Three Essays on Time Preference in Health Economics

Kumulative Dissertation

der Wirtschaftswissenschaftlichen Fakultät

der Universität Augsburg

zur Erlangung des Grades eines

Doktors der Wirtschaftswissenschaften

(Dr. rer. pol.)

Vorgelegt von

Philipp Maximilian Alexander Hübler
(Diplom-Volkswirt Univ.)

Abgabedatum: 14.05.2018

Erstgutachter: Prof. Dr. Robert Nuscheler

Zweitgutachter: Prof. Dr. Kerstin Roeder

Vorsitzender der mündlichen Prüfung: Prof. Dr. Jens O. Brunner

Tag der mündlichen Prüfung: 06.09.2018

Three Essays on Time Preference in Health Economics

Philipp Maximilian Alexander Hübler

Acknowledgements

I would like to thank everybody who helped me in writing this dissertation. First of all, I would like to thank my supervisor Robert Nuscheler for his encouragement and support. Chapter 2 of the thesis is based on joint work with Robert. In addition, he gave me the opportunity to present and discuss my research at several national and international workshops and conferences. Special thanks to Andreas Kucher for his commitment, dedication to continuous improvement and all the lively discussions we had. Chapter 3 of the thesis is based on joint work with Andreas. I also thank my second supervisor, Kerstin Roeder, for helpful comments and suggestions. My dissertation further benefited from discussions with friends and colleagues. In particular, I thank Martina Grunow, Simon Binder, Christine Leopold and Jakub Rybicki. The feedback provided by conference participants and discussants is also gratefully acknowledged. Particular thanks go to Marjon van der Pol. Furthermore, I appreciate the financial support for several conference participations generously provided by Peter Welzel and the Research Center Global Business Management at the University of Augsburg.

Contents

List of Tables

List of Figures

Chapter 1

Introduction

1.1 Outline

This thesis investigates the influence of economic preferences, in particular time preferences, on health behavior and health-related decision-making. In addition, it explores the sources of individual variation in time discounting. The thesis is composed of three stand-alone, but related chapters, in which multiple empirical strategies are applied. Each chapter focuses on a particular subject and contributes evidence to our knowledge on economic preferences in the field of health economics.

Chapter 2 examines the relationship between maternal (time) preferences and the demand for preventive health care services that favor child health. Specifically, the uptake of vaccinations and developmental screenings ('U Screenings') for children is analyzed. In Chapter 3, the link between time discounting and smoking behavior is addressed. However, the intergenerational context is retained which has been barely considered in the existing literature. Hence, we test whether parental time discounting is associated with child's smoking behavior. Finally, Chapter 4 examines whether preferences are rather exogenous or endogenous. In particular, the genetic variation in time preference is explored. The analysis sheds light on the discussion on whether people are born with innate preferences or whether economic preferences are mainly shaped by environmental factors.

In order to put the papers of the thesis into context, this short introduction is provided. In Section 1.2, examples of intertemporal choice are presented to introduce the topic. The most commonly used theoretical frameworks of time discounting in (behavioral) economics are briefly discussed in Section 1.3. Although we recognize that time discounting may be relevant for a variety of outcomes, we stick with the field of health economics (Section 1.4). In the

following Sections 1.5 to 1.7, each of the above mentioned chapters is addressed separately. The contribution of the thesis to the economics literature is summarized at the end of each of these sections.

1.2 Intertemporal choice

Intertemporal decision-making describes trade-offs between costs and (potential) benefits that occur at different points in time. People are frequently faced with various intertemporal choices. Examples include trade-offs between studying for an upcoming exam and watching TV or forgoing consumption today and saving money for retirement. Both decisions include immediate costs (learning efforts, reduction in consumption), but future benefits could be substantial (passing the exam with a good grade, sufficient financial resources for a comfortable retirement). Intertemporal choices may also have an impact on personal health. For instance, a person may decide between current satisfaction from tobacco consumption and good long-run health due to quitting or abstaining from smoking. Moreover, getting a vaccine can also be interpreted as an intertemporal decision. An individual faces the trade-off between costs of getting a flu shot in the present and health benefits from being vaccinated. All of these examples have in common that a decision has to be made between a sooner, smaller payoff and a larger, but later payoff. How people *solve* such intertemporal dilemmas highly depends on the relative value they assign to the outcomes that are realized at different points in time. In other words, it depends on their personal time preference (rate).

Another example of intertemporal choice is the so-called "Marshmallow Test". It is probably one of the most famous experiments on people's ability to delay gratification (Mischel et al., 1972, 1989). In the 1960s, the psychologist Walter Mischel and his colleagues tested the patience of preschoolers (4 years of age) at the Stanford University's childcare facility. The setting of the experiment was simple: a desirable object was offered to each child, e.g., a marshmallow. The instructor told the child that they could eat the marshmallow whenever they wanted, e.g., immediately. However, if they were able to wait to eat the marshmallow for a given amount of time (typically 15 minutes) until the experimenter came back, they would get another marshmallow as a reward. More technically, the children had to make a trade-off between a sooner, smaller reward (one marshmallow immediately) and a larger, later reward (two marshmallows in 15 minutes). Unsurprisingly, the achievements of the participants were mixed. Whereas many children could not wait to eat the marshmallow immediately or shortly after the experimenter

had left the child alone, others were able to fully resist the instant gratification.

Over the next decades, a number of follow-up studies were conducted that aimed to analyze the children's progress in life.[1] Interestingly, children who ended up with two marshmallows, and hence proved able to delay gratification, were more successful in life than their peers. According to these longitudinal findings, more patient individuals achieved higher Scholastic Aptitude Test (SAT) scores and higher levels of social competence in adolescence. Moreover, they were rated as more rational and planful and were able to better cope with stress and frustration (Mischel et al., 1988; Shoda et al., 1990). Significant associations were also found with regard to health outcomes. The longer the child delayed gratification, the lower the risk of becoming obese and using illicit drugs in adulthood (Schlam et al., 2013; Ayduk et al., 2000). Although these findings cannot necessarily be interpreted as causal relationships, the predictive power of people's ability to delay gratification is striking. This canonical experiment shows that self-control and future orientation are likely to play a crucial role for success in life.

Although the concept of delay of gratification is strongly related to the concept of time discounting/time preference, they are not identical. The main difference is that delay of gratification tasks measure the ability to sustain a choice for a later reward while a smaller instant reward is continuously available. By contrast, *standard* time discounting refers to how people actually make intertemporal choices (Reynolds and Schiffbauer, 2005). In the following, the emphasis of the thesis is on time discounting and time preference in the field of health economics. The influence of self-control abilities, however, will also be discussed whenever the context is appropriate. Before we present some empirical evidence regarding the relationship between time preference and different outcomes, Section 1.3 briefly discusses the theoretical concept of time preference in economics.

1.3 Economic theory of time preference

1.3.1 The Discounted Utility Model (DU Model)

The first generalized model of time discounting was proposed by Samuelson (1937). Individuals maximize their present utility function which is the weighted sum of the current utility in $t = 0$ and all future utility flows. This intertemporal utility function is defined as

$$U_0(c_0, ..., c_T) = \sum_{t=0}^{T} \delta^t u(c_t) = u(c_0) + \delta u(c_1) + \delta^2 u(c_2) + ... + \delta^T u(c_T)\,, \tag{1.1}$$

[1] See Mischel et al. (2011) for a comprehensive overview of systematic follow-up studies.

where $u(c_t)$ is the instantaneous utility function for each time period t, and δ^t represents the exponential discount function. Humans have the tendency to devalue the future and therefore discount future outcomes (Frederick et al., 2002). The personal discount factor δ $(0 < \delta < 1)$ defines the relative weight the individual attaches to future utility streams. Alternatively, the discount function can be written as $\left(\frac{1}{1+r}\right)^t$, with r being defined as the discount rate. Thus, the level of individual time preference can either be captured using the discount factor δ or the discount rate r. According to Frederick et al. (2002), the time preference parameters in this model comprise all the factors that influence time discounting. Hence, time preference is simply the preference for immediate utility over delayed utility. The lower the discount factor (the higher the discount rate), the greater the preference for immediate utility outcomes over delayed utility outcomes. In general, heterogeneity in time discounting/time preference exists (Barsky et al., 1997). Individuals with a relatively high discount rate focus on present consumption rather than on future outcomes. On the contrary, people who have low discount rates can be expected to assign more value to the future consequences of their current behavior. Hence, low time preference may be a key aspect of future orientation.

One of the main assumptions of the discounted utility model (DU model) is that the discount factor is constant over time.[2] That is the reason why the form of the discount function is exponential. The implication of this assumption is that the discounting behavior is consistent over time. In other words, preference reversals are not produced because the preferences are always the same, independent of the time horizon. For instance, if a person is willing to forgo immediate consumption in t in order to get a larger reward in $t + 1$, then this person will make the exact same choice again at any future point in time. However, this dynamic consistency was challenged by researchers who argued that this model was not capable of accurately describing human behavior. In fact, empirical evidence suggests that discount rates may not (always) be constant over time (e.g., Thaler, 1981). To address this "anomaly" of the DU model, Section 1.3.2 provides an alternative specification of the discount function.[3]

1.3.2 Hyperbolic discounting

Over the past decades, researchers have shown that humans are often prone to discount (approximately) hyperbolically rather than exponentially (see Ainslie and Haslam, 1992). Discounting in a hyperbolic fashion is primarily characterized by a relatively high discount rate over short

[2]See Frederick et al. (2002) for a summary of the features and assumptions of the DU model.
[3]For a detailed review of other "DU anomalies", please see Frederick et al. (2002).

time horizons and a low discount rate over long time horizons. Thus, the implicit discount rate is not perfectly constant but tends to decline over time (see, e.g., Thaler, 1981). This may lead to a conflict between current preferences and the preferences the individual holds in the future. Laibson (1997) set up a widely used model framework that captures the qualitative properties of hyperbolic discounting.[4] The intertemporal utility function of the DU model is slightly modified and can be written as

$$U_0(c_0, ..., c_T) = u(c_0) + \beta \sum_{t=1}^{T} \delta^t u(c_t) = u(c_0) + \beta\delta u(c_1) + \beta\delta^2 u(c_2) + ... + \beta\delta^T u(c_T)\,, \qquad (1.2)$$

where $\delta \in (0, 1)$ is the discount factor and $\beta \in (0, 1]$ indicates the individual's bias towards the present.[5] Assuming a present-biased individual ($0 < \beta < 1$), the discount rate declines sharply between the current and the next period. Thereafter, a constant discount rate is applied (Frederick et al., 2002). Hence, the β-δ framework implies time-inconsistent preferences for the near future and exponential/time-consistent preferences for the distant future (quasi-hyperbolic discounting). As a consequence, this can lead to preference reversals (e.g., Kirby and Herrnstein, 1995). People make far-sighted plans for the (distant) future which are supposed to be beneficial for them in the long run. However, they often fail to stick to their plans and go for short-run gratification when the future has become the present.

A simple example illustrates the dynamic inconsistency in intertemporal decision-making when (quasi-) hyperbolic discounting is present. Today, an individual faces the decision between exercising and watching a movie in the next period t+1 (e.g., tomorrow). Exercising produces immediate costs such as physical effort, commuting time to sport facility, etc. However, delayed rewards in terms of good physical health are gained in t+2. Alternatively, watching a movie generates an instant but small reward (joy of watching a movie, no physical or mental effort,...) while future benefits (for health) are usually negligible.

Table 1.1 summarizes the payoffs (utilities) under each option. Based on the quasi-hyperbolic discounting framework, a change in preferences, and hence a change in behavior, may be produced. When $\beta = \frac{1}{2}$ and $\delta = \frac{3}{4}$ (per day), the individual decides to exercise in the next period (t+1). From today's perspective, this is plausible since $U_{exercise} > U_{movie}$. More specifically, $U_{exercise} = u_0 - 2\beta\delta + 6\beta\delta^2 = u_0 + \frac{15}{16}$ is bigger than $U_{movie} = u_0 + 1\beta\delta + 0\beta\delta^2 = u_0 + \frac{6}{16}$. However, as time goes by, preferences change. When the near future becomes the present, a

[4] See Frederick et al. (2002) for an overview of generalized hyperbolic discount functions that have been proposed in the past.

[5] If $\beta = 1$, Equation 1.2 equals Equation 1.1. Hence, exponential discounting is nested within the framework of quasi-hyperbolic discounting.

Table 1.1: Quasi-hyperbolic discounting - an example

	t+1	t+2
Exercise	−2	6
Watching TV	1	0

preference reversal occurs. Now, the individual prefers watching a movie over doing a proper workout ($U_{exercise} = -2+6\beta\delta = \frac{1}{4} < U_{movie} = 1+0\beta\delta = 1$). Hence, hyperbolic discounting may explain why people procrastinate and change plans. In case of exponential discounters ($\beta = 1$), preferences are consistent over time. If such an individual plans to do sports tomorrow, it will stick to the initial plan and will be physically active when the moment of truth has arrived.[6] Further examples of dynamic inconsistency and procrastination are presented in Akerlof (1991) and O'Donoghue and Rabin (1999).[7]

Such behavior patterns may be familiar to most people. However, they are not compatible with the standard DU model from above. Apparently, preference changes that occur over time can reasonably be explained by (quasi-) hyperbolic discount functions. For instance, Read and van Leeuwen (1998) conducted an experiment to provide evidence on preference reversals in the context of health behavior. They showed that the majority of people who preferred healthy foods in advance changed their mind and switched to the unhealthy alternative at the time of actual consumption. Hence, these present-biased preferences may explain why people often fail to stick to their healthy eating plans or why smokers who are willing to quit smoking frequently fail to do so. Although the focus of the thesis is not on hyperbolic discounting and its (possible) implications, this theoretical background on time discounting will facilitate the understanding of previous findings from empirical studies linking time discounting to different (health) outcomes.

1.4 Time preference and outcomes

A number of studies have examined the relationship between time discounting and basic socioeconomic outcomes such as educational attainment, income and financial decisions (see, e.g., Golsteyn et al., 2014). Low discount rates are positively associated with longer investments in education (Harrison et al., 2002) and even higher cognitive abilities (Dohmen et al., 2010).

[6] In t=0: $U_{exercise} = u_0 + \frac{15}{8} > U_{movie} = u_0 + \frac{6}{8}$. In the next period: $U_{exercise} = 2{,}5 > U_{movie} = 1$.

[7] Please see O'Donoghue and Rabin (1999, 2001) for theoretical frameworks that explicitly distinguish between (partially) naive and sophisticated individuals.

Adams and Nettle (2009) identify a negative correlation between discount rate and financial planning horizon. In addition, Meier and Sprenger (2010) find that present-biased individuals are significantly more likely to borrow on their credit cards. For example, Golsteyn et al. (2014) investigated whether children's time preferences at age 13 were associated with social and economic outcomes in adulthood. The results show that time preference is predictive of many lifetime outcomes. A high discount rate is linked to weaker performance in school, lower earnings and disposable income and a higher probability of unemployment and welfare use. Sutter et al. (2013) analyzed the relationship between experimental measures of risk and time preferences and several behavior patterns among a sample of children and adolescents. Individuals with a high time preference rate (impatience) are less likely to save money and are more likely to live an unhealthy lifestyle. For instance, more impatient children and adolescents are more likely to have a higher body mass index (BMI) and to spend money on alcohol and tobacco. Interestingly, risk aversion is found to be only a minor predictor of these outcomes. In general, we can state that high time preference rates, and hence low levels of patience and future orientation, are robustly associated with unfavorable self-investments which may foster adverse social and economic outcomes.

In the scope of this thesis, however, we put special emphasis on the context of time preference and health. In recent years, the literature on time discounting and health behavior has grown substantially. For instance, several studies have shown that high discount rates are associated with smoking participation (see, e.g., Kang and Ikeda, 2014). In Section 1.6.2.3, we will present more evidence on time discounting and smoking.[8] Chapters 2 and 3 will specifically address the role of (time) preferences in decision-making regarding health prevention and smoking behavior, respectively (see Sections 1.5 and 1.6). As already mentioned above, Chapter 4 discusses a different research topic. In this chapter, we examine the formation of time preference in more detail (see also Section 1.7).

[8]See Story et al. (2014) for an extensive review of the association between time discounting and other health outcomes such as BMI/obesity, (excessive) alcohol consumption and illicit drug use.

1.5 Uptake of health prevention for children and adolescents - do parental (time) preferences matter?

1.5.1 Childhood health and future outcomes

Early-life health is supposed to be a major determinant of various outcomes in adulthood. Numerous studies have found a negative relationship between poor child health and health and/or economic outcomes later in life (see, e.g., Currie, 2011; Currie and Almond, 2011).[9] In what follows, we present a selection of relevant papers and their main findings. Blackwell et al. (2001) used data from the third wave of the Health and Retirement Study (HRS) to examine whether childhood conditions and health conditions at age 55-65 are correlated. They find that poor childhood health (e.g., autoimmune conditions and infectious disease) is associated with a variety of health problems such as cancer, lung disease and cardiovascular conditions in later life.

In their seminal paper, Case et al. (2005) studied the effects of childhood health on adult health, employment and socioeconomic status. They used data from the 1958 National Child Development Study (NCDS), a british cohort study which tracks individuals from childhood into adulthood. The number of chronic health conditions at ages 7 and 16 (e.g., general physical and mental impairments) is negatively associated with educational attainment, health status and social status as adults. More specifically, individuals who suffered from chronic conditions during childhood are not only in worse health in middle age, but they have passed fewer O-level exams by age 16, have lower probabilities of employment and a lower socioeconomic status at ages 33 and 42.

Using data from the Panel Study of Income Dynamics (PSID), Smith (2009) examined the link between child health and several financial and socioeconomic measures. He applied sibling fixed effects in order to control for unobserved family level heterogeneity. Excellent or very good health up to the age of 16 is positively associated with household income and wealth, individual earnings and weeks worked in adulthood.[10] Case and Paxson (2010) relied on data from the National Longitudinal Survey of Youth 1979 (NLSY79) and sibling fixed effects regressions. Height-for-age is being used as a marker of early-life health and nutrition. It turns out that children's height is positively associated with cognitive test score results and educational achievement within childhood (e.g., math score, reading comprehension score, and

[9] For specific information about the fetal and infant origins of adult disease, see Barker (1990) and Almond and Currie (2011).

[10] For further discussion on child health and socioeconomic outcomes, see Currie (2009).

scholastic competence score). In addition, an increase in height is shown to be predictive of higher earnings and a better self-reported health status in adulthood.

Similar findings are reported by Currie et al. (2010). Based on administrative data from public health insurance records for Canadian children/siblings, the authors investigate the effects of physical and mental health problems at different child ages on several young adult outcomes. Overall, the results indicate that major health problems at young ages (e.g., asthma, major injury, and ADHD/conduct disorder) are associated with a decrease in school performance and an increase in the probability of being on welfare. Interestingly, children who have a major physical condition early in life (ages 0-3 years) and then recover do not suffer adverse outcomes later. However, mental disorders and physical problems that occur in late adolescence or which persist over many years during childhood have particularly negative effects on schooling and welfare participation.[11]

Although causal inference may be subject to debate, these findings indicate that child circumstances and development are important predictors of economic and health outcomes in adulthood. Preventive health care for children and adolescents is supposed to be a key element to good health at young age and may help individuals to remain healthy throughout their lifetime.

1.5.2 Health prevention services in childhood and adolescence - definition, examples and evidence

Following the standard classification proposed by Caplan (1964), health prevention can be grouped into three main categories: primary, secondary and tertiary. Primary prevention aims to prevent injury or disease before it actually occurs. Hence, the main goal is to reduce the incidence of a specific condition in the population. Examples include vaccinations against infectious diseases or health promotion and education about living a healthy lifestyle. The goal of secondary prevention is the early detection and effective treatment of a disease that has already occurred. Thus, the focus is on the reduction of the prevalence of a specific condition by restoring health. For instance, this form of prevention includes regular and age-specific examinations and screenings such as mammograms and children's developmental screenings. The identification of an existing disease at early stage is essential in order to take appropriate actions to stop its progress. Finally, tertiary prevention focuses on the treatment of people that already suffer from

[11] See Currie and Vogl (2013) for more information on early-life health and long-term outcomes in developing countries.

an ongoing illness. Main goals are the reduction of physical and/or mental impairments associated with the disease, the limitation of medical complications and rehabilitation or (chronic) disease management programs in order to restore health and quality of life.[12]

In Chapter 2 of the thesis, we will focus on the demand for well-child visits and childhood immunizations in Germany. In the German Health Care System, these services are standard and free of charge for all children and adolescents no matter if they are covered by statutory or private health insurance. Although routine check-ups, screening tests and immunizations are generally not mandatory, they are highly recommended by public health authorities and medical experts. In fact, it is up to the parents if they take their offspring to the pediatrician (or general practitioner).[13,14] More information on the main preventive health care services offered to infants, children and adolescents will be presented next.

1.5.2.1 Developmental screenings

A series of free routine examinations is offered from birth to adolescence ('U screenings') (Federal Joint Committee (G-BA), 2017, 2016). In the first six years of life, nine screenings are offered to the children (U1-U9). A well-adolescent visit is also available for individuals aged 12-14 years (J1).[15] The examinations U1 and U2 are performed immediatly after birth and usually within the first week after birth, respectively. They include checks for reflexes, breathing, hypotonia (low muscle tone) and heart activity. Normally, they are directly carried out in the hospital. After leaving the hospital, the parents are primarily in charge of demanding the upcoming preventive health care services for their offspring. Of course, screening tests vary depending on the child's age. In general, however, child examinations focus on appropriate physical and mental development, motor skills and child's speech and language development. The juvenile health screening J1 is typically done between childhood and adulthood. Adolescents are screened for age-specific physical, mental and social problems. Moreover, symptoms of puberty, risky health behaviors and vaccination status are examined. Since children are continuously faced with physical, emotional and psychological changes as they grow, regular monitoring of child's

[12] In practice, however, a clear distinction between these prevention categories, especially between primary and secondary prevention, may not always be feasible.

[13] A comprehensive overview of the child health care system in Germany is provided by Ehrich et al. (2016).

[14] In recent years, there has been debate about making preventive health care visits obligatory. For instance, the majority of federal states has introduced reporting systems that inform parents and/or health authorities about screenings that have been missed. Lately, screening participation has become mandatory in some federal states (e.g., Bavaria). However, regarding our empirical analysis, participation was voluntary during our observation period (2003-2006) (Wissenschaftliche Dienste des Deutschen Bundestages, 2014).

[15] Additional check-ups (U10, U11 and J2) have been introduced recently. However, they are optional and costs are not covered by all health insurance companies. In Chapter 2 below, these examinations will not be taken into account. The introduction took place after our observation period.

development is essential. Frequent examinations of child development and health are important in order to detect developmental delays and disabilities as soon as possible. The early detection of (potential) disease allows the early implementation of appropriate intervention measures. Hence, the identification of health problems helps to keep the child healthy and may also enable him/her to become a healthy adult.

Over time, however, the use of these medical services decreases. According to representative survey data collected from 2003 to 2006, roughly 81% of children take part in all examinations that are provided until age 6 (U3-U9). In the first two years of life, the rate is always above 90% (95.3% at U3 and 92.4% at U7). However, the uptake rate decreases as children get older. Four years after birth, the rate is 89% at U8. Screening examination U9, which should be done in the sixth year of life, has a rate of only about 86% (Kamtsiuris et al., 2007). Regarding the well-adolescent visit J1, the uptake rate is only 38% among adolescents (Robert Koch Institute (RKI), 2008). Based on findings from a follow-up study (2009-2012), the overall use of health screenings has slightly increased. About 82% of children take part in all child examinations. Especially those screenings at later stage (U7-U9) have received considerably more interest and rates are all above 90%. Although the gap has become smaller, there are still differences in uptake across different groups. For instance, the participation rates across all U screenings are significantly lower for children from families with low socioeconomic status than for those with a higher socioeconomic background (Rattay et al., 2014). More evidence on factors that are associated with low uptake rates of preventive services for child health will be presented in Section 1.5.3 below.

1.5.2.2 Immunizations

The demand for routine childhood vaccinations represents another important pillar of health prevention. Protective vaccinations can be considered as one of the most important and effective health prevention measures. In the past, they have made substantial contributions to decrease the probability of contracting infectious disease and to reduce the severity of illness. In addition, they enabled the eradication of acute contagious diseases such as smallpox (see, e.g., Plotkin, 2014; Greenwood, 2014). Of course, the main purpose of a vaccination is to protect a person from contracting a disease. Although protection may not be perfect, a child who is sufficiently vaccinated is normally well protected against typical childhood diseases such as measles, mumps and rubella. For instance, measles infection can cause serious health complications among children including blindness, brain swelling and pneumonia. In the worst-case scenario, such

an infection can result in death (World Health Organization (WHO), 2018). Needless to say that unvaccinated (or undervaccinated) children are exposed to an unnecessarily high risk of contracting these preventable diseases.

However, vaccines do have positive externalities (see Kenkel, 2000, Section 4.1). That is, even unvaccinated individuals benefit from the vaccination decision of other individuals. Since vaccinated persons (e.g., children) will not contract the disease, they will not pass it on to others (herd immunity effect). Obviously, the incentive to get a vaccine decreases with the share of vaccinated people. In general, this leads to low immunization rates. In this case, government is frequently asked to take action. In order to increase demand for immunization, goods like standard vaccines are typically subsidized. But as the demand for vaccines increases, the risk of infection decreases because of lower disease prevalence. However, if the probability of contracting the disease gets lower, it becomes less attractive to get vaccinated and vaccination rates tend to decline again (prevalence effect). The possibility of free riding may induce some individuals to forgo vaccinations and the disease may return. Hence, externalities from vaccines are a common explanation for why it is so difficult to eradicate diseases. Public policy interventions such as vaccine subsidization and mandatory vaccine programs have been challenged in the past and are still subject of debate. Findings from previous studies have shown that such policies may not be able to fully solve the problem of low immunization rates. Compulsory vaccination may even reduce social welfare (Brito et al., 1991). Geoffard and Philipson (1997) provided a theoretical framework that demonstrates that the prevalence elasticity of demand precludes the ability of price subsidies or mandatory programs to increase the overall demand for vaccinations, and hence to achieve disease eradication.

In Germany, (child) immunization is voluntary. Recommendations for vaccinations are made by the German Standing Committee on Vaccinations (STIKO) which is based at the Robert Koch Institute. The latest version of the vaccination schedule for infants, children and adolescents includes standard vaccinations to protect against tetanus, diphtheria, pertussis, polio, hepatitis B, Hib, pneumococcal disease, rotavirus, meningococcal disease, measles, mumps, rubella, varicella and human papillomavirus. The latter is a special vaccine for girls aged 9 to 14 years. Some vaccines may be offered and administered at U screenings (Standing Committee on Vaccinations (STIKO), 2017). These routine vaccinations are fully covered by statutory health insurance. In general, private health insurance does not have to reimburse the costs of vaccination. However, private insurance companies normally do cover the recommended vaccines.

It is generally agreed that routine childhood immunization programs are highly cost-effective

(e.g., Zhou et al., 2014). Despite the remarkable and undeniable health benefits from immunization, vaccination rates are often too low. According to the World Health Organization (WHO), immunization coverage should exceed a minimum rate of 95% across all types of childhood vaccines. Taking Germany as an example, there is still a long way to go until all WHO goals are met. In general, primary immunization rates for diphtheria, tetanus and polio are reasonably high and close to 95%. However, compliance with booster vaccinations is substantially worse. With regard to tetanus and diphtheria, coverage rates regarding the first booster vaccination are about 83%. Compliance with the second booster vaccination drops down to roughly 30% (Poethko-Müller et al., 2007). Deficits in overall immunization coverage are also identified for other basic vaccines such as rotavirus (68.3%), pneumococcal disease (75.9%) and measles (86.5%, 2. dose) (Rieck et al., 2018). For example, more than 900 measles cases were reported in Germany in 2017; about one third of the reported cases occurred in children under five years of age (Robert Koch Institute (RKI), 2018).

Although it is generally agreed that (early) prevention is better than disease treatment, evidence suggests that compliance with preventive services such as child screenings and vaccination uptake could be better. This raises the question of why the uptake of prevention for children is often incomplete. As already mentioned above, Section 1.5.3 will give a brief overview of factors that have been found to be directly associated with low levels of childhood immunization and participation rates in recommended health examinations.

1.5.3 Common predictors of childhood vaccination and screening uptake

Numerous studies have investigated factors that are related to low compliance with the recommendations for childhood prevention. In the following, we briefly summarize the main factors that are likely to affect vaccine uptake in children. Findings from empirical studies suggest that low immunization rates are generally associated with different socioeconomic and sociodemographic variables such as low socioeconomic status as a whole, low parental education, low parental income, non-white race/ethnicity, young age of the mother/parents, large family size/number of siblings, older age of the child and marital status of the mother (unmarried or divorced).[16] Structural barriers in health care were also found to be associated with suboptimal compliance to immunization. They include the lack of health care structures, inadequate support from physicians and restricted access to preventive child health services. However, these

[16]The correlates of low vaccination rates and socioeconomic status (SES) often show an inverted U-shaped pattern. That is, rates are lower among low and high SES families (e.g., Poethko-Müller et al., 2007).

factors are likely to play a negligible role in vaccination uptake in Germany. Parental attitudes and beliefs may be additional risk factors for undervaccination. Psychological factors such as negative attitudes towards immunization, perceiving vaccines to cause adverse health effects and low perceived susceptibility to illness are negatively associated with vaccination uptake (see Falagas and Zarkadoulia, 2008; Danis et al., 2010; Smith et al., 2017).

Similar findings can be found with regard to the uptake of developmental screening examinations. As was already motivated in Section 1.5.2.1, children from families with low socioeconomic status are significantly less likely to have participated in U screenings than children from middle and high SES families (Kamtsiuris et al., 2007; Rattay et al., 2014). More specifically, Reinhold and Jürges (2012) show that participation rates in selective screening exams (U3, U6, and U9) increase with parental income. Hence, low-income parents use screenings significantly less often than parents with higher incomes. Moreover, migration background as well as childbirth at a young ($<$ 26 years old) and advanced maternal age ($>$ 35 years old) are also negatively associated with the full use of well-child visits (Kamtsiuris et al., 2007; Rattay et al., 2014).

Little is known, however, about the role of parents' economic preferences (e.g., time preferences) in this context. We put special emphasis on the investigation of parental preferences as determinants of vaccination and screening uptake in children. Before we further discuss the contribution of the thesis in Section 1.5.5, we provide insight into the relationship between time discounting and preventive health behaviors at the individual level.

1.5.4 Time preference and health prevention

1.5.4.1 Health prevention as an intertemporal investment decision

In general, preventive health behavior can reasonably be considered as an investment decision that includes an intertemporal trade-off. The uptake of vaccinations or health screenings is a decision problem that typically involves immediate efforts and/or monetary costs, whereas (potential) benefits are delayed. The individual is required to engage in activities he/she may not enjoy today. These may include scheduling an appointment with the doctor, efforts to get to the doctor's office and discomfort with getting vaccinated or tested. However, the expected future benefits from primary and secondary prevention may by far exceed the current costs and sacrifices. That is, the prevention of disease onset and the early detection of disease which allows early intervention before problems actually occur or get worse. It should be further noted that risk aversion may also play a role in health investments. It is shown that risk averse individuals are less likely to engage in risky health behaviors such as smoking and drinking

(Barsky et al., 1997; Anderson and Mellor, 2008). For instance, vaccination decisions may also be influenced by risk preferences. Vaccinations come at an additional risk of side effects (e.g., nausea or dizziness) and vaccine failure. However, not being vaccinated increases the risk of infection. Considering the possibility of becoming ill, a higher level of personal risk aversion should increase the demand for preventive health care (Picone et al., 1998). Theoretical models suggest that time preference may be a major determinant of investments in preventive health behaviors and therefore in health (Grossman, 1972; Ehrlich and Chuma, 1990). On average, people who discount the future less heavily (low time preference rate) are expected to demand more preventive health care services than people with high rates of time preference. Although the predictions made by economic theory are rather obvious, results from previous empirical studies are ambiguous. In the following, a short summary of these findings is presented.

1.5.4.2 Empirical evidence on time preference and preventive health care use

Evidence on the relationship between time discounting and preventive health behaviors is both scarce and mixed. Chapman and Coups (1999) found a weak relationship between monetary time preference and immunization. More future-oriented individuals were more likely to accept a free influenza vaccination. However, other measurements of time discounting (e.g., elicitation of individual discount rates in the flu context) failed to predict vaccination uptake. In another study, Chapman et al. (2001) analyzed the association between time discounting and several preventive health behaviors. Three health outcomes were under investigation: influenza vaccination uptake, adherence to medication for high blood pressure, and adherence to cholesterol-lowering medication. A lower time preference rate was positively associated with flu shot acceptance. However, this is only true for the time preference measure in the monetary domain. With regard to medication compliance, no meaningful relationship for any of the time preference measures was found.

Using data from the Health and Retirement Study (HRS), Picone et al. (2004) found significant effects of time preference on a number of medical tests such as breast self-examinations, mammograms and Pap smears. Women with a (relatively) short time horizon were less likely to perform self-exams or demand a mammography screening. However, a long time horizon was positively related to the demand for cervical smear tests. Risk aversion was only marginally associated with these health variables. Bradford (2010), using a different wave from the HRS, also found significant associations between individual discount rates and various health maintenance habits. People with a high discount rate were less likely to demand basic preventive

screenings (e.g., mammograms, prostate exams, cholesterol tests, dental visits) and were more likely to indulge in a sedentary lifestyle in the past two years.

The paper from Nuscheler and Roeder (2016) used a sample of individuals representative of the German population. Its focus is on time discounting and seasonal influenza uptake. A major contribution of this study is the analysis of behavioral anomalies (e.g., the role of present bias in prevention decisions). Interestingly, they found that the discount factor is *negatively* associated, at the 10% level, with the demand for vaccination. Furthermore, no significant effect of present bias was identified. Hence, the demand for flu shots does not differ across exponential discounters and present-biased individuals. However, individuals that put extra weight on future outcomes (future bias) have a higher probability of getting a flu shot than exponential discounters. Risk aversion was positively associated with the demand for vaccination. The authors note that the main results are primarily driven by men.

Using cross-sectional data from the Barriers to Care for People with Chronic Health Conditions (BCPCHC) survey, van der Pol et al. (2017) investigated the role of time and risk preferences in the adherence to physician advice among individuals with chronic disease. The financial planning horizon is used as a proxy for time preference. Personal risk preference is measured through self-assessed willingness to take risk. Individuals with a longer planning horizon, and hence with higher future orientation, are less likely to not adhere to advice on physical activity changes. Non-adherence to advice on dietary changes is negatively related to low time preference for men but not for women. Willingness to take risk had no impact on adherence behavior.

1.5.5 Contribution of the thesis

We have shown that individual time preference may be an important factor associated with several preventive health behaviors. In Chapter 2 of the thesis, we examine the relationship between parental decision-making and childhood vaccination and screening. This chapter is based on joint work with Robert Nuscheler. As already discussed in Section 1.5.3, different socioeconomic characteristics and attitudes of the parents have been linked to the demand for preventive child care. However, the influence of parental preferences is still not well understood. In this intergenerational setting, we focus on the role of economic preferences. They include parental time preferences, risk aversion and altruistic attitudes.

First, we present a theoretical framework for parental prevention efforts (Section 2.2). It allows us to analyze the impact of the preference parameters mentioned above on the uptake of

primary and secondary prevention services for children and adolescents. We derive important implications from our theoretical model. The results suggest that disentangling the influence of different preference parameters on prevention decisions is a major challenge. Moreover, the directional effect of parental preferences on prevention decisions is ambiguous. Finally, as we consider both primary and secondary prevention, interdependence of these types of preventive activities may arise. Thus, an econometric model may be required that takes into account the issue of simultaneity. Overall, the findings from the theoretical model provide a useful guideline for the econometric modeling of prevention decisions.

We then empirically test the relationship between economic preferences and vaccine uptake (primary prevention) and the demand for well-child visits (secondary prevention). Based on our theoretical framework, we opt for the joint estimation of the demand for the two modes of prevention. We rely on representative cross-section data of children and adolescents from Germany. As our main preference proxy, we use information on whether the mother smoked during pregnancy. We argue that such a behavior is associated with a high time preference rate, low risk aversion and, obviously, a low level of altruism. The results suggest that smoking during pregnancy is negatively associated with preventive investments in child health. For example, we find a significant reduction in the probability of having an up-to-date screening status of 3.5 percentage points (pp) when the mother smoked during pregnancy. Whereas maternal preferences are particularly relevant for screening decisions, they also show a negative impact on vaccination uptake for boys but not for girls (-3.6 pp for boys). In addition, stratifying by social status, negative impacts on screening status are primarily identified for children from low and medium SES families.

Chapter 2 of the thesis contributes to the health economics literature in many ways. First, our theoretical framework provides guidance for the appropriate econometric modeling of prevention decisions. Second, we present new empirical evidence on the determinants of parental investments in child health. Children from socially disadvantaged families (low parental SES and/or health) tend to be in worse health (e.g., Case et al., 2002). Maternal preferences might contribute to a better understanding of why this gradient is present and persistent over time. Third, our analysis further contributes to findings from previous studies which analyzed the link between time and risk preferences and the use of preventive medical care (see Section 1.5.4.2). However, we specifically address the intergenerational context. Since maternal preferences are significantly associated with the uptake of primary and secondary child prevention, our findings improve the understanding of why parents seek or do not seek preventive health services for their

offspring. Inadequate parental investments in child health that are driven by low levels of future orientation, risk aversion and altruism may have unfavorable consequences for the development of children's and adolescents' health capital, and thus for their human capital formation.

