Regulatory Capital Requirement for Supplementary Health Insurance: A Solvency Perspective

by

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Abstract

This thesis investigates how a solvency regulatory capital requirement, like that in Solvency II, affects the risk load of a supplementary health insurance product. Motivated by a credit risk model, we introduce a static structural model in which the latent variable represents an individual’s severity of a certain illness. In this model, we include the effects on an individual’s health from common shock, systematic risk and idiosyncratic risk. We derive the asymptotic distribution of the aggregate supplementary health claims, based on which the minimum risk load can be calculated in order to meet the solvency regulatory capital requirement. The main contributions of this thesis are first proposing an appropriate model for the aggregate supplementary health claims, and then finding the lower bound of the risk load from the solvency perspective.
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Chapter 1

Introduction

Health insurance is an essential element for a country. In some countries, the government provides a basic universal health insurance system to cover the cost of medical treatments. These basic health insurance systems are different in different countries. For example, the Canadian government provides an overall and universal basic health insurance system to cover almost all medical treatment fees. However, this kind of overall basic universal insurance system is not common. Some countries including China provide basic health insurance just covering a part of medical treatment fees. The costs to restore health can be considerable even though the basic insurance covers a part of the treatment fee and some people can’t afford these expensive treatment fees. Thus, individuals may wish to buy supplementary insurances from insurance market to cover these very expensive medical treatment costs. For insurance companies, they need to consider various risk factors which will influence the pricing or risk management of their health supplementary insurances. In traditional models, based on the Law of Large Number (LLN), insurance
pooling effect shows that the risk of supplementary insurances will decrease with the increase of policyholder size. A very important assumption for this pooling effect is that policyholders are independent with each other. However, the population health is exposed to common risks which affect population health simultaneously. Thus, the assumption of independence can’t be satisfied. In our thesis, we focus on the effects of common risk factors in health insurance claims for a large population. Specifically, we will introduce an aggregate health claims model that takes common risk factors into consideration and, based on the model, figure out a method to calculate the lower bound of the risk load of the supplementary health insurance from the solvency perspective.

In our thesis, risk factors are classified into common risk factors and idiosyncratic risk factors. The common risk factors include systematic risks and common shocks (Figure 1.1). There are a couple of differences between systematic risks and common shocks. First of all, systematic risks are the potential risk factors which will influence public health in a country as a whole. However, the common shocks just influence the people in a certain area. Next, the effects of common shocks will disappear gradually but the systematic risks will influence human health for a long time.

1.1 Systemic Risk

Systematic risk is the overall, ongoing and long-term risk that is inherent to the whole population. Persistent air pollution is a representative example of systematic risk factors and is significantly associated with human health. A lot of
evidence suggests that air pollution is the main attribution for some diseases including chronic obstructive, pulmonary disease, pneumonia and asthma. It will cause a long-term and overall health crisis which includes respiratory and cardiovascular diseases and even premature death. Lim et al. (2012) show that ambient fine particulate matter (PM2.5), ambient ozone pollution and household air pollution cause lower respiratory infections, trachea, bronchus, ischaemic heart dis-
ease, cerebrovascular disease and chronic obstructive pulmonary disease. In 2010, PM2.5 and ozone caused over 3.2 million respiratory and cardiovascular diseases and 150000 premature deaths respectively. There are approximately 4 million premature deaths related to household air pollution (Anenberg et al, 2016). Greco et al. (2016) used the mortality risk reduction caused by the reduction of PM2.5 concentration to show the health crisis brought from PM2.5. They found that the expected reduction of premature deaths with a 100-metric-ton reduction of PM2.5 is 48 deaths. The health crisis caused by PM2.5 has brought trillions of dollars of loss (North, 2016). Persistent air pollution as a very important element in systematic risks has obvious effects on population health and has been a major and worldwide concern.

Some other examples of systematic risk are long-term underwater contamination and long-term unimproved water and sanitation. Underwater contamination is a source of some diseases such as cholera and is a potential and common public health threat because people use underwater to drink or bath. The concentrations of trace metals in underwater will bring population with chronic health risks. Some studies show that Cr\(^{6+}\) exposure via oral route will lead to cancer. At the same time, some other trace metals including Cd\(^{62+}\), Ni\(^{2+}\) and Pb\(^{2+}\) expose to underwater resulting in higher carcinogenic risks. The high concentrations of these trace metals in underwater have brought significant health risks to people (Etchie et al., 2012). Moreover, unimproved water and sanitation problems still exist. They result in approximately 0.3 million deaths globally in 2010 (Lim et al., 2012). Besides long-term underwater contamination and persistent air pollution, chemicals
used in industrial applications also have brought serious effects on the population health. For example, Perfluorooctanoic acid (PFOA) is used in industrial and commercial applications as waterproof clothing and paper coatings used in food packaging for decades. However, this chemical has a significant association with kidney and testicular cancer (Barry et al., 2013; Benbrahim-Tallaa et al., 2014). In addition, poverty, undernutrition, unsafe sex and iron deficiency are systematic risks for some developing countries (WHO, 2002).

