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Chapter 1

Introduction

Take a look in the mirror – who do you see? The eyes of your mother, the forehead of your father, or the smile of your grandmother? Do you see someone confident like your grandfather? A risk-loving person like your brother? Future-oriented and inquisitive? How are you feeling right now and how are you perceiving yourself in your environment? Are you athletic and healthy? Do you smoke or drink alcohol with your friends? Do you pay attention to your dental care and the dental care of your children? What about weight management? Is your immunization record up to date?

As an empirical *social economist* in health economics in the sense of Becker and Murphy (2003), the content of my study is the individual's health behavior affected by his or her environment using methods of economics. My thesis moves in the field of tension between individual health behavior, prevention, and familial socialization. Family life is the daily formation and balancing of rules, norms, perceptions, and habits and is an important part of socialization. The core of the analyses are incentives and possible mechanisms that shape and guide child and parental behavior in the context of health investment decisions.

Being able to do research in these fields is fascinating and just as challenging: to think about mechanisms and interactions within the parent-child relationship from a theoretical economic point of view and than trying to check them with survey data quantitatively. So within my research I was faced and challenged with methodological boundaries and data limitations. Therefore the task is to investigate testable hypotheses from economic theory within empirical frameworks to get some insights. The results can be seen as inputs to health services and policy. In order to embed the results in a broader context, some aspects referring to health and health behavior are considered in more detail below.

1.1 An economic view on health and health behavior

Next to social and political factors such as prosperity, wealth, access to healthcare, and environmental factors, our genetic endowment and own individual behavior are determinants of our health. From a public health perspective, individual behavior plays an important role, as it directly and indirectly influences demand for healthcare services. However, it is recognized that public health intervention targeted toward changing individual lifestyle behaviors with the aim of reducing health risks is a notable challenge.

1.1.1 What determinates our health and health behaviors?

When investigating individual health, economists think in terms of Grossman's health production: receiving some health endowment at birth, a lifetime resulting from the health stock, depreciation, and the consumption of commodities that are in competition with each other. It should be noted that we are aware of this and can do something: we can *produce* health with inputs (market goods and our time). On the other hand, it also means that the question arises as to whether one should currently spend one's life with consumption (with benefits today and at the costs of a shorter lifespan) or whether one should invest in health today (with the benefits of a longer, healthier lifespan in the future and at the cost of consumption benefits today). For health investment decisions to be at an optimum, the costs of the last input combination into health production must correspond to the additional marginal return on the outcome, *i.e.*, our health stock. This individual intertemporal optimization problem depends on prices, restrictions and individual preferences for consumption, health and time (see Grossman (1972)).

According to this theoretical understanding of individual health production, health behaviors connect market goods and time to produce the commodity "good health": buying market goods (*e.g.*, medical services, drugs, athletic shoes, gym membership, food) in combination with other aspects of life. However, it is important to note that the first-order condition of health production also considers input decisions to consume goods and to spend time even if both are health-reducing, *e.g.*, cigarettes and smoking, unprotected sex, illegal drugs and their consumption, or less sleep. These decisions regard both individual health production *and* as other commodities the utility function: *e.g.*, smoking is an input of the health production and decreases the health stock, but as commodity "addiction

pleasure", it increases a person's utility directly. Therefore, unhealthy, risky behavior is justified as the optimal response of an intertemporal utility maximization problem with given preferences (see Becker and Murphy (1988) and Kenkel and Sindelar (2011)).[1] In this framework, the next section will take a closer look at preventive decisions.

1.1.2 Is prevention an investment in health?

Prevention should help one maintain an existing state of health for the future and avoid worsening through the onset of disease risk. Curative treatment restores a lost state of health after a disease has occurred. Both approaches have the goal of reducing the suffering from disease, but prevention begins before the onset of a disease or further damage, and treatment thereafter.

From an economic viewpoint, investments in the health stock are characteristically preventive; they could increase the health stock and lower illness probability, which itself depends on the health stock. Within the health production function, prevention would be seen as health investment that also increases resources for further economic decisions (see Grossman (1972)).

But should prevention always take place?

First, one should take a closer look at prevention. There are two known criteria for specifying prevention more precisely: *timing* (primary, secondary, tertiary, and quaternary) and *levels* (behavioral- and condition-oriented).[2] The different intervention phases (*timing*) of prevention are briefly explained.

Primary prevention begins before the onset of disease, *i.e.*, without medical diagnosis and when patients feel well. It considers causal factors for an event (*e.g.*, disease or risky behavior) and tries to avoid or reduce it, *e.g.*, preventing incidences by hygiene training, vaccination immunization against infectious diseases, prohibition of dangerous substances harmful to health (*e.g.*, asbestos), statutory health and safety regulations (*e.g.*, seat belt or smoking laws), or health promotion and information (*e.g.*, eating habits, exercising, risk assessment).

Secondary prevention aims to start at the earliest possible stage of a disease,

[1] An overview of theoretical frameworks and empirical evidence in the context of risky health behavior is given by Cawley and Ruhm (2011) in the handbook of health economics in 2011.

[2] The timing dimension for prevention was first established by Leavell and Clark (1958). See Rosenbrock and Gerlinger (2014), Chapter 3, for an overview of both criteria and Jamoulle (2015) for the idea of quaternary prevention. There are other categorizations and prevention models in the literature, *e.g.*, the continuum of care model of the Institute of Medicine (see Springer and Phillips (2007)).

where no symptoms have yet occurred for the patient (who feels well), albeit from the medical side. The goal here is to prevent the progression of a disease and to increase the chances of recovery through the early stage treatment. Screenings are the most widely known interventions (*e.g.*, mammograms for breast cancer). Other examples include drug treatments for prevention (*e.g.*, regular but low doses of aspirin for strokes), or adjustments to the workplace to allow employees to safely reenter after illness.

If a disease is present (the patient feels ill) and cannot be cured (for instance, the medical diagnosis of a chronic disease), measures to prevent further harm and to improve the quality of life are covered by *tertiary* prevention. These include rehabilitation programs in the vocational context or disease management programs for chronic diseases (*e.g.*, diabetes or chronic kidney disease). Measures of tertiary prevention partly overlap with curative treatments.

Finally, *quaternary* prevention has an eye on overmedicalization, overutilization and overtreatment and aims to reduce unnecessary costs from an economic and patient perspective ("the need to not harm", Jamoulle (2015, p. 62)). Interestingly, this phase is often the starting point for interventions, as patients feel ill, but whether there is any disease at all must be clarified. In most cases, this is not the case.

The other dimension, *levels*, divides prevention according to objectives: changing the frame conditions (for instance, living or working conditions) or the individual health behavior directly by intervention. For example, smoking bans in restaurants are condition-based prevention concerning the health risk of service workers exposed to second-hand smoke. Anti-smoking training for smokers is a behavioral prevention measure.

Returning to the question of whether prevention should always take place, there are some aspects to consider. Again, the classical time perspective induces a trade-off and uncertainty: investment in health with opportunity costs today, healthy time and longer life tomorrow. This view regards prevention as an intertemporal choice, so inherently individual preferences, such as time and risk preference, play an important role in the magnitude of demand and the implementation of prevention measures.

As part of an individual decision, not only health production through prevention but also opportunity costs are taken into account, which means the applied temporal and monetary resources as well as the possible side effects through prevention. For example, if the disutility of the commodity "suffering pain" from a

vaccine injection for primary immunization is large and exceeds the production gains for the health stock, the decision against the preventive measure is optimally taken. This weighting of the different commodities within the individual utility function is determined by individual preferences, among other things.

At the societal level, however, other weightings may apply to the costs and benefits, but "damages" are critical and should be questioned. Studying the effectiveness of prevention on individual and aggregate intervention level is a challenging task, as is achieving a balance between some few helpful (early) interventions and many (potential) side effects: *e.g.*, for secondary prevention such as screenings, there are many cases of overdiagnosis, false positive findings, and follow-up treatments.[3] Next to cost-benefit discussions, there are other ethical or political dimensions (*e.g.*, ethical and political aims such as distribution or healthcare access) that play a role (see Rosenbrock and Gerlinger (2014, p. 72)).

The two aforementioned dimensions of prevention can also be applied to families: parents provide the framework with certain health-related conditions in which a child develops health behaviors and health (condition-oriented), but parents also actively influence a child's health and health behaviors through (health) education, role-modeling, habit formation, and communication culture (behavioral-oriented). All forms of prevention regarding timing can also occur: *e.g.*, vaccination decisions, well-child visits, or dental care (primary), screenings for diabetes when there is a family history before clinical diagnosis (secondary), chronic disease management (tertiary), and overmedicalization (quaternary).

In this work, individual health behaviors within the family are examined in greater detail. They are all in the focus of global and state-funded prevention programs[4]: primary immunization, weight misperceptions, and smoking.

1.1.3 Health behavior and preferences

Looking at the formation of preferences, one might argue that genes, family, and environment together form a person's preferences, perhaps in early childhood.

[3]For example, for mammograms, there is such a debate (see Gøtzsche and Jørgensen (2013) and Lauby-Secretan et al. (2015)).

[4]Prevention of noncommunicable diseases (such as diabetes, lung and heart diseases) are the focus of the WHO with tobacco-free programs, health promotion and other approaches (see WHO (2013a)). In Germany, these programs often take the form of collaboration among the federal government, the federal states and social security funds. Since 2015, a prevention law has been in effect in Germany that explicitly anchored this prevention behavior as a goal of primary prevention (see Nationale Präventionskonferenz (2016) for government recommendations under the law.

Once set, these preferences "control" observable individual behavior, such as economic decisions for the consumption of goods or intertemporal investments. This kind of research focuses on models with given (exogenous) preferences that are often used in standard economic models and investigates their influence on outcomes next to confounding factors, *e.g.*, intertemporal choice models, lifecycle models, and hyperbolic discounting models (see, *e.g.*, Laibson (1997)). Due to the inherent temporal dimension of health-related decisions, whether beneficial or harmful, the influence of preferences is an important issue. There is a wide body of literature focused on the effect of *intrapersonal* preferences, such as risk and time preferences, on health outcomes and behaviors, including smoking, physical activity, or body weight. In essence, the majority of research assumes that time preference is inherently considered to be preventative, as they place relatively high emphasis on the future and therefore support investments that will generate returns in the future. However, the evidence is not straightforward. A literature overview is given by Cawley and Ruhm (2011, p. 130f), which discusses the role of time preference together with education on health behaviors and approaches to measure time preference.

There is another strand of research arguing that preferences might be endogenous and that preferences are shaped over the lifecycle by environmental factors and continuous adaption processes. The environment includes education and learning, role models and peer groups, interactions with others and the influence of political and economic institutions such as markets (see Becker and Mulligan (1997) and Bowles (1998)). In the literature, the decomposition of exogenous genetics and endogenous environment is summarized and often addressed as the nature-nurture debate (see Sacerdote (2011) in the Handbook of Social Economics). For German twin data, Hübler (2018) finds that approximately one-quarter of the variation in time preference is due to genetics. Consequently, in addition to this genetic transmission for preferences, there are other major influencing factors to investigate. For some answers to the introductory questions, one would prefer to hold genes accountable, for others preferences, a person's "character", or social networks. Some of the mentioned attitudes and traits are due to biology and are formed or shaped in childhood by role modeling, parental education, family ties and communication; others are the result of ongoing social norms and interaction. One of the most obvious factors suspected is parental socialization.

1.1.4 Health behavior and socialization

Health behavior is shown to be driven not only by *intrapersonal* preferences but also by social norms, networks and peer effects that influence it and vice versa; Manski's mirror, called the reflection problem, poses an additional challenge when considering individual traits and behavior. He claimed that in addition to the interaction between two actors, further endogenous (bidirectional within group), contextual (exogenous common group traits) and correlated (common environmental) effects must be discussed and distinguished, if possible (see Manski (1993)). Therefore, looking at individuals and their behavior and thinking about the formation process seems to be complex, since social interactions are usually bidirectional in nature, whether consciously or unconsciously. In analyzing a child's behavior, these endogeneity concerns might be reduced because the direction runs more straightforwardly from parents and the environment to the child.

In recent decades, Cavalli-Sforza and Feldman (1981) and Bisin and Verdier (2000 and 2001) established a theoretical framework of cultural transmission to highlight socialization mechanisms and to study the distribution of traits within the population in the long run. Within this theory, the individual formation of values, beliefs and preferences through the influence of parents and the environment is described. For example, it can be explained why a strong transfer of characteristics can be found among minorities, religion, fertility behavior, or gender roles. This framework has inspired and continues to inspire many researchers. It became the "workhorse" within many research fields, *e.g.*, family economics, education economics and health economics (see Bisin and Verdier (2011, section 3) for an empirical review).

As usual, this theory of socialization attempts to depict the essential mechanism, thus abstracting from many aspects that play a role in the socialization process. The core is the interaction and transmission effectiveness of two players, *i.e.*, parents and the child's environment. This environment includes all persons and institutions that can potentially influence a child: idols, respected individuals such as educators, teachers or trainers, and peers, but also the neighborhood and the media. Both have influence on child development. Parents have an intrinsic, direct incentive to *vertically* transmit, e.g. traits or opinions, and the environment a more reflective, indirect role, called *oblique* or *horizontal* transmission (see Bisin and Verdier (2000, p. 959)). Parents may be able to influence or select some of these environmental factors, while others may not. Examples are the place of

residence, educational institutions, or memberships and participation in sports clubs.

In addition, parents can make further efforts to shape their child's characteristics in their favor. While this may sound selfish to parents, ultimately, it is in the nature of things: parents who care about the well-being of their child are altruistic and want the best for their child. The definition of *the best*, however, is crucial, and here, the theory defines paternalistic, imperfect empathy on the parental side. This means that parental choices for the child are based on parental preferences, beliefs and norms and do not take into account their child's preferences (ibid., p. 961f). Parents might spend more time with a child, have more conversations, or take part in trips or activities together. In summary, all efforts by parents increase the likelihood of direct vertically transmission. This transfer can now be indirectly[5] supported or mitigated by the second player, *i.e.*, the environment.

This consideration is a question of context; for example, if parents want to impart a trait to the child that is socially accepted and desired, parents may be more supported in transmitting from the environment than in the case of non-acceptance.

In economic terms, the two sides would be complements in the first case and substitutes in the second case. This, in turn, is reflected in the level of parental effort, as one could expect in the case of substitutes for parents higher effort to secure their parental transmission success. Additionally, in some contexts, it could be that the transmitted trait by parents differs from the parental trait.

The literature has shown a significant positive intergenerational correlation for health behaviors that are both risky and healthy. The role of socialization, its magnitude and the mechanisms seems to be a reasonable challenge in the health context. Finally, parental efforts in transmission affects both behavior-oriented and condition-oriented prevention levels. The peculiarity of prevention within the family is that parents (have to) largely decide regarding demand for prevention for the child and not for themselves - with all the possible side effects and discomfort for the child. This already applies to a variety of screenings during pregnancy and a seemingly never-ending number of possible tests directly after birth in the first few days of life. Currently, 6 out of 10 U-examinations occur under the age of 5 in the first year of life, 8 of 14 vaccination series in the context of the primary immunization are completed around the first birthday, and another 5

[5]Indirectly is not to be understood as passive, because one can imagine, for instance, that the peer group influences also can be very active.

are completed at the end of the second year of life.[6] There are different phases in childhood in which the intensity of interactions with parents, peers and the environment is different: Imprinting and education is mostly parental in early childhood, since they are the main caregivers at the time. From birth, development begins through nonverbal interaction to identify the child's needs and to impart first social and cultural rules (see Van Egeren and Barratt (2003, p. 287)). In middle childhood, the childish focus is on the first peer relationships and school achievement (see Stafford (2003, p. 314)), and adolescence is the phase of building personal identity (see Laursen and Collins (2003, p. 314)).

This is why it is a desired goal to support parents and help them make such health decisions, feel understood seriously with their fears and concerns, and realize the importance of their parental role.

1.2 Health behavior, prevention and the family

All subsequent research projects are related to the influence of parental norms, attitudes and behavior on their children's health behavior and outcomes. They are sorted by phases in childhood: starting with parental vaccination decisions within the first years of a child's life, followed by shaping a child's weight misperceptions in middle childhood around puberty, and ending with a child's smoking decisions that usually occur in late adolescence.

Deriving political implications from the results is not easy. First, health behaviors are driven by preferences such as time and risk preferences. Second, the family is a "secured constitution". For both, the government's rights are legally limited, *e.g.*, the Basic Law or human rights. In policy discussions about legal mandates for health behaviors, individual freedom is used. Economic behavioral research systematically highlights incentive schemes and mechanisms where individual economic decisions make the outcomes on an aggregated level in need of improvement. That can inspire policy or decision makers to "nudge" within the realms of possibility. In a free society, therefore, communication, education, and information are the preferred tools of choice. Furthermore, behaviors cannot be fully tolerated if they give rise to external negative effects on third parties. It is therefore particularly important that parents receive support in their role and influence as role models for their children in order to reduce possible risks or avoid

[6] Only the human papillomavirus (HPV) vaccination is recommended in middle childhood. For an overview, see Gemeinsamer Bundesausschuss (2017) and STIKO (2018).

them altogether.

In my thesis I analyze the policy effects of vaccination policy, parental transmission efforts in the weight context, and underlying preference mechanisms in the context of smoking.

The remainder of the thesis is organized as follows. In Chapter 2, parental demand for the first and second measles vaccinations in conjunction with a recommendation adaption of timeliness is considered. Chapter 3 examines the incorrect weight status perceptions of children around the age of puberty, the transmission of parental norms and role of the intrafamily communication. Finally, Chapter 4 focuses on parental role-modeling and economic preferences, particularly on the time preference of both parents and child, in order to investigate how they affect a child's smoking decision. The next sections give a brief summary of the thesis.

1.2.1 Vaccination recommendations and timeliness

High vaccination rates have a social protection function in addition to individual prevention of infections and diseases. To reach herd immunity thresholds and to protect risk groups, the timeliness of immunization is an important goal for vaccination policy. There is currently no causal evidence of age-appropriate immunization for childhood vaccinations. This research investigates how changing the recommended timeliness of vaccination in childhood affects vaccination status, leaving the number of doses during the primary vaccination unchanged. I analyze an adaption in 2001 that shifts the timeliness two and a half years earlier within the second year of life. Using representative German survey data based on vaccination cards, I investigate a timeliness adaption of the measles primary vaccination in 2001, which changed the scheduled age of the second dose among young children aged 2 to 7 years, and use variation of the implementation across states. For adjusted timeliness of the second measles vaccination, the data imply a significant shift into earlier ages after the policy for the treatment group. In the short run, a difference-in-difference strategy implies causal evidence of the up-to-date vaccination probability at the end of the 7th year of life. Additionally, the adaption induced a significant timeliness effect on the up-to-date level of the first measles dose at the end of the second year of life. This effect can be seen as evidence that individuals, in this case parents, respond to nonbinding vaccination recommendation policies and that timing of vaccination is an important factor for reaching vaccination policy aims.

Contributions to policy evaluation and vaccination timeliness

For economists, a policy change as an exogenous source of variation is the first choice to evaluate causal timeliness effects of vaccination. I contribute evidence in a vaccination policy framework with only nonbinding recommendations. The analyses are unique with German data and within the empirical causal framework for timeliness adaption: state variation and a clear shift in the recommendation makes it possible. I find significant evidence that timeliness adaption increased up-to-date vaccination status and that timing of vaccinations is an important tool to achieve desired policy goals. For filling the immunization gaps, even recommendations work if the timing is optimal. Here, the timing of the second dose within the primary immunization for measles helped to reach additional percentage points for the first immunization threshold. However, it is unclear whether this improvement was driven by supply or demand side. Finally, the development of measles incidences supports the adaptation of the second measles dose.

1.2.2 Transmission of weight status (mis)perceptions

It is important that individuals recognize unhealthy weight status as a health risk in order to change their lifestyle behaviors effectively. Evidence suggests that actual weight and the perception of weight status often do not match, especially among adolescents. As unhealthy weight may be the result of weight status misperceptions, the formation of weight perceptions is an important topic for health policy makers. Building on Bisin and Verdier's (2001) framework, we argue that weight perceptions are being formed during childhood and adolescence. The model distinguishes between direct and indirect perception transmission. In the former, weight perceptions of parents are passed on to children. In the latter, the weight environment serves as a benchmark against which a child evaluates his or her own weight. The theory highlights the role of parental effort that influences the relative importance of the two transmission channels. Using a representative German dataset, we find overwhelming evidence for direct perception transmission and that parental effort, in our case family communication, plays an important role. On the one hand, conditional on correct weight perception of parents, family communication significantly reduces the probability of weight misperceptions of the child. This finding emphasizes the importance of correct weight perception on the side of parents. On the other hand, family communication increases the probability that misperceptions are transmitted. Public policy should thus in-

form both parents and children about the actual weight status of the child and, if needed, suggest weight management strategies.

Contributions to intergenerational transmission and social interactions

Socialization is the cultural basis of every society. However, not every development seems desirable. We adopt the cultural transmission framework for weight, weight norms and preferences to contribute to the increasing weight problem in the population, which is particularly noticeable in industrialized nations: the parental trait *weight perception* would be transmitted to the child regardless of whether it is right or wrong from a medical perspective. We argue that parental effort is communication about weight as the mediation factor in this intergenerational transmission and account for social network effects. More specifically, we not only argue for the theory of this transfer mechanism but also deduce testable hypotheses and, in our empirical strategy, form the effort interactions for both the parents and the environment.
Identification of social network effects in observational data is not without challenges. In addition, unobserved characteristics in the shared environment that affect all individuals in the social network may cause environmental confounding. In the empirical analyses, we solve endogeneity concerns by estimating reduced forms. Our instrument is a quality general measure of the parent-child relationship called 'familial communication' that is assumed to be correlated with the weight-specific communication and only affects a child's weight misperception through parental effort. Policy implementations must recognize that risk groups would only be reached when they are aware of being at risk. Here, parents have a crucial role for their children. This is important for campaigns and informations in the weight context, as well as for other health behaviors such as immunization and smoking.

1.2.3 Parental time discounting and child's smoking behavior

Intergenerational correlations of risky health behaviors such as tobacco consumption are well established. However, there is still limited empirical evidence about the underlying process through which the transmission is driven. Our research aims to analyze parental time discounting and its role in the intergenerational transmission of smoking. The mediation analysis is based on longitudinal data

from the German Socio-Economic Panel (SOEP) for the years 2006, 2008 and 2010. We use panel regression models to estimate a child's likelihood of being a current smoker. The SOEP contains many socioeconomic characteristics and meaningful measures of individual discounting behavior, namely, general patience and impulsivity; this information enables us to distinguish between time preference and self-control, respectively. We find significant effects of time preference for both mothers and fathers. That is, an increasing level of parental patience is associated with a lower smoking probability of a child. Regarding self-control, only the father's impulsivity has a similar decreasing impact. Stratifying the sample by gender reveals substantial mother-daughter, mother-son and father-son effects. We consider the influence of health-related behaviors of the parents such as smoking as possible pathways. Although parental health behaviors do not represent true mediating factors, their smoking behavior itself is positively related to offspring smoking. Finally, we examine the intensive margin of cigarette consumption and find no direct effect of either parental patience or impulsivity. However, beyond role modeling, parental time discounting is firmly relevant for a child's smoking behavior.

Contributions to intergenerational transmission and time preference

That preferences shapes individual health behavior is common knowledge and at first logical and unproblematic. However, the question is what to do when individual decisions are driven by the preferences of others. Thus far, there are only a few researchers who have, for instance, studied the influence of parental preferences in the *interpersonal* context of child outcomes such as savings or health behavior in an intergenerational context (see Brown and Pol (2014) and Gouskova, Chiteji, and Stafford (2010)). Explaining the established positive smoking correlation between parents and children, we investigate the underlying mechanism of parental time preference. Our research is characterized by the fact that we consider important points in order to obtain a more accurate idea of the mechanisms: different preference dimensions of intertemporal choice (time and risk preferences as well as hyperbolic discounting), the influence of both parents, and a mediation analysis. We use panel data and validated measures of patient and impulsiveness instead of revealed preferences such as saving behavior. We are able to control for expected mediating factors, parental health behaviors and socioeconomic status, as well as their role in the transmission process. At the end, there remains a direct parental time preference effect on a child's smoking decision, a 'puzzle' for future research.

Chapter 2

Vaccination recommendations and timeliness

The German measles case

2.1 Introduction

"*This century promises to be the century of vaccines, with the potential to eradicate, eliminate or control a number of serious, life-threatening or debilitating infectious diseases, and with immunization at the core of preventive strategies.*" (Global Vaccine Action Plan, World Health Organization WHO (2013b, p. 13))

For 14 out of 25[1] vaccine-preventable infections, the German immunization schedule recommended a primary vaccination within the first two years of life in 2018 (see STIKO (2018, p. 338)). Vaccination is a highly effective way that parents can protect their children from more than a dozen major infectious diseases, sequelae, hospitalization and even death[2]: looking at the measles vaccine, the field effectiveness of the first dose is on average 91 % regardless of a child's age or region. Studies for Europe showed an effectiveness between 79 and 99 % for the first dose and between 93 and 99 % for the second dose.[3] From 2000 to 2015, measles incidence rates declined by 75 %.

Although there is an effective vaccine, there were still 36 cases per million population contracted worldwide in 2015, the majority among children under the

[1]Number of infections with existing licensed vaccines (see WHO (2013b, p. 16)).

[2]For a short historical summary for the United States of America, McLean et al. (2013) described incidence, hospitalization and death rates for measles, rubella, and mumps.

[3]See Uzicanin and Zimmerman (2011) for a review of the field effectiveness for measles.

age of 5.[4] Based on this information, the measles death rate was estimated at 134,200 worldwide (see Patel et al. (2016, p. 1230f)). Additionally, there are risks for secondary diseases such as ear infections, bronchitis, and pneumonia as well as long-run risks such as brain inflammation of the type subacute sclerosing panencephalitis (called SSPE) (see Moss and Polack (2001, p. 298), McLean et al. (2013, p. 3) and Schönberger et al. (2013)). The virus still ranks first in the list of deaths for vaccine-preventable diseases worldwide[5]; therefore, a continuing priority of measles eradication has been pursued for decades (see Kabra and Lodha (2013, p. 2)). For both Germany and the European WHO region, this goal[6] should have already been reached in 2010, but it was postponed several times and is currently set for 2020 (see Federal Ministry of Health (2015)).

Why is it so difficult to eradicate the measles despite its high vaccine effectiveness?

The measles virus is highly contagious on contact (*e.g.*, air, droplet, or casual touching). Symptoms initially resemble a cold or flu until a typical skin rash appears. However, individuals are already infectious and transmit the virus before a skin rash outbreak (see Halloran, Longini Jr., and Struchiner (2010, pp. 11, 64, 220)). In addition to the aggressive nature of measles, challenges are driven by a shortened protection period after birth by maternal antibodies within the first year of life and the waning of vaccine-induced immunity[7], as well as vaccine fatigue[8]. Factors for fatigue are seen in the individual perception of incidence and secondary diseases[9], persisting misinformation and beliefs (*e.g.*, fraudulent evidence regarding autism in the late 1990s), or medical, religious and philosoph-

[4]For Germany, children under 5 years had highest incidence rates in 2001 (30 %) and 2017 (over 35 %) (Hellenbrand et al. (2003, p. S213) and Matysiak-Klose and Wicker (2017, p. 1769)).

[5]In 2000, measles caused 5 % of all child deaths under the age of 5 and were responsible for over 40 % of all deaths of vaccine-preventable infections for children under the age of 15 (see WHO (2002, p. 50)).

[6]Eradication of measles in the European region is defined by an immunization coverage over 95 %, an incidence under 1 per million population per region and year, and no deaths (see Ramsay (1999, pp. 1, 13)).

[7]In the past, the term of protection for newborns was longer because the mothers themselves had experienced measles. For vaccinated mothers, the protective function of the child is shortened (see Waaijenborg et al. (2013)). Similarly, a decrease in vaccine-induced immunity is anticipated, as the mechanism of lifelong immunity has yet not been fully understood (see Halloran, Longini Jr., and Struchiner (2010, p. 59) and Moss and Polack (2001, p. 302).

[8]This can be observed in all vaccine-preventable diseases and is not a specific measles problem (see Larson et al. (2011)).

[9]Local and/or regional success contrarily causes less perception of disease risk and risks of secondary diseases (see (Matysiak-Klose and Wicker (2017, p. 1767)).

ical exemptions (see Salmon et al. (1999)). All these factors increase contagion potential over the lifecycle.

To facilitate a successful implementation of the WHO strategic plan for the eradication of measles, a high overall nationwide vaccination coverage (over 95 %) with two doses of measles vaccine[10] should be achieved. Additionally, susceptible subgroups of people must be identified and targeted by vaccination programs in order to interrupt the transmission of measles (see WHO (2005 and 2013b), Halloran, Longini Jr., and Struchiner (2010, p. 59)).

However, which policy should the government implement to achieve its goals?

There is considerable heterogeneity: policy options (*e.g.*, mandates, recommendations, or mixed policies) are combined with vaccination programs, cultural or national features, legal frameworks, ethical justifications, and access to vaccinations. However, evidence is rare, and external validation is difficult.[11]

Empirical evidence for the causal effects of vaccination policies had only been established for certain select vaccinations. The empirical strategy is to use a natural experiment that randomly assigns the vaccination policy to the population. State-level introduction over time represents a suitable exogenous variation using a treatment-control group design to investigate national evidence.

To the best of my knowledge, Abrevaya and Mulligan (2011) and Lawler (2017) investigate U.S. vaccination policies in the last two decades in such a causal manner. Abrevaya and Mulligan (2011) show that vaccination rates for varicella (chicken pox) increased after introducing state-level mandates in 2000. For hepatitis A, Lawler (2017) finds evidence for two policies - recommendations and mandates - enacted in 1999 or later at the state level. Both policies significantly increased vaccination rates and are associated with decreasing incidence. Given the few studies examining the causal effect of the *introduction* of childhood vaccination policies, such as mandates or recommendations, on immunization rates exploiting state-level diversity, there is no causal evidence of the *timeliness* of childhood vaccinations. For European countries and the Commonwealth of Independent States, investigations of measles/measles-mumps-rubella (MMR) timeliness have been limited to cross-sectional and survival analyses without control groups: *e.g.*, for Germany (Fell, David, and Reintjes (2005) and Siedler et al. (2002)), for Sweden (Dannetun et al. (2004)), for the UK (Walton et al. (2017)), and for Armenia,

[10]With an average vaccine effectiveness of 91 %, even if all received one dose, the necessary threshold would not be reached.

[11]See MacDonald et al. (2018), which described factors that should be mentioned together with an implementation strategy such as mandates but that also apply to other policy strategies.

Kazakhstan, Kyrgyzstan and Uzbekistan (Akmatov et al. (2008)). The same applies to Canada (Périnet et al. (2018)).

Despite the importance of timely immunization, there has been extremely limited empirical research focusing on the effects of an age-appropriate implementation of vaccination policies.

On the one hand, to estimate the causal effect of an age-appropriate vaccination policy we have to exploit exogenous variation and random assignment of age-specific recommendations. Since reunification, the German vaccination policy has undergone a number of changes, *e.g.*, nationwide introduction of new vaccination (for instance, haemophilus influenzae type b) or a change in timing recommendations by several months. The reform that I seek to exploit is the adaption of the second measles dose from the fifth to second year of life in 2001. In this context, an exogenous source would be diverse responses within the public vaccination guidance of individual federal states because implementation of the vaccination recommendations is the responsibility of the federal states. The recommendation of the second measles dose of the primary immunization and its timeliness adaption by several years is one such *unique* case within the German vaccination system.

On the other hand, the main problem has been the availability of national vaccination records (see Fell, David, and Reintjes (2005, p. 29) for the German case). With the "The German Interview and Examination Survey for Children and Adolescents (KiGGS)" from the Robert Koch Institute (RKI), such data are now available. One limitation of the KIGGS data is that the data allow us to analyze the effect of the 2001 timeliness adaption for measles only in the short term since birth cohorts up to and including 2003 could be considered to end 4 years after the policy. Additionally, information about the vaccination status and timing for both measles doses are not exactly known: the measles status for the first dose at the age of 2 years and for the second dose at the age of 7 years. Since a pure recommendation policy without binding age limits is applied within this analysis, the latter point seems less critical because a certain delay in measurement does not appear problematic.

