Trends in overweight- and obesity-attributable mortality and its consequences for the life expectancy in the Netherlands between 1981 and 2012

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Abstract

Background Throughout the world, the prevalence of overweight and obesity has risen tremendously in the past three decades due to poor diet and increasingly sedentary lifestyles. Overweight and obese individuals have a higher risk of several chronic diseases and all-cause mortality. Quantification of overweight and obesity must be attempted to focus the debate on such a major cause of mortality burden as excess weight. **Objective** To investigate the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012. **Data & Methods** The number of deaths attributable to overweight and obesity are estimated using the population attributable fraction, which combines body mass index prevalence data with the relative risks of mortality associated with overweight and obesity. The relative risks are obtained from five published prospective cohort studies. Associated single decrement life tables were used to estimate the potential gain in life expectancy if obesity were eliminated. **Results** Upward trends in obesity-attributable deaths, from 2,785 deaths in 1981 to 6,425 deaths in 2012. It appears that being overweight could significantly increase (3,295 attributable deaths in 1981 to 4,315 deaths in 2012) or decrease (-5,264 attributable deaths in 1981 to -6,861 deaths in 2012) all-cause mortality rates. Furthermore, the potential gain in life expectancy if obesity were to be eliminated increased from 0.35 years in 1981 to 0.61 years in 2012. **Conclusion** Overweight- and obesity-attributable mortality and the potential gain in life expectancy if obesity were to be eliminated substantially increased between 1981 and 2012. Therefore, overweight and obesity are important public health problems for the Netherlands.

Keywords: obesity, overweight, BMI, prevalence, attributable mortality, the potential gain in life-expectancy, trends, Netherlands

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List of abbreviations

ASDT	Associated single decrement table
BMI	Body mass index
CPS I	Cancer Prevention Study I
EU	European Union
GBA	Gemeentelijke Basisadministratie Persoonsgegevens
HMD	Human Mortality Database
NHANES	National Health and Nutrition Examination Survey
NR-NCD	Nutrition related non-communicable disease
OECD	Organisation for Economic Co-operation and Development
PAF	Population attributable fraction
PGLE	Potential gain in life expectancy
QALY	Quality-adjusted life-years
RR	Relative risk
SES	Socio-economic status
SNCS	Swedish National March Cohort
U.K.	United Kingdom
U.S.	United States
WHO	World Health Organization
YPLL	Years of potential life lost

1. Introduction

1.1. Background

Throughout the world, the prevalence of overweight and obesity has risen tremendously in the past three decades (WHO, 2000). The increase in overweight and obese individuals has affected all population groups regardless of gender, age, ethnicity and socio-economic status (OECD, 2010). The WHO (2014a) estimated that 1.4 billion adults aged 20 or older were overweight (BMI ≥25) in 2008. Of these adults, over 200 million men and nearly 300 million women were obese (BMI ≥30). There are substantial difference in overweight and obesity prevalence, both between and within countries. For instance, the prevalence of overweight varies geographically from <10% in some Asian countries to >90% in several Pacific Island nations (Finucane et al., 2011).

In the European Union, 50.1% of the population aged 20 and above were overweight or obese in 2008. The prevalence of overweight (BMI ≥25) is the lowest in Switzerland (44.3%) and the highest in the United Kingdom (61.5%). The prevalence of obesity (BMI ≥30) has nearly doubled between 1980 and 2008 in most European countries (Finucane et al., 2011). On average, 15.5% of the adult population is obese in the European Union in 2008. The prevalence of obesity varies among countries, from less than 10% in Romania, Switzerland and Italy to 20% to 25% in the United Kingdom, Ireland, Malta and Iceland (WHO, 2014b).

Overweight and obesity are not caused by a single factor. There is a complex interplay of biologic, genetic and social factors. The main contributors to overweight and obesity are a poor diet, with high levels of fat, sugar- and processed foods, and physical inactivity (e.g., Aires et al., 2003; James et al, 2004). Genetics, socio-economic status, the environment, medication, and demographic factors are also important determinants. Overweight and obesity are directly associated with the risk of several diseases and health conditions (WHO, 2000). The health consequences range from chronic diseases such as type II diabetes, cardiovascular diseases- and several cancers to premature death.

Quantification of overweight and obesity can be attempted in varies ways. One possibility to quantify excess weight on the population level is attributable mortality. Attributable mortality refers to the fraction of deaths that are avoided if a risk factor is eliminated from the population (Flegal et al., 2005). Based on the Global Burden of Disease Study 2010, the WHO (2009) estimated that 4.8% of the total deaths (or 2,825,000 deaths) in the world were attributable to overweight and obesity in 2004. Of these deaths, 1,081,000 were in Europe, with 318,000 deaths taking place in high-income countries and 763,000 in low- and middle-income countries. Banegas et al. (2003) estimated deaths attributable to excess weight (BMI ≥ 25) for the European Union and for several European countries. The number of deaths attributable to excess weight among European adults was approximately 7.7% of the total deaths (or 279,000 deaths) in 1997. The highest numbers of attributable deaths were found in the United Kingdom (8.7%) and the lowest in France (5.8%). Konnopka et al. (2011) estimated that 4.5%

of the total deaths (or 36,653 deaths) in 2002 were attributed to overweight and obesity in Germany. A number of studies have examined mortality attributable to overweight and obesity outside the European Union, for example the United States (Allison et al., 1999; Flegal et al. 2005; McGinnis & Foege,1993; Mokdad et al. 2004, 2005) and Canada (Katzmarzyk & Ardern, 2004). Only one previous study has examined trends in overweight- and obesity-attributable mortality. Katzmarzyk & Ardern (2004) estimated that number of deaths attributable to overweight and obesity increased from 2,514 in 1985 to 4,321 in 2000 in Canada.

Estimating obesity-attributable deaths allows for the calculation of the potential gain in life expectancy if obesity were to eliminated. The potential gain in life expectancy is also an indicator that is used in quantifying premature deaths due to a disease on the population level. Lai & Hardy (1999) defined the potential gain in life expectancy as 'the added years of life expectancy for the population if the deaths from a particular cause were removed or eliminated as a competing risk of death' (pp. 895). Olshansky et al. (2005) calculated the potential decline in life expectancy due to obesity for the United States in 2000. They estimated that the potential decline in life expectancy at birth would ranged from 0.21 to 1.08 years depending on gender and ethnicity. The article by Olshansky et al. (2005) led to considerable concern because the researchers pointed out that the steady rise in life expectancy may come to an end in the United States due to the rising prevalence of overweight and obesity. There are three studies in the Netherlands that calculated the potential decline in life expectancy. Hoeymans et al. (2010) estimated for 2008 that the potential decline in life expectancy is 0.5 years if overweight (BMI 25-30) were to be eliminated, and 0.3 years for obesity (BMI ≥30). Van Baal et al. (2006) and In 't Panhuis-Plasmans et al. (2012) all estimated that the elimination of overweight (BMI ≥25) leads to a potential decline in life expectancy of 0.8 years at age 20. Trends in the potential decline in life expectancy have never been undertaken in previous research.

As indicated above, overweight and obese individuals have a higher risk of several chronic diseases and premature death (WHO, 2000). This damages individuals, but it is also affects the society. The Dutch government spent 1.6 billion Euros in 2010 for diseases caused by overweight and obesity, which was 2.2% of the total health care expenditure (In 't Panhuis-Plasmans et al., 2012). Quantification of overweight and obesity must be attempted in order to focus debate on excess weight, seeing as it is such a major contributor to both mortality and economic burdens. The overweight and obesity attributable deaths and the potential gain in life expectancy if obesity were to be eliminated will give insight in the extent of the burden of excess weight on mortality in the Netherlands. In addition, research on trends in overweight- and obesity-attributable mortality are rare and trends in the potential gain in life expectancy if obesity would be eliminated have never been undertaken.

1.2. Objective

The objective of this thesis is to investigate the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012, by estimating overweight- and obesity-attributable deaths and the potential gain in life expectancy when obesity were to be eliminated.

1.3. Research question

The main research question:

- What are the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012?

The main question is divided in three sub-questions:

- What is the prevalence of overweight and obesity in the Netherlands between 1981 and 2012?
- What are the deaths attributable to overweight and obesity in the Netherlands between 1981 and 2012?
- What is the potential gain in life expectancy when obesity were to be eliminated in the Netherlands between 1981 and 2012?

1.4. Relevance

1.4.1. Scientific relevance

Exploring the overweight- and obesity-attributable deaths and the potential gain in life expectancy if obesity were to be eliminated will give insight in the extent of the burden of excess weight on mortality in the Netherlands. The research in this thesis is innovative, as a slightly different method is used to calculate attributable deaths and the potential gain in life expectancy as compared to previous research. In addition, research on trends in overweight- and obesity-attributable mortality are rare and trends in the potential gain in life expectancy if obesity would be eliminated have never been undertaken.

1.4.2. Societal relevance

Overweight and obese individuals have a higher risk of several chronic diseases as well as premature death (WHO, 2000). This increased risk damages individuals, but it is also effects entire societies. Obese individuals have medical costs that are on average 30% higher than those for individuals with a ‘normal’ weight (Withrow & Alter, 2011). In ’t Panhuis-Plasmans et al. (2012) estimated that the Dutch government spent 1.6 billion Euros in 2010 on diseases caused by overweight and obesity which represented 2.2% of its total health care expenditure. The majority of these funds were spent on cardiovascular diseases, followed by diabetes.

Quantification of overweight and obesity must be attempted in order to focus debate on excess weight, seeing as it is such a major contributor to both mortality and economic burdens. Overweight and obesity are usually considered risk factors that predispose individuals to chronic diseases, rather than being diseases of themselves. As such, increases in their prevalence do not carry the same public

health message as increases in overweight- and obesity-attributable deaths or the potential gain in life expectancy if obesity were to be eliminated.

1.5. Structure of the thesis

This thesis is broken into four chapters. Chapter two contains the theoretical framework. It consists of a review of the main theory on overweight and obesity, followed by a literature review, the conceptual model and the hypotheses. Chapter three presents the data and methods and it provides information on the study design, the data sources and the methods used in the analysis. The results of the research questions are shown in chapter four. Finally, chapter five contains the conclusion and discussion including a summary of the results, an evaluation of the data and methods, an interpretation of the results, suggestions for further research and policy recommendations.

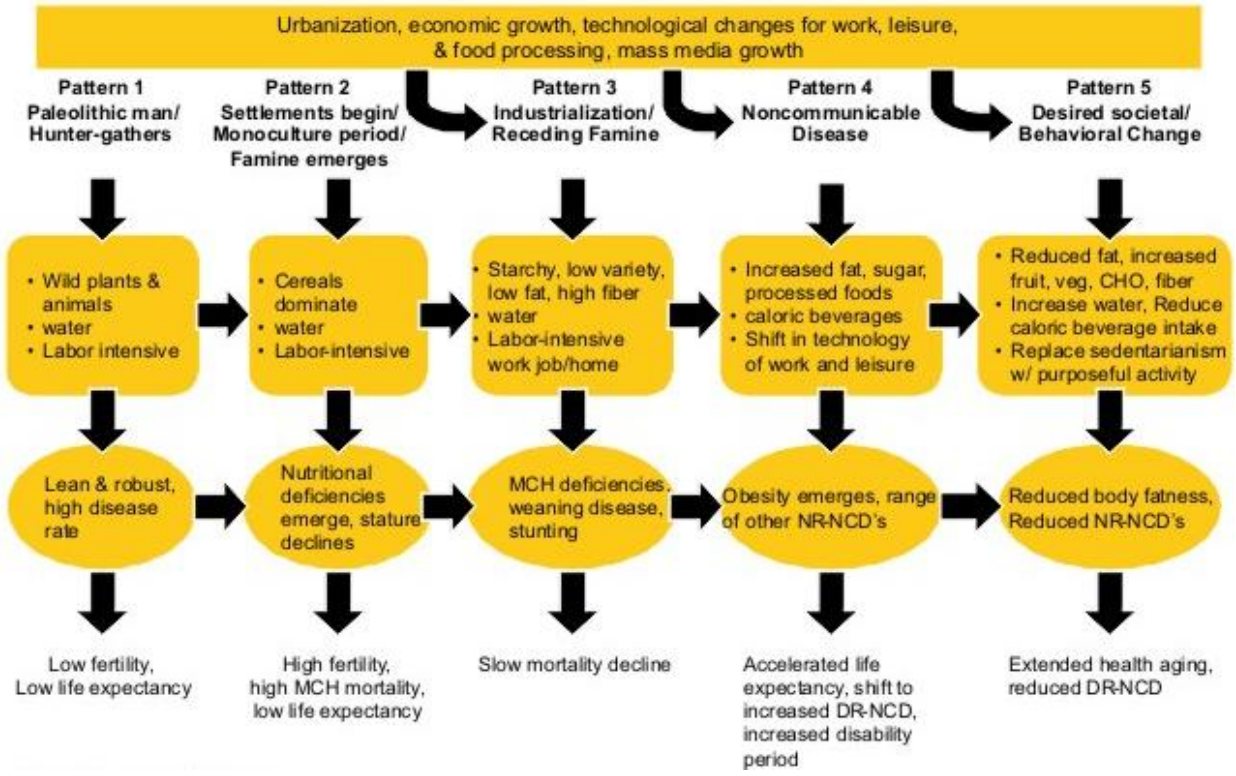
2. Theoretical framework

2.1. Nutrition Transition Theory

The Nutrition Transition Theory, which was introduced by Barry Popkin in 1993**,** explains changes in dietary intake and physical activity. These transitions are reflected in nutritional outcomes, such as the prevalence of overweight and obesity. The theory explains the increasing prevalence of overweight and obesity and their influence on morbidity, mortality and life expectancy. Since the objective of this research is about investigating trends in the prevalence of overweight and obesity and their influence on the mortality burden, the Nutrition Transition Theory is a useful framework to consider.

