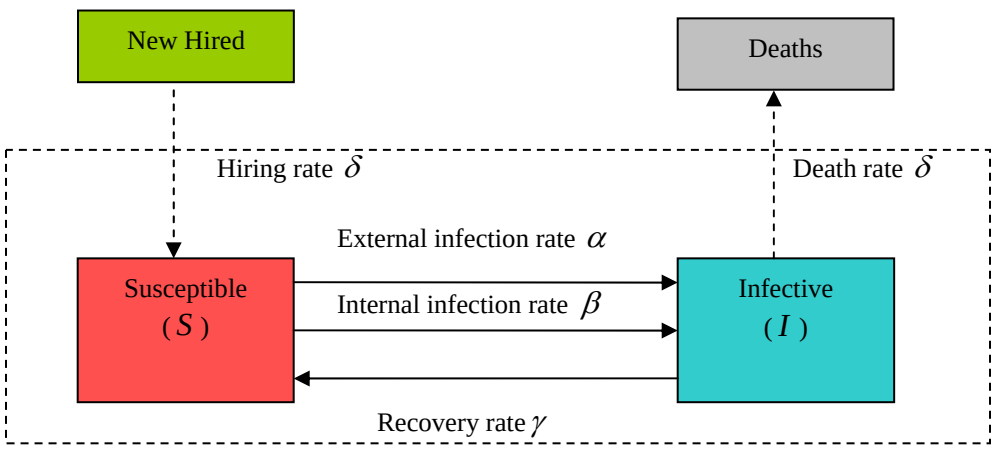
Figure 2.2: The Modified *SI* Model Allowing for External Contagions



2.2.3. The Modified SI Model Allowing for External Contagions and Deaths

When considering a mild epidemic and a relatively large population, the assumption that there is no death from the disease is reasonable, and the SI model can often be used to obtain acceptable approximations. However, if we are faced with a severe pandemic like the 1918 flu, or when we consider the disease transmission in a large business, we need to modify the classical SI model to allow for deaths caused by the disease.

Figure 2.3: The Modified *SI* Model Allowing for External Contagions and Deaths



Assume that the firm can hire new employees in order to make the working force constant, and the new employees fall into the class S. Let δ be the constant rate of fatality in the class I, the modified SI model allowing for external contagions and deaths is as follows:

$$dI_t = [\beta I_t(1-I_t) - \gamma I_t + \alpha(1-I_t) - \delta I_t]dt\,. \tag{2.4}$$

2.2.4. The Modified Stochastic SI Model

To convert the deterministic model to a stochastic model, we have to introduce an assumption concerning the diffusion function. The most reasonable assumption is that the random variation is greater in the center region than in the extreme cases. This suggests that the

variation term is proportional to $I_t(1-I_t)$. The change in I_t follows the rule

$$dI_t = [\beta I_t(1-I_t) - \gamma I_t + \alpha(1-I_t) - \delta I_t]dt + \sqrt{cI_t(1-I_t)}dW_t, \tag{2.5}$$

where c is a small positive constant, and W_t a standard Brownian motion.

2.2.5. The Regime-Dependent Modified Stochastic SI Model

The above discussions are based on the assumption that the firm is active and does not implement disease control policies. Note that the evolution of the epidemics may tie up with the strategic decisions the manager makes. Firms can use some disease control policies to alter the value of parameters such as β and γ, and reduce the spread of the contagion. For instance, when the epidemic breaks out, the firm may adopt some immunization programs to lower the infection rate between the infective and the susceptible. When some employees in the company get infected, the manager can screen the suspected infective and mandate the immediate full-paid leave. Particularly, if the number of the infective increases and hits a predetermined point, the firm can temporarily suspend the business and send all the employees back home. I focus on the analysis of the suspension-reactivation strategy in this study. Therefore, I adapt the modified *SI* model further to accommodate two regimes: active and inactive.

I denote by r the regime of the firm ($r=1$ if the firm is active and $r=2$ if the firm is inactive). Let γ_1 be the recovery rate when firm is active. The corresponding epidemic model is

$$dI_t = [\beta I_t(1-I_t) - \gamma_1 I_t + \alpha(1-I_t) - \delta I_t]dt + \sqrt{cI_t(1-I_t)}dW_t, \qquad \text{if } r=1. \tag{2.6}$$

When the firm is inactive, the person-to-person transmission from internal sources is cut off (i.e., $\beta = 0$). The contagion will be controlled and the infective will be recovered with a higher recovery rate γ_2 ($\gamma_2 > \gamma_1$). Assuming the external infection rate α and the death rate δ remain the same, the dynamics of the disease when the firm is inactive turns out to be

$$dI_t = [-\gamma_2 I_t + \alpha(1-I_t) - \delta I_t]dt + \sqrt{cI_t(1-I_t)}dW_t, \qquad \text{if } r = 2 . \tag{2.7}$$

To conclude, the epidemic model can be described as

$$dI_t = \mu(I_t, r)dt + \sigma(I_t, r)dW_t , \tag{2.8}$$

where

$$\mu(I_t, r) = \begin{cases} \beta I_t(1-I_t) - \gamma_1 I_t + \alpha(1-I_t) - \delta I_t, & \text{if } r = 1 \\ -\gamma_2 I_t + \alpha(1-I_t) - \delta I_t, & \text{if } r = 2 \end{cases}, \tag{2.9}$$

and

$$\sigma(I_t, r) = \sqrt{cI_t(1-I_t)} . \tag{2.10}$$

2.3. Stage II: Real Option Pricing and Regime-Switching Models

2.3.1. Real Option Pricing

In finance, the net present value (NPV) approach serves as the building-blocks for most analysis, such as investment decisions, capital structure decisions, and valuation of firms. However, as recognized by most researchers and practitioners, the traditional NPV approach fails to account for real options that are embedded in business strategic decisions, and thereby always underestimates firm value. Implicitly, the NPV approach assumes that "either the investment is reversible" or "if the investment is irreversible, it is a now or never proposition" (Dixit and Pindyck, 1994). That is, firms have the ability to withdraw the investment project and recover all the initial costs if the economic situation turns adversely. If the firm does not undertake the investment now, it will not be able to implement it in the future. However, investment opportunities in the real world do not always meet these conditions. Most investments are partially or completely irreversible. In other words, investment expenditures become sunk costs once firms make the move, and firms cannot retrieve the costs in full amounts later on. In

addition, firms have the managerial flexibility to defer the investment until new information arrives. Therefore, firms can analyze the desirability of the investment project further using the newly acquired information. The ability to delay an irreversible investment can affect the decision to invest, and thereby undermines the simple NPV rule (Dixit and Pindyck, 1994). A firm with an investment opportunity is analogous to holding a call option. It can either exercise the option (make the investment), or hold the option and wait for new information arriving. When the firm chooses to invest right now, it gives up the option value—possibility of waiting for new information that might affect the investment decision itself. The lost option value is an opportunity cost that must be included when we calculate the NPV. Real options are inherently present in any strategic decision where firms have the managerial flexibility to alter its course, i.e. expansion, contraction, delay, or abandonment (Brealey, Myers and Marcus, 2001).

2.3.2. Regime-Switching Models

Regime-switching models often arise in real option valuation, the history of which can be traced back to Mossin (1968). The simplest regime-switching models are optimal stopping problems, with the American option pricing as a well known example. More complicated models are later discussed by McDonald and Siegel (1986), allowing for movement back and forth among different regimes. Brennan and Schwarz (1985) apply the Black-Scholes formula to value the active and inactive firms. They argue that the inactive firm has the option to invest and its value is equivalent to the value of this call option, with strike price equal to the entry cost. Likewise, the active firm has the option to exit the market and its value is determined by its current profit and the option to abandon. Considering the value-matching conditions and the smooth-pasting conditions, Dixit (1989) further obtains a pair of price thresholds for the entry-

exit decisions. During the 1990's, the baseline Dixit's model was extended in many directions. Allowing for the possibility of laying-up or scrapping the project, Dixit and Pindyck (1994) consider four price thresholds for investment, laying-up, reactivation and scrapping. Ekern (1993) relaxes the extreme assumption of complete irreversibility, and assumes restricted number of switches between states. Brekke and Oksendal (1994) introduce diminishing production capacity over time into the model and solve the model as a special case of sequential optimal stopping problem. Bar-Ilan and Strange (1996) consider the time delay between the decision to invest and the start of the production, and include one more state of nature (under construction) into the model.

2.3.3. Regime-Switching in My Example

Basically, the essence of regime-switching models is to determine the optimal switching thresholds across different regimes in order to maximize a certain value function. Suppose that the manager cannot tell if an employee is infected individually (it may be due to lack of expertise, or too costly to do so), but he has some technique that can help him know the fraction of the infective I_t .[5] The manager wants to determine two optimal switching thresholds, I_H (mothballing threshold) and I_L (reactivation threshold), such that if I_t is above I_H, the manager suspends the production temporarily and offers full-paid leave for all employees[6], and if I_t is lower than I_L, the manger calls back all the employees and reactivates the production. I make the above assumptions in order to avoid adverse selection and a moral hazard problem. Otherwise, some of the infective would pretend to behave normally if they cannot get paid

[5] For example, he may use daily released data of disease cases, outpatient visits or hospitalization in this affected region to obtain a proxy of the fraction, which is acceptable especially when the disease is spreading rapidly in the region.

[6] The full-paid leave is suggested by the HHS and the CDC in the Business Pandemic Influenza Planning Checklist.

during the suspended period, and the non-infected employees would pretend to be infected in order to enjoy more leisure without being detected.

I normalize the productivity of a non-infected employee to unity, and assume that the productivity will drop to a given level $k < 1$ once the employee gets infected. N is the total number of employees in the firm. The price of the product, P, can be viewed as given, because I only consider one firm in a competitive market. The variable cost and fixed cost are denoted by VC and FC, respectively. Note that wages for the workers are contained in the fixed cost FC because I consider full-paid leaves. In addition, there is a penalty cost E to the firm for every infected employee when the firm is in the active regime. The penalty cost may come from the employees' complaints and reluctance to work, or the firm's loss of reputation in the future. Therefore the cash flow function can be defined as:

$$\pi(I_t, r) = \begin{cases} [k \times I_t \times N + (1 - I_t) \times N] \times (P - VC) - FC - E \times I_t \times N, & \text{if } r = 1 \\ -FC, & \text{if } r = 2 \end{cases} . \qquad (2.11)$$

It implies that the cash flow to the firm, $\pi(I_t, r)$, is a function of the fraction of the infective, and it also depends on the regime variable.

Suppose the firm incurs a mothballing cost M when switching from the active to inactive regime, and reactivation cost A when switching from the inactive to active regime, but there is no cost to remain in the current regime. Let C_{ij} denote the lump-sum cost of switching from regime i to j. The cost matrix is hence defined as $C = \begin{pmatrix} 0 & M \\ A & 0 \end{pmatrix}$.[7] The discount rate is given as ρ.

The manager desires to maximize the expected discounted cash inflows less any switching costs incurred over an infinite time horizon, by choosing the optimal regime at each moment.

[7] To avoid the possibility of "an infinite money machine", we assume $M + A \geq 0$.

That is,

$$\max_r V(I,r) = E\left[\int_0^\infty e^{-\rho t}\pi(I_t,r)dt - \sum_k \sum_{i=1}^{2}\sum_{j=1}^{2} e^{-\rho t_k^{ij}} C_{ij}\right], \tag{2.12}$$

where t_k^{ij} are the times at which the agent switches from regime i to j.

2.4. The Methodology: Dynamic Programming

2.4.1. Comparison between Dynamic Hedging and Dynamic Programming

To solve the regime-switching problems, we mainly have two techniques. "They are in fact closely related to each other, and lead to identical results in many applications. However, they make different assumptions about financial markets, and the discount rate that firms use to value future cash flows" (Dixit and Pindyck, 1994). The first one is the extension of financial option pricing theory. I call it dynamic hedging approach here. If we can find a portfolio of traded assets which can perfectly replicate the returns from the investment project at any time, the value of the investment project must be equal to the value of the replicating portfolio if there is no arbitrage opportunity. This approach requires a highly liquid market where the risks embedded in the investment opportunity can be dynamically hedged, which can be quite demanding. The second method is the so-called dynamic programming. The beauty of this approach is that decisions over a multiple period horizon can be broken up into a series of decisions over a single period horizon. This allows us to derive the individual's optimal decisions by starting at the last period and working backwards toward the present, if the planning horizon is finite. If the planning horizon is infinite, we can derive a recursive formula for every period. The solution always exists and is guaranteed through numerical approximation. If the underlying assets cannot be traded in markets, dynamic programming still works since the objective value function can simply reflect

the decision maker's subjective value of risk (Dixit and Pindyck, 1994). The disadvantage is that it assumes a constant discount rate ρ, which is specified exogenously.

2.4.2. The Recursive Dynamic Programming

In this section, I briefly discuss the dynamic programming approach. I refer interested readers to Miranda and Fackler (2002) and Fackler (2004) for further discussion. Suppose that there are m regimes (i.e., $r = \{1,2,...,m\}$). An agent obtains cash flow streams $\pi(x,r)$ per unit time, which depends on both the discrete regime variable r and on a continuous state variable x (x can also be a vector of state variables). The dynamics of the state variable(s) x can be described by:

$$dx_t = \mu(x,r)dt + \sigma(x,r)dW_t . \tag{2.13}$$

The agent can move from regime i to j at a lump-sum cost of C_{ij}, but there is no cost to remain in the current regime, i.e., $C_{ii} = 0$. Similarly, in order to avoid the possibility of infinite profits, it must be true that $C_{ij} + C_{ji} \geq 0$. The discount rate is ρ.

Dynamic programming is based on the principle of optimality. As Bellman (1957) says, "An optimal policy has the property that, whatever the initial state and decision are, the remaining decisions must constitute an optimal policy with regard to the state resulting from the first decision." The principle of optimality can formally be expressed in the form of the Bellman equation.

Let us consider for a short time period of length Δt. Denote $V(x,r)$ the maximum attainable sum of current and expected future rewards of time t, given that the agent is in regime r. In the interior of the non-switch regions, the Bellman equation for the discrete time problem

can be written as follows:[8]

$$V(x,r) = \pi(x,r)\Delta t + \frac{1}{1+\rho\Delta t} E_t\left[V(x_{t+\Delta t}, r)\right]. \tag{2.14}$$

Multiplying both sides of equation (2.14) by $(1+\rho\Delta t)/\Delta t$ and rearranging, we can get

$$\rho V(x,r) = \pi(x,r)(1+\rho\Delta t) + \frac{E_t\left[V(x_{t+\Delta t}, r) - V(x,r)\right]}{\Delta t}. \tag{2.15}$$

Taking the limits of this expression at $\Delta t \to 0$ yields the continuous time version of the Bellman equation:

$$\rho V(x,r) = \pi(x,r) + \frac{E_t dV(x,r)}{dt}. \tag{2.16}$$

Equation (2.16) has an intuitive economic interpretation. Note that V can be thought of as the value of an asset on a dynamic project. The Bellman equation states that the total rate of return on the asset in regime r, ρV, must equal the current income flow to the project, $\pi(x,r)$, plus the expected rate of capital gain, $\frac{E_t dV(x,r)}{dt}$.

Denote V_x and V_{xx} the first and the second derivatives of V, respectively. By Ito's Lemma,

$$\begin{aligned} dV(x,r) &= V_x(x,r)dx_t + \frac{1}{2}V_{xx}(x,r)dx_t dx_t, \\ &= V_x(x,r)\left[\mu(x,r)dt + \sigma(x,r)dW_t\right] + \frac{1}{2}V_{xx}(x,r)\sigma^2(x,r)dt \\ &= \left[\mu(x,r)V_x(x,r) + \frac{1}{2}\sigma^2(x,r)V_{xx}(x,r)\right]dt + \sigma(x,r)V_x(x,r)dW_t. \end{aligned} \tag{2.17}$$

Taking expectations on both sides of equation (2.17) and dividing by dt, we obtain

$$\frac{E_t dV(x,r)}{dt} = \mu(x,r)V_x(x,r) + \frac{1}{2}\sigma^2(x,r)V_{xx}(x,r). \tag{2.18}$$

Substituting equation (2.18) into (2.16) results in the following form of the Feynman-Kac

[8] In the interior of the no-switch regions, no action is taken during the interval Δt, so there is no maximization on the right-hand side of this equation.

equation:

$$\rho V(x,r) - \mu(x,r)V_x(x,r) - \frac{1}{2}\sigma^2(x,r)V_{xx}(x,r) = \pi(x,r)\,. \tag{2.19}$$

At the boundary point x^*, supposing it is optimal to switch from regime i to regime j, the value function must satisfy two conditions at such a point. The first is a value-matching condition, which will hold no matter whether the switching points are optimal or not. Namely, the value before switching must be equal to the value after switching less the switching cost:

$$V(x^*,i) = V(x^*,j) - C_{ij}(x^*)\,. \tag{2.20}$$

The second is a smooth-pasting condition that is satisfied at the optimal switching points. That is, the marginal value before switching must equal the marginal value after switching minus the marginal cost of switching. Let C' denote the marginal cost function, the smooth-pasting condition can be expressed as follows:

$$V_x(x^*,i) = V_x(x^*,j) - C_{ij}'(x^*)\,. \tag{2.21}$$

.

2.5. Numerical Illustrations

2.5.1. Parameter Calibration

The purpose of this study is to build a theoretical framework and provide a quantitative approach for business managers to prepare for pandemics. The precise calibration of parameters in the model is important, but beyond the scope of this study. Although “a large number of published sources offer empirical data from previous outbreaks of influenza in the U.S”, Finkelstein et al. (1981) mention that “the various data sources are uneven in quality and do not reflect readily comparable study designs and data collection methodology”. Therefore, I skip the step of parameter estimation from empirical epidemic data, and directly choose the disease

parameter values based on previous empirical work.

Smith and Moore (2000) study the spread of the Hong Kong flu (1968) in New York City and obtain the estimation of the infection rate $\beta = 0.6$.[9] Finkelstein et al. (1981) set β equal to 0.75 for "compatibility with empirical data reported for actual influenza epidemics occurring in 1957 and 1968". Till now, the human-to-human spread of H5N1 has been limited, inefficient and unsustained. Human beings possess little or no immune system against the H5N1 virus. Scientists are worried that the H5N1 virus could be able to mutate one day and spread easily among the population. In order to reflect scientists' concern, I set the infection rate β equal to 1 in this work.

As to the recovery rate, it is defined as the reciprocal of the average number of days of the infective period. The commonly reported duration of influenza ranges from 1 to 5 days, therefore the recovery rate should change from 0.2 to 1. Smith and Moore (2000) use the average duration of 3 days and obtain the recovery rate to be 0.33; Cobb (1998) use the value of 0.4; and Finkelstein et al. (1981) set it equal to 0.5. I select the recovery rate in the active regime, γ_1, to be 0.4, and that in the inactive regime, γ_2, equal to 0.6, to reflect the fact that the recovery rate should be higher when workers are separated from each other. The values of external transmission rate α and the volatility coefficient c are selected based on Cobb (1998), where he suggests $\alpha = 0.02$ and $c = 0.1$.

The Department of Homeland Security predicts 16 million deaths from the attack of a possible H5N1 avian influenza, assuming a mortality rate of 20 percent and 80 million illnesses.[10] Therefore, I set the fatality rate δ to be equal to 0.2.

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[9] Smith and Moore. 2005. The SIR Model for Spread of Disease.
Available at: http://www.math.duke.edu/education/ccp/materials/diffcalc/sir/index.html

[10] Available at: http://www.globalsecurity.org/security/ops/hsc-scen-3_flu-pandemic-deaths.htm

The values of the other parameters used in this paper are set as follows. I choose these values to make economic sense and render the sensitivity analysis in the following sections more evident: [11]

Discount rate: $\rho = 0.05$

Productivity of an infective: $k = 0.5$

Total number of employees: $N = 1000$

Price of the product: $P = 3$

Variable cost: $VC = 1$

Fixed cost: $FC = 500$

Penalty cost: $E = 5$

Mothballing cost: $M = 300$

Reactivation cost: $A = 300$.

2.5.2. The Stationary Probability Distribution of the Epidemic Process

People are concerned by what will happen to the disease if no action is taken, i.e. no regime switching nor other controls. Will it spread over the population or die out gradually? What is the possible fraction of people who are infected by the disease? In order to answer these questions, we need to examine the distribution of I_t and get some statistical information.

Generally, suppose we are working with the variable of interest, x_t. It behaves according to a stochastic differential equation:

[11] For example, we could choose a relatively bigger value for the switching costs. At this time, our economic rational on the sensitivity analysis is still correct. However, we may get a zero reactivation threshold and it remains unchanged when we try to increase the switching cost. We, therefore, may not see the expected effect of increasing the switching costs.

$$dx_t = \mu(x)dt + \sigma(x)dW_t . \tag{2.22}$$

The probability density function of such a random variable, $f(x,t)$, depends not only on the random variable itself, but also on time t. The evolution of the probability density function is presented in the form of the Kolmogorov forward equation:

$$\frac{\partial f}{\partial t} = \frac{\partial}{\partial x}(\mu(x) f(x,t)) + \frac{\partial^2}{\partial x^2}(\sigma^2(x) f(x,t)) .^{12} \tag{2.23}$$

An explicit solution to this equation is not always available in general. Therefore, I turn to the stationary probability density function when the process reaches equilibrium (i.e., $\frac{\partial f}{\partial t} = 0$). Wright (1938) has developed a formula to calculate the stationary probability density function:

$$f(x) = \frac{\psi}{\sigma^2(x)} \exp\left\{ \int_{-\infty}^{x} \frac{\mu(y)}{\sigma^2(y)} dy \right\}, \tag{2.24}$$

where ψ is a constant such that $\int_{-\infty}^{\infty} f(x)dx = 1$.

For the example here, I am interested in the stationary distribution of the variable I_t, where $\mu(I) = \beta I(1-I) - \gamma I + \alpha(1-I) - \delta I$ and $\sigma^2(I) = cI(1-I)$. Substituting into the Wright's formula, I obtain

$$\begin{aligned} f(I) &= \frac{\psi}{I(1-I)} \exp\left\{ \int_0^I \frac{\beta y(1-y) - \gamma y + \alpha(1-y) - \delta y}{cy(1-y)} dy \right\} \\ &= \psi I^{-1+\alpha/c}(1-I)^{-1+(\gamma+\delta)/c} e^{\beta I/c} . \end{aligned} \tag{2.25}$$

[12] See Cobb (1998) for more details.

Figure 2.4: The Stationary Probability Density Function

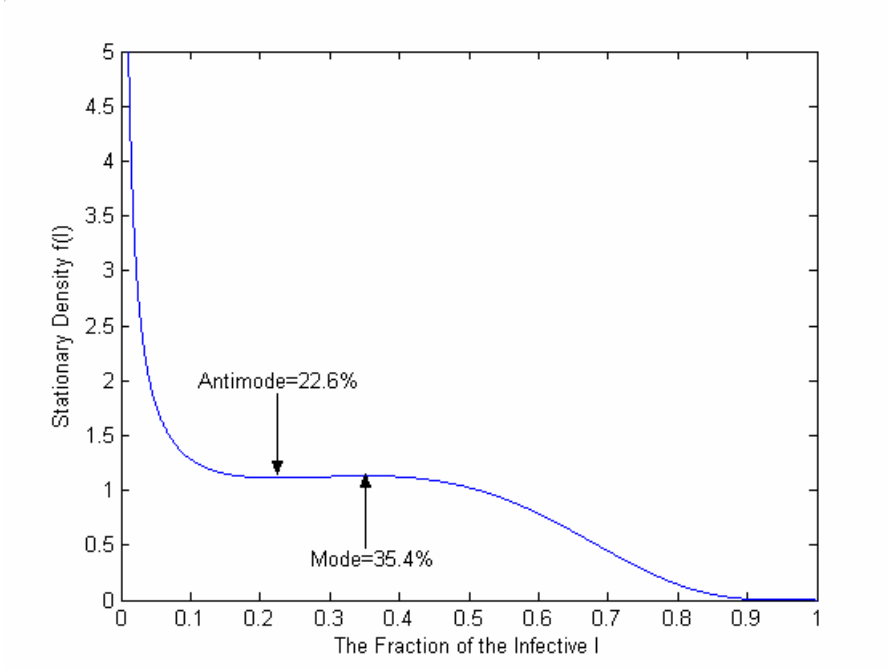


Figure 2.4 shows the stationary probability density function, $f(I)$. The antimode and mode of the stationary probability density function have important economic meanings.[13] The antimode indicates a threshold beyond which the epidemic is likely to spread, while the mode is the most likely fraction of the infective in the whole population. Given the parameter values, I obtain $I_1 = 22.60\%$ and $I_2 = 35.40\%$. It implies that the epidemic is unlikely to spread unless more than 22.6% of the workers are infected. It is most likely that 35.4% of the employees would get infected should the epidemic spread.

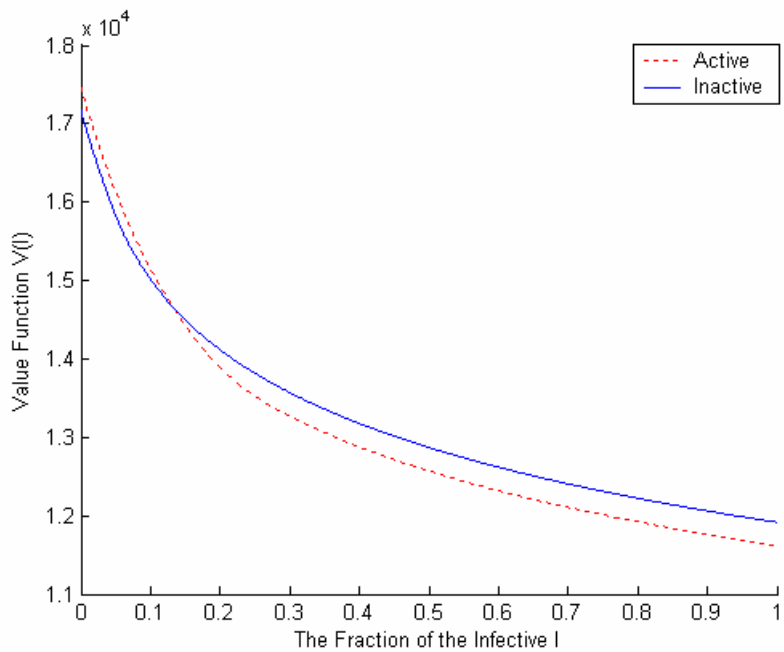
2.5.3. The Effect of Uncertainty: Stochastic versus Deterministic

[13] By solving the equation $f'(I) = 0$, we can get the antimode $I_1 = d - \sqrt{d^2 - (c-\alpha)/\beta}$, mode $I_2 = d + \sqrt{d^2 - (c-\alpha)/\beta}$, where $d = (\beta - \gamma - \delta - \alpha + 2c)/2\beta$

Based on the MATLAB implementation proposed by Fackler (2004)[14], I solve the above regime-switching problem by dynamic programming. The value functions and marginal value functions are displayed in Figures 2.5 and 2.6.

Given the parameters above, it is optimal for the firm in the active regime to suspend business temporarily when the fraction of infected employees is higher than 27%. In the inactive regime, however, it is optimal to reactivate anytime the fraction drops to 5%. If there is no uncertainty with regard to the dynamics of the epidemic, i.e. $c = 0$, the epidemic model in stage I becomes deterministic. The corresponding thresholds are 27% and 3% respectively, by my calculation.