1.2 Common Shock

Common shock symbolizes certain external events that cause serious but short-term population health crisis. The outbreak of epidemic diseases is a representative example of common shock. Some epidemic diseases including cholera, diarrhea, dengue fever, malaria and so on are still very popular now. The studies about modern epidemiology originated John Snow’s investigation of the 1854 cholera epidemic in London. The death due to cholera in London increased from 8 January 1842 to 28 December 1901 (Tien et al., 2011). Nowadays, cholera still is a serious and tricky disease for the areas without clean water. Some areas outbreaking cholera epidemics recently include Angola in 2006, Zimbabwe in 2008-2009 and Haiti in 2010.

Natural disasters are important examples of common shocks because various natural disasters may cause short-term air pollution, epidemic diseases or short-term groundwater contamination. A representative natural disaster is wildfire such as 2019 Amazon rainforest wildfires and 2019 Alberta wildfires. The cli-
mate changes have obviously increased the probability of wildfire (Albertson et al., 2010). Liu et al. (2015) reviewed the studies about the adverse health effects brought by wildfires for the people in a certain area. They summarize that the risks of respiratory morbidity and cardiovascular diseases are significantly associated with the smoke from wildfires. There is a 1.2 to 10 times increase in PM10 due to wildfire smoke. In addition, the wildfires also increase the exposure to a lot of hazardous gases including CO, SO$_2$, O$_3$ and NO$_x$. Another example is volcanic eruption. Some hazardous materials such as volcanic gases left by volcano eruption even have harmful effects on vegetation and infrastructure. The United States Geological Survey (USGS) (available at: https://volcanoes.usgs.gov/vhp/gas.html) mentions that 1991’s volcano eruption of Mt. Pinatubo injected more than 250 megatons of gas into the upper atmosphere on a single day. If the magma can’t reach the surface, the volcanic gases will get into the atmosphere from the soil resulting in negative effects on crops. At the same time, some other harmful gases including carbon dioxide, sulfur dioxide, hydrogen sulfide and hydrogen halides will release to the atmosphere resulting in short-term air pollution which is hazardous to humans, animals and agriculture (USGS, 2019). The natural disasters also result in short-term low underwater quality. It causes water-borne risk to health by some water-borne infectious diseases including diarrhea, typhoid, cholera, dysentery and infectious hepatitis (Hlavinék et al., 2008). In addition to wildfire and volcanic eruption, ocean tsunami also is a source of a health crisis. The Indian Ocean Tsunami in 2004 resulted in a wide range of flood in Africa which causes bacterial infection. Thus, some epidemic diseases such as cholera, diarrhea
and malaria spread in Africa.

1.3 Threshold

In our thesis, in order to judge if an individual is diagnosed with a certain illness, we assume that there exists a positive and fixed threshold and the individual is diagnosed with the illness when this individual’s severity of the illness is higher than the fixed threshold. We use a latent variable to represent an individual’s severity of the illness. The latent variable follows a static structural model containing common shock, systematic risk and idiosyncratic risk.

This threshold model is motivated by some models in the literature of credit risk. Bassamboo et al. (2008) propose a static structural model for a large portfolio of obligors. They assume an obligor defaults when the threshold is reached. They derive the estimation of sharp asymptotics of loss from portfolio defaults. One of the key assumptions in their paper is that the threshold for individual obligor increases to infinity as portfolio size increases.

Inspired by Bassamboo et al. (2008), Tang et al. (2019) use the same threshold model to study the default behavior of a large portfolio of obligors. They find that the occurrence of large losses can be attributed to either the common shock variable or the systematic risk factor, whichever has a heavier tail.

Unlike Bassamboo et al. (2008) and Tang et al. (2019), Liu (2018) uses a fixed threshold to define default. The default probability of an individual obligor does not change as the portfolio size increases. Furthermore, Liu (2018) does not allow portfolio defaults to be affected by common shock.
Our threshold model shares with Liu (2018) the idea that the threshold is fixed for individuals. Thus, the probability of an individual to be diagnosed with the illness does not change as the population increases. However, we consider common shock effects that influence the health of a population simultaneously.
Chapter 2

Models

2.1 Severity Model

We consider a certain population with \( n \) individuals that are exposed to the risk of a certain illness. We build a model to capture an individual’s severity of this illness. Precisely, we use latent variable \( v_j \) to represent individual \( j \)’s severity of the illness and individual \( j \)’s severity is affected by idiosyncratic risk, common shock and systematic risk:

\[
v_j = S \left( X + Y_j \right), \quad j = 1, 2, \ldots, n. \tag{2.1.1}
\]

In this model, \( X \) is a positive random variable representing the systematic risks which are inherent to the whole population, \( S \geq 1 \) captures the common shock effects on the population, and \( Y_j \) is a positive random variable symbolizing idiosyncratic risks.
An underlying assumption in this model is that everyone in this population is affected by the same common shock $S$ and systematic risk $X$. Note that when $S = 1$ there is no common shock effect. While when $S > 1$, the common shock effect applies on everyone in the population.