My paper contributes to the previous literature in several ways. First, I provide evidence for the effect of an age-appropriate recommendation policy, here the recommended timing of the second measles dose. The up-to-date measles status at the interview period between 2003 and 2005 allows me to analyze the age-specific vaccination shift of the recommendation adaption in a control group setting.

Second, using the state-level variation of the adaption of the age-appropriate timing in 2001 and the age-specific information about the up-to-date status at the age of 7 years before and after policy change, I estimate the causal adaption effect of the recommendation on the vaccination probability for the timely second measles dose and fill the lack of timeliness evidence in the vaccination context.

Third, in addition to the direct policy effect on the measles status of the second dose, I investigate the 'side effects' of the adaption on the first measles vaccination demand. Due to the shift into the second year of life temporally following the first vaccination in the context of the primary immunization, it is to be assumed that the 2001 adaption also affects the first dose status at the age of 2 years.

Fourth, these analyses will be extended with a regional stratification, a dynamic policy regression model for short-term dynamics, and a robustness section in which methodological and content-related aspects are additionally discussed.

Finally, the effect of the adaption on measles incidence rates in Germany is considered with reporting data and will be discussed.

Results at a glance: An up-to-date analysis shows a clear increasing trend from 2003 to 2005 over all ages from 2 to 6 years with constant rates within the control group at the same time. For the difference-in-differences (DD) framework, the common trend assumption holds and individuals who are affected by the adaption already respond in the short run. For both the first and second measles dose, there is a significant positive effect of over 9 percentage points.

The regional stratification differs for the second measles dose but is equal for the first dose. The dynamic policy results give a constant probability effect for the first dose and some increasing effect for the second dose.

The remainder of the paper proceeds as follows. In Section 2.2, economic theory on vaccination is considered. Section 2.3 summarizes the historical and institutional background and measles vaccination policy for the German case. Section 2.4 describes the data source and the variables used. Section 2.5 presents the empirical strategy. The main results are presented in Section 2.6. Section 2.7 gives some robustness analyses. Section 2.8 considers the incidence rates in the time period after the policy change. Finally, Section 2.9 concludes with a discussion of the main findings.

2.2 An economic view on vaccination decisions and policies

From a theoretical point of view, there is no clear statement about the effect or direction of individual vaccination decisions. The commodity 'protected against preventable infections' by vaccinations suffers from free-riding and external effects, decisions under uncertainty and misinformation[12].

At the individual level, there is a decision trade-off between costs (in particular, time and suffering from side effects) and benefits (individual protection). Society gains from decreasing transmission of diseases, which depends on the proportion of people being vaccinated in society (social protection): individual protection reduces the likelihood of infection of all unvaccinated persons and the population prevalence decreases (physical positive externality). Therefore, high vaccination rates give an incentive for non-vaccination because the individual profits from the transmission reduction and could avoid vaccination costs. Due to this externality, socially optimal demand is not reached, since the individual does not take into account the positive external effect and tends to free-ride.[13] (see Zweifel, Breyer, and Kifmann (2009, p. 157f)). The prevalence elasticity of demand for immunization plays here an important role. Geoffard and Philipson (1996 and 1997) showed that the demand for vaccines are prevalence dependent that prohibits the eradication totally of such infections.

Additionally, the vaccination decision is a decision under uncertainty: pay the costs today and receive the benefits (perhaps) tomorrow. Therefore, risk and time preference also play an important role in the health context and for vaccination decisions. The role of economic and other preferences in the individual vaccination context has been analyzed empirically with survey data (see, *e.g.*, Nuscheler and Roeder (2016)) or within laboratory experiments (see, *e.g.*, Binder and Nuscheler (2017)).

Misinformation and misperception relate, for example, to the effectiveness of

[12]There are other predictors that have also been named and recognized in the literature. From a medical point of view, missed appointments or the impossibility of vaccination due to acute illness and social neglect play a major role (see Schmitt (2001, S3)). Poethko-Müller et al. (2009) and Poethko-Müller, Kuhnert, and Schlaud (2007) analyze sociodemographic predictors, such as socioeconomic status (outcome and proxy of preferences) or migration status, and parental factors, such as beliefs, in Germany.

[13]There is empirical research that analyzes these points, *e.g.*, Ibuka et al. (2014) observed free-riding under laboratory conditions or the quantity of external effects by Boulier, Datta, and Goldfarb (2007).

vaccination and the assessment of the likelihood of infection in the context of vaccination.[14] Research had already shown that people generally and in the health context have problems with misleading beliefs and correct perception (*e.g.*, optimistic bias) (see Weinstein (1982 and 1987), *e.g.*, in the context of smoking behavior see Viscusi and Hakes (2008)). Many investigations have supported these points for individual vaccination decisions: For both patients and doctors, inadequate information and misjudgments about vaccinations and vaccines are important issues (see Favin et al. (2012), Nuscheler and Roeder (2016), and Smith et al. (2017)).

This consideration raises the social and political question of which vaccination policy and incentives can be pursued to support the individual vaccination decision and reach the social goals (*e.g.*, herd immunity, disease eradication).

The effectiveness of the measles vaccination is high, but not perfect (see 2.1). Looking at monetary incentives, Rosian-Schikuta et al. (2007) give an international review of costs and benefits for the MMR vaccination; for Germany, data are not available. Generally, the costs of the disease and estimated indices (such as cost-benefits) vary widely across countries and subgroups, but from a monetary point of view, vaccinations are preferable to the disease infection and treatment costs. The subsidization of costs by public health or health insurance is widespread. In Germany, the vaccine and the medical expenses of recommended vaccinations are covered by health insurance.

One might argue that because of the mentioned market failures, only vaccination mandates can achieve immunity and eradication goals, and they are the preferred choice instead of a recommendation policy. Geoffard and Philipson (1997) showed theoretically that even vaccination mandates could not reach disease eradication because of the nature of vaccination demand.[15] In addition to financial and market arguments, the enforcement of a vaccination policy depends on the country in view of the legal framework and historical conditions.

2.3 Institutional background

Historically, before the German reunification in 1990, the German Democratic Republic (DDR) instituted vaccination mandates for children and adults. In the

[14]Nuscheler and Roeder (2012) found a huge impact of misinformation next to the role of family doctors for the influenza vaccination with German survey data.

[15]See Brito, Sheshinski, and Intriligator (1991) for further theoretical investigations about the justification of mandates and comparison to other regimes such as free choice.

Federal Republic of Germany (FRG), there was only one general vaccination mandate: the pox vaccination between 1945 to 1983. After abolishment until today, health authorities pursued a simple nonbinding recommendation policy for citizens in contrast to other industrial countries, such the U.S. and other European countries.

2.3.1 Structural organization

In Germany, the Paul-Ehrlich Institute (PEI) is the agency responsible for evaluating the safety and efficacy of new vaccines. The Standing Committee on Vaccination[16] (STIKO) at the RKI[17] develops annual evidence-based national recommendations for the use of licensed vaccines by the PEI. These recommendations are age- and subgroup-specific, but they are not legally binding at the state level. By law, each federal state has to extend its public vaccination guidance based on the STIKO recommendations. Following the approval of a new vaccination or timeliness recommendations, individual states may conform with it or differ from it.[18] Participation in the program is voluntary, and the STIKO recommended vaccination are nowadays offered free of charge by sickness funds.[19] The majority of all vaccinations in childhood take place in the outpatient sector by pediatricians and family physicians.

2.3.2 Measles recommendations, status and research questions

In Germany, the measles vaccination has been recommended by the STIKO since 1974 (see STIKO (1974, p. 291)). Starting with a monovalent[20] live vaccine for measles, there is a trivalent MMR vaccine that has been licensed and recommended by the STIKO for West Germany since 1988 (see STIKO (1988, p. 412)). During

[16]The committee is a national immunization technical advisory group appointed by German Federal Ministry of Health. For detailed information on structure, working procedure and publications, see STIKO (2016).

[17]The RKI emerged from the Federal Health Agency ("*Bundesgesundheitsamt*"), which was dissolved in 1994.

[18]The separation of national and federal state levels is based mostly on monitoring, claim for damages of injuries from immunization (see §11 and §60 Infektionsschutzgesetz (IfSG))

[19]After the annual publication by the STIKO within a time span of a few months, the Federal Joint Committee decide to cover the payment for vaccination of the immunization schedule by the service catalog of the statutory health insurance.

[20]One licensed single measles vaccine is still listed in Germany (compared to 21 tri/tetravalent vaccines) (licensed vaccine list available under www.pei.de/impfstoffe)

the division of Germany, the measles vaccination was mandated for all children at 9 months in the DDR and was recommended at 12 months in the FRG; after 1980, it was recommended at 14 months (see STIKO (1980, p. 314)). After reunification, the FRG recommendations were adopted. Since that time, there was one adaption of the first measles dose: In 1997, the timing of the first dose was dated between 11 and 14[21] months of life (see STIKO (1997, p. 101)).

In 1991, the STIKO recommendation for a second measles dose[22] was published for all children aged 5 years and older and was adopted by all federal states (see STIKO (1991, p. 384)). This recommendation was motivated by evidence from the U.S. and some Scandinavian countries to improve the lack of immunity and to obtain adequate herd immunity on a population level.

For practical reasons, the school entry health exam[23] is mentioned as a good point in time for the second measles dose indication (see STIKO (1995, p. 109f)). Since 1998, the recommended age in the STIKO immunization schedules was cited at age 4 and older (see, *e.g.*, STIKO (1998, p. 104)) without any explanation or text mention. However, the controlling advice within the school entry health exam persisted in all STIKO recommendation publications until the 2001 recommendation adaption.

In 2001, the STIKO timing of the second dose was shifted to within the second year of life (15 to 23 months of age) in connection to the first dose (see STIKO (2001, p. 205)). The dating of the second dose into the second year of life was justified with the importance of an early and timely immunization and initial vaccinations without sufficient immune response (nonresponders) should be compensated as early as possible. Because in the first two years of life additional vaccinations and well-child visits are pending, there are comparatively more doctor contacts. The temporal coincidence supports the vaccination implementation.[24].

Fifteen federal states instituted the new timing of the second measles dose, but

[21]If admission to a childcare institution is considered, the MMR-series could start at 9 months. Then, a second dose is recommended at 14 months (see STIKO (1995, p. 109)).

[22]In the DDR, a second measles dose was mandated, and after reunification, was recommended for one year without the STIKO recommendation.

[23]This screening takes place nationwide and is obligatory for all preschool children. It is organized in the preschool year by the local Department of Health. In general, children are required to attend school if they have their 6th birthday before the deadline (country-specific between June and September) and will be enrolled at the earliest possible date. This data source is still used widely for official publications on the vaccination status of children and adolescents at school enrollment.

[24]In phases of recommended well-child visits within fixed-time windows, vaccination coverage increases (see figure 2 in Rieck et al. (2013, p. 3))

only the Free State of Saxony maintained its timing at 5 years and above until 2016.[25] The population living in the Free State of Saxony will be the control group in the data used for the policy evaluation of families that live in the treatment states. This STIKO timeliness adaption was the first adaption of more than 2 years of an existing recommendation, which means that the possible time span of a timely immunization is more than halved.

Since 2006, the varicella ('chicken pox') vaccination has been recommended and could be combined with MMR (*e.g.*, with a licensed tetravalent vaccine) (see STIKO (2006).)

An overview of the age-specific recommendations for the measles[26] vaccination since introduction of the second dose until today is listed in Table 2.1.

Table 2.1: STIKO measles recommendation for primary vaccination

Measle	**Recommendations and time span 1991–2019**			
dose	8/1991[1]–2/1997	3/1997[1]–2/1998	3/1998[1]–6/2001	7/2001[1]–2019
1st	14	11–14	11–14	11–14
2nd	60	60	(48–) 60	15–23

Notes: The age limit (*in months*) represents the recommended age that a child should be (at a given point in time) and be eligible for the first/ second measles dose. [1] Publication date of the vaccination recommendations. Sources: STIKO (1991, 1997, 1998, 2001, and 2018).

Since 2001, the states have reported infections and vaccination status in school entry health exams.[27]

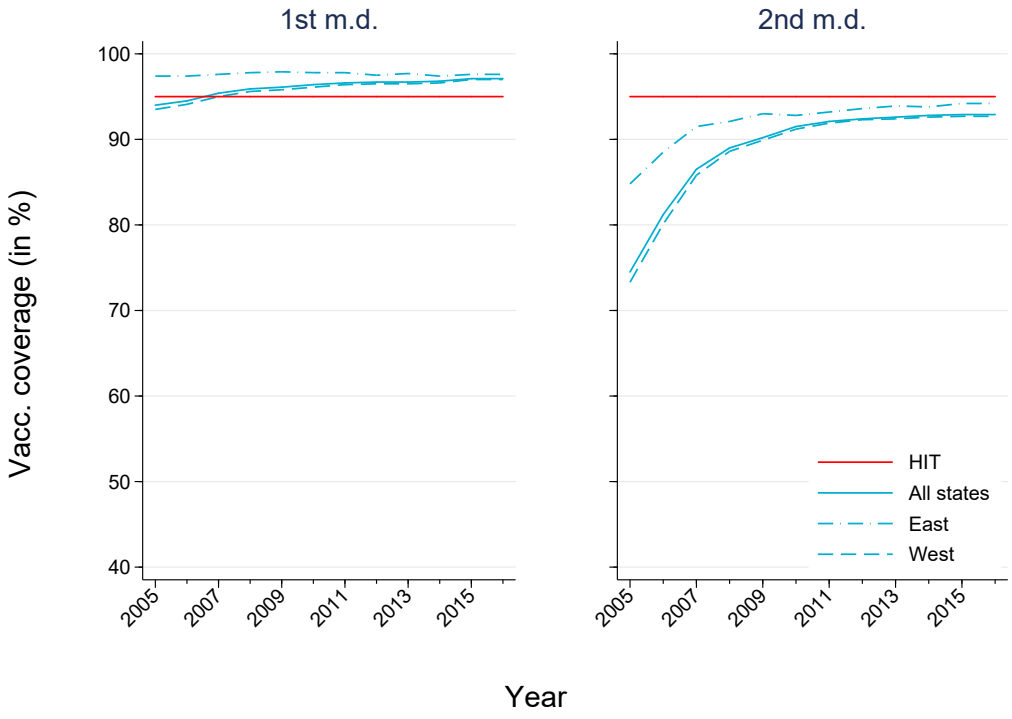
[25]Both the STIKO and the Free State of Saxony rely on evidence from the U.S. and other industrial countries for their timing recommendation. For example, in the U.S. the Center for Disease Control and Prevention and the Advisory Committee on Immunization Practices recommend a 2-dose series at 12–15 months and 4–6 years. The WHO recommended the second measles vaccination at school entry conditional on high vaccination rates for the first dose (> 90) and high school enrollment (> 95), and otherwise in the second year of life (see WHO (2017, p. 221)). Since 2017, the second measles recommendation in the Free State of Saxony has been at 45 months of age and older (Sächsiche Landesärztekammer (2018)). Nevertheless, the second dose is preferable for indication (*e.g.*, measles exposure; note that the minimum length of time to the first vaccination is 3 months).

[26]In the time span from 1991 until 2018, the first and second dose recommendations against measles, mumps and rubella are identical. Only for girls, a third rubella dose is recommended at age 11 and older.

[27]The law aimed to build up a nationwide, statutory reporting and central surveillance for all 431 county and 16 state health departments managed by the RKI (see §34 Abs. II IfSG). Before the law, there was voluntary, nonsystematic reporting to the RKI, and rates often were estimations on single survey data.

In 2016, the average German vaccination rates based on vaccination card information[28] are 97.1 % for one measles dose and 92.9 % for two doses at the age before school entry: The first rate reaches on average the critical herd immunity threshold of 95 %, and the second is just below. The regional variation varies for one shot from 95.2 % (Baden-Württemberg) to 98.3 % (Mecklenburg-West Pomerania and Saxony-Anhalt) and for two shots from 89.5 % (Baden-Württemberg) to 95.8 % (Mecklenburg-West Pomerania). This was the first year that all states exceeded the herd immunity threshold (HIT) at 95 % vaccination coverage at least for the first dose (see Robert Koch Institute (2018, p. 153)).[29]

Figure 2.1: Measles status at school entry health exam by *region*



Notes: Data from Robert Koch Institute (2008-2018), own calculations. The red line is the herd immunity threshold at 0.95.

[28]Rates are based on children with vaccination cards at the health check-up before school entry (on average between 90.9 and 92.6 % for the period 2005 to 2016; in east states, the annual card rates are some higher, between 0.1 and 2.8 percentage points; the east-west population ratio is approximately 1:8.)

[29]It should be remembered, however, that for more than 7 % of the preschool child population, the information was not documented, and the measles field effectiveness is below 100 % (see section 1.2.1).

In Figure 2.1 looking at the data from the school entry health exam in the period from 2005 to 2016, a continuous increase in West Germany can be seen for the first vaccination. As of 2008, the 95% threshold was exceeded in East and West Germany. For the second vaccination, the increase is very clear in the years 2005 to 2009, possibly due to the adjustment in 2001. Since then, the rates show a horizontal course.

Given the recommendation adaption in 2001, there are direct and indirect investigations of interest: Directly, the adaption aims to reach an earlier date of the second measles dose and to increase overall vaccination status at school entry. Indirectly, the new recommended time span for the primary measles immunization ending at the age of 2 could support a timely first immunization.

Whether and how strongly vaccination policies work is ultimately an empirical question. This applies to mandates, recommendations and adjustments of these policies. In summary, I investigate whether the recommendation adaption affects the measles vaccination status

(i) for the second dose at age 2 to 6 (*age shift* effect)

(ii) for the second dose at age 7 (*direct adaption* effect) and

(iii) for the first dose at age 2 (*indirect adaption* effect).

Finally, I take a closer look at reporting data on the incidence of measles, which must be legally recorded and reported by the health authorities in the federal states since 2001.

2.4 Data

Initially, I will describe the dataset generally and the sample selection. Next, I will go into more detail about the variables used in the empirical analyses, first and foremost the individual information for the measles vaccination. After sample stratification by birth cohorts, I will describe the covariates.

2.4.1 Survey data and sample selection

The KiGGS data from the RKI are a cross-sectional dataset collected between May 2003 and May 2006. The dataset is representative of the German population aged up to 18 and contains a massive set of sociodemographic individual and household variables, health inputs and outcomes and information on vaccinations

based on vaccination cards records. The KIGGS data cover birth cohorts from 1985 to 2006. In a first step, 167 sample locations were randomly selected at the community level (primary sample units). Then, age-specific random samples were drawn from population registries (secondary sample units).[30]

For the analyses, the survey years 2003 to 2005 are considered because there are no observations in 2006 for the control group. All children are mentioned that had a vaccination card, were born and have lived since birth in Germany, and reside with at least one biological parent. All child and household information is provided by at least one biological parent, and observations with missing values are dropped. The full sample has 6,272 observations. Table 2.2 shows the sample size after the selection criteria in the upper panel. The size of the samples depends on selected birth cohorts based on the research question and empirical analyses (see Section 2.4.2).

Table 2.2: Sample selection and size

Selection criterion	**Sample size**
None (full sample)	17,640
Survey years 2003 to 2005	14,988
birth cohorts 1993 to 2004	9,718
Child with vaccination card	9,265
Child born in Germany	8,988
Child lived with at least one biological parent	8,916
Questionnaire responder was biological parent	8,896
Observations without missing information	6,272

2.4.2 Vaccination status and timeliness

When analyzing the timeliness of vaccination, it is important to know the age of the child at the time of vaccination.

The most direct method of assessing the age shift of recommendation on timely up-to-date vaccination status is to compare the the age at vaccination before and after the adaption.[31] The KIGGS data provide such information with limitations. Retrospectively, especially for the pre-policy period, the age-specific timeliness

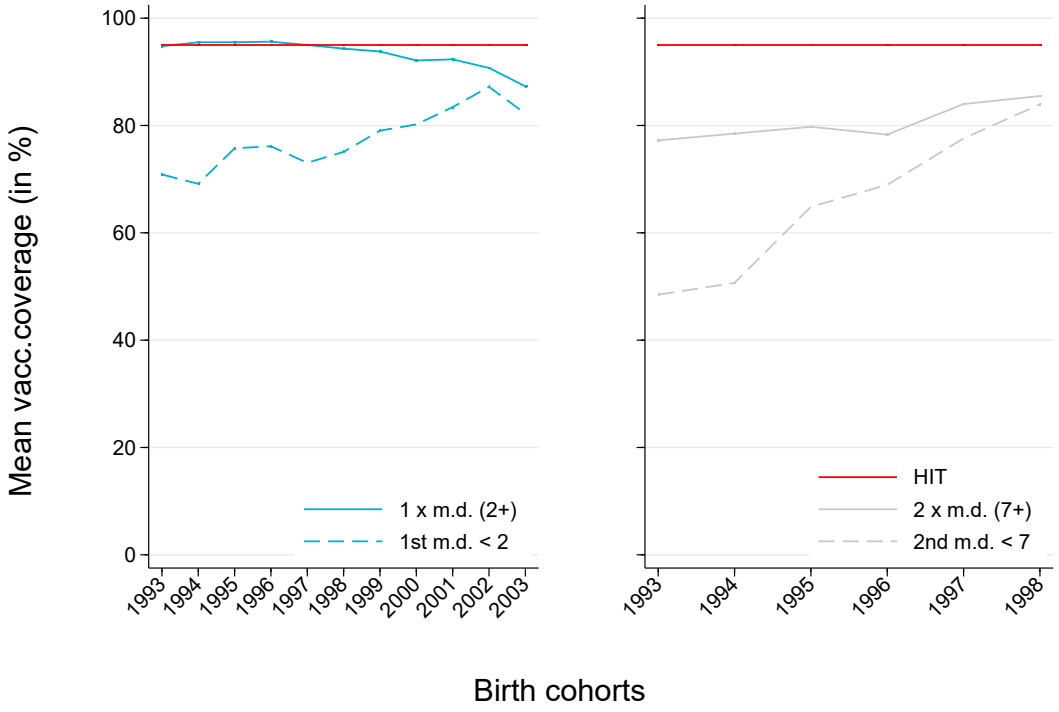
[30]For more details on the setting and the sampling procedure, see Kurth (2007a).

[31]For the considered time period the registry of reported data by the German health departments are not helpful for Germany because it started nationwide by law in 2001. Administrative data derived from health insurance claims could be an alternative source if the control group is well represented.

information on a child's vaccination status is not known exactly. For each individual, there is age-specific timeliness information if the child received the first (second) measles dose before the age of 2 (7) years. Additionally, the up-to-date information about the total sum of measles doses at the date of interview is known.

To obtain a more accurate picture at the timing of vaccination, the measles quota by birth cohorts totally and by age of vaccination are given in Figure 2.2. These data are based on the whole KiGGS data. On the left side, the totally up-to-date status (solid) and the timely status at the age of 2 (dashed) for the first measles dose are shown; shown in the right figure are the corresponding rates for the second dose totally and at the age of 7. At the beginning of the period considered, the gap between the overall rate and the age-recommended rate for both vaccinations was over 20 percentage points, showing a clear rapprochement over time. Interestingly, these data for the first vaccination show a steady decline for the birth cohort in 1997 and younger. This observation shows that some of the overall rates are achieved much later than recommended. Vaccine fatigue could of course also be behind this finding.

Figure 2.2: Measles status and timeliness by *birth cohort*



Source: KiGGS data, own calculations.

I use two samples to analyze the recommendation adaption: the timely up-to-

date sample and the pre-post sample.

The up-to-date sample involves the post-policy period from 2003 to 2005 to investigate the age- and group-specific up-to-date measles status of children aged 2 to 6 years for two measles doses. I also check the difference of children aged 1 to 2 years between treatment and control group for the first measles dose. The corresponding cohorts are listed in Table 2.3.

The pre-post sample contains all children aged 2 and older for the first measles dose or aged 7 years and older for the second measles dose before and after the recommendation adaption. For the pre-policy period, all children were taken into account, so the recommended vaccination age *and* the measured time of vaccination status are not affected by the adaption. This is also the case for the post-policy period if both recommendation and measurement of the vaccination come after the policy introduction. For earlier policy decisions or other adaption to have no effect, I start with the previous period in 1998. Children born between 1999 and 2000 (first dose) and in 1995 (second dose) were excluded because the (age-appropriate) vaccination recommendation or the school entry health exam is before the 2001 adaption, but the measurement of vaccination status falls into the post-period. Therefore, it is unclear whether the decision is driven by the 2001 adaption. In Table 2.3, birth cohorts considered in the analyses are defined by sample, measles doses, age, pre-policy, and post-policy.

Table 2.3: Sample birth cohorts

				Birth cohorts		
Sample	**Measles dose**	**Age** (*in years*)	**Vacc. age** (*in years*)	**Pre-policy** (*1998 – 2000*)	**Post-policy** (2003 – 2005)	***N***
Up-to-date	1st dose	1			2001 – 2004	625
		2			2000 – 2003	
	2nd dose	2			2000 – 2003	3,114
		3			1999 – 2002	
		4			1998 – 2001	
		5			1997 – 2000	
		6			1996 – 1999	
Pre-post policy	1st dose		< 2	1996 – 1998	2001 – 2003	3,075
	2nd dose		< 7	1993 – 1994	1996 – 1998	2,459

Source: Selected sample birth cohorts of the KiGGS data, own illustration.

2.4.3 Parental, household and offspring characteristics

The dataset used includes a wide range of individual and household characteristics to account for individually heterogeneity. These include proxies for a parent's socioeconomic status (education, occupation, income), migration background, household size, birth order, and regional structure.

There is also information on child care and school enrollment. Both are criteria that can play a role in the vaccination decision (see Section 2.3.2 and footnote 21). Furthermore, vaccination-specific and health-specific information is available in the dataset, including well-child visits, medical vaccination advice, parental reasons against vaccinations, chronic illnesses and timeliness of other vaccinations. The vaccination guidelines advise a steady control of the vaccination status of children as part of the well-child visits (so-called U-exams): For analyses of the first measles dose, the U6 (10 to 12 months old) and U7 (21 to 24 months) groups are included; for the second dose, the U8 (46 to 48 months) and U9 (60 to 64 months) groups are also included. In addition, medical advice and recommendation has an important role for the parental decision, for which, of course, individual reasons also play a role, *e.g.*, the individual opinion of the non-need for immunization. A child's health can also influence the decision. There is information about a set of chronic diseases and their first appearance by age: For the first measles dose, all chronic diseases that occurred in the first two years of life are considered, and for the second dose, the first three years are considered. Last but not least, it is possible to check for the revealed vaccination preferences of the parents. Therefore, a vaccination is considered that had taken place earlier: For the first measles dose, the timely primary immunization for hepatitis B[32] is used; the second dose is the timely first vaccination against measles. Table 2.13 in the Appendix lists all mentioned variables with attributes. The descriptive statistics for the full sample and both the treatment and control groups are provided in Tables 2.14, 2.15 and 2.16 in the Appendix.

2.5 Empirical strategy

My empirical exploration has two parts. First, investigating the age-specific development of timeliness, I compare the treatment and control groups (difference in mean) in an age-period regression framework after 2001 using sample 1. Second,

[32]I chose hepatitis B because it is still not a standard vaccination in the first year series such as diphtheria, tetanus, or pertussis.

I use a difference-in-differences (DD) regression framework that allows me to take advantage of the variation in the implementation of recommendation adaption between states using multilevel data. I estimate the policy effects of the STIKO recommendation adaption on the parental decision of a child's measles vaccination as an outcome variable using individual data from the KIGGS dataset. Using sample 2, I estimate the causal effect of the recommendation adaption in 2001 for the measles vaccinations level for the first dose at the end of the second year of life and the second dose at the end of the 7th year of life.

To take advantage of the available observed sociodemographic information at the individual and household level, the unit of analysis is an individual i to control for heterogeneity and improve the power of the estimates.

After model descriptions, I will discuss the identification strategy and challenges with serial correlation and clustering within policy evaluation.

2.5.1 Age shift regression model

The adaption contains a recommendation age shift into the second year of life. Analyzing this shift effect, I use a 3-way interactions regression model for the group-specific age-period vaccination timing. Formally, the outcome indicator for the parental choice of child i living in state s in period t to be vaccinated against measles is VAC_{ist}.

$$Pr(VAC_{ist} = 1|.) = F(age_i \text{ x } treated_s \text{ x } period_t, X_{ist}) \tag{2.1}$$

where X_{ist} captures individual and household controls and $treated_s$ is the indicator for the treatment, the age-adaption for the second measles vaccination within the second year of life. For flexible model specification I include indicators for age and period. For the distribution function F, the identity is assumed to be a linear probability model (LPM). For robustness, I will estimate the regressions with age to be continuous and squared assuming a positive increasing age-effect on vaccination probability within the age span from 2 to 6. Additionally, I assume the normal distribution for the distribution function to estimate a nonlinear probit model accounting for the binary nature of the dependent variable. I will provide graphical analyses of the partial effects because, even in linear models, these

effects require computation of the coefficients.[33] In this model, the age-period effects are of interest. I control for birth cohorts without additional restrictions using individual records because there is no exact linear dependency between age, period and cohort in the data (see Robertson and Boyle (1986, p. 530)): *e.g.*, individuals who are 2 years old at the date of interview in the survey year 2003 might be born in the year 2000 or 2001, in the survey year 2004 in 2001 or 2002, and so on.

2.5.2 Policy evaluation regression model

Now, let $VAC_{is\tau}$ be the outcome indicator for the parental choice of individual i living in state s born in year τ[34] to be vaccinated against measles.

The linear probability model within the DD framework then is

$$Pr(VAC_{is\tau} = 1|.) = \gamma_s + \lambda_\tau + \beta D_{s\tau} + X_{is\tau}\delta + \epsilon_{is\tau} \tag{2.2}$$

where γ_s and λ_τ are state and cohort fixed effects. Individual and household controls are captured by $X_{is\tau}$. $\epsilon^v_{is\tau}$ is the error term. γ_s captures group differences between treatment and control group before the policy change (with the assumption to be constant over time in the absence of treatment), and λ_τ implies the cohort effects of vaccination rates for both groups. $D_{s\tau}$ is an indicator whether state s recommended the adaption for birth cohort τ and the DD estimator for $D_{s\tau}$ is the parameter of interest.[35]

To obtain some dynamic policy insights, the DD model can be extended to birth cohort treatment effects:

$$Pr(VAC_{is\tau} = 1|.) = \gamma_s + \lambda_\tau + \sum_{j \in J} \beta_j \; D^j_{s\tau} + X_{is\tau}\delta + \epsilon_{is\tau} \tag{2.3}$$

where γ_s and λ_τ capture state and year of birth fixed effects. $D^j_{s\tau}$ is an indicator equal to 1 if cohort j, with $J = \{-m, .., -1, 1, .., n\}$, was affected by the policy ending with m 'leads' and n 'lags' treatment effects. The reference group will be

[33] For comparison with the nonlinear model, it is also easier and more meaningful (Ai and Norton (2003) and Greene (2010)).

[34] In this model, birth cohorts are the 'time' perspective (see Section 2.4.2).

[35] For robustness, a nonlinear DD model is estimated (Puhani (2012)).

the last pretreatment birth cohort (j=0). The coefficients β_j represent the cohort j-specific DD estimator. Assumptions and challenges of the DD framework are discussed in detail in the next section.

2.5.3 Identification and inference

I use variation across birth cohorts and state-specific age recommendations for vaccination to identify the effect of the vaccination timeliness adaption at the state level on individual vaccination probability. Next, to control for observed heterogeneity by adding powerful individual controls for the vaccination decisions, both the variance of residuals and the standard errors of the estimates are reduced, and the estimates become more precise. This approach is helpful in the DD framework if the covariates are not affected by the timeliness adaption themselves; otherwise, they are *bad controls* in the sense of Angrist and Pischke (2009, 22f, 64). The use of a timely first measles dose as a control variable in the regression of the second dose probability is unproblematic here since the first decision was completed prior to the policy for all birth cohorts used.