Figure 2.5: Value Functions of the Stochastic Model



[14] I use the Optimal Switching Solver in the CompEcon Toolbox. It is publicly available at http://www4.ncsu.edu/~pfackler/compecon/newtools.html

Figure 2.6: Marginal Value Functions of the Stochastic Model

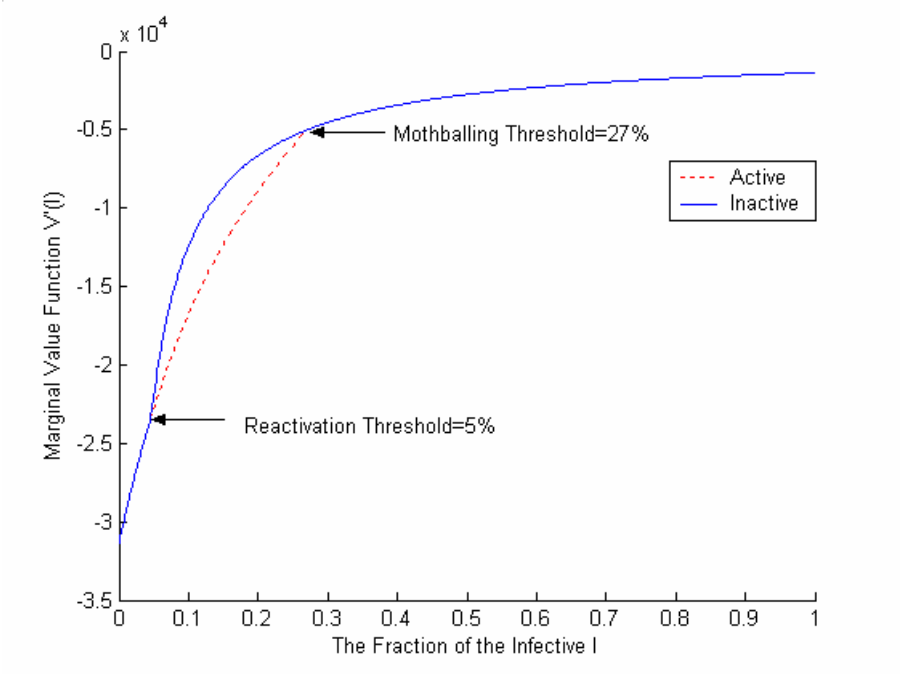
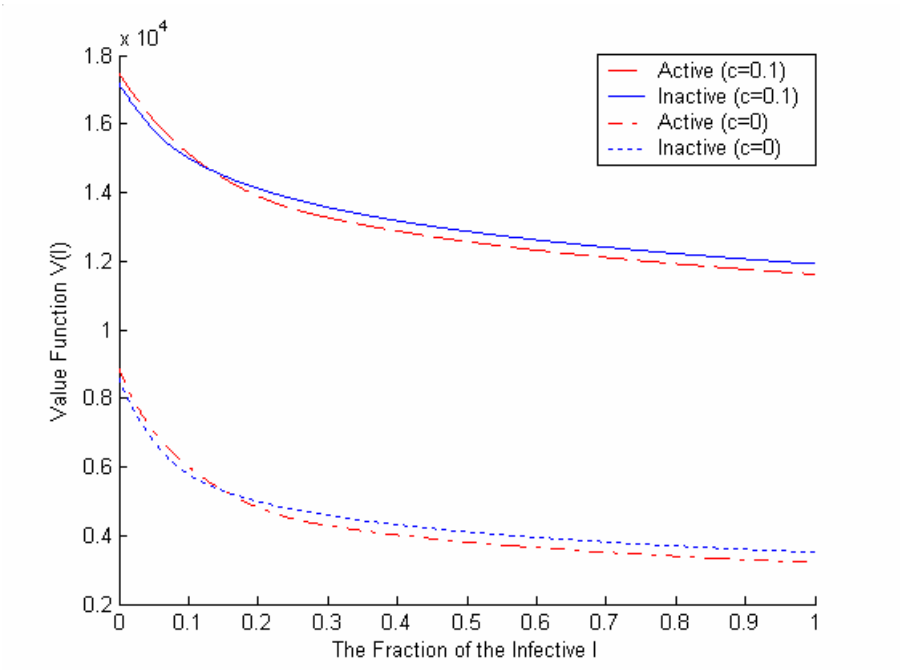


Figure 2.7: Comparison of Value Functions: Stochastic v.s. Deterministic



This observation implies that firms are more conservative on the suspension-reactivation decisions when they take into account the uncertainty in the future. In the stochastic case, firms will not come back to operation until the fraction drops by 2 points below 5% (the reactivation threshold in the deterministic case). The difference here also represents the effects of real options. It is the uncertainty in the future that makes the options valuable. It is the existence of real options which makes firms behave differently relative to the deterministic case. I graph the value functions in both the deterministic and stochastic models in Figure 2.7. The finding is consistent with the theory of real option valuation: A firm's value is usually underestimated under the traditional NPV approach.

2.5.4. The Effect of Switching Costs on Switching Thresholds

I now examine the effect of changing the mothballing cost and reactivation cost on the switching thresholds.

Table 2.1: The Effect of Changing the Switching Costs

Initial parameters M=300, A=300	Mothballing threshold (I_H =27%)	Reactivation threshold (I_L =5%)	Conclusions
M=400, A=300	28%	4%	I_H increases with M I_L decreases with M
M=300, A=400	28%	4%	I_H increases with A I_L decreases with A

First, let us consider the impact of an increase in the mothballing cost. As shown in Table 2.1, the mothballing threshold I_H increases to 28% and the reactivation threshold I_L drops to 4%, when M rises to 400 and A remains at the original level. The effect of the mothballing cost on the mothballing threshold I_H is straightforward: when the mothballing cost increases, the firm

needs to pay more when it switches to the inactive regime. Thus, the manager is more reluctant to suspend the business, and the mothballing threshold increases as a result. The influence of the mothballing cost on the reactivation threshold I_L might need further consideration. Intuitively, the increase in the mothballing cost M decreases the reactivation threshold I_L according to the mirror image effect. That is, the firm might reactivate the production with less willingness, if it has to pay a large amount of lump sum costs when the fraction of the infective increases in the future, otherwise it would rather stay in the inactive regime.

It seems that the change of the reactivation cost A has similar effects to the change of the mothballing cost M. The reactivation threshold I_L decreases with the reactivation cost A, since the firm is more reluctant to reactivate the production as the reactivation cost increases. The mothballing threshold I_H increases with the reactivation cost A. That is because the firm suspends the production with some reluctance to lose its option value. Considering the possibility that the fraction of the infective might drop in the near future, the firm could avoid paying the reactivation cost again by remaining in the active regime. Therefore, the larger the reactivation cost, the larger the option value and the greater the reluctance to suspend.

Figure 2.8 and 2.9 can help us observe the comparative results more clearly. The switching thresholds change like step functions, and the mothballing cost and the reactivation cost have almost the same effects on the switching thresholds.

Figure 2.8: The Effect of Increasing the Mothballing Cost

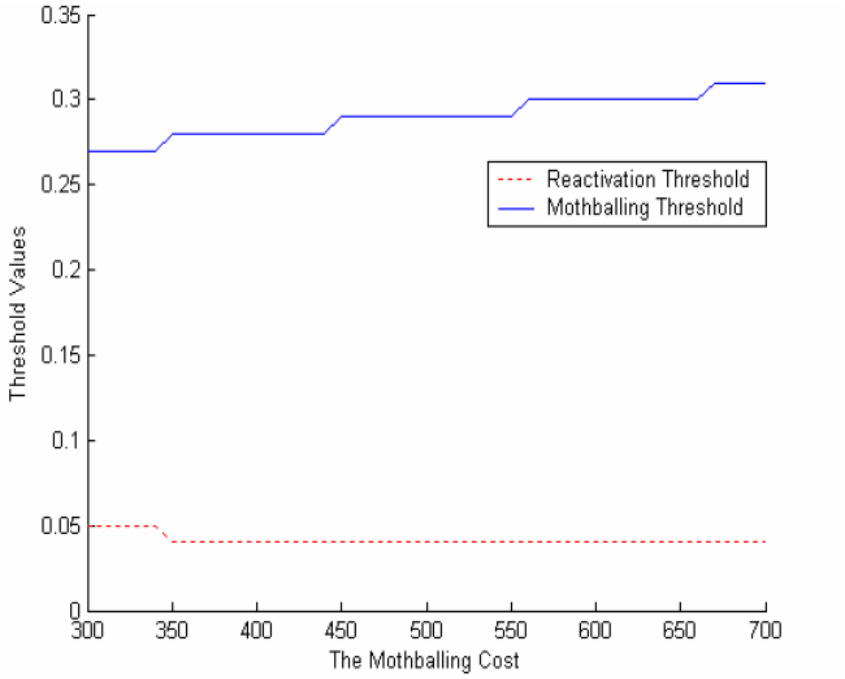
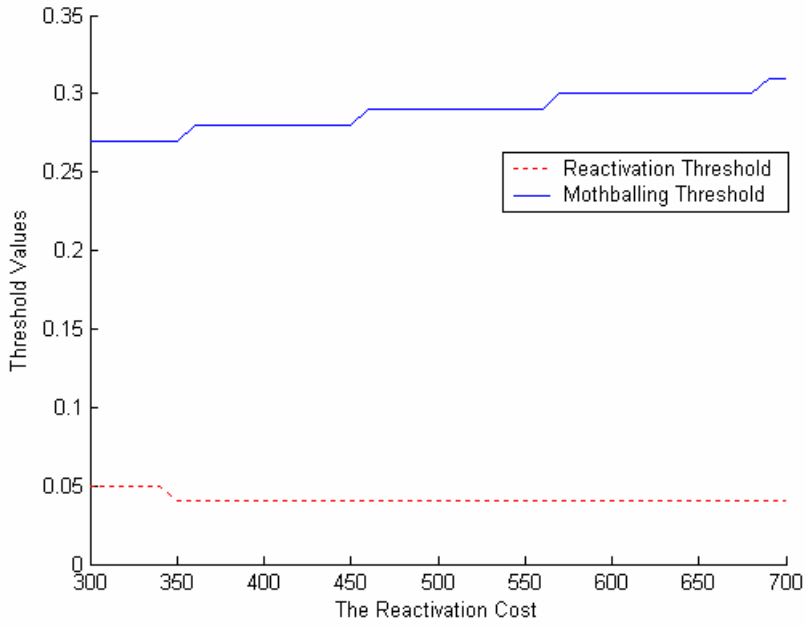


Figure 2.9: The Effect of Increasing the Reactivation Cost



2.5.5. The Effect of Disease Control Strategies

One of the purposes of modeling epidemics is to provide a rational basis for policies designed to control the spread of a disease. A firm could adopt different strategies which aim to alter the parameters in the epidemic model, so that the disease could be controlled. For instance, the firm could reduce the infectious contagion among its employees by adopting internet conferences and phone meetings. It can screen the suspected infective and mandate an immediate leave for those who are thought to pose a risk. It could immunize some or all of the employees by vaccination. It can also initiate an information session to raise public awareness of higher disease prevalence and inform its employees of some preventive measures. All these control strategies are aiming at

- decreasing the internal infection rate β;
- increasing the recovery rate γ;
- decreasing the external infection rate α;

Interestingly, firms can implement these control policies at little cost, but can benefit a lot from these strategies. As shown in Figure 2.10, when the firm adopts some strategies to decrease the internal infection rate β from 1 to 0.8, the value of the firm increases significantly in both regimes. Similar results can be obtained if I increase the rate of recovery γ or decrease the rate of external transmission α (see Figures 2.11 and 2.12, respectively). Therefore, firms can maximize their values at the same time as controlling the disease.

Figure 2.10: The Effect of Decreasing the Internal Infection Rate

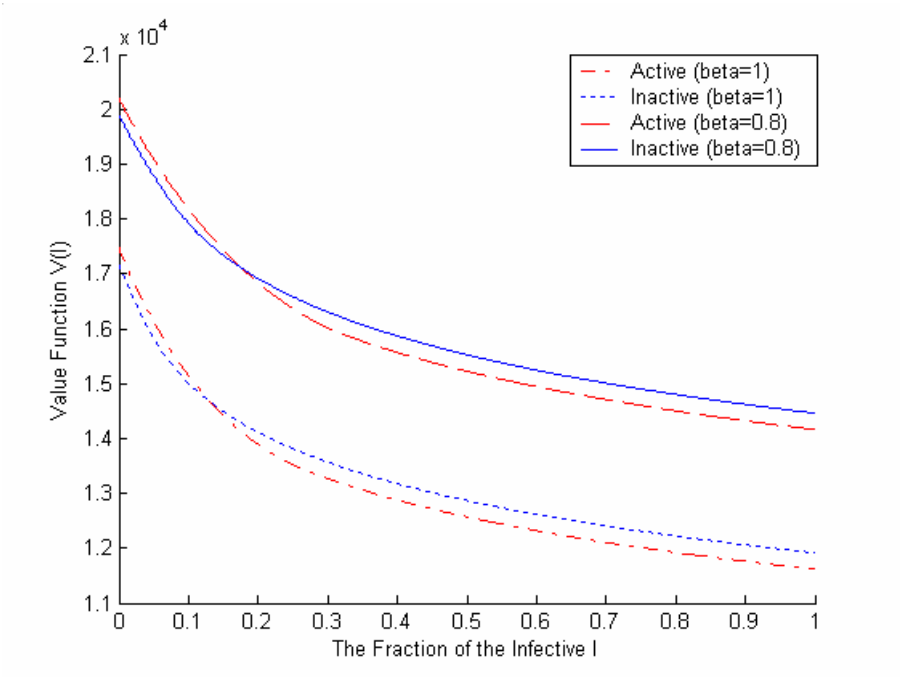


Figure 2.11: The Effect of Increasing the Recovery Rate

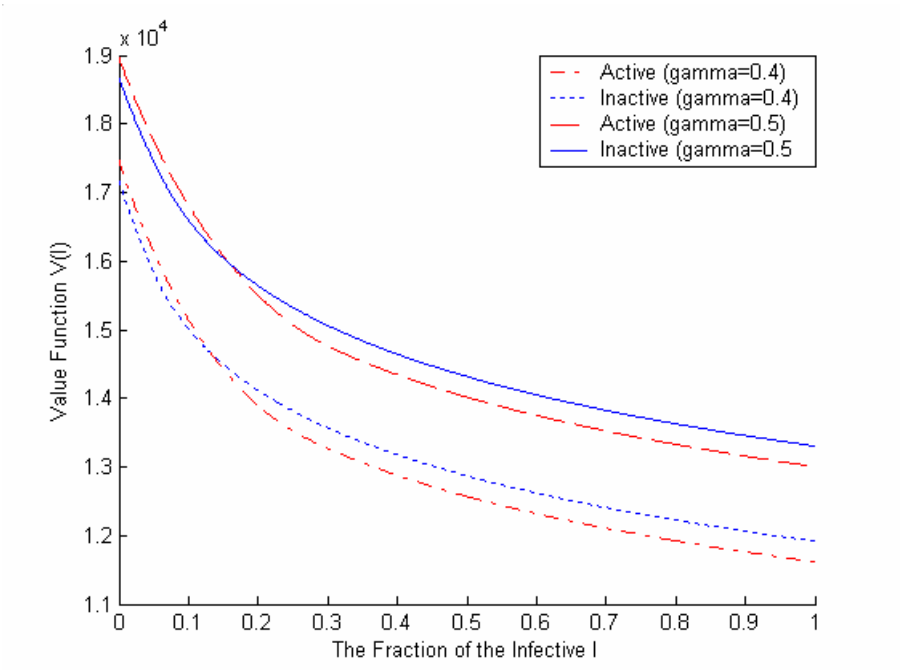
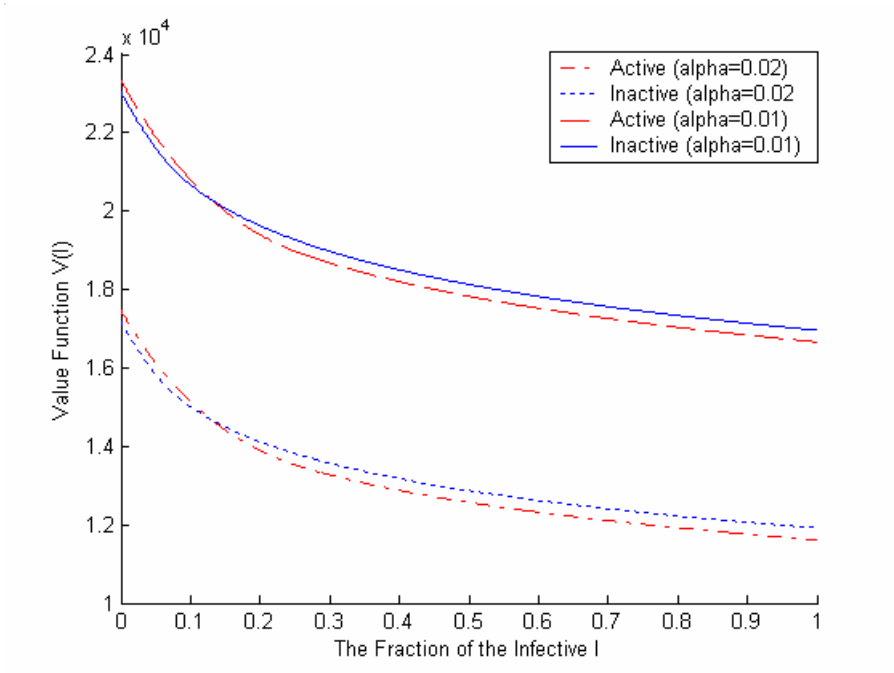


Figure 2.12: The Effect of Decreasing the External Infection Rate



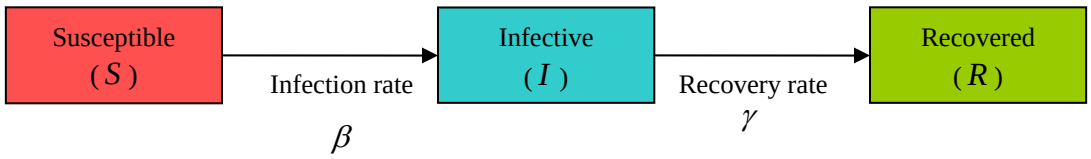
2.6. Extensions and Open Questions

2.6.1. The Modified SIR Model Allowing for External Contagions and Deaths

Previous analysis is based on the modified *SI* model, where I only consider two categories (susceptible and infective) and have one state variable (I_t). In this section, I extend the model to accommodate three classes (susceptible, infective and recovery) and deal with two state variables (S_t and I_t). The *SIR* model is originally proposed by Kermack and McKendrick (1927). They assume there is no entry into or departure from the population, and no birth or death in the course of the epidemics. In addition, they introduce a new class R (recovered): all the infective are assumed to get recovered and falls into this class. Figure 2.13 shows the transition dynamics among these three classes. Note that the evolving process of the disease in the *SIR* model is irreversible. Once a person gets infected, he/she will end up with recovery and obtain the

immunity to the disease permanently: the infective will not fall into the class S again.

Figure 2.13: The Classic *SIR* Model

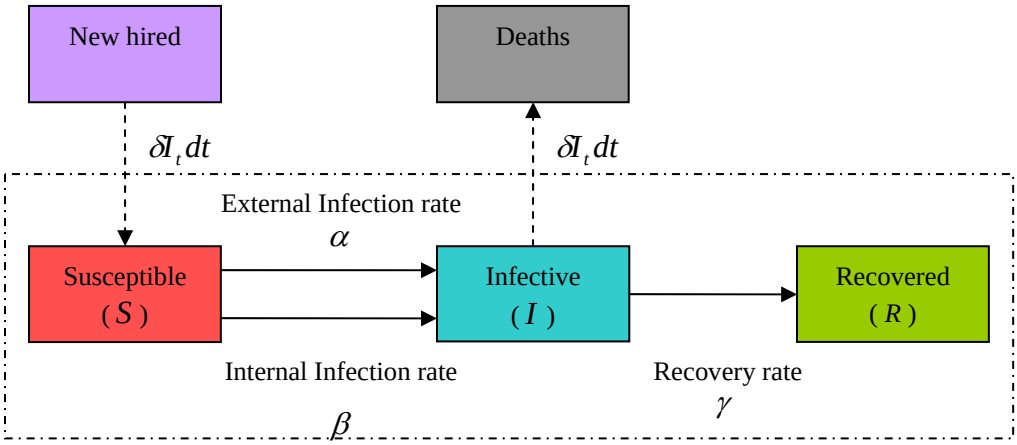


Let R_t denote the fractions of population that reside in the class R at time t, the qualitative relationships among S_t, I_t, and R_t are given by the following system of differential equations:

$$\begin{cases} dS_t = -\beta I_t S_t dt \\ dI_t = (\beta I_t S_t - \gamma I_t)dt. \\ dR_t = \gamma I_t dt \end{cases} \tag{2.26}$$

When considering infection from external sources and possible deaths from the disease, the transition of the disease among the three classes can be shown in Figure 2.14.

Figure 2.14: The Modified *SIR* Model Allowing for External Contagions and Deaths



Now, the modified SIR model can be expressed as follows:

$$\begin{cases} dS_t = (-\beta I_t S_t + \delta I_t)dt \\ dI_t = (\beta I_t S_t + \alpha S_t - \gamma I_t - \delta I_t)dt \\ dR_t = (\gamma I_t - \alpha S_t)dt \end{cases}. \tag{2.27}$$

2.6.2. Open Questions to the Stochastic SIR Models

In order to accommodate uncertainty in the evolution of the epidemics, we need to extend the above deterministic model to a stochastic one. Jia and Tsui (2005) use discrete random variables such as Poisson and binomial to capture the stochastic transitions between different states. I doubt the validity of their approach, since the discrete stochastic transitions are not inherently consistent with the continuity from the *SIR* model. One of the referees suggests that I add stochastic terms to S_t and I_t , driven by Brownian motions W_{1t} and W_{2t} , respectively. Questions arise as to the function forms of volatility terms for the *SIR* model. Different assumptions can be made, such as:

- The volatility is proportional to the state variable, i.e., $\sigma_1 S$ and $\sigma_2 I$, as in the geometric Brownian motion model;
- The volatility is proportional to the square root of the state variable, i.e., $\sigma_1\sqrt{S}$ and $\sigma_2\sqrt{I}$, as in the CIR model;
- The volatility is greater in the center region than in the extreme cases, i.e., $\sigma_1\sqrt{S(1-S)}$ and $\sigma_1\sqrt{I(1-I)}$;

In addition, we have to impose several restrictions such that $0 \le S \le 1$, $0 \le I \le 1$, and $0 \le S + I \le 1$. Although the third assumption about the random variation looks the most reasonable since it satisfies the first two restrictions, it doesn't guarantee the third restriction. Besides, the two Brownian motions W_{1t} and W_{2t} may be correlated with each other. We need to

figure out the appropriate correlation coefficient. Below, I just show a numerical example which generates seemingly reasonable results. More research is needed to explore a stochastic model which can better describe the dynamics of a disease.

2.6.3. An Example for Optimal Switching Boundaries

Suppose W_{1t} and W_{2t} are two independent Brownian motions (Again, W_{1t} and W_{2t} are not necessary to be independent). When the firm is active, the dynamics of the disease is

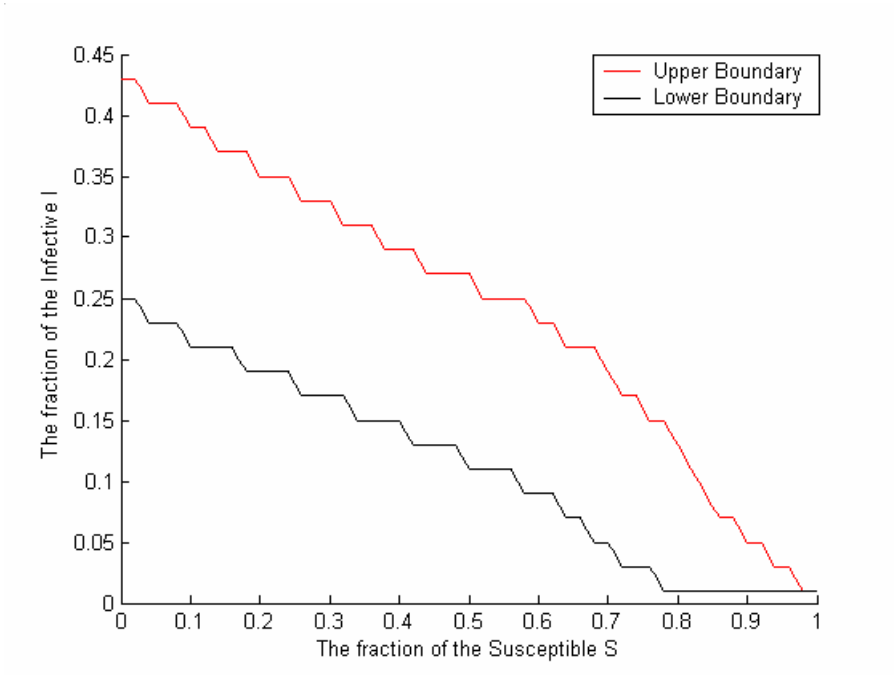
$$\begin{cases} dS_t = [-\beta I_t S_t + \delta I_t]dt + \sigma_1 \sqrt{S_t}\, dW_{1t} \\ dI_t = [\beta I_t S_t + \alpha S_t - \gamma_1 I_t - \delta I_t]dt + \sigma_2 \sqrt{I_t}\, dW_{2t} \end{cases}, \qquad \text{if } r = 1. \tag{2.28}$$

When the firm is inactive, it is

$$\begin{cases} dS_t = \delta I_t dt + \sigma_1 \sqrt{S_t}\, dW_{1t} \\ dI_t = [\alpha S_t - \gamma_2 I_t - \delta I_t]dt + \sigma_2 \sqrt{I_t}\, dW_{2t} \end{cases}, \qquad \text{if } r = 2\ . \tag{2.29}$$

When the system is controlled by two state variables, we can obtain two optimal switching boundaries, as shown in Figure 2.15. The lower line represents the points for which it is optimal to switch from the inactive to active regime, and the upper line represents the points for which it is optimal to switch from the active to inactive regime. I also examine the effect of changing switching costs on the optimal switching boundaries. As I expect, the upper boundary move upward when the reactivation cost increases, and move downward when the reactivation cost decreases (see Figure 2.16 and 2.17). The mothballing cost has similar impacts on optimal switching boundaries (see Figure 2.18 and 2.19).