Throughout this thesis, we have the following independence assumption:

**Assumption 2.1.1** The idiosyncratic risks, $\{Y_j : j = 1, \ldots, n\}$, are i.i.d. Furthermore, $(S, X)$ is independent with the idiosyncratic risks $\{Y_j : j = 1, \ldots, n\}$.

Under this assumption, similar to the portfolio default model built by Tang et al. (2019) and Bassamboo et al. (2008), the severity model is a conditional independent model given $(S, X)$.

Given individual $j$’s severity of the illness $v_j$, we assume that a positive and fixed threshold $\tau$ exists and, when individual $j$’s severity of the illness $v_j$ is higher than the threshold $\tau$, individual $j$ is diagnosed with the illness. The threshold $\tau$ can be estimated based on specific medical indexes for specific diseases or physicians’ diagnosis. For example, the medical index for hypertension is 144–159/90–99mmHg which means that, when the blood pressure of an individual is higher than this medical index, she is diagnosed with hypertension.

### 2.2 Utility

For simplicity, we assume that only one treatment is available for this illness and its cost is $\delta$. Although patients can get benefits from this medical treatment, using treatment without restriction may be a waste for the whole society. If the
medical treatment is used without restrictions, individual $j$ will use the treatment as long as she is diagnosed with the illness, i.e. $v_j > \tau$. We use utility function $U(v)$ to represent the utility gain of using the treatment when the patient’s severity is $v$. Here $U(\cdot)$ is a positive, continuous and strictly increasing function. Furthermore, the utility function $U(\cdot)$ should be a concave function (Schosser et al., 2016). Although an individual’s utility gain may be greater than the treatment’s cost, the social average utility gain obtained from the treatment may be lower than its cost.

**Assumption 2.2.1** *The treatment is not socially cost-effective if used without restriction:*

\[ \text{TVaR}_{U(v)}(U(\tau)) < \delta. \]

The TVaR of a r.v. is the average percentile above a threshold. Under the condition that individual $j$ is diagnosed with the illness ($v_j > \tau$), $\text{TVaR}_{U(v)}(U(\tau))$ symbolizes the conditional expectation of utility gain derived from the treatment for the whole society. When this conditional average utility gain is lower than the costs of the medical treatment, using treatment without restriction destroys social surplus in the sense that the cost exceeds the average utility gain. The treatment is not socially cost-effective.

There is an underlying assumption that, although a physician is able to determine whether or not an individual has the illness, they can not recognize the severity of the individual. In other words, insurers or planners just know whether an individual has the illness. But they can not verify the patient’s severity.
2.3 Health Insurance

We assume that people are risk-averse and the cost of the treatment is high enough so that people may need to buy insurance to cover the treatment cost. Furthermore, they do not have budget constraints. Our thesis considers a combined health insurance system where the government provides free universal basic insurance covering $\delta - \gamma$ in the total cost of the treatment and private insurance companies offer voluntary supplementary insurance. The $\gamma$ is determined by the government and people need to buy supplementary insurances from insurance market. Moreover, the supplementary insurance is bought in addition to basic insurance to cover further treatment costs. This kind of health insurance system combining governmental basic insurance and commercial insurance exists in many countries such as China, Japan, Belgium, etc.

Because we assume that the treatment is not socially cost-effective, the moral hazard exists. In our thesis, motivated by Boone (2018), the definition of moral hazard due to health insurance is that, if the treatment is used without restriction, the unnecessary treatment uses will destroy social surplus in the sense that the average utility gain is lower than the cost. Thus, in order to reduce the moral hazard and increase the cost-effectiveness of the treatment, we introduce a copayment $c$. In other words, an individual also needs to pay a copayment $c$ when she uses the treatment. In this case, the individual will not use the treatment if the utility gain of using the treatment is lower than the copayment $c$. Point A in Figure 2.1 is the minimal utility gain obtained from using treatment without restriction. Because of the copayment, an individual will use the treatment only when the utility gain
of using treatment is higher than $c$. In this case, the minimal utility gain point changes from A to B in Figure 2.1 resulting in a decrease of moral hazard. At the same time, the cost-effectiveness of the treatment also is improved. However, this method can not totally prevent cost-ineffectiveness for the whole society. It is because, even though the minimal utility gain increases, the average utility gain of using the treatment may still be lower than the cost of treatment $\delta$ (point C in Figure 2.1). Therefore, in order to eliminate the social ineffective use of the treatment, we assume:

**Assumption 2.3.1** There exists some $c$ in $(U(\tau), \delta)$ such that

$$\text{TVaR}_{U(v)}(c) = \delta.$$
Thus, the social average utility gain is equal to the cost of using the treatment by introducing copayment $c$. The moral hazard is prevented.

