Essays on Human Capital Investments: Pharmaceuticals and Education

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Abstract

This thesis consists of four essays on individual health behaviour and investment in higher education. Three of them consider different aspects of pharmaceutical utilization and aim at explaining some of the observed individual variation in access and adherence to treatment regimens. The fourth explores the potential link from health in young adulthood to subsequent university education.

The first essay develops a dynamic model of the individual's pharmaceutical drug treatment decisions, making the distinction between instantaneous utility effects and dynamic long-term health investment effects. The model provides an economic tool for understanding individual pharmaceutical-treatment decisions.

The second essay uses this model and examines whether people with higher treatment benefits are more adherent to long-term treatments for cardiovascular diseases and mental illness than people with lower benefits. The empirical results show a positive relationship between feasible treatment benefit and adherence to cardiovascular treatments and antidepressants. This finding is consistent with the model suggesting that adherence is an informed and rational decision reflecting treatment benefits and costs.

The third essay relates to inequalities in health care access by identifying disparities in pharmaceutical utilization between natives and immigrants in Sweden. Using the national pharmacy register and survey information on socioeconomic characteristics and individual health, we find differences in the pattern of pharmaceutical utilization between immigrants and natives. As immigrants are less likely than natives to access standard preventive pharmaceuticals for cardiovascular diseases, this may relate to and reinforce the observed health differences between the groups.

The fourth essay follows individuals, who were diagnosed with type 1 diabetes onset at age 17 to 20, in order to ascertain whether the onset affects their chances of a university education. The most important result is that type 1 diabetes onset among women in young adulthood exerts negative influences on both university education and motherhood. Comparisons of women with university education show that type 1 diabetes is negatively associated with having children, suggesting that type 1 diabetes increases the trade-off between these life goals.

Key words
Pharmaceutical utilization; Adherence; Human capital; Grossman model; Health; Education; Diabetes; Non-monotonic health investments; Steady-state and stable equilibria

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To my mother

Acknowledgments

The ending of a process, which has occupied substantial amounts of time and effort over years, frees up time for new objectives. The last time I was in such a situation, I was finalizing my pharmacy degree and preparing for the labour market transit. The acts of others affect us and we may move in unexpected directions. The frank reply "most of our employees hold a PhD" made me re-evaluate the academic track and later brought me to the unanticipated economic path. I am utterly grateful to the wonderful people who inspired and supported me in reaching my long-term goal of finalizing this thesis.

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Stockholm, October 2014

Sofie Gustafsson

Contents

Chapter 1

Introduction

The distribution and level of health and education relate to the economy of the nation by influencing, for instance, healthcare demand, labour market outcomes and economic growth. Moving from the population to the individual level, good health and education are important assets increasing the capacity to achieve goals in life. To provide equal opportunities for all residents, the Swedish welfare state heavily subsidizes healthcare and provides free education at all levels including university level. Yet, even with a generous welfare system, some groups in society are less likely than others to access adequate health care or attain higher education.

This thesis aims at explaining some of the individual variation in pharmaceutical utilization and university education in Sweden. This introduction briefly presents the conceptual framework of interest, the demand for health model and human capital theory, which make up the foundation in the essays in this thesis. Essay I (chapter 2) modifies the demand for health model to provide a tool for analysing pharmaceutical utilization behaviour. Essay II (chapter 3) tests this tool by empirically estimating whether people with higher treatment benefits are more adherent to pharmaceutical prescriptions than people with lower treatment benefits. Essay III (chapter 4) relates to the question of why there are inequalities in health care access by identifying disparities in pharmaceutical utilization between natives and immigrants in Sweden. Essay IV (chapter 5) focuses on educational human capital and explores whether the sudden onset of type 1 diabetes in young adults affects their chances of a university education.

1.1 Health and education in a human capital model

From an economic point of view, undergoing pharmaceutical treatment or taking on a university education may be regarded as human capital investment raising lifetime utility prospects. Skills and knowledge acquired in school enhance our capacity, and affect our productivity at work or in other areas we engage in. Health capital differs from ordinary human capital, because health affects the amount of time we can freely allocate across activities. This view is conceptualised in

the demand for health model (Grossman, 1972a; Grossman, 1972b), which has emerged as a standard economic model for analysing individual health-related-behaviour. The framework, which builds on the human capital model (Becker, 1962), has been extended to include, for instance, uncertainty (Dardanoni and Wagstaff, 1987, 1990; Selden, 1993; Chang, 1996; Liljas, 1998, 2000), social capital (Bolin et al., 2003), healthy and unhealthy consumption (Forster, 2001), the family rather than the individual as health producer (Jacobson, 2000; Bolin et al., 2001, 2002b), the employer as health producer (Bolin et al., 2002c) and, recently, behaviours with non-monotonic health returns (Bolin and Lindgren, 2012).

In short, the demand for health model views health as a capital good, which individuals are equipped with from birth. The amount of start-up capital varies across individuals and depends on factors such as genetics, chances and external causes. With time, health capital depreciates, and at an increasing rate. Reaching the lower critical health capital bound implies the end of an individual's life. The individual may take actions to slow down this process, and thereby extend the amount of time with good health, by making health investments. Health investments take various forms, for instance, a healthy lifestyle and the use of pharmaceutical treatments. All health investments requires time effort, while physical-activity-based health investments are time intensive pharmaceutical-based health investments require substantially less time effort. As time and other resources are limited, investments in health compete with other demands in life. The individual will therefore engage in activities at the intensity level that makes the best out of life according to own values. Consequently, better health may be traded off for other desires. To exemplify, even if too little sleep may lead to serious health concerns, people may sleep less than needed to free up time to work or enjoy the company of family and friends. For similar reasons the individual may, despite adverse long-term health outcomes, resort to pharmaceutical treatment regimens to avoid side-effects immediately diminishing quality-of-life. Essay I and essay II explore the relationship between pharmaceutical utilization and treatment effects on long-term health and short-term quality of life. Another reason that may cause people to refrain from adequate pharmaceutical treatment is the high cost in terms of the time and effort required for consulting a physician. For immigrants, unfamiliarity with the health care system or the native language may imply that this requirement is too high. If so, differences relating to non-monetary costs for prescriptions, which follow from correct diagnoses, may contribute to differences in pharmaceutical utilization between immigrants and natives. Essay III identifies differences in the pharmaceutical utilization pattern between natives and immigrants in Sweden, and seeks to categorize the underlying causes.

Regarding investment in education, people may refrain from taking on a university education because of unwillingness to sacrifice competing desires, such as current labour market earning. Thus, the individual enrols in university education only if he or she considers that the benefits outweigh those of the alternative options. The availability of new information that changes the set constraints may cause young adults to re-evaluate their life goals, which in turn may modify the university decision. Essay IV examines whether the sudden onset of type 1 diabetes in young adulthood has consequences for subsequent university education.

1.2 Summary of essay I

Advice and guidelines exist for a wide range of health-related behaviours. These may or may not be adhered to, however. Empirical observations clearly suggest that guidelines to promote a healthy lifestyle are frequently not followed, and the same is true for prescribed pharmaceutical treatments. As a consequence, treatable health conditions may receive too little or too much treatment, resulting in potential health levels not being realized. It is obvious that adherence to a prescribed pharmaceutical treatment varies. For instance, common sense suggests that patients are likely to adhere to suggested pharmaceutical treatments in case of life-threatening conditions – most cancer patients will follow what their oncologist recommends (disregarding any payment issues). On the other hand, not all patients will follow a recommendation to start using anti-depressants, and those patients who do follow that recommendation may not follow the pharmaceutical prescription to the letter. More precisely, in cases where there is room for patient autonomy, he or she may make own treatment decisions based on his or her assessment of effects and side effects. This essay develops a dynamic model of the individual's drug treatment decisions, making the distinction between instantaneous utility effects and dynamic long-term health investment effects. Physician decisions on treatment recommendation are exogenous. The model provides an economic tool for understanding individual pharmaceutical-treatment decisions.

The analysis illustrates, for instance, that public policies which uniformly subsidise a higher share of the pharmaceutical costs have either positive or negative effects on public health, depending on the type of pharmaceutical. Therefore, policy makers could modify the pharmaceutical reimbursement system, so that the pharmaceutical out-of-pocket cost steers individual decisions toward treatment intensities more beneficial for long-term health. Population health will increase if pharmaceutical treatments with long-term health objectives, particularly

when involving adverse quality of life effects, are subsidised more, and if treatments generating short-term utility by forgoing long-term health objectives are subsidised less or taxed.

1.3 Summary of essay II

This essay considers patient adherence to prescriptions standardly prescribed for long-term treatment of cardiovascular diseases and mental health concerns. To explore the link between treatment benefits and adherence, I use the Swedish Prescribed Pharmacy Register (SPPR) and detailed individual information on health, demographical and socioeconomic characteristics. The data offers a unique opportunity to study long-term adherence in the Swedish setting which, due to the cost-sharing subsidy rules, is suitable for exploring non-financial adherence determinants.

The extensive literature on adherence barriers provides mixed results. The homogeneity in measuring adherence, and the absence of conceptual frameworks that may integrate the results from studies across patient groups and pharmaceuticals (e.g., identified barriers are inconsistently associated with adherence across studies) make it difficult to draw firm conclusions for policy recommendations (Vermeire et al., 2001; Gellad et al., 2009; WHO, 2010). Despite the shortcomings, the literature identifies cost-sharing, regimen complexities, medication beliefs and depression as potential fruitful targets for improving adherence (RAND, 2009).

This essay focuses on a less studied link between achievable treatment benefits and adherence to pharmaceuticals standardly prescribed for long-term treatment of prevalent chronic diseases. This link is important because a positive association, i.e. people are more adherent when treatment benefits are higher, may suggest that adherence follows from informed and rational decisions reflecting treatment benefits and costs.

The empirical results show a significant and sizable positive relationship between adherence and the achievable treatment benefit. The over-all results are robust across the analysed pharmaceutical classes and for two different population groups. That people with higher treatment benefits are more adherent than people with lower health returns is an important finding. For example, such knowledge is useful for health professionals when drawing up treatment guidelines.

1.4. Summary of essay III

Immigrants in Sweden have poorer health, e.g., self-rated health (Lindström, Sundquist, and Östergren 2001), cardiovascular illness (Gadd et al., 2005), psychiatric illness (Johansson et al.

1997; Ferrada-Noli, 1997, Bayard-Burfield, Sundquist, and Johansson, 2001), and a higher overall mortality risk (Sundquist and Johansson 1997), compared to natives. Some of the health differences across these two groups seem to relate to the pattern for overall health care utilization (Westin et al. 2004; Wamala et al. 2007) and prescriptions (Nordin, Dackehag, and Gerdtham, 2013; Sundquist 1993). This essay considers pharmaceutical utilization differences between immigrants and natives in Sweden.

Despite the fact that immigrants constituted 15% of the Swedish population in 2011 (Statistics Sweden, 2011) and that the health of immigrants has substantial impact on general public health and healthcare expenditures, there are few analyses exploring differences in pharmaceutical utilization between immigrants and natives. For this purpose, we use the Swedish Prescribed Drug Register (SPDR) and the Swedish Survey of Living Conditions (ULF) to explore differences in dispensed pharmaceuticals between immigrants and natives. The detailed individual-level data also enables us to disentangle differences in utilization relating to disparities in health and socioeconomic characteristics from factors relating to immigration characteristics.

The results show that immigrants and natives have different access to several of the 20 most dispensed pharmaceutical subgroups. When focusing on prescriptions listed as first-line treatments by evidence-based treatment guidelines (e.g. Janus 2006; Läkemedelsrådet 2006) for prevalent chronic conditions, a uniform pattern emerges – immigrants are less likely than natives to access thiazide-diuretics (C03A), ACE inhibitors (C09A) and adrenergic inhalators (N02B). These pharmaceuticals make up the foundation in the prevention and treatment of cardiovascular-related diseases (high blood pressure, heart failure and kidney diseases) and asthmatic diseases. This may suggest that immigrants are less likely than natives to have adequate preventive treatment. As cardiovascular-related morbidity and mortality are leading public health concerns, and adequate pharmaceutical treatment substantially reduces such morbidity and mortality (WHO, 2012), effective intervention targeting immigrants with unmet medical needs may increase their health.

1.5 Summary of essay IV

Growing evidence shows that health in early life and childhood is important for adult outcomes such as academic achievements, but data limitations have made it difficult to study the importance of health in young adulthood. This essay investigates the interrelationships of young-adulthood health, university education and family formation, and analyses the link between health in young adulthood and university education by using the Econ-DISS database, which includes

detailed individual data on individuals who had type 1 diabetes onset in age 17 to 20 and matched population controls.

This link is interesting because children, when they reach young adulthood, gain wider responsibility for their own health behaviour and academic achievements compared to when they were younger, and the role of parents becomes less governing and more advisory. Therefore, the link between young-adulthood health and university achievements reflects the young adult's own decision making process, while the link between childhood health and education mirrors, to a higher extent, the level of parental involvement in their children's education through e.g. homework.

Comparing individuals with and without type 1 diabetes illustrates how an unexpected health shock may affect subsequent university education. Type 1 diabetes typically occurs rapidly without prior symptoms, the onset mimicking a before and after treatment setup. Focusing on onset in the age group 17 to 20, which is when young adults decide upon university enrolment, we minimize the influences on childhood academic achievements that also affect university enrolment.

The most important result is that type 1 diabetes onset among women in young adulthood exerts negative influences on both university education and becoming a parent. As the negative influences persist at age 35, the results are permanent drops rather than delays. Comparisons of women with university education show that type 1 diabetes is negatively associated with motherhood, suggesting that type 1 diabetes obstructs the achievement of both university education and motherhood, and that the trade-off between these life goals increases. In terms of redirecting life goals after onset, socioeconomic background is important. The group level analysis shows that young adult women with a high or low socioeconomic background choose motherhood over university education, while women with a middle socioeconomic background remain childless and prioritize university education. For men, we find no association between type 1 diabetes onset in young adulthood and subsequent university education.

Taken together, these results suggest that type 1 diabetes intensifies the conflict between motherhood and university education, and that socioeconomic background affects women's response to such conflict. These findings underline the need of further research on the complexity between type 1 diabetes onset in young-adulthood and subsequent university education. As family formation is important and men and women face different constrains (cultural and biological) when forming a family, policy makers should consider that (1) men and women respond differently to diabetes onset, (2) diabetes intensifies the conflict between

motherhood and university education and (3) socioeconomic background matters for the choice between motherhood and university education.

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Chapter 2

Pharmaceutical utilization in a demand-for-health framework – The trade-off between instantaneous quality-of-life and long-term health

With Kristian Bolin[a,b]

2.1 Introduction

The broad range of advice on how to behave to promote good health, from lifestyle advice to prescribed pharmaceutical treatments, may be adhered to more or less rigorously. There may be various reasons for this; for instance, uncertainty, lack of knowledge and/or conflict of interests. We focus on the implications for health and health-related good-health behaviour not being the individual's sole concern when making decisions on the allocation of resources. More specifically, we consider behaviours that influence both health and quality of life, making the distinction between health as a capital good and instantaneous utility or quality of life. For instance, a pharmaceutical treatment may have instantaneous quality-of-life effects by relieving symptoms, and at the same time influence health in the longer perspective by producing investments in health. Moreover, several health behaviours seem to be associated with a physiologically determined, individual-optimal level of activity, implying that activity levels below or above that level would result in less than the maximum attainable effect. The pharmaceutical example offers a plausible illustration of this non-monotonicity property: there is a maximum instantaneous quality-of-life effect that is attained for a specific treatment intensity, which may be smaller or larger than the intensity that maximizes the amount of health investment (Bolin and Lindgren,

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2012). We develop an economic-theoretical model along those lines, using pharmaceutical treatment as an illustration.

The purposes of following a pharmaceutical treatment regimen are to improve, or retain, health and/or to improve quality of life by curing disease and/or alleviating disease-related symptoms. Whenever these objectives cannot be perfectly accommodated simultaneously, the optimal course of action will involve a balance between the two. Thus, a prescribed pharmaceutical treatment, which is designed to maximize the (expected) positive health effects, will only be perfectly adhered to in the special case when this objective is not in conflict with the quality-of-life objective, and vice versa. Radical treatment examples include strong pain killers (e.g., morphine and methadone) which rapidly alleviate pain, but may involve adverse long-term health effects (e.g., respiratory depression), and chemotherapy, which has undesirable short-term quality-of-life effects (nausea, fatigue etc), but may restore health in a longer perspective. Certainly, in some cases short-term quality-of-life effects harmonize with long-term health effects, in which case there is no conflict between the two objectives. For instance, this may be the case for treatment situations that involve antihistamines alleviating allergy symptoms and preventing further allergic reactions.

The theoretical model outlined above facilitates economic-theoretical analysis of health-related behaviours in general, and the analysis of pharmaceutical-treatment adherence in particular. The importance of this is highlighted by the economic costs induced by alterable health-related lifestyle choices and pharmaceutical misuse. In both cases, empirical studies have found the costs to be substantial, even though, in the latter case, most studies do not distinguish between costs that arise due to side effects that occur, despite perfect adherence, and costs that can be attributed to adverse effects that arise due to less than perfect adherence (Cawley and Ruhm; Wester et al., 2008; Lazarou et al., 1998; Johnson and Bootman, 1995).

The notion of health behaviours producing non-monotonic effects follows Bolin and Lindgren (2012). Bolin and Lindgren (2012) argue that the intentions behind much health-related behaviour are twofold: to gain direct consumption utility and to improve health (or to decrease the risk of illness). Such behaviours include physical exercise, certain consumption and composition of food, alcohol consumption and, as a matter of fact, any recreational activity (art, literature, music, etc). We extend their idea by incorporating two *distinct* activity levels that maximize instantaneous quality of life and gross health investments, respectively.

The following section develops a dynamic human-capital model of health investments, incorporating the notion of an activity – exemplified by pharmaceutical treatment – yielding non-

monotonic effects (1) on the health stock, and (2) on instantaneous utility (quality of life).[1,2] The model is an extension of the demand-for-health model (Grossman, 1972),[3] and adopts its following basic features: (1) the individual demands health for its effect on the amount of healthy time (the investments motive – the individual has no consumption motive for holding health), (2) the demand for investments in health is derived from the more fundamental demand for health, (3) investments in health are produced by the individual, and (4) the stock of health depreciates at each point in time. Our model incorporates the additional complexities of health behaviours having (1) non-monotonic effects and (2) both instantaneous and investment effects. The model uses pharmaceutical treatment as a benchmark, but its applicability reaches beyond that particular situation.

The structure of the rest of the paper is as follows. Section two comprises the general structure of the model, including the individual's control problem. Section three explores the general properties of the model (steady-states, stability and dynamics). Conclusions and a discussion bring the paper to an end.

2.2 The model

General structure

We consider a theoretical model of the demand for pharmaceutical treatment, taking both short-term and long-term effects of that treatment into account. The short-term effect is the improvement in quality of life produced by the chosen treatment intensity, while the long-term effect is the influence on the health stock. Thus, we distinguish between quality of life on the one hand, and health on the other. Further, both effects are non-monotonic and single-peaked. Thus, in our model, pharmaceutical utilization has two objectives: (1) to improve instantaneous quality of life, and (2) to enhance future health $(H(t))$. Following in the tradition of the human-capital approach to health (Grossman, 1972a, b), the demand for pharmaceutical treatment is derived from the underlying fundamental demand for health and, in our case, from the instantaneous quality-of-life effect. More specifically, we consider a continuous-time demand-for-health model,

[1] The human-capital approach to health suggests that health behaviours are dynamic. Thus, a theoretical analysis needs to take changes in any such behaviour, and the resulting health outcomes, over time into account. The appropriate way of achieving this is by specifying a dynamic model either in a discrete-time multi-period or a continuous-time framework.

[2] The two concepts *instantaneous utility* and *quality of life* are used interchangeably.

[3] Since its introduction, the demand-for-health model has been extended in various ways. For instance, to incorporate uncertainty (Dardanoni and Wagstaff, 1987, 1990; Selden, 1993; Chang, 1996; Liljas, 1998, 2000), the family as producer of health (Jacobson, 2000; Bolin et al., 2001, 2002b), the employer as producer of health (Bolin et al., 2002c), social capital (Bolin et al., 2003), healthy and unhealthy consumption (Forster, 2001), decreasing returns to scale in the production of health investment (Ehrlich and Chuma, 1990; Galama, 2011) and non-monotonic health behaviours (Bolin and Lindgren, 2012).

in which the individual chooses an optimal pharmaceutical-utilization (treatment intensity) time path, $a(t) \geq 0 \; \forall t$, taking both instantaneous quality-of-life and investment motives into account.[4]

Treatment intensities

Pharmaceutical treatments are prescribed in order to restore and maintain health and/or quality of life. For simplicity, we assume that for each treatable health condition there is a unique pharmaceutical treatment associated, which is characterised by its influence on symptoms and capacity to restore health. We are concerned with situations in which (1) physicians prescribe pharmaceuticals according to this association only and (2) pharmaceuticals are only available as prescription drugs. In this scenario it seems reasonable to make the simplifying assumption that, for each pharmaceutical treatment, the treatment intensities that maximize the quality-of-life and health investment effects are independent of health. Thus, for each treatment the effects on health and quality of life are determined solely by the treatment intensity.[5] Differences between health conditions that demand pharmaceutical treatment are reflected by differing highest attainable treatment effects.

Formally, a particular pharmaceutical is characterised by its physiologically optimal treatment intensities with respect to instantaneous utility (quality of life) and health investments. Instantaneous utility is provided according to $\theta^s(a(t)) \equiv TE^s - \varphi^s \cdot (a(t) - a^s)^2$, where TE^s is the maximum instantaneous utility that can be achieved by that particular treatment, which occurs at $a(t) = a^s \geq 0$ (a^s is the short-run physiologically optimal treatment intensity). The parameter φ^s captures the rate at which deviating from the short-term physiologically optimal level of treatment reduces quality of life ($\varphi^s > 0$). Analogously, the chosen treatment intensity produces gross health investments according to $\theta^l(a(t)) \equiv TE^l - \varphi^l \cdot (a(t) - a^l)^2$, where TE^l is the maximum investment attainable at each t, and the parameter φ^l captures the rate at which the amount of health investments diminishes when deviating from the long-run physiologically optimal level, $a^l \geq 0$ ($\varphi^l > 0$). Further, we assume that no treatment, $a(t) = 0$, yields a zero effect on both instantaneous utility and health investments. Thus, $\theta^i(0) = 0$ ($i = s, l$), which means that $TE^i = \varphi^i \cdot \left(a^i\right)^2$. These specifications comprise both treatments with conflicting long- and short-term objectives as well as therapies which are beneficial both in the short and in the long run. The former type of treatments includes, for instance, cancer

[4] The two concepts *treatment intensity* and *pharmaceutical utilization* are used interchangeably

[5] The implications for health and health investments in health influencing the capacity of transforming time and resources into health capital has been analysed by Bolin and Lindgren (2014).

treatment with chemotherapy and pain treatment with morphine, which may have long-term adverse health effects. In the case of chemotherapy, side-effects adversely influence instantaneous quality of life, i.e., $\theta^s(a(t) > 0) < 0$. In the long run, however, chemotherapy constitutes an investment in health, i.e., $\theta^l(a(t) > 0) > 0$. The morphine example is directly analogous. The second type of treatments encompasses a less pronounced conflict between the two objectives. For instance, stomach-ulcer treatment with proton-pump inhibitors improves instantaneous quality of life, by reducing stomach pain, and constitutes an investment in health, by curing and preventing the underlying disease. Notice, however, that in the case when treatment has beneficial effects for instantaneous quality-of-life and produces health investments there may still be a range of conflict between the two objectives. Figure 1 illustrates the case in which there is no range of treatment intensities for which short- and long-term effects are simultaneously strictly positive (a chemotherapy treatment, for instance). Figure 2 illustrates the case in which there is a range of treatment intensities which produce both short- and long-term benefits (for instance, a treatment using a proton-pump inhibitor for ulcer treatment).

Figure 1. Illustration of the utility and the health-investment effects in the chemotherapy case. The specific choices of the parameters reflect a specific treatment situation. The dashed curve illustrates the health-investment effects, while the dotted curve shows the instantaneous-utility effect. The following specifications are used: $\varphi l = \varphi s = 1; al = 2; as = 0;\ TE^l = 4$ and $TE^s = 0$.

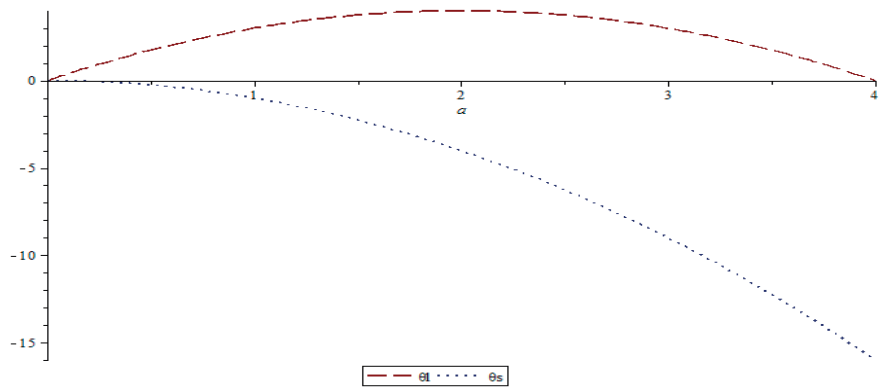
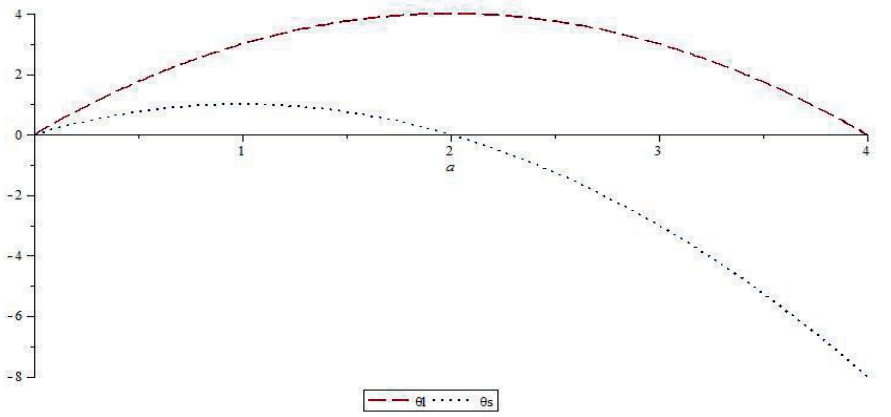


Figure 2. Illustration of the utility and the health-investment effects in the proton-pump case. The specific choices of the parameters reflect a specific treatment situation. The dashed curve illustrates the health-investment effects, while the dotted curve shows the instantaneous-utility effect. The following specifications are used: $\varphi l = \varphi s = 1; al = 2; as = 1;\ TE^l = 4$ and $TE^s = 1$



Preferences and the dynamics of health capital

The individual derives utility from consumption of a market good and from the quality of life produced by the chosen treatment intensity, $a(t)$. Health, $(H(t))$, is purely an investment capital good that determines market income and, hence, consumption and treatment opportunities. Disposable income, $y(t)$, is an increasing and strictly concave function of health $(y' > 0; y'' < 0)$, the price of consumption is normalised to 1, and the marginal cost of pharmaceutical treatment is constant and equal to π. Thus, consumption, $c(t)$, equals $y(H(t)) - \pi \cdot a(t)$. For simplicity, we assume that preferences can be represented by the following quasi-linear utility function, $U(c(t), a(t))$:[6]

$$U(c(t), a(t)) = y(H(t)) - \pi \cdot a(t) + \theta^s(a(t)). \quad (1)$$

Health investments produced by pharmaceutical treatment are partially offset by natural depreciation – at rate $\delta\ (0 < \delta < 1)$ – of the existing stock of health capital. For tractability of dynamic analysis we consider a model in which the rate of depreciation is time independent.[7] Thus, the motion of the health stock is given by:

$$\frac{dH(t)}{dt} = \theta^l(a(t)) - \delta \cdot H(t). \quad (2)$$

[6] Throughout the paper, a subscript indicates a partial derivative. The derivatives of single-variable functions follow Lagrange's notation and use ' for the first and " for the second derivative. The time-derivative operator is expressed as $\frac{d}{dt}$.

[7] We analyse time-paths and stability of equilibrium. This is in contrast to Grossman (1972), Muurinen (1982), Wagstaff (1986), Liljas (1998), Jacobson (2000), and Bolin et al. (2001), who all examined models with time-dependent rates of depreciation.

The individual's control problem

The individual faces the intertemporal problem of controlling the treatment-intensity time path in order to maximize his or her total utility over the planning period. This means that in order to find the optimal allocation of resources, the individual has to find the optimal dynamic balance between instantaneous quality of life and the size of the health stock. We assume that the individual has a fixed planning horizon, $t = T$, and, hence, if future utility is discounted at rate ρ, the individual acts according to the following (using $c(t) = y(H(t)) - \pi \cdot a(t)$):[8]

$$\max_{H,a} \int_0^T e^{-\rho \cdot t} \cdot U(y(H(t)) - \pi \cdot a(t), a(t))\, dt,$$

subject to: $\frac{dH(t)}{dt} = \theta^I(a(t)) - \delta \cdot H(t)$, $H_0 = \underline{H}$ $(\geq H_{min})$, and a transversality condition (H_{min} is the smallest permissible level of health). We focus on the fixed time horizon problem including a minimum target health-capital level, $\overline{H}$; formally the transversality condition is: $\lambda(T) \cdot \left(H(T) - \overline{H}\right) = 0$ ($H(T) \geq \overline{H}$, target health).[9] Regarding the problem at hand, these conditions describe the situation in which a specific treatment time and a minimum health level at the end of the treatment are decided at the outset of the treatment.

2.3 Optimality

Conditions for optimal treatment intensity and health time path

The maximum principle gives necessary and sufficient conditions for the (unique) optimal control of $a(t)$, since the Hamilton function is jointly concave in $(a(t), H(t))$ (this is straightforward to demonstrate; see appendix A). In what follows, $(a(t)^*, H(t)^*)$ denotes a path satisfying these conditions. The current-value Hamilton function for the maximisation problem is:

$$\mathcal{H}(t) = U(y(H(t)) - \pi \cdot a(t), a(t)) + \lambda(t) \cdot (\theta^I(a(t)) - \delta \cdot H(t)), \quad (3)$$

The maximum principle yields the following equations of motion for the stock of health:

$$\frac{d\lambda}{dt} = -\frac{\partial \mathcal{H}}{\partial H} + \rho \cdot \lambda^*(t) = -y'(H(t)^*) + (\delta + \rho) \cdot \lambda(t)^* \quad (4)$$

[8] The individual's life-time optimisation problem is formulated as a vertical time line problem by, for instance, Bolin et al (2001; 2002b, c), which means that the terminal time is fixed, but the terminal state is free (Chiang, 1992, p.182.

[9] This is often referred to as a truncated vertical terminal line problem. Life-long treatments could also be approached by a horizontal terminal line formulation, in which case the transversality condition is $\mathcal{H}(T) = 0$, and $H(T)$ fixed. ($\mathcal{H}(T)$ is the value of the Hamiltonian function at $t = T$).

The optimal control of the treatment intensity, $a(t)$, is given by the following first-order condition:

$$\left.\frac{\partial \mathcal{H}}{\partial a}\right|_{a(t)=a^*} = -\pi - 2 \cdot \varphi^s \cdot (a(t)^* - a^s) + \lambda(t)^* \cdot \left(-2 \cdot \varphi^l \cdot (a(t)^* - a^l)\right) = 0. \qquad (5)$$

The optimal choice of treatment intensity

First, notice that $\pi < 2 \cdot \varphi^s \cdot a^s$ is a sufficient condition for $a(t)^* > 0 \; t \in [0, T]$. This follows from the fact that $\lambda(t)^* > 0$ for $t \in [0, T)$,[10] which implies that $\left.\frac{\partial \mathcal{H}}{\partial a}\right|_{a(t)=0} > 0$. Similarly, if $\left.\frac{\partial \mathcal{H}}{\partial a}\right|_{a(t)=\frac{y(H^*(t))}{\pi}} < 0$, the market income will be divided between consumption and treatment, i.e., $a(t)^* < \frac{y(H(t))}{\pi}$. We will refer to this as an interior allocation. *Second*, equation (5) suggests that the individual chooses the intensity of a pharmaceutical treatment by balancing quality of life anddemand for health. The relationship between a^s and a^l determines the range of potentially optimal treatment intensities. This is summarized in Claim 1:

Claim 1: an interior treatment intensity, $a(t)^*$, is chosen as follows for $t \in [0, T]$:

(*i*) when $\pi = 0$, and $a^l \neq a^s$, then $a(t)^* \in (\min(a^s, a^l), \max(a^s, a^l))$;

(*ii*) when $\pi > 0$ and $a^l = a^s = a$, then $a(t)^* \in [0, a)$;

(*iii*) when $\pi > 0$ and $a^l > a^s$, then $a(t)^* \in [0, a^l)$;

(*iv*) when $\pi > 0$ and $a^s > a^l$, then $a(t)^* \in [0, a^s)$;

(*v*) when $0 < \pi \leq 2 \cdot \varphi^s \cdot (a^s - a^l)$ and $a^s > a^l$, then $a(t)^* \in [a^l, a^s)$;

Proof: see appendix A.

[10] See lemma 3.1, Caputo (2005), p 56. The Maximum principle guarantees that $\lambda(t)^*$ is a continuous function of t and, hence, $\lambda(T)^* \geq 0$.

Claim 1 summarizes the range of possible optimal choices of treatment intensity. We will illustrate the dynamics of these choices diagrammatically in the control-state space, starting in the next section, by characterising the location of existing points of equilibrium in the a-H plane. If there are any such points they will be located at the intersection(-s) between the $\frac{da(t)}{dt} = 0$ and the $\frac{dH(t)}{dt} = 0$ locus.

Equilibrium

Points of equilibrium (steady state) may be relevant merely as points of reference for the behaviour of the dynamic system. Moreover, specific transversality conditions may rule out equilibrium points as parts of the optimal time paths of control and state variables. In our case, however, the significance of equilibrium may go well beyond that in many other dynamic models. The reason is that pharmaceutical treatments often involve relatively long periods of maintenance treatment, which seems plausibly modelled as a steady state. Points of equilibrium that are consistent with the transversality condition that we have applied – $\lambda(T)^* \cdot \left(H(T) - \overline{H}\right) = 0$ – are characterised by either the steady-state shadow price of health capital being equal to 0, or by the fact that the equilibrium level of health capital equals $\overline{H}$.

