

**SELF-REPORT DIETING ATTEMPTS AND  
INTENTIONAL WEIGHT LOSS IN A GENERAL  
ADULT POPULATION**

ASSOCIATIONS WITH LONG-TERM WEIGHT GAIN AND  
RISK OF TYPE 2 DIABETES

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# ABSTRACT

Obesity is a worldwide, major public health problem. It contributes to the risk of several noncommunicable diseases (NCDs), health conditions, and mortality; impairs functional capacity and the quality of life, and places substantial burden on health care systems and economies. Along with the obesity epidemic, dieting has become popular, and indeed successful weight loss has been demonstrated to decrease the risk of many NCDs. Weight loss, however, is difficult, and further, weight maintenance after weight loss appears to be nearly impossible for the majority of individuals. Accordingly, self-report dieting attempts and intentional weight loss (IWL) have been indicated to predict subsequent weight gain in the long term. Weight loss is not limited solely to individuals with obesity, but dieting is common among individuals within each weight category. Despite dieting being common at the population level, the research on its associations in general adult populations is scarce.

The aims of this thesis were to study which factors (sociodemographic, lifestyle, metabolic, somatic health, and mental health) are associated with dieting; whether dieting modifies the association between quality of diet and obesity; whether dieting predicts long-term changes in body mass index (BMI) and waist circumference (WC), and the incidence of type 2 diabetes (T2D); and whether several background factors modify these associations.

The study was based on data from the nationally representative Health 2000 Survey and on its follow-up, the Health 2011 Survey, 11 years later. The study sample consisted of participants aged 30 and older at baseline (n=8028). Of this sample, 6771 individuals participated in health examinations in 2000. Of the individuals invited to participate in Health 2000, 6319 were invited to participate in Health 2011. Of this sample, 4006 participated in health examinations in 2011. In Health 2000/2011, information on health, wellbeing and functional capacity was collected through health examinations (including measurements and blood samples), interviews and questionnaires. Information on dieting attempts (having tried to lose weight), IWL (having tried and having lost weight), and changes in weight during the previous year was collected via a questionnaire at baseline. A validated food frequency questionnaire (FFQ) was used to measure the habitual diet of the participants, and dietary data were used to form a modified Alternate Healthy Eating Index (mAHEI), representing quality of diet. Height, weight and WC were measured during the health examinations in both Health 2000 and Health 2011. Information on incident T2D during a 15-year follow-up was drawn from national health registers. The statistical analyses were based on linear and logistic regression models, and on Cox's proportional hazards model.

In all, at baseline, 39% of women and 24% of men had attempted to diet and 15% of women and 10% of men had achieved IWL during the previous year. The dieters (BMI 29.1 [SD 4.97] kg/m<sup>2</sup>) were more obese than the non-dieters (BMI 25.7 [SD 4.17] kg/m<sup>2</sup>). Dieting attempts and IWL were associated with several sociodemographic, lifestyle, somatic health and psychological factors. Most of the factors had parallel associations among both sexes. However, dieting and BMI showed a stronger direct gradient in men than in women. Moreover, dieting was positively associated with having diabetes, hip or knee osteoarthritis, and short habitual sleep only among men. Dieting attempts modified the association between quality of diet and obesity; while among the non-dieters, no association emerged, among the dieters, higher quality of diet was indicatively associated with a higher likelihood of overweight or obesity.

Dieting attempts, weight loss, and weight fluctuation during the previous year were associated with an increase in BMI and WC during an 11-year follow-up. The increase was greater among dieters with normal weight than among non-dieters with normal weight. Conversely in those with initial obesity, the increases among dieters and non-dieters did not differ. During a 15-year register follow-up, 417 individuals developed T2D. IWL during the previous year predicted an increased risk of T2D during the follow-up. The association was indicatively accentuated in men, younger individuals, individuals with a lower educational level, individuals with some unfavorable lifestyle habits (low quality diet, low physical activity, and short habitual sleep), and in individuals with low energy intake.

The results suggest that at the population level, self-report dieting may be associated with greater subsequent weight gain and an increased risk of developing T2D in the long term. These findings may derive from dieting-activated biological mechanisms (e.g. lowered resting energy expenditure and increased appetite) that promote weight gain. Failed dieting may lead to repeated weight loss attempts and thus, to weight cycling. Alternatively, it is possible that dieters differ from non-dieters and are initially at an elevated risk of weight gain and of developing T2D due to other factors. The results that the risk of T2D is tentatively pronounced among those with IWL and some unfavorable lifestyle habits suggest that poorly conducted dieting in conjunction with unhealthy lifestyle habits in particular may be associated with disadvantageous consequences.

In spite of these results, individuals with obesity and medical needs for weight loss should not avoid dieting. Dieting, however, should be conducted properly with evidence-based strategies and an emphasis on weight maintenance after weight loss. Among individuals with normal weight, on the other hand, unnecessary dieting attempts should be avoided and instead, focus should be targeted to learning healthy lifestyle habits in order to maintain normal weight and to prevent weight gain in the future.

Keywords: Dieting; Dieting attempts; Intentional weight loss; Changes in weight; Body mass index; Waist circumference; Obesity; Type 2 diabetes; Quality of diet; Population study

# TIIVISTELMÄ

Lihavuus on merkittävä maailmanlaajuinen kansanterveysongelma. Lihavuus lisää useiden kroonisten kansantautien ja kuolleisuuden riskiä, heikentää toimintakykyä ja elämänlaatua, kuormittaa terveydenhuoltojärjestelmää ja aiheuttaa kuluja yhteiskunnalle. Lihavuusepidemian myötä myös laihduttaminen on yleistynyt, ja onnistuneen laihtumisen onkin todettu olevan yhteydessä useiden kansantautien pienentyneeseen riskiin. Laihduttaminen ja laihduttamisen jälkeinen painonhallinta on suurimmalle osalle kuitenkin haastavaa - usein miltei mahdotonta. Havainnoivissa pitkittäistutkimuksissa on osoitettu, että laihduttamisyrietykset ja tarkoituksellinen laihtuminen ovat yhteydessä myöhempään lihomiseen pitkällä aikavälillä. Laihduttaminen ei rajoitu pelkästään ylipainoisiin ja lihaviin, vaan se on yleistä myös normaalipainoisilla. Vaikka laihduttaminen on yleistä, sen yhteyksiä eri tekijöihin on tutkittu väestötasolla melko vähän.

Tämän väitöskirjan tavoitteena oli tutkia, mitkä tekijät (sosiodemografiset, elintapa-, metaboliset, somaattiseen terveyteen ja mielenterveyteen liittyvät tekijät) ovat yhteydessä itseraportoituun laihduttamiseen. Tavoitteena oli myös tutkia, eroaako ruokavalion terveellisyyden ja lihavuuden välinen yhteys niiden välillä, jotka olivat laihduttaneet ja eivät olleet laihduttaneet edellisen vuoden aikana. Lisäksi tutkittiin, ennustaako laihduttaminen painoindeksin (BMI) ja vyötärönympäryksen muutoksia ja tyypin 2 diabeteksen (T2D) ilmaantuvuutta pitkällä aikavälillä. Tutkimuksessa selvitettiin myös, onko taustatekijöillä (sosiodemografiset, elintapa-, metaboliset, somaattiseen terveyteen ja mielenterveyteen liittyvät tekijät) vaikutusta laihduttamisen ja lihavuusmittareiden muutosten tai T2D:n ilmaantuvuuden välisiin yhteyksiin.

Tutkimus perustui kansallisesti edustavaan Terveys 2000 -tutkimukseen ja sen seurantatutkimukseen, Terveys 2011 -tutkimukseen, joiden tavoitteina oli kerätä ajankohtaista tietoa suomalaisten terveydestä, tärkeimmistä kansansairauksista ja niihin yhteydessä olevista tekijöistä. Tässä tutkimuksessa käytettiin Terveys 2000 -tutkimuksessa 30 vuotta täyttäneiden otosta (n=8028), josta 6771 henkilöä osallistui terveystarkastukseen vuonna 2000. Terveys 2000 -tutkimukseen kutsutuista henkilöistä 6319 kutsuttiin myös seurantatutkimukseen vuonna 2011. Tästä otoksesta terveystarkastukseen osallistui 4006 henkilöä vuonna 2011. Tutkimukset sisälsivät terveystarkastuksen (sisältäen mittauksia ja verinäytteiden oton), haastatteluja ja kyselylomakkeita. Tieto laihduttamisyrietyksistä (yrittänyt laihduttaa), tarkoituksellisesta laihtumisesta (onnistunut laihduttamisessa) ja painonmuutoksista edellisen vuoden aikana kerättiin kyselylomakkeen avulla lähtötilanteessa. Validoidun frekvenssityyppisen ruoankäyttökyselyn (FFQ) avulla arvioitiin tutkittavien

tavanomaista ruoankäyttöä ja ravintoaineiden saantia. Näihin tietoihin pohjautuen muodostettiin ruokavalion terveellisyyttä kuvaava 'terveellisen syömisen indeksi' (modified Alternate Healthy Eating Index, mAHEI-indeksi). Tutkittavien pituus, paino ja vyötärönympäryys mitattiin molemmilla tutkimuskerroilla. T2D:een sairastuneet 15 vuoden seurannan aikana tunnistettiin käyttäen kansallisia terveyttä koskevia rekistereitä. Tilastolliset analyysit perustuivat lineaarisen ja logistiseen regressiomalliin sekä Coxin malliin.

Lähtötilanteessa kaikkiaan 39 % naisista ja 24 % miehistä oli yrittänyt laihduttaa ja 15 % naisista ja 10 % miehistä oli laihtunut tarkoituksellisesti edellisen vuoden aikana. Laihduttajat (BMI 29.1 [SD 4.97] kg/m<sup>2</sup>) olivat lihavampia kuin ei-laihduttajat (BMI 25.7 [SD 4.17] kg/m<sup>2</sup>). Laihduttamisyrietykset ja tarkoituksellinen laihtuminen olivat yhteydessä useisiin sosiodemografisiin, elintapa-, terveyteen liittyviin ja psykologisiin tekijöihin. Suurin osa yhteyksistä oli samankaltainen miehillä ja naisilla. Miehillä havaittiin kuitenkin naisia voimakkaampi suora yhteys laihduttamisen ja BMI:n välillä. Lisäksi laihduttaminen oli suoraan yhteydessä diabeteksen ja lonkan tai polven nivelrikon sairastavuuteen sekä lyhyeen unenpituuteen vain miehillä. Laihduttamisyrietykset vaikuttivat ruokavalion terveellisyyden ja lihavuuden väliseen yhteyteen; ei-laihduttajilla yhteyttä ei ilmennyt, kun taas laihduttajilla terveellisempi ruokavalio oli yhteydessä suurempaan todennäköisyyteen olla ylipainoinen tai lihava.

Laihduttamisyrietykset, laihtuminen ja painon vaihtelu edellisen vuoden aikana olivat yhteydessä BMI:n ja vyötärönympäryksen kasvuun 11 vuoden seurannan aikana. Kasvu oli suurempaa alun perin normaalipainoisilla laihduttajilla verrattuna normaalipainoisiin ei-laihduttajiin. Sen sijaan lihaviin kohdalla samanlaista eroa ei havaittu. T2D:een 15 vuoden rekisteriseurannan aikana sairastui 417 henkilöä. Edellisen vuoden aikainen tarkoituksellinen laihtuminen oli yhteydessä suurentuneeseen riskiin sairastua seurannan aikana. Tarkoituksellisen laihtumisen ja T2D:n ilmaantuvuuden välinen yhteys oli suuntaa antavasti korostunut miehillä, nuoremmilla henkilöillä, henkilöillä, joilla oli matalampi koulutus, henkilöillä, joilla oli epäterveellisiä elintapoja (epäterveellinen ruokavalio, vähäinen liikunta-aktiivisuus ja liian lyhyt unenpituus) ja henkilöillä, joiden päivittäinen energiansaanti oli matalaa.

Tulosten perusteella itseraportoitu laihduttaminen voi olla väestötasolla yhteydessä suurempaan riskiin lihoa ja sairastua T2D:een pitkällä aikavälillä. Tuloksia voivat selittää laihduttamisen aktivoimat biologiset mekanismit (mm. madaltunut aineenvaihdunta, näläntunteen lisääntyminen), jotka pyrkivät ehkäisemään laihtumista ja edesauttamaan lihomista. Epäonnistunut laihduttaminen voi johtaa toistuviin laihduttamisyrietyksiin ja painon "jojoiluun". Vaihtoehtoisesti on mahdollista, että laihduttajat eroavat ei-laihduttajista ja ovat alun perin muista tekijöistä johtuen suuremmassa vaarassa lihoa ja sairastua T2D:een. Tarkoituksellisen laihtumisen ja T2D:n ilmaantuvuuden suuntaa antavasti korostunut yhteys henkilöillä, joilla oli



epäterveellisiä elintapoja viittaa siihen, että etenkin kehnosti toteutettu laihduttaminen yhdistettynä kyseisiin elintapoihin voi olla yhteydessä haitallisiin seurauksiin.

Huolimatta tämän tutkimuksen tuloksista, henkilöiden, joille on laihtumisesta terveydellistä hyötyä, ei tule välttää laihduttamista. Laihduttaminen tulee kuitenkin toteuttaa suunnitelmallisesti myönteisiin elintapamuutoksiin perustuen painottaen laihduttamisen jälkeisen painonhallinnan tärkeyttä. Normaalipainoisten henkilöiden tulisi välttää turhia laihduttamisyriä, ja sen sijaan keskittyä terveellisten elintapojen omaksumiseen säilyttääkseen normaalipainonsa ja ehkäistäkseen painon nousu tulevaisuudessa.

Avainsanat: Laihduttaminen; Laihduttamisyriä; Tarkoituksellinen laihtuminen; Painonmuutokset; Painoindeksi; Vyötärönympäry; Lihavuus; Tyypin 2 diabetes; Ruokavalion terveellisyys; Väestötutkimus

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# LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following publications:

- I Sares-Jäske L, Knekt P, Männistö S, Lindfors O, Heliövaara M (2019). Self-Report Dieters: Who Are They? *Nutrients* 2:11(8). pii: E1789. doi: 10.3390/nu11081789.
- II Sares-Jäske L, Knekt P, Lundqvist A, Heliövaara M, Männistö S (2017). Dieting attempts modify the association between quality of diet and obesity. *Nutr Res* 45:63-72. doi: 10.1016/j.nutres.2017.08.001
- III Sares-Jäske L, Knekt P, Männistö S, Lindfors O, Heliövaara M (2019). Self-report dieting and long-term changes in body mass index and waist circumference. *Obes Sci Pract* 26;5(4):291-303. doi: 10.1002/osp4.336
- IV Sares-Jäske L, Knekt P, Eranti A, Kaartinen N, Heliövaara M, Männistö S. Intentional weight loss as a predictor of type 2 diabetes occurrence in a general adult population. (Accepted for publication in *BMJ Open Diabetes Research & Care* 07/2020).

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## ABBREVIATIONS

AHEI	Alternate Healthy Eating Index
AT	adaptive thermogenesis
BDI	Beck Depression Inventory
BMI	body mass index
BSDS	Baltic Sea Diet Score
CI	confidence interval
CHD	coronary heart disease
CVDs	cardiovascular diseases
DASH	the Dietary Approaches to Stop Hypertension Score
DPP	Diabetes Prevention Program
DPS	Diabetes Prevention Study
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders, 4th revised edition
ExEE	exercise energy expenditure
FFM	fat free mass
FFMI	fat free mass index
FFQ	food frequency questionnaire
FM	fat mass
FMI	fat mass index
GWAS	genome-wide association studies
HbA1c	glycated hemoglobin
HDL	high-density lipoprotein
Health 2000	Health 2000 Survey
Health 2011	Health 2011 Survey
HEI	Healthy Eating Index
HFII	Healthy Food Intake Index
HR	hazard ratio
ICD	International Statistical Classification of Diseases and Related Health Problems
IDF	International Diabetes Federation
IWL	intentional weight loss
kcal	kilocalories
Kela	The Social Insurance Institution of Finland
KTL	National Public Health Institute
LDL	low density lipoprotein
mAHEI	modified Alternate Healthy Eating Index
M-CIDI	German Composite International Diagnostic Interview
MetS	metabolic syndrome
mmHg	millimeter of mercury
mmol/l	millimole per liter
N	number of participants

NCDs	noncommunicable diseases
OR	odds ratio
PA	physical activity
REE	resting energy expenditure
SD	standard deviation
SOC	sense of coherence
T2D	type 2 diabetes
TDEE	total daily energy expenditure
TEF	thermic effect of food
THL	Finnish Institute for Health and Welfare
VLCD	very low calorie diet
WC	waist circumference
WHO	World Health Organization
WLW	weight loss without information on intentionality

# 1 INTRODUCTION

The obesity epidemic is increasing worldwide, with more than 39% of adults having overweight (body mass index [BMI]  $\geq 25$  kg/m<sup>2</sup>), and 13% of adults having obesity (BMI  $\geq 30$  kg/m<sup>2</sup>) in 2016 (World Health Organization 2018). According to the results of the FinHealth 2017 Study, almost two in three women and three in four men had at least overweight, and approximately one in four adults had obesity in Finland in 2017 (Koponen *et al.* 2018). Moreover, every other adult had central obesity (waist circumference [WC] in men  $>100$  cm and in women  $>90$  cm (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013)).