Figure 2.15: Optimal Switching Boundaries



Note: I set $\sigma_1 = \sigma_2 = 0.1$

Figure 2.16: The Effect of Increasing the Reactivation Cost on the Optimal Switching Boundaries

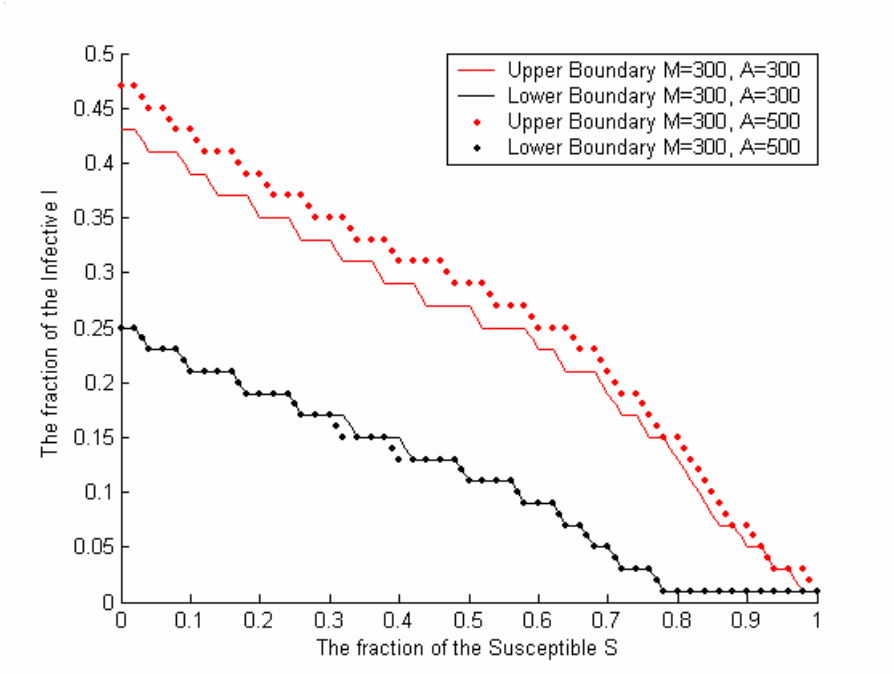


Figure 2.17: The Effect of Decreasing the Reactivation Cost on the Optimal Switching Boundaries

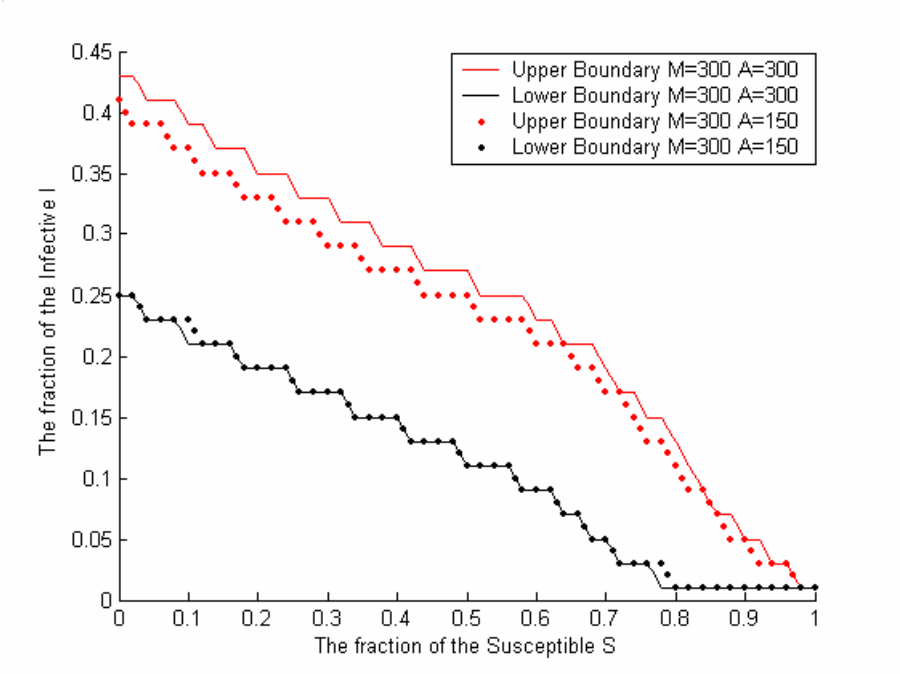


Figure 2.18: The Effect of Increasing the Mothballing Cost on the Optimal Switching Boundaries

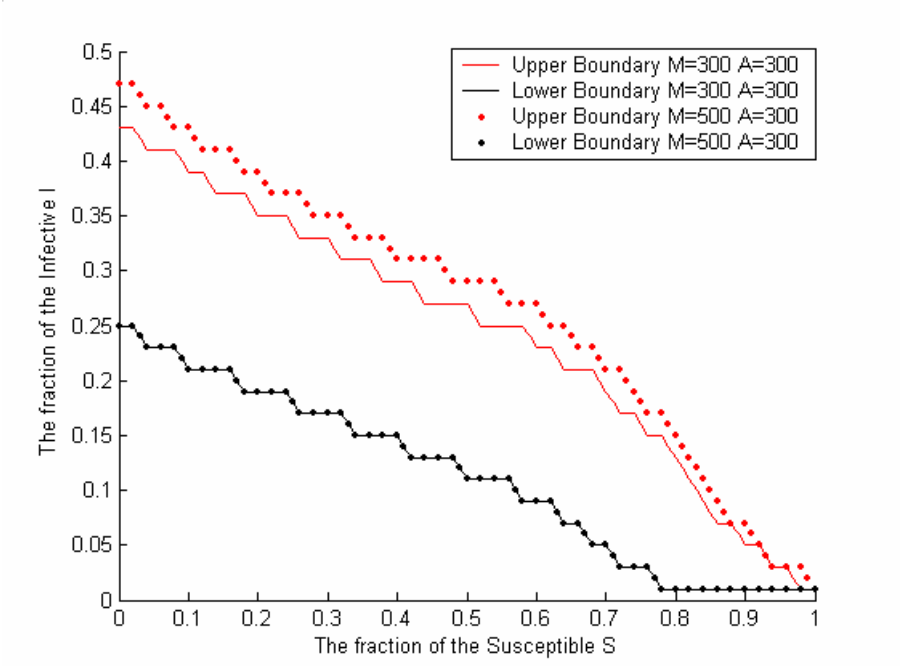
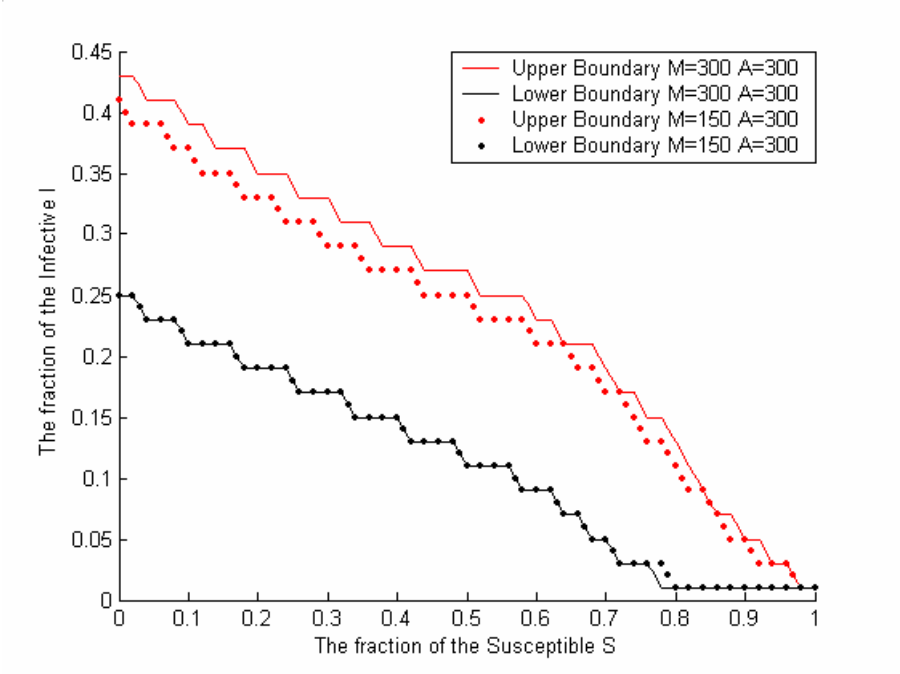


Figure 2.19: The Effect of Decreasing the Mothballing Cost on the Optimal Switching Boundaries



2.7. Conclusions and Discussions

The dynamics of human epidemics is an important topic in epidemiology and mathematical biology. A vast literature has developed in these fields, but it seems that little of it addresses epidemic risks for private enterprises with large numbers of employees. Epidemic models may provide some insights as to the effectiveness of control measures such as immunization, worker furloughs, and quarantines. Modeling may lead to optimal rules for implementing these strategies. In this chapter, I make the first attempt to build a two-stage model to analyze how firms can manage the risk associated with pandemics that can severely attack their workforce. In the first stage, I propose a simple regime-dependent epidemic model to allow for external contagion and deaths from the disease. In the second stage, I use the fraction of the infective in a given firm as a decision aid to construct an optimal suspension-reactivation strategy. Dynamic programming is

discussed and the optimal switching thresholds are found by numerical approximation. My two-stage model has practical implications for a large business to set up triggers for activating and terminating response plans in the event of an influenza pandemic. My approach is consistent with the CDC's recommendations and can be viewed as a quantitative implementations.

Given the parameter values, the firm should suspend the business (or part of its business) when the infected workers account for 27% of the total work force, and reactive the operation when the fraction of the infective drops to 5%. Certainly, these triggers of suspension or reactivation are firm-specific: they rely on many factors involved in this model and are different for different firms. However, the economic rationale is universal. When faced with uncertainty in the future, the existence of real options makes firms more valuable. At the same time, firms are more conservative about the decisions of suspension and reactivation. If firms incur lump sum costs when switching between different regimes, the mothballing threshold increases with the switching costs while the reactivation threshold decreases with the costs.

Firms are motivated by the goal of profit maximization. An infected employee not only has a lower productivity, but also delivers the virus to other people in the company. Firms need to implement different strategies to decrease the internal infection rate, increase the recovery rate, or decrease the rate from external transmission. While these strategies aim at the control of the epidemic, firms' values are also increasing in both regimes. Disease control and firms' value maximization can be obtained simultaneously in my model.

Many large businesses provide health and life insurance to employees. However, the insurance contract is normally for administrative services only. Firms may buy a stop-loss coverage, but even then they bear substantial risk of a flu epidemic. Therefore, firms are also motivated to control the disease in order to reduce their losses from the claims for medical and

death benefits. In the benefit/cost analysis, I only consider the lost productivity due to restricted activity and premature mortality. A further analysis should include other disease-related costs such as hospitalization, physician services, and prescription drugs.

3

Modeling Mortality with Jumps: Transitory Effects and Pricing Implication to Mortality Securitization

Option pricing theory is built on the assumption that the payoff of the option can be replicated using the underlying asset and riskless lending or borrowing. While this is perfectly justifiable in the context of listed options on traded stocks, it becomes less defensible when the underlying asset is not traded. I have shown in chapter 2 how to apply the real option theory to maximize a firm's value and determine regime switching triggers. In this chapter, I further explore how to price contingent claims with non-tradable underlying assets. This research is motivated by the rapid expansion of the mortality securitization market. The payoff of mortality bonds is based on a mortality index of a certain population, which is not tradable and has low or no correlation with financial market variables. Consequently, we cannot create a portfolio

consisting of bonds or stocks to replicate the payoff of mortality bonds. Therefore, the pricing of mortality bonds must be examined in an incomplete market framework.

In this chapter, I incorporate a jump-diffusion process into the original Lee-Carter model, and use it to forecast mortality rates and analyze mortality securitization. I compare alternative models with transitory versus permanent jump effects, and find that the transitory jump effect model is more appropriate for extreme mortality risk modeling. I use the Swiss Re mortality bond in 2003 as an example to illustrate how to apply the distortion measure approach to value mortality-linked securities in an incomplete market.

3.1. Introduction

Mortality risk management is fundamental to life insurance and pension industries. Mortality models are crucial as a means of quantifying these risks and providing the basis of pricing and reserving. Traditionally reinsurance, and more recently, securitization, provide a means of transferring or hedging mortality risks. Naturally mortality models are fundamental to these transactions.

A wide variety of stochastic models have been proposed for modeling the dynamics of mortality over time. Cairns, Blake and Dowd (2006a) provide a detailed overview and categorization. Most of the literature in this field is in the framework of short-rate models, among which continuous time models focus on the spot force of mortality and discrete time models concentrate on the spot mortality rates. Continuous time models (e.g., Milevsky and Promislow, 2001; Dahl, 2004; Dahl and Møller 2005; Miltersen and Persson 2005; Biffs, 2005; Schrager, 2006) help us understand the evolution of mortality rates over time, but are relatively intractable at present. I prefer discrete time models because they are easy to implement in

practice.

The Lee-Carter model is among the earliest discrete time models. Lee and Carter (1992) model the central mortality rates to be log-linearly correlated with a time-dependent mortality factor, and adjust for age-specific effects using two sets of age-dependent coefficients. In this way, the model captures both the mortality trend overall and the age-specific changes on different age groups. Thus it describes the development of the mortality curve over time quite well. The age adjustment is necessary, because mortality improvement varies across age groups. Moreover, the short-term mortality shocks, such as the 1918 influenza pandemic, attack different groups with different intensities. I will discuss these two points in detail in the data section. The Lee-Carter approach has been extended by Brouhns, Denuit and Vermunt (2002), Renshaw and Haberman (2003), Denuit, Devolder and Goderniaux (2007), and further revisited by Li and Chan (2007). Recently, Cairns, Blake and Dowd (2006b) propose a two-factor model for mortality modeling and morality-linked security pricing. The first factor equally affects mortality at all ages, whereas the second factor's effect on mortality is proportional to age. Based on their model setup, the mortality curve is increasing in ages, which does not reflect the fact that mortality rates of infants and children are much higher than those at middle ages. In addition, their model does not allow mortality jumps.

Mortality jumps must be taken into account in mortality securitization modeling, because the rationale behind selling or buying mortality-linked securities is to hedge mortality risks (Cox, Lin and Wang 2006). Nevertheless, most of papers on this topic, as in Cairns, Blake and Dowd (2006b), ignore mortality jumps (see Renshaw, Haberman and Hatzoupoulos, 1996; Sithole, Haberman and Verrall 2000; Milevsky and Promislow, 2001; Dahl, 2004; Denuit, Devolder and Goderniaux, 2007). Even if they recognize that short-term catastrophe shocks may cause

mortality jumps, they do not model mortality jumps explicitly. For example, Lee and Carter (1992) treat the 1918 influenza pandemic as a highly unusual event and employ an intervention model to remove its influence. Li and Chan (2007) regard pandemic events as non-repetitive exogenous intervention too, and implement outlier detection and adjustment to unveil the "true" model underlying the outlier-free mortality series.

To my knowledge, there are only a few papers considering mortality jumps in mortality securitization modeling. Biffis (2005) uses affine jump-diffusions to address the risk analysis and market valuation of life insurance contracts in the continuous time framework. Cox, Lin and Wang (2006) find that mortality jumps do have a significant effect on mortality modeling. They, however, model the age-adjusted death rates instead of the mortality curve. Thus their model fails to represent age-specific changes of mortality rates. In addition, they model the jump process with permanent effects on mortality rates, although many mortality jumps are caused by short-term catastrophic events and only have transitory effects.

In this chapter, I propose to incorporate a jump-diffusion process into the Lee-Carter model, restricting mortality jumps to have one-period effects. I fit the model to US age-specific mortality rates and forecast the development of the mortality curve. I show that the model with jumps outperforms that without jumps, and the model with permanent jump effects induces big deviations in parameter estimation compared with that with transitory jump effects. I then discuss the outlier-adjusted Lee-Carter model presented by Li and Chen (2007) to further explore the source of mortality jumps. I find that the so-called "outliers" are actually very important to our morality securitization modeling, and we cannot delete the outliers from our time-series data in order to establish a proper model for pricing mortality-linked securities. I use the Swiss Re mortality bond (2003) as an example of pricing the mortality-linked securities, and illustrate that

the model with permanent jump effects results in large pricing distortions.

In an incomplete insurance market, there are mainly two approaches for security valuation. One way is to adapt the arbitrage-free pricing framework of interest-rate derivatives to the valuation and securitization of mortality risk. Cairns, Blake and Dowd have a detailed discussion on this issue and give as an example the pricing of the EIB longevity bond (see Cairn, Blake and Dowd 2006a, 2006b). The second method is to use a distortion operator to create an equivalent risk-adjusted distribution, and obtain the fair value of the security under this risk-neutral measure. Examples of this approach, based on the Wang transform (Wang 2000, 2002), include Lin and Cox (2005), Dowd, Blake, Cairns and Dawson (2006), Denuit, Devolder and Goderniaux (2007). The Swiss Re mortality bond (2003) covers mortality risks across countries and over time, which makes the valuation problem very difficult. Previous research (e.g. Cox, Lin and Wang, 2006) employ the normalized multivariate exponential tilting, which is a generalization of the Wang transform, to take into account correlations across countries. They, however, modify the contract terms by linking the principal repayment with the maximum of the mortality index in three years, and ignore correlations over time. In this chapter, I employ the Wang transform and make the first attempt to account for correlations of the mortality index over time. The basic idea is to forecast the mortality index on paths and change the measure on each path to get the risk-adjusted mortality index.

The remaining of this chapter proceeds as follows. In section 3.2, I provide an overview of the mortality securitization market and examine the general design of mortality bonds. In section 3.3, I describe the data and demonstrate historical facts for further motivation of the problem. In section 3.4, I propose a generalized Lee-Carter model with transitory jump effects, and discuss the outlier-adjusted Lee-Carter model to justify the necessity of modeling mortality with jumps.

A numerical example of pricing the Swiss Re mortality bond (2003) is given in section 3.5. Concluding remarks and discussions are provided in section 3.6.

3.2. Mortality Securitization Overview

3.2.1. Functional Transform from Risk Warehousing to Risk Intermediation

In general, there are two types of mortality risks we need to consider. The first is longevity risk, which refers to the uncertainty in the future improvement in mortality rates. If the realized mortality rates are much lower than the assumed mortality rates in premium pricing and reserve calculations, annuity providers and pension plans will incur large losses. The second is short-term catastrophic shocks, which are caused by catastrophic events and result in much higher mortality rates than would normally be experienced. The 1918 Spanish flu killed up to 50 million people worldwide and 500,000 in the United States (Rasmussen 2005). The earthquake and tsunami in 2004 resulted in 300,000 dead and missing across southern Asia and eastern Africa (Cox, Lin and Wang 2006).

Traditionally, these types of risks are shared by insurers or reinsurers via reinsurance and retrocession, respectively. Insurers (or reinsurers) issue risk hedging products, warehouse the risks on balance sheets and bear the risks by holding equity capital. Although investors in the capital markets serve as the ultimate risk bearers by holding the stocks of insurance companies, they typically do not have the option of buying particular financial securities originated by the insurers (Cowley and Cummins, 2005). With the growth of insurance markets, insurers badly need additional risk-bearing capacity. However, capacity of the reinsurance market is often limited as possible transaction partners usually already have similar risks in their books. Also, retrocession would require a reinsurer to disclose its own business to potential competitors.

Therefore, raising new capital can be expensive.

The Alternative Risk Transfer (ART) provides theoretical fundamentals for a capital market solution. Insurers and reinsurers are now stepping away from the traditional risk warehousing function towards risk intermediation function. They repackage the risk hedging products and originate different types of financial securities that are passed through to the capital market. As a result, much less risk is retained in the business of insurers or reinsurers, which enables them to operate more efficiently as well as increase underwriting capacity (Froot, 2000; Cowley and Cummins, 2005; Lin and Cox, 2008). In the past few decades, insurance companies have successfully transferred the catastrophic risk in the property-liability business to financial markets by issuing CAT bonds (Cummins, Lalone and Phillips 2004). More recently, various mortality bonds (or longevity bonds) have been designed and/or issued to transfer mortality risk (or longevity risk) into the capital market. Mortality-linked securities have little or no correlations with the financial market, thereby providing new sources of diversification for investors.

3.2.2. Market Overview and General Design of Mortality Securitization

The idea of mortality securitization was first proposed by Samuel H. Cox in a talk in 1998. Cox, Pedersen and Fairchild (2000) mention this idea in their paper. However, Blake and Burrows (2001) may be the first to propose the structural design of longevity bonds explicitly. The bonds are designed so that coupon payments are contingent on the percentage of a certain population cohort surviving some further period. The longevity bonds allow annuity providers to hedge against aggregate longevity risk: if annuitants live longer than anticipated, the insurance companies would incur losses due to longer payment periods, but they would also receive greater

coupon payments for holding longevity bonds to offset the losses (Denuit, Devolder and Goderniaux, 2007). A more extensive discussion of mortality-linked derivatives is provided by Blake, Cairns and Dowd (2006), who present the various forms of longevity bonds, swaps, futures, and options, and investigate their potential uses.

The Swiss Re mortality bond issued by the Swiss Reinsurance company in 2003 is the first mortality securitization transaction. It was designed as a hedge for a life reinsurer: it expanded Swiss Re's capacity to pay catastrophic mortality losses. Possibly inspired by the successful securitization of mortality risks, the European Investment Bank (EIB) offered the first longevity bond in November 2004 to hedge the longevity risk for pension planners and annuity providers. Although this particular deal failed to be launched because of insufficient demand, it did attract public attention and provided an instructive case study (Blake, Cairns and Dowd, 2006).

Investors' demand to mortality bonds seems to be very high. Ever since the first issue of the Swiss Re mortality bond in 2003, insurers or reinsurers have made another four mortality bond transactions in order to reduce their extreme mortality exposures, with a total volume exceeding $1.2 billion (Bauer and Kramer, 2006). A detailed overview of mortality securitization transactions is summarized in Table 3.1. While they differ in many aspects, such as tranche sizes, credit ratings, trigger levels, premium spreads, maturities and covered areas, the basic structure is the same. Figure 3.1 demonstrates the general design of mortality securitizations.

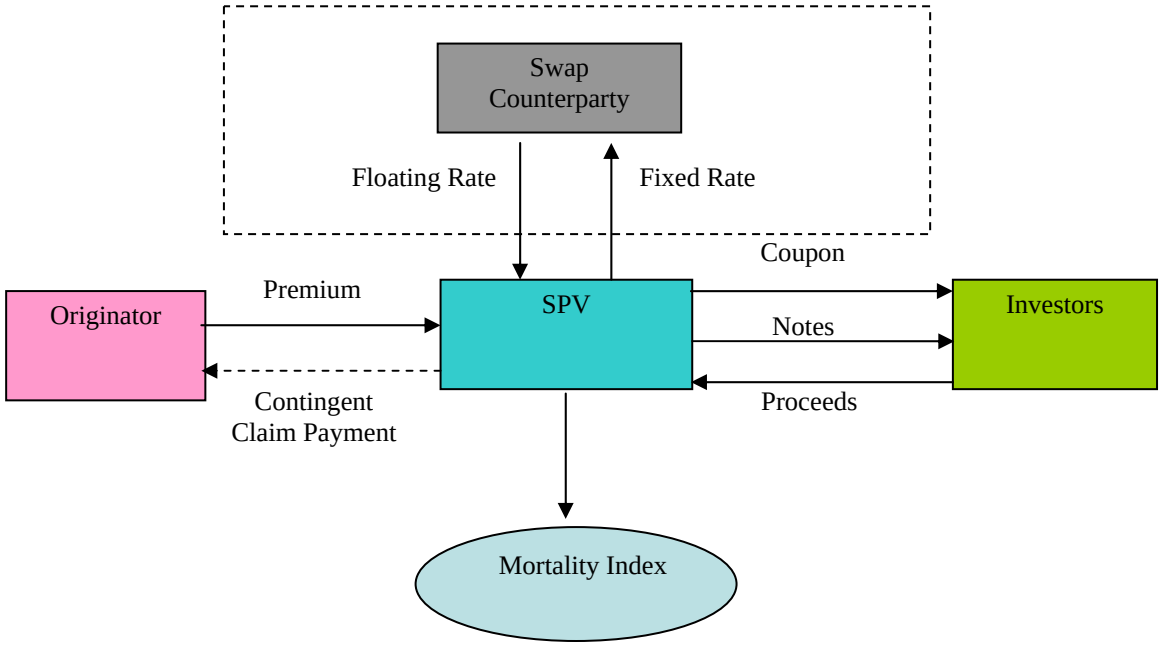
Table 3.1: Comparison of Five Mortality Securitization Transactions in the Market

Time	Nov. 2003	Apr. 2005			May 2006		Nov. 2006			
Issued by	Vita Capital Ltd.	Vita Capital II Ltd.			Tartan Capital Ltd.		Osiris Capital Ltd.			
Arranged by	Swiss Re	Swiss Re			Goldman Sachs		Swiss Re			
Protection for	Swiss Re	Swiss Re			Scottish Re		AXA			
Class	A	B	C	D	A	B	B1	B2	C	D
Tranche Size	$400m	$62m	$200m	$100m	$75m	$80m	€100m	€50m	€150m	€100m
Attachment	130%	120%	115%	110%	115%	110%	114%	114%	110%	106%
Detachment	150%	125%	120%	115%	120%	115%	119%	119%	114%	110%
Premium (bps)	135	90	140	190	19	300	20	120	285	500
Maturity	3 years	5 years			3 years		4 years			
Covered Areas	US 70%, UK 15%, F 7.5%, Italy 5%, CH 2.5%	US 62.5%, UK 17.5%, DE 7.5%, Japan 7.5%, CAN 5%			US 100%		F 60%, J 25%, US 15%			

Time	Dec. 2006								
Issued by	Vita Capital III Ltd.								
Arranged by	Swiss Re								
Protection for	Swiss Re								
Class	A-IV	A-V	A-VI	A-VII	B-I	B-II	B-III	B-V	B-VI
Tranche Size	$100m	$100m	€55m	€100m	$90m	$50m	€30m	$50m	€55m
Attachment	125%	125%	125%	125%	120%	120%	120%	120%	120%
Detachment	145%	145%	145%	145%	125%	125%	125%	125%	125%
Premium (bps)	21	20	21	80	110	112	110	21	22
Maturity	4 years	5 years	4 years	5 years	4 years	5 years	4 years	5 years	4 years
Covered Areas	US 62.5%, UK 17.5%, DE 7.5%, Japan 7.5%, CAN 5%								

Source: Revised from Bauer and Kramer (2007)

Figure 3.1: The General Design of Mortality Securitization



Source: Revised from Cowley and Cummins (2005)

The originator, which is usually an insurer or reinsurer, set up a special purpose vehicle (SPV) in order to accomplish the transaction. The SPV is a passive financial intermediary that exists to create a "pure play" security, issue the security to investors, insulate investors from the sponsor's credit risk, and provide tax and accounting benefits to the sponsor (Cowley and Cummins, 2005). The originator pays premiums to the SPV in return for a contingent claim payment. The SPV issues debt securities to investors in the capital markets, and pays periodic coupon payments to them. Securities issued by the SPV are usually structured to several tranches in order to attract different types of investors. The proceeds from the sale of the notes are used to buy high quality securities as collateral. In many cases, it is desirable to pay a floating rate of interest to debt investors, even though the underlying assets may pay interest at a fixed rate. In order to hedge the interest rate risk, the SPV usually enters into a swap transaction. The fixed interest rate is swapped for a floating rate tied to a widely used index such as LIBOR. Mortality

bonds are similar to CAT bonds, except that payoffs of mortality bonds are based on adverse mortality experiences. It is possible to write a securitization on a portfolio of insured lives. There are a large number of deals in which a closed block of life insurance policies or annuities are securitized. These are actually asset securitizations. The insurer is selling its future profits on the block. In this case, investors are concerned about moral hazard problems. This can be solved by hiring a third party to audit the losses of the insurer, but it would add a lot to the transaction costs. In today's market, "pure" mortality securitizations base the payoffs of mortality bonds on a public mortality index of a certain population. If the defined mortality index falls below a certain trigger level (or attachment point), parts of the principal will be withdrawn and paid to the originator, and any remaining principal will be returned to investors at maturity. As discussed by Cowley and Cummins (2005), there is always a tradeoff between the moral hazard problem and basic risk. Linking the payoff of mortality bonds to a population index has the advantage of reducing investors' concern about moral hazard problems, but it also introduces basis risk since the insurer's mortality experience could deteriorate significantly more than that of the index. For this reason, mortality bonds are likely to appeal to large, diversified insurers or reinsurers.

3.3. Data Descriptions and Historical Facts: Further Motivation

The mortality data is from the National Center for Health Statistics (NCHS). The NCHS reports the age-adjusted death rate and age-specific death rate per 100,000 population (2000 standard) for selected causes of death from 1900 to 2003.[15] Age-adjusted death rates are used to compare relative mortality risks across groups and over time; they are indices rather than direct measures. The age-specific death rates are tabulated for age 0, age group 1-4, then 10-year groups 5-14, 15-24, up to 75-84, and the age group 85 and over. Selected causes include heart

[15] Source: http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs.htm

disease, cancer, stroke, influenza and pneumonia.

Table 3.2 provides evidence of mortality improvement. Overall, the age-adjusted death rate by all causes decreased to 832.7 per 100,000 in 2003, which is 33.1% of the 1990's level (2518.0 per 100,000). However, the improving mortality has variant effects across age groups. The mortality rates for age group 1-4 fell to 1.6% of its initial value, but that for age group 85 and over only dropped to 55.9% of its initial value. These proportions differ by a factor of 35 at the extremes! A proper mortality model should capture this age-specific effect of mortality improving on all ages.

