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An Investigation into the Psychosocial Background of Obesity: The Role of Adverse Childhood Experiences, Common Mental Health Disorders and Socioeconomic Status

Psychosoziale Prädiktoren von Adipositas: die Rolle von Kindheitsbelastungen, Depression, Angststörungen und sozioökonomischem Status

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List of Abbreviations and Symbols

ACE	Adverse Childhood Experience
BMI	Body Mass Index
ß	Regression coefficient
CI	Confidence Interval
CQ	Childhood Questionnaire
DALY	Disability-adjusted Life-years
HPA	Hypothalamic-pituitary-adrenal
MHO	Metabolically Healthy Obesity
MSBI	Mainz Structured Biographical Interview
MUO	Metabolically Unhealthy Obesity
NAFDL	Non-alcoholic Fatty Liver Disease
NASH	Non-alcoholic Steatohepatitis
p	P-value
SCL-27-plus	Symptom-Check-List-27-plus
$se_ß$	Standard Error
SES	Socioeconomic Status
t	T-value
WHO	World Health Organization
YLD	Years Lived with Disability

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1 Introduction

For the first time in history, more of the world's population is overweight than underweight. Apart from a few exceptions in sub-Saharan Africa and Asia, obesity has reached epidemic proportions in all countries and has surpassed malnourishment as a major health concern both for the individuals affected and for society as a whole (WHO, 2018). Though most of human history has been characterized by food shortages and malnutrition, through industrialization and urbanization a surplus of food has become available and, combined with an increase in sedentary lifestyle, has triggered the trend of widespread obesity (Caballero, 2007). Once a first world problem, the most dramatic increases in obesity since the end of the 20th century have occurred in developing countries. Currently, 1.9 billion adults worldwide are overweight and 650 million are obese, comprising 39% and 13% of all adults, respectively (WHO, 2018).

Although the adverse health effects of obesity are widely acknowledged, the causes and the background are far more ambiguous. In view of the rapidly rising prevalence of obesity and lack of successful treatment options, obesity prevention is more important than ever. Prevention strategies, however, tend to concentrate solely on decreasing dietary intake and increasing physical activity, despite evidence indicating that it is a combination of individual predisposition toward weight gain and psychosocial factors that tip the scales toward overweight and obesity. Psychosocial factors that facilitate excess weight gain include a reciprocal association with socioeconomic status, a bidirectional link with common mental disorders, and a correlation with adverse childhood experiences. These complex and multifactorial psychosocial determinants of overweight and obesity are at the center of this thesis.

Just as the causes of obesity lie both in individual predisposition and societal structure, the consequences of obesity affect both the individual, through adverse physiological and psychological outcomes, and society as a whole, directly through medical expenses and indirectly through loss of productivity (Yates et al., 2016). The prevalence of overweight and obesity in both industrialized and developing countries over the past decades indicate that its individual and societal relevance will only continue to grow. Thus, the psychosocial background of obesity is a critical topic for further research, which may shed light on more effective strategies for prevention.

2 Literature Review

2.1 *Implications of the Obesity Epidemic*

In light of the global increase in obesity and its far-reaching consequences, it is important to gain a better understanding of the multifaceted background of obesity. The following chapters represent an overview of the current literature, highlighting the medical consequences and etiology of obesity before detailing the psychosocial factors fostering obesity. The focus of this thesis is on the association of overweight and obesity with socioeconomic status, depressive disorders, anxiety disorders, and adverse childhood experiences. The objective is to shed light on the psychosocial background of obesity and in doing so, to help us gain a better understanding of the psychological and environmental factors that contribute to obesity.

2.1.1 *Epidemiology of Obesity*

Obesity is defined as an accumulation of over 30% body fat in women and 20% body fat in men and is a significant risk factor for many non-communicable diseases and associated with a higher mortality rate. Moreover, obesity has itself been defined as a disease that can cause premature disability and death. The Body Mass Index (BMI) is a measurement used to approximate an individual's percentage of adipose tissue, and is calculated by dividing body weight, in kilograms, by height, in meters squared (Herpertz and Zipfel, 2018). The significance of the obesity epidemic is evident in epidemiological data, the prevalence of obesity has risen in every single county around the world—since 1975, the worldwide prevalence of obesity has nearly tripled in adults and risen tenfold in children and adolescents (N. C. D. Risk Factor Collaboration, 2017).

The human propensity toward weight gain is understandable from an evolutionary perspective. In times of food scarcity, it was those who could outlast long periods of famine who were able to reproduce. However, the current obesogenic environment—characterized by a sedentary lifestyle and large quantities of processed, high-caloric foods—have caused these traits to become counterproductive, the long-term energy imbalance causing many people around the world to gain excess weight (Blüher, 2019). It is no longer merely developed countries that are affected by the obesity epidemic, the prevalence of obesity has risen in every single country around the world. The adverse effects of obesity may be particularly far-reaching in low-income countries as they grapple with what has been identified by World Health Organization and others as the 'double burden of disease'—the perpetuation of infectious diseases and malnutrition, on the one hand, and the rise of noncommunicable diseases, on the other (WHO, 2018, Popkin et al., 2020).

The German population has easy access to inexpensive, high caloric foods and drinks. Evolutionarily, however, humans are far better equipped to deal with a lack of food than with an abundance thereof. Thus, in Germany today, 58.2% of the entire population are either

overweight (BMI ≥25kg/m²) or obese (BMI ≥30kg/m²). Of these, 20.8% are obese: 15.1% are obesity class I (BMI 30-35kg/m²), 4.1% are obesity class II (BMI 35-40kg/m²), and 1.5% are obesity class III (BMI ≥40kg/m²) (Ernährung, 2008). Approximately 15% of children and young adults in Germany are overweight, and of these 6.3% are obese (Zündorf, 2006). This trend of obesity in young people is particularly concerning as a possible predictor for the future of the obesity epidemic, considering that around half of the overweight children and the majority of overweight adolescents become overweight as adults (Weaver, 2017). Furthermore, the high levels of obesity in Germany generate expenditures between 2.7 and 5.7 million Euros every year—both directly, through medical costs, and indirectly, through occupational disability (Herpertz et al., 2011).

2.1.2 *Biological Determinants of Obesity*

A long-term energy imbalance between calorie intake, on the one hand, and metabolism, energy expenditure, and physical activity, on the other hand, lead to excess weight gain. Yet the interplay among the factors that lead to this imbalance are complex, and not conclusively explained—a number of interconnected biological, environmental, and psychosocial factors predispose an individual toward becoming overweight or obese. Due to an obesogenic environment, characterized by an increase in calorie dense foods and sedentary lifestyle, more people are overweight today than ever before. This section offers a brief overview of some biological determinants of obesity, including genetics, neuroendocrinological factors, metabolic imbalance, and gut dysbiosis.

The extent to which the imbalance between calorie intake and physical activity results in excess weight gain is largely **hereditary**. It is estimated that around 40-70% of a person's BMI can be attributed to genetics (Zaitlen et al., 2013). To date over 140 gene loci have been discovered that influence adiposity traits (Fall et al., 2017). However, save for a few notable exceptions, such as Prader-Willi Syndrome, the influence of specific genes on the development of obesity is not yet well understood (Hinney et al., 2010). Despite a plethora of evidence indicating that obesity, or at least the disposition toward obesity, is heritable, the genetic variants discovered thus far explain only 5% of BMI variation (Franks and McCarthy, 2016). This indicates on the one hand, that there is need for further research into the heritability of obesity and, on the other hand, that the explanation for BMI variation will not be found solely in an individual's genes.

Neuroendocrinological factors also play a role in weight gain, with the central nervous system playing an important role in energy and glucose homeostasis (Timper and Brüning, 2017). In humans, complex neuronal circuits in the hypothalamus and brain stem are in charge of appetite and satiety. These circuits, however, are subject to modulation by higher brain functions, such as the limbic region and the cerebral cortex (Ahima and Antwi, 2008). In other words, behavioral factors and our internal reward system can override our

sense of satiety, causing us to eat more than we need to. Thus rendering therapeutic solutions that attempt to directly regulate the hypothalamic circuit useless and explaining why the pharmacological treatment options for obesity are still extremely limited (Zwaan et al., 2011). Furthermore, the changes in biological mechanisms, caused by excess adipose tissue, may themselves prevent effective weight loss and even perpetuate weight gain. Underscoring the fact that short term behavioral or medical interventions will be insufficient to reverse the far-reaching effects of overweight and obesity (Blüher, 2019).

A relatively recent area of research into the etiology of obesity can be found in the human gut—more precisely the reduction in gut microbiome diversity. **Gut dysbiosis** has been consistently associated with several metabolic disorders, including obesity (Cunningham et al., 2021), type II diabetes, and non-alcoholic fatty liver disease (Vallianou et al., 2019). These discoveries may offer key insights for future individualized therapeutic solutions (Cunningham et al., 2021, Wei et al., 2021). However, there are currently still many uncertainties in the role that gut dysbiosis may play in the association between an individual's genetics and the development of obesity (Vallianou et al., 2019).

Research into the biological factors leading to excess weight gain is extensive and this chapter can offer only a brief excerpt of this field of study, nevertheless, these examples demonstrate that our biology plays an important role in fostering obesity.

2.1.3 *Medical Consequences of Obesity*

Obesity is a major modifiable risk factor for many non-communicable diseases, affecting almost all regions of the body. Understanding obesity as an inflammatory disease, defined by an increase in cytokines released by adipocytes, helps explain its far-reaching medical consequences—including, but not limited to, the cardiovascular, metabolic, musculoskeletal, and pulmonary systems (Finer, 2015). Moreover, the risk for certain types of cancer is elevated (Finer, 2015, Friedenreich et al., 2021). In Europe, one in seven premature deaths can be attributed to overweight and obesity—making it one of the leading causes of premature death, second only to tobacco (Global B. M. I. Mortality Collaboration et al., 2016). In discussing the medical consequences, it is important to distinguish between overweight and obese individuals, because the relationship between BMI and mortality is curvilinear—the higher an individual's BMI, the greater the risk of disease and mortality (Bray et al., 2017). A u-shaped curve has been used to describe the link between the two, with both a low and a high BMI associated with a higher mortality (Finer, 2015). The optimal BMI in regard to all-cause mortality is between 22.5 and 25kg/m^2—above 25kg/m^2 mortality rises by 30% for every extra 5 BMI points (Bray et al., 2017).

The medical consequences of obesity have long been in the public eye. When it was first published in 2002, the **Obesity Paradox**, which claimed that overweight individuals live longer than normal weight individuals, caused debate both in the media and medical

community (Gruberg et al., 2002). Despite the attention it received, these results were negated by two further studies that showed that the data was skewed. First, a 2018 study published in JAMA cardiology (Khan et al., 2018), reported that, although longevity was similar in overweight and normal weight individuals, overweight individuals (defined as BMI ≥25kg/m² and <30kg/m²) had a higher risk of developing cardiovascular diseases, were diagnosed at a younger age, and lived more years of their life with cardiovascular disease morbidity. In obese individuals (defined as BMI ≥30kg/m²), higher rates of both morbidity and mortality were confirmed. According to the authors of this study, the previously published positive effects of overweight on longevity could be explained by the fact that individuals with a higher BMI were often diagnosed earlier in life, thus living more years with the diagnosis of a cardiovascular disease without indeed having a lower rate of mortality. Second, a comprehensive study from The Global BMI Mortality Collaboration in 2016 debunked large parts of the Obesity Paradox by adjusting for smoking and chronic diseases. Almost 4 million people were identified who had never smoked and had no pre-existing chronic diseases, furthermore, those who died within the first five years of the study were also excluded. Without the effects of smoking and chronic disease, both of which can cause a decrease in BMI, overweight and all grades of obesity were associated with a higher rate of all-cause mortality. Moreover, results showed that the risks associated with overweight and obesity were particularly high in men and young people. Interestingly, the study also found that people at the lower end (18.5-20kg/m²) of what the World Health Organization categorizes as "normal" BMI (18.5-25kg/m²) had a similar all-cause mortality as people in the overweight category. Nonetheless, the finding of a curvilinear relationship between BMI and morbidity and mortality remained—the higher an individual's BMI the greater the risk of disease and the shorter the life expectancy (Global B. M. I. Mortality Collaboration et al., 2016).

The adverse health effects of obesity are twofold—first, the direct effects of excess adipose tissue on the organism, and second, the metabolic changes caused by an increase of adipocytes both in number (hyperplasia) and size (hypertrophy) (Bray et al., 2017). Large amounts of excess weight affect the musculoskeletal and respiratory systems and often go hand in hand with metabolic changes. Excess weight impacts the **ventilatory system** causing dyspnea and breathlessness. These symptoms are a result of both the increased weight compressing the chest wall, thus decreasing the end-expiratory lung volume, and the higher ventilatory demand created by a greater body weight, which is met by an increased breathing rate especially when exercising (Finer, 2015). Obstructive sleep apnea syndrome is also observed in approximately 30% of obese individuals and more frequently affects men. It is caused both through mechanical changes such as accumulation of fat in the pharynx and a decrease in muscle tone (Bray et al., 2018). The **musculoskeletal system** is similarly affected, with osteoarthritis more commonly observed in overweight and obese individuals,

affecting both weight bearing and non-weight bearing joints, suggesting that the cause is a combination of both increased pressure on joints and metabolic changes (Finer, 2015).

The pathogenic products produced by adipocytes and the resulting metabolic changes systematically affect the entire body making obesity a risk factor for, among others, cardiovascular diseases, type II diabetes, gastrointestinal diseases and cancers (Bray, 2004). Obesity increases the risk of **hypertension, stroke and cardiovascular disease**. Approximately one third of obese adults suffer from hypertension (compared to about one sixth of normal weight individuals) and are thus at a higher risk of developing congestive heart failure. Several factors contribute to the increased risk, including metabolic imbalances in lipids, hemostasis, and insulin that contribute to a structural change in the vascular system and the heart. Overall, cardiovascular mortality attributes to approximately half of the excess mortality of obesity (Finer, 2015).

The association between obesity, in particular an increase in visceral fat, and **type II diabetes** is particularly striking—75% of type II diabetics are overweight or obese (Finer, 2015). The effects of obesity on type II diabetes have been likened to the effects of tobacco on lung cancer (Yach et al., 2006)—for every 1kg/m^2 increase in BMI above 22kg/m^2 the risk of diabetes rises by 25%. Overall survival is reduced by a median of two to four years for individuals with a BMI of 30-35 kg/m^2 and by eight to ten years for those with a BMI of 40-45 kg/m^2 (Finer, 2015). However for individuals at risk of developing type II diabetes, this strong correlation can have a positive effect—even modest weight loss (of approximately 5-10%) can meaningfully improve health outcomes (Hallberg et al., 2019, Ryan and Yockey, 2017).

Furthermore, obesity negatively affects the **gastrointestinal system** especially the biliary system and the liver. Obese individuals are at a higher risk of developing gall-stones due to an excess of body fat which produces a surplus of cholesterol that must be secreted through the biliary tree (Finer, 2015). Even more deleterious is the effect of obesity on the liver. Akshintala et al. (Akshintala et al., 2019) describe the liver as "the 'mirror' of metabolic health", since the amount of fat in the liver reflects insulin resistance and adipocyte disfunction. In developed countries, parallel to the rise of obesity, there has been an increase in non-alcoholic fatty liver disease (NAFLD), characterized by an increase of liver fat. NAFLD is currently the most common chronic liver disease in developed countries. Around 40-50% of the obese population has NAFLD, and among obese individuals with type II diabetes this number reaches up to 70%. Nevertheless, NAFDL is most likely widely underdiagnosed due to the scarcity of reliable non-invasive diagnostic methods. A more severe form of liver disease is non-alcoholic steatohepatitis (NASH), characterized by lobular inflammation, necrosis and often fibrosis, which can lead to cirrhosis and eventually to hepatocellular carcinoma (Akshintala et al., 2019, Saitta et al., 2019). In developed countries NASH is a leading indication for liver transplantation, second only to hepatitis C. Moreover, if the

current trend in obesity remains, it is predicted to become the most common cause of end stage liver failure within the next decade (Akshintala et al., 2019).

Obesity is also associated with certain types of **cancers**, especially in women (Argyrakopoulou et al., 2021). In both sexes, the risk of colorectal, esophagus, gall bladder, renal, and liver cancers among others are elevated. Additionally, there is a higher risk of breast and endometrial cancer in women, due to the hormonal activity of excess adipose tissue, and a higher risk of prostate cancer in men (Finer, 2015). In 2012 an estimated 3.9% of all cancers worldwide could be causally attributed to overweight and obesity and this number is expected to continue to rise in future. This disproportionally affects women, who, compared to men, had more than double the number of cancer cases attributable to excess body weight (Sung et al., 2019, Argyrakopoulou et al., 2021).

Obesity is defined as an element of the **metabolic syndrome**; a co-occurrence of several cardiovascular risk factors, including abdominal obesity, dyslipidemia, insulin resistance, and hypertension. Notably, the distribution of body fat plays an important role as an indicator of health risk. In comparison to peripheral patterns of fat distribution as well as increased subcutaneous fat, excess visceral and intra-abdominal fat are more metabolically active and thus associated with a higher risk for insulin resistance. One of the drawbacks of the BMI as a measurement is that it is unable to distinguish between different types of body fat distribution (Jayedi et al., 2020) and, moreover, cannot differentiate between fat and lean tissue (Engin, 2017).

BMI is a simple, fast, and inexpensive measurement that can be easily employed in a clinical setting. Nevertheless, Tomiyama et al. discourage the use of BMI as the principal measure of metabolic health. According to the authors, the major drawback of relying entirely on **BMI as a proxy for health** is that BMI categories cannot accurately reflect the complex association between excess weight and health outcomes. In their study, examining the association of BMI and cardiometabolic health, they found that, while the percentage of metabolically healthy individuals decreased as BMI increased, there were nevertheless many overweight and obese individuals who were metabolically healthy and a significant proportion of normal weight individuals who were metabolically unhealthy. According to their results, roughly 30% of normal weight individuals, 53% of overweight individuals, 71% of obesity grade I individuals, and 84% of obesity grade II and III individuals were classified as metabolically unhealthy, indicating that not all overweight and obese individuals are uniformly unhealthy. The arbitrary assumption that all obese patients have poor metabolic health can have negative consequences for people of all BMI categories—overweight and obese individuals may face stigmatization under the assumption that they lead an unhealthy lifestyle, while normal weight individuals may not receive adequate preventative, diagnostic, and therapeutic measures as they are regularly assumed to be metabolically healthy (Tomiyama et al., 2016, Wijayatunga and Dhurandhar, 2021).

Recent studies, aimed at identifying the root of higher morbidity and mortality amongst overweight and obese individuals, have differentiated between **metabolically healthy obesity** (MHO) and metabolically unhealthy obesity (MUO). However, at present, there is controversy as to generally accepted criteria defining MHO and, in some cases, the very existence of MHO has been called into question. MHO has been referred to as the "honeymoon phase" of obesity, that progresses to MUO in time. Moreover, despite the fact that MHO is not associated with an increased risk of type II diabetes and cardiovascular morbidity, adverse health effects in regard to osteoarthritis, sleep apnea, and respiratory problems remain. Therefore, there is relatively wide consensus among the reviewed literature that weight loss should also be a primary goal for metabolically healthy obese individuals (Iacobini et al., 2019).

Defining weight loss as the primary therapeutic goal for all obese individuals is easier said than done—there are many factors, which underscore the fact that making individuals alone responsible for weight loss is not a viable solution to ending the obesity epidemic. Especially in light of the fact that, despite evolving knowledge on the etiology of obesity, treatment options for overweight and obese individuals remain extremely limited (Müller et al., 2021). Moreover, biology alone cannot explain the current extent of the obesity epidemic, and although genetic variants, neuroendocrinological factors, and gut dysbiosis may facilitate obesity, it is the interaction between individual propensity toward weight gain and an obesogenic environment that tip the scales toward overweight and obesity.

2.2 *The Psychosocial Background of Obesity*

In addition to the heterogeneous biological factors that contribute to obesity, there are psychosocial factors that dictate the extent to which those of us who are predisposed toward obesity gain wait. It is this multidimensional psychosocial background of obesity that is at the center of this thesis. The Oxford English Dictionary defines "psychosocial" as "pertaining to the influence of social factors on an individual's mind or behavior, and to the interrelation of behavioral and social factors" (Oxford English Dictionary, 1989). Psychosocial factors are multidimensional constructs that encompass social and cultural environment (such as socioeconomic status, interpersonal relationships, family, ethnicity, and religion), mood status (including, among others, anxiety and depression), and cognitive behavioral responses (for instance satisfaction, control, self-esteem, and self-efficacy) (Vizzotto et al., 2013).

In reviewing the literature, the following three psychosocial determinants of overweight and obesity emerged as particularly relevant: first, the importance of **socioeconomic status**; second, the bidirectional association between obesity and **common mental disorders**, namely symptoms of depression and symptoms of anxiety;

and third, the impact of **adverse childhood experiences** on the current obesity epidemic. The details and implications of each are outlined in the following sections (2.2.1-2.2.5).

2.2.1 *Socioeconomic Status*

Socioeconomic status (SES) is a latent construct that describes an individual's or family's economic and social status in relation to others and can be differentiated into objective and subjective SES. The more commonly used **objective SES** is defined by access to material resources, most commonly the three aspects of education, income, and occupation (Baker, 2014) Whereas **subjective SES** is defined as an individual's own subjective perception of their place on the social ladder. Evidence suggests that subjective socioeconomic status more strongly affects health and BMI than objective SES does (Hoebel and Lampert, 2020, Bradshaw et al., 2017). To date, socioeconomic status (SES) is the one of the most important predictors, not only of overall health (Stringhini et al., 2017), but also of overweight and obesity (Bradshaw et al., 2017).

The extent to which our biologically determined individual propensity toward excess weight causes obesity is mediated by our environment and our health behaviors and these are dictated overwhelmingly by our socioeconomic status. The importance of environmental and social factors in fostering obesity is indicated by the fact that obesity is not equally distributed among our society. There is a well-established inverse relationship between SES and overweight and obesity—individuals with a lower SES tend to have a higher BMI (Kuntz and Lampert, 2010). An increasing body of evidence indicates that, as a result of both social causation and reverse causality, there is a bidirectional association between SES and obesity.

2.2.1.1 *Current Inequalities and the Bidirectional Association between Obesity and SES*

Sobal and Stunkard pioneered research into the field of obesity science and, in doing so, shed light on the inverse **association between obesity and SES** in developed countries (Sobal and Stunkard, 1989). In the developing world, however, the likelihood of obesity was greater in more affluent socioeconomic groups until the end of the 20th century. Sobal and Stunkard reviewed 144 studies published between 1960 and 1980 and found that obesity was higher in individuals with a higher SES in developing countries (Sobal and Stunkard, 1989). However, this association was no longer found in studies published after 1989—as a country's gross national product increases, the burden of obesity progressively shifts towards groups with lower SES (Monteiro et al., 2004, Dinsa et al., 2012), creating the double burden of disease that has evolved as a particular problem of developing countries in recent years (Popkin et al., 2020).

Based on Sobal and Stunkards' original research, McLaren reviewed 333 scientific articles on the correlation between SES and overweight and obesity in developed countries. Consistent with previous findings, she observed that a lower SES correlates with a higher

BMI in women in 63% of the studies and in men in 37% of these studies, though the negative correlation was not as striking as the one published in 1989 (McLaren, 2007). A more recent review, published in 2017, reproduced the negative correlation between SES and obesity for women, but found inconsistent results for men. The authors note that the majority (13 out of 15) of the studies available examined developed countries, while little information was available for developing countries (Newton et al., 2017). While the levels of overweight and obesity in adults continue to rise across the globe, the levels of childhood and adolescent obesity seem to have reached a plateau in many high-income countries (Abarca-Gómez et al., 2017). Worryingly however, even in these countries the socioeconomic disparities regarding obesity continue to grow (Krueger and Reither, 2015, Mensink et al., 2013, Frederick et al., 2014).

These inequalities indicate the importance of further research into the bidirectional association between obesity and SES. The inverse relationship between SES and obesity has long been explained by **social causation**, namely that a variety of financial and social factors lead groups with a lower SES to be more vulnerable to obesity. However, in recent years, evidence of the importance of **reverse causality** has emerged—meaning that structural inequalities make it more likely for people with obesity to develop a lower SES. A meta-analysis published in 2018, found a statistically significant link for both lower income and subsequent obesity (social causation) and obesity and subsequent lower income (reverse causality). Although notably, after adjusting for publication bias, only the results indicating reverse causality remained relevant (Kim and von dem Knesebeck, 2018). In the following chapters, evidence supporting both of these hypotheses is presented and possible mechanisms are discussed.

2.2.1.1.1 The Social Causation Hypothesis

A poor food environment and decreased physical activity are the two most commonly discussed factors in the **social causation** of the obesity epidemic. In areas with fewer financial means there is often an environment that negatively influences health related behavior and fosters obesity, both through a lack of access to healthy food and a lack of opportunity for physical activity.

Physical activity is strongly associated with physical health and mental well-being and a lack of physical activity can contribute to the long-term positive energy imbalance that leads to overweight and obesity. Most evidence suggests that an increasingly sedentary lifestyle is a main contributor to the obesity epidemic. Physical activity seems to be influenced by our physical environment and areas with higher walkability and access to recreational facilities have been associated with lower body weight. Nevertheless, the causal relationship between our environment, consequent physical activity and the obesity epidemic remain controversial (Smith et al., 2019, Archer et al., 2018, Lam et al., 2021).

Diet quality and nutritional preferences, which are influenced by both financial and psychosocial aspects, also play an important role in developing overweight and obesity. The negative consequences of unhealthy diets, such as obesity, type II diabetes, and cardiovascular disease, are well established in scientific literature (WHO, 2003). However, within our society, groups with a low SES are disproportionately affected by poor diet quality and subsequently also disproportionately affected by obesity and poor health (Fekete and Weyers, 2016). The inequalities in diet quality and nutritional preferences can be explained by socioeconomic, structural, psychosocial, and sociocultural factors—these numerous factors can be subsumed into three determinants that at times overlap, namely the **affordability, availability, and accessibility of foods** (Mader et al., 2020).

Affordability of healthy nutritional options is dictated by socioeconomic factors, most importantly financial resources. Affordability refers to the monetary cost of a diet and is, at least in part, responsible for the socioeconomic disparities in diet quality. There is a substantial financial barrier to a nutritious diet, as healthy foods are often more expensive than unhealthy options (Darmon and Drewnowski, 2015). Thus, it is unsurprising that there is an inverse association between the consumption of healthy foods and SES (Appelhans et al., 2012), although it must be noted that in absolute terms the SES differences in regard to food purchasing are relatively small (Pechey et al., 2013).

Availability of foods is dictated by structural factors, including neighborhood SES and food insecurity. Interestingly, **neighborhood SES** is associated with a higher risk of overweight and obesity independent of individual SES, Mohammed et al. found 31% higher odds of overweight and 45% higher odds of obesity in low SES neighborhoods in their meta-analysis containing data from over 1 million individuals (Mohammed et al., 2019). There are several factors thought to contribute to the obesogenic nature of neighborhoods with a low SES. A common explanation why obesity disproportionately affects groups with low SES is a **poor food environment**—meaning energy dense foods are more readily available, while healthy food options are scarcer in neighborhoods with a low SES (Caldwell and Sayer, 2019). Furthermore, **food insecurity** may cause lasting changes in food preferences even when food is no longer scarce (Olson et al., 2007). According to a 2019 meta-analysis, adults, especially women, in food insecure households are at a higher risk of obesity. However, they also note that the results should be viewed critically, due to the large heterogeneity amongst the 31 studies examined (Moradi et al., 2019).

Despite these findings, food availability and diet preferences alone seem insufficient to explain the inequalities in SES in regards to overweight and obesity (Mader et al., 2020, Zagorsky and Smith, 2017). Moreover, the scientific results are at times contradictory. A meta-analysis from 2020, examined first, the **social causation of diet quality** and subsequent effects on BMI and second, the socioeconomic consequences of obesity. Utilizing longitudinal data of over 20,000 individuals of the general German population, they found

inconclusive results regarding the predictive power of SES on diet quality (in fact, the only factors that predicted healthy diets were female sex, higher educational attainment, and younger age). However, the authors did find that developing obesity was significantly associated with a decrease in SES, thus adding evidence to the theory of reverse causality (which is discussed in the following chapter) (Mader et al., 2020).

Accessibility is dictated by sociocultural factors and psychosocial factors, many of which additionally limit access to physical activity and negatively influence health behavior. **Sociocultural factors**, include culturally handed down nutritional traditions, exercise behaviors, and body-image (Fekete and Weyers, 2016), which pass on the predisposition toward overweight and obesity from one generation to the next (Kuntz and Lampert, 2010). Evidence indicates, that while **parental influences** affect weight gain, genetic determinants of obesity may be more relevant than family environment. Studies investigating twins separated at birth, found that when adopted by parents who were both overweight or obese, the children were significantly more likely also to be overweight or obese. Despite these findings, the association between biological parents and their offspring were significantly stronger than between adopted parents and their children (Silventoinen et al., 2010).