Obesity refers to excess fat mass (FM), which is a consequence of a long-term imbalance between energy intake and energy expenditure. Obesity predisposes to several noncommunicable diseases (NCDs) such as type 2 diabetes (T2D). According to the estimations of the World Health Organization (WHO), the prevalence of all types of diabetes combined rose from 4.7% in 1980 to 8.5% in 2014 among adults aged 18 and older (Roglic & World Health Organization 2016). The majority of the individuals affected by diabetes have T2D. The contribution of excess FM to the development of T2D is considerable; it has been estimated that 77% of incident T2D cases could be prevented if everyone had a BMI of less than 25 kg/m<sup>2</sup> (Laaksonen *et al.* 2010). Both obesity and T2D place a substantial burden on not only individual and public health but also on the public economy.

In parallel with ascending obesity and T2D rates, dieting has become increasingly popular. Worldwide, approximately 40% of adults have tried to lose weight during their lives (Santos *et al.* 2017). According to the FinHealth 2017 Study, in Finland in 2017, of women and men aged 25–74, 65% and 43%, respectively had tried to lose weight during their lives, and 35% of women and 22% of men were trying to lose weight at present.

Traditionally, weight loss has been used to lose excess weight. However, regardless of whether individuals have obesity, overweight or normal weight, they are affected by society's demand for leanness. Thus, individuals with normal weight with no medical needs for weight loss also practice dieting. For individuals with obesity, weight loss reduces the risk of many NCDs and alleviates symptoms or the prognosis of many existing diseases (Oster *et al.* 1999, Douketis *et al.* 2005, Eneli *et al.* 2008, LeBlanc *et al.* 2018). Intervention studies, with participants with obesity or other metabolic risk factors, have demonstrated that dietary changes and increased physical activity (PA) can result in weight loss (Franz *et al.* 2007, LeBlanc *et al.* 2018) and in a reduction of T2D incidence (Peirson *et al.* 2014, Haw *et al.* 2017, LeBlanc *et al.* 2018). In the seminal Finnish Diabetes Prevention Study (DPS), the individuals in the intervention group (4.2 [SD 5.1] kg) lost significantly more weight than the individuals in the control group (0.8 [SD



3.7] kg) during the first study year (Tuomilehto *et al.* 2001). Most of these lifestyle intervention studies, however, have had no or relatively short follow-ups after the actual intervention periods. A few studies have had longer follow-ups: for example, the DPS with a 13-year follow-up (Lindström *et al.* 2013) and the Diabetes Prevention Program (DPP) with a 10-year follow-up (Diabetes Prevention Program Research Group *et al.* 2009). Moreover, the support received in such interventions is not comparable to the conditions of average dieters in general populations. Accordingly, in most longitudinal observational studies, self-report dieting attempts and intentional weight loss (IWL) have been associated with subsequent weight gain (French *et al.* 1994b, Coakley *et al.* 1998, Korkeila *et al.* 1999, Stice *et al.* 1999, Kroke *et al.* 2002, Lowe *et al.* 2006, Viner & Cole 2006, Field *et al.* 2007, Savage *et al.* 2009, Pietiläinen *et al.* 2012, Neumark-Sztainer *et al.* 2012, van Strien *et al.* 2014, Siahpush *et al.* 2015, Brown *et al.* 2016, Kärkkäinen *et al.* 2018), especially among individuals with initial normal weight (Korkeila *et al.* 1999, Pietiläinen *et al.* 2012, Dulloo *et al.* 2015). The biological mechanisms activated by weight loss and energy deficit favor weight regain (Ochner *et al.* 2013, Sumithran & Proietto 2013), and consequently, weight maintenance seems to be the most demanding phase of weight loss. As weight maintenance commonly fails, and the dieter regains the weight (Sarlio-Lähteenkorva *et al.* 2000, Wing & Phelan 2005), it often leads to another attempt, and ultimately to weight cycling.

Conversely, most observational studies evaluating the associations between weight loss (without information on intentionality (WLW)) and the incidence of T2D have suggested that WLW predicts a reduced risk of T2D (Chan *et al.* 1994, Colditz *et al.* 1995, Resnick *et al.* 2000, Koh-Banerjee *et al.* 2004, Wannamethee *et al.* 2005, Kim *et al.* 2018, Stokes *et al.* 2018). In these studies, however, the actual weight loss, usually measured between time points over a longer time period, may differ from self-report dieting in terms of long-term success. Thus, self-report IWL may actually more accurately represent the dieting that occurs in general populations; people may lose weight, but usually they regain it in a relatively short time. Nevertheless, the few studies thus far conducted that have used self-report dieting (Monterrosa *et al.* 1995) or IWL (Williamson *et al.* 1995, Williamson *et al.* 1999, Will *et al.* 2002) as an exposure and T2D as an outcome have yielded mainly similar results to those of WLW studies, although their populations have been limited only to individuals with overweight or obesity. The evidence on the associations between self-report IWL and T2D incidence in the general population thus remains scarce and inadequate. Hence, it is important to clarify how self-report dieting associates with outcomes that are significant to public health.

Dieting is associated with several sociodemographic, lifestyle, and health-related factors, but the whole picture of the determinants of dieting is unclear. The factors associated with dieting may confound or modify the associations between dieting and subsequent weight change and the

incidence of NCDs. Thus, comprehensive scrutiny of such factors is important. Moreover, dieting itself may confound or modify the associations between other factors, for instance diet and obesity, and thus it is essential to reveal these possibilities in order to be able to study these associations in an unbiased way.

Due to the commonness of dieting at the population level, further research on its associations is needed. Dieting behavior can be a countermeasure to weight gain or a proxy of susceptibility to gain weight. As on the one hand dieting attempts have been shown to predict later weight gain, but on the other hand, weight loss has been shown to predict a reduced risk of T2D occurrence, the whole picture obviously has pieces missing. It seems that whether dieting has positive or negative consequences depends on the population in question and on the nature of the dieting or weight loss variables, but most importantly, on the long-term success of the weight loss.

As successful weight loss and weight maintenance after weight loss seem to be overpowering for the majority of individuals, the importance of preventing weight gain cannot be emphasized enough. Among individuals with normal weight, initially refraining from unnecessary dieting while learning a healthy lifestyle to support maintain normal weight could play a role in such prevention. In contrast, among individuals with a medical need for weight loss, dieting should not be avoided, but carefully conducted using adequate strategies and long-term support and follow-up.

The substudies of the present thesis examined dieting attempts and IWL at the population level in a general adult population, looked into their associations with several potential determinants, examined their modifying role in the association between quality of diet and obesity, determined their associations with subsequent changes in BMI and WC, and studied whether they predicted long-term incidence of T2D.

## 2 REVIEW OF THE LITERATURE

### 2.1 OBESITY

#### 2.1.1 DEFINITION

Obesity denotes having excess FM in the body, in terms of optimal health. No universal cut-off values can be set to define excess fat. Instead, the risk of NCDs increases gradually in parallel with the amount of excess FM. Moreover, the optimal level of FM varies inter-individually. However, various measures and their cut-off values have been developed and further studied to enable the categorization of individuals in clinical settings and epidemiological studies.

The most commonly used tool for assessing obesity is BMI ( $\text{kg}/\text{m}^2$ ). The basis for BMI was first developed by Belgian scientist Adolphe Quetelet between 1830 and 1850. The index was further named BMI by Keys and colleagues (1972). Later, higher BMI has been shown to associate with an increased risk of NCDs by, for example, the seminal Framingham Heart Study (Wilson *et al.* 2002). A BMI value of  $25 \text{ kg}/\text{m}^2$  or more defines overweight, a value of  $30 \text{ kg}/\text{m}^2$  or more defines obesity, whereas a value of less than  $18.5 \text{ kg}/\text{m}^2$  defines underweight, leaving a normal range of  $18.5\text{--}24.99 \text{ kg}/\text{m}^2$  (World Health Organization 2000). BMI, however, has been criticized, as it does not take into account the distribution and amount of FM and fat free mass (FFM) in the body but serves as a surrogate measure of body fatness (Prentice & Jebb 2001). Furthermore, BMI fails to recognize the localization of fat deposits (Chooi *et al.* 2019). Regardless, BMI is an easy, cheap, and well-known tool for categorizing people into groups according to weight status and thus, has preserved its position in general use.

WC measurement (cm) is used to assess abdominal obesity, which is associated with poorer metabolic health (Smith 2015). Various cut-off values exist to define abdominal obesity. WHO has established values of  $\text{WC} \geq 88$  cm for women and  $\text{WC} \geq 102$  cm for men (World Health Organization 2000), the International Diabetes Federation (IDF) uses values of  $\text{WC} \geq 80$  cm for women and  $\text{WC} \geq 94$  cm for men (Alberti *et al.* 2009), while in clinical practice in Finland, values of  $\text{WC} \geq 90$  cm for women and  $\text{WC} \geq 100$  cm for men are being used (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013).

Other measures to evaluate obesity and the distribution of FM are, for instance, waist to hip ratio ( $\text{WC cm} / \text{hip circumference cm}$ ), body fat percentage (%), fat mass index (FMI) ( $\text{fat kg}/\text{m}^2$ ), and sagittal abdominal diameter (cm). Moreover, fat free mass index (FFMI) ( $\text{fat free mass kg}/\text{m}^2$ ) is used to assess the proportion of muscle mass and also to recognize sarcopenic obesity (Schutz *et al.* 2002).

### 2.1.2 PREVALENCE AND TRENDS

Obesity rates have nearly tripled in the last 45 years worldwide (World Health Organization 2018). WHO estimated that globally in 2016, more than 39% of adults had overweight, and 13% of adults had obesity (World Health Organization 2018). In Finland, according to the results of the National FINRISK Study, the obesity rates of working-age Finnish adults have increased over the decades from the 1970s (Borodulin *et al.* 2015). In accordance, the results of the Mini-Finland Health Survey, conducted between 1978 and 1980, and the Health 2000 Survey, conducted between 2000 and 2001, show an increase in obesity rates over 20 years among adults aged 30 and over (Lahti-Koski *et al.* 2010). The increase, however, seemed to level off during 2002–2012, with approximately two in three men and every other woman having overweight and one in five adults having obesity during that time (Männistö *et al.* 2015). The obesity rates also appeared to stay relatively stable between 2000 and 2011 in the National Health 2000/2011 Survey in adults aged 30 and over (Table 1) (Aromaa & Koskinen 2004, Koskinen *et al.* 2012). According to the National FinHealth 2017 Study, however, the rates have continued to rise: Of adults aged 30 and over, 71.9% of men and 63.2% of women had overweight and 26.1% of men and 27.5% of women had obesity (Table 1) (Koponen *et al.* 2018).

**Table 1** Trends of obesity in Finland in 2000–2017 in men and women aged  $\geq 30$  (Aromaa & Koskinen 2004, Koskinen *et al.* 2012, Koponen *et al.* 2018).

Study	Men			Women		
	Mean BMI (kg/m <sup>2</sup> )	Obesity %	Abdominal obesity %	Mean BMI (kg/m <sup>2</sup> )	Obesity %	Abdominal obesity %
Health 2000	27.3	21.2	38.8	27.0	23.5	42.1
Health 2011	27.3	22.9	39.3	27.1	26.3	39.4
FinHealth 2017	27.7	26.1	45.6	27.5	27.5	46.0

Abbreviations: BMI, body mass index; WC, waist circumference.

Obesity: BMI  $\geq 30$  kg/m<sup>2</sup>; Abdominal obesity: WC  $\geq 100$  cm for men or  $\geq 90$  cm for women.

Results are age-adjusted.

On average, during 1972–2007, Finnish adults annually gained 300–500 g (Pajunen *et al.* 2012). The amount of annual gain remained stable in men, but showed a slight increase in women after the 1990s.

### 2.1.3 RISK FACTORS OF OBESITY

A long-term positive imbalance between energy intake and energy expenditure leads to the accumulation of FM and eventually to obesity. Generally, the increased standard of living and the obesogenic environment, the availability of energy-dense foods and sugar-sweetened beverages with big portion sizes, as well as sedentary lifestyles with a decreased need for physical work, lower leisure-time PA, and increased sitting time seem to play a major role (Swinburn *et al.* 2011, Fogelholm *et al.* 2012, Ng & Popkin 2012). While the high intake of meat, refined grains, and sugar-rich foods and drinks has been associated with subsequent weight gain, it has been indicated that a high intake of dietary fiber, nuts, and dairy products predicts less weight gain (Fogelholm *et al.* 2012). Meal frequency and meal timing are also associated with the risk of obesity; irregular eating patterns and skipping breakfast are unfavorable in terms of maintaining normal weight in the long term (St-Onge *et al.* 2017, Paixão *et al.* 2020). Moreover, eating behaviors that have loss of control over eating in common (e.g. emotional eating and disinhibition) have been linked to obesity (Vainik *et al.* 2019). Lack of PA with low daily energy expenditure contributes to the risk of obesity. Indeed, a high volume of PA has been shown to associate with less weight gain (Fogelholm & Kukkonen-Harjula 2000).

At the individual level, in addition to everyday lifestyle choices, genes play a role in the development of obesity (Elks *et al.* 2012, Li *et al.* 2014, Goodarzi 2018). In genome-wide association studies (GWAS), over 300 single-nucleotide polymorphisms (SNPs) have been identified to associate with obesity measures (e.g. BMI and WC) (Goodarzi 2018). One of the most studied obesity-related genes is FTO, whose variants have been shown to increase the risk of obesity by affecting food intake and food preferences (Loos & Yeo 2014). The effect of FTO on weight, however, is small; each additional risk allele is associated with only a 0.39 kg/m<sup>2</sup> higher BMI. Further, it has been estimated that 40–70% of inter-individual BMI variation can be explained by genetic factors. Genes may predispose a person to a greater risk of gaining weight. However, having unfavorable genes in this respect does not mean a definite judgement to become affected by obesity, but genetic susceptibility can be attenuated by favorable lifestyle choices such as participating in regular PA (Mustelin *et al.* 2009, Li *et al.* 2010, Lin *et al.* 2019) or making healthier dietary choices (Qi *et al.* 2012, Qi *et al.* 2014). Moreover, results based on DPS data have indicated that even though having an obesity-predisposing genotype in the FTO gene is associated with higher BMI, this does not hinder weight loss in lifestyle intervention (Lappalainen *et al.* 2009).

In addition to lifestyle and genes, some non-traditional risk factors of obesity have also been identified. These include, for example, short sleep duration (Itani *et al.* 2017), psychosocial stress (Wardle *et al.* 2011) and changes in gut microbiome (Torres-Fuentes *et al.* 2017).

#### **2.1.4 CONSEQUENCES OF OBESITY**

Obesity is a risk factor for several NCDs and conditions such as T2D (Singh *et al.* 2013), cardiovascular diseases (CVDs) (Singh *et al.* 2013), several cancers (Lauby-Secretan *et al.* 2016), musculoskeletal disorders (Anandacoomarasamy *et al.* 2008), and mental health problems – potentially partly mediated by experiences of obesity stigma and perceived discrimination (Williams *et al.* 2015). Higher BMI also predisposes one to an increased risk of multimorbidity (Wikström *et al.* 2015, Kivimäki *et al.* 2017). Furthermore, obesity impairs health-related quality of life (Fontaine & Barofsky 2001) and is associated with increased sick-leaves (Laaksonen *et al.* 2007, Neovius *et al.* 2009, Virtanen *et al.* 2018). In addition to the individual-level burden of such consequences, it also places a substantial burden on economies (von Lengerke & Krauth 2011, Kim & Basu 2016).

Besides increasing the risk of other diseases, obesity has been recognized as a disease itself (Kyle *et al.* 2016). Even in the absence of actual distinct diseases or conditions, obesity adversely affects multiple physical functions of the body. For instance, abnormal glucose metabolism (Kahn *et al.* 2006), low-grade chronic inflammation (Gregor & Hotamisligil 2011), and oxidative stress (Manna & Jain 2015) represent such effects. Abdominal (visceral) and ectopic (fat deposits inside skeletal muscles, heart, liver, pancreas, etc.) fat have been shown to be especially malign in regard to the development of metabolic illness (Tchernof & Despres 2013). Despite the existence of metabolically healthy individuals with obesity, and findings supporting the “obesity paradox” (i.e. patients with CVDs and overweight or obesity having a better prognosis) (Antonopoulos & Tousoulis 2017), findings regarding the associations between BMI, metabolic health status and subsequent long-term risk of T2D (Bell *et al.* 2014), and cardiovascular events and mortality (Kramer *et al.* 2013) suggest that individuals with obesity are at a greater risk of occurrence of such events, regardless of their initial metabolic health status.

## **2.2 DIETING ATTEMPTS AND INTENTIONAL WEIGHT LOSS**

Dieting is usually defined as an intentional attempt to lose weight. In some contexts, dieting refers to following a certain dietary plan without intention to lose weight. In this thesis, dieting is used to denote efforts to lose weight. Dieting as a concept, however, does not include information on the success of weight loss but only the intention to do so. Hence, the term “dieting attempts” is used to refer to dieting without information on such success. In addition, the term “intentional weight loss” (IWL) is used to refer to dieting with known success. The term “dieting” is used more loosely to describe any intentional dieting behavior.