Formally, the points of equilibrium are found when solving the equations system by setting both equations of motion equal to 0. The equation of motion of the stock of health is given by (2). Let:

$$\frac{dH(t)}{dt} = TE^l - \varphi^l \cdot (a(t)^* - a^l)^2 - \delta \cdot H(t)^* \equiv h(a(t)^*, H(t)^*). \qquad (6)$$

In order to obtain the corresponding equation of motion for $a(t)$, take the total time-derivatives of (5), set $\frac{d\lambda(t)^*}{dt} \equiv f(H(t)^*)$, and obtain: $f(H(t)^*) \cdot \left(-2 \cdot \varphi^l \cdot (a(t)^* - a^l)\right) - \frac{da(t)^*}{dt} \cdot \left(2 \cdot (\varphi^s + \varphi^l \cdot \lambda(t)^*)\right) = 0$. Solving for $\frac{da(t)^*}{dt}$ gives:

$$\frac{da(t)^*}{dt} = \frac{f(H(t)^*) \cdot \left(-2 \cdot \varphi^l \cdot (a(t)^* - a^l)\right)}{2 \cdot (\varphi^s + \varphi^l \cdot \lambda(t)^*)} \equiv \frac{g(a(t)^*, H(t)^*)}{2 \cdot (\varphi^s + \varphi^l \cdot \lambda(t)^*)}. \qquad (7)$$

The dynamics of the system is described by equations (6) and (7), and the stationary loci can be found using $h(a(t)^*, H(t)^*) = 0$ and $g(a(t)^*, H(t)^*) \equiv f(H(t)^*) \cdot \left(-2 \cdot \varphi^l \cdot (a(t)^* - a^l)\right) = 0$. Technically, a steady state, a^e, H^e , is defined by the equation system $[h(a^e, H^e) =$

$0, g(a^e, H^e) = 0]$. In appendix Awe show (1) that the $\frac{dH(t)^*}{dt} = 0$ locus is a parabola, and (2) that the $\frac{da(t)^*}{dt}$ locus is a hyperbola, which is downward (upward) sloping when $a^l \geq a^s$ ($a^l < a^s$ and $\pi \leq 2 \cdot \varphi^s \cdot (a^s - a^l)$);[11] and in both cases convex if y''' is sufficiently small. The following claim summarizes how the location of a steady state depends on (1) the out-of-pocket marginal cost of pharmaceuticals, (2) the rate at which the amount of gross health investment diminishes as treatment diverges from a^l, and (3) the rate at which the instantaneous utility diminishes as treatment diverges from a^s:

Claim 2: the effects on the location of a steady state of an increase in (1) π, (2) φ^l, and (3) φ^s are:

(*i*) in the case when $a^l > a^s$:

(1) $\frac{dH^e}{d\pi} < 0$ and $\frac{da^e}{d\pi} < 0$; (2) $\frac{dH^e}{d\varphi^l} <> 0$ and $\frac{da^e}{d\varphi^l} > 0$; and (3) $\frac{dH^e}{d\varphi^s} > (<) 0$ when $a^e < (>) a^s$ and $\frac{da^e}{d\varphi^s} > (<) 0$ when $a^e < (>) a^s$.

(*ii*) in the case when $a^l < a^s$, and $\pi < 2 \cdot \varphi^s \cdot (a^s - a^l)$:

(1) $\frac{dH^e}{d\pi} > 0$ and $\frac{da^e}{d\pi} < 0$; (2) $\frac{dH^e}{d\varphi^l} < 0$ and $\frac{da^e}{d\varphi^l} < 0$; and (3) $\frac{dH^e}{d\varphi^s} < 0$ and $\frac{da^e}{d\varphi^s} > 0$.

(*iii*) in the case when $a^l = a^s$ and $\pi > 0$:

(1) $\frac{dH^e}{d\pi} < 0$ and $\frac{da^e}{d\pi} < 0$; (2) $\frac{dH^e}{d\varphi^l} <> 0$ and $\frac{da^e}{d\varphi^l} > 0$; and (3) $\frac{dH^e}{d\varphi^s} > 0$ and $\frac{da^e}{d\varphi^s} > 0$.

(*iv*) in the case when $a^l = a^s$ and $\pi = 0$, the location of the equilibrium does not depend on π, φ^l or φ^s, since $a(t)^* = a^e = a^l = a^s$.

Proof: the proof of (*i*) and (*ii*) is a set of straightforward comparative statics calculations. The (*iv*) case follows trivially since (a) $a^e = a^l = a^s$ and (b) the stationary loci for H does not depend on the exogenous parameters of interest. See appendix A.

The intuition behind these results is as follows: *First*, an increase in the marginal cost of treatment, π, always leads to a lower treatment level, but may increase or decrease the equilibrium level of health capital depending on the relationship between a^l and a^s: when $a^l > a^s$ we know that the equilibrium treatment level is below a^l and, hence, that a reduction in the treatment level

[11] This means that an equilibrium, if it exists, is unique when $a^l \geq a^s$.

will lead to a smaller health stock. When $a^l < a^s$, the equilibrium health stock will increase for analogous reasons. *Second*, consider an increase in the rate of "punishment", φ^l, for deviating from a^l. Such an increase suggests that the balance between instantaneous utility and health investments occur at a higher treatment level when $a^l > a^s$, and at a lower level when $a^l < a^s$. In the first case, a treatment-intensity increase may only partly offset the effect of the increase of φ^l and, hence, the equilibrium health stock may increase or decrease. In the second case, the decrease in treatment intensity does not offset the increase in the rate "punishment", φ^l. *Third*, an increase in φ^s will affect the optimal treatment intensity in a directly analogous way; that is, the equilibrium intensity will decrease (increase) if the original equilibrium is above (below) a^s.

Type of equilibrium

The steady-state stability properties are determined by an examination of the Jacobian matrix of the system $[h(a^e, H^e) = 0, g(a^e, H^e) = 0]$, that is, $J = \begin{pmatrix} h_H & h_a \\ g_H & g_a \end{pmatrix}$. The determinant $|J|$ and the trace, $tr(J)$, provide the necessary information. We summarize the model's stability properties in Claim 3:

Claim 3: A steady state is saddle-point stable in the following case:

(i) $a^l > a^s$; and

(ii) $a^l = a^s$ and $\pi > 0$.

A steady state is locally stable in the following cases:

(iii) $a^l \leq a^s$ and $0 \leq \pi < 2 \cdot \varphi^l \cdot (a^s - a^l)$.

Proof: See appendix A.

Dynamics – phase diagrams

Figures 3 and 4 illustrate possible optimal streamlines in the state-control space.[12] The shape of each stationary locus is derived in appendix A. The partial derivatives h_H and g_H are used to determine the direction of the vector field in each section of the positive quadrant. Thus, in Figure 3, the rate of change of the health stock will decrease from bottom to top in a phase diagram with $H(t)$ on the vertical axis, and, in particular, it will be 0 at the $H(t)$ stationary locus.

[12] Figures 3 and 4, below, illustrate the stationary loci using the specification $y = ln(H(t))$. The $\frac{da}{dt}$=0 locus is a hyperbola with one branch at each side of the vertex $a(t) = a^s$, with some parts lying outside the admissible quadrant (both the health stock and the treatment intensity must be positive).

This means that the stock of health is increasing below the locus, and decreasing above it. Similarly, along a vertical line that crosses the $a(t)$ stationary locus, the treatment intensity is decreasing below and increasing above the locus. Using an analogous way of reasoning gives the state-phase diagrams in Figures 4 and 5.

In Figure 3, streamlines **A** and **B** are consistent with equations (2), (4) and (5), and with the transversality condition. It should be noticed, however, that the shadow-price of health capital may be strictly positive or equal to 0 at $t = T$ (this bears some significance, below, in the example illustrating the optimal time-path when $a^l > a^s$; Figure 6). The difference between the streamlines is the amount of time that they are associated with to go from $\underline{H}$ to $\overline{H}$. Streamline **C** is consistent with the transversality condition only when $\pi = 0$, which is obvious from equation (5) ($\lambda(T) = 0$ and $a(T) = a^s$). Similarly, in Figure 4, streamlines **D-E** are solutions to our problem for some *T*, while **F** may or may not violate the condition that $H(t) \geq H_{min}$. In Figure 5, the case when $a^l = a^s$ is illustrated. The streamlines **G** and **H** ($\pi > 0$) and **I** ($\pi = 0$) are all solutions to some specification of the optimization problem. Remember from *Claim 1*, i.e. $a(t) > a^l = a^s$ is not possible.

Figure 3. Illustration of the shape of the stationary loci, and trajectories for the truncated vertical terminal line problem, the case when $a^l > a^s$. The $\frac{dH_t}{dt} = 0$ locus is a parabola with an inflexion point at $a(t) = a^l$, and intersections with the a-axis at $a(t) = a^l \pm \sqrt{\frac{TE^l}{\varphi^l}}$. The $\frac{da_t}{dt} = 0$ locus is a hyperbola, with the vertex at $a(t) = a^s$. The equilibrium is saddle-point stable; the stable and unstable branches are marked.

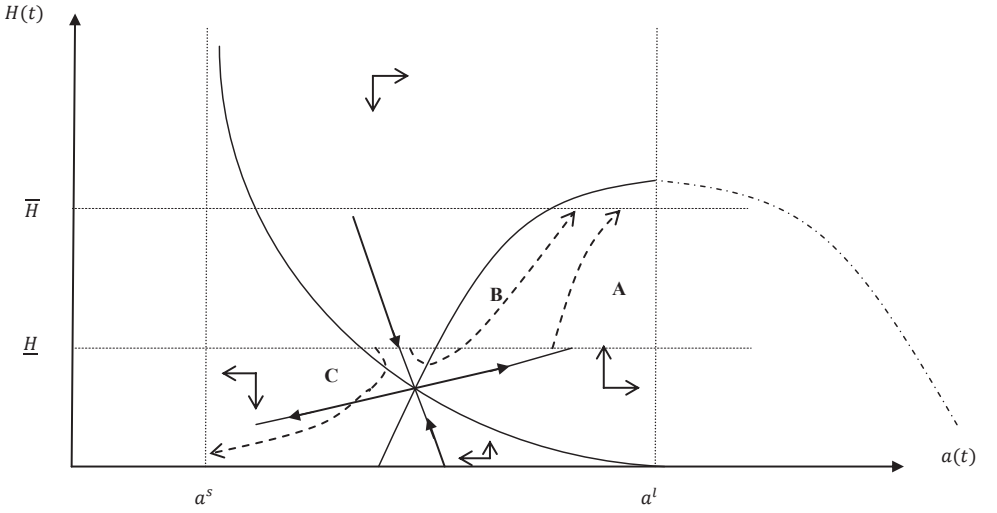


Figure 4. Illustration of the shape of the stationary loci, and trajectories for the truncated vertical terminal line problem, the case when $a^l < a^s$, and $\pi \leq 2 \cdot \varphi^l \cdot (a^s - a^l)$. The $\frac{dH_t}{dt} = 0$ locus is a parabola with an inflexion point at $a(t) = a^l$, and intersections with the a-axis at $a(t) = a^l \pm \sqrt{\frac{TE^l}{\varphi^l}}$. The $\frac{da}{dt} = 0$ loci is a hyperbola, with the vertex at $a(t) = a^s$. Note that the figure is drawn so that the steady state satisfies the transversality condition. The equilibrium is locally stable.

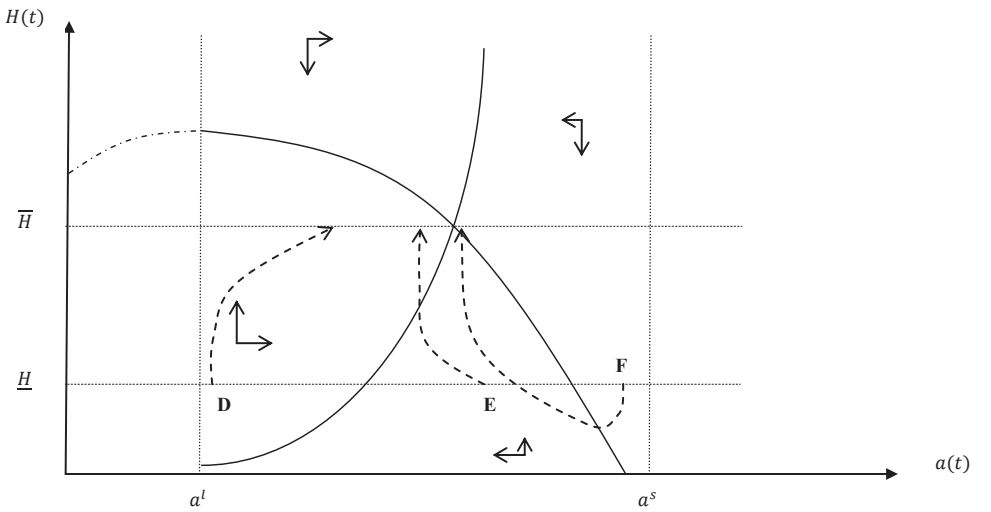
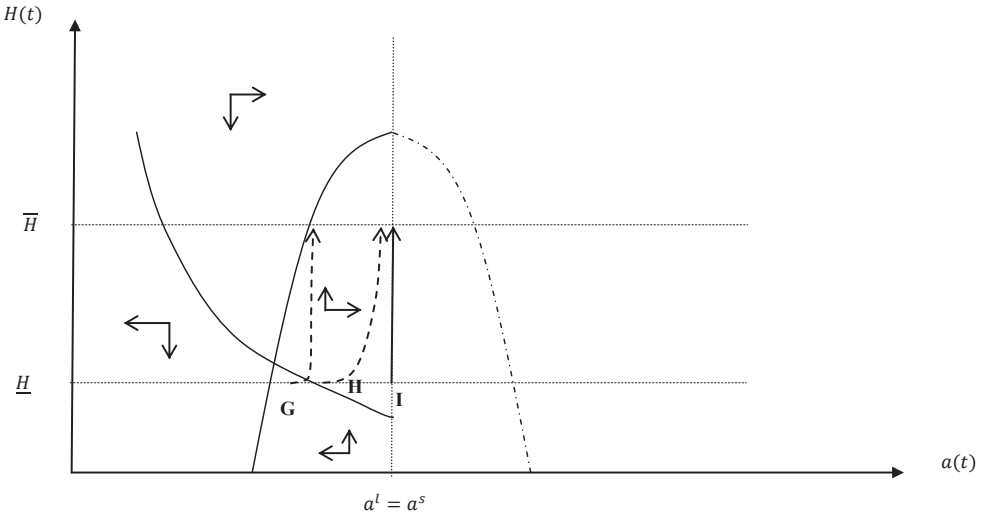


Figure 5. Illustration of the shape of the stationary loci, and trajectories for the truncated vertical terminal line problem, the case when $a^l = a^s$. The $\frac{dH_t}{dt} = 0$ locus is a parabola with an inflexion point at $a(t) = a^l$, and intersections with the a-axis at $a(t) = a^l \pm \sqrt{\frac{TE^l}{\varphi^l}}$. The $\frac{da}{dt} = 0$ loci is a hyperbola, with the vertex at $a(t) = a^s$. The equilibrium (at the intersection of the parabola and the vertical line) is locally stable.



The implications, which follow from these dynamics, for the practical problem at hand – optimal pharmaceutical utilization – are more thoroughly discussed in the discussion section.

The optimal time-path of treatment and health

Next, we characterise the optimal time-path of $a(t)^*$. From equation (5) it is clear that the optimal time-path of $a(t)^*$ mirrors that of the shadow price of health capital, $\lambda(t)^*$. For illustration purposes, we assume that the requirements in *Claim 1* are fulfilled for $\forall t$, and that $T = 10$. Then, the optimal treatment-intensity time-path can be illustrated using equation (5) directly. Solving for $a(t)$ in (5) gives: $a(t) = \frac{2 \cdot \varphi^s \cdot a^s + \lambda(t) \cdot 2 \cdot \varphi^l \cdot a^l - \pi}{2 \cdot \varphi^s + \lambda(t) \cdot 2 \cdot \varphi^l}$. Figures 6 and 7, below, illustrate the shape of the treatment time-path when $a^l > a^s$ and when $a^l < a^s$, respectively, letting λ go from 10 to 0, and using the substitution $t = 10 - \lambda$, i.e., $a(t)$ is plotted for $t = 0$ to $t = 10$ (implicitly, this assumes that the transversality condition is satisfied due to $\lambda(T) = 0$). The specific parametrizations used are reported in the figure heads. Taking the time derivative of

$a(t)$, gives $\frac{4\cdot\varphi^s\cdot\varphi^l\cdot(a^l-a^s)+2\cdot\pi\cdot\varphi^l}{\left(2\cdot\varphi^s+\lambda(t)\cdot2\cdot\varphi^l\right)^2}\cdot\frac{d\lambda(t)}{dt}$, which shows that whether or not the treatment intensity increases or decreases over time depends on the relation between a^l and a^s.

Figure 6. Optimal treatment-intensity levels, for the following specification:$\varphi^l=\varphi^s=1;\ a^s=1;\ a^l=2;\ \pi=1$.

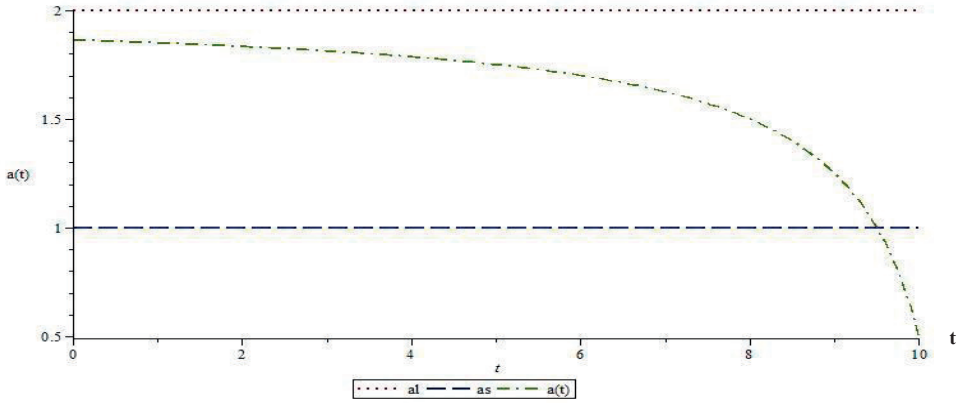
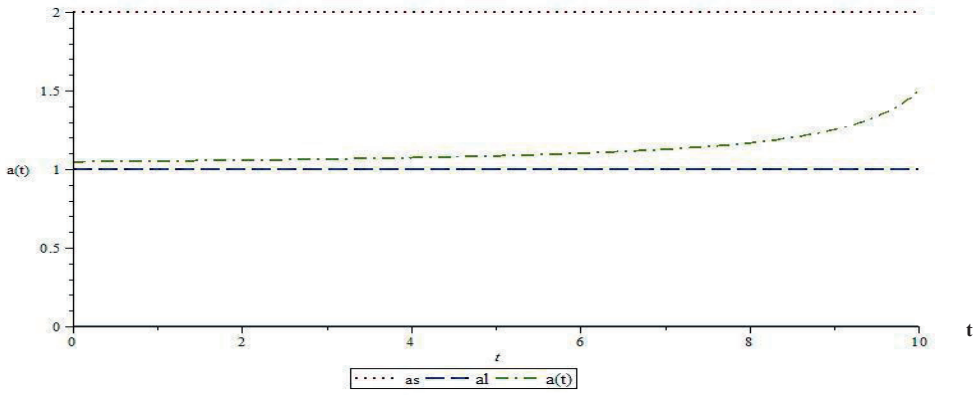


Figure 7. Optimal treatment-intensity levels, for the following specification:$\varphi^l=\varphi^s=1;\ a^s=2;\ a^l=1;\ \pi=1$.



From the expression for $\frac{da(t)}{dt}$, above, it is clear that when $a^l<a^s$ the time-path of the treatment intensity has either a positive or a negative slope, depending on the marginal cost of treatment. The time-path of $a(t)$, when $a^l=a^s$, always slopes downward, and is located below a horizontal line $a(t)=a^l=a^s$.

2.4 Discussion

In this paper, we have developed a dynamic theoretical model of individual health behaviours that are characterised by a double-peaked effect on individual wellbeing. This feature is present in several important behaviours that are related to health. The steady-state and the dynamic properties of the model have been demonstrated, and the model can readily be used for deriving comparative dynamics results necessary for thorough policy analyses. For this purpose, mathematical methods have been developed and applied to human-capital analyses (Oniki, 1973; Eisenring, 1999; Ehrlich and Chuma, 1990). Performing comparative dynamics analyses goes beyond the scope of this paper, however.

Several health-related behaviours may have separate short-term and long-term effects that produce non-monotonic effects. Our model makes the distinction between instantaneous utility and investments in health. For instance, doing physical exercise may provide improvements in current quality-of-life and add to the health stock. More specifically, we have developed a dynamic human-capital model of health-behaviour, making the distinction between two competing interests of the individual: instantaneous utility (short term), affecting quality of life, and investments in health (long-term). The fundamental notion is that each of these two objectives may be achieved by targeting a specific level of effort – the physiologically optimal level. Thus, the studied behaviour exerts a non-monotonic effect on both quality of life and health. The model and the analysis use pharmaceutical treatment as an example, but the scope of the model is wider. In fact, the demand for any behaviour or treatment that can be characterised by having both short- and long-term non-monotonic effects, i.e., quality-of-life and health-investment effects, may be analysed using this framework. Several preventive, curative and palliative treatments have these features.

Using this framework, and the pharmaceutical-treatment example, we have demonstrated that (1) the treatment intensity optimally falls into given ranges, depending on the relationship between long- and short-term physiologically optimal levels; (2) when the quality-of-life objective calls for a higher treatment intensity than what would be optimal from a health-investment perspective: (a) the chosen treatment is dynamically stable; and (b) a higher marginal out-of-pocket cost of treatment may be associated with better health.

So, what conclusions can be drawn regarding pharmaceutical-treatment adherence? To begin with, we have to distinguish between the two main treatment situations: $a^l > a^s$ and $a^l < a^s$. In the first case, we have all types of treatments for which there is no (or possibly negative) instantaneous utility, or treatments with positive non-monotonic instantaneous utility effects

where the intensity level optimal for utility is *below* the level maximizing health. Certainly, in that category there are several treatments which are monitored by health-care personnel and, hence, little room is left for patient discretion. For instance, chemotherapy treatment is either accepted or rejected; essentially, the individual does not have the option of partaking in the treatment at a level that he or she finds comfortable. However, for a wide range of pharmaceutical treatments the individual, in effect, has complete autonomy over the treatment intensity. *First*, consider the case when $a^l > a^s$ where the physician prescribes a preventative self-management pharmaceutical treatment with intensity a^l. When not supervised, the individual chooses a lower treatment intensity if perceiving that the costs (in terms of money or immediate quality of life effects) are not warranted in terms of the the health (long-term) benefit. If so, increasing the reimbursement level – or paying the individual – is the only intervention that unambiguously may increase the individual treatment intensity of choice. *Second*, consider the case when $a^l < a^s$: due to immediate quality of life gains, the individual may use higher treatment intensities than prescribed, despite being aware of the adverse health consequences. Then, lowering the reimbursement level – or taxing the individual – decreases the individual's preferred treatment intensity. Alternative interventions can be restricting sales (e.g., limiting prescribing or keeping sales records) or turning to incremental pharmaceutical innovations: Modifications that decrease φ^s, e.g., slow that release formulas decrease the treatment intensity and thus improve health.

In treatment situations with perfectly harmonising treatment objectives, i.e., the treatment intensity level maximizing health outcomes also maximizes instantaneous quality of life, the steady-state equilibrium is locally stable. This implies that the treatment's quality of life effects help the individual to maintain the treatment intensity, thus maximizing the health capital investment. Consequently, when a pharmaceutical treatment is intended for the long-term, such as for many treatments of chronic conditions, harmonising the treatment goals is ideal. Yet, if the out-of-pocket price for the pharmaceutical treatment is sufficiently high, the individual may use lower treatment intensities than what would be optimal from a health-investment perspective.

Model analyses of our modified version of the demand for health model illustrate that public policies which uniformly subsidise a higher share of the pharmaceutical costs have either positive or negative effects on public health, depending on the type of pharmaceutical. Therefore, policy makers could modify the pharmaceutical reimbursement system, so that the pharmaceutical out-of-pocket cost steers individual decisions toward treatment intensities more beneficial for long-term health. Population health will increase if pharmaceutical treatments with long-term health objectives, particularly when involving adverse quality of life effects, are subsidised more, and if

treatments generating short-term utility by forgoing long-term health objectives are subsidised less or taxed.

Moreover, incremental pharmaceutical innovations modifying older pharmaceuticals to become more user friendly (e.g., lower φ^s or φ^l)) have either positive or negatives effects on public health, depending on the type of pharmaceutical treatment.

Regarding pharmaceutical innovations offering more user friendly versions of old pharmaceuticals, the model may be used to carefully evaluate how new pharmaceutical properties affect individual treatment intensity decisions, and thus disclose the potential value of the innovation. Note that some innovations may affect pharmaceutical utilization behaviour in a health-impeding way.

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Appendix A

Concavity of the Hamiltonian

The Hamiltonian function, $\mathcal{H}$, is jointly and strictly concave in $(a(t), H(t))$. First, note that $\mathcal{H}_{Ha} = \mathcal{H}_{aH} = 0$. That leaves only the diagonal terms of the Hessian matrix for $\mathcal{H}$. These are: $\mathcal{H}_{HH} = y'' < 0$, and $\mathcal{H}_{aa} = -2 \cdot \varphi^s - \lambda \cdot 2 \cdot \varphi^l < 0$, since $\lambda(t) > 0 \; \forall \, t \in [0, T)$ (Caputo, 2005, p 56), which means that $|\mathcal{H}| > 0$ and, hence, that the Hamiltonian is jointly and strictly concave in $(a(t), H(t))$. When $H(T) = \overline{H}$, the transversality condition puts no restriction on $\lambda(T)$. However, due to (1) lemma 3.1 in Caputo (2005) and (2) continuity of $\lambda(t)^*$ (the Maximum principle; see, for instance, Caputo, 2005) it must be the case that $\lambda(T) \geq 0$.

Proof of Claim 1

First, $\lambda(T) \geq 0$ – see above. The (*i*) part follows immediately by assuming $a(t) \geq (\leq) \max(a^s, a^l)$ $(\min(a^s, a^l))$, which leads to a violation of $\frac{\partial \mathcal{H}}{\partial a} = 0$, when $a^l \neq a^s$. When $\pi > 0$, it is necessarily true that $a^*(t) < max(a^s, a^l)$; $a^*(t) = 0$ may be optimal for some combination of (a^s, a^l) and π (the (*ii*) – (*iv*) parts); notice, that when $a^l = a^s = a$, $\frac{\partial \mathcal{H}}{\partial a}\Big|_{a(t)=a} < 0$. The case when $a^l < a^s$: if $\pi \leq 2 \cdot \varphi^s \cdot (a^s - a^l) \Rightarrow \frac{\partial \mathcal{H}}{\partial a}\Big|_{a(t)=a^l} \geq 0$. Thus, $a^l \leq a(t) < a^s$ (the (*v*) part).

Shape of the steady-state loci

First, note that for $a^l \neq a^s$ the following applies (arguments omitted, for brevity): Let the left-hand side of equation (4) be denoted f, and use equation (5) to substitute for $\lambda(t)^*$. Then, we get: $f \equiv -y' + (\delta + \rho) \cdot \lambda = -y' - (\delta + \rho) \cdot \frac{(\pi + 2 \cdot \varphi^s \cdot (a(t) - a^s))}{2 \cdot \varphi^l \cdot (a(t) - a^l)}$. The partial derivatives of f are:

$f_H = -y'' > 0;$ $\qquad f_a = -(\delta + \rho) \cdot \frac{2 \cdot \varphi^s \cdot 2 \cdot \varphi^l \cdot (a(t) - a^l) - (\pi + 2 \cdot \varphi^s \cdot (a(t) - a^s)) \cdot 2 \cdot \varphi^l}{\left(2 \cdot \varphi^l \cdot (a(t) - a^l)\right)^2} = -(\delta +$

$\rho) \frac{4 \cdot \varphi^s \cdot \varphi^l \cdot (a^s - a^l) - 2 \cdot \pi \cdot \varphi^l}{\left(2 \cdot \varphi^l \cdot (a(t) - a^l)\right)^2}$, which is $< (>) \, 0$ for $a^s > (<) a^l$, when $\pi = 0$. If $\pi > 0$, $f_a > 0$ if $a^l \geq a^s$; and, if $a^l < a^s$, $f_a \leq 0$ if $\pi \leq 2 \cdot \varphi^s \cdot (a^s - a^l)$.

Finally, if $a^l = a^s$ and $\pi = 0$, then $f_a > 0$.

Second, the shape of the $\frac{dH(t)}{dt} = 0$ locus is given by rearranging the equation $h(a(t), H(t)) = 0$, which yields: $H(t) = \frac{TE^l - \varphi^l \cdot (a(t) - a^l)^2}{\delta}$; and the shape of the $\frac{da}{dt} = 0$ locus is found by differentiating $f = 0$, which gives: $f_H \cdot dH + f_a \cdot da = 0 \Rightarrow \frac{dH}{da} = -\frac{f_a}{f_H}$. Thus, when $\pi = 0$, $\frac{dH}{da} < (>)\, 0$ for $a^l > (<) a^s$. The case when $\pi > 0$ follows directly from the above. The curvature of the locus is given by $\frac{d^2H}{da^2}$:

$$\frac{d^2H}{da^2} = -(\delta + \rho) \cdot \frac{4 \cdot \varphi^s \cdot \varphi^l \cdot (a^s - a^l) - 2 \cdot \pi \cdot \varphi^l}{\left(\left(2 \cdot \varphi^l \cdot (a(t) - a^l)\right)^2 \cdot y''\right)^2} \left(4 \cdot \varphi^l \cdot (a(t) - a^l) \cdot y'' + \left(2 \cdot \varphi^l \cdot (a(t) - a^l)\right)^2 \cdot y'''\right) > 0$$

if $y''' \approx 0$, which means that in such a case the locus is convex.

Proof of Claim 2

We apply Cramer's rule for solving the system that results from differentiating $[h(a^e, H^e) = 0, g^s(a^e, H^e) = 0]$. The determinant of the Jacobian, $|J|$, matrix is: $\begin{vmatrix} h_H & h_a \\ f_H & f_a \end{vmatrix}$. Evaluated in equilibrium it becomes: $|J| = -\delta \cdot f_a + 2 \cdot \varphi^l \cdot (a^e - a^l) \cdot f_H$. Thus, $|J| < 0$ when $a^l > a^s$, due to the signs of f_a and f_H, and claim 1. When $a^l < a^s$, the sign of the determinant is ambiguous. Adding the assumption $\pi < 2 \cdot \varphi^s \cdot (a^s - a^l)$ yields $a^e > a^l$, and, hence, that $|J| > 0$. When $a^l = a^s$ and $\pi > 0 \Rightarrow |J| < 0$.

The effect of π*:* Differentiating the equation system that defines steady state gives:

$$\begin{pmatrix} h_H & h_a \\ f_H & f_a \end{pmatrix} \cdot \begin{pmatrix} \frac{dH^e}{d\pi} \\ \frac{da^e}{d\pi} \end{pmatrix} = \begin{pmatrix} -h_\pi \\ -f_\pi \end{pmatrix}.$$

Cramer's rule gives:

$$\begin{pmatrix} \frac{dH^e}{d\pi} \\ \frac{da^e}{d\pi} \end{pmatrix} = \frac{\begin{pmatrix} \begin{vmatrix} -h_\pi & h_a \\ -f_\pi & f_a \end{vmatrix} \\ \begin{vmatrix} h_H & -h_\pi \\ f_H & -f_\pi \end{vmatrix} \end{pmatrix}}{|J|}.$$

More explicitly, when $a^l > a^s$, we have $\frac{dH^e}{d\pi} = (0 \cdot f_a + h_a \cdot f_\pi) \cdot \frac{1}{|J|} = \frac{\delta + \rho}{|J|} < 0$, and $\frac{da^e}{d\pi} = (-h_H \cdot f_\pi + 0 \cdot f_H) \cdot \frac{1}{|J|} = -\frac{\delta \cdot (\delta + \rho)}{2 \cdot \varphi^l \cdot (a^e - a^l)} \cdot \frac{1}{|J|} < 0$, due to claim 1. Similarly, when $a^l < a^s$, we have $\frac{dH^e}{d\pi} = \frac{\delta + \rho}{|J|} > 0$, and $\frac{da^e}{d\pi} = -\frac{\delta \cdot (\delta + \rho)}{2 \cdot \varphi^l \cdot (a^e - a^l)} \cdot \frac{1}{|J|} < 0$, if $\pi < 2 \cdot \varphi^s \cdot (a^s - a^l)$. When $a^l = a^s$ and $\pi > 0$, $|J| < 0$, and, hence, $\frac{dH^e}{d\pi} < 0$ and $\frac{da^e}{d\pi} < 0$.

The effect of φ^l*:* Following the same procedure as above we get, when $a^l > a^s$: $\frac{dH^e}{d\varphi^l} = \left(-h_{\varphi^l} \cdot f_a + h_a \cdot f_{\varphi^l}\right) \cdot \frac{1}{|J|} = \left[(a^e - a^l)^2 \cdot f_a + (-4 \cdot \varphi^l \cdot (a^e - a^l)^2 \cdot (\delta + \rho) \cdot \frac{(\pi + 2 \cdot \varphi^s \cdot (a^e - a^s))}{\left(2 \cdot \varphi^l \cdot (a^e - a^l)\right)^2}\right] \cdot \frac{1}{|J|} <> 0$, since by (5) $\pi + 2 \cdot \varphi^s \cdot (a^e - a^s) > 0$; and $\frac{da^e}{d\varphi^l} = \left(-h_H \cdot f_{\varphi^l} + h_{\varphi^l} \cdot f_H\right) \cdot \frac{1}{|J|} = \left[\delta \cdot (\delta + \rho) \cdot 2 \cdot (a^e - a^l) \cdot \frac{(\pi + 2 \cdot \varphi^s \cdot (a^e - a^s))}{\left(2 \cdot \varphi^l \cdot (a^e - a^l)\right)^2} - (a^e - a^l)^2 \cdot f_H\right] \cdot \frac{1}{|J|} > 0$.