1.6 Tobacco consumption, intergenerational transmission and time discounting

1.6.1 The problem of tobacco smoking

Smoking is still one of the main causes of cancer development and heart and lung disease (e.g., lung cancer, stroke and chronic bronchitis) (Centers for Disease Control and Prevention (CDC), 2018a). Worldwide, around 7 million people die each year because of tobacco use. Almost 900 thousand of those deaths are the result of non-smokers being exposed to second-hand smoke (World Health Organization (WHO), 2017a,b). For example, about one in four Germans aged 15 or older smokes (Federal Statistical Office (Destatis), 2017). Roughly 121,000 people died as a result of smoking in Germany in 2013. This equals 13.5% of the total number of deaths. Annual costs of tobacco consumption are estimated to be approximately 79 billion Euros. This includes direct costs such as medical expenses to treat smoking-related diseases as well as indirect costs (e.g., reduced earning capacity or unemployment) (Pötschke-Langer et al., 2015).

Adverse health consequences and negative externalities of smoking are frequently considered to be the main reasons for policy interventions to curb smoking prevalence. Undoubtedly, smoking has negative impacts on the health of smokers. However, it is also bad for the health of others that do not smoke, and thus places an extra burden on society as a whole. Typically, smokers will only consider their private costs (e.g., monetary costs of buying tobacco products and (perhaps) personal health risks for smoking-related diseases) and benefits (e.g., pleasure from tobacco consumption). However, negative external effects such as bad air quality, involuntary but harmful passive smoking and health care costs to treat smoking-related health problems are imposed on third parties without adequate compensation.[17]

Taxation of tobacco products is a standard approach to make smokers pay the social costs of smoking ("Pigouvian taxes"). The intention of such a tobacco control strategy is to reduce tobacco (over-)consumption to a socially optimal and therefore more desirable level. In addition, excise taxes on tobacco could also correct smoking's "internalities". Individuals may

[17] As already stated by Gruber and Köszegi (2001), smoking during pregnancy and poor subsequent infant health are probably the most drastic example of negative externality in the context of tobacco consumption.

overlook (discount heavily) the adverse health effects of tobacco consumption they face themselves. Hence, if the smoker has to decide between enjoying the pleasures of smoking a cigarette and long-term health, he or she will choose to smoke. However, the smoker may prefer to stop smoking (e.g., quit smoking tomorrow). But when tomorrow becomes today (the future becomes the present), the smoker will face the exact same trade-off. And again, smoking a cigarette will be the preferred option. Thus, smoking continues although the smoker may deeply regret the decisions made. Such self-control problems among smokers may arise due to *unhealthy* time-inconsistent preferences (see Section 1.3.2).[18] Smokers who would like to quit but cannot may appreciate commitment devices such as (considerably) higher taxes to achieve a tobacco-free life (Gruber, 2002; Gruber and Köszegi, 2001, 2004). It should be noted, however, that the demand for addictive goods such as cigarettes is rather price inelastic. Normally, the price elasticity of demand for cigarettes or tobacco ranges from -0.3 to -0.5 (Chaloupka and Warner, 2000). However, it is an indication that taxation could reduce tobacco consumption.[19]

Around the globe, a number of tobacco control policies have been developed in order to increase the costs of smoking. While raising taxes is still suggested to be the most effective and cost-effective way to reduce smoking and encourage smokers to quit, other prevention policies have received more and more attention in the past. In addition to the steady elevation of tobacco prices by the use of consumption taxes, different measures such as health warning labels, anti-tobacco mass media campaigns, smoking bans, and the like have been introduced and improved over the past years (World Health Organization (WHO), 2017b). In fact, there is evidence that these policies can play an important role in reducing tobacco consumption (see, e.g., Nesson, 2017; DeCicca et al., 2008; Carpenter and Cook, 2008; Evans et al., 1999; Anger et al., 2011; Hammond, 2011; Emery et al., 2012; Blecher, 2008; Saffer and Chaloupka, 2000).[20] They may have contributed to an overall decrease in the prevalence of tobacco smoking. Global smoking prevalence has declined from 23.5% in 2007 to 20.7% in 2015. Although prevalence is still higher in developed countries than in most developing countries, average rates of current smoking decrease faster in high-income countries than in middle- or low-income countries. However, steady population growth is the reason why the number of smokers has remained constant over the past decade. According to the WHO, about 1.1 billion people worldwide were smokers in 2015 (World Health Organization (WHO), 2017b).

[18] Empirical evidence on time discounting and smoking behavior is provided in Section 1.6.2.3.

[19] See also Section 2.4 of Cawley and Ruhm (2011) for more information on the price elasticities of health behaviors.

[20] See Chaloupka and Warner (2000) for a review.

Although the rates of current smoking decline, the numbers are still considerably high. Hence, the factors from above (e.g., taxes or smoking bans) may represent some, but not all influences on the decision to smoke. In the following, social and economic determinants of smoking status are discussed which may, at least partly, explain why the "global tobacco epidemic" is not dead yet.

1.6.2 Determinants of smoking status and behavior

1.6.2.1 The SES-health gradient

The understanding of factors that influence smoking habits is of particular interest for health researchers, but may also have implications for policy makers. There is a large body of literature on the relationship between socioeconomic status (SES) and health. In general, people with a higher SES can be considered to be in better health and live longer (see Cutler et al. (2011) for a review). For instance, education is negatively associated with the probability of being a current smoker, the probability of being obese and the probability of being a heavy drinker (Cutler and Lleras-Muney, 2010).

Although the empirical evidence on the SES-health gradient is robust, many findings cannot necessarily be interpreted as causal relationships. For instance, poor health may also reduce productivity and therefore a person's educational attainment and income. Furthermore, third factors such as time preferences may affect both SES and health (Fuchs, 1982). However, causal effects of SES on health have been established in the past (see, e.g., Brunello et al., 2016). There is some evidence that education has a direct causal effect on smoking behavior. More educated individuals are less likely to smoke (see, e.g., de Walque, 2007). In fact, health behaviors such as smoking are identified as mediating factors and account, at least partly, for the causal effect of education on health (Brunello et al., 2016). Higher educated people tend to make better health decisions and therefore live a healthier lifestyle and abstain from risky health behaviors such as smoking. Moreover, they are more likely to manage health inputs more efficiently which improves health production (Grossman, 1972).

The gradient may have its roots already in childhood (Case et al., 2002). That is, lower parental SES, and thus poorer health, is predictive of poorer child health. As argued above (see Section 1.5.1), health disadvantages in childhood are likely to have further impacts on the child's economic status and health in adulthood (see, e.g., Case et al., 2005). In Section 1.5.3, we already discussed the income gradient in healthcare utilization (screening participation) for children. Hence, health problems in children living in low-income families are at higher risk

of remaining undetected. In order to shed more light on this vicious circle of poor SES and health, economists have increased their attention to the intergenerational transmission of health and health behaviors. Parental habits and characteristics may play an important role in the commonly observable persistence of specific health patterns (e.g., smoking behaviors passed down from generation to generation). In section 1.6.2.2, we discuss potential mechanisms of the transmission of health behaviors such as smoking.

1.6.2.2 Intergenerational transmission of smoking behavior

The positive relationship between parental and child smoking behavior is well documented in the literature (e.g., Wickrama et al., 1999; Bantle and Haisken-DeNew, 2002; Shenassa et al., 2003; Göhlmann et al., 2010; Melchior et al., 2010; Loureiro et al., 2010). Overall, these findings suggest that children of smokers are more likely to become smokers themselves. However, various mechanisms exist through which parents may transmit their smoking habits and therefore influence offspring's smoking behavior.[21]

Intergenerational similarities in smoking may be the result of the genetic inheritance of health (Thompson, 2014). Evidence suggests that tobacco consumption and nicotine dependence are partly determined by genetics (see, e.g., Shenassa et al., 2003). In addition to genetic effects, offspring's smoking behavior may also be influenced by parents serving as role models for their children. Based on their beliefs, parents may try to shape their offspring's values, attitudes and preferences through direct socialization efforts. However, rather unintended actions of the parents may further impact the child's decision to start smoking. Exposure to second-hand smoke or easy access to cigarettes at home may serve as examples. Hence, imitation of parental behavior may be an explanation for why children and adolescents decide to start smoking (e.g., Bantle and Haisken-DeNew, 2002; Loureiro et al., 2010; Göhlmann et al., 2010). In addition, there is some evidence that parental health shocks have an effect on offspring smoking behavior. A smoking-related cardiovascular shock to the parent is associated with a reduction in adult offspring's smoking participation and intensity. However, this effect is limited to women (Darden and Gilleskie, 2016).

Reconsidering more fundamental mechanisms, economic preferences such as risk and time preferences may be crucial. These hardly observable factors may be common to both parents and their children. In the intrapersonal context, individual risk attitudes and time preference

[21] In Chapter 3, we focus on parental influences on offspring smoking behavior. For further reading on the effects of peers (e.g., friends or classmates), see Cawley and Ruhm (2011, Section 3.1).

are widely considered as predictors of smoking behavior. Unsurprisingly, a higher level of risk aversion is associated with a lower probability of cigarette smoking (e.g., Anderson and Mellor, 2008). In addition, more future-oriented individuals are more likely to abstain from tobacco use (see Section 1.6.2.3 for more details). There is robust evidence for a positive correlation in risk and time preferences between parents and children (see, e.g., Dohmen et al., 2012; Gauly, 2017). Genetic inheritance and/or parental efforts to form these preferences may explain the observed correlations.[22] However, surprisingly little is known about the role of time preference in the intergenerational context of smoking.

1.6.2.3 Time discounting and smoking

Apparently, tobacco consumption is a major risk factor for smoking-related diseases (see Section 1.6.1). Moreover, it is another striking example of intertemporal choice for health. As already discussed in Section 1.5.4.1, we consider the demand for health screenings and vaccines as a trade-off between costs and benefits that occur at different points in time. In the context of smoking, similar considerations can be made. People weigh immediate and future rewards of tobacco consumption. Typically, they choose between the immediate pleasure from smoking a cigarette (e.g., stress relief and relaxation) and good health in the future from not smoking (e.g., decreased risk of lung cancer). According to economic theory, individuals will (continue to) smoke if the present gains from smoking exceed its future costs (Becker and Murphy, 1988). Individuals with a low discount rate (high patience/future orientation) are likely to abstain from smoking which will otherwise damage their health in the future. Hence, they place more value on the long-run gains from abstaining from tobacco use than on the short-run pleasures from smoking. However, those who have high discount rates (low patience/future orientation) tend to select the short-run rewards derived from tobacco consumption. The future benefits of living a tobacco-free life (or the delayed adverse health consequences of smoking) are discounted heavily. Hence, impatient individuals place more value on immediate pleasures and therefore sacrifice larger health benefits in the future. Moreover, individuals with present-biased preferences (hyperbolic discounters) are expected to smoke more than exponential discounters. Their discount rates are higher for immediate future choices than for choices in the distant future. As a consequence, preference reversals may occur because immediate gratification is consistently preferred over the larger future health reward (see also Section 1.6.1).

[22] As we will see in Section 1.7, Chapter 4 is dedicated to the empirical analysis of genetic and environmental influences on the formation and transmission of time preferences.

In the past, the relationship between time discounting and smoking behavior has received particular interest among behavioral and health economists. There is evidence that smokers discount future outcomes such as delayed monetary or health gains more heavily than non-smokers (e.g., Bickel et al., 1999; Odum et al., 2002; Scharff and Viscusi, 2011). Hence, the individual discount rate is typically positively associated with smoking behavior. In addition to the robust relationship between time preference rate and smoking, some studies are also able to relate tobacco consumption to hyperbolic discounting and time inconsistencies (see, e.g., Harrison et al., 2010; Kang and Ikeda, 2014). Based on a quasi-hyperbolic discount function, Ida (2014) shows that both time preference rate and present bias are significantly associated with the probability of smoking. The higher the time preference rate and the present bias, the higher the smoking probability. Furthermore, Kang and Ikeda (2014) find that discount rates and hyperbolic discounting are also correlated with smoking intensity. Among smokers, both factors are positively associated with the number of cigarettes consumed per day. The empirical analysis on discounting and smoking further extends to the topic of smoking cessation. Evidence suggests that future-oriented smokers are more likely to quit tobacco consumption and also more likely to permanently abstain from smoking (e.g., Brown and Adams, 2013; Goto et al., 2009).[23]

Although previous studies have linked time discounting parameters to smoking behavior (at the intrapersonal level) and discussed possible transmission mechanisms of smoking, we do hardly know anything about the role of parental time discounting in the intergenerational transmission of smoking. Brown and van der Pol (2014) have been the first to provide some evidence on this issue. For their analysis, they used survey data from Australia. A pooled probit model was estimated and no correlation between maternal time preference and offspring smoking probability was found. However, indirect effects are present. Sons of mothers who smoke and have a shorter planning horizon are 6% more likely to smoke than sons of mothers who smoke and have a longer planning horizon. The effects on females are similar. Daughters of mothers who smoke and have a shorter planning horizon are 7% more likely to smoke than daughters of mothers who smoke and have a longer planning horizon. It should be noted, however, that these correlations cannot necessarily be interpreted as causal relationships.

1.6.3 Contribution of the thesis

It is obvious that the existing literature on this topic is extremely limited. Chapter 3 of the thesis is based on joint work with Andreas Kucher and contributes to Brown and van der Pol

[23] For a review, see Barlow et al. (2017).

(2014) by further investigating the effect of parental time discounting on child smoking. We extend their rather tentative analysis in several ways. First and foremost, we apply validated measures in order to distinguish between self-control abilities and general time preference. This approach provides a more distinctive view on (slightly) different features of time discounting (long-term discount rate vs. present bias). In addition, we control for potential confounders such as risk aversion and parental altruism. For example, while immediate consequences are certain, the future is inherently risky. Hence, people's risk preferences may also play a role in smoking decisions that may cause serious health consequences in the distant future. Moreover, our analysis is not restricted to the influence of mothers. Characteristics of the father are also taken into account. This allows us to study potential gender differences in parent-child relationships in more detail. We further investigate if health behaviors such as past and current smoking act as mediating factors of parental time discounting. Finally, we explore if the number of cigarettes consumed is related to our time preference measures.

Our estimation results confirm findings from previous studies (see Section 1.6.2.3). Higher levels of personal impatience and impulsivity are associated with a higher likelihood of being a smoker. However, our findings further suggest that there is a direct effect of parental time discounting on child smoking. A one standard deviation increase in maternal and paternal patience is associated with a reduction in child's smoking participation of about 6-7%, respectively. Interestingly, these effects remain robust even after controlling for additional explanatory variables and potential mediating factors (e.g., education and parental smoking behavior). Although the coefficients of parental smoking status (ex-smoker and current smoker) are both highly significant and show the expected positive sign, none of the parental health behaviors (smoking, drinking, nutrition and sports) is identified as mediator between parental time preference and offspring smoking behavior. Furthermore, we do not find significant effects of parental time discounting on offspring cigarette consumption.

We can only speculate about what actually drives the direct effects of parental time preference on child smoking. The analysis of other potential pathways may be a promising direction for future research (e.g., parent-child communication). Due to limitations of our dataset, we are not able to investigate other transmission channels in more detail. To some extent, genetic and/or cultural linkages of time preference between parents and their offspring may further contribute to these findings. A closer examination of the formation and transmission of time preferences is therefore presented in Chapter 4. However, although more research on this issue is required, the possible impact of parental time preference on offspring tobacco use should not be ignored.

1.7 The formation and transmission of time preferences

The final chapter of the thesis covers a distinct topic in the area of time discounting. While Chapters 2 and 3 both link time discounting to specific health behaviors, Chapter 4 takes a different approach. As pointed out above, a large body of literature has examined the relationship between time preference and different health behaviors such as smoking, alcohol use and obesity (see Story et al. (2014) for a systematic review). However, our knowledge about the transmission and formation of time preference is still limited. In this chapter, we seek to open up this 'black box' and shed some light on the basic processes that lie behind preference formation.

Economic preferences such as time preferences may be shaped by genetic inheritance and/or learning and adaptation processes. Our study contributes to the discussion on whether time preferences can be taken as given/exogenous or as endogenously determined. Are people born with innate time preference rates or can they be shaped by environmental influences? In fact, the aim of our analysis is to examine the relative contribution of genes and environment to the total variance in time preference. Providing a better understanding of the origins of individual differences in time discounting is important because we have already seen that preferences are likely to act as underlying factors of various health behaviors. New insights into the composition and transmission of time preference may also be valuable for (health) economists and policy makers in order to evaluate how people's choices could be influenced.

1.7.1 Endogeneity of preferences

1.7.1.1 The nature-nurture debate

The formation of human behavior has been subject of extensive discussion in the past and the debate is still going on. Hundreds of years ago, people were already curious about the main determinants of human mind, traits and behavior patterns. In fact, they were searching for an answer to the fundamental question of whether nature or nurture exhibits a predominant influence on physical, behavioral and cognitive characteristics. Whereas "nature" refers to the formation of traits as a consequence of genetic inheritance, "nurture" sums up all the influences associated with other sources such as environmental factors, socialization efforts and/or learning effects. Philosophers of the 17th and 18th centuries have taken rather extreme views on the topic. The English philosopher John Locke (1632-1704) proposed the idea that a child is born without predispositions. Hence, the mind of the child is a "blank slate" (tabula rasa). Revealing a strong bias towards nurture, Locke argued that human characteristics are mainly influenced

by experiences and environmental effects. In contrast to Locke, the Swiss philosopher Jean-Jacques Rousseau (1712-78) emphasized the role of nature in human development. His theory suggests that children are endowed with goodness. They mainly develop according to "nature's plan" (natural predispositions) and the effects of nurture or experience are of minor importance (Goldhaber, 2012, p. 15; Harris and Butterworth, 2002, pp. 12-13).[24]

Based on these philosophical considerations, the stage was set for the emergence of the so-called nature versus nurture debate. Inspired by Charles Darwin's book *The Origin of Species* (1859), the English anthropologist Sir Francis Galton (1822-1911) was the first to phrase the nature-nurture problem which is still one of the most controversially discussed topics within psychology. Galton had observed that genius tended to run in families and inferred that intelligence was likely to be inherited (Galton, 1869).[25] Moreover, his thoughts on heredity led the way to the implementation of the twin method. Its roots trace back to the beginning of the 20th century and it is still the most widely used method in behavioral genetics (see, e.g., Rende et al., 1990).

Despite its long tradition in behavioral genetics, the use of quantitative genetic methods is still relatively new in health economics. These methods include twin and adoption designs in order to understand the relative importance of nature versus nurture. Genetic and environmental factors may both contribute to differences in traits across individuals. Quantitative genetics offers an approach to analyze the extent to which variance in a trait is due to genetic and/or environmental variation. The twin design, for example, compares the resemblance of monozygotic (identical) and dizygotic (fraternal) same-sex twins. It is generally agreed that both monozygotic and dizygotic twins share their environment to the same extent (e.g., born at the same time, share the same womb, are raised in the same home, are the same age and are of the same sex). However, the major difference between both twin types is the genetic relatedness among the twins of a twin pair. Fraternal twins share (substantially) less genetic material than genetically identical twins. If genetic influences on a trait are present, the greater genetic similarity of identical twins will make them more similar than fraternal twins with respect to the trait under investigation. By taking advantage of the genetic differences between both twin types, twin studies are able to decompose and estimate the relative contribution of genetic and environmental factors to a specific trait (see, e.g., Plomin and Daniels, 2011).[26]

Lately, however, economists have devoted increasing attention to the examination of genetic

[24] For more information on the diverging theories of Locke and Rousseau, see Gianoutsos (2006).
[25] See, e.g., Sherry (2004, pp. 85-89) for an overview of the history of the nature vs. nurture debate.
[26] The twin methodology will be discussed in more detail in Chapter 4.

mechanisms in health transmission. For instance, Thompson (2014) exploits data on biological and adopted children to quantify the extent to which the intergenerational transmission of health is due to genetic linkages between parents and their children. The focus of the analysis is on the comparison of intergenerational correlations in health among biological and adopted children. Since an adoptive child is genetically unrelated to the adoptive parents, the difference in the intergenerational health correlations between biological and adopted children can be interpreted as the proportion of parent-child transmission that is due to shared genetics. The main finding is that the genetic transmission of chronic health conditions like asthma and hay fever is around 20-30%. Thus, the majority of health transmission is due to environmental influences.[27]

However, behavioral genetic methods can also be applied to estimate the heritability of economic preferences such as risk attitudes and time preferences (see Section 1.7.3). Of course, from an economics perspective, the impact of the environment is at least as important as the genetic inheritance. In the following section, some theoretical and empirical considerations are made to highlight the (potential) endogeneity of time discounting. This allows us to hypothesize why nurture may be important with regard to time preference.

1.7.1.2 The endogenous determination of time preference

Standard economic literature assumes that (time) preferences can be taken as given or exogenous. Technically speaking, genetic inheritance would be primarily responsible for preference formation and transmission. However, this argument is questioned by researchers who treat preferences as cultural traits or learned behavior.[28] Preferences may be affected and formed by economic institutions, social interactions and cultural evolution (Bowles, 1998). For instance, the effects of markets and other economic institutions on preferences include market framing, the evolution of norms and the design of reward systems. The process of cultural transmission involves child-rearing, childhood socialization and schooling. According to the model framework developed by Bisin and Verdier (2001), preferences of children are directly influenced by their parents' socialization efforts (vertical socialization) and by social and cultural environments (oblique socialization).

In their seminal paper, Becker and Mulligan (1997) provide a theoretical framework of en-

[27] A selection of twin-based findings regarding the heritability of specific health conditions (e.g., diabetes and migraine headaches) is presented in Thompson (2014, Section 2).

[28] The paper by Fuchs (2000) discussed health economics as a behavioral science. His selection of promising areas for future research included the issue of endogenous preferences. He recognized the "attempts to uncover the endogenous aspects of [...] preferences in health and medical care" as "extremely fruitful" which "could enrich the mainstream literature"(Fuchs, 2000, p. 146).

dogenous time preference formation. The idea of the model is that individuals can invest resources in order to alter their discount rates, and therefore become more future-oriented. Such investments may include spending time and efforts to increase the value of future utilities. Other examples are the purchase of commitment devices to forgo current consumption (e.g., regular deposits into a savings account without the option to withdraw money from the account until a certain date) or activities that raise awareness of making provisions for old age (e.g., spending time with aging parents). However, the model also suggests that individual time preference may be influenced by nurture. Parents could directly invest resources in order to teach their children to better plan for the future. As a consequence, offspring discount rates should decrease.[29] According to this model, schooling is also supposed to make young people more future oriented by focusing students' attention towards the future. In addition, Becker and Mulligan (1997) argue that people make frequent experiences about what had been future utilities. This may facilitate their anticipation of the future.

The existing literature on endogenous time preferences is limited. However, there is some empirical evidence that time discounting could be influenced by environmental factors. Studies that analyze the effects of plausibly exogenous shocks on time preferences show that these preferences do change. Voors et al. (2012) conducted a series of field experiments in Burundi to investigate the effects of exposure to violent conflicts on economic behaviors. The findings suggest that exposure to violence increases individual discount rates.[30] Callen (2015) follows a different approach and explores whether time preferences respond to natural catastrophes. He relies on data from a sample of Sri Lankan wage workers and exploits random exposure to the 2004 Indian Ocean Earthquake tsunami. The results show that tsunami exposure increases patience.[31]

As already mentioned above, schooling may also play a role for time preference formation. Indeed, there is suggestive evidence of a causal effect of education on time preference. Perez-Arce (2017) ran an experiment where public college applicants in Mexico City were randomly assigned to either the treatment or the control group. Individuals who were successful in the lottery got immediate acceptance for admission to the college (treatment group). Those who were not picked in the lottery had to wait an entire year before enrollment was possible (control group). On average, the *immediate* admission group acquired more years of education than

[29]Based on the model proposed by Becker and Mulligan (1997), Gouskova et al. (2010) elaborated a model extension that explicitly considers parental investments in offspring's stock of future-oriented capital.

[30]In addition, the experience of violence appears to make people less risk-averse and more altruistic towards their neighbors.

[31]See Meier and Sprenger (2015) for empirical evidence on the temporal stability of time preference.

the *delayed* admission group. Based on a set of hypothetical intertemporal choice questions, the main finding is that the individuals who were successful in the admission lottery were more patient than those who did not get early access to more education. Hence, it seems that schooling has some influence on patience.

Although far from being conclusive, economic theory and experimental studies provide rather plausible evidence that individual time preference is not fully programmed in a person's genetic code but may be prone to social and environmental influences. Based on these considerations, we take a further look at studies that have analyzed the transmission of economic preferences between parents and their offspring.

1.7.2 Intergenerational correlation of economic preferences

In the recent past, research on the intergenerational transmission of economic preferences has gained increasing interest among economists. However, the empirical evidence is still limited. With regard to risk and trust attitudes, Dohmen et al. (2012) found positive correlations between (young) adult children and their parents. In a similar fashion, Gauly (2017) identified intergenerational correlations of proxy variables for time discounting, namely patience and impulsivity. On average, children report a higher level of patience as parents' level of patience increases. Moreover, children's impulsivity depends positively on the impulsivity of their parents. A recent paper by Brown and van der Pol (2015) provides additional evidence on the positive correlation in parental and offspring time and risk preferences. Although studies exist that do not find statistically significant correlations (see, e.g., Bettinger and Slonim (2007) for a study on patience), the majority of the literature reports positive correlations between children's economic preferences and those of their parents.[32]

However, a major limitation of these studies is that the estimation of intergenerational correlations is not informative about what actually drives the transmission process. Observed trait correlations for first-degree relatives (parents and children) can be due to shared heredity as well as shared family environment. However, the nature of the correlational research design makes it impossible to separate genetic effects from environmental effects. In Chapter 4 of the thesis, we address this issue in more detail. It aims to explore if economic preferences are influenced by genetics. In particular, we apply the classical twin design to estimate the genetic and environmental influences on time preference. Surprisingly, this topic has received little

[32]See Brown and van der Pol (2015) for a comprehensive summary of relevant papers that have analyzed the intergenerational correlations of time and risk preferences.

attention in the economics literature so far. But further progress in this area is important in order to gain a better understanding of how economic preferences such as time preferences are actually formed. This and other contributions of our analysis will be discussed next.

1.7.3 Contribution of the thesis

Chapter 4 contributes to the literature on the formation of economic preferences. More specifically, we study the genetic variation in individual time preference using a twin-based research design. We compare monozygotic twins who are genetically identical to dizygotic twins whose genetical structure is not perfectly correlated. This approach allows us to estimate the proportion of variance in time preference that is due to genetic, shared and unshared environmental influences.[33] In general, the (economic) literature on the genetic variation in risk and time preferences is scarce. Moreover, the results are ambiguous. With regard to risk preferences, the heritability estimates vary considerably, from roughly 20% up to more than 60% (Cesarini et al., 2009; Zhong et al., 2009; Zyphur et al., 2009).

Research on the heritability of time preference, however, seems to be still at the very beginning. The poor availability of twin data in combination with a striking lack of reliable time preference measures may perhaps be an explanation for this gap in the literature. Anokhin et al. (2011) conducted a longitudinal twin study in order to estimate the heritability of delay discounting in adolescence (initial sample at age 12: $n = 744$ twins; follow-up sample at age 14: $n = 606$ twins). The children were recruited from the general U. S. population through a twin registry. Participants were asked to choose between $7 in cash immediately or $10 in cash by mail in seven days. A significant heritability of delay discounting of 30% and even 51% was found at ages 12 and 14, respectively. Amongst other behavioral anomalies, Cesarini et al. (2012) investigated the genetic variation in short-term time preference. Using data from a subsample of the Swedish Twin Registry ($n = 11{,}418$ adult twins), survey participants had to choose between receiving an amount of money today and a larger amount of money in the future. While today's payoff remained constant (SEK 5,000), the larger later reward was either SEK 7,000, SEK 6,000 or SEK 5,500 in a week. However, the heritability estimate was not statistically significant.[34]

We conduct an empirical analysis to explore if individual time preferences are under genetic influence. Newly available twin data from Germany is used. Our final sample size is reasonably

[33] An alternative approach of behavioral genetic research is the adoption design. See Plomin and Daniels (2011) for a description of the adoptee strategy.

[34] To the best of our knowledge, there is no other relevant literature on this specific topic.

large and consists of roughly 3,000 twins, distributed in three age groups: children, adolescents and adults. In addition, we introduce a new time preference measure in this context. An experimentally validated survey question on general patience is used to measure time preference. The empirical results suggest that genetic differences explain about 23 percent of the individual variance in time preference. In fact, it is the estimate of the broad-sense heritability which describes the proportion of variance in time preference that is associated with all genetic influences. However, the non-shared environment of the twins in the same family is not only the major source of environmental variation, but also represents the main source of the total variation. Despite the obvious fact that more research on this important but yet under-researched topic is needed, our findings raise awareness of the relative importance of genetic and environmental factors in the intergenerational transmission of preferences. A rigorous understanding of the role and composition of economic preferences may also be interesting for policy makers that aim to break up the vicious cycle of negative (health) behavior patterns and persistent inequality.

Chapter 2

Maternal Preferences and Child Prevention

2.1 Introduction

Even in developed countries, where large parts of the population have reasonably good access to health care, there is substantial variation in individual health outcomes. Although it is well documented that health differences in adulthood can be traced back to health differences in early childhood, at birth, or even earlier (see, e.g., Case et al., 2005), the mechanisms are not well understood.

In principle, one can distinguish between biological, environmental, and behavioral factors that explain observed health differences. The perhaps most important *biological factor* is genetics. Children born with a chronic disease, for instance, will suffer from the same disease in adulthood. Combining biological and *environmental factors*, Barker (1995, 1997, 1998) offered a biological channel other than genetics. He suggested that in-utero conditions are an important determinant not only for child health but for health over the life cycle. In his so-called *fetal-origins hypothesis*, he argues that poor in-utero conditions, *i.e.* malnutrition, lead to a fetal programming that, among other things, changes the metabolic system that makes individuals more prone to develop chronic conditions later in life. Coronary heart disease and hypertension serve as examples (see Barker, 1998). That in-utero malnutrition might have long-lasting effects has attracted the interest of economists who asked whether the correlation underlying Barker's fetal origins hypothesis has a causal interpretation. The idea is to use a natural experiment that randomly assigns malnutrition to the population and thereby to the unborn babies of pregnant women. Famines offer such plausibly exogenous variation. Studying, for instance, the effects of

the Dutch potato famine in the 1840s and the Dutch hunger winter in the 1940s Scholte et al. (2015) and Lindeboom et al. (2010), respectively, find that this in-utero exposure to malnutrition increases mortality.

While the studies on the health consequences of famines have advanced our understanding regarding the importance of in-utero conditions for health outcomes, there is hardly any economic lesson learned. More interesting in this respect are *behavioral factors* that influence in-utero nutrition. One important factor is smoking during pregnancy. It is well documented that smoking during pregnancy harms the unborn baby. Malnutrition resulting in low birth weight, stillbirth or pre-term birth are possible consequences (see, e.g., Bruin et al., 2010). There is also evidence for an increase in the probability of developing mental or physical disorders (see, e.g., Castles et al., 1999; Cnattingius, 2004). The interesting question then is why disadvantaged children never catch up when they grow older. Case and Paxson (2002) investigate how the behavior of parents affects child health. Important factors are, for instance, whether someone in the household smokes and whether the child wears a seat-belt most of the time. The channel through which such behaviors affect child health are more (second-hand smoke) or less (accidents) obvious.[1] Case and Paxson (2002) report socio-economic differences in risky health behaviors. This is perhaps not too surprising as socio-economic status (or education and income) is – just like health – the outcome of investments in human capital. As was argued by Fuchs (1982) these investments and with it the positive correlation between socio-economic status and health can be explained by individual preferences, in particular, time preferences. That preferences indeed matter was demonstrated by Case and Paxson (2001) who investigated health behaviors of legal guardians. They found that legal guardians that are more likely to be altruistic towards children, e.g., adoptive mothers and foster parents, invest more in the health of children (measured in routine doctor visits, for instance) than legal guardians that are likely to be less altruistic, e.g., step mothers.

In our paper we bring all this together and consider smoking during pregnancy a proxy variable for maternal preferences and relate them to the demand for preventive health care for children and adolescents. More precisely, we investigate how maternal preferences affect the vaccination status of children (primary prevention) and the demand for well-child visits (secondary prevention). We thereby shed light on the potential channel that leads from poor health in early childhood to poor health later in life – mothers that attach a relatively low value to the health of their offspring invest comparably few resources in health production. Using the

[1] Case and Paxson (2002) do not investigate potential differences in health care consumption.

base survey from the 'German Interview and Examination Survey for Children and Adolescents' we find that children born to mothers who smoked during pregnancy are about 120 grams lighter than children of mothers who did not smoke during pregnancy. There are, thus, differences in health at birth and these differences persist in terms of parent-reported child health at the time of the interview. More precisely, while 43 percent of mothers who abstained from smoking during pregnancy report that their child is in very good health only 38 percent of mothers who smoked during pregnancy do so. We demonstrate that this pattern may well be explained by the differences in the demand for preventive health care.

We define four dependent variables, namely, two indicator variables measuring whether the vaccination status or the screening status is up-to-date. Motivated by our theoretical framework, we estimate a bivariate probit model with smoking during pregnancy being our most important proxy for maternal preferences. We find a significant reduction in the probability of having an up-to-date screening status of 3.5 percentage points when the mother smoked during pregnancy. In a gender-stratified analysis it turns out that the effect is about twice as large for boys than for girls (-4.6 and -2.3 percentage points, respectively). In the pooled sample, there is no significant association between maternal preferences and the demand for immunization. A gender-stratified analysis reveals that smoking during pregnancy significantly reduces the probability of boys having up-to-date vaccination records by 3.6 percentage points. There is no significant association between our preference proxy and the demand for immunization for girls. We find qualitatively similar results when considering our alternative dependent variables, the vaccination rate and the screening participation rate (both measured as the number of demanded services divided by the number of recommended services). We estimate this model using a bivariate Tobit. In further analysis we use alternate proxies for maternal preferences: smoking during the nursing period, current smoking status of the mother, and whether someone in the household smokes in the presence of the child. Our results are largely robust. Motivated by the growing literature on the socio-economic gradient in child health we investigate whether our suggested preference channel is particularly active in low socio-economic status groups and obtain a somewhat mixed picture. If anything boys born to mothers who smoked during pregnancy are most at risk when their family has a low or medium socio-economic status. Interestingly, we find that the above mentioned patterns do not obtain in families with a migration background. Our results suggest that public policies should target primarily boys living in socially disadvantaged families without migration background.

Our paper also relates to the literature studying the relationship between socio-economic

status and child health. The first paper in this strand of the literature is Case et al. (2002). The contribution of their paper is two-fold. First, by concentrating on the relationship between child health and household income (or socio-economic status) they mitigate the problem of reverse causality inherent in studies trying to pin down how adult income affects adult health: While Currie and Madrian (1999) emphasized that the causality is running from health to education (and, consequently, to income) the reverse causality is at the heart of Grossman's (1972) seminal paper on health production. The second contribution of Case et al. (2002) is that they find an income gradient in child health and that the gradient steepens as children grow older. The gradient was shown to exist in many developed countries including the UK (Currie et al., 2007; Propper et al., 2007; Case et al., 2005), Canada (Currie and Stabile, 2003), and Germany (Reinhold and Jürges, 2012). In some countries the gradient is found to become steeper when children age, in other countries the gradient is persistent. Notably, in none of the countries the gradient is found to become smaller when children grow older. Our paper suggests that maternal preferences might contribute to understanding why the gap never closes or, more dramatically, why the gap is persistent or even grows. Amongst these articles our paper is most related to Reinhold and Jürges (2012) as their analysis is based on the same data as ours. In their analysis they try to identify possible channels through which socio-economic status affects child health. In so doing they find a socio-economic gradient in the demand for well-child visits. Whether or not this contributes to socio-economic gradient in child health remains unclear. By concentrating on the demand for screening exams (and vaccinations) we zoom into the relationship between household characteristics and the demand for preventive care and relate them to individual preferences.[2] They also consider different health behaviors of parents that might affect child health, including smoking during pregnancy or smoking behavior more generally. Like us, they interpret these behaviors as proxies for parental preferences. They find that these behaviors are no mediating factors, that is, the income gradient in child health is robust to adding the respective explanatory variables in their regressions. They remain silent about the channel through which these preferences affect child health (except for second-hand smoke). This is where our study comes into play. As mentioned above, we argue that maternal preferences measured by smoking behavior affect the demand for preventive child care that is an important input in the production of child health.

The remainder of this chapter is organized as follows. In Section 2.2 we present a simple theoretical model to illustrate how maternal preferences affect the demand for preventive child

[2]Note that Reinhold and Jürges (2012) only consider a subset of the screening exams we are investigating. They look at three such exams, we look at 8.

care. The comparative static properties of the model inform the econometric modeling and guide the interpretation of results. The data set is introduced in Section 2.3 followed by a brief description of the econometric framework in Section 2.4. We present our main results in Section 2.5 and offer some extensions in Section 2.6. Section 2.7 concludes.

2.2 Theoretical framework

We consider a model family with one parent and one child in a two-period setting. In period 1 the parent earns an income $y > 0$ and may invest part of this income in prevention. This effort lowers the expected health loss $EL > 0$ of the child in period 2. We let the expected loss depend on primary prevention effort $e_1 \geq 0$ and secondary prevention effort $e_2 \geq 0$. Income, health loss, and preventive effort are measured in monetary units.

First and foremost, primary prevention is directed towards lowering the probability of disease occurrence, vaccinations being the prime example. Vaccinations are typically imperfect: they only offer partial protection, that is, they reduce the probability of illness but not to zero.[3] If vaccinated individuals are infected, however, the course of the disease is typically milder as compared to unprotected individuals.[4] Primary prevention may thus reduce the expected loss by lowering the probability of infection, by reducing the size of the loss conditional on infection, or by both.