Weight loss can be achieved by creating a negative energy balance i.e. expending more energy than is consumed. Generally, the recommendable weight loss rate is 0.5–1.0 kg/week, which can usually be achieved with a 500–1000 kcal energy deficit/day (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013). For individuals with obesity, the aim of weight loss is not necessarily to reach normal weight if this seems unrealistic, but to achieve a weight loss of 5% or more, which has been indicated as sufficient for the prevention and improvement of several conditions (World Health Organization 2000, Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013, Ryan & Yockey 2017).

Weight loss can be conducted in countless ways. For individuals with severe obesity, very low calorie diets (VLCD) can be of assistance at the beginning (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013). However, in order to achieve lasting weight loss results, the regimen used should be such that can also be applied after the actual weight loss phase. Taking personal preferences into account assists in adhering to the regimen and keeping up motivation. The new Current Care guidelines for obesity emphasize individualized guidance in weight loss.

Generally, it is essential to decrease energy intake in parallel with making healthier dietary choices. Such changes in lifestyle can be made by little steps and using, for instance, the food plate model and the food pyramid for assistance (National Nutrition Council 2014). Nutrient-dense foods should be favored, while energy-dense foods should be consumed in moderation. Regular meal frequency is also important, and ensuring adequate protein intake is essential in order to prevent excessive loss of muscle tissue. In the weight maintenance phase, energy consumption should correspond to the new energy expenditure, which is usually lower than the dieter's initial consumption. Adherence to Finnish dietary recommendations has been suggested to help in maintaining normal WC and body fat percentage (Kanerva *et al.* 2013a), although personal preferences should also be taken into account in order to be able to maintain the new healthy lifestyle.

PA is also an essential part of weight loss (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013, Bray *et al.* 2016). Even though the contribution of PA in creating a negative energy balance is usually more modest than that of decreasing energy intake, PA plays an important role in adopting a healthy lifestyle and in preserving muscle tissue (Cava *et al.* 2017, Dulloo 2017). In weight loss, PA worth 300 kcal/day (corresponding to 45–60 min of moderately strenuous endurance training) has been recommended as an adequate amount (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013). While endurance training helps increase energy expenditure and improve cardiorespiratory fitness, strength training helps maintain FFM, which is essential in avoiding lowering resting energy expenditure (REE)

(Dulloo 2017). When moving from the weight loss phase to the weight maintenance phase, the importance of sufficient PA becomes pronounced (Fogelholm & Kukkonen-Harjula 2000, Goldberg & King 2007, Bray *et al.* 2016, Swift *et al.* 2018). During weight maintenance, PA worth 2500–2800 kcal/week (corresponding to 60–90 min of moderately strenuous endurance training/day) has been estimated as an adequate amount (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013). In an American study of female adolescents and young adults, exercising was the sole strategy for succeeding in not gaining weight during a four-year follow-up (Field *et al.* 2010). The authors further concluded that combining exercise with dietary approaches is necessary in order to reduce long-term weight gain. A recent systematic review of studies using weight control registries also disclosed that increased PA is the most consistent predictor of weight maintenance (Paixão *et al.* 2020).

In addition to assisting in weight loss and weight maintenance, PA produces independent favorable health effects (Fogelholm *et al.* 2011). The UKK Institute's (Centre for Health Promotion Research) new recommendations for health-enhancing PA for adults suggest at least 2 h 30 min moderate-intensity aerobic PA or 1 h 15 min vigorous-intensity aerobic PA a week, strength training and balance training at least twice a week, and as much light PA as possible (UKK Institute 2019). Moreover, for the first time, these recommendations now also include taking breaks from sedentary activities and sleeping adequately.

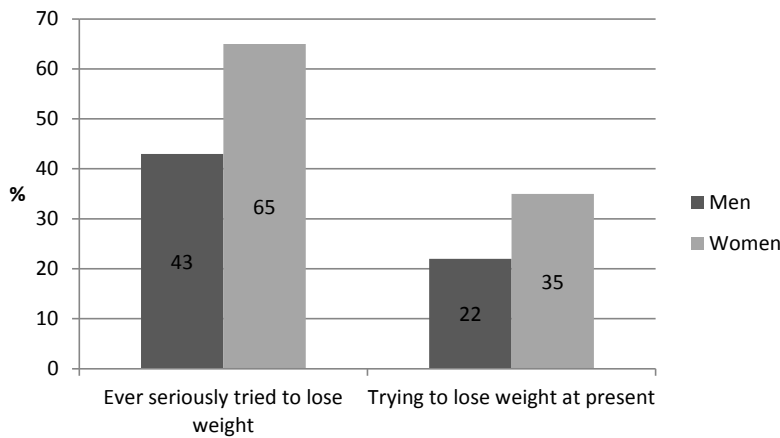
Poor lifestyle habits may reduce the probability of succeeding in weight loss and, especially, in maintaining lost weight. A review of the determinants of weight maintenance and weight regain concluded that healthy lifestyle habits were associated with greater success in weight maintenance (Elfhag & Rossner 2005). A more recent review affirmed these results with findings that a reduction in energy intake, an increase in energy expenditure and monitoring of this balance were associated with success in weight maintenance (Varkevisser *et al.* 2019). It has also been suggested that in “a high energy flux” state, when both energy intake and energy expenditure are balanced but higher, weight maintenance is easier than in “a low energy flux” state, when both energy intake and energy expenditure are lower, and appetite is usually more pronounced (Melby *et al.* 2017).

In addition, various psychological traits have been found to associate with successful weight loss and weight maintenance. An increase in flexible control over eating and a decrease in rigid control over eating seem to be associated with better success in weight maintenance (Sairanen *et al.* 2014). In clinical settings, behavioral and lifestyle counselling have also been shown to help achieve sustained results (Bray *et al.* 2016). In cases of severe obesity, weight loss medication and bariatric surgery are also options for losing weight. This thesis, however, focuses on behavior-based weight loss, and excludes scrutiny of weight loss conducted through such methods.



### 2.2.1 PREVALENCE AND TRENDS

Of the general adult populations worldwide, approximately 40% have tried to lose weight during their lifetimes (Santos *et al.* 2017). In Finland in 2002, roughly 60% of women and 35% of men aged 25–74 had tried to lose weight during their lives (Laatikainen 2003). The prevalence of dieting seems to have increased over recent decades, and according to the FinHealth 2017 Study in Finland in 2017, the percentages of women and men of the same age reporting weight loss attempts during their lives were 65% and 43%, respectively (Figure 1). Further, 35% of women and 22% of men reported that they were trying to lose weight at present.



**Figure 1** Prevalence (%) of weight loss attempts in National FinHealth 2017 Study.

### 2.2.2 REASONS FOR DIETING

Traditionally, dieting is used by individuals with obesity to lose excess weight. This demarcation, however, is long outdated, and dieting is no longer limited to those with obesity, but individuals with normal weight or underweight also report dieting attempts (Korkeila *et al.* 1999, Bendixen *et al.* 2002, Keski-Rahkonen *et al.* 2005, Montani *et al.* 2015). For individuals with obesity, dieting may refer to weight loss of dozens of kilograms, while for individuals with normal weight, dieting may denote efforts to lose “only the two kilograms gained during the summer holidays”, in order to stay at a normal weight. Hence, the term dieting, even when focusing on weight loss dieting, may cover very heterogeneous types.

For individuals with obesity, weight loss can decrease the risk of several NCDs or alleviate the symptoms or prognosis of an existing disease (Oster *et al.* 1999, Douketis *et al.* 2005, Eneli *et al.* 2008, LeBlanc *et al.* 2018).

However, in addition to health-related motives, the reasons for dieting may include appearance and sports performance-related factors. Moreover, sociocultural pressures to be lean impact individuals on the whole BMI scale. Eating disorders and distorted body image, as extreme reasons, also drive individuals to attempt to lose weight.

### **2.2.3 FACTORS RELATED TO DIETING**

Dieting has been shown to be more common among women (Bendixen *et al.* 2002, Keski-Rahkonen *et al.* 2005, Santos *et al.* 2017, Barebring *et al.* 2018) and younger age groups (Serdula *et al.* 1999, Hjartaker *et al.* 2001, Bendixen *et al.* 2002, Kruger *et al.* 2004, Weiss *et al.* 2006, Yaemsiri *et al.* 2011), although according to a recent large study with a 15-year follow-up (Haynos *et al.* 2018), dieting behavior seems to persist in adulthood and increase along with aging among men in particular. Findings also suggest that dieting is more common among individuals with a higher educational level (Stephenson *et al.* 1987, Serdula *et al.* 1999, Hjartaker *et al.* 2001, Kruger *et al.* 2004, Bish *et al.* 2005, Weiss *et al.* 2006), individuals with a higher income (Stephenson *et al.* 1987, Hjartaker *et al.* 2001, Machado *et al.* 2012, Barebring *et al.* 2018), physically more active individuals (French *et al.* 1994a, Green *et al.* 1997, Kabeer *et al.* 2001, Jeffery *et al.* 2013, Barebring *et al.* 2018), individuals with a higher BMI (Jeffery *et al.* 1991, Bendixen *et al.* 2002, Keski-Rahkonen *et al.* 2005, Santos *et al.* 2017, Barebring *et al.* 2018), and individuals with healthier dietary habits (Jeffery *et al.* 1991, French & Jeffery 1997, Neumark-Sztainer *et al.* 2000, Kant 2002, Andreyeva *et al.* 2010, Jeffery *et al.* 2013, Wolfson & Bleich 2015). However, some unhealthy dietary habits have also been found to relate to dieting behavior: Overeating, snacking and eating in response to visual and emotional cues have been shown to be more common among individuals with IWL (Keski-Rahkonen *et al.* 2005). Further, it has been suggested that dieting (in addition to e.g. obesity) is related to misreporting of dietary intake (Livingstone & Black 2003, Maurer *et al.* 2006, Poslusna *et al.* 2009, Castro-Quezada *et al.* 2015). Dieters seem to underestimate their energy intake and consumption of foods considered unhealthy, and conversely overestimate their consumption of foods considered healthy. Similarly, study findings imply that dieting behavior is related to underreporting of weight (Polivy *et al.* 2014).

#### ***Restrained eating***

Restrained eating is often considered one form of dieting. It can be measured by different dietary restraint scales (Williamson *et al.* 2007). Not all restrained eaters, however, try to lose weight. Restrained eating refers more to attempts to control eating and avoid overeating and thus, gaining weight (Lowe & Levine 2005). However, restrained eating is more common among

individuals with IWL than individuals without IWL (Keski-Rahkonen *et al.* 2005). The literature on the associations between restrained eating and obesity has been conflicting with most studies lacking a chronological association between restrained eating and subsequent changes in weight (Lowe *et al.* 2013, Konttinen *et al.* 2018). A large Finnish study, however, indicated that a higher BMI was associated with an increase in restrained eating during a seven-year follow-up (Konttinen *et al.* 2018). As restrained eating differs from dieting attempts and IWL, it has been excluded from further scrutiny in this thesis focusing on weight loss dieting.

#### **2.2.4 WEIGHT LOSS INTERVENTION STUDIES**

Several intervention studies have demonstrated that in individuals with obesity, restricting energy intake and/or increasing energy expenditure results in weight loss (Franz *et al.* 2007, LeBlanc *et al.* 2018). Even though the contents and procedures of the interventions have varied considerably, the main goal of the interventions has been weight loss through changes in diet and PA. In a pooled analysis by LeBlanc *et al.* (2018), the mean difference in weight loss during 12–18 months between intervention groups and control groups was -2.39 (95% confidence interval (CI) -2.86 to -1.93) kg. During the first year of the Finnish DPS, the intervention group and the control group lost 4.2 (SD 5.1) kg and 0.8 (SD 3.7) kg, respectively ( $P < 0.001$ ) (Tuomilehto *et al.* 2001).

#### **2.2.5 EPIDEMIOLOGICAL STUDIES ON DIETING ATTEMPTS AND SUBSEQUENT WEIGHT GAIN**

Even though numerous weight loss intervention studies with individuals with obesity have shown weight loss to be achievable by restricting energy intake and/or increasing energy expenditure (Franz *et al.* 2007, LeBlanc *et al.* 2018), these results cannot be generalized to concern individuals in general populations with a wider BMI range and mostly attempting to diet by themselves. Hence, this chapter focuses on epidemiological follow-up studies that examine the associations between self-report dieting and IWL and subsequent weight change. The criteria for the studies to be included in the review of this literature are presented in Table 2.

**Table 2** Criteria for inclusion of follow-up studies in literature review.

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1.	The outcome is a change in an obesity measure (e.g. BMI, weight, WC) or a risk of gaining or losing weight during follow-up.
2.	The exposure is an attempt to lose weight or IWL before baseline, at baseline or during follow-up.
3.	Participants with dieting attempts are compared to participants without dieting attempts, and participants with IWL are compared to participants without intention to lose weight and maintaining their weight.
4.	Participants are adults ( $\geq 18$ years) at baseline or proceed into adulthood during follow-up.

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Abbreviations: BMI, body mass index; IWL, intentional weight loss; WC, waist circumference.

The association between self-report dieting attempts and IWL and subsequent changes in obesity measures has been studied in some original articles (Table 3). Most of the studies have been conducted in the USA, but a few also in Europe or Australia. The study baseline in most studies was during the 1970s to the 1990s, with a few exceptions in which data collection was initiated in the 21st century (van Strien *et al.* 2014, Siahpush *et al.* 2015, Kärkkäinen *et al.* 2018). The number of participants in the studies ranged from under 100 to almost 20 000, and in most studies they comprised both women and men. A little more than half of the studies presented the two sexes' results separately.

Studies examining the association between dieting and subsequent changes in weight in children or teenagers were excluded from this literature review, unless the follow-up time proceeded into adulthood. Thus, the age ranges at the study baselines varied from teenagers to middle-aged and older adults. The dieting measures used included different variables concerning current or ever dieting attempts or IWL, and life-time frequency of dieting attempts or IWL. The considered outcome measures included changes in weight or BMI and the risks of gaining, losing or maintaining weight. Control for confounding factors varied from not reporting whether the results were adjusted for to comprehensive adjustments.

Most of the findings indicated that dieting predicts later weight gain (French *et al.* 1994b, Coakley *et al.* 1998, Korkeila *et al.* 1999, Stice *et al.* 1999, Kroke *et al.* 2002, Lowe *et al.* 2006, Viner & Cole 2006, Field *et al.* 2007, Savage *et al.* 2009, Pietiläinen *et al.* 2012, Neumark-Sztainer *et al.* 2012, van Strien *et al.* 2014, Siahpush *et al.* 2015, Brown *et al.* 2016, Kärkkäinen *et al.* 2018). A large American study with a sample of 10 554 men and women aged 45–64 and conducted using data from the Atherosclerosis Risk in Communities Study found that during a six-year follow-up, the odds of losing any weight were smaller among men and women attempting to diet than among corresponding non-dieters (Juhaeri *et al.* 2001). In the Finnish Twin Cohort Study, women aged 30–54 and men aged 18–29 and currently trying to lose weight were at an elevated risk of major weight gain (>10 kg) during a six-year follow-up compared to non-dieters, whereas men aged 18–

29 and men aged 30–54 were at a corresponding elevated risk during a 15-year follow-up (Korkeila *et al.* 1999). In a study conducted using data from the Health Professionals Follow-up Study (Coakley *et al.* 1998), a greater number of IWLs of  $\geq 2.3$  kg during the previous 16 years predicted a greater increase in weight during a four-year follow-up. In concordance, a large study with 18 001 participants and data from The European Prospective Investigation into Cancer and Nutrition (EPIC) Potsdam study indicated that an IWL of 5 kg or more during the two preceding years predicted an increased risk of weight gain during a two-year follow-up after comprehensive control for confounding factors in both men and women (Kroke *et al.* 2002). In an American study utilizing data from the Healthy Worker Project, previously participating in a formal weight loss program was associated with weight gain during follow-up (French *et al.* 1994b). Moreover, current dieting at baseline predicted weight gain in women. In contrast, ever dieting to lose weight was unrelated to later weight gain. In a British study by Viner and colleagues (2006), however, ever having tried to lose weight was associated with weight gain.

Five studies looking into the risk of gaining weight when proceeding into adulthood consistently suggested that dieting attempts (Stice *et al.* 1999, Viner & Cole 2006, Field *et al.* 2007, Neumark-Sztainer *et al.* 2012) and IWL (Pietiläinen *et al.* 2012) during adolescence or early adulthood were associated with an increased risk of later weight gain. One study with a relatively short follow-up of seven months, however, failed to find such an association (Delinsky & Wilson 2008). In the FinnTwin16 cohort study, lifetime IWLs of 5 kg or more before the end of follow-up were associated with a risk of becoming overweight during a nine-year follow-up (between ages 16 and 25) (Pietiläinen *et al.* 2012). In a study conducted with the same cohort and later data collection waves, lifetime IWLs of 5 kg or more before the mean age of 24 were associated with poorer odds of maintaining weight versus gaining weight during a 10-year follow-up (between ages of 24 and 34) (Kärkkäinen *et al.* 2018).

A study based on the CARDIA Study data found different dieting measures to predict both weight gain and weight loss (Bild *et al.* 1996). The percentages of those ever on a diet, now on a diet, ever lost 10 pounds, or lost 10 lb  $\geq 3$  times were significantly greater among both those who gained weight and those who lost weight during a two-year follow-up compared to those who maintained a stable weight. No observational studies, however, have demonstrated dieting to predict weight loss only.