The transition model consists of five subsequent stages, which are shown in figure 1. The first stage is called collecting food and characterizes hunter-gatherer populations whose diet was high in carbohydrates and low in fat. Their stature was lean and robust and nutritional deficiencies were uncommon. The second pattern is called famine. This diet is less varied than the diet of hunter-gatherers and there are often periods of food scarcity. As a result, diseases related to nutritional deficiencies emerge. The third pattern is receding famine. Periods of food scarcity are decreasing, and there is an increase in the consumption of fruits, vegetables- and animal protein that is accompanied by a decrease in starchy staples. Many nutritional deficiency-related diseases disappear. The fourth pattern is called degenerative diseases. The diet is high in fat, sugar and processed foods and low in fibre. This stage is often accompanied by an increasingly sedentary lifestyle. Obesity emerges, and alongside it, a range of nutrition-related non-communicable diseases (NR- NCD). The fifth and final pattern is behavioural change. People have the desire to prolong health and therefore have a diet low in fat and sugar and high in fruits and vegetables. Consequently, the prevalence of overweight and obesity and NR-NCD decreases and health is prolonged.

Figure 1. Stages of the Nutrition Transition.



Source: Popkin, 2000, p. 286

Simultaneously, two other transitions that affect the Nutrition Transition are also occurring. These are the Demographic Transition, whereby countries experience a transition from high to low fertility and high mortality rates, and the Epidemiological Transition, whereby countries undergo a change from a high prevalence of infectious diseases to a high prevalence of chronic and degenerative diseases (Popkin, 1993). Furthermore, the Nutrition Transition is also partly rooted in the processes of globalization, given that globalization influences changes in lifestyle.

It can be assumed that the Netherlands is now in the fourth stage of the Nutrition Transition, seeing as the prevalence of overweight and obesity had risen substantially since the 1980'-s in almost all Western countries. At the same time, life expectancy has increased in this time span and continues to do so. This thesis will identify trends in the prevalence of overweight and obesity, to confirm the placement of the Netherlands in the fourth stage of the Nutrition Transition. Furthermore, the impact of overweight and obesity on the life expectancy will be analysed to see to what extent there is a threat to the increasing life expectancy.

2.2. Literature review

2.2.1. Definition and assessment of overweight and obesity

The WHO (2014b) defined overweight and obesity as "abnormal or excessive fat accumulation that presents a risk to health" (p. 1). Efforts have been made to quantify adiposity (i.e., body fat) in

individuals and in the population. Measurements that are often used as indicators are body mass index (BMI), waist-hip-ratio, waist circumference, skin-fold thickness, and bio-impedance (Stein & Colditz, 2004). The most commonly reported measure is BMI, which is defined as weight (in kg) divided by square height (in m). BMI is age- and sex- dependent when used as an indicator of body fatness (Gallagher et al., 1996). International cut-points, which are recommended by the WHO, are often used as guidelines for determining overweight and obesity (table 1). The recommendations are based on evidence that morbidity and mortality significantly increases with BMI (Bellanger & Bray, 2005; Lawlor et al., 2006). A BMI of 25 kg/m^2 is the threshold generally accepted for identifying an individual at a higher risk for excess weight related morbidity and mortality.

Table 1. Classification of overweight and obesity.

Classification	**BMI (kg/m^2)**
Normal weight	18.5-24.9
Overweight	25.0-29.9
Obesity	
Grade I	30.0-34.9
Grade II	35.0-39.9
Grade III	≥40.0

Source: WHO, 2000, p. 24

The WHO (2000) defines overweight as a BMI of 25.0 to 29.9. An individual with a BMI of least 30.0 is classified as obese. The WHO distinguishes three grades of obesity: grade I refers to a BMI of at least 30.0 but less than 35.0, grade II to a BMI of 35.0 to 39.9 and grade III (which is also called morbid obesity) refers to a BMI of at least 40.0.

2.2.2. Prevalence overweight and obesity

Throughout the world, the prevalence of overweight and obesity has taken epidemic proportions (WHO, 2000). Finucane et al. (2011) estimated that the worldwide BMI increased on average by 0.4 kg/m^2 per decade for men and 0.5 kg/m^2 per decade for women between 1980 and 2008. As a result, the worldwide prevalence of obesity (BMI ≥30) has nearly doubled in this time span. In 2008, 10% of men and 14% of women in the world were obese, compared with 5% of men and 8% of women in 1980 (WHO, 2014c). The WHO (2014a) estimated that 1.4 billion adults aged 20 or older were overweight (BMI ≥25) in 2008. Of these adults, over 200 million men and nearly 300 million women were obese (BMI ≥ 30). There are substantial differences in overweight and obesity prevalence both between and within countries. For instance, the prevalence of overweight varies geographically from <10% in some Asian countries to >90% in several Pacific Island nations (Finucane et al., 2011).

The OECD (2010) estimated overweight and obesity prevalence in the European Union (EU) in 2008. The prevalence of overweight and obesity has doubled in the past two decades in most countries of the EU, independent of what the overweight and obesity prevalence were 30 years ago. The increase in overweight and obese individuals has affected all population groups regardless of gender, age, ethnicity and socio-economic status (Finucane et al., 2011). In the EU, 50.1% of the population aged 20 and above was overweight or obese in 2008. The prevalence of overweight and obesity is more than 50.0% in 15 of the 27 EU countries. Overweight and obesity prevalence are lowest in Switzerland (44.3%) and France (45.9%) and highest in the U.K. (61.5%) and Ireland (60.9%). On average, 15.5% of the adult population is obese in the EU. The prevalence of obesity varies among countries, from less than 10% in Romania, Switzerland and Italy to 20% to 25% in the U.K., Ireland, Malta and Iceland.

2.2.3. Causes of overweight and obesity

Overweight and obesity are not caused by a single factor. There is a complex interplay of biologic, genetic and social factors (WHO, 2003). The main contributors of overweight and obesity are a poor diet and physical inactivity. Genetics, the environment, socio-economic status, medication, and demographic factors are also important determinants. All of these variables will be discussed in the following sections.

Diet & nutrition

Diet and nutrition are important determinants of health status. Nowadays, there is a shift in diet towards more energy-dense foods and drinks. Diets increasingly consist of (processed) foods that are high in saturated fat, sugar and salt, combined with a low consumption of fruit, vegetables and dietary fibre (James et al, 2004a; Mokdad et al. 2004; Pereira & Ludwig, 2001). A high intake of energy-dense foods, such as butter, oils- and fried food, promotes weight gain. These foods are not only high in fat and sugar, but also poor in nutrients. A high intake of sugar-sweetened beverages, such as soda, also promotes weight gain (Matters, 1996; Harnack et al., 1999; Ludwig et al., 2001).

Physical activity

There is a shift to a more sedentary lifestyle due to changes in work and leisure, modes of transportation, and urbanization (WHO, 2003). Consequently, there is an increase in physical inactivity among adults. Physical activity is an important determinant of BMI (Aires et al., 2003; Haapanen et al., 1997; Norman et al., 2002). Most epidemiological studies show that being active (for at least 30 minutes a day) protects against weight gain (Fogelholm & Kukkonen-Harjula, 2000). In addition, both physical activity and physical fitness are important determinants of mortality and morbidity attributed to overweight and obesity. The risk of cardiovascular diseases and all-cause mortality is substantially reduced by moderate to high physical activity at all BMI levels (WHO, 2003).

Ortega et al. (2013) studied obese individuals who seem to be protected against diseases related to obesity. These individuals are referred to as being 'metabolically healthy but obese'. Their study suggested that metabolically healthy but obese individuals are physically fitter than metabolically abnormal obese individuals. The metabolically healthy but obese individuals had a 30% to 50% lower risk for obesity related morbidity and mortality than the metabolically abnormal obese individuals.

Genetics

Genetics likely reflects differences among individuals in their susceptibility to obesity, as some genes appear important in the development of most early-onset forms of obesity (Bell et al., 2005; Speakman, 2004). Genetic defects also affect the risk of obesity. The genetic defects found by Boutin & Froguel (2001) and Farooqi & O'Rahilly (2006) all affect the drive to eat. Maes et al. (1997) reviewed the literature on the family resemblance of BMI. Based on twin studies, they concluded that genetic factors explain approximately 50% to 90% of the variance in BMI. In addition, they estimated that 20% to 60% of the variation in BMI is attributed to genetic factors based on adoption studies.

Environment

The environments in which humans dwell are enormously complex. Evidence indicates that the environment has a significant effect on diet, physical activity and obesity. The obesogenic environment is perceived to be a driving force behind the obesity epidemic, since many environments become increasingly obesogenic (Swinburn & Egger, 2002). Swinburn et al. (1999) defined the obesogenic environment as "the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations" (p. 564). The environment can be related to health in several ways. First, through the physical design (i.e. the built environment), second, the socio-cultural rules that govern these environments (e.g. the availability and accessibility to food and food advertising and marketing for fast food) and third, the socio-economic status (SES) of these environments (Lake & Townshend, 2006).

Socio-economic status

The SES of an individual is often measured by income, educational level, occupation, or a combination of these factors. The relationship between SES and obesity is complex. Its underlying determinants are closely linked to the environment, especially to social, economic, employment and education policies (WHO, 2003). The classic pattern of overweight and obesity in a population starts with middle-aged women with a high SES. As the overweight and obesity epidemic advances, excess weight becomes more common in individuals with a low SES, especially in women (WHO, 2003). It is clear that a low SES is associated with a significantly higher prevalence of overweight and obesity among females in developed countries (McLaren, 2007). However, in these countries, the relationship for men and children is non-significant. A significant relationship does exist between SES and obesity among men and children in developing countries. Hulshof et al. (2003) studied differences in dietary intake between adults with different SES in the Netherlands, and concluded that dietary intake among

individuals with a higher SES tended to be closer to the recommendations of the Netherlands Food and Nutrition Council. Thus, as a result, people with a high SES had a lower BMI than individuals with a low SES. However, people who have a low SES may be more exposed to the obesogenic environment, and therefore, their eating and physical activity patterns are probably the default choices on offer (WHO, 2003).

Medication

Weight gain is a problematic side effect of many prescription drugs, which can lead to noncompliance with therapy and to the exacerbation of co-morbid conditions related to obesity (Ness-Abramof & Apovian, 2005). Examples are medications for seizures, diabetes, migraines- and high blood pressure (Schwartz et al., 2004). Most antipsychotic medications, such as clozapine and olanzapine, are also associated with substantial weight gain (Newcomer, 2006). In addition, these medications also alter insulin sensitivity and lipid metabolism, which increases the risk of diabetes and cardiovascular disease (Keck & McElroy, 2003).

Demographic determinants

In addition to the causes discussed above, there are demographic determinants of overweight and obesity that are worthy to mention. These are age, gender and ethnicity. In Western countries, BMI increases with age, both for males and females (Santos & Barros, 2003). Furthermore, although females have a higher prevalence of obesity than males in most Western countries, on average, males have a higher BMI than females because their prevalence of overweight is much higher (Gezondheidsraad, 2003). As regard to ethnicity, there are significant differences in obesity prevalence. For example, Ogden et al. (2006) estimated in the U.S. that Mexican-American and non-Hispanic black female adults were significantly more likely to be overweight compared with non-Hispanic white female adults. Among men, however, the prevalence of obesity did not differ between ethnic groups. The prevalence of overweight and obesity in the Netherlands is higher for the foreign population than for the native population (De Graaf et al., 2011). Antillean, Moroccan and Turkish females in particular have a higher risk of being overweight and obese.

2.2.4. Health consequences of overweight and obesity

Overweight and obesity are directly associated with the risk of several diseases and health conditions. Obese individuals have a lower healthy life expectancy than individuals with a ‘normal’ weight due to obesity-related morbidity and mortality (Visscher & Seidell, 2001). Worldwide, excess body weight is the sixth most important risk factor that contributes to the total burden of disease (Haslam & James, 2005). A study by James et al. (2004b) looked at the global and regional burden of disease attribution to selected major risk factors. The proportions of the global burden of disease attributable to excess weight were 58% for type II diabetes, 21% for ischaemic heart disease, 39% for hypertensive disease, 32% for endometrial cancer in women, 23% for ischaemic stroke, 12% for colon cancer and 8% for

postmenopausal breast cancer. The most common diseases associated with obesity (namely diabetes, cardiovascular diseases and cancer) will be discussed in the following sections.

Diabetes

An estimated 834,100 individuals in the Netherlands (or 5% of the population) had diabetes in 2011 (Baan et al., 2014). Type II diabetes accounted for approximately 90% of all diagnosed cases (Baan et al., 2014). The risk for developing diabetes increases as the degree of overweight increases. Thompson et al. (1999) estimated that mildly obese (BMI 27.5) individuals in most age and sex groups had an increased risk of 50% to 60% for contracting type II diabetes, moderately obese (BMI 32.5) had a twofold higher risk and severely obese (BMI 37.5) faced a risk that was three- to four-fold higher.