For secondary prevention the situation is very similar. Screenings primarily aim at early disease detection. While earlier treatment likely reduces the health loss coming along with the disease, a screening cannot affect the probability that the respective disease occurs (*e.g.*, prostate cancer). If the disease is a risk factor for another disease, though, early detection of the risk factor may reduce the probability of the other disease occurring. Consider, for instance, well-child visits. As part of the health exam, height and weight of children are measured. This allows the physician to assess the physical development of the child and to position the child in the age and sex specific weight distribution. Early detection of excess body weight or obesity may make parents aware of associated health problems like diabetes. This may induce parents to take measures that reduce the body weight of their child which, in turn, reduces the probability that their child develops diabetes.

[3] To keep the analysis focused, we abstract from potential side-effects of vaccinations.

[4] The mumps-measles-rubella (MMR) vaccine serves as an example. The CDC states that the efficacy of two doses of the MMR vaccine is 97 percent for the case of measles and that the course of the disease may indeed be milder for vaccinated individuals as compared to unprotected ones (Centers for Disease Control and Prevention (CDC), 2018b).

As primary and secondary prevention are likely to affect the expected loss via both a reduced probability of disease occurrence and a lower loss conditional upon developing or contracting a disease a very general expected health loss function $EL = EL(e_1, e_2)$ is required. We assume that the expected health loss is strictly decreasing with both, primary and secondary prevention, that is, $EL_i < 0$, $i = 1, 2$. As usual, we let the productivity of prevention be decreasing, $EL_{ii} > 0$, $i = 1, 2$. We do not make any assumptions regarding the size and sign of the cross partial derivatives $EL_{ij} = EL_{ji}$, $i \neq j$.[5]

To keep things simple the child is considered inactive in both periods and the parent inactive in period 2. To get positive prevention efforts e_1 and e_2 the parent needs to be altruistic towards its offspring. The parameter $\alpha \in (0, 1]$ reflects the degree of altruism with $\alpha = 1$ characterizing a parent who fully internalizes the child's health loss. As prevention costs are incurred in period 1 while the benefits may materialize in period 2, time preferences, measured by the discount factor $\delta \in (0, 1]$, matter. Finally, benefits are uncertain so that risk attitudes will also impact prevention decisions. We capture risk aversion by considering Yaari's (1987) dual theory. This essentially implies that risk averse individuals overstate the probability of bad outcomes. For the sake of illustration, we consider a constant over-statement factor $\rho \geq 1$ with $\rho = 1$ characterizing a risk neutral individual. We arrive at the following utility function of the parent

$$U(e_1, e_2) = y - e_1 - e_2 - \alpha\delta\rho EL(e_1, e_2). \tag{2.1}$$

This simple theory nicely demonstrates that disentangling the impact of different preference dimensions on preventive actions poses a major identification challenge. In fact, it is impossible to independently identify the three preference parameters α, δ, and ρ. More altruism (a higher α), more future orientation (a larger δ) and a higher degree of risk aversion (a larger ρ) all have the same directional effect on prevention levels. This is why we summarize them in a single parameter $\theta = \alpha\delta\rho$.

The parent maximizes utility (2.1) with respect to e_1 and e_2. Concentrating on interior solutions, the corresponding first order conditions are given by

$$\frac{\partial U}{\partial e_i} = 0 \quad \Leftrightarrow \quad -\theta EL_i(e_1, e_2) = 1, \qquad i = 1, 2. \tag{2.2}$$

Both first order conditions require that the marginal cost of prevention (the respective right-hand sides) are equal to the expected marginal benefit of prevention (the respective left-hand

[5]To ease notation we write $EL_i \equiv \frac{\partial EL}{\partial e_i}$ and $EL_{ij} \equiv \frac{\partial^2 EL}{\partial e_j \partial e_i}$, $i, j \in \{1, 2\}$.

sides). The first order conditions imply $EL_1 = -1/\theta = EL_2$.[6]

To analyze how a change in θ affects prevention efforts we totally differentiate the first order conditions. In matrix notation we obtain

$$-\theta \begin{pmatrix} EL_{11} & EL_{12} \\ EL_{12} & EL_{22} \end{pmatrix} \begin{pmatrix} de_1 \\ de_2 \end{pmatrix} = \begin{pmatrix} EL_1 \\ EL_2 \end{pmatrix} d\theta. \tag{2.3}$$

Applying Cramer's rule and using the first order conditions we find

$$\frac{de_1}{d\theta} = \frac{EL_{22} - EL_{12}}{\det(H_U)}, \tag{2.4}$$

$$\frac{de_2}{d\theta} = \frac{EL_{11} - EL_{12}}{\det(H_U)}. \tag{2.5}$$

As we made no assumption regarding the size and sign of the cross partial derivate of the expected loss we are unable to generally sign the comparative static effects. For the case of complements, $U_{ij} > 0$, we have $EL_{ij} < 0$ and both, primary and secondary prevention are strictly increasing with the parameter θ. This also applies when the cross derivative vanishes, that is, when primary and secondary prevention are additively separable in the expected loss. This would be the case if the different forms of prevention were directed towards different and independent diseases.[7] For the case of substitutes, $U_{ij} < 0$, we have $EL_{ij} > 0$ and the directional effect of θ on prevention is generally ambiguous. It is straightforward to see, however, that at least one preventive activity necessarily must be increasing with θ. To see this, suppose that primary and secondary prevention would drop with an increase in θ. Then $EL_{11} < EL_{12}$ and $EL_{22} < EL_{12}$ would have to hold simultaneously violating the second order condition – a contradiction.[8]

Our simple theoretical model carries three important messages that inform the econometric modeling of prevention decisions. First, as already mentioned above, the model reminds us of a fundamental identification challenge when it comes to measuring the impact of different preference dimensions on preventive actions. Second, the directional effect of a change in preference

[6] We assume throughout that the second order conditions for utility maximization are satisfied, that is, the Hessian of the utility function, H_U, needs to be negative definite: $U_{ii} < 0 \Leftrightarrow EL_{ii} > 0$, $i = 1, 2$, and $\det(H_U) = \theta^2(EL_{11}EL_{22} - EL_{12}^2) > 0$.

[7] With additive separability the expected loss may, for instance, take the form $EL = \pi_1(e_1)L_1 + \pi_2 L_2(e_2)$, with $\pi_1' < 0$, $\pi_1'' > 0$, $L_2' < 0$, and $L_2'' > 0$. Equations (2.4) and (2.5) then simplify to $de_1/d\theta = -\pi_1'/\theta\pi_1'' > 0$ and $de_2/d\theta = -L_2'/\theta L_2'' > 0$, respectively.

[8] An important example for substitutes is a situation where primary and secondary prevention effort target the same disease. The expected loss may then assume the following functional form: $EL(e_1, e_2) = \pi(e_1)L(e_2)$, with $\pi' < 0$, $\pi'' > 0$, $L' < 0$, and $L'' > 0$.

parameters on prevention decisions is not unambiguously determined so that it is an empirical exercise to pin down their (joint) effect. Third, primary and secondary prevention may well depend on one another calling for a simultaneous decision model.

2.3 Data and descriptive statistics

2.3.1 Data source and sample selection

We use data from the base survey of the 'German Interview and Examination Survey for Children and Adolescents (KiGGS)' conducted by the Robert Koch Institute (RKI) from 2003 to 2006. The survey sampled 17,641 children aged from 0 to 17 years and aimed at providing a representative cross-section of children and adolescents living in Germany. Children and their families were invited to participate using a two-stage randomization procedure. First, 167 communities were randomly selected (primary sampling units). Second, at the level of primary sampling units, population registries were used to randomly select families for an invitation to participate (secondary sampling units).[9] Parents were asked to answer a questionnaire as well as children aged 11 years and above. Moreover, children had to undergo a medical exam comprising physical measurements, laboratory tests, and a Computer Assisted Personal Interview by a physician. The medical exams and the interview covered, among other things, a number of diseases and detailed vaccination records.[10]

In the previous section we showed how the preferences of the mother affect the demand for preventive health care after the child is born. As the relevant preference dimensions, that is, risk aversion, time preferences, and altruism, are not directly observed, we use the health behavior of the mother before the child was born as a proxy for these preferences (we provide more details below). This approach requires that the child is raised by the biological mother. This restriction reduces the sample size by about 600 observations (see Table 2.1). We further restrict the sample to children where at least one biological parent answered the parental questionnaire. This guarantees more accurate information as compared to a situation where grandparents or even nannies filled out the questionnaire. We arrive at a sample of 16,994 observations. Due to missing information, mostly on control variables, the regression results presented in Section 2.5 are based on 11,826 observations.

[9] The response rate was 66.6 percent.

[10] For more information on the data set see Kurth et al. (2008).

Table 2.1: Sample selection

Selection criterion	*N*
none	17,641
child living with biological mother	17,047
biological parent(s) answered questionnaire	16,994
missings (full model)	11,826

2.3.2 Primary prevention – vaccinations

Vaccines stimulate the immune system and ideally help to develop immunity against the disease in question. Although the efficacy of the vaccines considered in our analysis is generally high, none of the vaccines is perfect in the sense that the probability of infection is reduced to zero. The German Standing Committee on Vaccination (Ständige Impfkommission, STIKO) at the RKI regularly issues vaccination recommendations.[11] Based on these recommendations, survey physicians checked vaccination records of sampled children as part of the medical exam. In total 9 diseases were considered, namely, Diphtheria, Polio, Tetanus, Mumps, Measles, Rubella, Pertussis, Hib, and Hepatitis B. For our analysis we concentrate on the first six diseases as information on the latter three is subject to substantial measurement error. We construct two variables to capture the vaccination status of the child. The first variable V_{UTD} is an indicator that assumes the value one whenever the vaccination status of the child is up-to-date at the time of the medical exam (considering the STIKO recommendations and the age of the child) and zero otherwise.[12] For two reasons this measure might be considered problematic. First, one may argue that it overstates the importance of vaccination timing. Second, the measure makes no difference between children that never got immunized against any of the diseases we are looking at and those who just missed one immunization. This is why we define the vaccination rate V_{RATE}. It is calculated by dividing the number of demanded vaccinations by the age-specific number of recommended vaccinations considering the six diseases mentioned above.

Only 55 percent of sampled children have an up-to-date vaccination record. Figure 2.1 below illustrates how this share relates to the age of children (the dashed line). Very young children are closely monitored (see also the next subsection) so that it is perhaps not too surprising to see the highest shares of up-to-date vaccination records in the group of children under 2 years. The solid line in the figure depicts the vaccination rate which, by definition, is everywhere

[11] For the current recommendations see Standing Committee on Vaccinations (STIKO) (2017).

[12] We do not have access to vaccination dates so that we cannot assess whether immunizations were demanded in the respective recommended age brackets.

above the dashed line. The considerable gap between these two curves shows that most children demand at least some immunization. While 4.3 percent of children have never received any immunization, 55.4 percent of children demanded all recommended vaccinations. We conclude that immunization rates in German children are shockingly low.

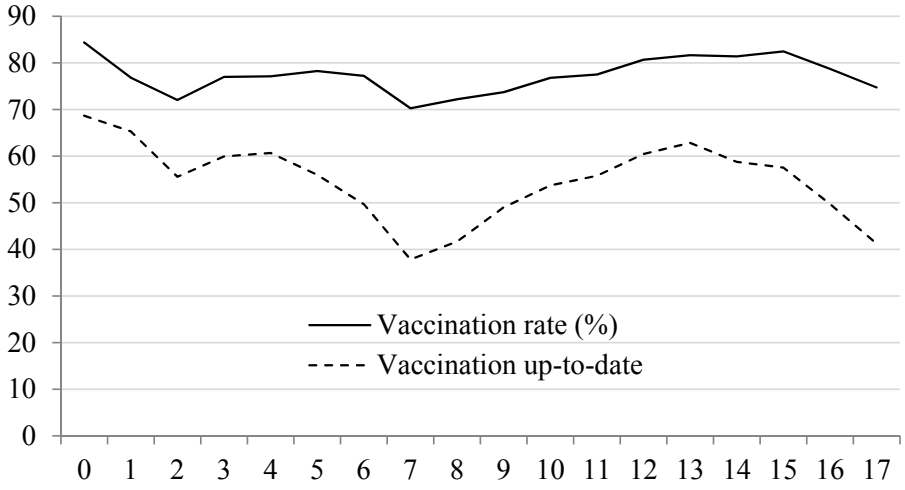

Figure 2.1: Vaccination status up-to-date and vaccination rates by age (in years)

2.3.3 Secondary prevention – screening examinations

The rationale behind regular screenings is that diseases or risk factors for diseases are detected earlier than without them. Early detection allows for better treatment or for adjustments in behavior to improve health prospects or to postpone the worsening of individual health. During the sampling period 10 screenings were scheduled for children under the age of 18 years. Back then participation was voluntary.[13] Table 2.8 in the Appendix shows the age brackets for screening examinations. Based on this schedule we define the indicator variable screening up-to-date, S_{UTD}. The variable assumes the value one whenever a child demanded the latest screening examination and zero otherwise (considering the age of the child and the screening schedule). Suppose, for instance, that a 4 months old child has already demanded screening U4. As this examination is scheduled for the third or fourth month of life the screening status is clearly

[13]In 2008 the regional state of Bavaria passed a law making participation mandatory. For details see https://www.stmas.bayern.de/kinderschutz/praevention/index.php (accessed on April 10, 2018). Similar laws have been implemented in the regional states of Hesse and Baden-Württemberg. However, the majority of regional states rely on reminders to inform parents and/or health authorities about screenings that have been missed. Enforcement remains an issue, though (see Zeit Online, 2011). For more details see https://www.bundestag.de/blob/406316/8f4c316937f69892c86fce34c6946d28/wd-3-143-14-pdf-data.pdf (accessed on April 10, 2018).

up-to-date. Even if the latest screening exam was U3, the status would be up-to-date as there is still time left to demand U4 as scheduled. By contrast, a five months old child would not have an up-to-date screening status if the latest examination was U3. We concentrate on examinations U3 through U9 and J1. Parents can hardly decide on whether or not to participate in U1 and U2. U1 is due in the first few hours after birth and U2 between the third and tenth day of life. While U1 is typically conducted right in the hospital, a midwife takes care of U2.[14]

Like with the vaccination up-to-date variable one may argue that the screening up-to-date variable over-emphasizes the importance of examination timing. Also children without any examination are not distinguished from children that just missed the last one (but demanded all other ones). This is why we define the screening rate, S_{RATE}, as the number of demanded screenings divided by the age-specific number of recommended screenings. Figure 2.2 shows that, apart from J1, there is no big difference between the alternative screening measures – compliance is generally high. Note that 2.3 percent of children never attended any screening exam and that 78.3 percent of children attended all scheduled screening exams.

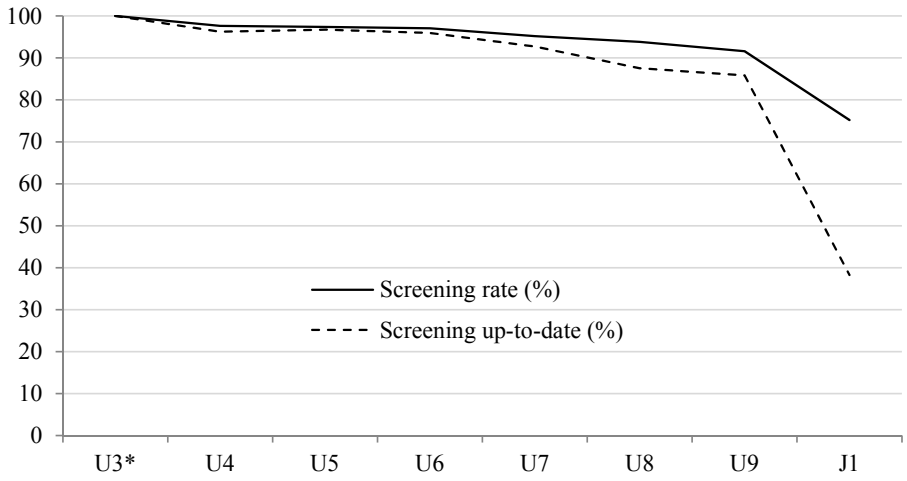

Figure 2.2: Screening status up-to-date and screening rates by age (in recommended screening brackets)

There are at least two reasons for the small difference between the two measures. First, the nature of age-dependent screening examinations is that pediatricians assess whether the child develops according to age. It makes no sense, for instance, to check whether a 6 year old child can crawl. This is part of U6 which is due between the 10th and 12th month of life.[15] Second,

[14] We only have two children under the age of 6 weeks in our sample so that this restriction is innocent.
[15] In Table 2.8 in the Appendix we provide details regarding the content of screening examinations.

physicians have no incentive to conduct screenings outside the recommended age bracket as remuneration is conditional upon timely examinations. This implies that one can hardly catch up on missed examinations.

2.3.4 Maternal preferences and health behaviors

One of the lessons of our theoretical framework is the impossibility to independently identify the effects of different preference dimensions on preventive behavior. Given this identification problem, the unavailability of preference information in the KiGGS data is a minor issue as long as proxies for our composite preference measure θ are available. We argue that a mother's smoking behavior is a good proxy for her individual preferences. Barsky et al. (1997), Anderson and Mellor (2008), and Dohmen et al. (2011) show that more risk tolerance, *i.e.* less risk aversion, is associated with higher smoking participation. Fuchs (1982) was the first to emphasize the importance of time preferences in health investment decisions. Ida and Goto (2009b), Kang and Ikeda (2014), and Scharff and Viscusi (2011) present evidence for low discount factors (present orientation) being associated with high smoking participation. Evidence on an association between altruism and smoking behavior is lacking. Khwaja et al. (2006), for instance, find no impact of altruism on smoking behavior considering altruism between spouses.[16] We are confident, however, that smoking behavior of the mother is correlated with altruism towards its offspring. The reason being that the case for a role of altruism in health behaviors is stronger in a parent-child environment than in partnership or marriage.

We measure maternal preferences using four alternative proxies, namely, smoking during pregnancy, smoking during the nursing period, whether the mother is a current smoker, and whether someone in the household smokes in the presence of the child. Accordingly, we define indicator variables for each behavior: $PREGNANCY$, $NURSING$, $CURRENT$, and $PRESENCE$. In our data set 14 percent of women smoked during pregnancy, 7 percent of mothers smoked during the nursing period, 27 percent of mothers are current smokers, and in 13 percent of all households someone smokes in the presence of the child. The pairwise correlations of our four preference proxies range from .22 to .55 and are, thus, in the medium range (see Table 2.2). Assuming the stability of preferences our descriptive statistics provide indirect evidence for altruistic motives affecting smoking participation. While 27 percent of mothers are current smokers much fewer women smoked when the child is directly affected, *e.g.*, when the child is in

[16] By contrast, there is evidence for a positive association between altruism (or pro-social behavior) and the demand for immunization (see, e.g., Böhm et al., 2016; Nuscheler and Roeder, 2016).

utero. Admittedly, this pattern is also consistent with smoking being under-reported whenever the child is directly affected.[17] Even if there was under-reporting we argue that those with a very strong preference for smoking, that is, mothers with little risk aversion, future orientation and altruism, are more inclined to report having smoked during pregnancy, during the nursing period, or that they smoke in the presence of the child than those with a weaker preference for smoking. In short, we conjecture that even in the case of under-reporting the relationship between preferences and reported smoking behavior remains intact.

Table 2.2: Pairwise correlations of preference proxies and prevention decisions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(1) $PREGNANCY$	1							
(2) $NURSING$	.55	1						
(3) $CURRENT$	.52	.36	1					
(4) $PRESENCE$	.35	.22	.37	1				
(5) V_{UTD}	−.00	−.02	−.00	−.01	1			
(6) S_{UTD}	−.03	−.02	−.06	−.11	.10	1		
(7) V_{RATE}	−.01	−.03	−.01	−.01	.83	.11	1	
(8) S_{RATE}	−.02	−.02	−.05	−.09	.08	.62	.09	1

In Table 2.2 we summarize all correlations between our preference proxies and the variables measuring the demand for prevention. As expected, we find negative (but small) associations between all preference proxies and all prevention measures. Figure 2.3 provides little more detail on the association between smoking during pregnancy, $PREGNANCY$, and the four variables measuring the demand for prevention by showing the conditional means. A clear picture emerges: no matter how the demand for prevention is measured, the demand is always lower for mothers who smoked during pregnancy. The differences in conditional means range from 3 percentage points for screening up-to-date to .6 percentage points for vaccination status up-to-date. Although the effects are small, $PREGNANCY$ will turn out significant in most regressions below.[18]

[17] Note, however, that Gruber and Köszegi (2001) found no under-reporting of smoking in pregnant women.

[18] For empirical evidence that time preferences affect the demand for prevention see, for instance, Bradford (2010), Chapman and Coups (1999), and Picone et al. (2004). Binder and Nuscheler (2017) show that more risk aversion is associated with a higher demand for vaccination.

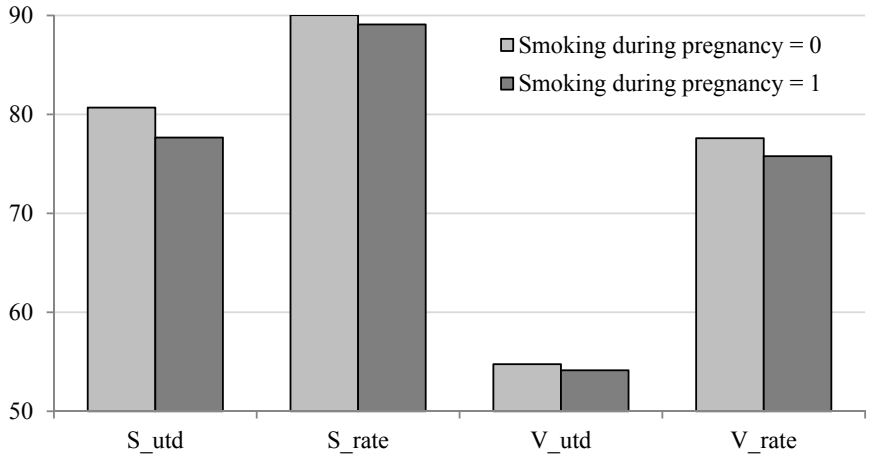

Figure 2.3: Screening examinations, vaccination decisions (both in %), and smoking during pregnancy

2.3.5 Individual heterogeneity

The KiGGS data include extensive information on individual characteristics of children, mothers, and fathers. We here provide a complete list of variables that we include as control variables in our econometric models below.[19]

Individual heterogeneity of parents is modeled considering age, marital status, education (8 indicator variables for educational achievement) and employment status (12 indicators), separately for mothers and fathers. Gender and age are the most important control variables for children. To capture the different development stages of children we include Tanner scores (4 stages). Differential access to health care is captured by a variable indicating private health insurance.[20] We also include an indicator that controls for the occurrence of health problems of the child within four weeks after delivery. Such problems are likely to affect the demand for screening exams and might also have an impact on the demand for immunizations. Note that we do not add any additional variables capturing the health status of children as this would – at least theoretically – bias our estimates (we discuss in more detail in the next section).

At the level of households, we include a dummy variable for East-German residence as East-German parents were exposed to a different health care system than West-German parents. There was, for instance, mandatory vaccination in East Germany but not in West Germany

[19]For the most important variables a description of variables and summary statistics are provided in Tables 2.9 and 2.10 in the Appendix.

[20]All vaccines considered here are covered by all health plans no matter whether they are public or private. Reimbursement rates are higher in the private system, though. This also applies to the screening exams.

(except for the smallpox vaccine). An urbanization dummy captures the presumably larger risk of infection in densely populated areas and better access to health care due to the higher physician density. Prevention decisions of parents (or mothers) may depend on the family structure. The number of children may play a role but also whether the sampled child has older or younger siblings. We also include net household income (in brackets) in our regression models. Finally, we include the migration background of the family.

2.4 Econometric framework

In our theoretical model we considered primary and secondary prevention simultaneous decisions. To mirror this in the empirical analysis we jointly estimate the demand for the two modes of prevention. Considering the comparative static properties, equations (2.4) and (2.5), the optimal level of primary prevention depends on the level of secondary prevention and *vice versa* unless the cross derivative vanishes. This calls for a simultaneous equation model with e_j being an explanatory variable for e_i, $i = 1, 2$ and $i \neq j$. Due to the lack of convincing exclusion restrictions we cannot identify the structural form parameters of our model so that we resort to estimating the reduced form, that is, we regress e_i on a set of plausibly exogenous variables, $i = 1, 2$. We capture the potential interdependence between primary and secondary prevention by allowing the error terms to be correlated across equations.

When considering the up-to-date variables, that is $e_1 = V_{UTD}$ and $e_2 = S_{UTD}$, our dependent variables are binary calling for a simultaneous binary response model and we opt for the bivariate probit. Alternatively we let $e_1 = V_{RATE}$ and $e_2 = S_{RATE}$. As both variables measure the actual demand for preventive health care as a proportion of the number of recommended vaccinations and screenings, respectively, they are continuous and naturally bounded between 0 and 1. We already saw above that censoring matters, especially right-censoring. Accordingly, we estimate a bivariate Tobit model taking into account left-censoring and right-censoring. In all models we cluster the standard errors at the level of primary sampling units.

Table 2.2 showed all unconditional correlations between our preference proxies and prevention variables. The table revealed that the correlations between the two up-to-date variables and the two rate-variables are relatively small. This suggests that the decisions for primary and secondary prevention might be independent rendering separate estimation feasible. We found only minor differences between coefficient estimates of the two approaches, namely, separate and simultaneous. As standard errors are marginally smaller for the latter we stick with the more

efficient simultaneous equation model.[21]

Before we turn to the results we should remind ourselves to interpret them cautiously. The decision to smoke during the nursing period, in the presence of the child or to smoke at all may well be viewed as being simultaneous to the decisions to demand vaccinations and screening exams for the child, respectively. We are confident, however, that the simultaneity bias is small if there is any. Simultaneity is not an issue when considering smoking during pregnancy as this behavior naturally precedes the demand for prevention. This is why we emphasize the results for smoking during pregnancy and view the results using the alternative preference proxies primarily as robustness checks. Differences in coefficient estimates may not be the result of simultaneity bias but rather reflect that our proxies measure slightly different things. As already mentioned above, when the child is directly affected by smoking behavior then altruism might play a larger role than otherwise. If someone smokes in the presence of the child, for instance, the child is exposed to second-hand smoke which is known to be harmful for the child. That smoking during pregnancy might have adverse health effects for the unborn baby is perhaps less clear. Differences between the Probit and Tobit models are likely to be rooted in the differences of the econometric approaches. They may also reflect, at least partially, that the dependent variables measure marginally different things: the up-to-date variables emphasize the correct timing of prevention, the rate-variables not so much. One would expect that time preferences play a larger role in the former than in the latter. This may translate into differences in the coefficients of our proxies for composite maternal preferences.

Finally, a discussion regarding the role of child health in the econometric analysis is in order. Our data comprise two prominent variables that are reasonably good measures of child health, namely, birth weight and self-assessed health status. It is well known that smoking during pregnancy is a risk factor for premature birth and low birth weight (see, e.g., Bruin et al., 2010). Indeed a naïve comparison of conditional means shows that children born to mothers who smoked during pregnancy are about 120 grams lighter than those born to mothers who abstained from smoking during pregnancy. Birth weight may thus be considered an outcome variable, that is, birth weight is a 'bad control' and including such a variable would introduce selection bias (Angrist and Pischke, 2009, pp. 64-68). When using our alternative preference proxies, birth weight is an unproblematic control variable as it is determined prior to the smoking behaviors under consideration. To facilitate the comparison of regression estimates we refrain from using birth weight as a control variable in these models as well. Self-assessed health would

[21] The results of the simultaneous equation model are shown in Table 2.4. The outcome with separate equations, Table 2.11, is relegated to the Appendix.

obviously introduce simultaneity bias. Both vaccinations and screening exams are inputs in the production of child health. This is particularly so as our dependent variables measure the demand for prevention in the past while self-assessed health measures current child health.[22] It is well established that poor health outcomes at birth (or early childhood) have long-lasting effects, including a negative impact on health (see, e.g., Case et al., 2005). But then maternal preferences not only affect child health at birth or early childhood but also later in life so that self-assessed health is likely to be 'bad control'. A regression of the demand for prevention on self-assessed health would, thus, introduce simultaneity *and* selection bias. We are confident that neglecting self-assessed health and birth weight does not introduce omitted variable bias into our analysis. The reason being that the demand for prevention is a more fundamental decision that – as the theoretical model suggests – is driven by the preferences of parents or, more precisely, of maternal preferences.[23]

2.5 Results

2.5.1 Smoking during pregnancy and prevention

2.5.1.1 Model specification

Our main results are shown in Table 2.3. The first column shows the average marginal effects of smoking during pregnancy on the two modes of prevention without any control variables. In columns 2 to 4 we successively add control variables. As the results are very robust, we concentrate on our preferred specification with the full set of control variables, namely, column 4.

Considering the bivariate probit estimates for the two up-to-date variables first we find that smoking during pregnancy has no statistically significant effect for vaccinations but for screening exams. Children of mothers who smoked during pregnancy have a 3.5 percentage points lower probability of having demanded the latest screening exam. A similar picture emerges for the two rate variables. The vaccination rate is not significantly associated with the mother's smoking behavior during pregnancy but the demand for screening exams is. Note that we report marginal

[22] A simple regression of an indicator for very good child health on V_{UTD} and S_{UTD} shows that having an up-to-date vaccination record increases the probability of the child being in very good health by 2 percentage points. The effect of an up-to-date screening status amounts to 10 percentage points. Both effects are highly statistically significant.

[23] Our regression results are robust to including birth weight and self-assessed health. Note that our set of control variables includes an indicator variable for post-pregnancy health problems of children (within four weeks after birth), e.g., jaundice. There is no significant correlation between this indicator and $PREGNANCY$ so that this control appears unproblematic. It just captures that such problems may make parents more health conscious.

Table 2.3: Maternal preferences and child prevention: model specification

PREGNANCY	(1)	(2)	(3)	(4)
V_{UTD}	-0.0016	-0.0078	-0.0020	-0.0089
	(0.0133)	(0.0131)	(0.0126)	(0.0124)
S_{UTD}	-0.0282^{***}	-0.0386^{***}	-0.0369^{***}	-0.0347^{***}
	(0.0102)	(0.0096)	(0.0098)	(0.0099)
ρ	0.1821^{***}	0.1778^{***}	0.1747^{***}	0.1748^{***}
	(0.0175)	(0.0193)	(0.0197)	(0.0197)
V_{RATE}	-0.0081	-0.0104	-0.0070	-0.0123
	(0.0084)	(0.0084)	(0.0080)	(0.0078)
S_{RATE}	-0.0165^{***}	-0.0205^{***}	-0.0202^{***}	-0.0183^{***}
	(0.0052)	(0.0049)	(0.0048)	(0.0049)
ρ	0.1615^{***}	0.1862^{***}	0.1930^{***}	0.1930^{***}
	(0.0144)	(0.0148)	(0.0145)	(0.0143)
Control variables:				
Child		yes	yes	yes
Household			yes	yes
Parents				yes
N	11,826	11,826	11,826	11,826

Notes: Average marginal effects of $PREGNANCY$. Clustered standard errors in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

effects in all tables. For both the bivariate probit and the bivariate tobit we calculate the average marginal effects. For the latter we concentrate on the marginal effects of the censored variable, that is, the marginal effects are given by the coefficient estimate times the probability that the dependent variable is uncensored. On average, this probability amounts to 40 percent for vaccinations and 19 percent for screening exams, respectively. We find that the vaccination rate is not significantly associated with a mother's smoking behavior during pregnancy but that the demand for screenings is: the total demand for screening exams of children with mothers who smoked during pregnancy is about 1.8 percentage points smaller than for children of mothers who did not smoke during pregnancy.

In Section 2.4 we argued that measures of child health would be 'bad controls' in a regression like ours. One may argue that most household and parent characteristics are also bad controls. After all, most variables measure education, income, or employment status. The robustness of our results shown in Table 2.3 are reassuring that any selection bias would be small if there is any. Also note that papers investigating the socio-economic gradient in child health, that ask whether parental behaviors like smoking during pregnancy are mediators, face the same problem (see, e.g., Case et al., 2005).

2.5.1.2 Gender effects

It is instructive to stratify the sample by gender. Table 2.4 shows that the effects are markedly stronger for boys than for girls. For girls the smoking behavior of the mother during pregnancy is not significantly associated with the demand for vaccinations. By contrast, boys of mothers that smoked during pregnancy have a 3.6 percentage points lower probability of having an up-to-date vaccination record. For screening exams we find that the effects of smoking during pregnancy are twice as large for boys than for girls. While boys of mothers who smoked during pregnancy have a 4.6 percentage points lower probability of having demanded the latest screening exam, the effect for girls is only 2.3 percentage points, though still statistically significant. The results for the bivariate tobit model demonstrate that these gender effects are not mere artifacts of improper prevention timing for sons but that the demand for preventive care of mothers who smoked during pregnancy is systematically smaller for their sons than for their daughters however measured.

Table 2.4: Maternal preferences and child prevention: gender effects

PREGNANCY	all	boys	girls
V_{UTD}	-0.0089	-0.0355^{**}	0.0173
	(0.0124)	(0.0176)	(0.0206)
S_{UTD}	-0.0347^{***}	-0.0464^{***}	-0.0231^{*}
	(0.0099)	(0.0134)	(0.0125)
ρ	0.1748^{***}	0.1593^{***}	0.1971^{***}
	(0.0197)	(0.0284)	(0.0253)
V_{RATE}	-0.0123	-0.0246^{**}	-0.0005
	(0.0078)	(0.0105)	(0.0125)
S_{RATE}	-0.0183^{***}	-0.0234^{***}	-0.0142^{**}
	(0.0049)	(0.0066)	(0.0063)
ρ	0.1930^{***}	0.1739^{***}	0.2151^{***}
	(0.0143)	(0.0204)	(0.0207)
N	11,826	5,994	5,832

Notes: Average marginal effects of $PREGNANCY$. Clustered standard errors in parentheses. All regressions include the full set of controls.
$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

2.5.2 Alternate preference proxies

In Table 2.5 we contrast our results for our preference proxy smoking during pregnancy, $PREGNANCY$, with alternative preference proxies, namely, smoking during the nursing period, $NURSING$, current smoking status of the mother, $CURRENT$, and whether someone in the household smokes in the presence of the child, $PRESENCE$.

Table 2.5: Alternate preference proxies

full sample	PREGNANCY	NURSING	CURRENT	PRESENCE
V_{UTD}	-0.0089	-0.0344^{**}	-0.0111	-0.0152
	(0.0124)	(0.0168)	(0.0103)	(0.0149)
S_{UTD}	-0.0347^{***}	-0.0234^{**}	-0.0364^{***}	-0.0447^{***}
	(0.0099)	(0.0109)	(0.0072)	(0.0083)
ρ	0.1748^{***}	0.1744^{***}	0.1737^{***}	0.1741^{***}
	(0.0197)	(0.0197)	(0.0197)	(0.0197)
V_{RATE}	-0.0123	-0.0265^{***}	-0.0151^{**}	-0.0153^{*}
	(0.0078)	(0.0101)	(0.0062)	(0.0086)
S_{RATE}	-0.0183^{***}	-0.0175^{***}	-0.0165^{***}	-0.0217^{***}
	(0.0049)	(0.0053)	(0.0037)	(0.0042)
ρ	0.1930^{***}	0.1928^{***}	0.1917^{***}	0.1921^{***}
	(0.0143)	(0.0143)	(0.0143)	(0.0142)
N	11,826	11,826	11,826	11,826
boys	PREGNANCY	NURSING	CURRENT	PRESENCE
V_{UTD}	-0.0355^{**}	-0.0761^{***}	-0.0216	-0.0174
	(0.0176)	(0.0239)	(0.0141)	(0.0213)
S_{UTD}	-0.0464^{***}	-0.0197	-0.0408^{***}	-0.0482^{***}
	(0.0134)	(0.0168)	(0.0101)	(0.0108)
ρ	0.1593^{***}	0.1602^{***}	0.1580^{***}	0.1606^{***}
	(0.0284)	(0.0284)	(0.0286)	(0.0283)
V_{RATE}	-0.0246^{**}	-0.0446^{***}	-0.0198^{**}	-0.0177
	(0.0105)	(0.0134)	(0.0081)	(0.0116)
S_{RATE}	-0.0234^{***}	-0.0152^{**}	-0.0177^{***}	-0.0212^{***}
	(0.0066)	(0.0075)	(0.0050)	(0.0053)
ρ	0.1739^{***}	0.1746^{***}	0.1728^{***}	0.1743^{***}
	(0.0204)	(0.0206)	(0.0205)	(0.0205)
N	5,994	5,994	5,994	5,994
girls	PREGNANCY	NURSING	CURRENT	PRESENCE
V_{UTD}	0.0173	0.0070	0.0010	-0.0102
	(0.0206)	(0.0263)	(0.0151)	(0.0186)
S_{UTD}	-0.0231^{*}	-0.0263^{*}	-0.0318^{***}	-0.0421^{***}
	(0.0125)	(0.0151)	(0.0096)	(0.0122)
ρ	0.1971^{***}	0.1966^{***}	0.1964^{***}	0.1951^{***}
	(0.0253)	(0.0253)	(0.0253)	(0.0254)
V_{RATE}	-0.0005	-0.0089	-0.0100	-0.0122
	(0.0125)	(0.0164)	(0.0099)	(0.0110)
S_{RATE}	-0.0142^{**}	-0.0204^{***}	-0.0155^{***}	-0.0221^{***}
	(0.0063)	(0.0076)	(0.0049)	(0.0061)
ρ	0.2151^{***}	0.2148^{***}	0.2139^{***}	0.2134^{***}
	(0.0207)	(0.0206)	(0.0207)	(0.0207)
N	5,832	5,832	5,832	5,832

Notes: Average marginal effects of the respective preference proxies. Clustered standard errors in parentheses. All regressions include the full set of controls. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

We find that the screening status of children is generally significantly negatively associated with the smoking behavior of the mother no matter how we actually measure the demand for screenings and the preferences of the mother. Considering the full sample and screening up-to-date we find that the 95 percent confidence intervals overlap for all preference proxies. It should be noted, however, that the point estimates differ by a factor of up to almost two (-0.0234 for $NURSING$ and -0.0447 for $PRESENCE$) and that there are no marked differences across gender. As argued above, these differences might reflect that our proxies measure slightly different things, *i.e.*, that the relevant preference dimensions of the mother enter the proxies with different weights. When measuring the demand for screenings using the screening rate we find that the marginal effects are very robust across preference proxies and across gender. As the main difference between the two alternative dependent variables is the timing of screening demand the higher variation in point estimates of maternal preferences when considering the up-to-date variable provides indirect evidence that time preferences indeed enter the preference proxies differently.