Evaluating the same expression when $a^l < a^s$ and $\pi < 2 \cdot \varphi^s \cdot (a^s - a^l)$, gives $\frac{dH^e}{d\varphi^l} < 0$ and $\frac{da^e}{d\varphi^l} < 0$, when. When $a^l = a^s$ and $\pi > 0$, we have $\frac{dH^e}{d\varphi^l} <> 0$ and $\frac{da^e}{d\varphi^l} > 0$.

The effect of φ^s*:* Following the same procedure as above we get, when $a^l > a^s$: we have $\frac{dH^e}{d\varphi^s} = \left(-h_{\varphi^s} \cdot f_a + h_a \cdot f_{\varphi^s}\right) \cdot \frac{1}{|J|} = \left(0 \cdot f_a + 2 \cdot \varphi^l \cdot (a^e - a^l) \cdot (\delta + \rho) \cdot \frac{2 \cdot (a^e - a^s)}{2 \cdot \varphi^l \cdot (a^e - a^l)}\right) \cdot \frac{1}{|J|} > (<) 0$ when $a^e < (>) a^s$, and $\frac{da^e}{d\varphi^s} = \left(-h_H \cdot f_{\varphi^s} + h_{\varphi^s} \cdot f_H\right) \cdot \frac{1}{|J|} = \left(-\delta \cdot (\delta + \rho) \cdot \frac{2 \cdot (a^e - a^s)}{2 \cdot \varphi^l \cdot (a^e - a^l)}\right) \cdot \frac{1}{|J|} > (<) 0$ when $a^e < (>) a^s$. Evaluating the same expression when $a^l < a^s$ and $\pi < 2 \cdot \varphi^s \cdot (a^s - a^l)$ gives $\frac{dH^e}{d\varphi^s} < 0$ and $\frac{da^e}{d\varphi^s} > (<) 0$, when $a^e > (<) a^l$. When $a^l = a^s$ and $\pi > 0$, we have $\frac{dH^e}{d\varphi^s} > 0$ and $\frac{da^e}{d\varphi^s} > 0$.

Proof of Claim 3

The elements of the Jacobian matrix are (evaluated in steady state): $h_H = -\delta < 0$; $h_a = -2 \cdot \varphi^l \cdot (a^e - a^l) \geq (<) 0$ for $a^e \leq (>) a^l$; from the above it is clear that $f_H > 0$. Thus, $f_a \geq (<) 0$ if $\pi = 0$ and $a^l > (<) a^s$; $f_a > 0$ if $\pi > 0$ and $a^l \geq a^s$; and $f_a < 0$ if $a^l \leq a^s$ and $0 \leq \pi < 2 \cdot \varphi^l \cdot (a^s - a^l)$. Further, we have: $tr(J) = -\delta + f_a \leq 0$ whenever $f_a \leq 0$. Thus, in the following cases the Jacobian determinant, $|J| = -\delta \cdot f_a + 2 \cdot \varphi^l \cdot (a^e - a^l) \cdot f_H$, has a definite sign: (*i*) $a^l \geq a^s$ and $\pi > 0 \Rightarrow |J| < 0$; (*ii*) $a^l < a^s$ and $\pi = 0 \Rightarrow |J| > 0$; (iii) $a^l < a^s$ and $0 < \pi < 2 \cdot \varphi^l \cdot (a^s - a^l) \Rightarrow |J| < 0$. The sign of the trace and determinant in these cases yield the conclusions in Claim 3; see, for instance, Caputo (2005) p 354-355. In the $a^l = a^s$ case, when $\pi = 0$, stability is guaranteed by $h_H = -\delta < 0$.

Chapter 3

Patient adherence to prescribed pharmaceuticals – An analysis based on Swedish real-life data

3.1 Introduction

Low patient adherence to treatment recommendations is a persisting problem. Evidence shows that every second person with chronic conditions in developed countries does not adhere to treatment recommendations, and adherence is even lower in developing countries, presumably due to unaffordability. As low adherence to prescribed pharmaceutical regimes attenuates treatment benefits and lowers health care efficiency, adherence attracts attention from policy makers and health managers around the world. Being a considerable public health concern, the world health organization considers that increasing adherence to long-term pharmaceutical regimens is essential to curb the growing burden of chronic diseases. (WHO, 2010)

The literature on adherence barriers has generated a vast amount of results. The homogeneity in measuring adherence and the absence of conceptual frameworks that may integrate the results from studies across patient groups and pharmaceuticals (e.g., identified barriers are inconsistently associated with adherence across studies) make it difficult to draw firm conclusions for policy recommendations (Vermeire et al., 2001; Gellad et al., 2009; WHO, 2010). Despite the shortcomings, the literature identifies cost-sharing, regimen complexities, medication beliefs and depression as potential fruitful targets for improving adherence (Gellad et al., 2009).

This essay focuses on a less studied link from attainable achievable treatment benefits to adherence for a set of pharmaceuticals standardly prescribed for long-term treatment of prevalent chronic diseases. This link is important because a positive association i.e., people are more adherent when treatment benefits are higher, may suggest that adherence follows from informed and rational decisions reflecting treatment benefits and costs. If so, conceptual economic frameworks such as the demand-for-health model may contribute to adherence literature and

guide policy solutions. For instance, the societal value of pharmaceutical innovations, which marginally improve treatment benefits, may be larger than projected if higher treatment benefits per se motivate adherence. Moreover, if low attainable treatment benefits discourage patients from following treatment recommendations, health care professionals and developers of clinical guidelines should take into consideration the fact that the internalized value of treating people with moderate benefits may be substantially lower than expected.

To explore the link between treatment benefits and adherence, I use the Swedish Prescribed Pharmacy Register (SPPR) and detailed individual information on e.g., health, demographical and socioeconomic characteristics. The data offers a unique opportunity to study long-term adherence in the Swedish setting which, due to the cost-sharing subsidy, is suitable for exploring non-financial adherence determinants[1].

The theoretical starting point is that adherence reflects treatment benefits and costs. By identifying specific health conditions augmenting the maximum therapeutic benefit from the particular treatment, I empirically estimate the relationship between specific health conditions and adherence. The following selection of standardly prescribed pharmaceuticals for maintenance treatment of chronic conditions suits my purpose. (1) Selective beta blockers (SBRB), angiotensin converting enzyme inhibitors (ACE), angiotensin receptor blockers (ARB) and diuretics for cardiovascular disease treatment and (2) antidepressants for mental health concerns. The selection of pharmaceuticals and the study design reduce measurement bias, which is a common flaw in adherence studies. Moreover, these pharmaceuticals have simple regimens, typically one dose a day, which rules out the fact that regimen complexity interferes with adherence. For theses pharmaceuticals, the achievable effect on long-term health depends on the specific health conditions, whereas treatment side-effects are mainly treatment-specific. The following examples demonstrate the relationship between specific health conditions and achievable therapeutic benefit. People with manifested coronary heart diseases benefit more from treatment with e.g., selective beta receptor blockers (SBRB) than people with hypertension (elevated blood pressure). The reason coronary heart disease amplifies the achievable therapeutic response is that SBRB, besides lowering hypertension, regulates the heart function which is disturbed in people with coronary heart diseases. Analogously, people with a history of depression benefit more from treatment with antidepressants than people who are depressed for the first time. This is because antidepressants alleviate illness symptoms and prevent recurrence, which is more common in people with a history of depression.

[1] The national health insurance in Sweden covers all residents and fully subsidizes pharmaceutical expenditures exceeding SEK 2200 in out-of-pocket payments (SEK 1800 before 2012) on a 12 month rolling basis.

3.2 Theoretical framework

Patient adherence to prescribed pharmaceutical treatment regimens are analysed within the demand-for-health framework. Building on Becker's human capital model (Becker, 1962), Grossman developed the demand-for-health framework (Grossman, 1972a, 1972b), which has become the standard economic model for analysing individual health-related behaviour. Since its introduction, the demand-for-health model has been extended in various ways; for instance, to incorporate uncertainty (Dardanoni and Wagstaff, 1987, 1990; Selden, 1993; Chang, 1996; Liljas, 1998, 2000), social capital (Bolin et al., 2003), healthy and unhealthy consumption (Forster, 2001), the family, instead of the individual, as health producer (Jacobson, 2000; Bolin et al., 2001, 2002b), and the employer as health producer (Bolin et al., 2002c). In short, the demand-for-health model differentiates health from regular human capital (e.g., education and skills). The rationale for the distinction is that regular human capital affects labour-market (or non-market) productivity, while health capital generates healthy time, enabling market participation per se. Besides this investment aspect, health is also demanded for consumption i.e., we enjoy good health, or put differently, we dislike poor health. Individuals contribute to their health capital level by producing health investments using means of own time and market goods such as medical care. The original demand-for-health model and most modifications postulate that the demand for markets goods entering the health production function derives solely from the underlying health demand.

Pharmaceutical treatments are a common medical-care good affecting long-term health and immediate quality-of-life (e.g., pain relief). Therefore, the consumption aspect of pharmaceutical treatments may also influence the demand. When treatments involve conflicting short- and long-term objectives (e.g., the treatment increases long-term health at the cost of side-effects lowering short-term quality of life), the individual may trade long-term health for immediate quality-of-life gains by using treatment intensities other than the optimal for long-term health. To exemplify, despite awareness of the consequences for long-term health, people may exceed the recommended dosage regimen of e.g., strong pain killers for immediate quality-of-life gains; or the reverse, i.e., people may not follow chemotherapy recommendations because side-effects immediately lower quality-of-life.

Recent versions of the demand-for-health model incorporate certain health activities that have non-monotonic health effects (Bolin and Lindgren, 2012; Bolin and Gustafsson, 2014), and explicitly state that various health behaviors have two distinct intensity levels maximizing short-term utility and long-term health, respectively (Bolin and Gustafsson, 2014). I follow this

theoretical line in assuming that pharmaceutical treatment pertains to an intensity level which maximizes short-term utility or long-term health. Accordingly, consumption aspects of pharmaceutical utilization also influence the demand for pharmaceutical treatment. The remaining assumptions follow essential demand-for-health (Grossman, 1972) concepts: (1) the individual demands health for the positive effect on the amount of healthy time (the investment motive) (2) the demand for health investments derives from the underlying demand for good health, (3) the individual produces health investments, and (4) the health-capital stock depreciates at each point in time.

Given that the prescribed treatment intensity maximizes the treatment effect on long-term health[2], any deviation of the daily dosage regimen results in diminishing health investments. When treatment side-effects increase proportionally with treatment intensity, no-treatment pertains to the maximum short-term quality-of-life level. Under these circumstances, the individual may trade-off long-term health for short-term quality of life gains by using lower treatment intensities than prescribed. As higher treatment intensities than prescribed result in diminishing health investments and increasing side-effects, the individual has no motive to exceed the prescribed treatment regimen.

The size of the achievable health capital investment with a particular pharmaceutical treatment depends on the health profile of the patient. As individuals with such specific health conditions may achieve higher treatment benefits than people without such conditions, the marginal cost for health investment in the particular treatment is higher for the latter group. In other words, the specific health conditions motivate higher adherence to the prescription, or the reverse, i.e., the specific health conditions increase the penalty in terms of forgone health if deviating from the prescription.

When estimating the link between feasible treatment benefits and adherence, individual heterogeneity in treatment cost may obscure the link. Thus, ideally, one should adjust for such heterogeneity that may follow from out-of pocket pharmaceutical expenses and non-monetary costs such as required time intensity (e.g., travel time, medical consultation and pharmaceutical management) and disutility costs from e.g. side-effects.

[2] Given that prescriptions are correct, it is realistic to assume that perfect adherence to the prescriptions maximizes the treatments' long-term health outcomes. In contrast, when pharmaceutical treatments generate immediate quality-of-life gains at the cost of long-term health, a lower treatment intensity than is prescribed pertains to higher long-term health.

3.3 Previous research

Since 1975, the empirical literature has studied the association between adherence and more than 200 variables. These variables, relating to, for instance, characteristics of the patient and the prescriber, economic factors and pathology factors, do not consistently predict adherence across studies, i.e., the association varies in magnitude and has opposite signs (Haynes et al., 1997; Donovan and Blake, 1992, Donovan 1995; Steiner and Vetter, 1994; Marinker, 1997; Haynes et al., 1979). Two reasons for inconsistency across studies may be that health behavior is difficult to predict in general, and the absence of conceptual frameworks that can integrate the results from different studies and thus contribute to the understanding of adherence behavior, which is inherently complex (Vermeire et al., 2001).

To understand adherence, the literature mainly applies theories from sociology and psychology. The commonly used health-belief-model suggests that low disease awareness and treatment misperception are common barriers to adherence. Yet, specific disease treatment programs, aiming at increasing adherence by educating patients in self-management, have moderate effects on long-term adherence (Schroeder et al. 2005; McDonald HP, Garg AX, and Haynes R 2002). While the health-belief-model may be applicable in some contexts, other models may be more suitable for explaining long-term adherence behavior.

The empirical literature indicates that adherence reflects treatment benefits and costs and that it may be suitable for analyses using economic theory. For example, people respond to economic incentives: Low patient co-payments are associated with higher adherence (Cole et al. 2006; Gibson, Ozminkowski, and Goetzel, 2005; Eaddy et al., 2012; Dor and Encinosa, 2004); when co-payments raise, people with low incomes (Stuart and Grana 1998; Smith and Kirking, 1992) or low treatment benefits (Blais et al., 2001; Pilote et al., 2002) downshift adherence more than high income people or people with higher treatment benefits; and when financial rewards for adherence are offered, people become more adherent (Giuffrida and Torgerson 1997). Besides monetary costs, treatment costs in terms of side-effects also decrease adherence (Lamiraud and Geoffard, 2007). Regarding education, economic theory suggests that better educated people are more efficient health producers than the less educated This is consistent with the results from the Goldman and Smith (2002) study, which demonstrates a positive association between education and adherence to complex, and essential, diabetes and HIV treatment regimens. Economic theory may help explain why education may not be positively associated with adherence across studies. For instance, when regimens are less complex or prescriptions may be substitutable with, e.g., medical surgery or general life-style changes, people across the socioeconomic strata may be

more or less likely to undergo non-pharmaceutical procedures, which in turn influences the necessity of pharmaceutical treatment. Thus, when studying adherence behavior, economic frameworks may help in identifying important confounders that, uncontrolled for, may obscure the studied relationship.

So far, the adherence literature applying economic theory is limited. Two of the few studies are Lamiraud and Geoffard (2007) and Koulayev and colleagues (2013). Lamiraud and Geoffard use clinical trial data to study adherence to two pharmaceutical HIV treatments with comparable therapeutic effects. Their results show that internalized treatment benefits and side-effects in one period affect adherence in the subsequent period. To gain knowledge about factors influencing adherence to long-term pharmaceutical therapies in out-patient care, we also need to study adherence in "real-life" practice, where circumstances such as health care information may be less than ideal. Using dispensed registry data from Denmark, Koulayev and colleagues study long-term adherence to cardiovascular treatments in real-life practice, but focus on patient-physician relationships rather than treatment benefits.

Heterogeneity in assessing, measuring and defining adherence also contributes to inconsistency in the adherence literature (see e.g., Gellad et al., 2009). There are roughly two ways to measure adherence, direct and indirect measures. When studying long-term adherence behavior, indirect measures are generally better suited than direct measures. This is because direct measures, such as supervising pharmaceutical intake or measuring chemical compounds in the body, have a positive influence on adherence behavior (i.e., white coat adherence), making the results difficult to extrapolate outside the study setting. Indirect measures are also imperfect; self-reported adherence or a pill count (i.e., counting returned tablets at the end of the study period) likely overestimates adherence as people may overrate adherence or not return remaining pharmaceuticals at the end of the study. The availability of national pharmacy registers offers a unique opportunity to indirectly measure adherence in large population groups unaware of being observed; thus the results are unflawed by white coat adherence or self-report bias. Although dispensed pharmaceuticals do not ensure pharmaceutical use, the measure is generally regarded as a valid proxy for consumed adherence (Vitolins et al., 2000).

3.4 Pharmaceuticals for treatment of cardiovascular diseases and mental disorders

Cardiovascular diseases and mental health concerns are prevalent chronic diseases where adherence to pharmaceutical recommendations is central for treatment success. The selection of pharmaceutical for treating these chronic conditions represents standardly prescribed pharmaceuticals for long-term maintenace use, i.e., the regimens do not allow "on-demand usage". As these pharmaceuticals increase long-term health at the cost of immediate short-term disutility from e.g. side-effects, the individual has no incentive to use higher treatment intensities than prescribed. Accordingly, excess pharmaceutical supply likely indicates stockpiling rather than higher treatment intensity levels than prescribed. Moreover, the simple treatment regimens, usually once-a-day, minimize heterogeneity in regimen complexity and could interfere with the results.

Pharmaceuticals for cardiovascular diseases

The selection of pharmaceuticals for cardiovascular disease treatment is (1) SBRB: ATC-code C07AB, (2) ACE and ARB: ATC-codes C09A and C09C, and (3) diuretics: ATC-codes C03.

Prior manifested hypertension and prior manifested heart diseases (e.g. coronary heart disease, heart failure and stroke) are health conditions specifically increasing the achievable therapeutic effect with cardiovascular pharmaceutical treatment. Therefore, (1) following treatment recommendations is more beneficial for people with prior manifested hypertension than people without cardiovascular illness history, and (ii) following treatment recommendations is even more beneficial for people with manifested heart diseases than people without cardiovascular illness history. The reason cardiovascular history affects achievable treatment benefit is that hypertension is a chronic condition that makes the cardiovascular system more and more vulnerable to coronary heart disease over time. When a coronary heart disease manifests itself, the heart function is already hampered and even more vulnerable. The pharmaceutical used in cardiovascular disease treatment alleviates hypertension and protects the heart function.

Individual heterogeneity in cardiovascular history enables me to analyze the link between adherence and achievable treatment benefit. To control for pharmaceutical specific side-effects diminishing short-term quality-of-life, SBRB, ACE/ARB and diuretics are analyzed separately. Because the cardiovascular disease patterns differ between men and women (e.g., disease onset and the infarction risk is generally higher for men than women (Lerner and Kannel, 1986)), they are analyzed separately.

Pharmaceuticals for mental disorders

For treatment of mental health concerns (e.g., generalized anxiety, depression, obsessive or compulsive behaviors), all antidepressants (ATC-code N06A) are selected. The specific health conditions, prior manifested anxiety and mental illness history increase the achievable long-term health effect with antidepressants. For people with mental health concerns, antidepressants may improve the mood and feeling of well-being. As antidepressants also alleviate anxiety and anxiety feeling commonly co-exist with depression, (1) people with manifested anxiety feelings benefit more from antidepressants than people with previous good mental health. Once symptoms are controlled, staying on treatment prevents illness recurrence. As depression is highly recurrent, and people with mental illness history have a higher risk of recurrence, (2) people with mental illness history benefit more from adhering to antidepressant treatments than people with previous good mental health.

Individual heterogeneity in mental illness history enables an analysis of the link between adherence and achievable treatment benefit. As antidepressants have comparable clinical therapeutic effects (e.g. see NICE 2004; CCOHTA 1997), they are jointly analyzed. Men and women are also analyzed separately because of potential differences in illness recurrence and health seeking behavior.

Pharmaceutical regulations

The selection of pharmaceuticals for analyses is prescription-only pharmaceuticals, which are exclusively dispensed by pharmacies in Sweden. When treatments are intended for long-term usage, physicians usually issue a prescription card entitling the patient a pharmaceutical supply for 12 months. However, for insurance coverage, the rules of the national health insurance permit each dispensing to cover a pharmaceutical supply for three months at most. Given a supply for three months has been dispensed, a new dispensing of pharmaceuticals is allowed after a two-month period has elapsed. Each insurance spell lasts 12 months and the level of reimbursement is a function of the accumulated out-of pocket payment for prescription pharmaceuticals. When the SEK 2,200 payment cap (SEK 1,800 before 2012) is reached, the individual receives full reimbursement for the remainder of the 12 month insurance period. Thus the rules of the insurance system introduce economic incentives to stockpile pharmaceuticals.

Therefore, in studies where dispensed adherence proxies consumed adherence, researchers must be aware of the risk of overestimating adherence following from stockpiling. Due to the

national health insurance rules in Swedish, people may stockpile pharmaceuticals when reimbursement is high, and then consume from the stock when reimbursement is low again. i.e., when facing a new insurance spell. Thus, I measure adherence for 18 months, which, by spanning more than one reimbursement spell, reduces the stockpiling-induced bias.

Excess pharmaceutical supply does not always imply stockpiling. For pharmaceuticals with desirable short-term utility gains such as tranquilizers or opioids, excess supply may reflect higher treatment intensities than prescribed. For this reason, dispensed adherence may be a more reliable proxy for consumed adherence when the pharmaceutical properties per se. discourage patients from exceeding the prescribed treatment intensity.

3.5 Data, variables and model specification

Data

The empirical analysis uses the database Health and Individuals. Longitudinal Data and When dispensed pharmaceuticals proxy the consumed amount, delay in time from dispensing to usage induces uncertainty. As stockpiling induces substantial delay in usage, or non-usage, an 18-month adherence is used to minimize uncertainty from stockpiling for two reasons. First, as the reimbursement period lasts 12 months, individuals with stockpiled pharmaceuticals are likely to consume from the stock before purchasing more initially unreimbursed pharmaceuticals. Second, as a prescription card is valid for 12 months and at most for a 12-month pharmaceutical supply, individuals need to renew their prescriptions to obtain pharmaceuticals for more than 12 months. This implies that people who have stockpiled pharmaceuticals, but do not continue their treatment, are less likely to be misclassified as adherents than in studies using shorter track periods. Because patient co-payment is a function of accumulated pharmaceutical out-of pocket spending, co-payment is highly correlated with adherence. Thus, I refrain from including co-payment in the analysis. By restricting the study population to individuals with a pharmaceutical dispensed before June 2006, I can follow individuals through 18 months. Starting from a baseline, which defines the date of the individual's initial dispensing, and thus varies across individuals, I track dispensed pharmaceuticals for each individual, i.e., someone with an initial dispensed pharmaceutical in June 2005 is followed through November 2006.

Analysis contains the Swedish biannual Survey of Living Conditions (ULF) waves 1980–1981, 1988-1989, 1996-1997 and 204-2005. Each wave interviews a nationally representative sample of approximately 16 000 people aged 16-84 with an average response rate of 80-85%. The survey

asks questions about living conditions, health and socioeconomic circumstances (e.g., educational level and labour market participation) and complements answers with register data on, for instance, income, tax transfers and in-patient hospitalization. Furthermore, respondents in wave 2004-2005 are linked to the Swedish Prescribed Pharmacy Register (SPPR) for the period July 2005 through November 2007. From July 2005 onwards, the nation-wide SPPR registers include personal identification numbers in addition to detailed information on all prescribed and dispensed pharmaceuticals in out-patient care in Sweden. The information includes such things as dispensed date and amount, the individual's unique prescribed dosage regimen and the pharmaceutical's Anatomical Therapeutic Chemical code (ATC) (for more details of the register see e.g., Wettermark et al., (2007)). The ATC-code groups pharmaceuticals according to the main active substance and its therapeutic, pharmacological and chemical properties (for more details, see WHO, 2012).

Using cross-sectional data on respondents in the ULF 2004–2005, I control for individual characteristics that may affect adherence behavior. As the morbidity pattern may vary between young-adults and older individuals, I exclude individuals younger than 30. In some cases, prescriptions are used for conditions other than long-term treatment of cardiovascular diseases and mental illness . Therefore, I exclude individuals with prescriptions where the dosage regimens permit on-demand usage, or state usage for treatment of illness other than long-term cardiovascular or mental illness. These criterions generate the following samples; SBRB (men: n=451 and women: n=513), ACE/ARB (men: n=385 and women: n=362), diuretics (men: n=257 and women: n=371) and antidepressants (men: n=178 and women: n=368).

I use the individually prescribed daily dosage regimens and dispensed pharmaceuticals to calculate the number of dispensed daily doses and assess the 18-month medication position ration (MPR), which denotes the percentage of days with pharmaceutical supply during an 18-month period. When dispensed pharmaceuticals exceed an 18-month supply, MPR takes on values greater than 100. Recall that treatment intensities higher than prescribed may diminish the treatment effect in the long term and induce more side effects. Hence, as MPR≥100 more likely reflects stockpiling than excess consumption, it is reasonable to define individuals with MPR≥100 as perfectly adherent.

Dependent variables

I use three adherence measures: (1) MPR, which is a continuous variable ranging from 0 to 100, (2) perfectly adherent, which is a dummy variable taking the value one if MPR≥100 and zero

otherwise and, (3) sufficiently adherent, which is a dummy variable taking the value 1 if MPR ≥80[3] and zero otherwise. (see Appendix B for the distribution of the untruncated MPR)

Main variables of interest

The main variables of interest are the treatment-specific health conditions enhancing the achievable therapeutic effect: Prior manifested hypertension or heart disease for pharmaceuticals for cardiovascular treatment, and a prior manifested anxiety or mental illness history for treatments with antidepressants. The main variables are specified as follows;

Hypertension and Heart disease are mutually exclusive dummy variables. Heart disease takes on the value one if the respondent reports a condition matching WHO's international Classification of Diseases, Ninth Revision (ICD-9), in the interval 410.0-429.9 (e.g., myocardial infarction, ischemic heart disease, pulmonary circulation disease) and 0 otherwise. For respondents without heart disease, Hypertension takes on the value one if the respondent reports a condition matching WHO's ICD-9 in the interval 401.0-405.9, (i.e., hypertension), and 0 otherwise.

Mental disorder and Anxiety are mutually exclusive dummy variables. Mental disorder takes on the value one if the respondent reports a condition matching WHO's ICD-9 in the interval 290.0-316.9 (e.g., depression and psychotic disorder), and 0 otherwise. For respondents without a mental disorder, Anxiety takes on the value one if the respondent reports worry or anxiety, and 0 otherwise.

The demand-for-health model postulates that age, general health, demographical and socioeconomic characteristics influence health behavior. To estimate the association between adherence and achievable therapeutic outcome, the empirical model includes a wide range of variables that may influence adherence behavior (see Appendix A for a detailed description of the control variables).

Empirical method and model specification

The association between MPR and specific health conditions is estimated with Ordinary Least Square (OLS) models and "trimming" OLS models. The association between specific health conditions and the perfect adherence and sufficient adherence measures is estimated with probit

[3] A cut-off point of 80 per cent is commonly used in adherence studies (see e.g. the Gellad et al 2009 review), and regarded as clinically relevant for predicting subsequent hospitalization in chronic diseases such as cardiovascular diseases (Karve et al 2009)

models using the Huber/White/sandwich estimate of variance to generate robust standard errors.

To analyse the association between specific health conditions and adherence to prescribed medical regimen, the empirical model is specified as follows:

$$y_{i,t}^{pharmj} \mid y_{i,t^*} > 0 = \alpha + \beta X_{i,t-1} + \gamma Z_{i,t-1} + \varepsilon_i, \quad (1)$$

where, y_i measures 18-month adherence to pharmaceutical j for individual i at time period t (July 2005 to November 2007) conditional on dispensing in t^* (July 2005 to May 2006). X_i is a vector of specific health-related conditions enhancing the achievable health benefit with pharmaceutical treatments. The Z_i vector contains personal characteristics variables that may influence the net treatment benefit, such as general health variables (e.g., age, hospitalization, self-care ability, respiratory disorder, neurological disorder, pain, diabetes, weight, and smoking status), socioeconomic variables (e.g., education level, full-time work, disposable household income and experience of financial stress), demographical variables (e.g., immigration status, marital and cohabiting status, living in a large city, county and having a child or children in the household).

As side-effects are mainly treatment-specific, I estimate the pharmaceutical classes separately. The time used for following the pharmaceutical regimens is minimal, physicians typically issue a prescription card entitling the patient to a 12-month pharmaceutical supply and the medication regimen is simple (usually once-a-day). As access and travel time to health care centers and pharmacies may vary between counties, I use county fixed effects in all regressions. In Sweden, patient out-of-pocket payments for prescriptions are moderate, and annual expenditures exceeding SEK 2200 (SEK 1800 before 2012) are fully reimbursed. As current out-of-pocket payment reflects adherence behavior, I refrain from including pharmaceutical out-of-pocket expenditures. Random variation is captured in the error term ε_i. The OLS and the probit model use the Huber/White/sandwich estimate of variance to produce robust standard errors.

3.6. Results

The descriptive statistics for the dependent variable are shown in table 1 (see appendix 3B for the distribution of adherence). About 50 percent of the men and women are less than perfectly adherent. The exact figure varies between treatments and is 60 per cent for antidepressants in

men and women and 46 per cent and 43 per cent for SBRB in men respectively women. The regression estimates (using eq 1) for pharmaceuticals used in cardiovascular disease treatment are shown in tables 3 to 7, and antidepressants for mental health conditions are shown in tables 8 and 9. The placebo test results, estimating the link between adherence to SBRB and health conditions specifically modifying the therapeutic effect of antidepressants, are shown in tables 10 and 11. As the OLS results are in line with the probit results and the clinical relevance of the probit estimates has more straightforward interpretation than the OLS estimates (e.g., the clinical effect of 7 per cent more adherence depends on the ultimate adherence level), I focus on the probit results.

Table 1: Descriptive statistics for men and women

		Men		
	MPR(mean)	MPR≥80%	MPR≥100%	No. of obs.
SBRB	87%	79%	54%	451
ACE/ARB	86%	55%	41%	358
Diuretics	81%	67%	47%	251
Antidepressants	67%	58%	40%	178

		Women		
	MPR(mean)	MPR≥80%	MPR≥100%	No. of obs.
SBRB	84%	76%	57%	513
ACE/ARB	83%	77%	58%	302
Diuretics	81%	67%	48%	373
Antidepressants	71%	59%	40%	368

3.6.1 Cardiovascular treatment

SBRB

As expected, the results in tables 2 and 3 show that specific health conditions, prior manifested hypertension and heart disease respectively, are positively associated with adherence to SBRB in men and women. For men, table 1 (columns 5 and 7) shows that (i) men with manifested heart disease are 16 per cent more likely to be sufficiently adherent than men without cardiovascular history, and (ii) men with prior manifested heart disease are 15 per cent more likely to be sufficiently adherent or 16 per cent more likely to be perfectly adherent. The perfectly adherent measure is informative as it illustrates that men with manifested heart disease, but not men with

prior manifested hypertension only, are more likely to be perfectly adherent than men without prior cardiovascular history.

Table 3 for women discloses the same pattern: (i) women with prior manifested hypertension are 17 per cent more likely to be sufficiently adherent or 12 per cent more likely to be perfectly adherent than women without cardiovascular history, and (ii) women with prior manifested heart disease are 13 per cent more likely to be sufficiently adherent or 15 per cent more likely to be perfectly adherent than women without cardiovascular history.

Interestingly, self-care ability is negatively associated with adherence in men, but positively associated for women, and marital status matters for adherence in men but not in women (. A possible explanation for these results may be that women supervise their husbands' prescriptions and possibly more so if the husband has self-care deficiencies.

Table 2: Adherence to selective beta receptor blockers (SBRB) in men
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	12.85***	12.78***	14.52***	0.08	0.09	0.16***	0.16***
	(3.44)	(3.47)	(3.80)	(0.06)	(0.06)	(0.04)	(0.04)
Heart disease	13.96***	13.78***	15.05***	0.16***	0.16***	0.15***	0.15***
	(3.29)	(3.39)	(3.71)	(0.06)	(0.06)	(0.04)	(0.04)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	451	451	405	448	448	451	451
R-squared	0.18	0.19	0.19				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C2 for full results.

Table 3: Adherence to selective beta receptor blockers (SBRB) in women
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	13.07***	12.46***	12.85***	0.13**	0.12**	0.18***	0.17***
	(3.06)	(3.09)	(3.18)	(0.05)	(0.05)	(0.04)	(0.04)
Heart disease	12.02***	11.50***	12.32***	0.15**	0.15**	0.14**	0.13**
	(3.59)	(3.64)	(3.77)	(0.07)	(0.07)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	513	513	493	512	512	512	512
R-squared	0.12	0.13	0.13				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1 See appendix C, table C3 for full results.

ACE inhibitors and ARBs

Once again, the specific health conditions are positively associated with adherence to ACE/ARB in men (table 4). Compared to men without cardiovascular history, column 7 shows that (i) men with prior manifested hypertension are 16 per cent more likely to be sufficiently adherent and, (ii) men with prior manifested heart disease are 18 per cent more likely to be sufficiently adherent or 13 per cent more likely to be perfectly adherent. Inconsistently, table 4 for women reveals no positive association between adherence and the specific health condition.

Turning to the ARB-dummy, the parameter estimate is insignificant for both men and women, i.e., there is no difference in adherence to ACE inhibitors and ARBs. This result is expected as these pharmaceuticals have almost identical therapeutic and side-effect profiles[4].

Table 4: Adherence to ACE-inhibitors (ACE) and angiotensin receptor blockers (ARB) in men
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS**$^{\text{Trimmed}}$ MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr MPR≥100	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	11.12***	10.60***	10.47***	0.10	0.10	0.16***	0.16***
	(3.96)	(3.90)	(3.97)	(0.06)	(0.06)	(0.05)	(0.05)
Heart disease	13.30***	12.95***	14.03***	0.14*	0.13*	0.19***	0.18***
	(4.18)	(4.19)	(4.41)	(0.07)	(0.07)	(0.06)	(0.06)
ACE				*Reference*			
ARB	-1.17	-0.69	-0.98	-0.05	-0.02	-0.04	-0.03
	(2.91)	(2.90)	(3.07)	(0.05)	(0.05)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	358	358	336	354	354	342	342
R-squared	0.15	0.17	0.17				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C4 for full results.

[4] The main difference between ACE inhibitors and ARBs and is that ARBs do not produce dry cough. As the prescribing of ARBs in Sweden is restricted to patients who have experienced dry cough with ACE, treatment benefits and side-effects are comparable in people with ACE and ARB.