Psychosocial factors include health literacy, self-efficacy, and social support and may mediate the link between low SES and obesity via psychosocial stress (Moore and Cunningham, 2012). Individuals living in low SES neighborhoods may be more exposed to psychosocial stressors, which negatively affect self-efficacy and socials support structures. Both low self-efficacy and lack of social support are associated with lower SES and with higher rates of obesity (Fekete and Weyers, 2016). Furthermore, higher SES is associated with higher **health literacy**, which promotes healthy lifestyle choices. In addition to financial constraints, knowledge of nutrition is an important aspect that influences diet quality and health behavior (Kim et al., 2017).

Job strain and the chronic stress it produces has also been found to predict the development of obesity (Brunner et al., 2007). As work stress and job strain are more frequent in individuals with lower SES it has been hypothesized to be a mediator between SES and obesity, however, results remain inconclusive. Two studies conducted in 2006 and 2007, found an association between higher levels of work stress and the development of obesity, abdominal obesity, and metabolic syndrome (Brunner et al., 2007, Chandola et al., 2006). Several others, including a meta-analysis by Wardle et al. and a review by Overgaard et al., however, showed weak or absent effects (Overgaard et al., 2004, Wardle et al., 2011, Rosengren et al., 2015, van Oostrom et al., 2021). Furthermore, the association could not be reproduced in a 2015 meta-analysis including eight studies on the subject (Kivimäki et al., 2015).

As mentioned above, **psychosocial stress** has been proposed as the mediating link between SES and obesity. Moreover, psychosocial stress makes individuals with a lower SES

disproportionately susceptible to **mental health disorders**, such as depression, which in turn increase the risk of overweight and obesity (see section 2.2.3 for details) (Schlax et al., 2019, Gary-Webb et al., 2011). Goodman et al. propose that subjective SES may mediate the link between objective SES and obesity via psychological stress, which can lead to social isolation, depression, and weight gain (Goodman et al., 2003). However, evidence supporting the premise that psychosocial stress predicts obesity is controversial and complicated due to the complex and multifactorial nature of the link.

2.2.1.1.2 Reverse Causality and the Obesogenic Effects of Weight Stigma

The social causation hypothesis (see chapter 2.2.1.1.1.) states that people living in areas with a low socioeconomic status (SES) are disadvantaged, especially regarding access to healthy nutritional options and physical activity, and thus disproportionately develop overweight and obesity. By contrast, mounting evidence suggests that, overweight and obesity can also lead to a decrease in SES—this is referred to as **reverse causality**. Weight stigma and structural discrimination are primarily to blame for reverse causality. Stigmatization leads to fewer material resources, disadvantages in education and occupation, changes in health promoting behavior, as well as psychological ramifications, such as stress, isolation, lower self-esteem, lower self-efficacy and internalized weight bias (Kim and von dem Knesebeck, 2018).

In this section, mechanisms are discussed through which weight bias and subsequent **structural discrimination** lead individuals with overweight or obesity to be disproportionately more likely to experience a decrease in SES. The belief that obesity is an individual life choice is deeply rooted in our society and, due to the fact that obesity is readily apparent, obese individuals are uniquely susceptible to **stigma and discrimination**. This continuous and pervasive discrimination affects almost all aspects of life and is harmful to physical and mental health (Bray et al., 2017, Rubino et al., 2020). Importantly, concentrating only on individual responsibility of overweight and obesity fails to address the ways in which weight stigma perpetuates obesity and leads to structural discrimination (Goldberg and Puhl, 2013).

The inverse association between SES and obesity is a relatively recent phenomenon—throughout most of history, overweight and obesity were considered a status symbol. In developed countries, excess weight was a prerogative of the elite and an indicator of personal wealth and health until the late 19th century. At the turn of the 20th century, however, it was no longer only the rich who had a surplus of food, as it became more readily available to the middle class as well. Thus, as overweight and obesity became more common the tide began to turn and fatness became associated with greed and gluttony. In this context, a thin body became a marker of self-control and restraint in a society characterized by excess. Interestingly, the stigmatization of the fat body predates health concerns regarding obesity. Cultural anxiety regarding body weight developed at a time when, through industrialization

and urbanization, concerns regarding overconsumption grew more prominent. This historic denigration contributes to the pervasiveness of weight stigma in today's society (Farrell, 2011).

Weight stigma is defined as the "devaluation and denigration of individuals because of their excess body weight" (Rubino et al., 2020) and can lead to weight-based stereotypes. Such preconceptions include the unfounded assumption that overweight and obese individuals are lazy, gluttonous, and lacking in self-discipline. Weight bias is widespread and includes **explicit bias**, which is overt and can be measured via self-report, **implicit bias**, which consists of unconscious stereotypes (Elran-Barak and Bar-Anan, 2018), and **internalized bias**, which may be particularly harmful as it causes self-blame and self-devaluation. According to estimates by Puhl et al., internalized weight bias is highly prevalent, affecting 40-50% of US adults with overweight or obesity (Puhl et al., 2018). As a consequence of weight bias, obese individuals face discrimination in almost all aspects of their lives, including the health care system, education, workplace, and social interactions (Wu and Berry, 2018).

In the public narrative and media coverage the misconception that obesity is an entirely individual responsibility is pervasive, and this plays an important role in reinforcing and legitimizing weight stigma (Puhl et al., 2013b, Rubino et al., 2020). Some medical ethicists, such as Callahan, have gone as far as to argue that stigmatization is an effective tool against the obesity epidemic—the common wisdom being that stigmatizing and ostracizing overweight and obese individuals will provide the necessary motivation needed to promote weight loss (Callahan, 2013). Besides being an ethically highly problematic suggestion (Goldberg and Puhl, 2013), current scientific literature indicates that the opposite is the case—not only does weight stigma fail to incite weight loss, it may be an important factor in reinforcing obesity (Hunger and Tomiyama, 2014, Rubino et al., 2020, Brewis et al., 2018). This was stated unequivocally by 36 members of an expert panel who authored a "Joint international consensus statement for ending stigma of obesity", published in Nature Medicine in 2020 (Rubino et al., 2020).

Weight stigma is associated with negative **physiological and psychological health outcomes** (Wu and Berry, 2018). Higher levels of weight stigma make individuals more susceptible to common mental health disorders (as detailed in section 2.3.3.2) and, conversely, lead to increased food intake, increased weight gain, and decreased physical activity. Furthermore, when compared to individuals who did not report weight stigma, the experience of weight stigma lead to higher levels of C-reactive protein, cortisol, cardiometabolic risk, and mortality (Rubino et al., 2020).

Weight stigma is particularly pervasive and particularly harmful in **the health care system.** 'Anti-fat' sentiments are widespread among medical health professionals (Sabin et al., 2012), even in clinicians who specialize in obesity issues (Tomiyama et al., 2015). In some

cases, concentrating solely on a patient's obesity has caused physicians to overlook other serious health conditions (Tomiyama et al., 2018). Unsurprisingly, stigmatization in the health care system can lead overweight and obese individuals to avoid or delay seeking medical attention (Puhl et al., 2013a, Puhl et al., 2021) and negatively impact the quality of care that higher weight individuals receive (Rubino et al., 2020).

Besides discrimination by individual health care providers, there is also **structural discrimination in the health care system**. Structural discrimination limits obese patients' access to bariatric surgery even when they are eligible and despite the fact that it is efficacious and cost-effective. Craig et al. conclude that these inequalities in the care of individuals with severe obesity are unethical and can be explained by pervasive weight bias that informs health policy (Craig et al., 2018). In some countries, such as the United States, lack of access to bariatric surgery disproportionately affects individuals with a low SES, further exacerbating the inequalities that already exist (Martin et al., 2010). Evidence suggests that individuals who are aware of the complex and multifactorial etiology of obesity are less likely to engage in weight stigma and discrimination (Rubino et al., 2020). The misconception of personal responsibility can cause individual and societal harm and, as these examples illustrate, even amongst health care professionals and policy makers there is a need for education on the multidimensional determinants of obesity.

Apart from the health care system, overweight and obese individuals face **professional, educational and interpersonal discrimination**—and throughout, the burden of weight stigma is felt more severely by women than men (Tronieri et al., 2017). On the one hand, women are subjected to more weight stigma than men, they are exposed to discrimination at an earlier age (Di Pasquale and Celsi, 2017) and at lower BMIs (27 kg/m^2 compared to 35 kg/m^2 in men) (Puhl et al., 2008). On the other hand, women seem to be more psychologically affected by weight discrimination and more likely to internalize weight bias (Tronieri et al., 2017). It has been hypothesized that this may be due to the greater value society places on female attractivity, thus penalizing women who do not fit conventional beauty ideals more harshly than men (Spahlholz et al., 2016). Subsequently, women with obesity complete fewer years of school, have a higher rate of poverty, are less likely to be upwardly mobile, and are less likely to be married than their normal weight counterparts. Men with obesity, by comparison, are merely less likely to be married (Finer, 2015).

In the **employment setting**, obese individuals are less likely to be considered qualified for a position, less likely to be invited for an interview, and less likely to be hired (Shinall, 2015, Flint et al., 2016, Campos-Vazquez and Gonzalez, 2020). Although workplace discrimination affects both men and women, the effects are more far reaching in women with obesity (Campos-Vazquez and Gonzalez, 2020, Shinall, 2015), who, if hired, are less likely to be employed in a setting that emphasizes personal interaction (the same is not true for men) (Shinall, 2015). People with obesity suffer from a considerable wage gap and this too is felt

more harshly by women than men—women earn an estimated 6.1% less than their normal weight peers, whereas men earn an estimated 3.4% less than normal weight men (Baum and Ford, 2004).

The health concerns regarding obesity are unquestionably valid and adapting appropriate preventative measures, such as improving food environment and promoting physical activity, could curb the far-reaching medical consequences of excess weight. However, the current culture of weight stigma and discrimination acts as an additional driver of the obesity epidemic, and the negative effects may disproportionally affect women. Stigmatization and structural discrimination cause further harm to individual and public health and exacerbate health inequalities along the socioeconomic gradient.

2.2.2 *The Role of Common Mental Disorders in the Obesity Epidemic*

Both obesity and mental health disorders are highly prevalent and lead to significant and far reaching public health implications. Common mental disorders are comprised of two diagnostic categories that are frequently comorbid: first, depressive disorders and, second, anxiety disorders (WHO, 2017). There is a bidirectional link between obesity and these common mental disorders, the significance of which is examined in the following sections.

The effects of **bariatric surgery** and consequent weight loss on mental health disorders and vice versa are an interesting and often reoccurring topic in current scientific literature. However, candidates for bariatric surgery have an increased prevalence of mental health disorders and, as such, are not representative of the general population (Jakobsen et al., 2018, Herpertz et al., 2017). For instance, approximately 50% of patients undergoing bariatric surgery were found to use antidepressants (de Zwaan et al., 2011). Moreover, bariatric surgery may directly affect mental health through various mechanisms (Brown et al., 2021). In light of these differences between candidates for bariatric surgery and the general population (Jakobsen et al., 2018, Herpertz et al., 2017) as well as the complex effects of bariatric surgery and consequent weight loss on symptoms of depression and anxiety (Brown et al., 2021), these areas of research are deliberately excluded from this literature review, as their inclusion would go beyond the scope of this thesis.

2.2.3 ***Depressive Disorders***

The two main subcategories of **depressive disorders** are major depressive disorder and dysthymia (WHO, 2017). Individuals with major depressive disorder often present with depressed mood and anhedonia, defined as an inability to enjoy experiences that would normally be enjoyable. Furthermore, symptoms of depression include fatigue, loss of interest, decreased concentration, altered sleep patterns (including both insomnia and hypersomnia), changes in appetite, feelings of worthlessness or excessive guilt, psychomotor changes (such as agitation or lack of initiative), and suicidal ideation. A depressive episode can be diagnosed

if symptoms persist for more than 2 weeks and can be categorized according to severity as either mild, moderate, or severe. Dysthymia, on the other hand, is defined as a persistent or chronic form of mild depression that continues over a minimum of two years (Lyness, 2019). Depressive disorders cannot, however, be diagnosed when there is a history of manic episodes, which differentiates them from bipolar disorder (Otte et al., 2016).

2.2.3.1 *Epidemiology and Implications of Depressive Disorders*

Depression is a common mental disorder that affects around 322 million people worldwide and is prevalent in high-, middle-, and low-income countries. According to the WHO, the estimated **global prevalence** of depression was 4.4% in 2015. Notably, depressive disorders are more common in women (5.1%, compared to 3.6% in men). Furthermore, between 2005 and 2015 there was a 18.4% increase in people living with depression. This development is explained both by an overall increase in the global population as well as an increased number of people in the age groups that most commonly experience depression. The WHO's data indicates that prevalence varies by age, with the median age of onset around 25 years (Otte et al., 2016) and the highest rates of depression amongst adults between 55 and 74 years old (WHO, 2017). Depressive disorders are the number one cause of non-fatal health loss worldwide. In 2015, depressive disorders led to over 50 million Years Lived with Disability (YLD) globally, comprising 7.5% of all YLDs (WHO, 2017).

In Germany, the prevalence of current depressive symptoms is 8.1% and the lifetime prevalence of a diagnosed depression is 11.6%. In both cases the prevalence is higher amongst women than men; with a prevalence of current depressive symptoms of 10.2% in women, compared to 6.1% in men, and a lifetime prevalence of a diagnosed depression of 15.4% in women, compared to 7.8% in men (Busch et al., 2013). According to WHO estimates, the prevalence of depressive disorders in Germany is 5.2%, comprising almost 700,000 YLD or 7.5% of total YLD (WHO, 2017).

Individuals affected by depressive disorders often have severe functional impairments and a decreased quality of life. Depressive disorders are also associated with many non-communicable diseases, including obesity, diabetes, stroke, acute myocardial infarction, and dementia, and confer a risk for several other chronic health conditions (Machado et al., 2018). Moreover, symptoms of depression may aggravate the negative health consequences of these comorbidities. For several somatic conditions, including breast cancer and acute coronary syndrome, depressive disorders have been shown to alter illness behavior, affecting patient compliance and decreasing adherence to treatment (Machado et al., 2018). Furthermore, depressive disorders may counteract adherence to positive lifestyle changes that are vital in counteracting negative health consequences (Sanchez-Villegas and Martinez-Gonzalez, 2013). As a public health concern, depressive disorders are responsible both for high health care costs and loss of productivity through work absence (Chisholm et al., 2016).

Fortunately, **treatment**—including pharmacotherapy, psychotherapy and supportive measures—is available for depressive disorders and these measures can lead to improved mental, emotional, and social functioning. Although depressive disorders affect many people around the world, there are large discrepancies regarding access to treatment. Globally, fewer than 50% of individuals with depressive disorders receive treatment and in low income countries fewer than 10% have access to treatment (Marcus et al., 2012). Furthermore, even in high-income countries, such as Germany, many people with depressive disorders do not receive adequate treatment (Wang et al., 2007, Thornicroft et al., 2017). The WHO identifies a scarcity of resources, lack of trained health-care providers, and societal stigma associated with mental health disorders as the main barriers to effective treatment of depressive disorders (WHO, 2020). Another barrier to treatment is inaccurate assessment with the result that many people with depression remain undiagnosed and thus untreated (WHO, 2020). Moreover, depressive disorders are often chronic and, although treatment is available, a considerable percentage of patients do not respond satisfactorily (Blackburn, 2019).

Due in part to a lack of access to treatment and adequate treatment options, depressive disorders are a leading cause of disability and a major public health concern. The high prevalence of depression as well as its far-reaching consequences on individual and public health indicate that it is an important topic of research. Particularly, the psychosocial background of depressive disorders and their interconnection with other major public health concerns such as the obesity epidemic.

2.2.3.2 *The Bidirectional Association between Obesity and Depressive Disorders*

Depressive disorders and obesity are both highly prevalent and thus commonly co-occur. Moreover, there is a **bidirectional association** between obesity and depressive disorders—they occur not only concurrently but also consecutively. On the one hand, mental disorders significantly increase an individual's risk of developing obesity, among others, due to increased sedentary behavior and changes in dietary habits (Martins et al., 2019). On the other hand, obesity and the associated metabolic disorders increase the incidence of depression. This co-occurrence is particularly troubling as it is linked to an increase in adverse health outcomes (Jantaratnotai et al., 2017).

Individuals who are obese have an approximately 55% increased odds of developing depression (Luppino et al., 2010). Notably, this association was higher when further metabolic risk factors were present and lower in metabolically healthy obese individuals (Jokela et al., 2014). A meta-analysis by de Wit et al. in 2010 found a similar reciprocal relationship between depression and obesity in women but not in men (de Wit et al., 2010a). Mannan et al. reconfirmed the bidirectional association between depression and obesity previously reported by Luppino et al. and de Wit et al. in 2010 (Mannan et al., 2016). In this

meta-analysis, the authors concluded that depression is more likely to predict overweight and obesity than vice versa, and that the association is particularly relevant in women of reproductive age (Mannan et al., 2016). Moreover, children who have depressive symptoms before the age of 17 are at a higher risk of developing weight problems as adults. In women, childhood depression was significantly associated with both overweight and obesity, while it was only associated with overweight in men (Hasler et al., 2005).

2.2.3.2.1 Some Biological and Psychosocial Pathways Linking Obesity and Depressive Disorders

The co-occurrence of depression and obesity may have both **biological and psychosocial underpinnings**. Biologically, plausible pathways mediating the link between obesity and depressive disorders involve inflammatory mediators and the immune system (neuroimmune theory), abnormal homeostasis of the hypothalamic-pituitary-adrenal axis (neuroendocrine theory), and behavioral mechanisms (Martins et al., 2019). Furthermore, certain antidepressants directly cause weight gain as a side effect (Verhaegen and Van Gaal, 2021). Psychologically, the stigma of obesity may play an important role in perpetuating depressive symptoms—both indirectly, through the structural effects of stigmatization and directly, through psychosocial stress (Brewis, 2014). Ouakinin et al. propose the stress response as a plausible mediator linking psychological and biological determinants of the two conditions (Ouakinin et al., 2018).

As mentioned above, biologically plausible mechanisms linking depressive disorders and obesity include the neuroimmune and the neuroendocrine theory. The **neuroimmune theory** proposes that an increase in immune mediators indirectly contributes to depressive disorders through disruptive effects on key functions, such as sleep, appetite, and cognition (Lee et al., 2016). Both depressive disorders and obesity are characterized by prolonged low-grade inflammation and, thus, an increase of inflammatory cytokines and other mediators that can promote neuroinflammation. This link is especially relevant in abdominal obesity, which correlates with higher rates of inflammation and is a more accurate predictor of depression than BMI alone (Hryhorczuk et al., 2013, Jantaratnotai et al., 2017). The **neuroendocrine theory** suggests that an overactivation of the stress response causes an imbalance in the hypothalamic-pituitary-adrenal (HPA) axis (Lee et al., 2016). Furthermore, there is mounting evidence that gut microbiome dysbiosis may play a role in changes in brain function through mediating effects on the inflammatory response and the HPA-axis (Martins et al., 2019).

Some behavioral mechanisms, involving **dietary habits and sleep abnormalities**, may also play a role. Mood disorders can lead to altered dietary habits, such as an increased craving for foods rich in carbohydrates (Ventura et al., 2014). Consumption of foods with high carbohydrate content can temporarily lift mood both due to an activation of the brain

opioid system and an increased serotonin production (Martins et al., 2019). Conversely, a diet high in calories and saturated fat is also directly associated with a higher risk of depression (Sanchez-Villegas and Martinez-Gonzalez, 2013). Moreover**,** sleep abnormalities, which are common in depressive disorders, alter neuroendocrine functions and may link depression to higher levels of obesity. Impaired sleep is also associated with dysfunctional glucose metabolism and increased hunger, mediated by changes in hormone balance (more precisely, an increase in ghrelin levels and a decrease in leptin levels) (Martins et al., 2019).

Importantly**, antidepressant medication**, which can be prescribed for moderate to severe depressive episodes, can also have far reaching consequences on metabolic health and is often directly responsible for weight gain. Many antidepressants, such as tricyclic antidepressants, some selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs) and mirtazapine (a tetracyclic antidepressant) are associated with a mean long-term increase of body weight upwards of 5% (Verhaegen and Van Gaal, 2021). Thus, antidepressant treatment is undoubtedly one reason why some individuals with depressive disorders become overweight or obese (Lee et al., 2016, Verhaegen and Van Gaal, 2021). However, there is currently no way to predict which individuals will be affected by weight gain, as it is independent of the severity of the underlying disorder and there is a large variation in individual weight gain. In a review by Verhaegen et al., the only consistent predictor of long-term weight gain was early weight gain, therefore it is important to screen for early weight gain (specifically an increase of over 5%) and, if possible, alter the antidepressant medication accordingly (Verhaegen and Van Gaal, 2021). The sharp rise in the prescription of antidepressants, which has risen by almost 400% since 1988, may be a contributing factor in the obesity epidemic (Lee et al., 2016).

In addition to the aforementioned biological and behavioral mechanisms, the bidirectional link between depressive disorders and obesity may be explained by psychosocial factors, primarily those that cause **psychosocial stress.** Psychosocial stress has been associated with negative physical and mental health outcomes although the interconnection between the two are not entirely understood (Beutel et al., 2018). In this context, the following two mechanisms have been proposed—on the one hand, mental health disorders may lead to poorer health behavior and as such reinforce the development of overweight and obesity and, on the other hand, the negative consequences of weight stigma may perpetuate symptoms of depression.

Previously, research found that psychosocial stress perpetuates weight gain in the general population through **poor health behavior** (Block et al., 2009, Harding et al., 2014). In one study this effect was stronger for women than it was for men, indicating that women may be more vulnerable to the effects of psychosocial stress (Chen and Qian, 2012). Psychosocial stress was associated with more uncontrolled eating, higher food craving, and physical inactivity, all of which promote weight gain. This effect of psychosocial stress on

health behavior seem to be exacerbated in those individuals who have mental health disorders such as depressive disorders. Within this cohort, as was also true in the general population, women were more affected by psychosocial stress (Beutel et al., 2018, Teychenne et al., 2010).

In a 2018 cross-sectional study by Beutel et al., which included over 2300 participants with mental disorders, the most commonly named stressor was "worries about health" among both men and women. The second most common stressor was "worries about weight or look[s]" among women and "financial problems" among men (Beutel et al., 2018)—indicating both the interdependence between the effects of weight-based stigma and psychosocial stress and the fact that women seem to be more vulnerable to them. In this study, higher levels of stress were associated with poorer health behavior. In women, higher levels of stress were associated with obesity, uncontrolled eating, and physical inactivity, whereas in men, higher levels of stress were associated with uncontrolled eating, physical inactivity, and smoking (Beutel et al., 2018). These results confirmed similar previous findings that stress leads to overeating, binge eating, food craving, and an increased intake in foods high in calories (Järvelä-Reijonen et al., 2016, Chao et al., 2015, Barrington et al., 2014)

As discussed in detail in chapter 2.2.1.1.2, obese individuals are uniquely susceptible to **societal stigma and discrimination** and the stigma of obesity plays an important role in perpetuating depressive symptoms both through psychosocial stress and through the structural effects of stigmatization (Brewis, 2014). Although women experience more weight-based discrimination than men at both an interpersonal and structural level, the effects of weight bias are felt by both genders across almost all facets of life and this continuous exposure to discrimination creates psychosocial stress (Tronieri et al., 2017). Increased levels of stress can contribute to depressive disorders by reinforcing inadequate dietary and exercise habits (Krzysztoszek et al., 2015). Moreover, the chronic stress in response to continuous stigmatization can lead to an increase in abdominal fat, which in turn may increase the mediators connected to both obesity and depression—starting anew the cycle of stigma, discrimination, and depression (Hryhorczuk et al., 2013). Hatzenbuehler et al. studied the effects of perceived weight discrimination in over 22,000 U.S. citizens and found that individuals who had experienced discrimination due to their weight were approximately 2.5 times more likely to experience mental disorders than those who did not. Interestingly, this association was independent of BMI, indicating that the detrimental effects of weight stigma may be independent of individual weight (Hatzenbuehler et al., 2009).

Both obesity and depression are isolating, stigmatizing, and have far reaching consequences on health and social functioning. A more holistic approach to the often co-occurring mental health disorders and obesity could improve both conditions. For instance, improved health behaviors, such as increased physical activity, can positively influence mental health through antidepressant effects and is also a cornerstone in the prevention of

obesity (Hallgren et al., 2016). Such strategies for the prevention of obesity may be particularly effective if directed at individuals seeking treatment for mental health disorders, as research indicates that they are uniquely susceptible to the effects of psychosocial stress (Beutel et al., 2018).

2.2.4 *Anxiety Disorders*

Anxiety disorders include a variety of disorders ranging from generalized anxiety disorders to specific phobias; symptoms range from mild to severe. Their common feature is excessive fear and anxiety that persists beyond the appropriate period and/or avoidance of perceived internal or external threats, additionally, panic attacks may occur. Commonly, anxiety disorders develop in childhood or early adulthood and persist if left untreated (American Psychiatric Association, 2013, Craske et al., 2017). Anxiety disorders tend to coexist with each other and are highly comorbid with other mental disorders. Moreover, they are associated with poor subsequent health outcomes, both somatic and mental (Schmitz et al., 2010, Ströhle, 2018). For instance, over 50% of individuals with an anxiety disorder suffer from multiple anxiety disorders. In addition, individuals with anxiety disorders are at a higher risk of developing depressive disorders and more likely to suffer from a more severe course of depression (Craske et al., 2017). Despite their common occurrence and their significance as a public health care problem, even in high-income countries, anxiety disorders often remain undiagnosed and untreated (Alonso et al., 2018).

2.2.4.1 *Etiology and Epidemiology of Anxiety Disorders*

There are several risk factors which predispose toward anxiety disorders. These include, female sex (women are twice as likely as men to develop anxiety disorders), a family history of anxiety or depressive disorders (children of parents with anxiety disorders are two- to four-times as likely to develop anxiety disorders), and adverse childhood events. Moreover, genetics play an important role—heritability has been estimated at around 30 to 40% and, especially when combined with environmental stress, can lead to the development of anxiety disorders. However, specifics regarding mechanisms of familial transmission or gene loci remain largely unknown. Neurobiological changes have also been implicated in the etiology of anxiety disorders both through alterations in specific brain regions and dysfunction in the hypothalamic-pituitary-adrenal axis (Craske et al., 2017).

Globally, approximately one out of four individuals either currently has an anxiety disorder or has had one in the past (Craske et al., 2017). Anxiety disorders are the most common mental disorder in Europe with a 12-month prevalence of 14% (WHO, 2017). Most anxiety disorders begin at a young age and the 12-month prevalence in children and adolescents is similar to that in adults (Craske et al., 2017). Among highly developed countries, anxiety disorders rank in fourth place as the cause of Years Lived with Disability (YLD) and the impact of these disorders on the lives of affected individuals is profound.

According to WHO estimates, the prevalence of anxiety disorders in Germany is 5.8%, comprising over 400,000 YLD or 4.5% of total YLD (WHO, 2017).

2.2.4.2 *The Bidirectional Link between Obesity and Anxiety Disorders*

The u-shaped curve used to describe the correlation between BMI and somatic health conditions has been reproduced for the link between BMI and anxiety disorders—with both overweight (BMI ≥25kg/m²) and underweight (BMI <18,5 kg/m²) individuals reporting higher levels of anxiety (Scott et al., 2008, DeJesus et al., 2016). Notably, this u-shaped curve could not be reproduced by a recent study investigating the relationship between BMI and anxiety disorders in the German population (Herhaus et al., 2020). Similar to the correlation between obesity and depression, there appears to be a reciprocal, **bidirectional link between obesity and anxiety disorders** (Puhl and Luedicke, 2012). However, there are very few longitudinal studies on this link and thus no clear conclusion can be made regarding the direction of the association (Rajan and Menon, 2017).

There have been two meta-analyses investigating the association between obesity and anxiety in adults, one conducted by Gariepy et al. in 2010 and one by Amiri and Behnezhad in 2019—although both found a positive association they also reported high levels of heterogeneity (Gariepy et al., 2010, Amiri and Behnezhad, 2019). The 2019 meta-analysis, including a majority of cross-sectional studies and three prospective cohort studies, found that overweight/obesity is positively associated with symptoms of anxiety. Amiri and Behnezhad examined emotional eating, weight stigma, and adverse childhood events as possbile pathways linking obesity and anxiety disorders (Amiri and Behnezhad, 2019). The 2010 meta-analysis by Gariepy et al. also found a positive association between obesity and anxiety disorders. The two pathways they consider most relevant in explaining this link are, first, the negative effect of weight-related discrimination on mental health outcomes and, second, the adverse effects of comorbidities associated with both obesity and anxiety disorders (Gariepy et al., 2010).