**Table 3** Summary of follow-up studies on the prediction of self-report dieting attempts or IWL on subsequent changes in obesity measures in general adult populations.

Study, BL year, country, (reference)	Study population	Follow-up period	Exposure	Outcome	Results <sup>1</sup>	Control for confounding
<b>Studies with risk of gaining, losing or maintaining weight as outcome and odds ratio (OR) or relative risk (RR) as measure of association</b>						
Atherosclerosis Risk in Communities Study, 1987, USA (Juthaeri et al. 2001)	10 554 M and W aged 45–64	6 years	Dieting to lose weight	Odds of losing weight (weight change <0 kg/year) calculated from measurements	Dieters compared to non-dieters: White W: <b>OR 0.53 (0.39–0.72)</b> White M: <b>OR 0.52 (0.31–0.86)</b> African-American W: OR 0.51 (0.26–1.00)	Age, study center, BMI, height, WHR, smoking, education, and PA
Australian Longitudinal Study on Women's Health, 1996, Australia (Brown et al. 2016)	4881 W aged 18–23	16	Dieting history in the last year: never, occasionally (1–4 times), or frequently (≥5 times)	Odds of maintaining a healthy BMI (18.5–24.9) compared to progressing to overweight or obesity calculated from self-reports	Occasionally vs. Never: <b>OR 0.81 (0.69–0.97)</b> Frequently vs. Never: OR 1.02 (0.78, 1.34)	Education, area of residence, country of birth, occupation, marital status, parity, smoking status, alcohol consumption, PA, takeout food, major illness, oral contraceptive pill
European Prospective Investigation into Cancer and Nutrition (EPIC), 1994, Germany (Kroke et al. 2002)	18 001 M and W aged 19–70	2 years	IWL of ≥5 kg during last 2 years	Odds of small (>1 to <2 kg/y) and large (≥2 kg/y) weight gain calculated from measurement at baseline and self-report at endpoint	In polytomous logistic regression model gain compared to stable weight: W: Small gain: <b>OR 2.28 (2.72–2.89)</b> Large gain: <b>OR 4.77 (3.70–6.15)</b> M: Small gain: <b>OR 1.95 (1.43–2.66)</b> Large gain: <b>OR 2.84 (2.00–4.05)</b>	W: age, BL BMI, education, PA, energy intake, fat intake, anti-depressive drugs, corticoid or pharmacological thyroid therapy during follow-up, psycho-pharmacological drug use before BL, dietary changes before BL, dietary changes during follow-up, and prevalent diabetes M: age, BL BMI, education, energy intake, fat intake, anti-depressive drugs, antiepileptic drugs, insulin use during follow-up, dietary changes before BL, dietary changes during follow-up and prevalent stroke

Table 3 continues

Study, BL year, country, (reference)	Study population	Follow-up period	Exposure	Outcome	Results'	Control for confounding
Finnish Twin Cohort, 1975, Finland (Korkeila <i>et al.</i> 1999)	7729 M and W aged 18–54	6 and 15 years	Currently trying to lose weight	Risk of major weight gain (>10 kg) calculated from self-reports	Dieters compared to non-dieters during 6-year follow-up / 15-year follow-up (respectively): W aged 18–29: OR 1.60 (0.95, 2.71) / OR 0.97 (0.68, 1.37) W aged 30–54: OR 2.42 (1.27, 4.61) / OR 1.41 (0.96, 2.09) M aged 18–29: OR 2.91 (1.54, 5.48) / OR 1.94 (1.18, 3.20) M aged 30–54: OR 1.43 (0.71, 2.91) / OR 2.30 (1.36, 3.20)	Age, BL BMI, smoking, alcohol, education, and social class
FinnTwin16 study, 1991, Finland (Pietiläinen <i>et al.</i> 2012)	4129 M and W aged 16	9 years	Lifetime IWL of 5 kg or more before age of 25	Risk of becoming overweight (BMI <sub>≥25</sub> kg/m <sup>2</sup> ) calculated from self-reports	Lifetime IWLs: Once / Twice or more (respectively) compared to Never: W: OR 2.72 (1.72–4.30) / OR 5.22 (3.17–8.60) M: OR 1.82 (1.27–2.60) / OR 2.04 (1.27–3.29)	BL BMI, PA, smoking, eating breakfast, number of children in females, mother's and father's overweight, and father's socioeconomic status
FinnTwin16 study, 2000, Finland (Kärkkäinen <i>et al.</i> 2018)	4679 M and W aged 22–28	10 years	Lifetime IWL of 5 kg or more before BL	Risk of weight maintenance (±5% of BL weight) vs. weight gain (gain of >5% of BL weight) calculated from self-reports	Lifetime IWL compared to never: W: OR 0.59 (0.39–0.88) M: OR 0.31 (0.16–0.60)	BL BMI, food and drink intake, PA, eating-related variables, education, life satisfaction, and self-rated health
Household Income and Labour Dynamics in Australia survey, 2009, Australia (Siahpush <i>et al.</i> 2015)	8824 M and W, age range NR	1 year	Frequency of dieting during last 12 months (Never, Once, More than once, Always).	A trichotomous BMI variable (Gained, Maintained, Lost) calculated from self-reports	Once, More than once, Always vs. Never: Odds of gaining versus maintaining: Once: OR 1.39 (1.11–1.75) More than once: OR 1.45 (1.08–1.94) Always: OR 2.58 (1.65–4.05) Odds of gaining versus losing: Once: OR 1.48 (1.24–1.77) More than once: OR 1.47 (1.20–1.80) Always: OR 1.35 (1.00–1.87)	Sampling design, BL obesity, self-rated health, mental health, psychological distress, PA, smoking, age, sex, English-speaking, marital status, education, equalized income

Table 3 continues

Study, BL year, country, (reference)	Study population	Follow-up period	Exposure	Outcome	Results <sup>1</sup>	Control for confounding
Study name NR, BL year NR, USA (Shice <i>et al.</i> 1999)	692 W aged 13.6–17.1	4 years	Currently on a diet, Exercise for weight control	Onset of obesity (BMI>25 kg/m <sup>2</sup> ) in those initially non-obese, calculated from measurements	Currently on a diet compared to not: <b>RR 3.24 (1.53–6.85)</b> Exercise for weight control: <b>RR 1.25 (1.08–1.45)</b>	Sexual maturity level
<b>Studies with weight change as outcome and regression coefficient as measure of association</b>						
The 1970 British Birth Cohort, 1986, UK (Viner & Cole 2006)	4461 M and W aged 16	14 years	Ever tried to lose weight by dieting (altering diet), Ever tried to lose weight by exercise	Change of zBMI calculated from measurement at BL and self-report at endpoint	Yes compared to no: <i>Ever tried to lose weight by dieting:</i> <b><math>\beta</math> 0.09 (0.01–0.18), P=0.04</b> <i>Ever tried to lose weight by exercise:</i> <b><math>\beta</math> 0.03 (-0.10–0.16), P=0.6</b>	Sex, social class, height at 16 and 30 years, and zBMI at 16 years
Health Professionals Follow-up Study, 1988, USA (Coakley <i>et al.</i> 1998)	19 478 M aged 40–75	4 years	Number of IWLs of $\geq 2.3$ kg during previous 16 years	Weight change calculated from self-reports	Number of IWLs of $\geq 2.3$ kg during previous 16 years: <b>Age 45–54y: <math>\beta</math> 0.17 (0.02), P&lt;0.001</b> <b>Age 55–64 y: <math>\beta</math> 0.07 (0.03), P&lt;0.01</b> <b>Age <math>\geq 65</math> y: <math>\beta</math> 0.23 (0.04), P&lt;0.001</b>	Weight, height, vigorous activity, TV/VCR viewing, high blood pressure, and high cholesterol
Healthy Worker Project, 1987, USA (French <i>et al.</i> 1994b)	3552 M and W mean age 38.1 (10.3)	2 years	Ever dieted to lose weight, Previously participated in formal weight loss program, Currently dieting	Weight change calculated from measurements	Yes compared to no: Ever dieted to lose weight: <b>M: B 0.80 (SE 0.58), P=0.17</b> <b>W: B 0.53 (SE 0.68), P=0.43</b> Participated in weight loss program: <b>M: B 4.83 (SE 1.12), P=0.0001</b> <b>W: B 1.74 (SE 0.64), P=0.006</b> Currently dieting: <b>M: B 0.61 (SE 0.83), P=0.47</b> <b>W: B 1.99 (SE 0.64), P=0.002</b>	BL body weight, age, education, occupation, marital status, smoking, treatment group, and company identification number
National Longitudinal Study of Adolescent Health, 1994, USA (Field <i>et al.</i> 2007)	8402 M and W aged 11–20	5 years	Dieting to lose or maintain weight during the past week in Wave I or II (prior to follow-up)	BMI change calculated from measurements	Dieting in Wave I or II compared to never: <b>W: <math>\beta</math> 0.39 (0.08–0.71)</b> <b>M: <math>\beta</math> 0.46 (-0.20–1.12)</b>	Age, BMI in Wave I, change in BMI from Wave I to II, activity level in adolescence, television viewing in adolescence, region of the country at Wave I, and race



Table 3 continues

Study, BL year, country, (reference)	Study population	Follow-up period	Exposure	Outcome	Results <sup>a</sup>	Control for confounding
Study name NR, BL year NR, USA (Savage <i>et al.</i> 2009)	163 W aged 24–46	6 years	Currently dieting to lose weight	Weight change calculated from measurements	Dieting at baseline by time: <b>B 0.36 (SE 0.15), P=0.02</b>	Interaction for time
Study name NR, 2007, The Netherlands (van Strien <i>et al.</i> 2014)	675 M and W aged 18–75	3 years	Frequency of one dietary restraint item “I diet”	BMI change calculated from self-reports	<b>B 0.162 (SE 0.038), <math>\beta</math> 0.176, P&lt;0.001</b>	NR
<b>STUDIES with other study question approaches</b>						
CARDIA study, 1985, USA (Bild <i>et al.</i> 1996)	4278 M and W aged 18–30	2 years	Weight loss practices: Ever on diet, Now on diet, Ever lost 10 lb, Lost 10 lb $\geq$ 3 times	Loss or gain of $\geq$ 5% weight calculated from measurements	Of those with Loss, Stable, Gain (respectively): % Ever on diet: <b>45.2</b> , 31.7, <b>38.6</b> % Now on diet: <b>11.7</b> , 6.2, <b>9.1</b> % Ever lost 10 lb: <b>82.7</b> , 70.3, <b>77.0</b> % Lost 10 lb $\geq$ 3 times: <b>48.4</b> , 35.9, <b>42.9</b> P value for all comparisons between Loss vs. Stable and Gain vs. Stable < <b>0.005</b>	Age, BL BMI, sex, race
Project EAT (Eating and Activity in Teens and Young Adults), 1998, USA (Neumark-Sztainer <i>et al.</i> 2012)	1902 M and W aged 11–18	10 years	Dieting during last year in Wave I or II: not dieting at either time point (No); not dieting in Wave I but dieting in Wave II (Starters); dieting in Wave I but not in Wave II (Stoppers); and dieting at both time points (Persistent)	BMI change calculated from self-reports	Mean increases in BMI (P value for difference compared to No): M: No: 3.45 (SE 0.18) Starters: <b>4.88 (SE 0.32), P&lt;0.001</b> Stoppers: 3.24 (SE 0.34), P=0.592 Persistent: <b>6.96 (SE 0.41), P&lt;0.001</b> W: No: 2.38 (SE 0.32) Starters: <b>3.49 (SE 0.37), P=0.022</b> Stoppers: <b>3.78 (SE 0.37), P=0.004</b> Persistent: <b>4.33 (SE 0.26), P&lt;0.001</b>	Ethnicity/race, SES, age cohort, and Time 1 BMI
Study name NR, BL year NR, USA (Lowe <i>et al.</i> 2006)	69 W aged 18–19	10 months	Currently dieting or ever been on a diet to lose weight (historical dieters)	Weight gain calculated from measurements	Mean weight gain (kg): Current dieters: 5.0 Historical dieters: 2.5 Never-dieters: 1.6 Due to small number in categories, differences not analyzed statistically.	-

Table 3 continues

Study, BL year, country, (reference)	Study population	Follow-up period	Exposure	Outcome	Results <sup>1</sup>	Control for confounding
Study name NR, BL year NR, USA (Delinsky & Wilson 2008)	149 W mean age 17.92 (0.50)	7 months	Currently on a weight loss diet	Weight change and BMI change calculated from self-reports	Dieters compared to non-dieters: Weight change: $t(144)=0.53$ , $P=0.594$ BMI change: $t(143)=0.33$ , $P=0.745$	NR

Abbreviations:  $\beta$ , standardized beta coefficient; B, unstandardized regression coefficient; BL, baseline; IWL, intentional weight loss; M, men; MZ, monozygotic; NR, not reported; OR, odds ratio; PA, physical activity; RR, relative risk; SE, standard error; SES, socioeconomic status, SR, self-report; W, women; WHR, waist-to-hip ratio; zBMI, BMI z-score

<sup>1</sup> Values in parentheses denote 95% confidence interval or standard deviation unless stated otherwise. Results in bold are statistically significant ( $P<0.05$ ).

A few studies have examined whether certain factors modify the associations between dieting and later weight gain. The results of these studies have been partly inconsistent. It appears that the elevated risk of dieters gaining weight is pronounced among young women (Field *et al.* 2007), middle-aged women (Korkeila *et al.* 1999) and young men (Korkeila *et al.* 1999). The findings also suggest that the risk is accentuated among dieters with normal weight (Korkeila *et al.* 1999, Pietiläinen *et al.* 2012).

Even though generally any measure of dieting appears to be related to subsequent weight gain, it seems that repeated dieting is associated with an even greater weight gain (Coakley *et al.* 1998, Neumark-Sztainer *et al.* 2012, Pietiläinen *et al.* 2012). Some divergent findings, however, also exist. In an Australian study with a 16-year follow-up, even though occasional dieting (1–4 times) during the last year was negatively associated with weight maintenance, no significant association emerged between frequent dieting (5 times or more) and weight maintenance (Brown *et al.* 2016). Moreover, in another Australian study with a rather short follow-up of one year, those reporting constant weight loss dieting had substantially greater odds of gaining weight than maintaining weight compared to those never dieting (Siahpush *et al.* 2015). Conversely, those reporting constant dieting had merely significant odds of gaining weight than losing weight compared to those never dieting. In the study by Viner *et al.* (2006), only weight loss attempts by dieting (altering diet) were related to subsequent weight gain, while weight loss attempts using PA were not. In contrast, in a study by Stice *et al.* (1999), both current dieting (altering diet) and exercising for weight loss predicted an elevated risk of onset of obesity.

A few review articles have been published on the association between self-report dieting attempts and IWL and later changes in obesity measures. A Medicare’s review by Mann *et al.* (2007) concluded that generally no evidence supports dieting as predicting sustained weight loss or improvements in health. In spite of finding a seemingly clear association between dieting and subsequent weight gain in their earlier review (Lowe *et al.* 2013), Lowe (2015) suggested that dieting would not predispose an individual to any greater weight gain that would have occurred in the absence of dieting, and in individuals without obesity dieting would be only a proxy risk factor of proneness to gaining weight in response to other reasons. In line with this, Hill (2004) concluded “it is not that dieting makes you fat, but that being fat makes you (more likely to) diet”. However, in contrast with this, an expanding amount of evidence suggests that dieting per se, especially in individuals with initially normal weight, has an independent effect on the development of obesity (Dulloo *et al.* 2015, Dulloo *et al.* 2018).

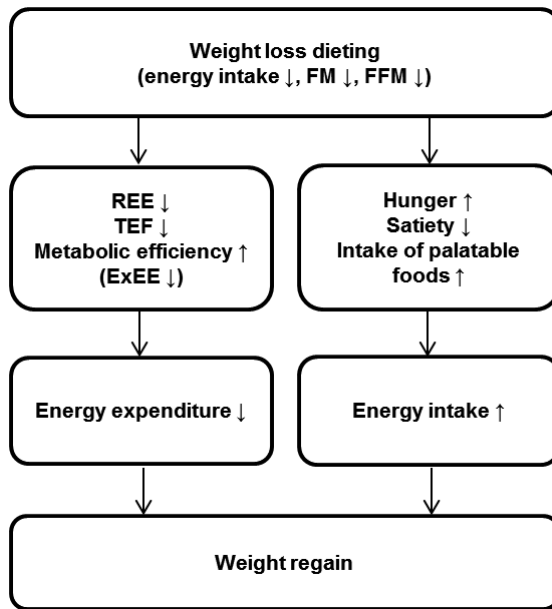
## 2.2.6 POTENTIAL MECHANISMS BETWEEN DIETING AND SUBSEQUENT WEIGHT GAIN

For many, weight maintenance turns out to be the most demanding phase of weight loss (Wing & Phelan 2005). According to the population-based National FINRISK 2012 Study, of adults who had succeeded to intentionally lose 5 kg or more during the last 10 years, approximately three in four reported regaining it all (Borodulin *et al.* 2013). It is common for weight regain to result in a higher post-dieting weight than the dieter's initial weight. Already in the classical Minnesota experiment (Keys 1950), results indicated that after a strict energy intake restriction diet, when returning to free access to food, the mean regain of the participants surpassed their initial weight. In 1985, Polivy and Herman also proposed that binge eating was usually preceded by dieting, and hence dieting may predispose people to disinhibition and overeating (Polivy & Herman 1985).