Table 5: Adherence to ACE-inhibitors (ACE) and angiotensin receptor blockers (ARB) in women
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS**^{Trimmed} MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	-3.76	-3.98	-4.52	-0.05	-0.05	-0.08	-0.09
	(3.73)	(3.83)	(4.10)	(0.07)	(0.06)	(0.06)	(0.06)
Heart disease	-0.59	-1.05	-1.32	-0.01	-0.01	-0.01	-0.02
	(5.15)	(5.13)	(6.01)	(0.09)	(0.09)	(0.08)	(0.08)
ACE				*Reference*			
ARB	0.64	0.08	0.69	-0.01	-0.02	0.03	0.03
	(3.37)	(3.49)	(3.81)	(0.06)	(0.06)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	302	302	278	301	301	294	294
R-squared	0.13	0.17	0.16				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1 See appendix C, table C5 for full results.

Diuretics

Interestingly, the results for diuretics are the opposite of the results for ACE inhibitors and ARBs. That is, adherence to diuretics is positively associated with the specific health conditions in women (table 6) but not in men (table 6).Compared to women without cardiovascular history (i) women with prior manifested hypertension are 12 per cent more likely to be sufficiently adherent and (ii) women with prior manifested heart disease are 18 per cent more likely to be sufficiently or perfectly adherent.

Turning to the loop-diuretic dummy, the parameter estimate shows that men and women adhere less to loop-diuretics than other diuretics. Lower adherence to loop-diuretics may be related to the more pronounced side-effects, e.g., frequent urination, with loop-diuretics than other diuretics

Table 6: Adherence to diuretics in men
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	0.85	0,8	-1.41	-0.02	-0.08	-0.00	-0.04
	(4.27)	(4.48)	(5.09)	(0.08)	(0.08)	(0.07)	(0.07)
Heart disease	7.51	5.47	6.45	0.11	0.08	0.12	0.09
	(5.15)	(5.22)	(5.80)	(0.09)	(0.10)	(0.08)	(0.08)
Non-loop duretics				*Reference*			
Loop diuretics	-11.71***	-11.16**	-12.12**	-0.19**	-0.19**	-0.20***	-0.19***
	(4.42)	(4.55)	(5.12)	(0.08)	(0.08)	(0.07)	(0.07)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	257	257	230	246	246	252	252
R-squared	0.20	0.24	0.24				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C6 for full results.

Table 7: Adherence to diuretics in women
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	9.43***	8.08**	9.11**	0.05	0.05	0.13**	0.12**
	(3.46)	(3.49)	(4.11)	(0.06)	(0.06)	(0.05)	(0.05)
Heart disease	19.25***	18.01***	31.39***	0.17**	0.18**	0.18**	0.18**
	(4.59)	(4.53)	(4.99)	(0.08)	(0.08)	(0.07)	(0.07)
Non-loop duretics				*Reference*			
Loop diuretics	-15.78***	-14.69***	-20.33***	-0.24***	-0.24***	-0.21***	-0.19***
	(3.44)	(3.44)	(4.20)	(0.05)	(0.06)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	371	370	304	371	371	371	371
R-squared	0.21	0.22	0.24				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C7 for full results.

3.6.2 Antidepressants

For antidepressants as well, the results show that the specific health conditions are associated with higher adherence in men and women. For men, table 8 shows that (i) men with prior anxiety are 21 per cent more likely to be sufficiently or perfectly adherent than men with prior good mental health and, (ii) men with mental illness history are 30 per cent more likely to be sufficiently adherent or 21 per cent more likely to be perfectly adherent than men with prior good

mental health. Similarly for women, table 8 shows that women with mental illness history are 14 per cent more likely to be sufficiently adherent than women with previous good mental health.

It is worth mentioning that having some higher education is negatively associated with adherence in men but positively in women, and, again, being married (or cohabiting) is positively associated with adherence in men whereas no such link is apparent in women see table C8 in appendix C).

Table 8: Adherence to antidepressants in men
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimme}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
Previous good mental health				*Reference*			
Anxiety	12.33	12.26	10.69	0.21**	0.21***	0.20*	0.21**
	(7.80)	(7.65)	(8.25)	(0.08)	(0.08)	(0.10)	(0.10)
Mental illness	19.03***	18.42**	21.28***	0.16**	0.21**	0.28***	0.30***
	(6.74)	(7.07)	(7.93)	(0.08)	(0.08)	(0.08)	(0.09)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	178	178	164	168	168	162	162
R-squared	0.26	0.31	0.31				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C8 for full results.

Table 9: Adherence to antidepressants in women
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimme}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
Previous good mental health				*Reference*			
Anxiety	1.26	1.40	1.89	0.05	0.04	-0.01	-0.02
	(3.85)	(4.00)	(4.15)	(0.06)	(0.06)	(0.06)	(0.06)
Mental illness	13.30***	13.24***	16.53***	0.06	0.04	0.15**	0.14**
	(3.71)	(3.72)	(4.17)	(0.06)	(0.06)	(0.06)	(0.06)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	368	368	345	360	360	368	368
R-squared	0.21	0.24	0.23				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1. See appendix C, table C9 for full results.

3.6.3 Placebo test

As expected, the placebo results (tables 10 and 11) show no positive association between adherence to SBRB and the specific health conditions enhancing the therapeutic response with antidepressant treatments. Instead, the association between adherence to SBRB and mental illness history is negative in men.

Table 10: Men, placebo test with adherence to selective beta receptor blockers (SBRB) and specific health conditions augmenting the treatment benefits with antidepressants Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
Previous good mental health				*Reference*			
Anxiety or mental illness history	-7.78**	-8.40**	-10.77**	-0.13**	-0.14**	-0.07	-0.07
	(3.75)	(3.79)	(4.20)	(0.06)	(0.07)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	451	451	388	448	448	451	451
R-squared	0.14	0.16	0.16				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1.

Table 11: Women, placebo test with adherence to selective beta receptor blockers (SBRB) and specific health conditions augmenting the treatment benefits with antidepressants

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr (MPR≥100)	(5) **Probit** Pr (MPR≥100)	(6) **Probit** Pr (MPR≥80)	(7) **Probit** Pr (MPR≥80)
Previous good mental health				*Reference*			
Anxiety or mental illness histor	4.36*	4.36*	4.49*	0.05	0.05	0.07	0.07
	(2.55)	(2.61)	(2.72)	(0.05)	(0.05)	(0.05)	(0.05)
Control for health and education	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*	*Yes*
control for demographics and socioeconomics	*No*	*Yes*	*Yes*	*No*	*Yes*	*No*	*Yes*
No. of observations	513	513	493	512	512	512	512
R-squared	0.08	0.10	0.09				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1.

3.7. Discussion

The results confirm that patient adherence to prescribed pharmaceutical regimens is suitable for analysis within the Bolin and Gustafsson version of the demand-for-health model. The empirical findings in this paper and the Bolin and Gustafsson's theoretical framework may contribute to the applied adherence literature specifically by demonstrating that patient adherence to prescribed pharmaceuticals for maintenance treatment reflects the trade-off between long-term health and short-term quality-of-life objectives i.e., ceteris paribus, people are more adherent when attainable therapeutic returns are greater.

Overall, the specific health conditions are positively associated with adherence, which is in line with economic theory. The OLS and "trimmed" OLS results are in line with the probit estimates. The latter are easier to interpret because the clinical benefit of being, for instance, 10 per cent more adherent is different if adherence increases to 60 per cent or 80 per cent. While 80 per cent adherence may have considerable therapeutic benefits, increasing adherence to 60 per cent may have marginal or no effects on the therapeutic benefit.

This essay shows that people are more adherent when treatment benefits are high, and this holds for cardiovascular diseases and mental health concerns. For example, men and women with prior manifested cardiovascular heart diseases are up to 16 per cent more likely to be perfectly adherent to prescribed SBRB than those without cardiovascular history. I find the same pattern for antidepressants: men with mental illness history are 30 per cent more likely to be sufficiently adherent than men with prior good mental health; the corresponding figure for women is 14 per cent.

There are two exceptions where the associations are statistically insignificant: diuretics in men and ACE/ARB in women. One explanation may be related to the observed differences in treatment pattern: Women are more often dispensed diuretics and no ACE/ARB, while men are more often dispensed ACE/ARB and no diuretics.[5] Diuretics or ACE/ARB are first-line treatments for hypertension control; if the hypertension control is insufficient, diuretics and ACE/ARB can be combined. Given that women predominantly initiate treatment with diuretics and men with ACE/ARB, ACE/ARB complements treatment for women with insufficient hypertension control, and diuretics complement treatment for men. Being partial substitutes, combined usage implies that the second agent has lower marginal utility than the first. As prior cardiovascular events increase the importance of adequate treatment, the specific health

[5] One reason for the differences may be that diuretics alleviate cardiovascular related oedema, (i.e., swelling of e.g., legs and ankles) which is more prevalent in women than in men (Evans et al. 1999)

conditions may be positively associated with combined therapy and, therefore, partly explain the insignificant associations with adherence to diuretic in men, and with ACE/ARB in women.

The placebo test shows no positive association between specific health conditions augmenting treatment outcome with antidepressants and adherence to SBRB for cardiovascular treatment. Instead, the association is negative in men. This supports the assumption that specific health conditions augment particular therapeutic outcomes rather than exerta positive effect on health behaviour in general. If the specific health conditions primarily influence adherence because the marginal returns from health investments are higher when the health capital stock is lower, then we may expect a positive association between the specific health conditions and adherence across all pharmaceutical groups. This is disproved by the placebo regression though. The reason for not conducting additional placebo tests, i.e., estimating the association between cardiovascular illness history and adherence to antidepressants, is that cardiovascular events such as heart attack and stroke may trigger depression and thereby affect adherence positively.

The variables in the model explain up to 31 per cent of the variation in adherence. Overall, the general variables are inconsistently associated with adherence across the pharmaceutical groups. For instance, there is no consistency between level of education and adherence. The link is positive for some pharmaceuticals and negative for others. One reason for the inconsistency may be that there are alternative ways, besides pharmaceutical treatment, to invest in health, and people with different levels of education may be more or less inclined to choose them. For example, life-style changes such as a healthy diet and physical exercise have positive health effects and can, partly, substitute cardiovascular treatment. Alternatives to treatment with antidepressants are e.g,. other medical procedures and psychology sessions.

Regarding other control variables, being married (or cohabitation) or having low self-care ability is generally associated positively with adherence in men. No such difference is apparent between married (or cohabitation) and single women, and self-care ability in women is associated positively with adherence. One imaginable reason may be that women care for their own medication, while wives care for their husband's medication, and do so even more for husbands with self-care deficits. Indicators of low health, such as hospital overnight stay, is positively associated with adherence in men and women, and neurological disorders are positively associated with adherence in women. Age is usually positively associated with adherence but at a decreasing rate.

Using dispensed adherence as a measure for consumed adherence introduces uncertainty about the actual consumed amount. Stockpiling is a major uncertainty factor. This study handles stockpiling bias by estimating 18 adherences. As a prescription card entitles the patient to no

more than a 12-month pharmaceutical supply, patients who stockpile pharmaceuticals and do not remain on therapy are unlikely to renew their prescription. A related concern is that an oversupply of pharmaceuticals can imply that individuals consume higher quantities than prescribed and are therefore not adherent. To avoid such misclassification, the selections of analysed pharmaceuticals are non-addictive, and exceeding the prescribed dosage regimen generates no positive effects; instead, side-effects increase.

That people with higher treatment benefits are more adherent than people with lower health returns is an important finding. Such knowledge is useful for e.g., health professionals when drawing up treatment guidelines. In controlled trials, the trial environment affects adherence behaviour. Thus, the results from such studies may not be suitable for drawing conclusions about factors influencing adherence to long-term therapies in real life. As adherence to pharmaceutical treatments has substantial public health ramifications, affecting public health and the overall disease burden, improving adherence is on the agenda of health policy-makers around the world.

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Appendix A

The control variables are specified as follows:

Underweight, Normal weight and *Overweight* are mutually exclusive dummy variables that take on the value 1 if the respondent is (1) underweight (BMI<18.5), (2) normal weight (18.5≤BMI<25), and (3) overweight (BMI≥25), respectively. In all cases, the variables are 0 otherwise.

Smokes daily is a dummy variable which takes the value 1 if the respondent reported smoking daily in 2005-2005 and 0 otherwise.

Hospital stay 1 overnight and *Hospital stay ≥2 overnights*, are dummy variables. *Hospital stay 1 overnight* takes on the value 1 if the respondent had one overnight hospital admission, and *Hospital stay ≥2 overnights* if the respondent had two or more overnight hospital admissions. In all cases, the variables are 0 otherwise. As overnight obstetrics and gynecology hospital admission may imply maternity care for women 30-45 years of age, such admission is excluded.

Self-care ability is a dummy variable which takes on the value 1 if the respondent reported having self-care ability, and 0 otherwise.

Mobile impairment is a dummy variable which takes on the value 1 if the respondent reported having mobility impairment and 0 otherwise.

Pain is a dummy variable which takes on the value 1 if the respondent reported having pain (e.g. back pain), and 0 otherwise.

In addition, the respondents were asked questions about specific diseases. In cases of a disease, the ULF survey categorized the disease according to WHO's ICD-9 classification.

Respiratory disorder is a dummy variable which takes on the value 1 if the respondent reported a respiratory disease corresponding to at least one diagnosis in the interval 460.0-519.9 according to WHO´s ICD-9, and 0 otherwise.

Skeleton disorder is a dummy variable which takes on the value 1 if the respondent reported a skeleton disease corresponding to at least one diagnosis in the interval 710.0-739.9 according to WHO´s ICD-9, and 0 otherwise.

Neurological disorder is a dummy variable which takes on the value 1 if the respondent reported a neurological disease corresponding to at least one diagnosis in the interval 320.0-389.9 according to WHO´s ICD-9, and 0 otherwise.

Diabetes is a dummy variable which takes on the value 1 if the respondent reported a cardiovascular disease corresponding to at least one diagnosis in the interval 250.0-250.9 according to WHO´s ICD-9, and 0 otherwise.

Age is the respondent's age in year 2006 and the square of age, Age^2 is included to control for a potential nonlinear relationship between age and the dependent variables.

Immigrant is a dummy variable which takes on the value 1 if the respondent was born outside Sweden to non-Swedish born parents, and 0 otherwise.

Primary education, Secondary education, Higher education are mutually exclusive dummy variables that take on the value 1 if the respondent's education level was (1) up to primary school, (2) up to secondary school, (3) some higher education. In all cases, the variables are 0 otherwise.

Married or cohabiting is a dummy variable which takes on the value 1 if the respondent was married or cohabiting, and 0 otherwise.

Log disp. income is the respondent´s logged disposable yearly income (in SEK 100) after taxes and social transfers. For married or cohabiting respondents, Log disp. income is the mean of the spouses' Log disp. income.

Child(ren) in household is a dummy variable which takes on the value 1 if the respondent had one or more children in the household, and 0 otherwise.

Live in a large city is a dummy variable which takes on the value 1 if the respondent lived in Stockholm, Gothenburg or Malmo, and 0 otherwise.

Work full time is a dummy variable which takes on the value 1 if the respondent reported working 40 hours a week or more, and 0 otherwise.

No income from work is a dummy variable which takes on the value 1 if the respondent did not have income from work, and 0 otherwise.

Financial stress is a dummy variable which takes on the value 1 if the respondent reported being unable to pay for unexpected expenses corresponding to 15,000 SEK with one week's notice, and 0 otherwise.

The county variables: *Stockholm; Västra Götaland; Skåne; Östergötland; Jönköping; Uppsala; Gävleborg; Dalarna; Örebro; Halland; Värmland; Norrbotten; Västmanland; Västerbotten; Södermanland; Västernorrland; Kalmar; Kronoberg; Blekinge; Jämtland* and *Gotland* are mutually exclusive dummy variables that take on the value 1 if the respondent lives in the county, and 0 otherwise.

Appendix B

Figure 1 shows the adherence to cardiovascular treatments measured as MPR (untruncated) by number of dispensed pharmaceuticals for men and women

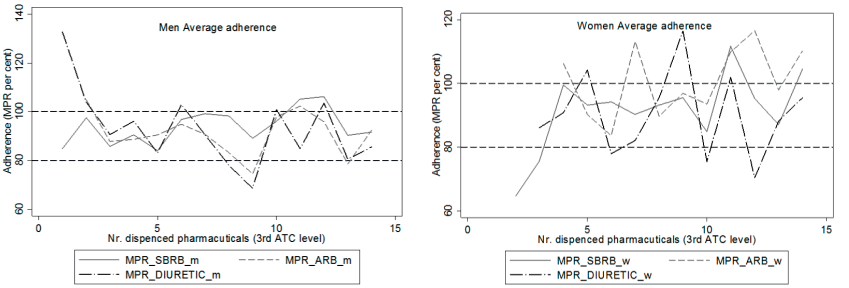
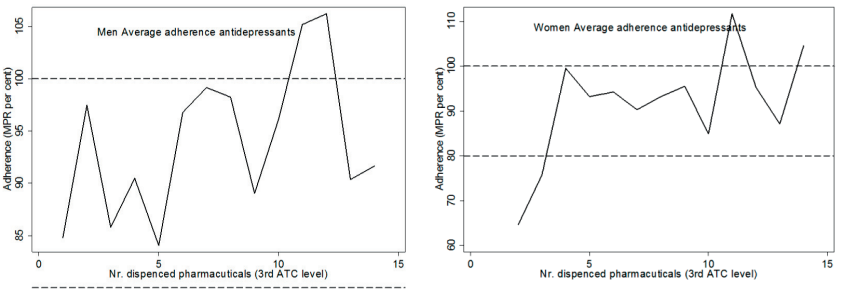


Figure 2 shows the adherence to antidepressants measured as MPR (untruncated) by number of dispensed pharmaceuticals for men and women



Generally, MPR vales greater than 100 seems to be more common in invividuals with multiple pharmacuticals treatments than in individuals with fewer treatments. Given that multiple pharamcutical treatments associates poitively with highe reimbursment levels, MPR-values greater than 100 may indivcate stockpiling.

Appendix C

Table C2: Adherence to selective beta receptor blockers (SBRB) in men
Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	**12.85*** **	**12.78*** **	**14.52*** **	**0.08**	**0.09**	**0.16*** **	**0.16*** **
	(3.44)	(3.47)	(3.80)	(0.06)	(0.06)	(0.04)	(0.04)
Heart disease	**13.96*** **	**13.78*** **	**15.05*** **	**0.16*** **	**0.16*** **	**0.15*** **	**0.15*** **
	(3.29)	(3.39)	(3.71)	(0.06)	(0.06)	(0.04)	(0.04)
Primary education				*Reference*			
Secondary education	-2.87	-2.34	-2.32	0.04	0.06	-0.03	-0.03
	(2.57)	(2.62)	(2.88)	(0.05)	(0.05)	(0.04)	(0.04)
Higher education	-0.57	-0.11	-0.44	0.03	0.05	-0.02	-0.02
	(3.09)	(3.22)	(3.82)	(0.07)	(0.07)	(0.05)	(0.06)
Smokes daily	0.49	0.60	0.76	-0.08	-0.08	0.09	0.09
	(3.30)	(3.37)	(3.92)	(0.07)	(0.07)	(0.06)	(0.06)
Hospital stay 1 overnight	4.80	4.03	4.22	0.15*	0.13*	0.06	0.05
	(4.53)	(4.57)	(5.45)	(0.08)	(0.08)	(0.06)	(0.06)
Hospital stay ≥2 overnights	0.07	0.15	0.06	-0.01	-0.02	0.01	0.01
	(2.39)	(2.46)	(2.64)	(0.05)	(0.05)	(0.04)	(0.04)
Self-care ability	-8.64**	-9.25**	-11.90**	-0.18	-0.21*	-0.10	-0.12
	(4.11)	(4.25)	(5.82)	(0.11)	(0.12)	(0.08)	(0.08)
Mobility impairment	-0.32	-0.88	-1.02	0.04	0.02	0.03	0.04
	(2.36)	(2.46)	(2.75)	(0.06)	(0.06)	(0.05)	(0.05)
Respiratory disorder	-0.14	-1.01	-2.63	0.06	0.05	-0.00	-0.02
	(5.80)	(5.97)	(7.03)	(0.11)	(0.11)	(0.09)	(0.09)
Skeleton disorder	-1.00	-0.54	-0.83	-0.05	-0.03	-0.09*	-0.08*
	(3.13)	(3.13)	(3.48)	(0.06)	(0.06)	(0.05)	(0.05)
Neurological disorder	-5.76	-5.61	-6.74	-0.06	-0.05	-0.10*	-0.09*
	(3.91)	(4.07)	(4.37)	(0.07)	(0.07)	(0.05)	(0.05)
Pain	-1.60	-1.85	-2.54	-0.02	-0.02	-0.04	-0.04

	(2.38)	(2.41)	(2.70)	(0.05)	(0.05)	(0.04)	(0.04)
Diabetes	1.56	1.44	2.05	0.10	0.10	0.05	0.04
	(2.77)	(2.81)	(3.42)	(0.06)	(0.06)	(0.05)	(0.05)
Normal weight				*Reference*			
Overweight or Obese	2.53	2.60	2.82	0.02	0.02	0.07*	0.06
	(2.75)	(2.76)	(3.01)	(0.06)	(0.05)	(0.04)	(0.04)
Underweight	6.40	6.96	11.53	-0.07	-0.05	0.05	0.06
	(5.14)	(5.37)	(7.30)	(0.18)	(0.18)	(0.13)	(0.13)
Age	2.21**	1.92*	2.04*	-0.01***	-0.01**	-0.00**	-0.01**
	(0.92)	(1.05)	(1.08)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	-0.02***	-0.02**	-0.02**				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		0.38	0.38		0.01		0.03
		(2.63)	(2.79)		(0.07)		(0.05)
Full-time work		-2.80	-4.08		-0.02		-0.03
		(3.19)	(3.61)		(0.07)		(0.06)
No income from work		-0.33	-2.28		0.05		-0.04
		(3.60)	(4.12)		(0.08)		(0.07)
Financial stress		-4.51	-7.43		-0.20**		-0.08
		(4.06)	(5.82)		(0.10)		(0.07)
Immigrant		-5.98	-7.15		-0.05		-0.06
		(4.79)	(5.05)		(0.08)		(0.06)
Married or Cohabiting		2.28	2.79		0.10*		0.04
		(2.51)	(2.74)		(0.05)		(0.04)
Child(ren) in household		-4.29	-5.51		-0.11		-0.06
		(5.35)	(5.41)		(0.10)		(0.08)
Large city		0.79	1.11		-0.08		-0.05
		(4.22)	(4.43)		(0.08)		(0.06)
Constant	14.00	28.96	32.35				
	(30.60)	(37.73)	(39.22)				
No. of observations	451	451	405	448	448	451	451
R-squared	0.18	0.19	0.19				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C3: Adherence to selective beta receptor blockers (SBRB) in women

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	**13.07*** **	**12.46*** **	**12.85*** **	**0.13** **	**0.12** **	**0.18*** **	**0.17*** **
	(3.06)	(3.09)	(3.18)	(0.05)	(0.05)	(0.04)	(0.04)
Heart disease	**12.02*** **	**11.50*** **	**12.32*** **	**0.15** **	**0.15** **	**0.14** **	**0.13** **
	(3.59)	(3.64)	(3.77)	(0.07)	(0.07)	(0.05)	(0.05)
Primary education				*Reference*			
Secondary education	0.15	0.15	0.51	0.08	0.08	0.02	0.03
	(2.52)	(2.54)	(2.68)	(0.05)	(0.05)	(0.04)	(0.04)
Higher education	-0.07	0.17	0.77	0.03	0.04	0.02	0.03
	(3.41)	(3.49)	(3.65)	(0.07)	(0.07)	(0.06)	(0.06)
Smokes daily	4.22	4.63	5.39	-0.02	-0.01	0.05	0.04
	(3.36)	(3.44)	(3.85)	(0.06)	(0.06)	(0.06)	(0.06)
Hospital stay 1 overnight	-2.50	-2.61	-3.06	-0.07	-0.07	-0.03	-0.03
	(4.26)	(4.38)	(4.42)	(0.07)	(0.08)	(0.07)	(0.07)
Hospital stay ≥2 overnights	5.14*	5.17*	5.63*	0.04	0.04	0.05	0.05
	(2.72)	(2.77)	(3.01)	(0.05)	(0.05)	(0.04)	(0.04)
Self-care ability	4.09	3.83	4.11	0.09	0.09	0.09	0.10*
	(3.75)	(3.81)	(3.89)	(0.07)	(0.07)	(0.06)	(0.06)
Mobility impairment	-1.11	-1.10	-1.80	0.06	0.05	-0.01	-0.02
	(2.85)	(2.83)	(2.94)	(0.06)	(0.06)	(0.05)	(0.05)
Respiratory disorder	-1.32	-1.08	-1.08	0.07	0.07	-0.04	-0.03
	(5.12)	(5.02)	(5.33)	(0.09)	(0.09)	(0.07)	(0.07)
Skeleton disorder	-0.19	-0.14	0.08	-0.02	-0.01	-0.03	-0.03
	(2.69)	(2.72)	(2.82)	(0.05)	(0.05)	(0.04)	(0.04)
Neurological disorder	-1.70	-1.54	-1.61	-0.02	-0.02	-0.05	-0.05
	(3.94)	(3.97)	(4.14)	(0.06)	(0.06)	(0.06)	(0.06)
Pain	2.10	1.37	1.57	0.04	0.03	0.06	0.05
	(3.09)	(3.09)	(3.19)	(0.05)	(0.05)	(0.05)	(0.05)

Diabetes	-1.12	-0.84	-0.80	-0.09	-0.09	-0.01	-0.00
	(3.81)	(3.86)	(4.04)	(0.07)	(0.07)	(0.06)	(0.06)
Normal weight				*Reference*			
Overweight or Obese	2.50	2.27	2.60	-0.03	-0.04	0.02	0.02
	(2.59)	(2.59)	(2.71)	(0.05)	(0.05)	(0.04)	(0.04)
Underweight	-2.15	-2.12	-2.01	-0.15	-0.16*	0.00	0.00
	(5.19)	(5.27)	(5.44)	(0.09)	(0.09)	(0.08)	(0.08)
Age	1.08	1.03	1.07	0.00	0.00	0.00	0.00
	(0.94)	(1.15)	(1.16)	(0.00)	(0.00)	(0.00)	(0.00)
Age2	-0.01	-0.01	-0.01				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		0.49	0.73		-0.02		-0.00
		(3.22)	(3.25)		(0.05)		(0.04)
Full-time work		-1.07	-1.38		0.00		-0.02
		(4.46)	(4.60)		(0.07)		(0.06)
No income from work		4.31	4.45		0.03		0.09
		(3.30)	(3.43)		(0.06)		(0.06)
Financial stress		4.42	4.69		0.04		-0.01
		(4.20)	(4.30)		(0.07)		(0.06)
Immigrant		5.72*	6.97*		0.12*		0.04
		(3.32)	(3.88)		(0.07)		(0.07)
Married or Cohabiting		0.75	1.13		0.03		0.04
		(2.83)	(2.93)		(0.05)		(0.05)
Child(ren) in household		2.97	2.81		-0.00		0.12*
		(6.02)	(6.08)		(0.10)		(0.06)
Large city		1.52	2.28		-0.01		0.04
		(4.03)	(4.26)		(0.08)		(0.07)
Constant	40.95	34.02	31.39				
	(30.25)	(47.00)	(47.90)				
No. of observations	513	513	493	512	512	512	512
R-squared	0.12	0.13	0.13				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C4: Adherence to ACE-inhibitors (ACE) and angiotensin receptor blockers (ARB) in men

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	**11.12*** **	**10.60*** **	**10.47*** **	**0.10**	**0.10**	**0.16*** **	**0.16*** **
	(3.96)	(3.90)	(3.97)	(0.06)	(0.06)	(0.05)	(0.05)
Heart disease	**13.30*** **	**12.95*** **	**14.03*** **	**0.14***	**0.13***	**0.19*** **	**0.18*** **
	(4.18)	(4.19)	(4.41)	(0.07)	(0.07)	(0.06)	(0.06)
ACE				*Reference*			
ARB	**-1.17**	**-0.69**	**-0.98**	**-0.05**	**-0.02**	**-0.04**	**-0.03**
	(2.91)	(2.90)	(3.07)	(0.05)	(0.05)	(0.05)	(0.05)
Primary education				*Reference*			
Secondary education	1.66	2.23	2.82	-0.05	-0.06	0.03	0.04
	(3.35)	(3.37)	(3.70)	(0.06)	(0.06)	(0.06)	(0.05)
Higher education	0.65	2.37	1.93	0.02	0.00	-0.01	-0.01
	(3.75)	(3.82)	(4.10)	(0.07)	(0.07)	(0.06)	(0.07)
Smokes daily	-0.73	-0.27	-0.72	-0.11	-0.09	-0.10	-0.09
	(3.32)	(3.49)	(3.72)	(0.07)	(0.07)	(0.06)	(0.06)
Hospital stay 1 overnight	-1.83	-1.48	-2.56	-0.00	-0.01	-0.05	-0.06
	(4.83)	(4.77)	(5.24)	(0.09)	(0.09)	(0.08)	(0.08)
Hospital stay ≥2 overnights	-0.29	0.06	-0.11	-0.03	-0.03	-0.02	-0.02
	(2.77)	(2.74)	(3.00)	(0.06)	(0.06)	(0.05)	(0.05)
Self-care ability	-2.60	-0.00	-0.93	-0.12	-0.09	-0.08	-0.04
	(7.62)	(7.89)	(8.45)	(0.13)	(0.12)	(0.11)	(0.11)
Mobility impairment	-3.20	-1.78	-2.18	0.04	0.07	-0.08	-0.05
	(3.39)	(3.39)	(3.69)	(0.07)	(0.07)	(0.06)	(0.06)
Respiratory disorder	-4.17	-4.45	-4.93	0.01	0.01	-0.02	-0.03
	(6.88)	(6.87)	(7.57)	(0.11)	(0.11)	(0.09)	(0.09)
Skeleton disorder	-0.25	-0.65	-0.83	0.03	0.03	0.01	0.01
	(3.64)	(3.66)	(4.00)	(0.07)	(0.07)	(0.06)	(0.06)

Neurological disorder	-4.49	-5.39	-5.08	-0.06	-0.07	-0.07	-0.08
	(4.30)	(4.21)	(4.72)	(0.08)	(0.08)	(0.07)	(0.07)
Pain	-1.87	-1.60	-2.14	-0.01	-0.01	-0.05	-0.05
	(2.89)	(2.94)	(3.14)	(0.06)	(0.06)	(0.05)	(0.05)
Diabetes	0.20	0.25	0.07	0.02	0.02	0.01	0.01
	(3.24)	(3.27)	(3.47)	(0.06)	(0.06)	(0.05)	(0.05)
Normal weight				*Reference*			
Overweight or Obese	-6.04**	-5.90*	-7.08**	-0.09	-0.09	-0.11**	-0.11**
	(3.06)	(3.05)	(3.35)	(0.06)	(0.06)	(0.06)	(0.06)
Underweight	-23.99**	-25.40**	-26.26**	-0.45***	-0.45***	-0.41***	-0.43***
	(11.24)	(11.62)	(11.75)	(0.17)	(0.17)	(0.14)	(0.14)
Age	2.66**	2.49*	2.54*	-0.00	-0.00	-0.00	-0.00
	(1.25)	(1.30)	(1.32)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	-0.02**	-0.02*	-0.02*				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100			1.73		0.10		0.07
			(3.78)		(0.06)		(0.06)
Full-time work		2.51	2.73		-0.07		-0.01
		(4.16)	(4.52)		(0.08)		(0.07)
No income from work		4.09	4.70		0.07		0.05
		(3.79)	(4.17)		(0.08)		(0.07)
Financial stress		8.48	9.11		0.14		0.08
		(5.96)	(6.22)		(0.11)		(0.10)
Immigrant		-8.17*	-7.65		-0.07		-0.09
		(4.76)	(5.02)		(0.08)		(0.07)
Married or Cohabiting		-1.78	-2.30		0.04		-0.04
		(3.36)	(3.64)		(0.06)		(0.06)
Child(ren) in household		-0.60	-0.60		-0.18*		-0.04
		(4.73)	(4.97)		(0.10)		(0.10)
Large city		-0.99	-1.38		-0.09		-0.11
		(4.24)	(4.51)		(0.09)		(0.08)
Constant	3.01	-3.60	-16.54				
	(42.84)	(44.56)	(54.61)				
No. of observations	358	358	336	354	354	342	342
R-squared	0.15	0.17	0.17				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C5: Adherence to ACE-inhibitors (ACE) and angiotensin receptor blockers (ARB) in women

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history					*Reference*		
Hypertension	**-3.76**	**-3.98**	**-4.52**	**-0.05**	**-0.05**	**-0.08**	**-0.09**
	(3.73)	(3.83)	(4.10)	(0.07)	(0.06)	(0.06)	(0.06)
Heart disease	**-0.59**	**-1.05**	**-1.32**	**-0.01**	**-0.01**	**-0.01**	**-0.02**
	(5.15)	(5.13)	(6.01)	(0.09)	(0.09)	(0.08)	(0.08)
ACE					*Reference*		
ARB	**0.64**	**0.08**	**0.69**	**-0.01**	**-0.02**	**0.03**	**0.03**
	(3.37)	(3.49)	(3.81)	(0.06)	(0.06)	(0.05)	(0.05)
Primary education					*Reference*		
Secondary education	-3.23	-4.65	-4.84	-0.02	-0.08	-0.01	-0.04
	(4.08)	(4.11)	(4.52)	(0.07)	(0.07)	(0.06)	(0.06)
Higher education	0.00	-2.83	-3.15	-0.01	-0.09	-0.02	-0.07
	(4.67)	(5.10)	(5.41)	(0.08)	(0.09)	(0.07)	(0.07)
Smokes daily	-5.73	-6.84	-7.69	-0.12	-0.13	-0.09	-0.12
	(5.32)	(5.42)	(5.81)	(0.09)	(0.09)	(0.07)	(0.07)
Hospital stay 1 overnight	0.27	0.96	0.28	0.01	0.02	-0.03	-0.02
	(5.37)	(5.29)	(5.47)	(0.08)	(0.08)	(0.08)	(0.07)
Hospital stay ≥2 overnights	1.64	1.46	1.91	0.05	0.06	-0.01	-0.01
	(4.06)	(4.13)	(4.78)	(0.07)	(0.07)	(0.06)	(0.06)
Self-care ability	6.00	7.88	7.40	0.16*	0.17*	0.09	0.13
	(5.73)	(5.81)	(6.28)	(0.09)	(0.09)	(0.07)	(0.08)
Mobility impairment	-5.03	-3.77	-5.37	0.10	0.14*	-0.08	-0.06
	(4.43)	(4.51)	(5.14)	(0.07)	(0.07)	(0.07)	(0.07)
Respiratory disorder	7.76	6.20	14.74	0.16	0.14	0.11	0.08
	(6.32)	(6.35)	(8.98)	(0.13)	(0.12)	(0.11)	(0.11)
Skeleton disorder	3.60	2.69	2.71	0.02	0.00	0.04	0.04
	(3.93)	(4.04)	(4.46)	(0.07)	(0.07)	(0.06)	(0.06)