This interpretation is supported by the role of maternal preferences in the demand for immunization. Considering vaccination up-to-date as dependent variable we find no significant impact of maternal preferences when using the proxies $PREGNANCY$, $CURRENT$, or $PRESENCE$. By contrast, the fact that the mother smoked during the nursing period, $NURSING = 1$, significantly reduces the probability of the child having an up-to-date vaccination status by 3.4 percentage points. There are dramatic differences across gender. For girls maternal preferences are not significantly associated with the demand for immunization, that is, the effect we found in the full sample is entirely driven by boys. Indeed, when the mother smoked during the nursing period the probability of sons having an up-to-date vaccination record is reduced by 7.6 percentage points. The pattern is similar but less pronounced when considering the vaccination rate instead of the up-to-date status.

2.5.3 Sample selection based on age

In the descriptive analysis above we found that the demand for screening exams dramatically drops once screening J1 is due. More than 90 percent of children have up-to-date screening records when the latest scheduled exam was U9 or earlier. Looking at the oldest children in our sample where exam J1 should have been demanded, less than 40 percent actually have. To assess the robustness of our main results we exclude all children that are or were eligible for J1, that is, we exclude all children aged 169 months (14 years) and above from our analysis sample.

We find that the results based on the smaller sample are generally weaker, that is, the marginal effects of smoking during pregnancy on the demand for preventive care are smaller in absolute terms but, for the most part, still statistically significant (see Table 2.12 in the Appendix). This suggests that maternal preferences have a particularly strong effect in older children. Recall that all vaccinations considered here and screening exams (U1 through U9; J1) are covered by all German health insurers. Thus, parents do not face any financial burden from demanding these preventive services. As children get older, however, the gap between two subsequent examinations increases. There are as much as 9 years between exams U9 and J1. Parents may lose track of the screening schedule, in particular for J1, unless they are very risk averse, future oriented or altruistic. In other words, maternal preferences play a larger role for older children.

2.6 Extensions

In this section we briefly investigate effect heterogeneity regarding the perhaps most important dimensions when it comes to smoking behavior and health status or, more precisely, maternal preferences and the demand for preventive child care: socio-economic status and migration status.[24]

2.6.1 Socio-Economic Status

There is an extensive economic literature on the income gradient or socio-economic status (SES) gradient in child health (see, e.g., Case et al., 2002). The literature finds that disadvantaged children are born in worse health than advantaged children. As this gap is rather persistent – and in some countries even increases over the life-cycle – one wonders whether our proposed channel is particularly active in disadvantaged groups.

Table 2.6 breaks down the association between smoking during pregnancy and the demand for prevention by SES groups. Considering the full sample we find no association between maternal preferences and the demand for vaccination for any SES group. It is irrelevant whether we measure the demand for vaccination using the indicator for an up-to-date vaccination record or the vaccination rate. Stratification by gender offers a richer picture. For boys the association

[24]Note that we interact the socio-economic status indicators or the migration indicator with our preference proxy $PREGNANCY$. As was argued by Ai and Norton (2003) interaction terms in non-linear models only measure the interaction effect of the respective variables over and above the interaction that is implied by the non-linearity of the econometric approach. Stratification by socio-economic status and gender or migration status and gender, however, is not feasible as the low number of observations per cell prevents us from obtaining the maximum likelihood estimates in some instances.

Table 2.6: Maternal preferences and child prevention: socio-economic status

		full sample		boys		girls	
V_{UTD}	low	−0.0013	(0.0190)	−0.0507*	(0.0283)	0.0541*	(0.0317)
	medium	−0.0077	(0.0173)	−0.0248	(0.0245)	0.0050	(0.0264)
	high	−0.0313	(0.0315)	−0.0320	(0.0429)	−0.0305	(0.0490)
S_{UTD}	low	−0.0332**	(0.0145)	−0.0365*	(0.0207)	−0.0323	(0.0205)
	medium	−0.0405***	(0.0128)	−0.0619***	(0.0168)	−0.0179	(0.0177)
	high	−0.0178	(0.0246)	−0.0211	(0.0299)	−0.0132	(0.0365)
ρ		0.1749***	(0.0197)	0.1599***	(0.0284)	0.1977***	(0.0253)
V_{RATE}	low	−0.0155	(0.0118)	−0.0441**	(0.0171)	0.0163	(0.0200)
	medium	−0.0054	(0.0108)	−0.0104	(0.0147)	−0.0031	(0.0157)
	high	−0.0263	(0.0186)	−0.0229	(0.0242)	−0.0319	(0.0302)
S_{RATE}	low	−0.0157**	(0.0070)	−0.0156	(0.0104)	−0.0190*	(0.0099)
	medium	−0.0220***	(0.0062)	−0.0307***	(0.0085)	−0.0120	(0.0082)
	high	−0.0126	(0.0109)	−0.0195	(0.0135)	−0.0057	(0.0178)
ρ		0.1931***	(0.0143)	0.1746***	(0.0204)	0.2154***	(0.0207)
N		11,826		5,994		5,832	

Notes: Average marginal effects of $PREGNANCY \times$SES-group. Clustered standard errors in parentheses. All regressions include the full set of controls. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

between maternal preferences and the demand for vaccinations is only significant in the low SES group. Note that the effect is only significant at the 10 percent level and, additionally, that the estimated marginal effects are statistically indistinguishable. Thus, our result only offers weak evidence for a socio-economic gradient in the preference channel. Considering a uniform effect between maternal preferences and immunization across socio-economic groups above, we found no significant effects for girls (see Table 2.4). We here find a positive association in the low SES group and no effect in the other two groups – a surprising result that is in conflict with the predictions of our theoretical model. But again, the effects across SES groups are statistically indistinguishable. When considering the vaccination rate this surprising result vanishes while the (weak) evidence of a SES gradient in the association between maternal preferences and the demand for vaccinations remains.

No matter how we measure the demand for screening examinations the association between maternal preferences and prevention is only statistically significant in low or medium SES groups. The point estimates tend to be larger for boys than for girls. For both genders the point estimates of the marginal effects do not statistically differ across SES groups so that our results, at best, only offer weak evidence for a SES gradient in the maternal preference channel.

2.6.2 Migration status

About 10 percent of families in our analysis sample have a migration background. Table 2.7 breaks down the association between maternal preferences and the demand for prevention by migration status and a very clear picture emerges. In none of the regressions maternal preferences of families with a migration background are significantly associated with the demand for prevention. Due to the small number of observations standard errors are relatively large so that we have somewhat imprecise zeros. Note that the signs of all marginal effects are positive rather than negative as in our main specification shown in Table 2.4. An immediate consequence is that the effects for families without a migration background are larger than in the main specification. Qualitatively the results remain intact, that is, maternal preferences only matter in the demand for vaccination for boys but not girls. For screening exams there is a significantly negative association and, as before, the effects for boys are about twice as large than those for girls.[25]

Table 2.7: Maternal preferences and child prevention: migration background

		full sample		boys		girls	
V_{UTD}	$MIG = 1$	0.0295	(0.0358)	0.0019	(0.0517)	0.0725	(0.0638)
	$MIG = 0$	-0.0137	(0.0129)	-0.0402^{**}	(0.0188)	0.0107	(0.0208)
S_{UTD}	$MIG = 1$	0.0331	(0.0234)	0.0578^{*}	(0.0318)	0.0144	(0.0352)
	$MIG = 0$	-0.0450^{***}	(0.0104)	-0.0623^{***}	(0.0145)	-0.0288^{**}	(0.0129)
ρ		0.1742^{***}	(0.0197)	0.1578^{***}	(0.0283)	0.1967^{***}	(0.0253)
V_{RATE}	$MIG = 1$	0.0167	(0.0215)	0.0055	(0.0292)	0.0367	(0.0380)
	$MIG = 0$	-0.0159^{**}	(0.0080)	-0.0288^{***}	(0.0110)	-0.0049	(0.0127)
S_{RATE}	$MIG = 1$	0.0173	(0.0124)	0.0227	(0.0177)	0.0127	(0.0159)
	$MIG = 0$	-0.0242^{***}	(0.0052)	-0.0309^{***}	(0.0071)	-0.0186^{***}	(0.0067)
ρ		0.1922^{***}	(0.0142)	0.1728^{***}	(0.0203)	0.2142^{***}	(0.0207)
N		11,826		5,994		5,832	

Notes: Average marginal effects of $PREGNANCY \times MIG$. Clustered standard errors in parentheses. All regressions include the full set of controls. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

2.7 Conclusion

There is a large body of evidence showing that health in early childhood has long-lasting effects. Poor health in early childhood is, for instance, associated with low educational achievement and poor health in adulthood. The mechanisms through which poor child health translates into poor

[25] It is important to note that the demand for child prevention is substantially lower in families with a migration background. The vaccination rate (74%) and the screening rate (79%) are 4 percentage points and 15 percentage points, respectively, smaller in families with migration background than in families without such a background.

adult health are not well understood. In this article we suggest that maternal preferences – as proxied by adverse health behaviors of the mother – not only explain poor health at birth or in early childhood but also the low demand for preventive child care and with it poor health in adulthood.

In our analysis we concentrated on the relationship between smoking during pregnancy and the demand for vaccinations and screening exams. We found sons born to mothers who smoked during pregnancy have a significantly lower probability of having up-to-date vaccination records as compared to sons of mothers who abstained from smoking during pregnancy. There is no such pattern for daughters. The demand for screening exams is significantly lower for both genders when the mother smoked during pregnancy. The effects, however, were found to be twice as large for boys than for girls. This pattern is remarkably robust. It holds also when using alternative measures for the demand for prevention, namely, the vaccination rate and the screening rate, and for alternative proxies for maternal preferences, that is, smoking during the nursing period, current smoking status of the mother, and whether someone in the household smokes in the presence of the child. We found weak evidence that our suggested mechanism is particularly active in boys living in households with low or medium socio-economic status. Finally, we found that our results only obtain in families without a migration background. There is no association between maternal preferences and the demand for child prevention in families with a migration background.

Our results contribute to the understanding why health differences observed in early childhood are rather persistent over the life cycle – maternal preferences not only affect child health at birth and early childhood but via the demand for preventive health care also health of their children in adolescence and adulthood. Public policies should, thus, break the link between maternal preferences and child prevention. The most drastic measure would be to make vaccinations and screening exams mandatory or, in the latter case, enforce the mandate.

2.8 Appendix

Table 2.8: Content and age brackets of screening examinations

	Period	Measures
U1	after birth	Check skin color, respiration, cardiac rhythm, and reflexes
U2	3rd to 10th day of life	Examine internal and sensory organs, child's metabolism and hearing
U3	4th to 5th week of life	Check size, weight, nutritional state; test hip joints, eye reaction and hearing
U4	3rd to 4th month of life	Examine kinesic behavior, seeing, hearing, growth; check vaccination status
U5	6th to 7th month of life	Check physical development (e.g., independent turning), teeth and vaccination status
U6	10th to 12th month of life	Check physical development (e.g., crawling, first steps), sexual plus sensory organs, speech development; check vaccination status
U7	21st to 24th month of life	Test physical and mental development (e.g., running, fine motor skills, body control, speaking); check vaccination status
U8	43rd to 48th month of life	Test physical dexterity, hearing, seeing, speaking development; check vaccination status
U9	within 6th year of life	Test physical and mental development, correct movement, hearing, seeing, speaking; check vaccination status
J1	within 15th year of life	Examine posture anomalies, health behavior (e.g., smoking, alcohol consumption), motoric skills, sexual behavior, specific problems during puberty; vaccination status review (e.g., diphtheria, tetanus, polio)

Notes: Table shows all well-child visits fully covered by mandatory health insurance. U7a, U10, U11, J2 are not listed since not being part of the questionnaire. U1 and U2 are excluded from the analysis. Both are conducted in the first days after birth, thus maternal preferences can hardly have any effect. For further information we recommend https://www.g-ba.de/informationen/richtlinien/15/, https://www.g-ba.de/informationen/richtlinien/14/ or www.kindergesundheit-info.de (accessed on May 02, 2018).

Table 2.9: Explanation of variables

Dependent variables	
V_{UTD}	= 1 if vaccination status is up-to-date, 0 else
V_{RATE}	vaccination rate
S_{UTD}	= 1 if screening status is up-to-date, 0 else
S_{RATE}	screening rate
Maternal preferences	
PREGNANCY	= 1 if mother smoked during pregnancy, 0 else
NURSING	= 1 if mother smoked during nursing period, 0 else
CURRENT	= 1 if mother is a current smoker, 0 else
PRESENCE	= 1 if so. in the household smokes while child is present, 0 else
Control variables	
female	= 1 if female, 0 else
age[a, b]	age of child (in months of life)
Tanner	four categories of physical development in children
postpregprob	= 1 if specific health condition reported within first weeks after birth, 0 else
homecare	= 1 if child care performed by family during preschool age, 0 else
PKV	= 1 if insured in the private health care system, 0 else
MIG	= 1 if child has migration background, 0 else
EastGermany	= 1 if East German household, 0 else
urban	= 1 if urban place of residence, 0 else
oldersibshh	number of older siblings in the household
youngersibshh	number of younger siblings in the household
sameagesibshh	number of siblings of the same age in the household
income[c]	seven categories for monthly net household income ranging from 0 to 4000+ Euros
msingle	= 1 if single mother, 0 else
page[a, d]	parent's age (in years)
education[d]	seven categories for educational achievement (degrees)
unemp[d]	= 1 if currently unemployed, 0 else
pleave[d]	= 1 if on parental leave, 0 else
parttime[d]	= 1 if employed part time, 0 else
fulltime[d]	= 1 if employed full time, 0 else
bluecollar[d]	= 1 if blue-collar worker, 0 else
selfemp[d]	= 1 if self-employed, 0 else
whitecollar[d]	= 1 if white-collar worker, 0 else
civil[d]	= 1 if civil servant, 0 else
homemaker[d]	= 1 if homemaker, 0 else

[a] The squared age term is included as additional control variable.
[b] For the descriptive statistics, the variable age is represented in years.
[c] Income brackets (in Euros): 0-499, 500-999, 1000-1499, 1500-1999, 2000-2999, 3000-3999, and 4000+. For the descriptive statistics, the variable income was assigned the middle of the income bracket except for the highest bracket where we set household income equal to 4000.
[d] The set of control variables is the same for mothers and fathers.

Table 2.10: Summary statistics

Variable	N	Mean	Std. Dev.	Min	Max
Dependent variables					
V_{UTD}	11,826	0.554	0.497	0	1
V_{RATE}	11,826	0.779	0.298	0	1
S_{UTD}	11,826	0.833	0.373	0	1
S_{RATE}	11,826	0.923	0.203	0	1
Maternal preferences					
PREGNANCY	11,826	0.143	0.352	0	1
NURSING	11,826	0.071	0.257	0	1
CURRENT	11,826	0.275	0.447	0	1
PRESENCE	11,826	0.127	0.333	0	1
Control variables[a]					
female	11,826	0.493	0.500	0	1
age	11,826	8.082	5.047	0	17
MIG	11,826	0.097	0.297	0	1
EastGermany	11,826	0.334	0.472	0	1
income	11,826	2514.291	906.348	250	4000
SESlow[b]	11,826	0.213	0.409	0	1
SESmedium[b]	11,826	0.493	0.500	0	1
SEShigh[b]	11,826	0.295	0.456	0	1
PKV	11,826	0.132	0.336	0	1

[a] A selection of control variables is presented. The summary statistics on the full set of control variables is available upon request.
[b] Indicator variables for low, medium and high socio-economic status (SES) were only used for stratification purposes.

Table 2.11: Maternal preferences and child prevention: separate regressions

PREGNANCY	all	boys	girls
V_{UTD}	-0.0092	-0.0353^{**}	0.0167
	(0.0124)	(0.0176)	(0.0206)
S_{UTD}	-0.0348^{***}	-0.0464^{***}	-0.0233^{*}
	(0.0100)	(0.0134)	(0.0125)
V_{RATE}	-0.0121	-0.0245^{**}	-0.0004
	(0.0078)	(0.0105)	(0.0124)
S_{RATE}	-0.0185^{***}	-0.0234^{***}	-0.0144^{**}
	(0.0050)	(0.0066)	(0.0064)
N	11,826	5,994	5,832

Notes: Average marginal effects of $PREGNANCY$. Clustered standard errors in parentheses. All regressions include the full set of controls.
$^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table 2.12: Maternal preferences and child prevention: age ≤ 168 months

PREGNANCY	all	boys	girls
V_{UTD}	-0.0093	-0.0305	0.0098
	(0.0136)	(0.0197)	(0.0216)
S_{UTD}	-0.0252^{***}	-0.0321^{**}	-0.0178
	(0.0093)	(0.0126)	(0.0123)
ρ	0.1896^{***}	0.1701^{***}	0.2168^{***}
	(0.0252)	(0.0325)	(0.0357)
V_{RATE}	-0.0125	-0.0210^{*}	-0.0056
	(0.0087)	(0.0120)	(0.0134)
S_{RATE}	-0.0153^{***}	-0.0190^{***}	-0.0118^{**}
	(0.0044)	(0.0059)	(0.0057)
ρ	0.2173^{***}	0.1930^{***}	0.2483^{***}
	(0.0177)	(0.0242)	(0.0250)
N	9,633	4,902	4,731

Notes: Average marginal effects of $PREGNANCY$. Clustered standard errors in parentheses. All regressions include the full set of controls. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Chapter 3

Parental time discounting and child's smoking behavior

3.1 Introduction

Catchy phrases such as "The apple doesn't fall far from the tree." or "Like father, like son." are commonly used in the analysis of family characteristics. Fundamental attitudes and behavior patterns are likely to be passed on from parents to their children. Besides the well-known biological transmission of parental genetics, parents are usually the primary caregivers for their children and act as role models. Bisin and Verdier (2000, 2001), for instance, argue that mothers and fathers exert vertical socialization efforts through which the child adopts parental traits. After birth, a child's preference structure may seem to be only loosely framed. However, parents are supposed to be the child's first teacher. By doing so, parental characteristics are likely to be picked up by the child. The effects of such influences may even last for a lifetime. This may explain why family patterns and habits often persist over multiple generations.[1]

Over the last decade, a growing number of studies have provided some empirical evidence on preference and trait transmission. Dohmen et al. (2012) show that risk and trust attitudes are passed on from one generation to the next.[2] Furthermore, significant correlations exist with regard to intertemporal choice. Hence, parental time preferences are positively mirrored in the offspring's preference structure (e.g., Brown and van der Pol, 2015; Gauly, 2017). Instead of using direct survey measures such as self-assessed patience or impulsivity, some studies focus on saving decisions as a proxy for a person's time preference rate (Knowles and Postlewaite, 2005; Webley and Nyhus, 2006). Relying on pension participation as an approximation of

[1] See Section 2 of Darden and Gilleskie (2016) for a summary of the basic mechanisms of the intergenerational transmission of (smoking) behavior.

[2] Amongst others, additional evidence on the intergenerational transmission of risk is provided by Arrondel (2013), De Paola (2013) and Necker and Voskort (2014).

time preference, an adult child's pension participation is positively associated with the father's pension participation (Gouskova et al., 2010). Conducting an experiment, Kosse and Pfeiffer (2012, 2013) show that especially the mother's short-run patience is significantly related to the preschool child's ability to delay gratification.

Some authors try to capture the transmission of preferences across generations by investigating preferences for distinct activities. For instance, similarities are identified for a number of leisure time activities like doing sports, watching TV, going to the cinema or theater, food consumption, or socializing (Volland, 2013). The transmission of adverse health behaviors such as smoking is no exception. In the context of tobacco consumption, substantial evidence is provided for the strong relationship between parental smoking habits and the smoking status of the offspring.[3,4] Children living in families with at least one parent who smokes are at increased risk of becoming regular smokers themselves. The effect gets even stronger when both parents smoke (e.g., Bantle and Haisken-DeNew, 2002). Taking Germany as an example, Göhlmann et al. (2010) explicitly focus on smoking initiation. Using a discrete time hazard model, the results indicate that parental smoking significantly increases the probability that the child starts to smoke. Loureiro et al. (2010) go one step further. They show that the well-established association mentioned above is indeed causal. To identify this causal effect, they use an instrumental variable approach in order to overcome the potential endogeneity of parental smoking. Indicators of the socioeconomic status of the children's grandparents are used as instruments. The results provide further evidence for the transmission of smoking and highlight the importance of both mother and father. Whereas daughters are primarily influenced by their mothers, father's smoking behavior is more relevant for sons. While the vast majority of the corresponding literature focuses on the transfer of smoking across two generations, Vandewater et al. (2014) analyze the transmission link across multiple generations. In fact, they show that smoking behavior is transmitted from the grandparents to their grandchildren. Thus, the parent generation acts as a mediating factor. However, by identifying a transmission link across three generations, they also validate previous findings regarding the analysis of two generations.

Another important strand of the literature has its focus on the direct association between individual preferences and the person's health behaviors and outcomes. In general, individuals can improve health by positively investing in their health capital (Grossman, 1972). Here, by

[3]See, for example, Melchior et al. (2010), Chassin et al. (2008), Powell and Chaloupka (2005), Shenassa et al. (2003), Bantle and Haisken-DeNew (2002), Wickrama et al. (1999).

[4]The intergenerational transmission of risky health behaviors is not restricted to smoking. For instance, Schmidt and Tauchmann (2011) show that parental drinking has a significant influence on child's alcohol consumption.

contrast, the consumption of cigarettes or other tobacco products represents a highly unfavorable health investment. Instead of increasing the health stock, smoking deteriorates health gradually. Moreover, smoking behavior involves intertemporal decision-making. Hence, a person's time discounting is important. A trade-off has to be made between a sooner, smaller reward (pleasure of smoking a cigarette) and a larger, later reward (good health). Many empirical studies show that time preference and impulsivity are significantly correlated with smoking behavior.[5] In the *intra*personal context, individuals with lower discount factors smoke more than others. For instance, smokers discount future outcomes more steeply than non-smokers (e.g., Friedel et al., 2014). Beyond that, discounting also influences smoking cessation.[6] More specifically, quitting smoking involves both short-term costs like suffering from cigarette cravings as well as long-term benefits such as improvements in lung function. Those who exhibit relatively high levels of future orientation are more likely to stop smoking successfully and keep abstaining from it in the future. Consequently, a high discount rate impairs a person's attempt to forgo tobacco consumption.

Inevitably, this raises the question whether time discounting is also relevant in the *inter*personal context of smoking. Thus, the purpose of this chapter is to analyze the intergenerational transmission of smoking in more detail by simultaneously considering the role of time discounting of the child and the parents. In particular, we explore the link between parental discounting behavior and child's probability of being a smoker.

To the best of our knowledge, there has been only one empirical study examining the role of parental time preference in the intergenerational transmission of smoking. Brown and van der Pol (2014) rely on data from the Household Income Labour Dynamics of Australia (HILDA). They focus on mothers and their young adult children aged between 16 and 25 years.[7] Five waves (2002, 2003, 2004, 2006 and 2008) are selected which include information on both smoking indicators and time preference. A question on the financial planning horizon is used as a proxy for (long-term) time preference. A dummy variable "longer planning horizon" is created. It equals one if time periods of one year or more are most important to the respondent and zero otherwise. Control variables for basic offspring and household characteristics are also taken into account. Compiling an unbalanced panel, the final sample consists of 1,901 mothers and 3,167 children. Estimating a pooled probit model, they do not find any direct effect of maternal

[5] See, for example, Kang and Ikeda (2014), Ida (2014), Scharff and Viscusi (2011), Harrison et al. (2010), Ida and Goto (2009b), Khwaja et al. (2007), Reynolds et al. (2004), Ohmura et al. (2005), Baker et al. (2003), Odum et al. (2002), Mitchell (1999), and Bickel et al. (1999).

[6] See Adams (2009), Goto et al. (2009), and Ida and Goto (2009b).

[7] The sample only consists of individuals that reside with their mothers.

time preference on young adult smoking. After creating interaction terms of maternal time preference and her smoking behavior, they find significant indirect effects. Hence, daughters (sons) of mothers who smoke and have a shorter planning horizon are 7% (6%) more likely to smoke than daughters (sons) of mothers who smoke and have a longer planning horizon.

This chapter contributes to the previous literature in several ways. First, we disentangle different aspects of intertemporal discounting. We explicitly distinguish between two dimensions, self-control and time preference. For the sake of illustration, both characteristics of time discounting can best be represented by a quasi-hyperbolic discounted utility function U with $U(x_0, ..., x_t) = u_0 + \beta \sum_{t=1}^{T} \delta^t u(x_t)$. It assumes that an individual places higher weight on present payoffs relative to payoffs in the future. Hence, β corresponds to present-biased preferences ($\beta < 1$), whereas δ represents the long-run discount factor (see, e.g., Laibson, 1997). The relevance of the β-δ-framework has been validated by neuroeconomics. Relying on findings from McClure et al. (2004, 2007) and Tanaka et al. (2004), the parameter β is associated with the limbic brain system which has its focus on immediate outcomes and instant rewards. In contrast, the δ-component is strongly linked to the lateral prefrontal and parietal brain areas which are responsible for the planning and making of far-sighted decisions.[8] Throughout this chapter, we relate the short-run and long-run components of this convenient and fairly realistic approach to impulsivity and patience, respectively. Whereas impulsivity primarily refers to fundamental self-control abilities such as the ability to delay instant gratification, a person's general patience is linked with today's decisions which are followed by consequences in a more distant future (see, e.g., Peretti-Watel et al., 2013).[9]

Second, when it comes to the elicitation of preferences, measurement is not straightforward. In particular, dealing with survey data based on individual questionnaires, it is a challenge to identify high quality measures of economic preferences. We rely on a direct survey measure of a person's general patience. In addition, it is validated with an incentivized experiment (Vischer et al., 2013). Within the scope of this experiment, the question regarding impulsivity is validated indirectly. Moreover, it forms part of common impulsivity scales such as the famous Barratt Impulsiveness Scale (BIS) (Barratt, 1959). Hence, patience and impulsivity represent meaningful proxies for both components of intertemporal decision-making.

Third, we control for other confounding preferences that are related to discounting decisions

[8] See Kalenscher and Pennartz (2008) for an extensive review.

[9] Except for those who try it for the first time, smoking a cigarette generates immediate pleasure in the short run. However, current smokers hazard the adverse health consequences of regular tobacco consumption that may occur later in life (long-run outlook).

and adverse health behavior. Above all, we argue that personal risk attitudes may be another influencing factor. In fact, time and risk preferences measure different economic aspects but are highly intertwined when making intertemporal decisions under uncertainty (Andreoni and Sprenger, 2012). Whereas the present is known, the future is apparently risky. As already mentioned above, tobacco consumption is a classic example of an intertemporal trade-off. But, in addition, it is obvious that smoking puts a smoker's health at considerable risk. For instance, tobacco consumption substantially increases the probability of suffering lung cancer later in life. Ida and Goto (2009a,b) show that the likelihood of smoking is associated with both a higher time preference rate (lower level of patience) and a lower degree of risk aversion. Thus, taking individual risk attitudes into account is necessary to state more precisely the influence of the time discounting parameters in the transmission process of smoking. Omitting risk preferences is likely to bias the effects of (parental) patience and/or impulsivity upwards.[10]

Forth, we analyze the impact of both mother and father. This allows us to investigate gender-specific differences. Apart from that, merely focusing on one parent might neglect the potential influence of the other parent. This may result in a biased estimation of the influence of the parent who enters the analysis, regardless of whether it is the mother or the father. For example, excluding the father would be highly questionable. Although the overall prevalence of smoking has declined considerably over the last decades, around one in four Germans over 15 years old is a tobacco smoker (24.5%). The share of occasional and regular male smokers is still higher than the share of female smokers. According to the 2013 Census data, 20.3% of the female and 29.0% of the male population in Germany smoke (Federal Statistical Office (Destatis), 2017).[11]

Fifth, we study the role of parental time discounting on child's smoking behavior while analyzing the influence of possible (health) mechanisms. We argue that parental impulsivity and patience are likely to result in certain behavior patterns of the parents which for their part could affect a child's (health) behavior. According to Baron and Kenny (1986), we suppose the presence of multiple mediating factors. Thus, possible mediators of parental time discounting on the smoking status of the child are taken into account. Primarily, we focus on relevant health behaviors of the parents that may be or may have been influenced by their time discounting behavior. For instance, the smoking status of the parents is likely to be affected by their own

[10]Despite availability, Brown and van der Pol (2014) did not add information about other economic preferences such as risk preferences to their analysis.

[11]For more information, please visit `www.gbe-bund.de`. The homepage of the Information System of the Federal Health Monitoring (*Gesundheitsberichterstattung des Bundes*) offers abundant health data of the German population.

attitudes towards intertemporal choice. Controlling for the smoking status is expected to have a direct positive effect on our dependent variable. Moreover, if the observed parental health behaviors represent true mediating factors, we would expect a considerable reduction in size of the coefficient estimates of our main discounting variables. Hence, we do not only show the *raw* intergenerational transmission of smoking behavior but also highlight the potential mechanism(s) of parental time discounting on child's smoking status. Unfortunately, our analysis of possible mediating influences is limited. Although we can control for individual heterogeneity and parental attitudes towards health, we are not able to properly observe the influence of other potentially relevant factors with the data available. For example, we could think of the role of mothers' and fathers' parenting style as well as parental engagement in health promotion and education. Communication about (future) health risks and consequences of tobacco consumption may be a promising and effective determinant of child's decision to smoke or not to smoke.[12] Similarly, the potential impact of peer groups, especially at young age, cannot be investigated properly. At the end of this chapter, we will discuss these and some other limitations of our paper in more detail.

Sixth, we disentangle three different types of parental smoking habits. In principle, we differentiate between current smokers, ex-smokers and non-smokers. Thus, the smoking history of the parents is taken into account. It allows us to consider the importance of parental role modeling when the offspring was younger and prone to start smoking. This is important because it can be reasonably assumed that intergenerational transmission has already taken place at earlier stages in life (e.g., in childhood or adolescence). Finally, as an extension of the analysis of the extensive margin of smoking, we further address the role of parental impulsivity and patience on the intensive margin of tobacco consumption. Conditional on being a smoker, we examine how the number of cigarettes smoked per day gets affected by the parameters of intertemporal decision-making of the parents.

In line with the literature, we show that children who are more impulsive and less patient have a higher likelihood of being a smoker than more future-oriented individuals. But, most importantly, our results show significant direct effects of mother's as well as father's time preference. Overall, a one standard deviation increase in the level of parental patience reduces the child's probability of smoking by 6-7%. A one standard deviation increase in father's impulsivity reduces the smoking probability by roughly 6%. The coefficient of maternal impulsivity is not statistically significant. Controlling for parental smoking status, we further confirm the positive

[12]For instance, Kucher et al. (2014) investigate the role of family communication referring to another health risk, namely weight misperception.

transmission of smoking habits from parents to their child through role modeling. However, even after adding all parental health behaviors to our regression model, the relevant preference parameters remain robust. On the contrary, parental time discounting does not have a meaningful effect on child's smoking intensity. In addition to the positive correlation in intergenerational smoking habits, we conclude that parental time preferences also play a role for child's smoking decision. Hence, especially time preferences should be further considered by researchers as well as public health authorities when dealing with health behavior formation.

The remainder of this chapter is organized as follows. Section 3.2 describes the data. Section 3.3 introduces our empirical strategy. The main results are presented in Section 3.4. Finally, Section 3.5 concludes with a discussion of the main findings.

3.2 Data

3.2.1 Survey data and sample selection

We use data from the German Socio-Economic Panel (SOEP) which is an annual panel survey conducted since 1984. Each wave contains information on more than 22,000 individuals of the adult population in Germany living in approximatly 12,000 households. The SOEP provides both general household information as well as rich socio-economic data about each household member (Wagner et al., 2007). We exploit the panel structure of the survey and focus on data from waves 2006, 2008 and 2010. Whereas information on smoking is available every other year[13], the survey questions about personal impulsivity and patience are not an integral part of the individual questionnaire. Up to now, the 2008 wave is the only wave containing precise and comparable measures for the parents' and offspring's time discounting and their respective smoking behavior. As was demonstrated by Meier and Sprenger (2015), time preferences are rather stable over short periods of time. Assuming that this holds true for the German case, we use the 2008 time discounting parameters for the waves 2006 and 2010 allowing us to analyze three waves.[14]

We select parents and their biological children who are still living together with them in the same household and those children who have already moved out and live in their own household at the time of the interview.[15] We drop observations that have missing information on the variables used for the upcoming regression analysis. Our final sample contains information on

[13]The survey question on smoking behavior was introduced in 2002.

[14]See Chuang and Schechter (2015) for a literature review on the stability of time preferences. The authors also examine the stability of risk attitudes and other social preferences such as altruism.

[15]Children living with foster parents or in children's homes are not part of our sample.

2,456 children and their parents (n=1,739). Since we have panel data, the number of observations sums up to 5,817 individuals.

The children in our sample are 18 years of age or older at the time of the interview.[16] Despite the non-availability of appropriate information of younger individuals, this circumstance is neither a disadvantage nor problematic. At this advanced stage of life, we argue that the intergenerational transmission of personality traits as well as smoking has already taken place. In Germany, for instance, the average age of smoking initiation is around 17.8 years (Federal Statistical Office (Destatis), 2014).[17] This is not surprising because many young people try the first cigarette during adolescence, but not all of them convert into regular smokers afterwards. In general, more than 80% of adult smokers report having started regular tobacco consumption before they turned 18 years old (Kuntz and Lampert, 2013). Thus, it is fair to say that if public health authorities are willing and able to keep the youth tobacco free, the vast majority of these young people will most likely abstain from smoking during adulthood.

3.2.2 Smoking

Based on the question "Do you currently smoke, be it cigarettes, a pipe or cigars?", we generate a binary variable ("current smoker") to measure the smoking status of each individual. It equals one if the survey participant reports any level of tobacco consumption and equals zero otherwise. According to this specification, 29% of the children in our final sample smoke. On average, sons smoke more than daughters (32% vs. 26%). The share of mothers and fathers who currently smoke is 21% and 27%, respectively.

The parent-child smoking correlation is about 0.18 (p-value = 0.000) for both mothers and fathers. The correlation between maternal and paternal smoking status is also positive and highly significant ($\rho = 0.28$, p-value = 0.000). However, a limitation of the variable "current smoker" is that it ignores any parental smoking history. In our sample, the average age of the parents is about 55 years. According to the 2013 Census data, the overall smoking participation considerably decreases after reaching the age of 50 years (Federal Statistical Office (Destatis), 2017). Therefore, we apply a second dummy variable, namely "ex-smoker". This variable allows us to capture parental smoking behavior in the past. It takes the value 1 if the individual has smoked more than 100 cigarettes or other tobacco products in his/her life[18] and is a non-smoker

[16] Strictly speaking, only persons under the age of 14 can be considered as children. However, throughout the chapter, daughters and sons are commonly entitled as offspring or children, independent of their rather advanced age. 50% of our sample are not older than 25 years. 75% are not older than 31 years.

[17] According to the latest Surgeon General's Report, similar results are reported for the United States (U.S. Department of Health and Human Services, 2014).

[18] The exact wording of the question is as follows: "Have you ever smoked? In other words, have you smoked

throughout the sample period and 0 otherwise. According to this definition, 36% of the mothers are classified as "ex-smoker". The respective share of paternal ex-smokers sums up to 49%. Hence, we are able to differentiate between rigorous non-smokers and non-smokers that engaged in tobacco consumption in the past.

In order to further examine the association between parental time discounting and child's smoking behavior, we shed some light on the intensive margin of child smoking. We replace child's current smoking status as our main dependent variable with the smoking intensity measured by the number of cigarettes smoked per day. Current smokers were asked to report their daily average of cigarettes smoked in the last week. Conditional on being a smoker, the children in our sample smoke roughly 13 cigarettes on a daily basis. Females smoke 11.5 cigarettes, whereas male smokers have a mean cigarette consumption of almost 14 cigarettes.

3.2.3 Time discounting

The 2008 questionnaire contains two questions which enable us to elicit individual time discounting. In order to identify different dimensions of intertemporal decision-making, we explicitly distinguish between (short-run) self-control/present bias and (long-run) patience (see, e.g., Peretti-Watel et al., 2013). Maximum comparability is ensured because both parents and their children answer the exact same questions independently of each other. First, each respondent is asked to rate his or her personal level of patience on an 11-point scale. The wording of the question is as follows: "How would you describe yourself: Are you generally an impatient person, or someone who always shows great patience? Please tick a box on the scale, where the value 0 means: 'very impatient' and the value 10 means: 'very patient'. You can use the values in between to make your estimate." Hence, self-reported patience is used as a proxy for individual time preference. In our sample, the average patience level of the child is 5.89, whereas sons are slightly more patient than daughters (5.94 vs. 5.82). In general, mothers are more patient than fathers (6.37 vs. 6.00).