Cardiovascular diseases

Cardiovascular disease is the second leading cause of death in the Netherlands (Poos, 2013). The most prevalent cardiovascular diseases are coronary artery disease, heart failure and stroke. BMI has been found to be consistently associated with an increased risk of cardiovascular diseases (Dalton et al., 2003; Field et al., 2001). Van Kreijl et al. (2004) estimated that adults with obesity in the Netherlands have a two to four times higher risk of cardiovascular disease. Obesity has an association with cardiovascular disease, probably through the impact it has on related risk factors, including hypertension, dyslipidemia, impaired glucose tolerance- and type II diabetes (Eckel, 1997 in Billington et al., 2000). However, some studies have also found that overweight has a direct impact on cardiovascular diseases and all-cause mortality, even after correction for other risk factors (Rabkin et al., 1977; Garrison & Castelli, 1985).

Cancer

Cancer is the leading cause of death in the Netherlands (Poos, 2013). In 2011, 44.083 individuals died of cancer, which represented 32% of total deaths. Excess body weight is directly associated with the risk of several cancers, including colon, breast (in postmenopausal women), endometrium, oesophagus, kidney (Bianchini et al., 2002; Key et al., 2002), leukemia (Lichtman, 2010), liver (Sun & Karin, 2012; Yoshimoto et al., 2013) and gallbladder (Hundal & Shaffer, 2014). Diet is estimated to account for approximately 30% of all cancers in Western countries (Doll & Peto, 1996; Key et al., 2002).

Other morbidities

In addition to diabetes, cardiovascular diseases and cancer, being overweight or obese also increases the risk of several other diseases and health conditions. The most common are hypertension (e.g., Chow et al., 2000; Thompson et al., 1999), obstructive sleep apnoea (e.g., Guilleminault et al., 1988; Lavie et al., 2007; Marshall et al., 2011), gallbladder disease (e.g., Khare et al., 1995; Ruhl & Everhart, 2001), osteoarthritis (e.g., Coggon et al., 2001; Davis et al., 1990; Felson, 1996; Hart & Spector, 1993), infertility in both sexes (e.g., Grodstein et al., 1994; Nguyen et al., 2007; Rich-Edwards et al., 2002)- and depression (e.g., De Wit et al., 2009; Johnston et al., 2004).

2.2.5. Mortality consequences of overweight and obesity

Mortality

The relationship between BMI and mortality can be presented by a U-shaped curve, with increased mortality at both lower and higher levels of BMI (Calle et al., 1999; Katzmarzyk et al., 2001; Seidell et al., 1999; Sempos et al., 1998; Schulte et al., 1999). The U-shaped curve is the source of considerable debate. Some researchers believe that the association represents causation, while others think that the elevated mortality rate at the low end of the BMI continuum represents a form of confounding (Geller et al., 2007 in Childers & Allison, 2010).

Previous research has shown that obesity (BMI ≥30) significantly increases all-cause mortality as compared to individuals with a 'normal' weight. All large-scale prospective epidemiological studies from the U.S., including the Framingham Study (Hubert et al., 1983), the Nurses' Health Study (Manson et al., 1990) and the National Health and Nutrition Examination Survey (Flegal et al., 2005), have shown that obesity is associated with increased mortality in both men and women. Overweight (BMI 25-30) shows controversy regarding all-cause mortality. Several studies found a significant increase in mortality for overweight individuals (e.g., Ärnlöv et al., 2010; Carlsson et al., 2011; Manson et al., 1995), some studies found no significant effect (e.g., Benders, 1998; Jonsson et al., 2002; Katzmarzyk et al., 2012) and other studies estimated that being overweight is associated with significantly lower all-cause mortality (e.g., Flegal et al., 2005, 2013; Taylor & Østbye, 2001), suggesting that the overweight category might even have protective qualities regarding morbidity and mortality.

Attributable mortality

Previous studies on excess deaths associated with overweight and obesity has centred on the concept of attributable mortality. Attributable mortality refers to the fraction of deaths that are avoided if a risk factor is eliminated from the population (Flegal et al., 2005). To paraphrase, by deaths attributable to obesity it is meant that given the number of people who were alive in a particular year, how many fewer would have died by the end of that year if all of the obese people alive at the beginning of the year had not been obese and had the hazard of those with a 'normal' weight.

Based on the Global Burden of Disease Study 2010, the WHO (2009) estimated that 4.8% of the total deaths (or 2,825,000 deaths) in the world were attributable to overweight and obesity in 2004. For males, this represented 4.2% of all global deaths (or 1,319,000 deaths) and for females 5.4% (or 1,506,000 deaths). Of these deaths, 1,081,000 were in Europe, with 318,000 deaths taking place in high-income countries and 763,000 in low- and middle-income countries.

Banegas et al. (2003) estimated deaths attributable to excess weight (BMI ≥ 25) in the EU for 1997. They used BMI prevalence data that was based on self-reported weight and height from the Pan-EU in 1997 for individuals aged 25 and above, which they combined with the RRs from the Cancer Prevention Study I by Allison et al. (1999). The RRs were not adjusted for smoking and history of disease. The number of annual deaths attributable to excess weight among EU adults was approximately 279,000. This represents 7.7% of all deaths registered among adults aged 25 years and older. More attributable deaths occurred among obese (175,000 deaths) subjects than it did among those who were overweight (104,000 deaths), reflecting the higher RR associated with obesity. The highest numbers of attributable deaths were found in the UK (8.7%) and the lowest in France (5.8%). Approximately 7.7% of the total death in the Netherlands were attributable to overweight and obesity. If adjusted RR would have been used, the attributable deaths would have been higher.

A number of other studies have examined mortality attributable to obesity and overweight. Konnopka et al. (2011) estimated that 4.5% (or 36,653 deaths) of the total deaths in 2002 were attributed to overweight and obesity among adults aged 18 and above in Germany. Allison et al. (1999) estimated overweight (BMI ≥25) attributable deaths in the U.S. in 1991. Their subjects were adults aged 18 years and older who were non- or never-smokers. The mean estimate of deaths attributable to obesity was 280,184 (236,111-341,153). McGinnis & Foege wrote an article called 'Actual Causes of Death in the United States' in 1993 in which they also estimated attributable deaths. However, they used diet and physical activity instead of obesity as a risk factor for mortality. They estimated that 300,000 deaths in 1991 were attributable to poor diet and physical inactivity. Mokdad et al. (2004) updated the work of McGinnis & Foege (1993) and estimated the actual causes of death in the U.S. in 2000. They estimated that 400,000 deaths were attributable to excess weight (BMI ≥25), an increase of more than 100,000 deaths in nine years compared to the estimate of McGinnis & Foege (1993). They later corrected their calculation (Mokdad et al., 2005) to 365,000 deaths, resulting in a 65,000 increase. In 2005, Flegal et al. published an article that prompted considerable controversy about the issue of excess death associated with overweight and obesity. Flegal et al. (2005) estimated that only 111,909 deaths in the U.S. in 2000 were attributable to adults being obese (BMI≥30). The estimate was considerably smaller than the finding of Mokdad et al. (2004, 2005). Furthermore, according to their research, overweight (BMI 25-29.9) was not associated with excess mortality (-86,094 deaths).

Trends in attributable mortality

Since the prevalence of overweight and obesity is increasing throughout the world, it can be assumed that overweight- and obesity-attributable mortality is also increasing. Unfortunately, studies on trends in attributable mortality due to excess weight are rare. Only one previous study (Katzmarzyk & Ardern, 2004) has examined trends in overweight- and obesity-attributable mortality. This study looked at overweight- and obesity-attributable deaths in Canada between 1985 and 2000 for adults

aged 20 to 64. The annual number of deaths attributable to overweight and obesity increased from 2,514 (966-4,061) in 1985 to 4,321 (2,114-6,542) in 2000.

There are studies with attributable deaths due to excess weight for the same country for different years that could be compared, like the study of McGinnis & Foege (1993) and Mokdad et al. (2004) for the U.S. in the previous section. The problem is that these estimations were performed by various researchers who used different data sources and methods to estimate attributable mortality, which makes it difficult or even impossible to compare results.

Life expectancy

The WHO (2014d) defines life expectancy as "the average number of years a person can expect to live, if in the future they experience the current age-specific mortality rates in the population" (p. 1). Life expectancy at birth has risen in all developed countries in the world since the beginning of the 20th century (Kinsella, 1992). In 2012, life expectancy at birth for both sexes globally was 70 years, ranging from 62 years in low-income countries to 79 years in high-income countries (WHO, 2014e). Life expectancy differs substantially by gender (e.g., Kinsella, 1992). Women have a lower mortality for every age group and for most causes of death compared to males, which results in a higher life expectancy at birth for females. Women in the EU outlive men by 6.5 years (OECD, 2010).

Lai & Hardy (1999) distinguish two types of indicators that are widely used in quantifying premature deaths due to a particular disease. The first is the potential gain in life expectancy (PGLE), which is measured on the population level; the second is the years of potential life lost (YPLL), which is measured on the individual level. Lai & Hardy (1999) define the potential gain in life expectancy as 'the added years of life expectancy for the population if the deaths from a particular cause were removed or eliminated as a competing risk of death' (pp. 895). The PGLE is based on multiple-decrement life table techniques that properly takes into account competing risks of death and the age structure of the population. The YPLL is the years of life lost by those who died. Fontaine et al. (2003) defined YPLL due to obesity as 'the difference between the number of years a person would be expected to live, if he/she was not obese and the number of years expected to live if the person were obese' (pp. 187). This type of method fails to take into account competing risks and is heavily influenced by population age structures. The study by Lai & Hardy (1999) indicated that the YPLL does not always provide similar or consistent results to those obtained from the PGLE.

Years of potential life lost

Peeters et al. (2003) estimated YPLL in the U.S. for 40-year-old non-smoking adults. Mortality rates specific for the age and BMI group were derived using data from the Framingham Heart Study, and life expectancy was analysed using life tables. They estimated that females lost 7.1 years and males lost 5.8 years due to obesity. Chang et al. (2013) predicted YPLL associated with morbid obesity (BMI >40) for U.S. non-smoking adults aged 40 to 49 years. These researchers extracted data from the

National Health Interview Survey and used a simulation approach instead of the generally used life table approach to project life years. Obesity led to 0.2 to 11.7 YPLL depending on gender, race, BMI, and age. Black males lost 5.4 life years on average, white males 5.2 years, black females 5.0 years and white females 4.7 years. Fontaine et al. (2003) looked at the YPLL in the U.S. due to morbid obesity (BMI ≥45). Data from NHANES I/II/III (which included participants aged 18 to 85 years) was used. White men and women aged 20 with BMIs greater than 45, are estimated to have 13 YPLL and eight YPLL due to obesity respectively, in comparison to people with a BMI of 24.

Potential gain in life expectancy

Olshansky et al. (2005) calculated the potential decline in life expectancy due to obesity for the U.S. for 2000. They considered individuals aged 20 to 85, with a maximum BMI of 45, who participated in the NHANES III to estimate life expectancy. They estimated that the PGLE at birth would be 0.33 to 0.93 years for white males, 0.30 to 0.81 years for white females, 0.30 to 1.08 years for black males, and 0.21 to 0.73 years for black females if obesity did not exist. The article by Olshansky et al. (2005) led to considerable concern because the researchers point out that the steady rise in life expectancy may come to an end in the U.S. due to rising overweight and obesity levels.

Preston & Stokes (2011) estimated the PGLE at age 50 due to obesity (BMI ≥30) for several European countries in 2006. They used the Prospective Studies Collaboration study to estimate PAF and self-reported weight and height from the Survey of Health, Ageing and Retirement in Europe (2004-2007). The countries included were Austria, Belgium, Czech Republic, Denmark, France, Germany, Israel, Italy, Netherlands, Poland, Spain, Sweden and Switzerland. For females, PGLE at age 50 ranged from 0.50 years (Switzerland) to 1.19 years (Poland). The PGLE for Dutch females was 0.69 years. The PGLE for males ranged from 0.73 years (Netherlands) to 1.37 years (Poland).

There are three studies in the Netherlands that calculated the PGLE when obesity were to be eliminated. They all use the RRs from the RIVM *Chronische Ziekten Model* (i.e., RIVM Chronic Disease Model) to calculate the PAR. The model simulates the prevalence of risk factors and the incidence of diseases, by age and gender. The model uses an alternative version of the Markov-type multi states model, developed by Hoogenveen et al. (2010) to calculate the RRs. Hoeymans et al. (2010) estimated the PGLE for 2008. The PGLE would be 0.5 years for overweight (BMI 25-30) and 0.3 years for obesity (BMI ≥30). An additional 0.4 years are gained if all physically inactive persons would adopt physically active lifestyles. Van Baal et al. (2006) and In 't Panhuis-Plasmans et al. (2012) all estimated that the elimination of overweight (BMI ≥25) leads to a PGLE of 0.8 years at age 20. The year for which these studies estimate the PGLE is unknown.