Now, when a patient uses the treatment, the basic universal insurance covers $\delta - \gamma$, the supplementary insurance covers $\gamma - c$ and the patient herself pays a copayment $c$. After introducing the copayment $c$, the probability that an individual uses the treatment becomes $\Pr(v_j > U^{-1}(c))$ rather than $\Pr(v_j > \tau)$. Therefore, denoting by $\eta$ the risk load, the premium of the supplementary health insurance is

$$p(\eta) = (1 + \eta)(\gamma - c) \Pr(v_j > U^{-1}(c)).$$

(2.3.1)
Chapter 3

Regulatory Capital and Risk Load

3.1 Asymptotic Distribution of Aggregate Claims

Based on the models and assumptions in Chapter 2, we denote the whole population’s aggregate claim payment for the supplementary health insurance as

\[ T_n = (\gamma - c) \sum_{j=1}^{n} 1(U(v_j) > c). \]  

(3.1.1)

We first derive the asymptotic distribution of \( T_n \) when \( n \to \infty \). Motivated by Tang et al. (2019), we have the following result:

Proposition 3.1.1 Consider \( T_n \) defined above. Assume that \( (S, X) \) is jointly continuously distributed and that \( \text{supp}(F_Y) \), the support set of \( F_Y \), is a non-empty interval when \( Y \) is not degenerate. Then, for any fixed \( b \in (0, \gamma - c) \),

\[ \lim_{n \to \infty} \Pr(T_n/n > b) = \int_{r(s,x) > b} \Pr(S \in ds, X \in dx), \]  

(3.1.2)
where

\[ r(s, x) = (\gamma - c) F_Y \left( \frac{U^{-1}(c)}{s} - x \right). \]

Note that for a non-decreasing function \( f : \mathbb{R} \to \mathbb{R} \), its support set, \( \text{supp}(f) \), consists of points \( x \in \mathbb{R} \) such that \( f(x) \) is not constant in its neighbourhood. Thus, the assumption about the support set of \( F_Y \) can ensure \( F_Y \) is a strictly increasing function when \( Y \) is not degenerate.

**Proof of Proposition 3.1.1** Due to the independence between \((S, X)\) and \(\{Y_j : j = 1, \cdots, n\}\) it holds that

\[
\Pr \left( \frac{T_n}{n} > b \right) = \int_{[1,\infty) \times \mathbb{R}_+} \Pr \left( \frac{T_n}{n} > b \mid S = s, X = x \right) \Pr(S \in ds, X \in dx). \tag{3.1.3}
\]

Based on the LLN, given \( S = s \) and \( X = x \), it holds almost surely as \( n \to \infty \) that

\[
\frac{T_n}{n} = \frac{\gamma - c}{n} \sum_{j=1}^{n} 1(\upsilon_j > c) \to (\gamma - c) \Pr(\upsilon_j > U^{-1}(c)) = (\gamma - c) \bar{F}_Y \left( \frac{U^{-1}(c)}{s} - x \right) := r(s, x).
\]
For arbitrarily fixed small $\epsilon > 0$, we have

\[
\Pr \left( \frac{T_n}{n} > b \middle| S = s, X = x \right) = \Pr \left( \frac{T_n}{n} > b \middle| S = s, X = x \right) (1_{r(s,x) \geq b-\epsilon} + 1_{r(s,x) < b-\epsilon}) \\
\leq 1_{r(s,x) \geq b-\epsilon} + \Pr \left( \frac{T_n}{n} > r(s,x) + \epsilon \middle| S = s, X = x \right) \\
\to 1_{r(s,x) \geq b-\epsilon}.
\]

Similarly,

\[
\Pr \left( \frac{T_n}{n} > b \middle| S = s, X = x \right) \geq \Pr \left( \frac{T_n}{n} > b \middle| S = s, X = x \right) 1_{r(s,x) > b+\epsilon} \\
\geq \Pr \left( \frac{T_n}{n} > r(s,x) - \epsilon \middle| S = s, X = x \right) 1_{r(s,x) > b+\epsilon} \\
\to 1_{r(s,x) > b+\epsilon}.
\]

Thus, for arbitrarily small $\delta > 0$, there exists some large positive integers $n_0$ such that, for all $n \geq n_0$,

\[
1_{r(s,x) > b+\epsilon} - \delta \leq \Pr \left( \frac{T_n}{n} > b \middle| S = s, X = x \right) \leq 1_{r(s,x) \geq b-\epsilon} + \delta.
\]