Additionally, the second question refers to a person's self-control abilities. The respondent is asked to specify his or her general level of impulsivity. The wording of the question is as follows: "How would you describe yourself: Do you generally think things over for a long time before acting – in other words, are you not impulsive at all? Or do you generally act without thinking things over for long time – in other words, are you very impulsive? Please tick a box on the scale, where the value 0 means: 'not at all impulsive' and the value 10 means: 'very impulsive'. You

more than 100 cigarettes or other tobacco products in your life?" We retrieve this retrospective information from wave 2012. Unfortunately, only a few former smokers have provided details about when exactly they quit smoking.

can use the values in between to make your estimate." According to the descriptive statistics, females are more impulsive than males (5.35 vs. 5.09). Overall, children show a mean level of impulsivity of about 5.21. In comparison to fathers, mothers are more impulsive (5.03 vs. 4.85).

The behavioral relevance of our time preference measure has been explicitly validated. Vischer et al. (2013) conducted an incentivized experiment with 977 participants forming a representative sub-sample of the adult population to the 2006 wave of the SOEP. Subjects were asked to indicate their preferences in a choice over a 12-month time horizon.[19] The results show that those who rate themselves as 'more impatient' in the survey in 2008 also exhibit a higher degree of impatience in the experiment in 2006. Hence, this simple and ultra-short survey measure of patience turns out to be a meaningful proxy for time preference. The findings remain robust even after controlling for impulsivity.[20] Indeed, this shows that the questions on general patience and impulsivity measure different parameters of intertemporal choice. Thus, a respondent's misinterpretation of the more future-oriented (long-term) aspects underlying the general question on patience can be ruled out. Moreover, the question on personal impulsiveness is a basic part of the most common scales used to measure this personality trait.[21] Therefore, we reasonably assume that the survey question eliciting impulsivity represents a true and rigorous measure of present bias/self-control.

The raw *intra*personal correlation of time preference and self-control is -0.17, -0.19 and -0.17 for the offspring, mother and father, respectively. Each correlation coefficient is highly significant (p-value = 0.000). Considering the *inter*generational correlation of these variables, the raw correlation in parent-child impulsivity is 0.11 (p-value = 0.000) for the mother and 0.14 (p-value = 0.000) for the father. The corresponding coefficients for patience are smaller in size: 0.06 (p-value = 0.001) for the mother and 0.09 (p-value = 0.000) for the father. In general, these findings are in line with the corresponding literature (see Gauly, 2017). Comparing children living in their own households with children still living together with their parents, the

[19] In the experiment, choice tables with the typical price list decision format were used. The participants had to indicate their preferences by choosing between an immediate (left column) or delayed payment (right column). The immediate payment was continuously fixed (€200). However, the delayed payment varied in each of the 20 choice situations and increased by 2.5 percentage points (compounded semi-annually) from row to row. Switching from left to right (and sticking to the delayed payment in all subsequent rows) indicates the bounds of the discount rate the respondent claims in order to wait an additional time period of 12 months for payout. Before the start of the experiment, the participants were informed that one of their choices would be randomly selected for payment. Using a second random device, one out of nine participants was actually paid by check according to the previous choice.

[20] In addition, controlling for personal risk attitudes does not affect the results either.

[21] Examples of common impulsivity scales are the Barratt Impulsiveness Scale: see, e.g., Barratt (1959), Patton et al. (1995), Stanford et al. (2009), Steinberg et al. (2013) and Coutlee et al. (2014); the Eysenck Impulsiveness Scale: see, e.g., Eysenck et al. (1985); the Dickman Impulsivity Inventory: see, e.g., Dickman (1990) and Boutwell and Beaver (2010); and the UPPS Impulsive Behavior Scale: see, e.g., Whiteside and Lynam (2001).

latter show slightly higher correlation coefficients. For instance, regarding the *inter*personal correlations of patience, we obtain 0.06 vs. 0.07 for mothers and 0.07 vs. 0.10 for fathers. The correlation coefficients are statistically significant at least at the 5% level.

3.2.4 Parental and offspring characteristics

The SOEP provides rich information on the socioeconomic status and other individual and household characteristics. We adjust for individual heterogeneity by adding a number of control variables for the children and their parents. Summary statistics for the children are shown in Table 3.1. Offspring characteristics include basic biological information about age and gender. Moreover, we add information about the migration background and generate a new variable that indicates if the child still lives together in the same household with at least one biological parent. We further consider the educational attainment (highest school degree achieved) and the log annual net household income as proxies for the socioeconomic status.[22] In Table 3.2, descriptive statistics for the parents' demographics are reported for mothers and fathers separately. We include variables such as parental age and migration background. In addition, the level of parental education serves as a proxy for the socioeconomic status of the family.

As already mentioned above, we analyze the influence of time discounting on smoking behavior, taking into account the importance of personal risk attitudes. Individual risk is highly correlated with impulsivity. For the offspring, mother and father, we find a significant correlation of about 0.40 (p-value = 0.000), respectively. Hence, risk preferences are included as additional control variables for both children and their parents.[23] However, it could be argued that the effects of parental risk and/or time discounting are (partly) confounded with the impact of other preferences. Thus, we additionally control for parental altruism. It is obvious that the altruistic attitudes of the parents are a key element of the social interactions within the family. The proxy variable for altruism equals one if a parent has indicated that it is very important to him/her to "be there for others" and zero otherwise. For instance, maternal altruism is significantly correlated with her patience ($\rho = 0.11$). The correlation between paternal altruism and patience is slightly lower ($\rho = 0.08$), but also highly significant (p-value = 0.000).[24]

To account for regional differences, we control for the 16 federal states (*Bundesländer*) in

[22]The annual net household income is lagged by one year. It corresponds to the household the child lives in.

[23]The original wording of the survey question to elicit personal risk attitudes is as follows: "How would you describe yourself: Are you generally willing to take risks, or do you try to avoid risks? Please tick a box on the scale, where the value 0 means: 'risk averse' and the value 10 means: 'fully prepared to take risks'. You can use the values in between to make your estimate." In line with the implementation of our time discounting variables, information on risk preference is retrieved from wave 2008 and also imputed to the years 2006 and 2010.

[24]Information on parental altruism is retrieved from wave 2008 and also imputed to the waves 2006 and 2010.

Germany. Indicator variables for each state account for potential regional confounders. Moreover, year dummies are used to control for time trends. They account for common trends in smoking behavior over time. Over the past decades, increased taxes on cigarettes and a growing health consciousness among the general population are supposed to be two important factors that have contributed to an overall reduction in tobacco consumption in Germany. By adding indicator variables for each federal state and year, we also control for the implementation or expansion of different anti-tobacco policies (e.g., smoking bans) that varied across states and/or over time.

In order to further investigate the impact of role modeling, we look at parental behavior patterns that could further represent mediating factors between parental time discounting and the smoking status of the child. For this purpose, we focus on a comprehensive set of parental health behaviors. On the one hand, we include adverse health behaviors such as parental smoking and alcohol consumption.[25] We use a dummy variable to measure alcohol consumption that equals one if the parent drinks any kind of alcohol (e.g., beer, wine, spirits or mixed drinks) on a regular basis and zero otherwise. On the other hand, we analyze two positive health investments. First, we use information on whether the parents live a healthy lifestyle or not. The variable equals one if the person follows a health-conscious diet "very much" or "much" and zero otherwise. Second, we generate another indicator variable that measures physical activity. It equals one if the individual takes part in active sports "daily" or "at least once a week" and zero otherwise.

[25] Please see Section 3.2.2 for the definition of our smoking variables.

Table 3.1: Summary statistics (Children)

	Children (N=5,817)				Daughters (N=2,788)				Sons (N=3,029)			
Variable	**Mean**	**S.D.**	**Min**	**Max**	**Mean**	**S.D.**	**Min**	**Max**	**Mean**	**S.D.**	**Min**	**Max**
Dependent variables												
Current smoker	0.291	0.454	0	1	0.263	0.44	0	1	0.316	0.465	0	1
Smoking consumption[1]	12.772	7.224	1	50	11.532	6.552	1	40	13.737	7.569	1	50
Time discounting and risk												
Impulsivity[2]	5.214	2.104	0	10	5.350	2.125	0	10	5.089	2076	0	10
Patience[2]	5.886	2.232	0	10	5.824	2236	0	10	5.942	2.227	0	10
Risk[2]	4.941	2.179	0	10	4.506	2.145	0	10	5.341	2.133	0	10
Control variables												
Female	0.479	0.5	0	1								
Age	27.735	7.622	18	60	27.310	7098	18	56	28.125	8.056	18	60
German	0.947	0.225	0	1	0.949	0.219	0	1	0.944	0.23	0	1
Living with parent(s)	0.506	0.5	0	1	0.437	0.496	0	1	0.570	0.495	0	1
Lower secondary school	0.162	0.368	0	1	0.120	0.325	0	1	0.200	0.4	0	1
Intermediate secondary school	0.322	0.467	0	1	0.341	0.474	0	1	0.304	0.46	0	1
Specialized upper secondary school	0.069	0.253	0	1	0.066	0.249	0	1	0.071	0.257	0	1
Upper secondary school	0.335	0.472	0	1	0.361	0.48	0	1	0.311	0.463	0	1
Other school degree	0.016	0.125	0	1	0.012	0.11	0	1	0.019	0.138	0	1
No school degree	0.011	0.106	0	1	0.010	0.1	0	1	0.013	0.111	0	1
Not yet finished	0.086	0.28	0	1	0.089	0.285	0	1	0.083	0.275	0	1
Household income[3] (log)	10.485	0.679	3.912	13.346	10.443	0.711	5.273	13.346	10.524	0.646	3.912	13.010

Notes: [1] Number of cigarettes smoked per day. Number of smokers=1,657 (Daughters=725, Sons=932); [2] Information from wave 2008; [3] Log annual household income lagged by one year.

Table 3.2: Summary statistics (Parents)

	Mother				Father			
Variable	**Mean**	**S.D.**	**Min**	**Max**	**Mean**	**S.D.**	**Min**	**Max**
Time discounting and risk								
Impulsivity[1]	5.032	2.149	0	10	4.850	2.121	0	10
Patience[1]	6.370	2.125	0	10	6.002	2.232	0	10
Risk[1]	3.840	2.142	0	10	4.661	2.252	0	10
Health behaviors								
Current smoker	0.207	0.405	0	1	0.274	0.446	0	1
Ex-smoker[2]	0.357	0.479	0	1	0.485	0.500	0	1
Regular alcohol	0.099	0.299	0	1	0.300	0.458	0	1
Healthy nutrition	0.603	0.489	0	1	0.410	0.492	0	1
Regular sport	0.414	0.493	0	1	0.332	0.471	0	1
Control variables								
Age	54.410	8.336	35	86	57.292	8.819	35	93
German	0.919	0.273	0	1	0.920	0.271	0	1
Altruism[1]	0.287	0.452	0	1	0.174	0.379	0	1
Lower secondary school	0.350	0.477	0	1	0.385	0.487	0	1
Intermediate secondary school	0.371	0.483	0	1	0.273	0.445	0	1
Specialized upper secondary school	0.026	0.159	0	1	0.036	0.187	0	1
Upper secondary school	0.134	0.340	0	1	0.187	0.39	0	1
Other school degree	0.076	0.266	0	1	0.087	0.282	0	1
No school degree	0.043	0.202	0	1	0.032	0.175	0	1
Household income[3] (log)	10.603	0.550	8.170	13.409	10.609	0.558	5.338	13.409

Notes: [1] Information from wave 2008; [2] Generated variable based on information from wave 2012; [3] Log annual household income lagged by one year; number of parents=1,739.

3.3 Empirical strategy

The empirical analysis is based on three main steps. First, we estimate the direct association between parental time discounting and child's smoking status using a linear probability model (LPM). In step 2, we additionally control for parental health behaviors. This allows us to examine if certain health behaviors of the parents (e.g., parental smoking status) act as mediating factors between their time discounting and the smoking participation of the offspring. In the third and last step, we use a different dependent variable. We replace the current smoking status of the child with the number of cigarettes smoked per day. This enables us to further analyze the intensive margin of child smoking.

3.3.1 Regression models

The regression equation for step 1 and 2 looks as follows:

$$S_{it} = \beta_0 + \beta_1 I_{it}^p + \beta_2 P_{it}^p + \beta_3 I_{it} + \beta_4 P_{it} + \beta_5 X_{it}^{'} + \lambda_t + \alpha_i + u_{it} \tag{3.1}$$

where i represents the child and t the year of observation. The superscript p indicates that the variables relate to the parents. S_{it} is equal to 1 if the child smokes and zero otherwise. I_{it}^p includes information on mother's and father's general level of impulsivity, whereas P_{it}^p covers their level of patience. I_{it} indicates child's impulsivity and P_{it} represents child's patience. $X_{it}^{'}$ includes additional offspring and parental characteristics (see Section 3.2.4). In step 1, the regression model is estimated without controls for parental health patterns. These variables are added to the regression in step 2. Moreover, we include year dummies (λ_t) in all our regressions. The child-specific effect is represented by α_i. u_{it} is the individual-specific error term. The LPM with random effects is implemented by generalized least squares (GLS).[26] All time discounting and risk preference variables were standardized before entering the Equation (3.1). Standard errors are clustered at the family level because our sample includes families with one or more children. The vectors of parameters β_1 and β_2 are of particular interest. They measure how parental self-control and time preference are related to the smoking status of the child, respectively.

Finally, we estimate a two-part model to investigate the association between parental time discounting and child's smoking intensity (step 3). This regression approach is widely used in the context of tobacco consumption (e.g., Kang and Ikeda, 2014). First, we estimate child's smoking participation as shown in Equation (3.1) with a probit model and obtain the average marginal

[26] Considering the well-known limitations of the LPM, we compare the results to a panel probit estimation in Section 3.4.3.

effects of our variables of interest. This estimation approach provides a built-in robustness check of the LPM. Second, the number of smoked cigarettes is estimated with a truncated regression. All non-smokers are dropped because their tobacco consumption equals zero.[27] We prefer the two-part model over the standard Tobit model because the former implements and estimates the decision to smoke and the decision about how many cigarettes to consume daily separately. This provides a higher degree of flexibility. For instance, the determinants of the probability of being a smoker and the determinants of smoking intensity do not necessarily have to show the same signs. Moreover, both parts of the model are not required to include the exact same set of variables on the right hand side of the regression equation. However, in the tobacco demand literature, the decision to smoke and the amount of cigarettes consumed can also be interpreted as a joint decision and not as two separate decisions. In this case, a standard Tobit model would be the appropriate choice from an econometric point of view (Pfeifer, 2012). In Section 3.4.3, we provide a statistical test that supports the use of the two-part model.

3.3.2 Specifications and mediation analysis

The focus of our empirical strategy is on the relationship between parental time discounting and child's smoking behavior. However, we are keen to explore if there are mechanisms through which our discounting variables might have an indirect impact on the dependent variable(s). The influence of parental smoking behavior is of special interest in this context. At first glance, the elimination of parental health behaviors from our baseline regression does not seem to be particularly convincing. Previous literature has shown that a positive transmission of smoking habits from parents to their offspring exists (e.g., Loureiro et al., 2010). However, including variables such as the smoking status or the educational level of the parents are likely to be outcomes of their own impulsivity and/or patience. This might cause a so-called bad control problem (Angrist and Pischke, 2009). Basically, the same argument can be applied to all variables that are associated with child's socioeconomic status (education and income). Even controlling for child's preferences (patience, impulsivity, risk) is not straightforward. Due to intergenerational transmission, we have to consider that they are influenced by their parents' preferences (Gauly, 2017).

Being aware of potential endogeneity concerns, we decide to run several specifications of our econometric model and gradually add more (problematic) controls. Starting off with the

[27] For both regression equations, we use Roodman's cmp command in Stata (Roodman, 2011). We continue to exploit the panel structure of our data and estimate random effects models. Moreover, it allows us to obtain clustered standard errors.

fully specified model would mask the role of existing mechanisms driving the relationship under investigation. Initially, we include parental preferences along with plausibly exogenous control variables such as age and child's gender. The regression model is extended by adding child's time discounting variables and the degree of personal risk aversion. In the following step, we additionally control for educational attainment of both parents and the child. Higher education is supposed to be a powerful determinant of positive investments in health capital. However, we reckon that a person's level of education may be endogenous in our context because it was probably affected by a person's time preference when visiting school. It is obvious that a person with relatively high future orientation will invest more in educational attainment and, by doing so, cumulates more years in school than someone who is more present-oriented. The latter is likely to leave school earlier. Hence, when we control for the levels of education of the child (and both parents), we also account for the underlying correlations between (parental) time discounting and (parental) educational attainment. But, even if low time preference has had a beneficial effect on the educational attainment, school education is finished for all individuals by the end of our analysis period. This should reduce the endogeneity concerns regarding the inclusion of education. The same considerations apply to the role of the offspring's personal income because it is likely to be a consequence of the educational level achieved. Therefore, it may also represent an indirect outcome of child's time discounting. However, when analyzing the link between parental time discounting and child smoking, our stepwise approach takes these econometric issues into account.

Considering the mediating effect of parental behavior patterns, time preference and/or impulsivity are likely to influence different health behaviors (e.g., consumption of tobacco products). Given our data, we select four potential health mechanisms through which parental impulsivity and patience might affect the smoking behavior of the child: parental smoking status (current smoker and ex-smoker), regular alcohol consumption, health-conscious nutrition, and regular physical activity (see Sections 3.2.2 and 3.2.4 for detailed information on the health behavior variables). Smoking and alcohol consumption are both examples of rather unhealthy lifestyles which represent negative health investments.[28] The commitment to healthy nutrition and engagement in regular physical activity act as proxies for beneficial investments in health capital. The attitudes towards eating a healthy diet is supposed to capture healthy eating habits and dietary behavior in general. Thus, a person that recognizes the benefits of a well-balanced diet is unlikely to (excessively) consume unhealthy products such as junk food or soft drinks. It can

[28]Smoking and alcohol consumption are closely related to each other. See McKee and Weinberger (2013) for further reading on alcohol and tobacco co-use.

be observed that, in general, more impulsive or less patient individuals are more likely to indulge in adverse health investments than people who are more concerned about the future health consequences of their behaviors. If living in good future health is personally important for current decision-making, such an individual will live a relatively healthy lifestyle. Self-control and future orientation are key components of beneficial investments in a person's (future) stock of health capital. For instance, a person who is future-oriented and aware of the adverse health effects of smoking will be less likely to smoke. Evidence on this well-known intrapersonal relationship between discounting behavior and smoking participation has already been given in Section 1.6.2.3 and in the introductory section of this chapter. Hence, in case of parental non-smokers, it is likely that the same pattern is transmitted to the offspring who will neither turn into a smoker (parental role model). The same argument can be applied to regular alcohol consumption and living a healthy lifestyle. Discount rates are positively associated with frequent alcohol consumption (see, e.g., Rossow, 2008). Moreover, relationships between obesity and high time preference rates or even hyperbolic discounting exist (see Komlos et al., 2004; Scharff, 2009).

More specifically, we are interested in how the coefficients of parental time discounting change after the inclusion of these designated mediating factors. Possible changes after the inclusion of parental smoking are of particular interest because it is supposed to be a major determinant of child smoking anyway. On the one hand, we could observe a (considerable) reduction or even elimination of the direct effects of parental impulsivity and/or patience. In this case, we would have identified a *true* mediator. Hence, we could infer that parental time discounting has an influence on child's health/smoking behavior (mainly) through one or several parental health mechanisms. On the other hand, even though parental health behaviors show significant effects, the coefficients of interest could remain robust. In this case, we would see no reason not to control for these variables. Thus, we would have identified a meaningful influence of parental health patterns on child's smoking status that does not alter our estimated time discounting effects (Baron and Kenny, 1986). Hence, our suspected control problem would turn out to be less *bad*.

3.4 Results

3.4.1 Parental time discounting and child's smoking participation

Results from Equation (3.1) are presented in Table 3.3.[29] Column (1) shows that father's patience is negatively associated with child's probability of being a smoker. The coefficient of maternal patience is weakly significant at the 10% level. After controlling for child's time discounting and risk preference in column (2), the negative effects of parental patience remain basically the same. The coefficients of child's preferences are all significant and show the expected signs. Father's impulsivity turns weakly significant when we add dummy variables for parental education in column (3). According to the F-test on joint significance of parental education, we can reject the null hypothesis at the 1% level (p-value = 0.005). In column (4), the incorporation of other potentially endogenous variables such as child's highest school degree and income has negligible effects on the previous findings. The test on the joint significance of child's education variables and personal income clearly rejects the null hypothesis (p-value = 0.000).[30] Since we consider the model in column (4) as the most comprehensive specification, we interpret these results in more detail.

Our results confirm the findings from previous studies regarding the association between individual time discounting and smoking (*intra*personal context). A one standard deviation increase in child's impulsivity increases the likelihood of smoking by 2.8 percentage points or 10% (according to the mean). A one standard deviation increase in patience is associated with an average decrease of 1.7 percentage points or roughly 6% in the probability of being a smoker. Hence, as expected, a person that exhibits more future orientation and acts less impulsively is significantly more likely to abstain from smoking than someone who is (very) impatient and impulsive. But more importantly, we find direct effects of parental time discounting on the offspring's probability of being a smoker. For mothers, the level of impulsivity lacks statistical

[29] To support the choice of a random effects model, we test for random effects. Since we deal with an unbalanced panel, we apply the modified Breusch-Pagan Lagrange multiplier (LM) test for random effects (Baltagi and Li, 1990). The null hypothesis is that variances across individuals are zero. Thus, there is no panel effect because no significant differences across individuals exist. We can reject the null hypothesis ($Prob > chi2 = 0.0000$). Hence, random effects are appropriate. Alternatively, we apply the one-sided test. This supports our findings from the two-sided test. This test is conducted for all regressions, if necessary and wherever appropriate. In each case, the random effects model is preferred over the pooled OLS regression. Unfortunately, we cannot test fixed effects versus random effects. Using a fixed effects model is not appropriate in our case because we rely on time discounting parameters that are assumed to be time-invariant over the observation period.

[30] An additional significance test for the equality of children's education dummies rejects the null hypothesis of equal coefficients (p-value = 0.000). For instance, we observe that individuals with a high school degree are significantly less likely to smoke than those who achieved a relatively modest level of education. Hence, this finding supports the extensive evidence on the education gradient in health and health-related behaviors (see, e.g., Conti et al., 2010).

significance, while patience has a preventive effect. A one standard deviation increase in maternal patience reduces the likelihood of smoking by 2.1 percentage points. This is a reduction of about 7%. For fathers, both components of time discounting are significant. A one unit increase in paternal impulsivity is associated with a negative impact of 1.8 percentage points. Regarding his long-term time preference, we also find a prevention effect. It is similar to the effect obtained for the mother. If his level of patience increases by one standard deviation, the likelihood of smoking decreases by 1.9 percentage points. Economically, both effects are substantial because they imply a reduction in smoking of approximately 6-7%, respectively.[31]

Our results suggest that especially parental future orientation is able to prevent the offspring from engaging in adverse health behaviors such as smoking (prevention effect). In contrast, the effect of father's impulsivity (negative sign) may not be that intuitive at first sight and, therefore, needs some additional remarks. In principle, it would be delusive to expect the same sign as in the *intra*personal context. The positive effect of impulsivity in the intrapersonal context does not automatically imply that we get similar results when we turn to the *inter*personal context. For instance, we could think of the role of parental impulsivity for social interactions within the family. In the family environment, there could be uncertainty about parental behavior which might explain why the coefficient of paternal impulsivity has a negative sign. We argue that decision-making and actions taken by impulsive parents are extremely difficult to anticipate for the offspring. Therefore, children of parents with relatively low self-control are likely to think twice before doing something (e.g., engaging in smoking) which might cause trouble at home. They want to avoid negative attention because following such a lifestyle is likely to provoke immediate but rather ambiguous reactions of the parent(s). We refer to this finding as the 'slap-effect'. However, the coefficient is only weakly significant. We conclude that particular caution is required when interpreting this effect.

[31] In order to check the robustness of our results, we re-estimate the model using dichotomous versions of the time and risk preference measures. Individuals are classified as being patient, impulsive and willing to take risks if their response values are greater than the median on the patience, impulsivity and risk scale, respectively. Alternatively, we do a mean split on each of these variables. These specifications yield qualitatively similar results. Moreover, we extend our analysis to four observation periods by adding wave 2012. Although we still get similar results, we do not want to impose an extra burden on the assumption of stable preferences.

Table 3.3: Parental time discounting and child's smoking participation

		Dep. var.: child's smoking status					
		(1)	(2)	(3)	(4)	(5) Daughters	(6) Sons
Mother	Impulsivity	0.005 (0.010)	0.000 (0.010)	0.001 (0.010)	0.001 (0.009)	0.014 (0.012)	-0.012 (0.013)
	Patience	-0.016* (0.010)	-0.017* (0.009)	-0.018** (0.009)	-0.021** (0.009)	-0.025** (0.012)	-0.022* (0.013)
Father	Impulsivity	-0.004 (0.010)	-0.011 (0.010)	-0.017* (0.010)	-0.018* (0.009)	-0.003 (0.013)	-0.031** (0.013)
	Patience	-0.019** (0.009)	-0.018** (0.009)	-0.018** (0.009)	-0.019** (0.009)	-0.011 (0.012)	-0.025** (0.012)
Child	Impulsivity		0.032*** (0.009)	0.033*** (0.009)	0.028*** (0.009)	0.041*** (0.012)	0.020 (0.013)
	Patience		-0.014* (0.008)	-0.015* (0.008)	-0.017** (0.008)	-0.017 (0.011)	-0.015 (0.012)
Parental education				✓	✓	✓	✓
Child education & income					✓	✓	✓
N		5,817	5,817	5,817	5,817	2,788	3,029
R^2		0.037	0.050	0.060	0.106	0.140	0.102

Notes: Random effects GLS regressions. Cluster-robust standard errors at the family level in parentheses. Impulsivity, patience and risk are measured in standard deviations. All specifications include wave and state dummies, control variables child (gender, age, age squared, migration status, home indicator, risk preference), and control variables parents (age, migration background, risk preference, altruism). Significance: *p<0.1, **p<0.05, ***p<0.01.

As discussed in Section 3.2.4, we want to make a brief comment on the impact of risk preferences. With regard to the influence of individual risk attitudes, we find a significant intrapersonal effect. Hence, a one standard deviation increase in child's risk attitude increases the likelihood of smoking by 2.2 percentage points (8%). However, the corresponding coefficients of the parents are not statistically significant (results not shown).[32]

In order to identify possible gender differences, we estimate Equation (3.1) separately for daughters and sons. Results are shown in columns (5) and (6) of Table 3.3. The overall effects of child's time discounting and risk preference are primarily driven by women.[33] For men, the point estimates are generally smaller in size and statistically insignificant but show the same signs. Regarding the influence of parental time discounting, we identify significant same-sex as well as cross-sex effects. A one standard deviation increase in mother's patience lowers the likelihood of smoking by 2.5 percentage points for daughters and by 2.2 percentage points for sons. Again, maternal impulsivity is not significant. The time discounting variables of the father influence the smoking behavior of male offspring only. A one standard deviation increase of paternal impulsivity (patience) reduces the likelihood of smoking for men by 3.1 (2.5) percentage points.

In addition to gender, Table 3.6 in the Appendix of this chapter replicates the regression from column (4) of Table 3.3 and stratifies by child's home. If the child lives together with the parents, mother's patience is associated with a significant decrease in child's smoking probability. A similar effect can be found with respect to children who no longer share the household with their parents. Interestingly, father's time discounting variables are only significant for those children who already live in their own household. Table 3.7 in the Appendix replicates the regression from column (4) of Table 3.3 and stratifies by different age groups. Parental patience as well as child's preferences show the expected signs and are significant across almost all specifications.

3.4.2 Role of parental health behaviors

Table 3.4 presents the estimation results after adding parental smoking behavior and other health variables to the regression model. Column (1) replicates the results from the model specification without health mediators. First, we focus on the impact of parental smoking behavior in column (2). In line with previous findings regarding the intergenerational transmission of smoking, we find a positive and highly significant relationship between parental smoking habits and child's smoking status. Furthermore, the economic significance is substantial. If the mother is a current

[32] As a robustness check, we replace the general risk measure with the willingness to take risks in the health domain from wave 2009. Our results remain qualitatively similar.

[33] The estimated coefficient of female patience is -0.017 and the t-value is about 1.55.

smoker, the likelihood of child smoking increases by more than 13 percentage points (or 45%). If the mother is a former smoker, the increase is about 5 percentage points (or 17%). With regard to father's smoking habits, the size of the coefficients as well as the level of significance are similar. However, in comparison to column (1), the coefficients of interest remain highly robust.

In column (3), we control for all parental health behaviors simultaneously. Again, our main findings remain largely unchanged. The impact of parental smoking is still highly significant. With the exception of father's healthy lifestyle, all other health variables are not statistically significant.[34] In comparison to the baseline regression, the overall changes in coefficients are negligible. With respect to mother's patience, however, it may seem that a partial mediation effect of minor size is present. But, in fact, this is not enough evidence to infer that parental smoking is a rigorous mediator of parental time discounting. Another valuable finding is that the previously discussed bad control problem is obviously not that *bad*. In columns (4) and (5), we stratify by gender. We find same-sex as well as slightly weaker cross-sex effects of parental smoking status. Although the coefficient of maternal patience is no longer significant for sons, the effects of parental time discounting show similar patterns as in Table 3.3.

Although parental smoking habits do not represent a true mediating factor of time discounting, we can conclude that they are meaningful determinants of child's smoking status. Hence, parents' smoking patterns should not be omitted from the regression. In general, the findings are in line with the findings from our previous specifications without controlling for certain health behaviors. The coefficients of interest change only marginally after taking into account parental health variables. Irrespective of whether we control for parental smoking or parental smoking and all the other health investments, a one standard deviation increase in mothers' patience is associated with a reduction in child smoking of around 6%. The effect of fathers' patience on child's smoking status is very similar in sign and magnitude. The impact of paternal impulsivity is still significant. A one standard deviation increase in fathers' impulsivity is associated with a decrease in the likelihood of smoking of roughly 7%.

[34] Joint significance tests show that the parental health variables are jointly significant for the mother and the father, respectively. Excluding parental smoking, the remaining health variables (alcohol consumption, nutrition and physical activity) are jointly significant for fathers but not for mothers.

Table 3.4: Role of parental health behaviors

		Dep. var.: child's smoking status				
		(1)	(2)	(3)	(4)	(5)
			parental smoking	all behaviors	all behaviors	
					Daughters	Sons
Mother	Impulsivity	0.001 (0.009)	-0.007 (0.009)	-0.007 (0.009)	0.004 (0.012)	-0.018 (0.013)
	Patience	-0.021** (0.009)	-0.018** (0.009)	-0.017* (0.009)	-0.022* (0.012)	-0.017 (0.012)
	Current smoker		0.134*** (0.024)	0.136*** (0.024)	0.162*** (0.032)	0.112*** (0.034)
	Ex-smoker		0.049*** (0.019)	0.050*** (0.019)	0.089*** (0.025)	0.021 (0.027)
	Regular alcohol			0.004 (0.018)	0.016 (0.020)	-0.011 (0.030)
	Healthy nutrition			-0.001 (0.010)	0.010 (0.015)	-0.010 (0.015)
	Regular sport			-0.001 (0.011)	-0.004 (0.017)	0.005 (0.016)
Father	Impulsivity	-0.018* (0.009)	-0.019** (0.009)	-0.019** (0.009)	-0.004 (0.013)	-0.033*** (0.013)
	Patience	-0.019** (0.009)	-0.018** (0.008)	-0.017** (0.008)	-0.012 (0.011)	-0.021* (0.012)
	Current smoker		0.130*** (0.023)	0.125*** (0.023)	0.082*** (0.031)	0.157*** (0.033)
	Ex-smoker		0.061*** (0.020)	0.059*** (0.020)	0.027 (0.027)	0.078*** (0.028)
	Regular alcohol			0.017 (0.011)	0.014 (0.015)	0.022 (0.017)
	Healthy nutrition			-0.020** (0.010)	-0.017 (0.013)	-0.020 (0.015)
	Regular sport			-0.013 (0.011)	-0.005 (0.015)	-0.020 (0.017)
Child	Impulsivity	0.028*** (0.009)	0.026*** (0.009)	0.026*** (0.009)	0.038*** (0.012)	0.019 (0.013)
	Patience	-0.017** (0.008)	-0.015* (0.008)	-0.014* (0.008)	-0.011 (0.011)	-0.014 (0.012)
N		5,817	5,817	5,817	2,788	3,029
R^2		0.106	0.138	0.141	0.175	0.136

Notes: Random effects GLS regressions. Cluster-robust standard errors at the family level in parentheses. Impulsivity, patience and risk are measured in standard deviations. Column (1) replicates the overall results from Table 3.3. All specifications include wave and state dummies, control variables child (gender, age, age squared, migration status, home indicator, income, education, risk preference), and control variables parents (age, migration background, altruism, risk preference, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

3.4.3 Intensive margin of smoking

In our final step, we examine the intensive margin of smoking. So far, we have shown that parental time discounting is directly associated with child's decision to smoke or not to smoke. This refers to the extensive margin of smoking. In the following, we are interested in how parental impulsivity and patience influence the actual demand for tobacco products of the offspring. Conditional on being a smoker, child's smoking intensity is measured as the number of cigarettes smoked per day. A two-part model is specified to estimate child's smoking participation and the level of tobacco consumption.[35]

Results from the two-part model are reported in Table 3.5. As shown in column (1), the average marginal effects of the probit regression are in line with the previous findings from the LPM (extensive margin). With respect to smoking intensity, the coefficients of parental time discounting are no longer significant (see column (2)). However, there is a positive correlation between parental smoking and child's cigarette consumption. Children whose mothers (fathers) are current smokers smoke, on average, 1.6 (2.1) cigarettes more than children whose parents have been non-smokers. If the father is an ex-smoker, child's tobacco consumption is associated with an increase of about 2 cigarettes. The effect of former maternal smoking is not statistically significant. Interestingly, the stratification by gender yields substantial cross-sex effects. Fathers' smoking habits are significant for daughters but not for sons, whereas mothers' current smoking status is only significant for sons. These findings differ from those presented in Table 3.4. With regard to child's smoking participation, we have identified cross-sex and same-sex effects. Finally, the impulsiveness of the child is positively related to the number of cigarettes smoked on a daily basis (see column (2)). A one standard deviation increase in child's impulsivity is associated with an average increase in tobacco consumption of 0.5 cigarettes. According to columns (3) and (4) of Table 3.5, this effect is mainly driven by sons.

We infer that parental time discounting is primarily relevant for child's smoking participation. In particular, parental future orientation plays a significant role. With regard to smoking intensity, parents' smoking habits represent the main contributing factor.

[35] We test the Tobit model against the two-part model using a likelihood ratio test. We reject the null hypothesis that the Tobit model is appropriate at the 1% level (see Smith and Brame, 2003).

Table 3.5: Intensive margin of smoking

		(1)		(2)		(3)		(4)	
		Participation		Intensity		Intensity			
						Daughters		Sons	
Mother	Impulsivity	-0.004	(0.009)	0.350	(0.311)	0.107	(0.456)	0.683	(0.442)
	Patience	-0.018**	(0.008)	-0.297	(0.272)	-0.362	(0.388)	-0.198	(0.385)
	Current smoker	0.145***	(0.025)	1.550*	(0.838)	0.358	(1.239)	2.291**	(1.114)
	Ex-smoker	0.062***	(0.019)	0.722	(0.721)	0.896	(1.137)	0.045	(0.931)
	Regular alcohol	0.007	(0.028)	0.601	(1.236)	1.217	(1.962)	-0.190	(1.516)
	Healthy nutrition	-0.004	(0.017)	-0.301	(0.899)	-0.122	(1.316)	-0.316	(1.171)
	Regular sport	0.001	(0.019)	-0.944	(0.930)	-1.000	(1.482)	-0.668	(1.190)
Father	Impulsivity	-0.022**	(0.009)	-0.051	(0.309)	-0.059	(0.458)	-0.186	(0.414)
	Patience	-0.019**	(0.008)	0.342	(0.290)	-0.039	(0.370)	0.363	(0.411)
	Current smoker	0.144***	(0.027)	2.114**	(0.899)	2.678**	(1.232)	1.502	(1.245)
	Ex-smoker	0.078***	(0.022)	2.080**	(0.837)	2.699**	(1.218)	1.403	(1.164)
	Regular alcohol	0.020	(0.018)	0.204	(0.835)	0.217	(1.084)	0.260	(1.204)
	Healthy nutrition	-0.205	(0.018)	-0.268	(0.883)	0.316	(1.107)	-0.532	(1.268)
	Regular sport	-0.018	(0.019)	0.108	(0.955)	-0.276	(1.269)	0.175	(1.362)
Child	Impulsivity	0.031***	(0.009)	0.539*	(0.291)	0.065	(0.346)	0.955**	(0.475)
	Patience	-0.017**	(0.008)	-0.320	(0.274)	-0.197	(0.377)	-0.414	(0.415)
N		5,817		1,657		725		932	

Notes: Two-part model: Probit regression for smoking participation (full sample). Truncated regression for smoking intensity (subsample of current smokers). Both regressions are run separately using the cmp command. Average marginal effects are reported in column (1). Cluster-robust standard errors at the family level in parentheses. Impulsivity, patience and risk are measured in standard deviations. All regressions include wave and state dummies, control variables child (gender, age, age squared, migration status, home indicator, risk preference, income, education), and control variables parents (age, migration background, risk preference, altruism, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

3.5 Discussion and conclusion

The main purpose of this chapter is the analysis of the link between parental time discounting and child's likelihood of being a smoker. We use self-reported impulsivity and patience as meaningful proxies for self-control and time preference, respectively. We confirm previous evidence of an association between individual time discounting and smoking (*intra*personal context). That is, those individuals who are less impulsive and/or more patient are also significantly less likely to smoke.