Neurological disorder	-8.96	-9.01	-10.86*	-0.02	-0.02	-0.13*	-0.14**
	(5.79)	(5.73)	(6.12)	(0.08)	(0.08)	(0.07)	(0.07)
Pain	-1.17	-1.38	-1.20	0.06	0.05	0.00	0.01
	(4.21)	(4.29)	(4.52)	(0.07)	(0.07)	(0.06)	(0.06)
Diabetes	-1.78	-0.30	-1.24	0.02	0.06	-0.06	-0.03
	(4.65)	(4.67)	(5.05)	(0.08)	(0.08)	(0.06)	(0.06)
Normal weight				*Reference*			
Overweight or Obese	0.62	0.49	0.67	-0.02	-0.03	-0.00	-0.01
	(3.63)	(3.67)	(3.94)	(0.06)	(0.06)	(0.06)	(0.05)
Underweight	-16.72*	-15.81*	-16.97*	-0.03	-0.01	-0.15	-0.15
	(8.96)	(9.09)	(9.27)	(0.11)	(0.11)	(0.09)	(0.10)
Age	0.62	-0.17	-0.02	0.00	0.00	0.00	0.00
	(1.21)	(1.38)	(1.45)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	-0.00	0.00	0.00				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		6.89***	8.07***		0.18**		0.10*
		(2.58)	(2.72)		(0.08)		(0.06)
Full-time work		4.94	4.84		-0.01		0.06
		(5.25)	(5.63)		(0.09)		(0.08)
No income from work		3.54	3.26		-0.00		-0.00
		(4.97)	(5.23)		(0.09)		(0.08)
Financial stress		-3.04	-3.25		0.09		-0.01
		(4.81)	(5.01)		(0.09)		(0.08)
Immigrant		10.24**	17.73***		0.22**		0.17**
		(4.37)	(6.40)		(0.10)		(0.09)
Married or Cohabiting		0.65	2.50		0.04		0.01
		(4.06)	(4.40)		(0.06)		(0.06)
Child(ren) in household		-5.64	-5.20		-0.22*		-0.19
		(7.02)	(7.12)		(0.12)		(0.13)
Large city		-9.85*	-10.82*		-0.13		-0.13*
		(5.20)	(5.88)		(0.10)		(0.08)
Constant	63.01*	35.39	18.46				
	(37.19)	(44.59)	(47.09)				
No. of observations	302	302	278	301	301	294	294
R-squared	0.13	0.17	0.16				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C6: Adherence to diuretics in men

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors (obtained using the Huber/White/sandwich estimate) in parentheses.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	**0.85**	**0,8**	**-1.41**	**-0.02**	**-0.08**	**-0.00**	**-0.04**
	(4.27)	(4.48)	(5.09)	(0.08)	(0.08)	(0.07)	(0.07)
Heart disease	**7.51**	**5.47**	**6.45**	**0.11**	**0.08**	**0.12**	**0.09**
	(5.15)	(5.22)	(5.80)	(0.09)	(0.10)	(0.08)	(0.08)
Non-loop duretics				*Reference*			
Loop diuretics	**-11.71*** **	**-11.16** **	**-12.12** **	**-0.19** **	**-0.19** **	**-0.20*** **	**-0.19*** **
	(4.42)	(4.55)	(5.12)	(0.08)	(0.08)	(0.07)	(0.07)
Primary education				*Reference*			
Secondary education	-7.97**	-6.77*	-8.12*	-0.02	0.02	-0.09	-0.06
	(3.97)	(3.83)	(4.24)	(0.08)	(0.08)	(0.07)	(0.07)
Higher education	-2.26	-0.13	-0.32	0.06	0.12	0.04	0.06
	(5.31)	(5.58)	(6.08)	(0.10)	(0.10)	(0.08)	(0.09)
Smokes daily	-14.52**	-10.45	-11.54	-0.14	-0.11	-0.23**	-0.19*
	(6.26)	(6.92)	(7.03)	(0.11)	(0.11)	(0.09)	(0.10)
Hospital stay 1 overnight	-2.19	-1.35	-2.95	0.05	0.05	0.09	0.12
	(5.26)	(4.95)	(5.79)	(0.11)	(0.11)	(0.09)	(0.08)
Hospital stay ≥2 overnights	0.18	1.42	1.21	-0.04	-0.02	0.04	0.08
	(4.14)	(4.18)	(4.72)	(0.08)	(0.08)	(0.07)	(0.07)
Self-care ability	-10.46*	-13.55**	-14.70*	0.05	0.04	-0.23*	-0.23*
	(6.22)	(6.58)	(7.89)	(0.14)	(0.14)	(0.14)	(0.14)
Mobility impairment	-6.86	-5.51	-6.04	-0.07	-0.07	-0.08	-0.06
	(4.81)	(4.77)	(5.20)	(0.08)	(0.08)	(0.07)	(0.07)
Respiratory disorder	4.48	4.56	4.76	0.01	-0.01	-0.02	-0.01
	(4.87)	(4.60)	(6.75)	(0.13)	(0.12)	(0.10)	(0.09)
Skeleton disorder	7.62*	8.23*	10.27*	0.06	0.07	0.07	0.07
	(4.56)	(4.70)	(5.65)	(0.08)	(0.08)	(0.07)	(0.07)
Neurological disorder	0.74	2.04	2.16	0.12	0.13	-0.00	0.02
	(5.40)	(5.22)	(5.99)	(0.10)	(0.10)	(0.09)	(0.09)
Pain	-0.99	-1.48	-2.57	0.03	0.02	0.02	0.02
	(3.85)	(3.87)	(4.34)	(0.07)	(0.07)	(0.07)	(0.06)
Diabetes	1.96	3.37	2.11	0.10	0.13	0.07	0.09

	(4.75)	(4.87)	(5.69)	(0.08)	(0.08)	(0.08)	(0.08)
Normal weight				*Reference*			
Overweight or Obese	6.68	6.18	7.65*	0.04	0.05	0.06	0.05
	(4.33)	(4.34)	(4.62)	(0.07)	(0.07)	(0.07)	(0.06)
Underweight	-13.17	-14.06	-11.94	-0.14	-0.10	-0.26	-0.25
	(12.21)	(13.91)	(14.65)	(0.23)	(0.23)	(0.19)	(0.20)
Age	-1.15	-0.13	-0.05	0.00	0.01	0.00	0.01
	(1.28)	(1.42)	(1.53)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	0.01	0.00	0.00				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		2.12	2.42		-0.09		0.06
		(2.95)	(3.20)		(0.09)		(0.09)
Full-time work		7.68	10.13		0.21**		0.14
		(5.96)	(6.87)		(0.11)		(0.10)
No income from work		-4.17	-3.88		0.09		-0.04
		(7.28)	(7.83)		(0.11)		(0.10)
Financial stress		0.21	1.10		0.10		-0.05
		(6.71)	(7.35)		(0.11)		(0.09)
Immigrant		-5.80	-5.29		-0.11		-0.18
		(9.20)	(10.15)		(0.14)		(0.12)
Married or Cohabiting		2.14	0.83		0.00		-0.04
		(4.17)	(4.62)		(0.08)		(0.06)
Child(ren) in household		6.34	12.13		-0.04		0.11
		(7.22)	(9.14)		(0.15)		(0.12)
Large city		-12.91*	-14.90*		-0.26**		-0.24**
		(7.05)	(7.69)		(0.11)		(0.10)
Constant	127.56***	72.68	65.29				
	(46.50)	(58.26)	(63.72)				
No. of observations	257	257	230	246	246	252	252
R-squared	0.20	0.24	0.24				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C7: Adherence to diuretics in women

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80). Heteroscedasticity robust standard errors in parentheses.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	**OLS**	**OLS^{Trimmed}**	**Probit**	**Probit**	**Probit**	**Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
No cardiovascular history				*Reference*			
Hypertension	**9.43*** **	**8.08** **	**9.11** **	**0.05**	**0.05**	**0.13** **	**0.12** **
	(3.46)	(3.49)	(4.11)	(0.06)	(0.06)	(0.05)	(0.05)
Heart disease	**19.25*** **	**18.01*** **	**31.39*** **	**0.17** **	**0.18** **	**0.18** **	**0.18** **
	(4.59)	(4.53)	(4.99)	(0.08)	(0.08)	(0.07)	(0.07)
Non-loop duretics				*Reference*			
Loop diuretics	**-15.78*** **	**-14.69*** **	**-20.33*** **	**-0.24*** **	**-0.24*** **	**-0.21*** **	**-0.19*** **
	(3.44)	(3.44)	(4.20)	(0.05)	(0.06)	(0.05)	(0.05)
Primary education				*Reference*			
Secondary education	-8.21**	-8.51**	-14.27***	-0.07	-0.07	-0.12**	-0.11**
	(3.26)	(3.31)	(4.02)	(0.06)	(0.06)	(0.05)	(0.05)
Higher education	-6.66	-7.01	-12.47**	-0.07	-0.07	-0.13*	-0.12*
	(4.16)	(4.27)	(4.98)	(0.07)	(0.08)	(0.07)	(0.07)
Smokes daily	-1.22	-0.56	-1.25	-0.19**	-0.20**	-0.05	-0.05
	(4.71)	(4.95)	(5.50)	(0.08)	(0.08)	(0.07)	(0.08)
Hospital stay 1 overnight	-2.96	-3.54	-4.50	0.01	0.00	-0.06	-0.08
	(4.88)	(5.09)	(5.71)	(0.08)	(0.08)	(0.08)	(0.08)
Hospital stay ≥2 overnights	-1.57	-1.38	-2.07	0.01	0.01	-0.03	-0.03
	(3.95)	(3.97)	(4.74)	(0.06)	(0.06)	(0.06)	(0.06)
Self-care ability	5.04	4.87	8.42	0.08	0.08	0.06	0.06
	(5.24)	(5.38)	(6.26)	(0.08)	(0.08)	(0.08)	(0.08)
Mobility impairment	5.98*	7.32**	14.60***	0.15***	0.16***	0.07	0.08
	(3.38)	(3.57)	(4.66)	(0.06)	(0.06)	(0.06)	(0.06)
Respiratory disorder	3.03	1.97	2.70	-0.04	-0.04	0.05	0.04
	(4.56)	(4.63)	(5.18)	(0.08)	(0.08)	(0.07)	(0.07)
Skeleton disorder	-7.42**	-7.96**	-10.76**	-0.07	-0.07	-0.13**	-0.13**
	(3.43)	(3.44)	(4.20)	(0.06)	(0.06)	(0.06)	(0.06)
Neurological disorder	12.30***	12.10***	15.03***	0.22***	0.23***	0.20***	0.21***

	(3.96)	(3.93)	(5.41)	(0.08)	(0.07)	(0.08)	(0.07)
Pain	1.31	1.99	4.01	0.01	0.01	0.02	0.02
	(3.24)	(3.26)	(3.84)	(0.06)	(0.06)	(0.06)	(0.06)
Diabetes	4.92	5.64	8.34	0.17**	0.19**	0.07	0.07
	(5.43)	(5.39)	(5.86)	(0.08)	(0.08)	(0.08)	(0.08)
Normal weight				*Reference*			
Overweight or Obese	-0.93	-0.99	-2.31	0.01	0.01	0.02	0.01
	(3.28)	(3.27)	(3.68)	(0.06)	(0.06)	(0.05)	(0.05)
Underweight	3.76	2.75	4.77	0.01	0.02	0.10	0.10
	(5.01)	(4.94)	(6.51)	(0.10)	(0.10)	(0.09)	(0.09)
Age	-0.72	-1.09	-0.64	-0.00	-0.00	-0.00	0.00
	(1.30)	(1.56)	(1.74)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	0.00	0.01	0.00				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		3.19	4.02		0.01		-0.01
		(4.31)	(4.69)		(0.07)		(0.07)
Full-time work		0.11	-2.59		-0.01		0.05
		(4.79)	(5.70)		(0.09)		(0.08)
No income from work		-5.67	-9.17		-0.05		-0.06
		(4.94)	(5.67)		(0.08)		(0.07)
Financial stress		4.18	8.01		0.04		-0.05
		(5.55)	(6.25)		(0.08)		(0.07)
Immigrant		1.62	1.84		-0.16**		0.02
		(4.16)	(4.92)		(0.08)		(0.07)
Married or Cohabiting		2.16	-0.05		-0.04		0.06
		(3.31)	(3.91)		(0.06)		(0.05)
Child(ren) in household		-3.35	-0.26		0.09		-0.05
		(9.29)	(10.45)		(0.14)		(0.14)
Large city		3.07	1.66		0.04		0.11
		(5.07)	(5.88)		(0.08)		(0.08)
Constant	102.44**	86.52	68.18				
	(44.36)	(62.18)	(68.13)				
No. of observations	371	370	304	371	371	371	371
R-squared	0.21	0.22	0.24				

Table C8: Adherence to antidepressants in men

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80 Heteroscedasticity robust standard errors in parentheses.

VARIABLES	(1) **OLS** MPR [0-100]	(2) **OLS** MPR [0-100]	(3) **OLS^{Trimmed}** MPR [0-100]	(4) **Probit** Pr(MPR≥100)	(5) **Probit** Pr(MPR≥100)	(6) **Probit** Pr(MPR≥80)	(7) **Probit** Pr(MPR≥80)
Previous good mental health				*Reference*			
Anxiety	**12.33**	**12.26**	**10.69**	**0.21****	**0.21*****	**0.20***	**0.21****
	(7.80)	(7.65)	(8.25)	(0.08)	(0.08)	(0.10)	(0.10)
Mental illness	**19.03***	**18.42****	**21.28*****	**0.16****	**0.21****	**0.28*****	**0.30*****
	(6.74)	(7.07)	(7.93)	(0.08)	(0.08)	(0.08)	(0.09)
Primary education				*Reference*			
Secondary education	-6.55	-7.52	-10.75	-0.16**	-0.22***	-0.09	-0.14
	(7.57)	(7.78)	(8.50)	(0.08)	(0.08)	(0.09)	(0.09)
Higher education	1.23	-2.56	-6.90	0.00	-0.09	-0.01	-0.12
	(7.52)	(7.87)	(8.60)	(0.09)	(0.09)	(0.10)	(0.10)
Smokes daily	2.89	5.91	4.87	0.26***	0.30***	0.07	0.11
	(7.87)	(8.17)	(8.68)	(0.08)	(0.08)	(0.10)	(0.10)
Hospital stay 1 overnight	-3.88	-1.14	-2.17	-0.11	-0.11	-0.00	0.07
	(11.52)	(12.75)	(13.83)	(0.11)	(0.11)	(0.14)	(0.13)
Hospital stay ≥2 overnights	4.90	4.74	7.88	-0.18**	-0.18**	0.12	0.10
	(7.21)	(7.39)	(7.54)	(0.08)	(0.08)	(0.10)	(0.09)
Self-care ability	-14.65	-16.55	-24.13*	0.08	0.05	-0.06	-0.07
	(11.77)	(11.60)	(12.85)	(0.17)	(0.17)	(0.20)	(0.18)
Mobility impairment	-15.99*	-19.75**	-24.69**	-0.07	-0.09	-0.15	-0.15
	(9.25)	(9.85)	(10.70)	(0.12)	(0.12)	(0.13)	(0.13)
Respiratory disorder	-3.00	-0.67	0.15	0.09	0.06	-0.05	-0.02
	(10.90)	(11.28)	(11.71)	(0.12)	(0.10)	(0.13)	(0.12)
Skeleton disorder	6.60	3.25	5.07	0.14	0.16	0.16	0.12
	(8.45)	(8.38)	(9.40)	(0.11)	(0.11)	(0.12)	(0.11)
Neurological disorder	0.03	-1.92	-3.56	0.02	0.03	-0.00	-0.02
	(8.12)	(8.30)	(11.22)	(0.09)	(0.09)	(0.11)	(0.10)
Pain	-3.56	-4.57	-5.53	-0.09	-0.14*	-0.11	-0.13
	(6.50)	(7.09)	(8.00)	(0.07)	(0.07)	(0.09)	(0.08)

Diabetes	6.61	5.04	3.09	-0.09	-0.11	0.13	0.11
	(13.31)	(13.36)	(13.87)	(0.20)	(0.18)	(0.17)	(0.15)
Normal weight	*Referenc*						
Overweight or Obese	7.50	5.54	8.41	0.12*	0.11	0.14*	0.09
	(6.63)	(6.88)	(7.45)	(0.07)	(0.07)	(0.08)	(0.08)
Underweight	5.18	6.06	10.83				
	(15.15)	(16.04)	(18.95)				
Age	-0.55	-1.25	-1.59	0.00	0.00	-0.00	-0.01
	(1.50)	(1.57)	(1.74)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	0.00	0.01	0.01				
	(0.01)	(0.01)	(0.02)				
Log disp. income in SEK 100		9.47**	10.64***		0.21**		0.22**
		(3.82)	(3.92)		(0.09)		(0.09)
Full-time work		-3.47	-3.94		0.08		-0.08
		(7.76)	(8.45)		(0.09)		(0.10)
No income from work		10.83	17.55*		0.14		0.09
		(8.58)	(9.64)		(0.10)		(0.12)
Financial stress		0.08	0.50		0.09		-0.07
		(8.71)	(9.26)		(0.11)		(0.11)
Immigrant		0.47	0.46		0.21*		0.05
		(12.51)	(13.93)		(0.13)		(0.15)
Married or Cohabiting		12.59*	15.62*		0.04		0.20**
		(7.05)	(8.02)		(0.07)		(0.08)
Child(ren) in household		-5.93	-6.33		-0.10		-0.16*
		(7.39)	(7.98)		(0.08)		(0.09)
Large city		-5.69	-5.94		0.00		-0.00
		(9.84)	(10.20)		(0.09)		(0.11)
Constant	85.66*	35.92	41.61				
	(40.00)	(47.77)	(51.12)				
No. of observations	178	178	164	168	168	162	162
R-squared	0.26	0.31	0.31				

Note: All regressions controls for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Table C9: Adherence to antidepressants in women

Average marginal effects (A.M.E.) on 18-month adherence measured as Medication Position Ration (MPR), the probability of being perfectly adherent (MPR≥100) and sufficiently adherent (MPR≥80 Heteroscedasticity robust standard errors in parentheses.

	(1) **OLS**	(2) **OLS**	(3) **OLS^{Trimmed}**	(4) **Probit**	(5) **Probit**	(6) **Probit**	(7) **Probit**
VARIABLES	MPR [0-100]	MPR [0-100]	MPR [0-100]	Pr(MPR≥100)	Pr(MPR≥100)	Pr(MPR≥80)	Pr(MPR≥80)
Previous good mental health				*Reference*			
Anxiety	**1.26**	**1.40**	**1.89**	**0.05**	**0.04**	**-0.01**	**-0.02**
	(3.85)	(4.00)	(4.15)	(0.06)	(0.06)	(0.06)	(0.06)
Mental illness	**13.30*** **	**13.24*** **	**16.53*** **	**0.06**	**0.04**	**0.15** **	**0.14** **
	(3.71)	(3.72)	(4.17)	(0.06)	(0.06)	(0.06)	(0.06)
Primary education				*Reference*			
Secondary education	12.25***	13.23***	16.64***	0.06	0.06	0.16**	0.18***
	(4.37)	(4.46)	(4.85)	(0.06)	(0.06)	(0.06)	(0.06)
Higher education	7.77	9.38*	12.16**	0.07	0.06	0.08	0.10
	(4.97)	(5.48)	(5.80)	(0.07)	(0.07)	(0.07)	(0.07)
Smokes daily	2.30	1.78	1.95	0.09	0.09	0.09	0.09
	(4.11)	(4.17)	(4.40)	(0.06)	(0.06)	(0.06)	(0.06)
Hospital stay 1 overnight	6.67	6.23	6.03	-0.06	-0.05	0.15*	0.15*
	(5.78)	(5.54)	(5.98)	(0.09)	(0.08)	(0.08)	(0.08)
Hospital stay ≥2 overnights	9.21**	9.07**	9.03**	0.08	0.06	0.11*	0.09
	(3.94)	(4.00)	(4.24)	(0.06)	(0.06)	(0.06)	(0.06)
Self-care ability	-4.18	-3.00	-2.09	-0.09	-0.08	-0.07	-0.07
	(5.96)	(5.83)	(6.19)	(0.09)	(0.09)	(0.10)	(0.09)
Mobility impairment	7.01	7.75*	9.61*	0.02	0.03	0.04	0.05
	(4.32)	(4.44)	(4.99)	(0.07)	(0.07)	(0.07)	(0.07)
Respiratory disorder	-4.32	-3.33	-3.64	-0.05	-0.04	-0.13*	-0.12
	(5.24)	(4.89)	(4.92)	(0.08)	(0.08)	(0.08)	(0.07)

Skeleton disorder	-2.71	-3.00	-3.31	-0.03	-0.03	-0.04	-0.05
	(3.95)	(3.93)	(4.16)	(0.06)	(0.06)	(0.06)	(0.06)
Neurological disorder	9.50*	8.65*	10.96*	0.10	0.10	0.16*	0.14
	(5.15)	(5.17)	(6.57)	(0.09)	(0.08)	(0.09)	(0.09)
Pain	-2.68	-1.96	-3.47	-0.02	-0.02	-0.03	-0.02
	(4.02)	(4.06)	(4.42)	(0.06)	(0.06)	(0.06)	(0.06)
Diabetes	-0.17	-0.79	1.31	0.09	0.12	0.03	0.02
	(8.54)	(8.82)	(9.79)	(0.13)	(0.13)	(0.13)	(0.13)
Normal weight				*Reference*			
Overweight or Obese	1.38	1.11	0.72	-0.03	-0.02	0.06	0.05
	(3.48)	(3.47)	(3.70)	(0.05)	(0.05)	(0.05)	(0.05)
Underweight	1.64	3.32	2.78	-0.03	-0.07	0.08	0.07
	(7.33)	(7.07)	(7.49)	(0.13)	(0.13)	(0.11)	(0.11)
Age	1.49*	1.96**	1.97**	0.01***	0.01***	0.01***	0.01***
	(0.78)	(0.86)	(0.93)	(0.00)	(0.00)	(0.00)	(0.00)
Age^2	-0.01	-0.01*	-0.01				
	(0.01)	(0.01)	(0.01)				
Log disp. income in SEK 100		-9.01	-9.97		-0.03		-0.14
		(6.01)	(6.40)		(0.09)		(0.09)
Full-time work		-1.97	-1.24		0.04		-0.01
		(4.31)	(4.61)		(0.06)		(0.06)
No income from work		-1.55	-0.58		-0.13*		-0.05
		(4.32)	(4.74)		(0.07)		(0.07)
Financial stress		-0.01	0.93		-0.16**		-0.07
		(4.29)	(4.59)		(0.07)		(0.07)
Immigrant		-15.11***	-16.65***		-0.18**		-0.21***
		(5.30)	(5.44)		(0.08)		(0.08)
Married or Cohabiting		-5.17	-5.26		-0.07		-0.08
		(3.63)	(3.91)		(0.05)		(0.05)
Child(ren) in household		1.89	1.72		-0.05		-0.00
		(4.63)	(4.79)		(0.07)		(0.07)
Large city		5.01	5.76		0.07		0.06
		(6.02)	(6.22)		(0.08)		(0.08)
Constant	7.98	66.76	67.06				
	(23.60)	(46.25)	(47.75)				
No. of observations	368	368	345	360	360	368	368
R-squared	0.21	0.24	0.23				

Note: All regressions control for county fixed effects. *** p<0.01, ** p<0.05, * p<0.1

Chapter 4

Pharmaceutical-based health investment differences between immigrants and natives in Sweden

With Thomas Eriksson*

4.1 Introduction

Differences in healthcare utilization (Rue et al., 2008; Wamala et al., 2007) and health (Denktaş et al., 2010; Morgan et al., 2011) have been observed between immigrants and natives worldwide. In the Swedish case, health insurance covers all (legal) residents in order to reduce financial barriers to healthcare. The county councils fully subsidized individual payments exceeding SEK 2,200 (~USD 330) for prescribed pharmaceuticals on a 12-month rolling basis in 2012. Nevertheless, differences in the utilization of prescribed pharmaceuticals (Nordin, Dackehag, and Gerdtham., 2013; Sundquist, 1993), as well as health care utilization in general (Westin et al., 2004; Wamala et al., 2007), are observed between natives and immigrants in Sweden. These findings may imply that the goal of Swedish health policy, i.e., all (legal) residents should have equal access to medical care according to need, is unachieved.

Pharmaceutical treatment is the dominating medical intervention available for several health conditions, and is often immensely important for the course of the disease. For instance, pharmaceutical treatments substantially reduce cardiovascular related morbidity and mortality (WHO, 2012). Therefore, disparities in pharmaceutical utilization between population groups may have significant public health consequences.

Despite the fact that immigrants constituted 15% of the Swedish population in 2011 (Statistics Sweden, 2011), and that immigrants' health has considerable consequences for general public health and healthcare expenditures, little has been written to explore differences in pharmaceutical utilization between immigrants and natives in Sweden.

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To explore the immigrant effect on pharmaceutical utilization, we need to adjust for underlying health differences. Studies not doing this (e.g., Sundquist, 1993) are less suited as a basis for designing public health policies to prompt access according to medical need. Nordin, Dackehag, and Gerdtham (2013) adjust for such health differences, but focus on pharmaceutical utilization by socioeconomic status rather than immigration. Using the Swedish Prescribed Drug Register (SPDR) and the Swedish Survey of Living Conditions (ULF), our paper analyzes differences in dispensed pharmaceuticals between immigrants and natives in Sweden, after controlling for health and socioeconomic characteristics. The detailed individual-level data enables us not only to explore the differences in pharmaceutical access between natives and immigrants, but also to disentangle differences related to health and socioeconomic status, and from other factors related to immigration.

The paper proceeds as follows: Section 4.2 provides a brief background of immigrants in Sweden. Section 4.3 introduces the demand-for-health framework. Section 4.4 presents the dataset and the pharmaceutical classification system. Section 4.5 describes the empirical specification. Section 4.6 contains the results and section 4.7 concludes the paper.

Heterogeneity in region of origin across immigrants

"The healthy immigrant effect" (Marmot et al. 1984) – where immigrants, due to self-selection, are on average healthier than natives – has been proposed as an explanation for observed disparities in healthcare utilization between immigrants and natives. However, this has been refuted by findings showing that immigrants are disadvantaged in several health-related aspects. For instance, compared with natives, immigrants have (1) lower self-rated health (Lindström, Sundquist, and Östergren, 2001), (2) higher risk of cardiovascular diseases (Gadd et al., 2005), (3) higher overall mortality (Sundquist and Johansson, 1997), (4) higher rate of suicide (Johansson et al., 1997; Ferrada-Noli, 1997), and (5) higher prevalence of psychiatric illness (Bayard-Burfield, Sundquist, and Johansson 2001).

Instead, we propose that differences in prescribed pharmaceutical utilization between immigrants and natives may follow country-specific knowledge differences, such as the ability to speak the native language and knowledge of the healthcare system.

We also expect heterogeneity in access to prescription pharmaceuticals among immigrant groups. The reason is labor market integration differences among immigrant groups, and that labor market participation is of importance for the "value of healthy time". To illustrate, the main reason for migration to Sweden has changed over time. Between the Second World War and up

to the early 1970s, Sweden experienced a labor shortage. As immigration was predominantly labor-related, immigrants were largely employed in the Swedish labor market. Since the 1970s, though, the Swedish labor shortage has decreased, and the composition of immigrants has changed into mainly refugees and family-related immigrants. In contrast to the labor-related immigrants, refugees more often face harsh labor-market opportunities in Sweden. (Ekberg, 2011)

4.3 Theoretical framework

We specify and interpret the empirical analyses within the demand-for-health framework (Grossman, 1972), which is the dominating economic theory of individual health-related behavior. The demand-for-health framework refines Becker´s human-capital theory (1964) by distinguishing health from the educational component of human-capital. The ground for the distinction is that health is considered to govern the flow of productive time the individual has available for market and non-market participation – the investment aspect of health – while education affects the obtained efficiency per participated time unit.

The health-capital stock wears down with time. To maintain health, and thus avoid or postpone shortfalls in the flow of productive time, the individual can produce health investments by means of combining own time with market goods such as medical care. Although the input of own time may seem negligible for producing, e.g., pharmaceutical-based health investments, time effort is essential beyond pharmaceutical intake per se, and includes, for instance, health information gathering and medical consultations.

Regarding productivity, the demand-for-health framework postulates that education influences the ability to combine and transform own time and market goods into health. In terms of pharmaceutical-based health investments, education may help the individual to comprehend complex medical information and navigate the healthcare system; thus, obtaining a specific pharmaceutical treatment may be less demanding for individuals with more education than for those with less education. Education should be interpreted broadly as incorporating not only formal education, but also country-specific knowledge like the ability to speak the native language. Language barriers and inadequate healthcare system knowledge have been identified as barriers to healthcare access (Priebe et al., 2011). To illustrate, if language difficulties hinder the patient from articulating relevant health information, the physician's may be less able to correctly diagnose and treat the patient. Such misdiagnosis may lead to additional consultations or fewer

prescriptions. Accordingly, pharmaceutical-based health investments may be more effort-demanding for immigrants than for natives.

Discrimination is another factor that may affect pharmaceutical access. For instance, perceived discrimination in healthcare has been shown to discourage some immigrants from seeking health care (Wamala et al., 2007), while discrimination against immigrants in the Swedish labor market (Carlsson and Rooth, 2007) implies that they have fewer opportunities to convert health capital into salaried labor supply, in turn lowering the incentive for them to invest in health.

4.4 Data and pharmaceutical classification system

Data

We used the HILDA (Health and Individuals Longitudinal Data and Analysis) dataset, which combines the Swedish Survey of Living Conditions (ULF) and the Swedish Prescribed Drug Register (SPDR). The ULF survey asks about socioeconomic and labor-market situations, among other things. The responses are complemented with national registry data on such things as taxes and monetary transfers. The ULF-survey years 2004 and 2005 focused on health by asking a wide array of health-related questions. Such detailed health information, together with national register data, opened up the possibility of analyzing disparities in pharmaceutical utilization by population groups with respect to differences in health. Disregarding health differences might imply that the observed disparities in pharmaceutical utilization reflect differences in need rather than population groups with comparable health receiving unequal treatment.

The ULF-survey years 2004 and 2005 had a 75% response rate, which generated 10,179 respondents (Statistics Sweden, 2012). At the time of the survey, respondents were aged 16 or older and representative of the Swedish population in that corresponding age segment. Because education affects health behavior, we control for level of education in all our analyses. As the youngest respondents were too young to enter our supreme educational level, *two or more years of higher education*, we focus on respondents in the more feasible age group 25 and older. This restriction generates a working sample of 8,488 respondents, 48% and 11% of whom are men and immigrants, respectively.

The SPDR registers *all* prescribed, dispensed pharmaceuticals in Sweden, with detailed information on e.g. substance, volume, and prescriber information (e.g. profession and practice)[1].

[1] The SPDR registers do not include information on unclaimed prescriptions. A previous study shows that the proportion of unclaimed electronic prescriptions at pharmacies in Sweden is low (2.4 per cent) (Ekedahl and Månsson, 2004)

As of July 2005, the register includes the patients' personal identification number, which enables merging the data with the ULF-survey. For a more detailed description of the SPDR register, see for example Wettermark et al., (2007). At the time of data collection, the SPRD with personal identification numbers was available for July 2005 through November 2007.

Pharmaceutical classification system

As we are interested in pharmaceutical-based health investment quantities, we utilize the standardized pharmaceutical measurement unit, defined daily doses (DDD). The WHO Collaborating Centre for Drug Statistics Methodology (WHOCC) assigns active pharmaceutical substances as DDD, which by definition is the assumed daily maintenance dose when the pharmaceutical substance is used for its main therapeutic purpose by adults. When measuring health investment quantities, the DDD measure is superior to other common measurement units like the number of unique pharmaceuticals, because it gives the number of daily doses. To illustrate, the number of attended gym classes is supposedly a more suitable health investment measure than the number of health club memberships.

In addition to the health investment quantities, we are interested in the type of pharmaceutical, as pharmaceuticals have different abilities to affect health. We distinguish between pharmaceutical types by adopting WHOCC's Anatomical, Therapeutic and Chemical (ATC) classification system. This five-level ATC code hierarchically groups pharmaceuticals according to chemical properties and the target system or organ. The first ATC-level specifies the pharmaceuticals' main target anatomical system (e.g. cardiovascular system, nervous system), and the second level systemizes the pharmaceuticals in therapeutic subgroups. Still, the second level is broad, encompassing pharmaceuticals with wide-ranging therapeutic indications. The third ATC-level gives the pharmacological subgroup and contains pharmaceuticals with more homogenous indications. For example, despite the fact that Aspirin® (acetylsalicylsyra) and Alvedon® (paracetamol) are not fully interchangeable, both offer pain relief and belong to the same third ATC-level. The fourth and fifth ATC-levels further separate pharmaceuticals according to their chemical subgroup and substances. As we are interested in the pharmaceuticals' ability to affect health, rather than the chemical compound per se, distinguishing between pharmaceuticals at the third ATC-level suits our purpose. Partly due to sample size restrictions, using a higher ATC-level would produce empirical results which are difficult to interpret.

When using dispensed pharmaceuticals as a proxy for consumed pharmaceuticals, the length of the study period affects the validity of the proxy. Generally, the validity is higher for longer

study periods than for shorter ones. This is because dispensed pharmaceuticals are typically consumed over a period of time, which may stretch to month after the actual dispensing. If patients stockpile pharmaceuticals, the time gap between dispensed and consumed pharmaceuticals could be even greater.

Having data on dispensed pharmaceuticals from July 2005 through November 2007, we use the entire period in our analyses. This study period length is reasonable when considering that the Swedish reimbursement system for prescription pharmaceuticals, on a 12 month rolling basis, exempts patients from further copayment on reaching the copayment cap, and that pharmaceutical stockpiling is more common in patients excepted from, than with, co-payment. Given that financial objectives motivate stockpiling behavior, patients likely consume from the stock before purchasing more pharmaceuticals with initial full copayment. As our study period comprises several reimbursement periods, our results are less sensitive to variations in dispensing behavior than studies with shorter study periods.