In standard difference-in-differences models, identification relies on the *common trend assumption* that in the absence of the policy, outcomes in the treated states would have evolved as in the control group. More precisely, to achieve identification, I assume linearity, and any unobserved time-varying state characteristics that affect outcomes are uncorrelated with treatment. Checking this trend assumption, I need at least two periods before the recommendation adaption for both groups, *i.e.*, treatment and control groups. With equation 2.3, the significance of all leads[36] of the DD estimator checks the common trend assumption and should be zero (Granger causality test, see Angrist and Pischke (2009, p. 177)).

Another point is the individual home decision. The federal state where the family lives is a decision variable that is (at least partly) under individual control. In response to the state's policy decisions, parents might move. In my context, it is unlikely that a nonbinding vaccination recommendation and its adaption directly influence parental home decisions; both measles doses were already introduced and are free of charge as they are statutory health insurance benefits. Therefore, it can be assumed that when parents insist on a second vaccination, they could also go to another state to obtain the vaccination. In the case of measles exposure, even in Saxony, an earlier second vaccination was recommended. In addition, I cannot

[36]Here, leads are birth cohorts that are not affected by the recommendation adaption.

control for German internal migration since the place of residence is known only at the time of the interview and not at the time of vaccination. Public statistics show that the majority of German internal migration takes place within a federal state, which is not a problem in this framework of policy analyses. In addition, migration within the states of the treatment group would not be a problem either.[37].

For valid inference of the t-statistics in my regression frameworks, two pitfalls must be considered: clustering and serial correlation (see Bertrand, Duflo, and Mullainathan (2004) or Angrist and Pischke (2009, chapter 8)).

One challenge is the calculation of the 'correct' standard errors choosing the 'right' clusters[38]: The level of clustering is the highest level of aggregation determined by the data used[39] or the empirical strategy. Within the DD framework, the timeliness adaption policy affects both states and birth cohorts and must be used as the level of clustering. This consideration accounts for the presence of a common random effect at the state and birth cohort levels (see Bertrand, Duflo, and Mullainathan (2004), Donald and Lang (2007), and Moulton (1986)); the individual age-specific vaccination decision in the same state tends to be correlated because individuals in the same state are exposed to the same policy environment, *e.g.*, federal-state (vaccination) policies and education system.

Within the clustered data, the assumption for the error term is $E[\epsilon^v_{ist}\epsilon^v_{jst}|X] = \rho\sigma^2_\epsilon > 0$ for individual i and j in the same state, s, and the same year of birth, t, where ρ is the intraclass correlation coefficient (ICC) and σ^2_ϵ is the residual variance (see Angrist and Pischke (2009, 231f)). There is no standard ICC method for binary outcome variables[40], but within regression frameworks, the influence of ICC depends on the level of clustering. Additionally, the regression model 2.3 with state-fixed effects allows state-specific intercepts that account for within-group error correlation next to cluster robust standard errors (so-called 'cluster-specific effects models' in Cameron and Miller (2016, p. 8)). Thus, clustering and cluster fixed effects are common practice (see Cameron, Gelbach, and Miller (2011, p. 242)).

[37]Since reunification, annual German internal migration has been approximately 5 %, of which $\frac{3}{4}$ takes place within the federal states (Destatis (2016, pp. 14, 20)).

[38]Normally, in ordinary-least-square regressions, standard errors are underestimated (overstates t-statistics) if the data are clustered (see Cameron and Miller (2016)).

[39]The KiGGS study design is a two-stage cluster sampling scheme (nested clusters). The first stage includes 167 study locations over all states; at the second stage, a random sample from the population register is drawn (see Kamtsiuris, Lange, and Rosario (2007)).

[40]Wu, Crespi, and Wong (2012) compared five ICC estimation procedures for binary data and discussed the shortcomings.

The other challenge in DD models is policy autocorrelation inducing a serial correlation problem. For example, the persistence of regional structural factors or a regional shock could induce time-series correlation at the state level. A practical solution suggested by Bertrand, Duflo, and Mullainathan (2004, p. 267) is to collapse data into a pre-period and post-period. This is mentioned in the regression equation 2.2. Additionally, here, another quick fix is to go to the next higher clustering level (*states*) for the calculation of the standard errors and to allow residual correlation over time within states (see Angrist and Pischke (2009, p. 319)). However, this approach has its price[41] in a reduction of clusters: with higher cluster aggregation and smaller group number, a common and simple correction[42] for the inference is to use a $T(G\text{-}1)$ distribution for p-values and critical values instead of the standard normal distribution (see Cameron and Miller (2016, p. 11)). In Section 2.7.1, I will review the robustness of the results depending on the chosen level of aggregation over the DD regression models.

2.6 Results

2.6.1 Age shift

The linear predictions by measles vaccination, age group and groups are presented in Table 2.4. For the first dose, both treatment and control group reach approximately 90 % for children aged under 3 years. Within treated states, the probability for the second dose is approximately 70 % for both age groups, 2 to 4 years and 5 to 6 years. Within in the younger age group, the probability is over 5 times larger and approximately 15 percentage points higher for the older children compared to those in the control group. The control group had a probability of 10 (49) % for age group 2 to 4 years (5 to 6).

For visualization of the three-way interaction regression results, I plot age- and group-specific marginal effects over periods. The left graph in Figure 2.3 plots the

[41] With clustering on the highest possible aggregation level, the state level here, the number of clusters is 16, raising the question of whether the standard errors are also calculated incorrectly. For Germany, there is research using similar evaluation frameworks for policy analyses on state-level variation, *e.g.*, to analyze bans (see Anger, Kvasnicka, and Siedler (2011) and Marcus and Siedler (2015)) or schooling duration (Pischke (2007)). An unfinished discussion exists about the minimum number of clusters and some 'solution' procedures (see Angrist and Pischke (2009, 231f), Hansen (2007a,b)). One example is a bias correction with the bias-reduced linearization procedure by Bell and McCaffrey, but it does not work in a DD model (Angrist and Pischke (2009, p. 239)).

[42] Stata takes into account the number of clusters and chooses the distribution for critical values.

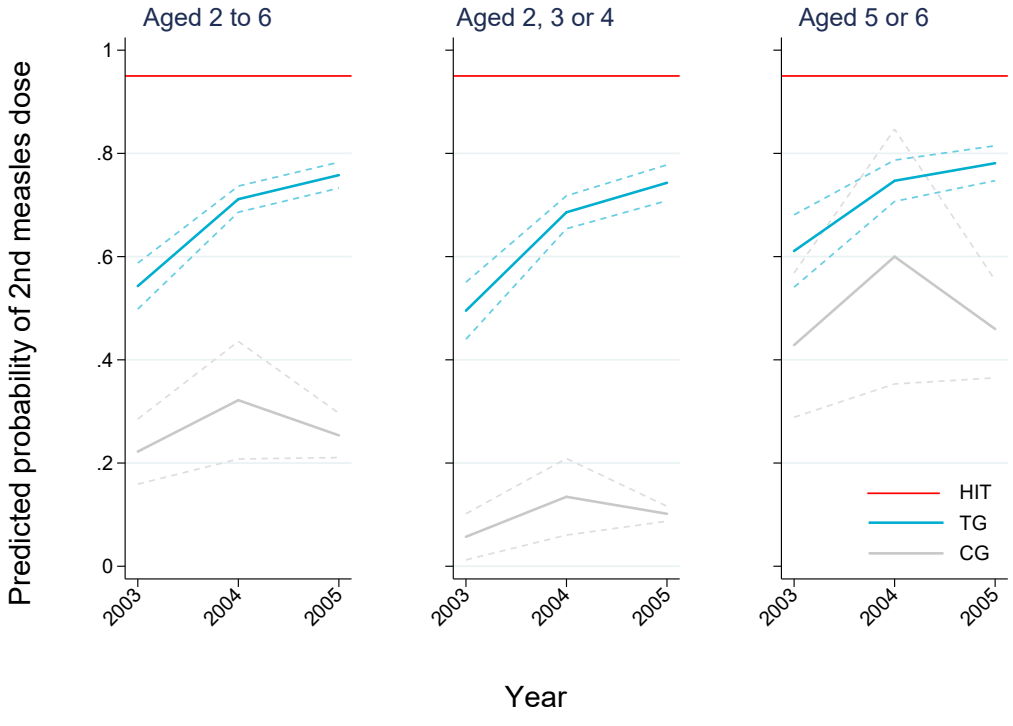
Table 2.4: Predicted vaccination probability by *dose*, *group* and *age*

Measles dose	**Age-group**	**TG** *Margin*	[95% *CI*]		**CG** *Margin*	[95% *CI*]	
Pr(*1 x m.d.* = 1)	1 – 2	0.897	0.866	0.930	0.890	0.847	0.934
Pr(*2 x m.d.* = 1)	2 – 4	0.676	0.647	0.704	0.102	0.077	0.128
	5 – 6	0.735	0.703	0.768	0.487	0.400	0.573

Notes: Average predicted probabilities calculated as the average of the probability among individuals in the LPM with *state* x *year of birth* clustered S.E. using *Stata*'s .margins.

predicted vaccination probability over the three survey periods: the average rate of all 2- to 6-year-old children increases from below 60 % in 2003 to 70 % in 2004 and 75 % in 2005 in the treatment group and no time effects within the control group. Looking at the age-specific rates by survey periods in the middle and right graphs, this upwards shift holds for both age groups in the treatment group. For the control group, predicted probabilities stay constant over time, with a higher level in the older age group. In the older age group, there are only significant differences in 2005.

Figure 2.3: 2nd measles vaccination by *agegroups* and *period*



Source: KiGGS data, own calculations.

Aside from the positive significant development within the treated states, one should note that the necessary herd immunity threshold of 95 % is still missed by 20 percentage points within this age group.

Assuming age squared and supplementary estimating a probit model, the results remain similar, and Figure 2.7 and 2.8 in the Appendix show the same pattern: the predicted probabilities are constant in the treatment group and increase in the control group with age (on the left). Over time, the probabilities increase for the treatment group for age 3 to approximately 5 but remain constant for the control group (on the right).

Additionally, the results raise the question as to whether this development directly causes the 2001 adaption, and if so, how strongly. For an age-specific causal interpretation of the policy effects, information on the second measles dose is required for the same age groups for the policy, but is not available. Therefore, a causal analysis for the direct policy effect is possible for children aged 7 years and older before and after the adaption. Fortunately, the indirect policy effect could be explored in this manner as well.

2.6.2 Policy evaluation

Figure 2.4 shows the means of vaccination rates by birth cohorts and groups for both measles vaccinations based on children with vaccination cards. The vertical black line is the 2001 recommendation adaption at the first affected birth cohorts and separates pre-period and post-period; the 2000 birth cohort was the first for which the primary vaccination should be finished at age 2, the 1996 birth cohort was the first for which school entry health exam and 7th birthday were after the 2001 adaption.

The DD regression results with one collapsed pre-cohorts and post-cohorts[43] (indicator variable $POST$ for post-cohorts) for both the first measles dose at age 2 and the second at age 7 are presented in Table 2.5. Column $M1$ is the DD model without additional explanatory variables. In specification $M2$ the parental, household and child controls are included. In $M3$ state fixed effects allow state-specific intercepts to account for differences in levels and for intracluster correlations.

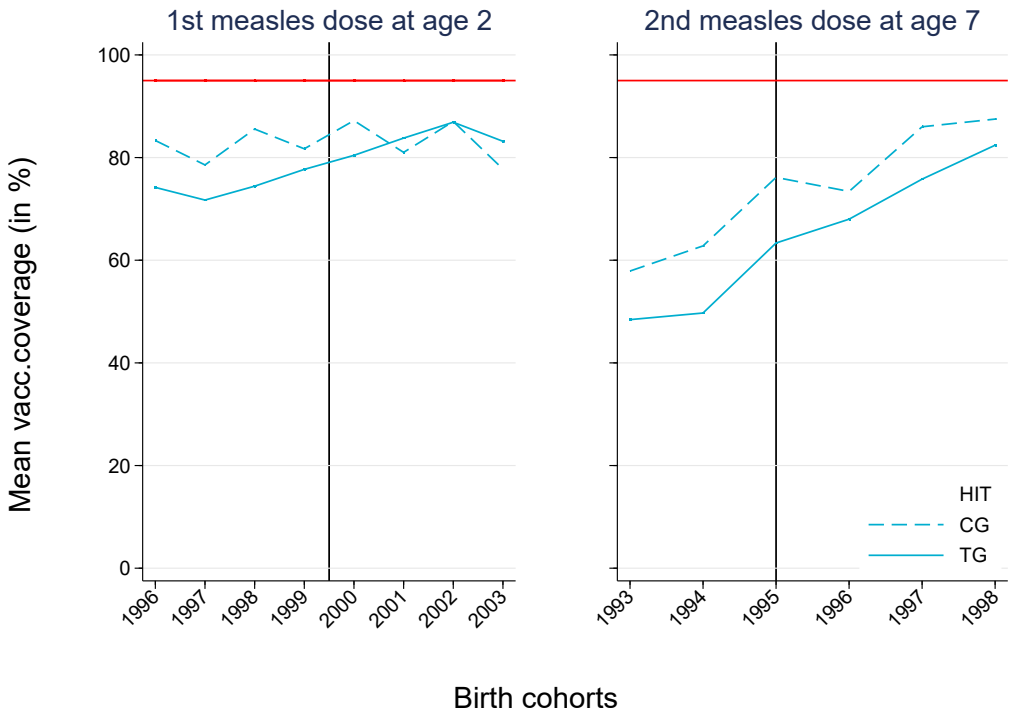
The temporal development in the vaccination probability (coefficient for $POST$ in $M3$) shows no change over time for the first measles dose and an increase of

[43]See Section 2.4.2 for period classification.

over 13 percentage points for the second dose, which implies a rather large secular trend.

The same for the average outcome levels in both groups in the pre-period that is estimated by the TG indicator: After including controls, there is no difference between control and treatment group for the first dose but a significant level difference of 7.5 percentage points in favor of the control group ($M2$). Specification $M3$ allows state-specific intercepts to account for possible within-group correlation, but they are not suitable for interpretation at the state level, as there are, for example, a number of small states.

Figure 2.4: 2nd measles vaccination by *age* and *period*



Notes: KiGGS data, own calculations. The 2001 adaption is indicated by the vertical line (black).

The parameters of interest, the DD coefficients (TG x POST), indicate the adaption policy effects: for both the first and second measles dose, the adaption increases the likelihood of a timely vaccination status by over 9 percentage points significantly at the 1% and 5% significance levels.[44] The average vaccination rate in the pre-period was 83.9 % (74.5 %) for the first measles dose and 60.4 %

[44]The results of nonlinear probit estimations for $M2$ are similar, calculating margins and the effect of interest as differences of cross differences (see Table 2.17 in the Appendix).

(48.4 %) for the second dose in the treatment (control) group. If one then sets the DD coefficients in relation to the counterfactual conditional in order to calculate the effect size, the result for the first vaccination is an increase of 12.2 % and for the second vaccination of 12.6 %.[45]

Table 2.5: Pre-post estimates - *1st and 2nd measles dose*

	Dep. var.: child's vaccination status		
	Pr(*1st measles dose at age 2* = 1)		
	$M1$	$M2$	$M3$
POST	-0.019 (0.023)	-0.038 (0.025)	-0.036 (0.025)
TG	-0.098*** (0.021)	-0.027 (0.022)	
TG x POST	0.119*** (0.028)	0.097*** (0.029)	0.098*** (0.027)
$N(df)$	3,075 (3,072)	3,075 (3,018)	3,075 (3,004)
adj. R^2	0.013	0.237	0.237
	Pr(*2nd measles dose at age 7* = 1)		
	$M1$	$M2$	$M3$
POST	0.176*** (0.031)	0.127*** (0.035)	0.133*** (0.038)
TG	-0.119*** (0.023)	-0.075*** (0.025)	
TG x POST	0.073 (0.045)	0.098** (0.044)	0.093** (0.044)
$N(df)$	2,459 (2,456)	2,459 (2,397)	2,459 (2,383)
adj. R^2	0.064	0.196	0.202
Controls		√	√
State FE			√

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. Controls are control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of hepatitis B/first measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

Taking into account the level difference in the previous period, this means a higher timely vaccination probability for the first vaccination in the treatment group after adaption and a resolution to the level of the control group, which itself experienced a significant increase in this time.

Next, the DD framework is stratified with two treatment regions: to consider the different immunization policies until reunification all 'old' (*West* Germany) and all 'new' federal states except the control group (*East* Germany) are grouped

[45] first m.d.: $\frac{9.8}{83.9-3.6}$; second m.d.: $\frac{9.3}{60.4-13.3}$.

together. Despite the policy history, both groups respond similarly to the adaptation: For both measles doses, the point estimate is greater in the old states. For the second dose, the DD point estimate of the East states is 6 percentage points, but not significantly different from the massive and significant time effect of the control group (0.133). The results are shown in Table 2.6 column *East* and *West*.

Table 2.6: Pre-post estimates - *east/west*

	Dep. var.: child's vaccination status		
	Pr(*1st measles dose at age 2* = 1)		
	All	*East*	*West*
POST	-0.036 (0.025)	-0.036 (0.025)	
TG x POST	0.098*** (0.027)	0.085*** (0.030)	0.103*** (0.028)
adj. R^2	0.234	0.237	
	Pr(*2nd measles dose at age 7* = 1)		
	All	*East*	*West*
POST	0.133*** (0.038)	0.133*** (0.038)	
TG x POST	0.093** (0.044)	0.060 (0.054)	0.104** (0.046)
adj. R^2	0.157	0.202	

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of hepatitis B/first measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

As the adaption is shown to have positive significant effects on both vaccination decisions, possible short-term dynamic effects within the post-period are considered below.

In Table 2.7, the last column are the results of the dynamic DD regression with several cohorts before and after policy adaption to analyse (short-term) dynamic policy patterns. The middle column contains the previous results for comparison.

Table 2.7: Pre-post estimates - *policy dynamics*

	Dep. var.: child's vaccination status	
	Pr(*1st measles dose at age 2* = 1)	
TG x POST	0.098*** (0.027)	
TG x 1996 (pre)		0.017 (0.019)
TG x 1997 (pre)		0.034 (0.023)
TG x 1998 (pre)		reference group
TG x 2001 (post)		0.120*** (0.016)
TG x 2002 (post)		0.103*** (0.023)
TG x 2003 (post)		0.114*** (0.025)
adj. R^2	0.237	0.238
Common trend		$F(2, 93)$ =
$F(n, m)(Prob > chi2)$		1.21 (0.3031)
	Pr(*2nd measles dose at age 7* = 1)	
TG x POST	0.093** (0.044)	
TG x 1993 (pre)		0.022 (0.027)
TG x 1994 (pre)		reference group
TG x 1996 (post)		0.098*** (0.027)
TG x 1997 (post)		0.084*** (0.028)
TG x 1998 (post)		0.147*** (0.037)
adj. R^2	0.207	0.207
Common trend		$F(1, 78)$ =
$F(n, m)(Prob > chi2)$		0.72 (0.3992)

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of hepatitis B/first measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

First, looking at the significance of the pre-policy interaction terms, the *common trend assumption* for the first measles dose (the years 1996 and 1997 (TG x 1996 and TG x 1997) compared to 1998, the last-pretreatment period (reference group)) and the second (TG x 1993 compared to reference group 1994) holds: the null hypothesis that the temporal development between the control and treatment groups is equal before the policy introduction could not be rejected.

Looking at the dynamics of policy, for all post-cohorts affected by the adaption, the vaccination probability increases significantly: the first dose point estimates of the post-policy period appear to be constant for the birth cohorts 2001 to 2003 (TG x 2001=.12 to TG x 2003=.114) and increasing, but are not statistically different, for the cohorts 1996 to 1998 (TG x 1996=.098 to TG x 1998=.147).

Comparing these results for the second dose with the group differences of the age shift in Section 2.6.1 the effects here are clearly smaller, but causal. As previously described within the DD analyses, older cohorts had to be included, and for that, the recommendation adaptation replaces the upcoming previous vaccination recommendation: when the adaption came into force in 2001, the post-period cohorts (1996 to 1998) were already 3 years and older, and for them, the vaccination for the second dose was immediately due. Additionally, the pre-policy practice in both groups is fully considered to control the vaccination status in the context of the School Medical Entry and, if necessary, to initiate vaccination.

2.7 Robustness

In this section, I address concerns regarding my empirical strategy as mentioned before. First, the clustering level will be changed, allowing different error relationships within the chosen units and calculating the corresponding standard errors. Second, instead of birth cohorts, I use school cohorts for the second measles dose, because the school entry health exam is appointed in the preschool year for the forthcoming school beginners. Third, the parental vaccination decision for measles might be influenced by other contemporaneous vaccination decisions (here rubella and mumps). Therefore, I estimate a three-equation model as seemingly unrelated. Fourth, I check for parallel events that possibly influence vaccination demand. Finally, I perform a placebo test on a preventive health examination.

2.7.1 Clustering within DD

Avoiding potential biases in the estimation of the standard errors (see the discussion in Section 2.5.3), it is important to account for the possible correlation of the errors ϵ_{ist} across time and/or space.

Checking the robustness of the main results, I apply different clustering levels. I allow for any covariance structure within regional areas over time by computing the standard errors clustered at the federal state (policy aggregation level) and the sample points (sampling structure) level. Additionally, I compute standard errors clustered at the state-time level with only two cohort periods (pre-post) and compare these standard errors with the chosen level in the main specification, the state and year of birth level. Finally, robust and OLS standard errors are calculated. Shown in Table 2.8 are the standard errors at the different clustering levels.

In the upper panel in Table 2.8, the policy effect for the first measles dose appears robust over all specifications. For the second dose, the standard errors are increasing with lower aggregation assumption for the clustering.

Table 2.8: Pre-post estimates - *clustering*

		Dep. var.: child's vaccination status				
	Coef.	**Pr**(*1st measles dose at age 2* = 1)				
TG x POST	0.098	***(0.014)	***(0.010)	***(0.027)	***(0.038)	**(0.042)
No. of clusters		16	32	94		
		Pr(*2nd measles dose at age 7* = 1)				
TG x POST	0.093	***(0.019)	***(0.014)	** (0.044)	* (0.056)	* (0.056)
No. of clusters		16	32	79		
Cluster-standard errors at level		*state*	*state x pre-post*	*state x year of birth*	*robust*[1]	*OLS*

Notes: LPM with level-clustered standard errors are in parentheses. [1] White heteroskedastic. Significance: *p<0.1, **p<0.05, ***p<0.01. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of hepatitis B/ first measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

2.7.2 School cohorts

The STIKO recommendations for the second measles dose suggested School Medical Entry in the preschool year as the practical point in time to control the up-to-date vaccination status. One might consider school cohorts as the affected policy cohort. Next to public health policy, education and school policy is administered by the federal states. Therefore, school cohorts differ slightly between states because the school age cut-off[46] is different within a three month span: There are 8 states with a cut-off at June 30, 2 states with an August 30 cut-off and 6 with a September 30 cut-off. These dates were considered in the analysis. The policy adaption in 2001 was released in July next to the start of school, so the 2002/2003 school cohort, born in the second half of 1995 and the first half of 1996, was the first cohort that was affected by the adaption.

The results for the school cohorts are robust with the standard and the dynamic policy model; with school cohorts, the increasing dynamics are slightly steeper.

Table 2.9: Pre-post estimates - *cohorts*

Dep. var.: child's vaccination status **Pr**(*2nd measles dose at age 7* = 1)			
Birth cohorts (*N*=2,459)		*School cohorts*(*N*=2,669)	
TG x POST	0.093** (0.044)	TG x POST	0.113** (0.051)
adj. R^2	0.212	*adj.* R^2	0.183
TG x 1993 (pre)	0.022 (0.027)	TG x 1993/94 (pre)	-0.032 (0.020)
TG x 1994 (pre)	reference group	TG x 1994/95 (pre)	reference group
TG x 1996 (post)	0.098***(0.027)	TG x 1995/96 (post)	0.068** (0.028)
TG x 1997 (post)	0.084***(0.028)	TG x 1996/97 (post)	0.085***(0.026)
TG x 1998 (post)	0.147***(0.037)	TG x 1997/98 (post)	0.233***(0.029)
adj. R^2	0.252	*adj.* R^2	0.199
		Common trend $F(n,m)(Prob > chi2)$	$F(1,78)$ = 2.51 (0.1175)

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* and at *state* x *school cohort* level. Significance: *p<0.1, **p<0.05, ***p<0.01. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of hepatitis B/first measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

[46]The child starts school in the year in which his or her 6th birthday is before the cut-off.

2.7.3 MMR - seemingly unrelated

Since the mid-1980s, the trivalent combination vaccine measles-mumps-rubella has been initially recommended for simultaneous vaccinations in addition to the existing monovalent vaccines. Therefore, the measles vaccination can also be viewed as a conditional, non-independent decision with the other two vaccinations.

The seemingly unrelated regression (SUR) model[47]:

$$Pr(VAC^{v}_{is\tau} = 1|.) = \gamma_s + \lambda_\tau + \beta D_{s\tau} + X_{is\tau}\delta + \epsilon^{v}_{ist}$$

with $VAC^{v}_{is\tau}$ is an indicator for whether individual i living in state s in cohort τ has been vaccinated against $v = 1, .., V$ with $V = 3$ for set (measles, mumps, rubella).

In Table 2.10 the results highlight a similar pattern for all three vaccinations: starting from a higher level in the control group, an increase over time for both the control and treatment groups could be observed with an additional policy effect of over 10 percentage points for all vaccinations.

Table 2.10: Pre-post estimates - *seemingly unrelated*

	Dep. var.: child's vaccination status **Pr**(*2nd ... dose at age 7* = 1)		
	measles	*mumps*	*rubella*
POST	0.122***(0.037)	0.120***(0.037)	0.114***(0.035)
TG	-0.073***(0.025)	-0.075***(0.025)	-0.081***(0.025)
TG x POST	0.101** (0.044)	0.101** (0.044)	0.101** (0.044)
N	2,459		

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. Controls are control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of corresponding first dose), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

[47] I used Roodman's Stata .cmp that uses a maximum likelihood estimation and allows for clustering (see Roodman (2009)).

2.7.4 Outbreak or policy?

The causal inference within DD regression frameworks persists on the common trend assumption and that there are no other secular trends or events influencing the vaccination decision. Regional outbreaks could be such events. Therefore, I divide the treatment states into two groups, one with a documented measles outbreak in the post-policy period and the other with no such event. Database are publications of the European Centre for Disease Control and Prevention (ECDC) describing regional outbreaks (see Hellenbrand et al. (2003), Siedler (2005), Siedler, Hellenbrand, and Rasch (2002), and Siedler et al. (2006)): For the period 2001 to 2005, there were outbreaks in regions of Bavaria (November 2001 and March-July 2005), Lower Saxony (November 2001-March 2002), North Rhine Westphalia (January-March 2002), Hesse (January-May 2005), and Schleswig-Holstein (January-April 2001). Measles outbreaks and their reports are usually restricted on local areas, *e.g.*, counties. However, there is no local information in the KiGGS data within the states. Therefore, the separation can be made only very roughly at the level of the federal states.

Table 2.11 shows the results for both measles vaccinations: For the first and the second measles dose, there is no difference between the two groups.

Outbreaks may affect demand, but the question is whether this occurrence is only regional. The reporting of measles epidemics is often very medial, so it is not limited to local print media in Germany, especially in the considered time period. Furthermore, measles outbreaks are not uncommon, since due to their infection potential at low vaccination rates, outbreaks occur regularly and reoccur partly regionally.

In Section 2.8, I will take a closer look at incidences in the period after policy adaption.

Table 2.11: Pre-post estimates - *outbreaks*

	Dep. var.: child's vaccination status		
	Pr(*1st measles dose at age 2* = 1)		
	All	*Outbreaks*[1]	*No outbreaks*
TG x 2001 (post)	0.120***(0.016)	0.146***(0.017)	0.092***(0.024)
TG x 2002 (post)	0.103***(0.023)	0.094***(0.032)	0.108***(0.030)
TG x 2003 (post)	0.114***(0.025)	0.101***(0.022)	0.125***(0.038)
adj. R^2	0.235	0.238	
N	3,075	3,075	
	Common trend	$F(2, 93) =$	$F(2, 93) =$
	$F(n, m)(Prob > chi2)$	2.16 (0.1216)	0.10 (0.9070)
	Pr(*2nd measles dose at age 7* = 1)		
	All	*Outbreaks*[1]	*No outbreaks*
TG x 1996 (post)	0.098***(0.027)	0.133***(0.057)	0.056* (0.032)
TG x 1997 (post)	0.084***(0.028)	0.070* (0.054)	0.100***(0.034)
TG x 1998 (post)	0.147***(0.037)	0.157***(0.056)	0.131***(0.036)
adj. R^2	0.167	0.208	
N	2,459	2,459	
	Common trend	$F(1, 78) =$	$F(1, 78) =$
	$F(n, m)(Prob > chi2)$	0.01 (0.9052)	2.30 (0.1335)

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. [1] Bavaria, Hesse, Lower Saxony, North Rhine Westphalia, and Schleswig-Holstein. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of Hepatitis B/first Measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

2.7.5 Placebo test

As a final robustness analysis and to assess whether my results might be driven by a secular trend, I look at the adaption effects of a placebo health outcome. The outcome variable of the policy adaption, the measles status of the child, is replaced by another prevention measure that was not the content of the adaption but is done in parallel. One possibility for this would be a child health screening such as well-child visits. The $U9$ scheduled for the age of 5 years (between the 60th and 64th month of life) or $U10$ after the 7th birthday would be suitable. After the U10, which has been offered in Germany since 2006, no information is available on the birth cohorts used. In contrast to age-recommended vaccinations, the execution of these examinations must take place within a certain time interval and cannot be made up after being exceeded (see Gemeinsamer Bundesausschuss (2017, p. 7)).

Table 2.12: Pre-post estimates - *placebo*

	Dep. var.: child's vaccination status	**Dep. var.: child's screening status**
	Pr(*2nd m.d. at age 7* = 1)	**Pr**($U9$ *screening* = 1)
TG x 1993 (pre)	0.022 (0.027)	-0.022 (0.023)
TG x 1994 (pre)	reference group	reference group
TG x 1996 (post)	0.098***(0.027)	0.026 (0.016)
TG x 1997 (post)	0.084***(0.028)	0.010 (0.017)
TG x 1998 (post)	0.147***(0.037)	-0.001 (0.023)
adj. R^2	0.167	0.065
N	2,459	2,459
Common trend $F(n, m)(Prob > chi2)$		$F(1, 78)$ = 0.95 (0.3321)

Notes: LPM with clustered standard errors (in parentheses) at the *state* x *year of birth* level. Significance: *p<0.1, **p<0.05, ***p<0.01. [1] Bavaria, Hesse, Lower Saxony, North Rhine Westphalia, and Schleswig-Holstein. All specifications include state FE and control variables child (gender, medical vaccination advice, reasons against vaccination, chronic diseases, well-child visits, child care and/or school entry, timely vaccination record of Hepatitis B/first Measles), and control variables household (income, ISCED-97, parental occupation, migration background, no. of children in household, firstborn indicator, local living area).

Table 2.12 presents the results of the placebo outcome regression. For this child screening, there are no trend differences between the both groups in the

considered period of time either before timeliness adaptation in 2001 or in the years thereafter. This and the previous outbreak analysis give no indication of a parallel policy trend and support the previous causal analysis.

2.8 A look at incidence rates in the long-run

The long-term expectations of a vaccination policy are high immunization rates and associated declining disease rates in the population. One goal of the timeliness adaption for the second measles vaccination in the context of primary immunization is to increase the protection of children under the age of 5 years.