Now applying Fatou’s lemma to (3.1.3), we have

\[
\liminf_{n \to \infty} \Pr \left( \frac{T_n}{n} > b \right) \geq \int_{1,\infty} \int_{\mathbb{R}^+} 1_{r(s,x) > b+\epsilon} \Pr \left( S \in ds, X \in dx \right) - \delta \\
= \int_{r(s,x) > b+\epsilon} \Pr \left( S \in ds, X \in dx \right) - \delta \tag{3.1.4}
\]
and

\[
\limsup_{n \to \infty} \Pr\left( \frac{T_n}{n} > b \right) \leq \int\int_{[1, \infty) \times \mathbb{R}_+} 1_{r(s, x) \geq b - \varepsilon} \Pr(S \in s, X \in x) + \delta \\
= \int\int_{r(s, x) \geq b - \varepsilon} \Pr(S \in ds, X \in dx) + \delta.
\]

Combining (3.1.4) and (3.1.5) and letting \( \delta \) decreases to 0, we obtain

\[
\int\int_{r(s, x) > b + \varepsilon} \Pr(S \in ds, X \in dx) \leq \liminf_{n \to \infty} \Pr\left( \frac{T_n}{n} > b \right) \\
\leq \limsup_{n \to \infty} \Pr\left( \frac{T_n}{n} > b \right) \\
\leq \int\int_{r(s, x) \geq b - \varepsilon} \Pr(S \in ds, X \in dx).
\]

By the arbitrariness of \( \varepsilon \), from (3.1.6) we immediately have

\[
\int\int_{r(s, x) > b} \Pr(S \in ds, X \in dx) \leq \liminf_{n \to \infty} \Pr\left( \frac{T_n}{n} > b \right) \\
\leq \limsup_{n \to \infty} \Pr\left( \frac{T_n}{n} > b \right) \\
\leq \int\int_{r(s, x) \geq b} \Pr(S \in ds, X \in dx).
\]

When \( Y \) is not degenerate, it is obvious that \( F_Y \) is non-decreasing in both \( s \in [1, \infty) \) and \( x \in \mathbb{R}_+ \). By assuming that \( \text{supp}(F_Y) \) is a non-empty interval, we know that \( F_Y \) is a strictly increasing function. Thus, \( r(s, x) \) also is a strictly increasing function. This guarantees that \( r(s, x) = b \) does not allow a rectangle for \( (s, x) \). Thus, we have that \( \Pr(r(s, x) = b) = 0 \) for every \( b \in (0, \gamma - c) \). When \( F_Y \) is degenerate, variable \( Y \) has a single possible value, \( \Pr(r(s, x) = b) = 0 \) automatically holds. Thus, (3.1.7)
reduces to (3.1.2). This completes the proof. ■

### 3.2 Regulatory Capital Requirement

Generally speaking, the government formulates some regulations to protect policyholders from the price competition between insurance companies and also to provide appropriate incentives for good risk management. It is mainly because some insurance companies may lower insurance prices to attract more customers. It will result in a decrease in the reserve prepared to pay future claims and increase the risk that an insurance company can not afford claims. These regulations may be directly or indirectly related to the aggregate claim payment of an insurance policy $T_n$. In our thesis, we consider a regulation in Europe called Solvency II and assume that the reserve of a business line only derives from the premium revenues from this business line. An important capital requirement in Solvency II is solvency capital requirement (SCR). The SCR is a Value at Risk at the 99.5% confidence level of "basic own funds". The "basic own funds" refers to broadly assets minus best estimate of liability and risk margin. See Solvency II – Health Insurance (available at [https://www.actuaries.org.uk/system/files/field/document](https://www.actuaries.org.uk/system/files/field/document)).

In our thesis, based on above assumptions, "basic own funds", denoted by $W_n$, symbolizes the difference between premium revenues and claim amount:

$$W_n = np(\eta) - T_n = np(\eta) - (\gamma - c) \sum_{j=1}^{n} 1_{U(v_j) > c}.$$  \hspace{1cm} (3.2.1)

Consider $W_n$ defined above. If the government sets the minimum SCR per capita,
denoted by $\xi$, then, in order to complete with other insurance companies and meet the minimum SCR per capita, an insurance company will lower price until the price satisfies that the minimum SCR per capita is the 99.5 percentile of the "basic own funds". In other words:

$$\Pr(W_n/n < \xi) = 0.995 \iff \Pr(T_n/n > p(\eta) - \xi) = 0.995.$$  \hfill (3.2.2)

It is because insurance companies want to lower the price of the business line to improve competitiveness. However, lowering the premium will decrease reserve resulting in a higher probability that an insurance company does not have enough money to pay claims under extreme situations. Thus, the government sets the minimum SCR per capita to restrict the minimum premium of the business line. Moreover, setting the minimum SCR per capita can help the government to estimate the losses paid by the government when insurance companies can not afford their claims under extreme situations.