Table 3.2: Mortality Improvements by Different Age Groups

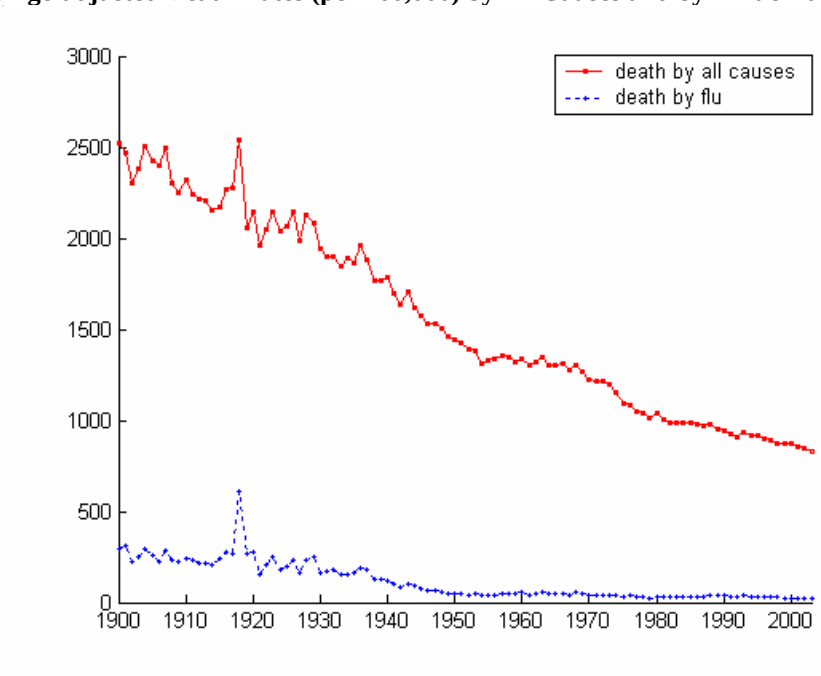
Age groups	1900	2003	Ratio	Age groups	1900	2003	Ratio
All	2518.0	832.7	0.331	35-44	1023.1	201.6	0.197
<1	16244.8	700.0	0.043	45-54	1495.4	433.2	0.290
1-4	1983.8	31.5	0.016	55-64	2723.6	940.9	0.345
5-14	385.9	17.0	0.044	65-74	5636.1	2255.0	0.400
15-24	585.5	81.5	0.139	75-84	12330.0	5463.1	0.443
25-34	819.8	103.6	0.126	>=85	26088.2	14593.3	0.559

Note: The "all" row is the age-adjusted death rate by all causes per 100,000, from NCHS reports HIST293 and GMWK293R. The other rows are the age-specific death rates per 100,000, from NCHS reports HIST290 and GMWK290R**.** The mortality improvement ratio is calculated by the author.

The trend of mortality improvement is further demonstrated in Figure 3.2. In addition, Figure 3.2 compares the dynamics of age-adjusted death rates by all causes to that by influenza and pneumonia from 1900 to 2003. Although the death rates caused by influenza and pneumonia become small after 1950 (less than 0.00005), which makes the comparison difficult to visualize, we can still observe the similar pattern of fluctuations of death rates by all causes and by flu in the first half of the past century. The two curves even jump at the same time, which is remarkably evidenced in year 1918. The correlation coefficient between the two curves is 0.9116,

which also indicates a close correspondence between flu-caused deaths and all deaths. Deaths caused by flu account for 9.4% of all deaths before 1950 on average, 3.4% from 1950 to 2003, and 6.3% for the whole period examined. In 1918, this proportion reaches its historic peak at 24.1%. The high correlation between the two curves and high portion of deaths caused by flu suggest that we should not exclude flu events when modeling the mortality.

Figure 3.2: U.S. Age-adjusted Death Rates (per 100,000) by All Causes and by Influenza and Pneumonia

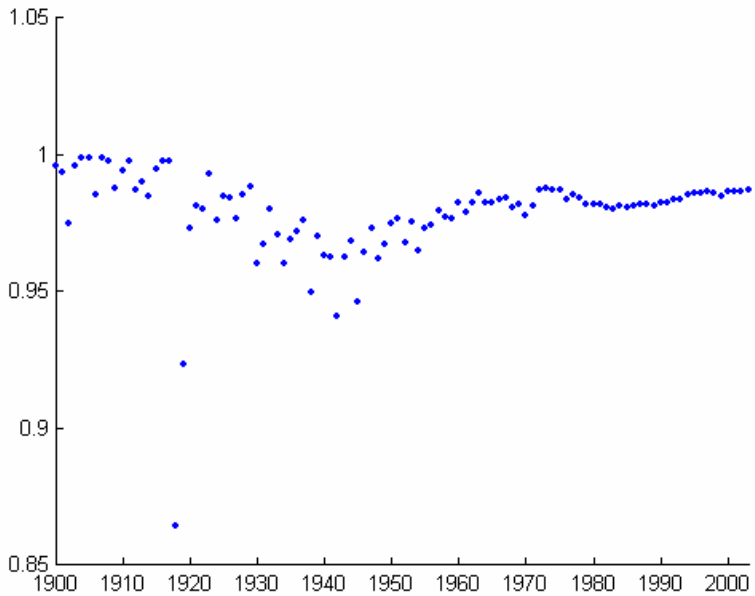


Note: Data are from NCHS reports HIST293 and GMWK293R

The high correlation between deaths by flu and all deaths is more evident if we examine age-specific death rate data. I calculate the correlation coefficient between death rates by all causes and death rates by flu across different age groups for each year from 1900 to 2003, which is shown in Figure 3.3. The correlation is above 0.95 for most of the time with only a few exceptions, and it averages to 0.9787. Interestingly, the correlation falls to the lowest value of 0.86 in 1918, which indicates the 1918 Spanish flu has different effects on the death rates of different age-groups. The age-specific effect of the flu attack on death rates is revealed in more

detail in Table 3.3. The 1918 influenza pandemic raised the mortality rate by 30% overall. It affected the age groups 15-24 and 25-34 the most, whereas for individuals aged 55 and over the death rates decreased a little bit. A proper mortality model should reflect the age-specific effect of short-term catastrophic shocks on mortality.

Figure 3.3: Correlation Coefficients between Age-specific Death Rates (per 100,000) by All Causes and by Influenza and Pneumonia



Note: Age-specific death rates are from NCHS reports HIST290 and GMWK290R. The correlation coefficients are calculated by the author.

Table 3.3: Changes of Death Rates (per 100,000) for Each Age Group

Age groups	1917	1918	Ratio	Age groups	1917	1918	Ratio
All	1397.1	1810	1.296	35-44	900.8	1339.3	1.487
<1	10457.2	11167.2	1.068	45-54	1385.6	1524.1	1.1
1-4	1066	1573.5	1.476	55-64	2678.6	2648.1	0.989
5-14	256	412.8	1.613	65-74	5728.4	5505	0.961
15-24	468.9	1070.6	2.283	75-84	12386.2	11295.7	0.912
25-34	649.1	1643.5	2.532	>=85	24593.6	22213.5	0.903

Note: Data are from NCHS report HIST290 and GMWK290R. I calculate the mortality improvement ratios.

3.4. Mortality Modeling: the Generalized Lee-Carter Model

3.4.1. The Lee-Carter Model Overview

Ever since Lee and Carter presented their original work in 1992, the Lee-Carter model has been widely used in mortality trend fitting and projection. The Census Bureau population forecast has used it as a benchmark for the long-run forecast of U.S. life expectancy. The two most recent Social Security Technical Advisory Panels have suggested the Trustees to adopt this method or other methods consistent with it (Lee and Miller, 2001).

Let $m_{x,t}$ denote the central death rate for age x at time t. The model decomposes this time series of age-specific death rates into two sets of age-specific constants a_x and b_x, and a time-varying mortality factor k_t (Lee and Carter referred to k_t as the mortality index. In order to distinguish k_t and the mortality index defined in securitization contracts, I call k_t the mortality factor thereafter). Mathematically, the Lee-Carter model can be represented as follows:

$$\ln(m_{x,t}) = a_x + b_x k_t + e_{x,t}, \tag{3.1}$$

where a_x represents the age pattern of death rates, b_x represents age-specific reactions to k_t, and $e_{x,t}$ is the error term which captures the age-specific effects not reflected in the model.

The Lee-Carter model cannot be fitted by the ordinary least square approach, because all variables on the right side of equation (3.1) are unobservable. Moreover, this model is overparameterized, that is, the solution is determined up to a linear transformation (Lee and Carter, 1992). To obtain a unique solution, normalization conditions are imposed such that the b_x terms sum to unity and the k_t terms sum to zero, i.e.,

$$\sum_x b_x = 1 \text{ and } \sum_t k_t = 0. \tag{3.2}$$

Under the normalized conditions, a_x becomes the average value of $\ln(m_{x,t})$ over time, i.e.,

$$a_x = \frac{1}{T}\sum_{t=1}^{T}\ln(m_{x,t}), \tag{3.3}$$

where T is the length of the time series of mortality data.

Lee and Carter suggest a two-stage procedure to solve this problem. In the first stage, the singular value decomposition (SVD) method is applied to the matrix of $\ln(m_{x,t}) - a_x$, to obtain estimates of b_x and k_t. In the second stage, the k_t factors are re-estimated by iteration given the values of a_x and b_x obtained in the first step, such that the implied number of deaths equals the actual number of deaths, i.e.,

$$D_t = \sum_x \left(Pop_{x,t}\exp(a_x + b_x k_t)\right), \tag{3.4}$$

where D_t is the actual total number of deaths at time t, and $Pop_{x,t}$ is the population in age group x at time t.

Based on the U.S. mortality data for different age groups from 1900 to 2003, I implement this two-stage procedure, report the fitted values of a_x and b_x for 11 age groups in Table 3.4, and plot the final estimates of the mortality factor k_t in Figure 3.4. Generally, the mortality rates of young age groups respond more rapidly when the mortality factor k_t changes. As I expect, the mortality factor k_t is decreasing over time, which shows the trend of mortality improvement. The big jump around 1918 is caused by the severe influenza pandemic in that year.

Figure 3.4: Dynamics of the Mortality Factor k_t from the Lee-Carter Model

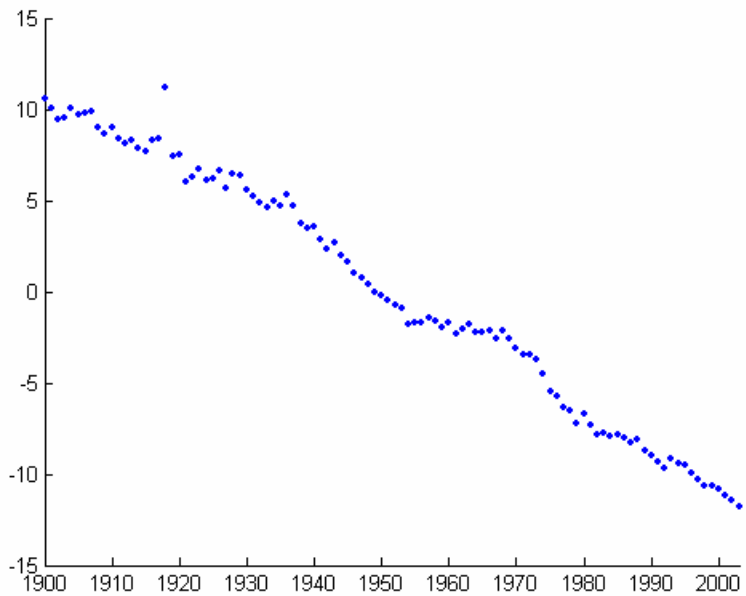


Table 3.4: Fitted Values of a_x and b_x (SVD) from the Lee-Carter Model

Age group	a_x	b_x
Under 1	-3.3935	0.14501
1-4	-6.2072	0.19673
5-14	-7.1833	0.14942
15-24	-6.2877	0.10037
25-34	-5.9837	0.10531
35-44	-5.4745	0.085801
45-54	-4.7734	0.060443
55-64	-4.0071	0.046024
65-74	-3.2289	0.041927
75-84	-2.4146	0.040345
85 over	-1.6084	0.028619

3.4.2. Modeling k_t with a Jump-Diffusion Process: Permanent v.s. Transitory Effects?

To make forecasts of the mortality factor k_t, we need to choose an appropriate model to fit k_t. Cox, Lin and Wang (2006) combine a geometric Brownian motion and a compound Poisson process to model the age-adjusted mortality rates for the U.S. and the U.K. Their model cannot be applied here. First, the mortality factor k_t decreases from positive to negative values. This property restrains us from modeling it with a geometric Brownian motion, because a geometric Brownian motion will never become negative when starting from a positive value. Instead, a process driven by a standard Brownian motion may be applicable. Second, Cox, Lin and Wang (2006) include jumps into the stochastic differential equation, which makes the jumps have persistent effects on mortality rates. However, most of mortality deteriorations are caused by short-term catastrophic events such as the 1918 Spanish flu and the 2004 earthquake and tsunami, and only have transitory effects on mortality rates. We can observe mortality rates pop up when the catastrophic events happen, and fall back to the normal level when the events end. I agree that we need to include mortality jumps in mortality securitization modeling, but I believe we need to assume different jump effects in different pricing scenarios. A model with permanent jump effects may be suitable for pricing longevity derivatives, but it is not appropriate for pricing mortality bonds. Therefore, I model the mortality factor k_t with a standard Brownian motion and a discrete Markov chain with jumps which only have transitory effects. For the purpose of comparison, I also present the model with permanent jump effects in Appendix 3A.

Let N_t be the total number of jumps during the time interval $(0,t)$, and suppose there is at most one jump event in each time period $(t-h,t)$. Therefore, N_t can be expressed as a discrete Markov chain with $N_0 = 0$ and the transition path:

$$N_{t+h} = \begin{cases} N_t + 1, & \text{with probability } p \\ N_t, & \text{with probability } 1-p \end{cases}. \tag{3.5}$$

Let $N_{[t-h,t]} = N_t - N_{t-h}$ be the number of jumps occurring in the period $(t-h,t)$, then $N_{[t-h,t]}$ is a Bernoulli random variable with the probability of jump p.

Denote $\tilde{k}_t$ the mortality factor when there is no jump events. It can be driven by a standard Brownian motion:

$$d\tilde{k}_t = \mu dt + \sigma dW_t, \tag{3.6}$$

where μ and σ are the instantaneous rate of change and the instantaneous volatility of the mortality factor when there is no jumps, and W_t is a standard Brownian motion with mean 0 and variance t.

If a jump occurs in the interval $(t-h,t)$, i.e., $N_{[t-h,t]} = 1$, I assume the jump sizes $Y_{[t-h,t]}$ are identically independently distributed normal variables with mean m and standard deviation s, and $Y_{[t-h,t]}$ is independent of the Brownian motion W_t. The jump $Y_{[t-h,t]}$ makes the actual mortality factor k_t change from $\tilde{k}_t$ to $\tilde{k}_t + Y_{[t-h,t]}$, that is,

$$k_t = \tilde{k}_t + Y_{[t-h,t]}. \tag{3.7}$$

If there is no jump in the interval $(t-h,t)$, i.e., $N_{[t-h,t]} = 0$, we have

$$k_t = \tilde{k}_t. \tag{3.8}$$

(3.7) and (3.8) can be combined and written in one equation

$$k_t = \tilde{k}_t + Y_{[t-h,t]} N_{[t-h,t]}. \tag{3.9}$$

Therefore, the dynamics of the mortality factor k_t can be completely expressed as

$$\begin{cases} d\tilde{k}_t = \mu dt + \sigma dW_t \\ k_t = \tilde{k}_t + Y_{[t-h,t]} N_{[t-h,t]} \end{cases}. \tag{3.10}$$

By integrating the first equation in (3.10) from t to $t+h$, we obtain

$$\tilde{k}_{t+h} = \tilde{k}_t + \mu h + \sigma[W_{t+h} - W_t]. \tag{3.11}$$

From the second equation in (3.10), we can derive

$$\begin{aligned} k_{t+h} &= \tilde{k}_{t+h} + Y_{[t,t+h]} N_{[t,t+h]} \\ &= \tilde{k}_t + \mu h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]} N_{[t,t+h]} \\ &= k_t - Y_{[t-h,t]} N_{[t-h,t]} + \mu h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]} N_{[t,t+h]} \end{aligned} \tag{3.12}$$

Let $z_t = k_{t+h} - k_t$. If we have a time series of K observations of k_t, there will be $K-1$ observations of z's values with time interval equal to $h=1$. z_t and z_{t+h} can be expressed as

$$z_t = \mu h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]} N_{[t,t+h]} - Y_{[t-h,t]} N_{[t-h,t]}, \tag{3.13}$$

$$z_{t+h} = \mu h + \sigma[W_{t+2h} - W_{t+h}] + Y_{[t+h,t+2h]} N_{[t+h,t+2h]} - Y_{[t,t+h]} N_{[t,t+h]}. \tag{3.14}$$

If $N_{[t,t+h]} = 0$, then z_t is independent of z_{t+h}. If $N_{[t,t+h]} = 1$, then z_t is correlated with z_{t+h} because of the $Y_{[t,t+h]}$ part. It is noteworthy that we cannot use the traditional maximum likelihood estimation to calibrate the parameters when the data are not independent.[16] Instead, we should use conditional probabilities to derive the log-likelihood function, which is so-called Conditional Maximum Likelihood Estimation (CMLE). Detailed derivation of the log-likelihood function is included in Appendix 3B.

[16] Lin and Cox (2008) try to combine a geometric Brownian motion with a Markov chain to capture the transitory effect of mortality jumps. However, they don't take into account the correlations of the data, which may bring big errors in their maximum likelihood estimation and cause the estimation results to deviate from the true values. If we don't consider the correlations of the data, then the parameter estimates are u = -0.2172, σ = 0.4018, m = -3.2391, s = 0, p = 0.0098.

Table 3.5: Parameter Estimates via Conditional Maximum Likelihood Estimation

Model with jumps -transitory effect: ln(L) = -62.52			
Parameter	Estimate	Parameter	Estimate
μ	-0.2173	σ	0.3733
m	0.8393	s	1.4316
p	0.0436	LRT Statistics	63.49
Model with jumps -permanent effect: ln(L) = -65.47			
Parameter	Estimate	Parameter	Estimate
μ	-0.2172	σ	0.3872
m	-0.3062	s	2.3133
p	0.0396	LRT Statistics	57.60
Model without jumps : ln(L) = -94.27			
Parameter	Estimate	Parameter	Estimate
μ	-0.2172	σ	0.6043

Note: The critical value for the chi-square distribution (d.f =3, alpha=0.01) is 11.34. Therefore, our likelihood ratio test rejects the model without jump at the significance level of 0.01.

The upper panel of Table 3.5 reports the parameter estimates for the model with transitory jump effects. The expected rate of change of the mortality factor, μ, is -0.2173, which implies the mortality factor k_t decreases by 0.2173 per year on average. The negative sign of μ is consistent with the fact that the U.S. population mortality improves over time. The instantaneous volatility is equal to 0.3733. The probability that there is a jump in a given year is equal to 0.0436.

I also estimate the parameters for the model with permanent jump effects,[17] the results of which are shown in the middle panel of Table 3.5. The instantaneous mean and volatility of the mortality factor are roughly the same as before, while there are significant differences in the

[17] See Appendix 3A for the model with permanent jump effects.

mean and variance of the jump severity distribution between two models. The frequency of jumps decreases from 0.0436 to 0.0396. I will show later that modeling jumps with permanent effects brings a large pricing error in mortality securitizations.

The estimation results for the model without jumps are in the lower panel of Table 3.5. The instantaneous mean of the mortality factor is unchanged, while the instantaneous volatility increases to 0.6043, which is an increment by 61 percent, because the model without jumps incorporates the variations caused by the jump process into the volatility term. I report the values of the log-likelihood functions for different models in Table 3.5, and perform the likelihood ratio test. The test rejects the model without jumps at the significance level of 1%.

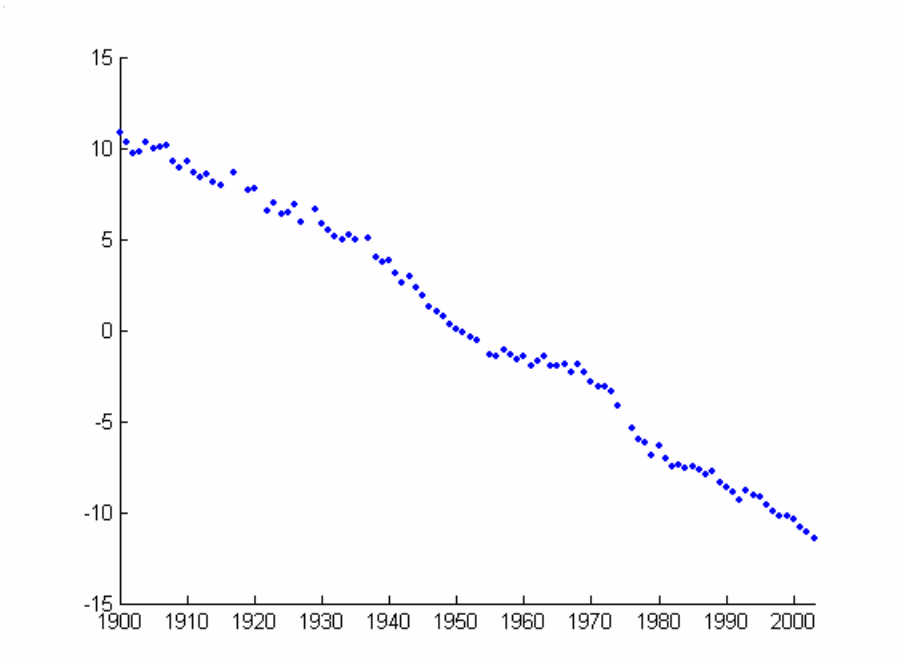
3.4.3. Evidence from the Outlier-adjusted Lee-Carter Model: Do Outliers Matter?

I have already shown that a jump-diffusion process fits the mortality factor k_t better than the model without jumps. The next question is where the mortality jumps come from. Before answering this question, let us briefly review the work done by Li and Chan (2005, 2007). They argue that mortality series are often contaminated with discrepant observations, which may result from recording or typographical errors, or from non-repetitive exogenous interventions such as pandemics or hostilities. In order to reveal the "true" mortality trend, they perform a systematic time-series outlier analysis for the mortality data in the U.S. and Canada, and fit the adjusted outlier-free mortality series to the Lee-Carter model. For the U.S. data from 1900 to 2000, they find 7 outliers, which occurred in year 1916, 1918, 1921, 1928, 1936, 1954 and 1975, respectively. These outliers are closely related to or resulted from influenza epidemics according to their explanations, except for the data in 1954 and 1975.

Do the mortality jumps in my model mainly come from the flu events? Do these outliers

really stand outside the mortality trend? I delete the outliers found by Li and Chan from the original mortality data, estimate the mortality factor k_t again (Figure 3.5), and compare the results for the model with transitory jump effects and that without jumps (Table 3.6). I find that after eliminating the outliers the mortality factor k_t declines more smoothly and does not show significant jumps in the evolution process. In addition, when I fit the mortality factor using the model with transitory jump effects, the probability of a jump in a given year p becomes zero, which actually makes the model with jumps equivalent to the model without jumps. I therefore infer that the mortality jumps arise from these so-call "outliers", which are mostly caused by flu epidemics.

Figure 3.5: Dynamics of the Mortality Factor k_t from the Outlier Adjusted Lee-Carter Model



Note: Mortality data in year 1916, 1918, 1921, 1928, 1936, 1954, and 1975 are deleted according to the outliers analysis of Li and Chan (2007)

Table 3.6: Parameter Estimates via Maximum Likelihood Estimation, using the Outlier-free Mortality Data

Model with jumps -transitory effect: ln(L) = -48.38			
Parameter	Estimate	Parameter	Estimate
μ	-0.2317	σ	0.4005
m	-0.3774	s	0.9316
p	0		
Model without jumps : ln(L) = -48.38			
Parameter	Estimate	Parameter	Estimate
μ	-0.2317	σ	0.4005
Likelihood Raito Test (LRT) statistics =0			

Note: Based on the likelihood ratio test, I cannot reject the model without jumps at the 99% significance level. Actually, for the model with transitory jump effects, the estimated values of m and s are not very stable if I choose different initial values. However, the value of the log-likelihood function remains unchanged.

Is it appropriate to exclude the outliers when we model the death rates for the purpose of pricing mortality-linked securities? As Chan (2002) recognizes, "Whether or not it is appropriate to adjust the data for outliers depends on the purpose to which the model so derived will be used… If…the model will be used in an application for which extreme stochastic fluctuations are important (such as pricing catastrophe risks…), then a model which is sympathetic to outliers in the data ought to be used."[18] We have witnessed the high correlation between deaths caused by flu and deaths by all reasons in the data section, and recognized that mortality jumps mostly result from influenza epidemics. I believe that outliers should not be neglected and that mortality jumps should be explicitly modeled in mortality securitization, since the rationale behind selling or buying mortality securities is to hedge mortality risks. Historic data shows that influenza pandemics happen with frightening regularity, occurring every 30 to 50 years (Knapp, 2006). We should not view the pandemic as a one-time event which will never happen again.

[18] See Chan (2002), page 559-560

3.5. Mortality-Linked Securities Pricing: A Numerical Example

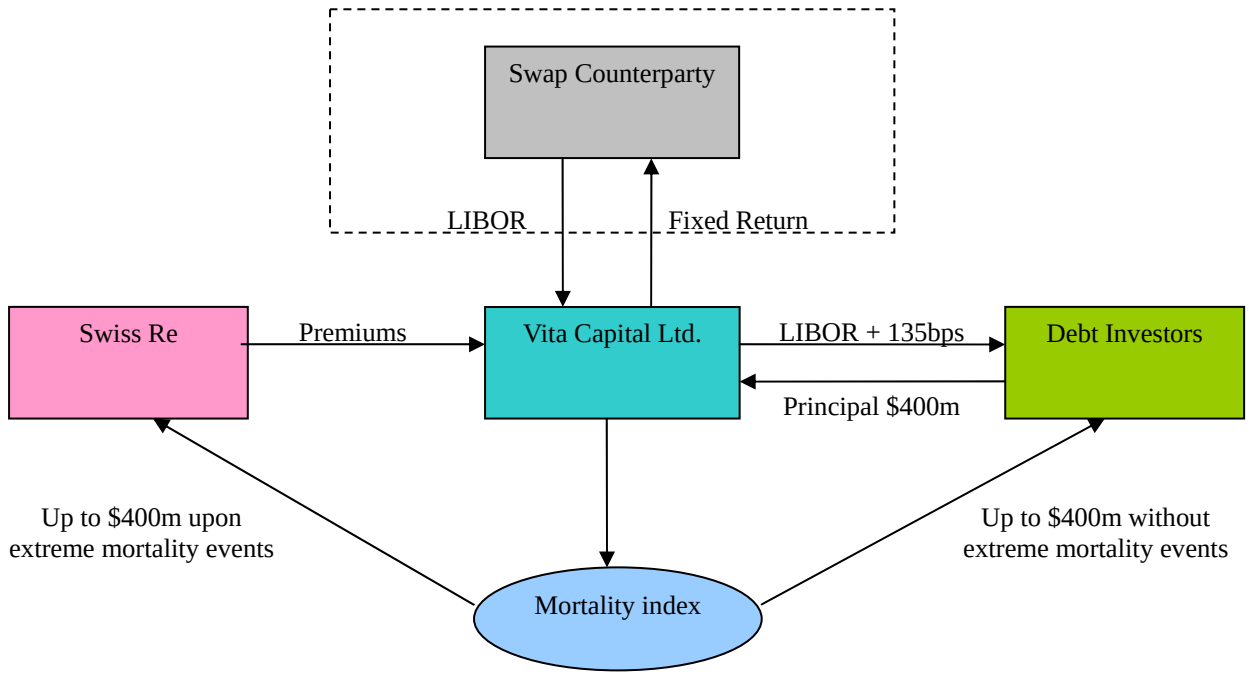
3.5.1. The Swiss Re Mortality Bond and Pricing Difficulties

Modeling mortality jumps seems more important for securities linked to short-term catastrophic risks. Ignoring mortality jumps may cause us to underestimate the probability of having a catastrophic event, and overestimate the variation of the mortality factor (see Table 3.5), which may bring large errors in pricing mortality-linked securities and calculating risk premiums. I, therefore, take the Swiss Re mortality bond (2003) as an example to show how to apply the model here to mortality securitizations.