Since 2001, it has been a legal obligation to report all notifiable disease cases and pathogens to the RKI. In Figure 2.5, the incidence cases per million population in Germany are plotted from 2001 to 2016.[48] In addition, the proportion of incidence cases that occurred in the age groups 0 to less than 5 years and 0 to less than 15 years are calculated. In line with the policy change, the two groups from the previous analyzes are again compared; the left figure shows the average over all federal states without Saxony, and the right contains the Saxon measles cases.

First, it can be seen that there were still regular outbreaks during this period, which occurred at a distance of 2 to 4 years (gray bars). It can also be stated that in 7 of the 16 years, Saxony had an incidence rate of less than 1 case per million population and generally below-average incidence rates. In recent years, however, the local rates in Saxony are increasing with an above-average high in 2016.

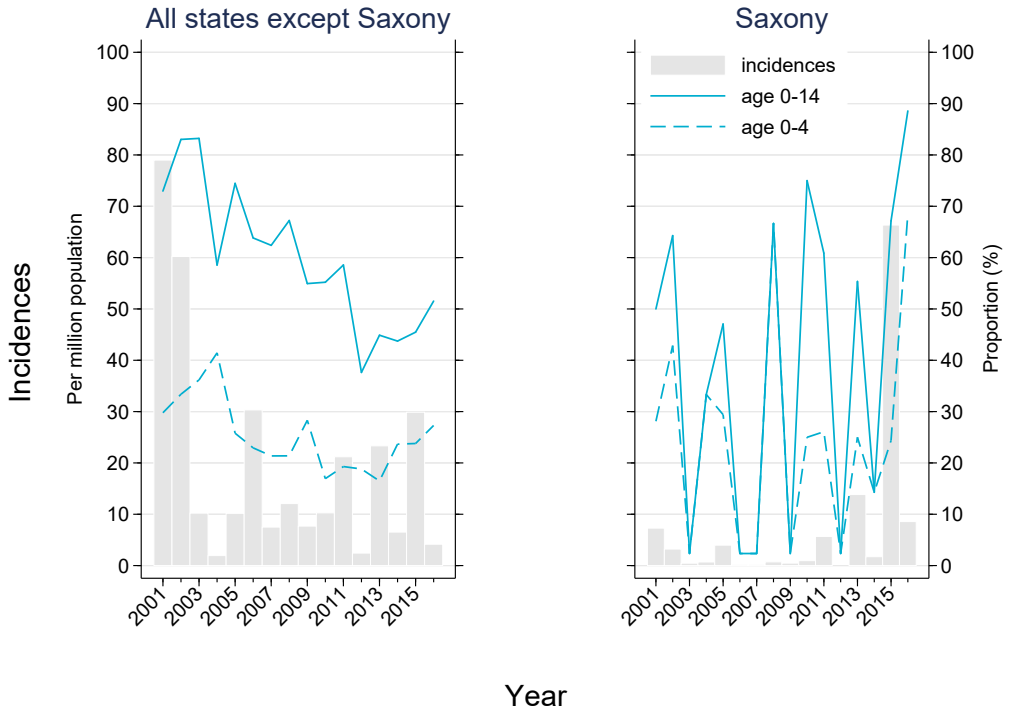
Second, in the states where the measles timeliness adaption was implemented in 2001, there is a steady decline in the proportion of disease cases in both age groups. For Saxony, in the years in which the incidence rate was over 5 cases per million population, the proportion of cases among children under 15 years was 50 % or even higher.

The average German incidence rates, however, are regionally very different: for instance, in Germany, the absolute incidences were over 1,500 cases[49] in 2001, 2002, 2006, 2011, 2013, 2015, and 2017. For example, in North-Rhine Westphalia, in 2 of these 7 years, the regional quota was over 50 % (53 in 2017; 74 in 2006), and in 2 of these 7 years, the quota was over 25 % (26 in 2001; 34 in 2002).

[48] Data for measles incidences are available until 2018, but population statistics end in 2016.

[49] Relative to the German population, with approximately 82 million inhabitants, the cases are over 18 per million population, which is half of the worldwide average in the year 2015 and over 18 times above the measles eradication definition (see Section 2.1).

Figure 2.5: Measles cases by *per million* and *proportions of age groups*



Source: Data from the Robert Koch Institute *SurvStat@RKI* 2.0, and from *Destatis*, own calculations.

2.9 Conclusion

According to current recommendations, timely primary vaccination should be completed for all children in the first two years of life. Almost all recommended immunizations require a certain number of vaccinations to ensure the necessary immune response.

The results here, representing further analyses of health department's reporting data and claims data of health insurance, show a positive development of vaccination status for Germany in the last 15 years. The introduction of the adaption already shows initial success in the short and long run. Rieck et al. (2013) analyzed claims data for the birth cohorts 2004 to 2006 regarding age and vaccination status: their results show high first measles dose status and an increasing second measles dose status for 2- and 3-year-old children, which are in line with my findings. Compared to the rates at the school entry health exam, the level of the first dose is reached already at a young age (see Figure 2.1). For the second dose, the difference between the rates at 3 years and before school entry differs between 10 and 15 percentage points when including the Free State

of Saxony in the data.

It can be assumed that the opportunity costs for measles vaccinations decreased by the 2001 adaption because the vaccinations take place next to the well-child visits in the first years of life. Due to higher parental screening compliance in the first years, doctor visits are increased, time costs are reduced, and the probability of having already experienced an infection is lower under otherwise identical conditions. These results give reason to certify a nonbinding vaccination policy; however, the incentives should be used as best as possible, especially with a recommendation policy framework.

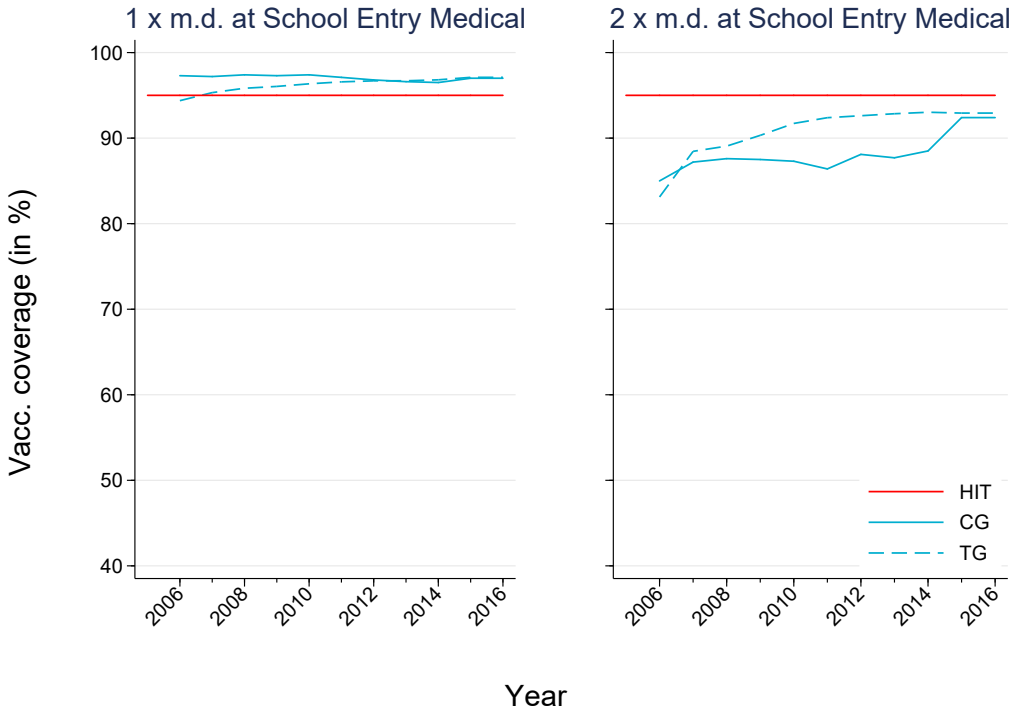
From an economic point of view, it is of great benefit to know how the distribution of those policy effects is accomplished on both market sides, *i.e.*, vaccination demand by parents and doctoral supply efforts. This consideration is important to transfer and implement policy adaption efficiently. Limited by the data, this remains here an open question.

Despite these positive findings, the coverage rates are still not sufficient to prevent outbreaks, and in 2019, we are still far from having the nationwide coverage required to build adequate protection and advance the elimination of the measles virus.

The main problem is that a relatively high immunization rate is needed in the population and a pure recommendation vaccination policy reaches its limits. Whether in the case of measles Germany should finally achieve a vaccination mandate, such as France and Italy, or intervene in other ways remains an exciting political discussion.

2.10 Appendix

Figure 2.6: Measles status by *year of school entry health exam*



Source: Data from Robert Koch Institute (2008-2018), own calculations.

Table 2.13: Explanation of variables

Dependent variables	
1st m.d.[b]	measles vaccination status: 1 = child got 1st measles vaccination timely; 0 = else
2nd m.d.[b]	measles vaccination status: 1 = child got 2nd measles vaccination timely; 0 = else
$U9$[b]	screening status: 1 = child got $U9$ well-child visit; 0 = else
Control variables	
Child characteristics	
Gender[b]	1 = child is female; 0 = else
Child care under age 2[b]	1 = child care in the first/second year of life; 0 = else
School enrollment (4 cat.)	regular; delayed; earlier; not yet
Well-child visit[b] (U6 to U9)	1 = child visited the screening; 0 = else
Medical advice against vaccination (4 cat.)	MMR or one single component; all; single vaccination; non
Chronic illness under 2[b] (N=14)	1 = child has illness within the first second years of life; 0 = else
Chronic illness under 3[b] (N=14)	1 = child has illness within the first three years of life; 0 = else
Reasons against vaccination (3 cat.)	yes; no; don't know
Vaccination side effects (3 cat.)	yes; no; don't know
Timely hepatitis B vaccination[b]	1 = child got vaccination at age of 2; 0 = else
Parental and household characteristics	
Professional qualification (8 cat., mother and father)	apprenticeship; professional training school, vocational college; specialist college; technical college, engineering college; university, polytechnic; other training qualification; No professional qualification; still in professional training.
Education (3 cat.)	max. household ISCED (1997): low, middle, high
Income (4 cat.)	0 < 1,500; 1,500 < 2,250; 2,250 < 3,000; 3,000 and more
Migration background (3 cat.)	both sides; one-sided; non
Children in household (4 cat.)	single child; two; three; four and more
Birth order[b]	1 = child is firstborn, 0 = else
Region (4 cat.)	rural (<5' inhabitants), small town (5' < 20'), middle town (20' < 100'), urban (> 100')

[b] binary

Table 2.14: Descriptive statistics - *part* 1

	full sample	**Treatment group (TG)**	**Control group (CG)**
N	6,272	5,571	701
Dependent variables			
1st measles dose[1]	0.94	0.93	0.95
2nd measles dose[1]	0.72	0.70	0.26
1st measles dose (< 2)[2]	0.77	0.76	0.82
2nd measles dose (< 7)[2]	0.62	0.61	0.69
$U9$[2]	0.64	0.64	0.65
Control variables			
Female	0.49	0.49	0.52
Age	12.9	12.9	12.6
Child care under age 2	0.19	0.18	0.32
Timely hepatitis B vaccination[2]	0.59	0.57	0.74
School enrollment			
Regular	0.44	0.44	0.43
Delayed	0.03	0.03	0.04
Earlier	0.03	0.03	0.01
Not yet	0.50	0.50	0.51
Well-child visit			
U6	0.96	0.96	0.98
U7	0.94	0.94	0.95
U8	0.73	0.73	0.73
Medical advice against vaccination			
MMR or one component	0.01	0.01	0.00
All	0.00	0.00	0.00
Single vaccinations	0.03	0.04	0.00
Non	0.95	0.95	0.98
Parental reasons against vaccination			
Yes	0.09	0.09	0.05
No	0.91	0.91	0.95
Don't know	0.00	0.00	0.00
Vaccination side effects			
Yes	0.02	0.02	0.02
No	0.98	0.98	0.98
Don't know	0.00	0.00	0.00

Notes: [1] Mean values refer to cohorts of the up-to-date sample, [2] Mean values refer to cohorts of the pre-post sample.

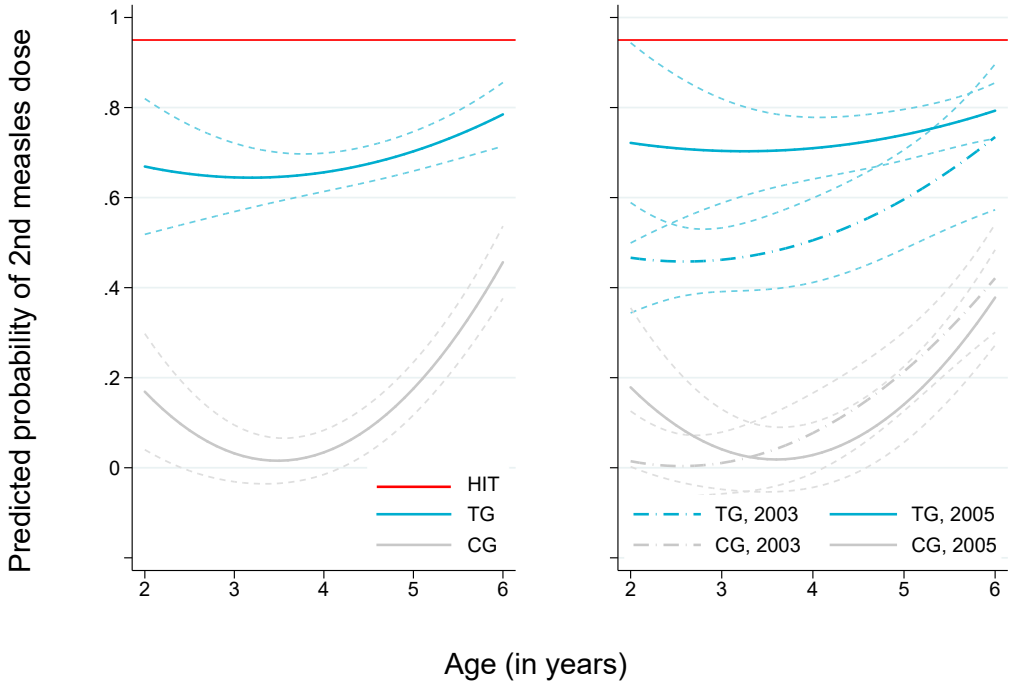
Table 2.15: Descriptive statistics - *part* 2

	full sample	**Treatment group (TG)**	**Control group (CG)**
N	6,272	5,571	701
Professional qualification (mother/ father)			
Apprenticeship	0.43/0.42	0.44/0.41	0.40/0.47
Professional training school	0.16/0.11	0.16/0.11	0.17/0.17
Vocational college	0.12/0.15	0.12/0.15	0.16/0.11
Engineering college/ applied university	0.06/0.09	0.06/0.09	0.09/0.08
University	0.11/0.15	0.11/0.16	0.14/0.14
Other training qualification	0.03/0.02	0.03/0.03	0.01/0.01
No professional training	0.08/0.05	0.08/0.05	0.01/0.01
Still in professional training	0.01/0.00	0.01/0.00	0.01/0.00
Household ISCED education			
low	0.03	0.03	0.01
middle	0.48	0.48	0.50
high	0.49	0.49	0.49
Household income			
0 < 1,500	0.17	0.16	0.21
1,500 < 2,250	0.27	0.26	0.36
2,250 < 3,000	0.30	0.30	0.25
3,000 and more	0.27	0.28	0.18
Migration background			
both sides	0.09	0.10	0.01
one-sided	0.08	0.08	0.02
non	0.83	0.81	0.97
Children in household			
Single child	0.18	0.17	0.22
Two	0.52	0.53	0.47
Three	0.20	0.20	0.20
Four and more	0.10	0.09	0.11
Firstborn	0.47	0.47	0.47
Region			
Rural	0.26	0.25	0.38
Small town	0.27	0.26	0.31
Middle town	0.25	0.26	0.15
Urban	0.22	0.23	0.17

Table 2.16: Descriptive statistics - *part* 3

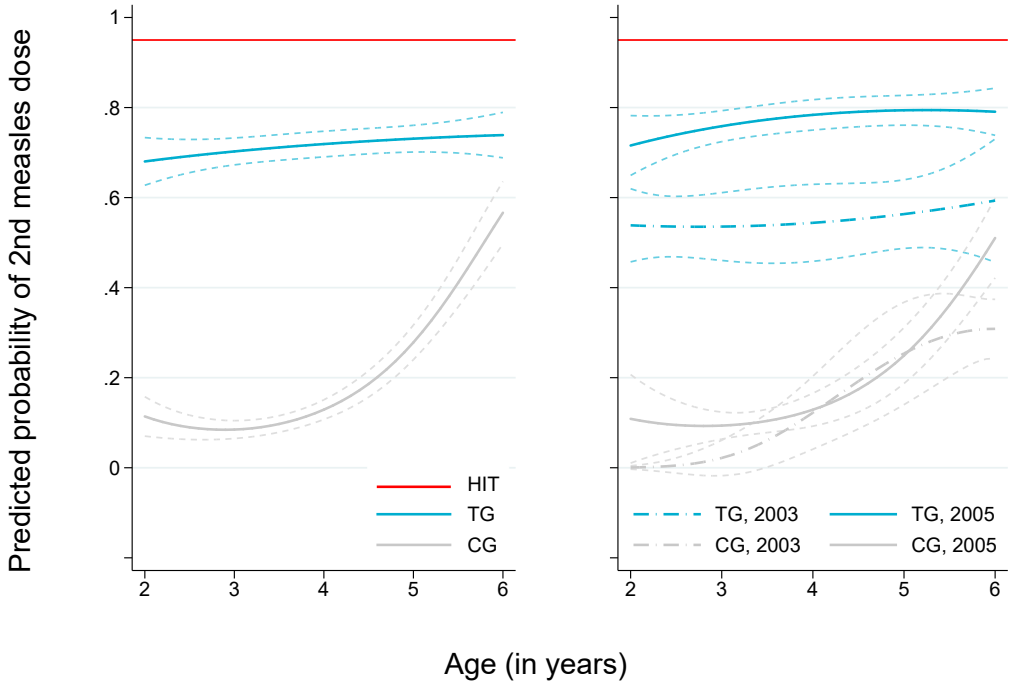
	full sample	**Treatment group (TG)**	**Control group (CG)**
N	6,272	5,571	701
Chronic disease under 3 *years*			
Allergic rhinitis/ conjunctivitis	0.01	0.01	0.01
Atopic dermatitis/ eczema	0.12	0.12	0.12
Asthma	0.01	0.01	0.01
Chronic obstructive pulmonary disease	0.10	0.11	0.07
Pneumonia	0.06	0.06	0.04
Otitis media	0.33	0.33	0.32
Heart disease	0.02	0.02	0.02
Anemia	0.01	0.01	0.01
Epileptic seizure	0.03	0.03	0.03
Thyroid	0.00	0.00	0.00
Diabetes mellitus	0.00	0.00	0.00
Scoliosis	0.00	0.00	0.00
Migraine	0.00	0.00	0.00
Other diseases	0.13	0.13	0.16
Chronic disease under 2 *years*			
Allergic rhinitis/ conjunctivitis	0.01	0.01	0.00
Atopic dermatitis/ eczema	0.10	0.10	0.09
Asthma	0.01	0.01	0.01
Chronic obstructive pulmonary disease	0.08	0.08	0.06
Pneumonia	0.04	0.05	0.04
Otitis media	0.22	0.22	0.21
Heart disease	0.02	0.02	0.01
Anemia	0.01	0.01	0.01
Epileptic seizure	0.02	0.02	0.02
Thyroid	0.00	0.00	0.00
Diabetes mellitus	0.00	0.00	0.00
Scoliosis	0.00	0.00	0.00
Migraine	0.00	0.00	0.00
Other diseases	0.09	0.09	0.11

Figure 2.7: 2nd measles vaccination by *age* and *period*



Source: KiGGS data, own calculations.

Figure 2.8: 2nd measles vaccination by *age* and *period* - *probit*



Source: KiGGS data, own calculations.

Table 2.17: Predicted vaccination probability by *group* and *time* - *probit*

	Pr(*1 x m.d.* = 1)			**Pr**(*2 x m.d.* = 1)		
	Margin	[95% *CI*]		*Margin*	[95% *CI*]	
Pre # CG	0.798	0.762	0.834	0.572	0.545	0.599
Pre # TG	0.769	0.750	0.789	0.504	0.469	0.539
Post # CG	0.754	0.710	0.798	0.708	0.639	0.776
Post # TG	0.832	0.812	0.851	0.730	0.691	0.769
H_0 : Pre # CG=Pre # TG		0.1850			0.0002	
H_0 : Pre # CG=Post # CG		0.1131			0.0002	
H_0 : Pre # TG=Post # TG		0.0000			0.0000	
H_0 : Post # CG=Post # TG		0.0009			0.5853	
	Contrast	[95% *CI*]		*Contrast*	[95% *CI*]	
TIME#TG	0.107***	0.048	0.167	0.091**	0.0017	0.179

Notes: Average predicted margins calculated among group and time using *Stata*'s .margins with contrast option for the difference of cross-differences.

Chapter 3

Transmission of weight status (mis)perceptions

Is family contagious?[1]

3.1 Introduction

Unhealthy weight is associated with a number of diseases. In the case of overweight or obesity individuals face an increased risk of, for instance, cardiovascular diseases, diabetes, and arthropathy (see Cawley and Meyerhoefer (2012), Doolen, Alpert, and Miller (2009) and Wabitsch et al. (2005).[2] In the industrialized world, population weight irresistibly increases across all population groups (see, *e.g.*, OECD/EU (2016, pp. 96–100) including children and adolescents (see Deckelbaum and Williams (2001) and Wabitsch et al. (2005))) making the obesity epidemic a concern for public health. But also underweight puts individuals at risk. Strong underweight, that may be the result of malnutrition or eating disorders, is associated with a lower probability of hormonal balance and might also have an impact on growth and the functioning of the immune system (see Fairburn (2008, p. 155)).[3] To identify suited policy measures to fight unhealthy weight a better understanding of the determinants of body weight is needed.

Ali, Amialchuk, and Renna (2011, p. 828) argued that – in a genetically stable population – the increase in obesity rates can only be attributed to an increase

[1]This chapter based on a joint research project with Robert Nuscheler and Kerstin Roeder.

[2]Doolen, Alpert, and Miller (2009, Table 2, p. 161) provide a presumably complete list of diseases that are associated with overweight and when they are likely to occur in the lifecycle.

[3]There are adverse effects of unhealthy weight outside the health realm. Han, Norton, and Stearns (2009), for instance, show that there is a negative association between BMI and wages and that this relationship is particularly strong in occupations with intense social interactions.

in calorie intake, a reduction in physical activity, or any combination of the two. Although these behavioral factors are uncontested, it remains unclear why, on average, insufficient measures are being taken to lose weight once individuals have reached a critical weight. Christakis and Fowler (2007) suggest that a heavier weight environment increases the tolerance towards obesity. This mechanism leads them to conjecture that obesity is contagious, that is, that it may spread through social networks. Indeed, they find that the likelihood of an individual becoming obese significantly increases when the partner or close friends became obese. This interpretation was challenged by Cohen-Cole and Fletcher (2008), who showed that unbiased estimation of the Christakis-Fowler model renders their transmission process statistically insignificant.

Among others, Kuchler and Variyam (2003) argued that weight perceptions are crucial for healthy weight management, *e.g.*, if an obese individual perceives its weight status as uncritical nothing will be done about body weight. In fact, such misperceptions may explain the ineffectiveness of obesity prevention programs (Müller et al. (2005). But also healthy-weighed individuals' misperceptions may lead to adverse outcomes once unhealthy dietary restraints are taken (see, *e.g.*, Greiner, Schillmöller, and Färber (2010) and Ursoniu, Putnoky, and Vlaicu (2011)).[4] It is, thus, crucial to understand how weight perceptions are being formed. Weight status perceptions are likely to be developed during childhood and adolescence pointing to the key role of parents, first, in the formation of weight status perceptions in their children and, second, for the effectiveness of intervention strategies or preventive actions (see Warschburger and Kröller (2012)).[5] Ali, Amialchuk, and Renna (2011), Burke, Heiland, and Nadler (2010), Maximova et al. (2008), and Röhrig, Giel, and Schneider (2012) found that the weight environment of an individual is an important determinant of individual weight perception: If an individual's peers are growing bigger and bigger, the benchmark against which the own weight is evaluated increases leading to a rise in the likelihood of weight under-perception. The literature appears incomplete as it only analyzes *indirect* perception transmission (from the weight environment to the child) but neglects *direct* transmission, *i.e.*, that parents transmit their weight perception to their offspring.

Bisin and Verdier (2001) developed a theoretical model that nicely combines

[4]While weight misperceptions are bad in terms of physical health they may be good for mental health as they might protect an individual's self-esteem (Neubauer (1976, p. 118)).

[5]For an overview on transmission of health within the family see Ahlburg (1998).

direct and indirect transmission channels. Moreover, they allow parents to tilt the balance between these two channels by exerting some costly effort. Arguing that weight perceptions are rooted in weight preferences, we adapt Bisin and Verdier's (2001) model to explain weight perception formation in children and adolescents. The effort parents can exert to increase the probability of direct transmission is what we call *communication*. There are three perception types, namely, correct perceivers, under-perceivers, and over-perceivers. We derive the probability distribution of children over these three perceptions conditional on the perception type of their parents. It turns out that these probabilities depend on the parents' type, the communication level, and the weight environment. This also applies to the marginal effects with respect to communication and the weight environment suggesting three-way-interactions in the econometric specification. Furthermore, we show that the optimal level of communication depends on the parents' type and on the weight environment. If, for instance, parents over-perceive the weight status of their child, then an increase of the weight environment reduces the probability that the child over-perceives its own weight. Parents then counter this worsening of the weight environment by increasing their efforts to transmit their weight perception. This optimal communication response to changes in the weight environment raises two issues. First, empirical models not controlling for communication (or, more generally, parental effort) may suffer from omitted variable bias.[6] Second, using perception specific communication as a regressor introduces an endogeneity bias. We solve this endogeneity problem by estimating reduced form models where general family communication serves as an instrument for weight specific communication.

Using a representative German data set and considering children aged between 11 and 17 years and their families we find overwhelming evidence for direct perception transmission from parents to children and that communication plays an important role therein. Though statistically significant, the results regarding indirect perception transmission are less clear cut. Notably, we find that direct perception transmission works largely independent of indirect perception transmission, that is, any influence of the weight environment can, in principle, be fostered or mitigated by parents and their communication efforts. More precisely, if parents correctly perceive the weight status of their child, then the probability that the child misperceives its own weight status is significantly lower when

[6]This is a potential shortcoming of the analysis offered by Ali, Amialchuk, and Renna (2011), Burke and Heiland (2007), Maximova et al. (2008), and Röhrig, Giel, and Schneider (2012).

family communication is intact as compared to when it is not. Girls tend to be more responsive to family communication. Independent of communication, however, direct perception transmission works better for boys. One may suspect that the influence of parents is particularly strong when children are young and that peers are more influential the older children are (see, *e.g.*, Steinberg and Monahan (2007)). Interestingly, both transmission channels, direct and indirect, are largely independent of the age of the child. If anything, direct transmission of weight over-perception is more likely in younger children than in older ones. Our analysis closes with a detailed sensitivity analysis that demonstrates the robustness of our results to competing econometric models, alternative codings of (transmission) variables, and model specifications.

Our evidence regarding direct and indirect perception transmission identifies two important factors in the formation of weight status (mis)perceptions in children and adolescents and thereby provides health policy makers with important information for the design of public policy measures. We found that children of correctly perceiving parents are less likely to misperceive their weight status than children of misperceiving parents. There are two immediate implications for health policy. First, parents should be informed of what constitutes a healthy weight for children so that they are able to correctly assess the weight status of their child. Second, children of misperceiving parents should also be provided with accurate information regarding their weight status to avoid that any misperception originating in the false weight assessment of their parents translates into unhealthy weight. As weight status misperceptions are usually not observed, the targeting of these measures is a major challenge. This is why parents and children should generally be provided with objective information regarding the actual weight status of the child by, *e.g.*, school teachers or doctors, and, if necessary, be informed about weight management strategies. This could be done during the already existing screening exams for children and adolescents. Participation in these exams, however, is voluntary and participation rates strongly decrease with age.[7] One potential explanation is that some of these exams are not included in public health insurance plans. Mandating participation and public financing might be a sensible option for public policy.[8]

Our paper relates to the health economics literature on network effects in the

[7]In our sample, the participation rate in the J1 exam (for children between 12 and 14 years) is 34 percent.

[8]We provide more details regarding the screening examinations further on below.

realm of body weight and body weight perceptions. As already mentioned above, the existing empirical literature that aims at explaining weight perception formation in children and adolescents concentrated on the indirect transmission channel, that is, on the influence of the weight environment on a child's weight perception (see Ali, Amialchuk, and Renna (2011), Maximova et al. (2008), and Röhrig, Giel, and Schneider (2012)). These studies neglect direct perception transmission and are, thus, incomplete. Based on our results, the more important channel is ignored.[9] We also found that the two channels are closely connected so that the results reported in the aforementioned papers may well be biased. Christakis and Fowler (2007) and Cohen-Cole and Fletcher (2008) asked whether obesity is contagious. While the former paper finds that obesity spreads through social networks, the latter paper challenges this result. Both papers argue that a heavier weight environment may increase the tolerance towards over-weight or obesity which, in turn, lowers the 'price' of gaining weight (see also Burke and Heiland (2007) and Burke, Heiland, and Nadler (2010)).[10] Our approach is more indirect as we do not directly link the weight environment to individual weight status but to weight perceptions that may then, down the road, affect individual weight. We thereby highlight the mechanism through which obesity may spread. Both papers also neglect the potential influence of parents on a child's weight status. By contrast, we show that weight perception transmission from parents to children, *i.e.* direct transmission, is more important than indirect transmission. It is, thus, less the environment that is contagious it is the family.

More generally, our paper relates to the literature on the transmission of traits in social networks and, most importantly, the papers by Bisin and Verdier (2000 and 2001). Our theoretical model is based on their 2001 article. The most important difference between their theory and ours is that we extend the model from two to three types. Both papers lack a rigorous empirical application. By contrast, Dohmen et al. (2011) provide an empirical analysis of the intergenerational transmission of risk and trust attitudes and find strong evidence for successful transmission. Parental effort, however, is not modeled. To the best of our knowledge, Patacchini and Zenou (2016) is the only empirical paper that integrates parental effort. They argue that participation in religious service or religious

[9]Röhrig, Giel, and Schneider (2012) also use the KiGGS data and regress weight misperception of children on a set of household characteristics, including the body mass index of parents but neither misperception of parents nor parental effort. Al Sabbah et al. (2009) found evidence that family communication is related to weight dissatisfaction.

[10]Closely related to this literature are Chang and Christakis (2003) 2001, and Johnson et al. (2008) who analyze whether a shift in social weight norms affect population weight.

events is a suitable measure for parental effort and find that, indeed, effort plays an important role in the intergenerational transmission of religious traits. They do not address the potential endogeneity of effort, though. We estimate reduced form models where family communication serves as an instrument for weight specific family communication so that our analysis is not subject to endogeneity bias.

The remainder of the paper is organized as follows. The economic framework is laid out in Section 3.2. In Section 3.3 we provide information on our data source and conduct a descriptive analysis. The empirical strategy is discussed in Section 3.4. Our main regression results are presented in Section 3.5 followed by a robustness analysis in Section 3.6. Section 3.7 offers some concluding remarks.