### 3.3 A Numerical Example

In this section, we give a numerical example in which, given the solvency capital requirement per capita, the minimum required premium and risk load have closed-form formulas. The model specifications are listed below:

1. The common shock, $S(\geq 1)$, follows a two-point mixture of 1 and a condi-
tional inverse exponential distribution with parameter $\theta_S$. Thus,

$$F_S(s) = \begin{cases} 
q, & \text{if } s = 1, \\
q + \frac{1-q}{1-\exp(-\theta_S)} \left[ \exp(-\theta_S/s) - \exp(-\theta_S) \right], & \text{if } s > 1.
\end{cases}$$

2. The systematic risk factor $X$ follows an exponential distribution with pdf $f_X(x) = \exp(-x/\theta_X)/\theta_X$ and a generic idiosyncratic risk factor $Y$ follows an exponential distribution with pdf $f_Y(y) = \exp(-y/\theta_Y)/\theta_Y$.

3. The common shock $S$ and the systematic risk factor $X$ are independent.

4. The utility function $U(\cdot)$, defined on $[\tau, \infty)$, is given by a concave function with

$$U(v) = U(\tau) + b(v - \tau)^{1/a}.$$ 

where $a > 1$ and $b > 0$ are fixed.

Using Proposition 3.1.1 for large $n$ the right-hand side of (3.2.2) can be calculated asymptotically as the tail probability of $T_n/n$. So,

$$\Pr(W_n/n < \xi) \doteq \int \int_{r(s,x) > p(\eta) - \xi} \Pr(S \in ds, X \in dx). \quad (3.3.1)$$
We also have

\[ r(s, x) > p(\eta) - \xi \]
\[ \iff (\gamma - c) F_Y \left( \frac{U^{-1}(c)}{s} - x \right) > p(\eta) - \xi \]
\[ \iff (\gamma - c) \exp \left( \frac{x - U^{-1}(c)/s}{\theta_Y} \right) > p(\eta) - \xi \]
\[ \iff x > U^{-1}(c)/s + \theta_Y \ln \left( \frac{p(\eta) - \xi}{\gamma - c} \right) . \]  \hfill (3.3.2)

For simplicity, let us denote \( g(\eta) = \theta_Y \ln \left( \frac{p(\eta) - \xi}{\gamma - c} \right) \). By combining (3.3.1) and (3.3.2) and using the independence between \( S \) and \( X \), we have

\[
\Pr(\frac{W_n}{n} < \xi) \]
\[ = \int_1^\infty F_X \left( \frac{U^{-1}(c)}{s} + g(\eta) \right) \Pr(S \in ds) \]
\[ = \int_1^\infty \exp \left( - \left( \frac{U^{-1}(c)}{s} + g(\eta) \right) / \theta_X \right) \Pr(S \in ds) \]
\[ = \exp \left( - \frac{g(\eta)}{\theta_X} \right) \left[ \int_1^\infty \exp \left( \frac{-U^{-1}(c)}{s\theta_X} \right) f_S(s) ds + q \exp \left( \frac{-U^{-1}(c)}{\theta_X} \right) \right] . \]  \hfill (3.3.3)

From (3.3.3), by plugging in the conditional inverse exponential pdf of \( S \), we further derive that

\[
\left[ \Pr(\frac{W_n}{n} < \xi) \exp \left( \frac{g(\eta)}{\theta_X} \right) - q \exp \left( \frac{-U^{-1}(c)}{\theta_X} \right) \right] \frac{1 - \exp(-\theta_S)}{\theta_S(1 - q)} \]
\[ = \int_1^\infty \exp \left( - \left( \frac{U^{-1}(c)}{\theta_X} + \theta_S \right) / s \right) / s^2 ds \]
\[ = \left( \frac{U^{-1}(c)}{\theta_X} + \theta_S \right)^{-1} \left[ 1 - \exp \left( - \left( \frac{U^{-1}(c)}{\theta_X} + \theta_S \right) \right) \right] , \]
which implies
\[
\frac{g(\eta)}{\theta_X} = \ln \left( \frac{l(c, \theta_X, \theta_S, q)}{\Pr(W_n/n < \xi)} \right)
\]
and thus
\[
p(\eta) = (\gamma - c) \left( \frac{l(c, \theta_X, \theta_S, q)}{\Pr(W_n/n < \xi)} \right)^{\theta_X/\theta_Y} + \xi, \quad (3.3.4)
\]
where
\[
l(c, \theta_X, \theta_S, q) = \frac{(1 - q)\theta_S}{1 - \exp(-\theta_S)} \cdot \frac{1 - \exp \left( -\left( \frac{U^{-1}(c)}{\theta_X} + \theta_S \right) \right)}{\frac{U^{-1}(c)}{\theta_X} + \theta_S} + q \cdot \exp \left( -\frac{U^{-1}(c)}{\theta_X} \right).
\]

So, if the SCR per capita \( \xi \) is defined as the 99.5 percentile of the basic own funds per capita \( W_n/n \), as required in Solvency II, then \( (3.3.4) \) provides a closed-form formula to approximate the minimum required premium for a large population.