More importantly, our results show that parental time discounting, especially parental time preference, has a significant direct effect on the likelihood of child smoking (*inter*personal context). A one standard deviation increase in parental patience is associated with a reduction in the likelihood of child smoking of around 6-7%. Hence, parental future orientation has a preventive effect on child's decision to engage in risky health behaviors such as smoking. Parents with a high level of future orientation are supposed to be well informed about the adverse health effects of smoking. Therefore, they may have an influence on child's smoking status by pointing out the negative health consequences of smoking. With regard to father's self-control, a one standard deviation increase in impulsivity is associated with a 7% reduction in child's smoking probability. However, the interpretation of this effect is not straightforward. Children whose parents are impulsive could have difficulties in properly anticipating parental actions and/or consequences after they have learned about specific child behaviors. Hence, those children could act with caution and rather think twice before they indulge in the consumption of health deteriorating goods such as cigarettes. We admit, however, that this interpretation may be rather imperfect.

The direct relationship between parental time discounting and child smoking remains robust to the inclusion of potential mediating factors such as socioeconomic outcomes and different health behaviors. When we control for parental smoking, our findings are in line with results from the previous literature on the transmission of smoking habits from parents to their children. Hence, we confirm the well-known positive correlation between parental and child smoking. In general, parental time discounting and parental smoking patterns are both significantly related to child's smoking participation. Thus, the direct effects of parental time discounting on child smoking do not vanish after controlling for parental smoking. However, the results from the two-part model reveal that parental time discounting is not significantly associated with child's smoking intensity.

In addition, our findings provide evidence that the influence of the father is important in this context and should not be ignored. Hence, focusing only on mothers (e.g., Brown and van der Pol, 2014) may result in potentially misleading inference. Stratifying by gender, we find differences in the effects of parental time discounting. The influence of maternal patience on child smoking is significant for both daughters and sons, whereas father's time discounting is primarily relevant for boys. Our findings are generalizable to the adult population in Germany because we rely on a representative sample of adult individuals/children. Moreover, we control for whether the offspring still lives together with at least one biological parent or if the child has already moved out. In general, our findings encourage future research to control for both risk preference and time discounting measures (if available) when analyzing the mechanisms of health (behavior) transmission from one generation to another.

However, our study has some limitations. As already mentioned above, our mediating factor analysis is only a partial one. We focus on health behaviors through which parental time preference or impulsivity might affect our dependent variable. In fact, we are not able to identify a true (health) mediator. But, once more, this highlights and supports the presence of a direct relationship between parental time discounting and child smoking. Nonetheless, there may exist other latent channels through which the direct effects of parental discounting could be absorbed. We suggest that parenting style could be an appropriate candidate variable. Children of parents that care about good (child) health are unlikely to smoke because their parents are likely to properly invest in their children's health capital. Health education and family communication may be of particular importance. Unfortunately, we are not able to address this issue with our data.

Another disadvantage is that we are not able to properly capture peer effects such as the influence of friends or other social environments. Furthermore, due to data limitations, we cannot systematically address the actual formation of time preference during childhood and adolescence. Information on time discounting is not reported until the offspring turns into an adult and answers the corresponding questions of the standard individual questionnaire. However, evidence suggests that delay discounting is affected by both genetic and environmental influences (e.g., Anokhin et al., 2011).[36] But, our sample lacks sufficient information on twins and/or adoptees to examine the role of genetic factors in more detail.

Furthermore, we only include those children in our sample for whom information on both parents was available. First, we focus on both parents to examine if neglecting one parent (e.g.,

[36]For further reading, see MacKillop (2013).

the father) is a clever idea. Obviously, it is not. Second, we do not have adequate information on a single parent's living circumstances. Normally, those influences are hardly observable. For instance, we lack information on when exactly parents split up or for how long a parent is a single mother or father. Moreover, we cannot account properly for the potential influence of a new partner. We simply do not know if children from single-parent families still have regular contact with the biological parent who left the family home.

Although we analyze a relatively short period of time, the assumption of stable preferences may be another point of criticism. However, we make this assumption in order to set up a panel data analysis. Survey questions on impulsivity and patience asked in 2008 are not part of the standard individual questionnaires from waves 2006 and 2010. Nevertheless, we get qualitatively similar results even after running a simple cross-section analysis based on data from wave 2008. Finally, we cannot completely rule out potential endogeneity bias. Dealing with preferences, it is always a challenge to identify causal effects. However, we argue that our empirical approach of gradually adding potentially problematic control variables is able to reduce endogeneity concerns. Obviously, our regression results remain robust, regardless of whether we control for these variables or not.

Undoubtedly, smoking is a preventable health risk and (still) a major public health concern. Our analysis provides new findings which are potentially relevant for public health authorities that are concerned with the prevalence of tobacco consumption. In line with the previous literature, individual time discounting is significantly related to the decision to smoke. In addition, we find significant associations between parental time discounting and child's smoking status even after controlling for parental smoking and other health mediating factors. Nevertheless, the possibility exists that the inclusion of other mechanisms (e.g., family communication) could wash out the direct effects of parental impulsivity and/or patience. We refrain from giving extensive policy implications because the empirical results cannot necessarily be interpreted as causal relationships.

However, it is important to brief parents about the influence they have when they act as role models and primary health mentors. This information is crucial if public health services intend to prevent (young) people from starting to smoke or to help them quit tobacco consumption. The support of smoking cessation efforts in young adults, the next parent generation, should also be considered. The improvement of self-control techniques may help individuals to successfully abstain from smoking. Especially future orientation seems to be a key parameter to break up the vicious cycle of adverse health behaviors that are passed from generation to generation.

However, more research is needed to fully explore the role of time discounting and risk preferences in the intergenerational transfer of smoking and to provide more rigorous advice to public health authorities.

3.6 Appendix

Table 3.6: Stratification by home

		Dep. var.: child's smoking status		
		(1)	(2)	(3)
			With parents	Own household
Mother	Impulsivity	0.001 (0.009)	-0.006 (0.012)	0.015 (0.013)
	Patience	-0.021** (0.009)	-0.026** (0.011)	-0.026** (0.012)
Father	Impulsivity	-0.018* (0.009)	-0.008 (0.012)	-0.025* (0.013)
	Patience	-0.019** (0.009)	-0.012 (0.011)	-0.028** (0.012)
Child	Impulsivity	0.028*** (0.009)	0.027** (0.012)	0.021 (0.013)
	Patience	-0.017** (0.008)	-0.027** (0.011)	-0.019 (0.012)
N		5,817	2,944	2,873
R^2		0.106	0.116	0.148

Notes: Random effects GLS regressions. Cluster-robust standard errors at the family level in parentheses. Column (1) replicates the results from column (4) of Table 3.3. Impulsivity, patience and risk are measured in standard deviations. All regressions include wave and state dummies, control variables child (gender, age, age squared, migration status, education, income, risk preference), and control variables parents (age, migration background, risk preference, altruism, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

Table 3.7: Stratification by age

		Dep. var.: child's smoking status						
		(1)	(2)	(3)	(4)	(5)	(6)	(7)
			Under 20	Under 25	Under 30	Under 35	Under 40	Under 45
Mother	Impulsivity	0.001 (0.009)	-0.024 (0.016)	0.005 (0.013)	0.002 (0.011)	-0.001 (0.010)	0.003 (0.009)	0.002 (0.009)
	Patience	-0.021** (0.009)	-0.053*** (0.015)	-0.026** (0.012)	-0.024** (0.010)	-0.025*** (0.009)	-0.022** (0.009)	-0.022** (0.009)
Father	Impulsivity	-0.018* (0.009)	-0.017 (0.016)	-0.018 (0.013)	-0.013 (0.011)	-0.016 (0.010)	-0.018* (0.010)	-0.018* (0.009)
	Patience	-0.019** (0.009)	-0.018 (0.014)	-0.016 (0.012)	-0.023** (0.010)	-0.028*** (0.009)	-0.022** (0.009)	-0.020** (0.009)
Child	Impulsivity	0.028*** (0.009)	0.043*** (0.016)	0.030** (0.012)	0.025** (0.011)	0.028*** (0.010)	0.030*** (0.009)	0.029*** (0.009)
	Patience	-0.017** (0.008)	-0.026* (0.015)	-0.020* (0.011)	-0.010 (0.009)	-0.015* (0.009)	-0.016* (0.008)	-0.016* (0.008)
N		5,817	1,101	2,740	3,989	4,790	5,369	5,700
R^2		0.106	0.148	0.140	0.121	0.117	0.113	0.113

Notes: Random effects GLS regressions. Cluster-robust standard errors at the family level in parentheses. Column (1) replicates the results from column (4) of Table 3.3. Impulsivity, patience and risk are measured in standard deviations. All regressions include wave and state dummies, control variables child (gender, age, age squared, migration status, home indicator, risk preference, education, income), and control variables parents (age, migration background, risk preference, altruism, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

Chapter 4

Heritability of time preference: Evidence from German twin data

4.1 Introduction

In economics, individual preferences are highly important since they affect people's decision-making.[1] Next to the exhaustively studied role of risk preferences, the economic concept of time preference is particularly relevant. Time preferences are significantly involved in a person's intertemporal choices. In general, they influence decisions that include a trade-off between costs today and (potential) benefits in the future (see Frederick et al. (2002) for an excellent review of the concept of time discounting). Time preferences are related to a variety of crucial lifetime outcomes such as educational attainment and personal income. For instance, Golsteyn et al. (2014) show that patience predicts success in school as well as higher earnings in the long run. In addition, present-biased individuals are less likely to save money for the future. They are even more likely to run into debt (Meier and Sprenger, 2010). Moreover, a high discount rate (low level of patience) is associated with a number of adverse health behaviors and outcomes such as smoking (Kang and Ikeda, 2014), frequent alcohol consumption (Rossow, 2008) and obesity (Komlos et al., 2004).[2]

In recent years, the empirical evidence on the intergenerational transmission of time preference has increased. For instance, Gauly (2017) finds a positive correlation between parental and offspring time preference using representative household survey data from Germany. Other studies show similar results.[3] Despite all these findings, a fundamental question remains unan-

[1] This chapter is a slightly extended version of Hübler (2018).

[2] The expressions time preference, time discounting, discount rate, patience and future orientation are used synonymously throughout the course of this chapter.

[3] See, for example, Brown and van der Pol (2015), Arrondel (2013), Kosse and Pfeiffer (2012), Gouskova et al. (2010), Reynolds et al. (2009), Webley and Nyhus (2006), and Knowles and Postlewaite (2005).

swered. How are time preferences actually formed? Are people born with innate time preferences or can they be shaped by the environment? In other words, are time preferences exogenous or endogenous? Providing answers to these questions is crucial if (behavioral) economists and policy makers want to develop effective interventions to encourage forward-looking behavior.

Apparently, time preferences differ across individuals (e.g., Barsky et al., 1997). But where does this variation come from? Inspired by psychology, behavioral genetics pioneered the introduction of the nature-nurture debate which is concerned with the decomposition of genetic and environmental influences on human traits and phenotypes (Galton, 1869). "Nature" refers to the relative contribution made by genetic inheritance and related biological factors. "Nurture" represents the influence that comes from external sources such as parents' socialization efforts, imitation/learning or unique experiences. In fact, previous studies have found that both inherited and acquired characteristics determine behavioral traits and/or outcomes such as the socioeconomic status (see, e.g., Björklund et al., 2007).

In this chapter, we want to shed light on the fraction of variance in time preference that is explained by genetics. Relying on twin data from Germany, the comparison of identical/monozygotic (MZ) and fraternal/dizygotic (DZ) twins enables us to estimate the proportion of variation in individual time preference that is due to genetic and environmental factors. In twin studies, it is assumed that both types of twin pairs share their environments to the same degree. But fundamental differences in the genetic relatedness exist between MZ and DZ twins. Hence, any excess similarity of MZ twin pairs compared to DZ twin pairs with respect to a specific phenotype or trait indicates the presence of genetic effects. Studies that merely analyze the *raw* intergenerational correlation in a trait are not able to adequately decompose this variation.

The literature on the endogeneity of preferences has developed theoretical frameworks which show that nurture may be important with regard to the determination of time preference. Becker and Mulligan (1997) argue that parents can invest resources and time to make their children more future-oriented. Hence, teaching their children to plan for the future directly affects the children's time preference rate. Following a different approach, the model in Bisin and Verdier (2001) shows the impact of the parental and social environment on children's preferences. However, empirical evidence on the heritability of time preference is scarce. Direct association studies link genes to certain behavior patterns or preferences (e.g., Carpenter et al., 2011). However, these studies are lacking to quantify the relative importance of nature and nurture with regard to the total variation in time preference.

To the best of our knowledge, there have been a limited number of studies addressing a

similar research question. Anokhin et al. (2011) analyze the heritability of delay discounting in a longitudinal twin design. Using a basic delay of gratification method, they find that genetic factors contribute 30% and 51% to the variation in delay discounting at ages 12 and 14, respectively. Cesarini et al. (2012) use data from the Screening Across the Lifespan Twin survey, Younger cohort (SALTY) which is part of the Swedish Twin Registry (STR). They find that the heritability estimate for short-term time preference, measured using hypothetical choice questions in the monetary domain, is not significantly different from zero.[4] Cronqvist and Siegel (2015) also rely on a set of MZ and DZ twins from the STR. However, they focus on a person's saving behavior and relate it to individual time preference. According to their findings, genetic differences explain roughly one third of the total variation in savings propensities across individuals. Interestingly, evidence on the genetic variation in economic risk preferences is more conclusive. However, the relative contribution of genetics to risk attitudes varies considerably across studies. Whereas Cesarini et al. (2009) show that heritability is around 20%, Zhong et al. (2009) and Zyphur et al. (2009) find magnitudes of 57% and 63%, respectively.

In line with the aforementioned literature, our study employs the classical twin methodology. However, we contribute to the literature in several ways. We are the first to use a novel twin data set from Germany to estimate the genetic variation in time preference. Cross-sectional data are available for adolescents and young adults aged 10-25 years. In comparison to the vast majority of twin studies, the sample size is relatively large. Our final sample contains information on roughly 3,000 twins. Hence, we are able to address some common limitations of twin studies based on laboratory experiments such as a limited age range of the participants and/or a relatively modest sample size (see Anokhin et al., 2011). In general, this impairs the statistical power and limits the scope of the empirical analysis. Moreover, we use a direct measure of time preference which is new in this area of research. A simple and short survey question on general patience acts as a proxy for individual time discounting. An incentivized experiment showed that self-assessed patience is a meaningful proxy for time preference as elicited using the multiple price list (MPL) decision format (Vischer et al., 2013). The general level of patience of an individual is likely to evaluate the personal discount rate more comprehensively than relying on his/her savings behavior alone. The latter is supposed to be an outcome of time discounting rather than a straightforward measurement of time preference (see Cronqvist and Siegel, 2015).

Our empirical analysis provides evidence that time preferences are partly heritable. According to our best-fitting model, we find that differences in genetics explain about 23 percent of

[4]Benjamin et al. (2012), using the same dataset (SALTY) but different estimation techniques, also find no support for genetic variation in time preference.

the variation in patience across individuals. The results reveal a minor influence of the twins' shared environment. Hence, the remaining variation in time preference is accounted for by the unique environment of the individual. The remainder of this chapter is organized as follows. Section 4.2 describes the twin data and the measurement of time preference. Section 4.3 provides information on the basic twin methodology and the statistical analysis. Results are presented in Section 4.4. Finally, Section 4.5 concludes with a discussion of the main findings.

4.2 Data

4.2.1 Twin data

We use novel twin data from the German TwinLife project. The main purpose of this twin family study is to improve the understanding of the development of social inequalities over the life course. Next to its longitudinal design, TwinLife combines a multi-cohort cross-sequential and an extended twin family design. It observes four birth cohorts of MZ and DZ twins over a 12-year period. Twins born in the years 1990-1993, 1997/1998, 2003/2004 and 2009/2010 as well as their parents and siblings (if available) are included. The project began in 2014 and is supposed to end with the last survey wave in 2023. Interviews are conducted on a yearly basis. Overall, this twin survey will provide data of a representative sample of about 4,000 German twin families. In this study, we rely on data from the first household interview for the first partial wave on 2,009 twin families. In order to prevent distortions due to gender differences, only twin pairs of the same sex were surveyed. Furthermore, only such twins are examined that have grown up or are still growing up in the same family. More details on the conception and design of the TwinLife study can be found elsewhere (Hahn et al., 2016).[5]

For the empirical analysis, we exclude all twins from the youngest birth cohort. Twins born in 2009/2010 were about five years old during the first survey wave in 2014 and therefore too young to answer the survey question on time preference. Further restrictions are necessary because not all individuals have provided information on their respective time preference. Hence, we exclude all respondents with missing information on the variable of interest. The remaining twins are children, adolescents and young adults aged 10-25 years. Approximately, all three cohorts comprise a similar number of twins. The first cohort of twins contains 990 twins aged 10-12 years, the second comprises 1,034 twins aged 16-18 years and the third includes 932 twins aged 22-25 years. It allows us to cover a wide range of ages. This is of particular interest because this early period of life is characterized by fundamental decisions on education, employment,

[5] Additional information is also available online: `http://www.twin-life.de/en`.

health, etc., where time preferences are frequently involved. All these decisions made will affect lifetime outcomes (e.g., income) in the future.

The zygosity of twins was verified with a specific zygosity questionnaire. In addition, all results obtained from the standard questionnaire items were validated with a saliva test. This DNA test is a standard procedure to tell if the twins are monozygotic or dizygotic and is generally considered to be highly reliable. Our final sample contains information on 703 MZ twin pairs (1,406 twins) and 775 DZ twin pairs (1,550 twins). Hence, the total sample size sums up to 2,956 individuals. Table 4.1 shows the number of twins separated by zygosity and sex. As previously mentioned, opposite-sexed dizygotic twins are not part of the survey.

Table 4.1: Number of twins by zygosity and gender

	female	male	
DZ twins	856	694	1,550
MZ twins	806	600	1,406
	1,662	1,294	2,956

4.2.2 Measuring time preference

The twin questionnaire contains a convenient survey question to elicit individual time discounting. Each respondent has to rate his or her personal level of patience according to an 11-point scale. The exact wording of the question is as follows: "How would you describe yourself: Are you generally an impatient person, or someone who always shows great patience? Please tick a box on the scale, where the value 0 means: 'very impatient' and the value 10 means: 'very patient'. You can use the values in between to make your estimate." Hence, self-reported patience is used as a proxy for individual time preference. In our sample, the average level of patience is 5.75.

The behavioral relevance of our time preference measure has been explicitly validated. This ultra-short survey measure of patience was first introduced in the 2008 questionnaire of the German Socio-Economic Panel (SOEP). The SOEP is an annual panel survey conducted since 1984. Each year more than 20,000 individuals (12,000 households) representative of the German population are surveyed (see Wagner et al., 2007). Vischer et al. (2013) conducted an incentivized experiment with 977 participants forming a representative sub-sample of the adult population to the 2006 wave of the SOEP. Subjects were asked to indicate their preferences in a choice over a

12-month time horizon.[6] The results show that those who rank themselves as 'more impatient' in the survey in 2008 also exhibit a higher degree of impatience in the experiment in 2006. Hence, this simple and ultra-short survey measure of patience turns out to be a meaningful proxy for time preference. The findings remain robust even after controlling for impulsivity.[7] This shows that the measure of general patience captures (long-term) patience, and not (short-run) impulsivity. Thus, a respondent's misinterpretation of the more future-oriented aspects underlying this question on patience can be ruled out. Table 4.2 reports the summary statistics of our twin sample. Apparently, Figure 4.1 indicates that variation in self-assessed patience is present across individuals.

Table 4.2: Summary statistics of the final twin sample (n=2,956)

Variable	**Mean**	**S. D.**	**Min.**	**Max.**
patience	5.753	2.624	0	10
female	0.562	0.496	0	1
age	17.095	5.085	10	25
MZ	0.476	0.499	0	1

4.3 Twin methodology

The classical twin design contains information on both MZ and DZ twins. This enables us to decompose the observed variance in a specific trait (e.g., patience) into genetic and environmental components. According to the polygenic model (ADCE model) proposed by Fisher (1918), four latent factors can be taken into account. The genetic variation in a phenotype or trait can be divided into two components. The additive genetic effect (A) describes the influence of different alleles which are added up when being passed from the parents to the offspring. In other

[6] In the experiment, choice tables with the typical price list decision format were used. The participants had to indicate their preferences by choosing between an immediate (left column) or delayed payment (right column). The immediate payment was continuously fixed (€200). However, the delayed payment varied in each of the 20 choice situations and increased by 2.5 percentage points (compounded semi-annually) from row to row. Switching from left to right (and sticking to the delayed payment in all subsequent rows) indicates the bounds of the discount rate the respondent claims in order to wait an additional time period of 12 months for payout. Before the start of the experiment, the participants were informed that one of their choices would be randomly selected for payment. Using a second random device, one out of nine participants was actually paid by check according to the previous choice.

[7] In addition to impulsivity, including a control variable for personal risk attitudes does not affect the results either.

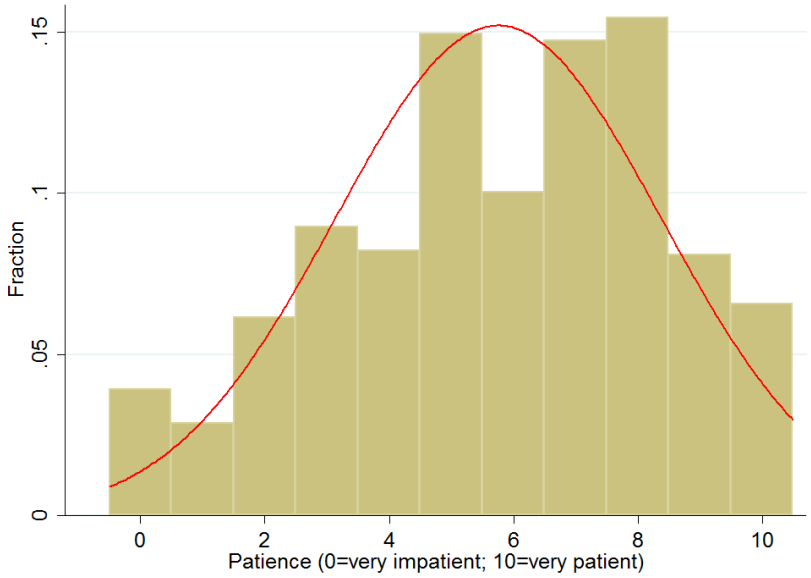

Figure 4.1: Distribution of patience with the plot of a normal-density curve

words, it is an additive function of many genes that code for a specific trait. The second genetic component consists of non-additive/dominant genetic effects (D). These effects are characterized by allelic interactions within genes. Hence, these genetic influences are transmitted from parents to their offspring in a dominant/recessive way.

With respect to the environmental components, shared and non-shared environmental effects can be distinguished. The common environment component (C) captures all influences shared by the twins reared in the same family. Thus, both twins of a twin pair are equally affected by this component. It comprises the same prenatal environment, home environment, parenting style, socioeconomic status of the parents/family, and the like. On the contrary, the unique environmental effects (E) are not shared by the twins. They are different across the twins of a twin pair. These individual-specific influences refer to unique experiences of illness or injury, unique experiences with friends and sexual partners, and the like.

Based on this logic, the standard twin model decomposes the total variance of patience into the four components A, D, C, and E. The corresponding ADCE model of patience (Pat) for twin j ($j = 1, 2$) in family/twin pair i can be written as a variance component model

$$Pat_{ij} = X_{ij}^{'}\beta + A_{ij} + D_{ij} + C_i + E_{ij}\,, \tag{4.1}$$

where $X_{ij}^{'}\beta$ is a set of independent covariates and their parameters, $A_{ij} \sim \mathrm{N}(0, \sigma_A^2)$ is the additive genetic component, $D_{ij} \sim \mathrm{N}(0, \sigma_D^2)$ is the dominant genetic component, $C_i \sim \mathrm{N}(0, \sigma_C^2)$

is the common environmental component, and $E_{ij} \sim \mathrm{N}(0, \sigma_E^2)$ is the non-shared environmental component.[8] The expected value (mean) of patience is $E(Pat_{ij}) = X_{ij}^{'}\beta = \mu_{ij}$. Thus, the total variance of patience is represented by the sum of the four mutually independent variance components

$$\mathrm{Var}(Pat_{ij}) = \sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2 \,. \tag{4.2}$$

Genetic theory shows that differences in the genetic relatedness between MZ and DZ twins exist (see Neale and Maes, 2004). MZ twins develop from the splitting of the same fertilized egg into two, whereas DZ twins develop from two different eggs fertilized by two different sperm cells. Hence, MZ twins share all their genes. On the contrary, DZ twins share (on average) only half their genes. Among DZ twins, this results in a correlation of 0.5 for additive genetic effects and a correlation of 0.25 for dominant genetic effects. Since the shared environment is reasonably assumed to be the same for both members of a twin pair, the correlation is perfect (1.0) across twins, regardless of zygosity. Non-shared environmental factors are unique to each twin and therefore uncorrelated across twins of a twin pair, regardless of zygosity. Due to these genetic differences, the variance-covariance matrices vary by type of twin pair. For MZ twin pairs, the covariance matrices can be written as

$$\sum\nolimits_{Pat}^{MZ} = \begin{pmatrix} \sigma_A^2 & \sigma_A^2 \\ \sigma_A^2 & \sigma_A^2 \end{pmatrix} + \begin{pmatrix} \sigma_D^2 & \sigma_D^2 \\ \sigma_D^2 & \sigma_D^2 \end{pmatrix} + \begin{pmatrix} \sigma_C^2 & \sigma_C^2 \\ \sigma_C^2 & \sigma_C^2 \end{pmatrix} + \begin{pmatrix} \sigma_E^2 & 0 \\ 0 & \sigma_E^2 \end{pmatrix} , \tag{4.3}$$

and the respective covariance matrices for DZ twin pairs can be written as

$$\sum\nolimits_{Pat}^{DZ} = \begin{pmatrix} \sigma_A^2 & \frac{1}{2}\sigma_A^2 \\ \frac{1}{2}\sigma_A^2 & \sigma_A^2 \end{pmatrix} + \begin{pmatrix} \sigma_D^2 & \frac{1}{4}\sigma_D^2 \\ \frac{1}{4}\sigma_D^2 & \sigma_D^2 \end{pmatrix} + \begin{pmatrix} \sigma_C^2 & \sigma_C^2 \\ \sigma_C^2 & \sigma_C^2 \end{pmatrix} + \begin{pmatrix} \sigma_E^2 & 0 \\ 0 & \sigma_E^2 \end{pmatrix} , \tag{4.4}$$

where the variances of the twins are situated in the diagonal and the corresponding covariance between twin 1 and twin 2 is situated in the off-diagonal of each matrix.[9] Since both twin

[8] Throughout the biometric analysis, we use the standardized version of patience. The genetic and environmental components are assumed to be uncorrelated across twin pairs. For notational convenience, the random error term ϵ_{ij} is replaced by E_{ij} in Equation (4.1). Hence, the measurement error is part of the non-shared environment component.

[9] It is assumed that the twin pairs are unrelated. The general notation of a variance-covariance matrix in the twin context is

$$\sum = \begin{pmatrix} \text{variance of twin 1} & \text{covariance of twins} \\ \text{covariance of twins} & \text{variance of twin 2} \end{pmatrix}.$$

types are supposed to experience the same degree of similarity in their environments (equal environment assumption), any excess similarity in patience between MZ twins must be due to the greater proportion of genes shared by MZ twins compared to DZ twins. The estimate of interest is the (broad) heritability coefficient:

$$H_{Pat} = \frac{\sigma_A^2 + \sigma_D^2}{\sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2}\,. \tag{4.5}$$

It provides the degree to which genetic factors contribute to the total variation in patience across individuals.

The polygenic model is estimated under the standard assumptions of biometric modeling (see, e.g., Rijsdijk and Sham, 2002). MZ twins are considered to be genetically identical and the equal environment assumption holds true for MZ and DZ twins. No gene-environment correlations or interactions are present for the trait of interest. Another non-technical assumption is the absence of non-random pairing of the twins' parents. The most important technical assumptions are equal mean and variance of twin 1 and twin 2 and MZ and DZ twins. This is required to estimate the model because the same path coefficients are applied for both MZ and DZ twins. Moreover, the maximum likelihood principle is used for estimation and inference. It assumes bivariate normality of the paired observations (see the Appendix of this chapter for further details).

A disadvantage of this biometric approach is that it is impossible to estimate all four components of the ADCE model simultaneously with classic twin data alone. Normally, only information on MZ and DZ twins reared together is available. Thus, the effect of the shared environment (C) and the influence of genetic dominance (D) are confounded in the classic twin study design (Neale and Maes, 2004).[10] Retaining the additive genetic component (A) and the non-shared environmental component/measurement error (E), the remaining components make opposite predictions about the relative difference between MZ and DZ correlations. Common environmental influences make DZ correlations more similar to MZ correlations. However, the presence of dominant genetic effects makes DZ correlations less similar to MZ correlations. The primary reason for that is that D correlates perfectly for MZ twin pairs whereas the correlation is only 0.25 for DZ twin pairs (Rijsdijk and Sham, 2002). Therefore, in practice, ACE or ADE

All four entries in a matrix are the same when the correlation among the twins of a twin pair is considered to be perfect ($\rho = 1$) and certain assumptions are made. Assuming equal mean and variance for twin 1 and twin 2 and MZ and DZ twins, the covariance of the twins is equal to the variance of each twin.

[10] When A and E are both retained in the model, the estimation of C and D at the same time results in an underidentification problem. Covariance differs across twin types: $Cov_{MZ} = \sigma_A^2 + \sigma_D^2 + \sigma_C^2$ vs. $Cov_{DZ} = \frac{1}{2}\sigma_A^2 + \frac{1}{4}\sigma_D^2 + \sigma_C^2$. But two informative equations are not enough to solve for three unknown parameters. Hence, we have to assume $D = 0$ or $C = 0$ to proceed with the biometric analysis.

models and their respective submodels (e.g., AE or DE models) are estimated. For example, the ADE model is obtained after eliminating C_i from Equation (4.1). Which model to choose highly depends on the information directly retrieved from the twin data.

Genetic heuristics predict the existence of genetic effects when the trait correlation among MZ twin pairs is higher than the correlation among DZ twin pairs. If the covariance of MZ twin pairs is larger than the covariance of DZ twin pairs, but the DZ covariance is more than half the size of the MZ covariance, C is a meaningful contributing factor to the variation in patience ($2\rho_{\text{DZ}} > \rho_{\text{MZ}} > \rho_{\text{DZ}}$). Hence, an ACE model should be estimated. By contrast, genetic dominance is present if $\rho_{MZ} > 2\rho_{DZ}$. In other words, a correlation among MZ twin pairs that is more than two times larger than the corresponding correlation among DZ twin pairs suggests a genetic dominance effect (D) on time preference. In this case, the influence of the shared environment component is set to zero and the ADE model should be fitted (see Plomin et al., 2013; Neale and Maes, 2004).

To provide evidence on the heritability of time preference/patience, we follow three basic steps. First, we start with a mean comparison across MZ and DZ twin pairs. Second, we compare the intraclass correlation coefficients across both twin types. This should provide us with a guideline for the biometric analysis. Finally, we apply maximum-likelihood based structural equation modeling to elicit the best-fitting polygenic model and report the relative contributions of the variance components. The statistical tool (twinlm) in the Analysis of Multivariate Events *mets*-package in R is used to conduct the structural equation analysis (Holst and Scheike, 2017).

4.4 Results

We start with reporting basic statistics on patience for MZ and DZ twins. In Figure 4.2, we plot a histogram of the distribution for patience, separately, for MZ and DZ twins. In general, identical twins seem to be more patient than fraternal twins. This initial finding is supported by the comparison of the average level of patience between both twin types. The mean levels of patience are reported in Table 4.3.

On average, MZ twins report a general level of patience of about 5.9. DZ twins are slightly more impatient. The corresponding level of patience is roughly 5.6. However, the difference between means is highly significant. Testing the equality of means rejects the null hypothesis of equal means at the 1% level.[11] In Table 4.4, we report the MZ and DZ twin pair correlations

[11]The significant difference in patience across MZ and DZ twins may be surprising. For this reason, we run

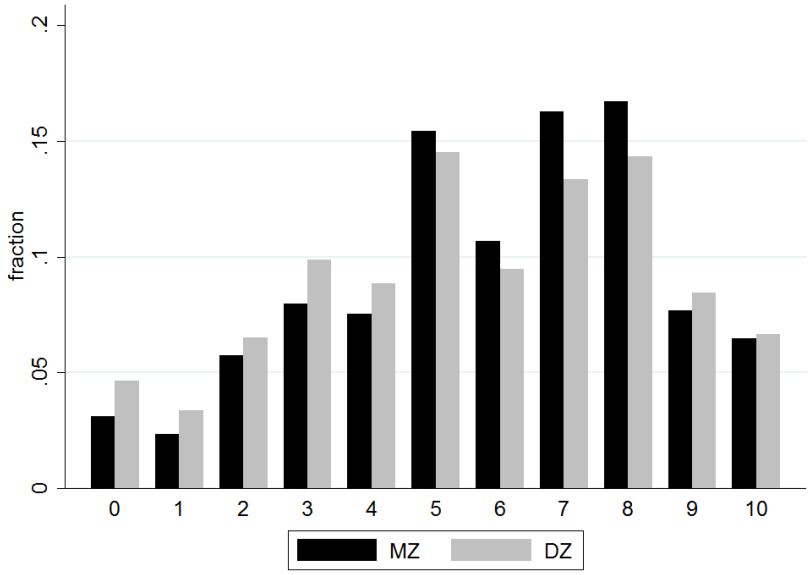

Figure 4.2: Distribution of patience, by zygosity

Table 4.3: Self-reported patience

		MZ twins	**DZ twins**	**p-value**
Patience	Mean	5.907	5.614	<.01
	S.D.	2.522	2.707	
	n	1,406	1,550	

Notes: p-value from two-sample t-test. Null hypothesis: Same mean of patience for MZ and DZ twins.

of patience. Spearman's correlation coefficient for MZ twin pairs is 0.241 and highly significant. The respective correlation for DZ twin pairs is basically zero.[12] These findings provide first evidence that genetics seem to play a role in the variance of individual patience. The substantial difference in MZ and DZ correlations can also be considered graphically. In Figure 4.3, we plot twin's patience against co-twin's patience. It supports the calculated correlations from above.

tests of equality separately for each age group. Interestingly, we fail to reject the null hypothesis for children aged 10-12 years (p = 0.234) and young adults aged 22-25 years (p = 0.469). However, for adolescents (16-18 years of age), we reject the null hypothesis that the MZ and DZ means are equal at the 1% level. When we exclude the adolescents from the sample, we cannot reject the null hypothesis at the 5% level. These findings may be explained by a phase of neural imbalance between brain areas associated with time discounting in adolescence. Cognitive and emotional changes frequently lead to poor decision-making which is likely to be responsible for the significant differences in this cohort (e.g., Konrad et al., 2013).

[12]The Spearman correlations are very similar to the Pearson correlations. Pearson's correlation for MZ twin pairs is 0.233 (p < .01). Pearson's correlation for DZ twin pairs is 0.021 and not statistically significant.

Table 4.4: Correlations for MZ and DZ twin pairs

		MZ twin pairs	**DZ twin pairs**	**p-value of diff.**
Patience	Spearman	0.241***	0.006	<.01
		(0.170-0.310)	(−0.064-0.077)	
	n	703	775	

Notes: Significance: *p<0.1, **p<0.05, ***p<0.01. 95% confidence intervals for Spearman's rank correlations in parentheses. p-value from bootstrapped equality test for equal correlation coefficients. Null hypothesis: Same correlations of patience for MZ and DZ twin pairs. Bootstrapped replications = 1,000.

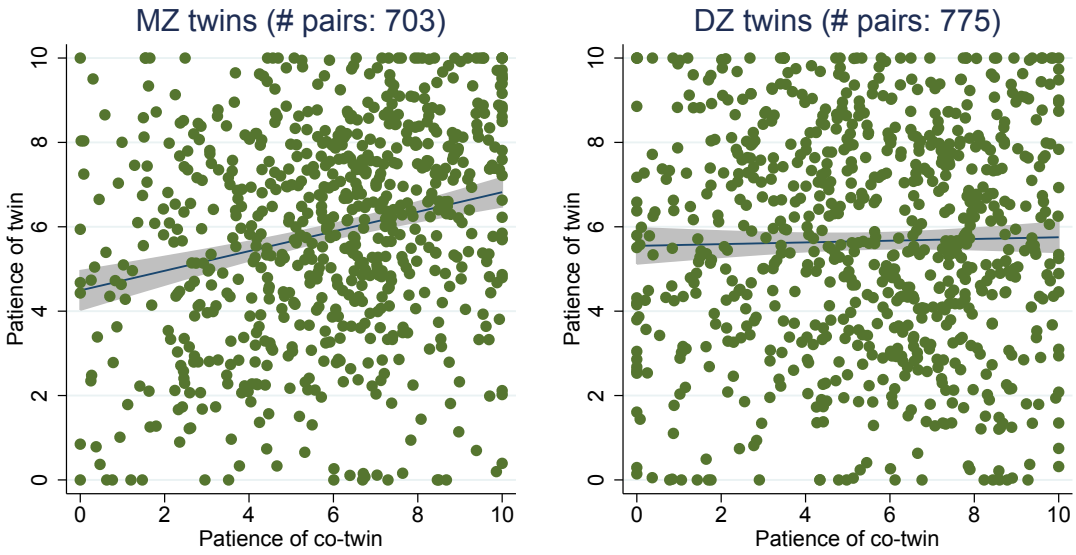

Figure 4.3: Twin-twin plots of patience, by zygosity (Fitted regression lines and 95% confidence intervals are reported. Scatterplots are jittered for expositional clarity.)