3.2 The economic framework

Evidence suggests that individual weight perception is an important factor in healthy weight management (see, *e.g.*, Kuchler and Variyam (2003)). The idea is that an individual who feels too heavy will take measures to reduce weight. Similarly, thinking of oneself as being too thin sets incentives to gain weight. We consider normal weight the healthy or socially optimal weight status (in terms of current and, in particular, future health outcomes). We argue that individual weight perceptions are rooted in weight preferences. In the event of normal weight being the weight preference of an individual, weight perception will be correct no matter what the actual weight status is. Overweight, for instance, will always be perceived as being too heavy so that measures are taken to lose weight. Similarly, correct perception of under-weight as being too thin leads to a weight gain. Thus, correct weight perception implies that individual weight converges, however slowly, to the healthy range. Things are markedly different when the individual weight preference departs from normal weight. Suppose the preferred weight of an individual is underweight. Then weight status will be over-perceived along the whole weight distribution and weight will converge to the underweight range (which the individual considers optimal). By contrast, if the preferred weight is overweight, then weight status will generally be under-perceived and, in equilibrium, the individual will be too heavy. The key assumption is that individuals are not aware of their potentially distorted weight preference, that is, individuals believe that their weight preference constitutes the healthy weight. To summarize, for any given weight status an individual may correctly perceive its own weight status (C), under-perceive (U) or over-perceive (O) it. While correct perceptions

are just fine, weight misperceptions are problematic as equilibrium weight status will not be healthy, *i.e.* will depart from normal weight. A better understanding of the evolvement of individual weight – and with it the weight distribution of the population – requires to take due account of the influence of weight perceptions on actual weight. As weight perceptions, *i.e.* weight preferences, are formed early in life, we focus on the formation of such preferences in children and adolescents highlighting the role of the environment a child lives in.

Taking ethnic and religious traits as an example, Bisin and Verdier (2001) suggest that individual preferences can be transmitted from one individual to another. To be more precise, there may be intergenerational transmission of traits from parents to their children (vertical or *direct* transmission). In addition, the cultural/social environment of the child may play a role (horizontal or *indirect* transmission). To tilt the balance between direct and indirect transmission parents can exert a (costly) effort. In our context, the 'trait' is a weight preference or weight norm. Assuming that parents are altruistic towards their children and given that parents are unaware of any distorted weight preference they might have, the best parents can do is to (try to) transmit their weight preference to their offspring and with it their weight perception. According to our categorization from above, there are three parent types $i = U, C, O$ and we denote by π_i the probability of successful transmission of weight perceptions from parents to children. Like in Bisin and Verdier (2001), parents can exert an effort $e \geq 0$ to increase the probability of successful direct transmission, that is, $\pi_i'(e) > 0$ and $\pi_i''(e) < 0$. In the following we call this effort *communication*.

Several studies have shown that the (social) weight environment of a child also matters for perception formation (*e.g.*, Ali, Amialchuk, and Renna (2011) and Maximova et al. (2008)). This environment is made up of a child's parents, other adults, and children of similar age and same sex (*i.e.*, their peers). The weight status of these groups may become a benchmark against which a child evaluates its own weight. This may lead to distorted perceptions on the side of children whenever the weight environment departs from normal weight. Let Δ be a measure of the weight environment where large values are associated with a heavy environment and small values with a light environment.[11] Independent of direct perception transmission, a child under-perceives its own weight with probability q_U. We let this probability depend on the weight environment and conjecture

[11] To fully capture indirect transmission, Δ necessarily is a vector including peer, parental and adult weight. In the theoretical model we do not distinguish between the different dimensions but keep this for the empirical part.

that $q'_U(\Delta) > 0$. The probability that, for instance, a normally weighted child will perceive its weight status as 'too thin' is higher the heavier its weight environment. The probability of over-perception is denoted q_O and we let $q'_O(\Delta) < 0$. The probability of correct weight perception of the child is then given by the residual probability $q_C(\Delta) = 1 - q_U(\Delta) - q_O(\Delta)$, where $q'_C(\Delta) = -[q'_U(\Delta) + q'_O(\Delta)]$ cannot be signed without further assumptions.

Combining direct and indirect perception transmission we can calculate the transition probabilities, that is, the conditional probabilities of type-i parents having type-j children, $i, j \in \{U, C, O\}$. We get

$$p_{j|i}(e;\Delta) = \begin{cases} \pi_i(e) + (1 - \pi_i(e))q_i(\Delta) & \text{for } i = j, \\ (1 - \pi_i(e))q_j(\Delta) & \text{for } i \neq j. \end{cases} \tag{3.1}$$

Consider the upper branch of equation (3.1) which displays the probability of a type-i parent having a child of the same kind. Parents and children share the same weight perception if direct *or* indirect transmission is successful. The former obtains with probability π_i and the latter with probability q_i. As we considered the transmission channels to be stochastically independent we get $p_{ii} = \pi_i + q_i - \pi_i q_i$. For unsuccessful perception transmission, $i \neq j$ (the lower branch of equation (3.1)), direct transmission must not work. The probability of this event occurring is $1 - \pi_i(e)$. At the same time there needs to be successful indirect transmission of a weight perception other than i.

It is instructive to investigate the comparative static properties of transition probabilities. For a given weight environment we find that more communication increases the probability that the child shares the weight status perception with its parents:

$$\frac{\partial p_{j|i}(e;\Delta)}{\partial e} = \begin{cases} \pi'_i(e)(1 - q_i(\Delta)) > 0 & \text{for } i = j, \\ -\pi'_i(e)q_j(\Delta) < 0 & \text{for } i \neq j. \end{cases} \tag{3.2}$$

In the empirical part of the paper we estimate the conditional probability distributions of children over the three perceptions, namely, under-, correct-, and over-perception. The theoretical framework suggests that the weight environment affects weight perceptions rendering Δ an important regressor. Equation (3.2) shows that the impact of the weight environment goes beyond the direct influence on weight perceptions, Δ is a moderator: the marginal effect of communication effort e depends on both, i and Δ, so that the empirical model should include interaction terms between parents' perception type, communication effort, *and*

weight environment. The importance of three-way interactions also follows from the comparative static effects of the weight environment:

$$\frac{\partial p_{j|i}(e;\Delta)}{\partial \Delta} = (1-\pi_i(e))q_j'(\Delta). \tag{3.3}$$

We find that, for a given effort level, a marginal increase in the weight environment affects the transition probabilities where size and sign of the effect depend on communication effort e and weight environment Δ. As the sign of the first factor of equation (3.3) is always positive, the sign of the partial derivative is determined by the second factor. Independent of the parents' perception type i the directional effects of an increase in Δ on the respective (conditional) perception probabilities of children directly follow from our assumptions on q_U and q_O. More specifically, the probability of child over(under)-perception falls (increases) with Δ. The effect on the probability of correct weight perception cannot be signed.

To determine the optimal communication effort of parents, we let their utility depend on both, the own perception of the child's weight and the child's perception. When parent and child have the same perception, the utility of the parent is u_0. If parental and child perception are off one category, then the utility is u_1. In case they are off two categories the utility is u_2. Reflecting our assumptions on parental altruism and the unawareness of potential weight misperceptions we let $u_0 > u_1 > u_2 > 0$.[12] This gives rise to the following expected utility $EU_i(e;\Delta)$ of a type-i parent, $i = U, C, O$:

$$EU_U(e;\Delta) = p_{U|U}(e;\Delta)u_0 + p_{C|U}(e;\Delta)u_1 + p_{O|U}(e;\Delta)u_2 - H(e), \tag{3.4}$$

$$EU_C(e;\Delta) = p_{C|C}(e;\Delta)u_0 + \left[p_{U|C}(e;\Delta) + p_{O|C}(e;\Delta)\right]u_1 - H(e), \tag{3.5}$$

$$EU_O(e;\Delta) = p_{O|O}(e;\Delta)u_0 + p_{C|O}(e;\Delta)u_1 + p_{U|O}(e;\Delta)u_2 - H(e), \tag{3.6}$$

where, for $e > 0$, the costs of communication are measured by $H(e) > 0$ with $H'(e) > 0$ and $H''(e) > 0$. To guarantee interior solutions we further assume that $H(0) = H'(0) = 0$. Parents maximize their utility with respect to the level of communication with the child. Optimal communication, e_i^*, is characterized by

[12] To simplify the analysis we let the utility levels only depend on the 'distance' in weight perceptions across generations and not on their absolute values. Patacchini and Zenou (2016) also assume that parents enjoy a higher utility when the child is of the same kind as them, in their case religious activity.

the following first order conditions[13]

$$\pi'_U\left((1-q_U)[u_0-u_1]+q_O[u_1-u_2]\right)=H'\Rightarrow e^*_U, \tag{3.7}$$

$$\pi'_C(q_U+q_O)[u_0-u_1]=H'\Rightarrow e^*_C, \tag{3.8}$$

$$\pi'_O\left((1-q_O)[u_0-u_1]+q_U[u_1-u_2]\right)=H'\Rightarrow e^*_O, \tag{3.9}$$

for under-perceiving, correctly perceiving, and over-perceiving parents, respectively.[14] A comparative static analysis reveals that the different forms of transmission interact with one another. Total differentiation of the first order conditions from above with respect to e^*_i and Δ gives

$$\frac{\mathrm{d}e^*_U}{\mathrm{d}\Delta}=\frac{\pi'_U\left(q'_U[u_0-u_1]-q'_O[u_1-u_2]\right)}{SOC^e_U}<0, \tag{3.10}$$

$$\frac{\mathrm{d}e^*_C}{\mathrm{d}\Delta}=-\frac{\pi'_C(q'_U+q'_O)[u_0-u_1]}{SOC^e_C}\lesseqgtr 0, \tag{3.11}$$

$$\frac{\mathrm{d}e^*_O}{\mathrm{d}\Delta}=\frac{\pi'_O\left(q'_O[u_0-u_1]-q'_U[u_1-u_2]\right)}{SOC^e_O}>0. \tag{3.12}$$

A type-U parent responds to an increase in the child's weight environment by reducing costly communication with the child. When the weight environment is getting heavier, the probability that the child under-perceives its own weight status increases, $q'_U>0$, allowing the parent to reduce communication, that is, to lower the efforts to transmit under-perception. A lighter weight environment intensifies type-U parents' communication. The effect of an increase in weight environment on communication between type-C parents and their offspring is ambiguous. On the one hand, the probability of over-perception is reduced, $q'_O<0$, benefitting the parent. On the other hand, the probability of under-perception increases, $q'_U>0$. Which of the two effects dominates depends on how the weight environment elasticities compare to one another. Finally, type-O parents intensify communication when the weight environment is getting heavier. A heavier weight environment reduces the probability that the child over-perceives its weight, $q'_O<0$, triggering more communication efforts from the parent to counter the 'worsening' of the

[13]To simplify notation we drop the argument of the probability and cost functions.

[14]The second-order conditions are satisfied and given by

$$\begin{aligned}
SOC^e_U =&\pi''_U\left((1-q_U)[u_0-u_1]+q_O[u_1-u_2]\right)-H''<0,\\
SOC^e_C =&\pi''_C(q_U+q_O)[u_0-u_1]-H''<0,\\
SOC^e_O =&\pi''_O\left((1-q_O)[u_0-u_1]+q_U[u_1-u_2]\right)-H''<0.
\end{aligned}$$

weight environment. Our theoretical framework, thus, makes very clear that, first, the different channels of preference transmission, direct and indirect, are closely connected and, second, that the relationship between the two is influenced by the intensity of communication between parents and their offspring.

Finally, one might ask how a change in the weight environment affects the transition probabilities when factoring in the optimal communication response of parents to such changes as given in equations (3.10) through (3.12). We find that the directional effect of the weight environment is ambiguous:[15]

$$\frac{\mathrm{d}p_{j|i}}{\mathrm{d}\Delta} = \frac{\partial p_{j|i}}{\partial e}\frac{\mathrm{d}e_i^*}{\mathrm{d}\Delta} + \frac{\partial p_{j|i}}{\partial \Delta} \lessgtr 0. \tag{3.13}$$

In the empirical part of the paper, we investigate the impact of a change of a single variable on the transition probability for *given* values of the remaining variables, that is, we conduct a *ceteris paribus* analysis. This implies that equations (3.2) and (3.3) offer testable hypotheses regarding the directional effects of communication and weight environment on the transition probabilities, respectively. By contrast, equation (3.13) is not a *ceteris paribus* analysis as a change in the weight environment implies a change in the communication effort. This resembles a situation where transition probabilities are regressed on parental perception type and weight environment but not communication. Comparison of equations (3.2) and (3.13) reveals that a regression without communication effort as explanatory variable will suffer from omitted variable bias (the first term of equation (3.13)). In cases where the second term of equation (3.13) can unambiguously be signed, the first term works in the opposite direction rendering the overall effect ambiguous. As communication is chosen optimally given the parents' type and the child's weight environment a naïve regression with transmission specific communication as an additional explanatory variable will suffer from endogeneity bias. We discuss further on below how we address this endogeneity problem.

3.3 Data and descriptive statistics

After a general description of the data set, this section introduces the variables that are being used in the empirical analysis. Most importantly, we explain how we measure the objective weight status of children and how we construct the perception types of both, children and their parents. We explain how we approximate

[15]The explicit calculations for each probability can be found in Appendix A.

a child's weight environment and motivate a familial communication measure that serves as an instrument for communication effort.

3.3.1 Data source and sample selection

For our empirical analysis we use the base survey of 'The German Interview and Examination Survey for Children and Adolescents (KiGGS),' a cross-sectional data set collected by the Robert-Koch-Institute (RKI) between 2003 and 2006. The study aims at providing a representative picture of health and health care needs of all children and adolescents in Germany aged between 0 and 17 years (excluding those residing in institutions or hospitals). In a first step, 167 sample locations were randomly selected at the community level (primary sample units). Then age-specific random samples were drawn from population registries (secondary sample units).[16] Selected children and their parents or legal guardians were invited to participate in the KiGGS study (response rate: 66,6 %). Children had to undergo a medical exam, laboratory tests, and a physician guided medical computer-assisted personal interview.[17] Parents or legal guardians of all sampled families had to answer a questionnaire. In addition, children aged between 11 and 17 years had to fill out a child questionnaire. Our analysis draws on information from both questionnaires so that we only selected families with children aged 11 years and above into the analysis sample. Furthermore, we restrict the sample to children that live with at least one biological parent and where a biological parent answered the questionnaire. Finally, we drop all observations with missing values to arrive at an analysis sample size of 4,611 observations. Table 3.1 shows how selection criteria affect sample size.

3.3.2 Objective weight status measurement

Following Bisin and Verdier (2001), parents have an interest in transmitting their weight preference to their offspring. Our data contain no direct but indirect information regarding these preferences. Using an objective weight status measure and relating it to subjective weight perception measures allows us to define the

[16]For more details on the setting and the sampling procedure see Kurth (2007b).

[17]The RKI executed a second survey round (wave 1) between 2009 and 2012 via telephone interviews and recently made the data available as a public use file. The design of wave 1 departs in several ways from the base survey so that we are unable to use the more recent data. Most importantly, there is no medical exam in wave 1. Wave 2 interviews are currently being administered (2014-2017). The respective data will be made available as public use file four years after interviews have been finalized. For more information, please visit http://www.rki.de.

Table 3.1: Sample selection and size

Selection criterion	**Sample size**
None (full sample)	17,641
Children aged 11 to 17 years	6,813
Child lived with at least one biological parent	6,577
Questionnaire responder was biological parent	6,549
Observations without missing information	4,611

three perception types of the theoretical model, namely *U*-, *C*-, and *O*-types, for both, parents and children. In this subsection we discuss our objective weight status measure and then, in the next subsection, turn to weight perceptions and to the construction of perception types.

The Body-Mass-Index (BMI) is the perhaps most popular objective weight status measure. It relates an individual's weight to its height which appears plausible.[18] An important reason for its popularity is that the BMI is easy and inexpensive to measure. There are a number of shortcomings, though. For survey data it is typically argued that BMI values may be subject to reporting bias. Such a bias is ruled out in our analysis as weight and height measurement are part of the medical exam. Another common objection is that the BMI does not distinguish between fat and muscle mass. Alternative measures, *e.g.*, the waist-to-hip ratio or skin-fold thickness, are more suited in this respect. The problem with these alternatives is weight status categorization: To classify a child's weight status, threshold values are needed to separate the respective weight status categories. For the alternative measures such thresholds do not exist for children and adolescents.

By contrast, BMI thresholds are well established. Adults can be categorized as having strong underweight (BMI below 16), underweight (16-18.5), normal weight (18.5-25), overweight (25-30), and severe overweight or obesity (above 30). These thresholds are not suited to categorize children as they do not reflect the development stage of a child. To account for that, age- and gender-specific BMI thresholds need to be defined.[19] One possibility is to use the well-known Kromeyer-Hauschild (K-H) scheme for Germany (see Kromeyer-Hauschild (2005) and Kromeyer-Hauschild et al. (2001)). This scheme uses four percentiles of age- and gender-specific weight distributions, namely the 3rd, 10th, 90th, and 97th

[18]BMI = weight in kilograms divided by height in meters squared.

[19]No matter how these thresholds are constructed, they will always be arbitrary. See Cole et al. (2000) for an attempt to establish an international standard for the weight status of children.

percentile, to separate the five weight status categories from above. As the K-H BMI thresholds are calculated based on an outdated weight distribution, we recalculate them using the age- and gender-specific weight distributions of the KiGGS data. Table 3.15 and 3.16 in the Appendix contrast the K-H with the KiGGS thresholds. At the lower end of the weight distribution differences between thresholds are negligible. The difference, however, increases with the considered percentile. The obesity threshold for girls (the 97th percentile), for instance, is 3 to 4 BMI points larger when using the KiGGS weight distribution rather than the K-H weight distribution. For boys the respective difference is between 2 and 3 BMI points.[20] The empirical analysis uses the KiGGS thresholds. Results do not change qualitatively when considering the K-H thresholds (see Section 3.6.2).

In order to facilitate comparison across age and gender BMI values need to be standardized. As the BMI distribution is non-normal and skewed a more sophisticated normalization method than z-standardization is usually being applied, the LMS-method by Cole (1990). Denoting by r_j the age- and gender-specific reference group of individual j, the BMI in standard deviation scores is calculated as

$$SDS_j = \frac{[BMI_j/M(r_j)]^{L(r_j)} - 1}{L(r_j)S(r_j)}, \tag{3.14}$$

where $M(r_j)$ is the median BMI of the reference group, $L(r_j)$ the Box-Cox power transformation and $S(r_j)$ the coefficient of variation. For the age- and gender-specific values of L and S see Table 3.15 and 3.16 in the Appendix.

3.3.3 Weight perception

The construction of perception types according to the theory requires one objective and two subjective weight status measures (one for parents and one for children.) Despite its disadvantages, the BMI is the best available measure for objective weight status categorization. We contrast this categorization with weight perceptions regarding the child's weight status. These perceptions are elicited separately for parents and children (via the respective questionnaires) and are measured using the following five categories: far too thin, too thin, about right, too heavy, far too heavy.

[20]These observations are well in line with the obesity 'pandemic' (see Neuhauser et al. (2013, 32f)). (Burke and Heiland (2007)) found a similar pattern for the United States and argue that the shift in the weight distribution is self-enforcing as the increase in average weight increases the tolerance towards over-weight.

There are five objective weight status categories (OWS), ordered from strong underweight (=1) to obesity (=5), and five subjective weight status categories (SWS_k), ordered from much too thin (=1) to much too heavy (=5) with $k = C$ for children and $k = P$ for parents. We say that there is an *under*-perception of weight status if the perceived weight status falls into a lower category than the actual weight status. This results in two under-perception indicators defined as $UPk = \mathbf{1}_{\{OWS>SWS_k\}}$. Similarly, we define two *over*-perception indicators: $OPk = \mathbf{1}_{\{OWS<SWS_k\}}$.[21] The definition of misperception variables $MPk = OPk - UPk \in \{-1, 0, 1\}$ allows us to incorporate their natural ordering into our analysis.[22] For completeness, we also define indicators for correct weight perception: $CPk = 1 - UPk - OPk$.

Table 3.2 provides an overview regarding the accuracy of weight perceptions for both, children and parents. Across all weight categories only about 48 percent of children correctly perceive their own weight status. With 41 percent misperceptions disproportionately fall on over-perceptions. The remaining 11 percent under-perceive their weight status. The table reveals that the perception distribution strongly depends on weight status. The share of correctly perceiving children increases from 21 percent in the lowest weight status category to 67 percent in the second highest category. 58 percent of obese children correctly rate their own weight, *i.e.* as far too heavy. Notably, as strong underweight is the lowest weight status category and obesity the highest, the former cannot under-estimate their weight status while the latter cannot over-estimate it. The share of over-perceiving children monotonically decreases from 79 percent in the lowest weight status category to 28 percent in the highest relevant category. Weight status under-perception is most pronounced for obese children. In the remaining weight status categories the share of under-perceiving children is less prevalent (between 4 and 11 percent). Although the numbers differ, the same pattern emerges for weight perceptions of parents regarding the weight status of their child. With an overall share of 67 percent correct weight perceptions, however, the weight assessment of parents is considerably more accurate than the self-assessment of children. We find that girls have a considerably higher tendency to over-perceive

[21] In this definition we follow Röhrig, Giel, and Schneider (2012). They do not consider parental misperception at all and, in their empirical analysis, concentrate on weight over-perception of children.

[22] An alternative way to model weight misperception is to compute the difference between standardized BMI values and standardized perception scores (see, *e.g.*, Ali, Amialchuk, and Renna (2011) and Maximova et al. (2008)). As our weight perception variable is measured using an ordinal scale rather than a metric one this approach is not applicable in our setting.

their weight status (51 percent) than boys (32 percent). While 53 percent of boys correctly perceive their own weight only 43 percent of girls do so. These gender differences are much smaller in parental perceptions: for daughters (sons) weight perception is correct in 69 (65) percent of cases.

To shed some light on the role of direct perception transmission from parents to children we investigate the joint probability distribution of weight perceptions and, more importantly, the distribution of child perception conditional on parental perception. The respective frequencies are shown in Table 3.3, with the numbers in square brackets being the conditional frequencies. First, we note that weight perceptions of parents and their children are rarely off two categories. Second, weight perceptions coincide in 61 % of the cases (59 % for girls and 62 % for boys). Third, looking at the conditional probabilities, perception transmission works particularly well in the event of over-perception. Considering all children, 81 % of children over-perceive their weight status conditional on having over-perceiving parents. With 58 % and 39 % for correct perceptions and under-perceptions, respectively, transmission is less likely. Fourth, there are some interesting differences across gender. While the transmission of over-perception works better for girls than for boys it is the other way round for correct perception and under-perception. This calls for a gender-stratified econometric analysis.

Finally, note that a naïve regression of weight perceptions of children on weight perceptions of parents without controlling for the weight status of children would find a (strong) positive correlation by construction (see Table 3.2). To avoid this, we include 4 body measures in weight category splines, most importantly the BMI. As empirical evidence suggests that body measures which consider body fat or body fat distributions affect weight perceptions independently of BMI (see Burke, Heiland, and Nadler (2010)), we also control for the waist-to-hip ratio (WHR), the waist-to-height ratio (WHtR), and the sum of triceps and back skin fold thickness (SFT). All of these measures were part of the medical exam and are, as usual, measured in standard deviation scores.

3.3.4 Weight environment

An important part of the weight environment of a child is the weight status of its peers. We measure peer weight by calculating sample point (*i.e.* cluster) SDS means over all children aged 11 to 17 years. For each of the 167 clusters we get one

Table 3.2: Perception distribution by *weight classification* and *gender*

		Weight classification					
Perception children		*Strong underweight*	*Under-weight*	*Normal-weight*	*Over-weight*	*Obese*	Total (%)
	U	-	0.08	0.11	0.04	0.42	484 (0.11)
	C	0.21	0.43	0.48	0.67	0.58	2,234 (0.48)
All	*O*	0.79	0.49	0.41	0.28	-	1,893 (0.41)
	Total	145	327	3,689	324	126	4,611
	(%)	(0.03)	(0.07)	(0.80)	(0.07)	(0.03)	(1.00)
	U	-	0.05	0.05	0.03	0.28	128 (0.06)
	C	0.20	0.34	0.43	0.61	0.72	978 (0.43)
Girls	*O*	0.80	0.61	0.52	0.37	-	1,149 (0.51)
	Total	66	149	1,830	150	60	2,255
	(%)	(0.03)	(0.07)	(0.81)	(0.07)	(0.03)	(1.00)
	U	-	0.10	0.16	0.06	0.55	356 (0.15)
	C	0.22	0.51	0.53	0.73	0.45	1,256 (0.53)
Boys	*O*	0.78	0.39	0.31	0.21	-	744 (0.32)
	Total	79	178	1,859	174	66	2,356
	(%)	(0.03)	(0.08)	(0.79)	(0.07)	(0.03)	(1.00)
		Weight classification					
Perception parents		*Strong underweight*	*Under-weight*	*Normal-weight*	*Over-weight*	*Obese*	Total (%)
	U	-	0.05	0.13	0.06	0.33	548 (0.12)
	C	0.23	0.50	0.70	0.73	0.67	3,089 (0.67)
All	*O*	0.77	0.45	0.18	0.21	-	974 (0.21)
	Total	145	327	3,689	324	126	4,611
	(%)	(0.03)	(0.07)	(0.80)	(0.07)	(0.03)	(1.00)
	U	-	0.02	0.08	0.03	0.33	180 (0.08)
	C	0.20	0.45	0.72	0.77	0.67	1,553 (0.69)
Girls	*O*	0.80	0.53	0.20	0.20	-	522 (0.23)
	Total	66	149	1,830	150	60	2,255
	(%)	(0.03)	(0.07)	(0.81)	(0.07)	(0.03)	(1.00)
	U	-	0.07	0.17	0.08	0.33	368 (0.16)
	C	0.27	0.54	0.67	0.70	0.67	1,536 (0.65)
Boys	*O*	0.73	0.39	0.15	0.22	-	452 (0.19)
	Total	79	178	1,859	174	66	2,356
	(%)	(0.03)	(0.08)	(0.79)	(0.07)	(0.03)	(1.00)

Note: U = Under-, C = Correct and O = Over-perception.

Table 3.3: Children's and parents' perception by *gender*

		p_{Ui}	$[p_{U\|i}]$	p_{Ci}	$[p_{C\|i}]$	p_{Oi}	$[p_{O\|i}]$		
All children									
	U	0.05	**[0.39]**	0.06	[0.49]	0.01	[0.12]	0.12	[1]
Parents (i)	C	0.06	[0.08]	0.39	**[0.58]**	0.23	[0.34]	0.67	[1]
	O	0.00	[0.01]	0.04	[0.18]	0.17	**[0.81]**	0.21	[1]
		0.11		0.48		0.41		1	
Girls									
	U	0.03	**[0.32]**	0.04	[0.49]	0.01	[0.18]	0.08	[1]
Parents (i)	C	0.03	[0.04]	0.36	**[0.53]**	0.30	[0.43]	0.69	[1]
	O	0.00	[0.01]	0.03	[0.14]	0.20	**[0.85]**	0.23	[1]
		0.06		0.43		0.51		1	
Boys									
	U	0.07	**[0.43]**	0.08	[0.49]	0.01	[0.09]	0.15	[1]
Parents (i)	C	0.08	[0.13]	0.41	**[0.63]**	0.16	[0.24]	0.66	[1]
	O	0.00	[0.02]	0.04	[0.23]	0.14	**[0.75]**	0.19	[1]
		0.15		0.53		0.32		1	

Notes: U = Under-, C = Correct and O = Over-perception. p_{ji} and $p_{j|i}$ with child j and parent i,$j, i = U, C, O$.

peer SDS value:[23] positive values identify a cluster with an average peer SDS above the gender- and age-specific mean of all sample children. Negative values obtain for clusters with an average peer SDS below the mean.[24] Additional variables capturing the weight environment of the child are the BMIs of its mother and father and the average BMI of all mothers as well as the average BMI of all fathers in the respective cluster. We use gender-, but no age-specific standardization for parental BMI values. Rather plausibly, this implies that, for instance, the impact of overweight of a parent on the propensity of perception transmission is independent of the age of the parent. Computing gender-specific cluster means

[23]This is only a proxy for a child's peer group SDS as the variable considers all sample point children rather than children the child interacts with.

[24]Note that BMI values of children – when measured in standard deviation scores – have mean zero and standard deviation one. The standard deviation between cluster SDS values are necessarily smaller than one as deviations of 167 means from the overall mean are considered instead of deviations of all SDS values from the overall mean (see Table 3.13).

we get a proxy for adult BMI environment in each cluster.[25]

3.3.5 Communication

The theory presented in the previous section highlighted the importance of communication in the process of perception transmission. As part of the KiGGS questionnaire children aged between 11 and 17 years and all parents (or other caring individuals) were separately asked whether the statement "In our family, everyone has the feeling that you listen and be responsive to each other" is either not true, rarely true, rather true, or exactly true. Based on the responses we construct two indicator variables, one communication indicator for the child (CC) and one for the parents (CP). These variables assume the value one if the respective response to the above question was rather true or exactly true. Communication within the family can hardly work if sender or receiver answer the above question with rarely true or worse. This is why we consider the indicator $C = CC * CP$ a meaningful measure of communication intensity (and quality) within the family. One potential objection against this measure is that it draws on information provided by children and parents whereas in our theory communication is a choice variable of parents. We argue that parents are responsible for family communication and that their communication effort is reflected in their own response to the above statement *and* in the response of their children.

In Table 3.4 below we provide more information regarding our communication variable and its underlying variables. The correlation between the communication responses between parents and their children using the 4-point scale from the questionnaire is 0.25. In 47 % of families, parents and children identically rate the quality of communication within the family. In 30 (22) % of families parents rate communication better (worse) than children.[26] The absolute frequencies set in bold face are the cases in which our communication indicator C assumes the value 1 (76 %). Using the binary measure the correlation between communication variables between parents and children drops to 0.16.[27]

It should be noted that the variable C is a rather general measure of communication. Our data do not contain any information regarding weight specific

[25] An alternative to capturing the weight environment of peers and adults by cluster specific means is to use cluster fixed effects (see Section 3.5.1).

[26] Due to rounding percentages do not sum up to 100.

[27] One could certainly think of alternative ways to model family communication. In an extension section we discuss how these alternative measures affect our results (see Section 3.6.1).

Table 3.4: Family communication

	Children				
Parents	*not true*	*rarely true*	*rather true*	*exactly true*	Total
not true	6	16	25	13	60
rarely true	31	99	121	55	306
rather true	102	472	**1,276**	**797**	2,647
exactly true	26	149	**625**	**798**	1,598
Total	165	736	2,047	1,663	4,611

communication within the family, that is, we do not have a precise measure of the communication effort e. We conjecture, however, that C and e are sufficiently correlated to render C a suitable proxy for e. In fact, a regression of children's weight perceptions on parental perceptions and e would suffer from reverse causality. Provided that general family communication C is exogenous – which we believe is the case – using C as an instrument for e would allow us to arrive at consistent estimates. Instrumental variables estimation is not feasible as e is unobserved. In this sense, a regression using C as a proxy for e yields a reduced form estimate of the effects of weight specific communication (*i.e.* effort).

To ease the interpretation of regression results we use our communication indicator to create 6 types of parents: There are three different perception types (that is, UPP, CPP, and OPP) and the communication indicator can be switched on or off. Table 3.12 provides an overview of the definition of transmission variables and Table 3.13 shows the corresponding summary statistics. Both tables are relegated to the Appendix.

3.3.6 Control variables

The KiGGS data set offers a rich selection of individual and household characteristics that allow us to control for individual heterogeneity. Most importantly, we have information on socio-demographic factors like age (6 dummies), sex (dummy), education (school type, 7 dummies), migration background (dummy), puberty (Tanner stages 1-6, 2 dummies), parents' socio-economic status (SES, 2 dummies), household size, residential status (both parents or one parent, 3 dummies), region (East/West, dummy; rural/small town/middle town/urban, 3 dummies) and questionnaire respondent (3 dummies). Summary statistics of these variables are provided in Table 3.14 in the Appendix. While most of these vari-

ables are fairly self-explanatory, our education measures deserve some discussion. Primary school (Grundschule) ranges from grade 1 to 4 in most regional states. There are a few states with 6 years of primary school education.[28] German states used to have a three-tired school system for secondary school with Hauptschule being the lowest tier, Realschule the middle tier, and Gymnasium the most advanced tier.[29] While the former two end after completion of grade 9 or 10, the latter leads to an university-entrance diploma after grade 12 or 13. While these different tiers are typically operated by different schools, Gesamtschulen are a combination of all three, where, depending on performance, pupils can advance to a higher tier or be relegated to a lower one. The Orientierungsstufe is essentially a Gesamtschule for 5th- and 6th-graders that exist in some regional states. Finally, Foerder- and Sonderschulen are schools for children with special needs.