Dependent variables

We measure pharmaceutical-based health investments by the access-no access dichotomy and, if accessed, the accessed number of DDDs.

Independent variables

We define immigrant as a Swedish resident born abroad to non-Swedish-born parents. We use two immigration measures: (1) *immigrant,* which is a dummy variable taking the value 1 if immigrant and 0 otherwise, and (2) immigrant region of origin i.e. *Nordic origin*, *Western origin* and *non-Western origin,* which for immigrants is a set of mutually exclusive dummy variables; *Nordic origin* takes the value 1 if born in Denmark, Finland, Iceland or Norway and 0 otherwise; *Western origin*[2] takes the value 1 if born in Belgium, France, Greece, Ireland, Italy, Great Britain and Northern Ireland, Germany, Austria, USA, Portugal, The Netherlands, Switzerland or Spain and 0 otherwise; *non-Western origin* takes the value 1 if immigrant region of origin is neither *Nordic* nor *Western* and 0 otherwise.

On a conceptual level, individual characteristics like age, education and health-state influence the "price" and "benefit" for health, and accordingly the demand for prescribed pharmaceuticals. Thus, we include controls for age, education and health-state (e.g. self-assessed health, life style

[2] We use the same definition of western immigrant as Andersson and Wadensjö (2007) with the addition of immigrants from the United States.

factors and the occurrence of specific medical conditions). Because financial and time budget constraints also influence such demand, we control for disposable household income, full-time work and children in the household. Moreover, we control for additional factors (married or cohabitant, residency in larger cities and 2nd generation immigrant) that may be associated with health-related behavior and attitude to pharmaceutical utilization. The control variables are described more in depth in Appendix A.

Descriptive statistics

Table 1 reports the descriptive statistics. Our working sample consists of 8,488 individuals, of whom 48% are men and 11% are immigrants. For immigrants, the most common region of origin is non-Western (54%), followed by Nordic (33%) and Western (12%), and the most common countries or birth are Finland (22%), former Yugoslavia (12%), Iraq (6%) and Iran (5%).

In terms of dispensed pharmaceuticals, the respondents in our sample are comparable to the population in general[3]. Comparing population groups within our sample, the descriptive statistics indicate some differences between immigrants and natives. For instance, despite the fact that the immigrants in our sample reported lower self-assessed health than natives on average, immigrant women accessed on average 1,650 DDDs (unconditional on access), whereas native women accessed 1,896 DDDs on average.

[3] In 2005, the Swedish national mean was 1,542 DDDs per 1000 individuals and per day (The Swedish National Board of Health and Welfare, 2008), which corresponds to 1,434 DDDs per individual and per 31 months. As older people on average utilize more pharmaceuticals than younger people, the figure is presumably larger for individuals aged 25 or older.

Table 1: Descriptive statistics for men and women

		Women (N=4410), mean or percentage				**Men** (N=4078), mean or percentage			
Variables	Description	Natives N=3907		Immigrants N=503		Natives N=3639		Immigrants N=439	
Dependent variable (June 2005-Nov 2007)									
DDD[a,b]	Daily Defined Doses	1,896	(3,344)	1,650	(3,151)	1,414	(2,867)	1,455	(2,736)
Independent variables (2004-2005)									
Age[a]	Age (in years) and age^2	52.7	(17.3)	50.4	(14.9)	51.3	(16.2)	49.2	(14.5)
Ln(dispinc)[a]	log of disposable income	7.4	(0.5)	7.3	(0.8)	7.4	(0.5)	7.1	(0.9)
SAH 1	Very good health	34.0%		29.2%		39.4%		27.3%	
SAH 2	Good health	36.3%		34.5%		37.4%		40.8%	
SAH 3	Fair or low health	29.5%		35.9%		23.1%		31.9%	
Underweight	BMI<18.5	5.2%		5.8%		1.2%		1.4%	
Normal weight	18.5≤BMI≤25	56.5%		50.6%		42.8%		39.2%	
Overweight	BMI>25	38.2%		43.7%		56.0%		59.5%	
Exercise 1	Never exercises	9.6%		15.1%		10.7%		15.9%	
Exercise 2	Less than once a week	32.4%		33.5%		31.4%		34.9%	
Exercise 3	Once a week	13.0%		12.3%		14.8%		13.4%	
Exercise 4	More than once a week	44.9%		39.1%		43.1%		35.8%	
Smoker	*Currently smokes*	17.5%		21.0%		13.0%		29.2%	
Mobility impairment	Mobility impairment	23.2%		25.6%		13.8%		17.3%	
Self_care	Self-care ability	93.6%		92.9%		97.8%		97.5%	
Psychiatric	Psychiatric diagnosis	5.5%		5.4%		3.2%		6.4%	
Neurology	Neurologic disease	7.8%		7.1%		7.4%		5.0%	

Cardiovasc	Cardiovascular disease	18.5%	17.9%	18.5%	15.7%
Respiratory	Respiratory system disease	8.1%	7.5%	7.1%	5.2%
Skeleton	Skeleton disease	24.5%	27.0%	15.8%	21.0%
Pain	Pain diagnosis	59.3%	64.6%	50.8%	58.5%
Other_disease_1	One other disease	21.1%	21.8%	17.0%	14.6%
Other disease_1+	Two or more other diseases	8.4%	6.0%	7.9%	9.3%
2nd gen immigrant	Born in Sweden, parents foregin born	1.9%	0%	1.9%	0%
Primary edu	At most primary education	21.4%	21.6%	20.4%	19.8%
Secondary edu	At most upper secondary	42.9%	39.5%	45.8%	42.8%
Higher edu ≤2	At most two years of higher education	16.0%	14.5%	15.0%	13.9%
Higher edu >2	More than two years of higher education	19.7%	22.4%	18.8%	23.0%
Married_cohab	Married or cohabiting	67.5%	65.9%	73.1%	77.7%
Child_1	Have one child	12.0%	21.0%	11.6%	15.3%
Child_2+	Have two or more children	22.3%	22.8%	20.5%	25.7%
Large_city	Live in any of the three biggest	32.2%	51.0%	32.0%	50.3%
Work_full	Work full time	39.4%	39.7%	62.2%	53.8%

[a]Mean value, standard error in parentheses
[b]Conditional on access, the average accessed DDDs was; native women: 2,146; immigrant women: 1,929; native men: 1,862, and immigrant men. 1,836.

4.5 Empirical specification

The distribution of the dependent variable has two key characteristics: (1) the presence of non-users (18% had no pharmaceutical access), and (2) a long right-tail (10% of those who accessed most, accessed 25% of the DDDs). As the presence of non-users causes the standard Ordinary Least Squares (OLS) to yield inconsistent results (Cameron and Trivedi 2005), we turn to alternative models. There are three obvious empirical candidate models to address the presence of non-users: the Tobit model, the Two-part model and the Heckman model.

The Tobit model is a standard econometric model addressing dependent variables taking a mixture of zero and positive values. The Tobit model assumes that the same mechanism generates the non-zeros and the size of the positive values for non-zeros. In our case, this would imply that the mechanism for pharmaceutical access would equal the mechanism for the accessed amount. This assumption may be violated as people are more or less reluctant to seek healthcare. Thus, when seeking care, people who are more reluctant to seek healthcare may be sicker and, accordingly, prescribed more pharmaceuticals than their less reluctant counterparts.

Two-part model is more general than the Tobit model, as the two-part model assumes that one mechanism generates the non-zeros and a different mechanism generates the size of the positive values for non-zeros. A restriction of the Two-part model is if the residuals from the two parts, after controlling for independent variables, are correlated, in which case selection on unobservables causes selection bias. As the residuals from the two parts in our analysis are correlated, the selection bias prevents us from using the Two-part model.

The Heckman two-step model handles such selection by using an exclusion-criterion (Heckman 1979). For our analysis, an exclusion-criterion would be a variable that influences pharmaceutical access but not the accessed amount. As our dataset lacks such a variable, we are unable to meet the Heckman model exclusion-criterion.

Given that the three empirical models for addressing the presence of non-users rely on assumptions we may violate, we estimate pharmaceutical utilization in two separate steps: (1) the probability of pharmaceutical access using a probit model, and (2) conditional on access, the accessed amount of DDDs. One way to handle the long right-tailed distribution of DDD in the second step is to estimate log DDD with OLS, which yields log-scaled coefficients. Log scaled results are not interesting per se, though; to simplify inference drawing and facilitate comparability with other studies, the log-scaled coefficients are often retransformed to un-scaled coefficients with, for instance, the Duan smearing factor (Duan, 1983). Still, this retransformation

requires that the residuals are heteroscedastic to the explanatory variables; otherwise the retransformation generates biased coefficients (Ai and Norton, 2000). In our case, the White-test (White 1980) shows that our residuals are heteroscedastic (i.e., the retransformation generates biased coefficients); therefore we refrain from using log OLS and turn to the Generalized Linear Model (GLM).

As GLM yields un-scaled coefficients via a link function, retransformation is unnecessary. In our case, the long right-tailed distribution of DDD calls for a log-link function, and the Park test (see Manning and Mullahy, 2001 for a description) classifies the distribution as a member of the Gaussian family. Accordingly, to estimate the second step i.e., the accessed amount of DDD conditional on access, we use GLM with a Gaussian distribution family and log-link function. For comparability reasons only, we also estimate the unconditional OLS model (including non-users), the Tobit model and the Two-part model with the joint effect. We use STATA version 12.2 and the Huber/White/sandwich estimate of variance to obtain robust standard errors for all analyses.

4.5 Results

The main results are presented in tables 2 to 6. Tables 2 and 3 analyze overall pharmaceutical-based health investments, and table 4 analyzes specific pharmaceutical-based health investments with the 20 most commonly dispensed pharmaceutical subgroups individually. Extensions of the models for overall pharmaceutical-based health investments are presented in tables 5 to 8.

4.5.1 Overall pharmaceutical utilization

fewer DDDs than native men. Table 3 shows the results for women. Controlling for immigrant region of origin and conditioning on access, column 6 shows that non-Western immigrant women access fewer DDDs than native women. This difference persists after controlling for health (column 7) and socioeconomic status (column 8). Appendix C shows the full model specifications and estimates for the regressions analyzing overall pharmaceutical-based health investments (columns 4 and 8 in tables 2 and 3, respectively). In general, for both men and women; (1) low self-assessed health is positively associated with pharmaceutical access and accessed amount, (2) being married (or cohabiting), having some higher education and a higher disposable household income are positively associated with pharmaceutical access and, (3) children in the household are negatively associated with pharmaceutical access.

Table 2: Overall pharmaceutical utilization, results for men

Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and conditional on access, accessed number of defined daily doses (DDD Heteroscedasticity robust standard errors (Std. Err).

	Probit on access											
	(1)			(2)			(3)			(4)		
	A.M.E.	Std. Err.		A.M.E.		Std. Err.	A.M.E.		Std. Err.	*A.M.E.*		Std. Err.
Native Swedish	*reference*			*reference*			*reference*			*reference*		
All immigrants	0.03	0.02										
Nordic origin				0.01		0.04	-0.01		0.04	-0.00		0.04
Western origin				-0.01		0.06	0.02		0.05	0.02		0.05
non-Western origin				0.05	**	0.03	0.06	**	0.02	0.07	***	0.02
Controls for health	*No*			*No*			*Yes*			*Yes*		
Controls for human capital	*No*			*No*			*No*			*Yes*		
No. of observations	4,078			4,078			4,078			4,078		
	GLM on DDD|access											
	(5)			(6)			(7)			(8)		
	A.M.E.	Std. Err.		A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*			*reference*					
All immigrants	-87.71	175.67										
Nordic origin				520.31		324.56	82.32		263.3	190.20		279.92
Western origin				-915.82	***	237.81	-196.31		391.79	-191.29		411.57
non-Western origin				-171.26		222.28	188.82		290.23	115.78		254.1
Controls for health	*No*			*No*			*Yes*			*Yes*		
Controls for human capital	*No*			*No*			*No*			*Yes*		
No. of observations	3,114			3,114			3,114			3,114		

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capital include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, work full time and ln disposable income. Appendix C presents full results for the models in columns 4 and 8. Full results for the remaining models are available from the authors upon request. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Table 3: Overall pharmaceutical utilization, results for women
Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and conditional on access, accessed number of defined daily doses (DDD). Heteroscedasticity robust standard errors (Std. Err).

	Probit on access							
	(1)		(2)		(3)		(4)	
	A.M.E.	Std. Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.
Native Swedish	*Reference*		*reference*		*reference*		*reference*	
All immigrants	-0.02	0.01						
Nordic origin			-0.03	0.03	-0.04	0.03	-0.04	0.03
Western origin			0.02	0.04	0.02	0.04	0.02	0.04
non-Western origin			-0.03	0.02	-0.03	0.02	-0.03	0.02
Controls for health	*No*		*No*		*Yes*		*Yes*	
Controls for human capital	*No*		*No*		*No*		*Yes*	
No. of observations	4,410		4,410		4,410		4,410	

	GLM on DDD|access											
	(5)			(6)			(7)			(8)		
	A.M.E.	Std. Err.		A.M.E.	Std. Err.		A.M.E.	Std. Err.		A.M.E.	Std. Err.	
Native Swedish	*Reference*			*reference*			*reference*			*reference*		
All immigrants	-248.28	188.75										
Nordic origin				-49.13	296.22		-83.41	354.29		-137.57	373.37	
Western origin				291.98	413.05		-252.67	316.44		-224.58	328.2	
non-Western origin				-482.24	220.89	**	-629.55	355.49	*	-797.07	323.09	**
Controls for health	*No*			*No*			*Yes*			*Yes*		
Controls for human capital	*No*			*No*			*No*			*Yes*		
No. of observations	3,877			3,877			3,877			3,877		

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capital include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. Appendix C presents full results for the models in columns 4 and 8. Full results for the other models are available from the authors upon request. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

4.5.2 Specific pharmaceutical utilization, the 20 most dispensed pharmaceutical subgroups

Using a probit model with controls for immigrant region of origin, health and socioeconomic status, we separately estimate the likelihood of accessing the 20 most dispensed pharmaceutical subgroups (in the third ATC-level) for men and women. Table 4 shows the pharmaceutical subgroups where immigrants and natives have statistically different (on the 10% level) probabilities of pharmaceutical access (see Appendix B for full results).

Immigrant and native men have different access probabilities in 6 out of the 20 analyzed pharmaceutical subgroups. Compared with native men: (1) Nordic immigrant men are less likely to access pharmaceuticals with the ATC-codes A02B (for e.g. peptic ulcer) and D07A (corticosteroids), but more likely to access C07A (beta blockers for e.g. cardiovascular diseases) and A10B (anti-diabetics, excluding insulin), (2) Western immigrant men are less likely to access pharmaceuticals with ATC-codes C09A (ACEs for cardiovascular diseases), C07A (beta blockers for e.g. cardiovascular diseases) and C01D (vasodilators for cardiac diseases) and (3) non-Western immigrant men are more likely to access pharmaceuticals with ATC-codes A02B (for e.g. peptic ulcer), A10B (anti-diabetics, excluding insulin) and C01D (vasodilators for cardiac diseases).

Turning to the women, immigrants and natives have significantly different access probabilities in 9 out of the 20 analyzed pharmaceutical subgroups: Compared with native women (1) Nordic immigrant women are less likely to access pharmaceuticals with ATC-codes D02A (for e.g., dry skin) and C03A (thiazide diuretics for e.g., cardiovascular diseases), (2) Western immigrant women are less likely to access pharmaceuticals with the ATC-code C09A (ACEs for cardiovascular diseases), and (3) non-Western immigrant women are less likely to access pharmaceuticals with ATC-codes G03A (oral hormonal contraceptives) and R03A (adrenergic inhalants for obstructive airways), but more likely to access A02B (for e.g., peptic ulcer), M01A (for e.g. pain and inflammation), N02B (e.g. analgesics) and A06A (laxatives for constipation).

Table 4: Specific pharmaceutical utilization for men and women
The average marginal effects (A.M.E.) on the probability of accessing the 20 most dispensed pharmaceuticals. Native Swedish is the reference category, and results are only reported when at least one of the regions of origin immigrant coefficient is statistically significant at the 10% level. Heteroscedasticity robust standard errors (Std. Err).

Men - Probit on access

ATC		A.M.E		Std. Err .	ATC		A.M.E		Std. Err.
C09A *ACE inhibitors*	Nordic origin	-0.01		-0.03	*A10B* *Blood glucose lowering agents, excl. insulin*	Nordic origin	0.04	*	-0.02
	Western origin	-0.07	***	-0.03		Western origin	0.05		-0.04
	non-Western origin	0		-0.02		non-Western origin	0.03	**	-0.02
C07A *Beta blocking agents*	Nordic origin	0.09	***	-0.03	*D07A* *Corticosteroids*	Nordic origin	-0.06	**	-0.03
	Western origin	-0.09	**	-0.04		Western origin	0.02		-0.05
	non-Western origin	-0.02		-0.02		non-Western origin	-0.02		-0.02
A02B *Agents for peptic ulcer and gastro-oesophageal reflux*	Nordic origin	-0.05	**	-0.03	*C01D* *Vasodilators used in cardiac diseases*	Nordic origin	0.03		-0.02
	Western origin	-0.06		-0.04		Western origin	-0.04	*	-0.02
	non-Western origin	0.05	*	-0.03		non-Western origin	0.05	**	-0.02

Women - Probit on access

ATC		A.M.E.		Std. Err.	ATC		A.M.E		Std. Err.
D02A Emollients and protectives	Nordic origin	-0.05	***	-0.02	**N02B** Analgesics and antipyretics	Nordic origin	-0.01		-0.03
	Western origin	0.01		-0.04		Western origin	0.01		-0.06
	non-Western origin	0.03		-0.02		non-Western origin	0.05	*	-0.03
C09A ACE inhibitors	Nordic origin	-0.01		-0.02	**A06A** Laxatives	Nordic origin	-0.01		-0.02
	Western origin	-0.07	***	-0.02		Western origin	0.05		-0.05
	non-Western origin	-0.02		-0.02		non-Western origin	0.07	**	-0.03
A02B Peptic ulcer and gastro-oesophageal reflux	Nordic origin	-0.03		-0.03	**R03A** Adrenergic inhalants	Nordic origin	-0.02		-0.02
	Western origin	0.07		-0.06		Western origin	-0.04		-0.02
	non-Western origin	0.07	**	-0.03		non-Western origin	-0.04	**	-0.02
G03A Hormonal contraceptives, systemic use	Nordic origin	-0.01		-0.03	**C03A** Thiazide diuretics	Nordic origin	-0.02	*	-0.01
	Western origin	0.05		-0.05		Western origin	-0.01		-0.03
	non-Western origin	-0.06	***	-0.02		non-Western origin	0		-0.02
M01A NSAID (anti-inflammatory and antirheumatic agents)	Nordic origin	0		-0.04					
	Western origin	0		-0.07					
	non-Western origin	0.1	***	-0.03					

Note: Health variables includes: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses, age and age squared. Human capital variables include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. Full results are available upon request. *** p<0.01, ** p<0.05, * p<0.1.

4.5.3 Extensions

Tables 5 to 8 present the results from four extensions of the overall pharmaceutical-based health investments model, controlling for immigration status, health, and socioeconomic status. Table 5 controls for immigrant status by using years of Swedish residence. As contraceptives have a different purpose than improving health, table 6 omits oral hormonal contraceptives and re-estimates the regressions for women. Due to conceivable differences in the educational level of respondents within the lowest educational category, up to primary schooling, table 7 omits these respondents and re-estimates the regressions. Table 8 extends the analysis by estimating the models using three alternative econometric approaches – the unconditional OLS (including non-users), Tobit and Two-part model.

Controlling for years of Swedish residency

Country specific-knowledge, such as the ability to speak the native language, affects individual productivity in terms of, for instance, health seeking behavior and labor market outcomes. While residing in Sweden, immigrants can acquire country-specific skills, which may increase productivity. On a conceptual level, given that immigrants acquire country specific-knowledge while residing in Sweden, the productivity of immigrants and natives will converge over time. As productivity relates both to the effort demanded when accessing pharmaceuticals and the value of health in terms of e.g., salaried labor supply, immigrants with longer years of Swedish residence and natives should become more alike in terms of pharmaceutical-based health investments.

We empirically explore the link between pharmaceutical-based health investments and years of residing in Sweden by using controls indicating years of Swedish residence. We categorize immigrants into four groups on the basis of years since immigration to Sweden: 0 to 5, 6 to 10, 11 to 20 and more than 20 years. One reason for using category variables, instead of a continuous residence variable, is that the marginal effect of going from one to two years of residence likely differs from going from 10 to 11 years.

Table 5 presents the resulting estimates from models controlling for years of Swedish residence, health and socioeconomic status. For men, column 1 shows that immigrants with 11 to 20 years of residence are 8% more likely to access pharmaceuticals than natives, and column 2 shows that immigrants with up to 5 years of residence access 1,252 fewer DDDs than natives when conditioning on access. Correspondingly for women, column 3 shows that immigrants with up to 5 years of residence are 19% less likely to access pharmaceuticals than natives, and column 4 shows that immigrants with 6 to 10 years of residence access 1,115 fewer DDDs than natives

when conditioning on access, and immigrants with more than 20 years of residence access 513 fewer DDDs than natives when conditioning on access.

Table 5: Overall pharmaceutical utilization after controlling for years of Swedish residence, results for men and women.
Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD). Heteroscedasticity robust standard errors (Std. Err).

	Men				Women			
	(1)		(2)		(3)		(4)	
	Probit on access		**GLM on DDD|access**		**Probit on access**		**GLM on DDD|access**	
	A.M.E.	Std.Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.
Native Swedish	*reference*		*reference*		*reference*		*reference*	
In Sweden -5	0.03	0.05	-1,251.62 ***	245.6	-0.19 ***	0.06	2,415.43	1,838.60
In Sweden 6-10	0.03	0.06	218.75	456.32	-0.08	0.05	-1,114.9 ***	385.87
In Sweden 11-20	0.08 ***	0.03	-289.09	289.69	-0.01	0.03	-436.80	460.51
In Sweden 21-	0.01	0.03	208.62	209.99	-0.00	0.02	-512.64 **	213.72
Control for health	*Yes*		*Yes*		*Yes*		*Yes*	
Control for H.C.	*Yes*		*Yes*		*Yes*		*Yes*	
No. of observations	4,078		3,114		4,410		3,877	

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capital (H.C.) include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. See appendix D for full results. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

The results are in line with the hypothesis that country-specific knowledge matters for pharmaceutical access; the results show a smaller discrepancy in pharmaceutical access between natives and immigrants with more than 10 Swedish residence years compared to natives and immigrants with fewer residence years.

Excluding oral hormonal contraceptives for women

Contraceptives differ from other pharmaceuticals in that the main use is to prevent pregnancy rather than improving health per se. To ascertain if disparities in pharmaceutical access between immigrant and native women originate from differences in contraceptive practice, we omit oral hormonal contraceptives and re-estimate the models.

The new result, that non-Western immigrant women, conditional on access, accessed 788 fewer DDDs than native women (table 6, column 2), is qualitatively and quantitatively similar to the old result (table 3, columns 4 and 8), suggesting that hormonal contraceptive use alone does not explain the overall discrepancy in pharmaceutical access between immigrant and native women.

Table 6: Overall pharmaceutical utilization after excluding oral hormonal contraceptives, results for women
Average marginal effects ((A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD) after exclusion of hormonal contraceptive (ATC: G03A). Heteroscedasticity robust standard errors (Std. Err).

	-1			-2		
	Probit on access			**GLM on DDD|access**		
	A.M.E.	Std. Err.		*A.M.E.*		Std. Err.
Native Swedish	*reference*			*reference*		
Nordic origin	-0.04	0.03		-135.49		375.03
Western origin	0.04	0.04		-242.59		326.03
non-Western origin	-0.00	0.02		-787.73	**	329.11
Controls for health	*Yes*			*Yes*		
Controls for human capital	*Yes*			*Yes*		
No. of observations	4,410			3,774		

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capital include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. See appendix F for full results. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Excluding respondents with no more than primary school

In 1936, 7 years of education became mandatory for children in Sweden, implying that the majority of natives have at least 7 years of education. As educational rules vary between countries, some immigrants may have substantially less, or no, formal education. To establish if observed differences in pharmaceutical access between immigrants and natives derive from respondents belonging to the lowest educational group, we omit respondents with up to primary school and re-estimate the models. The omission does not essentially change the results for men (table 7, columns 1 and 2).

After the omission in women (table 7, column 3 to 4), non-Western immigrants still access fewer DDDs conditional on access than natives, but the discrepancy is smaller and no longer statistically significant (for comparison see table 3, column 8). These results suggest that immigrant women in the lowest educational group contribute to the observed discrepancy in pharmaceuticals access between immigrants and natives, but do not explain the entire difference.

Table 7: Excluding individuals with at most primary education, results for man and women
Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD). Heteroscedasticity robust standard errors (Std. Err).

	Men				**Women**			
	(1)		(2)		(3)		(4)	
	Probit on access		**GLM on DDD|access**		**Probit on access**		**GLM on DDD|access**	
	A.M.E.	Std.Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.
Native Swedish	*Reference*		*reference*		*reference*		*reference*	
Nordic origin	-0.03	0.05	545.47	347.69	-0.03	0.03	453.58	552.47
Western origin	0.02	0.05	-20.41	409.44	0.00	0.04	69.21	431.69
non-Western origin	0.07 ***	0.03	204.10	242.42	-0.03	0.03	-441.70	445.33
Controls for health	*Yes*		*Yes*		*Yes*		*Yes*	
Controls for H.C.	*Yes*		*Yes*		*Yes*		*Yes*	
No. of observations	3,245		2,442		3,457		3,053	

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capita (H.C.) include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. See appendix G for full results.***p-value<0.01, **p-value<0.05 and *p-value<0.1 level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. See appendix G for full results.***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Alternative econometric approaches

For comparability reasons (see discussion in the empirical specification), we also estimate overall pharmaceutical-based health investments with three additional econometric models: (1) unconditional OLS (including non-users), (2) Tobit, and (3) the joint two-part.

For women (table 8), all the alternative econometric approaches uniformly show that non-Western immigrant women have lower pharmaceutical access than native women. These results are in line with the estimations in two separate steps (i.e., the probability of pharmaceutical access using a probit model, and, conditional on access, the accessed amount of DDDs).

For men, (table 8), the marginal estimates for immigrant region of origin are significant, which generally overlaps the results from the two separate steps. The signs of the estimates, however, have different directions across the alternative economic approaches. This inconsistency may imply that the estimates are biased.

Table 8. Overall pharmaceutical utilization using alternative econometric approaches, results for men and women. The combined marginal effect (A.M.E.) of access and, conditional on access, accessed number of defined daily doses (DDD) estimated with OLS (columns 1 and 4), Tobit (columns 2 and 5) and the Two-part model (columns 3 and 6). Heteroscedasticity robust standard errors (Std. Err).

	Men					
	(1)		(2)		(3)	
	OLS DDD		**Tobit DDD**		**Two-part model DDD**	
	A.M.E.	Std. Err.	A.M.E.	Std. Err.	A.M.E.	Std. Err.
Native Swedish	*reference*		*reference*		*reference*	
Nordic origin	268.24	234.75	261.66	286.63	147.77	46383.65
Western origin	-66.19	154.16	60.13	230.71	-134.61	539.71
non-Western origin	-57.29	143.88	119.64	171.43	184.06	2652.26
Controls for health	*Yes*		*Yes*		*Yes*	
Controls for H.C. capital	*Yes*		*Yes*		*Yes*	
No. of observations	4,078		4,078		3,114	

	Women								
	(4)			(5)			(6)		
	OLS DDD			**Tobit DDD**			**Two-part model DDD**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*			*reference*		
Nordic origin	-306.96		230.17	-390.54		255.41	-251.35		219.50
Western origin	7.54		341.37	-70.48		387.98	-139.23		363.10
non-Western origin	-479.35	***	173.99	-627.25	***	200.32	-531.58	***	175.23
Controls for health	*Yes*			*Yes*			*Yes*		
Controls for H.C.	*Yes*			*Yes*			*Yes*		
No. of observations	4,410			4,410			3,877		

Note: Controls for health include: self-assessed health, BMI, frequency of exercise, smoking status, mobility impairment, self-care ability, medical diagnoses and age. Controls for human capital (H.C) include: 2nd generation immigrant, educational level, whether married or cohabitant, children in household, residency in a large city, full-time work and ln disposable income. See appendix G for full results.***p-value<0.01, **p-value<0.05 and *p-value<0.1

4.6 Discussions

In general, our results regarding the effect of health and education on health-related investments are in line with findings in previous studies (Merlo et al. 2003; Bolin, Lindgren, and Rössner 2006; Nordin, Dackehag, and Gerdtham 2013). In addition, the results show that immigrants and natives differ in (1) the likelihood of accessing prescribed pharmaceuticals and (2) accessed number of DDDs when conditioning on access. For women, the disparity between natives and non-Western immigrants is considerable, conditioning on access – non-Western immigrant women access about 800 fewer DDDs than native women. For comparison, the corresponding average for women is 1,866 DDDs.

Pharmaceutical access differs between immigrants and natives across several of the 20 most dispensed pharmaceutical subgroups (on the 3rd ATC-level). The directions of the disparity in

pharmaceutical access between natives and immigrants from different regions of origin are mixed – natives have higher access to some pharmaceuticals whereas immigrants have higher access to others. Focusing on first-line pharmaceuticals listed by evidence-based treatment guidelines (e.g. Janus 2006; Läkemedelsrådet 2006), a uniform pattern emerges – immigrants are less likely than natives to access the first-line pharmaceuticals: thiazide-diuretics (C03A), ACE inhibitors (C09A) and adrenergic inhalators (N02B). As these first-line pharmaceuticals make up the pharmaceutical foundation in the prevention and treatment of common cardiovascular-related diseases (high blood pressure, heart failure and kidney diseases) and asthmatic diseases, immigrants may be less likely than natives to have adequate preventive pharmaceutical treatment. As cardiovascular-related morbidity and mortality are leading public health concerns in Sweden, and pharmaceuticals substantially reduce such related morbidity and mortality (WHO 2012), disparities in access to these pharmaceuticals between immigrants and natives may have significant public health consequences. Non-Western immigrant women are more likely than native women to access pharmaceuticals commonly used for treating pain and inflammatory conditions (M01A and N02B), which may imply that immigrants, more often than natives, receive treatment alleviating pain symptoms rather than treatment for the underlying cause. To illustrate, chronic physical pain may indicate other underlying medical causes such as depression or cancer. The results also show that non-Western immigrant women are less likely to access hormonal contraceptives. Given that immigrants in Sweden are overrepresented among women requesting induced abortion (Helström et al. 2003), the lower hormonal contraceptive practice in non-Western immigrant women suggests that they face higher barriers to accessing contraceptives rather than needing less.

The empirical results regarding pharmaceutical-based health investments are consistent with the predictions obtained within the demand-for-health framework. In brief, low health and education are positively related to pharmaceutical-based health investments, whereas time constraints (measured as full-time work and children in the household) are negatively related. While living in a new country, immigrants acquire country-specific knowledge, such as the ability to master the native language. Such knowledge may be regarded as educational capital, which makes pharmaceutical-based health investments less costly. In line with theory, a longer stay in Sweden is positively related to pharmaceutical-based health investments in immigrants and, generally, immigrants with more residence years in Sweden are more similar to natives with regard to pharmaceutical-based health investments than immigrants with fewer residence years.

Limitations

The nature of our data creates two caveats that should be mentioned. First, when using perceived self-assessed health as a proxy for true health, cultural differences affecting the perception of health may yield biased estimates. To reduce such bias, we complement self-assessed health with more objective health measures (e.g. mobility impairment and the occurrence of cardiovascular disease). Second, sample selection may arise due to systematic differences in characteristics between respondents and non-respondents.

Our sample comprises 11% immigrants, whereas the corresponding figure was 14% in 2004 (Statistics Sweden, 2013). A study of non-respondents in the ULF survey year 2000 (Statistics Sweden, 2003) showed that the majority of non-respondent immigrants were outside the labor force. Given that immigrants are selected into the labor force on the basis of, for instance, country-specific skills such as mastering the native language, immigrants inside and outside the labor force may differ in terms of such skills. As educational capital, including country-specific skills, is positively related to pharmaceutical-based health investments, we may underestimate the discrepancy in pharmaceutical-based health investments between immigrants and natives.

Policy implications and future studies

Our results indicate that the Swedish health policy goal – that all (legal) residents should have equal access to medical care according to need – is unachieved. When creating policies for tackling inequalities in the utilization of prescribed pharmaceuticals, policymakers can either address general socioeconomic inequalities or specific vulnerable groups (e.g. women immigrants with low educational level).

Disparities in pharmaceutical utilization between population groups may reinforce present and future health inequalities. To ascertain the consequences of differences between immigrants and natives in pharmaceutical utilization for public health, more research is needed. More specifically, given that physicians are gatekeepers to prescribed pharmaceuticals, future research should explore the role of discrimination, for instance by analyzing if physicians' prescribing patterns for immigrants and natives differ

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Appendix A – Specification of control variables

SAH 1, SAH 2, and *SAH 3* are mutually exclusive dummy variables that take the value 1 if the respondents' self-assessed health is (1) very good, (2) good, and (3) low, respectively. In all cases, the variables are 0 otherwise.

Underweight, Normal weight and *overweight* are mutually exclusive dummy variables that take the value 1 if the respondent is (1) underweight (BMI<18.5), (2) normal weight (18.5≤BMI<25), and (3) overweight (BMI≥25), respectively. In all cases, the variables are 0 otherwise.

Exercise 1, Exercise 2, Exercise 3, and *Exercise 4* are mutually exclusive dummy variables and take the value 1 if the respondent reports (1) never exercise, (2) exercise, but less than once a week, (3) exercise once a week, and (4) exercise more than once a week, respectively. In all cases, the variables are 0 otherwise.

Smoker is a dummy variable which takes the value 1 if the respondent reports that he or she smokes daily, and 0 otherwise.

Mobile impairment is a dummy variable which takes the value 1 if the respondent reports having a mobility impairment, and 0 otherwise.

Pain is a dummy variable which takes the value 1 if the respondent reports experienced pain (e.g. back pain), and 0 otherwise.

Self-care ability is a dummy variable which takes the value 1 if the respondent reports self-care ability, and 0 otherwise.

