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# Chapter 5

## Summary

This thesis presents work on the intersection of two fields: insurance and health economics. First, I study asymmetric information in insurance contracts. A typical example of asymmetric information is adverse selection, which occurs when high risk individuals are more likely to self-select into insurance. Another example is moral hazard - if being insured changes individual's behaviour, which may lead to higher accident rates. It is well documented that asymmetric information in insurance markets can lead to nonoptimal market outcomes or even market failures. Therefore, it needs to be taken into account when contracts are designed and when insurance markets are designed and/or regulated.

In Chapter 2, I test for moral hazard among firms that have insured themselves against sickness absenteeism. This moral hazard is non-standard because it arises on the employer's side: due to the costs of monitoring of sick workers. If most of the worker's sick pay is covered by the insurance company, which is true for firms insured with high coverage, the monitoring costs might be higher than the firm's expected benefits. This creates incentives for firms not to monitor, which from the insurer's point of view results in moral hazard reflected in longer than needed sickness spells. In this chapter, I present an empirical framework that allows me to identify the effect of a change in the monitoring policy of the insurance company on the duration of sickness spells reported by insured firms. Using a data from a main Dutch insurance company, I show that sickness insurance contracts reduce firms' incentives to monitor sick workers and result in a classical moral hazard problem. Moreover, I present a model which explains why smaller firms buy more comprehensive insurance coverage.

Moral hazard and adverse selection can sometimes be concealed by the existence of other effects that can influence the observed correlation between claims and coverage. In Chapter

3, I try to incorporate an example of such an effect: learning about one's own level of risk in the context of car insurance, into a typical theoretical framework that models an agent's decision on insurance coverage. The common belief in the existing literature is that switching contracts is a result of asymmetric learning about the insuree's type. I show that, contrary to this hypothesis, changes of contracts can also be caused by nonproportional pricing or asymmetric updating between the insurer and the insuree. I prove that, when the price of insurance is proportional to the estimated accident rate of an insuree, when agents are characterized by a CARA utility function and when an insuree and an insurer learn symmetrically and use the same estimate of the agent's accident risk, then the insuree's decision on contract choice depends only on the level of his risk aversion and not on the estimated level of his accident risk. I test the predictions of this baseline model with data on car insurance contracts and show that the null hypothesis of no incentives for contract changes must be rejected.

The second focus of the thesis is health economics, and, in particular, testing the hypothesis of fetal origins. The hypothesis states that adverse nutritional conditions in utero or early in life might result in inadequate development of vital organs in a human body. As a consequence, this can lead to increased risk of chronic diseases at old ages. Testing this hypothesis is difficult, because it requires linking data on early life events with long-term outcomes. Moreover, there are often confounding factors influencing later life outcomes. Unobserved factors at the individual and family level, like parental poverty or biological factors, may jointly affect conditions early in life and later life outcomes and this may hamper the assessment of causal relationships. Moreover, early life exposure to adverse conditions can lead to increased mortality risk at earlier ages, which can result in selection that may mask long-run effects.

In Chapter 4, based on Lindeboom et. al 2014, I test the hypothesis of fetal origins using unique data on birth and death certificates of people born around the time of the Dutch Potato Famine (1846-1847) in Zeeland, The Netherlands. The famine provides exogenous variation on the nutritional status of those exposed to the famine. The sibling information allows us to control for other usually unobserved environmental factors at the family level that may affect later life health. For instance, family specific biological or socio-economic factors may mitigate or enforce the impact of nutritional shocks early in life and failing to control for this may bias estimates of the effect of the nutritional shock. We use a unique historical dataset that follows siblings in a family from the time of birth until death. This dataset is merged with data on regional food prices and calories available per capita in

the nineteenth century in the Netherlands. The calories data allow us to assess effects of the diet composition (proteins and proteins from animals intake) in utero and early life on longevity. We show that conditions during both utero and early in life (ages 1-5) are important for mortality patterns at younger ages (1-10) and at older ages (50-60) and these results remain significant if we correct for unobserved family specific effects. We also find that children born in families of unskilled farm workers and in families with low social status were particularly strongly affected by the famine. A further analysis of the family fixed effects reveals large mortality differences between families. Moreover, the impact of a nutritional shock also differs per family type. The effect of a nutritional shock is quantitatively large for families with higher family fixed effects (families that face high mortality rates in general) and negligible for families with lower fixed effects. Thus, nutritional shocks early in life increase inequality in mortality and longevity at later ages.