The possibility of **weight-based discrimination** as a mechanism linking obesity and anxiety disorders is discussed in both meta-analyses (Gariepy et al., 2010, Amiri and Behnezhad, 2019). Obese and overweight individuals report a higher incidence of being bullied, more instances of social isolation, and lower self-esteem and are thus at a higher risk of developing anxiety disorders (Puhl and Luedicke, 2012). Moreover, individuals with obesity tend to have a lower SES (see chapter 2.2.1.1), which is associated with poorer social support and social networks and these, in turn, are also associated with poorer mental health (Gariepy et al., 2010). Interestingly, one experimental study, investigating how SES and wealth inequality influence caloric intake, found that wealth inequality leads to increased calorie consumption via social anxiety (Bratanova et al., 2016).

There are **gender differences** in the association between anxiety disorders and obesity, with women being disproportionately affected (Amiri and Behnezhad, 2019, Sharafi et al., 2020). These differences seem to be due to the fact that women with obesity are more severely affected by weight stigma and structural discrimination (see section 2.2.1.1.2. for details). Levy and Pilver investigated lingering effects of past weight stigma on mood disorders. The authors found that the likelihood of anxiety and depressive disorders among formerly obese individuals was almost equal to that of individuals who were presently obese. Thus, indicating that internalized weight bias may play an important role perpetuating negative mental health (Levy and Pilver, 2012).

Emotional eating has been proposed as the mediating link between anxiety disorders and obesity (Amiri and Behnezhad, 2019). Ostrovsky et al. examined the association between anxiety disorders and emotional and binge eating in overweight and obese individuals and found statistically significant results (Ostrovsky et al., 2013). However, a 2016 study examining this association in youths found that, although there was a positive association between the degree of adiposity and anxiety, emotional eating was not a mediating factor (Fox et al., 2016). Despite these inconsistencies, further research into this topic is important—anxiety disorders, and the accompanying emotional overeating, may be a severe hindrance toward seeking treatment for obesity and obesity treatment may be ineffective if the underlying anxiety disorders are not addressed as well (Ostrovsky et al., 2013).

Another pathway hypothesized to mediate the bidirectional link between obesity and anxiety disorders is their co-occurrence with **poor somatic health and an unhealthy lifestyle**. On the one hand, individuals with anxiety disorders tend to engage in fewer social and physical activities, thus increasing their risk of developing obesity (de Wit et al., 2010b). Anxiety disorders are also independently associated with several physical disorders and these are significantly associated with poor quality of life as well as disability and, thus, can predispose toward obesity (Sareen et al., 2006). On the other hand, an obesogenic lifestyle, including lack of physical activity and an unhealthy diet may promote and sustain anxiety disorders (De Moor et al., 2008). A recent study found that social anxiety symptoms mediated the link between obesity and insulin resistance as well as elevated inflammation—indicating that these health risks may be more severe in those individuals who suffer from both symptoms of anxiety and a higher BMI (Jaremka and Pacanowski, 2019).

Adverse childhood experiences (ACEs) have been associated with anxiety disorders (Hovens et al., 2012, Carr et al., 2013, Gardner et al., 2019a). In Europe, more than a quarter of cases of depression and anxiety could be attributed to ACEs, creating a combined annual cost of approximately 51 billion US dollars (Bellis et al., 2019). Although Gariepy et al. propose ACEs as a possible link between obesity and anxiety disorders (Gariepy et al., 2010), little is known regarding the mediating effect of ACEs on this association. Furthermore, there

may be an underlying **neurobiological link** between anxiety and obesity, these discoveries, however, have their basis in preclinical trials. In animal models studying rodents, a diet high in calories and sugar altered both the structure and function of the prefrontal cortex, the amygdala and the hippocampus, thus fundamentally altering emotional behavior (Giustino and Maren, 2015, Kalyan-Masih et al., 2016, Baker et al., 2017).

Although several pathways have been proposed to explain the recurring link between anxiety disorders and obesity, the association is less robust than the one between obesity and depressive disorders (Rajan and Menon, 2017). Moreover, the underlying mechanisms remain unclear and further research into this topic is needed in order to better understand the complexity of the mental health implications of overweight and obesity.

2.2.5 *Adverse Childhood Experiences*

Adverse childhood experiences (ACEs) are characterized by disruptive and traumatizing events in a young person's life, such as abusive parenting, sexual abuse, parent separation, caregiver mental disorders, parental substance abuse, death of a caregiver, food insecurity and many more (Shonkoff et al., 2012). Parents or guardians are responsible for the vast majority of childhood mistreatment, and besides direct physical harm, the consequences of ACEs include long-term effects on "neurological, cognitive, and emotional development and overall health" (Norman et al., 2012). Nevertheless, it was only in the course of the industrial revolution that childhood was recognized as a particularly vulnerable period in one's life and attention was paid to the consequences of childhood trauma and mistreatment (Kappis and Hardt, 2016). ACEs are commonplace around the world and they tend to be highly clustered within families. Moreover, ACEs are a risk factor for chronic ill health and many mental disorders, with the negative ramifications persisting throughout a lifespan (Jorm and Mulder, 2018). Throughout the scientific literature, ACEs have been consistently linked to various adverse health outcomes with most studies reporting a dose-response relationship—as the number of ACEs increase so does the risk for negative health outcomes such as obesity (Hemmingsson et al., 2014, Bellis et al., 2019, Norman et al., 2012, Schroeder et al., 2021).

2.2.5.1 *Prevalence and Societal Implications of ACEs*

First published in a 1998 landmark study, Felitti et al. found that individuals exposed to ACEs were at higher risk of developing many chronic diseases, including obesity (Felitti et al., 1998). Following this 1998 study, the CDC examined the prevalence of ACEs in the United States and their impact as a public health problem. They found first, that ACEs are widespread, with at least one in seven children in the U.S. having suffered from ACEs within the past year; second, that children of low socioeconomic status are disproportionally affected; and third, that the economic burden of ACEs is high, creating an estimated 124 billion U.S. dollars expenditure annually in the United States (CDC, 2019). Furthermore,

ACEs seem to be highly clustered—individuals who reported one ACE were more likely also to have suffered additional ACEs (Anda et al., 2010).

The epidemiological data and implications of ACEs are similar in Germany. According to a 2017 study, almost every third individual in Germany had experienced at least one form of childhood maltreatment in their lifetime, with women reporting higher levels of sexual and emotional abuse. As in previous studies, ACEs were associated with lower educational attainment, employment status, and income (Witt et al., 2017). Not only do ACEs have far reaching consequences for chronic ill health, and thus cause a burden to the health care system, they also create a high economic burden. In Germany, the annual costs caused by maltreatment have been estimated between 11 and 30 billion Euros annually (Habetha et al., 2012). Thus, promoting safe and nurturing environments for children is important from both a health care and an economic perspective. According to a 2019 meta-analysis published in the Lancet, reducing the prevalence of ACEs in Europe by 10% could reduce the number of disability-adjusted life-years (DALYs) by around 2 million and consequently reduce the annual expenditure by 49 billion US dollars. Moreover, these numbers may be even higher if the effect of ACEs on social issues, such as unemployment and imprisonment, were also taken into account (Bellis et al., 2019).

2.2.5.2 *ACEs and the Development of Obesity*

The largest meta-analysis to date, published in 2017 by Hughes et al. (Hughes et al., 2017), found a positive association between ACEs and the development of obesity, thus, confirming results published previously and since (Danese and Tan, 2014, Hemmingsson et al., 2014, Norman et al., 2012, Fleischer et al., 2021). Hughes et al. examined 37 studies, published prior to 2016, in regard to the multiple health effects of ACEs. The study compared health outcomes in adults with at least four ACEs to adults without any ACEs and found a modest association with overweight and obesity. Eight of the analyzed studies (cumulative sample size: 84,840) specifically identified overweight and obesity as an adverse health outcome (pooled odds ratio of 1.39) among these studies those that examined a higher BMI reported higher odds ratios. Additionally, the authors noted a dose-response—individuals who reported at least four ACEs were at an approximately four-times higher risk of developing overweight or obesity. There was a high heterogeneity throughout the studies, possibly due to the lack of comparability in the studies regarding type and extent of exposure to ACEs. Despite this heterogeneity, there remained a consistent positive association between ACEs and negative health outcomes (Hughes et al., 2017).

Interestingly, Westermair et al. focused not only on the effects of ACEs on weight gain, but also on the ramifications of two commonly used types of analysis, namely the selective versus the cumulative approach. The ‘selective approach’ focuses solely on one ACE without controlling for others, while the ‘cumulative approach’ combines all categories of

ACEs into one and thus is unable to differentiate between them. The cumulative approach is problematic, because, although the different categories of ACEs are highly interconnected, they are not necessarily interchangeable. The author argues that different ACEs may have very specific outcomes mediated by varying mechanisms that we are unable to differentiate using a cumulative approach (Westermair et al., 2018). On the other hand, due to the cumulative nature of ACEs, studies investigating only the effect of a single ACE may likely overestimate its impact on public health (Anda et al., 2010). One systematic review on the association between ACEs and childhood obesity, for instance, found that the effect of sexual abuse was greater than that of other ACEs while also noting the likely cumulative effect of ACEs on the development of childhood obesity (Schroeder et al., 2021).

2.2.5.3 *Pathways Linking ACEs and Obesity*

A recent meta-analysis, published in 2020 by Wiss and Brewerton, investigated the wide variety of potential mechanisms that have been proposed to explain the link between adverse childhood experiences and health, specifically overweight and obesity. The following pathways are most commonly discussed in the relevant literature: first, a chronic stress response, and second, changes in health behaviors (Wiss and Brewerton, 2020).

ACEs have both **biological and psychological manifestations**. Biologically, ACEs cause chronic stress and thus an increase in inflammatory cytokines, a hyper-activation of the HPA-axis, and an increase in cortisol levels and suppression of the immune system (Hemmingsson et al., 2014). Besides increasing biomarkers of inflammation, ACEs have also been shown to affect health through shortened telomers (Bellis et al., 2019). Psychologically, ACEs have been linked to depression and anxiety, which may subsequently lead to weight gain (see sections 2.2.3 and 2.2.4) (Gardner et al., 2019a). Moreover, ACEs have been associated with anorexia nervosa, which may cofound the estimates between ACEs and obesity (Wiss and Brewerton, 2020).

In the early 2000s the *Centers for Disease Control and Prevention* (CDC) coined the term '**toxic stress**' to explain the consistent link between ACEs and obesity—the state of chronic stress caused by adversity can cause both hormonal imbalances and reactive overeating that adversely affects health and weight in adulthood (Shonkoff et al., 2012). Tomiyama proposes stress as the main driver of a vicious cycle in which chronic stress contributes to obesogenic processes and the consequent weight stigma, in turn, perpetuates chronic stress (Tomiyama, 2019). One study found that ACEs predicted adolescent obesity independent of caregiver weight-status, physical activity, and dietary habits. Notably, the effect was more pronounced amongst children in low-income households. The authors postulate that it is the combined stress of living in poverty and exposure to ACEs that cause increased weight gain amongst these youths (Gardner et al., 2019b).

ACEs have been linked to **changes in health behavior**, these include both mental disorders (the effects of which are discussed in chapters 2.2.3 and 2.2.4) and health harming activities, such as smoking tobacco, excessive alcohol and food consumption, and substance abuse (Bellis et al., 2019, Norman et al., 2012). Bellis et al. argue that the effects of health-harming activities due to ACEs may contribute considerably to the development of non-communicable diseases, and yet, ACEs are generally overlooked as risk factors (Bellis et al., 2019). Additionally, ACEs may have a negative effect on sleeping patterns and promote unhealthy dietary and exercise choices, including an increased craving for calorie dense "comfort-food" (Jastreboff et al., 2013).

The high prevalence of ACEs across the globe (Stoltenborgh et al., 2014), their far reaching impact on psychological and physiological health, and the ensuing high economic burden demonstrate the importance of adverse childhood experiences as a public health problem. Moreover, the far-reaching consequences of ACEs highlight the importance of childhood as a period of life in which the foundations of good mental health are established (Bellis et al., 2019). In regard to obesity, clinicians often screen overweight individuals for overeating and lack of physical activity and disregard the important topic of ACEs. However, as the above results indicate, obesity is not solely a consequence of individual life-choices—there are significant ramifications resulting from childhood adversity and the subsequent psychological and biological consequences. Thus, in treating obesity in individuals with ACEs, it may be vital to understand the underlying issues in order to identify individuals at risk and address the public health problem that ACEs create (Elsenburg et al., 2017).

2.2.6 *Defining Obesity as a Disease*

The differences in weight gain among people living in the same environment may make it appear as though each person is entirely individually responsible for overweight and obesity. The etiology of obesity, however, is far more complex and once weight has been gained there are "powerful homeostatic mechanisms that hinder weight loss and promote further weight gain" (Blüher, 2019). Currently, the most widely accepted solution to the obesity epidemic is weight loss—achieved by limiting caloric intake and increasing physical activity. Although, a well-balanced diet and physical activity are important measures to counteract obesity on a societal level, these measures alone are inadequate to achieve long-term weight loss in those individuals who are already obese (Blüher, 2019, Franz et al., 2007). Furthermore, the results of most scientific literature indicate that only around 5% of obese individuals successfully lose weight long term without bariatric surgery (Tsai and Wadden, 2005). Thus, any solution that is directed solely at obese individuals and disregards our environment is going to fall short of its goal (Herpertz et al., 2011).

In 2017 the World Obesity Federation released a statement with a similar conclusion. They define obesity as a "chronic relapsing disease process**"**, instead of a risk factor for other

diseases, in an attempt to relieve those suffering from obesity from individual responsibility and sensitize the public that obesity is a topic that concerns us all (Bray et al., 2017). This definition of obesity has important implications both for policy and treatment options. Nevertheless, as previously discussed, it is problematic to define obesity as a disease while measuring obesity solely in BMI categories (Rubino et al., 2020). A combination of approaches is needed, focusing both on individual and population-wide strategies in order to achieve meaningful advancements in population-wide health (Stephens et al., 2014). In light of the global increase in obesity and its far-reaching consequences, it is apparent that gaining a better understanding of the background of obesity is important and may help find a solution that is as multifaceted and complex as the problem.

2.3 *Formulation of the Hypotheses*

In view of the far-reaching consequences of the obesity epidemic, the importance of research, not only into the consequences of obesity, but, especially, into its etiology—more precisely the psychosocial factors fostering obesity—becomes apparent. Moreover, the adverse medical, societal, and economic consequences of widespread obesity are expected to increase the future. In reviewing the literature, the following factors—socioeconomic status, depression, social phobia, and adverse childhood events—have emerged as particularly relevant and are thus examined in the hypotheses at the center of this thesis.

Hypothesis 1: Individuals with a lower socioeconomic status are at a higher risk of being overweight or obese.

Hypothesis 2: Individuals who experience symptoms of depression are at a higher risk of being overweight or obese.

Hypothesis 3: Individuals who experience symptoms of social phobia are at a higher risk of being overweight or obese.

Hypothesis 4: Individuals who experienced adverse childhood experiences are at a higher risk of being overweight or obese.

3 Materials & Methods

3.1 *Data Collection and Sample Description*

The data was collected using surveys and was conducted in two separate waves. The first wave of data was collected in 2008, whereas the second wave was collected in 2018 through 2019—these will be referred to as *Wave 1* and *Wave 2* respectively. In total, 780 participants were recruited and the data of 718 participants was evaluated in the statistical analysis. The surveys were simultaneously conducted in Poland (*Wave 1* and *Wave 2*) and England (*Wave 2*). The data from these countries, however, is not considered in this thesis as the comparison between countries is the subject of a different thesis.

The participants of *Wave 1* were made up of 377 German university students, and ultimately the data of 356 participants was statistically analyzed. The survey was conducted on paper and participants were recruited at the Universities in Düsseldorf and Mainz. The 237 students who took part in the survey in Düsseldorf received 5 Euros in compensation, while the 148 students who took part in Mainz were not monetarily compensated for their time.

In *Wave 2* a total of 403 participants took part in the survey and the data of 362 participants was ultimately statistically analyzed. The second wave of data was collected via recruitment of participants mainly at schools and universities in Mainz. The link to the online survey, which was hosted by the platform *SoSci Survey* (https://www.soscisurvey.de/), could be found on the University's homepage for the *Institute of Medical Psychology and Sociology*. Participation was anonymous, voluntary and was not monetarily compensated. The overall feedback from the participants in both waves was positive, with most reporting that the questions were interesting and engaging. The main criticism was however, that the survey was too lengthy and thus time consuming.

Due perhaps in part to the length of the survey, estimated at around 30 minutes, there were several participants who did not complete all items in the questionnaire. Thus, for a participant's data to be included in the statistical analysis, there were following prerequisites: they had specified their age, gender, height, and weight and had answered at least a minimum number of items in the categories *Symptoms of Depression/Social Phobia* and *Socioeconomic Status* (as detailed in Table 3.1). Furthermore, only participants with a BMI between 17 and 45 kg/m² were included. Although both a low and a high BMI are associated with a higher mortality (see chapter 2.1.3), underweight is not the subject of the current study and its inclusion would have gone beyond the scope of this thesis. The survey also contained one participant with a BMI of 52 kg/m², who's data was also excluded from statistical analysis. As the data was collected via online-survey and no clinical examination of the participants took place, it was not possible to investigate possible underlying physical conditions that may have led to this high BMI. It is possible that the inclusion of this

individual's data may have had a disproportionately strong influence on the results. Thus, the data from this participant was excluded. However, due to the large sample size, the exclusion of this one outlier had only very limited effect on the results.

The minimum requirements for all of these subsets of data had to be met, in order for a participant to be included in the analysis. Thus, all data from one participant was eliminated as soon as a single subset of data was missing. This was necessary in order to ensure that the ordered sequences of regressions could be correctly analyzed. Using these criteria, a total number of 62 participants were excluded from the statistical analysis due to missing data. In Table 3.1., the numbers of participants excluded within each subset are listed in the order of elimination. Of the 780 participants who initially took part in the survey, the data of 718 participants was statistically analyzed.

Table 3.1 Criteria for exclusion and number of participants excluded in the order of elimination

Criteria for Exclusion	Number of Participants Excluded
Age not specified	6
Gender not specified	5
Weight not specified	10
Height not specified	2
BMI > 45 kg/m²	1
BMI < 17 kg/m²	4
Symptoms of Depression / Social Phobia: < 7 out of 9 items specified	11
Socioeconomic Status: < 4 out of 5 items specified	23
Total number of participants excluded	**62**

3.2 *Description of the Questionnaires*

The survey is comprised of items from three different questionnaires: The Symptom-Check-List-27-plus, the Mainz Structured Biographical Interview, and the Childhood Questionnaire. These questionnaires are briefly outlined in this chapter. For detailed information regarding all variables examined in the statistical analysis, and the questions they contain, see chapter 3.3.

The subset of items for the questions regarding the secondary response, *Symptoms of Social Phobia and Depression*, are derived from the Symptom-Check-List-27-plus (SCL-27-plus). This questionnaire is an abbreviated form of the original Symptom Checklist-90-Revised and was validated by Hardt. It acts as a "short, multidimensional screening instrument for mental health problems" (Hardt, 2008). A total of five scales are included in the SCL-27-plus. Two of these scales pertain to symptoms of depression and symptoms of social phobia, they are described in greater detail in chapter 3.3.2.

The subset of items regarding the tertiary response, *Childhood Adversities*, are derived from the Mainz Structured Biographical Interview (MSBI). The MSBI was developed by Hardt and Egle and contains seven sections, the items from one of these sections are included in the statistical analysis and described in chapter 3.3.3 (Egle and Hardt, 2004).

The subset of items regarding the *Socioeconomic Status* are derived from the Childhood Questionnaire (CQ). This questionnaire is based on the Adult Attachment Interview and retrospectively examines the relationship adults had to their parents growing up as well as their socioeconomic situation during this time. A shortened version of this questionnaire was cross validated in 2012 and the items examined in this thesis are described in chapter 3.3.5 (Hardt et al., 2012). Additionally, the variable *Socioeconomic Status* was amended by one item from the MSBI, details regarding this item can also be found in chapter 3.3.5 (Egle and Hardt, 2004).

3.3 *Description of Examined Variables and Corresponding Items*

The variables included in the statistical analysis are subsets of items contained in the three questionnaires SCL-27-plus, MSBI, and CQ. According to their role in the statistical analysis these are defined as the primary response (*Body Mass Index*), the secondary response (*Symptoms of Social Phobia and Depression*), the tertiary response (*Childhood Adversities*) and solely explanatory variables (*Age, Gender, Wave and Socioeconomic Status*). Most of the questions are answered on a Likert scale, with only occasional free text responses for items such as *Age, Height, and Weight*. Figure 3.1 offers an overview of the variables included in the statistical analysis, the corresponding questions for all items pertaining to these variables are detailed in the subchapters below.

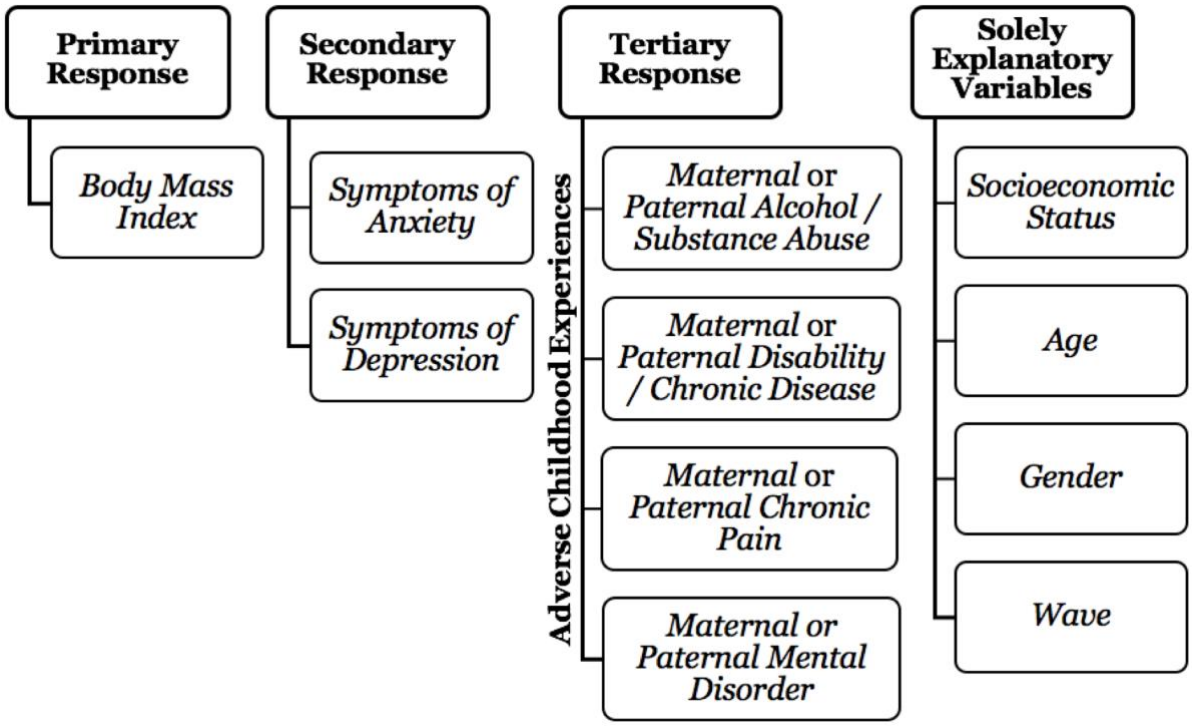

Figure 3.1 Overview of the Variables Included in the Statistical Analysis

3.3.1 *Items for the Primary Response: Body Mass Index*

In order to calculate the primary response, *Body Mass Index*, participants were asked to indicate their weight and height at the beginning of the survey.

1. *"Please state your weight in kilograms: _____ kg"*
2. *"Please state your height in centimeters: _____ cm"*

3.3.2 *Items for the Secondary Response: Symptoms of Social Phobia and Depression*

The items regarding the secondary response, *Symptoms of Social Phobia and Depression*, are derived from the Symptom-Check-List-27-plus. Participants answered the following five items regarding symptoms of social phobia within the past two weeks:

"How often did you experience the following symptoms?"

1. *"Fear of saying something embarrassing"*
2. *"Feeling others do not like me"*
3. *"Feeling inhibited when dealing with others"*
4. *"Feeling insecurity when others look at me"*
5. *"Feeling of being unwanted"*

Participants answered the following four items regarding symptoms of depression within the past two weeks:

"How often did you experience the following symptoms?"

6. *"Melancholy"*
7. *"Feeling blank inside"*
8. *"Hopelessness"*
9. *"Loss of joy"*

The possible answers on a five-point Likert scale were as follows:

- *"never"*
- *"seldom" (defined as 1-2 days within the past two weeks)*
- *"sometimes" (defined as 3-7 days within the past two weeks)*
- *"often" (defined as 8-12 days within the past two weeks)*
- *"very often" (defined as 13-14 days within the past two weeks)*

These answers were transformed to a scale ranging from 0 to 100, in which higher values indicate more symptoms of depression and social anxiety. The internal consistency (Cronbach's α) for this subset of items was calculated as 0.88 for *Wave 1* and 0.90 for *Wave 2*.

3.3.3 *Items for the Tertiary Response:* Childhood *Adversities*

The items regarding the tertiary response, *Childhood Adversities*, are derived from the Mainz Structured Biographical Interview. Participants answered the following eight items regarding childhood adversities during their childhood and adolescence:

"*Think back on your first 14 years of life:"*

1. *"Was your mother chronically ill or physically handicapped during this time?"*
2. *"Was your father chronically ill or physically handicapped during this time?"*
3. *"Did your mother suffer from chronic pain during this time?"*
4. *"Did your father suffer from chronic pain during this time?"*
5. *"Did your mother suffer from mental problems or nervous disorders during this time (e.g. anxiety, depression, thoughts of suicide, psychiatric illnesses, …)?"*
6. *"Did your father suffer from mental problems or nervous disorders during this time (e.g. anxiety, depression, thoughts of suicide, psychiatric illnesses, …)?"*
7. *"Did your mother have an alcohol problem or other addiction during this time?"*
8. *"Did your father have an alcohol problem or other addiction during this time?"*

The possible answers for questions **1.-8.** were as follows:

- *"Yes"*
- *"No"*

These answers were transformed to a scale ranging from 0 to 1. The answers were interpreted as follows: the answer "*yes*" was coded as 1, the answer *"no"* was coded as 0, and when the item was not specified it was coded as 0.5. No response to this question may stem from uncertainty about the presence or absence of the parental problem or even from not knowing the parent at all. The value 0.5 was chosen in these cases so that the participants who were unable to answer the question were not completely lost for the analysis. Listed in Table 3.2 is an overview of the answers reported for the subset of questions regarding *Childhood Adversities* including those who were unable to make a specification.

Table 3.2 Overview of the Responses to the Items Regarding Childhood Adversities

		"Yes" (≙ 1)	***"No"*** (≙ 0)	**Not specified** (≙ 0.5)
Alcohol/ Substance Abuse	**Maternal**	5.99 %	90.25 %	3.76 %
	Paternal	10.72 %	85.10 %	4.18 %
Disability / Chronic Disease	**Maternal**	7.80 %	88.44 %	3.76 %
	Paternal	8.36 %	87.33 %	4.32 %
Chronic Pain	**Maternal**	7.52 %	88.16 %	4.32 %
	Paternal	6.96 %	87.05 %	5.99 %
Mental Disorder	**Maternal**	16.85 %	80.50 %	2.65 %
	Paternal	8.91 %	87.19 %	3.90 %

3.3.4 *Items for the Explanatory Variables: Age and Gender*

Participants answered the following two items regarding their age and gender:

1. *"Please state your age: _____"*
2. *"Please state your gender: _____"*

3.3.5 *Items for the Explanatory Variables: Socioeconomic Status*

The items regarding the *Socioeconomic Status* are derived from the Childhood Questionnaire and the Mainz Structured Biographical Interview. Participants answered the following five items regarding their socioeconomic status during their childhood and adolescence:

1. *"What was your family's financial situation like during your first 7 years of life?"*

"These questions [2.-5.] *pertain to your home life with your parents from your childhood to your adolescence (up to the age of 14):"*

2. *"I come from a family with a high social status."*
3. *"Money was pretty tight in my family."*
4. *"During my childhood we lived in a really small, cramped place."*
5. *"I come from a wealthy family."*

The possible answers on a four-point Likert scale for question **1.** was as follows:

- *"meagre"*
- *"unstable"*
- *"unproblematic"*
- *"good"*

The possible answers on a four-point Likert scale for questions **2.-5.** were as follows:

- *"not true at all"*
- *"hardly true"*
- *"rather true"*
- *"absolutely true"*

These answers were transformed to a scale ranging from 0 to 100, in which higher values indicate a higher socioeconomic status and lower values indicate a lower socioeconomic status. The internal consistency (Cronbach's α) for this subset of items was calculated as 0.87 for *Wave 1* and 0.84 for *Wave 2*.