Potential mechanisms explaining the weight regain, commonly following dieting, and the weight regain exceeding initial weight have been suggested. In response to a decrease in energy intake, FFM and FM, the body's biological anti-weight loss mechanisms become activated (Ochner *et al.* 2013, Sumithran & Proietto 2013). Such mechanisms primarily aim to prevent and replenish the loss of FFM i.e. protect the muscles and organs, even in the presence of excess FM (Dulloo 2017). Behavioral changes induced by dieting may also play a role in weight regain.

During and after dieting and weight loss, the restriction of energy intake and diminishing FM and FFM contribute to the decline in total daily energy expenditure (TDEE) (mostly consisting of decreased REE, the decreased thermic effect of food (TEF) and increased metabolic efficiency in daily activities, but often also of decreased exercise energy expenditure (ExEE)), an increase in appetite, a decrease in satiety, and an increased intake of palatable foods (Bosy-Westphal *et al.* 2013, Polidori *et al.* 2016, Melby *et al.* 2017). These adaptations contribute to weight regain. This process is roughly illustrated in Figure 2.

The Minnesota experiment was the first study to measure the decline in REE after weight loss (Keys 1950). Decline in REE attenuates weight loss and hastens weight regain, as the daily energy deficit needs to be even greater to achieve weight loss or avoid regaining the weight (Bosy-Westphal *et al.* 2013, Dulloo & Schutz 2015). Decline in REE is not entirely accounted for in the decrease of energy intake and diminishing of FM and FFM. In turn, beyond their effect, adaptive thermogenesis (AT) – the body's metabolic adaptation mechanism that varies inter-individually – contributes to the decline (Dulloo & Schutz 2015). By lowering REE, AT attenuates weight loss results and hastens weight regain.



**Figure 2** Process from weight loss to weight regain.

Abbreviations: ↓ decrease; ↑ increase; ExEE, exercise energy expenditure; FFM, fat free mass; FM, fat mass; REE, resting energy expenditure; TEF, thermic effect of food.

During the weight loss period, both FM and FFM are lost. Consequently, biological mechanisms, including an increase in appetite (hyperphagia) are activated in order to replenish the lost tissues, FFM in particular. Hyperphagia has been indicated to persist until total recovery of the tissues (MacLean *et al.* 2015, Dulloo *et al.* 2018). Hence, it has been suggested that weight regain beyond initial weight results because of a slower rate of replenishment of FFM when regaining weight. Thus, when FM has recovered to correspond to the level before weight loss, FFM is not entirely replenished. Consequently, as biological mechanisms driving the replenishment of FFM persist, FM continues to increase beyond its initial level. This phenomenon is called fat overshooting (Dulloo *et al.* 2015) or collateral fattening (Dulloo *et al.* 2018).

Both hormonal and neural systems contribute to energy homeostasis and weight regulation. Dozens of gut, adipose tissue, and central nervous system-secreted hormones, neuropeptides and neurotransmitters have been identified as affecting appetite (Lean & Malkova 2016). Part of these factors have anorexigenic functions by increasing satiety, suppressing energy intake, and increasing energy expenditure, while others have opposite orexigenic functions. Leptin is an adipocyte-secreted hormone whose primary function is to signal the amount of peripheral energy storages (i.e. FM) to the

receptors in the hypothalamus and promote satiety. Leptin levels decrease in response to energy restriction and in response to a decrease in FM. Reduction of the leptin level triggers biological mechanisms that result in an increase in hunger and food intake, i.e. hyperphagia, and a decrease in REE (Ochner *et al.* 2013). Ghrelin is a hunger-stimulating hormone secreted mainly from the stomach. Ghrelin levels have been shown to increase during diet-induced weight loss and to remain elevated for even a year after weight loss (Lean & Malkova 2016). In addition to leptin and ghrelin, various other hormones (e.g. peptide YY (PYY) and cholecystokinin) contribute to the body's tendency to regain lost weight.

The neural homeostatic system with the hypothalamus regulates energy homeostasis by hunger and satiety according to the body's energy needs. During weight loss in particular, the hedonic food reward system, however, has been indicated to easily override the homeostatic system, attenuate satiety, and favor an intake of energy-dense foods and thus, promote weight gain (Ochner *et al.* 2013). Moreover, this tendency towards the activity of the food reward system associates with responsivity to food cues, and appears to persist after the weight loss period, facilitating weight regain.

Another factor that potentially explains weight regain is related to adipose tissue. With initial excess weight gain, both adipocyte size (hypertrophy) and number (hyperplasia) increase. During weight loss, adipocytes become smaller, but their number, proliferated during earlier weight gain, remains at an elevated level. Thus, it has been suggested that the greater amount of newly-reduced adipocytes hinders weight loss and promotes weight regain (Ochner *et al.* 2013). Adipocytes have been shown to communicate with the brain during and after weight loss and to promote appetite beyond biological energy needs (MacLean *et al.* 2015).

In individuals with less initial body fat, weight loss consists of relatively more FFM than FM compared to individuals with higher initial body fat (Forbes 2000, Dulloo *et al.* 2018). The relative loss of FFM is most notable in individuals with the least body fat and levels out in parallel with more body fat (Forbes 2000). Accordingly, when individuals with low initial body fat lose weight, and more FFM is lost, when weight regain occurs, fat overshooting (as described above) persists longer, enabling greater FM gain (Dulloo *et al.* 2015).

As dieting commonly fails, unsuccessful dieting attempts often lead to the next attempt, and eventually to weight cycling. Weight cycling refers to multiple cycles of decreasing and increasing weight. No unequivocal exact definition has been established, but usually weight cycling denotes several intentional weight loss episodes followed by unintentional weight regain episodes. A Finnish study estimated that during the preceding 10 years, approximately 7% of men and 10% of women had experienced severe weight cycling (weight loss of  $\geq 5$  kg at least three times with regain), and 11% of men and 19% of women had experienced mild weight cycling (weight loss of  $\geq 5$  kg one to two times with regain) (Lahti-Koski *et al.* 2005). Weight cycling has

been associated with subsequent weight gain (Kroke *et al.* 2002, Field *et al.* 2004, Saarni *et al.* 2006). It is possible that each weight cycle results in a little higher body weight and a more unfavorable body composition, as during the weight loss phase, relatively more FFM is lost and during the weight regain phase, relatively more FM is accumulated. However, evidence on unfavorable changes in body composition remains inconclusive (Mackie *et al.* 2017). Chronic dieting may be associated with lower dieting self-efficacy contributing to overeating and unhealthy food choices (French & Jeffery 1997), which could even further promote the tendency to gain weight.

In sum, various physiological and behavioral potential pathways exist by which dieting and weight loss contribute to weight regain and substantially hinder weight maintenance.

## **2.3 TYPE 2 DIABETES**

### **2.3.1 DEFINITION**

Diabetes denotes chronic and progressive diseases that include elevation of blood glucose concentration. Such a condition may result from the malfunctioning or entirely suppressed ability of pancreatic  $\beta$ -cells in the islets of the Langerhans to secrete insulin or from impaired insulin sensitivity and, thus, uptake of glucose in the peripheral tissues, i.e. insulin resistance (Forbes & Cooper 2013). Depending on the type of diabetes, both causes can occur simultaneously.

Diabetes comprises a relatively heterogeneous group of diseases (Tuomi *et al.* 2014). T2D is the most common type of diabetes overall, especially in adulthood in the Western world (Roglic & World Health Organization 2016, Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018, World Health Organization 2019). According to WHO's new classification of types of diabetes, other types include type 1 diabetes (T1D), hybrid forms of diabetes (slowly evolving immune-mediated diabetes of adults and ketosis-prone T2D), other specific types of diabetes (e.g. monogenic diabetes and different types of diabetes induced by diseases, disorders, drugs, or infections), and hyperglycemia first detected during pregnancy (diabetes mellitus in pregnancy and gestational diabetes mellitus) (World Health Organization 2019). Traditionally, the most common types of diabetes in addition to T1D and T2D have been categorized as LADA (Latent Autoimmune Diabetes in Adults), MODY (Maturity-Onset Diabetes of the Young), and gestational diabetes (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). Apart from T2D, incidence of the other types of diabetes in adulthood is relatively rare (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018, World Health Organization 2019), and their

pathophysiology differs from that of T2D (Zaccardi *et al.* 2016, McCabe & Perng 2017). Moreover, of the different types of diabetes, onset of T2D appears to be susceptible to the effect of unfavorable lifestyle factors, whereas in other types of diabetes, apart from gestational diabetes, lifestyle does not play such a marked role (World Health Organization 2019).

The clinical diagnostic criteria for T2D are based on population studies; the cut-off values in the criteria have been defined according to the average impaired prognosis and the increase in complications. The criteria have been defined by WHO and the IDF (World Health Organization & International Diabetes Federation 2006, World Health Organization 2019), and have been updated over the years. According to the latest criteria, T2D is diagnosed with a fasting plasma glucose value of  $\geq 7.0$  mmol/l, a 2-h post-load (75 g) plasma glucose value of  $\geq 11.1$  mmol/l, an HbA<sub>1c</sub> value of  $\geq 48$  mmol/mol (6.5%) (representing poor long-term glycemic balance), or a random blood glucose value of  $\geq 11.1$  mmol/l with the presence of symptoms (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018, World Health Organization 2019). The symptoms may include thirst, increased urination, fatigue, and unexplainable weight loss. In the absence of symptoms, and when using only a fasting plasma glucose test or a random glucose test, a repeat of the test is recommended. When using the HbA<sub>1c</sub> test, it is important to keep in mind that in the presence of a condition causing a shortened or lengthened life cycle of the erythrocytes, the test may give false concentrations. In addition, the absence of ketones in the blood or in the urine (presence indicating T1D) and the measurement of C-peptide concentration can be used to draw the line between the types of diabetes (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). Occasionally, however, the type of diabetes only becomes clear with follow-up.

In epidemiological studies, fasting plasma glucose value is often used as the only measure to determine T2D due to the glucose tolerance test being more time-consuming and expensive to conduct in large population samples. HbA<sub>1c</sub> levels can also be utilized (World Health Organization 2011a), but are considered less reliable than the glucose tolerance test.

### 2.3.2 PATHOPHYSIOLOGY

In obesity, adipose tissue-released non-esterified fatty acids, glycerol, hormones (e.g. leptin and adiponectin), and proinflammatory cytokines contribute to the development of insulin resistance (Kahn *et al.* 2006). Usually in T2D, insulin insufficiency is relative, as in the pre-stages of the disease insulin secretion works properly, and insufficiency derives from insulin resistance in the peripheral insulin-sensitive tissue cells (e.g. skeletal muscle, adipose tissue and liver) (Forbes & Cooper 2013). In a healthy condition, a feedback loop between the insulin-sensitive cells and pancreatic



$\beta$ -cells secure the homeostasis in the glucose balance; peripheral cells respond to insulin in the blood by increasing their uptake of glucose, whereas  $\beta$ -cells release a sufficient amount of insulin in response to blood glucose concentration (Laakso 2015). With impaired insulin sensitivity, the peripheral cells' ability to respond to insulin and take up glucose is reduced. Such insulin resistance leads to elevated blood glucose concentration, to which the pancreatic  $\beta$ -cells respond by increasing a secretion of insulin in order to secure homeostatic blood glucose balance. Over time, insulin secretion starts to decline, and the secretion rate fails to keep up with increasing needs, facilitating hyperglycemia and the development of T2D. Further, hyperglycemia impairs the  $\beta$ -cells' ability to secrete insulin, and thus, a vicious circle emerges. In T2D, in addition to meal-induced hyperglycemia in the postprandial stage, hyperglycemia also occurs during the fasting state due to relative hypoinsulinemia.

Pathophysiological pathways to T2D, however, are relatively heterogeneous. Most of the risk genes increase the risk through reduced beta cell function, although some do so through the mediation of obesity, lipodystrophy or liver-lipid metabolism, leading to insulin resistance (Udler *et al.* 2018).

T2D may be symptomless for years and is often diagnosed by chance as part of routine laboratory screening or once complications appear. In the National FinHealth 2017 Study, 3.1% of men and 1.4% of women aged 30 and older, who were unaware of having diabetes, were diagnosed as potential diabetics based on elevated HbA1c and fasting glucose determinations (Koponen *et al.* 2018). Depending on its severity, T2D can be treated with oral or injection medication, insulin, or with lifestyle modifications (Roglic & World Health Organization 2016, Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018).

### **2.3.3 PREVALENCE AND TRENDS**

Rising T2D rates are a global problem. WHO estimated that the prevalence of all types of diabetes combined in adults grew from 4.7% in 1980 to 8.5% in 2014 (Roglic & World Health Organization 2016). In Finland, according to estimations, over 500 000 individuals have diabetes (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). In the National FinHealth 2017 Study, 15% of men and 9% of women aged 30 and over reported having diabetes diagnosed by a doctor, or were diagnosed during the study based on elevated HbA1c or fasting glucose concentrations (Koponen *et al.* 2018). Based on new diabetes drug reimbursement cases and population HbA1c concentrations, the increase in the incidence rate of T2D, however, seems to have attenuated during the last 10 years (Lundqvist *et al.* 2019).

### 2.3.4 RISK FACTORS AND PREVENTION OF TYPE 2 DIABETES

Major lifestyle risk factors that contribute to the development of T2D are obesity, lack of PA, and an energy-dense and unhealthy diet (Laaksonen *et al.* 2010, Kahn *et al.* 2014, Kolb & Martin 2017). Healthy dietary patterns and high scores in diet quality indices, as well as a high consumption of vegetables, fruits, wholegrain, fiber, non-tropical vegetable oils, and fish, and a low consumption of red meat and sugar-sweetened beverages are associated with a lowered risk (Montonen *et al.* 2005, Esposito *et al.* 2010, Jannasch *et al.* 2017, Neuenschwander *et al.* 2019). In addition to PA level, cardiorespiratory fitness and muscle strength have also been shown to have an inverse association with T2D risk (Tarp *et al.* 2019). Genes and family history of diabetes predispose to the development of the disease (Kahn *et al.* 2014, Kwak & Park 2016, Kolb & Martin 2017). At least 243 independent loci have been identified as contributing to the risk (Mahajan *et al.* 2018). Some of the gene variants predispose to T2D by interactions with lifestyle factors, which modulate gene expression via epigenetic mechanisms. Moreover, several other factors such as increasing age, ethnicity, stress, and inadequate sleep have been linked to an increased risk of T2D (Kahn *et al.* 2014, Itani *et al.* 2017, Kolb & Martin 2017). Various biomarkers have also been found to associate with a risk of T2D (Laakso 2019).

However, the rising incidence of T2D is mostly attributable to the obesity epidemic, and it has been estimated that 77% of incident T2D cases could be avoided if everyone had a BMI of 25 kg/m<sup>2</sup> or less (Laaksonen *et al.* 2010). Visceral and ectopic fat in particular have been associated with an elevated risk (Neeland *et al.* 2012, Sattar & Gill 2014). It has also been suggested that weight cycling may increase the risk of T2D, although the consensus on this issue remains under debate (Rhee 2017, Kodama *et al.* 2017). Nevertheless, the pathways by which weight cycling has been suggested to increase the risk of cardiometabolic diseases include fluctuations in metabolic milieu and functions (Montani *et al.* 2015, Rhee 2017). In addition to obesity, the accumulation of unfavorable changes in metabolic factors (blood pressure, concentrations of serum fasting glucose, serum HDL and serum triglycerides) is strongly associated with the risk of T2D (Shin *et al.* 2013).

In clinical practice, the prevention of T2D among individuals at an elevated risk consists of lifestyle changes including a healthy diet and sufficient PA. For individuals with normal weight, weight maintenance is essential, while for individuals with overweight or obesity, weight loss is recommended (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). Individuals with obesity can benefit from VLCD at the beginning of weight loss. VLCD, however, is not sufficient alone; healthy lifestyle changes must be adopted when targeting long-term weight maintenance (Lean 2019).

A large number of weight loss and lifestyle intervention studies have indicated that intervention reduces the risk of T2D, the first of which being conducted in Finland (Tuomilehto *et al.* 2001, Peirson *et al.* 2014, Haw *et al.*

2017, LeBlanc *et al.* 2018). In these studies, the interventions targeted weight loss through alterations in diet and increased PA. Some of the studies also included actions to promote changes in attitudes and behavior or to increase motivation to change lifestyle through individual or group sessions. The studies' intervention periods ranged between 0.5 and 6.3 years (Haw *et al.* 2017).

In the Finnish DPS, the aims for the participants with overweight or obesity were to lose 5% or more of their weight, to lower their intake of fat to less than 30% and saturated fat to less than 10% of their daily energy intake, to increase their intake of fiber to 15 g or more per 1000 kcal, and to participate in moderate exercise for at least 4 h per week. In addition, consumption of whole-grain products, vegetables, fruits, low-fat milk and meat products, soft margarines, and vegetable oils rich in monounsaturated fatty acids was encouraged (Tuomilehto *et al.* 2001). In this study, the risk of developing T2D during the average follow-up of 3.2 years was reduced by 58% in the lifestyle intervention group in comparison to the control group. In accordance, in the DPP, during the mean 2.8-year follow-up, the risk was also reduced by 58% in the intervention group in comparison to the control group (Knowler *et al.* 2002). Both of these seminal intervention studies also showed that the favorable consequences lasted for over 10 years after the trial periods (Diabetes Prevention Program Research Group *et al.* 2009, Lindström *et al.* 2013), suggesting that sustained weight loss and lifestyle changes indeed reduce the risk of T2D incidence.