Trends in potential gain in life expectancy

Studies on past trends in the PGLE due to the elimination of overweight and obesity have never been undertaken. However, Stewart et al. (2009) forecasted the effect of obesity on U.S. life expectancy

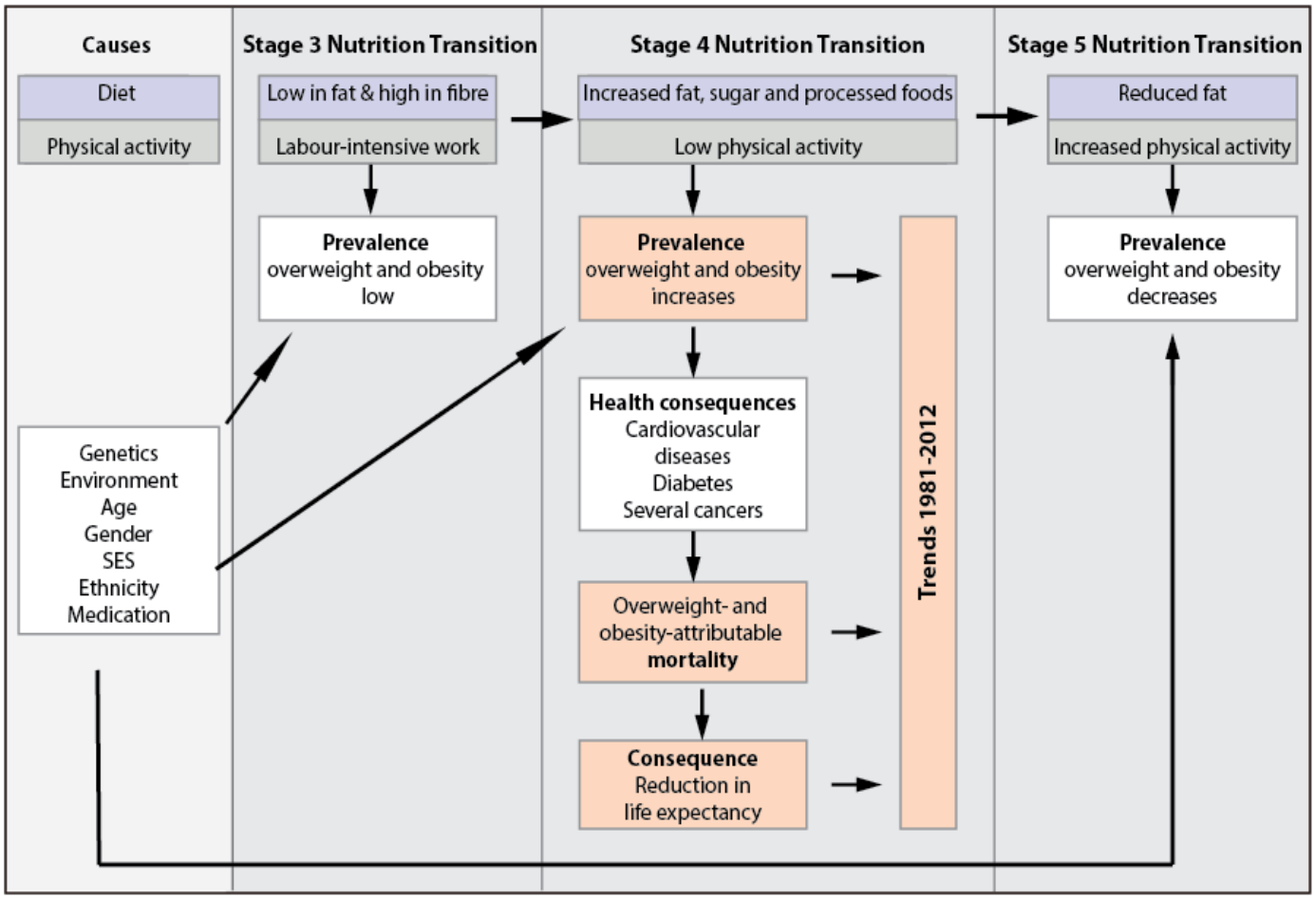
between 2005 and 2020. If increases at the rates seen over the past 15 years in BMI continue, gains in life expectancy would be reduced by 1.02 years between 2005 and 2020.

Jia & Lubetkin (2010) estimated losses in the quality-adjusted life-years (QALYs) due to obesity for U.S. adults from 1993 to 2008. The QALYs lost contributed by a risk factor is the sum of QALYs lost due to morbidity in the current year and future QALYs lost in expected life-years due to premature deaths. The prevalence of obese people increased significantly, from 14.5% to 26.7% during this period. The QALYs lost was 0.0204 per adult population (i.e., 7.5 days of healthy life lost) in 1993. In 2008, obesity resulted in 0.0464 QALYs lost per adult population, which is an increase of 127%. The QALYs lost contributed by obesity consistently increased with the increase in the percentage of obesity from 1993 to 2008.

2.3. Conceptual model

Figure 2 shows a conceptual model that is intended to demonstrate the main concepts and their interrelations of this thesis while incorporating the theory and the literature, in order to investigate the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012. On the left side of the model are the causes of overweight and obesity, and they determine the prevalence of individuals being overweight and obese in the Netherlands. The causes are diet, physical activity, genetics, the environment, age, gender, SES, ethnicity and medication. Diet and physical activity are determinants of the Nutrition Transition as well. The last three stages of the Nutrition Transition (namely stage three, stage four and stage five) and their characteristics are also shown. The diet in stage three is low in fat and high in fibre. On average, individuals are physically active due to labour-intensive work. These characteristics in diet and physical activity lead to a low prevalence of overweight and obesity. Over time, countries will flow from the third to the fourth stage. The fourth stage is characterized by a diet that includes increased amounts of fat, sugar and processed foods combined with low physical activity. This leads to increasing prevalence of overweight and obesity. The main health consequences of overweight and obesity according to the literature are cardiovascular diseases, diabetes and several cancers. Subsequently, these health consequences are associated with overweight- and obesity-attributable mortality. Finally, overweight and obesity have an influence on life expectancy. The higher the prevalence of overweight and obesity, the higher the reduction of the life expectancy. The orange blocks in the figure contain the outcome variables and reflect the research questions. The three variables are overweight and obesity prevalence, overweight- and obesity-attributable mortality and the reduction in life expectancy. All three of these factors will be estimated for the years 1981 to 2012, and trends will be analysed. After the fourth stage of the Nutrition Transition, countries will end up in the final fifth stage. There is a reduction in dietary fat and there is an increase in physical activity. This leads to a decrease in the prevalence of overweight and obesity.

Figure 2. Conceptual model.



Source: Adapted from Popkin, 2000, p. 286

2.4. Hypotheses

According to the Nutrition Transition, countries in stage four, the non-communicable disease pattern, experience an increase in the prevalence of overweight and obesity. This is what we expect to be the case in the Netherlands as well between 1981 and 2012. More specifically, it would be expected that the prevalence have doubled during that period, as it did in most European countries (OECD, 2010). According to the literature, people who are overweight and obese have a higher risk of all-cause mortality as compared to those with a 'normal' weight. Therefore, with an increasing prevalence of overweight and obesity, we expect an upward trend in overweight- and obesity-attributable mortality between 1981 and 2012 in the Netherlands, similar to the trend in attributable mortality in Canada (Katzmarzyk & Ardern, 2004), where attributable mortality significantly increased between 1985 and 2000. We expect a much lower number of attributable deaths than in the U.S. (Allison et al., 1999; Flegal et al., 2005; McGinnis & Foege, 1993; Mokdad et al., 2004, 2005), since the prevalence of overweight and obesity is expected to be much lower. A similar estimate is made by Banegas et al. (2003) for the Netherlands in 1997 is expected for that year. Finally, the PGLE if obesity were

eliminated is expected to increase as well between 1981 and 2012 in the Netherlands, as a result of the increase in the deaths attributable to obesity. The PGLE ranges from 0.21 to 1.37 years in previous studies (Hoeymans et al., 2010; In 't Panhuis-Plasmans et al., 2012; Olshansky et al., 2005; Preston & Stokes, 2011; Van Baal et al., 2006), and we expect the PGLE to be within or close to this range.

Summarized, the hypotheses are as follows:

- There is an upward trend in the prevalence of overweight and obesity in the Netherlands between 1981 and 2012.
- There is an upward trend in the deaths attributable due to overweight and obesity in the Netherlands between 1981 and 2012.
- There is an upward trend in the potential gain in life expectancy when obesity is eliminated in the Netherlands between 1981 and 2012.

3. Data & methods

3.1. Study design

The main research question of the thesis is 'What are the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012?'. Descriptive research is used to answer this question, as the focus is on describing trends in the prevalence of overweight and obesity, overweight- and obesity-attributable deaths and the PGLE if obesity were to eliminated. Quantitative research will be conducted and secondary data analysis will be performed. Analysis will be performed on the population level. The data for this analysis is primarily derived from Statistics Netherlands.

3.2. Data sources

Several sources with prevalence data on overweight and obesity were considered. These studies included Statistics Netherlands, the LISS-panel data, the MORGEN-project, NL-de Maat and the European Community Household Panel. The data from Statistics Netherlands was chosen because it is the only source with nationally representative prevalence data. In addition, because the focus of the thesis is on trends over time, data over a long time span was preferred. Only Statistics Netherlands provides more than five subsequent years of prevalence data on overweight and obesity.

In accordance with the objective of the study, aggregated prevalence data for overweight and obesity over the period 1981-2012, by age (20-30, ..., 50-55, 55-65,...,75+) and sex were obtained. The time span 1981- 2012 and age category 20 years and above were chosen for analysis due to the availability of the data. Statistics Netherlands derived prevalence data by gathering information from the following interview surveys: the *Gezondheidsenquête* (1981-1996), *POLS gezondheid* (1997-2009) and the *Gezondheidsenquête* (2010-2012). The sample of the *Gezondheidsenquête* (1981-1996) and *POLS gezondheid* (1997-2009) consist each year of approximately 10,000 individuals, of all ages. The response rate is approximately 35% to 40% for each year. The sample of the *Gezondheidsenquête*

(2010-) consists of 15,000 individuals (of all ages) and the response rate is 60% to 65% for each year. The sample is adjusted for age, sex, marital status, level of urbanization and province. Respondents with a BMI of <14 and >45 are deleted from the sample. According to Statistics Netherlands, the data is nationally representative.

Other data that was obtained from Statistics Netherlands included the population numbers on July 1 for males and females for the years 1981-2012 by age (0, 1-4, 5-9, 10-14….90-94, 95+). The data consists of every person who is included in a population register of a Dutch municipality, also called the *jure* population (CBS, 2014). Aggregated number of deaths by all-causes for the years 1981-2012, by age (20-24,25-29...90-94, 95+) and sex were obtained from the Cause of Death Statistics (*Doodsoorzakenstatistiek*) from Statistics Netherlands as well. The population taken into account is the group that has passed away and was registered in the *Gemeentelijke Basisadministratie Persoonsgegevens* (GBA). Everyone who legally stays in the Netherlands for more than four months will be taken into the GBA of their municipality (CBS, 2014).

Abridged life table information was obtained from the Human Mortality Database (HMD) by age (0, 1-4, 5-9, 10-14...110+) for the years 1981-2009. Unfortunately, life table data after 2009 was not available.

3.3. Operationalization

By far the most commonly used measure of overweight and obesity is the BMI, which is calculated by dividing weight (in kg) by square height (in m). BMI will be used as a measure of overweight and obesity in this thesis as well, since the data from Statistics Netherlands on the prevalence of overweight and obesity is already expressed in BMI. The international cut-points (recommended by the WHO, 2000) of BMI are used as a guidelines for determining overweight and obesity. Overweight is classified as a BMI of 25 to 29.9. The WHO (2000) distinguishes three levels of obesity: grade I is described as a BMI of at least 30.0 but less than 35.0, grade II refers to a BMI of 35.0 to 39.9 and grade III refers to a BMI of at least 40.0. Within this thesis, obesity will be defined as any BMI of at least 30 (thus incorporating all three grades).

3.4. Methods

3.4.1. Attributable deaths

The underlying method used to estimate attributable deaths involves the concept of the population attributable fraction (PAF). Mark (2005) defines the PAF (which is also referred to as the attributable or etiologic fraction) as "the proportion of morbidity or mortality in a population that can be attributed to a particular cause or risk factor" (p. 1918). It provides a quantitative estimate of the fraction of deaths that would be avoided in a situation where a risk factor or cause (in this case overweight and obesity) is reduced or eliminated from the population (Mehta & Chang, 2009). The following formula (based on Kleinbaum et al., 1982) will be used to calculate the sum of the category specific PAF:

$PAF = \sum Pe_i(RR_i - 1)/(1 + (Pe_i(RR_i - 1)))$

This formula was chosen since it is preferred when there is confounding (Rockhill et al., 1998) and it is known from the literature that there is confounding in the association between excess weight and mortality (Lawlor et al., 2006; Fulton & Shekelle, 1997). To explain the formula, Pe_i is the proportion of the population exposed to the factor (in this case, the prevalence of overweight and obesity) for the *i*th exposure category, and RR is the adjusted relative risk of mortality associated with overweight and obesity for the *i*th exposure category. The category specific attributable fraction for the unexposed group, which is *i*=0 is 0, since that RR is 1. Subsequently, attributable deaths are calculated according to the following formula:

AD = PAF x M

Were the PAF is multiplied with the total number of deaths (M).

3.4.2. Relative risks

As mentioned above, to calculate the PAF, the relative risk (RR) of dying for overweight and obese individuals relative to individuals with a 'normal' weight is used. Ideally, this would involve a nationally representative prospective study on overweight, obesity and mortality, to estimate the RR. However, such studies are not (yet) being carried out in the Netherlands. Therefore, RR estimates from published literature are used to make an indirect estimate of the RR in the Netherlands.

Published studies on the association between excess weight and all-cause mortality were identified through a systematic literature search of the electronic database Web of Science, using the selection criterion of articles only from 1990 onwards. Studies that reported on the relationship between excess weight and outcomes other than all-cause mortality, or those that did not use a prospective cohort study, were excluded from consideration. Studies were included if they provided RR estimates with the characteristics presented in box 1.

Box 1. Selection criteria for the relative risks.