3.4 *Overview of the Examined Variables*

The following table represents an overview of the data obtained for the variables in wave 1 and wave 2.

Table 3.3 Overview of the Examined Variables in Wave 1 and Wave 2

					Wave 1 n=356		**Wave 2** n=362	
Description		Possible Values	Min.	Max.	Mean	SD	Mean	SD
Age		–	13	51	24.31	4.00	22.88	5.38
Gender (female)		–	0	1	64.57%	–	67.40%	–
Height (in cm)		–	150	200	173.84	9.18	171.83	9.21
Weight (in kg)		–	43	130	68.47	13.24	68.19	14.26
BMI (in kg/m²)		17–41	17	41	22.56	3.49	23.00	3.86
Socioeconomic Status		0–100	6.25	100	69.86	19.41	66.12	21.23
Symptoms of Depression		0–100	0	100	18.21	17.56	26.09	23.55
Symptoms of Social Phobia		0–100	0	100	28.05	18.21	27.99	25.13
Alcohol/ Substance Abuse	**Maternal**	0–1	0	1	5.20%	0.19	10.50%	0.30
	Paternal	0–1	0	1	12.08%	0.30	13.54%	0.34
Disability/ Chronic Disease	**Maternal**	0–1	0	1	7.02%	0.23	12.29%	0.32
	Paternal	0–1	0	1	7.72%	0.24	13.26%	0.33
Chronic Pain	**Maternal**	0–1	0	1	6.32%	0.22	12.98	0.32
	Paternal	0–1	0	1	6.88%	0.22	12.98%	0.32[1]
Mental Disorder	**Maternal**	0–1	0	1	14.19%	0.33	22.19%	0.41
	Paternal	0–1	0	1	9.55%	0.26	12.15%	0.32

[1] Please note that the identical values of the standard deviations in wave 1 and 2 as well as the mean in wave 2 for the variables *Maternal/ Paternal Chronic Pain* are incidental and have been verified by the author.

3.5 *Statistical Analysis*

The survey responses were statistically analyzed by means of ordered sequences of regressions. This method is an extension of the path analysis first developed by the Geneticist Wright and was developed by Wermuth and Cox (Wermuth and Cox, 2013). Wermuth and Cox designed this technique similarly to Graphical Markov Models in order to enable a method for testing complex interrelations between variables, while ensuring that the significant results can be concisely illustrated (Wermuth and Cox, 2015).

The model uses a linear regression for continuous variables, such as *Age, BMI, and Socioeconomic Status,* and a logistical regression for binary variables. Since the responses regarding the subclass of variables within *Childhood Adversities* were coded as 0, 0.5, and 1, all responses in this model are continuous and thus only linear regressions were used. The variables are defined as the primary response (*Body Mass Index*), the secondary response (*Symptoms of Social Phobia and Depression*), the tertiary response (*Childhood Adversities*) and solely explanatory variables (*Age, Gender, Wave and Socioeconomic Status*). The only binary variables examined, *Wave* and *Gender*, are solely explanatory variables and thus not tested as responses in the regressions.

First, linear regressions were performed for all primary, secondary, and tertiary responses. All quadratic effects and all two-way interactions were tested successively, while including all significant and non-significant main effects. The quadratic effects and two-way interactions with significant results (defined as $p<0.01$) were entered. Both the quadratic effects as well as the two-way interactions only remained in the statistical analysis if the term itself as well as the variables contained were significant. In order to test the level of significance for the variables contained, a Chi^2-test was performed using 2 degrees of freedom for a quadratic term and 3 degrees of freedom for an interaction term. Finally, all main effects were removed, that were neither significant nor contained in an interaction or quadratic term. The statistical analysis was performed using the software *Stata 16*.

Due to the large sample size (n=718) the threshold of significance (α) was defined as ≤1%. This reduces the number of significant effects that are not clinically relevant. In order to improve clarity and comprehensiveness, tables and figures contain only those effects with a p-value <0.01.

3.6 *Presentation of the Results*

In the Results section (chapter 4), all significant results are illustrated in tables and figures. As mentioned above, results with a p-value >0.01 are not displayed. Each of the subchapters (4.1-4.3) follows the same outline: First, a graphical overview is given, of all significant interactions and effects between variables as determined by the ordered sequences of regressions. These figures are labelled as *Overview of all Significant Effects and Interactions* and are described in detail in the following paragraph. Second, the significant results are illustrated in a table and third, each significant effect and interaction is individually depicted in a figure. The details of these are also described below. The Pearson's correlations of the residuals are illustrated in a separate subchapter at the end of the *Results* section.

First, in the figures that represents an *Overview of all Significant Effects and Interactions*, the responses and variables are graphically represented. As described in the *Statistical Analysis*, the variables are defined as the primary response (*Body Mass Index*), the secondary response (*Symptoms of Social Phobia and Depression*), the tertiary response (*Childhood Adversities*) and solely explanatory variables (*Age, Gender, Wave and Socioeconomic Status*). The first box from the left represents the primary response, the second represents the secondary response, the third represents the tertiary response, and the fourth contains solely explanatory variables. Within the boxes, continuous variables are represented by circles and binary variables are represented by dots. Due to the fact that the variables of the tertiary response, *Childhood Adversities,* are coded as 0, 0.5, and 1 (see section 3.5 *Statistical Analysis* for details) these too are treated as continuous variables and therefore represented by circles. Arrows between the variables indicate the significant main effects, quadratic effects, and two-way interactions as determined by the ordered sequences of regressions. Thus, these figures (4.1., 4.5, 4.11, 4.16, 4.19, 4.22, and 4.27) offer an overview of all significant effects and interactions for all variables of the primary, secondary, and tertiary responses. These overviews can be found at the beginning of each subchapter.

Second, following the graphical overview, each subchapter contains a tabulated overview of all significant results (defined as p-value <0.01) for each response as determined in the linear regression analyses. Tables 4.1-4.11 display the regression coefficient, standard error, p-value, t-value, total explained variance, and Cohen's f^2 for all significant results. The p-values reported in the tables, figures and the text are derived from the linear regressions. As mentioned in the *Statistical Analysis* (chapter 3.5), all individual variables contained in significant effects and interactions were also tested for significance at a level of p-value <0.01 using a Chi²-test, however, these p-values are not separately reported.

Third, each of the significant two-way interactions, main effects and quadratic effects are illustrated in a separate figure (4.2–4.4, 4.6–4.10, 4.12–4.15, 4.17, 4.18, 4.20, 4.21, 4.23–4.26, 4.28, and 4.29). In these figures the x-axis represents the explanatory variable and the y-axis represents the estimated value of the response including the corresponding 99%

confidence interval. The curves themselves represent only 90% of the data, with the highest and the lowest 5% excluded in order to avoid over-interpretation in quadratic and interaction effects. The tertiary response, *Childhood Adversities,* are coded as 0, 0.5, and 1 (see chapter 3.5 *Statistical Analysis* for details) and although these values are included in the regression analysis, the value 0.5 is not displayed in the graphics.

Finally, the Pearson's correlations of the residuals are illustrated in a separate chapter at the end of the Results section. The residuals are defined as the variance that remains, after the impact of influencing variables is deducted. Due to the nature of the statistical analysis—in which regressions are calculated for each of the variables within one box for all variables in the boxes to its right—the correlations for the variables within their own box has not yet been analyzed. Thus, in chapter 4.4, Pearson's correlation coefficients of the residuals within each box are tabulated for all significant results ($p<0.01$). The correlation of the residuals was calculated only for the second and third boxes (secondary and tertiary responses), as the first box (primary response) contains only a single variable and the fourth box contains only solely explanatory variables.

4 Results

4.1 *Results for the Primary Response:* ***Body Mass Index***

In this subchapter all statistically significant predictors of the primary response, *Body Mass Index,* are described and graphically illustrated.

4.1.1.1 Body Mass Index*: Overview of all Significant Effects*

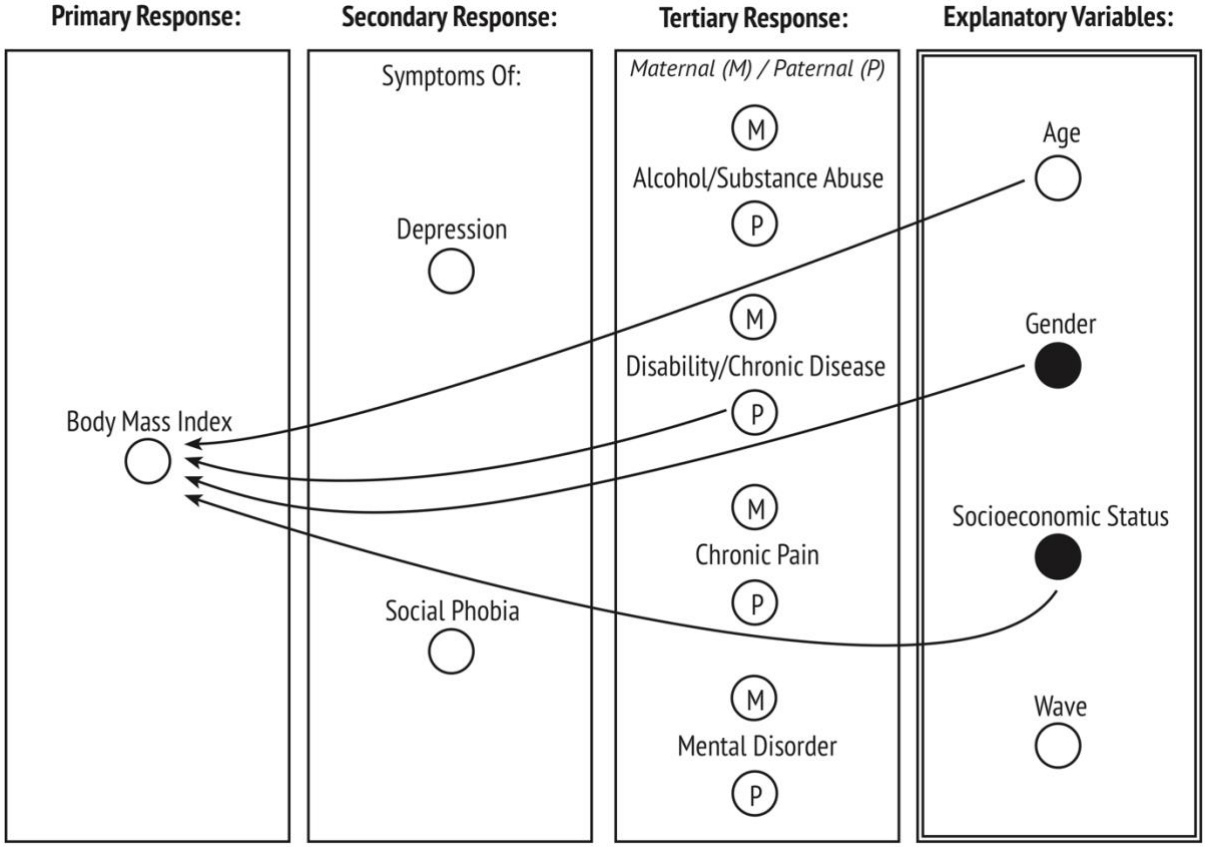

Figure 4.1 Primary Response: Body Mass Index—Overview of all Significant Effects

Regarding the primary response, *Body Mass Index*, there are four significant predictors. There is an interaction between reported *Paternal Chronic Disease* and *Age* (1 and 2), a quadratic effect for *Socioeconomic Status* (3), and a main effect for *Gender* (4). All significant effects are illustrated by arrows in Figure 4.1.

4.1.1.2 *Body Mass Index: Linear Regression Analysis*

Table 4.1 displays the regression coefficient, standard error, t-value, p-value, and total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.15 and thus, as suggested by Cohen, equivalent to a medium effect size (Cohen, 1992).

Table 4.1 Body Mass Index: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Paternal Chronic Disease	-6.62	2.10	-3.16	0.002
Age	0.15	0.03	5.23	<0.001
Paternal Chronic Disease *Age	0.32	0.09	3.64	<0.001
Socioeconomic Status	-0.11	0.03	-3.42	0.001
Socioeconomic Status^2	0.00	0.00	2.78	0.006
Gender	1.72	0.27	6.40	<0.001
Constant	22.34	1.20	18.67	<0.001
Total Explained Variance: 16.94%				
Cohen's f^2: 0.204				

4.1.1.3 *Body Mass Index: Interaction between Paternal Chronic Disease and Age*

The two-way interaction between *Paternal Chronic Disease* and *Age* in regard to the primary response, *Body Mass Index,* is depicted in Figure 4.2. With a p-value <0.001, the interaction is statistically highly significant.

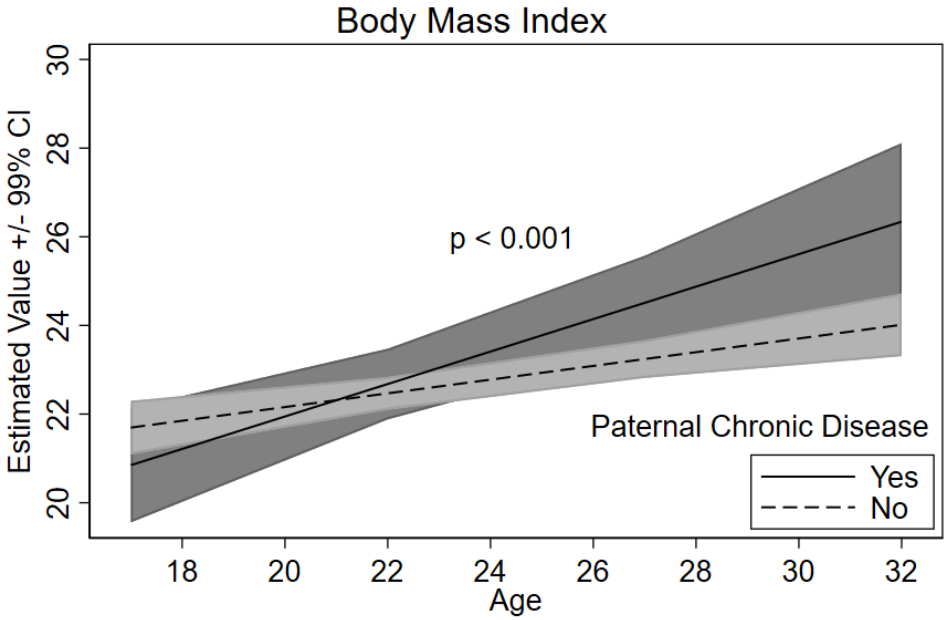

Figure 4.2 Primary Response: Body Mass Index Explained by the Interaction between Paternal Chronic Disease and Age

The figure plots and the age of the participants on x-axis and their BMI in kg/m² including the corresponding 99% confidence interval on the y-axis. The solid line represents participants who reported paternal chronic disease, while the dashed line represents those who reported no paternal chronic disease. Both in participants with and without paternal chronic disease, their BMI was positively associated with age. Notably however, participants who reported paternal chronic disease or disability in the first 14 years of their life had a significantly sharper rise in their BMI. The broader confidence interval (CI) for the group of participants with paternal chronic disease is explained by the smaller number of cases. The CI is defined as the quotient of the standard error and the root of the number of cases, therefore fewer cases result in a broader CI.

4.1.1.4 Body Mass Index*: Quadratic Effect of* Socioeconomic Status

The quadratic effect of *Socioeconomic Status* in regard to the BMI is depicted in Figure 4.3. The effect is statistically significant, with a p-value ≤0.006.

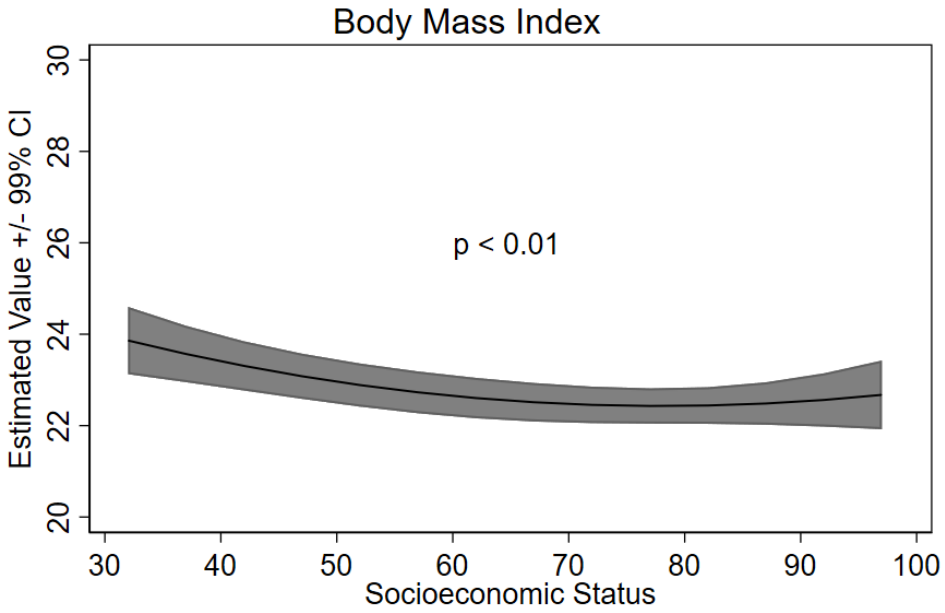

Figure 4.3 Primary Response: Body Mass Index Explained by the Quadratic Effect of Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and BMI, in kg/m^2, is plotted on the y-axis. As seen in the figure, a lower socioeconomic status during childhood and adolescence is associated with a higher BMI. However, as indicated by the quadratic effect, SES only predicted high BMI for individuals with a low SES while the same was not the case for individuals with a medium to high SES. The apparent repeated increase of the BMI values at the at the high end of the socioeconomic status, however, is an artefact due to fitting a quadratic term.

4.1.1.5 Body Mass Index*: Main Effect of* Gender

The main effect of *Gender* in regard to the BMI is depicted in Figure 4.4. The effect is statistically highly significant, with a p-value <0.001.

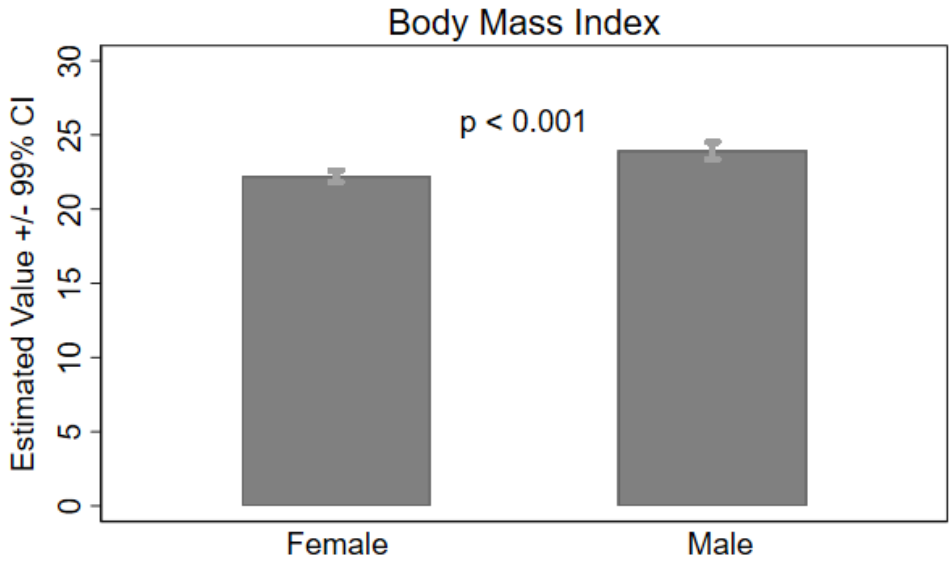

Figure 4.4 Primary Response: Body Mass Index Explained by the Main Effect for Gender

In the figure, gender is plotted on the x-axis and BMI, in kg/m², is plotted on the y-axis. Male participants of the survey reported a higher BMI than female participants. Cohen's d was calculated as -0.495 and thus equivalent to a small effect size (defined as >0.20 and <0.50), as suggested by Cohen (Cohen, 1992).

4.2 *Results for the Secondary Responses:* ***Symptoms of Depression and Social Phobia***

In this subchapter all statistically significant two-way interactions and main effects of the two variables, *Symptoms of Depression* and *Symptoms of Social Phobia,* of the secondary response are described and graphically illustrated.

4.2.1 *Results for Symptoms of Depression*

4.2.1.1 *Symptoms of Depression: Overview of all Significant Effects*

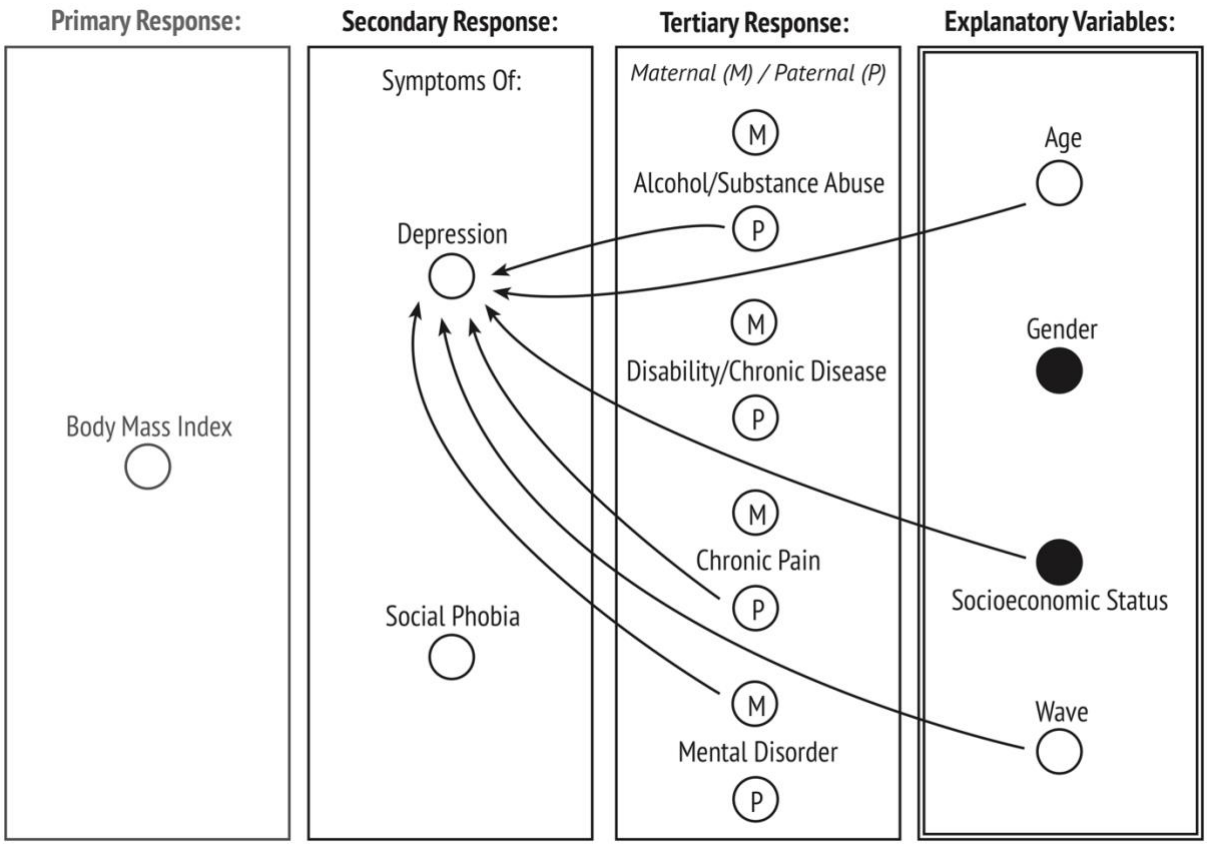

Figure 4.5 Secondary Response: Symptoms of Depression—Overview of all Significant Effects and Interactions

Regarding the secondary response, *Symptoms of Depression*, there are six significant predictors. There is an interaction between *Paternal Alcohol Abuse* and *Paternal Chronic Pain* (1 and 2), a main effect for *Maternal Mental Disorder (3),* a main effect for *Socioeconomic Status (4),* a main effect for *Age (5),* and a main effect for *Wave (6)*. All significant effects are depicted by arrows in Figure 4.5.

4.2.1.2 *Symptoms of Depression: Linear Regression Analysis*

Table 4.2 displays the regression coefficient, standard error, t-value, p-value, and total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.15 and thus, as suggested by Cohen, equivalent to a medium effect size (Cohen, 1992).

Table 4.2 Symptoms of Depression: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Paternal Alcohol Abuse	4.30	2.66	1.61	0.107
Paternal Chronic Pain	1.20	3.16	0.38	0.704
Paternal Alcohol Abuse *Paternal Chronic Pain	23.13	7.30	3.17	0.002
Maternal Mental Disorder	5.46	2.04	2.67	0.008
Socioeconomic Status	0.15	0.03	-4.03	<0.001
Age	0.58	0.15	-3.69	<0.001
Wave	5.68	1.51	3.75	<0.001
Constant	41.52	5.34	7.77	<0.001
Total Explained Variance: 13.31%				
Cohen's f^2: 0.154				

4.2.1.3 *Symptoms of Depression: Interaction of Paternal Alcohol Abuse and Paternal Chronic Pain*

The two-way interaction between *Paternal Alcohol Abuse* and *Paternal Chronic Pain* on the secondary response, *Symptoms of Depression is* depicted in Figure 4.6. The effect is statistically significant, with a p-value ≤0.002.

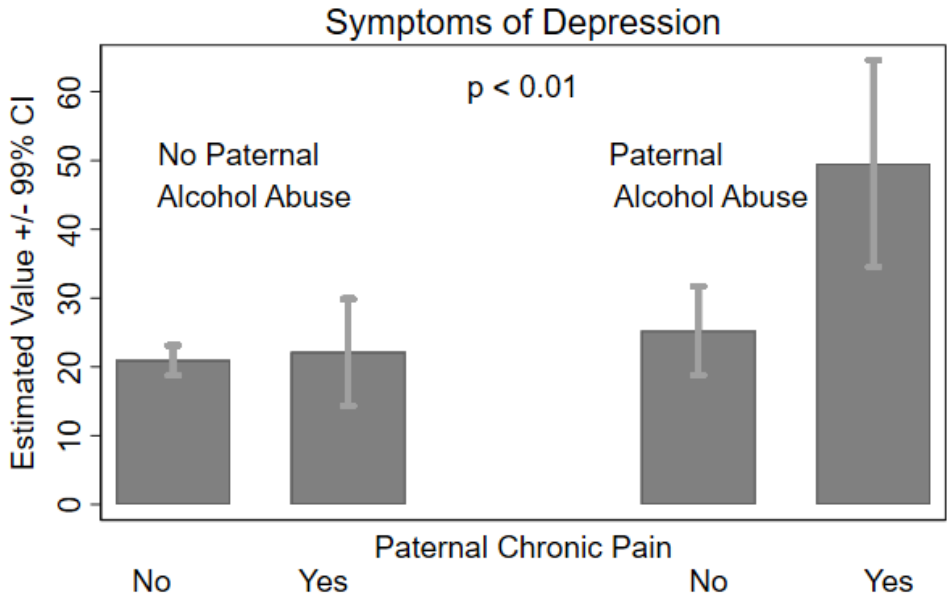

Figure 4.6 Secondary Response: Symptoms of Depression Explained by the Interaction of Paternal Alcohol Abuse and Paternal Chronic Pain

In the bar diagram, the responses to the items *Paternal Chronic Pain* and *Paternal Alcohol Abuse* are plotted on the x-axis, while the estimated value for the symptoms of depression are plotted on the y-axis. As can be seen in the diagram, only those participants who reported both paternal chronic pain and paternal alcohol abuse were more likely to suffer from symptoms of depression. The presence of only one of the two items, however, did not have a significant effect on symptoms of depression.

4.2.1.4 *Symptoms of Depression: Main Effect for Age*

The main effect of *Age* in regard *to Symptoms of Depression is* depicted in Figure 4.7. The effect is statistically highly significant, with a p-value <0.001.