The Swiss Re transaction is diagramed in Figure 3.6. The Swiss Reinsurance company is the largest reinsurance company in the world. In order to reduce its exposure to catastrophic mortality risks, it issued its first pure mortality bond transaction through a SPV called Vita Capital. The issue size was $400 million. It is a three-year deal: the bond was issued in December 2003 and matured on 1 January 2007. Coupons are paid quarterly at a rate of three-month U.S. dollar LIBOR plus a spread of 135 basis points. Vita Capital executed a swap transaction to swap Swiss Re's fixed premium payment for LIBOR. The principal repayment is at risk and contingent on a weighted mortality index based on five countries, males and females, and different age-groups[19]. If the mortality index does not exceed 1.3 times the 2002 base level during any of the three years, the principal is fully repayable. Otherwise, investors will receive a reduced principal repayment if the mortality index exceeds this threshold, and will get nothing back if the index is above 1.5 times the base level.

[19] The five countries are U.S., U.K., France, Italy and Switzerland. The weights assigned to each country are: U.S. 70%, U.K. 15%, France 7.5%, Italy 5%, Switzerland 2.5%.

Figure 3.6: The Contract Design of the Swiss Re Mortality Bond (2003)



Source: Revised from Blake, Cairns and Dowd (2006)

Let q_t denote the mortality index at year t, and q_0 be the 2002 base level of the mortality index, then the payoff schedule of this bond is shown as follows:

$$f(t) = \begin{cases} LIBOR + spread, & t = 2004,2005 \\ LIBOR + spread + \max\left(1 - \sum_{t=2004}^{2006} loss_t, 0\right), & t = 2006 \end{cases}, \tag{3.15}$$

where the loss ratio in year t is defined as

$$loss_t = \begin{cases} 0, & q_t \le 1.3q_0 \\ \dfrac{q_t - 1.3q_0}{0.2q_0}, & 1.3q_0 < q_t \le 1.5q_0 \\ 1, & q_t > 1.5q_0 \end{cases}, \tag{3.16}$$

There are two difficulties we need to deal with in order to value the Swiss Re mortality bond. First, the mortality index defined in the contract is a weighted average across five countries. The correlation of mortality risks across countries makes the pricing problem difficult. Cox, Lin and Wang (2006) solve this problem by adopting the normalized multivariate

exponential tilting to take into account correlations across countries. Second, the principal repayment of the Swiss Re mortality bond is based on the experience of the mortality index in three consecutive years. The correlation of the mortality index over time makes the problem even more difficult. Cox, Lin and Wang (2006) take the maximum of the mortality index in three years and link the principal repayment to this maximum value. In this way, they actually ignore the correlation over time and change the multiple-period problem into a single-period problem.

In this chapter, I adopt the Wang transform and find a way to take into account correlations of the mortality index over time. For simplicity, I assume the mortality index only depends on the U.S. mortality rates and is weighted across different age groups. The weight is determined by the year 2000 standard population. However, my methodology can be easily extended to the case where the mortality index is weighted by countries and age-groups, using multivariate exponential tilting.

3.5.2. The Methodology: from Exponential Tilting to the Wang Transform

Exponential tilting, as a general method for neutralizing the statistical distribution, is broadly consistent with much of the current literature on no-arbitrage pricing of contingent claims (Duffie, 1992; Heston, 1993; Karatzas and Shreve, 1992; Gerber and Shiu, 1996). It is widely applicable in pricing risks embedded in loan defaults, mortgage refinancing, electricity trading, weather derivatives, and catastrophic insurance (Wang, 2007).

Consider two risks X and Y. The exponential tilting of X with respect to Y is defined as:

$$f_X^*(x) = f_X(x)\frac{E\left[\exp(\lambda Y) \mid X = x\right]}{E\left[\exp(\lambda Y)\right]}, \tag{3.17}$$

where f_X and f_X^* represent the probability density function of X before and after the exponential tilting, respectively.

The ratio

$$RN(x) = \frac{f_X^*(x)}{f_X(x)} = \frac{E[\exp(\lambda Y) \mid X = x]}{E[\exp(\lambda Y)]} \tag{3.18}$$

is the Radon-Nikodym derivative of f_X^* with respect to f_X.

For the exponential tilting in equation (3.17), we do not have a consistent interpretation of the λ parameter; the scale and shape of the reference variable Y can significantly impact the result of the exponential tilting. In order to get a consistent interpretation of λ, Wang (2003) propose a normalization procedure of the reference variable Y through percentile-matching. That is, he defines $Z = \Phi^{-1}(F_Y(Y))$ as a normalized variable of Y, and implement a normalized exponential tilting of X with respect to reference Y as follows:

$$f_X^*(x) = f_X(x)\frac{E[\exp(\lambda_0 Z) \mid X = x]}{E[\exp(\lambda_0 Z)]}, \tag{3.19}$$

where λ_0 is the parameter corresponding to Z.

Under the assumption that X and Y have bivariate normal copula with a correlation coefficient of ρ, the normalized exponential tilting in equation (3.19) is reduced to the Wang transform:

$$F_X^*(x) = \Phi\left[\Phi^{-1}(F_X(x)) - \lambda\right], \tag{3.20}$$

where $\lambda = \rho\lambda_0$. See Wang (2003) for a proof and further discussions.

One important feature of the Wang transform is that it preserves the normal and lognormal distribution. Specifically, if X has a normal (μ, σ^2) distribution, then after the Wang transform X is still normally distributed with $\mu^* = \mu + \lambda\sigma$ and $\sigma^* = \sigma$. If X has a lognormal (μ, σ^2) distribution, then X is still a lognormal variable with $\mu^* = \mu + \lambda\sigma$ and $\sigma^* = \sigma$ after the Wang transform. This property enables the Wang transform to replicate the CAPM if the return for an

underlying asset has a normal distribution and recover the Black-Scholes formula if the return for the underlying asset is lognormally distributed. For this reason, Wang refers to λ as the market price of risk. I will follow him and call λ the market price of risk thereafter.

The Wang transform produces a risk-adjusted CDF $F_X^*(x)$ for a given asset X with CDF $F_X(x)$. Calculating $E^*[X]$, the expected value of X under $F_X^*(x)$, and discounting it back to time zero using the risk-free interest rate, we can get the fair value of the asset X.

3.5.3. The Pricing Framework and Numerical Results

Recall that in the model section, I work with the following dynamics under the physical probability measure P:

$$\begin{cases} d\tilde{k}_t = udt + \sigma dW_t \\ k_t = \tilde{k}_t + Y_{[t-h,t]} N_{[t-h,t]} \end{cases}, \tag{3.21}$$

where $W_t \sim N(0,t)$, $Y_{[t-h,t]} \sim N(m, s^2)$ and $N_{[t-k,t]} = \begin{cases} 0, & prob = 1-p \\ 1, & prob = p \end{cases}$ under P.

Assuming the Brownian motion W_t, the jump severity Y, and the jump frequency N are independent with each other, I apply the Wang transform to W_t, Y, and N respectively. Under the risk-adjusted measure Q, W_t^* is normally distributed with mean $\lambda_1 t$ and variance t, $Y_{[t-h,t]}^*$ is normally distributed with mean $m + \lambda_2 s$ and variance s^2, and $N_{[t-h,t]}^*$ is a Bernoulli random variable with the probability of jumps p^*, where $p^* = 1 - \Phi[\Phi^{-1}(1-p) - \lambda_3]$.[20] [21]

[20] Here I denote the variables after the Wang transform with subscript *, which indicates that the distribution of the variable changes in the risk-adjusted measure.

[21] Note that the market price of risk increases with the time horizon, i.e., $\lambda = \lambda_1 \sqrt{t}$, where λ_1 represents the market price of risk per annum. $W_t^* \sim N(\lambda\sqrt{t}, t) \sim N(\lambda_1\sqrt{t} \times \sqrt{t}, t) \sim N(\lambda_1 t, t)$. See Wang (2002) for more detailed discussions on this issue.

Mathematically, the dynamics of the mortality factor under Q becomes

$$\begin{cases} d\tilde{k}_t^* = \mu dt + \sigma dW_t^* \\ k_t^* = \tilde{k}_t^* + Y_{[t-h,t]}^* N_{[t-h,t]}^* \end{cases}, \tag{3.22}$$

where $W_t^* \sim N(\lambda_1 t, t)$, $Y_{[t-h,t]}^* \sim N(m + \lambda_2 s, s^2)$, and $N_{[t-h,t]}^* = \begin{cases} 0, & prob = 1 - p^* \\ 1, & prob = p^* \end{cases}$

Here the parameter λ_1, λ_2 and λ_3 represent the market prices of risk associated with the Brownian motion, the jump severity and the jump frequency, respectively. Because we have an incomplete market for mortality-linked securities, the values of λ_1, λ_2 and λ_3, and thus the choice of the risk-adjusted measure Q, are not unique.

I use the following procedure to calculate the market prices of risk of the Swiss Re mortality bond (2003) based on the Wang transform.

- Because the mortality factor k_t is correlated over time, we cannot simulate k_t for each year independently. We need to simulate the path of the mortality factor k_t (t = 2004, 2005, and 2006). I do this by 10,000 times, using the jump-diffusion process (3.21) and the parameter estimates shown in the upper panel of Table 3.5.

- I use the Wang transform to change the mortality factor k_t from the physical probability measure P to the risk-adjusted probability measure Q, and obtain the values of k_t^* on each path under Q, given initial values of the market prices of risk λ_1, λ_2 and λ_3.

- I calculate the mortality rates for different age groups according to the formula $m_{x,t}^* = \exp(a_x + k_t^* b_x)$ under Q. The year 2000 standard population and corresponding weights are used to compute the weighted average mortality index q_t^* under Q for each

year. [22]

- I calculate the loss ratio, $loss_t^*$, under Q by the equation (3.16), and compute the risk-adjusted expected value of the principal repayment of the Swiss Re mortality bond as of the period T.

$$E_T^*[repayment] = 400 \text{ million} \times \left(\frac{1}{10{,}000} \sum_{i=1}^{10{,}000} \left[\max(1 - \sum_t loss_{i,t}^*, 0) \right] \right), \qquad (3.23)$$

- I discount the coupon payments each period and the principal repayment back to the beginning of year 2004, using the risk-free rates.[23] Setting the discounted expected payoff equal to the issue size of the Swiss Re mortality bond $400 million, I obtain the market prices of risk, λ_1, λ_2 and λ_3, via the numerical iteration such as the Quasi-Newton method.

Note that we have one mortality bond price and need to estimate three market prices of risk. Therefore, we cannot solve λ_1, λ_2 and λ_3 simultaneously. As Cairns, Blake and Dowd (2006b) demonstrate, we can estimate λ_1, λ_2 and λ_3 by fixing two of them and solving for the third. We can also assume the market prices of risk are equal, i.e., $\lambda_1 = \lambda_2 = \lambda_3 = \lambda$, and solve for λ consequently, which is the method used by Cox, Lin and Wang (2006).[24]

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[22] The year 2000 standard population and corresponding weights can be obtained from the technique notes of the NCHS report GMWK293R. The weight is 0.013818 for age under 1 year, 0.055317 for age 1-4, 0.145565 for age 5-14, 0.138646 for age 15-24, 0.135573 for age 25-34, 0.162613 for age 35-44, 0.134834 for age 45-54, 0.087247 for age 55-64, 0.066037 for age 65-74, 0.044842 for age 75-84, 0.015508 for age 85 and over.

[23] I use the US Treasury yield rates on December 30, 2003 as the risk-free rates. I calculate the coupon payment by assuming it is paid annually, because I don't have quarterly data for US Treasury yield rates.

[24] The purpose of this paper is to propose an appropriate mortality model and develop a valuation strategy to account for the pricing difficulties of the Swiss Re mortality bond. For simplicity, I only consider one transaction which occurred in 2003. Actually, the Swiss Reinsurance company issued another 3 mortality bonds in 2006. My model and pricing strategy can be easily extended to multiple-transaction situations. One can find optimal λ_1, λ_2 and λ_3 by minimizing the target function $\sum [\hat{P}_i(\lambda_1, \lambda_2, \lambda_3) - P_i]^2$, where $\hat{P}_i(\lambda_1, \lambda_2, \lambda_3)$ is the modeled price of the i-th issue depending on the parameters λ_1, λ_2 and λ_3, and P_i is the actual market quote for the i-th transaction.

Table 3.7: Implied Market Prices of Risk for Different Models

Model with transitory jumps	λ_1	5.1449	0	1.5000
	λ_2	0	3.4808	1.5000
	λ_3	0	0	1.5000
Model with permanent jumps	λ_1	4.6408	0	0.8072
	λ_2	0	2.0006	0.8072
	λ_3	0	0	0.8072
Model without jumps	λ_1	2.9921		

Based on the par spread of the Swiss Re bond 1.35%, the implied market prices of risk for different models are shown in Table 3.7.[25] It is noteworthy that what I estimate here are the market prices of risk associated with the Brownian motion, the jump severity, and the jump frequency for the mortality factor k_t, instead of the true mortality rates. Because the mortality factor k_t has a much larger scale than the true mortality rates (k_t changes from 10.6 in 1900 to -11.8 in 2003, and the age-adjusted mortality rate changes from 0.02518 in 1990 to 0.00833 in 2003), it is not surprising to see that the market prices of risk estimated here have high values.

Under the model with transitory jump effects, if I assume that the risk associated with the jump process is diversifiable, i.e., $\lambda_2 = \lambda_3 = 0$, the market price of risk associated with the Brownian motion is 5.1449. If there is no systematic risk of the Brownian motion and the jump frequency, i.e., $\lambda_1 = \lambda_3 = 0$, the market price of risk associated with the jump severity is 3.5004. Of course, these can be regarded as the extreme cases. If I assume $\lambda_1 = \lambda_2 = \lambda_3 = \lambda$, I can solve for $\lambda = 1.5$.

I notice that when I switch to the model with permanent jump effects, the estimated market

[25] I don't report the case where there is only systematic risk associated with the jump frequency. For the model with transitory jumps, our estimation results show that it is not enough to only adjust the distribution of the jump frequency.

prices of risk drop dramatically in each case. This can be explained by the large difference in the volatility of the jump severity distribution and the difference in the intrinsic model setup. First, if I model mortality jumps to have permanent effects, the jump effects accumulate over time. Therefore, the forecasted mortality rates are more inclined to reach the predetermined threshold level, which indicates a higher risk on the principal repayment. Second, the volatility of the jump severity is 2.3133 in the model with permanent jump effects, while it is 1.4316 in the model with transitory jump effects (see Table 3.5). The higher volatility of the jumps in the former model raises the risk further. When the par spread of the Swiss Re is fixed at 1.35%, the higher risk imposed on the principle repayment, the lower market price of risk I obtain. Therefore, modeling mortality rates with permanent jump effects underestimates the market prices of risk.

I come to the model without jumps at last. The estimated market price of risk associated with the Brownian motion is 2.9921, which is much lower than that for the model with jumps, whether the jumps have permanent or transitory effects. The model without jumps overestimates the variation of the mortality factor while underestimating the probability of catastrophic events. I suspect that the effect of overestimating the variation dominates the effect of underestimating the catastrophic probability, which brings down the market price of risk associated with the Brownian motion.

3.6. Conclusions and Discussions

In this chapter, I give a deep discussion of mortality modeling and mortality-linked security pricing. A good stochastic mortality model for pricing mortality-linked securities should meet the following criteria, while none of the previous research addresses all these problems.

- The model should capture both the mortality trend over time and the age-specific

changes for different age groups. Modeling the age-adjusted mortality rates is not enough, since the payoffs of mortality-linked securities sometimes depend on a mortality index based on different age groups.

- The model should incorporate a mortality jump process explicitly. Mortality jumps caused by short-term catastrophic events, such as the 1918 Spanish Flu, cannot be ignored because the rationale of mortality securitizations is to hedge extreme mortality risks.
- Adverse mortality jumps have transitory effects on mortality rates. It is inappropriate to model adverse mortality jumps with permanent effects, especially when we value mortality bonds, because most of adverse mortality jumps are caused by short-term catastrophic events and the effects should fade away after one or several periods.
- The model with transitory jump effects introduces correlations of the data. When estimating the parameters in the model, we cannot simply assume the data are independent and use the traditional maximum likelihood estimation.

I extend the work of Cox, Lin and Wang (2006), and address all of the problems mentioned above. I make the first attempt to incorporate a jump process into the Lee-Carter model and discuss in detail how to model the mortality factor k_t with transitory jump effects. Second, I derive the conditional log-likelihood function and estimate the parameters via CMLE, provided that the data are correlated over time. Big estimation errors occur if we assume the data are independent, which is the problem in previous studies (e.g., Lin and Cox, 2008). Third, I compare the model with permanent jump effects to that with transitory jump effects, and demonstrate how the difference will cause a large pricing distortion. Finally, this study contributes to the existing literature by showing how to account for correlations of the mortality

index over time when pricing the Swiss Re mortality bond (2003). The basic idea is to simulate the paths of the mortality index and change measures on paths.

A line of future research may focus on how to decide an “optimal” transform in an incomplete market. As suggested by Cox, Lin and Wang (2006), although the change of measures is not unique in an incomplete market, we can try to apply the minimum martingale transform to find a strategy that minimizes the variance of the payoff risk. Second, I ignore the issue of parameter uncertainty in this paper. I simulate the mortality index using estimates of the parameters, assuming these parameter estimates are true values without ambiguity. Another line of future research is to relax this assumption and consider the valuation and hedging of mortality-linked securities under parameter uncertainty.

4

Valuation of the Non-Recourse Provision in Reverse Mortgages: Understanding and Modeling the Risks

Although we cannot predict the mortality deterioration caused by some randomly occurring catastrophic events, we have seen a steadily increasing life expectancy over the last several decades. In the United States, the recorded life expectancy at birth was 60.95 in 1933. It increased to 77.87 in 2004[26]. Since the 1970's, the reduction of death rates among the elderly has become the main factor driving continued gains in life expectancy in developed countries. These mortality improvements place strains not only on fiscal sustainability of social security systems, but also on individuals' capacity to accumulate enough private savings to finance their retirement (Wang, Valdez and Piggott, 2007). The reverse Mortgage, recognized as an important financial vehicle to supplement current social security systems for the elderly, has attracted considerable

[26] According to the data from Human Mortality Database.

attention recently. In this chapter, I investigate the structural design and embedded risks in reverse mortgages. As a step toward further discussion of pricing mortality-linked securities in an incomplete market, I employ the conditional Esscher transform to value the non-recourse provision of a reverse mortgage.

4.1. Introduction

According to the statistics from the U.S. Census Bureau, more than 34 million Americans live above age 65. That number is expected to increase to 71 million by the year 2030, which will account for 19.6% of the population.[27] With the retirement of baby-boomers and the increase in the share of the elderly in the population, the retirement programs in the U.S. face an "aging-population tsunami" and significant future imbalances consequently. In addition, the shift of pension plans from defined benefit (DB) to defined contribution (DC) and the declining contribution levels from employers impose big challenges on financial budgets of the aged population after their retirement. The elderly may not only receive reduced monthly incomes, but also experience rising health-care costs and decreasing pension plan benefits. It is increasingly difficult for them to maintain financial independence and the standard of living. However, the American Housing Survey shows that more than 12.5 million elderly have no mortgage debt, and the median value of the unmortgaged homes is $127,959.[28] "House rich and cash poor" is the phrase often used to describe their dilemma.

Since the 1970s, academics and practitioners have sought to create mortgage instruments to enable elderly homeowners to borrow by using the equity in their homes as collaterals, which are

[27] U.S. Census Bureau. International database. Table 094.
Available at http://www.census.gov/population/www/projections/natdet-D1A.html

[28] American Housing Survey for the United States: 2005, Current Housing Reports. H150/05. U.S. Department of Housing and Urban Development and U.S. Census Bureau. Aug. 2006, P156

referred to as reverse mortgages. Providers of reverse mortgages advance a lump sum or periodic payments to elderly homeowners. The loans accrue with interests and are settled only when borrowers die, sell or vacate their homes to live elsewhere. Borrowers enjoy the favorable merits of reverse mortgages in several aspects. First, it allows homeowners to convert their home equity into cash flows without having to move out of the properties. Second, borrowers have no obligation to repay the loans as long as they are alive or reside in their homes. Finally, the repayments are capped with the proceeds from the sale of the properties. When a loan is terminated, if the loan balance is larger than the property value (usually referred to as crossover risk), the provider recovers only up to the sale price of the property. This is the so-called non-recourse provision.

In 1989, after the Department of Housing and Urban Development (HUD) introduced the Home Equity Conversion Mortgage (HECM) program, reverse mortgages became widely available in the United States. There are three major reverse mortgage programs in the U.S. market, which are the HUD's HECM, Fannie Mae's Home Keeper program, and Financial Freedom's Cash Account Plan. In late 2006, the Lifestyle Plan was introduced in the states of California, Oregon and Washington, and the plan was expected to be available nationally during 2007. Among them, the HECM program is considered the safest and the most popular program in the U.S., since it is insured by the U.S. federal government and accounts for 95 percent of the market share. For this reason, I focus on analyzing the HECM program in this study.

The main purpose of this study is to develop a framework to model the embedded risks, value the non-recourse provision, and calculate the present value of insurance premiums in the HECM program. As pointed out by Phillips and Gwin (1992), the lending feature of reverse mortgages subject loan providers to multiple risks. An increase of the lifespan of the loan

resulted from mortality improvement or reduced mobility rates will impose a higher crossover risk. A rise in interest rates will speed up the rate at which the loan accumulates, and will possibly hit the crossover point earlier. Finally, a depressed real estate market will worsen the value of the home. Most of the existing literature on risk modeling in the HECM program has two major flaws. First, some of them use period life tables, and thus neglect the dynamics of mortality rates (see Tse, 1995; Zhai, 2000; Weinrobe, 1988; Szymanoski, 1994). Moreover, almost all of previous studies ignore longevity risk and do not model the mortality improvement jumps explicitly. I employ the generalized Lee-Carter model proposed in Chapter 3 to model the mortality rates and construct dynamic life tables for pricing the non-recourse provision and calculating insurance premiums. It is noteworthy that I adapt the Lee-Carter model to incorporate permanent jump effects instead of transitory jump effects, since the main concern of reverse mortgage providers is the longevity risk, which is persistent over time. Second, traditional HECM models assume house prices are driven by a geometric Brownian motion (e.g., Szymanoski, 1994; Ma, Kim and Lew, 2007; Wang, Valdez and Piggott, 2007). However, I find that house price returns exhibit strong autocorrelation and varying volatility over time, which is inconsistent with the “memoryless” feature of geometric Brownian motions. Therefore, I follow Li, Boyle, Hardy and Tan (2007)’s work by using an ARIMA-GARCH model to fit house price returns.

The non-recourse provision in reverse mortgages can be viewed as offering borrowers a series of European exchange options with different times to maturity (Chinloy and Megbolugbe, 1994). Valuation of the non-recourse provision requires us to create an equivalent martingale measure in an incomplete market, since multiple risks are involved in reverse mortgages. The adoption of the GARCH model for house price returns suggests that the change of probability

measure via the conditional Esscher transform be applicable in this circumstance. Till now, there is a substantial literature for option valuations in the GARCH framework since the pioneering work of Duan (1995). He introduces the notion of locally risk-neutral valuation relationship (LRNVR), which preserves the one-period-ahead (local) conditional variances of the stock-price dynamics. Siu, Tong and Yang (2004) adopt the conditional Esscher transform to the valuation of derivatives under the general class of GARCH models. They recover Duan's pricing results, assuming the GARCH innovations are conditionally normal. Li, Boyle, Hardy and Tan (2007) apply Siu et al.'s pricing framework, when the conditional mean equation is given by an ARMA process, to price the No-Negative-Equity-Guarantee in equity release markets in the U.K. Their approach can be extended slightly here to value the non-recourse provision of reverse mortgages when the mean equation follows an ARIMA process.

In order to protect lenders of reverse mortgages from possible losses, the Federal Housing Administration (FHA), which is one part of HUD's Office of Housing, charges insurance premiums from borrowers, and pays insurance claims to lenders in case the loan balance exceeds the equity value at the time of settlement. Theoretically, the present value of expected premiums should be equal to the value of the non-recourse provision, under the actuarial equivalence principle. I examine the premium structure of HECM loans and investigate whether insurance premiums are adequate to cover expected claims. I find that the collected premiums exceed the actuarial present value of claim payments to lenders. The HECM program is sustainable.

The rest of this chapter is organized as follows. In section 4.2, I review the basic facts of reverse mortgages, using HECM as an example, and develop the pricing formula for the non-recourse provision and insurance premiums. In section 4.3, I discuss various risks involved in the HECM program. I build a model for longevity risk and provide details on how to construct a

dynamic life table in section 4.4. I review the literature on house price dynamics and model house price returns as an ARIMA-GARCH process in section 4.5. In section 4.6, I discuss the conditional Esscher transform and provide the pricing framework. I value the non-recourse provision and calculate the present value of insurance premiums consequently. Finally, section 4.7 concludes.

4.2. The HECM Program and the Non-Recourse Provision

The HECM program was authorized by HUD in the Housing and Community Development Act of 1987. Since 1990, there are more than 308,000 elderly homeowners taking advantage of the HECM program.[29] The market even booms at an incredible rate recently. According to the national Reverse Mortgages Lenders Association, 37,829 HECM loans were originated in 2004, representing a 109% jump over the previous year and nearly 500% growth since 2001. The growth continued in 2005 when 43,131 HECM loans were approved (Wang, Valdez and Piggott, 2007). Currently, it has become the most popular program of its kind in the U.S. Over 95 percent of all reverse mortgage borrowers choose the HECM products (Ma and Deng, 2006). This section reviews the basic facts of the HECM program, to prepare for further discussions in modeling and pricing sections later on.

4.2.1. Borrower Requirements

Under the HECM program, borrowers must be at least 62 years of age, living in a single-family property that meets HUD's minimum property standard. They must own their homes free and clear, which implies that any home purchase mortgage must be fully repaid either prior to

[29] Sioris, N. Over 300,000 HECM Reverse Mortgages Closed. June 28, 2007.
Available at http://www.letyourhomepayyou.com/2007/06/over-300000-hecm-reverse-mortgages.html

HECM or from the initial proceeds of the HECM product. Before closing, borrowers must attend counseling where third parties will explain the financial implications of entering into the HECM program as well as other options that may be available.

4.2.2. Payment Options

These are the payment options that borrowers can choose from:

- Lump sum – The borrower receives the entire available principal limit at closing.
- Line of credit – Installments are made at times and in amounts of the borrower's choosing until the line of credit is exhausted.
- Tenure – Equal monthly payments are made as long as the borrower lives and continues to occupy the property as a principal residence.
- Term – Equal monthly payments are made for a fixed period of months selected by the borrower.
- Modified tenure – Combination of line of credit with monthly payments for as long as the borrower remains in the home.
- Modified term – Combination of line of credit with monthly payments for a fixed period of months selected by the borrower.

4.2.3. Initial Principal Limit

The initial principal limit (IPL) is the initial loan amount that may be extended to a borrower by a lender, which equals the present value of the monthly payments offered to borrowers (Deutsche Bank, 2007). In the case of a lump sum option, it is the amount of cash advance that a borrower can get at the time of loan origination. It is determined by the age of the

borrower, the expected interest rate, and the adjusted property value, which is the minimum of the appraised value of the property and the mortgage limit imposed by FHA for one-family house in that specific area.