Preferred characteristics, in order of preference
1. RR for adults aged 20 and above
2. Adjustment for smoking
3. RR by age categories
4. RR by gender
5. Recent prospective cohorts studies
6. RR from European prospective cohorts studies

First, RRs are preferred to be derived from participants aged 20 and above, because the focus of this thesis is on that age group. Second, adjustment for smoking. Smoking is an important confounder on the association between BMI and mortality (Lawlor et al., 2006; Fulton & Shekelle, 1997). Estimates of deaths attributable to overweight and obesity can be biased if confounding is not properly taken into account (Flegal et al., 2004, 2005). No adjustment for smoking may overestimate the risk of mortality (Lawlor et al., 2006). Other possible confounders are age, alcohol consumption, pre-existing illness and SES, although they are controversial and are not backed by any considerable evidence (Mehta & Chang, 2009). Third, RRs by age categories, because obesity-attributable mortality declines with age at all levels of obesity (Bender et al., 1999; Stevens et al., 1998). In addition, the age composition also differs across populations. Fourth, RRs by gender. The RR, as well as the prevalence of overweight and obesity, are quite different for males and females (Bellocco et al., 2010; Borrell & Samuel, 2014). Fifth, the most recent prospective cohorts studies. There is controversy regarding RR and its relation with time. Flegal et al. (2005) suggested that the RR of obesity might have declined since the early 1970s, based on data from successive NHANES surveys. However, other studies concluded that RR does not change dramatically over time (Troiano et al, 1996; Allison et al, 1999; Lew & Garfinkel, 1979; Calle et al, 1999; Murray & Lopez, 1996). Finally, RRs derived from European prospective studies are preferred. RRs can change from one population to another, and it is assumed that European RRs are more similar to those for the Dutch population than are RRs from other countries in the world.

No study met all of the criteria mentioned above. The studies that best suited the research were eventually chosen. Five studies that explored the association between excess weight and all-cause mortality were included. These studies were the Cancer Prevention Study I (CPSI) based on Allison et al. (1999), the Swedish National March Cohort (SNMC) based on Bellocco et al. (2010), National Health and Nutrition Examination Survey III (NHANES III) based on Borrell & Samuel (2014), the National Health and Nutrition Examination Survey I/II/III (NHANES I/II/III) based on Flegal et al. (2005) and a meta-analysis by Berrington de Gonzalez et al. (2010).

Characteristics of these studies can be found in Appendix I. The NHANES and CPS I include data from U.S. participants, the SNMC from Sweden and the meta-analysis combines studies from the U.S., Sweden and Australia. The study size ranged from 36,859 participants (NHANES III) to 1,462,958 participants (meta-analysis). All of the studies use self-reported weight and height except for NHANES, which uses measured weight and height. All of the studies also use the Cox proportional hazards regression model to calculate the RR and all adjust for smoking. Allison et al. (1999) adjusted for age and sex as well. Bellocco et al. (2010) adjusted for age, alcohol, vitamin and mineral supplements, education and contraceptive pills and hormone replacement therapy for females. Borrell & Samuel (2014) adjusted for age, sex, race, education and leisure-time. Flegal et al. (2005) adjusted for sex, race and alcohol consumption. Berrington de Gonzalez et al. (2010) adjusted for alcohol consumption, education, marital status, physical activity and pre-existing illness.

RR estimates were obtained for overweight and obesity separately. In cases where the authors presented RR estimates in different exposure categories, categories are combined and a weighted average based on the population number is calculated. Table 2 shows the RRs for overweight and obesity, which are derived from the five studies. It can be seen that large differences in RRs exist between the studies. The RR for obesity varies between 1.097 and 2.787, while the RR for overweight lies between 0.660 and 1.830. Some RRs for overweight have a value smaller than one, meaning that the risk of dying from overweight is lower than the risks of dying with a 'normal' weight. The studies are combined to calculate an unweighted average of the RRs of dying from overweight and obesity, as compared to the risk of dying with a 'normal' weight. One RR will be calculated for obesity, and two RRs for overweight. The studies with a RR>1 for overweight (CPS I, SNMC and the meta-analysis) will be combined, as well as the studies with a RR<1 (NHANES I/II/III and NHANES III).

Table 2. Risk of all-cause mortality associated with overweight and obesity.

Source	Gender	Characteristic participants	Age	RR Overweight	RR Obesity
CPS I [a]	Both sexes	Non-smokers only	≥30	1.112	1.560
SNMC [b]	Male		20-94.2	1.120	1.620
	Female		20-94.2	1.020	1.350
NHANES III [c]	Male		≥18	0.780	1.163
	Female		≥18	1.100	1.420
	Both sexes		18-29	1.830	2.787
	Both sexes		30-44	0.720	1.450
	Both sexes		45-64	1.080	1.810
	Both sexes		≥65	0.890	1.097
NHANES I/II/III [d]	Both sexes	Never-smoker only	25-59	0.660	1.010
	Both sexes		60-69	0.810	1.755
	Both sexes		≥70	0.900	1.250
Meta-analysis [e]	Both sexes	Healthy never-smokers only	20-49	1.370	2.657
	Both sexes		50-59	1.160	2.130
	Both sexes		60-69	1.090	1.793
	Both sexes		70-84	1.095	1.580

a Based on the Cancer Prevention Study I (Allison et al., 1999)
b Based on the Swedish National March Cohort (Bellocco et al., 2010)
c Based on the National Health and Nutrition Examination Survey III (Borrell & Samuel, 2014)
d Based on the National Health and Nutrition Examination Survey I/II/III (Flegal et al., 2005)
e Based on a meta-analysis by Berrington de Gonzalez et al. (2010)

The attributable deaths are estimated for each study separately. After completing calculations, an unweighted mean estimate of the attributable deaths is determined by adding the five estimations for each year, then dividing them by the number of studies. An unweighted approach is chosen instead of a weighted average by study size, because the quality of the data and methods of the studies differs

substantially and no order of study preference can be indicated. It is followed by a calculation of the standard deviation. The following formula is used:

$$s = \sqrt{\frac{\sum(X - Xm\ )^2}{N}}$$

Where s is the standard deviation, ∑ means the 'sum of', x is the estimate for attributable mortality of each study, Xm is the unweighted mean of the five studies, and N is the number of studies, which is five is this case.

3.4.3. Potential gain in life expectancy

To see the effect of obesity on life expectancy, the PGLE when obesity were to be eliminated is estimated. A period life table is used to calculate the actual life expectancy between 1981 and 2012 in the Netherlands. Life table information is available from the HMD from 1981 to 2009. The ${}_na_x$ from 2009 is therefore used to calculate the life table for 2010 to 2012. Other life table information for 2010-2012 is calculated with the population numbers and number of deaths obtained from Statistics Netherlands for 2010-2012. The HMD has smoothed observed death rates at age 80 and above (Wilmoth et al., 2007). The death rates were not smoothed for the years 2010 to 2012, as it was unclear which smoothing technique was used by the HMD.

An associated single decrement table (ASDT), where the decrement is obesity, is used to calculate the life expectancy when obesity had been eliminated from the population of the Netherlands between 1981 and 2012. The PGLE is the difference between the two estimated life expectancies. The obesity-attributable deaths are used to calculate the life expectancy in the ASDT. The approach by Chiang (1968 in Preston et al., 2001) is chosen to calculate the ASDT, as it is suitable when relatively small causes of decrement are deleted (Preston et al., 2001). This approach assumes that the force of decrement function from cause *i* is proportional to the force of decrement function from all causes combined in each age interval. Usually, life expectancy is calculated for males and females separately. This is because women tend to have a lower mortality rate at every age, which results in a higher life expectancy as compared to males (Kinsella, 1992). Unfortunately, RRs for males and females separately by five-year age categories were not available. Therefore, life expectancy will be calculated for males and females combined.

4. Results

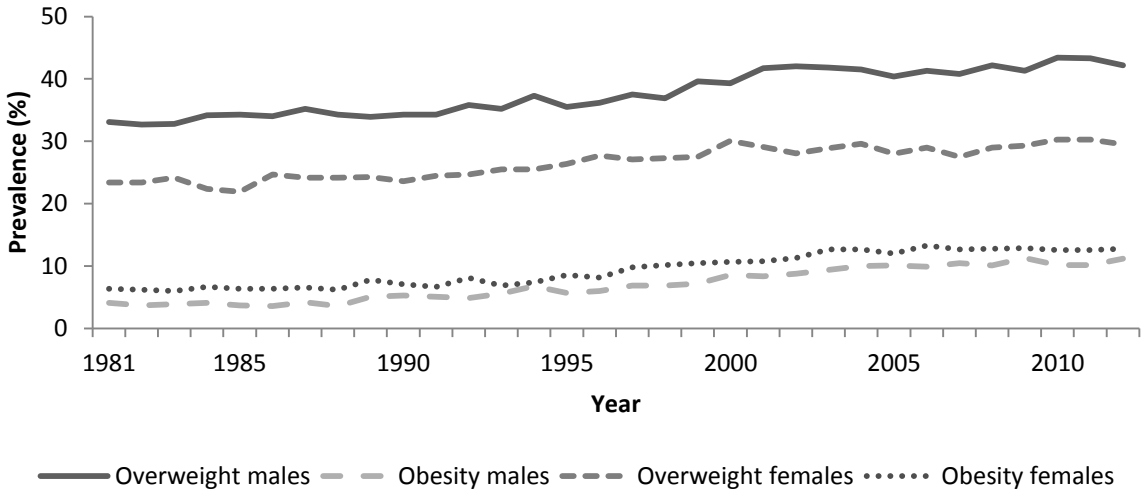
4.1. Prevalence of overweight and obesity

Overweight and obesity levels in the Netherlands have increased in the last 30 years from a combined percentage of 33.4% in 1981 to 47.9% in 2012: obesity increased from 5.3% to 12.0%, while

overweight increased from 28.2% to 35.9%. The most significant increase was from 1981 to 2003. After 2003, increase in overweight and obesity prevalence has been levelling off.

If we look at gender differences and overweight and obesity separately (figure 3), in 1981, 37.2% of the male adult population was overweight and only 4.1% was obese. In 2012, these figures increased respectively to 53.4% and 11.2%. For females, 29.8% of the population was overweight and 6.4% obese in 1981; in 2012 these numbers increased respectively to 42.3% and 12.8%. During the last 30 years, males, as compared to females, have always had a higher percentage of overweight, for obesity, the situation is reversed. Age-specific overweight and obesity prevalence data can be found in Appendix II. The highest prevalence of overweight males are those aged 40 and above. The strongest increase between 1981 and 2012 is in the age categories of 30-40 years (12.5%) and 40-50 years (8.8%). The prevalence of overweight in the age category 75+ made a remarkable drop from 51.4% in 1981 to 30.2% in 1982. In 2012, the prevalence of overweight was 44.7%. This is the only age group that experienced a decrease between 1981 and 2012. All male age groups experienced a strong decrease in obesity between 1981 and 2012. The age category 20-30 experienced the largest increase, with a near quintupling from 1.1% to 5.3%. The age categories 50-55 and 65-75 increased three to four-fold. Overweight females in the age category 20-30 experienced the greatest increase, from 7.9% in 1981 to 19.1% in 2012. Only two categories decreased in the prevalence of overweight. The age category 50-55 decreased from 30.7% to 29.8%, and the age category 65-75 from 44.3% to 38.2%. In all females age categories obesity increased between 1981 and 2012. The age category 30-40 experienced the greatest increase, as it quadrupled between 1981 and 2012.

Figure 3. The prevalence of overweight and obese males and females aged 20 and above in the Netherlands, 1981-2012.



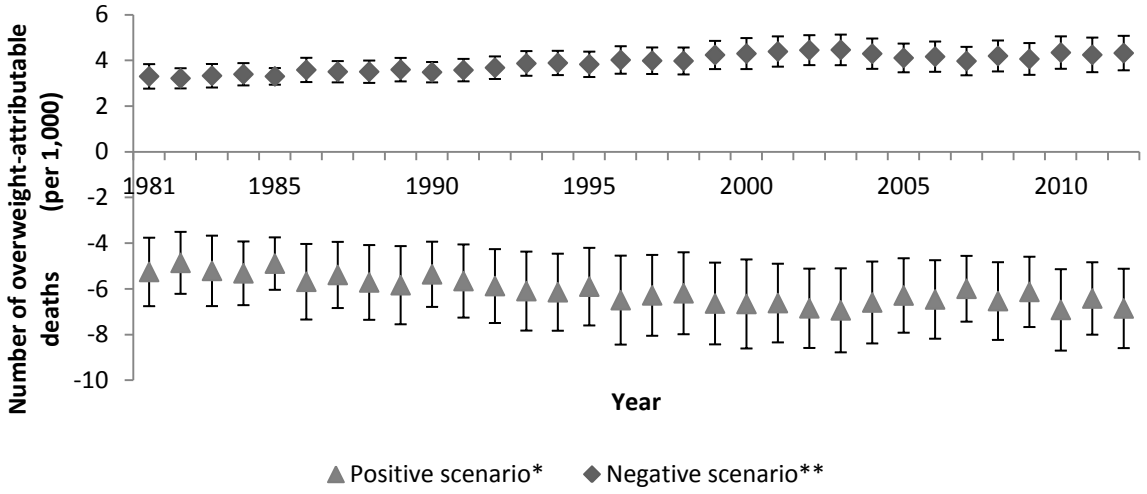
Source: Statistics Netherlands, 2013.