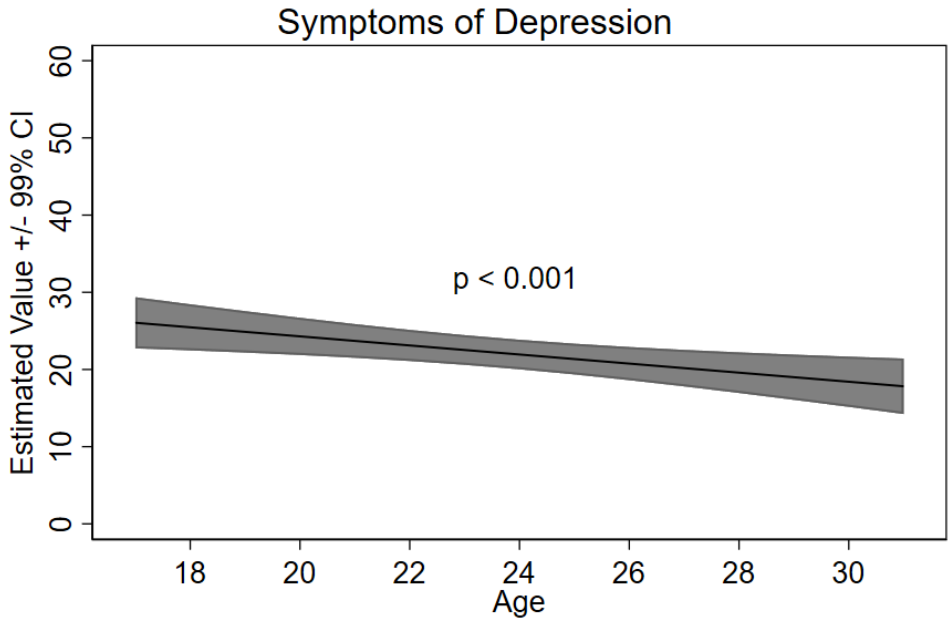

Figure 4.7 Secondary Response: Symptoms of Depression Explained by the Main Effect for Age

The figure plots the age of the participants on the x-axis and the estimated value for symptoms of depression on the y-axis. As seen in the figure, the symptoms of depression continually decrease as the age of the participants increases.

4.2.1.5 *Symptoms of Depression: Main Effect for Maternal Mental Disorder*

The main effect of *Maternal Mental Disorder* in regard *to Symptoms of Depression is* depicted in Figure 4.8. The effect is statistically significant, with a p-value of ≤0.008.

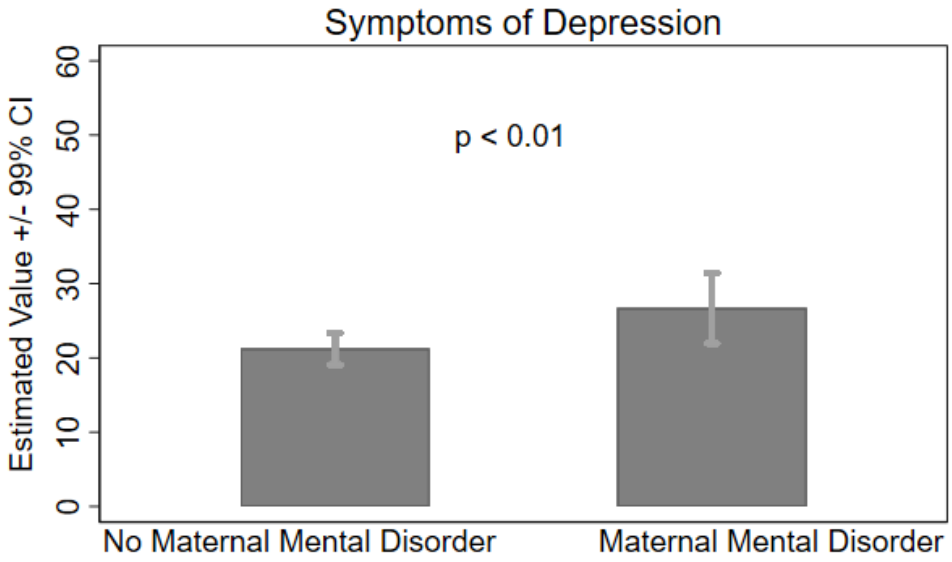

Figure 4.8 Secondary Response: Symptoms of Depression Explained by the Main Effect for Maternal Mental Disorder

In the bar diagram, the response to the item *Maternal Mental Disorder* is plotted on the x-axis and the estimated value for the symptoms of depression are plotted on the y-axis. As seen in the figure, those participants who reported maternal mental disorders had higher rates of symptoms of depression.

4.2.1.6 *Symptoms of Depression: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Symptoms of Depression is* depicted in Figure 4.9. The effect is statistically highly significant, with a p-value <0.001.

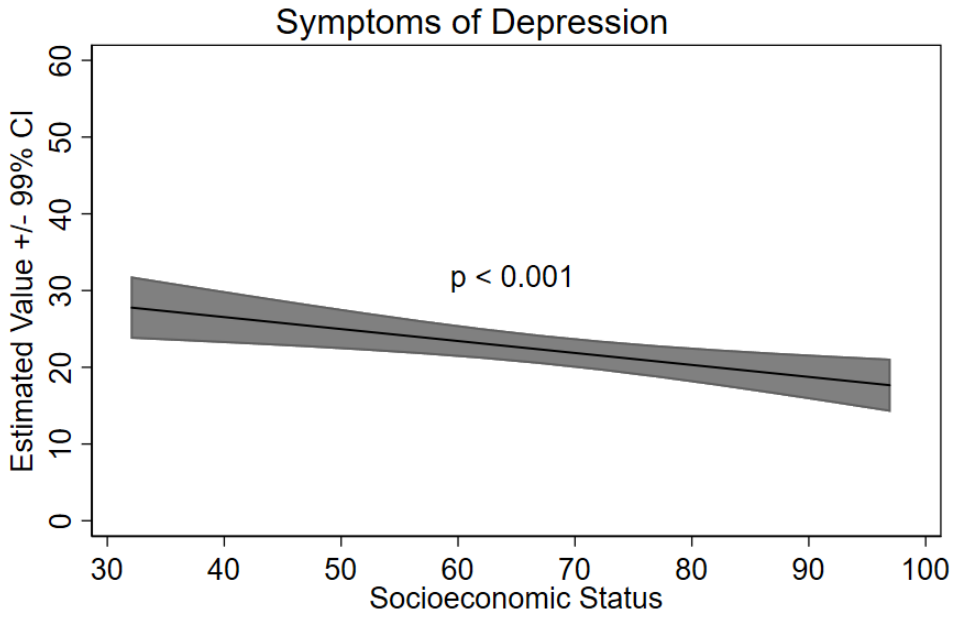

Figure 4.9 Secondary Response: Symptoms of Depression Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for symptoms of depression are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with more reported symptoms of depression. The symptoms of depression continuously decrease as the socioeconomic status increases.

4.2.1.7 *Symptoms of Depression: Main Effect of* Wave

The main effect of *Wave* in regard *to Symptoms of Depression is* depicted in Figure 4.10. The effect is statistically highly significant, with a p-value <0.001.

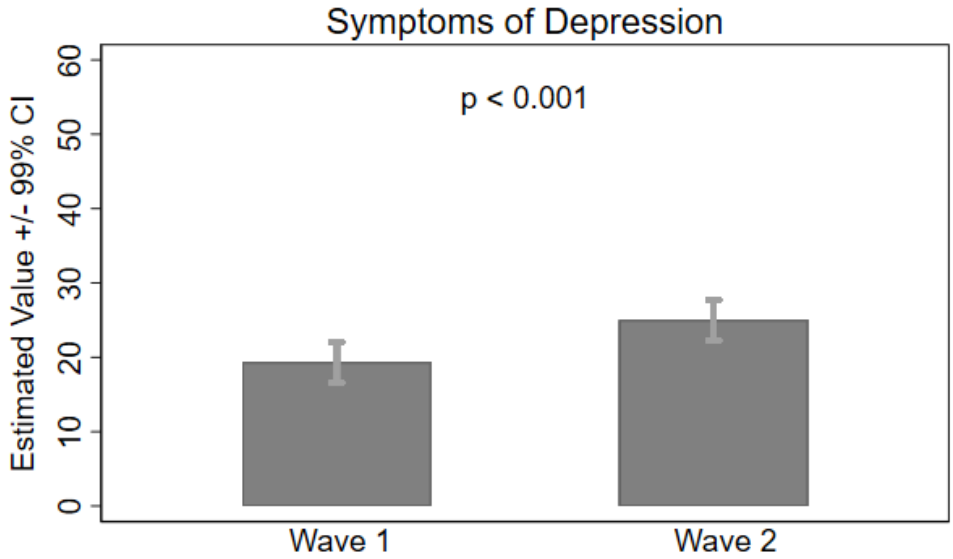

Figure 4.10 Secondary Response: Symptoms of Depression Explained by the Main Effect for Wave

In the bar diagram, the wave is plotted on the x-axis and the estimated value for the symptoms of depression are plotted on the y-axis. As seen in the figure, participant in wave 1 reported fewer symptoms of depression than those in wave 2. Cohen's d was calculated as -0.379 and thus equivalent to a small effect size (defined as >0.20 and <0.50), as suggested by Cohen (Cohen, 1992).

4.2.2 *Results for Symptoms of Social Phobia*

4.2.2.1 *Symptoms of Social Phobia: Overview of all Significant Effects*

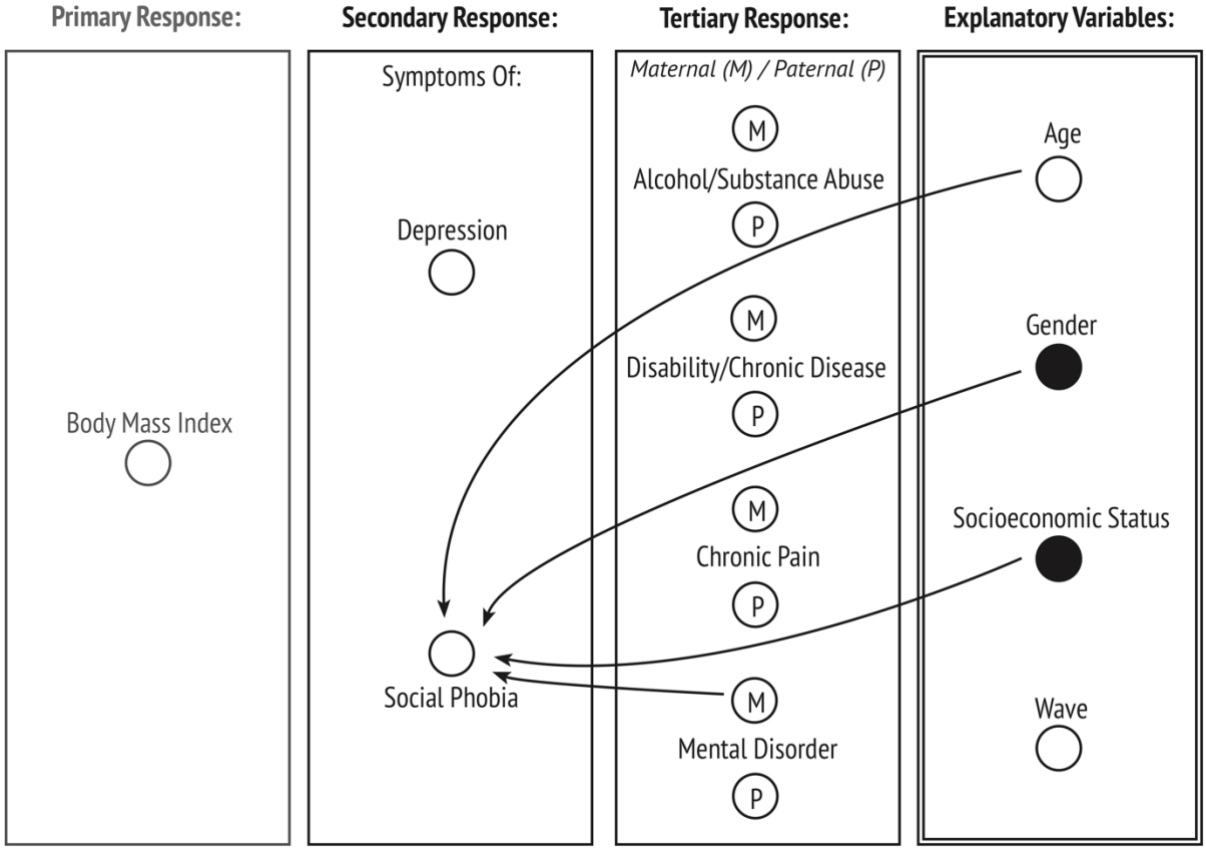

Figure 4.11 Secondary Response: Symptoms of Social Phobia—Overview of all Significant Effects and Interactions

Regarding the secondary response, *Symptoms of Social Phobia*, there are four significant predictors. There is a main effect for *Socioeconomic Status (1),* a main effect for *Maternal Mental Disorder (2),* a main effect for *Age (3),* and a main effect for *Gender (4)*. All significant effects are depicted by arrows in Figure 4.11.

4.2.2.2 *Symptoms of Social Phobia: Linear Regression Analysis*

Table 4.3 displays the regression coefficient, standard error, t-value, p-value, and total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.3 Symptoms of Social Phobia: Linear Regression Analysis

Variable	**ß**	**se$_{ß}$**	**t**	**p**
Socioeconomic Status	-0.16	0.03	-4.27	<0.001
Maternal Mental Disorder	7.68	2.10	3.65	<0.001
Age	-0.43	0.16	-2.60	0.010
Gender	-9.59	1.66	-5.76	<0.001
Constant	51.39	5.25	9.77	<0.001
Total Explained Variance: 10.04%				
Cohen's f^2: 0.112				

4.2.2.3 *Symptoms of Social Phobia: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Symptoms of Social Phobia is* depicted in Figure 4.12. The effect is statistically highly significant, with a p-value <0.001.

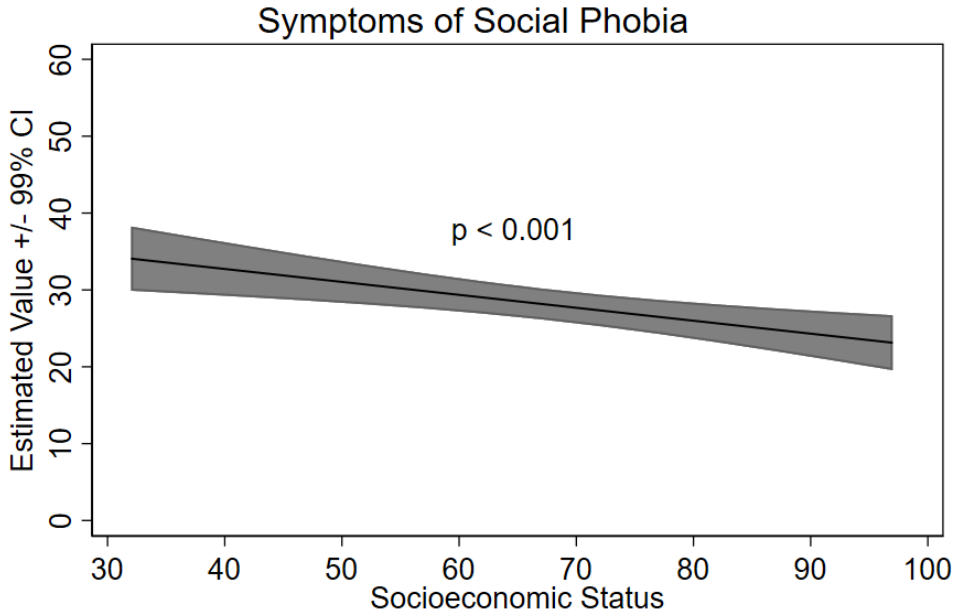

Figure 4.12 Secondary Response: Symptoms of Social Phobia Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for symptoms of social phobia are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with more reported symptoms of social phobia. The symptoms of social phobia continuously decrease as the socioeconomic status increases.

4.2.2.4 *Symptoms of Social Phobia: Main Effect for Maternal Mental Disorder*

The main effect of *Maternal Mental Disorder* in regard *to Symptoms of Social Phobia is* depicted in Figure 4.13. The effect is statistically highly significant, with a p-value of <0.001.

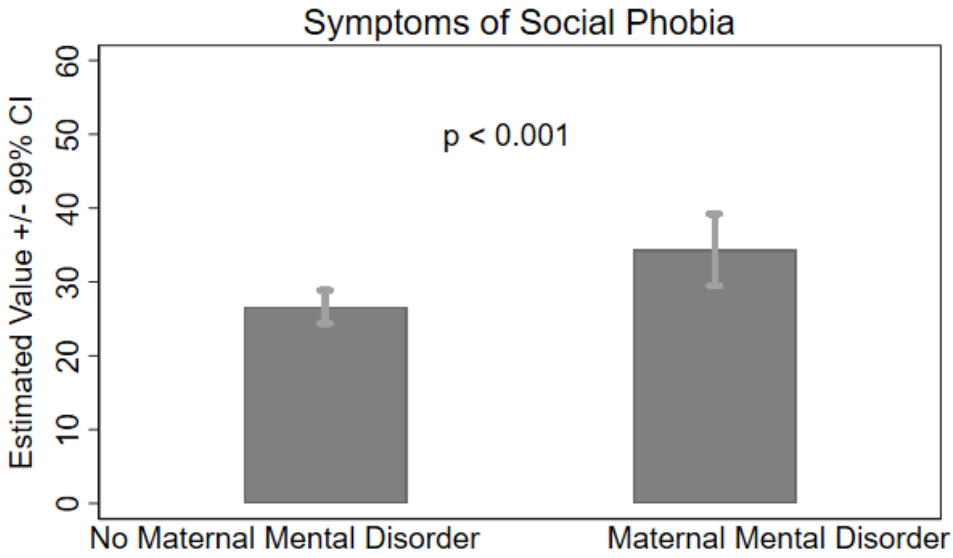

Figure 4.13 Secondary Response: Symptoms of Social Phobia Explained by the Main Effect for Maternal Mental Disorder

In the bar diagram, the response to the item *Maternal Mental Disorder* is plotted on the x-axis and the estimated value for the symptoms of social phobia are plotted on the y-axis. As seen in the figure, those participants who reported maternal mental disorders had higher rates of symptoms of social phobia compared to those who reported no maternal mental disorder.

4.2.2.5 *Symptoms of Social Phobia: Main Effect for Age*

The main effect of *Age* in regard *to Symptoms of Social Phobia is* depicted in Figure 4.14. The effect is statistically significant, with a p-value ≤0.010.

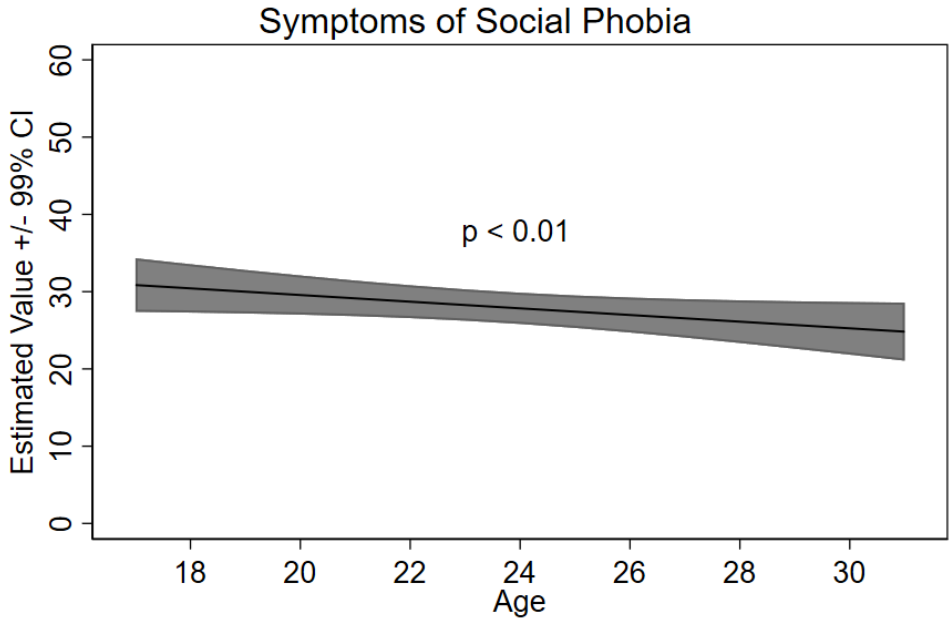

Figure 4.14 Secondary Response: Symptoms of Social Phobia Explained by the Main Effect for Age

The figure plots the age of the participants on the x-axis and the estimated value for symptoms of social phobia on the y-axis. As seen in the figure, the symptoms of social phobia continually decrease as the age of the participants increases.

4.2.2.6 *Symptoms of Social Phobia: Main Effect for Gender*

The main effect of *Gender* in regard to the BMI is depicted in Figure 4.15. The effect is statistically highly significant, with a p-value <0.001.

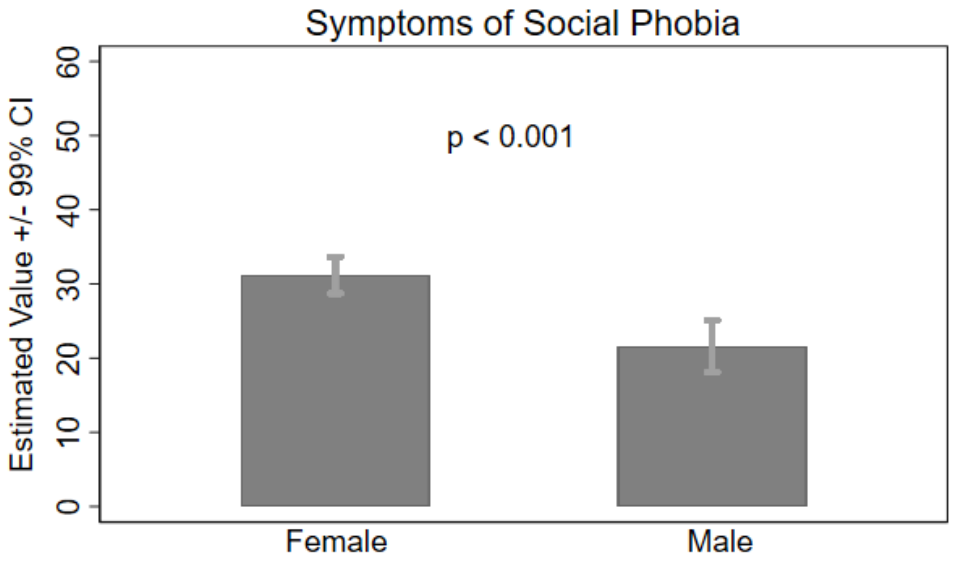

Figure 4.15 Secondary Response: Symptoms of Social Phobia Explained by the Main Effect for Gender

In the figure, gender is plotted on the x-axis the estimated values for symptoms of social phobia are plotted on the y-axis. Female participants of the survey reported higher symptoms of social phobia than male participants. Cohen's d was calculated as 0.477 and thus equivalent to a small effect size (defined as >0.20 and <0.50), as suggested by Cohen (Cohen, 1992).

4.3 *Results for the Tertiary Response:* ***Adverse Childhood Experiences***

In this subchapter all statistically significant two-way interactions, main effects and quadratic effects of the eight variables included in the tertiary response, *Childhood Adversities,* are described and graphically illustrated.

4.3.1 *Maternal and Paternal Alcohol/ Substance Abuse: Overview of all Significant Effects*

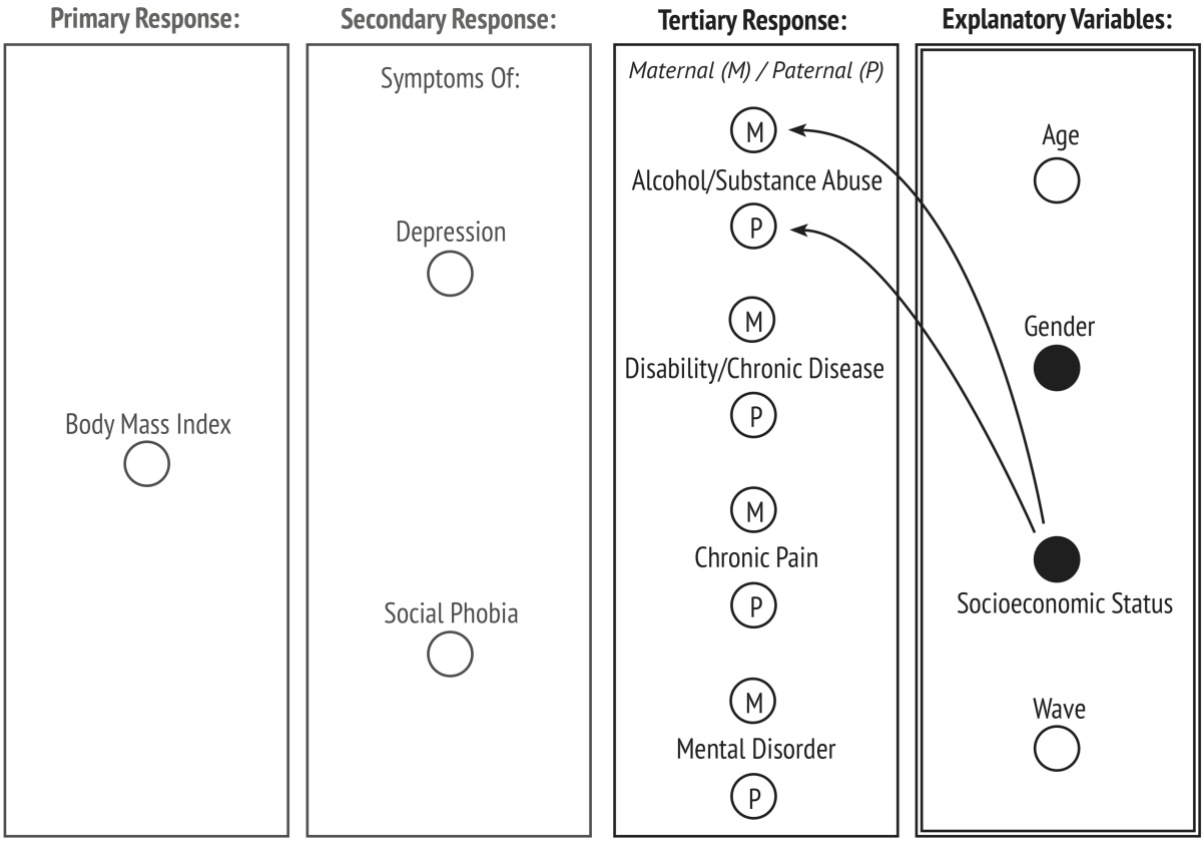

Figure 4.16 Tertiary Response: Maternal and Paternal Alcohol/ Substance Abuse—Overview of all Significant Effects

Regarding the variables *Maternal* and *Paternal Alcohol/ Substance Abuse* of the tertiary response there are a total of two significant predictors. For *Maternal Alcohol/ Substance Abuse* there is one significant predictor; a main effect for *Socioeconomic Status* (1). For *Paternal Alcohol/ Substance Abuse* there is also one significant predictor; a quadratic effect for *Socioeconomic Status* (2). All significant effects are illustrated by arrows in Figure 4.16.

4.3.1.1 *Maternal Alcohol/ Substance Abuse: Linear Regression Analysis*

Table 4.4 displays the regression coefficient, standard error, t-value, p-value, and total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.4 Maternal Alcohol Abuse: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-4.00	<0.001
Constant	0.20	0.03	6.27	<0.001
Total Explained Variance: 2.18%				
Cohen's f^2: 0.022				

4.3.1.2 *Maternal Alcohol/ Substance Abuse: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Maternal Alcohol Abuse* is depicted in Figure 4.17. The effect is statistically highly significant, with a p-value <0.001.

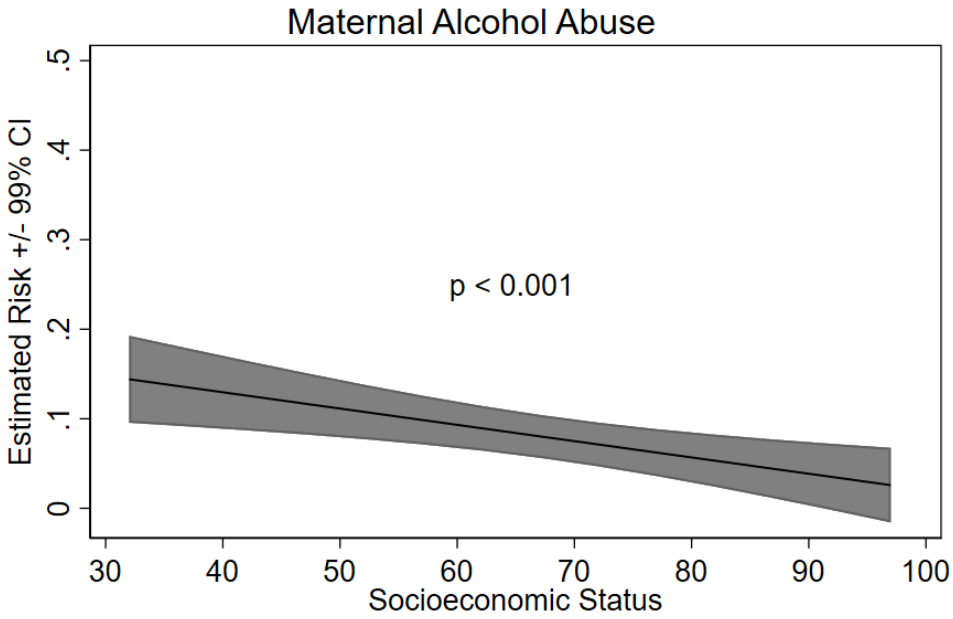

Figure 4.17 Tertiary Response: Maternal Alcohol Abuse Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for maternal alcohol abuse during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported maternal alcohol abuse. The reported maternal alcohol abuse continuously decreases as the socioeconomic status increases.