3.4 Empirical strategy

We aim at explaining the role of parents in the formation of weight status perceptions in their children considering the (social) environment the children live in. Building on our theoretical model, we estimate the probability of a child being type-j conditional on having type-i parents, where $i, j \in \{U, C, O\}$. We concentrate on the conditional probabilities of over-perception and under-perception. The probability of correct perception is then residually determined. This gives rise to the following probability models

$$Pr(MPC = j) = F_j(MPP, C, \Delta, \text{interactions}, \text{controls}), \qquad j = U, O. \quad (3.15)$$

The explanatory variables of interest are the transmission variables, namely, parental weight perception MPP, family communication C, and the weight environment Δ. Our theory suggests that there are important interactions between transmission variables. The communication intensity likely interacts with both parental weight status (mis)perception and the weight environment. For instance, in the event of parental over-perception we expect to find a significantly positive effect of communication on the probability that the child over-estimates its own weight. By contrast, in the event of correct parental perception or under-perception we expect to find a negative effect on the probability the child over-estimates its own

[28] Our sample children are aged between 11 and 17 years so that most of them grew out of primary school already.

[29] Recently, some states implemented a two-tier system.

weight. For the child's under-perception probability we expect the exact opposite. Communication is also expected to interact with the weight environment. As a response to the child's weight environment parents may adjust communication intensity so as to counter or foster unfavorable or favorable transmission of weight perceptions, respectively. These effects are picked up by the interactions of communication intensity, parents' perception, and weight environment.

To estimate the empirical model we consider two separate linear probability models, an under-perception model ($j = U$) and an over-perception model ($j = O$). The main advantage of the linear probability model is the straightforward interpretation of coefficients including the ones of interaction terms.[30] To assess the robustness of our results we contrast the outcomes of the linear probability models with the ones of non-linear models: the probit model and the generalized ordered probit model. We have to keep in mind, though, that the magnitude of interaction effects in non-linear models cannot be inferred from the coefficient estimates of interaction terms alone but crucially depend on the size of the index function (see, *e.g.*, Ai and Norton (2003) and Greene (2010)). In other words, even without explicit interaction terms non-linear models include interactions by construction.

The estimation of an ordered model is of particular interest as it allows us to use the information included in the natural ordering of the dependent variable MPC. This might improve the precision of our estimates. The downside is that, to avoid censoring, we have to exclude the bottom three percent *and* the top three per cent of the weight distribution.[31] Note that a simple ordered probit model is too restrictive for the problem at hand. The reason is what is typically called the "parallel lines assumption" (see, *e.g.*, Peterson and Harrell Jr. (1990) and Williams (2006)). It refers to the fact that the impact of an explanatory variable on the latent variable is the same for all categories of the dependent variable. This assumption will certainly be violated in our setting.[32] Suppose a parent correctly perceives the weight status of its child. Then our theory suggests that this perception is transmitted to the child with a higher probability the more intense communication within the family (see equation (3.2)). More precisely,

[30] As already mentioned above, the weight status of children with strong underweight cannot be under-estimated and the one of obese children cannot be over-estimated. To avoid censoring, we exclude the former from the under-perception model and the latter from the over-perception model. In both cases we lose about three per cent of observations.

[31] Our results are robust to ignoring censoring. Results available upon request.

[32] We test this assumption using Wolfe and Gould's STATA `omodel` command (see Long and Freese (2006, p. 199)) and reject the parallel lines assumption at the one per cent level.

the probability of both, an under-perception and an over-perception of the child's own weight status is expected to drop. But this is ruled out when fitting a simple ordered probit model. Let more communication have a negative effect on the child's latent over-perception. Then the probability that the child over-estimates its own weight status drops, while the probability of under-estimating its own weight status increases. Nevertheless, the probability of the child correctly perceiving its own weight might increase, albeit for the wrong reasons. This is why we estimate a generalized ordered probit model that estimates two coefficient vectors, one for the over-perception category and one for the under-perception category. This lowers the degrees of freedom and with it the precision of coefficient estimates.[33] We may be able to improve on efficiency by re-introducing the parallel lines assumption for the control variables.[34] Given our theoretical model and the arguments from above we refrain from reintroducing the parallel lines assumption for the transmission variables.

One may argue that our regression suffers from reverse causality. A child's perception of its weight status might have an impact on actual weight status. Indeed, as is often suggested, an actual change in weight status requires the understanding that the current weight status is sub-optimal. This reasoning, however, points to a time lag: current self-perception might impact *future* weight status. As our variables are measured at the same point in time simultaneity bias is unlikely. As we analyze behavior in social groups one may wonder whether Manski's (1993) reflection problem challenges our approach. The reflection problem refers to the difficulty to identify the impact of the behavior of a group on the behavior of a member of the group. Is it the behavior of a member that shapes the behavior of the group or is it the other way round? We are confident that the reflection problem is not of concern in our setting. First, consider direct perception transmission. The reflection problem would require that weight perception of parents affect the weight perception of the child and vice versa. It is hard to imagine a family where an 11 to 17 years old child socializes parents. Second, indirect perception transmission would only be problematic in Manski's sense if we considered weight perceptions of peers as explanatory variables. As we model the indirect transmission channel using the weight environment rather than weight

[33]Note that, except for the thresholds, a set of two binary regressions estimates the same number of parameters as a generalized ordered probit model where all variables are freed from the parallel lines assumption.

[34]For implementation in Stata see Williams (2006). Brant tests are conducted to test for the equality of coefficients across outcomes (see Long and Freese (2006)).

perceptions identification is not an issue.[35]

Finally, as observations from one and the same cluster may be correlated, we use clustered standard errors at the level of primary sample units (167 clusters) in all models. The correlation may be rooted in cluster-specific unobserved heterogeneity or may stem from using explanatory variables that are aggregated at the level of clusters, *i.e.*, the weight environment of peers and adults.

3.5 Results

3.5.1 Model specification

The regression results of the linear probability models (LPM) for weight over-perception and weight under-perception are shown in Tables 3.5 and 3.6, respectively, and we discuss them in turn. In this section we focus on model specification and, for the most part, keep the interpretation of coefficients for the next subsection.

In our first model (M1, Table 3.5) we investigate the role of *direct perception transmission* for the probability that a child over-perceives its own weight. Using correctly perceiving parents with disturbed family communication as reference category, we include the remaining five parental perception-communication types. All types turn out highly significant (p-value for joint significance = 0.0000). It should be noted that model M1 already includes all control variables except for the body measures of children. We argued above that the weight perception of parents is correlated by construction with the weight perception of their children when not controlling for the weight category of children (see Section 3.3.3 and Table 3.2). Thus, the coefficient estimates of the direct transmission variables are biased away from zero in model M1. Accordingly, model M2 controls for the weight category of children. To see that weight categories alone are insufficient to control for the role of body measures consider two children, one with a BMI at the upper end of a weight category and one with a BMI at the lower end of a weight category. Then we would expect that parents and children over(under)-perceive the weight status of the child with a higher (lower) probability in former than in the latter case. To capture this, we interact the BMI and all additional body measures with weight

[35]Recall that our 'peer' groups comprise children and adults a child does not interact with so that we do not see a link from the weight perception of a child to the average weight status in the cluster.

categories, that is, we add the body measures in weight category splines.[36] Although the direct transmission effects are, as expected, much smaller in absolute size, they are still highly statistically significant (p-value for joint significance = 0.0000). Note that direct transmission works particularly well for over-perceiving parents. As compared to correctly perceiving parents with disturbed family communication the positive impact of over-perceiving parents amounts to a 15 percentage point increase in the over-perception probability of children (0.149), whereas we find a much smaller absolute drop for under-perceiving parents (−0.075). With a 28 percentage points increase (0.193 − (−0.090)) and a two percentage points drop (−0.111 − (−0.090)) the results for over- and under-perceiving parents, respectively, are even more pronounced when family communication is intact.

In the next step, we add 5 weight environment measures including all interactions with parental perception and family communication (30 variables in total). With a p-value of .0554 the weight environment is only marginally significant, that is, *indirect perception transmission* seems to play a minor role. By contrast, direct perception transmission is still active and we see hardly any change in results between models M2 and M3, that is, sign and size of the direct transmission effects do not depend on wether or not we control for the weight environment and all its interactions. A likely reason for the minor impact of the weight environment is its (in part) imprecise measurement. Remember, we approximated the weight of peers using all children in the same cluster. Children within a cluster are very unlikely to know each other so that it appears perhaps a bit farfetched to expect peers effects. The same holds true for the weight environment of adults (males and females). As these variables are identical for all children of a cluster, we can alternatively capture these variables considering cluster fixed effects. The according model M4 shows no significant differences in coefficient estimates as compared to model M3. We prefer model M3 for two reasons. First, efficiency. Due to the higher degrees of freedom in model M3 standard errors are smaller than in model M4. Second, our theoretical framework. The theory suggested that the weight environment matters and that interactions with parental perception and family communication are likely to be important factors as well. In contrast to the cluster-level weight environment, the weight environment within the family is unproblematic. Our theory claimed that an increase in the body weight of parents leads to a drop in the probability of over-perception on the side of children

[36]Note that this specification also captures instances where an increase (decrease) in the BMI moves a child up (down) to the next weight category.

Table 3.5: Over-perception model

		$M1$	$M2$	$M3$	$M4$
Over-perception model $\mathbf{Pr}(OPC = 1)$	CPP (C=0)	*reference group*			
	CPP (C=1)	-0.091*** (0.021)	-0.090*** (0.020)	-0.089*** (0.021)	-0.094*** (0.021)
	UPP (C=0)	-0.224*** (0.036)	-0.075** (0.034)	-0.068* (0.034)	-0.072** (0.035)
	UPP (C=1)	-0.268*** (0.027)	-0.111*** (0.030)	-0.116*** (0.031)	-0.116*** (0.032)
	OPP (C=0)	0.346*** (0.035)	0.149*** (0.035)	0.153*** (0.035)	0.139*** (0.036)
	OPP (C=1)	0.410*** (0.025)	0.193*** (0.026)	0.189*** (0.026)	0.186*** (0.026)
	CPP (C=0) x BMI_M			0.020 (0.016)	0.017 (0.017)
	CPP (C=1) x BMI_M			-0.031*** (0.010)	-0.031*** (0.010)
	UPP (C=0) x BMI_M			0.020 (0.029)	0.000 (0.032)
	UPP (C=1) x BMI_M			0.001 (0.016)	0.000 (0.016)
	OPP (C=0) x BMI_M			-0.033 (0.028)	-0.019 (0.030)
	OPP (C=1) x BMI_M			-0.008 (0.013)	-0.007 (0.013)
	CPP (C=0) x BMI_F			0.037** (0.017)	0.034** (0.017)
	CPP (C=1) x BMI_F			0.003 (0.010)	0.006 (0.010)
	UPP (C=0) x BMI_F			-0.027 (0.025)	-0.004 (0.026)
	UPP (C=1) x BMI_F			-0.005 (0.016)	-0.003 (0.017)
	OPP (C=0) x BMI_F			0.029 (0.027)	0.013 (0.027)
	OPP (C=1) x BMI_F			-0.025 (0.016)	-0.032* (0.017)
	Intercept	0.244*** (0.039)	0.301*** (0.039)	0.305*** (0.038)	0.255*** (0.040)
	$N(df)$	4,485 (4,454)	4,485 (4,435)	4,485 (4,405)	4,485 (4,261)
	R^2	0.226	0.314	0.315	0.316
Controls		√	√	√	√
Child's BM (in splines)			√	√	√
Weight environment Δ				√	√
Cluster-fixed effects					√
Specification-tests		*Parental types*	*Child's BM*	Δ	
	$F(n, m)$	F(5, 166)	F(19, 166)	F(30, 166)	
	$(Prob > chi2)$	268.14 (0.0000)	30.91 (0.0000)	1.51 (0.0554)	

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

(see equation (3.3)). In this respect our results offer a mixed picture. Only 6 out of 12 coefficients show the expected sign and only one of those reaches statistical significance. Overall 2 out of 12 coefficients are statistically different from zero implying that one of the two points in the 'wrong' direction. We conclude that there is overwhelming evidence for the direct transmission channel to be active and that the results regarding indirect transmission are, despite their joint statistical significance, somewhat inconclusive. This is why we concentrate on the direct channel in the analysis to come controlling for the impact of the weight environment.

The results of the under-perception model are shown in Table 3.6 below. The general picture is very similar to the one of the over-perception model. The direct transmission channel is highly active (and statistically significant, model M1) and it is, again, crucial to control for the body measures of children (model M2). Like in the over-perception model we find that perception transmission works particularly well in the event of successful transmission, here under-perception. The effects of over-perceiving parents do not even reach statistical significance. Adding the 5 weight environment measures and all its interactions with the 6 perception-communication types demonstrates the robustness of direct perception transmission (model M3). Indirect perception transmission is highly significant (p-value for joint significance = 0.0001). Our theoretical model suggests that an increase in the weight environment leads to an increase in the probability of under-perception. 9 out of the 12 shown coefficients have the expected sign, though, only one of them reaches statistical significance. Finally, we contrast the results of model M3 with a cluster fixed effects specification (model M4) and find no significant differences between the two.

3.5.2 Perception transmission and gender

In this subsection we discuss the results of both models, over-perception and under-perception, in more detail, including the most obvious case for effect heterogeneity: gender. Girls may simply respond differently than boys to both the perception type of their parents and family communication. Table 3.7 provides the results of a gender-stratified analysis.

For the over-perception model, the pooled analysis showed that the probability of over-perception of the child conditional on having correctly perceiving parents is 9 percentage points smaller when family communication is intact as compared to a family where it is not (-0.089). The stratified analysis reveals a significantly

Table 3.6: Under-perception model

		$M1$	$M2$	$M3$	$M4$
Under-perception model $\mathbf{Pr}(UPC = 1)$	CPP (C=0)	*reference group*			
	CPP (C=1)	-0.031** (0.013)	-0.027** (0.012)	-0.031** (0.012)	-0.029** (0.012)
	UPP (C=0)	0.283***(0.038)	0.193***(0.039)	0.178***(0.039)	0.183***(0.041)
	UPP (C=1)	0.267***(0.028)	0.180***(0.027)	0.179***(0.027)	0.174***(0.027)
	OPP (C=0)	-0.080***(0.018)	0.025 (0.017)	0.020 (0.017)	0.028 (0.018)
	OPP (C=1)	-0.092***(0.013)	0.016 (0.012)	0.012 (0.012)	0.017 (0.013)
	CPP (C=0) x BMI_M			-0.006 (0.010)	-0.010 (0.011)
	CPP (C=1) x BMI_M			0.006 (0.006)	0.006 (0.006)
	UPP (C=0) x BMI_M			0.021 (0.032)	0.034 (0.030)
	UPP (C=1) x BMI_M			-0.023 (0.026)	-0.016 (0.026)
	OPP (C=0) x BMI_M			0.005 (0.013)	0.007 (0.015)
	OPP (C=1) x BMI_M			0.006 (0.004)	0.003 (0.005)
	CPP (C=0) x BMI_F			-0.007 (0.012)	-0.007 (0.012)
	CPP (C=1) x BMI_F			0.004 (0.005)	0.006 (0.005)
	UPP (C=0) x BMI_F			0.003 (0.035)	-0.018 (0.032)
	UPP (C=1) x BMI_F			0.021 (0.024)	0.016 (0.025)
	OPP (C=0) x BMI_F			0.004 (0.013)	0.003 (0.013)
	OPP (C=1) x BMI_F			0.016***(0.004)	0.013** (0.005)
	Intercept	0.119***(0.026)	0.096***(0.026)	0.101***(0.026)	0.187***(0.025)
	$N(df)$	4,466 (4,435)	4,466 (4,416)	4,466 (4,386)	4,466 (4,242)
	R^2	0.142	0.209	0.212	0.216
Controls		√	√	√	√
Child's BM (in splines)			√	√	√
Weight environment Δ				√	√
Cluster-fixed effects					√
Specification-tests		Parental types	Child's BM	Δ	
	$F(n, m)$	F(5, 166)	F(19, 166)	F(30, 166)	
	$(Prob > chi2)$	74.75 (0.0000)	17.76 (0.0000)	2.53 (0.0001)	

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

larger effect for girls than for boys. While the probability drops by 12 percentage points for girls (-0.117), the effect for boys is about half the size (-0.063). This suggests that girls are more responsive to family communication than boys. Indeed, the pattern remains unchanged when looking at misperceiving parents. In the case of under-perceiving parents the effect of communication in the pooled model amounts to a 5 percentage point drop in the over-perception probability ($-0.116 - (-0.068)$). With a drop of 10 percentage points ($-0.161 - (-0.064)$) the change is much larger for girls than for boys (an insignificant drop of 2 percentage points, $-0.070 - (-0.048)$). Again, girls are more responsive to family communication. When considering children of over-perceiving parents, the pooled effect of communication is a 4 percentage point increase in the probability of over-perception ($0.189 - 0.153$). The probability that a girl over-perceives its own weight is 7 percentage points larger when family communication is intact as compared to a situation where it is not ($0.113 - 0.040$). The effect for boys is a mere percentage point ($0.246 - 0.234$). In all instances the empirical results match the predictions of our theory (see equation (2)).

It is also instructive to investigate differences in type transmission conditional on communication. In the pooled model, we find that, for restrained communication, the probability of the child over-perceiving its own weight is 15 percentage points higher when parents are over-perceivers as compared to correctly perceiving parents (0.153). With intact family communication, the effect is almost twice as high (about 28 percentage points, $0.189 - (-0.089)$). There are pronounced differences across gender: with intact family communication girls with over-perceiving parents are 23 percentage points more likely to be over-perceivers as compared to girls with correctly perceiving parents ($0.117 - (-0.113)$). For boys the effect is as large as 31 percentage points ($0.246 - (-0.063)$). Even more pronounced is the difference in the respective effects when the communication indicator is switched off. While the effects for girls is about 5 percentage points and indistinguishable from zero (0.040), the effect for boys amounts to a 23 percentage points increase (0.234). In the pooled model, conditional on restrained family communication, children of under-perceiving parents are 7 percentage points less likely to over-perceive their own weight (-0.068). The effects for girls and boys are slightly smaller but statistically indistinguishable from both the pooled estimate and zero. Things change when the communication indicator is switched on. There are no significant differences in the probability of over-perception between children of correctly perceiving parents and those of under-perceiving parents. This holds true

for the pooled and the stratified analysis.[37] To summarize, in the transmission of weight status over-perception intact family communication is an important factor for girls, not so much for boys. Independent of family communication, however, direct transmission of over-perception works better for boys than for girls.

Table 3.7: Effect heterogeneity - *gender*

		All	*Girls*	*Boys*
Over-perception model $\mathbf{Pr}(OPC = 1)$	CPP (C=0)		*reference group*	
	CPP (C=1)	-0.089*** (0.021)	-0.117*** (0.031)	-0.063** (0.027)
	UPP (C=0)	-0.068* (0.034)	-0.064 (0.069)	-0.048 (0.042)
	UPP (C=1)	-0.116*** (0.031)	-0.161*** (0.054)	-0.070** (0.034)
	OPP (C=0)	0.153*** (0.035)	0.040 (0.049)	0.234*** (0.052)
	OPP (C=1)	0.189*** (0.026)	0.113*** (0.039)	0.246*** (0.035)
	Intercept	0.305*** (0.038)	0.483*** (0.052)	0.308*** (0.051)
	N	4,485	2,195	2,290
Under-perception model $\mathbf{Pr}(UPC = 1)$	CPP (C=0)		*reference group*	
	CPP (C=1)	-0.031** (0.012)	-0.007 (0.012)	-0.051*** (0.019)
	UPP (C=0)	0.178*** (0.039)	0.114** (0.058)	0.186*** (0.058)
	UPP (C=1)	0.179*** (0.027)	0.243*** (0.047)	0.129*** (0.034)
	OPP (C=0)	0.020 (0.017)	0.044** (0.021)	0.015 (0.024)
	OPP (C=1)	0.012 (0.012)	0.011 (0.012)	0.014 (0.021)
	Intercept	0.101*** (0.026)	0.002 (0.030)	0.109** (0.044)
	N	4,466	2,189	2,277

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. All regressions include transmission variables (weight environment, interactions between parental perception types and weight environment) and controls. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

In the under-perception model, the pooled effect of communication conditional on correct weight perception of parents is a drop of 3 percentage points (−0.031). Interestingly, this effect is driven by boys. Their probability of weight under-perception drops by 5 percentage points when family communication is intact as compared to a situation where it is not (−0.051). The effect for girls is statistically not distinguishable from zero (−0.007). This suggests that, in the under-perception model, boys may be more responsive to communication than girls. Considering children of misperceiving parents, however, we see that this is not

[37]Recall that there are only very few families where weight perceptions between parents and children are off two categories (about 1 per cent, see Table 3.2). The respective results should be interpreted with caution.

generally the case. In the pooled model, the effect of communication on the probability of under-perception, conditional on under-perceiving parents, is virtually zero ($0.179 - 0.178$). Considering only girls, the effect is as large as 13 percentage points ($0.243 - 0.114$). With a drop of 6 percentage points, the effect for boys is negative ($0.129 - 0.186$) but statistically indistinguishable from zero. There is no significant effect of communication for families with over-perceiving parents. Other than in the over-perception model the results of the under-perception model are less clear cut. This may well be rooted in the relatively few children that under-perceive their weight status (see Table 3.2).

Finally, we investigate type transmission in the under-perception model conditional on communication. With disturbed family communication, the under-perception probability increases by 18 percentage points in the pooled analysis when parents are under-perceivers as compared to correct perceivers (0.178). The effect for boys is 7 percentage points larger ($0.186 - 0.114$) than for girls. With intact family communication, the pooled estimate is a 21 percentage point increase in the probability of under-perception ($0.179 - (-0.031)$). Broken down by gender, the effect for girls amounts to 25 percentage points ($0.243 - (-0.007)$) and 18 percentage points for boys ($0.129 - (-0.051)$). The probability that under-perception of parents is passed on to their offspring depends on communication and gender. In the event of disturbed communication the effect is larger for boys than for girls. We find the exact opposite when family communication is intact. This result is in contrast to the over-perception model, where parental over-perception is passed on to boys with a higher probability than for girls irrespective of family communication. Neither for the pooled analysis nor for the stratified analysis we find a significant effect of parental over-perception on the probability that the child under-perceives its weight status.

We can conclude that girls are more responsive to family communication than boys. We only found one instance where boys were more responsive. One should keep in mind, though, that this result obtained in the under-perception model that tends to produce 'weaker' results. We found the strongest effects for successful direct transmission of weight perception. To be more precise, in the over-perception model the absolute effect of over-perceiving parents as compared to correctly perceiving parents is larger than the effect of under-perceiving parents. In the under-perception model under-perceiving parents have an absolutely larger impact on children than over-perceiving parents.

3.5.3 Perception transmission and age

Another potential case for effect heterogeneity is age. The receptiveness of children to socialization efforts of their parents and influences from the (social) environment may depend on age. To investigate this we split the sample in two strata, the 11 to 13 years old and the 14 to 17 years old.

In the over-perception model there is no heterogeneity in the effect of communication for correctly perceiving parents; there is a drop in the probability of 8 percentage points for the younger stratum (-0.083) and a 10 percentage point drop in the older stratum (-0.097, see Table 3.8). Conditional on having under-perceiving parents, we find a remarkable difference in the effect of communication. In the younger group there is a drop of 12 percentage points ($-0.119 - 0.003$) whereas there is virtually no effect in the older group ($-0.098 - (-0.092)$). Since there are only very few families where weight perceptions are off two categories, this results should be interpreted with great caution (see footnote 37). More interesting in this respect is the case of over-perceiving parents. Here the effect of communication in the younger group is zero ($0.237 - 0.236$) and an increase of 5 percentage points in the older group ($0.136 - 0.089$). Given the standard errors, the differential effects of communication do not reach statistical significance.

It remains to investigate direct transmission conditional on communication. With disturbed family communication the probability of over-perception when having under-perceiving parents is, in the older group, 9 percentage points lower than for children with correctly perceiving parents (-0.092). There is no effect in the younger group (0.003). When the communication indicator is switched on, there is a drop of 4 percentage points in the younger group ($-0.119-(-0.083)$) and no change in the older group ($-0.098 - (0.097)$). Again, this mixed picture may well be rooted in the small number of observations (see the previous paragraph and footnote 37). There are substantial differences in point estimates across age groups when considering over-perceiving parents. With disturbed family communication, younger children are 24 percentage points more likely to over-perceive their weight status (0.236). In older children this effect only amounts to 9 percentage points (0.089). With an increase of 32 percentage points for younger children ($0.237 - (-0.083)$) and 23 percentage points for older children ($0.136-(-0.097)$), the same pattern emerges when family communication is intact.

Finally, as a brief look at the coefficient estimates reveals, there is no effect heterogeneity with respect to age in the under-perception model. We can summarize that there is no strong case for effect heterogeneity with respect to age.

Table 3.8: Effect heterogeneity - *age*

		All	11-13 *years*	14-17 *years*
Over-perception model $\mathbf{Pr}(OPC = 1)$	CPP (C=0)		*reference group*	
	CPP (C=1)	-0.089***(0.021)	-0.083***(0.031)	-0.097***(0.027)
	UPP (C=0)	-0.068* (0.034)	0.003 (0.063)	-0.092** (0.043)
	UPP (C=1)	-0.116***(0.031)	-0.119***(0.043)	-0.098** (0.039)
	OPP (C=0)	0.153***(0.035)	0.236***(0.052)	0.089* (0.048)
	OPP (C=1)	0.189***(0.026)	0.237***(0.037)	0.136***(0.035)
	Intercept	0.305***(0.038)	0.270***(0.054)	0.419***(0.057)
	N	4,485	2,092	2,393
Under-perception model $\mathbf{Pr}(UPC = 1)$	CPP (C=0)		*reference group*	
	CPP (C=1)	-0.031** (0.012)	-0.018 (0.019)	-0.039** (0.016)
	UPP (C=0)	0.178***(0.039)	0.177***(0.065)	0.178***(0.055)
	UPP (C=1)	0.179***(0.027)	0.185***(0.043)	0.189***(0.037)
	OPP (C=0)	0.020 (0.017)	0.023 (0.023)	0.016 (0.024)
	OPP (C=1)	0.012 (0.012)	0.009 (0.018)	0.013 (0.017)
	Intercept	0.101***(0.026)	0.057* (0.031)	0.115***(0.036)
	N	4,466	2,077	2,389

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. All regressions include transmission variables (weight environment, interactions between parental perception types and weight environment) and controls. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

If anything, we find that – independent of family communication – the direct over-perception transmission works marginally better for younger than for older children. This result provides indirect evidence for a more prominent role of peer effects in older children.

3.6 Extensions and robustness

3.6.1 A closer look at communication

One may argue that our communication indicator is too rough a measure to adequately capture the role of communication intensity in a transmission framework like ours. Based on our two variables, CP and CC, there are, indeed, many possibilities to construct more sophisticated measures. We here suggest a measure that, instead of two, distinguishes between four levels of communication intensity, namely, disturbed, good, very good, and excellent. We defined four respective

indicator variables. The indicator for disturbed family communication assumes the value 1 if parents and children answered the communication question worse than 'rather true' (this is identical to $C = 0$ in our main specification). There is good family communication when both answered 'rather true.' We say that family communication is very good if one answer was 'rather true' and the other 'exactly true.' Finally, excellent communication is given when we got two 'exactly true' answers.

Table 3.9 contrasts the results of the more sophisticated model with our baseline model. In the over-perception model, communication still has a significantly negative effect for correctly perceiving parents. We find, as predicted by our theory (see equation (3.2)), that the probability of over-perception monotonically decreases with communication intensity. The respective coefficients are not statistically different from one another, though. We obtain the same result for misperceiving parents. In the under-perception model and correctly perceiving parents we also find a monotone relationship of communication intensity. Again, the respective coefficients do not statistically differ. The same holds true for misperceiving parents. These findings imply that our binary communication measure is well suited to capture the impact of family communication on perception transmission and that there is not much to be gained from more precise modeling.[38]

3.6.2 BMI thresholds and the reference weight distribution

The construction of our misperception variables required one objective weight status measure and two subjective weight status measures. Subjective information is available in five categories ranging from far too thin to far too heavy. Accordingly, we classified a child's bodyweight also using five categories ranging form strong underweight to obesity. Rather plausibly, we argued that there is an under(over)-perception of weight status, whenever the subjective assessment fell into a lower (higher) category than the objective measure. To categorize the actual weight of children, we used their relative position in the weight distribution of our sample children and applied the percentile thresholds that are usually being used in Germany, namely, the 3rd, 10th, 90th, and 97th percentile. These thresholds are somewhat arbitrary and it seems particularly odd to consider as many as 80 % of children as normally weighed. Table 3.10 shows that our results are remark-

[38] We tried numerous alternative communication definitions but none of them produced results that differed from the ones presented in this subsection.

Table 3.9: Communication intensity

		Communication		
		Indicator		*Flexible*
Over-perception model **Pr**$(OPC = 1)$	CPP (C=0)	*reference group*	CPP (C=0)	*reference group*
	CPP (C=1)	-0.089***(0.021)	CPP (C=good)	-0.075***(0.024)
			CPP (C=better)	-0.087***(0.022)
			CPP (C=best)	-0.120***(0.030)
	UPP (C=0)	-0.068* (0.034)	UPP (C=0)	-0.067* (0.034)
	UPP (C=1)	-0.116***(0.031)	UPP (C=good)	-0.140***(0.040)
			UPP (C=better)	-0.099***(0.036)
			UPP (C=best)	-0.119***(0.043)
	OPP (C=0)	0.153***(0.035)	OPP (C=0)	0.152***(0.035)
	OPP (C=1)	0.189***(0.026)	OPP (C=good)	0.190***(0.033)
			OPP (C=better)	0.191***(0.033)
			OPP (C=best)	0.183***(0.035)
	Intercept	0.305***(0.038)	Intercept	0.302***(0.038)
	N	4,485	N	4,485
Under-perception model **Pr**$(UPC = 1)$	CPP (C=0)	*reference group*	CPP (C=0)	*reference group*
	CPP (C=1)	-0.031** (0.012)	CPP (C=good)	-0.024 (0.015)
			CPP (C=better)	-0.027** (0.014)
			CPP (C=best)	-0.049***(0.015)
	UPP (C=0)	0.178***(0.039)	UPP (C=0)	0.180***(0.039)
	UPP (C=1)	0.179***(0.027)	UPP (C=good)	0.182***(0.043)
			UPP (C=better)	0.138***(0.034)
			UPP (C=best)	0.255***(0.054)
	OPP (C=0)	0.020 (0.017)	OPP (C=0)	0.018 (0.017)
	OPP (C=1)	0.012 (0.012)	OPP (C=good)	0.008 (0.012)
			OPP (C=better)	0.015 (0.015)
			OPP (C=best)	0.005 (0.013)
	Intercept	0.101***(0.026)	Intercept	0.109***(0.026)
	N	4,466	N	4,466

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. All regressions include transmission variables (weight environment, interactions between parental perception types and weight environment) and controls. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

ably robust to changes in the normal weight range. To ease comparison, the first column repeats the regression results of our main specification (see model M3 in Tables 3.5 and 3.6). In the second column, the normal weight category ranges from the 16th to the 84th percentile, while the third column considers all children between the 25th and the 75th percentile as normally weighed. We find only minor differences in coefficient estimates across columns. The over-perception model appears slightly more robust than the under-perception model but even in the latter differences in coefficients are far from reaching statistical significance.