### **2.3.5 CONSEQUENCES OF TYPE 2 DIABETES**

T2D predisposes individuals to macrovascular (e.g. coronary heart disease (CHD), stroke) and microvascular (e.g. retinopathy, neuropathy, nephropathy) complications (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). In good therapeutic equilibrium, T2D does not usually cause acute symptoms, but with inadequate treatment and long-term hyperglycemia, the risk of macro- and microvascular complications becomes elevated (Emerging Risk Factors Collaboration *et al.* 2010, Forbes & Cooper 2013, Stehouwer 2018). Treatment for hyperglycemia reduces the risk of microvascular complications but plays only a minor role in preventing macrovascular complications. Thus, treatment of hypertension and lipid disturbances is usually essential for the prevention of such conditions among individuals with T2D.

Moreover, diabetic complications have been shown to associate with a decreased quality of life and functional capacity, and an increased risk of depressive and anxiety disorders (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). Further, mortality is substantially greater among individuals with diabetes than in the general population (Forssas *et al.* 2010).

In addition to the public health burden of obesity, T2D is another major culprit stressing public health care and the economy. Of the total public health care costs in Finland, 15% are accounted for by the direct medical care expenses of diabetes (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018). According to the estimations of the Finnish diabetes association (Koski *et al.* 2018), the combined total costs incurred by diabetes increased by 49% during 2002–2011. Thus, prevention of T2D and research on the disease are essential.

### 2.3.6 EPIDEMIOLOGICAL FOLLOW-UP STUDIES ON THE PREDICTION OF WEIGHT LOSS ON THE RISK OF TYPE 2 DIABETES

In spite of the results of intervention studies, the scenario of maintained weight at the population level is relatively rare, as described in section 2.2. Moreover, intervention studies have mostly been conducted using subsamples with obesity, overweight, or otherwise at an initially elevated risk of developing T2D, as well as participants receiving support to lose and maintain weight. A clear distinction must be drawn between weight loss conducted within intervention studies or weight loss programs organized by professionals, and the weight loss efforts of the general population in relation to the subsequent risk of T2D. To discover whether IWL is associated with T2D incidence at the population level, population-based data must be used. Hence, this chapter focuses in more detail on epidemiological follow-up studies looking into the associations between WLW, IWL, dieting attempts and T2D incidence. The criteria for studies to be included in the review of this literature are presented in Table 4.

**Table 4** *Criteria for inclusion of follow-up studies in literature review.*

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1.	The outcome is T2D incidence or T2D-related death.
2.	The exposure is IWL, WLW, or dieting attempts before baseline or at baseline, but time period of weight change or weight loss attempts precedes the follow-up of T2D incidence.
3.	Participants with IWL, WLW, or dieting attempts are compared to participants maintaining their weight or not attempting to diet.
4.	Participants are free from T2D at baseline.
5.	Participants are adults ( $\geq 18$ years) at baseline or proceed into adulthood during the follow-up.

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Abbreviations: IWL, intentional weight loss; T2D, type 2 diabetes; WLW, weight loss without information on intentionality.

Several studies have examined whether WLW predicts the occurrence of T2D (Table 5). Most of such epidemiological follow-up studies have provided support for the preventive association of weight loss (Chan *et al.* 1994, Colditz *et al.* 1995, Resnick *et al.* 2000, Koh-Banerjee *et al.* 2004, Wannamethee *et al.* 2005, Kim *et al.* 2018, Stokes *et al.* 2018). For instance, in a large male study conducted using the Health Professionals Follow-up Study data (Chan *et al.* 1994), a retrospectively self-reported weight loss of 3 kg or more between the age of 21 and the beginning of the study predicted a decreased risk of incident T2D during a five-year follow-up in comparison to men who maintained their weight within a  $\pm 2$  kg margin. However, another study utilizing the Health Professionals Follow-up Study data from later study points and further outlining it to exclude those with existing diseases found no significant association between the same weight loss measure and T2D incidence (Koh-Banerjee *et al.* 2004). They did, however, find that a weight loss of 6 kg or more during the prior 10 years predicted a lowered risk of T2D in comparison to those maintaining their weight. Three American studies using data from different points from the National Health and Nutrition Examination Survey reached partly contradictory results (Ford *et al.* 1997, Resnick *et al.* 2000, Stokes *et al.* 2018). In a study by Resnick *et al.* (2000), the reduction in the risk of T2D grew in parallel with the amount of weight lost during the preceding 10 years. Stokes *et al.* (2018) concluded that a shift from obesity ( $\text{BMI} \geq 30 \text{ kg/m}^2$ ) to non-obesity ( $\text{BMI} < 30 \text{ kg/m}^2$ ) during the preceding 10 years predicted risk reduction in comparison to maintaining obesity. In contrast, in a study by Ford *et al.* (1997), inclusion of the confounding factors in the model attenuated the association to non-significant. Moreover, a study based on the Nurses' Health Study, consisting of 114 281 women, showed that weight loss in earlier adulthood prior to the baseline of the study predicted a lowered risk of T2D (Colditz *et al.* 1995). The results suggested that the greater the weight loss, the smaller the risk of developing T2D during a 14-year follow-up.

Despite these studies with results that support the preventive consequences of weight loss, some studies, however, have lacked significant findings (Ford *et al.* 1997, Moore *et al.* 2000, Oguma *et al.* 2005, Mishra *et al.* 2007, Waring *et al.* 2010, Nanri *et al.* 2011). No clear pattern on how these studies differed from the studies finding an association could be observed.

In addition to epidemiological studies with WLW, only three epidemiological follow-up studies have looked into the association between IWL and the incidence of T2D, all conducted using the extensive American Cancer Society's Cancer Prevention Study data (Williamson *et al.* 1995, Williamson *et al.* 1999, Will *et al.* 2002) (Table 5). One of these studies examined whether IWL predicted the occurrence of T2D during a 13-year follow-up (Will *et al.* 2002). In two other studies, the authors examined the prediction of IWL on T2D-related mortality separately in men and women

(Williamson *et al.* 1995, Williamson *et al.* 1999). All of these studies concluded that IWL was associated with a reduced risk. Not all the comparisons in these studies, however, found an association, and a reduced risk of T2D-related mortality only occurred in men and women with pre-existing health conditions (i.e. history of heart disease, stroke, diabetes, or high blood pressure, or reported current severe shortness of breath or severe pain in the chest) but not in men and women without such conditions. In addition to the IWL studies, in the San Antonio Heart Study data, greater efforts to control weight by dieting were associated with a tentatively decreased risk of developing T2D during an eight-year follow-up among women (Monterrosa *et al.* 1995). Among men, no association emerged in the final model when BMI was adjusted for. When BMI was excluded from the model, however, a tentatively elevated risk emerged in men. Apart from this one indicative finding, however, neither IWL, WLW nor dieting attempts predicted a significant increase in the risk in any of the studies.

**Table 5** Summary of follow-up studies that have assessed the prediction of IWL, dieting attempts or WLW on T2D or T2D-related death occurrence in general adult populations.

Study, BL year, country, (reference)	N/n	Study population	Exposure		Outcome <sup>2</sup>	Results <sup>3</sup>	Control for confounding	
			Follow-up (years)	Weight loss measurement <sup>1</sup>				Definition of weight loss
<b>Intentional weight loss or dieting attempts</b>								
Cancer Prevention Study, 1959, US (Williamson <i>et al.</i> 1995)	43 457/ 693	Never-smoking, W with overweight (BMI $\geq 27$ kg/m <sup>2</sup> ) aged 40–64	13	Self-reported	IWL of 0.5–9 kg, IWL of $\geq 9.1$ kg compared to no weight change	Death and cause of death (diabetes) from registers	No health conditions: IWL 0.5–9 kg: RR 0.83 (0.42–1.62) IWL $\geq 9.1$ kg: RR 1.02 (0.59–1.76) Pre-existing health conditions: IWL 0.5–9 kg: <b>RR 0.56 (0.38–0.82)</b> IWL $\geq 9.1$ kg: <b>RR 0.69 (0.53–0.90)</b>	Age, BL BMI, alcohol, education, PA, health conditions
Cancer Prevention Study, 1959, US (Williamson <i>et al.</i> 1999)	49 337/ 803	M with overweight (BMI $\geq 27$ kg/m <sup>2</sup> ) aged 40–64	13	Self-reported	IWL of 0.5–9 kg, IWL of $\geq 9.1$ kg compared to no weight change	Death and cause of death (diabetes) from registers	No health conditions: IWL 0.5–9 kg: RR 0.78 (0.40–1.50) IWL $\geq 9.1$ kg: RR 1.48 (0.93–2.33) Pre-existing health conditions: IWL 0.5–9 kg: <b>RR 0.68 (0.48–0.95)</b> IWL $\geq 9.1$ kg: <b>RR 0.64 (0.51–0.80)</b>	Age, BL BMI, smoking status, alcohol, education, PA, health conditions
Cancer Prevention Study, 1959, US (Will <i>et al.</i> 2002)	212 570/ 12 446	M and W with overweight (BMI $\geq 25$ kg/m <sup>2</sup> ) aged $\geq 30$	13	Self-reported	IWL (any amount) compared to no weight change	T2D self-reported at 4 time points after BL	M: <b>IDR 0.79 (0.73–0.86)</b> W: <b>IDR 0.72 (0.68–0.77)</b>	Age, BL BMI, race, education, intakes of fats and carbohydrates, alcohol, smoking, PA, heart disease, stroke, hypertension, cancer, cirrhosis, symptoms, and general health status
San Antonio Heart Study, 1979, US (Monterrosa <i>et al.</i> 1995)	844/57	M and W aged 25–64	8	Self-reported	Efforts to control weight through dieting (scale 0–2)	T2D self-reported and determined (laboratory measurements) at endpoint	I-point increase: M: OR 1.64 (0.78–3.40), W: OR 0.53 (0.28–1.00)	Age, SES, BL BMI, and structural assimilation

Table 5 continues

Study, BL year, country, (reference)	N/n	Study population	Follow-up (years)	Exposure		Outcome <sup>2</sup>	Results <sup>3</sup>	Control for confounding
				Weight loss measurement <sup>1</sup>	Definition of weight loss			
<b>Weight loss without information on intentionality</b>								
Australian Longitudinal Study on Women's Health, 1996, Australia (Mishra <i>et al.</i> 2007)	7 239/ 118 <sup>6</sup>	W aged 45– 50	3	Calculated from self-reports (5 y prior to follow-up)	Loss per y: ≥5%, 5–2.5%, 2.5–1.5% compared to loss or gain of <1.5%	Diabetes (including T1D) self-reported at endpoint	≥5%: OR 0.56 (0.22–1.41) 5–2.5%: OR 1.32 (0.82–2.14) 2.5–1.5%: 0.63 (0.32–1.21)	BMI, age, PA, smoking, education, menopause status, area of residence
The British regional heart study, 1978, UK (Wanamethee <i>et al.</i> 2005)	6 798/ 449	M aged 40– 59	15	Calculated from a measurement and a self-report (5 y prior to follow-up)	Loss of ≥4% compared to loss or gain of <4%	T2D self-reported at 2 times after BL (health record confirmation) and drawn from registers	<b>RR 0.62 (0.42–0.90)</b>	Age, social class, smoking, PA, alcohol, antihypertensive treatment, undiagnosed CHD, FEV, systolic blood pressure, total cholesterol, and BL BMI
The College Alumni Health Study, 1962, US (Oguma <i>et al.</i> 2005)	20 187/ 1223	M mean age 45.9	36	Calculated from a measurement and a self-report (27 y prior to follow-up)	BMI change per decade from university entry to BL: >5 kg/m <sup>2</sup> compared to loss or gain of ≤5 kg/m <sup>2</sup>	T2D self-reported at 5 or 3 time points after BL	RR 0.88 (0.59–1.32)	Age, PA, smoking, hypertension, family history of diabetes, and BMI at university entry
Framingham Heart Study, 1948, US (Moore <i>et al.</i> 2000)	618/85	M and W with overweight (BMI ≥27 kg/m <sup>2</sup> ) aged 30–50	20	Calculated from measurements (first 8 y and second 8 y prior to follow-up)	First 8 y (loss of ≥8 lb was considered a loss)/second 8 y: lost/gained, lost/stable, lost/lost compared to stable/stable	T2D self-reported and determined (laboratory measurements) biennially	Lost/gained: RR 1.30 (0.70–2.40) Lost/stable: RR 0.71 (0.37–1.40) Lost/lost: RR 0.45 (0.15–1.30)	Age, sex, education, BL BMI, height, PA, smoking, alcohol
Framingham Heart Study, 1948, US (Waring <i>et al.</i> 2010)	1 476/ 217	M and W aged 40	NR	Calculated from biennial measurements (10 y prior to follow-up)	Loss (score in quintile 1 on PC function reflecting weight change) compared to stable (score in quintile 2)	T2D self-reported and determined (laboratory measurements) biennially	HR 1.1 (0.7–1.8)	Weight status at age of ≥5, gender, ever use of hormones, alcohol, smoking, and education



Table 5 continues

Study, BL year, country, (reference)	N/n	Study population	Follow-up (years)	Exposure		Definition of weight loss	Outcome <sup>a</sup>	Results <sup>b</sup>	Control for confounding
				Weight loss measurement <sup>c</sup>	Weight loss				
The Health Professionals Follow-up Study, 1986, US (Chan <i>et al.</i> 1994)	27 983/ 272	M aged 40–75	5	Self-reported (time period between age of 21 and beginning of follow-up)	Loss of ≥3 kg compared to loss or gain of ≤2 kg	T2D self-reported at 3 time points after BL	<b>RR 0.3 (0.1-0.8)</b>	Age, family history of diabetes, smoking, and quintiles of BMI at age of 21	
The Health Professionals Follow-up Study, 1986, US (Koh-Banerjee <i>et al.</i> 2004)	22 171/ 305	M aged 50–85	4	Self-reported (time between age of 21 and BL), and calculated from self-reports (10 y prior to BL)	Between age of 21 and BL: loss of ≥3 kg vs. loss or gain of ≤2 kg 10 y prior to BL: Loss of ≥6 kg / loss of 3–5 kg vs. loss or gain of ≤2 kg	T2D self-reported at 2 time points after BL	Between age of 21 and BL: loss of ≥3 kg: RR 0.4 (0.2-1.1) 10 y prior to BL: Loss of ≥6 kg: <b>RR 0.5 (0.3-0.9)</b> loss of 3–5 kg: RR 1.0 (0.7-1.5)	Between age of 21 and BL: smoking, alcohol, PA, family history of diabetes, dietary fiber, and BMI at age of 21 y. 10 y prior to the BL: smoking, alcohol, PA, family history of diabetes, dietary fiber, BL BMI	
The Japan Public Health Center-based Prospective Study, 1990, Japan (Nanri <i>et al.</i> 2011)	52 014/ 989	M and W aged 45–75	5	Self-reported (time between age 20 and beginning of follow-up), and calculated from self-reports (5 y prior to follow-up)	Between age of 20 and beginning of follow-up: loss of ≥5 kg compared to loss or gain of <5 kg. 5 y prior to follow-up: loss of ≥5 kg, loss of 2.5–4.9 kg compared to loss or gain of <2.5 kg	T2D self-reported at endpoint.	Between age of 20 and beginning of follow-up: M: OR 0.91 (0.62-1.32) W: OR 0.70 (0.43-1.16) 5 y prior to follow-up: M: loss of ≥5 kg: OR 1.03 (0.77-1.39), loss of 2.5–4.9 kg: OR 1.02 (0.76-1.37) W: loss of ≥5 kg: OR 1.36 (0.99-1.87), loss of 2.5–4.9 kg: OR 1.16 (0.83-1.62)	Age, area, BL BMI, smoking, alcohol, PA, hypertension, family history of diabetes, coffee consumption. Additionally in analysis with weight change from 20 y to BL: weight change during 5-y survey, and in analysis with weight change during 5 y; weight change from 20 y to BL	
National Health and Nutrition Examination Survey (NHANES I and Epidemiologic Follow-up Study), 1971, US (Ford <i>et al.</i> 1997)	8 545/ 487	M and W aged ≥25	9	Calculated from measurements (10 y prior to follow-up)	Loss of ≥11 kg, loss of 5–<11 kg compared to loss or gain of <5 kg	T2D self-reported at 3 times after BL and drawn from registers (hospitalization and cause of death)	≥11 kg loss: HR 0.80 (0.46-1.40) 5–<11 kg loss: HR 1.13 (0.72-1.80)	Age, age <sup>a</sup> , sex, race, education, education <sup>a</sup> , smoking <sup>a</sup> , cholesterol <sup>a</sup> , cholesterol <sup>b</sup> , systolic blood pressure, systolic blood pressure <sup>a</sup> , antihypertensive medication, BL BMI, and alcohol <sup>a</sup> squared term	