4.3.1.3 *Paternal Alcohol/ Substance Abuse Linear Regression Analysis*

Table 4.5 displays the regression coefficient, standard error, p-value, t-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is equivalent to a small effect size, defined as >0.02 as suggested by Cohen (Cohen, 1992).

Table 4.5 Paternal Alcohol Abuse: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	-0.02	0.03	-6.69	<0.001
Socioeconomic Status^2	0.00	0.00	5.45	<0.001
Constant	0.78	0.83	9.38	<0.001
Total Explained Variance: 9.42%				
Cohen's f^2: 0.104				

4.3.1.4 *Paternal Alcohol/ Substance Abuse: Quadratic Effect for Socioeconomic Status*

The quadratic effect of *Socioeconomic Status* in regard to *Paternal Alcohol Abuse* is depicted in Figure 4.18. The effect is statistically highly significant, with a p-value <0.001.

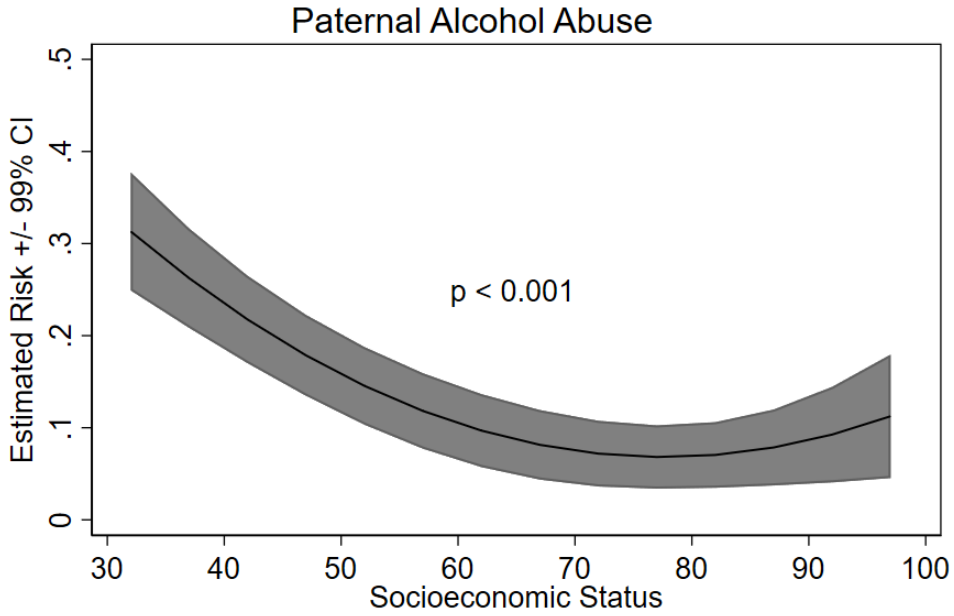

Figure 4.18 Tertiary Response: Paternal Alcohol Abuse Explained by the Quadratic Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for maternal alcohol abuse during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status during childhood and adolescence is associated with higher values of reported paternal alcohol abuse. The apparent repeated increase of paternal alcohol abuse at the at the high end of the socioeconomic status, however, is an artefact due to fitting a quadratic term.

4.3.2 *Maternal and Paternal Disability/Chronic Disease: Overview of all Significant Effects*

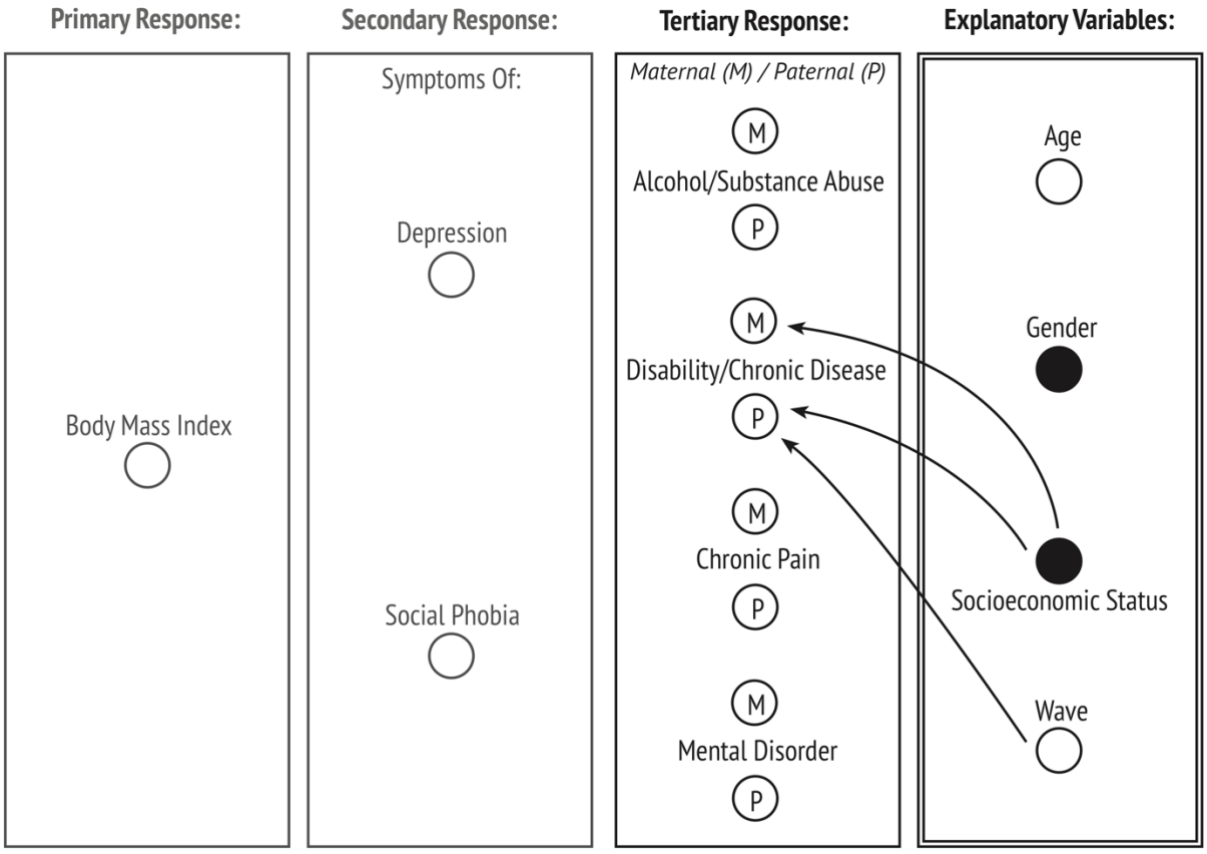

Figure 4.19 Tertiary Response: Maternal and Paternal Disability/ Chronic Disease—Overview of all Significant Effects

Regarding the variables *Maternal* and *Paternal Disability/Chronic Disease* of the tertiary response there are a total of three significant predictors. For *Maternal Disability/Chronic Disease* there is one significant predictor; a main effect for *Socioeconomic Status* (1). For *Paternal Disability/Chronic Disease* there are two significant predictors; an interaction between *Wave* and *Socioeconomic Status* (2 and 3). All significant effects are illustrated by arrows in Figure 4.19.

4.3.2.1 *Maternal Disability/Chronic Disease: Linear Regression Analysis*

Table 4.6 displays the regression coefficient, standard error, t-value, p-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is <0.02 and thus, as suggested by Cohen, indicates that there is no effect (Cohen, 1992).

Table 4.6 Maternal Chronic Disease: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-3.51	<0.001
Constant	0.22	0.04	6.05	<0.001
Total Explained Variance: 1.69%				
Cohen's f^2:0.017				

4.3.2.2 *Maternal Chronic Disease Explained by the Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Maternal Chronic Disease* is depicted in Figure 4.20. The effect is statistically highly significant, with a p-value <0.001.

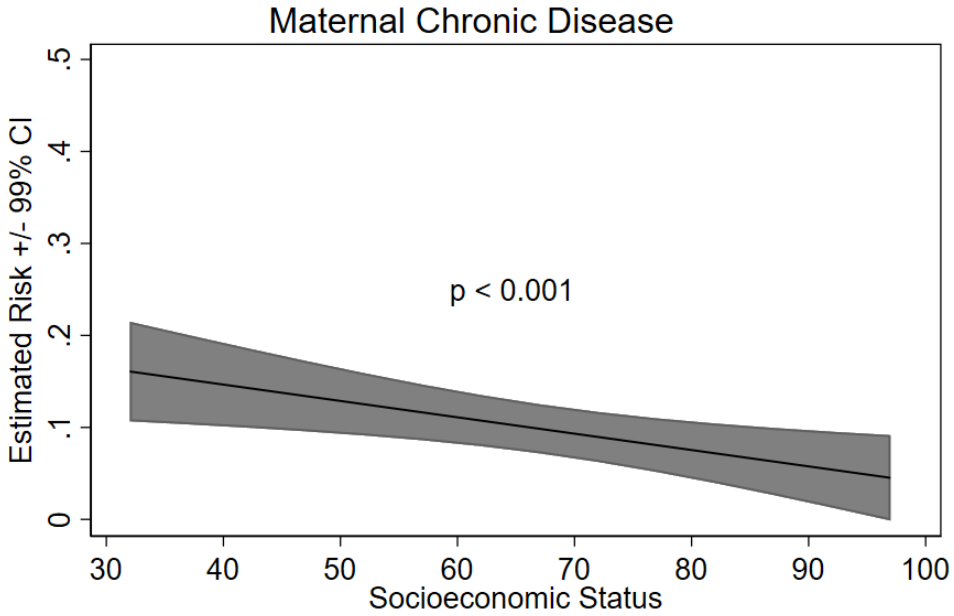

Figure 4.20 Tertiary Response: Maternal Chronic Disease Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for maternal chronic disease during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported maternal chronic disease. The reported maternal chronic disease continuously decreases as the socioeconomic status increases.

4.3.2.3 *Paternal Disability/ Chronic Disease Linear Regression Analysis*

Table 4.7 displays the regression coefficient, standard error, p-value, t-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.7 Paternal Chronic Disease: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-0.98	0.327
Wave	0.26	0.07	3.58	<0.001
Socioeconomic Status * Wave	0.00	0.00	-3.08	0.002
Constant	0.13	0.06	2.33	0.020
Total Explained Variance: 5.31%				
Cohen's f^2:0.056				

4.3.2.4 *Paternal Chronic Disease: Interaction between Socioeconomic Status and Wave*

The two-way interaction between *Socioeconomic Status* and *Wave* in regard to the tertiary response, *Paternal Chronic Disease,* is depicted in Figure 4.21. With a p-value ≤0.002, the interaction is statistically significant.

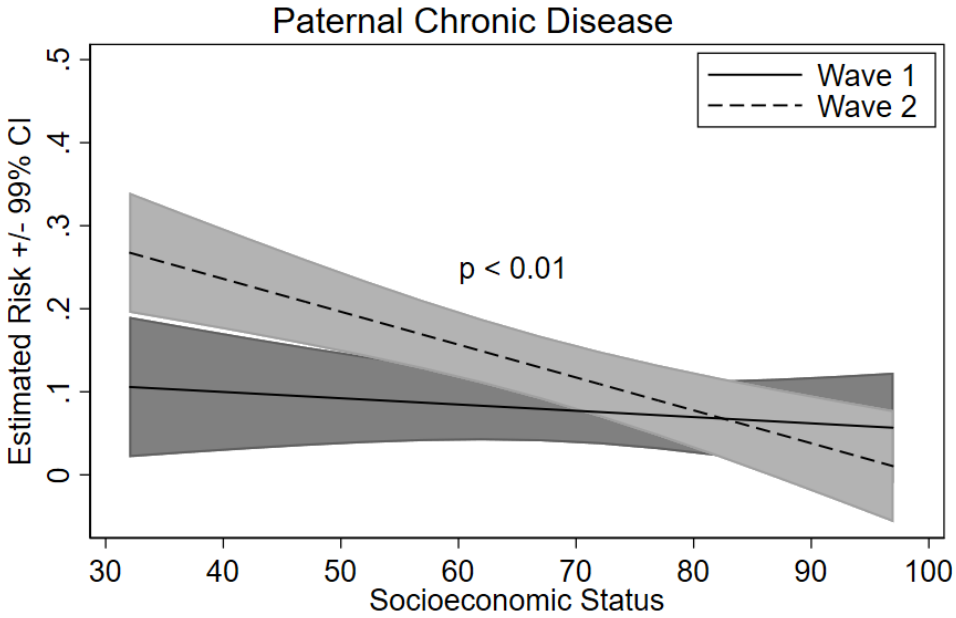

Figure 4.21 Tertiary Response: Paternal Chronic Disease Explained by the Interaction between Socioeconomic Status and Wave

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for paternal chronic disease during the first 14 years of life are plotted on the y-axis. The solid line represents participants in wave 1, while the dashed line represents those in wave 2. As seen in the figure, socioeconomic status is negatively associated with reported paternal chronic disease in both waves. The reported paternal chronic disease continuously decreases as the socioeconomic status increases. Notably however, the decline is significantly steeper in the participants of wave 2.

4.3.3 *Maternal and Paternal Chronic Pain: Overview of all Significant Effects*

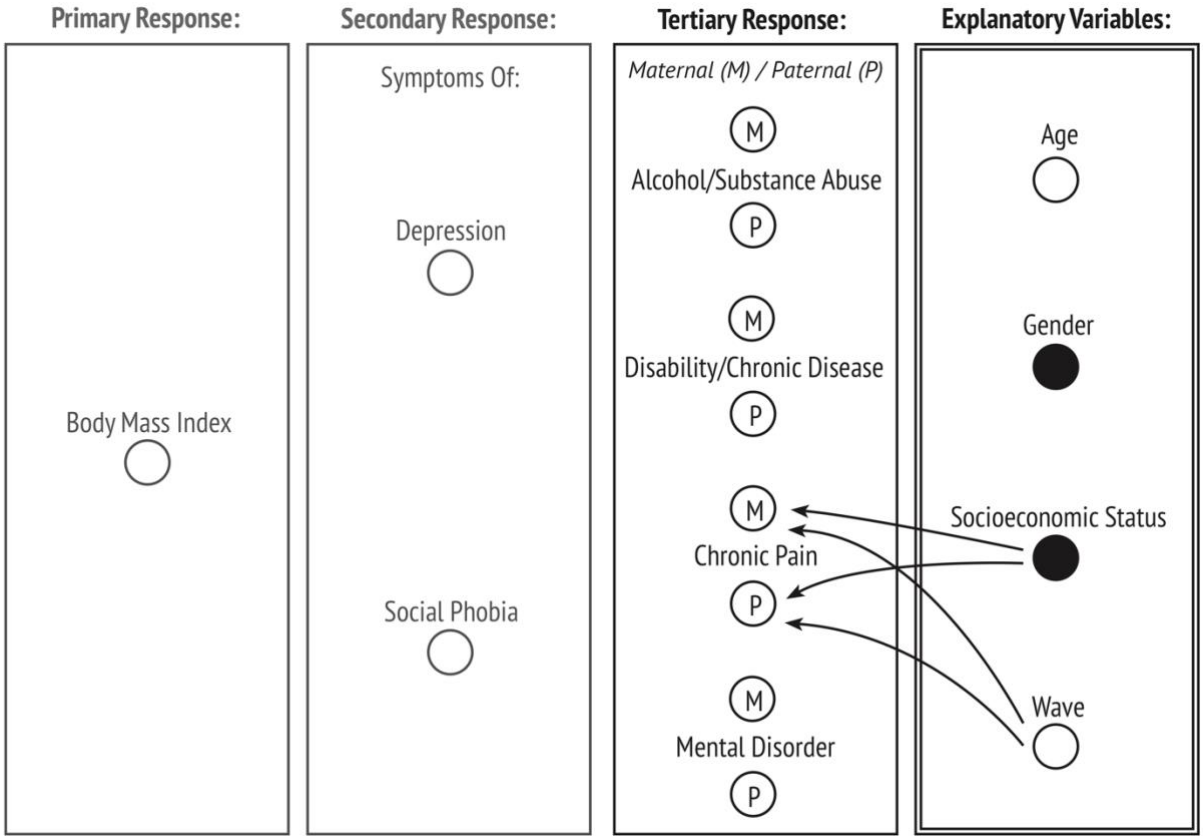

Figure 4.22 Tertiary Response: Maternal and Paternal Chronic Pain—Overview of all Significant Effects

Regarding the variables *Maternal* and *Paternal Chronic Pain* of the tertiary response there are a total of four significant predictors. For *Maternal Chronic Pain* there are two significant predictors; a main effect for *Socioeconomic Status* (1) and a main effect for *Wave* (2). Similarly, there are two significant predictors for *Paternal Chronic Pain*; a main effect for *Socioeconomic Status* (3) and a main effect for *Wave* (4). All significant effects are illustrated by arrows in Figure 4.22.

4.3.3.1 *Maternal Chronic Pain: Linear Regression Analysis*

Table 4.8 displays the regression coefficient, standard error, t-value, p-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.8 Maternal Chronic Pain: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-3.75	<0.001
Wave	0.06	0.02	2.92	0.004
Constant	0.19	0.04	5.13	<0.001
Total Explained Variance: 3.35%				
Cohen's f^2: 0.035				

4.3.3.2 *Maternal Chronic Pain: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Maternal Chronic Pain* is depicted in Figure 4.23. The effect is statistically highly significant, with a p-value <0.001.

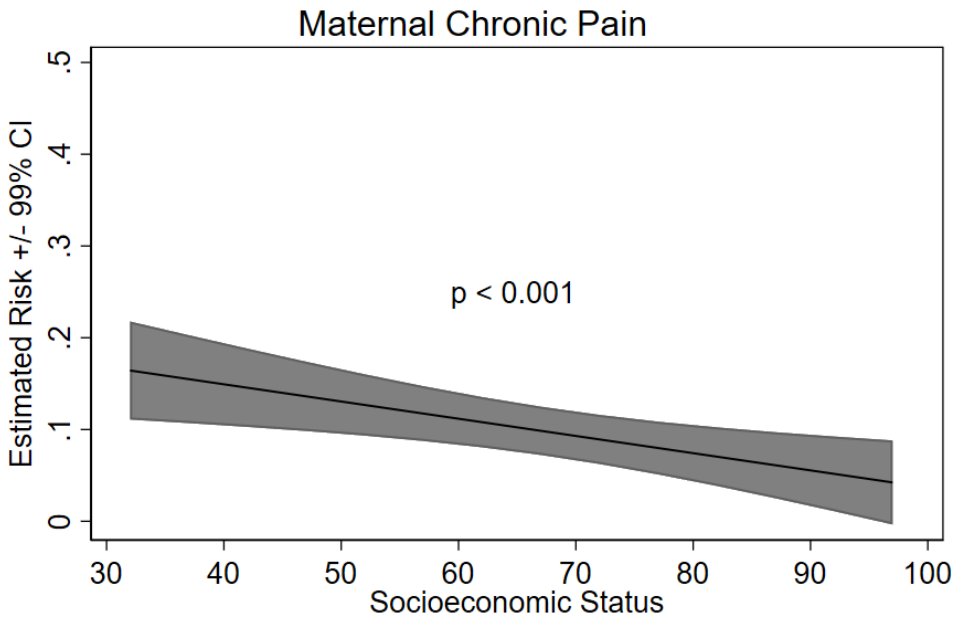

Figure 4.23 Tertiary Response: Maternal Chronic Pain Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for maternal chronic pain during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported maternal chronic pain. The reported maternal chronic pain continuously decreases as the socioeconomic status increases.

4.3.3.3 *Maternal Chronic Pain: Main Effect for Wave*

The main effect of *Wave* in regard to *Maternal Chronic Pain is* depicted in Figure 4.24. The effect is statistically significant, with a p-value ≤0.004.

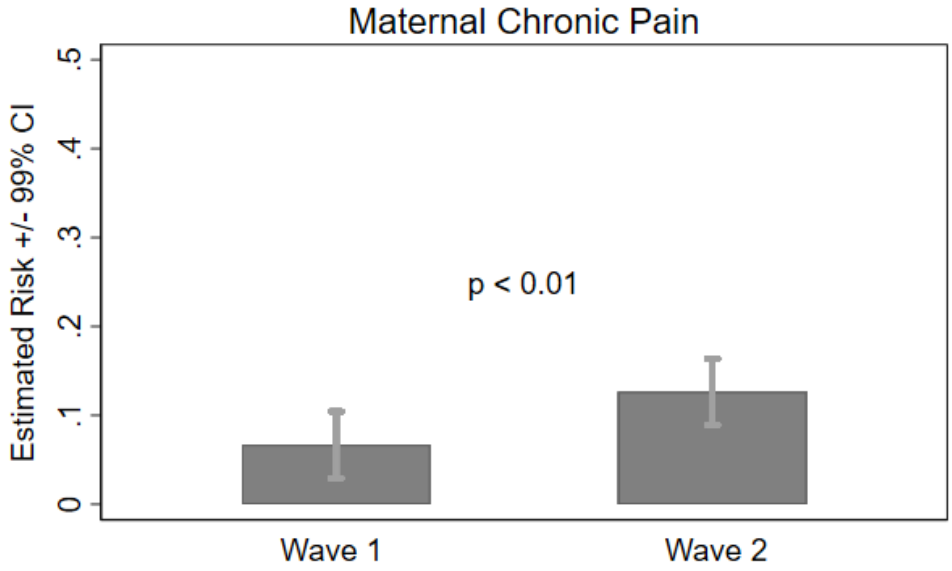

Figure 4.24 Tertiary Response: Maternal Chronic Pain Explained by the Main Effect for Wave

In the bar diagram, the wave is plotted on the x-axis and the estimated values for maternal chronic pain during the first 14 years of life are plotted on the y-axis. As seen in the figure, participants in wave 1 were less likely to report maternal chronic pain than those in wave 2. Cohen's d was calculated as -0.242 and thus equivalent to a small effect size (defined as >0.20 and <0.50), as suggested by Cohen (Cohen, 1992).

4.3.3.4 *Paternal Chronic Pain: Linear Regression Analysis*

Table 4.9 displays the regression coefficient, standard error, p-value, t-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.9 Paternal Chronic Pain: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-4.09	<0.001
Wave	0.05	0.02	2.65	0.008
Constant	0.21	0.04	5.62	<0.001
Total Explained Variance: 3.50%				
Cohen's f^2: 0.036				

4.3.3.5 *Paternal Chronic Pain: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Paternal Chronic Pain* is depicted in Figure 4.25. The effect is statistically highly significant, with a p-value <0.001.

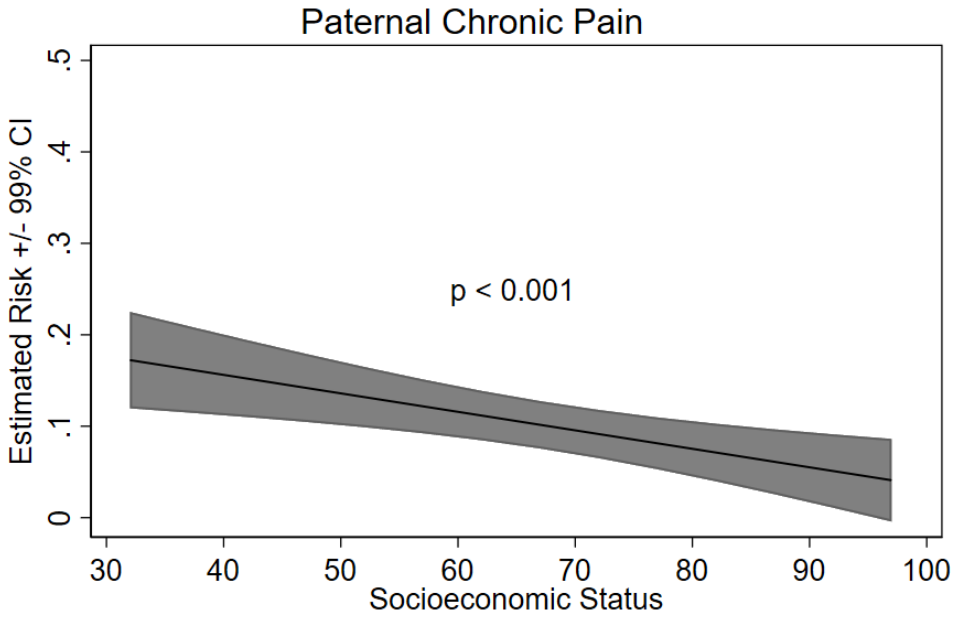

Figure 4.25 Tertiary Response: Paternal Chronic Pain Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for paternal chronic pain during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported paternal chronic pain. The reported paternal chronic pain continuously decreases as the socioeconomic status increases.

4.3.3.6 *Paternal Chronic Pain: Main Effect for Wave*

The main effect for *Wave* in regard to *Paternal Chronic Pain is* depicted in Figure 4.26. The effect is statistically significant, with a p-value ≤0.008.

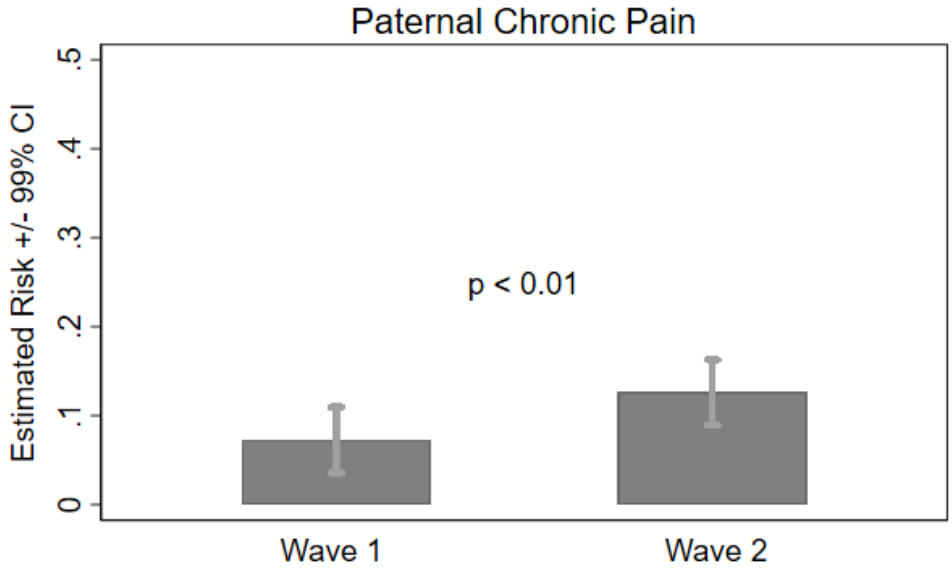

Figure 4.26 Tertiary Response: Paternal Chronic Pain Explained by the Main Effect for Wave

In the bar diagram, the wave is plotted on the x-axis and the estimated values for paternal chronic pain during the first 14 years of life are plotted on the y-axis. As seen in the figure, participants in wave 1 were less likely to report paternal chronic pain than those in wave 2. Cohen's d was calculated as -0.224 and thus equivalent to a small effect size (defined as >0.20 and <0.50), as suggested by Cohen (Cohen, 1992).