We can further derive a closed-form formula for the risk load. Actually, by combining \( (2.3.1) \) and \( (3.3.4) \) we immediately have the following formula for \( \eta \)
\[
\eta = \left[ \left( \frac{l(c, \theta_X, \theta_S, q)}{\Pr(W_n/n < \xi)} \right)^{\theta_X/\theta_Y} + \frac{\xi}{\gamma - c} \right] / \Pr \left( v > U^{-1}(c) \right) - 1.
\]

In the following we derive an explicit formula for the survival function of \( v \).

Since \( X \) and \( Y \) are independent, \( X + Y \) follows the gamma distribution with parameters \( \alpha = 2 \) and \( \theta = \theta_X \) when \( \theta_X = \theta_Y \). If \( \theta_X \neq \theta_Y \) it is straightforward to
find the moment generating function of $X + Y$ as

$$M_{X+Y}(t) = (1 - t\theta_X)^{-1} \cdot (1 - t\theta_Y)^{-1}$$

$$= \frac{\theta_X}{\theta_X - \theta_Y} (1 - t\theta_X)^{-1} + \frac{\theta_Y}{\theta_Y - \theta_X} (1 - t\theta_Y)^{-1}$$

$$:= \omega (1 - t\theta_X)^{-1} + (1 - \omega) (1 - t\theta_Y)^{-1}.$$ 

So $X + Y$ follows a two-point mixture distribution of two exponential distributions with parameters $\theta_X$ and $\theta_Y$. The corresponding weights are $\omega$ and $1 - \omega$. In summary, if we denote by $G(\cdot)$ the cdf of $X + Y$, then

$$\overline{G}(t) = \begin{cases} 
(1 + t/\theta_X) \exp (-t/\theta_X), & \text{if } \theta_X = \theta_Y, \\
\omega \exp (-t/\theta_X) + (1 - \omega) \exp (-t/\theta_Y), & \text{otherwise}.
\end{cases}$$

Thus, when $\theta_X = \theta_Y,$

$$\Pr(v > t)$$

$$= q\overline{G}(t) + \int_1^{\infty} \overline{G}(t/s) f_S(s) \, ds$$

$$= q\overline{G}(t) + \frac{(1 - q)\theta_S}{1 - \exp(-\theta_S)} \left( \frac{t}{\theta_X} + \theta_S \right)^{-2} \times \left\{ \theta_S + \frac{2t}{\theta_X} - \exp \left( - \left( \frac{t}{\theta_X} + \theta_S \right) \right) \left[ \theta_S + \frac{2t}{\theta_X} + \frac{t}{\theta_X} \left( \theta_S + \frac{t}{\theta_X} \right) \right] \right\}.$$
and, when \( \theta_X \neq \theta_Y \),

\[
\Pr(v > t) = qG(t) + \frac{(1 - q)\theta_S}{1 - \exp(-\theta_S)} \times \left\{ \omega \left( \frac{t}{\theta_X} + \theta_S \right)^{-1} \left[ 1 - \exp \left( - \left( \frac{t}{\theta_X} + \theta_S \right) \right) \right] + (1 - \omega) \left( \frac{t}{\theta_Y} + \theta_S \right)^{-1} \left[ 1 - \exp \left( - \left( \frac{t}{\theta_Y} + \theta_S \right) \right) \right] \right\}.
\]

Based on the above derivations, we input the following values to demonstrate one numerical example. Note that all monetary amounts are in thousand dollars.

- The total cost of the treatment \( \delta = 100 \).
- The copayment \( c = 27 \), which accounts for 27\% of the total cost.
- \( \gamma = (c + \delta) / 2 = 63.5 \). The universal basic insurance and the supplementary health insurance each covers 36.5\% of the total cost.
- The severity model specifications: \( \theta_X = \theta_Y = \theta_S = 5 \) and \( q = 0.4 \).
- Threshold \( \tau = 112.3 \).
- The utility function specifications: \( a = 1.65 \), \( b = 1.6 \) and \( U(\tau) = 5 \).
- The SCR per capita \( \xi = 3 \).

We perform the calculations using R software (with R code in Appendix A) and obtain the following results.

1. The probability that an individual is diagnosed the illness is 20\%. But only 67\% of individuals diagnosed the illness would choose to use the treatment.
2. TVaR_{U(\tau)}(U(\tau)) = 72.5 and TVaR_{U(v)}(c) = 100. So both Assumptions 2.2.1 and 2.3.1 are satisfied.

3. The premium of the insurance is 5.6 thousand dollars per year and the minimum risk load required by \( \xi \) is 15\%.
Chapter 4

Concluding Remarks

4.1 Summary

Based on an insurance system consisting of government health insurance and private health insurance, I study the minimum risk load of a health insurance product under a certain regulatory capital requirement in Solvency II and design a co-payment to make sure the social cost-effectiveness of the treatment covered by the insurance product. The probability that a policyholder is diagnosed with the illness is estimated through a threshold model. In other words, the probability that a policyholder is diagnosed with the illness is the chance that the policyholder’s severity of the illness is higher than a fixed threshold. Moreover, the severity model includes three risk factors, common shock, systematic risk and idiosyncratic risk. Based on this risk model, the pure premium is estimated when the number of policyholders is large. Then, we calculate the premium of the health insurance product. Furthermore, motivated by the fact that the government has a motiva-
tion to control the minimum risk load for health insurance, I derive a method to calculate exact minimum premium and minimum risk load of the health insurance products.