4.2.4. Interest Rate Options

The interest rate charged on the loan may be fixed or adjustable, with annual or monthly adjustments linked to the one-year Treasury bill rate. However, 99% of the HECM loans issued to date had an adjustable interest rate, largely because Fannie Mae (Federal National Mortgage Association), which has purchased nearly all of the loans issued under the program, does not purchase fixed-rate loans.

4.2.5. The Termination Time

To prevent involuntary displacement of elderly homeowners, the HECM loan does not require any repayment until a borrower sells the property, moves out, or dies. A borrower also has the option of prepaying the loan without any penalty. A foreclosure can only take place when a borrower discontinues paying monthly property taxes and insurance, or fails to maintain the property up to a minimum maintenance level.

4.2.6. The Non-Recourse Provision

The HECM loan is a non-recourse debt. When the loan terminates, if the net proceed from the sale of the property is sufficient to pay the outstanding loan balance, the remaining cash usually belongs to the borrower or his/her beneficiaries. If the sale proceed is not enough to cover the loan balance, the non-recourse provision prevents the lender from pursuing other assets

belonging to the borrower, apart from the house.

Denote L_t the outstanding balance of the loan and H_t the value of the mortgaged property at a random time t. If the loan is due at time t, the borrower pays L_t if $H_t \geq L_t$, and H_t if $H_t < L_t$, under the non-recourse provision. The cash flow function of the borrower can be written as follows:

$$repayment = \begin{cases} -H_t, & H_t < L_t \\ -L_t, & H_t \geq L_t \end{cases}$$

$$= -L_t + \max(L_t - H_t, 0). \tag{4.1}$$

Equation (4.1) means the non-recourse provision is equivalent to writing the borrower an European exchange option which changes the mortgaged property value H_t for the loan outstanding balance L_t. The borrower, therefore, is holding a debt position and an exchange option. At the time of termination, the borrower needs to pay the loan balance, but he or she gets the payoff from the exchange option at the same time. Note the termination time t is random here. More precisely, the non-recourse provision is equivalent to writing the borrower a series of European exchange options with different times to maturity (Chinloy and Megbolugbe, 1994; Li, Boyle, Hardy and Tan, 2007). Let ω be the highest attained age, the payoff from the non-recourse provision written on a cohort group aged x can be expressed as follows:

$$\text{Value} = \sum_{t=0}^{\omega-x-1} E_Q\left[{}_t p_x q_{x+t} e^{-rt} \max(L_t - H_t, 0)\right], \tag{4.2}$$

where r is the risk-free interest rate, ${}_t p_x$ is the probability that an individual aged x will survive another t years, q_{x+t} is the probability that an individual aged $x+t$ will die in one year, and E_Q denotes the expectation under the risk-adjusted measure Q.

4.2.7. Insurance Premiums

In order to protect lenders from possible losses if non-repayment occurs, as well as to guarantee borrowers receiving monthly payments if lenders default on the loans, HUD provides mortgage insurance for the HECM program. Two insurance options are available for lenders to choose from, which are the assignment option and the shared premium option. However, none of the lenders choose the shared premium option because Fannie Mae does not purchase these loans. With the assignment option, FHA collects all the insurance premiums and the lender is allowed to assign the loan to FHA when the loan balance equals the adjusted property value. FHA takes over the HECM loan and pays an insurance claim to the lender covering his/her losses. By choosing this option, lenders are effectively shifting the collateral risk to HUD.

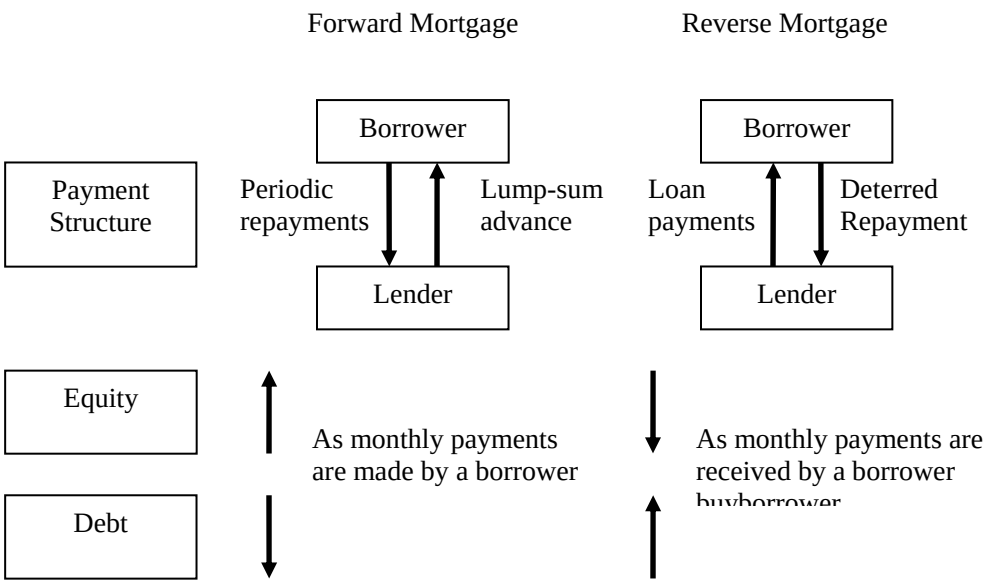
The mortgage insurance premiums (MIP) are paid by borrowers and include an upfront premium of 2% of the adjusted property value and an annual rate of 0.5% of the loan outstanding balance as long as the loan is active. Mathematically, the insurance premiums can be calculated as

$$\text{Premium} = 0.02H_0 + \sum_{t=1}^{\omega-x-1} {}_t p_x e^{-rt}\left(0.005L_t\right). \tag{4.3}$$

4.3. Modeling Insurance Risks in the HECM Loans

Reverse mortgages differ from traditional forward mortgages in the way that the outstanding loan balance grows due to principal advances, interest accruals, and other loan charges over the life of the loan, which can been observed from Figure 4.1. The loan balance may grow to exceed the property value at the time of termination because of multiple risks.

Figure 4.1: Comparison between a Forward Mortgage and a Reverse Mortgage



Source: This figure is revised from Deutsche Bank Report (2007)

4.3.1. The Termination Risk

If a borrower lives a longer time than the expected lifespan, the principal advances and interest accruals will continue, which may lead the loan balance above the sale proceed of the property. In other words, lenders of reverse mortgages are faced with longevity risk. The mobility rate has the same effect on reverse mortgage products. Borrowers may move out of their homes because of their health conditions, marriage, divorce, death of the spouse, disasters, or simply the desire to live in another place. The mobility rate for the U.S. population is observed to decrease with age initially, but starts to increase after a certain old age such as 80 (Zhai, 2000). However, there is little data for us to calibrate the mobility rate for HECM borrowers. HUD simply assumes that the mobility rates are approximately 30 percent of the mortality rates in the HECM model (see, e.g., Rodda, Lam and Youn, 2004; Deutsche Bank Report, 2007). In other words, the termination rates are roughly 1.3 times the death rates. I use the same assumption in

this study.

4.3.2. The Interest Rate risk

HECM loans almost exclusively opt for adjustable interest rates, therefore the variation of interest rates imposes additional uncertainty on HECM insurers. The rise of interest rates can result in a higher rate of interest accruals on the loan balance than anticipated, which increases the possibility of non-repayment when the loan eventually terminates. In this study, I choose a fixed interest rate with a risk adjustment, as most of the HECM models did. The question is: what is the appropriate risk premium in order to equate the value of the non-recourse provision to the present value of insurance premiums? I will answer this question in section 4.6.

4.3.3. The House Price Depreciation Risk

The uncertainty in house price depreciation rates is another risk we need to consider. If the house price remains stagnant or grows at a lower rate than anticipated, the outstanding loan balance at maturity may exceed the sale proceed of the property. Lenders may suffer from the losses and file insurance claims with the FHA. The house price depreciation risk is only partially diversifiable, because pooling mortgage products nationally only minimizes the risk of regional economic recession, but cannot diversify the risk of national economic recession. Additionally, the house price index is not a stationary time-series variable, which implies a simple risk adjustment is not applicable (Szymanoski, 1994).

4.4. Modeling the Longevity Risk

Longevity risk is defined as the uncertainty of mortality improvement in the future. It exists

in both individual and aggregate levels (MacMinn, Brockett and Blake, 2006). Individuals may face longevity risk of outliving their resources. However, they can insure against the risk through the social security systems, defined benefits plans, and private annuity products. Pension plans and insurers can diversify the longevity risk at the individual level following the law of large numbers, assuming lives are independent. In the aggregate sense, longevity risk is referred to as the fact that people of some certain population might live longer, on average, than expected. For instance, Hardy (2005) points out that the life expectancy for men aged 60 is 5 years' longer in 2005 than it was anticipated in the mortality projection made in the 1980s. Such risk breaks down the risk pooling mechanism and becomes non-diversifiable, making the provision of risk management tools increasingly difficult.

The traditional HECM model uses period life tables and therefore fails to capture the dynamics of longevity risk over time. In addition, it does not model the mortality improvement jumps explicitly. In the HECM program, an unexpected mortality improvement will increase the life expectancy and affect the premium pricing persistently. For example, heart disease is one of the leading causes of death in the United States. It accounts for 28.5 percent of the total death in 2002 according to the report of National Center of Health Statistics (Vol. 53, No. 5, October 2004). If there were a breakthrough of medicine protecting people from heart attack, the mortality rates would decrease substantially and the effects of mortality improvement would last for a long period, even forever. In chapter 3, I built a jump-diffusion process in the Lee-Carter framework and explored the models with permanent versus transitory jump effects. I found that the model with transitory jump effects is more appropriate for mortality risk (or short-term catastrophic risk), because most of adverse mortality jumps are caused by catastrophic events and only have short-term effects. Likewise, I believe that the generalized Lee-Carter model with

permanent jump effects is applicable for longevity risk, since the main concern of reverse mortgage lenders is the aggregate mortality improvement risk that seems to be due to lasting factors which impact the whole population for a long time.

Because I have already discussed the generalized Lee-Carter model with jumps in detail in chapter 3, I only briefly state the steps I am doing here:

- Model the central death rates $m_{x,t}$ by the classical Lee-Carter Model,

$$\ln(m_{x,t}) = a_x + b_x k_t + e_{x,t}, \tag{4.4}$$

  and estimate the parameters a_x, b_x and k_t following the two-stage procedure.

- Model the time series k_t by a jump-diffusion process with permanent jump effects,

$$dk_t = (\mu - pm)dt + \sigma dW_t + Y_t N_t, \tag{4.5}$$

  and estimate the parameters μ, σ, p, m and s via MLE.

- Forecast the mortality factor k_t ($t = 1,2,3,...$) in 1,000 paths according to the discrete approximation

$$k_{t+1} = k_t + \mu - pm + \sigma Z_{t+1} + Y_{t+1} N_{t+1}; \tag{4.6}$$

  where Z_t is a standard normal random variable.

- Obtain the forecasts of central death rates in 1000 paths

$$m_{x,t} = \exp(a_x + b_x k_t). \tag{4.7}$$

- Construct the dynamic complete life table for every year. That is, calculate $q_{y,t}$ ($t = 1,2,3,...$), the probability that an individual aged y will die in one year at time t (see Appendix 4A for details).

4.5. Modeling the House Price Depreciation Risk

4.5.1. The House Price Index

The underlying asset of the HECM product is the mortgaged property value. However, the mortgaged property is infrequently traded in the market and we seldom have historical data on each individual mortgaged property, which leaves us some difficulties on the pricing issue. Fortunately, we can mitigate this problem by resorting to the house price indices that reflect changes in residential property in a particular geographical region, since what we really care about is house price returns. In this study, I choose the nationwide House Price Index (HPI) from 1975 to 2007. The HPI is published by the Office of Federal Housing Enterprise Oversight (OFHEO) on a quarterly basis, and measures average price changes on single-family residential properties. It is based on a modified version of the weighted, repeated sales methodology, which was first proposed by Bailey, Muth and Nourse (1963), and later extended by Case and Shiller (1987, 1989). Repeated transactions of single-family houses are reviewed so that for each property, at least two mortgages were originated and subsequently purchased by Freddie Mac or Fannie Mae since January 1975. The use of repeated sales on the same property helps to control for differences in the quality of the houses in the sample. For this reason, the HPI is a "constant quality" house price index. As of December 1995, there were over 6.9 million repeated transactions in the national sample. Because of the breadth of the sample, it provides more information than is available in other house price indices. For more questions and comparison with other house price indices, I refer interested readers to the new released 2007 Q4 House Price Index Report.[30]

[30] Office of Federal Housing Enterprise Oversight (OFHEO). 2008. 4Q 2007 House Price Index Report, available at http://www.ofheo.gov/media/pdf/4q07hpi.pdf

4.5.2. Housing Price Dynamics Review

In recent years, numerous studies model the dynamics of house prices. The main analytical issues examined by these studies include testing housing market efficiency, pricing contingent claims embedded in mortgages and mortgage-backed securities, and establishing a hedging mechanism for house price volatility (Cho, 1996). The consensus in the literature is that the house value is arguably not a stationary series. Variation of individual property values around the average increases over time.

Cunningham and Hendershott (1984), Kau, Keenan and Muller (1993) model property values as a geometric Brownian motion. The HUD also adopts this model framework in its HECM program, and assumes that property values appreciate with a 4 percent mean and a 10 percent standard deviation (Quercia, 1997). Under this model setup, nonstationarity arises from the fact that the cumulative house price appreciation rate over time is normally distributed, with mean and standard deviation growing over time. In addition, the house price appreciation is a random walk and has no memory, which means that previous values do not help in the prediction of future values.

However, tests of efficient market hypothesis (EMH) in the real estate markets provide us with contradictory results. The history of the housing literature on this topic goes back only about 20 years to the studies by Hamilton and Schwab (1985) and Linneman (1986). Since then, numerous studies have tested the EMH in the housing markets in the United States and other countries. Case and Shiller (1989) reject the weak-form efficiency in the U.S. housing market by pointing out the positive autocorrelation effects in both the changes in house prices and after-tax excess returns. Hosio and Pesando (1991), Ito and Hirono (1993) investigate the Toronto and Tokyo housing markets, respectively, and get similar results. The Institute of Actuaries (2005)

also finds that there are positive autocorrelations in the Nationwide House Price Index in the U.K. Autocorrelations in the house price index actually give the price series some memory and allows speculative price bubbles, as well as mean reversion, to occur (Szymanoski, 1994).

Therefore, it is natural to apply time-series analysis to the real estate market. Particularly, the development of ARCH (autoregressive conditional heteroskedasticity) models relaxes the assumption of constant error variance, which can nicely explain the increasing volatility of house price dynamics. ARCH models were introduced by Engle (1982) and generalized as GARCH (Generalized ARCH) by Bollerslev (1986). These models are widely used in various branches of econometrics, especially in financial time series analysis. Chinloy, Cho and Megbolugbe (1997), Nothaft, Gao and Wang (1995) apply GARCH models in analyzing house price volatilities.

4.5.3. ARMA(R,M) – GARCH(P,Q)

To developing a GARCH model, we have to consider two specifications—one for the conditional mean and one for the conditional variance.

The conditional mean model $ARMA(R,M)$ can be expressed as

$$Y_t = c + \sum_{i=1}^{R} \phi_i X_{t-i} + \sum_{j=1}^{M} \theta_j \varepsilon_{t-j} + \varepsilon_t ,^{31} \tag{4.8}$$

where R is the order of the autocorrelation terms, M the order of the moving average terms, ϕ_i the ith-order autocorrelation coefficient, θ_j the jth-order moving average coefficient, and ε_t the Gaussian innovations.

Let Φ_{t-1} be the information set containing all the information up to time $t-1$. Denote σ_t^2

[31] The eigen values $\{\lambda_i\}$ associated with the characteristic AR polynomial $\lambda^R - \phi_1\lambda^{R-1} - \phi_2\lambda^{R-2} - \ldots - \phi_R$ must lie inside the unit circle to ensure stationarity. Similarly, the eigen values associated with the characteristic MA polynomial $\lambda^M + \theta_1\lambda^{M-1} + \theta_2\lambda^{M-2} + \ldots + \theta_M$ must lie inside the unit circle to ensure invertibility.

the conditional variance of the innovations given Φ_{t-1}, i.e., $\sigma_t^2 = E[\varepsilon_t^2 \mid \Phi_{t-1}]$. The conditional variance model $GARCH(P,Q)$ for the innovations can be written as

$$\sigma_t^2 = d + \sum_{i=1}^{P} \alpha_i \sigma_{t-i}^2 + \sum_{j=1}^{Q} \beta_j \varepsilon_{t-j}^2 \text{ ,}^{32} \tag{4.9}$$

where P is the order of the GARCH terms, Q the order of the ARCH term, α_i the ith-order GARCH coefficient, and β_j the jth-order ARCH coefficient.

Let X_t be the time series of the quarterly HPI from 1975 Q1 to 2007 Q4. I transform the data into the log-arithmetical return series, $Y_t = \ln(X_t) - \ln(X_{t-1})$, in order to analyze the house price returns. From Figure 4.2, the log return series is obviously not stationary. I implement the Augmented Dickey-Fuller (ADF) test, as well as the Phillips-Perron (PP) test. Both the ADF statistic (-0.1975) and PP statistic (4.1293) are higher than the critical values at the significance level of 5%, which means that the tests fail to reject the null hypothesis of a unit root in the return series. Therefore, we need to difference the return series to account for the order of integration. The return series becomes stationary after taking the first difference (see Figure 4.3), and the unit root tests confirm this result (ADF=-10.36413, PP=-18.64109).

[32] We require $k > 0$, $\alpha_i \geq 0$ for $i = 1,2,...,P$, $\beta_i \geq 0$ for $j = 1,2,...,Q$, $\sum_{i=1}^{P} \alpha_i + \sum_{j=1}^{Q} \beta_j < 1$.

Figure 4.2: The HPI Log Returns

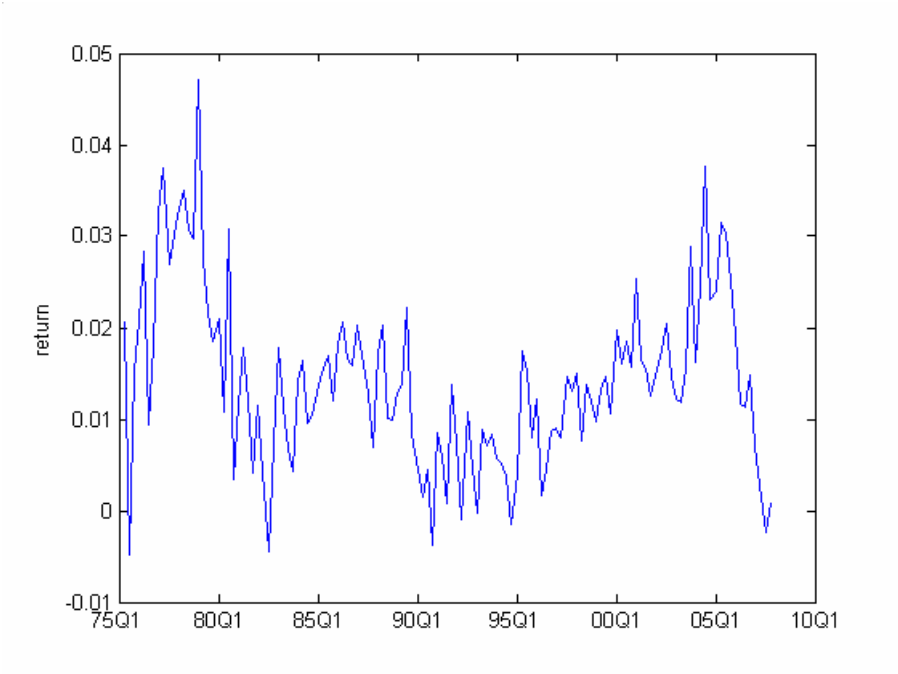
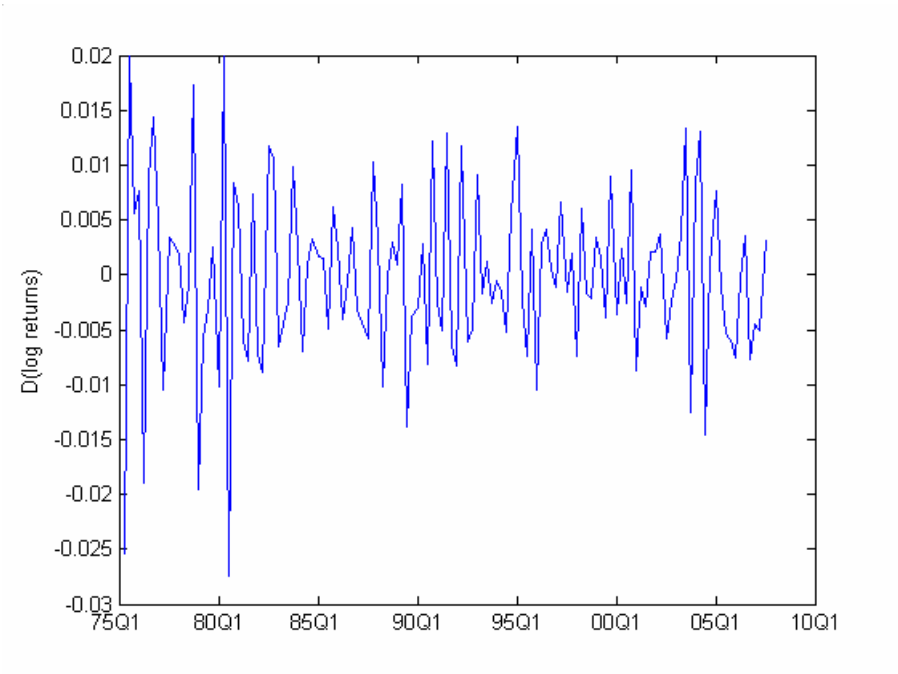


Figure 4.3: The First Difference of HPI Log Returns



The Box-Jenkins (1976) approach gives us a guide on how to identify an appropriate ARMA(R,M) model for the first difference of log returns (denoted as DY_t thereafter). I plot the

sample autocorrelation coefficients (ACF) and partial autocorrelation coefficients (PACF) of DY_t in Figure 4.4 and 4.5, respectively. The sample ACFs are almost zero after one lag, and the sample PACFs die off after two lags. It seems that ARMA (2,1) would be appropriate for the DY_t series. However, after fitting DY_t with ARMA(2,1), I find that the constant term, AR(1) term, and MA(1) term are statistically insignificant at the 5% significance level. Therefore, more trials with other combinations of R and M are needed. The Akaike (AIC) and Bayesian (BIC) information criterion can be used as a guide for the appropriate lag order selection. After several trials, I find that ARMA (2,0) without the constant term makes a good fit. The coefficients of AR(1) and AR(2) terms are both highly significant. In addition, the AIC and BIC reach the lowest values (see Table 4.1).

Figure 4.4: ACFs of the First Difference of HPI Log Returns

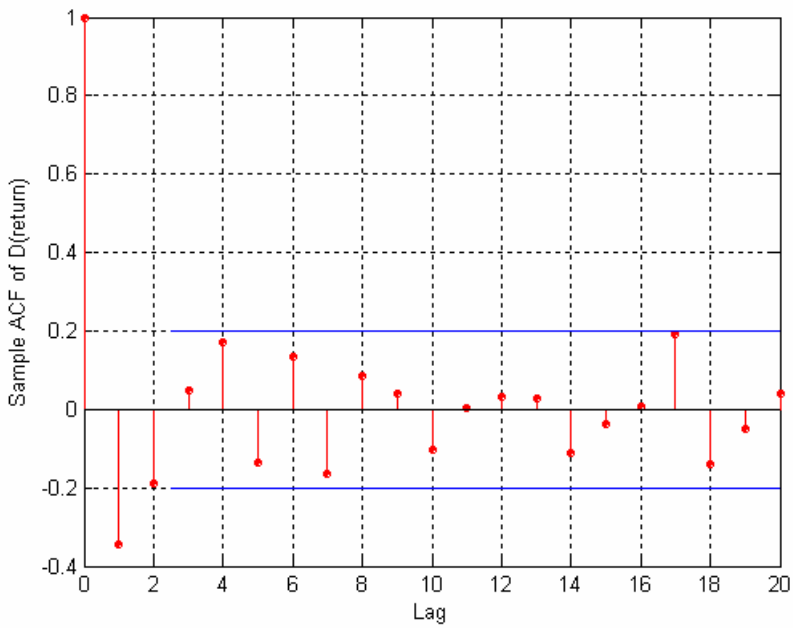


Figure 4.5: PACFs of the First Difference of HPI Log Returns

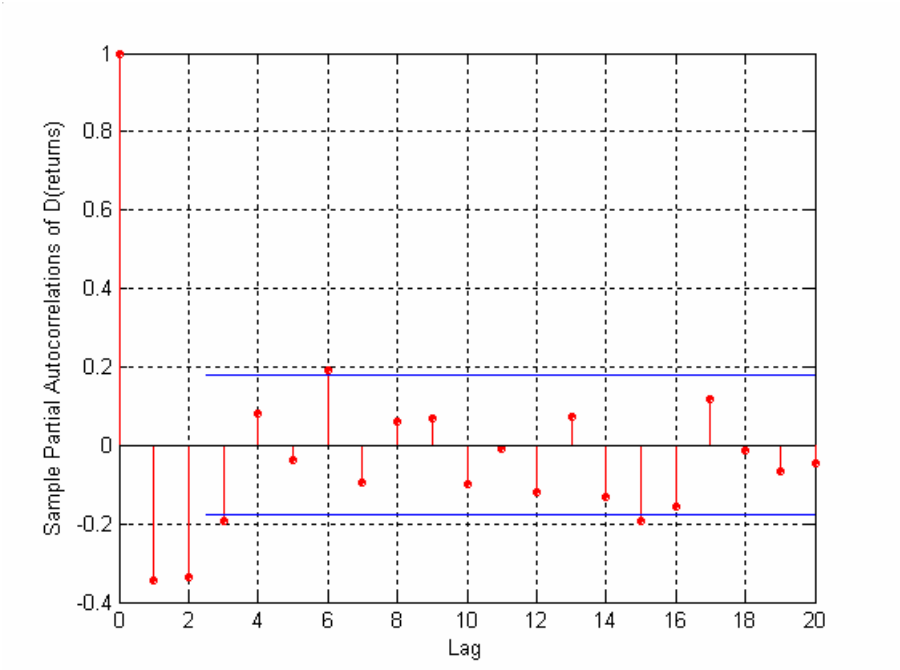
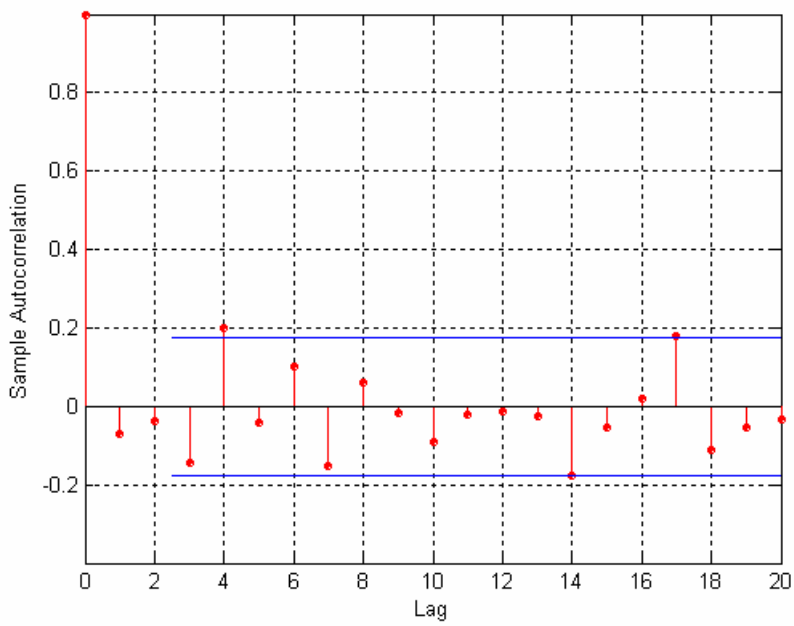


Table 4.1: Parameter Estimates of Several ARMA Models

Parameter	Value	Standard Error	T-statistic
ARMA(2,0) without constant term			
AR(1)	0.46137	-0.068178	-6.7672
AR(2)	-0.34567	0.074713	-4.6267
AIC	-911.8226	BIC	-903.2200
ARMA (2,0)			
C	-0.00028119	0.00063945	-0.4397
AR(1)	-0.46248	0.069209	-6.6824
AR(2)	-0.34666	0.074457	-4.6559
AIC	-910.0270	BIC	-898.5569
ARMA(2,1)			
C	-0.0001719	0.00043235	-0.3976
AR(1)	-0.16342	0.17813	-0.9174
AR(2)	-0.25641	0.10365	-2.4739
MA(1)	-0.35463	0.18916	-1.8747
AIC	-911.7503	BIC	-897.4126

After fitting a candidate ARMA specification, we should verify that there are no remaining autocorrelations that the model has not accounted for. Figure 4.6 plots the autocorrelations of innovations in the selected model. Almost all the ACFs are within the 95% confidence interval, which indicates that ARMA(2,0) without the constant term captures most of the autocorrelation effects in the DY_t series. I further verify this by implementing the Ljung-Box-Pierce Q-Test (see the upper panel of Table 4.2). When examined for up to 1, 5, and 10 lags of the ACFs at the 5% significance level, no significant correlation is present after fitting the DY_t series by ARMA(2,0) without a constant,

Figure 4.6: ACFs of Innovations of ARMA(2,0) without the Constant Term



I also plot the ACFs of the squared innovations in Figure 4.7, in order to examine the existence of ARCH effects. It demonstrates that, although the ACFs of the innovations exhibit little correlation, the ACFs of the squared innovations are still significantly correlated. The Ljung-Box-Pierce Q-Test on the squared residuals (the middle panel of Table 4.2) and Engle's ARCH test on the residuals (the lower panel of Table 4.2) show significant evidence in support

of ARCH effects.