4.2. Overweight- and obesity-attributable deaths

Figure 4 and 5 respectively the deaths attributable to overweight and obesity. A distinction is made between a positive and negative scenario for overweight-attributable deaths. The positive scenario uses the RR from the NHANES I/II/III and NHANES III. The RRs from both studies are smaller than one, which means that the risk of dying for an individual with a 'normal' weight is higher than for those who are overweight. Being overweight significantly decreases all-cause mortality according to these studies. This results in a negative number of deaths. The number of deaths in the positive scenario increased between 1981 and 2012. The mean estimate of the total number of overweight-attributable deaths in 1981 was -5,264, while the corresponding figure for 2012 was -6,861. This means that compared to 1981, there were 1,597 fewer deaths. For the NHANES III, the estimated number of overweight-attributable deaths decreased from -3,679 in 1981 to -4,806 in 2012 and NHANES I/II/III, decreased from -6,675 in 1981 to -8,279 in 2012.The standard deviation is quite large in this scenario because the attributable death from the NHANES I/II/III and NHANES III are quite different. The attributable deaths from the NHANES I/II/III are double the attributable deaths from NHANES III.

The negative scenario includes the CPS I, the SNMC and the meta-analysis. According to these studies, being overweight significantly increases all-cause mortality. The mean estimate of the total number of overweight-attributable deaths in this negative scenario in 1981 was 3,295, while in 2012 it was 4,315. Compared to 1981 there were 1,020 more deaths. The estimated number of overweight-attributable deaths from the CPS I increased from 3,400 to 5,374, the attributable deaths of SNMC from 2,592 to 3,666 and the meta-analysis from 3,893 to 3,906. The standard deviation is small, especially compared to the positive scenario.

Figure 4 The number of overweight-attributable deaths among males and females aged 20 and above in the Netherlands , 1981-2012.



*Includes the NHANES III and NHANES I/II/III
**Includes the American Cancer Society Cancer Prevention Study I, the Swedish National March Cohort and the Meta-analysis

The number of obesity-attributable deaths experienced a significant increase between 1981 and 2012, especially in the first 23 years. The mean estimate of the total number of obesity-attributable deaths was 2,785 in 1981 and 6,425 in 2012. Approximately 3,640 more obesity-attributable deaths occurred in 2012 than in 1981. The estimate of the total number of obesity-attributable deaths of CPS I increased from 3,201 to 8,754, NHANES III from 1,085 to 1,740, SNMC from 2,642 to 7,471, Meta-analysis from 4,507 to 8,326 and the NHANES I/II/III, from 2,488 to 5,834. A total of 153,472 deaths can be attributed to obesity within 31 years. Results pertaining to age-specific obesity-attributable deaths can be found in Appendix III. In general in 2012, the higher the age category, the higher the attributable deaths. The only exception is the age category 70-74, which has a lower number of deaths than age categories of 65-69 and 75-79. The situation was different in 1981. The attributable deaths increased with each age category until the age category 65-69; thereafter it decreased and it remains stable around 370 deaths for each of the remaining age categories. All age categories experienced an increase between 1981 and 2012. The age category 85+ experienced the largest increase, namely from 382 deaths to 2,220 deaths, representing a difference of 1,838 deaths.

Figure 5. The number of obesity-attributable deaths among males and females aged 20 and above in the Netherlands , 1981-2012.

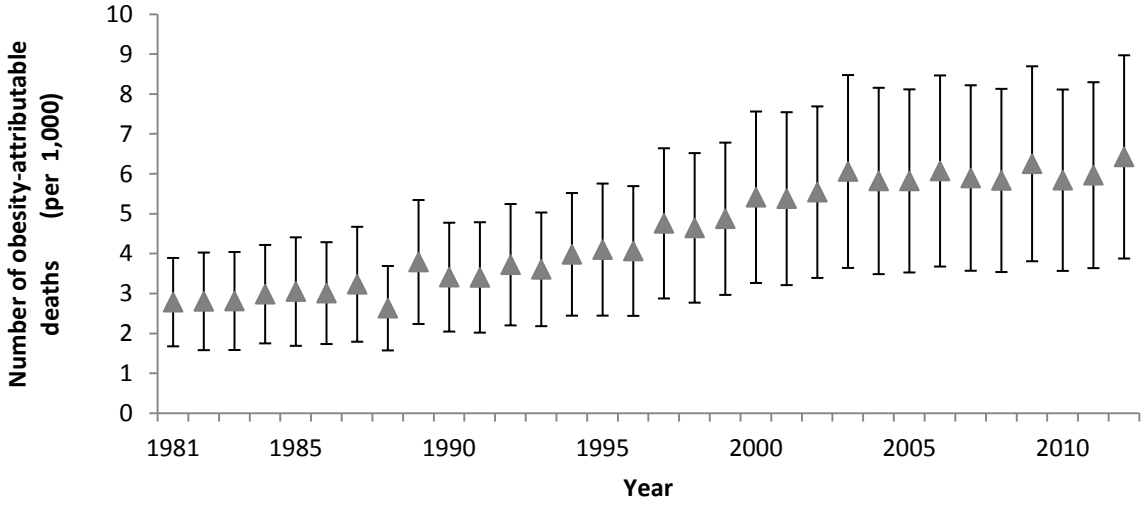
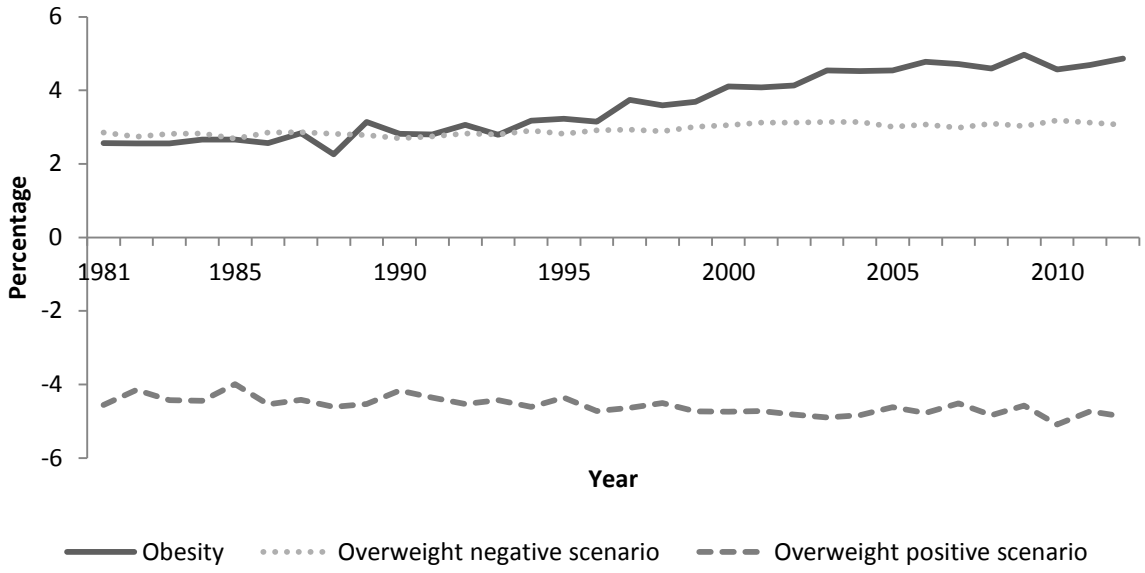


Figure 6 shows overweight- and obesity-attributable deaths as a percentage of the total deaths (all ages). There is a slight increase in the percentage of overweight (negative scenario), from 2.9% in 1981 to 3.1% in 2012. The other overweight category (positive scenario) also shows a slight decrease in overweight, from 4.6% in 1981 to 4.9% in 2012. The percentage of obesity remained stable from 1981 to 1990, hovering around 2.6%. From 1990 to 1996 a slight increase occurred, followed by a much sharer increase after 1996, which ultimately results in a percentage of 4.9% in 2012.

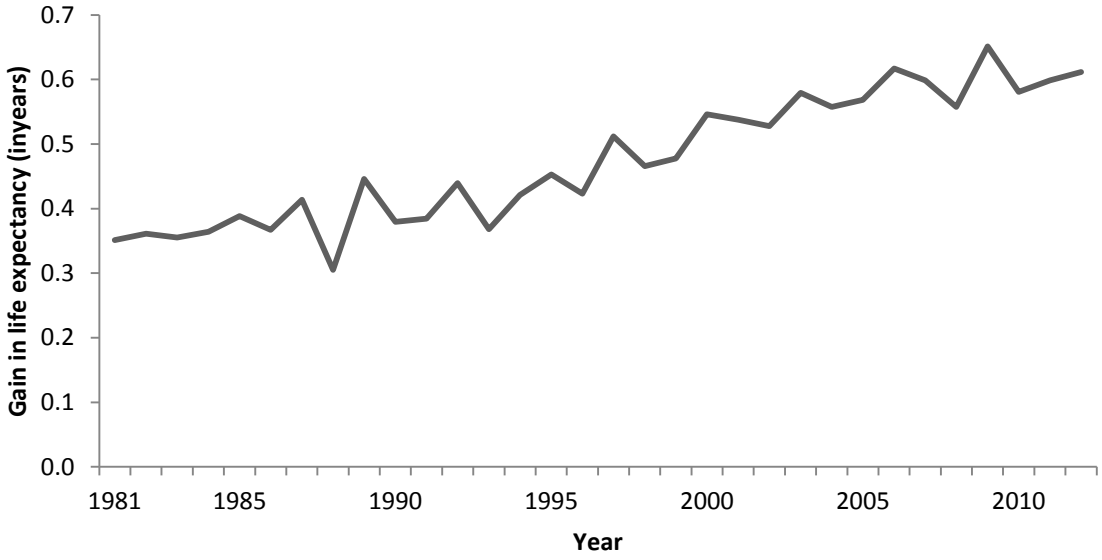
Figure 6. Percentage overweight- and obesity-attributable deaths of the total deaths in the Netherlands, 1981-2012.



4.3. Potential gain in life expectancy

Figure 7 presents the PGLE if obesity was eliminated in the Netherlands between 1981 and 2012. The PGLE in 1981 was 0.35 years. It steadily increases thereafter to 0.61 years in 2012 (with the exception of a large drop to 0.31 in 1988). The PGLE experienced an increase of 174% between 1981 and 2012.

Figure 7. The potential gain in life expectancy among males and females if obesity would be eliminated in the Netherlands, 1981-2012.



5. Conclusion and discussion

5.1. Summary of results

The aim of this thesis was to investigate the trends in the mortality burden attributed to overweight and obesity in the Netherlands between 1981 and 2012, by estimating overweight- and obesity-attributable deaths and the potential gain in life expectancy when obesity were to be eliminated. The results demonstrate an upward trend in obesity-attributable deaths from 2,785 in 1981 to 6,425 in 2012. There is controversy regarding overweight-attributable deaths. According to some literature, overweight increases all-cause mortality. If the population attributable fraction is calculated with relative risks supporting this assumption, overweight-attributable deaths are increasing from 3,295 in 1981 to 4,315 in 2012. If the population attributable fraction is calculated with relative risks which contradicts this, the mean estimate of the total number of overweight-attributable deaths in 1981 was -5,264 and in 2012, -6,861. Obese individuals have a lower life expectancy compared to individuals with a 'normal' weight. The potential gain in life expectancy if obesity were to be eliminated increased from 0.35 years 1981 to 0.61 years in 2012.

5.2. Evaluation of data and methods

This study has several strengths and weaknesses. The first limitation is that body mass index is not the most suitable measure of adiposity. Body mass index does not distinguish between muscle and fat and provides no information about the body fat distribution (Aronne, 2002). Other measures such as waist circumferences and waist-to-hip ratio are more suitable (Janssen et al., 2004; Savva et al., 2000). However, most studies use body mass index as a measure of weight status because of its ease of collection. In addition, data sources in the Netherlands often provide overweight and obesity prevalence in using body mass index.

Second, the prevalence of overweight and obesity used in this thesis is estimated from self-reported data. Self-reported height and weight is known to underestimate overweight and obesity prevalence, as individuals tend to over-report their height and under-report their weight (Flegal et al., 2013; Karasu, 2013). Several researchers have tried to create a calibration model, which uses a linear regression equation to correct this self-reported data (Kuskowska-Wolk et al., 1989; Nyholm et al., 2007). The calibration model has been debated, as it is difficult to use and does not correct completely for the systematic bias in self-reported weight and height in population studies (Nyholm et al., 2007). A general set of correction factors, based on data such as coefficients from regression models, is not currently available to correct for self-reported body mass index (Gosse, 2014).

Third, estimates of deaths attributable to obesity can be biased if confounding and effect modification are not properly taken into account or if the relative risks are not estimated accurately. Appropriate confounding if important since estimates of attributable deaths are very sensitive to minor changes in relative risk estimates (Flegal et al., 2005). Debate over the importance of overweight and obesity for

all-cause mortality generally focuses on whether it is appropriate to adjust (or exclude) the analysis for smoking, age, alcohol consumption, pre-existing illness and socio-economic status. Bias may also result from failure to control for unknown confounders that are associated with body weight and mortality. The relative risks used from the published literature may be biased because age, alcohol consumption, pre-existing illness and socio-economic status are not taken into account for each study. However, there is controversy regarding these factors and no considerable evidence that they are confounders (Mehta & Chang, 2009).

Fourth, there is controversy over the accuracy of the population attributable faction estimates to describe the percentage of total mortality due to overweight and obesity (Bender et al., 1998; Levine, 2008). Attributable mortality generally assumes a causal link between a risk factor and an outcome (Rockhill et al., 1998), but the associations are not necessarily causal. Even if body weights were reduced to the reference level, risks might not return to the level of the reference category (Flegal et al., 2005; Levine, 2008). In the case of obesity, this means that all obese individuals would instantaneously possess the same risk of dying as 'normal' weight individuals, thus ignoring any previous effects of obesity earlier in life. However, the population attributable faction is the only method available for making an estimation of overweight and attributable deaths, and researchers frequently use it.