Psychiatric is a dummy variable which takes the value 1 if the respondent reports psychiatric disorders corresponding to at least one diagnosis in the interval 290.0-316.9 in WHO´s ICD-9, and 0 otherwise.

Neurology is a dummy variable which takes the value 1 if the respondent reports neurological disease corresponding to at least one diagnosis in the interval 320.0-389.9 in WHO´s ICD-9, and 0 otherwise.

Cardiovasc is a dummy variable which takes the value 1 if the respondent reports cardiovascular disease corresponding to at least one diagnosis in the interval 390.0-405.9 or 410.0-429.9 in WHO´s ICD-9, and 0 otherwise.

Respiratory is a dummy variable which takes the value 1 if the respondent reports respiratory disease corresponding to at least one diagnosis in the interval 460.0-519.9 according to WHO´s ICD-9, and 0 otherwise.

Skeleton is a dummy variable which takes the value 1 if the respondent reports skeletal disease corresponding to at least one diagnosis in the interval 710.0-739.9 in WHO´s ICD-9, and 0 otherwise.

Other_disease_1 is a dummy variable which takes the value 1 if the respondent reports diseases corresponding to one of the following categories: (1) diabetes, infections, tumors, (2) diseases of the eye, ear, skin, joints, (3) diseases in the blood, digestive, congenital, endocrine and urogenital systems, or (4) morbidity from external causes, and 0 otherwise.

Other_disease_2+ is a dummy variable which takes the value 1 if the respondent reports diseases corresponding to two or more of the following categories: (1) diabetes, infections, tumors, (2) diseases of the eye, ear, skin, joints, (3) diseases in the blood, digestive, congenital, endocrine and urogenital systems, or (4) morbidity from external causes, and 0 otherwise.

Age consists of age and age^2 (measured year 2006). Age^2 controls for potential nonlinear relationships between age and the dependent variable.[4]

2nd gen immigrant is a dummy variable which takes the value 1 if the respondent was born in Sweden, but neither of the respondent´s parents was born in Sweden, and 0 otherwise.

Primary school, Secondary school, higher education≤2 and *higher education>2* are mutually exclusive dummy variables that take the value 1 if the respondent's education level is (1) up to primary school, (2) up to secondary school, (3) some, but less than two years, higher education, and (4) at least two years of higher education, respectively. In all cases, the variables are 0 otherwise.

Married_cohab is a dummy variable which takes the value 1 if the respondent is married or cohabiting, and 0 otherwise.

ln(dispinc) is the respondent´s logged disposable income after taxes and social transfers. For married or cohabiting respondents, ln(dispinc) is the mean of the household's disposable income after taxes and social transfers[5].

[4] Allowing for additional functional forms i.e, age^3, age^3, etc., does not significantly change the results.

[5] The response rate for hourly wage was low (about 60 per cent) and we observed systematic differences in wage rate between responders and non-responders, thus we refrain from using hourly wage in the analyses.

Child_1 and *Child_2+* are mutually exclusive dummy variables. Child_1 takes the value 1 if the respondent has one child in the household, and Child_2+ takes the value 1 if the respondent has two or more children in the household. In all cases, the variables are 0 otherwise.

Large_city is a dummy variable which takes the value 1 if the respondent lives in Stockholm, Gothenburg or Malmo, and 0 otherwise.

Work_full_time is a dummy variable which takes the value 1 if the respondent reports working 40 hours a week or more, and 0 otherwise.

Appendix B. The 20 most dispensed pharmaceuticals (third level ATC) in Sweden 2006 for men and women .

Men		Women	
ATC	**pharmaceutical subgroups**	**ATC**	**pharmaceutical subgroups**
B03B	Vitamin B12 and folic acid	B03B	Vitamin B12 and folic acid
C10A	Lipid modifying agents	D02A	Emollients and protectives
D02A	Emollients and protectives	G03A	Hormonal contraceptives for systemic use
B01A	Antithrombotic agents	N06A	Antidepressants
C09A	ACE inhibitors	C10A	Lipid modifying agents
C07A	Beta blocking agents	B01A	Antithrombotic agents
N06A	Antidepressants	N05C	Hypnotics and sedatives
C08C	Selective calcium channel blockers with mainly vascular	C07A	Beta blocking agents
C03C	High-ceiling diuretics	C03C	High-ceiling diuretics, Loop diuretics
N05C	Hypnotics and sedatives	C09A	ACE inhibitors, plain
A02B	Agents for peptic ulcer and gastro-oesophageal reflux	M01A	Non-steroid anti-inflammatory and antirheumatic agents
C09C	Angiotensin II antagonists	A02B	Agents for peptic ulcer and gastro-oesophageal reflux
A10A	Insulins and analogues	C08C	Selective calcium channel blockers with mainly vascular
M01A	Non-steroid anti-inflammatory and antirheumatic agents	H03A	Thyroid preparations
A10B	Blood glucose lowering agents, excluding insulin	N02B	Analgesics and antipyretics
D07A	Corticosteroids	A06A	Laxatives
R03A	Adrenergic inhalants	R03A	Adrenergic inhalants
C01D	Vasodilators used in cardiac diseases	C09C	Angiotensin II antagonists
A06A	Laxatives	R06A	Antihistamines for systemic use
G04C	Drugs used in benign prostatic hypertrophy	C03A	Low-ceiling diuretics, Thiazides

Source: National Board of Health and Welfare, 2007

Appendix C1: Overall pharmaceutical utilization, full results for men.

Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD). Heteroscedasticity robust standard errors in parenthesis.

	Men					
	(1)			(2)		
	Probit on access			**GLM on DDD | access**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*		
Nordic origin	**-0.00**		**0.04**	**190.20**		**279.92**
Western origin	**0.02**		**0.05**	**-191.29**		**411.57**
non-Western origin	**0.07**	*******	**0.02**	**115.78**		**254.1**
SAH 1	*reference*			*reference*		
SAH 2	0.06	***	0.02	351.34	**	139.29
SAH 3	0.08	***	0.02	633.14	***	192.57
Underweight	-0.05		0.06	1,083.82		751.83
Normal weight	*reference*			*reference*		
Overweight	0.03	**	0.01	318.43	**	129.8
Exercise 1	*reference*			*reference*		
Exercise 2	0.01		0.02	29.42		177.49
Exercise 3	0.04		0.03	-214.15		209.98
Exercise 4	0.05	*	0.02	-22.44		186.55
Smoker	0.01		0.02	-62.31		174.51
Mobility impairment	-0.01		0.03	459.96	***	152.55
Self_care	0.19	***	0.05	-334.26		323.17
Psychiatric	0.06		0.04	820.68	***	304.05
Neurology	0.07	*	0.03	-392.14	*	209.84
Cardiovasc	0.21	***	0.02	1,269.72	***	156.96
Respiratory	0.08	***	0.03	483.94	**	232.81
Skeleton	0.06	**	0.02	-151.74		139.33
Pain	0.02		0.01	-56.81		142.36
Other_disease_1	0.10	***	0.02	340.72	**	159.06
Other disease_2+	0.12	***	0.03	1,035.47	***	233.67
Age	0.00	***	0	25.70	***	7
2nd gen immigrant	0.12	**	0.05	-1,259.78	**	511.22
Primary education	*reference*			*reference*		
Secondary education	0.02		0.02	155.17		149.03
Higher edu. ≤2	0.05	**	0.02	257.44		248.53
Higher edu. >2	0.03		0.02	448.72		282.35
lndispinc	0.04	***	0.01	221.71		177.22
Married_cohab	0.05	***	0.02	-146.59		134.24
Child_1	0.01		0.02	-57.94		338.35
Child_2+	-0.05	***	0.02	-174.45		375.03
Large_city	0.00		0.01	-81.16		146.18
Work_full_time	-0.02		0.02	-463.84	***	154.81
No. of observations	4,078			3,114		

Note: Conditional on access, the average accessed number of DDDs is 1,859 DDDs for men. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Appendix C2: Overall pharmaceutical utilization, full results for women

Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD). Heteroscedasticity robust standard errors in parenthesis.

	Women					
	(3)			(4)		
	Probit on access			**GLM on DDD|access**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*		
Nordic origin	**-0.04**		**0.03**	**-137.57**		**373.37**
Western origin	**0.02**		**0.04**	**-224.58**		**328.2**
non-Western origin	**-0.03**		**0.02**	**-797.07**	******	**323.09**
SAH 1	*reference*			*reference*		
SAH 2	0.03	***	0.01	184.89		146.48
SAH 3	0.07	***	0.01	792.83	***	194.52
Underweight	-0.04	*	0.02	35.72		303.69
Normal weight	*reference*			*reference*		
Overweight	0.01		0.01	68.90		180.88
Exercise 1	*reference*			*reference*		
Exercise 2	0.02		0.02	-383.68		360.51
Exercise 3	0.01		0.02	-189.04		438.44
Exercise 4	0.02		0.02	-403.56		381.1
Smoker	-0.00		0.01	15.03		359.29
Mobility impairment	-0.02		0.01	710.40	***	178.26
Self_care	0.09	***	0.02	-661.52	**	289.8
Psychiatric	0.04	*	0.02	92.53		334.05
Neurology	0.01		0.02	53.76		285.02
Cardiovasc	0.10	***	0.01	1,099.98	***	173.6
Respiratory	0.06	**	0.02	627.90	**	277.78
Skeleton	0.02		0.01	159.96		169.56
Pain	0.02	**	0.01	226.31		161.39
Other_disease_1	0.06	***	0.01	184.45		159.2
Other disease_2+	0.06	***	0.02	698.06	**	338.64
Age	-0.00	***	0	5.20		9.88
2nd gen immigrant	-0.03		0.03	214.27		1,130.07
Primary education	*reference*			*reference*		
Secondary education	0.02	*	0.01	33.48		172.95
Higher edu. ≤2	0.02		0.02	454.20		671.77
Higher edu. >2	-0.01		0.02	406.65		349.37
lndispinc	0.02	***	0.01	-212.45	*	114.46
Married_cohab	0.02	**	0.01	-125.19		186.97
Child_1	0.01		0.01	-318.95		305.25
Child_2+	-0.05	***	0.02	-554.72	**	252.27
Large_city	0.04	***	0.01	161.40		180.93
Work_full_time	-0.01		0.01	-272.53		216.78
No. of observations	4,410			3,877		

Note: Conditional on access, the average accessed number of DDDs is 2,122 DDDs for Women. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Appendix D: Overall pharmaceutical utilization after controlling for years of Swedish residency, full results for men and women

Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD). The models control for years of Swedish residency among immigrants. Heteroscedasticity robust standard errors in parenthesis.

	Men						**Women**					
	(1)			(2)			(3)			(4)		
	Probit on access			**GLM on DDD | access**			**Probit on access**			**GLM on DDD | access**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*			*reference*			*reference*		
In Sweden -5	**0.03**		**0.05**	**-1,251.62**	*******	**245.6**	**-0.19**	*******	**0.06**	**2,415.43**		**1,838.60**
In Sweden 6-10	**0.03**		**0.06**	**218.75**		**456.32**	**-0.08**		**0.05**	**-1,114.96**	*******	**385.87**
In Sweden 11-20	**0.08**	*******	**0.03**	**-289.09**		**289.69**	**-0.01**		**0.03**	**-436.80**		**460.51**
In Sweden 21-	**0.01**		**0.03**	**208.62**		**209.99**	**-0.00**		**0.02**	**-512.64**	******	**213.72**
SAH 1	*reference*			*reference*			*reference*			*reference*		
SAH 2	0.06	***	0.02	346.74	**	140.12	0.03	***	0.01	150.45		145.06
SAH 3	0.08	***	0.02	625.46	***	194.17	0.07	***	0.01	769.36	***	197.08
Underweight	-0.05		0.06	1,036.17		751.24	-0.04	*	0.02	-41.27		317.29
Normal weight	*reference*			*reference*			*reference*			*reference*		
Overweight	0.03	**	0.01	319.76	**	130.18	0.00		0.01	105.04		186.84
Exercise 1	*reference*			*reference*			*reference*			*reference*		
Exercise 2	0.01		0.02	39.29		176.13	0.02		0.02	-360.74		374.46
Exercise 3	0.04		0.03	-216.21		209.5	0.01		0.02	-110.51		444.77
Exercise 4	0.04	*	0.02	-19.76		186.13	0.02		0.02	-372.87		389.01
Smoker	0.01		0.02	-50.06		176.83	-0.01		0.01	47.78		359.38
Mobility impairment	-0.01		0.03	464.77	***	152.76	-0.02		0.01	703.49	***	172.67
Self_care	0.19	***	0.05	-320.86		330.11	0.09	***	0.02	-619.17	**	277.66
Psychiatric	0.06		0.04	885.96	***	315.76	0.05	*	0.02	78.69		318.87
Neurology	0.07	*	0.03	-401.67	*	209.7	0.01		0.02	50.11		290.66

Cardiovasc	0.21	***	0.02	1,250.40	***	160.36	0.10	***	0.01	1,089.94	***	172.96
Respiratory	0.07	***	0.03	489.13	**	236.85	0.05	**	0.02	637.24	**	273.72
Skeleton	0.06	**	0.02	-158.80		139.89	0.01		0.01	158.34		179.15
Pain	0.02		0.01	-47.93		143.27	0.03	**	0.01	202.11		161.31
Other_disease_1	0.10	***	0.02	347.47	**	158.58	0.06	***	0.01	211.62		162.26
Other disease_2+	0.12	***	0.03	1,058.69	***	238.56	0.06	***	0.02	763.66	**	349.14
Age	0.00	***	0	24.67	***	7.02	-0.00	***	0	9.22		10.38
2nd gen immigrant	0.12	**	0.05	-1,261.20	**	498.8	-0.03		0.03	208.50		1,145.57
Primary education		*reference*			*reference*			*reference*			*reference*	
Secondary education	0.02		0.02	149.92		150.87	0.02		0.01	33.90		170.84
Higher education ≤2	0.05	**	0.02	258.67		247.15	0.02		0.02	437.96		678.86
Higher education >2	0.03		0.02	442.61		280.89	-0.01		0.02	303.17		343.75
lndispinc	0.04	***	0.01	204.46		175.38	0.02	**	0.01	29.51		121.01
Married_cohab	0.05	***	0.02	-138.89		132.73	0.02	**	0.01	-108.62		198.96
Child_1	0.01		0.02	-72.27		345.05	0.01		0.01	-395.40		311.61
Child_2+	-0.05	***	0.02	-154.34		389.46	-0.05	***	0.02	-693.96	**	293.25
Large_city	0.01		0.01	-79.56		147.1	0.04	***	0.01	67.49		194.24
Work_full_time	-0.02		0.02	-472.4	***	155.45	-0.01		0.01	-266.69		205.29
No. of observations	4,078			3,114			4,410			3,877		

Note: Years of Swedish residency are dummy variables. For instance, "In Sweden -5" takes on the value 1 for immigrants with up to 5 years of Swedish residency and 0 otherwise. ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Appendix F: Overall pharmaceutical utilization after excluding oral hormonal contraceptives, full results

Average marginal effects (A.M.E) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD) after exclusion of hormonal contraceptive (ATC: G03A). Heteroscedasticity robust standard errors (obtained using the Huber/White/sandwich estimator) in parenthesis.

	(1)			(2)		
	Probit on access			**GLM on DDD|access**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish		*reference*			*reference*	
Nordic origin	**-0.04**		**0.03**	**-135.49**		**375.03**
Western origin	**0.04**		**0.04**	**-242.59**		**326.03**
non-Western origin	**-0.00**		**0.02**	**-787.73**	******	**329.11**
SAH 1		*reference*			*reference*	
SAH 3	0.09	**	0.02	800.18	**	198.11
Underweight	-0.05	*	0.03	31.76		305.56
Normal weight		*reference*			*reference*	
Overweight	0.00		0.01	62.94		185.42
Exercise 1		*reference*			*reference*	
Exercise 2	0.05	**	0.02	-381.22		366.76
Exercise 3	0.04		0.02	-188.58		443.91
Exercise 4	0.04	**	0.02	-400.99		387.3
Smoker	0.01		0.01	28.53		362
Mobility impairment	-0.02		0.02	707.67	**	180.64
Self_care	0.10	**	0.03	-670.80	**	290.48
Psychiatric	0.05	*	0.03	82.23		340.03
Neurology	0.01		0.02	51.93		289.91
Cardiovasc	0.11	**	0.02	1,096.55	**	174.45
Respiratory	0.08	**	0.02	614.63	**	282.6
Skeleton	0.02		0.02	168.90		172
Pain	0.02	*	0.01	225.60		165.77
Other_disease_1	0.08	**	0.01	175.62		159.89
Other disease_2+	0.07	**	0.02	688.10	**	343.04
Age	0.00		0	7.37		10.79
2nd gen immigrant	-0.02		0.04	188.22		1,194.22
Primary education		*reference*			*reference*	
Secondary education	0.02		0.01	31.90		175.83
Higher education ≤2	0.03		0.02	467.31		694.8
Higher education >2	-0.01		0.02	411.84		353.52
ln(dispinc)	0.02	**	0.01	-207.14	*	116.31
Married_cohab	0.03	**	0.01	-127.78		190.37
Child_1	0.00		0.01	-273.13		333.75
Child_2+	-0.03	**	0.02	-551.21	**	274.86
Large_city	0.05	**	0.01	159.56		185.73
Work_full_time	-0.01		0.01	-245.76		228.77
No. of observations	4,410			3,774		

Note: ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Appendix G: Excluding individuals with at most primary education, full results for men and women

Average marginal effects (A.M.E.) of the probability of accessing pharmaceuticals and, conditional on access, accessed number of defined daily doses (DDD)

	Men						Women					
	(1)			(2)			(3)			(4)		
	Probit on access			**GLM on**			**Probit on access**			**GLM on DDD|access**		
	A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.	A.M.E.		Std. Err.
Native Swedish	*reference*			*reference*			*reference*			*reference*		
Nordic origin	-0.03		0.05	545.47		347.69	-0.03		0.03	453.58		552.47
Western origin	0.02		0.05	-20.41		409.44	0.00		0.04	69.21		431.69
non-Western origin	0.07	***	0.03	204.10		242.42	-0.03		0.03	-441.70		445.33
SAH 1	*reference*			*reference*			*reference*			*reference*		
SAH 2	0.06	***	0.02	462.98	***	136.52	0.03	**	0.01	9.93		169.09
SAH 3	0.10	***	0.02	659.18	***	169.5	0.08	***	0.02	540.55	**	216.37
Underweight	-0.02		0.09	1,594.16	**	700.48	-0.03		0.03	44.36		377.06
Normal weight	*reference*			*reference*			*reference*			*reference*		
Overweight	0.04	**	0.01	-27.27		167.35	0.00		0.01	344.71	**	170.84
Exercise 1	*reference*			*reference*			*reference*			*reference*		
Exercise 2	-0.00		0.03	-45.57		261.19	-0.02		0.02	-635.01	*	328.76
Exercise 3	0.02		0.03	-178.81		281.4	-0.03		0.02	-41.86		517.64
Exercise 4	0.03		0.03	3.80		239.87	-0.01		0.02	-668.42	**	328.67
Smoker	0.01		0.02	269.64		236.4	-0.01		0.01	233.98		315.47
Mobility impairment	0.01		0.04	412.86	**	186.78	-0.02		0.02	667.94	***	214.86
Self_care	0.31	***	0.08	-677.12	**	297.12	0.11	***	0.03	-492.29		364.35
Psychiatric	0.09	*	0.05	591.10		416.53	0.05		0.03	-23.76		437.12
Neurology	0.08	*	0.04	-36.02		283.58	0.01		0.02	-358.74		282.78
Cardiovasc	0.21	***	0.03	1,353.66	***	152.01	0.14	***	0.02	1,185.48	***	163.35
Respiratory	0.08	***	0.03	269.05		204.56	0.08	***	0.03	533.63	*	313.46
Skeleton	0.05	*	0.03	-173.35		158.27	0.01		0.02	221.58		190.39
Pain	0.01		0.02	31.44		189.63	0.02	*	0.01	0.16		157.14
Other_disease_1	0.11	***	0.02	180.21		156.8	0.06	***	0.01	396.18	**	183.02
Other disease_2+	0.17	***	0.03	1,042.20	***	305.48	0.06	***	0.02	1,294.94	***	336.26
Age	0.00	***	0	23.52	***	6.41	-0.00	***	0	1.95		7.37
2nd gen immigrant	0.10	*	0.05	-545.43		369.6	-0.03		0.03	-1,053.81		806.69
Secondary education	*reference*			*reference*			*reference*			*reference*		
Higher education ≤2	0.03	*	0.02	95.63		193.92	0.01		0.01	503.30		371.65
Higher education >2	0.02		0.02	170.80		153.62	-0.02	*	0.01	286.63		306.99
lndispinc	0.03	***	0.01	292.35	*	150.96	0.03	***	0.01	-10.41		126.81
Married_cohab	0.04	**	0.02	-71.18		172.73	0.01		0.01	-109.74		173.99
Child_1	0.02		0.02	-29.95		428.45	0.01		0.01	-324.91		314.66
Child_2+	-0.05	**	0.02	-244.80		365.17	-0.04	***	0.02	-666.24	***	224.93
Large_city	0.00		0.02	-36.68		189.26	0.02	**	0.01	20.08		168.98
Work_full_time	0.00		0.02	-400.03	**	167.93	-0.00		0.01	-263.77		210.62
No. of observations	3,245			2,442			3,457			3,053		

Note: ***p-value<0.01, **p-value<0.05 and *p-value<0.1.

Chapter 5

Onset of Type 1 Diabetes in Young Adults and Investments in University Education

With Ida Lovén[a], Lenarth Nyström[b,c], and Mona Landin-Olsson[c, d]

5.1 Introduction

Growing evidence shows that health in early life and childhood is important for adult outcomes such as academic achievements (c. f. Almond and Currie, 2011; Rees and Sabia 2011). Evidence also establishes that parents contribute to their child's skill formation (Currie, 2009; Cunha and Heckman, 2008; Becker and Tomes, 1976; Behrman et al., 1982). When young children experience health problems, their parents' caregiving role, which is an essential part of parenting, intensifies and may become crucial for any long-term consequences regarding the children's health. The increased need for caregiving, in turn, may affect parents' possibilities of engaging in their children's schooling and other family activities. If so, then the link between university education and childhood health will partly reflect the degree of parental involvement, as early education is important for subsequent academic achievements.

This paper focuses on the less explored link between health in young adulthood and subsequent university education. This link is interesting as young adults are themselves responsible for their health behavior and their academic aspirations, while parents' roles are more advisory. Accordingly, the link reflects how health influences university education when ruling

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out the influence of earlier academic achievements. In general, young adults face the choice of entering the labor market or continuing to university education to increase their future employability and labor earnings. This decision relates to other choices in life. For example, university education has been found to delay family formation.[1] (Boschini et al., 2011; Lundin et al., 2008; Bjorklund, 2006), as both university education and family formation require substantial investments in time and effort (apart from the monetary costs). An unexpected health shock, such as the sudden onset of a lifelong disease, also requires time and effort invested to restore and maintain health. Such a change in life constraints can cause young adults to reevaluate previously set university aspirations and other choices in life.

Using data on individuals with type 1 diabetes onset in the age group 17-20 and population controls, this paper analyses the link between young-adulthood health and university education at age 30. We account for heterogeneity in socioeconomic background and sex by including control variables for parental level of education, and by conducting separate regressions for men and women and when relevant, for the socioeconomic groups. We account for an extensive set of fixed effects and explore if family formation mediates the link between type 1diabetes and university education.

The individual's perceived tradeoff between university education and other choices in life likely depends on personal characteristics, such as socioeconomic background and sex. Preferences that relate to socioeconomic factors and gender may, therefore, contribute to the observed link between parents' and descendants' educational level (Chevalier, 2004; Black et al., 2003; Mulligan, 1999; Dearden et al., 1997) and educational differences by gender (Boschini et al., 2011; Lundin et al., 2008; Bjorklund, 2006). Similarly to the intergenerational transition of human capital (Chevalier, 2004; Black et al., 2003; Mulligan, 1999; Dearden et al., 1997), having better off parents is likely also positive for health-related behavior, including disease-coping strategies when disease management is as complex as it is with type 1 diabetes. The associations of socioeconomic characteristics, health and health-related behavior are well known (c.f., Smith et al. 1979). Studies show, for instance, that socioeconomically advantaged individuals have (1) higher survival rates when it comes to cancer and cardiovascular illness (Schrijvers and Mackenbach, 1994; Smith et al., 1979; Peltonen et al., 2000), (2) adhere better to complex self-management treatments of HIV and diabetes (Goldman and Smith, 2002), and are (3) earlier adopters of new medical technologies (Rosvall et al., 2008; Glied and Lleras-Muney, 2008) than individuals of lower socioeconomic status.

As disease self-management is essential in diabetes care and, as previous studies show, adherence to disease management regimens varies across the socioeconomic strata, the degree and severity of subsequent diabetes-related complications may also vary with socioeconomic background. Furthermore, having different networks and prior experience, people across the socioeconomic strata may be more or less likely to assimilate the long-term consequences of type 1 diabetes for both health and work. Many diabetes-related complications first appear several years after onset, but their severity and timing are influenced by current lifestyle choices. Also, current educational choices may impact on one's future work situation and ability to incorporate health impairments into one's everyday life. Such potential differences in diabetes self-management and assimilation of long-term diabetes-related consequences, along with differences in other relevant constraints, suggest that individuals of different socioeconomic background respond differently to type 1 diabetes onset in terms of university aspirations.

Men and women may also respond differently to type 1 diabetes onset. One reason may be that family formation and university education are complements for men, but substitutes for women (e.g., Boschini et al., 2011; Lundin et al., 2008; Bjorklund, 2006). Another reason may be that type 1 diabetes increases the medical risks during pregnancy for both mother and child. Such risks may discourage some women with type 1 diabetes from choosing university education over family formation, while the elevated risks, which increase with age, may hasten other women's decisions to start a family and maybe forgo their academic career. Regardless of whether socially and/or biologically induced, these differences imply that the young-adulthood onset of type 1 diabetes will affect men's and women's educational decisions differently.

Comparing individuals with and without type 1 diabetes provides a good illustration of how an unexpected health shock affects subsequent university education. Type 1 diabetes typically appears as a rapid onset without prior symptoms, mimicking a before and after treatment study design. By focusing on type 1 diabetes onset in the age group 17-20, when compulsory education and the track in upper secondary education is already set, we reduce the influences onset may have on the young adults' eligibility for university education. [2] The disease is a lifelong, severe auto-immune disorder with both immediate and long-term negative health effects (Centers for Disease Control and Prevention, 2005). Destroying the body's ability to produce the insulin needed to maintain a normal blood glucose level, type 1 diabetes requires time-consuming management with regular glucose controls, daily insulin injections, a healthy diet, and physical

[2] For the time studied, children entering upper secondary education were generally aged 15 or 16 and chose among theoretical programs for further studies or vocational programs for labor market entrance.

exercise (Centers for Disease Control and Prevention, 2005). Consequently, Type 1 diabetes affects everyday life and increases future health insecurity, as severe diabetes-complications may develop despite proper diabetes management. Long-term diabetes complications involve, for instance, blindness, kidney failure, heart disease, stroke, nerve damage, and foot amputation. Besides, type 1 diabetes amplifies the risk of severe pregnancy-related complications for both the mother and child. Thereby, type 1 diabetes adds to the risks all young women face when delaying childbearing, and even more so with increasing age (Jonasson et al., 2007; Casson et al., 1997).

Despite extensive research, the exact combination of environmental and genetic factors, together with the chain of events, triggering type 1 diabetes onset remains unclear. Lifestyle factors (i.e., obesity and physical inactivity) that are associated with low education (Devaux and Sassi, 2013; Cutler et al., 2003; Molarius, 2003; Molarius et al., 2000; Lissner et al., 2000; Lahmann et al., 2000) do not appear to affect the lifetime risk of onset (American Diabetes Association, 2008). More likely, factors outside the individuals' control, such as genetics, cold climate, and virus infection in early life seem to be at play (Atkinson and Eisenbarth, 2001; Lernmark, 1999; The TEDDY Study Group, 2007). Due to this complexity and the sudden onset, type 1 diabetes is generally seen as an unanticipated health chock, which the individual is unable to influence beforehand (Persson et al., 2013; Minor, 2011; Steen Carlsson et al., 2010).

Still, a correlation between type 1 diabetes and university education does not necessarily imply a causal relationship; correlation may appear due to third factors that affect the likelihood of type 1 diabetes onset and the probability of a university education, e.g., innate ability or socioeconomic characteristics The presence of a third factor that affects university attainment likely involves additional, systematic, group level differences. Socioeconomics, for instance, could affect educational decisions and life style factors that, in turn, may increase the risk of disease development. However, recall that lifestyle factors do not appear to impact on the lifetime risk of type 1 diabetes. Moreover, none of the observable variables in our sample (measured pre type 1 diabetes onset) are associated with type 1 diabetes. This lack of association supports the notion that socioeconomic factors do not affect type 1 diabetes onset. 3[3] Nevertheless, we have no means for testing if e.g. the genetics of type 1 diabetes correlate with educational achievements. Epidemiologic studies have shown, however, that despite the heredity of type 1 diabetes, 90 percent of all newly diagnosed children with type 1 diabetes in Sweden have no close family member with type 1 diabetes (Dahlquist and Mustonen, 2000).

[3] We test for differences in means between individuals with and without type 1 diabetes for all background variables available in our data, and estimate probit regression models to test whether any of these variables predict type 1 diabetes onset. These analyses are detailed in section 4.

Previous research, using detailed register data for all individuals diagnosed with type 1 diabetes in Sweden, reveals that individuals with type 1 diabetes have education and labor market disadvantages. Children who were aged 0-14 at the onset of type 1 diabetes have lower grades from compulsory education (Persson et al., 2013; Dahlquist et al., 2007) and from theoretical upper secondary programs for university, and have a higher risk of unemployment later in life (Persson et al., 2013). Adults who were aged 14-34 the onset of type 1 diabetes have a higher risk of unemployment and lower annual labor earnings (Steen Carlsson et al., 2010). These studies, however, do not reveal if individuals change their university aspirations after the onset of type 1 diabetes, or if it is only their prerequisites for higher education that change.

Most other previous studies on diabetes and economic outcomes depend on small sample surveys (Milton et al., 2006), or cannot discriminate between type 1 and type 2 diabetes (Kahn, 1998; Bastida and Pagan, 2002; Brown et al., 2005; Latif, 2009; Vijan et al., 2004; Zhang et al., 2009; Harris, 2008). The two types of diabetes have fundamentally different pathogenesis and expected impact on university education. For instance, old age (when education is already completed) and life-style factors, such as obesity and physical inactivity (factors that are associated with low education), substantially increase the risk of developing type 2 diabetes (Stumvoll et al., 2005; Clausen et al., 1996; Prentice and Jebb, 1995), while the risk of type 1 diabetes onset depends on other factors (American Diabetes Association, 2008).

Using detailed register data for individuals diagnosed with type 1 diabetes for the age group 17-20 in Sweden, our results show no difference in university education at age 30 between men with and without type 1 diabetes. Contrarily, our results show that women with type 1 diabetes are less likely to have a university education than their peers at age 30. As the educational disparity persists at age 35, the gap portrays a permanent drop in university education rather than educational delay. Comparing women who have a university education, those with type 1 diabetes become mothers to a lesser extent than other women, suggesting that type 1diabetes sharpens the tradeoff between these two life choices-i.e., diabetes makes it more difficult to have both a university education and one or more children. In terms of redirecting life choices after diabetes onset, socioeconomic background seems to be of importance. After diabetes onset, women of high or low socioeconomic background appear to choose having children over university education, while women of middle socioeconomic background prioritize university education over motherhood.

These findings underline the importance of further research to gain knowledge of the mechanisms behind the interplay between the young-adulthood onset of type 1 diabetes and

subsequent university education. Family formation is one possible reason for men and women reacting differently after onset. It also suggests that any policy interventions should take into account the fact that type 1 diabetes may intensify the conflict between motherhood and university education, and that women of different socioeconomic background may respond differently to such a conflict.

The structure of the paper is as follows. Section 2 theoretically describes how type 1 diabetes might affect educational decisions. Section 3 presents the data and descriptive statistics. Section 4 details the econometric strategy. Section 5 contains the main results, and section 6 the sensitivity analyses. Section 7 discusses the results.

5.2 Theoretical framework

University education can be regarded as a human-capital investment generating welfare or utility prospects in terms of greater long-term labor market returns such as employability, career-track, wage-rate, and working conditions (e.g., time and work-hour flexibility, and workplace flexibility fostering safety and health). Becker's seminal human-capital-model (Becker, 1962) conceptualizes the demand for human capital, and proposes that the individual, subject to own preferences and resource constraints, opts for the educational level that maximizes his or her lifetime utility. In other words, the individual balances his or her perceived forgone welfare from investing in education against future (time-discounted) university welfare returns. The forgone welfare concerning educational investments refers to the fact that resources allocated to education have alternative uses. For instance, the individual can use time and effort to earn labor income, rear children or any other activity that generates welfare.

Becker's household time allocation model (Becker, 1965) and more recent life cycle family models (c.f., Greenwood et al., 2003) illustrate that the division of labor within the household implies that women's career, and thus university decision, may conflict with childbearing and childrearing. Evidence show that university educated women who postpone motherhood (Boschini et al., 2011; Gustafsson and Adriaan, 2006; Adsera, 2011), have fewer children or are more often childless (Boschini et al., 2011) than less educated women. University educated men also postpone fatherhood (Boschini et al., 2011; Gustafsson and Adriaan, 2006; Adsera, 2011) but have more children and are more seldom childless (Boschini et al., 2011) than less educated men. Empirical evidence thus indicates that university education delays parenthood and proposes a tradeoff between family formation and women's career aspirations.

Conceptually, the university decision can be regarded as a process where the young adult weighs the perceived educational costs again benefits, and only invests if the benefits outweigh the costs. The individual's perception of costs and benefits is governed by time-preference (i.e., willingness to forgo immediate utility for forthcoming payoffs) and preferences for university education contra preferences for other objectives in life, such as family formation. In short, myopic time preferences (i.e., present time orientation) or strong family preferences (i.e., aversion to childlessness) lower university aspirations. It has been suggested that there are socioeconomic differences in parents' ability to invest in their children's time preferences (c.f., Becker and Mulligan 1997); thus the intergenerational transmissions of human capital may partly operate via time preferences.