Table 5 continues

Study, BL year, country	Exposure			Definition of weight loss	Outcome <sup>2</sup>	Results <sup>3</sup>	Control for confounding
	N/n	Study population	Follow-up (years)				
National Health and Nutrition Examination Survey (NHANES I and NHANES Epidemiologic Follow-up Study), 1971, US (Resnick <i>et al.</i> 2000)	1929/251	M and W with overweight (BMI $\geq 27.3$ for W and BMI $\geq 27.8$ for M) aged 25–74	10	Annual weight loss (kg/y) between BL and first follow up compared to no change	T2D self-reported at 2 time points after BL and drawn from registers (hospitalization and cause of death)	Annual loss (kg/y): -2.0: <b>OR 0.45 (0.34–0.60)</b> -1.5: <b>OR 0.55 (0.44–0.68)</b> -1.0: <b>OR 0.67 (0.58–0.78)</b> -0.5: <b>OR 0.82 (0.76–0.88)</b> -0.1: <b>OR 0.96 (0.95–0.97)</b>	Age, age <sup>a</sup> , BMI, sex, race, skinfold ratio and systolic blood pressure. <sup>a</sup> quadratic term
National Health and Nutrition Examination Survey (NHANES III and Continuous NHANES), 1988, US (Stokes <i>et al.</i> 2018)	21 554/1 877	M and W aged 40–74	10	Losing (BMI at age of $\geq 5$ $\geq 30$ kg/m <sup>2</sup> and BMI at beginning of follow-up $< 30$ kg/m <sup>2</sup> ) compared to stable obese and stable non-obese	T2D and age at diagnosis self-reported at endpoint	Compared to stable obese: <b>HR 0.33 (0.14–0.76)</b> Compared to stable non-obese: HR 1.47 (0.65–3.36)	Education, race, sex, family history of diabetes, and age
The National Health Insurance Service, National Sample Cohort, 2002, Korea (Kim <i>et al.</i> 2018)	51 405/2 749	M and W aged $\geq 20$	median 7	Loss of $> 9\%$ , loss of 9–6%, loss of 6–3% compared to loss or gain of $\leq 3\%$	T2D determined (laboratory measurements) biannually and drawn from registers	Those with BMI $< 25$ kg/m <sup>2</sup> Loss $> 9\%$ : <b>HR 0.51 (0.35–0.74)</b> loss 9–6%: <b>HR 0.66 (0.50–0.88)</b> loss 6–3%: <b>HR 0.81 (0.67–0.98)</b> Those with BMI $\geq 25$ kg/m <sup>2</sup> Loss $> 9\%$ : <b>HR 0.74 (0.59–0.95)</b> loss 9–6%: <b>HR 0.83 (0.68–1.03)</b> loss 6–3%: <b>HR 0.89 (0.77–1.04)</b>	Age, sex, current smoking, alcohol, exercise, income, hypertension, dyslipidaemia, and family history of diabetes
Nurses' Health Study, 1976, US (Colditz <i>et al.</i> 1995)	114 281/2204	W aged 30–55	14	Loss of $\geq 20.0$ kg, loss of 11.0–19.9 kg, loss of 5.0–10.9 kg compared to loss or gain of $\leq 4.9$ kg	T2D self-reported at 4 time points after BL	$\geq 20.0$ kg loss: <b>RR 0.13 (0.1–0.3)</b> 11.0–19.9 kg loss: <b>RR 0.23 (0.1–0.4)</b> 5.0–10.9 kg loss: <b>RR 0.54 (0.4–0.8)</b>	Age and BMI at age 18 y

*Table 5 continues*

Abbreviations: BL, baseline; BMI, body mass index; CHD, coronary heart disease; FEV<sub>1</sub>, forced expiratory volume; HR, hazard ratio; IDR, incidence density ratio; IWL, intentional weight loss; M, men; N, number of participants; n, number of cases; NR, not reported; PA, physical activity; PC, principal component; OR, odds ratio; RR, relative risk; SES, socio economic status; T1D, type 1 diabetes; T2D, type 2 diabetes; W, women; WLW, weight loss without information on intentionality; y, year.

<sup>1</sup> Whether weight loss is calculated using self-reported weight at two time points (calculated from self-reports), calculated using measured weight at two time points (calculated from measurements) or retrospectively self-reported (self-reported).

<sup>2</sup> In more detail, presented in Appendix of Substudy IV.

<sup>3</sup> Values in parentheses denote 95% confidence interval.

<sup>4</sup> Pre-existing health condition: history of heart disease, stroke, diabetes, or high blood pressure, or reported current severe shortness of breath or severe pain in chest.

<sup>5</sup> Symptoms including: pain in chest, shortness of breath, fatigue, loss of appetite, blood in stool, or blood in urine.

<sup>6</sup> Calculated from incidence % presented in study.

## 2.4 QUALITY OF DIET

Quality of diet refers to the healthfulness of a diet. A healthy diet helps protect from both malnutrition and the incidence of NCDs. At the population level, quality of diet can be evaluated against dietary recommendations. The main objectives of dietary recommendations are to improve population health, prevent NCDs and nutritional deficiencies, and generally provide guidance in forming a healthy diet (Nordic Council of Ministers 2014, National Nutrition Council 2014, US Department of Health and Human Services & US Department of Agriculture 2015).

Reduction of energy intake in relation to energy expenditure is essential in weight loss. In terms of quality of diet, however, the sources of energy nutrients are more meaningful than the amount of energy intake.

### 2.4.1 DIET INDICES

As individual nutrients and food items interact and have different effects on human physiology, epidemiological studies on the associations of individual nutrients and disease outcomes are prone to confounding by other components of the diet (Hu 2002). Thus, during the last decades, studies on dietary patterns have become more popular. To be able to study the associations of the whole diet, *a priori* and *a posteriori* methods have been utilized. In *a posteriori* methods, dietary patterns are statistically derived on the basis of existing data. In *a priori* method dietary patterns, i.e. indices or scores are theoretically based on knowledge on healthy diet. Several diet indices have been formulated to represent the intake of the most important factors of a healthy diet. Usually such indices are based on dietary recommendations or other healthy diets. Some of the indices are based on nutrients, some on foods or food groups and others on the combination of both (Waijers *et al.* 2007). Adherence to the indices is scored either by categorization of intakes (e.g. quartiles) or by cut-off values based on dietary recommendations or other knowledge on the issue. Different indices include e.g. the Healthy Eating Index (HEI) (Kennedy *et al.* 1995), the Alternate Healthy Eating Index (AHEI) (McCullough *et al.* 2002), AHEI-10 (a modification to the AHEI) (Chiuve *et al.* 2012), the Dietary Approaches to Stop Hypertension Score (DASH) (Fung *et al.* 2008), the Mediterranean Diet Score (Trichopoulou *et al.* 2003), the Baltic Sea Diet Score (BSDS) (Kanerva *et al.* 2014b), and the Healthy Food Intake Index (HFII) (Meinilä *et al.* 2016).

The strength of dietary indices is that they aim to mimic the actual combinations of foods and nutrients that people consume. Some weaknesses, however, also exist (Waijers *et al.* 2007). Even though the indices include the

most important components relative to their purpose, some other components may be missing, and a certain index may not be applicable to other uses. For example, an index aiming to evaluate adherence to dietary guidelines should differ from one targeted at evaluating the association between diet and the incidence of diabetes. Moreover, the prevailing dietary culture in each country or region sets frames for the suitability of an index.

The following chapter addresses the AHEI, a basis for the modified index used in this thesis, in more detail.

### ***AHEI***

The AHEI (McCullough *et al.* 2002) is a commonly used index. It was created on the basis of the Healthy Eating Index (HEI) (Kennedy *et al.* 1995), which is based on US dietary guidelines (Kennedy *et al.* 1996). The original AHEI consists of nine components: vegetables, fruits, nuts and soy protein, ratio of white to red meat, cereal fiber, trans fat, ratio of polyunsaturated fatty acids to saturated fatty acids, duration of multivitamin use, and alcohol. The components are scored, and the scores are summarized, with a higher score representing better adherence to a healthy diet.

## **2.4.2 QUALITY OF DIET, DIETING ATTEMPTS AND OBESITY**

Even though dieting has been associated with certain healthy dietary components or habits (Jeffery *et al.* 1991, French & Jeffery 1997, Neumark-Sztainer *et al.* 2000, Kant 2002, Andreyeva *et al.* 2010, Jeffery *et al.* 2013, Wolfson & Bleich 2015), as well as with some unhealthy habits (Keski-Rahkonen *et al.* 2005), no previous studies exist on the associations between dieting and overall diet quality defined by dietary indices.

In various but not all epidemiological studies, scoring high in different dietary indices (e.g. HEI, DASH Mediterranean Diet Score and BSDS) has been indicated to reduce risk of several NCDs and mortality, albeit that the magnitude of the reduction has been mostly modest (Kant 2004, Kant 2010, George *et al.* 2014, Kanerva *et al.* 2014a, Schwingshackl *et al.* 2018, Neuenschwander *et al.* 2019). Similarly, a high AHEI or AHEI-10 score has been associated with a reduced risk of CVDs, CHD, stroke, T2D, cancer, and mortality (McCullough *et al.* 2002, Akbaraly *et al.* 2011, Chiuve *et al.* 2012, George *et al.* 2014, Neuenschwander *et al.* 2019). In addition to NCD risk, a high quality diet has been shown to associate with a lower risk of obesity in most but not all comparisons (Kanerva *et al.* 2013b, Asghari *et al.* 2017, Kanerva *et al.* 2018). However, only few studies have looked into the associations between AHEI or AHEI-10 and obesity. Three studies have demonstrated an inverse association between AHEI or AHEI-10 and obesity or weight gain in diverse populations (The Black Women's Health Study (Boggs *et al.* 2013), the Health Professionals Follow-Up Study, the Nurses'

Health Study, and the Nurses' Health Study II (Fung *et al.* 2015), as well as among Chinese T2D patients (Cheung *et al.* 2018)). In contrast, in a study based on the Multi-Ethnic Study of Atherosclerosis the associations remained non-significant (Otto *et al.* 2015). Moreover, a longitudinal study on African American women by Boggs *et al.* (2013) showed an inverse association with obesity incidence only in women with normal weight, whereas in women with initial overweight, no association emerged.

Due to the inconsistencies in the associations between quality of diet and obesity, quality of diet and dieting, as well as dieting and obesity, the three factors should be studied simultaneously. Positioning dieting attempts as an effect modifying factor in the association between quality of diet and obesity is one way to examine such complex associations.

## 2.5 RATIONALE OF THE THESIS

Even though several lifestyle intervention studies with weight loss aiming to prevent T2D have been conducted, dieting at the population level, in spite of its frequency and possible contradictory consequences, is insufficiently studied. Several factors have been shown to associate with dieting, but a more complete picture of its determinants, however, is needed in order to be able to unbiasedly study how dieting predicts subsequent changes in obesity measures and the incidence of NCDs. Identifying individuals who report dieting and thus are potentially at an elevated risk of future weight gain is essential in light of preventive work against obesity and its co-morbidities.

In addition to the importance of the determination of the factors associated with dieting, the information on whether dieting per se affects the associations between other factors, for instance quality of diet and obesity, is needed in order to understand their complex associations. It is well known that individuals with obesity misreport their dietary intake. However, as dieters are also prone to misreporting (Livingstone & Black 2003, Maurer *et al.* 2006, Poslusna *et al.* 2009, Castro-Quezada *et al.* 2015) it is essential to take dieting into account when studying associations between dietary factors and the incidence of obesity or NCDs. Knowledge of dieting as an effect modifying factor, however, is nonexistent.

Even though the literature on dieting and its associations with weight gain is accumulating, most studies have been conducted in non-representative and somehow defined populations; only a few studies have used a follow-up time of more than 6 years, and in the majority of the studies, adjustment for lifestyle factors such as diet or PA has been insufficient. In addition, in a little more than half of the studies, the obesity measures have been self-reported, at least at some measurement point. Even though it has been suggested that self-reported weight correlates closely with measured weight (Spencer *et al.* 2002), individuals with obesity in particular seem to underestimate their

weight (Sherry *et al.* 2007, Maukonen *et al.* 2018) and thus, the same may hold true for individuals with dieting attempts. Consequently, such biased values in obesity measures in individuals with obesity and dieting attempts may distort the results. Moreover, no studies have comprehensively looked into whether various potential background factors modify the associations between dieting and subsequent weight gain. In addition, no studies using more than one measure of obesity seem to exist. Hence, epidemiological follow-up studies using representative adult population samples, long follow-ups, multiple obesity measures, and which look into potential effect-modifications are needed.

Evidence on successful weight reduction to decrease the risk of T2D in intervention settings is widely studied and solid (Peirson *et al.* 2014, Haw *et al.* 2017, LeBlanc *et al.* 2018). However, even though most of the epidemiological literature supports the hypothesis of IWL and WLW to reduce the risk of T2D, not all studies and not all comparisons in certain subgroups have found an association. Moreover, publications with IWL as an exposure are scarce and conducted in populations with overweight or obesity only. Hence, due to dieting being frequent at the population level in each BMI category, studies assessing its association with the incidence of T2D in general adult population samples are warranted. Moreover, there is a need for studies that comprehensively examine whether various sociodemographic, lifestyle-, somatic health-, and mental health-related factors modify this association.

### **3 AIMS**

The overall aims of this study were to evaluate whether self-report dieting during the previous year predicts long-term changes in BMI and WC, and incidence of T2D, which factors are cross-sectionally related to dieting, and whether dieting has a modifying role in the association between quality of diet and obesity. The more specific objectives were:

1. To study which sociodemographic, lifestyle, metabolic, somatic health, and mental health factors are associated with dieting attempts and IWL (I)
2. To study whether self-report dieting attempts modify the association between quality of diet and obesity (II)
3. To study whether self-report dieting attempts and previous changes in weight are associated with subsequent changes in BMI and WC during an 11-year follow-up (III)
4. To study whether IWL predicts T2D incidence during a 15-year follow-up (IV)