Fifth, the estimate of overweight- and obesity-attributable mortality is based on the assumption that Relative risks calculated from past cohorts apply to the current population, which may not be true. Flegal et al. (2005) suggested that the relative risk of obesity might have declined since the early 1970s, based on data from successive NHANES surveys. Gregg et al. (2005) provided indirect evidence supporting a declining risk of mortality among those with a body mass index of 25.0. However, other studies concluded that relative risks do not change dramatically over time (Allison et al, 1999; Calle et al, 1999; Lew & Garfinkel, 1979; Murray & Lopez, 1996; Troiano et al, 1996).

While the overall effect of the above limitations on the estimates of overweight- and obesity-attributable deaths are not very clear, quantification must nevertheless be attempted to focus debate on such a major cause of mortality burden as excess weight. Overweight and obesity are usually considered risk factors that predispose individuals to chronic diseases, rather than being diseases of themselves. As such, increases in their prevalence do not carry the same public health message as increases in overweight- and obesity-attributable deaths or the potential gain in life expectancy if obesity were to be eliminated.

This research has also several strengths. One major strength is the ability to estimate changes in the number of overweight- and obesity-attributable deaths and the potential gain in life expectancy if obesity were to be eliminated over a time span of 31 years, based on prevalence data of overweight and obesity, the relative risks and life table data. In addition to previous studies (e.g., Banegas et al.,

2003; Katzmarzyk & Ardern, 2004), not merely one prospective study as a data source for the relative risk is used, but five studies are combined to estimate an unweighted average of the relative risks. Estimates of attributable fractions, and thus numbers of deaths, are very sensitive to minor changes in relative risks estimates (Flegal et al., 2005). Thus, results are affected by the precision and bias in relative risk estimates. Through combining several studies, a more reliable outcome is pursued. In addition, to this researchers knowledge, none of the previous studies have examined trends in overweight- and obesity-attributable deaths and the potential gain in life expectancy if obesity were to be eliminated in the Netherlands.

5.3. Interpretation results

The prevalence of overweight and obesity has risen tremendously in the past decades. The increase in the prevalence between 1981 and 2012 confirms that the Netherlands in now in the fourth stage of the Nutrition Transition. Although the Netherlands has reached high prevalence levels of overweight and obese, the prevalence is still relatively low in comparison with other European countries. In the EU, 50.1% of the adult population was overweight or obese in 2008. The prevalence of overweight and obesity among adults exceeds 50.0% in 15 of the 27 EU countries. In the Netherlands, 47.1% of the adults were overweight or obese, lower than the European average (OECD, 2010). In 2008, 11.5% of males and females were obese in the Netherlands. Except for Sweden, Romania, Switzerland, Norway, Italy, France and Denmark, no other country in Europe has such a low prevalence of obesity (WHO, 2014b). The prevalence of obesity ranges from 7.9% Romania to 24.5% in the United Kingdom. The prevalence of overweight and obesity has more than doubled over the past 20 years in most European countries. In the Netherlands, obesity more than doubled as well (from 5.3% in 1981 to 12.0% in 2012), as was expected. However, the increase in the prevalence of overweight lagged behind (from 28.2% in 1981 to 35.9% in 2012).

Both the Nutrition Transition Theory and the literature showed that the increasing prevalence of overweight and obesity had several health consequences. The literature shows that the association between overweight and mortality remains controversial. This is in line with the results of this research. It is unclear whether overweight results in a significant decrease or increase of all-cause mortality. This research estimated a decrease from -5,264 deaths to -6,861 deaths in 2012, or an increase from 3,295 deaths in 1981 to 4,315 deaths in 2012. Evidence suggested that obesity significantly increases all-cause mortality as compared to individuals with a ‘normal’ weight. As a consequence of the increasing prevalence of obesity in the Netherlands, obesity-attributable deaths has also shown an upward trend between 1981 and 2012. The only year that is an exception in 1988, which shows a strong decline in the estimated number of obesity-attributable deaths: the number of attributable deaths dropped from 3,23 in 1987 to 2,63 in 1988, and increased to 3,79 in 1989. This significant decrease can be explained by a large decrease in the prevalence of obesity in this year, especially in the age category 75+. In 1988, the prevalence of obesity in this age category was 4.3%,

while in both 1987 and 1989, the prevalence was 9.4%. The drop in 1988 seems an measurement error, since it not reasonable that more than 50% of the obese individuals aged 75 and above in 1987 all lose a lot of weight in on year, after which a lot of individuals gain a lot of weight in the subsequent year. In addition, if the decrease were true, a substantial increase in the prevalence of overweight would be expected, and this is not seen in 1988. Overall, an upward trend is seen in overweight- and obesity-attributable deaths. These results support the study by Katzmarzyk & Ardern (2004) that obesity-attributable mortality is increasing. These researchers estimated an increase in overweight- and obesity-attributable deaths in Canada between 1985 and 2010 for adults aged 20 to 64. The annual number of deaths attributable to overweight and obesity increased from 2,514 in 1985 to 4,321 in 2000.

Banegas et al. (2003) estimated deaths attributable to excess weight (BMI ≥25) in the EU for 1997. Approximately 7.7% of the total deaths in the Netherlands were attributable to overweight and obesity. In this thesis, 4.3% of all deaths in 1997 were attributed to obesity, while overweight was the underlying cause of 2.9% or -4.6% of all deaths. If it is assumed that overweight significantly decreases all-cause mortality, it can be estimated that 7.2% of all deaths in 1997 are attributed to obesity and overweight. Although the results seem similar, it is quite difficult to compare these numbers because Banegas et al. (2003) uses a different age category (age 25+), source of body mass index prevalence (the PAN-EU study) and relative risks (CPS I by Allison et al. (1999)). The main contributor to the difference is the relative risks, since estimates of attributable deaths are very sensitive to minor changes in relative risk estimates. The relative risks used by Banegas et al. (2003) are also unadjusted while adjusted relative risks are used in this thesis. If adjusted relative risks would have been used, than the attributable deaths would be higher.

Konnopka et al. (2011) estimated that 4.5% of the total deaths in 2002 were attributed to overweight and obesity among adults aged 18 and above in Germany. This thesis has shown that 4.4% of the total deaths in 2002 in the Netherlands were attributable to obesity. Depending on the role of overweight (positive or negative effect on all-cause mortality), the proportion of obesity-attributable deaths is much lower or much higher in the Netherlands. The prevalence of obesity in Germany is 12.9% in 2002, and compared to the 9.7% for the same year in the Netherlands much higher. Therefore, it is expected that obesity-attributable mortality would be higher in Germany than in the Netherlands,

The number of attributable deaths in the Netherlands is at all times lower than the attributable deaths in the U.S., independent of the study (Allison et al., 1999; Flegal et al., 2005; McGinnis & Foege, 1993; Mokdad et al. 2004,2005). This is attributable to a larger population and a much higher prevalence of overweight and obesity found in the U.S. According to the WHO (2009), the prevalence of overweight (BMI ≥25) in the U.S. was 69.4% and 31.8% for obesity (BMI ≥30) in 2008.

Olshansky et al. (2005) calculated the potential gain in life expectancy if obesity were to be eliminated in the U.S. for 2000. They estimated that the potential gain in life expectancy at birth if obesity did not exist, would be 0.33 to 0.93 years for white males, 0.30 to 0.81 years for white females, 0.30 to 1.08 years for black males, and 0.21 to 0.73 years for black females. It was estimated that the potential gain in life expectancy in 2000 in the Netherlands was 0.55 years for both sexes. As expected, this estimation lies between the estimation for each category of Olshansky et al. (2005).

Preston & Stokes (2011) estimated the potential gain in life expectancy at age 50 due to obesity (BMI ≥30) as an proportion of all-cause mortality attributable to obesity for adults aged 50 to 59 years for several European countries in 2006. They estimated a potential gain in life expectancy at age 50 of 0.69 years for females and 0.73 years for males in the Netherlands. The current study estimates the potential gain in life expectancy at birth at 0.62 years for both sexes in 2006. The potential gain in life expectancy at age 50 would be even lower than 0.62 years if that would have been estimated. Therefore, there is a significant difference between the results from this thesis and the study by Preston & Stokes (2011) in the estimation of the potential gain in life expectancy. Although the exact relative risks used by Preston & Stokes (2011) are unknown, the difference can probably explained by a gap in the relative risks used in their study and the relative risks used in this thesis.

Hoeymans et al. (2010) estimated the potential gain in life expectancy if obesity (BMI ≥30) were to be limited for the Netherlands in 2008. The potential gain in life expectancy would be 0.3 years. The potential gain in life expectancy in the current study is estimated to be 0.56 years in 2008, almost twofold of the estimation of Hoeymans et al. (2010). The difference can be explained be the use of different relative risks, as Hoeymans et al. (2010) used relative risks from the RIVM *Chronische Ziekten Model*. Van Baal et al. (2006) and In 't Panhuis-Plasmans et al. (2012) all estimated that the hypothetical elimination of overweight (BMI ≥25) would lead to a potential gain in life expectancy of 0.8 years at age 20. Because their year of calculation is unknown, a comparison cannot be made with the current study.

Studies on past trends in the potential gain in life expectancy if obesity were to be eliminated have never been undertaken. However, it can be assumed than if there is an upward trend in the prevalence of obesity, and subsequently an upward trend in obesity-attributable mortality, an increase in the potential gain in life expectancy if obesity were be eliminated is a logical consequence. Therefore, the increase in the potential gain in life expectancy if obesity were be to eliminated that we see in the Netherlands (from 0.35 years in 1981 to 0.61 years in 2012) is what could also be expected to happen in all countries that experience a rise in the prevalence of obesity.

5.4. Overall conclusion and implications

The population attributable fraction and the potential gain in life expectancy are useful methods to quantify overweight and obesity and to see the magnitude of their deleterious effects on health on the

population level. Overweight- and obesity-attributable mortality and the potential gain in life expectancy if obesity were eliminated substantially increased between 1981 and 2012. Obesity accounted for approximately 153,472 deaths between 1981 and 2012 and decreases life expectancy between 0.63 and 1.05 years. Therefore, overweight and obesity are an important public health problem for the Netherlands.

5.4.1. Further research

There are gaps in the current knowledge about overweight and obesity and their association with mortality. The effects of overweight on mortality also remain controversial, as have been shown in the literature review. A better understanding of the relationship between excess weight and mortality will lead to a more accurate estimation of overweight- and obesity-attributable, as well as the potential gain in life expectancy if obesity were to be eliminated.

More research is necessary to see if overweight is protective against mortality of that being overweight increases the risk of mortality. Knowledge is also not complete regarding the health effects of differing lifetime trajectories of body weight, given that studies evaluating risks of obesity usually assess weight at a single point in time rather than throughout life (Mark, 2005). Further research is required to understand the full range of short- and long-term health risks associated with overweight and obesity. More research into confounders of the relation between excess weight and mortality is also essential to make more accurate relative risk estimate. Research can be done in already existing probable confounders such as pre-existing illness and alcohol consumption, or into unknown confounders.

The time lag effect of overweight and obesity on mortality has received little attention in the literature. This effect means that present mortality rates are the consequence of past exposure to risk factors. Being overweight or obese for an extended period of time is required before an effect is evident on health problems and mortality rates. Law & Wald (1999) found a time lag between an increase in fat consumption and its effect on heart disease risk of at least 25 years. Dyer et al. (2004) have argued that it takes 15 years or even more for obesity to have its full impact on cardiovascular mortality. Although we can already see some effect on attributable deaths and the life expectancy due to excess weight in the Netherlands, it is not clear how long it will take to see the full effect of increasing overweight and obesity prevalence since the 1980s on health outcomes at the population level. Some researchers think that that the current increases in diseases related to excess weight represent the tip of the iceberg and expect that the real impact of obesity will not be realized until younger obese individuals begin to age and develop obesity related diseases (Kumanyika, 2001; Sturm et al., 2004).

Finally, a national representative prospective cohort study on excess weight and mortality does not exist in the Netherlands. A prospective cohort study would give more insight in the relationship between excess weight and morbidity and mortality, and relative risks of dying from overweight and

obesity could be calculated with these data. Preferably, data on height and weight would be measured instead on self-reported in the study. It would therefore be ideal if such a study could be undertaken.

5.4.2. Policy implications

It is clear from the results of the current research that overweight and obesity are an important public health problem. The Dutch National Health Council confirmed the seriousness of the epidemic of overweight and obesity in 2003 (Gezondheidsraad, 2003). The goals of the government since 2006 have been to reduce the prevalence of overweight and obesity among children, and to stabilize the prevalence among adults (VWS, 2006). The main focus of related policies is to increase physical activity, for instance by encouraging individuals (by advertisements and by subsidizing community sport coaches) to do moderate physical activities for at least 30 minutes a day (VWS, 2011). In addition, the policies also try to stimulate a healthy lifestyle. Even though the government is going in the right direction to battle overweight and obesity among the Dutch population, the economic burden is still tremendously high, as 2.2% of the total health care expenditure in 2010 was spend on diseases attributable to excess weight (In ’t Panhuis-Plasmans et al., 2012). To decrease these health care costs, as well as the decrease in the morbidity and mortality burden for individuals, more effort has to be put into the prevention of individuals becoming overweight or obese, and eventually decreasing the prevalence of overweight, and especially obesity. Focus should not only be on adults, but on children as well. This goal can be reached in several ways; for example research on relevant issues, through campaigns providing information on healthy diet and warning for the individual health consequences of excess weight and subsidizing healthy foods at school.