The sudden onset of type 1 diabetes changes life-constraints and imposes greater uncertainty about future health, which may, for instance, form more myopic time preferences. Alternatively, the permanent up-shift in demand for health investments necessary to maintain health may affect the relevant resource constraint, increasing the perceived value of the remaining time available for other activities. Consequently, young adults may modify their university decision and other prior-set life-goals.

First, we assume that type 1 diabetes affects time-preferences due to higher short- and long-term morbidity and mortality risks. Given that type 1 diabetes affects time-preferences, (1) university investments increase if e.g. the responsibilities of disease management foster future-oriented time preferences, making the young adult more willing to forgo present utility for future educational returns, or (2) university investments decrease if e.g. the higher risk of adverse health outcomes forms myopic time-preferences, making the individual more present-oriented.

Second, we assume that type 1 diabetes increases women's cost of university education as enrollment postpones parenthood, and that it amplifies the risk of fertility-related problems when postponing motherhood. In other words, prioritizing a university education and delaying motherhood implies a greater risk for fertility-related complications for women with type 1 diabetes than for other women. Given that type 1 diabetes modifies women's fertility decision (i.e., fertility transit and or overall fertility) (1) university investments increase if type 1 diabetes, by suppressing family aspirations, lowers women's forgone cost for university enrollment, or (2) university investments decrease if type 1 diabetes lowers the transit age for motherhood to minimize the risk of type 1 diabetes-related fertility problems, and raises the alternative cost of university enrollment. Given that type 1 diabetes specifically affects fertility in women, we expect that type 1 diabetes onset affects university education differently for men and women.

The positive link between the parents' educational level and their child's prospects for university education is well established. Having better-off parents is also beneficial in terms of financial in-vivo transfers to adult children (c.f., Henretta et al., 2002; Grundy, 2005. As the better-off parents have more financial resources, they are better able to support their adult child. If parents want to help their adult child after type 1 diabetes onset, young adults with a more advantaged socioeconomic background may receive more support than young adults with a less advantaged socioeconomic background. Given that, e.g., financial transfers (by relaxing monetary constraints) affect the individual's decision-making process regarding university education contra other life objectives, we expect socioeconomic heterogeneity in the effect of type 1 diabetes on university education. The better-off parents may also access and transfer relevant health information enabling their adult children to make more informed decisions.

5.3 Data

This study uses the Econ-DISS database, which combines the national Diabetes Incidence Study in Sweden (DISS) with national population registers. Since 1983, DISS has registered all diagnosed diabetes cases in the age group 15 to 34 in Sweden (Ostman et al., 1986, 2008). The reporting physician classifies the diabetes type according to current clinical diabetes criteria [1983-91 (WHO, 1980, 1985); 1992 onwards (CDC The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1997)]. Using a case-control framework, Statistics Sweden matches each individual in the DISS to four control individuals by age, sex, and municipality of residence at the time of diagnosis, and identifies the parents of all individuals from the Multi-Generation Register (Statistics Sweden, 2009). Statistics Sweden then adds yearly data on demographic, socioeconomic, and work-related variables from the LISA database (Statistics Sweden, 2011) for the period 1990-2005. For details see Steen Carlsson et al. (2010).[4]

Ideally, we should limit this study to individuals with type 1 diabetes onset just after completing upper secondary education. Due to the limited disease frequency, however, such a restriction results in too small samples. Therefore, we select individuals diagnosed with type 1 diabetes in the age group 17 to 20 (n = 1,034) (in years 1983-1995) and their matched controls (n = 4,136) and study outcomes for the period 1993-2005. At age seventeen, most young people in Sweden are about to complete upper secondary education. Consequently, the lower age limit rules out that type 1 diabetes affects either educational achievements at the compulsory level or

4 The research program was approved by the Regional Ethical Review Board in Lund, Sweden (dnr 393/ 2005).

the choice between theoretical and vocational program for upper secondary education. In other words, we reduce potential disparities in academic prerequisites between individuals with and without type 1 diabetes that may affect university decisions. At the age of twenty, many young people are still facing the choice of university education. For the period studied here, the median age for first-time university enrollment was 22 and the median age when earning a degree was 27-28 (Statistics Sweden, 2008).

To exclude supplementary training and retraining following from unemployment later in life, we measure higher education at age 30 (and at age 35 in the sensitive analyses). Due to data restrictions, we exclude individuals with missing data on own education (3 with diabetes, 14 controls), individuals born outside Sweden or if their parent(s) were born outside Sweden (145 with diabetes, 779 controls). After these exclusions, the sample consists of 886 individuals with type 1 diabetes and 3,343 controls.

Table 1: Descriptive statistics for men at age 30

Standard deviation (SD) in parentheses.

	Diabetes		Controls	
	Mean %	(SD)	Mean	(SD)
Education				
Compulsory	14.0	(34.7)	11.8	(32.3)
Upper secondary	55.8	(49.7)	54.5	(49.8)
University	30.2	(46.0)	33.7	(47.3)
Covariates				
Married	22.6	(41.9)	21.9	(41.4)
Divorced	2.2	(14.7)	1.6	(12.5)
No. of children	0.66	(0.99)	0.65	(0.94)
Background factors				
Mothers' education				
Compulsory	35.1	(47.8)	34.8	(47.7)
Upper secondary	42.8	(49.5)	41.3	(49.3)
University	18.4	(38.8)	21.2	(40.9)
Missing data	3.7	(18.9)	2.7	(16.1)
Fathers' education				
Compulsory	39.1	(48.8)	38.2	(48.6)
Upper secondary	34.2	(47.5)	37.2	(48.3)
University	17.7	(38.2)	18.8	(39.1)
Missing data	9.1	(28.8)	5.9	(23.6)
Observations	407		1577	

Table 2: Descriptive statistics for women at age 30
Standard deviation (SD) in parentheses.

	Diabetes		Controls	
	Mean	(SD)	Mean	(SD)
Education				
Compulsory	12.9	(33.6)	8.9	(28.5)
Upper secondary	55.4	(49.8)	51.0	(50.0)
University	31.7	(46.6)	40.1	(49.0)
Covariates				
Married	31.3	(46.4)	32.5	(46.9)
Divorced	3.8	(19.0)	2.9	(16.7)
No. of children	0.89	(0.97)	1.10	(1.04)
Background factors				
Mothers' education				
Compulsory	33.8	(47.4)	33.0	(47.1)
Upper secondary (%)	43.3	(49.7)	41.7	(49.3)
University	18.8	(39.1)	22.0	(41.4)
Missing data	4.2	(20.0)	3.3	(17.9)
Fathers' education				
Compulsory	36.7	(48.3)	35.8	(48.0)
Upper secondary (%)	36.7	(48.3)	38.1	(48.6)
University	17.5	(38.1)	20.1	(40.1)
Missing data	9.2	(28.9)	6.0	(23.8)
Observations	240		911	

Note: Missing data indicates if educational data is missing. Significant mean differences (on at least the 10 % level) between women with and without type 1 diabetes for the following variables: Compulsory, University, No. of children, and Fathers' missing data.

Dependent variable

The dependent variable University education is a dummy variable indicating if the individual has university education at age 30. University education is defined as having credits from a Swedish university or university college corresponding to at least 20 weeks of full-time studies. [5] Descriptively, Table 1 shows no significant differences in university education at age 30 for men, whereas Table 2 shows significant differences, on the 10 % level, between women with and without type 1 diabetes. The differences in education originate at onset (ages 17-20) and no differences are evident before onset (see Figure A.1 in Appendix A showing years of education by age).

[5] We use a rather crude definition of university education as the Swedish educational system changed during the studied period. Certain types of education have become longer and we are unable to track these changes in our data. Still, our results are robust to defining university education as having credits corresponding to more than two years of full-time studies (see Section 4).

Independent variables

We control for socioeconomic background and family related variables. Socioeconomic background is pre-onset while starting a family (generally) is post-onset. The influence of socioeconomic factors is well recognized in the health-education literature (c.f., Currie, 2009), and socioeconomic background is frequently measured by parents' level of education. Better educated parents, on average, earn more income and are therefore more likely to live in areas providing high quality schooling. The better educated parents may also have higher (acquired and or innate) ability, enabling them to better support their children's learning by, for instance, helping out with homework. We use the parents' level of education (compulsory, upper secondary, and university level) to indicate *socioeconomic background* (SEB) in two ways.[6] First, we use mother's and father's level of education separately as control variables in the regression analysis. Second, we combine mother's and father's education into low, middle, and high SEB for the graphical analysis and the stratified regression analysis. We define SEB as low SEB if the highest educated parent has at most compulsory education, middle SEB if the highest educated parent has at most upper secondary education, and high SEB if the highest educated parent has university education.

If SEB correlates with onset of type 1 diabetes, these background factors will differ between two individuals with and without diabetes. However, no significant differences appear in the descriptive statistics in Tables 1 and 2. [7] Nevertheless, we account for SEB either by controlling for parental level of education in the regression analysis, or by stratifying parts of our analysis according to low, middle, and high SEB.

To illustrate the heterogeneity in university attendance related to SEB, Figure 1 shows the proportion of women and men with university education separately for individuals with and without type 1 diabetes and stratified into low, middle, and high SEB. Figure 1 indicates that (1) a lower proportion of people with type 1 diabetes have university education and (2) these differences in university education are particularly pronounced among women with low SEB or high SEB.

Life-defining choices, such as family formation, may compete with university education. We therefore look into differences in own family status (measured as number of children and marital status at age 30) as potential mediators in the link between type 1 diabetes and university

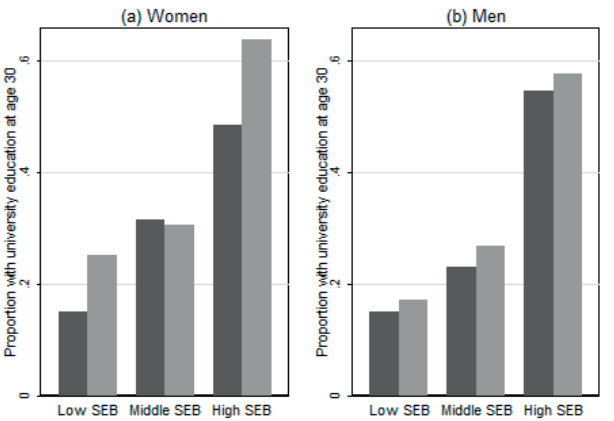
[6] Ideal is to measure SEB in childhood. Due to data limitations we use the fist available SEB observation in 1990 (when individuals are 16-27 years old) which is reasonable as SEB is rather constant over time.

[7] Except for having a father with missing data on education, but this variable is only relevant for a small number of people.

education. Descriptively, the family status of men with type 1 diabetes is no different from men without type 1 diabetes (Table 1), while women with type 1 diabetes have fewer children (significant on at least the 10% level, Table 2) compared to women without type 1 diabetes. Figure A.2 in Appendix A, showing the proportion of women with one or more children, indicates that these differences are concentrated to women with medium SEB.

Figure 1: Proportion of university educated women (a) and men (b) with type 1 diabetes (black bars) and controls (gray bars) by socioeconomic background (SEB)

SEB is defined as *low* if the highest educated parent has at most compulsory education, *middle* if the highest educated parent has at most upper secondary education, and *high* if the highest educated parent has university education.



5.4 Empirical methods

We estimate the relationship of type 1 diabetes onset at the age of 17 to 20 and university education at age 30 separately for women and men, using the following probit model specification using the Huber/White/sandwich estimate of variance to generate robust standard errors:

$$Y_i = \alpha + \beta D_i + \gamma X_i + \lambda Year + \varepsilon_i, \qquad \text{(eq. 1).}$$

The dependent variable, Y_i, is university education at age 30. α is the common constant. D_i, is a dummy indicating individuals with type 1 diabetes onset at the age of 17 to 20. X_i is a set of dummy variables indicating SEB. Z_i is a set of variables measuring family status at age 30 (i.e., number of children and dummies for marital status). *Year* is a year dummy set (i.e., calendar time-fixed effects) controlling for factors such as education policies, and economic or technology changes that may influence educational attainment. ε_i is the idiosyncratic error term.

The data is based on a case-control study design where four controls are matched to each case with type 1 diabetes by age, sex, and municipality of residence at the time of diagnosis. This design introduces at least three sets of fixed effects implicitly included as control variables in all the analyses. The first fixed effect, year of birth, is a proxy for cohort effects that controls for differences in composition and size of each cohort. The second, sex, controls for gender differences, and the third, municipality of residence at the time of diagnosis (inclusion for controls), controls for access to universities and to the local labor market at the time of onset (inclusion).

Given that these fixed effects capture all potential differences (unobservable and observable) between the groups prior to onset, any differences past onset will be effects of type 1 diabetes. That the fixed effects in fact wipe out all differences between the groups prior to onset is, however, a strong and non-testable assumption. Still, we test if the groups differ in observable background characteristics by running a regression on the probability of being in the diabetes group (in the sensitivity analysis in section 6). Moreover, we introduce control variables stepwise to evaluate if any background factor correlates with the type 1 diabetes dummy in our main analysis. First, we control for the fixed effects (via *Year* and the matching of controls), and then we add controls for SEB (X_i). Any differences in the estimated diabetes coefficients in the two specifications (i.e., indicating correlation) could mean that SEB is a confounder relating to both

type 1 diabetes onset and education. Lastly, we also add controls for family status (Z_i) to assess if these variables are potential mediators through which type 1 diabetes may affect education.

To allow for socioeconomic heterogeneity in the link between university education and type 1 diabetes, we repeat the analysis stratified into low, middle, and high SEB. [8] To account for differences in family status, we look further into differences in motherhood between women with and without type 1 diabetes. Specifically, we estimate the probability of having one or more children stratified into low, middle, and high SEB. Then, we estimate the probability of having both university education and one or more children.

To assess the robustness of the results, we perform several sensitivity analyses (in section 6). First, we test the results' sensitivity to how we define university education by redefining it as having more than years of full-time university studies. Similar estimates for both definitions indicate that the differences between the groups (diabetes and controls) are not only related to enrollment but also to continuing studies. Second, we perform a placebo test to test if any other group level differences, apart from onset of type 1 diabetes, appear to influence the results. This test repeats the main analysis for individuals with onset (inclusion) at the age of 24-26, by when they ought to have made their choice of university education. Third, we test if the results are driven by the youngest at the time of onset and exclude individuals with onset (inclusion) at age 17. Fourth, we test for educational delays by estimating the link between type 1 diabetes and education at age 35 instead of 30. Fifth, we test for later family formation by estimating the link between type 1 diabetes and motherhood at age 35. Sixth, we test if the probability of being in the type 1 diabetes group is associated with the background variables in our data and thereby indicates endogeneity problems.

Additionally, we test alternative empirical models (e.g., ordered probit, generalised ordered probit, and ordinary least square) and an alternative definition of SEB.[9] These results are in line with the overall findings of this study and are available on request.

[8] We use the same definition as in the graphical analysis i.e., *low SEB* if the highest educated parent has at most compulsory education, *middle SEB* if the highest educated parent has at most upper secondary education, and *high SEB* if the highest educated parent has university education

9 Low SEB if both parents have at most compulsory education, middle SEB if the lowest educated parent has at most upper secondary education, and high SEB if both parents have university education.

5.5 Results

Estimated probabilities of university education

Using equation 1, table 3 shows probit estimations for women (columns 1 to 3) and men (columns 4 to 6). Appendix B reports the marginal effects for the control variables. Controlling for fixed effects, columns (1) and (4) show the average difference in the likelihood of having university education between individuals with and without type 1 diabetes. The remaining columns show the results from estimations after adding controls for SEB (columns 2 and 5) and family status (columns 3 and 6). Independent of specification, the results show that women with type 1 diabetes are less likely to have university education (on at least the 5% significance level), but no such difference is evident for men.

Controlling for fixed effects, women with type 1 diabetes are 8.9% less likely to have university education than other women. This significant and sizable relationship also remains after controlling for SEB (the likelihood decreases somewhat from 8.9% to 8.3%) and family status (the likelihood increases from 8.3% to 10.4%) (column 3). The increase in the type 1 diabetes coefficient when including family status indicates that type 1 diabetes correlates with both family status and university education. Still, this change in estimates is only marginal and the relationship between type 1 diabetes and university education appears robust to both SEB and family status.

Table 3: Probit (average marginal effects) estimation of university education at age 30

	Women			Men		
	(1)	(2)	(3)	(4)	(5)	(6)
	University	University	University	University	University	University
Diabetes (d)	-0.0898***	-0.0827**	-0.104***	-0.0368	-0.0283	-0.0274
	(0.0342)	(0.0357)	(0.0353)	(0.0256)	(0.0261)	(0.0262)
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1151	1151	1151	1984	1984	1984

Marginal effects, (d) for discrete change of dummy variable from 0 to 1.Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls)

Estimated probabilities of university education by socioeconomic background

Table 4 shows the results when including fixed effects and stratifying the sample by SEB for women (columns 1-3) and men (columns 4-6). Columns (1) and (4) show the estimates for individuals with low SEB, columns (2) and (5) for individuals with middle SEB, and columns (3) and (6) for individuals with high SEB. When allowing for SEB related heterogeneity, the results show a strong negative link between education and type 1 diabetes for women with low or high SEB. Women with type 1 diabetes and low SEB are on average 10.4% less likely to have university education. Correspondingly, women with type 1 diabetes and high SEB are 17.2% less likely to have higher education. In contrast, the link between education and type 1 diabetes is small and insignificant for women with middle SEB. For men, there is still no relationship between type 1 diabetes and university education regardless of SEB.

Table 4: Probit (average marginal effects) estimation of university education at age 30 by socioeconomic background (SEB)

	Women			Men		
	(1)	(2)	(3)	(4)	(5)	(6)
SEB	Low	Middle	High	Low SEB	Middle	High
Diabetes (d)	-0.104*	0.00641	-0.172**	-0.0241	-0.0397	-0.0396
	(0.0557)	(0.0500)	(0.0700)	(0.0394)	(0.0347)	(0.0519)
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Observation	262	524	350	451	928	580

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls)

Table 5: Probit (average marginal effects) estimation of having one or more children at age 30 by socioeconomic background (SEB) for women

	(1)	(2)	(3)
	Low SEB	Middle SEB	High SEB
Diabetes (d)	-0.0360	-0.173***	-0.0638
	(0.0747)	(0.0534)	(0.0682)
Fixed effects	Yes	Yes	Yes
Observations	262	524	350

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls).

Estimated probabilities of having children

We explore the interplay of type 1 diabetes, university education, and motherhood in two ways: First, we estimate differences in the probability of having one or more children. Second, we estimate differences in the probability of having both university education and one or more children.

Table 5 presents the results for the first analysis, controlling only for fixed effects and stratifying the sample by SEB. Women with type 1 diabetes and middle SEB are, on average, 17.3% less likely mothers compared to women without type 1 diabetes. This link is small and insignificant for women with low or high SEB.

Combining these results (Table 5) with the stratified results for the probability of having university education (Table 4) indicates that type 1 diabetes sharpens the competition between motherhood and university education. Women with type 1 diabetes and low or high SEB have a lower likelihood of higher education, but their likelihood of motherhood appears unaffected, while women with type 1 diabetes and middle SEB have a lower likelihood of motherhood, but their likelihood of higher education appears unaffected. Drawing on this finding together with the recollection that type 1 diabetes increases the risk of fertility-related problems, women with type 1 diabetes and low or high SEB have children to the same extent as women without type 1 diabetes, possibly at the expense of abstaining from higher education. On the other hand, women with type 1 diabetes and middle SEB have university education to the same extent as women without type 1 diabetes, possibly at the expense of abstaining from having children.

Table 6 presents the results from the second analysis, assessing differences in the probability of having both university education and one or more children. These results also suggest that type 1 diabetes sharpens the tradeoff between university education and motherhood. The type 1 diabetes coefficient is significant and sizable and remains so when controlling for SEB as well.

Table 6: Probit (average marginal effects) estimation of having both university education and one or more children at age 30 for women

	(1) University*Child	(2) University*Child
Diabetes (d)	-0.0865***	-0.0815***
	(0.0248)	(0.0247)
Fixed effects	Yes	Yes
SEB	No	Yes
Observations	1151	1151

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls).

5.6 Sensitivity analyses

When redefining the outcome variable as having two or more years of university education, the estimates for women only decreases slightly (Table 7). This finding indicates that the differences between women with and without type 1 diabetes persist past enrollment. The result from the placebo test (Table 8), estimating the relationship between university education and type 1 diabetes onset at the age of 24-26, shows no association. This test rejects that the negative link between type 1 diabetes and university education is driven by any (observable or unobservable) group level difference other than type 1 diabetes. The results are robust for omitting individuals with type 1 diabetes onset at age 17 (and controls) (Table 9). This robustness ensures that the youngest individuals are not the ones driving the result. The result from the test that estimates the relationship between type 1 diabetes onset in the age group 17-20 and university education at age 35 generates similar results to the analysis for university education at age 30 (Table 10). This similarity indicates a permanent shortfall in education rather than educational delay. We come to the same conclusion when estimating the probability of having one or more children at age 35 (Table11). We also test for the probability that being in the type 1 diabetes group is associated with the background variables in our data and thereby indicates endogeneity problems (Table 12). The results show no such association, suggesting that the individuals' or the parents' behavior does not influence onset. 9[10]

Table 7: Probit (average marginal effects) estimation of having more than two years of university education at age 30

	Women			Men		
	(1)	(2)	(3)	(4)	(5)	(6)
	Universi	Universi	Universi	Universi	Universi	Universi
Diabetes (d)	-0.0610*	-0.0530*	-0.0682**	-0.0101	-0.00372	-0.00310
	(0.0316)	(0.0322)	(0.0312)	(0.0232)	(0.0231)	(0.0231)
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1151	1151	1151	1984	1984	1984

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls)

[10] There is a positive association between missing data on the father's level of education and type 1 diabetes. As only a few individuals have missing data on the father's level of education, the association is likely of lower importance.

Table 8: Probit (average marginal effects) estimation of university education at age 30 for onset (inclusion) ages 24-26

	Women			Men		
	(1) Universi	(2) Universi	(3) Universi	(4) Universi	(5) Universi	(6) Universi
Diabetes (d)	-0.0212	-	-0.0224	0.0163	0.00929	0.00103
	(0.0363)	(0.0381)	(0.0381)	(0.0261)	(0.0265)	(0.0262)
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	1069	1069	1069	1855	1855	1855

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls)

Table 9: Probit (average marginal effects) estimation of university education at age 30 excluding those with onset (inclusion) at age 17

	Women			Men		
	(1) Universit	(2) Universit	(3) Universit	(4) Universit	(5) Universit	(6) Universit
Diabetes (d)	-0.0749*	-0.0726*	-0.0892**	-0.0318	-0.0220	-0.0219
	(0.0392)	(0.0406)	(0.0409)	(0.0287)	(0.0294)	(0.0296)
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	896	896	896	1537	1537	1537

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls).

Table 10 Probit (average marginal effects) estimation of university education at age 35

	Women			Men		
	(1) University	(2) University	(3) University	(4) University	(5) University	(6) University
Diabetes (d)	-0.0984**	-0.0842*	-0.0864*	-0.0384	-0.0333	-0.0329
	(0.0448)	(0.0464)	(0.0468)	(0.0334)	(0.0338)	(0.0337)
Fixed Effects	Yes	Yes	Yes	Yes	Yes	Yes
SEB	No	Yes	Yes	No	Yes	Yes
Family status	No	No	Yes	No	No	Yes
Observations	653	653	653	1124	1124	1124

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses,

*** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls.)

Table 11 Probit (average marginal effects) estimation of having one or more children at age 35 by socioeconomic background (SEB) for women

	(1) Low SEB	(2) Middle SEB	(3) High SEB
Diabetes (d)	-0.128	-0.240***	-0.0828
	(0.0802)	(0.0735)	(0.0934)
Fixed effects	Yes	Yes	Yes
Observations	183	247	184

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1.Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls.)

Table 12 Probit (average marginal effects) estimation of being in the type 1 diabetes group

	Women Diabetes	Men Diabetes
Compulsory M (d)	-0.00453	-0.00936
	(0.0288)	(0.0209)
University M (d)	-0.0274	-0.0271
	(0.0325)	(0.0254)
Missing M (d)	0.0125	0.0220
	(0.0711)	(0.0597)
Compulsory F (d)	0.00802	0.0158
	(0.0289)	(0.0218)
University F (d)	-0.0112	0.0111
	(0.0351)	(0.0285)
Missing F (d)	0.0781	0.0902*
	(0.0587)	(0.0461)
Fixed effects	Yes	Yes
Observations	1151	1984

Average Marginal effects, (d) for discrete change of dummy variable from 0 to 1. Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls).

5.7 Discussions

Using Swedish nation-wide register data over individuals with young-adulthood onset of type 1 diabetes (ages 17-20), this paper shows that women are less likely to have a university education (at age 30) compared to women without type 1 diabetes, while no such difference appear for men. The negative link between type 1 diabetes and university education for women remains after controlling for socioeconomic background, family status, and fixed effects (year, cohort, and municipality of residence respectively). The link persists when estimating the relationship at age 35. This persistence indicates a permanent shortfall in education rather than educational delay. Although the underlying mechanisms remain unknown, sex differences in fertility constraints

appear to be one prominent reason why type 1 diabetes is associated negatively with university education for women but not for men. Given that university studies delay family formation (Boschini et al., 2011; Gustafsson and Adriaan, 2006; Adsera, 2011) and that type 1 diabetes amplifies the risk of fertility-related problems when delaying fertility (Jonasson et al., 2007; Casson et al., 1997), it is not surprising that our result portrays an enhanced tradeoff between university education and motherhood after type 1 diabetes onset.

Empirical findings show that better educated people adhere better to complex self-management treatments (Goldman and Smith, 2002) and adopt new medical technologies faster (Rosvall et al., 2008) than the less educated. Intuitively, our finding may appear striking as it demonstrates a reversed U-shaped socioeconomic pattern between type 1 diabetes and university education, where women with a high SEB are least likely to have a university education. However, the underlying reason for not attending university after type 1 diabetes onset could vary across the socioeconomic strata. For example, as type 1 diabetes imposes increased uncertainty about future health, and as most educational returns are future gains, women with different SEB may respond differently to these uncertainties. Furthermore, SEB may (through e.g. differing networks) relate to access to diabetes-specific information. If women with a high SEB are more likely than other women to assimilate the knowledge that diabetes induces pregnancy-related complications, and that these complications increase with age, then women with a high SEB who wish to become mothers may be less inclined, than women with a lower SEB, to postpone motherhood for university education.

Our results show a drop in the likelihood of university education for women with type 1 diabetes and high SEB, but they have the same likelihood of being mothers as other women with a high SEB, in spite of their higher risk of pregnancy-related complications. We find the same pattern among women with a low SEB, though the drop in university education is somewhat smaller for women with type 1 diabetes and a low SEB. Contrarily, women with type 1 diabetes and a middle SEB do not show a drop in university education, but they are less likely mothers compared to women with a middle SEB. As individuals with higher SEB receive more financial support from their parents throughout life (c.f., Henretta et al., 2002; Grundy, 2005), women with type 1 diabetes and a high SEB may be more willing than other women with type 1 diabetes to forgo future earnings premiums from university education, and still have the financial means to start a family.

Taken together, these results suggest that type 1 diabetes may intensify the conflict between motherhood and university education, and that women of different socioeconomic backgrounds

may respond differently to such a conflict. Still, the uncertainty in the exact mechanisms governing women's educational decisions calls for prudency in policy interventions and encourages further research.

The magnitude of the link between type 1 diabetes and university education is likely contextual and affected by the education and health care systems. In Sweden, university education and health care are primarily publicly funded and the pharmaceutical reimbursement system fully subsidizes insulin for individuals with type 1 diabetes. As diabetes management is costly, one would expect a larger negative link between type 1 diabetes and university education in privately funded health care settings with substantial out of pocket payments for pharmaceutical and medical care. The reason is that present health care expenditure (and inadequate financial markets for borrowing) likely increases the demand for present labor market earnings.

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Appendix A

Figure A.l: Years of education by age for women (a) and men (b) with type 1 diabetes (black line black diamonds) and

controls (gray line with hollow diamonds)

Secondary education is often registered with a lag; therefore, the data points at age 16-18 should be interpreted cautiously.

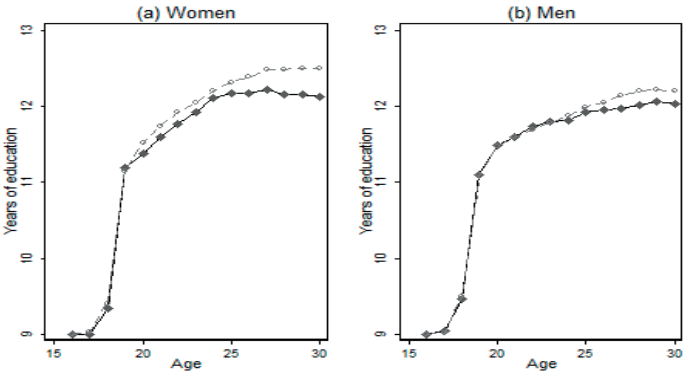
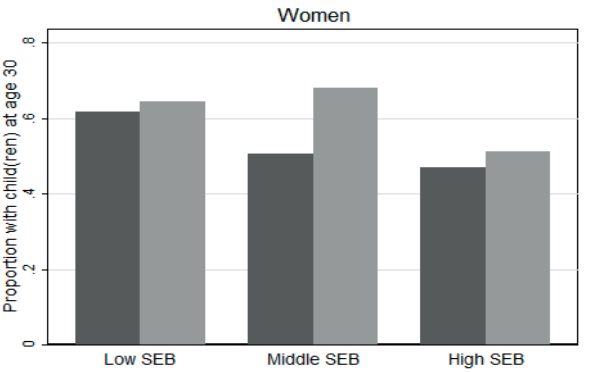


Figure A.2: Proportion of women with one or more children among women with type 1 diabetes (black bars) and controls (gray bars) by socioeconomic background SEB

SEB is defined as *low* if the highest educated parent has at most compulsory education, *middle* if the highest educated parent has at most upper secondary education, and *high* if the highest educated parent has university education.



Appendix B

Table 13: Probit (average marginal effects) estimation of university education at age 30, full results for men and women

	Women			Men		
	(1)	(2)	(3)	(4)	(5)	(6)
	University	University	University	University	University	University
Diabetes (d)	-0.0898***	-0.0827**	-0.104***	-0.0368	-0.0283	-0.0274
	(0.0342)	(0.0357)	(0.0353)	(0.0256)	(0.0261)	(0.0262)
1993 (d)	ref.	ref.	ref.	ref.	ref.	ref.
1994 (d)	-0.138	-0.169*	-0.152*	-0.0185	-0.0432	-0.0473
	(0.0974)	(0.0894)	(0.0922)	(0.0762)	(0.0736)	(0.0727)
1995 (d)	-0.158*	-0.202***	-0.202***	-0.00636	-0.0339	-0.0356
	(0.0820)	(0.0750)	(0.0741)	(0.0711)	(0.0692)	(0.0679)
1996 (d)	-0.0764	-0.169**	-0.176**	-0.0369	-0.0552	-0.0543
	(0.0885)	(0.0791)	(0.0777)	(0.0695)	(0.0680)	(0.0670)
1997 (d)	-0.155*	-0.186**	-0.173**	-0.0165	-0.0439	-0.0491
	(0.0805)	(0.0767)	(0.0784)	(0.0704)	(0.0683)	(0.0669)
1998 (d)	-0.0607	-0.145*	-0.160*	-0.0298	-0.0529	-0.0602
	(0.0977)	(0.0878)	(0.0848)	(0.0706)	(0.0688)	(0.0671)
1999 (d)	-0.136	-0.204***	-0.193**	-0.104	-0.150***	-0.147***
	(0.0865)	(0.0762)	(0.0784)	(0.0649)	(0.0577)	(0.0571)
2000 (d)	0.00390	-0.113	-0.118	0.0644	0.0262	0.0212
	(0.101)	(0.0909)	(0.0897)	(0.0764)	(0.0750)	(0.0736)
2001 (d)	0.00645	-0.0689	-0.0643	-0.0161	-0.0919	-0.0975
	(0.0981)	(0.0930)	(0.0932)	(0.0736)	(0.0658)	(0.0639)
2002 (d)	0.0958	0.00919	0.00357	-0.0243	-0.0891	-0.0985
	(0.101)	(0.0990)	(0.0985)	(0.0695)	(0.0634)	(0.0610)
2003 (d)	-0.0196	-0.0820	-0.0677	0.0998	0.0151	0.00232

	(0.0966)	(0.0914)	(0.0925)	(0.0760)	(0.0727)	(0.0708)
2004 (d)	0.0633	-0.0504	-0.0530	0.0850	-0.0177	-0.0316
	(0.0999)	(0.0950)	(0.0946)	(0.0751)	(0.0698)	(0.0676)
2005 (d)	0.0409	-0.0552	-0.0593	0.166**	0.0955	0.0718
	(0.0973)	(0.0930)	(0.0920)	(0.0798)	(0.0796)	(0.0777)
Compulsory M (d)		-0.0233	-0.0198		-0.0577**	-0.0536**
		(0.0362)	(0.0368)		(0.0253)	(0.0254)
Upper secondary M (d)		ref.	ref.		ref.	ref.
University M (d)		0.261***	0.234***		0.169***	0.160***
		(0.0412)	(0.0425)		(0.0322)	(0.0322)
Missing M (d)		-0.0310	-0.0458		-0.158***	-0.158***
		(0.0831)	(0.0841)		(0.0524)	(0.0531)
Compulsory F (d)		-0.0815**	-0.0847**		-0.0906***	-0.0923***
		(0.0352)	(0.0354)		(0.0251)	(0.0251)
Upper secondary F (d)		ref.	ref.		ref.	ref.
University F (d)		0.153***	0.122***		0.227***	0.217***
		(0.0443)	(0.0451)		(0.0340)	(0.0342)
Missing F (d)		-0.00644	-0.0319		-0.0182	-0.0122
		(0.0639)	(0.0631)		(0.0462)	(0.0474)
Children			-0.113***			-0.0775***
			(0.0169)			(0.0144)
Married (d)			0.140***			0.120***
			(0.0372)			(0.0319)
Divorced (d)			-0.149			-0.0707
			(0.0951)			(0.0834)
Constant						
Observations	1151	1151	1151	1984	1984	1984

Average marginal effects, (d) for discrete change of dummy variable from 0 to 1.Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.Fixed effects for year, cohort, and municipality of residence at the time of onset (inclusion for controls).

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