Another potential shortcoming of our misperception measures is that the objective weight status categorization rests on the weight distribution of sampled children and may thus be subject to the obesity epidemic (see also Burke and Heiland (2007)). As a result, children that would have been considered over-weight when the 'healthy' weight distribution was used, for instance, may be categorized as normally weighed. Although we are unaware of what constitutes the healthy weight distribution we can use the weight distribution before the onset of the obesity pandemic to categorize children, the Kromeyer-Hauschild BMI thresholds.[39] The respective regression results are shown in the fourth column of Table 3.10. Again, there are no significant changes in coefficient estimates. Finally, instead of using the German percentiles (3, 10, 90, and 97) for weight status categorization we conducted our analysis for the CDC percentiles used in the United States: 5, 85, and 95. Notably, the CDC categorization only distinguishes between 4 weight categories. In order to construct our misperception measures we lump together the two lowest subjective weight status assessments, namely, 'far too thin' and 'too thin.' The corresponding results (fifth column of Table 3.10) demonstrate the robustness of our main specification report in the first column.

3.6.3 Ignoring direct perception transmission

Ali *et al.* (2011) analyzed weight perceptions of children and found that the weight environment has some explanatory power. Our theory suggests that direct perception transmission plays an important role and that direct and indirect transmission are closely connected. To shed some more light on the relationship between the two transmission channels, we ran a regression of child misperception indicators on the full set of controls and our five weight environment measures (see Table 3.17 in the Appendix). Like in Ali *et al.* (2011) the weight environ-

[39]These thresholds are the basis of the growth charts that are displayed in Germany's screening exam booklets for children.

Table 3.10: Normal weight percentiles and reference weight distribution

		lower and upper normal weight percentile boundaries			***K-H***[1]	***CDC***[1]
		10th-90th	*16th-84th*	*25th-75th*		
Over-perception model **Pr**$(OPC = 1)$	CPP (C=0)			*reference group*		
	CPP (C=1)	-0.089*** (0.021)	-0.077*** (0.020)	-0.093*** (0.021)	-0.076*** (0.020)	-0.077*** (0.020)
	UPP (C=0)	-0.068* (0.034)	-0.073** (0.036)	-0.092*** (0.035)	-0.042 (0.032)	-0.042 (0.033)
	UPP (C=1)	-0.116*** (0.031)	-0.116*** (0.029)	-0.132*** (0.028)	-0.096*** (0.028)	-0.104*** (0.028)
	OPP (C=0)	0.153*** (0.035)	0.139*** (0.035)	0.144*** (0.035)	0.187*** (0.040)	0.174*** (0.042)
	OPP (C=1)	0.189*** (0.026)	0.195*** (0.027)	0.181*** (0.029)	0.220*** (0.027)	0.211*** (0.028)
	Intercept	0.305*** (0.038)	0.292*** (0.040)	0.349*** (0.040)	0.278*** (0.040)	0.276*** (0.039)
	N	4,485	4,485	4,485	4,303	4,385
Under-perception model **Pr**$(UPC = 1)$	CPP (C=0)			*reference group*		
	CPP (C=1)	-0.031** (0.012)	-0.026** (0.012)	-0.026** (0.012)	-0.037*** (0.012)	-0.035*** (0.012)
	UPP (C=0)	0.178*** (0.039)	0.156*** (0.042)	0.156*** (0.044)	0.141*** (0.037)	0.170*** (0.036)
	UPP (C=1)	0.179*** (0.027)	0.170*** (0.027)	0.139*** (0.027)	0.180*** (0.023)	0.194*** (0.022)
	OPP (C=0)	0.020 (0.017)	-0.006 (0.017)	-0.016 (0.018)	-0.008 (0.017)	0.002 (0.015)
	OPP (C=1)	0.012 (0.012)	-0.020 (0.012)	-0.034** (0.013)	0.002 (0.014)	0.016 (0.013)
	Intercept	0.101*** (0.026)	0.087*** (0.025)	0.106*** (0.025)	0.097*** (0.030)	0.081*** (0.029)
	N	4,466	4,466	4,466	4,507	4,360

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. [1] Kromeyer-Hauschild: older German BMI thresholds, CDC US-percentile boundaries. All regressions include transmission variables (weight environment, interactions between parental perception types and weight environment) and controls. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

ment turns out statistically significant (F-test p-values = 0.0341 and 0.0534, for the over-perception model and the under-perception model, respectively). To contrast this regression with our approach, we add the direct transmission channel (6 perception-communication types). The F-tests on the weight environment variables cannot reject the null hypothesis that the weight environment is irrelevant (p-values = 0.1514 and 0.1973). There is, thus, an important relationship between transmission channels that Ali *et al.* (2011) are unable to capture.

To arrive at model M3 of Tables 3.5 and 3.6 we finally add three-way interactions, namely, all interactions of the 5 weight environment measures with the 6 perception-communication types, where correctly perceiving parents with disturbed family communication serve as a reference category. The three-way interactions turn out significant in the over-perception model (p-value = 0.0682) but insignificant in the under-perception model (p-value = 0.1810).[40] We already know from Section 5.1, however, that the weight environment including all its interaction is highly statistically significant, that is, the indirect transmission channel is active but only when considering its interaction with direct transmission variables.

3.6.4 Non-linear probability models

The linear probability model is our preferred econometric model. The main reason is that coefficient estimates can readily be interpreted. This is of particular importance in a model that includes a great many of interactions. To assess the robustness of our results we contrast the outcome of the linear probability models (the first column in Table 3.11) with alternative non-linear models.

In a first step, we ran two probit models (second column). Neither the directional effects nor the significance changes. The average marginal effects show that there are only moderate differences in quantitative effects.[41] In our setting, the generalized ordered probit model is the perhaps most interesting non-linear model. As already argued above, to avoid censoring, we have to exclude the heaviest three per cent and the lightest 3 per cent from our sample. For the sake of comparison we reestimate, as a second step, the two probit models of the second

[40]Due to the weight environment interactions of model (3) in Table 3.17 the coefficients of the direct transmission variables cannot directly be compared to the other two columns. While model (3) shows the effects for the reference group (correctly perceiving parents with disturbed family communication), models (1) and (2) show the average effect across all perception-communication types.

[41]To save space, we do not present average marginal effects for the remaining models as they are very similar in size.

Table 3.11: LPM and non-linear models

		LPM **Pr**($OPC=1$)		**Probit** **Pr**($OPC=1$)	**Probit** **Pr**($OPC=1$)	**GOP** (free) **Pr**($MPC=1$)	**GOP** (**restricted**) **Pr**($MPC=1$)
weight status categories		4 out of 5 *Coef.*	*AME*	4 out of 5 *Coef.*	3 out of 5 *Coef.*	3 out of 5 *Coef.*	3 out of 5 *Coef.*
Over-perception model **Pr**($OPC=1$)	CPP (C=0)			*reference group*			
	CPP (C=1)	-0.089*** (0.021)	-0.087	-0.276*** (0.065)	-0.277*** (0.066)	-0.270*** (0.066)	-0.275*** (0.066)
	UPP (C=0)	-0.068* (0.034)	-0.076	-0.276* (0.150)	-0.276* (0.150)	-0.291* (0.155)	-0.283* (0.154)
	UPP (C=1)	-0.116*** (0.031)	-0.132	-0.452*** (0.130)	-0.448*** (0.131)	-0.431*** (0.129)	-0.433*** (0.128)
	OPP (C=0)	0.153*** (0.035)	0.130	0.429*** (0.130)	0.441*** (0.133)	0.430*** (0.133)	0.426*** (0.133)
	OPP (C=1)	0.189*** (0.026)	0.190	0.595*** (0.090)	0.591*** (0.091)	0.598*** (0.091)	0.585*** (0.091)
	Intercept	0.305*** (0.038)		-0.704*** (0.139)	-0.730*** (0.140)	-0.725*** (0.138)	-0.682*** (0.129)
	N	4,485		4,485	4,340	4,340	4,340
		Pr($UPC=1$)		**Pr**($UPC=1$)	**Pr**($UPC=1$)	**Pr**($MPC=-1$)	**Pr**($MPC=-1$)
weight status categories		4 out of 5 *Coef.*	*AME*	4 out of 5 *Coef.*	3 out of 5 *Coef.*	3 out of 5 *Coef.*	3 out of 5 *Coef.*
Under-perception model **Pr**($UPC=1$)	CPP (C=0)			*reference group*			
	CPP (C=1)	-0.031** (0.012)	-0.028	-0.231*** (0.085)	-0.239*** (0.088)	-0.231*** (0.088)	0.251*** (0.087)
	UPP (C=0)	0.178*** (0.039)	0.072	0.434*** (0.139)	0.480*** (0.149)	0.476*** (0.147)	-0.494*** (0.144)
	UPP (C=1)	0.179*** (0.027)	0.076	0.456*** (0.109)	0.474*** (0.116)	0.490*** (0.116)	-0.488*** (0.114)
	OPP (C=0)	0.020 (0.017)	-0.004	-0.065 (0.248)	-0.072 (0.250)	-0.066 (0.242)	0.082 (0.250)
	OPP (C=1)	0.012 (0.012)	-0.069	-0.816*** (0.285)	-0.829*** (0.289)	-0.832*** (0.317)	0.862*** (0.331)
	Intercept	0.101*** (0.026)		-1.632*** (0.209)	-1.623*** (0.226)	-1.633*** (0.224)	1.517*** (0.166)
	N	4,466		4,466	4,340	4,340	4,340

Notes: Clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01. All regressions include transmission variables (weight environment, interactions between parental perception types and weight environment) and controls. Controls are child's age, gender, puberty, school type, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

column only including children from three weight status categories instead of four (third column of Table 3.11). The sample size drops by about three per cent3 from 4485 to 4340 in the over-perception model and from 4466 to 4340 in the under-perception model. There are no significant changes in coefficient estimates. In a third step, we fit a generalized ordered probit model where the probability of over-perception and under-perception are estimated simultaneously. The results are shown in the fourth column of Table 3.11. This model allows all coefficients to differ between over- and under-perception, that is, all variables are freed from the parallel lines assumption of the standard ordered probit model. In a final step, we reintroduce the parallel lines assumption for those control variables where the Brant test suggests equality of coefficients as this might improve efficiency. The respective regression results are shown in the last column of Table 3.11. Again, there is hardly any difference in coefficient estimates. Notably, considering the standard errors of the restricted generalized ordered probit model, there are no efficiency gains, neither over the more flexible generalized ordered probit model of column four nor over the two separate probit models of column 3. In fact, the standard errors of the two probit models of column two tend to be smaller than the standard errors of the generalized ordered probit models, that is, the potential gain in efficiency of the generalized ordered probit model tends to be (more than) offset by efficiency losses originating in the drop in sample size. After all, the results of our linear probability model are highly robust to alternative non-linear specifications.

3.7 Conclusion

Given the irresistible and almost worldwide increase in population weight, policy measures are needed to get the obesity epidemic under control. Kuchler and Variyam (2003) argued that correct weight perception is an important factor in healthy weight management, that is, unhealthy weight may be the result of weight status misperceptions. For health policy makers it is, thus, decisive to understand how weight perceptions are being formed.

We argue that these perceptions are likely to be developed during childhood and adolescence and that both, parents and the social environment of the child play an important role therein. Adapting Bisin and Verdier's (2001) framework we provide a theoretical foundation for the formation of weight perceptions. The model distinguishes between direct and indirect perception transmission. The

former transmission channel points to the key role of parents in the socialization process of their offspring. The indirect channel acknowledges that the weight environment of the child might shape weight perception. To tilt the balance between the two transmission channels parents can exert some costly effort, in our case, family communication. Using a representative German data set we simultaneously analyze both transmission channels highlighting the importance of family communication. So far empirical studies on the formation of weight misperceptions in children and adolescents concentrated on indirect perception transmission, that is, on the influence the weight environment has on weight perception (see, *e.g.*, Ali, Amialchuk, and Renna (2011) and Maximova et al. (2008)). Though valuable, these studies are incomplete as they miss the likely more important direct transmission channel. Furthermore, the theoretical model suggests that transmission channels are closely connected and that parents have an influence on their relative strength or, more, precisely, that there are important interactions amongst direct transmission, indirect transmission, and effort. We also contribute to the more general literature on the intergenerational transmission of preferences by employing a convincing measure for parental effort, namely, family communication. Finally, we let a theoretical model guide the econometric specification and find that, for the most part, the theoretical predictions are supported by our empirical results, most importantly, the effects of communication as well as the importance of three-way interactions amongst transmission variables.

We find overwhelming evidence for direct perception transmission from parents to their offspring and for the role of communication therein. In particular, conditional on having correctly perceiving parents, the probability that a child misperceives its weight status is significantly lower when family communication is intact as compared to a family where it is not. Although the variables capturing indirect perception transmission, including all their interactions, reach joint statistical significance, no clear pattern emerges. We found some interesting heterogeneities in perception transmission. Girls seem to be more responsive to communication than boys. Conditional on communication, however, direct transmission works considerably better for boys. Transmission effects are largely independent of the age of the child. If anything, direct transmission of weight over-perception is more likely in younger children than in older ones. We investigated a number of variants of our main specification and found that our results are remarkably robust. Neither a more detailed analysis of communication, nor different thresholds for weight status categories or an alternative reference weight distribution changed

our results.

Given that our regression models account for substantial individual heterogeneity we are confident that our transmission variables are as good as randomly assigned, that is, we conjecture that our regression results can be interpreted causally. Good health policy should, thus, aim at preventing or at least at mitigating the transmission of weight status misperceptions. School teachers and doctors could, for instance, inform children and their parents about the actual weight status of the child and, if necessary, suggest measures for healthy weight management. Medical check-ups and screening examinations are potential settings. In fact, weight and height are measured as part of the screening exams in Germany. Overall there are 14 such exams and 12 of those are for children aged 10 years and below.For our sample children, there are two such exams, namely, the J1 (12 to 14 years) and the J2 (16 to 17 years).[42] Participation in these screening examinations is voluntary and the share of children actually undergoing them decreases with age. While almost all children receive the first exams, the participation rate drops to 34 % for the J1 in our sample. Also note that not all exam are included in the minimum benefit package of Germany's public health insurance scheme: the U7a, U10, U11, and J2 are not generally covered. In order to reach all families making participation in (a subset of) these exams compulsory and facilitating health insurance coverage seems a sensible option for public policy. If necessary, parents (and children) should be informed about weight management strategies.[43] Based on our results we are unable to assess the likely impact of such a policy on perception transmission. It appears nevertheless worthwhile to consider it, at least for a pilot study. We leave this for future research.

[42]There are 12 'U-exams', U1 to U11 (plus U7a), and two 'J-exams.'

[43]In 2008 the Federal Ministry of Food and Agriculture and the Federal Ministry of Health launched "Germany's national initiative to promote health diets and physical activity." (see http://www.in-form.de)

3.8 Appendix

A. Transition probabilities and the comparative static effects of the weight environment

$$\frac{\partial p_{UU}}{\partial \Delta} = \frac{\partial p_{UU}}{\partial e^*_U}\frac{\partial e^*_U}{\partial \Delta} + (1-\pi_U)q'_U \lesseqgtr 0$$
$$\frac{\partial p_{UN}}{\partial \Delta} = \frac{\partial p_{UN}}{\partial e^*_U}\frac{\partial e^*_U}{\partial \Delta} - (1-\pi_U)\left(q'_U + q'_O\right) \lesseqgtr 0$$
$$\frac{\partial p_{UO}}{\partial \Delta} = \frac{\partial p_{UO}}{\partial e^*_U}\frac{\partial e^*_U}{\partial \Delta} + (1-\pi_U)q'_O \lesseqgtr 0$$
$$\frac{\partial p_{NU}}{\partial \Delta} = \frac{\partial p_{NU}}{\partial e^*_N}\frac{\partial e^*_N}{\partial \Delta} + (1-\pi_N)q'_U \lesseqgtr 0$$
$$\frac{\partial p_{NN}}{\partial \Delta} = \frac{\partial p_{NN}}{\partial e^*_N}\frac{\partial e^*_N}{\partial \Delta} - (1-\pi_N)(q'_U + q'_O) \lesseqgtr 0$$
$$\frac{\partial p_{NO}}{\partial \Delta} = \frac{\partial p_{NO}}{\partial e^*_N}\frac{\partial e^*_N}{\partial \Delta} + (1-\pi_N)q'_O \lesseqgtr 0$$
$$\frac{\partial p_{OU}}{\partial \Delta} = \frac{\partial p_{OU}}{\partial e^*_O}\frac{\partial e^*_O}{\partial \Delta} + (1-\pi_O)q'_U \lesseqgtr 0$$
$$\frac{\partial p_{ON}}{\partial \Delta} = \frac{\partial p_{ON}}{\partial e^*_O}\frac{\partial e^*_O}{\partial \Delta} - (1-\pi_O)(q'_U + q'_O) \lesseqgtr 0$$
$$\frac{\partial p_{OO}}{\partial \Delta} = \frac{\partial p_{OO}}{\partial e^*_O}\frac{\partial e^*_O}{\partial \Delta} + (1-\pi_O)q'_O \lesseqgtr 0$$

Table 3.12: Explanation of transmission variables

Dependent variables		
MPC	Child's misperception (three categories): -1 = Child under-evaluated its own weight, 0 = Child evaluated its own weight correctly, 1 = Child under-evaluated its own weight	
UPC	Child's under-perception[b]: 1 = Child under-evaluated its own weight, 0 else	
CPC	Child's correct perception[b]: 1 = Child evaluated its own weight correctly, 0 else	
OPC	Child's over-perception[b]: 1 = Child over-evaluated its own weight, 0 else	
Explanatory variables		
Direct transmission variables		
UPP	Parents' under-perception[b]: 1 = Parents under-evaluated their child's weight, 0 else	
CPP	Parents' correct perception[b]: 1 = Parents evaluated their child's weight correctly, 0 else	
OPP	Parents' over-perception[b]: 1 = Parents over-evaluated their child's weight, 0 else	
C	Familial communication effort[b]: 1 = if child and parents rate family communication sufficiently good, 0 else	
Parents types		
UPP(C=0)	Parents' under-perception with no good communication[b]:	$UPP * C$
UPP(C=1)	Parents' under-perception with good communication[b]:	$UPP(1-C)$
CPP(C=0)	Parents' correct perception with no good communication [b]:	$CPP * C$
CPP(C=1)	Parents' correct perception with good communication[b]:	$CPP(1-C)$
OPP(C=0)	Parents' over-perception with no good communication [b]:	$OPP * C$
OPP(C=1)	Parents' over-perception with good communication[b]:	$OPP(1-C)$
Weight variables - Child's body measures		
BMI child	Child's Body-mass index (SDS)[c]	
WHR child	Child's waist-to-hip ratio (SDS)[c]	
WHtR child	Child's waist-to-height ratio (SDS)[c]	
SSF child	Child's sum of skin folds - triceps and back - (SDS)[c]	
Weight variables - Environment		
BMI peer	Average BMI of all children within a cluster (SDS)[c]	
BMI mother	Mother's BMI (standardized)[c]	
BMI father	Father's BMI (standardized)[c]	
BMI female	Average BMI of all female adults within a cluster (standardized)[c]	
BMI male	Average BMI of all male adults within a cluster (standardized)[c]	

Notes: [b] = binary, [c] = continuous, BMI = body-mass index, SDS = Standard-Deviation Score

Table 3.13: Summary statistics - *transmission* variables

Variables	**Mean**	**S.E.**	**Min**	**Max**
Child's perception of its weight status				
UPC	0.105	0.307	0	1
CPC	0.484	0.500	0	1
OPC	0.411	0.492	0	1
Parents' perception of child's weight status				
UPP	0.119	0.324	0	1
CPP (reference group)	0.670	0.470	0	1
OPP	0.211	0.408	0	1
Child's, parents' and familial communication				
CC	0.805	0.397	0	1
CP	0.921	0.270	0	1
C (CC*CP)	0.758	0.428	0	1
Parents types				
UPP(C=0)	0.032	0.176	0	1
UPP(C=1)	0.087	0.281	0	1
CPP(C=0) (reference group)	0.160	0.366	0	1
CPP(C=1)	0.510	0.500	0	1
OPP(C=0)	0.050	0.218	0	1
OPP(C=1)	0.161	0.368	0	1
Child's body measures				
BMI	20.713	3.806	11.940	44.571
BMI (SDS)	-0.044	0.993	-4.010	2.867
WHR	0.789	0.055	0.634	1.096
WHR (SDS)	-0.056	0.982	-4.069	4.449
WHtR	0.426	0.047	0.324	0.757
WHtR (SDS)	-0.073	0.985	-4.244	2.931
SSF	27.122	12.741	8	85.2
SSF (SDS)	-0.036	0.998	-3.21	2.438
Weight environment				
BMI mother	24.862	4.782	15.060	67.578
BMI mother (standardized)	0	1.000	-2.050	8.933
BMI father	26.505	3.590	17.182	57.36
BMI father (standardized)	0	1.000	-2.597	8.596
BMI peer	-0.044	0.208	-0.601	0.498
BMI female	0	0.198	-0.396	0.799
BMI male	0	0.214	-0.590	0.710

Note: N=4,611

Table 3.14: Summary statistics - *control* variables

Variables	**Mean**	**S.E.**	**Min**	**Max**
Child's age, gender and background				
Age (continuous)	14.294	1.970	11.001	17.982
Aged 11 (reference group)	0.161	0.368	0	1
Aged 12	0.151	0.358	0	1
Aged 13	0.154	0.361	0	1
Aged 14	0.148	0.355	0	1
Aged 15	0.147	0.355	0	1
Aged 16	0.129	0.335	0	1
Aged 17	0.110	0.312	0	1
Girls	0.489	0.500	0	1
Migration	0.104	0.305	0	1
Puberty status				
Infantile (Tanner I) (reference group)	0.066	0.249	0	1
Early puberty (Tanner II and III)	0.239	0.427	0	1
Late puberty (Tanner IV to VI)	0.694	0.461	0	1
School type				
Grundschule	0.028	0.164	0	1
Orientierungsstufe	0.016	0.127	0	1
Foerder-/Sonderschule	0.021	0.144	0	1
Hauptschule	0.142	0.349	0	1
Realschule	0.319	0.466	0	1
Gesamtschule	0.079	0.270	0	1
Gymnasium (reference group)	0.394	0.489	0	1
Houshold				
No. of persons in houshold	4.203	1.064	1	14
No of persons in houshold (centered)	0	1.064	-3.203	9.797
Low SES (reference group)	0.205	0.403	0	1
Middle SES	0.509	0.500	0	1
High SES	0.286	0.452	0	1
Child resides with ...				
both parents (reference group)	0.875	0.331	0	1
one parent and partner	0.085	0.278	0	1
one parent	0.040	0.197	0	1
Region				
East Germany	0.333	0.471	0	1
Rural	0.239	0.427	0	1
Small urban	0.267	0.442	0	1
Middle urban (reference group)	0.284	0.451	0	1
Urban	0.210	0.407	0	1
Questionnaire responder				
Mother (reference group)	0.845	0.362	0	1
Father	0.108	0.310	0	1
Both	0.048	0.214	0	1

Note: N=4,611

Table 3.15: KiGGS percentile values by *gender* and *age*

	Boys									Girls								
Age	*P*3	*P*10	*P*25	*P*50	*P*75	*P*90	*P*97	*L*	*S*	*P*3	*P*10	*P*25	*P*50	*P*75	*P*90	*P*97	*L*	*S*
11	14.36	15.28	16.41	17.99	20.11	22.78	26.75	-1.8589	0.1491	14.06	15.06	16.28	18.00	20.29	23.18	27.4	-1.6351	0.1617
11.5	14.52	15.48	16.65	18.30	20.50	23.26	27.32	-1.7843	0.1522	14.30	15.33	16.60	18.37	20.75	23.74	28.11	-1.6009	0.164
12	14.70	15.69	16.90	18.60	20.87	23.71	27.86	-1.726	0.1546	14.59	15.65	16.95	18.77	21.21	24.27	28.73	-1.5791	0.1647
12.5	14.89	15.91	17.16	18.90	21.23	24.13	28.36	-1.6839	0.1562	14.91	15.99	17.31	19.17	21.65	24.75	29.24	-1.5706	0.1641
13	15.11	16.15	17.43	19.21	21.59	24.54	28.83	-1.6563	0.1571	15.25	16.35	17.69	19.57	22.07	25.18	29.67	-1.5758	0.1623
13.5	15.34	16.40	17.70	19.52	21.93	24.93	29.26	-1.6402	0.1572	15.60	16.71	18.06	19.94	22.45	25.55	30.02	-1.5943	0.1598
14	15.59	16.67	17.99	19.83	22.28	25.30	29.65	-1.6319	0.1567	15.95	17.06	18.41	20.30	22.79	25.88	30.32	-1.6252	0.1568
14.5	15.86	16.95	18.29	20.15	22.61	25.65	29.99	-1.6279	0.1557	16.29	17.39	18.74	20.62	23.10	26.17	30.58	-1.6672	0.1536
15	16.14	17.24	18.59	20.47	22.94	25.98	30.30	-1.6259	0.1542	16.60	17.70	19.04	20.91	23.37	26.42	30.79	-1.7187	0.15'03
15.5	16.43	17.54	18.90	20.79	23.26	26.29	30.58	-1.6246	0.1525	16.90	17.98	19.31	21.16	23.60	26.62	30.97	-1.7778	0.1471
16	16.72	17.84	19.21	21.10	23.58	26.60	30.83	-1.6239	0.1505	17.16	18.23	19.55	21.37	23.79	26.79	31.12	-1.8427	0.1442
16.5	17.01	18.15	19.52	21.42	23.89	26.89	31.07	-1.6242	0.1484	17.38	18.45	19.74	21.55	23.95	26.92	31.25	-1.9117	0.1414
17	17.30	18.44	19.82	21.72	24.19	27.17	31.29	-1.6257	0.1464	17.58	18.63	19.91	21.70	24.07	27.03	31.36	-1.983	0.1389
17.5	17.59	18.74	20.12	22.03	24.49	27.45	31.52	-1.6281	0.1443	17.76	18.8	20.06	21.83	24.18	27.12	31.46	-2.0556	0.1366
18	17.87	19.02	20.41	22.31	24.77	27.71	31.73	-1.6308	0.1424	17.93	18.95	20.20	21.95	24.27	27.20	31.55	-2.1259	0.1345

Notes: For the KiGGS data see Neuhauser et al. (2013), p. 40f

Table 3.16: K-H percentile values by *gender* and *age*

	Boys									**Girls**								
Age	$P3$	$P10$	$P25$	$P50$	$P75$	$P90$	$P97$	L	S	$P3$	$P10$	$P25$	$P50$	$P75$	$P90$	$P97$	L	S
11	14.11	14.97	16.00	17.41	19.24	21.43	24.45	-1.77	0.14	13.95	14.88	15.99	17.50	19.4	21.61	24.51	-1.43	0.14
11.5	14.3	15.18	16.24	17.70	19.58	21.84	24.96	-1.75	0.14	14.18	15.14	16.28	17.83	19.78	22.04	25.00	-1.39	0.14
12	14.5	15.41	16.50	17.99	19.93	22.25	25.44	-1.72	0.14	14.45	15.43	16.6	18.19	20.18	22.48	25.47	-1.36	0.14
12.5	14.73	15.66	16.77	18.30	20.27	22.64	25.88	-1.69	0.14	14.74	15.75	16.95	18.56	20.58	22.91	25.92	-1.33	0.14
13	14.97	15.92	17.06	18.62	20.62	23.01	26.28	-1.66	0.14	15.04	16.07	17.30	18.94	20.98	23.33	26.33	-1.3	0.14
13.5	15.23	16.19	17.35	18.94	20.97	23.38	26.64	-1.63	0.14	15.35	16.40	17.64	19.30	21.36	23.71	26.7	-1.27	0.14
14	15.50	16.48	17.65	19.26	21.30	23.72	26.97	-1.61	0.14	15.65	16.71	17.97	19.64	21.71	24.05	27.01	-1.25	0.14
14.5	15.77	16.76	17.96	19.58	21.63	24.05	27.26	-1.58	0.14	15.92	17.00	18.27	19.95	22.02	24.35	27.26	-1.23	0.14
15	16.04	17.05	18.25	19.89	21.95	24.36	27.53	-1.55	0.14	16.18	17.26	18.53	20.22	22.28	24.59	27.45	-1.2	0.14
15.5	16.31	17.33	18.55	20.19	22.26	24.65	27.77	-1.52	0.13	16.40	17.49	18.76	20.45	22.50	24.77	27.57	-1.18	0.13
16	16.57	17.60	18.83	20.48	22.55	24.92	27.99	-1.49	0.13	16.60	17.69	18.96	20.64	22.67	24.91	27.65	-1.16	0.13
16.5	16.83	17.87	19.11	20.77	22.83	25.18	28.2	-1.47	0.13	16.78	17.87	19.14	20.81	22.82	25.02	27.69	-1.13	0.13
17	17.08	18.13	19.38	21.04	23.10	25.44	28.4	-1.44	0.13	16.95	18.04	19.31	20.96	22.95	25.11	27.72	-1.11	0.13
17.5	17.32	18.39	19.64	21.31	23.36	25.68	28.6	-1.41	0.13	17.11	18.20	19.47	21.11	23.07	25.20	27.74	-1.09	0.13
18	17.56	18.63	19.89	21.57	23.61	25.91	28.78	-1.39	0.13	17.27	18.36	19.62	21.25	23.19	25.28	27.76	-1.07	0.12

Notes: For KH data see Kromeyer-Hauschild et al. (2001, 812f)

Table 3.17: Weight environment

		(1)	(2)	(3)
Over-perception model $\mathbf{Pr}(OPC = 1)$	BMI_mother	-0.021*** (0.007)	-0.015** (0.007)	0.020 (0.016)
	BMI_father	-0.000 (0.007)	0.003 (0.007)	0.037** (0.017)
	BMI_peers	-0.009 (0.037)	0.002 (0.035)	-0.023 (0.095)
	BMI_female_adults	-0.044 (0.040)	-0.052 (0.037)	-0.055 (0.103)
	BMI_male_adults	-0.003 (0.033)	-0.001 (0.033)	-0.060 (0.087)
	Intercept	0.284*** (0.035)	0.302*** (0.039)	0.305*** (0.038)
	$N(df)$	4,485 (4,433)	4,485 (4,428)	4,485 (4,403)
	R^2	0.283	0.314	0.315
Under-perception model $\mathbf{Pr}(UPC = 1)$	BMI_mother	0.005 (0.004)	0.002 (0.004)	-0.006 (0.010)
	BMI_father	0.007 (0.005)	0.005 (0.004)	-0.007 (0.012)
	BMI_peers	-0.001 (0.027)	-0.007 (0.026)	0.051 (0.061)
	BMI_female_adults	0.005 (0.025)	0.008 (0.025)	-0.135** (0.058)
	BMI_male_adults	0.036* (0.022)	0.034 (0.021)	0.077 (0.053)
	Intercept	0.124*** (0.026)	0.099*** (0.027)	0.101*** (0.026)
	$N(df)$	4,466 (4,414)	4,466 (4,409)	4,466 (4,384)
	R^2	0.169	0.209	0.212
Parent types (6)			√	√
3-*way-interactions*				√
Specification-tests	$F(n, m)$	Δ F(5, 166)	Δ F(5, 166)	3-*way-interactions* F(25, 166)
$\mathbf{Pr}(OPC = 1)$	$(Prob > chi2)$	2.48 (0.0341)	1.64 (0.1514)	1.51 (0.0682)
$\mathbf{Pr}(UPC = 1)$	$(Prob > chi2)$	2.23 (0.0534)	1.48 (0.1973)	1.28 (0.1810)

Notes: LPM with clustered standard errors in parentheses. Significance: *p<0.1, **p<0.05, ***p<0.01.All regressions includes controls: child's age, gender, puberty, schooltype, migration, parents' SES, persons in household, residential status, region (East; area), parents' responder.