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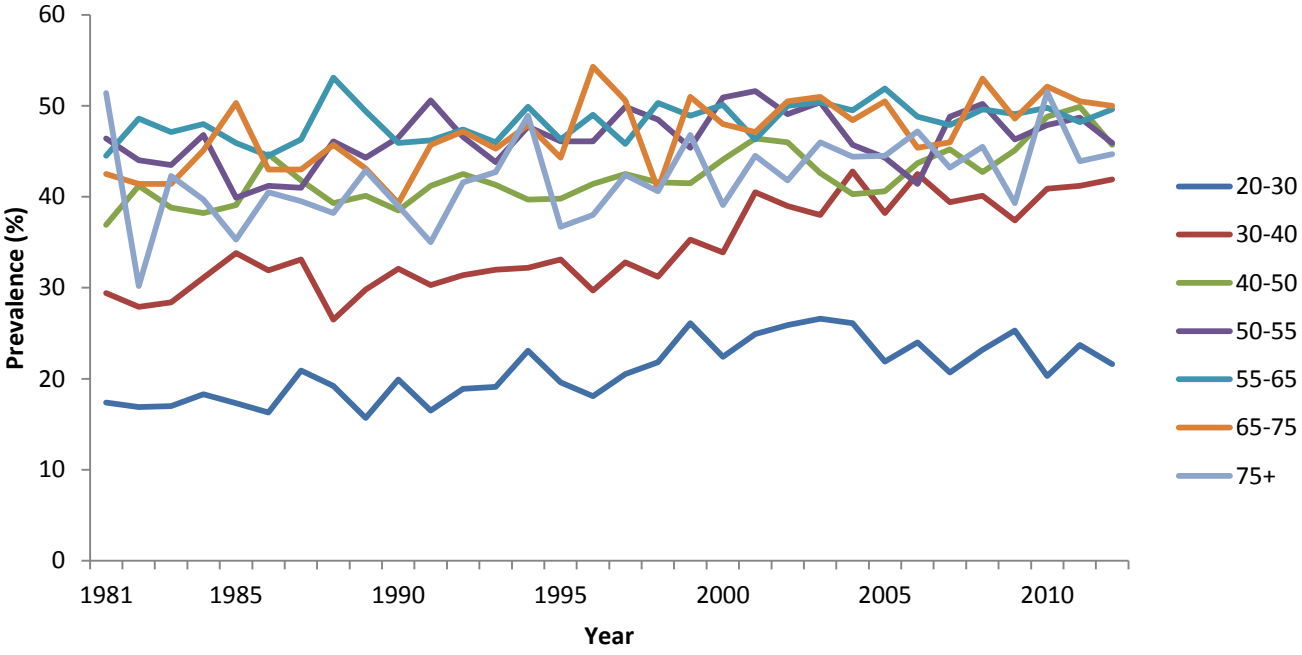
Appendix I: Characteristics of the data sources

Source	Year	Study	N	Adjustment	Country	Age	% female	Weight and height	Date	Reference category
Berrington de Gonzalez et al.	2010	Different cohort studies*	1462958	Smoking status, alcohol, education, marital status, physical activity, pre-existing illness	USA, Sweden, Australia	19-84	58	Self-reported	1972-2002 (study entry)	22.5- 25
Bellocco et al.	2010	Swedish National March Cohort	40729	Age, smoking, alcohol, vitamin and mineral supplements, education, contraceptive pills and hormone replacement therapy (female only)	Sweden	20-94.2	64	Self-reported	1997-2007	18.5-25
Flegal et al.	2005	National Health and Nutrition Examination Survey I, II,III	36859	Sex, smoking status, race, alcohol	USA	25+	NA	Measured	I: 1971-1975 II: 1976-1980 III: 1988-1994	18.5-25
Borrell & Samuel	2014	National Health and Nutrition Examination Survey III	16868	Age, sex, race, education, smoking, leisure-time	USA	18+	52	Measured	1988-1994	18.5-25
Allison et al.	1999	Cancer Prevention Study I	410817	Age, sex, smoking status	USA	30+	80	Self-reported	1959-1973	23-25

* NIH-AARP Diet and Health Study, Adventist Health Study 1, Agricultural Health Study, Breast Cancer Detection Demonstration Project Follow-up Study, California Teachers Study, CLUE I & II, Cohort of Swdish Men, Health Proffesionals Follow-up Study, Iowas, Women's Health Study, Melbourne Collaborative Cohort Stuy, Nurses' Health Study, NYU Women's Health Study, Physicians' Health Study, Postate Lung Colorectal and Ovarian Cancer Screening Trial, Swedisch Mammography Cohort, US Radiologic Technicians Cohort, VITamins and Lifestyle Study, Women's Health Study, Women's Lifestyle and Health Study

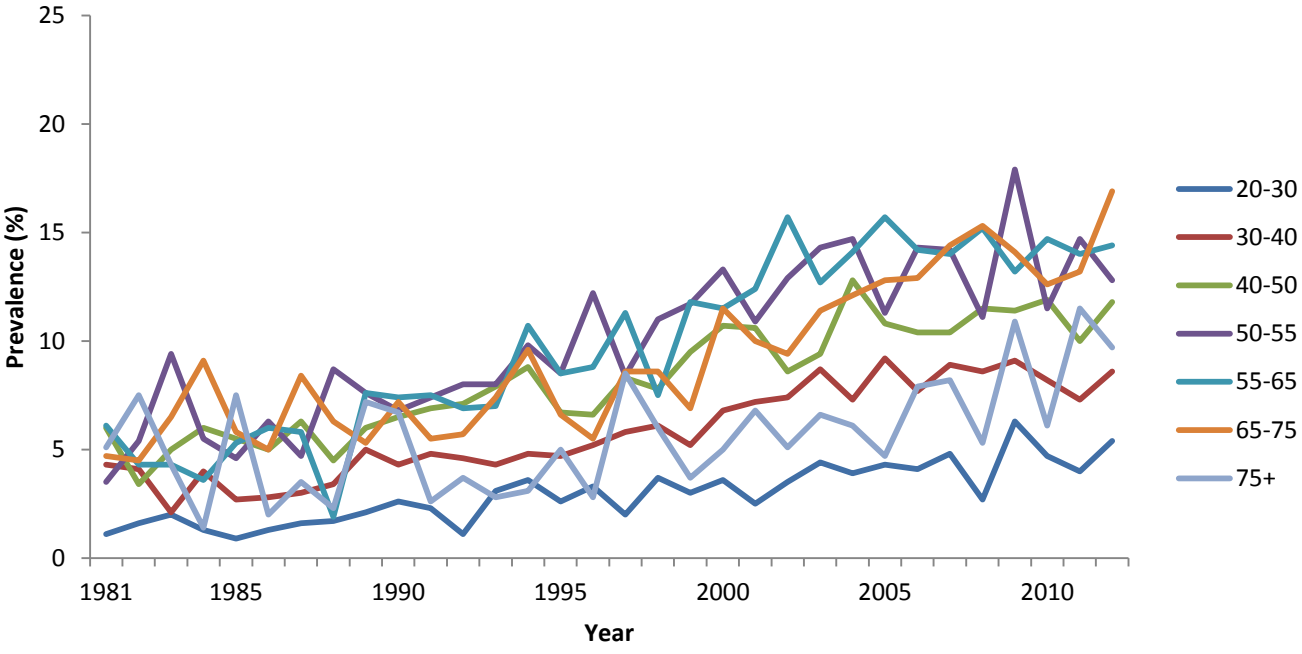
Appendix II Age-specific prevalence of overweight and obesity

Age-specific overweight prevalence among males in the Netherlands, 1981-2012



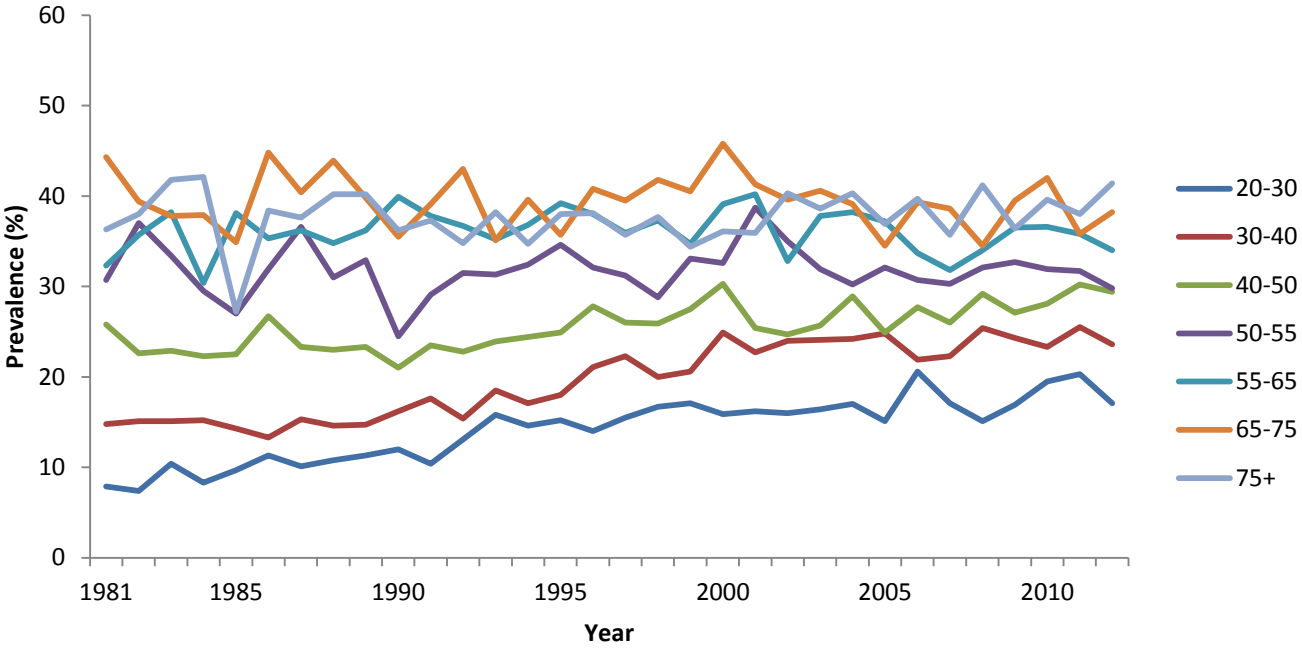
Source: Statistics Netherlands, 2013.

Age-specific obesity prevalence among males in the Netherlands, 1981-2012



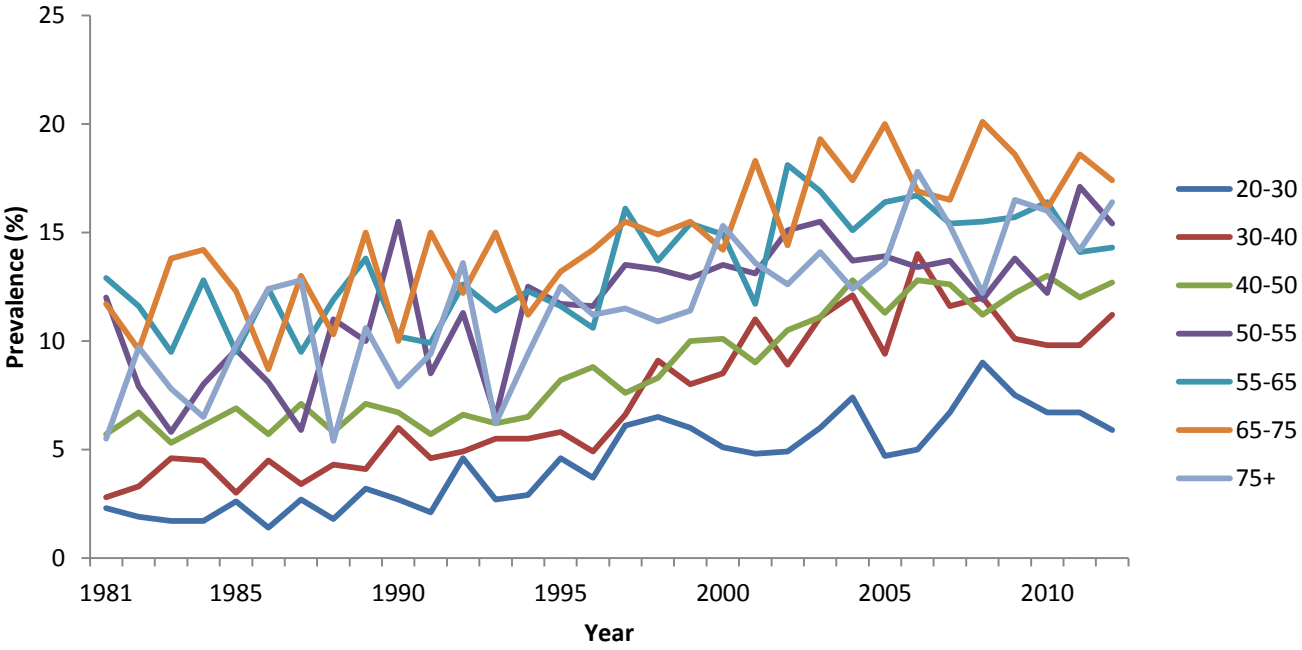
Source: Statistics Netherlands, 2013.

Age-specific overweight prevalence among females in the Netherlands, 1981-2012.



Source: Statistics Netherlands, 2013.

Age-specific obesity prevalence among females in the Netherlands, 1981-2012.



Source: Statistics Netherlands, 2013.

Appendix III: Age-specific obesity-attributable deaths