4.3.4 *Maternal and Paternal Mental Disorder: Overview of all Significant Effects*

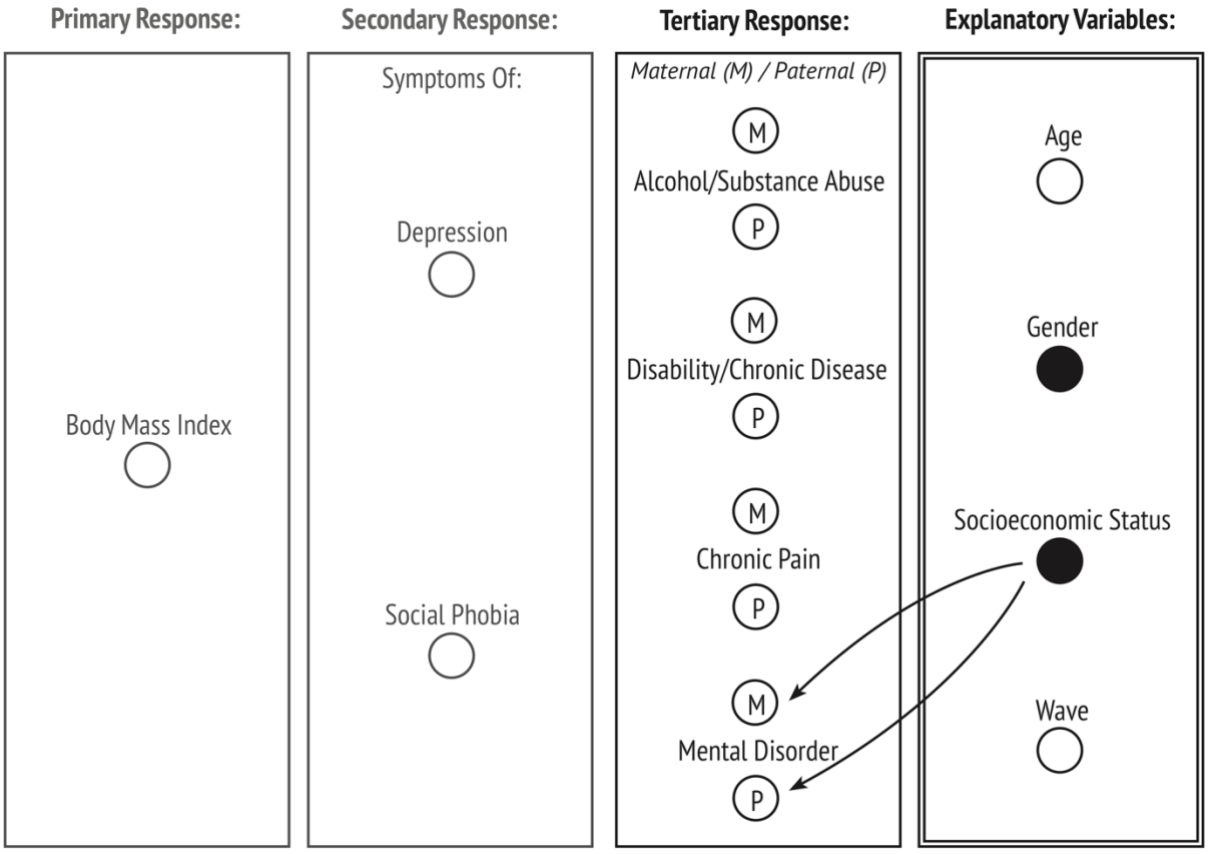

Figure 4.27 Tertiary Response: Maternal and Paternal Mental Disorder—Overview of all Significant Effects

Regarding the variables *Maternal* and *Paternal Mental Disorder* of the tertiary response there are a total of two significant predictors. For *Maternal Mental Disorder* there is one significant predictor; a main effect for *Socioeconomic Status* (1). Similarly, there is also one significant predictor for *Paternal Mental Disorder*; a main effect for *Socioeconomic Status* (2). All significant effects are illustrated by arrows in Figure 4.27.

4.3.4.1 *Maternal Mental Disorder: Linear Regression Analysis*

Table 4.10 displays the regression coefficient, standard error, t-value, p-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is >0.02 and thus, as suggested by Cohen, equivalent to a small effect size (Cohen, 1992).

Table 4.10 Maternal Mental Disorder: Linear Regression Analysis

Variable	**ß**	**se$_{ß}$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-4.49	<0.001
Constant	0.39	0.05	8.07	<0.001
Total Explained Variance: 2.74%				
Cohen's f^2: 0.028				

4.3.4.2 *Maternal Mental Disorder: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Maternal Mental Disorder* is depicted in Figure 4.28. The effect is statistically highly significant, with a p-value <0.001.

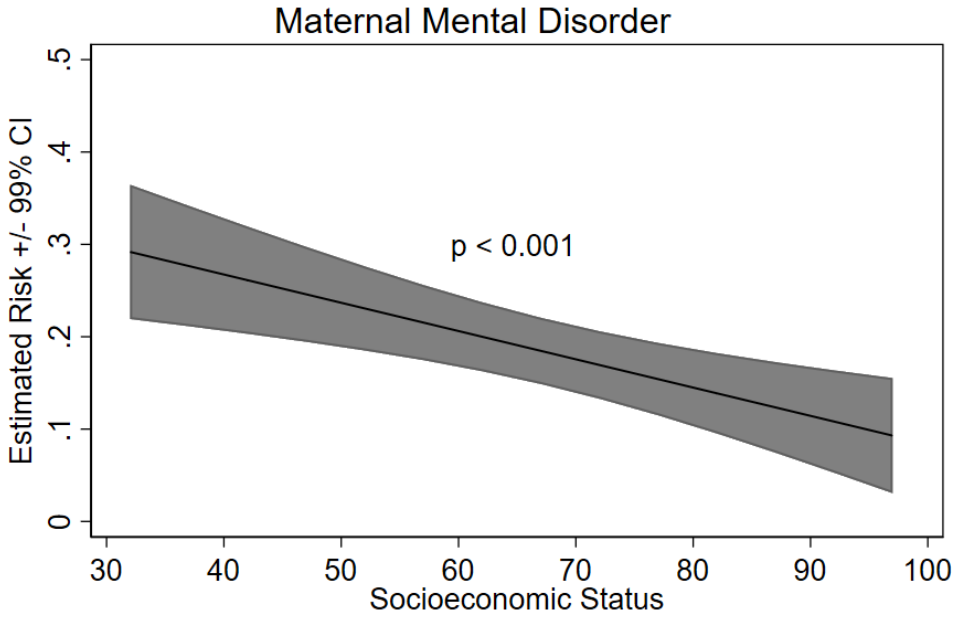

Figure 4.28 Tertiary Response: Maternal Mental Disorder Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for maternal mental disorder during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported maternal mental disorder. The reported maternal mental disorder continuously decreases as the socioeconomic status increases.

4.3.4.3 *Paternal Mental Disorder: Linear Regression Analysis*

Table 4.11 displays the regression coefficient, standard error, p-value, t-value, and the total explained variance for the statistically significant results of the linear regression. The value of Cohen's f^2 is <0.02 and thus, as suggested by Cohen, indicates that there is no effect (Cohen, 1992).

Table 4.11 Paternal Mental Disorder: Linear Regression Analysis

Variable	**ß**	**$se_ß$**	**t**	**p**
Socioeconomic Status	0.00	0.00	-2.85	0.004
Constant	0.21	0.04	5.58	<0.001
Total Explained Variance: 1.12%				
Cohen's f^2: 0.011				

4.3.4.4 *Paternal Mental Disorder: Main Effect for Socioeconomic Status*

The main effect for *Socioeconomic Status* in regard *to Paternal Mental Disorder* is depicted in Figure 4.29. The effect is statistically significant, with a p-value ≤0.004.

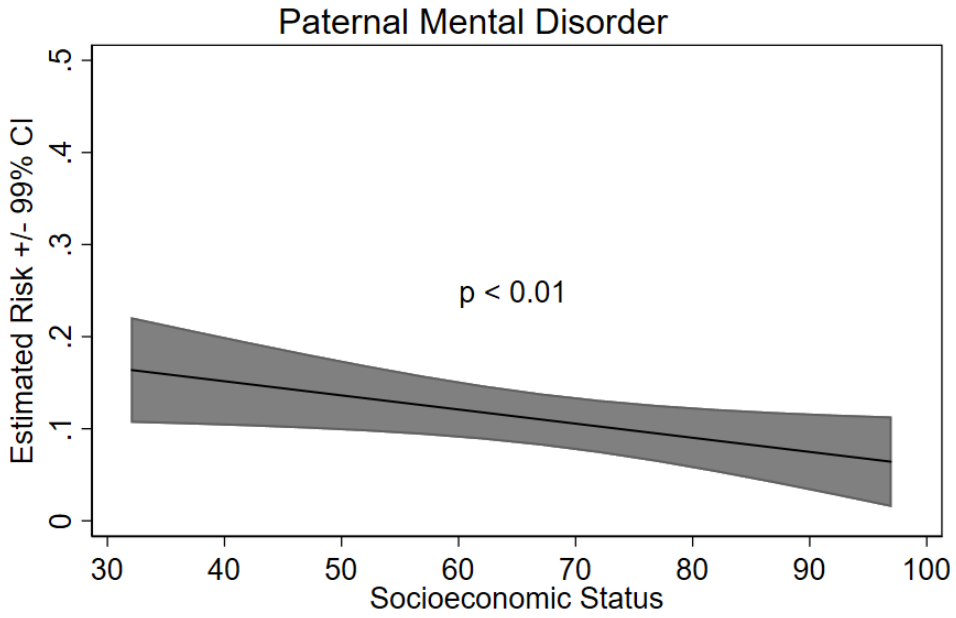

Figure 4.29 Tertiary Response: Paternal Mental Disorder Explained by the Main Effect for Socioeconomic Status

In the figure, the estimated values for socioeconomic status are plotted on the x-axis and the estimated values for paternal mental disorder during the first 14 years of life are plotted on the y-axis. As seen in the figure, a lower socioeconomic status is associated with higher values of reported paternal mental disorder. The reported paternal mental disorder continuously decreases as the socioeconomic status increases.

4.4 *Results for the Residuals*

In this chapter, the Person's correlations of the residuals are illustrated for the secondary and tertiary responses (see chapter 3.6, Presentation of the Results, for details). As depicted in Table 4.12 a significant correlation was found between all variables of the secondary and tertiary response, with the sole exception of *Paternal Chronic Pain* and *Maternal Alcohol/ Substance Abuse*. Most of the correlations have small (>0.10) to medium (>0.30) effect sizes. There are three correlations that have a large effect size (>0.05): one correlation regarding the secondary response (*Symptoms of Depression* and *Symptoms of Social Phobia)* and two correlations regarding the tertiary response (*Maternal Chronic Pain* and *Maternal Chronic Disease* as well as *Paternal Chronic Pain* and *Paternal Chronic Disease*) (Cohen, 1992).

Table 4.12 Residuals: Pearson's Correlation Coefficients for the Secondary and Tertiary Response

Secondary Response								
		Symptoms of Depression						
Symptoms of Social Phobia		0.54						
Tertiary Response								
		Alcohol/ Substance Abuse		**Disability/ Chronic Disease**		**Chronic Pain**		**Mental Disorder**
		Maternal	**Paternal**	**Maternal**	**Paternal**	**Maternal**	**Paternal**	**Maternal**
Alcohol/ Substance Abuse	**Paternal**	0.26						
Disability/ Chronic Disease	**Maternal**	0.20	0.17					
	Paternal	0.10	0.22	0.39				
Chronic Pain	**Maternal**	0.15	0.13	0.58	0.28			
	Paternal	n.s.	0.11	0.29	0.52	0.34		
Mental Disorder	**Maternal**	0.35	0.18	0.37	0.14	0.29	0.13	
	Paternal	0.15	0.31	0.20	0.45	0.15	0.26	0.28

Note: In the above table, only significant correlations are displayed. Correlations with a p-value greater than 0.01 are labelled as not significant (n.s.).

The significant Pearson's correlations between the variables of the secondary response as well as the tertiary response are graphically illustrated by dashed lines in figure 4.30 below.

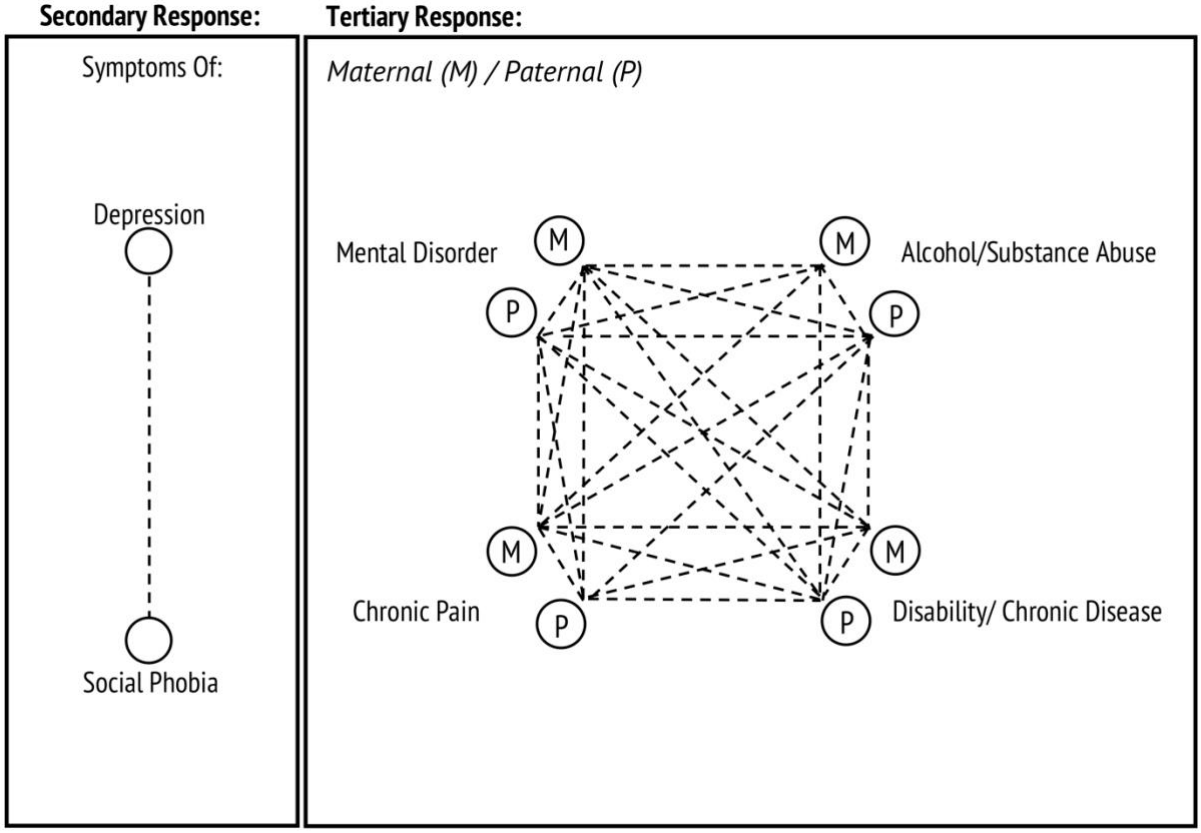

Figure 4.30 Graphic Representation of the Pearson's Correlations Coefficient

As illustrated in both table 4.12 and figure 4.30, all but one of the correlations are significant. The single Pearson's correlation >0.01, and thus non-significant correlation, is between *Paternal Chronic Pain* and *Maternal Alcohol/Substance Abuse*.

5 Discussion

5.1 *Interpretation of the Hypotheses*

The four hypotheses at the center of this thesis revolve around the primary response, Body Mass Index (BMI), and are presented in chapter 2.3. Fourteen variables were examined as predictors of BMI and, of these, four were found to be statistically significant at the significance level of 0.01. The results indicate that a lower socioeconomic status (SES), older age, male gender, and experience of paternal disability or chronic disease during childhood predicted a higher BMI. Only two of these variables, SES and paternal disability or chronic disease during childhood, are relevant for the interpretation of the hypotheses, which are examined in this section. Additional results for the primary response are discussed further on in section 5.2.1. Moreover, in the process of a comprehensive statistical analysis, all variables were also analyzed regarding the secondary and tertiary response, these are presented in sections 5.2.2 and 5.2.3.

5.1.1 *Interpretation of the Results for **Hypothesis 1***

Hypothesis 1: Individuals with a lower socioeconomic status are at a higher risk of being overweight or obese.

The results (presented in chapter 4.1.1.4) demonstrate a statistically significant quadratic effect of socioeconomic status (SES) on BMI. As such, a low SES is significantly associated with a high BMI and, therefore, the null hypothesis was rejected in favor of the alternative hypothesis, hypothesis 1. However, as indicated by the quadratic effect, SES only predicted high BMI for individuals with a low SES while the same was not the case for individuals with a medium to high SES.

These results are in line with current scientific literature. As discussed in chapter 2.2.1, there is a bidirectional association between a low SES and a high BMI—individuals with a lower SES are disproportionately affected by overweight and obesity. This association could also be found in our sample, consisting mainly of young university students. Those individuals who reported a low SES during the first fourteen years of life were more likely to be currently overweight or obese. The magnitude of the effect, however, is smaller than expected. This is perhaps due to the young mean age of the participants, which was approximately 24 years in wave 1 and 23 years in wave 2. In addition, sample bias may have affected the results—due to the sociodemographic structure of the university student body in Germany, it is plausible that individuals with lower SES are underrepresented (Autorengruppe Bildungsberichterstattung, 2020). As a result of the cross-sectional study design, it is not possible to interpret the causality of these results, only their correlation. Previous research, however, has indicated both bidirectionality and causality of the link between BMI and SES (Sobal and Stunkard, 1989, McLaren, 2007, Newton et al., 2017, Kim

and von dem Knesebeck, 2018). The results found in this study support previous research and highlight the importance of our environment in fostering obesity.

5.1.2 *Interpretation of the Results for* ***Hypothesis 2***

Hypothesis 2: Individuals who experience symptoms of depression are at a higher risk of being overweight or obese.

The results (presented in chapter 4.1.1.1) did not find symptoms of depression to be a significant predictor of BMI. Therefore, the null hypothesis was retained and the alternative hypothesis, hypothesis 2, was rejected. These results are contrary to the majority of results in current scientific literature, which indicate a strong bidirectional association between BMI and symptoms of depression (Luppino et al., 2010, de Wit et al., 2010a, Mannan et al., 2016). The lack of significant effect is unexpected. One possible explanation for the lack of association is found in the statistical analysis; due to the linear regression analysis, in which many possible confounders were tested, it is possible that other significant predictors of BMI may have masked the bivariate association between BMI and depression. On the other hand, it is possible that studies which do not control for these confounders may detect associations that are not relevant, thus reporting false results. Further research with a more representative sample selection is needed to answer this question.

Sample selection is this study's primary limitation. Consequently, the most plausible explanation for the absence of a significant correlation between symptoms of depression and BMI is sample bias. A study has indicated that German medical students experience higher rates of depressive disorders and anxiety disorders than the general German population (Seliger and Brähler, 2007). Yet individuals with symptoms of depression may be particularly underrepresented in this sample, as indicated by the low mean values on the scale for symptoms of depression. These are presented in table 3.3: on a scale from 0–100 the mean values for symptoms of depression are approximately 18 in wave 1 and 26 in wave 2. In the present survey, participants were asked to report symptoms of depression during the previous two weeks of their life. However, it is likely that individuals currently experiencing a depressive episode lack the energy and motivation to take part in an online survey, this may be exacerbated in individuals with severe symptoms of depression. Research demonstrates that, as the severity of depressive disorders increased, BMI also increased (Pratt and Brody, 2014). Thus, inadvertent exclusion of individuals with severe mental health disorders may negatively affect the generalizability of this result.

In addition, the sample selection affected the mean age of the participants included; participants in this study had a mean age of approximately 24 years in wave 1 and 23 years in wave 2 (detailed in table 3.3). Although the age of onset for depressive disorders has a wide variation, symptoms of depression commonly develop from mid-to-late adolescents through early adulthood (Otte et al., 2016) and the prevalence of depressive disorders continuously

increases with increasing age (Steffen et al., 2020). Moreover, the median age of onset for both men and women is around 25 years (Otte et al., 2016), and thus slightly higher than the mean age in this sample. Therefore, some of the surprising findings in this study may be explained by the young age of the participants.

The low mean score for depressive symptoms in this sample may also be explained by a **response bias**, due to the anonymous survey situation, it is possible that symptoms of depression were underreported (Rogler et al., 2001). Moreover, as a result of the self-reported data, although symptoms depression and social phobia could be identified, it was not possible to diagnose the clinical conditions themselves. In light of these limitations, future research should investigate if similar results can be found in a more representative cohort of young adults.

5.1.3 *Interpretation of the Results for **Hypothesis 3***

Hypothesis 3: Individuals who experience symptoms of social phobia are at a higher risk of being overweight or obese.

The results (presented in chapter 4.1.1.1) did not find symptoms of social phobia to be a significant predictor of BMI. Thus, the null hypothesis was retained and the alternative hypothesis, hypothesis 3, was rejected. As detailed in section 2.2.4.2, the results on the association between BMI and symptoms of anxiety disorders in scientific literature are inconclusive. Although two recent meta-analyses found a positive association, they also reported high levels of heterogeneity (Gariepy et al., 2010, Amiri and Behnezhad, 2019). In addition, a meta-analysis that examined the effects of stress and anxiety specifically in university students also found that results concerning the association to BMI were ambiguous (Haidar et al., 2018). Accordingly, the lack of significant association between symptoms of social phobia and overweight and obesity are in line with some of the current scientific literature. Moreover, some sources have described a u-shaped curve between anxiety disorders and bodyweight (Scott et al., 2008, DeJesus et al., 2016, Haidar et al., 2018). In the statistical analysis, non-linear associations were also tested, yet this study was unable to confirm a u-shaped curve between BMI and symptoms of social phobia. This result is in concordance with evidence from a recent study on anxiety disorders conducted in Germany, which was also unable to confirm a u-shaped curve between anxiety disorders and BMI (Herhaus et al., 2020).

These findings must be seen in the light of some limitations. Parallel to the limitations discussed above for symptoms of depression, symptoms of social phobia were not clinically diagnosed, but self-reported. It is possible that response bias in the anonymous online survey caused symptoms of social phobia to be underreported. Anxiety disorders tend to develop early in life (Craske et al., 2017) and the prevalence of social phobia decreases with age (Jacobi et al., 2014). Therefore, in contrast to the potential negative effects of the

participants' young age discussed previously regarding symptoms of depression, the young mean age of the participants, should not negatively affect the results regarding anxiety disorders.

The low mean values on the scale for symptoms of social phobia, indicate that individuals with symptoms of social phobia may be underrepresented in this study. As presented in table 3.3, on a scale from 0–100 the mean values for symptoms of social phobia are approximately 28 in both waves. As the recruitment of participants took place in a university setting, individuals with severe social phobia may be particularly underrepresented—as, due to the nature of their disorder, they may be less likely to be present at university. Inadvertent exclusion of individuals with severe social phobia may have negatively impacted the generalizability of the results. Considering these limitations, the results found in this study, indicate that there is no significant correlation and no u-shaped curve between BMI and symptoms of social phobia. Thus, these results add weight to the body of evidence indicating that anxiety disorders do not predict BMI.

5.1.4 *Interpretation of the Results for* ***Hypothesis 4***

Hypothesis 4: Individuals who experienced adverse childhood experiences are at a higher risk of being overweight or obese.

Eight different adverse childhood experiences (ACEs) were examined in regard to the primary response, *BMI*. Of these eight, only one variable, *Paternal Disability/ Chronic Disease*, significantly predicted a higher BMI. Therefore, the null hypothesis was retained and the alternative hypothesis, hypothesis 4, was rejected. These results are contrary to most scientific literature, which despite considerable heterogeneity, consistently reports a positive association between ACEs and the development of overweight and obesity (as discussed in section 2.2.5.2).

Several factors may explain the divergence of this study's results from prior scientific literature. It is possible that the effects of ACEs in scientific literature are overestimated, as confounders such as SES, gender, and age may not be considered. Studies not controlling for these confounders may report false results, i.e. detect associations that are not relevant. Further research is needed to answer this question. In this study, however, SES, gender, and age were partialled out and, thus, through linear regression analysis these confounders were taken into account. Moreover, as indicated by Cohen's f^2, which is approximately 0.2 and thus equivalent to a medium effect size (Cohen, 1992), as well as the total explained variance, which is roughly 17% (details presented in table 4.1), the variables examined in this study explain a considerable amount of variance for the primary response, BMI.

Despite the relatively large sample size (n=718), it is also plausible that the effects in this sample were simply too weak to show a significant association. Moreover, the sample may not be representative of the general population, as recruitment for the study took place

in a university setting. Due to the fact that ACEs are more common in lower-income households (Witt et al., 2017) and individuals with lower SES are underrepresented in German universities (Autorengruppe Bildungsberichterstattung, 2020), it is possible that ACEs are underrepresented in this sample. As presented in table 3.3, on a scale from 0–100 the mean values for SES were approximately 70 in wave 1 and 66 in wave 2 and, thus, considerably higher than the theoretical mid-point on the scale. This indicates that the discrepancies in SES in university enrollment may have similarly affected the composition of this sample.

The only significant effect of ACEs on BMI is an interaction between the two variables *Paternal Disability/ Chronic Disease* and *Age*, the implications of this effect are interpreted below, in section 5.2.1. Notably, there are plausible biological mechanisms through which paternal disease and disability may affect offspring's BMI, indicating that the psychological effects of ACEs on overweight and obesity may have been overestimated in prior literature. Nevertheless, these findings must be viewed in light of several limitations and the results should be replicated in a more representative sample of the German population before further interpretation.

5.2 *Interpretation of Additional Results*

5.2.1 *Interpretation of Additional Results for the Primary Response:* ***Body Mass Index***

In addition to the significant quadratic effect for *Socioeconomic Status* (see the interpretation of hypothesis 1 in section 5.1.1), a significant **interaction between the variables *Paternal Chronic Disease* and *Age*** as well as a significant **main effect for *Gender*** were found in regard to the primary response BMI, these are interpreted below. There was no significant association between *Wave* and *BMI*, indicating that the results for the primary response were stable across both waves of data collection.

In this study, male participants reported a higher BMI than female participants (results are presented in section 4.1.1.5), although the equivalent effect size was small (with a Cohen's d of -0.495). These results reflect data previously published in the Lancet (Ng et al., 2014), which found that German men were more likely to be overweight than women both as adolescents and as adults. According to this data, in Germany, 20.5% of men under the age of 20 and 64.3% of men over the age of 20 are overweight, compared to 19.4% and 49.0% of women respectively. The rates of obesity are 5.5% for men under the age of 20 and 21.9% for men over the age of 20, compared to 5.3% and 22.5% for women respectively (Ng et al., 2014).

The results in this study indicate, that BMI increases with age and that this effect is exacerbated when paternal chronic disease or disability was present during childhood

(presented in section 4.1.1.3). Prior literature has demonstrated that BMI increases as we grow older (Ng et al., 2014) and could be reproduced in this study both for individuals with and without paternal chronic disease and disability. Yet individuals whose fathers had a chronic disease or disability during their childhood were disproportionately affected by weight gain with increasing age. There are several possible explanations for this effect: First, paternal influences may impact their offspring's health both through biological and psychosocial mechanisms. Sharp and Lawlor postulate that paternal influences may impact the development of type II diabetes and obesity in their offspring through genetic and epigenetic factors as well as postnatal environment (Sharp and Lawlor, 2019). Second, there is some evidence indicating that paternal obesity may impact their offspring's metabolic health (Raad et al., 2017). Perhaps some of the participants, who answered the item *Paternal Chronic Disease/ Disability* in the affirmative, were reporting paternal obesity. This, however, is somewhat speculative. One limitation of this study is that, due to the design of the online-survey, participants could not specify what kind of disease or disability their father suffered from. Further studies should replicate these results prior to further interpretation of these results. If replicated, these results might hold implications for paternal influences as a potential target for public health interventions to improve children's health (Sharp and Lawlor, 2019).

5.2.2 *Interpretation of the Results for the Secondary Response:* ***Common Mental Disorders***

This section focuses on results for the secondary response, symptoms of common mental disorders. These were not at the center of this thesis yet were evaluated in the process of a comprehensive statistical analysis using ordered sequences of regression. Twelve variables were examined as predictors of *Symptoms of Depression*/ *Symptoms of Social Phobia* and, of these, six variables were found to be statistically significant for *Symptoms of Depression* and four were found to be statistically significant for *Symptoms of Social Phobia* at the significance level of 0.01 (presented in sections 4.2.1 and 4.2.2 respectively). The six significant predictors of *Symptoms of Depression* include an interaction between *Paternal Alcohol Abuse* and *Paternal Chronic Pain* (1 and 2), a main effect for *Maternal Mental Disorder (3),* a main effect for *Socioeconomic Status (4),* a main effect for *Age (5),* and a main effect for *Wave (6)*. The four significant predictors of *Symptoms of Anxiety* include a main effect for *Socioeconomic Status (1),* a main effect for *Maternal Mental Disorder (2),* a main effect for *Age (3),* and a main effect for *Gender (4)*. The most important interpretations and implications of significant and non-significant results are discussed below.

5.2.2.1 *The Effects of **SES**, **Wave**, **Gender**, and **Age** on Symptoms of Mental Health Disorders*

The inequalities in health across the social gradient affect not only somatic, but also mental health (Hoebel and Lampert, 2020, Lampert et al., 2014, Allen et al., 2014). These disparities in mental health were reproduced in this study, in which there was an inverse correlation between SES and symptoms of depression as well as symptoms of social phobia (presented in sections 4.2.1.6 and 4.2.2.3 respectively). Moreover, participants in the second wave of data collection reported more symptoms of depression than in the first wave (presented in section 4.2.1.7), while the incidence of symptoms of social phobia remained stable over both waves of data collection. In addition, older age significantly predicted both symptoms of depression and symptoms of social phobia (presented in sections 4.2.1.4 and 4.2.2.5 respectively) and female gender significantly predicted symptoms of social phobia (presented in section 4.2.2.6), but surprisingly, did not predict symptoms of depression.