In the following, we present our findings from the biometric analysis. The results from the model selection process and from the variance component analysis are shown in Table 4.5. All models include control variables for age and sex.[13] In order to appropriately test the overall fit of each polygenic model, we use the saturated model as a benchmark. The saturated model provides the means, variances and covariances without imposing any restrictions. Hence, it is the least restrictive model since no constraints/technical assumptions are made. Comparing the genetic twin models (e.g., the ADE model) to the saturated model allows us to evaluate the actual model fit. If the main assumptions are fulfilled, no significant drop in model fit should be observed.

We decide to test the ADE model against the saturated model.[14] The existing differences in correlation between the MZ and DZ twin pairs (0.241 vs. 0.006) predict the presence of a dominant genetic effect on the variance in patience. Hence, twin theory suggests that a standard ACE model would not be appropriate. According to Plomin et al. (2013), dominant genetic effects preclude an influence of the shared environment of the twins. Here, we reject the hypothesis that the fit of the ADE model is not significantly worse than the fit of the saturated model. In other words, the ADE model shows a reduction in model fit compared to the benchmark model ($p < .05$). However, we insist on the assumptions mentioned above although our data do not fully support these.[15] It is noteworthy that this issue occurs frequently in twin studies, especially in those with relatively large sample sizes. Even minor differences in variances between twin groups can be highly significant (see, e.g., Waszczuk et al., 2015). Moreover, in fairly large twin samples the consideration of the BIC criterion is advised. It takes into account the underlying sample size. The BIC criterion is considerably smaller for the ADE model, indicating a better fit than the saturated model (see, e.g., Dale et al., 2015).[16]

[13] We treat our outcome variable as a continuous variable. The survey question on general patience is measured on a scale from 0 to 10 (see Section 4.2.2). The response to this question represents the basic degree of patience of the individual. Hence, we argue that the underlying concept of this variable can reasonably be interpreted as being continuous. Moreover, the intervals between the point values are equal. However, we also conduct the biometric analysis treating patience as ordinal. Unfortunately, this has the disadvantage that the cell probability matrix contains some empty cells when controlling for the full set of standard covariates (age and sex). That circumstance limits the scope of analysis. However, we are able to provide some evidence that our results are robust to the application of alternative estimation techniques. For instance, the results are very similar after excluding age from the regression, regardless of whether the dependent variable is treated as continuous or ordinal. The robustness of our results is in line with the findings from Cesarini et al. (2009).

[14] Effectively, all theoretical twin models represent submodels of the saturated model.

[15] The fit of the biometrical model is better if we drop the group of adolescents from the sample. In this case, the ADE model is not significantly worse than the saturated model ($p = .103$). However, our results are almost identical if we exclude all twins aged 16-18 years (see Table 4.8 in the Appendix of this chapter).

[16] Table 4.9 in the Appendix of this chapter compares the ACE model with the saturated model as well as with the ADE model. According to the Likelihood ratio test, the ACE model is clearly outperformed by the saturated model. After comparing the ACE model with the ADE model, the ADE model gives the better fit by AIC and BIC. As already suggested by the intraclass correlation coefficients, these findings lend additional support to the

We continue with the model selection procedure and test the ADE model against other more parsimonious models (AE vs. ADE; DE vs. ADE). Dropping the D component from the ADE model significantly reduces model fit (LRT=6.077, $p < .01$). Eliminating the additive genetic component (A) from the ADE model does not worsen the model fit (LRT<.01, $p > .10$).[17] This is not surprising because A is estimated zero in the ADE model. These findings suggest that dominant genetic effects are present. Hence, we select the DE model as our preferred polygenic model. It does not perform significantly worse than the less parsimonious ADE model. Moreover, it shows a slightly better fit according to the standard criteria. In general, a lower AIC/BIC indicates better model fit. The results of the DE model suggest a heritability of patience of roughly one quarter. In other words, we find that differences in genetics account for 23 percent of the overall variation in general patience across individuals. Additive genetic effects are not supported by the data. Consequently, the largest proportion of variance is attributed to unique environmental influences (E). They account for approximately three quarters of the total variation in patience. However, it is important to keep in mind that the measurement error is also included in E.

notion that the ADE model suits the data better than the ACE model.

[17]The Likelihood ratio tests are performed unter the null hypothesis that the more parsimonious model is not significantly worse than the ADE model. The original p-values obtained from these tests were too conservative. According to Dominicus et al. (2006), we made the recommended adjustments and calculated the appropriate values.

Table 4.5: Results of the biometric analysis of patience

	LL	df	LRT	Δdf	p-value	AIC	BIC	A	D	C	E
Saturated model	−4155.544	18				8347.088	8442.46				
ADE	−4167.294	6	23.499	12	0.024†	8346.588	8378.378	0.000	0.234	—	0.766
								(0.000-0.000)	(0.162-0.306)		(0.694-0.838)
AE	−4170.332	5	6.077	1	<.01‡	8350.664	8377.156	0.194	—	—	0.806
								(0.127-0.261)			(0.739-0.873)
DE	−4167.294	5	<.01	1	>.10‡	8344.588	8371.08	—	0.234	—	0.766
									(0.162-0.306)		(0.694-0.838)

Notes: Results from the variance component analysis. LL=log Likelihood, df=degrees of freedom, LRT=Likelihood ratio test statistic, Δdf=difference in degrees of freedom, AIC=Akaike's information criterion, BIC=Sample size adjusted Bayesian's information criterion. † Compared to the saturated model. ‡ Compared to the ADE model. Adjusted p-values (Dominicus et al., 2006). 95% confidence intervals in parentheses.

In the following, we present the results from different sample stratifications. First of all, we stratify by gender. According to Table 4.2, roughly 56% of all twins in our sample are female. On average, males are slightly more patient than females (5.86 vs. 5.67). Running a t-test, the difference between these two means is significant at the 5% level. For female twins, the results from the biometric analysis are reported in Panel A of Table 4.6. For male twins, the results are reported in Panel B of Table 4.6. All variance component models control for age. Again, the DE models have the best fit in comparison to the other polygenic models. For female twins, the heritability of patience is about 20 percent. For their male counterparts, the genetic variation in patience accounts for 29 percent of the total trait variation.[18] Our findings are similar to the results from Cronqvist and Siegel (2015). Using the individual's savings rate as proxy for time preference, they find that the saving behavior among males is more attributable to genetic factors than the saving behavior among females (35% and 23%, respectively). However, the confidence intervals of our estimates of genetic non-additivity (D) show a significant overlap. The heritability coefficients for male and female individuals do not differ at the 5% level.

[18]The intraclass correlation coefficients (Spearman) are 0.213 and −0.010 for female MZ twin pairs and DZ twin pairs, respectively. The intraclass correlation coefficients (Spearman) are 0.277 and 0.024 for male MZ twin pairs and DZ twin pairs, respectively. Whereas the MZ twin pair correlations are always significant at the 1% level, the DZ twin pair correlations are not statistically significant. For both female and male twins, the MZ and DZ correlation coefficients differ significantly at the 1% level. The ACE models are continuously outperformed by the ADE models by AIC and BIC.

Table 4.6: Results of the biometric analysis of patience by sex

	LL	df	LRT	Δdf	p-value	AIC	BIC	A	D	C	E
Panel A: Female twins											
Saturated model	−2372.275	14				4772.551	4838.667				
ADE	−2376.775	5	11.671	9	0.437†	4763.551	4787.164	0.000	0.198	—	0.802
								(0.000-0.000)	(0.103-0.292)		(0.708-0.897)
AE	−2378.111	4	2.6706	1	0.051‡	4764.221	4783.112	0.165	—	—	0.835
								(0.077-0.253)			(0.747-0.923)
DE	−2376.775	4	<.01	1	>.10‡	4761.551	4780.441	—	0.198	—	0.802
									(0.103-0.292)		(0.708-0.897)
Panel B: Male twins											
Saturated model	−1774.455	14				3576.909	3639.522				
ADE	−1784.608	5	20.307	9	0.016†	3579.216	3601.578	0.000	0.290	—	0.710
								(0.000-0.000)	(0.180-0.401)		(0.599-0.820)
AE	−1786.672	4	4.1286	1	0.021‡	3581.345	3599.234	0.235	—	—	0.765
								(0.130-0.339)			(0.661-0.870)
DE	−1784.608	4	<.01	1	>.10‡	3577.216	3595.106	—	0.290	—	0.710
									(0.180-0.401)		(0.599-0.820)

Notes: Results from the variance component analysis. LL=log Likelihood, df=degrees of freedom, LRT=Likelihood ratio test statistic, Δdf= difference in degrees of freedom, AIC=Akaike's information criterion, BIC=Sample size adjusted Bayesian's information criterion. † Compared to the saturated model. ‡ Compared to the ADE model. Adjusted p-values (Dominicus et al., 2006). 95% confidence intervals in parentheses.

Moreover, we stratify by birth cohorts. The mean level of patience is 5.51 for children aged 10-12 years, 5.76 for adolescents aged 16-18 years and 6.01 for young adults aged 22-25 years. We conduct a one-way ANOVA and a Kruskal-Wallis H test to analyze if significant differences in patience exist among the three birth cohorts. The results obtained from the one-way ANOVA show a statistically significant difference in means between age groups ($F(2,2953)=8.78$, $p < .001$). In particluar, the Tukey post-hoc test reveals that patience is significantly higher in the oldest birth cohort (1990-1993) compared to the youngest birth cohort (2003/2004) ($p < .001$). Supporting the findings from the one-way ANOVA, the nonparametric Kruskal-Wallis H test shows a statistically significant difference in patience between the three birth cohorts, $\chi^2(2) = 15.477$, $p < .001$.

Separate biometric analyses are conducted for each birth cohort.[19] The results are reported in Table 4.7. All variance component models control for age and sex. In Panel A of Table 4.7, the results for the youngest birth cohort (2003/04) are reported. According to the DE model, genetic differences explain about 31 percent of the total variation in patience across the members of this age group. With respect to the other birth cohorts, the genetic influence is smaller. Panels B and C of Table 4.7 show the results for the birth cohort born in 1997/98 and for the oldest birth cohort (1990-93), respectively. For both cohorts, neither the AE nor the DE model is statistically worse than the ADE model. Although the AE model is not significantly worse than the ADE model ($p > .10$), we select the DE model as our preferred model because it gives the best fit by the AIC (BIC). We obtain a heritability coefficient of roughly .17 (.20) in the DE models that include individuals aged 16-18 (22-25) years. Hence, at young age, the proportion of genetic variation in patience seems to be larger than at later stages in life. However, we do not identify significant differences in heritability across the groups. Focusing on the considerable overlaps of the confidence intervals, the coefficients of D do not differ at the 5% level.

[19]The intraclass correlation coefficients (Spearman) are 0.299 and -0.002 for MZ twin pairs and DZ twin pairs in the youngest birth cohort (2003/2004), respectively. The intraclass correlation coefficients (Spearman) are 0.164 and -0.025 for MZ twin pairs and DZ twin pairs in the mid birth cohort (1997/1998), respectively. The intraclass correlation coefficients (Spearman) are 0.247 and 0.026 for MZ twin pairs and DZ twin pairs in the oldest birth cohort (1990-1993), respectively. Whereas the MZ twin pair correlations are always significant at the 1% level, the DZ twin pair correlations are not statistically significant. Across all birth cohorts, the MZ and DZ correlation coefficients differ significantly at least at the 5% level. The ACE models are continuously outperformed by the ADE models by AIC and BIC.

Table 4.7: Results of the biometric analysis of patience by birth cohorts

	LL	df	LRT	Δdf	p-value	AIC	BIC	A	D	C	E
Panel A: Birth cohort 2003/04											
Saturated model	−1459.305	18				2954.61	3030.292				
ADE	−1466.407	6	14.205	12	0.288†	2944.815	2970.042	0.000	0.312	—	0.688
								(0.000-0.000)	(0.182-0.442)		(0.558-0.818)
AE	−1468.689	5	4.5627	1	0.016‡	2947.377	2968.4	0.240	—	—	0.760
								(0.117-0.363)			(0.637-0.883)
DE	−1466.407	5	<.01	1	>.10‡	2942.815	2963.837	—	0.312	—	0.688
									(0.182-0.442)		(0.558-0.818)
Panel B: Birth cohort 1997/98											
Saturated model	−1367.334	18				2770.669	2847.133				
ADE	−1379.485	6	24.301	12	0.019†	2770.97	2796.458	0.000	0.173	—	0.827
								(0.000-0.000)	(0.044-0.301)		(0.699-0.956)
AE	−1380.157	5	1.3438	1	0.123‡	2770.313	2791.554	0.137	—	—	0.863
								(0.021-0.254)			(0.746-0.979)
DE	−1379.485	5	<.01	1	>.10‡	2768.97	2790.21	—	0.173	—	0.827
									(0.044-0.301)		(0.699-0.956)
Panel C: Birth cohort 1990-93											
Saturated model	−1299.373	18				2634.746	2709.341				
ADE	−1304.915	6	11.083	12	0.523†	2621.829	2646.694	0.000	0.204	—	0.796
								(0.000-0.000)	(0.089-0.319)		(0.681-0.911)
AE	−1305.311	5	0.7923	1	0.187‡	2620.622	2641.342	0.185	—	—	0.815
								(0.075-0.294)			(0.706-0.925)
DE	−1304.915	5	<.01	1	>.10‡	2619.829	2640.55	—	0.204	—	0.796
									(0.089-0.319)		(0.681-0.911)

Notes: Results from the variance component analysis. LL=log Likelihood, df=degrees of freedom, LRT=Likelihood ratio test statistic, Δdf=difference in degrees of freedom, AIC=Akaike's information criterion, BIC=Sample size adjusted Bayesian's information criterion. † Compared to the saturated model. ‡ Compared to the ADE model. Adjusted p-values (Dominicus et al., 2006). 95% confidence intervals in parentheses.

Before we further discuss our main findings, we make some comments on the most important assumptions underlying the polygenic models from above. First, the equal environment assumption (EEA) states that environmentally caused similarity is the same for MZ and DZ twin pairs. This seems plausible since both twin types share the womb at the same time, are the same age and are raised together in the same family environment. But this assumption is not free of criticism. Some researchers cast doubt on the general validity of the EEA. They argue that MZ twins are, in fact, treated more similarly by their environments (parents, teachers, peers, ...) than DZ twins (see, e.g., Joseph, 1998). This would increase their correlations relative to the correlations of DZ twins. As a result, the genetic effects would be overestimated and the shared environmental effect would be underestimated (Rijsdijk and Sham, 2002). However, many studies exist that have rigorously tested the EEA (see, e.g., Plomin et al., 2013). Focusing on personality traits, there is evidence that it can be considered as a valid assumption of the standard twin method (e.g., Borkenau et al., 2002). More specifically, research has shown that any potential bias due to violations of the EEA is not of first order importance (Bouchard, 1998). Moreover, Bouchard et al. (1990) estimated similar heritability coefficients, regardless of whether using MZ twins reared together or apart. Thus, although MZ twins are likely to grow up in more similar environments than DZ twins, this is not the cause of their greater similarity regarding a specific trait but, rather, a consequence of their genetic identity (Martin et al., 1997). Unfortunately, the TwinLife study does not include twins that were reared apart. This would have allowed us to directly compare the correlations for patience of MZ twins reared apart and those reared together. Similar correlations between both groups would have lent additional support to the validity of this assumption.

Second, we test the assumption of random mating of mothers and fathers. In comparison to the vast majority of twin studies, we have the possibility to analyze the correlation of patience between parents. The parents of the twins were asked to rate their level of patience on the same 11-point scale. We merged their answers with the corresponding answers provided by the twins. Unfortunately, our sample size shrinks drastically because many parents did not answer the question on patience. We end up with 785 twin pairs (345 MZ twin pairs, 440 DZ twin pairs) where full information on parental patience is present. Hence, the sample composition largely differs from the set of observations used for the previous biometric analysis. However, the results point towards random mating. We find a *negative* correlation between maternal and paternal patience that is not statistically significant ($\rho = -0.02$, $p > .10$). In general, there is very limited evidence on assortative mating regarding time preference. To the best of our knowledge,

Gauly (2017) is the only study explicitly addressing this issue. Her findings suggest a weakly significant correlation which is also negative. Hence, investigating the role of assortative mating in the field of time discounting seems to be a promising area for future research.

In general, standard twin models assume the lack of any gene-environment interactions. In fact, it is (almost) impossible to provide conclusive evidence that this assumption holds completely true. To test this assumption properly, a dataset incredibly rich in information would be needed. Depending on the subject under investigation, longitudinal trait data, information on non-adoptees and adoptees or very precise measures of the environment may be required (see Rijsdijk and Sham (2002) for a critical discussion of the underlying assumptions). However, as it is common practice in the twin literature, we assume that the absence of gene-environment interactions holds true. All in all, we are confident that our biometrical analyses are not biased due to fundamental violations of the standard assumptions of the twin models.

4.5 Discussion and conclusion

The main purpose of this study is to provide evidence on the general formation of time preference. In particular, we focus on the degree to which time preference is heritable (broad-sense heritability). We rely on novel twin data from the German TwinLife project. It is a large-scale twin study in Germany. We use self-reported patience as a meaningful proxy for individual time discounting. Standard biometric analyses are conducted. According to the model selection process, the DE model gives the best fit. In this model, the dominant genetic effect is estimated around 23 percent. An additive genetic effect is not supported by the data. Hence, almost one quarter of the total variance in patience is attributed to genetic influences. The largest proportion of variance is attributed to the unique environmental effect (E). In line with the previous literature on the genetic variation in economic preferences and behavior patterns, the shared environment effect (C) is negligible (see, e.g., Cronqvist and Siegel, 2015; Cesarini et al., 2009; Zyphur et al., 2009; Zhong et al., 2009). Although a series of studies has frequently estimated ACE models, they only find marginal or even no contribution of C at all (e.g., Cronqvist and Siegel, 2015; Cesarini et al., 2009). Stratifying by gender, the genetic variation in patience is larger for male than for female individuals (29% and 20%, respectively). After applying the sample stratification by birth cohorts, the largest estimate of genetic influence is found among members of the youngest cohort (.31). However, meaningful differences between genders or birth cohorts do not exist.

Unlike the findings from Anokhin et al. (2011) and Cronqvist and Siegel (2015), this is the

first paper that reveals a substantial contribution of the dominant genetic component (D) to the overall variation in time preference. According to our data, MZ correlations for patience are more than twice the size of the DZ correlations. This is a strong indication for non-additive genetic variation to be present. Based on their findings on risk assessment, Cesarini et al. (2009) come to the conclusion that "there is probably nonadditive variation in personality and attitudes. The low DZ correlations we observe suggest that a similar situation obtains for economic preferences" (p. 833-834). Again, with regard to the genetics of risk preferences, Zyphur et al. (2009) and Zhong et al. (2009) find similar results. Both conclude that the attitude towards risk seems to be a non-additive trait which is genetically coded in a dominant/recessive way.

Inevitably, this raises the question about candidate genes which are related to time discounting. In recent years, molecular genetics has spared no efforts to disentangle the complex genetic architecture of human traits. For example, Eisenberg et al. (2007) and Carpenter et al. (2011) found that the Dopamine Receptor D4 gene (DRD4) predicts impulsivity and time preference, respectively. Furthermore, links between serotonin (5-hydroxytryptamine, 5-HT) activity and personality traits such as impulsivity have been established (e.g., Carver and Miller, 2006; Oades et al., 2008; Miyazaki et al., 2012). We have to admit that most of these studies focus on impulsivity and not explicitly on (long-term) time preference/patience. However, although impulsivity is a slightly different concept in the context of time discounting, it is directly related to patience (see Kalenscher and Pennartz (2008) for an extensive review of the neuroeconomics of intertemporal decision-making). For example, Gauly (2017) shows a significant negative intrapersonal correlation between general patience and impulsiveness. Overall, there is some evidence that dominant/recessive genes account for differences in serotonin and/or dopamine levels that are further related to time discounting.

Our results imply that researchers should be aware of genetic non-additivity. Especially in the context of economic preferences, the additive genetic component (A) may not always be the main source of genetic variation. In case of sufficiently large differences in trait correlations across twin types, biometric models that include D seem to be the appropriate choice. But more research is needed to provide conclusive evidence on the role of non-additivity in the genetics of time preference. From an economist's point of view, however, the relative contribution of A and D to the overall genetic variation may be of minor importance. With the nature-nurture debate in mind, we focus on disentangling the aggregate genetic variance from the variance that is caused by environmental influences. In fact, the estimated impact of the environment is probably what matters most from a behavior change perspective. The smaller the extent to

which time preferences are innate, the larger the potential for modifications of people's preference structure.

However, our study has some limitations. As already discussed above, adherence of our data to the technical assumptions of the classical twin model could be better. Unfortunately, we are also not able to exploit the longitudinal design of the TwinLife project. While writing this paper, only data on the first partial wave with information on time preference were available for researchers. However, for future research, it could be promising to expand the cross-sectional analysis with panel data. This would allow for further investigation of possible changes in the degree of heritability over the twins' lifetime.

A further limitation is that the survey question on general patience is a rather universal measure of a person's time preference. But the possibility exists that time preferences may be domain-specific (Tsukayama and Duckworth, 2010). Thus, the degree of heritability of time preference may vary across different domains (e.g., monetary vs. health domain). For instance, Bickel et al. (1999) show that discount rates in the health domain are higher than discount rates in the monetary domain. Researchers are strongly encouraged to dive into the genetics of time preference in different domains.

Yet another shortcoming is that we cannot completely rule out misreporting among children. Our youngest cohort consists of twins aged 10-12 years. The survey question on general patience is measured on a standard 11-point scale for all respondents. In general, the possibility should be considered that some children may have experienced difficulties in answering this question appropriately (Mellor and Moore, 2014). Thinking about your own personality and providing the corresponding answer on a Likert scale requires a sufficiently high level of abstract thinking of these children. Thus, it is likely that at least some twins of this particular birth cohort have not fully developed their abstract thinking skills at this stage of life (Maćkiewicz and Cieciuch, 2016). However, we are confident that potential measurement bias is negligible. The overall response pattern to the question on self-assessed patience is basically the same across all three birth cohorts. Moreover, we exclude all twins falling into the respective age range and re-estimate the corresponding polygenic models. Although we lose a substantial number of observations (roughly one third of the sample size), we still obtain a heritability coefficient of around .20. However, using a uniform measurement of patience for all respondents ensures consistency and comparability across birth cohorts.

Despite the limitations mentioned above, we conclude that our findings provide evidence that time preferences are considerably heritable. However, the majority of variation in patience can

be attributed to unique environmental factors (and random error). From a policy perspective, this is an interesting starting point. We infer that people can effectively be targeted by public policies that intend to make them more future-oriented. For instance, traits or preferences that are widely heritable would only leave a narrow scope for intervening actions. Appropriate interventions may include information campaigns about the adverse health consequences of smoking, a proper design of commitment devices to save more money for the future or school subsidies to show children that today's investments in education will result in higher earnings in the (distant) future. Hence, our results indicate that public policy interventions with the aim of nudging people towards more future orientation and away from rather seductive instant gratification may have a good prospect of success. Since the literature on the heritability of time preference is still scarce, we want to encourage future research to dedicate more time on the investigation of the genetics of time discounting. However, it is already fair to say that the role of genetics in intertemporal decision-making should no longer be ignored.

4.6 Appendix

Maximum Likelihood (ML) Method:

- Bivariate normal density function for independent twin pair $i = 1, ..., N$:

  $f(Pat) = \frac{1}{2\pi}(\det\Sigma)^{-\frac{1}{2}}\exp[-\frac{1}{2}(Pat_i - \mu_i)^T\Sigma^{-1}(Pat_i - \mu_i)]$,

  where

  $$\sum = \begin{pmatrix} \sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2 & \sigma_A^2 + \sigma_D^2 + \sigma_C^2 \\ \sigma_A^2 + \sigma_D^2 + \sigma_C^2 & \sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2 \end{pmatrix}$$

  for MZ twins, and

  $$\sum = \begin{pmatrix} \sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2 & 0.5\sigma_A^2 + 0.25\sigma_D^2 + \sigma_C^2 \\ 0.5\sigma_A^2 + 0.25\sigma_D^2 + \sigma_C^2 & \sigma_A^2 + \sigma_D^2 + \sigma_C^2 + \sigma_E^2 \end{pmatrix}$$

  for DZ twins.

- Likelihood function:

  $L(Pat|\mu, \Sigma) = \prod_{i=1}^{N} f(Pat)$

- Log-likelihood function:

  $LL(Pat|\mu, \Sigma) = \sum_{i=1}^{N} \ln L(Pat|\mu, \Sigma) =$

  $\sum_{i=1}^{N} \left(-\ln(2\pi) - \frac{1}{2}\ln(\det\Sigma) - \frac{1}{2}(Pat_i - \mu_i)^T\Sigma^{-1}(Pat_i - \mu_i)\right)$

Table 4.8: Results of the biometric analysis of patience - twins aged 16-18 years excluded

	LL	df	LRT	Δdf	p-value	AIC	BIC	A	D	C	E
Saturated model	−2769.03	18				5574.061	5661.684				
ADE	−2778.251	6	18.442	12	0.103†	5568.503	5597.71	0.000	0.259	—	0.741
								(0.000-0.000)	(0.172-0.345)		(0.655-0.828)
AE	−2778.251	5	4.693	1	0.030‡	5571.195	5595.535	0.217	—	—	0.783
								(0.134-0.299)			(0.701-0.866)
DE	−2778.251	5	<.01	1	>.10‡	5566.503	5590.842	—	0.259	—	0.741
									(0.172-0.345)		(0.655-0.828)

Notes: Results from the variance component analysis (n=1,922). LL=log Likelihood, df=degrees of freedom, LRT=Likelihood ratio test statistic, Δdf=difference in degrees of freedom, AIC=Akaike's information criterion, BIC=Sample size adjusted Bayesian's information criterion. † Compared to the saturated model. ‡ Compared to the ADE model. Adjusted p-values (Dominicus et al., 2006). 95% confidence intervals in parentheses.

Table 4.9: Polygenic model selection - ACE vs. ADE

	LL	df	LRT	Δdf	p-value	AIC	BIC
Saturated model	−4155.544	18				8347.088	8442.46
ACE	−4170.332	6	29.576	12	0.003†	8352.664	8384.455
ADE	−4167.294	6	23.499	12	0.024†	8346.588	8378.378

Notes: Results from the variance component analysis. LL=log Likelihood, df=degrees of freedom, LRT=Likelihood ratio test statistic, Δdf=difference in degrees of freedom, AIC=Akaike's information criterion, BIC=Sample size adjusted Bayesian's information criterion. † Compared to the saturated model. Comparing the ACE with the ADE model, the ADE model gives lower AIC and BIC.

Bibliography

Adams, J. (2009). The Role of Time Perspective in Smoking Cessation Amongst Older English Adults. *Health Psychology 28*(5), 529–534.

Adams, J. and D. Nettle (2009). Time perspective, personality and smoking, body mass, and physical activity: An empirical study. *British Journal of Health Psychology 14*(1), 83–105.

Ai, C. and E. C. Norton (2003). Interaction terms in logit and probit models. *Economics Letters 80*(1), 123–129.

Ainslie, G. and N. Haslam (1992). Hyperbolic Discounting. In G. Loewenstein and J. Elster (Eds.), *Choice Over Time*, pp. 57–92. New York: Russell Sage Foundation.

Akerlof, G. A. (1991). Procrastination and Obedience. *American Economic Review 81*(2), 1–19.

Almond, D. and J. Currie (2011). Killing Me Softly: The Fetal Origins Hypothesis. *Journal of Economic Perspectives 25*(3), 153–172.

Anderson, L. R. and J. M. Mellor (2008). Predicting health behaviors with an experimental measure of risk preference. *Journal of Health Economics 27*(5), 1260–1274.

Andreoni, J. and C. Sprenger (2012). Risk Preferences Are Not Time Preferences. *American Economic Review 102*(7), 3357–3376.

Anger, S., M. Kvasnicka, and T. Siedler (2011). One last puff? Public smoking bans and smoking behavior. *Journal of Health Economics 30*(3), 591–601.

Angrist, J. D. and J.-S. Pischke (2009). *Mostly Harmless Econometrics: An Empiricist's Companion*. Princeton University Press.

Anokhin, A. P., S. Golosheykin, J. D. Grant, and A. C. Heath (2011). Heritability of delay discounting in adolescence: a longitudinal twin study. *Behavior Genetics 41*(2), 175–183.

Arrondel, L. (2013). Are "daddy's boys" just as rich as daddy? The transmission of values between generations. *The Journal of Economic Inequality 11*(4), 439–471.

Ayduk, O., R. Mendoza-Denton, W. Mischel, G. Downey, P. K. Peake, and M. Rodriguez (2000). Regulating the interpersonal self: Strategic self-regulation for coping with rejection sensitivity. *Journal of Personality and Social Psychology 79*(5), 776–792.

Baker, F., M. W. Johnson, and W. K. Bickel (2003). Delay Discounting in Current and Never-Before Cigarette Smokers: Similarities and Differences Across Commodity, Sign, and Magnitude. *Journal of Abnormal Psychology 112*(3), 382–392.

Baltagi, B. H. and Q. Li (1990). A lagrange multiplier test for the error components model with incomplete panels. *Econometric Reviews 9*(1), 103–107.

Bantle, C. and J. P. Haisken-DeNew (2002). Smoke signals: The intergenerational transmission of smoking behavior. *DIW Berlin: Discussion Papers Nr. 277*.

Barker, D. J. P. (1990). The Fetal and Infant Origins of Adult Disease. *The BMJ 301*(6761), 1111.

Barker, D. J. P. (1995). Fetal origins of coronary heart disease. *British Medical Journal 311*(6998), 171–174.

Barker, D. J. P. (1997). Maternal Nutrition, Fetal Nutrition, and Disease in Later Life. *Nutrition 13*(9), 807–813.

Barker, D. J. P. (1998). In utero programming of chronic disease. *Clinical Science 95*(2), 115–128.

Barlow, P., M. McKee, A. Reeves, G. Galea, and D. Stuckler (2017). Time-discounting and tobacco smoking: a systematic review and network analysis. *International Journal of Epidemiology 46*(3), 860–869.

Baron, R. M. and D. A. Kenny (1986). The Moderator-Mediator Variable Distinction in Social Psychological Research: Conceptual, Strategic, and Statistical Considerations. *Journal of Personality and Social Psychology 51*(6), 1173–1182.

Barratt, E. S. (1959). Anxiety and Impulsiveness Related to Psychomotor Efficiency. *Perceptual and Motor Skills 9*(3), 191–198.

Barsky, R. B., F. T. Juster, M. S. Kimball, and M. D. Shapiro (1997). Preference Parameters and Behavioral Heterogeneity: An Experimental Approach in the Health and Retirement Study. *Quarterly Journal of Economics 112*(2), 537–579.

Becker, G. S. and C. B. Mulligan (1997). The Endogenous Determination of Time Preference. *Quarterly Journal of Economics 112*(3), 729–758.

Becker, G. S. and K. M. Murphy (1988). A Theory of Rational Addiction. *Journal of Political Economy 96*(4), 675–700.

Benjamin, D. J., D. Cesarini, M. J. H. M. van der Loos, C. T. Dawes, P. D. Koellinger, P. K. E. Magnusson, C. F. Chabris, D. Conley, D. Laibson, M. Johannesson, and P. M. Visscher (2012). The genetic architecture of economic and political preferences. *Proceedings of the National Academy of Sciences of the United States of America 109*(21), 8026–8031.

Bettinger, E. and R. Slonim (2007). Patience among children. *Journal of Public Economics 91*(1-2), 343–363.

Bickel, W. K., A. L. Odum, and G. J. Madden (1999). Impulsivity and cigarette smoking: delay discounting in current, never, and ex-smokers. *Psychopharmacology 146*(4), 447–454.

Binder, S. and R. Nuscheler (2017). Risk-taking in vaccination, surgery, and gambling environments: Evidence from a framed laboratory experiment. *Health Economics 26*(S3), 76–96.

Bisin, A. and T. Verdier (2000). "Beyond the Melting Pot": Cultural Transmission, Marriage, and the Evolution of Ethnic and Religious Traits. *Quarterly Journal of Economics 115*(3), 955–988.

Bisin, A. and T. Verdier (2001). The Economics of Cultural Transmission and the Dynamics of Preferences. *Journal of Economic Theory 97*(2), 298–319.

Björklund, A., M. Jäntti, and G. Solon (2007). Nature and Nurture in the Intergenerational Transmission of Socioeconomic Status: Evidence from Swedish Children and Their Biological and Rearing Parents. *The B. E. Journal of Economic Analysis and Policy 7*(2), Article 4.

Blackwell, D. L., M. D. Hayward, and E. M. Crimmins (2001). Does childhood health affect chronic morbidity in later life? *Social Science & Medicine 52*(8), 1269–1284.

Blecher, E. (2008). The impact of tobacco advertising bans on consumption in developing countries. *Journal of Health Economics 27*(4), 930–942.

Böhm, R., C. Betsch, and L. Korn (2016). Selfish-rational non-vaccination: Experimental evidence from an interactive vaccination game. *Journal of Economic Behavior & Organization 131*(Part B), 183–195.

Borkenau, P., R. Riemann, A. Angleitner, and F. M. Spinath (2002). Similarity of childhood experiences and personality resemblance in monozygotic and dizygotic twins: a test of the equal environments assumption. *Personality and Individual Differences 33*(2), 261–269.

Bouchard, T. J. (1998). Genetic and Environmental Influences on Adult Intelligence and Special Mental Abilities. *Human Biology 70*(2), 257–279.

Bouchard, T. J., D. T. Lykken, M. McGue, N. L. Segal, and A. Tellegen (1990). Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart. *Science 250*(4978), 223–228.

Boutwell, B. B. and K. M. Beaver (2010). The Intergenerational Transmission of Low Self-control. *Journal of Research in Crime and Delinquency 47*(2), 174–209.

Bowles, S. (1998). Endogenous Preferences: The Cultural Consequences of Markets and other Economic Institutions. *Journal of Economic Literature 36*(1), 75–111.

Bradford, W. D. (2010). The Association Between Individual Time Preferences and Health Maintenance Habits. *Medical Decision Making 30*(1), 99–112.

Brito, D. L., E. Sheshinski, and M. D. Intriligator (1991). Externalities and compulsory vaccinations. *Journal of Public Economics 45*(1), 69–90.

Brown, H. and J. Adams (2013). The role of time preference in smoking cessation: a longitudinal analysis of data from the Household Income and Labour Dynamics of Australia survey, 2001-08. *Addiction 108*(1), 186–192.

Brown, H. and M. van der Pol (2014). The Role of Time Preferences in the Intergenerational Transfer of Smoking. *Health Economics 23*(12), 1493–1501.

Brown, H. and M. van der Pol (2015). Intergenerational transfer of time and risk preferences. *Journal of Economic Psychology 49*, 187–204.

Bruin, J. E., H. C. Gerstein, and A. C. Holloway (2010). Long-Term Consequences of Fetal and Neonatal Nicotine Exposure: A Critical Review. *Toxicological Sciences 116*(2), 364–374.

Brunello, G., M. Fort, N. Schneeweis, and R. Winter-Ebmer (2016). The Causal Effect of Education on Health: What is the Role of Health Behaviors? *Health Economics 25*(3), 314–336.

Callen, M. (2015). Catastrophes and time preference: Evidence from the Indian Ocean Earthquake. *Journal of Economic Behavior & Organization 118*, 199–214.

Caplan, G. (1964). *Principles of preventive psychiatry.* New York: Basic Books.

Carpenter, C. and P. J. Cook (2008). Cigarette taxes and youth smoking: New evidence from national, state, and local Youth Risk Behavior Surveys. *Journal of Health Economics 27*(2), 287–299.

Carpenter, J. P., J. R. Garcia, and J. K. Lum (2011). Dopamine receptor genes predict risk preferences, time preferences, and related economic choices. *Journal of Risk and Uncertainty 42*(3), 233–261.

Carver, C. S. and C. J. Miller (2006). Relations of serotonin function to personality: Current views and a key methodological issue. *Psychiatry Research 144*(1), 1–15.

Case, A., A. Fertig, and C. Paxson (2005). The lasting impact of childhood health and circumstance. *Journal of Health Economics 24*(2), 365–389.

Case, A., D. Lubotsky, and C. Paxson (2002). Economic Status and Health in Childhood: The Origins of the Gradient. *American Economic Review 92*(5), 1308–1334.

Case, A. and C. Paxson (2001). Mothers and others: who invests in children's health? *Journal of Health Economics 20*(3), 301–328.

Case, A. and C. Paxson (2002). Parental Behavior And Child Health. *Health Affairs 21*(2), 164–178.

Case, A. and C. Paxson (2010). Causes and consequences of early-life health. *Demography 47*(Supplement 1), S65–S85.