Figure 4.7: ACFs of Squared Innovations of ARMA(2,0) without the Constant Term

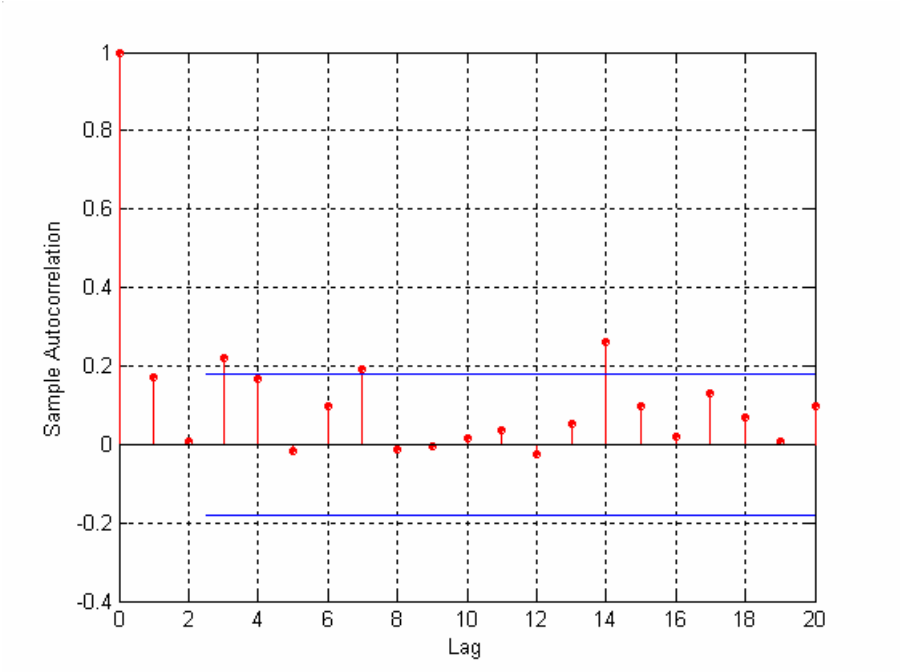


Table 4.2: Q-Test and ARCH Test for the ARMA(2,0) Model without the Constant Term

	Lag	Statistic	Critical Value	P-Value
Ljung-Box-Pierce Q-Test for the Innovations	1	0.6773	3.8415	0.4105
	5	9.2737	11.0705	0.0986
	10	15.6112	18.3070	0.1113
Ljung-Box-Pierce Q-Test for the Squared Innovations	1	3.9002	3.8415	0.0483
	5	14.1623	11.0705	0.0146
	10	20.5822	18.3070	0.0242
Engle's ARCH Test for the Innovations	1	5.9467	3.8415	0.0147
	5	10.1580	11.0705	0.0709
	10	11.2282	18.3070	0.3400

In most applications, GARCH(1,1) is enough to capture the ARCH effects. I keep ARMA(2,0) without the constant term for the conditional mean, and use GARCH(1,1) to model

the conditional variance. Parameter estimates and corresponding statistics are reported in Table 4.3. All the coefficients are highly significant except for d, the constant term in the GARCH model. Figure 4.8 shows that the standardized innovations (the innovations divided by the conditional standard deviations) become uncorrelated. In addition, almost all the ACFs of the squared standardized innovations fall in the 95 percent confidence interval (see Figure 4.9), which indicates that my model sufficiently explains the ARCH effects. The results of hypothesis testing in Table 4.4 confirm the qualitative checks above. The Q-test on the standardized innovations fails to reject the null hypothesis that there is no autocorrelation up to 10 lags. The Q-test on the squared standardized innovations and the ARCH test on the standardized innovations come to the same conclusion that conditional heteroskedasticity has disappeared after the model fitting.

Table 4.3: Parameter Estimates for the ARMA(2,0) – GARCH(1,1) model

Parameter	Value	Standard Error	T-statistic
AR(1)	-0.42727	0.083495	-5.1174
AR(2)	-0.34979	0.086188	-4.0584
d	4.0059e-006	3.3928e-006	1.1807
GARCH(1)	0.79897	0.12364	6.4620
ARCH(1)	0.10319	0.069692	1.4806

Figure 4.8: ACFs of Standardized Innovations of ARMA(2,0) + GARCH(1,1)

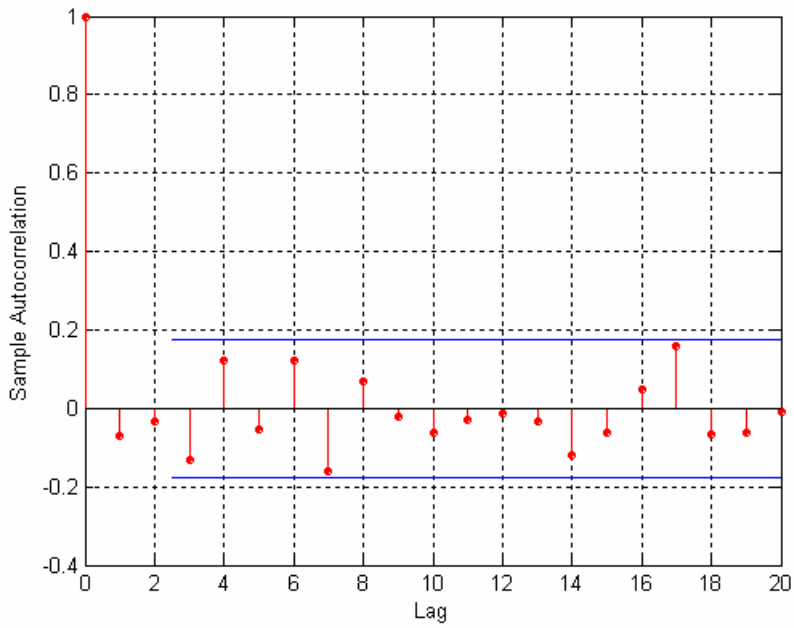


Figure 4.9: ACFs of Squared Standardized Innovations of ARMA(2,0) + GARCH(1,1)

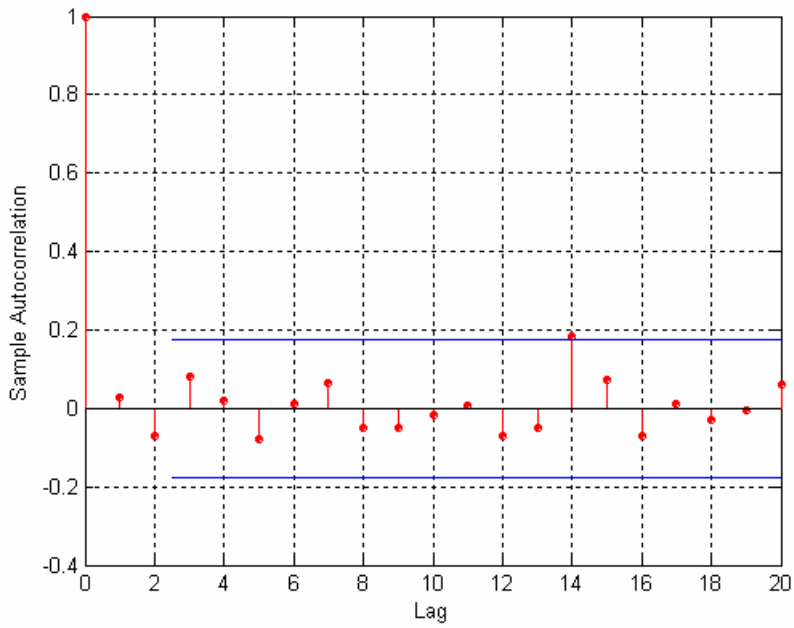


Table 4.4: Q-Test and ARCH Test for the ARMA(2,0) – GARCH(1,1) Model

	Lag	Statistic	Critical Value	P-Value
Ljung-Box-Pierce Q-Test for the Standardized Innovations	1	0.6668	3.8415	0.4142
	5	5.5319	11.0705	0.3545
	10	12.4285	18.3070	0.2574
Ljung-Box-Pierce Q-Test for the Squared Standardized Innovations	1	0.1049	3.8415	0.7461
	5	2.4678	11.0705	0.7813
	10	3.7699	18.3070	0.9571
Engle's ARCH Test for the Standardized Innovations	1	0.1639	3.8415	0.6856
	5	2.2089	11.0705	0.8196
	10	2.0212	18.3070	0.9962

4.6. The Pricing Framework

4.6.1. From Exponential Tilting to Conditional Esscher Transform

Recall that in chapter 3, I discussed the exponential tilting of X with respect to the reference variable Y, which is defined as

$$f_X^*(x) = f_X(x)\frac{E[\exp(\lambda Y) \mid X = x]}{E[\exp(\lambda Y)]}, \tag{4.10}$$

where f_X and f_X^* represent the probability density function of X before and after the exponential tilting, respectively.

We leave much flexibility in the choice of reference variable Y. Particularly, if we choose the reference Y to be the risk X itself, the exponential tilting is reduced to the famous "Esscher transform" formula:

$$f_X^*(x) = f_X(x)\frac{E[\exp(\lambda X) \mid X = x]}{E[\exp(\lambda X)]} = f_X(x)\frac{\exp(\lambda x)}{E[\exp(\lambda X)]}. \tag{4.11}$$

The Esscher transform was introduced by Esscher (1932) and has become a time-honored

tool in actuarial science. More recently, it has been applied to pricing financial and insurance securities in an incomplete market. Creating an equivalent martingale measure by the Esscher transform is justified by maximizing the expected power utility of an economic agent (Gerber and Shiu, 1994). See Pafumi (1997), Shiryaev (1999), Yao (2001), McLeish and Reesor (2003), and Yang (2004) for a rigorous theoretical background and more discussions.

Buhlmann, Delbaen, Embrechts and Shiryaev (1996) generalize the Esscher transform to stochastic processes and introduce the concept of the conditional Esscher transform. In terms of probability density functions, the conditional Esscher transform is defined as

$$f^*_{X_t}(x \mid \Phi_{t-1}) = f_{X_t}(x \mid \Phi_{t-1}) \frac{\exp(\lambda_t x)}{E\left[\exp(\lambda_t X_t) \mid \Phi_{t-1}\right]}. \tag{4.12}$$

The pricing results under the conditional Esscher transform can be justified within the dynamic framework of utility maximization problems. Siu, Tong and Yang (2004) employ the conditional Esscher transform to price derivatives, assuming the underlying asset returns follow GARCH processes. Li, Boyle, Hardy and Tan (2007) follow the same line to price the No-Negative-Equity-Guarantee in the U.K. equity release market, which is similar to the non-recourse provision of reverse mortgage programs in the U.S. I adapt their pricing framework when the underlying asset return follows an ARIMA-GARCH process.

4.6.2. Change of Measure Based on the Conditional Esscher Transform

Let (Ω, Φ, P) be a complete probability space, where P is the data-generating probability measure. Define two filtrations $F = (F_t)_{t \in \{0,1,,,,T\}}$ and $G = (G_t)_{t \in \{0,1,,,,T\}}$ on this probability space. The first filtration F is related to the development on the house price index, and F_t can be interpreted as the information obtained from observing the index up to time t. The second

filtration G contains the information about the borrower of the revere mortgage, and G_t can be interpreted as the morality experience up to time t. In addition, the third filtration is defined as $\Phi = (\Phi_t)_{t\in\{0,1,,,,T\}}$, where $\Phi_t = F_t \vee G_t$. It means that Φ_t is the smallest σ-algebra that includes both F_t and G_t. This has the usual interpretation that, at time t, we have access to the combined information concerning both the development of the house price index and the mortality experience up to time t. Hence, Φ includes all the available information.

Let $T(x)$ denote the termination time of a reverse mortgage initiated to an individual aged x. For simplicity, assume $T(x)$ is stochastically independent of the housing price index, H_t, under the physical probability measure P. On the one hand, because the IPL is predetermined at the time of loan origination, the scheduled cash advances to borrowers are not reduced when property values fall. Smart borrowers will take advantage of this feature of reverse mortgages and won't have the incentive of refinancing during a recession. On the other hand, when property values rise, although borrowers have the incentive of refinancing, high release costs on the old mortgage and high closing costs on a new mortgage severely reduce the cash advances available under a new mortgage, which dampens borrowers' incentive of refinancing. See Lin and Tan (2003), Rodda, Lam and Youn (2004) for more discussions.

In section 4.5, I fit the first difference of house price returns, DY_t, using an ARMA(2,0)-GARCH(1,1) process, i.e.,

$$DY_t = \phi_1 DY_{t-1} + \phi_2 DY_{t-2} + \varepsilon_t ,$$

where $\varepsilon_t \mid \Phi_{t-1} \sim N(0, \sigma_t^2)$ and $\sigma_t^2 = d + \alpha_1 \sigma_{t-1}^2 + \beta_1 \varepsilon_{t-1}^2$.

Let $\mu_t = \phi_1 DY_{t-1} + \phi_2 DY_{t-2}$, then DY_t is normally distributed with mean μ_t and variance σ_t^2, given the information set Φ_{t-1}. That is, $DY_t \mid \Phi_{t-1} \sim N(\mu_t, \sigma_t^2)$. Note that μ_t and σ_t are not

random given the information Φ_{t-1}. Consequently, the house price return series $Y_t \mid \Phi_{t-1} \sim N(\hat{\mu}_t, \sigma_t^2)$ under the physical measure P, where $\hat{\mu}_t = \mu_t + Y_{t-1}$.

Define a sequence $\{\Lambda_t\}_{t\in\{0,1,...,T\}}$ with $\Lambda_0 = 1$, and for $t \geq 1$,

$$\Lambda_t = \prod_{k=1}^{t} \frac{\exp(\lambda_k Y_k)}{E[\exp(\lambda_k Y_k) \mid \Phi_{k-1}]}, \tag{4.13}$$

for some constants $\lambda_1, \lambda_2, ..., \lambda_t$. Buhlmann, Delbaen, Embrechts and Shiryaev (1996) prove that $\{\Lambda_t\}_{t\in\{0,1,...,T\}}$ is a martingale.

Let P_t be the restriction of the measure P on the information Φ_t, where $P_T = P$. The fact $\{\Lambda_t\}_{t\in\{0,1,...,T\}}$ is a martingale allows us to construct a family of measures $\{Q_t\}_{t\in\{1,2,...,T\}}$ such that $dQ_t = \Lambda_t dP_t$, $Q_t = Q_{t+1} \mid \Phi_t$, and a probability measure $Q = Q_T$ on the sample space (Ω, Φ). See Siu, Tong and Yang (2004), Li, Boyle, Hardy and Tan (2007) for more details.

In order for Q to be a risk-neutral probability measure which is equivalent to the physical measure P on the same sample space (Ω, Φ), I choose a sequence of parameters $\lambda_t^q = \frac{r - \mu_t}{\sigma_t^2} - \frac{1}{2}, (t = 1,2,...,T)$, such that the following set of equations are satisfied:

$$E_{Q_t}[\exp(Y_t); \lambda_t^q \mid \Phi_{t-1}] = \exp(r), \tag{4.14}$$

where r is the risk-free interest rate.[33]

Under the risk-adjusted measure Q, we have the following properties:

- $Y_t \mid \Phi_{t-1} \sim N\left(r - \frac{1}{2}\sigma_t^2, \sigma_t^2\right)$;
- $\Pr^Q(T(x) > t) = \Pr^P(T(x) > t) = {}_t p_x$;

[33] I refer interested readers to Buhlmann et al. (1996) for a detailed proof.

- $T(x)$ is stochastically independent of H_t under Q;

The first property states exactly that the dynamics of Y_t under the risk-adjusted measure is the same as that under the physical measure, except that the mean is shifted by an amount of $-\hat{\mu}_t + r - \frac{1}{2}\sigma_t^2$ (see Appendix 4B for proof). The second property indicates the change of measure from P to Q does not affect the marginal distribution of the remaining lifetime of the reverse mortgage. Finally, the last property states that the independence between the termination time of loans and a priori given real estate market is preserved under Q. [34]

4.6.3. Numerical Illustrations

Recall the value of the non-recourse provision can be expressed as

$$\text{Value} = \sum_{t=0}^{\omega-x-1} E_Q\left[{}_t p_x q_{x+t} V_t\right], \tag{4.15}$$

where $V_t = e^{-rt}\max(L_t - H_t, 0)$ is the discounted payoff of an European exchange option matured at time t, exchanging H_t for L_t.

Suppose all home exits occur in the mid-year and let δ denote the average delay in time from the point of home exit until the actual sale of the property. Therefore, the time to maturity of the European exchange option is $t + 0.5 + \delta$. The value of the non-recourse provision becomes

$$\text{Value} = \sum_{t=0}^{\omega-x-1} E_Q\left[{}_t p_x q_{x+t} V_{t+0.5+\delta}\right]. \tag{4.16}$$

When the borrower dies in the future, the lender needs to sell the property and pay the transaction cost. Suppose the transaction cost is κ percent of the property value, then the

[34] Property 2 and 3 arise from the fact that the Radon-Nikodym derivative, $\Lambda_t = dQ_t / dP_t$, used to obtain the probability measure Q is a function of Y_t, and hence is independent of $T(x)$.

discounted payoff of the European exchange option is

$$V_{t+0.5+\delta} = e^{-r(t+0.5+\delta)} \max(L_{t+0.5+\delta} - (1-\kappa)H_{t+0.5+\delta}, 0). \tag{4.17}$$

Further considering the rental yields from the property as dividends, we can adjust the option formula to accommodate rental yields. Assuming g is the rental yield per year, the discounted payoff of the European exchange option becomes

$$V_{t+0.5+\delta} = e^{-r(t+0.5+\delta)} \max\left(L_{t+0.5+\delta} - (1-\kappa)e^{-g(t+0.5+\delta)}H_{t+0.5+\delta}, 0\right). \tag{4.18}$$

At maturity, the house value is

$$H_{t+0.5+\delta} = H_0 e^{\sum_{i=1}^{4(t+0.5+\delta)} Y_i}, \tag{4.19}$$

and the loan balance is

$$L_{t+0.5+\delta} = \left(0.02H_0 + L_0\right)e^{u(t+0.5+\delta)}, \tag{4.20}$$

where u is the interest rate charged on the mortgage loan.[35]

Substituting (4.19) and (4.20) into equation (4.18), I obtain:

$$V_{t+0.5+\delta} = e^{-r(t+0.5+\delta)} \max\left(\left(0.02H_0 + L_0\right)e^{u(t+0.5+\delta)} - (1-\kappa)H_0 e^{-g(t+0.5+\delta)+\sum_{i=1}^{4(t+0.5+\delta)} Y_i}, 0\right). \tag{4.21}$$

The key assumptions that I make for the numerical illustration are as follows.

- The average delay in time from the point of home exit until the actual sale of the property is six months, i.e., $\delta = 0.5$.
- The transaction cost of selling the house is 6 percent of the property value, i.e., $\kappa = 6\%$.
- In the HECM program, the risk-free interest rate is usually the 10 year U.S. Treasury rate. It is 3.91% per annum at 01/02/2008, which is equivalent to an interest rate of 3.84%, compounded continuously, i.e., $r = 3.84\%$.

[35] In the HECM program, the loan balance increases with interest accruals and insurance premiums.

- The interest rate charged on the loan is 4.71% per annum, which is equal to the one year constant maturity Treasury (CMT) rate of 2.71% plus a lender's spread margin of 150 bps and an additional HUD mortgage insurance premium of 50 bps. It is equivalent to 4.60% on a continuously compounding basis, i.e., $u = 4.6\%$.
- The rental yield is 2% per annum, compounded continuously, i.e., $g = 2\%$.
- The initial house value is assumed to be $300,000, i.e., $H_0 = \$300{,}000$.
- I consider the lump sum payment option. Therefore, the loan amount L_0 is equal to the initial principle limit (IPL). IPL can be obtained using the online reverse mortgage calculator on Financial Freedom's website, assuming the property is located in Philadelphia, Zip code 19104.[36]

I first simulate the DY_t series for 1000 paths and transform DY_t to Y_t on each path. Applying the pricing framework discussed above, I change the probability measure from P to Q on each path of Y_t, and then calculate the value of the non-recourse provision according to equation (4.16) and (4.21).

I also calculate the total insurance premiums collected by FHA for the purpose of comparison. Recall the insurance premiums consist of two parts, both paid by the borrowers: an up-front charge of 2% of the adjusted property value[37], and an annual rate of 0.5% of the outstanding loan balance for the life of the loan. Mathematically, it can be expressed as

$$\text{Premium} = 0.02H_0 + \sum_{t=1}^{\omega-x-1} {}_t p_x e^{-rt}\left(0.005 \times (0.02H_0 + L_0)e^{ut}\right). \tag{4.23}$$

[36] Available at http://www.financialfreedom.com/calculator/Input_new.asp

[37] The adjusted property value is the lesser of the appraised property value and the maximum loan limit. The maximum loan limit is placed by FHA depending on the geographical area of the borrower's house, taking into account the value of the house. For simplicity, we assume here the property value is always less than the maximum loan limit.

The simulation results are shown in the panel of Scenario 1 in Table 4.6. Several observations here:

- Initial loan amount increases as the age at loan origination increases. This is reasonable because other conditions equal, the risk of reverse mortgages ultimately depends on when the maturity event occurs and for how long the loan has been accruing. The elder borrower has a shorter life expectancy, therefore the lender faces less longevity risk and can advance more cash amounts to the borrower.
- For the same reason, the value of the non-recourse provision decreases dramatically as the age at loan origination goes up. When the initial age is 62, the non-recourse provision is worth $8,219. This figure drops to $915 for a borrower at age 90.
- The insurance premiums also decrease with the age at loan origination. We can see that when the initial age increases, the borrower gets more cash advances, pays less insurance premiums, and can spend more money to improve the living standard of his or her life.
- Theoretically, the value of the non-recourse provision should equal the insurance premiums the borrower pays to FHA in order to limit his/her liability to the property value. However, the present value of insurance premiums calculated based on current premium structure is far more than the actuarial present value of benefits. I report the ratio of the present value of insurance premiums to the value of non-recourse provision in Table 4.6. The ratio is 2.90 when the initial age is 62. It increases at an accelerating rate with the initial age of the borrower. When the borrower is 90 years old at the time of closing, the present value of insurance premiums collected by the FHA reaches 13.42 times the value of the non-recourse provision.

The U.S housing market has been experiencing the biggest slump recently. According to OFHEO Director James B. Lockhart, "The year 2007 showed the first four-quarter decline in the purchase-only index since its earliest data in 1991." Prices fell between the third and fourth quarters of 2007 in every state except Maine. When compared with the data in the fourth quarter of 2006, house prices in the fourth quarter of 2007 depreciated by 6.6% in California, 5.9% in Nevada, and 4.7% in Florida (OFHEO, 2008).[38] It is interesting to ask what would happen if the value of the property had a sudden decrease at time of loan origination.

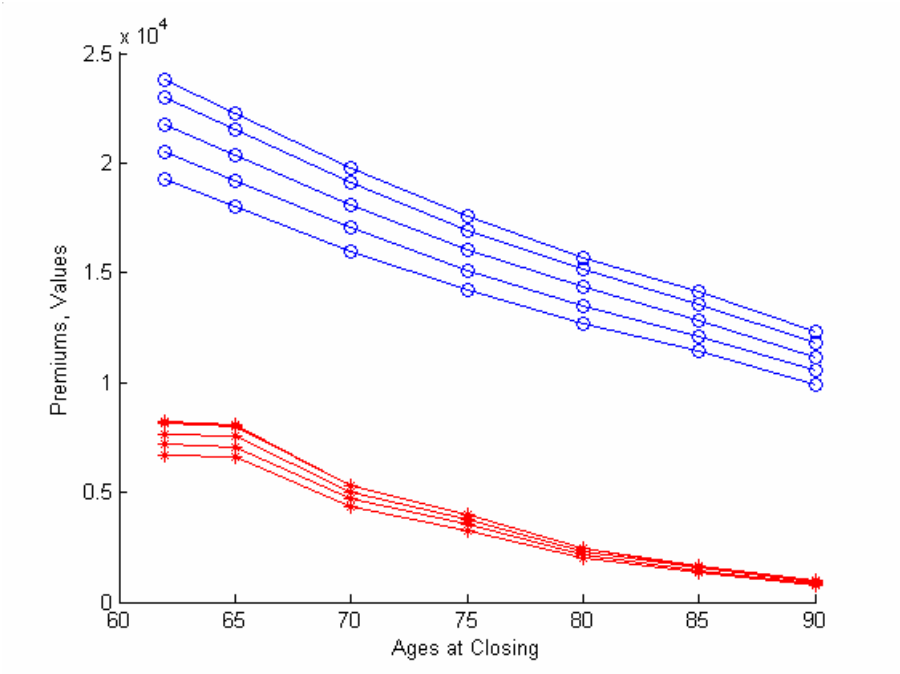
I analyze the effects of initial property values on the values of non-recourse provision and insurance premiums under different scenarios, assuming that the house price depreciates by 5%, 10%, 15% and 20%, respectively, at the time of closing. I predict that the present value of insurance premiums should decrease with the initial property value. That is because the house price depreciation at time of closing brings down the initial loan amount the borrower can obtain. The present value of insurance premiums decreases consequently, as the premium in each period is proportional to the outstanding loan balance. I predict the value of non-recourse provision should decrease with the initial property value, too, since it is simply the counterpart of insurance premiums and should behave similarly in response to the house price depreciation. Figure 4.10 confirms my predictions. However, it is noteworthy that the house price depreciation has little effect on the ratios of insurance premiums to the values of non-recourse provision (see Figure 4.11).