In consonance with the results presented herein, the prevalence of anxiety disorders in the German population seems to have remained relatively stable over the past decades—data from 1998 indicates a prevalence of 19.5% in women and 9% in men (Wittchen and Jacobi, 2004) compared to data from 2014, which indicates a prevalence of 21.3% and 9.3% respectively (Jacobi et al., 2014). Data on the trend of depression in Germany over time are inconsistent and a perceived increase in the prevalence of depression has been mainly attributed to a shift in age-distribution of the population (Bretschneider et al., 2018, WHO, 2017). Bretschneider et al., who compared data from 1997-1999 to data from 2009-2012, reported no overall increase in the prevalence of depressive disorders in Germany. Nevertheless, they found changes in prevalence among some subgroups of the German population, specifically, an increased prevalence among women between the ages of 18 and 34 and a decreased prevalence in women between the ages of 50 and 65 (Bretschneider et al., 2018). A recent study, which evaluated epidemiological data in Germany between 2009 and 2017 found that there has been an increase in the prevalence of depression especially among adolescents and young adults aged 15-35 and, although the prevalence increased among both sexes, it was more pronounced in men (Steffen et al., 2020). Concurrently, in this sample, consisting mainly of young adults, the participants in wave 2 (data collection 2018-2019) reported higher rates of depression than those in wave 1 (data collection 2008). Thus, data from this study supports the evidence that the prevalence of reported symptoms of depression are increasing in young adults. It remains unclear, however, whether the prevalence of depressive disorders is increasing or whether the observed effect is due to a greater acceptance of mental disorders in our society (Angermeyer et al., 2014).

In accordance with current scientific literature, which reports higher rates of anxiety disorders in women than men (Amiri and Behnezhad, 2019, Sharafi et al., 2020, Jacobi et al., 2014), women in this study reported more symptoms of social phobia than men. The finding

that gender did not predict symptoms of depression, however, is inconsistent with current scientific literature. Despite the most marked increase in depression being amongst young men between the ages of 15-25 (Steffen et al., 2020), the overall prevalence of depressive disorders among women is consistently higher than among men (WHO, 2017, Bretschneider et al., 2018, Steffen et al., 2020). As previously discussed, there are several limitations to the sample selection, which may have caused considerable sample bias. The surprising results may be due to due to the fact that the sample selection in this study does not accurately represent the general population. It is also possible that the prevalence of individuals with symptoms of depression in this sample was too small to cause a significant association between gender and symptoms of depression. Moreover, the absence of a significant effect may be found in a drawback of the linear regression analysis—wherein it is possible for other significant predictors of symptoms of depression to mask the effect of gender on symptoms of depression.

In this sample, symptoms of both depression and social phobia were inversely associated with age (presented in sections 4.2.1.4 and 4.2.2.5 respectively). Younger participants reported more symptoms of depression and social phobia than older participants. In regard to symptoms of depression, the data from this study is contrary to other scientific literature, which indicates a steady increase in the prevalence of depression until the overall highest incidence of depressive symptoms is reached between the ages of 60 and 64 (Steffen et al., 2020). The results for symptoms of social phobia in this study, on the other hand, are in accordance with results from Jacobi et al. on the prevalence of anxiety disorders in Germany by age group. Jacobi et al. found a continuous decrease in the prevalence of anxiety disorders with increasing age, both for anxiety disorders in general (from 18.0% for ages 18-34 to 11.0% for ages 65-79) as well as for social phobia in specific (from 4.6% for ages 18-34 to 0.7% for ages 65-79) (Jacobi et al., 2014).

5.2.2.2 *The Effects of **Adverse Childhood Experiences** on Symptoms of Common Mental Health Disorders*

Evidence from scientific literature indicates that adverse childhood experiences (ACEs) are a significant risk factor for developing mental disorders later in life (Hovens et al., 2012, Carr et al., 2013, Gardner et al., 2019a, Bellis et al., 2019). In this study, only *Maternal Mental Disorders* predicted both symptoms of depression and social phobia (presented in sections 4.2.1.5 and 4.2.2.4 respectively). Symptoms of depression were additionally predicted by a combination of *Paternal Alcohol/ Substance Abuse* and *Paternal Chronic Pain* (presented in section 4.2.1.3).

A meta-analysis from 2014 found that offspring of parents with severe mental illness were more than twice as likely to develop mental disorders as adults (Rasic et al., 2013). Moreover, a previous study found that maternal depression during early childhood predicted

depression in their offspring later in life (Raposa et al., 2014). In concordance with the above-named literature, the results in this study found that individuals who reported maternal mental disorders during their childhood were more likely also to report symptoms of depression and symptoms of social phobia. As both disorders have a genetic component (Craske et al., 2017, Otte et al., 2016) there is also a plausible biological explanation for the link. Notably, the effect was only found for maternal mental disorders and not for paternal mental disorders. This finding is contrary to the results of a prior study, which found that while parental depression was associated with higher rates of depression in their offspring, there was no difference between maternal and paternal mental health (Lieb et al., 2002).

In addition, participants who reported both *Paternal Chronic Pain* and *Paternal Alcohol/ Substance Abuse* were more likely to experience symptoms of depression. The same effect was not found in participants who only reported either *Paternal Chronic Pain* or *Paternal Alcohol/ Substance Abuse* separately (results are presented in section 4.2.1.3). Children who grew up with a parent with alcohol abuse, substance abuse or a mental disorder are statistically three to four times as likely to develop a mental disorder themselves (Die Drogenbeauftragten der Bundesregierung, 2019). Moreover, there is evidence of a dose response between ACEs and adverse health outcomes (Hughes et al., 2017). Thus, it is plausible that only the combined effect of both paternal alcohol or substance and paternal chronic pain caused symptoms of depression. It is notable, that the same effect was not found for *Maternal Alcohol/ Substance Abuse* and *Maternal Chronic Pain*, possibly this association was too weak to show a significant effect in this sample. The lack of effect may also be explained by the higher levels of alcohol and substance abuse among men than among women in Germany (Hardt et al., 2011). These results may reflect the importance of paternal influences in the development of symptoms of depression later in life. Nevertheless, given the sample bias in the current study, these results should be replicated prior to further interpretation.

5.2.3 *Interpretation of the Results for the Tertiary Response:* ***Adverse Childhood Experiences***

Of the eight variables included in the tertiary response, *Adverse Childhood Experiences,* all eight were significantly predicted by *Socioeconomic Status* and three (*Maternal* and *Paternal Chronic* Pain, as well as *Paternal Chronic Disease/ Disability*) were additionally predicted by *Wave*. The tertiary response includes six variables pertaining to parental health and two variables pertaining to parental alcohol or substance abuse. The results for these variables are interpreted separately in the subsequent sections: first, the **effects of SES and Wave on indicators of parental health** are discussed (in section 5.2.3.1) and, second, the **effects of SES on parental alcohol and substance abuse** are discussed (in section 5.2.3.2).

5.2.3.1 *Socioeconomic Status and Health Inequalities*

Disease and disability are not equally distributed among our society, within Germany there is a social gradient whereby people with a lower SES have higher rates of disease and disability and report poorer subjective health (Robert Koch-Institute, 2016). In concordance with these findings, this study recorded higher rates of parental health problems in children who grew up in low-income households—these participants reported higher rates of *Maternal* and *Paternal Chronic Disease/ Disability* (presented in sections 4.3.2.2 and 4.3.2.4), higher rates of *Maternal* and *Paternal Chronic Pain* (presented in sections 4.3.3.2 and 4.3.3.5), as well as higher rates of *Maternal* and *Paternal Mental Disorder* (presented in sections 4.3.4.2 and 4.3.4.4).

The data collection of waves 1 and 2 are approximately a decade apart (*Wave 1* in 2008 and *Wave 2* in 2018/2019) and the participants (whose mean age is around 24 years) reported events from their childhood. Accordingly, significant effects predicted by the solely explanatory variable *Wave* indicate changes in reported parental health over time. Regarding the development of health inequalities in Germany over time, Lampert et al. found that over the past 20 to 30 years health inequalities have remained relatively stable or perhaps increased (Lampert et al., 2018).

No significant association was found between *Wave* and *Maternal Chronic Disease/ Disability* as well as *Maternal/ Paternal Mental Disorder* indicating that the results for these variables were stable across both waves of data collection. *Maternal* and *Paternal Chronic* Pain, as well as *Paternal Chronic Disease/ Disability*, on the other hand, were predicted by *Wave*. Participants in wave 2 reported higher rates of both *Maternal* and *Paternal Chronic Pain* than those in wave 1 (results presented in sections 4.3.3.3 and 4.3.3.6 respectively), indicating an increased prevalence over time. Moreover, in regard to the variable *Paternal Chronic Disease/ Disability*, there was a significant interaction between the two variables *SES* and *Wave*. The results (presented in chapter 4.3.2.4) indicate that a lower SES predicted higher rates of reported paternal disease or disability both in wave 1 and wave 2, however the association is more pronounced in the second wave of data.

It is unclear why the correlation between SES and paternal disease and disability was stronger in the second wave of data, which was collected approximately a decade after the first wave. Perhaps, these results indicate that the social inequalities regarding chronic disease and disability of German men have increased during this period. Yet is also possible that discrepancies between male and female health behavior may explain this effect—besides the social gradient of health inequalities there are also disparities between men and women. For instance, women have a higher life expectancy. This is due, at least in part, to differing health behavior, which is informed by typical male and female gender roles (Faller and Lang, 2019). It is therefore possible, that the paternal rates of disease and disability were similar in both waves and that the results reflect a gradual change in gender stereotypes, whereby

fathers in the second wave of data collection were more likely to be open about their health toward their children, who in turn were more likely to report paternal disease and disability. However, before engaging in further interpretation, these results ought to be verified in a sample that more accurately reflects the general population.

5.2.3.2 *Socioeconomic Status and Disparities in Parental Alcohol and Substance Abuse*

The results presented herein indicate that SES is a significant predictor of both *Maternal* and *Paternal Alcohol/ Substance* Abuse, this is in concordance with some previously published research. According to Jones et al., while both high and low SES report drinking similar amounts of alcohol, people with a low SES seem to be disproportionally affected by the adverse health outcomes of alcohol consumption. Possible explanations for these discrepancies include differences in drinking patterns, the cumulative effect of risky health behaviors, and lack of access to health care (Jones et al., 2015). However, data published by the Robert Koch-Institute in 2016 did not reproduce these trends in the German population. They found that, in Germany, women with a high SES were considerably more likely to engage in risky alcohol consumption, while in men there was no association between alcohol consumption and SES (Lange et al., 2016). The prevalence of children in Germany living with parents or caregivers who suffer from alcohol or substance abuse is not known, thus any estimates on the effects rely solely on extrapolation (Die Drogenbeauftragten der Bundesregierung, 2017). Literature indicates, however, that families in which the caregiver suffers from alcohol or substance abuse are more likely to have a lower SES and higher rates of unemployment (Serec et al., 2012). Moreover, a review of epidemiological data conducted in 2016, indicates that, in Germans under the age of 25, there is a strong negative association between substance abuse and with SES (Henkel and Zemlin, 2016).

In light of these previous findings, the association between childhood SES and parental alcohol and substance abuse found in this study appear plausible. The results indicate an inverse linear correlation between the variables *SES* and *Maternal Alcohol/ Substance Abuse* (presented in section 4.3.1.3). Individuals who reported growing up in a low-income household were more likely to have a mother or female caregiver who suffered from alcohol or substance abuse. Additionally, there was a significant quadratic association between the two variables *SES* and *Paternal Alcohol/ Substance Abuse* (presented in section 4.3.1.5). Children who grew up in low-income households reported higher rates of paternal alcohol and/ or substance abuse. As indicated by the quadratic effect, there is a negative correlation for the lower portion of the SES scale, which becomes roughly linear around the mean SES value in this sample and then remains consistently low beyond that point. The apparent repeated rise in paternal alcohol or substance abuse for the highest SES is due to an artefact by fitting a quadratic term in the regression analysis.

5.2.4 *Interpretation of the Results for the Residuals*

The most relevant results for the Person's correlations of the residuals (presented in chapter 4.4) are discussed below. All but one of the correlations between the variables of the secondary and tertiary responses were significant at the 1% level. In addition, all significant correlations were positively correlated, meaning that participants who answered one item in the affirmative were more likely to answer other items in the same category (either within the secondary or tertiary response) in the affirmative as well. For the secondary response, common mental disorders, there is a significant correlation between the two variables *Symptoms of Depression* and *Symptoms of Social Phobia*. Moreover, this correlation has a large effect size. These results are plausible, as common mental disorders are highly comorbid (WHO, 2017) and it is therefore likely that participants, who reported symptoms of depression also reported symptoms of social phobia.

For the tertiary response, significant correlations were found between all but two of the variables (*Paternal Chronic Pain* and *Maternal Alcohol/ Substance Abuse*). These results are also plausible, as adverse childhood experiences tend to be highly clustered within families (Jorm and Mulder, 2018, Anda et al., 2010). Most of the correlations have small to medium effect sizes. In addition, there are two correlations with large effect sizes, these are *Maternal Chronic Pain* and *Maternal Chronic Disease* as well as *Paternal Chronic Pain* and *Paternal Chronic Disease*. Conceivable explanations for the large effect size between these variables are as follows: First, many chronic diseases, for instance low back pain (Vlaeyen et al., 2018), coincide with chronic pain and therefore the two variables are likely to have considerable overlap. Second, in the questionnaire, participants were asked to recall events from their childhood, it is plausible that a child's perception of parental chronic pain and chronic disease is similar, making it difficult for participants to differentiate between the two. Therefore, those who reported parental chronic disease were also more likely to report parental chronic pain.

5.3 *Strengths and Limitations*

This study's greatest **strength** is that the **statistical analysis** was conducted using ordered sequences of regression, as such it was possible to evaluate many explanatory variables. Moreover, in the statistical analysis, possible confounders (such as age, gender, and SES) were partialled out while examining the association between the explanatory variables and the responses. It is possible that some of the surprising findings in this study can be explained by the many confounders that could thus be taken into account.

Even after the elimination of 62 participants due to lack of data, the study is based on a comparatively **large sample size**, comprised of 718 individuals. Due to the large sample size, the threshold of significance was defined as ≤1%. Therefore, all p-values exceeding this 1% threshold were excluded and no trends in the data were examined. This reduces the number of effects that may have been statistically relevant yet are not clinically relevant. Moreover, the **total explained variance** was calculated for all responses. Although the majority of effect sizes were small, a considerable amount of variance was explained for the primary response, *BMI*, and the secondary response, *Symptoms of Depression*—Cohen's f^2 for these two variables was equivalent to a medium effect size.

The data herein was self-reported and collected via **online survey**. The measurement used to evaluate the primary response is the Body Mass Index (BMI), which is commonly used to approximate overweight and obesity. The BMI has several advantages that have led to its widespread implementation; it is easy to calculate, can be determined using self-reported data, can be easily compared, and allows stratification into various categories of overweight and obesity. Furthermore, online surveys, such as the one used in the current study, are highly economical, objective, and anonymous. Although the anonymity in this type of survey can have some drawbacks, it can also be advantageous when addressing difficult topics, such as adverse childhood experiences (Kappis and Hardt, 2016). Regarding the internal consistency for items within the survey, those items for which Cronbach's α could be calculated (*Symptoms of Depression, Symptoms of Social Phobia,* and *SES*), all had good internal consistency for both waves of data collection, with Cronbach's α ranging from 0.84 to 0.90 (for details see chapters 3.3.2 and 3.3.5). Nevertheless, both the use of the online survey as well as BMI has some drawbacks, the implications of these as well as other limitations, are discussed below.

This research is subject to several **limitations**, the primary of which is the **sample selection**. The recruitment of participants for the online survey was conducted primarily in a medical university setting. Therefore, a majority of the participants are medical students, who may not constitute a representative sample for several reasons. As discussed in detail throughout the interpretation of the results (sections 5.1 and 5.2), this sample may diverge from the general population due to their young age, an underrepresentation of mental

disorders, and comparatively high SES. Thus, the results presented herein must be seen in light of substantial sample bias.

Moreover, there are some limitations regarding the **data collection**; as the study was conducted via online survey, all data is self-reported. This may impact the validity of the data for BMI, symptoms of mental health disorders, and ACEs as described below. The primary response, BMI, is commonly used and was calculated using self-reported height and weight. The reliability of self-reported anthropometrics, however, has been called into question. One literature review by Maukonen et al. published in 2018, found that self-reported height was systematically overestimated, while self-reported weight was underestimated, thus resulting in a lower BMI. Interestingly, this bias was particularly pronounced in overweight and obese participants (Maukonen et al., 2018). Therefore, self-reported height and weight may have a negative impact on the accuracy of the results. More reliable results for BMI could be obtained if height and weight were measured as opposed to self-reported. Moreover, the clinical significance of the results could be increased, if, in addition to BMI, measurements of central adiposity (such as waist-to-hip ratio or waist circumference) were included in the analysis, as they more accurately correlate with health risks than overall adiposity (Jayedi et al., 2020).

Regarding the secondary response, symptoms of mental disorders, the following factors may influence the generalizability of the results. Symptoms depression and social phobia were self-reported and the clinical conditions themselves could not be diagnosed. Additionally, although all items may be subject to **response bias**, the validity of results for symptoms of mental health disorders may be disproportionately affected (Rogler et al., 2001). Moreover, participants were asked to report symptoms from the past two weeks, this may have affected the results, particularly in regard to symptoms of depression. It is plausible that individuals currently experiencing a depressive episode were less likely to take part in an online survey and may therefore be underrepresented in this sample.

Regarding the tertiary response, the eight items concerning ACEs allowed only a binary response (“Yes” or “No”), neither length of exposure nor age at the time of exposure were taken into account. Moreover, due to the nature of the online questionnaire, possible misunderstandings and uncertainties from the participants, could not be addressed. Especially concerning ACEs, some participants may have benefited from personal conversation (Kappis and Hardt, 2016). For instance, it is possible that participants who were unable to answer the questions regarding ACEs with a definitive “Yes” or “No”, simply left the item unanswered. In this study, in order to account for possible uncertainties regarding the presence or absence of a parental problem, unanswered questions were coded as 0.5 on a scale from 0 to 1 (see chapter 3.3.3 for details). However, future studies may benefit from answer choices on a Likert-scale or a third answer choice (such as “Unknown”) as opposed to the binary answer choices used herein.

The **total explained variance** was calculated for all responses, and values vary between approximately 1% for *Paternal Mental Disorder* and 17% for *BMI*. For most of the variables, only a limited amount of variance was explained. This is reflected by the values of Cohen's f^2, the majority of which are equivalent to a small effect size. Furthermore, no effect was found for the variables *Maternal Disability/ Chronic Disease* and *Paternal Mental Disorder*. These results indicate that, in addition to the variables examined in this study, further factors may play an important role in predicting these responses.

As with all cross-sectional studies, only correlation, but not causation, could be determined. A limitation of the retrospective study design is that participants may be subject to **recall bias**. This is particularly relevant for the tertiary response, ACEs, for which individuals were asked to recall events from their childhood. There is evidence that retrospective reporting of ACEs is more likely to lead to false-negative than false-positive reports, thus causing an underestimation rather than an overestimation of effects (Hardt and Rutter, 2004). Moreover, although commonly used, retrospective studies on the association between ACEs and mental health outcomes may be biased, as mental health factors may affect participants' reporting of adverse childhood experiences (Colman et al., 2016). Future studies may benefit from a longitudinal prospective study design.

5.4 *Evaluation of the Results*

Four hypotheses, revolving around the psychosocial predictors of overweight and obesity, are at the center of this research. Of these, only hypothesis 1 was supported by the results—in accordance with previous scientific literature there was a significant negative association between SES and BMI. The remaining three hypotheses were not supported by the results—no significant effect was found for symptoms of depression, symptoms of social phobia, or adverse childhood experiences as a predictor of BMI and, thus, the null hypotheses were retained. In summary, the results both reinforce the importance of SES in the development of overweight and obesity and challenge the importance of ACEs and common mental disorders as predictors of BMI.

Meta-analyses on the association of anxiety disorders and overweight and obesity reported high levels of heterogeneity (Gariepy et al., 2010, Amiri and Behnezhad, 2019), and the results of this study support evidence indicating that symptoms of anxiety do not predict BMI. Furthermore, in accordance with some previous literature (Herhaus et al., 2020), no u-shaped association was found between the two. The findings regarding symptoms of depression and ACEs, however, were unexpected and largely contrary to previous scientific literature which found a bidirectional link between obesity and depressive disorders (Luppino et al., 2010, de Wit et al., 2010a, Mannan et al., 2016) as well as a positive association between ACEs and obesity (Danese and Tan, 2014, Hemmingsson et al., 2014,

Norman et al., 2012, Hughes et al., 2017). ACEs were analyzed including many explanatory variables yet, surprisingly, the only variable that significantly predicted BMI was *Paternal Chronic Disease/ Disability*. For all other ACEs no significant correlation with BMI was found. Given all limitations, most importantly concerning the sample selection, the quintessence of this study is that the evidence for ACEs and symptoms of common mental disorders as predictors for overweight and obesity could not be reproduced.

This study revealed several interesting results beyond the scope of the hypotheses. The only explanatory variable that significantly predicted every single result was *SES*, indicating that even in this sample made up of young German university students, there are substantial inequalities along the social gradient. Moreover, *Maternal Mental Disorder* was the only ACE that predicted higher rates of both symptoms of depression and symptoms of social phobia. Notably, this result has a plausible biological explanation as there is a genetic component to both mental disorders. In addition, the combination of *Paternal Alcohol/ Substance Abuse* and *Paternal Chronic Pain* was significantly associated with *Symptoms of Depressive Disorders*. If reproduced, these results could provide stimulus for future areas of research.

Importantly, this study is subject to considerable sample bias, which may explain some of the surprising results. However, the divergence from prior research may also be a result of the statistical analysis. Due to the use of ordered sequences of regressions many possible confounders could be partialled out in this study. It is possible that previous studies, which did not partial out these possible confounders, found a spurious effect of depressive disorders, anxiety disorders, and ACEs on BMI. Moreover, these limitations in individual studies may have consequently affected the results of meta-analyses. Future studies should consider using ordered sequences of regression to avoid overestimating possible confounders.

6 Conclusion

Over 1.9 billion adults around the world are overweight or obese (WHO, 2018). Consequently, both individuals and society as a whole are affected, for example, through adverse health outcomes (Finer, 2015), medical expenses (Herpertz et al., 2011), and loss of productivity (Yates et al., 2016). The prevalence of overweight and obesity continues to increase (N. C. D. Risk Factor Collaboration, 2017) and in light of limited treatment options and ineffective preventative strategies this topic remains highly relevant. The etiology of obesity is complex and multifactorial, besides genetic and biological factors, societal and environmental influences dictate the extent to which predisposed individuals gain weight (Blüher, 2019). Previous research indicates that psychosocial factors such as socioeconomic status (SES), common mental disorders, and adverse childhood experiences (ACEs) are important in the development of obesity and therefore crucial to the hypotheses posed in this research.

This study aimed to identify these psychosocial predictors in a cohort of over 700 young adults predominantly from a German university. The four hypotheses examined whether a low childhood SES, current symptoms of depression, current symptoms of social phobia, and ACEs during the first fourteen years of life significantly predicted overweight and obesity in adulthood. Utilizing ordered sequences of regression, a comprehensive statistical analysis was performed to analyze the data. Of the fourteen explanatory variables included, four significantly predicted Body Mass Index (BMI) of which only two variables were relevant for the interpretation of the hypotheses.

The first hypothesis was supported by the results–in concordance with previous scientific literature there was a significant negative association between SES and BMI. The remaining three hypotheses, on the other hand, were not supported. Despite some heterogeneity, most current scientific literature reports a positive association between anxiety disorders and obesity. This study, however, found neither a significant correlation nor a u-shaped curve between symptoms of social phobia and BMI. Furthermore, in contrast to the majority of current literature, no association was found between BMI and symptoms of depression or BMI and ACEs. Notably, of the ACEs examined, only paternal chronic disease or disability significantly predicted BMI. Some unexpected results in this study may be due to sample bias. This sample may diverge from the general population due to the young age, the low prevalence of mental disorders, and a comparatively high socioeconomic status of the survey participants.

While the sample selection limits the generalizability of these results, this study suggests that the mediating role of common mental disorders and ACEs in the development of overweight and obesity may be smaller than previously indicated in scientific literature. However, the results also reinforce the significance of SES in the development of obesity thus indicating that societal inequalities are highly relevant to weight gain.

7 Deutschsprachige Zusammenfassung

Global sind über 1,9 Milliarden Menschen von Übergewichtigkeit und Adipositas betroffen (WHO, 2018). Infolgedessen ergeben sich sowohl persönliche wie auch gesellschaftliche Folgen, beispielsweise durch Folgeerkrankungen (Finer, 2015), medizinische Kosten (Herpertz et al., 2011) und Produktivitätsverluste (Yates et al., 2016). Die Prävalenz von Übergewicht und Adipositas nimmt weiterhin zu (N. C. D. Risk Factor Collaboration, 2017) und angesichts begrenzter Behandlungsmöglichkeiten und ineffektiver Präventionsstrategien ist dieses Thema nach wie vor von hoher Relevanz.

Die Ätiologie der Adipositas ist multifaktoriell und komplex. Neben genetischen und biologischen Faktoren bestimmen gesellschaftliche und Umwelteinflüsse das Ausmaß der Gewichtszunahme prädisponierter Personen. Wissenschaftliche Literatur weist darauf hin, dass psychosoziale Faktoren wie der sozioökonomische Status (SES), Depressionen, Angststörungen und Kindheitsbelastungen bei der Entstehung von Adipositas eine wichtige Rolle spielen. Auf Grundlage der genannten psychosozialen Faktoren wurden vier Hypothesen erarbeitet und evaluiert. Im Rahmen der Hypothesen wurde untersucht, ob ein niedriger Sozioökomischer Status in der Kindheit, aktuelle Symptome einer Depression oder sozialen Phobie, sowie Kindheitsbelastungen in den ersten vierzehn Lebensjahren signifikante Prädiktoren für Übergewicht und Adipositas darstellten.

Ziel dieser Studie ist es, diese psychosozialen Prädiktoren von Übergewicht und Adipositas in einer Kohorte junger Erwachsener in Deutschland zu identifizieren. Eine umfassende statistische Analyse wurde mittels geordneten Folgen von Regressionen durchgeführt, um die Daten von über 700 Personen hinsichtlich vierzehn erklärender Variablen zu analysieren. Vier der Variablen korrelierten signifikant mit dem Body Mass Index (BMI), von denen nur zwei für die Interpretation der Hypothesen relevant waren.

Die erste Hypothese wurde durch die Ergebnisse gestützt. In Übereinstimmung mit der bisherigen wissenschaftlichen Literatur gab es eine signifikante negative Assoziation zwischen sozioökonomischem Status und BMI. Die übrigen drei Hypothesen konnten dagegen nicht bestätigt werden. Trotz einer gewissen Heterogenität der Ergebnisse wird in der aktuellen wissenschaftlichen Literatur mehrheitlich eine positive Assoziation zwischen Angststörungen und Adipositas berichtet. Wohingegen diese Studie weder eine signifikante Korrelation noch einen u-förmigen Zusammenhang zwischen Symptomen der sozialen Phobie und BMI fand. Darüber hinaus wurde, im Gegensatz zur Mehrheit der aktuellen Literatur, kein Zusammenhang zwischen BMI und Symptomen der Depression sowie BMI und Kindheitsbelastungen gefunden. Bemerkenswert ist, dass von den untersuchten Kindheitsbelastungen nur väterliche chronische Erkrankung oder Behinderung den BMI signifikant vorhersagte. Die unerwarteten Ergebnisse bezüglich Symptome der Depression und sozialen Phobie könnten auf eine Verzerrung der Stichprobe zurückzuführen sein. Die

Teilnehmenden dieser Studie weichen durch ihr junges Alter, einer geringen Prävalenz psychischer Störungen und einem vergleichsweisen hohen sozioökonomischen Status von der Allgemeinbevölkerung ab.

Wenngleich die Stichprobenauswahl die Repräsentativität der Ergebnisse einschränkt, legt diese Studie nahe, dass die vermittelnde Rolle von Depressionen, Angststörungen und Kindheitsbelastungen bei der Entstehung von Übergewicht und Adipositas geringer ist als bisher in der wissenschaftlichen Literatur angenommen. Andererseits stützen die Ergebnisse die Bedeutung des sozioökonomischen Status bei der Entwicklung von Adipositas und weisen darauf hin, dass gesellschaftliche Ungleichheiten für die Gewichtszunahme von hoher Relevanz sind.

8 Bibliography

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