4.2 Future Research

Several extensions of our work are worthy of pursuit in the future. First of all, although the copayment is able to make the medical treatment is socially cost-effective, it can’t make sure the utility gain that every single medical treatment brings to every policyholder is equal to or higher than the cost of every treatment. Future research can focus on finding methods to eliminate moral hazard further.

Secondly, it is valuable to find out the optimal payment the government should pay for every treatment. My thesis focuses on how to design and price the supplementary insurance. Future research can pay more attention to the payment of basic universal insurance.
Appendix A

R code for the numerical example

```r
# Define functions
#################################################
#### Find tau ####
G_bar <- function(t) {
  if(th_X==th_Y) return((1+t/th_X)*exp(-t/th_X)) else {
    omg <- th_X/(th_X-th_Y)
    return(omg*exp(-t/th_X)+(1-omg)*exp(-t/th_Y))
  }
}

v_surv <- function(t) {
  term1 <- q*G_bar(t)
  if(th_X==th_Y) {
    term2 <- exp(-t/th_X-th_S)
    term2 <- term2*(th_S+2*t/th_X+t/th_X*(th_S+t/th_X))
    term2 <- th_S+2*t/th_X-term2
    term2 <- term2*(1-q)*th_S/(1-exp(-th_S))/(th_S+t/th_X)^2
    return(term1+term2)
  } else {
    omg <- th_X/(th_X-th_Y)
    term2 <- omg/(t/th_X+th_S)*(1-exp(-t/th_X-th_S))
    term2 <- term2+(1-omg)/(t/th_Y+th_S)*(1-exp(-t/th_Y-th_S))
    term2 <- term2*(1-q)*th_S/(1-exp(-th_S))
    return(term1+term2)
  }
}
```
Appendix A: R code for the numerical example

```r
threshold<- function(p) {
  f1 <- function(t) (v_surv(t)-p)^2
  return(optim(1,f1,method="L-BFGS-B",lower=0,upper=Inf)$par)
}

#### Find copayment ####
tvar_uti <- function(t) {
  f2 <- function(s) v_surv(U_inv(s))
  result <- integrate(f2,t,Inf)$value
  return(result/f2(t)+t)
}
copay <- function(dlta) {
  if(tvar_uti(U_tau)<dlta){
    f3 <- function(t) tvar_uti(t)-dlta
    return(uniroot(f3,c(U_tau,dlta),tol=0.0001)$root)
  } else{
    return(U_tau)
  }
}

#### premium as a function of SCR per capita ####
l <- function(co) {
  term1 <- (1-q)*th_S/(1-exp(-th_S))*(1-exp(-U_inv(co)/th_X-th_S))/(U_inv(co)/th_X+th_S)
  term2 <- q*exp(-U_inv(co)/th_X)
  return(term1+term2)
}

prem_req <- function(xi,perc) {
  (ins-cpay)*(l(cpay)/perc)^(th_X/th_Y)+xi
}

#### risk load as a function of SCR per capita ####
riskload_req <- function(xi,perc) {
  result <- prem_req(xi,perc)/(ins-cpay)/v_surv(U_inv(cpay))-1
  return(result)
}
```

########################################################################
Appendix A: R code for the numerical example

```r
### numerical input #########################
th_X <- 5 ## X follows exponential
th_Y <- 5 ## Y follows exponential
th_S <- 5 ## S follows inverse exponential tail
q <- 0.4 ## Pr(S=1)
prob_dis <- 0.2 ## the prob of being diagnosed the illness
## the severity threshold of being diagnosed the illness
tau <- threshold(prob_dis)

####### the utility curve depends on tau ###############
ttcost <- 100 ## in thousands
a <- 1.65 ## utility shape parameter a>1
b <- 1.6 ## utility scale parameter b>0
U_tau <- 5 ## utility at tau, less than ttcost
U <- function(t) U_tau+(t-tau)^(1/a)*b
U_inv <- function(u) ((u-U_tau)/b)^a+tau

#########################################
#### testing ############################
cpay<-copay(ttcost)
v_surv(U_inv(cpay));cpay;tvar_uti(cpay)

SCR <- 3
percent <- 0.995
ins <- (cpay+ttcost)/2 ## gamma should be between copay and delta
prem_req(SCR,percent)
riskload_req(SCR,percent)
```
Bibliography


cinogenicity of perfluorooctanoic acid, tetrafluoroethylene, dichloromethane, 1, 2-dichloropropane, and 1, 3-propane sultone. *Lancet Oncol*, 15, 924-925.


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