Chapter 4

Parental time discounting and child's smoking behavior

Ashes to ashes, time to time[1]

4.1 Introduction

Catchy phrases such as "The apple doesn't fall far from the tree." or "Like father, like son." are commonly used when analyzing the structure and characteristics of families. Fundamental attitudes and behavior patterns are most 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 and act as role models for their children. Bisin and Verdier (2000 and 2001), for instance, argue that mothers and fathers exert vertical socialization efforts through which the child adopts parental traits. Obviously, after birth, a child's preference structure is only loosely framed. Hence, parents are normally a child's first teacher. By doing so, the parental preferences picked up by the child are likely to last a lifetime. This may explain why family patterns and habits persist over multiple generations.[2]

Empirical evidence for preference and trait transmission can be found in many respects. Dohmen et al. (2011) show that risk and trust attitudes are passed on from one generation to the next.[3] Furthermore, significant correlations exist with respect to intertemporal discounting behavior. Hence, parental time preferences are positively mirrored in their offspring's preference structure (*e.g.*, Brown and

[1]This chapter based on a joint research project with Philipp Hübler.

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

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

Pol (2015), Gauly (2017)). Instead of using direct survey measures such as self-assessed patience or impulsivity, some studies proxy a person's time preference rate by focusing on saving decisions (Knowles and Postlewaite (2005), Webley and Nyhus (2006)). Indicating future orientation, a child's pension participation choice is also positively associated with the father's pension participation Gouskova, Chiteji, and Stafford (2010). Conducting an experiment, Kosse and Pfeiffer (2012 and 2013) show that especially the mother's short-run patience is significantly related to the preschool child's ability to delay gratification.

Some authors 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.[4,5] 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)). Using a discrete time hazard model, Göhlmann, Schmidt, and Tauchmann (2010) explicitly focus on smoking initiation taking Germany as an example. The results indicate that parental smoking significantly increases the child's probability of starting with tobacco consumption. Loureiro, Sanz-de-Galdeano, and Vuri (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 manage 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

[4]See, for example, Melchior et al. (2010), Chassin et al. (2008), Powell and Chaloupka (2004), Shenassa et al. (2003), Bantle and Haisken-DeNew (2002), Wickrama et al. (1999).

[5]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.

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 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 is one of those health behaviors which 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.[6] 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.[7] 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 the paper is to analyze the intergenerational transmission of smoking in more detail by considering the role of time discounting of both, the child and the parents simultaneously. 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

[6]See, for example, Kang and Ikeda (2014), Ida (2014), Scharff and Viscusi (2011), Harrison, Lau, and Rutström (2010), Ida and Goto (2009b), Khwaja, Silverman, and Sloan (2007), Reynolds et al. (2004), Ohmura, Takahashi, and Kitamura (2005), Baker, Johnson, and Bickel (2003), Odum, Madden, and Bickel (2002), Mitchell (1999), and Bickel, Odum, and Madden (1999).

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

smoking. Brown and Pol (2014) rely on data from the Household Income Labour Dynamics of Australia (HILDA). They focus on mothers and their children aged 16 to 25 years old.[8] Five waves (2002, 2003, 2004, 2006 and 2008) are selected which include information on both smoking indicators and time preference. A question about the financial planning horizon is used to proxy (long-term) time preference. A dummy variable "longer planning horizon" is constructed. It equals one if time periods of one year or more are most important to the respondent and zero otherwise. Basic offspring, mother and household characteristics are controlled for. Compiling an unbalanced panel, the final sample consists of 1901 mothers and 3167 children. Estimating a pooled probit model, they do not find any direct effects of maternal time preference on young adult smoking. After constructing 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.

Our paper 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 facets 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. (2007 and 2004) 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 area which is responsible for the planning and making of far-sighted decisions.[9] Throughout the paper, 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, L'Haridon, and

[8]The children share the same household with their respective mother.

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

Seror (2013)).[10]

Second, when it comes to the elicitation of economic preferences, measurement is not straightforward. In particular, dealing with survey data based on individual questionnaires, it is not always easy to identify high quality indicators for personal 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 and adverse health behavior. Above all, we argue that personal risk attitudes might be potentially influencing this process as well. In fact, time and risk preferences measure different economic aspects but are highly and inherently 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 one of the classic examples regarding intertemporal trade-offs. 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 active smoking participation 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 is likely to bias the effects of (parental) patience and/or impulsivity upwards.[11]

Forth, we analyze the impact of both mother and father. This allows us to investigate potential 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

[10]Except for those who try it for the first time, smoking a cigarette is accompanied by immediate pleasure in the short run. Instead, smokers normally hazard the adverse health consequences later in life due to regular tobacco consumption (long-run outlook).

[11]Despite availability, Brown and Pol (2014) do not add information about other economic preferences such as risk preferences to the analysis.

father would be highly questionable. Although the overall prevalence of smoking has declined considerably in the last decades, almost one quarter of the German population smokes nowadays (24.5%). The share of occasional and regular male smokers is still higher than the share of the female counterparts. According to the 2013 Census data, 20.3% of the female and 29.0% of the male population in Germany smokes Statisisches Bundesamt (2017).[12]

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 assume the existence 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 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 of the coefficient estimates of our discounting variables of interest. In this way, we do not only highlight the *raw* intergenerational transmission of smoking behavior but also the potential mechanism(s) of parental time discounting on child's smoking status. Unfortunately, our analysis of possible mediating influences is incomplete. Whereas 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 about the role of parenting style exerted by the mother and the father as well as their engagement in health promotion and education in the family. 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.[13] Similarly, the potential impact of peer groups especially at young age cannot be investigated properly. At the end of the paper, we will discuss these and some other limitations of our

[12]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.

[13]For instance, in Chapter 3 of this thesis the role of family communication referring to another health risk, namely weight misperception, is investigated.

study 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 considered. It allows us to take into account the importance of parental role modeling when the offspring was younger and prone to start smoking. This insight is important since it can be plausibly assumed that intergenerational transmission has already taken place at earlier stages in life (childhood/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 whether the child smokes at all, we examine how the number of cigarettes smoked per day gets affected by the intertemporal decision-making parameters of the parents.

In line with the literature, we show that children who are more impulsive and/or 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, an increase by one standard deviation in the level of parental patience reduces the child's probability of smoking by 6-7%. An increase by one standard deviation 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 transmission of smoking habits from parents to their child through role modeling. After the inclusion of the parental health behavior variables, our relevant preference parameters remain highly robust. However, parental time discounting does not have a meaningful effect on child's smoking intensity. Next to the *classic* intergenerational transmission of smoking habits, we conclude that parental time preference plays an important role for child's smoking decision, too. Hence, especially time preferences should be highly considered by researchers as well as public health authorities when dealing with health behavior formation.

The remainder of the paper is the following. Section 4.2 describes the data source and variables used. Section 4.3 presents our empirical strategy. The main 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 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 approximately 12,000 households. The SOEP provides both general household information as well as rich socio-economic data about each household member Wagner, Frick, and Schupp (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[14], questions regarding personal impulsivity and patience do not represent an inherent 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 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.[15]

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.[16] We drop observations that have missing information on the variables used for the upcoming regression analyses. Our final sample contains information on 2456 children and their respective parents (n=1739). Since we have panel data, the number of observations sums up to 5817 individuals.

All children are aged 18 years and above at the time of the interview.[17] Despite the non-availability of appropriate information of younger individuals, this circumstance is neither a disadvantage nor problematic. We argue that at these

[14]The current question on smoking behavior was introduced in 2002.

[15]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.

[16]Other family circumstances or living conditions (*e.g.*, foster parents) or children living in a children's home are not considered.

[17]Strictly speaking, only persons under the age of 18 are children. However, throughout the paper, daughters and sons are commonly entitled as offspring or children, independent of their rather advanced age. 50% of the sample are not older than 25 years. 75% are not older than 31 years.

rather advanced stages of life the intergenerational transmission of personality traits as well as smoking has already taken place. In Germany, for instance, the overall mean age of smoking initiation is around 17.8 years of age Statisisches Bundesamt (2014).[18] This is not surprising since many young people try their 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 daily smoking during adulthood.

4.2.2 Smoking

Based on the question "Do you currently smoke, be it cigarettes, a pipe or cigars?" we construct a binary variable ("current smoker") to measure the smoking status of each individual. It equals one if the respondent indicates 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. The average age of the parents in our sample is about 55 years. According to the 2013 Census data, we know that overall smoking participation considerably decreases after reaching the age of 50 years Statisisches Bundesamt (2017).

Therefore, we apply a second dummy variable, namely "ex-smoker", to capture past smoking behavior of the parents more precisely. It takes on the value 1 if the individual has smoked more than 100 cigarettes or other tobacco products in his/her life[19] and is a non-smoker throughout the sample period and 0 otherwise. According to this definition, 36% of the mothers are classified as "ex-smoker".

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

[19] The exact wording of the question is as follows: "Have you ever smoked? In other words, have you smoked more than 100 cigarettes or other tobacco products in your life?" We retrieve this retrospective information from wave 2012. Unfortunately, only a minority of the ex-smokers in our sample provided details about when exactly they quit smoking.

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.

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 requested to state their daily average of cigarettes smoked in the last week. On average, the children in our sample smoke roughly 13 cigarettes on a daily basis. Females smoke 11.5 cigarettes, whereas males have a mean cigarette consumption of almost 14 cigarettes.

4.2.3 Time discounting

The 2008 questionnaire contains two variables 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, L'Haridon, and Seror (2013)). Overall, a maximum of comparability is guaranteed since parents as well as their children independently answer exactly the same questions. First, each respondent has to rate his or her personal level of patience according to a 11-point scale. The exact wording of the corresponding 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 to proxy 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, our second variable refers to a person's self-control abilities. The respondent has to indicate his or her general level of impulsivity. Here, the wording of the question is the following: "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 can use the values in between to make your estimate."

According to the descriptive statistics, female offspring are more impulsive than male offspring (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.[20] 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.[21] Indeed, this demonstrates that the questions on general patience and impulsivity measure different aspects of intertemporal choice. Thus, a respondent's misinterpretation of the more future-oriented (long-term) aspects underlying the general question about patience can be ruled out. The wording of the question on personal impulsiveness is a basic part of the most common scales used to measure this particular personality trait.[22] 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

[20]In the experiment, choice tables with the typical price list decision format were used. The participants had to declare 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 for pay-out an additional time period of 12 months. Before the start of the experiment, the participants were informed that one of their choices would be randomly selected for payment. In the second random step one out of nine participants were actually paid by check according to the previous choice.

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

[22]Examples of common impulsivity scales are the Barratt Impulsiveness Scale: see, *e.g.*, Barratt (1959), Patton, Stanford, and Barratt (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).

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. Basically, these findings are in line with the corresponding literature (see Gauly (2017)). In comparison to those children who set up their own household, these correlations are slightly higher for children that still live together with their parents. 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 stratified by home are at least significant at the 5% level.

4.2.4 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 child are shown in Table 4.1. Offspring characteristics include basic biological information about age and gender. Moreover, we add information about the migration background and construct a home 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 of the socioeconomic status.[23] In Table 4.2, parents' descriptive statistics are reported for mothers and fathers separately. We include variables such as parental age and migration background. The highest school degree achieved of each parent serves as a proxy for the socioeconomic status of the family.

As already mentioned above, we recognize the importance of personal risk attitudes while analyzing the influence of time discounting in this particular context of smoking. 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, parental and child risk attitudes are included as additional controls.[24]

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

[24]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." Equally to 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.

To rule out that the effects of parental risk and/or time discounting are (partly) confounded with the impact of other preferences, we control for parents' altruism. It is pretty obvious that altruistic attitudes of the parents are a key element within the interaction process of a family. Therefore, our proxy equals one if a parent has indicated that it is very important to him/her to "be there for others" and zero otherwise. Above all, 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).[25]

To account for regional differences, we control for the 16 federal states in Germany. This contributes to a more detailed geographical segmentation of Germany and accounts for regional confounders. In addition, we control for time trends by adding wave dummies. All in all, this allows us to capture common trends behind changes in smoking participation. In the past, rising health consciousness and steadily increasing taxes on cigarettes are supposed to be two factors that have contributed to an overall reduction in tobacco consumption in Germany. By adding states as well as time dummies, we further control for the implementation or expansion of different anti-tobacco policies (*e.g.*, smoking bans) that vary across states and/or over time.

In order to explain the influence of parental time preference and impulsivity as best as possible, it is essential to look at relevant behaviors of the parents which might act as mediating factors between their 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 have a look at adverse health behaviors such as parental smoking status to further investigate the impact of role modeling and eased access to cigarettes as well as alcohol consumption. The latter equals one if a 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 a person's healthy lifestyle. The variable is set to a value of 1 if a mother or a father follows a health-conscious diet "very much" or "much" and zero otherwise. Second, we construct a binary variable representing physical activity. It equals one if the individual takes part in active sports "daily" or "at least once a week".

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

Table 4.1: Summary statistics - *children*

	Children (N=5,817)				**Daughters** (N=2,788)				**Sons** (N=3,029)			
	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
Explanatory variables												
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 4.2: Summary statistics - *parents*

	Mother				**Father**			
Explanatory variables	*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[3]	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[4] (log)	10.603	0.550	8.170	13.409	10.609	0.558	5.338	13.409

Notes: [1] Information from wave 2008; [2] Information from wave 2012; [3] Self-generated variable with information from wave 2012; [4] Log annual household income lagged by one year; number of parents=1,739.

4.3 Empirical strategy

The empirical analysis is based on three steps. First, we apply a linear probability model (LPM) to estimate the direct effect of parental time discounting on child's smoking status. In step 2, we additionally control for the parental health behaviors mentioned above. This allows us to clarify 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 investigate the intensive margin of child smoking.

4.3.1 Regression models

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

$$S_{it} = \beta_0 + \beta_1 I^p_{it} + \beta_2 P^p_{it} + \beta_3 I_{it} + \beta_4 P_{it} + \beta_5 X^{'}_{it} + \lambda_t + \alpha_i + u_{it} \tag{4.1}$$

where i represents the child and t the year of observation. The superscript p indexes the parents. S_{it} is equal to 1 if the child smokes and zero otherwise. I^p_{it} includes information on mother's and father's general level of impulsivity, whereas P^p_{it} 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 4.2.4). Whereas the regression in step 1 is run without any health variables of the parents, these are included as additional controls 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.

Generalized least squares (GLS) with random effects are used to estimate the linear probability model.[26] All time discounting and risk preference variables that enter Equation (4.1) are standardized. Standard errors are clustered at the family level since we have families with one or more children. The vectors of parameters β_1 and β_2 are of particular interest. They measure how a mother's and father's 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

[26]Considering the well-known limitations of the linear probability model, we compare the results to a panel probit estimation in Section 4.4.3.

parental time discounting and child's smoking intensity (step 3). This regression technique 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 (4.1) with a probit model and obtain the average marginal effects of our variables of interest. These estimation results provide a built-in robustness check of our LPM. Second, the number of smoked cigarettes is estimated with a truncated regression. Here, all non-smokers are dropped since their tobacco consumption equals zero.[27] We prefer the two-part model over the standard Tobit model since it models and estimates the decision to smoke and the decision of how many cigarettes to smoke separately. This allows a higher degree of flexibility. For instance, the determinants of the probability of being a smoker and the determinants of smoking intensity do not have to show the same signs. Moreover, both parts of the model do not have 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 could also be interpreted as a joint decision instead of 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 4.4.3, we also come up with a statistical test that supports our choice in favor of the two-part model.

4.3.2 Specifications and mediation analysis

Our empirical strategy is focused on the relationship between parental time discounting and child's smoking behavior. However, we are curious to see if there are mechanisms through which the discounting variables might have an indirect impact on our dependent variable(s). The influence of parental smoking behavior is of special interest in our context. At first sight, excluding all parental health behaviors from our baseline regression model seems to be little convincing. Previous literature has shown that a positive transmission of smoking habits from parents to their offspring exists (*e.g.*, Loureiro, Sanz-de-Galdeano, and Vuri (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 holds true for all variables that are associated with

[27]For both equations, we use Roodman's .cmp in Stata (see Roodman (2009)). It allows us to make use of the panel structure of the data and to keep estimating random effects models. Moreover, it enables us to obtain clustered standard errors.

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 extensively influenced by their parents' preferences (see Gauly (2017)).

Being aware of such potential endogeneity concerns, we decide to run several specifications of our econometric model which gradually add more (problematic) controls. Starting off with a fully specified model would mask the role of existing mechanisms driving the relationship under investigation. At the very beginning, we include parents' preferences along with plausibly exogenous control variables such as age and child's sex. Expanding the regression model, we add child's time discounting variables and the personal level of risk attitude. Following this, we do control for educational attainment for both parents and the child. Higher education is declared 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 since 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 consider 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 is finished for all individuals by the end of our analysis period. This fact should at least dampen the endogeneity concerns regarding the inclusion of education. Same considerations are largely true with respect to offspring's personal income since it is a consequence of the educational level achieved. Therefore, it represents an indirect result of child's time discounting. However, our stepwise approach copes with this issue.

Addressing the health variables, time preference and/or impulsivity are likely to influence different (health) behaviors (*e.g.*, consumption of tobacco products). Given our data, we select four 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 Section 4.2.4 for more details on the exact variable definitions). Smoking and alcohol consumption are both exam-

ples of rather unhealthy lifestyles that reflect negative health investments.[28] The appraisal of healthy nutrition and engagement in regular physical activity act as proxies for beneficial investments in health capital. The attitude towards a healthy diet is supposed to capture healthy eating habits in general. Thus, a person who puts weight on healthy nutrition is unlikely to (excessively) consume bad foods such as junk food or soft drinks. It can 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. As opposed to low self-control, future orientation is a key component for 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 personal discounting behavior and smoking participation was already given in the introduction section of this paper. Hence, in case of a parental non-smoker, 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, Smith, and Bogin (2004) and Scharff (2009)).

In fact, we are interested in how the coefficients of parental time discounting vary with the inclusion of these designated mediating factors. Possible changes after the inclusion of parental smoking are of particular interest since it is supposed to be a major determinant of child smoking anyway. On the one hand, this could result in a meaningful mechanism which leads to 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. So, we could infer that parental time discounting has an influence on child's health/smoking behavior (mainly) through one or even more parental health patterns. On the other hand, although a parental health pattern shows a significant effect, the coefficients of interest could remain basically the same. In this case, we would see no reason not to control for

[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.

these variables. Thus, we would have identified a meaningful influence on child's smoking status that does not 'vaporise' our previously estimated time discounting effects Baron and Kenny (1986). Hence, our suspected control problem will *not* turn out to be a seriously *bad* problem.

4.4 Results

4.4.1 Parental time discounting and child's smoking participation

Results from Equation (4.1) are presented in Table 4.3.[29] Column (1) shows that father's patience is negatively associated with child's probability of being a smoker. The coefficient of mother's patience is weakly significant at the 10% level. After controlling for child's time discounting and risk 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 result of the F-test on joint significance, 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.

[29]To underpin our 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 since no significant differences across individuals exist. We can reject the null hypothesis since $Prob > chi2 = 0.0000$. 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 where appropriate. In each case, random effects 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 since 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 child'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 supports the extensive evidence on the education gradient in health and health-related behaviors (see, *e.g.*, Conti, Heckman, and Urzua (2010)).

At first glance, we confirm the findings of previous studies regarding the association between individual time discounting and smoking (*intra*personal context). An increase by one standard deviation in child's impulsivity leads to a 2.8 percentage points or 10% increase (according to the mean) in the likelihood of smoking. Conversely, an increase by one standard deviation 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.

Above all, 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 significance, while patience has a preventative effect. An increase by one standard deviation in maternal patience reduces the likelihood of smoking by 2.1 percentage points. This amounts to a reduction of 7 %. Regarding the father, both components of time discounting are significant. An one unit increase in paternal impulsivity has a negative impact of 1.8 percentage points. With respect to his long-term time preference, we find a prevention effect, too. 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 since they imply a reduction of approximately 6-7 %, respectively.[31]

Hence, these results suggest that especially parental future orientation is able to prevent the offspring from exerting adverse health behaviors such as smoking (prevention effect). In contrast, the effect of father's impulsivity might not be that intuitive at first sight and, therefore, needs some additional remarks. By default, expecting the same sign as in the *intra*personal context might by delusive. The intrapersonal impact of impulsivity does not necessarily imply getting the same results when turning to the *inter*personal context. We could think about the interaction between impulsivity and human relations. In the family context, there could be uncertainty about parental behavior which might explain why the coefficient of paternal impulsivity has a negative sign. We argue that decision-

[31]To check the robustness of our results, we re-estimate the model using dichotomous versions of the original time as well as risk preference measures. Individuals are classified as being patient, impulsive and willing to take risks if they respond a value greater than the median or mean of the relevant survey question, respectively. The regression estimates for both alternative specifications yield qualitatively similar results. Moreover, we extend our analysis to 4 observation periods by adding wave 2012. Although we still get similar results, we do not want to push our assumption on stable preferences to the limit.

Table 4.3: Parental time discounting and child's smoking participation

		Dep. var.: child's smoking status **Pr**(*child smokes currently* = 1)					
		(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.

making and actions taken by impulsive parents might be 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.*, engage in smoking) that might cause trouble at home. They want to avoid negative attention since following such a lifestyle is likely to provoke immediate as well as ambiguous *reactions* of the parent(s). Regarding the influence of parental impulsivity, we call this the 'slap-effect'.

As pointing out the possible role of risk attitudes at the outset, we have a brief comment on the impact of risk preferences as well. With respect to the influence of individual risk attitudes, we find a significant intrapersonal effect. Hence, an increase by one standard deviation 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 (4.1) separately for daughters and sons. Results are shown in columns (5) and (6) of Table 4.3. 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. An increase by one standard deviation 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. An increase by one standard deviation of paternal impulsivity (patience) reduces the likelihood of smoking for men by 3.1 (2.5) percentage points. Once more, this highlights the significant role of the father in this context.

In addition to gender, Table 4.6 of Appendix A replicates the regression from column (4) of Table 4.3 and stratifies by child's home. When the child lives together with the parents, mother's patience is associated with a significant decrease in child's smoking probability. The effect size is the same for children who no longer share the same household with their parents. Interestingly, father's time discounting reduces smoking only for those who set up their own household. Table 4.7 of Appendix A replicates the regression from column (4) of Table 4.3

[32]As a robustness check, we replace this 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 about -0.017 and has a t-statistic of 1.55.

and stratifies by different age groups. Especially parental patience as well as child's preferences show the expected signs and are significant across almost all specifications.

4.4.2 Role of parental health behaviors

Table 4.4 presents the estimation results after adding parental smoking behavior and the other health variables to the initial equation. Column (1) replicates the results from the specification without any 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 as well as highly significant relationship between parental smoking habits and child's smoking status. Furthermore, the economic significance is huge. If the mother is a current smoker, the likelihood of child smoking increases by more than 13 percentage points (or 45%). In case of an ex-smoker, the increase is about 5 percentage points (or 17%). Regarding the father, the size of the coefficients as well as the levels of significance are very 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. Our main findings remain basically unchanged. Parental smoking is by far the most meaningful health behavior. With the exception of father's healthy lifestyle, all other health variables are not statistically significant.[34] Comparing it to the estimates from the baseline regression, the changes in coefficient size and significance levels are negligible. However, with respect to the influence of mother's patience, there may be a partial mediation effect of moderate size. But, in fact, it is not enough to infer that parental smoking is a true mediator of parental time discounting in this context. Another valuable insight is that the previously addressed bad control problem is in fact not that serious. In columns (4) and (5), we stratify by gender. Parental smoking behavior shows same-sex as well as slightly weaker cross-sex effects. Although the coefficient of maternal patience is no longer significant for sons, the effects of parental time discounting show a similar pattern as in Table 4.3.

We conclude that parental smoking status, although not representing a fully convincing mediator, is definitely a meaningful determinant of child's smoking

[34]Joint significance tests show that all four parental health patterns together are statistically significant for the mother and the father, respectively. Excluding parental smoking, the variables on alcohol, nutrition and physical activity are jointly significant for fathers but not for mothers.

status. Hence, especially parental smoking should be controlled for. Overall, the results confirm the findings from the specifications without parental health variables. The coefficients of interest change only marginally in size. No matter if parental smoking alone or all health investments together are incorporated, mother's patience is estimated to reduce child smoking by around 6%. Paternal impulsivity reduces the likelihood of smoking by 7%. His patience shows a diminishing effect of about 6%.

4.4.3 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. Now, we are further interested in how parental impulsivity and patience influence the actual demand for tobacco products of the offspring. Child's smoking intensity is measured as the number of cigarettes smoked per day, conditional on whether the offspring smokes at all. A two-part model is used to estimate child's smoking participation and the relevant consumption level.[35]

Results from the two-part model are reported in Table 4.5. In column (1), the average marginal effects (AMEs) of the probit regression (extensive margin) are in line with the previous findings from the LPM. 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 with a mother (father) who is a current smoker smoke, on average, 1.6 (2.1) cigarettes more than children where the parents are strict non-smokers. A paternal ex-smoker is associated with an increase in child's tobacco consumption of about 2 cigarettes. The effect of former maternal smokers is insignificant. Interestingly, the stratification by gender yields substantial cross-sex effects. This is contrary to the findings in Table 4.4 where we identified same-sex effects, too. As a side note, an increase by one standard deviation in child's impulsivity is associated with a rise in consumption of 0.5 cigarettes more per day. This overall effect is mainly driven by sons as can be seen in column (4).

We infer that parental time discounting is primarily relevant for child's smoking participation. Here, especially parental future orientation plays a significant role. With respect to smoking intensity, basic role modeling seems to be the main factor.

[35]Testing the Tobit model versus the two-part model, we can reject the null hypothesis that the Tobit model is appropriate at the 1% significance level (see Smith and Brame (2003)).

Table 4.4: Role of parental health behaviors

Dep. var.:		**parental smoking**		**all behaviors**		
child's smoking status					Daughters	Sons
Pr(*child smokes currently* = 1)		(1)	(2)	(3)	(4)	(5)
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 4.3. 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.

Table 4.5: Extensive and intensive margin of smoking

		Participation	**Intensity**		
				Daughters	Sons
		(1)	(2)	(3)	(4)
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 with probit regression for smoking participation (Dep. var.: $\mathbf{Pr}(\textit{child smokes currently} = 1)$, full sample) and truncated regression for smoking intensity (Dep. var.: $\mathbf{Pr}(\textit{child's cigarette consumption}|S_{it} = 1)$, subsample of current smokers). Both regressions are run separately using the Stata .cmp. 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.

4.5 Discussion and conclusion

The main purpose of the paper was to analyze the link between parental time discounting and child's likelihood of being a smoker. We used self-assessed impulsivity and patience as meaningful proxies for self-control and time preference, respectively. First, we confirm previous evidence on the association between individual time discounting and smoking (*intra*personal context). That is, individuals with lower impulsivity and/or higher levels of patience are less likely to smoke than people that show the opposite characteristics.

More importantly, our results show that the time discounting variables of the parents - especially time preference - have significant direct effects on the likelihood of child smoking (*inter*personal context). Increasing patience of mothers as well as fathers reduces the likelihood of smoking by around 6-7%. Hence, future orientation of parents 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 adverse health consequences of smoking. With respect to father's self-control, increasing impulsivity reduces the likelihood of smoking by roughly 7%. Interpreting this effect is not straightforward. We argue that it is hard for children with impulsive parents (fathers) to properly anticipate their actions and/or consequences after they have learned about specific child behaviors. Hence, those children may act with caution and rather think twice before they indulge in the consumption of health deteriorating goods such as cigarettes.

The direct effects of parental time discounting do not vanish after including potential mediating factors such as socioeconomic outcomes and different health behaviors. Controlling for parental smoking, our findings are in line with results from the previous literature concerning the transmission of smoking habits from parents to their children. Hence, we confirm the well known positive relationship between parental and child smoking. However, the main results obtained from the basic regressions without mediating health factors remain firmly stable. Overall, parental time discounting as well as parental smoking patterns are both significantly related to child's smoking participation. Thus, parental time discounting seems to play a role in the intergenerational transmission of smoking behavior. In contrast, the results from the two-part model reveal that parental time discounting is not associated with child's smoking intensity.

Our findings provide further evidence that the influence of the father is sub-

stantial in this context and should not be ignored. Hence, focusing on mothers only (*e.g.*, Brown and Pol (2014)) may result in potentially misleading inference. Stratifying by gender, effects of (parental) time discounting differ in sex. Whereas maternal patience shows an effect for both daughters and sons, father's discounting variables seem to be relevant only for sons. Since we have data on adult children and additionally control for offspring that either still live with at least one biological parent or live in an own household, our findings are thoroughly generalizable to the adult population in Germany. Moreover, our findings encourage future studies to control for both, risk preference as well as time discounting measures (if available) when analyzing the mechanisms of health (behavior) transmission from one generation to the other.

However, we have to admit some limitations of our paper. 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 persistence of the direct relationship between parental time discounting and child smoking. However, there may exist other latent channels through which the direct effects of parental discounting could be absorbed. We suggest that parenting style might be such a candidate variable. Children of parents that care about good (child) health are unlikely to smoke since their parents are likely to properly invest in their children's health capital. This might work especially through appropriate health education and communication within the family. Unfortunately, we are not able to fully 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 restrictions, we cannot address systematically 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 a heritability of delay discounting at up to 50% (Anokhin et al. (2011)).[36] But, our sample lacks considerable information on twins and/or adoptees to examine the role of parental genetics in more detail.

Regardless of whether mother and father live together or not, we only include those children where we have the appropriate information on both parents. First,

[36]MacKillop (2013) provides a review of the heritability of discounting behavior.

we focus on both parents to see if neglecting one parent (*e.g.*, the father) is a clever idea. Obviously, it is not. Second, we do not have full information on a single parent's living circumstances since these factors are hardly observable. For instance, we do not know when exactly parents split up or for how long she actually is a single mother. Furthermore, we are not aware of the actual influence of a new partner. Hence, we cannot observe if the child of a single parent still has regular contact with the other biological parent.

The assumption of stable preferences even over this relatively short period of time may be questionable, too. In order to set up a panel, we have to make this assumption. Survey questions on impulsivity and patience asked in 2008 are not part of the standard individual questionnaires from waves 2006 and 2010. However, we get qualitatively similar results even after running a simple cross-section analysis with data from wave 2008. Finally, we cannot completely rule out an endogeneity bias. Dealing with preferences, it is always a tough challenge to identify causal effects. However, we think that our comparative approach by gradually adding the *bad* control variables is straightforward. In fact, results with and without these explanatory variables do not differ substantially.

Undoubtedly, smoking is (still) one of the most prominent public health concerns with respect to preventable health risks. Our findings may provide further insights for public health authorities concerning the prevalence of smoking. However, since our effects can hardly be interpreted as causal, we are reluctant to give generous policy implications. In line with the literature, individual time discounting is related to the decision to smoke or not 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 reader should keep in mind that the inclusion of unconsidered mechanisms (*e.g.*, family communication) could fade away the direct effects of parental impulsivity and/or patience.

Overall, it is important to brief parents about their powerful influence as role model and primary health educator. This information is crucial if public health services intend to prevent people from starting to smoke or to help them quit tobacco consumption. The support of smoking cessation efforts in adults (the next parent generation) is another step. Improving self-control techniques may help individuals to abstain from smoking successfully. Especially future orientation seems to be a key 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 preference in the intergenerational context of smoking in order to provide more target-oriented advice for public health authorities.

4.6 Appendix

Table 4.6: Effect heterogeneity - *home*

		Dep. var.: child's smoking status $\mathbf{Pr}(\textit{child smokes currently} = 1)$		
		(1)	(2) With parents	(3) 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 4.3. 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), and control variables parents (age, migration background, altruism, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

Table 4.7: Effect heterogeneity - *age*

		Dep. var.: child's smoking status $\mathbf{Pr}(\textit{child smokes currently} = 1)$						
		(1)	(2) Under 20	(3) Under 25	(4) Under 30	(5) Under 35	(6) Under 40	(7) 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 4.3. Impulsivity, patience and risk are measured in standard deviations. All regressions include wave and state dummies, control variables child (gender, migration status, home indicator, education, income), and control variables parents (age, migration background, altruism, education). Significance: *p<0.1, **p<0.05, ***p<0.01.

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