[38] OFHEO. 2008. 4Q 2007 House Price Index Report, available at http://www.ofheo.gov/media/pdf/4q07hpi.pdf

Table 4.5: Values of the Non-recourse Provision and Insurance Premiums in Different Scenarios

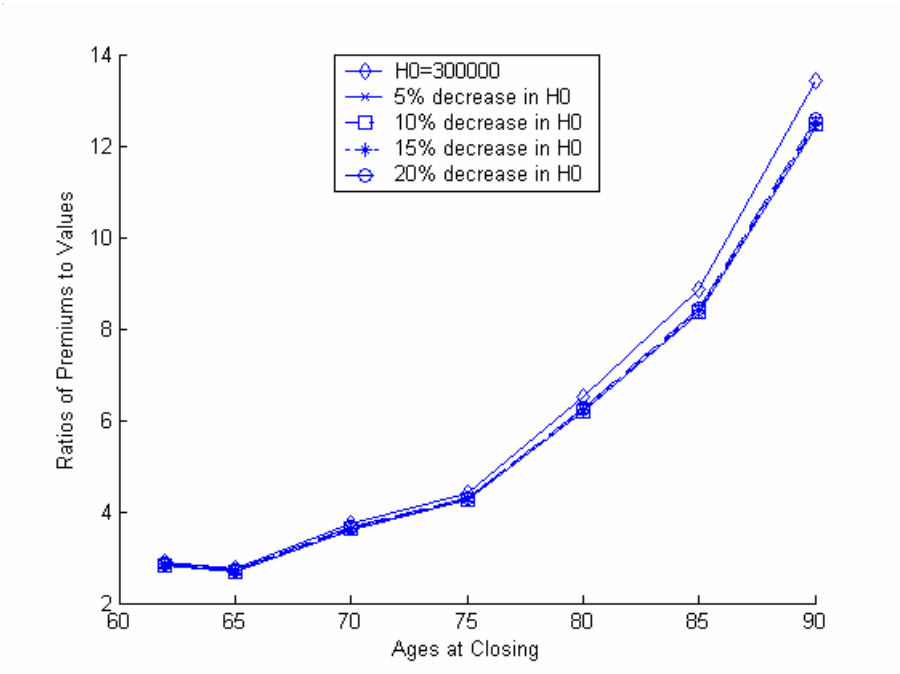
Age at Origination	62	65	70	75	80	85	90
Scenario 1: Initial Property Value H_0 = $300,000							
L_0	161,293	168,470	180,498	193,513	206,964	220,316	233,047
Value	8,219	8,071	5,319	3,967	2,414	1,592	915
Premium	23,811	22,286	19,796	17,550	15,713	14,110	12,282
Premium/Value	2.90	2.76	3.72	4.42	6.51	8.87	13.42
Scenario 2: Initial Property Value Decreases by 5%. H_0 = $285,000							
L_0	156,835	163,828	175,549	188,233	201,347	214,368	226,791
Value	8,125	7,990	5,292	3,973	2,448	1,631	952
Premium	23,004	21,525	19,107	16,927	15,144	13,586	11,809
Premium/Value	2.83	2.69	3.61	4.26	6.19	8.33	12.40
Scenario 3: Initial Property Value Decreases by 10%. H_0 = $270,000							
L_0	148,135	154,768	165,889	177,928	190,382	202,758	214,581
Value	7,658	7,532	4,989	3,745	2,307	1,537	898
Premium	21,746	20,351	18,071	16,013	14,330	12,859	11,181
Premium/Value	2.84	2.70	3.62	4.28	6.21	8.37	12.46
Scenario 4: Initial Property Value Decreases by 15%. H_0 = $255,000							
L_0	139,435	145,708	156,229	167,628	179,417	191,148	202,371
Value	7,191	7,074	4,685	3,518	2,166	1,444	843
Premium	20,488	19,177	17,034	15,100	13,516	12,133	10,552
Premium/Value	2.85	2.71	3.64	4.29	6.24	8.41	12.52
Scenario 5: Initial Property Value Decreases by 20%. H_0 = $240,000							
L_0	130,735	136,648	146,569	157,318	168,452	179,538	190,161
Value	6,724	6,616	4,382	3,289	2,025	1,350	789
Premium	19,230	18,004	15,997	14,185	12,702	11,406	9,923
Premium/Value	2.86	2.72	3.65	4.31	6.27	8.45	12.58

Figure 4.10: Values of the Non-recourse Provision and Insurance Premiums in Different Scenarios



Note: I graph 5 scenarios with the initial property value equal to $300,000, 285,000, 270,000, 255,000 and 240,000, respectively. The -o- line denotes present values of insurance premiums at different ages. It decreases with the initial property value. The -*- lines denotes the values of the non-recourse provision at different ages. It decreases with the initial property value, too.

Figure 4.11: Ratios of Insurance Premiums to Values of the Non-recourse Provision in Different Scenarios



In the above analysis, I assume the lender's margin is 150 bps. Here comes the next question: what is the actuarially fair risk premium that the lender should assess? In other words, what is the lender's margin that makes the present value of insurance premiums equal to the value of the non-recourse provision? Obviously, it depends on the initial age of the borrowers. I use Broyden's method to find out the actuarially fair risk premium and report it in Table 4.6. The finding is that the lender can achieve a much higher margin based on the current HECM insurance premium structure. The margin increases from 442 bps to 885 bps when the borrower's initial age changes from 62 to 90. It is explained by the fact that FHA charges more insurance premiums than the actuarial present value of claim payments. The intrinsic risk implied by high insurance premiums is correspondingly high. As a result, the lender can charge a higher risk margin on the loan balance.

Table 4.6: The Lender's Margin When H_0 = $300,000

Age at Origination	62	65	70	75	80	85	90
Lender's Margin (bps)	442	437	513	560	646	768	885

Note: Assume the initial property value equals $300,000, located in Philadelphia, Zip code 19104.

4.7. Conclusions and Discussions

The market for reverse mortgage products has started growing rapidly as an effective solution to the "Home Rich and Cash Poor" dilemma. As pointed out in the Deutsche Bank Report (2007), "A larger number of originators, increased interest in securitization, and the aging population will lead to a more competitive and efficient origination." The HECM program has favorable features and provides protection to both borrowers and lenders. On the one hand, borrowers benefit from the non-recourse provision which limits borrowers' liability to the mortgaged property value. On the other hand, lenders are protected from the possible losses by

the FHA insurance program. In this study, I model the various risks embedded in the HECM program and create an equivalent martingale measure to price the non-recourse provision. I further compare the value of the non-recourse provision with the present value of insurance premiums. I find that the premiums of HECM loans are adequate to cover expected claims. The FHA makes profits on the expected value basis.

The complexity of valuation problems comes from the fact that the HECM products are involved with multiple risks. I analyze longevity risk, mobility risk, and house price depreciation risk in this study. However, there are other risks we need to take into account, for example, basis risk, interest rate risk, and refinancing risk. First, the heart of pricing the non-recourse provision lies on modeling the dynamics of the underlying asset: the property value. I used the House Price Index to model house price returns, since each mortgaged property is infrequently traded in the market and does not have enough historical data available for us to analyze. Basis risk arises because the fluctuation of individual mortgaged property value may deviate from the HPI dynamics. Second, I assume a constant interest rate plus a lender's margin for the life of the HECM loans so that I can determine the conditional Esscher parameters and create the risk-adjusted probability measure for pricing purposes. However, we do not live in a simple world with a flat term structure subject only to additive shifts (Boehm and Ehrhardt, 1994). Therefore, we do need to model the stochastic interest rates with a more realistic term structure, for instance, the Vasicek model (Vasicek, 1977) or CIR model (Cox, Ingersoll and Ross, 1985). Finally, the termination time of a reverse mortgage should be determined by a multiple-decrement model, considering mortality risk, mobility risk and refinancing risk. Similar to the refinancing of forward mortgages, reverse mortgages become refinanceable when interest rates decline or house prices increase. I assumed that the refinancing rate is zero in this study. However, as the reverse

mortgage market expands and matures, we will see higher refinancing rates. Further research based on a multiple decrement termination model is warranted.

Appendix 2A: Numerical Approximation for the Optimal Switching Models

Optimal switching models generally require numerical approximations. We can approximate the value function by a linear combination of n known basis functions.That is,

$$V(x,r) \approx \sum_{j=1}^{n} \theta_j(r)\phi_j(x,r), \tag{2A.1}$$

where $\phi_j(x,r)$ defines the basis function and $\theta_j(r)$ is the corresponding coefficient in regime r $(j = 1,...,n)$. If we take the switching points as given, the values of $\theta_j(r)$ can be obtained by solving the Feynman-Kac equation:

$$\sum_{j=1}^{n} \theta_j(r)[\rho\phi_j(x_i,r) - \mu(x_i,r)\phi_j'(x_i,r) - \frac{1}{2}\sigma^2(x_i,r)\phi_j''(x_i,r)] = \pi(x_i,r), \quad i = 1,...,n \tag{2A.2}$$

at n collocation nodes x_i $(i = 1,...,n)$, along with the non-optimality side conditions.

The basis functions can be chosen such as monomial basis (i.e., $1, x, x^2, x^3,...$), Chebychev polynomial basis, linear spline basis, and cubic spline basis. And the optimality conditions can be used to solve the switching points by a root-finding algorithm.

In principle, the collocation equation may be solved using any nonlinear equation solution method. For example, one can write the collocation equation as a fix-point problem $\theta = \Phi^{-1}\upsilon(\theta)$ and employ function iteration, which uses the iterative update rule

$$\theta \leftarrow \Phi^{-1}\upsilon(\theta). \tag{2A.3}$$

Alternatively, one may write the collocation equation as a root finding problem $\Phi\theta - \upsilon(\theta) = 0$ and solve for θ using Newton's method, which employs the iterative update rule

$$\theta \leftarrow \theta - \left[\Phi - \upsilon'(\theta)\right]^{-1}\left[\Phi\theta - \upsilon(\theta)\right]. \tag{2A.4}$$

Here $\upsilon'(\theta)$ is the $n \times n$ Jacobian of the collocation function υ at θ.

Appendix 3A: The Log-likelihood Function for the Model with Permanent Jump Effects

If I assume that the jump events have permanent effects on mortality modeling, the dynamics of k_t can be expressed as

$$dk_t = \begin{cases} (\mu - pm)dt + \sigma dW_t \\ \quad \text{if the jump event does not occur at time t, i.e. } N_{[t,t+dt]} = 0 \\ (\mu - pm)dt + \sigma dW_t + Y_{[t,t+dt]} \\ \quad \text{if the jump event occurs at time t, i.e., } N_{[t,t+dt]} = 1 \end{cases} \tag{3A.1}$$

It can be simplified to

$$dk_t = (\mu - pm)dt + \sigma dW_t + Y_{[t,t+dt]} N_{[t,t+dt]}. \tag{3A.2}$$

By integrating both sides from t to $t+h$, we can get

$$k_{t+h} = k_t + (\mu - pm)h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]} N_{[t,t+h]}. \tag{3A.3}$$

Let $z_t = k_{t+h} - k_t = (\mu - pm)h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]} N_{[t,t+h]}$. (3A.4)

If $N_{[t,t+h]} = 0$ the variable $z_t \left| \left(N_{[t,t+h]} = 0 \right) \right.$ will be normally distributed with mean $M_n = (\mu - pm)h$, and variance $S_n^2 = \sigma^2 h$.

If $N_{[t,t+h]} = 1$ the variable $z_t \left| \left(N_{[t,t+h]} = 1 \right) \right.$ will be normally distributed with mean $M_y = (\mu - pm)h + m$, and variance $S_y^2 = \sigma^2 h + s^2$.

The density function of z_t, denoted by $f(z_t)$, can be written in terms of conditional probabilities:

$$f(z_t) = f(z_t \left| N_{[t,t+h]} = 0 \right.) \Pr(N_{[t,t+h]} = 0) + f(z \left| N_{[t,t+h]} = 1 \right.) \Pr(N_{[t,t+h]} = 1)$$

$$= \frac{1}{\sqrt{2\pi} S_n} \exp\left(-\frac{(z_t - M_n)^2}{2S_n^2} \right) \times (1-p) + \frac{1}{\sqrt{2\pi} S_y} \exp\left(-\frac{(z_t - M_y)^2}{2S_y^2} \right) \times p \tag{3A.5}$$

If I have a time series of K observations of k_t, there will be $K-1$ observations of z's values with time interval equal to h=1. The log-likelihood function can be expressed as follows:

$$\sum_{i-1}^{K-1} \log f(z_i)$$

$$= \sum_{i=1}^{K-1} \ln\left(\frac{1}{\sqrt{2\pi} S_n} \exp\left(-\frac{(z_t - M_n)^2}{2S_n^2} \right)(1-p) + \frac{1}{\sqrt{2\pi} S_y} \exp\left(-\frac{(z_t - M_y)^2}{2S_y^2} \right) p \right). \qquad (3A.6)$$

Appendix 3B: The Log-likelihood Function for the Model with Transitory Jump Effects

Recall in section 3.4.2, I defined

$$z_t = \mu h + \sigma[W_{t+h} - W_t] + Y_{[t,t+h]}N_{[t,t+h]} - Y_{[t-h,t]}N_{[t-h,t]}, \tag{3B.1}$$

$$z_{t+h} = \mu h + \sigma[W_{t+2h} - W_{t+h}] + Y_{[t+h,t+2h]}N_{[t+h,t+2h]} - Y_{[t,t+h]}N_{[t,t+h]}, \tag{3B.2}$$

If $N_{[t,t+h]} = 0$, then z_t is independent on z_{t+h}. If $N_{[t,t+h]} = 1$, then z_t is correlated with z_{t+h} because of the $Y_{[t,t+h]}$ part. Under the conditional maximum likelihood estimation, the likelihood function can be obtained as follows:

$$\begin{aligned}
& f(z_1, z_2, ...z_{K-1}) \\
= \; & f(z_{K-1} | z_1, z_2, ...z_{K-2}) f(z_1, z_2, ..., z_{K-2}) \\
= \; & f(z_{K-1} | z_{K-2}) f(z_{K-2} | z_1, z_2, ...z_{K-3}) f(z_1, z_2, ...z_{K-3}) \\
= \; & f(z_{K-1} | z_{K-2}) f(z_{K-2} | z_{K-3}) ... f(z_2 | z_1) f(z_1),
\end{aligned} \tag{3B.3}$$

Therefore, the log-likelihood function is:

$$\begin{aligned}
& \ln f(z_1, z_2, ...z_{K-1}) \\
= & \ln f(z_{K-1} | z_{K-2}) + \ln f(z_{K-2} | z_{K-3}) + ... + \ln f(z_2 | z_1) + \ln f(z_1),
\end{aligned} \tag{3B.4}$$

Next, I will derive $f(z_{t+h} | z_t)$ and $f(z_1)$ respectively.

If $N_{[t,t+h]} = 0$, then $z_{t+h} = \mu h + \sigma[W_{t+2h} - W_{t+h}] + Y_{[t+h,t+2h]}N_{[t+h,t+2h]})$. (3B.5)

$z_{t+h} | (N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 0)$ will be normally distributed with mean $M_{nn} = \mu h$, and variance $S_{nn}^2 = \sigma^2 h$. $z_{t+h} | (N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 1)$ will be normally distributed with mean $M_{ny} = \mu h + m$, and variance $S_{ny}^2 = \sigma^2 h + s^2$.

If $N_{[t,t+h]} = 1$, I add equations (3B.1) and (3B.2) together and simplify to get

$$z_{t+h} = -z_t + 2\mu h + \sigma[W_{t+2h} - W_t] + Y_{[t+h,t+2h]}N_{[t+h,t+2h]} - Y_{[t-h,t]}N_{[t-k,t]}. \tag{3B.6}$$

If no mortality jump event occurs during the period $(t-h,t)$ and $(t+h,t+2h)$, the variable $z_{t+h}\big|(z_t, N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0)$ will be normally distributed with mean $M_{nyn} = -z_t + 2\mu h$ and variance $S^2_{nyn} = 2\sigma^2 h$.

Similarly, $z_{t+h}\big|(z_t, N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0)$ will be normally distributed with mean $M_{yyn} = -z_t + 2\mu h - m$ and variance $S^2_{yyn} = 2\sigma^2 h + s^2$.

$z_{t+h}\big|(z_t, N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1)$ will be normally distributed with mean $M_{nyy} = -z_t + 2\mu h + m$ and variance $S^2_{nyy} = 2\sigma^2 h + s^2$.

$z_{t+h}\big|(z_t, N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1)$ will be normally distributed with mean $M_{yy} = -z_t + 2\mu h$ and variance $S^2_{yy} = 2\sigma^2 h + 2s^2$.

The conditional density function of $z_{t+h}\big|z_t$, denoted by $f(z_{t+h}\big|z_t)$, can be written as

$$\begin{aligned}
&f(z_{t+h}\big|z_t) \\
&= f(z_{t+h}\big|N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 0)\Pr(N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 0) \\
&+ f(z_{t+h}\big|N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 1)\Pr(N_{[t,t+h]} = 0, N_{[t+h,t+2h]} = 1) \\
&+ f(z_{t+h}\big|z_t, N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0)\Pr(N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0) \\
&+ f(z_{t+h}\big|z_t, N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0)\Pr(N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 0) \\
&+ f(z_{t+h}\big|z_t, N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1)\Pr(N_{[t-h,t]} = 0, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1) \\
&+ f(z_{t+h}\big|z_t, N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1)\Pr(N_{[t-h,t]} = 1, N_{[t,t+h]} = 1, N_{[t+h,t+2h]} = 1)
\end{aligned}$$

$$= \frac{1}{\sqrt{2\pi}S_{nn}} \exp\left(-\frac{(z_{t+h}-M_{nn})^2}{2S_{nn}^2}\right)(1-p)^2 + \frac{1}{\sqrt{2\pi}S_{ny}} \exp\left(-\frac{(z_{t+h}-M_{ny})^2}{2S_{ny}^2}\right)p(1-p)$$

$$+ \frac{1}{\sqrt{2\pi}S_{nyn}} \exp\left(-\frac{(z_{t+h}-M_{nyn})^2}{2S_{nyn}^2}\right)p(1-p)^2 + \frac{1}{\sqrt{2\pi}S_{yyn}} \exp\left(-\frac{(z_{t+h}-M_{yyn})^2}{2S_{yyn}^2}\right)p^2(1-p)$$

$$+ \frac{1}{\sqrt{2\pi}S_{nyy}} \exp\left(-\frac{(z_{t+h}-M_{nyy})^2}{2S_{nyy}^2}\right)p^2(1-p) + \frac{1}{\sqrt{2\pi}S_{yyy}} \exp\left(-\frac{(z_{t+h}-M_{yyy})^2}{2S_{yyy}^2}\right)p^3 \qquad \text{(3B.7)}$$

The variable $z_1 \big| (N_{[0,1]} = 0, N_{[1,2]} = 0)$ will be normally distributed with mean $\hat{M}_{nn} = \mu h$, and variance $\hat{S}_{nn}^2 = \sigma^2 h$.

The variable $z_1 \big| (N_{[0,1]} = 1, N_{[1,2]} = 0)$ will be normally distributed with mean $\hat{M}_{yn} = \mu h - m$, and variance $\hat{S}_{yn}^2 = \sigma^2 h + s^2$

The variable $z_1 \big| (N_{[0,1]} = 0, N_{[1,2]} = 1)$ will be normally distributed with mean $\hat{M}_{ny} = \mu h + m$, and variance $\hat{S}_{ny}^2 = \sigma^2 h + s^2$

The variable $z_1 \big| (N_{[0,1]} = 1, N_{[1,2]} = 1)$ will be normally distributed with mean $\hat{M}_{yy} = \mu h$, and variance $\hat{S}_{yy}^2 = \sigma^2 h + 2s^2$

The density function of z_1, which is denoted by $f(z_1)$, can be written as:

$$f(z_1)$$

$$= f(z_1 \big| N_{[0,1]} = 0, N_{[1,2]} = 0)\Pr(N_{[0,1]} = 0, N_{[1,2]} = 0)$$

$$+ f(z_1 \big| N_{[0,1]} = 1, N_{[1,2]} = 0)\Pr(N_{[0,1]} = 1, N_{[1,2]} = 0)$$

$$+ f(z_1 \big| N_{[0,1]} = 0, N_{[1,2]} = 1)\Pr(N_{[0,1]} = 0, N_{[1,2]} = 1)$$

$$+ f(z_1 \big| N_{[0,1]} = 1, N_{[1,2]} = 1)\Pr(N_{[0,1]} = 1, N_{[1,2]} = 1)$$

$$= \frac{1}{\sqrt{2\pi}\hat{S}_{nn}} \exp\left(-\frac{(z_1 - \hat{M}_{nn})^2}{2\hat{S}_{nn}^2} \right)(1-p)^2 + \frac{1}{\sqrt{2\pi}\hat{S}_{yn}} \exp\left(-\frac{(z_1 - \hat{M}_{yn})^2}{2\hat{S}_{yn}^2} \right) p(1-p)$$

$$+ \frac{1}{\sqrt{2\pi}\hat{S}_{ny}} \exp\left(-\frac{(z_1 - \hat{M}_{ny})^2}{2\hat{S}_{ny}^2} \right)(1-p)p + \frac{1}{\sqrt{2\pi}\hat{S}_{yy}} \exp\left(-\frac{(z_1 - \hat{M}_{yy})^2}{2\hat{S}_{yy}^2} \right) p^2 . \qquad (3B.8)$$

Substituting the formulas of $f(z_{t+h}|z_t)$ and $f(z_1)$ into the log-likelihood function (3B.4), I can calculate the log-likelihood function numerically.

Appendix 4A: Construction of the Dynamic Complete Life Table

We have 11 age groups:

$g_0 = \{0\}$,
$g_1 = \{1,2,3,4\}$,
$g_2 = \{5,6,...,14\}$,
.....,
$g_{10} = \{75,76,...,84\}$,
$g_{11} = \{85,86,...\}$

The question is: given $m_{k,t}$**,** the central-death-rates for the age group g_k at time t, how to obtain $q_{y,t}$, the probability that an individual aged y will die in one year at time t?

I use the following notations here (for simplicity, I omit the subscript t).

ω: The highest attainable age. Suppose ω = 110;

m_k**:** Central-death-rate for the age group g_k;

x_k: The first age in the age group g_k;

n_k : $x_{k+1} - x_k$;

l_y: Expected number of survivors to age y;

d_y: Expected number of deaths between ages y and $y+1$;

${}_nL_y$: The total expected number of years lived between ages y and $y+n$;

q_y: Probability that an individual aged y will die in one year.

Assuming that the function l_y is linear over the interval $x_k \le y \le x_{k+1}$, we have:

$$l_y = l_{x_k} - \frac{y - x_k}{n_k}(l_{x_k} - l_{x_{k+1}}), \tag{4A.1}$$

and

$$d_y = \frac{l_{x_k} - l_{x_{k+1}}}{n_k} = d_{x_k}\,, \tag{4A.2}$$

for all $y \in g_k$

Now we need to use the central death rate m_k to determine q_{x_k}.

$$_{n_k}L_{x_k} = \int_{x_k}^{x_{k+1}} l_y dy = \int_{x_k}^{x_{k+1}} \left(l_{x_k} - \frac{y - x_k}{n_k}(l_{x_k} - l_{x_{k+1}}) \right) dy\,,$$

$$= n_k l_{x_k} - d_{x_k} \int_0^{n_k} t dt = n_k l_{x_k} - d_{x_k} n_k^{\,2} / 2\ . \tag{4A.3}$$

Then

$$m_k = \frac{l_{x_k} - l_{x_{k+1}}}{_{n_k}L_{x_k}} = \frac{n_k d_{x_k}}{n_k l_{x_k} - d_{x_k} n_k^{\,2} / 2} = \frac{q_{x_k}}{1 - n_k q_{x_k} / 2}\,. \tag{4A.4}$$

Solving for q_{x_k}, I obtain $q_{x_k} = \dfrac{m_k}{1 + n_k m_k / 2}$

The probability q_y for age $y \in g_k$ is determined by q_{x_k}:

$$q_y = \frac{d_y}{l_y} = \frac{d_{x_k}}{l_{x_k} - \frac{y - x_k}{n_k}(l_{x_k} - l_{x_{k+1}})} = \frac{d_{x_k}}{l_{x_k} - (y - x_k) d_{x_k}} = \frac{q_{x_k}}{1 - (y - x_k) q_{x_k}} \tag{4A.5}$$

Appendix 4B: Change of Measure via the Conditional Esscher Transform

Given $Y_t \mid \Phi_{t-1} \sim N\left(\hat{\mu}_t, \sigma_t^2\right)$ under P, prove $Y_t \mid \Phi_{t-1} \sim N\left(r - \frac{1}{2}\sigma_t^2, \sigma_t^2\right)$ under Q via the conditional Esscher Transform.

Proof:

The conditional Esscher transform can be described as:

$$f_{Q_t}(y \mid \Phi_{t-1}) = f_{P_t}(y \mid \Phi_{t-1}) \frac{\exp(\lambda_t y)}{E\left[\exp(\lambda_t Y_t) \mid \Phi_{t-1}\right]} \tag{4B.1}$$

It immediately follows that the moment generating function of Y_t given Φ_{t-1} under the measure Q is given by

$$E_{Q_t}\left[\exp(zY_t); \lambda_t \mid \Phi_{t-1}\right] = E_{P_t}\left[\exp(zY_t) \frac{\exp(\lambda_t Y_t)}{E_{P_t}\left[\exp(\lambda_t Y_t) \mid \Phi_{t-1}\right]} \mid \Phi_{t-1}\right]$$

$$= \frac{E_{P_t}\left[\exp((z+\lambda_t)Y_t) \mid \Phi_{t-1}\right]}{E_{P_t}\left[\exp(\lambda_t Y_t) \mid \Phi_{t-1}\right]}. \tag{4B.2}$$

Because $Y_t \mid \Phi_{t-1} \sim N\left(\hat{\mu}_t, \sigma_t^2\right)$ under P, the moment generating function of Y_t given Φ_{t-1} under the measure P is given by

$$E_{P_t}[\exp(zY_t) \mid \Phi_{t-1}] = \exp\left(\mu_t z + \frac{1}{2}\sigma_t^2 z^2\right). \tag{4B.3}$$

Substituting equation (4B.3) into equation (4B.2)), we obtain

$$E_{Q_t}\left[\exp(zY_t); \lambda_t \mid \Phi_{t-1}\right] = \frac{\exp\left(\mu_t(z+\lambda_t) + \frac{1}{2}\sigma_t^2(z+\lambda_t)^2\right)}{\exp\left(\mu_t\lambda_t + \frac{1}{2}\sigma_t^2\lambda_t^2\right)}$$

$$= \exp\left((\mu_t + \sigma_t^2 \lambda_t) z + \frac{1}{2}\sigma_t^2 z^2 \right) . \qquad \text{(4B.4))}$$

Therefore,

$$E_{Q_t}\left[\exp(Y_t); \lambda_t^q \mid \Phi_{t-1}\right] = \exp\left(\mu_t + \sigma_t^2 \lambda_t^q + \frac{1}{2}\sigma_t^2 \right). \qquad \text{(4B.5)}$$

In order for Q to be an equivalent martingale measure, we need to have:

$$E_{Q_t}\left[\exp(Y_t); \lambda_t^q \mid \Phi_{t-1}\right] = \exp(r) . \qquad \text{(4B.6)}$$

Equating the above two equations, we can solve

$$\lambda_t^q = \frac{r - \mu_t}{\sigma_t^2} - \frac{1}{2}, (t = 1,2,...,T) . \qquad \text{(4B.7)}$$

Substituting equation (4B.7) into (4B.5), we obtain

$$E_{Q_t}\left[\exp(zY_t); \lambda_t^q \mid \Phi_{t-1}\right] = \exp\left(\left(r - \frac{1}{2}\sigma_t^2 \right) z + \frac{1}{2}\sigma_t^2 z^2 \right), \qquad \text{(4B.8)}$$

which means the random variable Y_t, given the information Φ_{t-1}, is normally distributed with mean $r - \frac{1}{2}\sigma_t^2$ and variance σ_t^2 under Q.

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