Castles, A., E. K. Adams, C. L. Melvin, C. Kelsch, and M. L. Boulton (1999). Effects of smoking during pregnancy: Five meta-analyses. *American Journal of Preventive Medicine 16*(3), 208–215.

Cawley, J. and C. J. Ruhm (2011). The Economics of Risky Health Behaviors. In M. V. Pauly, T. G. Mcguire, and P. P. Barros (Eds.), *Handbook of Health Economics*, Volume 2, pp. 95–199. Elsevier B. V.

Centers for Disease Control and Prevention (CDC) (2018a). Fast Facts and Fact Sheets: Health Effects of Cigarette Smoking. `https://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/index.htm` [Accessed 19 March 2018].

Centers for Disease Control and Prevention (CDC) (2018b). MMR and MMRV Vaccine Composition and Dosage. `https://www.cdc.gov/vaccines/vpd/mmr/hcp/about.html` [Accessed 21 February 2018].

Cesarini, D., C. T. Dawes, M. Johannesson, P. Lichtenstein, and B. Wallace (2009). Genetic Variation in Preferences for Giving and Risk Taking. *Quarterly Journal of Economics 124*(2), 809–842.

Cesarini, D., M. Johannesson, P. K. E. Magnusson, and B. Wallace (2012). The Behavioral Genetics of Behavioral Anomalies. *Management Science 58*(1), 21–34.

Chaloupka, F. J. and K. E. Warner (2000). The Economics of Smoking. In A. J. Culyer and J. P. Newhouse (Eds.), *Handbook of Health Economics*, Volume 1, pp. 1539–1627. New York: Elsevier Science B. V.

Chapman, G. B., N. T. Brewer, E. J. Coups, S. Brownlee, H. Leventhal, and E. A. Leventhal (2001). Value for the future and preventive health behavior. *Journal of Experimental Psychology: Applied 7*(3), 235–250.

Chapman, G. B. and E. J. Coups (1999). Time Preferences and Preventive Health Behavior: Acceptance of the Influenza Vaccine. *Medical Decision Making 19*(3), 307–314.

Chassin, L., C. Presson, D.-C. Seo, S. J. Sherman, J. Macy, R. J. Wirth, and P. Curran (2008). Multiple Trajectories of Cigarette Smoking and the Intergenerational Transmission of Smoking: A Multigenerational, Longitudinal Study of a Midwestern Community Sample. *Health Psychology 27*(6), 819–828.

Chuang, Y. and L. Schechter (2015). Stability of experimental and survey measures of risk, time, and social preferences: A review and some new results. *Journal of Development Economics 117*, 151–170.

Cnattingius, S. (2004). The epidemiology of smoking during pregnancy: Smoking prevalence, maternal characteristics, and pregnancy outcomes. *Nicotine & Tobacco Research 6*(Supplement 2), S125–S140.

Conti, G., J. Heckman, and S. Urzua (2010). The Education-Health Gradient. *American Economic Review 100*(2), 234–238.

Coutlee, C. G., C. S. Politzer, R. H. Hoyle, and S. A. Huettel (2014). An Abbreviated Impulsiveness Scale Constructed Through Confirmatory Factor Analysis of the Barratt Impulsiveness Scale Version 11. *Archives of Scientific Psychology 2*(1), 1–12.

Cronqvist, H. and S. Siegel (2015). The Origins of Savings Behavior. *Journal of Political Economy 123*(1), 123–169.

Currie, A., M. A. Shields, and S. W. Price (2007). The child health/family income gradient: Evidence from England. *Journal of Health Economics 26*(2), 213–232.

Currie, J. (2009). Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development. *Journal of Economic Literature 47*(1), 87–122.

Currie, J. (2011). Inequality at Birth: Some Causes and Consequences. *American Economic Review 101*(3), 1–22.

Currie, J. and D. Almond (2011). Human capital development before age five. In D. Card and O. Ashenfelter (Eds.), *Handbook of Labor Economics*, Volume 4, Part B, Chapter 15, pp. 1315–1486. Amsterdam: Elsevier B. V.

Currie, J. and B. C. Madrian (1999). Health, health insurance and the labor market. In O. Ashenfelter and D. Card (Eds.), *Handbook of Labor Economics*, Volume 3, Part C, Chapter 50, pp. 3309–3416. Amsterdam: Elsevier Science.

Currie, J. and M. Stabile (2003). Socioeconomic Status and Child Health: Why Is the Relationship Stronger for Older Children? *American Economic Review 93*(5), 1813–1823.

Currie, J., M. Stabile, P. Manivong, and L. L. Roos (2010). Child Health and Young Adult Outcomes. *The Journal of Human Resources 45*(3), 517–548.

Currie, J. and T. Vogl (2013). Early-Life Health and Adult Circumstance in Developing Countries. *Annual Review of Economics 5*, 1–36.

Cutler, D. M. and A. Lleras-Muney (2010). Understanding differences in health behaviors by education. *Journal of Health Economics 29*(1), 1–28.

Cutler, D. M., A. Lleras-Muney, and T. Vogl (2011). Socioeconomic Status and Health: Dimensions and Mechanisms. In S. Glied and P. C. Smith (Eds.), *The Oxford Handbook of Health Economics*. Oxford Handbooks.

Dale, P. S., M. G. Tosto, M. E. Hayiou-Thomas, and R. Plomin (2015). Why does parental language input style predict child language development? A twin study of gene-environment correlation. *Journal of Communication Disorders 57*, 106–117.

Danis, K., T. Georgakopoulou, T. Stavrou, D. Laggas, and T. Panagiotopoulos (2010). Predictors Of Childhood Vaccination Uptake: A Cross-sectional Study In Greece. *Procedia in Vaccinology 2*(1), 86–91.

Darden, M. and D. Gilleskie (2016). The Effects of Parental Health Shocks on Adult Offspring Smoking Behavior and Self-Assessed Health. *Health Economics 25*(8), 939–954.

Darwin, C. (1859). *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life*. London: J. Murray.

De Paola, M. (2013). The Determinants of Risk Aversion: The Role of Intergenerational Transmission. *German Economic Review 14*(2), 214–234.

de Walque, D. (2007). Does education affect smoking behaviors? Evidence using the Vietnam draft as an instrument for college education. *Journal of Health Economics 26*(5), 877–895.

DeCicca, P., D. Kenkel, and A. Mathios (2008). Cigarette taxes and the transition from youth to adult smoking: Smoking initiation, cessation, and participation. *Journal of Health Economics 27*(4), 904–917.

Dickman, S. J. (1990). Functional and Dysfunctional Impulsivity: Personality and Cognitive Correlates. *Journal of Personality and Social Psychology 58*(1), 95–102.

Dohmen, T., A. Falk, D. Huffman, and U. Sunde (2010). Are Risk Aversion and Impatience Related to Cognitive Ability? *American Economic Review 100*(3), 1238–1260.

Dohmen, T., A. Falk, D. Huffman, and U. Sunde (2012). The Intergenerational Transmission of Risk and Trust Attitudes. *Review of Economic Studies 79*(2), 645–677.

Dohmen, T., A. Falk, D. Huffman, U. Sunde, J. Schupp, and G. G. Wagner (2011). Individual Risk Attitudes: Measurement, Determinants, and Behavioral Consequences. *Journal of the European Economic Association 9*(3), 522–550.

Dominicus, A., A. Skrondal, H. K. Gjessing, N. L. Pedersen, and J. Palmgren (2006). Likelihood Ratio Tests in Behavioral Genetics: Problems and Solutions. *Behavior Genetics 36*(2), 331–340.

Ehrich, J., U. Grote, A. Gerber-Grote, and M. Strassburg (2016). The Child Health Care System of Germany. *The Journal of Pediatrics 177*(Supplement), S71–S86.

Ehrlich, I. and H. Chuma (1990). A Model of the Demand for Longevity and the Value of Life Extension. *Journal of Political Economy 98*(4), 761–782.

Eisenberg, D. T. A., J. MacKillop, M. Modi, J. Beauchemin, D. Dang, S. A. Lisman, J. K. Lum, and D. S. Wilson (2007). Examining impulsivity as an endophenotype using a behavioral approach: a DRD2 TaqI A and DRD4 48-bp VNTR association study. *Behavioral and Brain Functions 3*(2).

Emery, S., Y. Kim, Y. K. Choi, G. Szczypka, M. Wakefield, and F. J. Chaloupka (2012). The Effects of Smoking-Related Television Advertising on Smoking and Intentions to Quit Among Adults in the United States: 1999-2007. *American Journal of Public Health 102*(4), 751–757.

Evans, W. N., M. C. Farrelly, and E. Montgomery (1999). Do Workplace Smoking Bans Reduce Smoking? *American Economic Review 89*(4), 728–747.

Eysenck, S. B. G., P. R. Pearson, G. Easting, and J. F. Allsopp (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences 6*(5), 613–619.

Falagas, M. E. and E. Zarkadoulia (2008). Factors associated with suboptimal compliance to vaccinations in children in developed countries: a systematic review. *Current Medical Research and Opinion 24*(6), 1719–1741.

Federal Joint Committee (G-BA) (2016). Richtlinie des Gemeinsamen Bundesausschusses zur Jugendgesundheitsuntersuchung (Jugendgesundheitsuntersuchungs-Richtlinie). `https://www.g-ba.de/informationen/richtlinien/14/` [Accessed 26 February 2018].

Federal Joint Committee (G-BA) (2017). Richtlinie des Gemeinsamen Bundesausschusses über die Früherkennung von Krankheiten bei Kindern (Kinder-Richtlinie). `https://www.g-ba.de/informationen/richtlinien/15/` [Accessed 26 February 2018].

Federal Statistical Office (Destatis) (2014). Mikrozensus 2013 – Fragen zur Gesundheit – Rauchgewohnheiten der Bevölkerung. `https://www.destatis.de/DE/Publikationen/Thematisch/Gesundheit/Gesundheitszustand/Rauchgewohnheiten.html` [Accessed 19 October 2017].

Federal Statistical Office (Destatis) (2017). Smoking habits by age-groups and sex. `https://www.destatis.de/EN/FactsFigures/SocietyState/Health/HealthStatusBehaviourRelevantHealth/Tables/SmokingHabits.html` [Accessed 19 October 2017].

Fisher, R. A. (1918). The Correlation between Relatives on the Supposition of Mendelian Inheritance. *Transactions of the Royal Society of Edinburgh 52*(2), 399–433.

Frederick, S., G. Loewenstein, and T. O'Donoghue (2002). Time Discounting and Time Preference: A Critical Review. *Journal of Economic Literature 40*(2), 351–401.

Friedel, J. E., W. B. DeHart, G. J. Madden, and A. L. Odum (2014). Impulsivity and cigarette smoking: discounting of monetary and consumable outcomes in current and non-smokers. *Psychopharmacology 231*(23), 4517–4526.

Fuchs, V. R. (1982). Time Preference and Health: An Exploratory Study. In V. R. Fuchs (Ed.), *Economic Aspects of Health*, pp. 93–120. Chicago: University of Chicago Press.

Fuchs, V. R. (2000). The future of health economics. *Journal of Health Economics 19*(2), 141–157.

Galton, F. (1869). *Hereditary genius: An inquiry into its laws and consequences*. London, United Kingdom: Macmillan Publishers.

Gauly, B. (2017). The Intergenerational Transmission of Attitudes: Analyzing Time Preferences and Reciprocity. *Journal of Family and Economic Issues 38*(2), 293–312.

Geoffard, P.-Y. and T. Philipson (1997). Disease Eradication: Private versus Public Vaccination. *American Economic Review 87*(1), 222–230.

Gianoutsos, J. (2006). Locke and Rousseau: Early Childhood Education. *The Pulse 4*(1), 1–23.

Göhlmann, S., C. M. Schmidt, and H. Tauchmann (2010). Smoking initiation in Germany: the role of intergenerational transmission. *Health Economics 19*(2), 227–242.

Goldhaber, D. (2012). *The Nature-Nurture Debates: Bridging the Gap*. Cambridge University Press.

Golsteyn, B. H. H., H. Grönqvist, and L. Lindahl (2014). Adolescent Time Preferences Predict Lifetime Outcomes. *The Economic Journal 124*(580), F739–F761.

Goto, R., Y. Takahashi, S. Nishimura, and T. Ida (2009). A cohort study to examine whether time and risk preference is related to smoking cessation success. *Addiction 104*(6), 1018–1024.

Gouskova, E., N. Chiteji, and F. Stafford (2010). Pension Participation: Do Parents Transmit Time Preference? *Journal of Family and Economic Issues 31*(2), 138–150.

Greenwood, B. (2014). The contribution of vaccination to global health: past, present and future. *Philosophical Transactions of the Royal Society B: Biological Sciences 369*(1645).

Grossman, M. (1972). On the Concept of Health Capital and the Demand for Health. *Journal of Political Economy 80*(2), 223–255.

Gruber, J. (2002). Smoking's 'Internalities'. *Regulation 25*(4), 52–57.

Gruber, J. and B. Köszegi (2001). Is Addiction "Rational"? Theory and Evidence. *Quarterly Journal of Economics 116*(4), 1261–1303.

Gruber, J. and B. Köszegi (2004). Tax incidence when individuals are time-inconsistent: the case of cigarette excise taxes. *Journal of Public Economics 88*(9-10), 1959–1987.

Hahn, E., J. Gottschling, W. Bleidorn, C. Kandler, M. Spengler, A. E. Kornadt, W. Schulz, R. Schunck, T. Baier, K. Krell, V. Lang, F. Lenau, A.-L. Peters, M. Diewald, R. Riemann, and F. M. Spinath (2016). What Drives the Development of Social Inequality Over the Life Course? The German TwinLife Study. *Twin Research and Human Genetics 19*(6), 659–672.

Hammond, D. (2011). Health warning messages on tobacco products: a review. *Tobacco Control 20*(5), 327–337.

Harris, M. and G. Butterworth (2002). *Developmental Psychology: A Student's Handbook*. Psychology Press Ltd.

Harrison, G. W., M. I. Lau, and E. E. Rutström (2010). Individual discount rates and smoking: Evidence from a field experiment in Denmark. *Journal of Health Economics 29*(5), 708–717.

Harrison, G. W., M. I. Lau, and M. B. Williams (2002). Estimating Individual Discount Rates in Denmark: A Field Experiment. *American Economic Review 92*(5), 1606–1617.

Holst, K. K. and T. Scheike (2017). Package 'mets': Analysis of Multivariate Event Times. R package version 1.2.1. `https://cran.r-project.org/web/packages/mets/index.html` [Accessed 28 January 2017].

Hübler, P. M. A. (2018). Heritability of Time Preference: Evidence from German Twin Data. *Kyklos 71*(3), 433–455.

Ida, T. (2014). A quasi-hyperbolic discounting approach to smoking behavior. *Health Economics Review 4:5*.

Ida, T. and R. Goto (2009a). Interdependency among addictive behaviors and time/risk preferences: Discrete choice model analysis of smoking, drinking, and gambling. *Journal of Economic Psychology 30*(4), 608–621.

Ida, T. and R. Goto (2009b). Simultaneous measurement of time and risk preferences: Stated preference discrete choice modeling analysis depending on smoking behavior. *International Economic Review 50*(4), 1169–1182.

Joseph, J. (1998). The Equal Environment Assumption of the Classical Twin Method: A Critical Analysis. *The Journal of Mind and Behavior 19*(3), 325–358.

Kalenscher, T. and C. M. A. Pennartz (2008). Is a bird in the hand worth two in the future? The neuroeconomics of intertemporal decision-making. *Progress in Neurobiology 84*(3), 284–315.

Kamtsiuris, P., E. Bergmann, P. Rattay, and M. Schlaud (2007). Inanspruchnahme medizinischer Leistungen – Ergebnisse des Kinder- und Jugendgesundheitssurveys (KiGGS). *Bundesgesundheitsblatt – Gesundheitsforschung – Gesundheitsschutz 50*(5/6), 836–850.

Kang, M.-I. and S. Ikeda (2014). Time discounting and smoking behavior: Evidence from a panel survey. *Health Economics 23*(12), 1443–1464.

Kenkel, D. S. (2000). Prevention. In A. J. Culyer and J. P. Newhouse (Eds.), *Handbook of Health Economics*, Volume 1, Part B, Chapter 31, pp. 1675–1720. North Holland.

Khwaja, A., D. Silverman, and F. Sloan (2007). Time preference, time discounting, and smoking decisions. *Journal of Health Economics 26*(5), 927–949.

Khwaja, A., F. Sloan, and M. Salm (2006). Evidence on preferences and subjective beliefs of risk takers: The case of smokers. *International Journal of Industrial Organization 24*(4), 667–682.

Kirby, K. N. and R. J. Herrnstein (1995). Preference reversals due to myopic discounting of delayed rewards. *Psychological Science 6*(2), 83–89.

Knowles, J. and A. Postlewaite (2005). Do Children Learn to Save from Their Parents? *Population Aging Research Center (PARC) Working Paper Series 05-07*.

Komlos, J., P. K. Smith, and B. Bogin (2004). Obesity and the rate of time preference: Is there a connection? *Journal of Biosocial Science 36*(2), 209–219.

Konrad, K., C. Firk, and P. J. Uhlhaas (2013). Brain development during adolescence: neuroscientific insights into this developmental period. *Deutsches Ärzteblatt International 110*(25), 425–431.

Kosse, F. and F. Pfeiffer (2012). Impatience among preschool children and their mothers. *Economics Letters 115*(3), 493–495.

Kosse, F. and F. Pfeiffer (2013). Quasi-hyperbolic time preferences and their intergenerational transmission. *Applied Economics Letters 20*(10), 983–986.

Kucher, A. P., R. Nuscheler, and K. Roeder (2014). Is Family Contagious? Transmission of Weight Status (Mis-)Perceptions. mimeo.

Kuntz, B. and T. Lampert (2013). Educational Differences in Smoking among Adolescents in Germany: What is the Role of Parental and Adolescent Education Levels and Intergenerational Educational Mobility? *International Journal of Environmental Research and Public Health 10*(7), 3015–3032.

Kurth, B.-M., P. Kamtsiuris, H. Hölling, M. Schlaud, R. Dölle, U. Ellert, H. Kahl, H. Knopf, M. Lange, G. B. M. Mensink, H. Neuhauser, A. S. Rosario, C. Scheidt-Nave, L. Schenk, R. Schlack, H. Stolzenberg, M. Thamm, W. Thierfelder, and U. Wolf (2008). Study protocol – The challenge of comprehensively mapping children's health in a nation-wide health survey: Design of the German KiGGS-Study. *BMC Public Health 8*(196).

Laibson, D. (1997). Golden Eggs and Hyperbolic Discounting. *Quarterly Journal of Economics 112*(2), 443–477.

Lindeboom, M., F. Portrait, and G. J. van den Berg (2010). Long-run effects on longevity of a nutritional shock early in life: The Dutch Potato famine of 1846-1847. *Journal of Health Economics 29*(5), 617–629.

Loureiro, M. L., A. Sanz-de-Galdeano, and D. Vuri (2010). Smoking Habits: Like Father, Like Son, Like Mother, Like Daughter? *Oxford Bulletin of Economics and Statistics 72*(6), 717–743.

Maćkiewicz, M. and J. Cieciuch (2016). Pictorial Personality Traits Questionnaire for Children (PPTQ-C) – A New Measure of Children's Personality Traits. *Frontiers in Psychology 7*(498), 1–11.

MacKillop, J. (2013). Integrating behavioral economics and behavioral genetics: delayed reward discounting as an endophenotype for addictive disorders. *Journal of the Experimental Analysis of Behavior 99*(1), 14–31.

Martin, N., D. Boomsma, and G. Machin (1997). A twin-pronged attack on complex traits. *Nature Genetics 17*(4), 387–392.

McClure, S. M., K. Ericson, D. Laibson, G. Loewenstein, and J. Cohen (2007). Time Discounting for Primary Rewards. *The Journal of Neuroscience 27*(21), 5796–5804.

McClure, S. M., D. Laibson, G. Loewenstein, and J. Cohen (2004). Separate Neural Systems Value Immediate and Delayed Monetary Rewards. *Science 306*(5695), 503–507.

McKee, S. A. and A. H. Weinberger (2013). How Can We Use Our Knowledge of Alcohol-Tobacco Interactions to Reduce Alcohol Use? *Annual Review of Clinical Psychology 9*, 649–674.

Meier, S. and C. Sprenger (2010). Present-Biased Preferences and Credit Card Borrowing. *American Economic Journal: Applied Economics 2*(1), 193–210.

Meier, S. and C. D. Sprenger (2015). Temporal Stability of Time Preferences. *The Review of Economics and Statistics 97*(2), 273–286.

Melchior, M., J.-F. Chastang, D. Mackinnon, C. Galéra, and E. Fombonne (2010). The intergenerational transmission of tobacco smoking – the role of parents' long-term smoking trajectories. *Drug and Alcohol Dependence 107*(2-3), 257–260.

Mellor, D. and K. A. Moore (2014). The Use of Likert Scales With Children. *Journal of Pediatric Psychology 39*(3), 369–379.

Mischel, W., O. Ayduk, M. G. Berman, B. J. Casey, I. H. Gotlib, J. Jonides, E. Kross, T. Teslovich, N. L. Wilson, V. Zayas, and Y. Shoda (2011). 'Willpower' over the life span: decomposing self-regulation. *Social Cognitive and Affective Neuroscience 6*(2), 252–256.

Mischel, W., E. B. Ebbesen, and A. R. Zeiss (1972). Cognitive and attentional mechanisms in delay of gratification. *Journal of Personality and Social Psychology 21*(2), 204–218.

Mischel, W., Y. Shoda, and P. K. Peake (1988). The nature of adolescent competencies predicted by preschool delay of gratification. *Journal of Personality and Social Psychology 54*(4), 687–696.

Mischel, W., Y. Shoda, and M. L. Rodriguez (1989). Delay of gratification in children. *Science 244*(4907), 933–938.

Mitchell, S. H. (1999). Measures of impulsivity in cigarette smokers and non-smokers. *Psychopharmacology 146*(4), 455–464.

Miyazaki, K., K. W. Miyazaki, and K. Doya (2012). The Role of Serotonin in the Regulation of Patience and Impulsivity. *Molecular Neurobiology 45*(2), 213–224.

Neale, M. C. and H. H. M. Maes (2004). *Methodology for Genetic Studies of Twins and Families*. Dordrecht, The Netherlands: Kluwer Academic Publishers B. V.

Necker, S. and A. Voskort (2014). Intergenerational transmission of risk attitudes – A revealed preference approach. *European Economic Review 65*, 66–89.

Nesson, E. (2017). Heterogeneity in Smokers' Responses to Tobacco Control Policies. *Health Economics 26*(2), 206–225.

Nuscheler, R. and K. Roeder (2016). To Vaccinate or to Procrastinate? That is the Prevention Question. *Health Economics 25*(12), 1560–1581.

Oades, R. D., J. Lasky-Su, H. Christiansen, S. V. Faraone, E. J. S. Sonuga-Barke, T. Banaschewski, W. Chen, R. J. L. Anney, J. K. Buitelaar, R. P. Ebstein, B. Franke, M. Gill, A. Miranda, H. Roeyers, A. Rothenberger, J. A. Sergeant, H.-C. Steinhausen, E. A. Taylor, M. Thompson, and P. Asherson (2008). The influence of serotonin- and other genes

on impulsive behavioral aggression and cognitive impulsivity in children with attention-deficit/hyperactivity disorder (ADHD): Findings from a family-based association test (FBAT) analysis. *Behavioral and Brain Functions 4*(48).

O'Donoghue, T. and M. Rabin (1999). Doing It Now or Later. *American Economic Review 89*(1), 103–124.

O'Donoghue, T. and M. Rabin (2001). Choice and Procrastination. *Quarterly Journal of Economics 116*(1), 121–160.

Odum, A. L., G. J. Madden, and W. K. Bickel (2002). Discounting of delayed health gains and losses by current, never- and ex-smokers of cigarettes. *Nicotine & Tobacco Research 4*(3), 295–303.

Ohmura, Y., T. Takahashi, and N. Kitamura (2005). Discounting delayed and probabilistic monetary gains and losses by smokers of cigarettes. *Psychopharmacology 182*(4), 508–515.

Patton, J. H., M. S. Stanford, and E. S. Barratt (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology 51*(6), 768–774.

Peretti-Watel, P., O. L'Haridon, and V. Seror (2013). Time preferences, socioeconomic status and smokers' behaviour, attitudes and risk awareness. *European Journal of Public Health 23*(5), 783–788.

Perez-Arce, F. (2017). The effect of education on time preferences. *Economics of Education Review 56*, 52–64.

Pfeifer, C. (2012). A Note on Smoking Behavior and Health Risk Taking. *Nordic Journal of Health Economics 1*(2), 135–151.

Picone, G., F. Sloan, and D. Taylor Jr. (2004). Effects of Risk and Time Preference and Expected Longevity on Demand for Medical Tests. *Journal of Risk and Uncertainty 28*(1), 39–53.

Picone, G., M. Uribe, and R. M. Wilson (1998). The effect of uncertainty on the demand for medical care, health capital and wealth. *Journal of Health Economics 17*(2), 171–185.

Plomin, R. and D. Daniels (2011). Why are children in the same family so different from one another? *International Journal of Epidemiology 40*(3), 563–582.

Plomin, R., J. C. DeFries, V. S. Knopik, and J. M. Neiderhiser (2013). *Behavioral Genetics (6th ed.)*. New York, NY: Worth Publishers.

Plotkin, S. (2014). History of vaccination. *Proceedings of the National Academy of Sciences of the United States of America 111*(34), 12283–12287.

Poethko-Müller, C., R. Kuhnert, and M. Schlaud (2007). Durchimpfung und Determinanten des Impfstatus in Deutschland. Ergebnisse des Kinder- und Jugendgesundheitssurveys (KiGGS). *Bundesgesundheitsblatt – Gesundheitsforschung – Gesundheitsschutz 50*(5/6), 851–862.

Pötschke-Langer, M., S. Kahnert, K. Schaller, V. Viarisio, C. Heidt, S. Schunk, U. Mons, and K. Fode (2015). *Tabakatlas Deutschland 2015*. Pabst Science Publishers.

Powell, L. M. and F. J. Chaloupka (2005). Parents, public policy, and youth smoking. *Journal of Policy Analysis and Management 24*(1), 93–112.

Propper, C., J. Rigg, and S. Burgess (2007). Child health: evidence on the roles of family income and maternal mental health from a UK birth cohort. *Health Economics 16*(11), 1245–1269.

Rattay, P., A. Starker, O. Domanska, H. Butschalowsky, J. Gutsche, P. Kamtsiuris, and KiGGS Study Group (2014). Trends in der Inanspruchnahme ambulant-ärztlicher Leistungen im Kindes- und Jugendalter. Ergebnisse der KiGGS-Studie – Ein Vergleich von Basiserhebung und erster Folgebefragung (KiGGS Welle 1). *Bundesgesundheitsblatt 57*(7), 878–891.

Read, D. and B. van Leeuwen (1998). Predicting Hunger: The Effects of Appetite and Delay on Choice. *Organizational Behavior and Human Decision Processes 76*(2), 189–205.

Reinhold, S. and H. Jürges (2012). Parental income and child health in Germany. *Health Economics 21*(5), 562–579.

Rende, R. D., R. Plomin, and S. G. Vandenberg (1990). Who Discovered the Twin Method? *Behavior Genetics 20*(2), 277–285.

Reynolds, B., K. Leraas, C. Collins, and S. Melanko (2009). Delay discounting by the children of smokers and nonsmokers. *Drug and Alcohol Dependence 99*(1-3), 350–353.

Reynolds, B., J. B. Richards, K. Horn, and K. Karraker (2004). Delay discounting and probability discounting as related to cigarette smoking status in adults. *Behavioural Processes 65*(1), 35–42.

Reynolds, B. and R. Schiffbauer (2005). Delay of Gratification and Delay Discounting: A Unifying Feedback Model of Delay-Related Impulsive Behavior. *The Psychological Record 55*(3), 439–460.

Rieck, T., M. Feig, A. Siedler, and O. Wichmann (2018). Aktuelles aus der KV-Impfsurveillance – Impfquoten ausgewählter Schutzimpfungen in Deutschland. *Epidemiologisches Bulletin 1*, 1–14.

Rijsdijk, F. V. and P. C. Sham (2002). Analytic approaches to twin data using structural equation models. *Briefings in Bioinformatics 3*(2), 119–133.

Robert Koch Institute (RKI) (2008). Gesundheit und Inanspruchnahme des Gesundheitssystems. In *Erkennen – Bewerten – Handeln: Zur Gesundheit von Kindern und Jugendlichen in Deutschland*. Robert Koch Institute (RKI), Bundeszentrale für gesundheitliche Aufklärung (BZgA).

Robert Koch Institute (RKI) (2018). Aktuelle epidemiologische Situation der Masern und Röteln in Deutschland. `https://www.rki.de/DE/Content/Infekt/Impfen/Praevention/elimination_04_01.html` [Accessed 19 March 2018].

Roodman, D. (2011). Fitting fully observed recursive mixed-process models with cmp. *The Stata Journal 11*(2), 159–206.

Rossow, I. (2008). Alcohol consumption and discounting. *Addiction Research and Theory 16*(6), 572–584.

Saffer, H. and F. Chaloupka (2000). The effect of tobacco advertising on tobacco consumption. *Journal of Health Economics 19*(6), 1117–1137.

Samuelson, P. A. (1937). A Note on Measurement of Utility. *The Review of Economic Studies 4*(2), 155–161.

Scharff, R. L. (2009). Obesity and Hyperbolic Discounting: Evidence and Implications. *Journal of Consumer Policy 32*(1), 3–21.

Scharff, R. L. and W. K. Viscusi (2011). Heterogeneous rates of time preference and the decision to smoke. *Economic Inquiry 49*(4), 959–972.

Schlam, T. R., N. L. Wilson, Y. Shoda, W. Mischel, and O. Ayduk (2013). Preschoolers' delay of gratification predicts their body mass 30 years later. *The Journal of Pediatrics 162*(1), 90–93.

Schmidt, C. M. and H. Tauchmann (2011). Heterogeneity in the intergenerational transmission of alcohol consumption: A quantile regression approach. *Journal of Health Economics 30*(1), 33–42.

Scholte, R. S., G. J. van den Berg, and M. Lindeboom (2015). Long-run effects of gestation during the Dutch Hunger Winter famine on labor market and hospitalization outcomes. *Journal of Health Economics 39*, 17–30.

Shenassa, E. D., J. M. McCaffery, G. E. Swan, T. V. Khroyan, S. Shakib, C. Lerman, M. Lyons, M. Mouttapa, R. S. Niaura, S. L. Buka, F. Leslie, and S. L. Santangelo (2003). Intergenerational transmission of tobacco use and dependence: A transdisciplinary perspective. *Nicotine & Tobacco Research 5*(Supplement 1), S55–S69.

Sherry, J. L. (2004). Media Effects Theory and the Nature/Nurture Debate: A Historical Overview and Directions for Future Research. *Media Psychology 6*(1), 83–109.

Shoda, Y., W. Mischel, and P. K. Peake (1990). Predicting adolescent cognitive and self-regulatory competencies from preschool delay of gratification: Identifying diagnostic conditions. *Developmental Psychology 26*(6), 978–986.

Smith, D. A. and R. Brame (2003). Tobit Models in Social Science Research: Some Limitations and a More General Alternative. *Sociological Methods and Research 31*(3), 364–388.

Smith, J. P. (2009). The Impact of Childhood Health on Adult Labor Market Outcomes. *The Review of Economics and Statistics 91*(3), 478–489.

Smith, L. E., R. Amlôt, J. Weinman, J. Yiend, and G. J. Rubin (2017). A systematic review of factors affecting vaccine uptake in young children. *Vaccine 35*(45), 6059–6069.

Standing Committee on Vaccinations (STIKO) (2017). Empfehlungen der Ständigen Impfkommission (STIKO) am Robert Koch-Institut. *Epidemiologisches Bulletin 34*.

Stanford, M. S., C. W. Mathias, D. M. Dougherty, S. L. Lake, N. E. Anderson, and J. H. Patton (2009). Fifty years of the Barratt Impulsiveness Scale: An update and review. *Personality and Individual Differences 47*(5), 385–395.

Steinberg, L., C. Sharp, M. S. Stanford, and A. T. Tharp (2013). New tricks for an old measure: The development of the Barratt Impulsiveness Scale-Brief (BIS-Brief). *Psychological Assessment 25*(1), 216–226.

Story, G. W., I. Vlaev, B. Seymour, A. Darzi, and R. J. Dolan (2014). Does temporal discounting explain unhealthy behavior? A systematic review and reinforcement learning perspective. *Frontiers in Behavioral Neuroscience 8*(76).

Sutter, M., M. G. Kocher, D. Glätzle-Rützler, and S. T. Trautmann (2013). Impatience and Uncertainty: Experimental Decisions Predict Adolescents' Field Behavior. *American Economic Review 103*(1), 510–531.

Tanaka, S. C., K. Doya, G. Okada, K. Ueda, Y. Okamoto, and S. Yamawaki (2004). Prediction of immediate and future rewards differentially recruits cortico-basal ganglia loops. *Nature Neuroscience 7*(8), 887–893.

Thaler, R. (1981). Some empirical evidence on dynamic inconsistency. *Economics Letters 8*(3), 201–207.

Thompson, O. (2014). Genetic mechanisms in the intergenerational transmission of health. *Journal of Health Economics 35*, 132–146.

Tsukayama, E. and A. L. Duckworth (2010). Domain-specific temporal discounting and temptation. *Judgment and Decision Making 5*(2), 72–82.

U.S. Department of Health and Human Services (2014). *The Health Consequences of Smoking: 50 Years of Progress. A Report of the Surgeon General.* U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Printed with corrections.

van der Pol, M., D. Hennessy, and B. Manns (2017). The role of time and risk preferences in adherence to physician advice on health behavior change. *The European Journal of Health Economics 18*(3), 373–386.

Vandewater, E. A., S. E. Park, F. R. Carey, and A. V. Wilkinson (2014). Intergenerational Transfer of Smoking Across Three Generations and Forty-five Years. *Nicotine & Tobacco Research 16*(1), 11–17.

Vischer, T., T. Dohmen, A. Falk, D. Huffman, J. Schupp, U. Sunde, and G. G. Wagner (2013). Validating an ultra-short survey measure of patience. *Economics Letters 120*(2), 142–145.

Volland, B. (2013). On the intergenerational transmission of preferences. *Journal of Bioeconomics 15*(3), 217–249.

Voors, M. J., E. E. M. Nillesen, P. Verwimp, E. H. Bulte, R. Lensink, and D. P. van Soest (2012). Violent Conflict and Behavior: A Field Experiment in Burundi. *American Economic Review 102*(2), 941–964.

Wagner, G. G., J. R. Frick, and J. Schupp (2007). The German Socio-Economic Panel Study (SOEP): Scope, Evolution and Enhancements. *Schmollers Jahrbuch 127*(1), 139–169.

Waszczuk, M. A., H. M. S. Zavos, E. Antonova, C. M. Haworth, R. Plomin, and T. C. Eley (2015). A multivariate twin study of trait mindfulness, depressive symptoms, and anxiety sensitivity. *Depression and Anxiety 32*(4), 254–261.

Webley, P. and E. K. Nyhus (2006). Parents' influence on children's future orientation and saving. *Journal of Economic Psychology 27*(1), 140–164.

Whiteside, S. P. and D. R. Lynam (2001). The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. *Personality and Individual Differences 30*(4), 669–689.

Wickrama, K. A. S., R. D. Conger, L. E. Wallace, and G. H. Elder (1999). The Intergenerational Transmission of Health-Risk Behaviors: Adolescent Lifestyles and Gender Moderating Effects. *Journal of Health and Social Behavior 40*(3), 258–272.

Wissenschaftliche Dienste des Deutschen Bundestages (2014). Einführung verpflichtender Kindervorsorgeuntersuchungen? Überblick über die Verfassungs- und Rechtslage. `https://www.bundestag.de/blob/406316/8f4c316937f69892c86fce34c6946d28/wd-3-143-14-pdf-data.pdf` [Accessed 15 March 2018].

World Health Organization (WHO) (2017a). Fact sheet – Tobacco (Updated May 2017). `http://www.who.int/mediacentre/factsheets/fs339/en/` [Accessed 13 February 2018].

World Health Organization (WHO) (2017b). *WHO report on the global tobacco epidemic, 2017: monitoring tobacco use and prevention policies*. Geneva: World Health Organization.

World Health Organization (WHO) (2018). Fact sheet – Measles (Reviewed January 2018). `http://www.who.int/mediacentre/factsheets/fs286/en/` [Accessed 27 February 2018].

Yaari, M. E. (1987). The Dual Theory of Choice under Risk. *Econometrica 55*(1), 95–115.

Zeit Online (2011, 9 June). Vorsorgeuntersuchungen: Nutzlose Rasterfahndung. `http://www.zeit.de/2011/24/Familie-Kindervorsorgeuntersuchungen` [Accessed 27 March 2018].

Zhong, S., S. H. Chew, E. Set, J. Zhang, H. Xue, P. C. Sham, R. P. Ebstein, and S. Israel (2009). The Heritability of Attitude Toward Economic Risk. *Twin Research and Human Genetics 12*(1), 103–107.

Zhou, F., A. Shefer, J. Wenger, M. Messonnier, L. Y. Wang, A. Lopez, M. Moore, T. V. Murphy, M. Cortese, and L. Rodewald (2014). Economic Evaluation of the Routine Childhood Immunization Program in the United States, 2009. *Pediatrics 133*(4), 1–9.

Zyphur, M. J., J. Narayanan, R. D. Arvey, and G. J. Alexander (2009). The Genetics of Economic Risk Preferences. *Journal of Behavioral Decision Making 22*(4), 367–377.