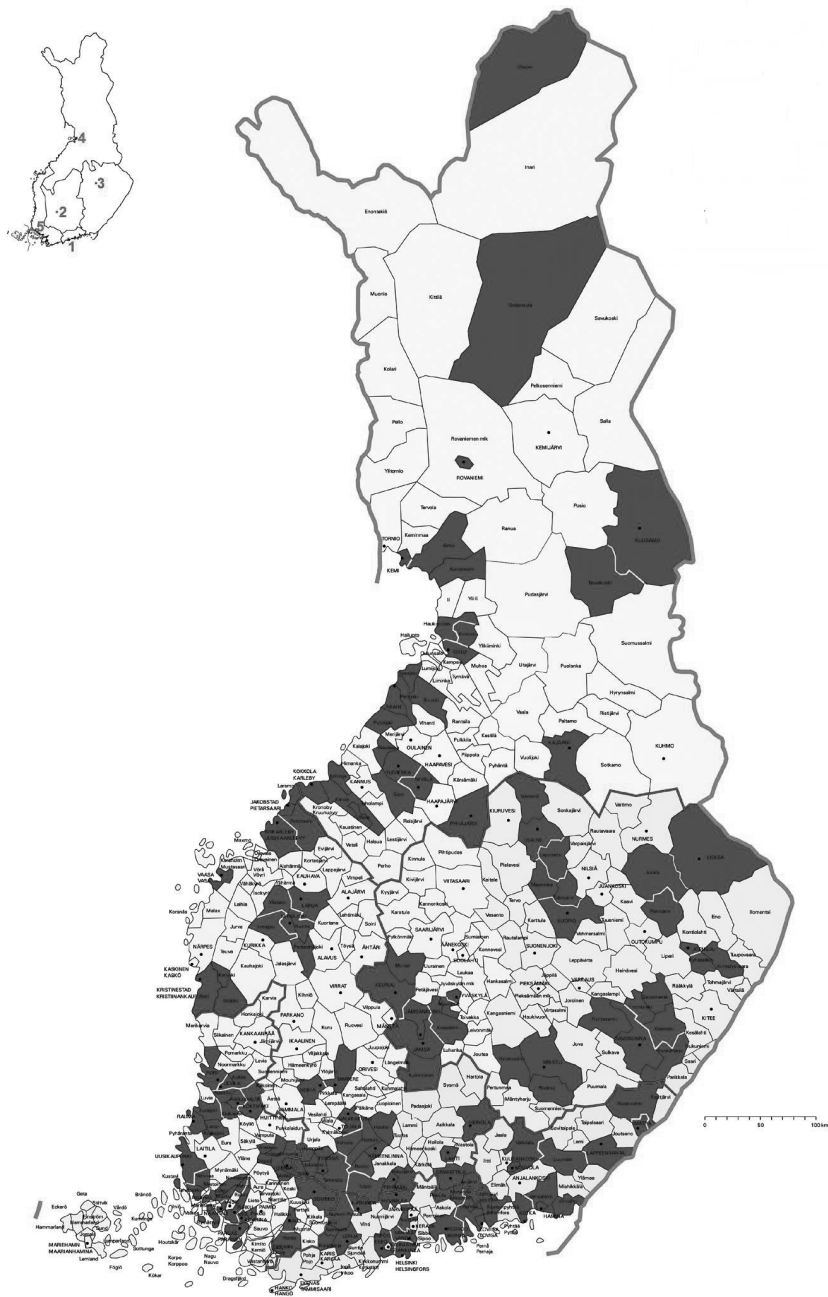


## 4 STUDY POPULATION AND METHODS

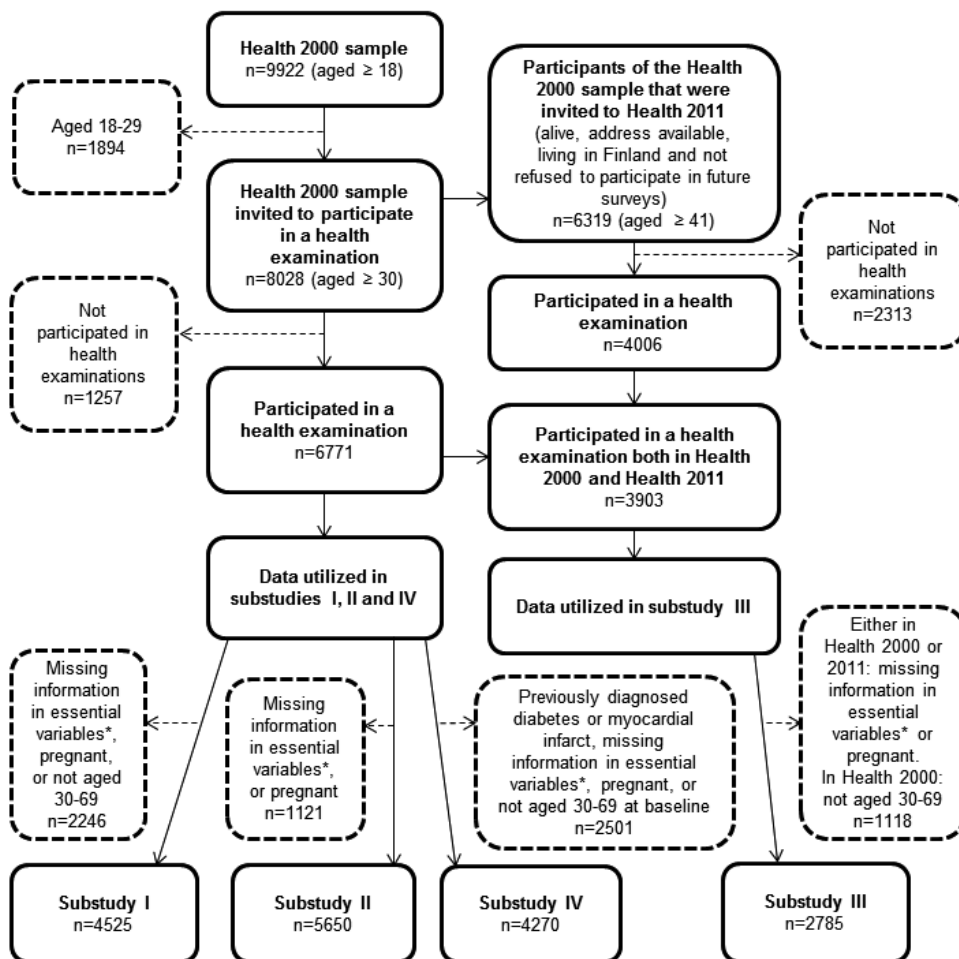
### 4.1 STUDY POPULATION

This thesis comprised data from the nationally representative Finnish Health 2000/2011 Survey (Health 2000/2011) (BRIF8901). The survey was conducted at the Finnish Institute for Health and Welfare (THL) (formerly National Institute for Health and Welfare (THL) and National Public Health Institute (KTL)) in co-operation with partners. The Health 2000 Survey (Health 2000) was carried out in 2000–2001 (Heistaro 2008), and its follow-up survey, the Health 2011 Survey (Health 2011), was carried out in 2011–2012 (Lundqvist & Mäki-Opas 2016). The main aim of the survey was to obtain information on the prevalence, causes, and distributions of public health problems and the most important NCDs. The aims also included producing information on the health, wellbeing, work ability and functional capacity of the Finnish adult population.

The drawing of the representative sample was planned in co-operation between Statistics Finland and KTL and was based on a two-stage stratified cluster sampling design. During the first stage, 80 out of 249 health care districts in mainland Finland were chosen, based on five university hospital regions. During the second stage, a random sample was drawn from within the 80 districts (Figure 3). The total original Health 2000 sample included 9922 individuals aged 18 and over. Of these, 8028 were 30 and older. Of this sample, 84% (n=6771) participated in a health examination (Figure 4). The majority of the participants attended the health examination at survey locations (e.g. health centers) (n=6354), and for those not able to participate there, a shorter home health examination was carried out (n=417).



**Figure 3** Survey areas of Health 2000: five university hospital regions and 80 districts. Source of figure: (Heistaro 2008)



**Figure 4** Flowchart of Health 2000/2011 and definitions of the data used in the substudies.  
\* The essential variables in each substudy are specified in Table 6.

All participants of Health 2000 were invited to also participate in Health 2011, excluding those who had died, were living abroad, had no contact details available, or had refused to participate in further studies. Of the adults aged 30 and older during Health 2000, this totaled 6319 individuals. Of the invited sample, 63% (n=4006) participated in a health examination (Figure 4). This thesis utilizes data on those who were 30 and older during Health 2000 and participated in the health examination. Figure 4 illustrates definition of the Health 2000/2011 data that were used in this thesis, and Table 6 presents the further inclusion criteria in more detail as well as the different study designs used.

**Table 6** Study designs and inclusion criteria of thesis substudies.

	Substudies			
	I	II	III	IV
<b>Study design</b>	Cross-sectional	Cross-sectional	Longitudinal follow-up, 11 years	Cohort, 15 years
<b>Data</b>	Health 2000	Health 2000	Health 2000 and Health 2011	Health 2000 and information from national health registers <sup>1</sup>
<b>Inclusion criteria</b>	No missing information on measured BMI, dieting attempts or IWL, not pregnant, aged 30–69	No missing information on BMI, dieting attempts or FFQ, not pregnant, aged ≥30	During both Health 2000 and 2011: no missing information on measured BMI, dieting attempts or previous weight change, not pregnant. During Health 2000: aged 30–69	No previously diagnosed diabetes or myocardial infarct, no missing information on IWL or the potential effect-modifying factors studied, not pregnant, aged 30–69 at baseline
<b>n</b>	4525	5650	2785	4270

Abbreviations: BMI, body mass index; FFQ, food frequency questionnaire; IWL, intentional weight loss.

<sup>1</sup> The Drug Reimbursement Register, The Finnish Hospital Discharge Register, The National Causes of Deaths Register

#### 4.1.1 ETHICAL QUESTIONS

The Health 2000/2011 was conducted in accordance with the guidelines laid down in the Declaration of Helsinki. Health 2000 was approved by the Ethical Committee for Research in Epidemiology and Public Health of the Hospital District of Helsinki and Uusimaa (HUS), and Health 2011 by the Coordinating Ethics Committee of the Hospital District of Helsinki and Uusimaa (HUS). Written informed consent was obtained from all survey participants during both study years.

## 4.2 METHODS

Health 2000/2011 were comprehensive health examination surveys that collected information on health, wellbeing and functional capacity via interviews, questionnaires, and health examinations, including measurements and blood samples. Information was also drawn from national health registers.

#### **4.2.1 DIETING ATTEMPTS, PREVIOUS CHANGES IN WEIGHT AND INTENTIONAL WEIGHT LOSS**

In Health 2000, information on dieting attempts, previous changes in weight and IWL was collected using a self-administered questionnaire. The following questions were used to form the variables used:

1. Have you tried to lose weight during the last 12 months? No/Yes
2. Have you lost weight during the last 12 months? No/Yes
  1. If yes: about how many kilograms?
3. Have you gained weight during the last 12 months? No/Yes

The first question was used as a “dieting attempts” variable (Substudies I, II, and III) so that the respondents were divided into those who had not tried to lose weight during the last 12 months (“non-dieters”) and those who had (“dieters”).

To create the “weight change during the previous year” variable (Substudy III) the second and third questions were combined to form a four-class variable containing the categories “no”, “lost”, “gained”, and “weight fluctuation”. Weight fluctuation was defined as having both lost and gained weight during the previous year.

The IWL variable (Substudies I and IV) consisted of Questions 1 and 2 and was formed as follows: Participants with both dieting attempts during the previous year and weight loss during the previous year were categorized as having IWL, and thus “yes”, while the others were categorized as “no”.

For additional and sensitivity analyses, several additional IWL variables considering the percentual amount of previously lost weight were formed using Question 2.1. (lost weight (kg)). Percentual weight loss was calculated as  $(\text{lost weight (kg)} / [\text{weight at baseline (kg)} + \text{lost weight (kg)}]) * 100$  and cut-off values of  $\geq 2\%$ ,  $\geq 5\%$ ,  $\geq 7\%$ ,  $\geq 10\%$ ,  $\geq 15\%$  and  $\geq 20\%$  were determined.

#### **4.2.2 OBESITY AND WEIGHT GAIN DURING FOLLOW-UP**

During the health examinations in Health 2000 and Health 2011, the participants’ height (cm), weight (kg), WC (cm), FM (kg), and FFM (kg) were measured by educated study nurses. The participants had been asked not to eat for at least four hours before the health examination. Height was measured using a wall-mounted stadiometer with the participant standing bare-footed and with their feet together, head up and back against the wall, and was recorded with a precision rate of 0.5 cm in Health 2000, and with a precision rate of 0.1 cm in Health 2011. Weight (kg), FM (kg), and FFM (kg) were measured while wearing light clothing but no shoes, using eight-polar bioimpedance devices (in Health 2000: InBody 3.0, Biospace, Soul, South-Korea; in Health 2011: Seca mBCA Model 5154 with Software Seca analytics 115, Seca, Hamburg, Germany) or, in the absence of bioimpedance devices,

weight was measured using a normal scale. The results were recorded with a precision rate of 0.1 kg at both time points. In Substudies I and III, only a measured weight was used. In Substudies II and IV, self-report weight, elicited in a questionnaire or an interview, was also utilized (n=276 in Substudy II; n=131 in Substudy IV).

BMI was calculated as weight (kg) divided by the square of height (m<sup>2</sup>). Normal weight was defined as BMI <25 kg/m<sup>2</sup>, overweight as BMI 25–<30 kg/m<sup>2</sup>, and obesity as BMI ≥30 kg/m<sup>2</sup> (World Health Organization 2000). In analyses using overweight as a dummy variable, overweight was defined as BMI ≥25 kg/m<sup>2</sup>. As the number of individuals with underweight (BMI <18.5 kg/m<sup>2</sup>) was small (e.g. in Substudy I n=29), those with underweight were combined with those with normal weight in each study. FMI and FFMI were calculated as FM or FFM divided by the square of height (m<sup>2</sup>), respectively.

WC (cm) was measured in standing position on the bare skin at the mid-point between the lowest rib bones and the high point of the iliac crest using a regular, flexible tailor's measuring tape during light expiration. The results were recorded with a precision rate of 0.5 cm in Health 2000 and with a precision rate of 0.1 cm in Health 2011.

Abdominal obesity was determined as WC ≥88 cm for women and ≥102 cm for men in Substudy III according to WHO's definition (World Health Organization 2011b), and as WC ≥80 cm for women and WC ≥94 cm for men in Substudy IV according to the IDF Metabolic syndrome (MetS) criteria (Alberti *et al.* 2009).

In Substudy III, the change in BMI and WC during an 11-year follow-up was determined by calculating [measure in 2011] – [measure in 2000].

For the new analyses conducted for this thesis, variables concerning ≥5% and ≥10% weight gain during the 11-year follow-up were created by first calculating percentual weight change during the follow up ([weight in 2011] – [weight in 2000]) / [weight in 2000] \* 100 and subsequently categorizing the dummy variables with the respective cut-off values.

#### **4.2.3 TYPE 2 DIABETES CASE ASCERTAINMENT AND FOLLOW-UP**

In Substudy IV, T2D incidence was used as an outcome. National health registers covering information on the use of medication, hospitalization, and cause of death were used to identify the incident T2D cases occurring during a 15-year follow-up. The Social Insurance Institution of Finland (Kela) keeps a register of patients receiving diabetes medication reimbursement. Individuals with diabetes and with a certified diagnosis from an attending physician and approval from Kela are entitled to reimbursements of the costs of the medication in Finland under the Sickness Insurance Act (Reunanen *et al.* 2000). Records from the Finnish Hospital Discharge Register (Heliövaara *et al.* 1984) and the National Causes of Deaths Register (Reunanen *et al.* 1983) were also utilized. Linkage of the study participants to these registers

was performed via the unique social security numbers identifying each Finnish citizen. Presence of any of the ICD-10 codes from E10 to E14 in the information from the registers was considered an incident T2D case. The follow-up period (days) was defined from the date of the Health 2000 examination to the date of incident T2D, death, or end of follow-up, whichever came first. A total of 417 individuals (241 men and 176 women) developed T2D during the 15-year follow-up.

#### **4.2.4 DIET**

The habitual diet of the participants was assessed using a validated self-administered, semi-quantitative, 128-item food frequency questionnaire (FFQ). The FFQ was initially designed in 1996, validated against food records, and updated regularly thereafter, also for use in Health 2000 (Männistö *et al.* 1996, Paalanen *et al.* 2006, Montonen *et al.* 2008, Kaartinen *et al.* 2012). The aim of the FFQ was to evaluate participants' habitual food use and dietary intake over the preceding 12 months. The FFQ comprised 128 foods and mixed dishes from 12 categories: "dairy products", "cereals", "spreads", "vegetables", "potatoes, rice and pasta", "meat", "fish", "chicken, turkey and eggs", "fruits and berries", "desserts", "sweets and snacks" and "beverages". Moreover, after each category, space for frequently consumed foods not listed in the food list of FFQ was provided. The portion sizes were pre-defined as commonly used units of the items at issue (e.g. teaspoon, piece, glass or slice), and participants were asked to compare their typical portion size to the pre-defined one and, to compensate the smaller or larger portion with frequency, if necessary. Nine frequency options ranging from "never or seldom" to "six or more times per day" were provided. Average daily intakes of foods, nutrients and energy were calculated using the National Food Composition Database (Fineli®), maintained by THL, and in-house software (Finessi) (Reinivuo *et al.* 2010). Energy intake (kcal/day) was both used as a continuous variable and categorized into sex-specific quintiles.

A validity study conducted in a subpopulation of 294 participants with a three-day food record as the reference method indicated that the nutrient intakes were generally higher in the FFQ method than in the food record method (Paalanen *et al.* 2006). This difference was accentuated among women, which possibly resulted from the fact that in the FFQ used, the portion sizes of the food items were not separately predefined for men and women. The energy-adjusted Pearson correlation coefficients for nutrient intakes varied between 0.14 and 0.66 in men and between 0.20 and 0.70 in women. The corresponding values for food consumption varied between 0.09 and 0.89 in men, and between 0.01 and 0.85 in women. The validity between these methods in the present study thus corresponds to the level of other studies (Paalanen *et al.* 2006). The repeatability of the FFQ was examined with a subpopulation of 180 participants 5–9 months after Health 2000

(Montonen *et al.* 2008). The reliability coefficients for nutrient intakes varied between 0.40 and 0.69, and for food groups between 0.32 and 0.68, indicating that the repeatability of the FFQ corresponds to the level of other studies.

The AHEI (McCullough *et al.* 2002) was chosen to represent quality of diet. The modified index (Jääskeläinen *et al.* 2013) was named the modified Alternate Healthy Eating Index (mAHEI) and it was formed by imitating the original AHEI as accurately as possible while allowing for the variables available in the data and the characteristics of Finnish dietary habits and culture (Table 7). Accordingly, rye, representing the use of foods high in fiber in the Finnish diet, was used instead of dietary fiber. This substitution was also due to the fact that the dietary fiber variable available in the data consisted of fiber both from cereal and plant products, in which case it would have overlapped with the “vegetables” and “fruit and berries” components. Use of multivitamins was excluded from the index as multivitamin use is not recommended for the general population in Finland. Moreover, in this thesis, alcohol was used as an individual measure and was thus excluded from the index to avoid overlap. How components of the mAHEI were formed is described in Table 7. For forming the index, each mAHEI component was divided into sex-specific quintiles. The quintiles were scored (1–5 points); the trans fat component received points in descending order, whereas the other components received points in ascending order. Finally, the scores of the components were summed (7–35 points) and the sum was divided into sex-specific quintiles. In the analyses, the higher quintile score represented higher quality of diet.

**Table 7** Contents of the original Alternate Healthy Eating Index (AHEI) and the modified Alternate Healthy Eating Index (mAHEI) used in the thesis (McCullough *et al.* 2002, Jääskeläinen *et al.* 2013).

Components of the original AHEI	Components of the mAHEI	Clarification of the components of the mAHEI
Vegetables	Vegetables	excluding potatoes and legumes
Fruits	Fruits and berries	fresh or frozen
Nuts and soy protein	Legumes and soy products	including nuts and seeds
Ratio of white and red meat	Ratio of white and red meat	white meat including poultry, fish and shellfish; red meat including beef, pork, lamb and processed meat
Cereal fiber	Rye	replacing cereal fiber
Trans fatty acids	Trans fatty acids	contrary to the other components, component received points in descending order
Ratio of PUFA and SFA	Ratio of PUFA and SFA	
Duration of multivitamin use	-	Excluded from the mAHEI
Alcohol	-	Excluded from the mAHEI

Abbreviations: AHEI, Alternate Healthy Eating Index; mAHEI, modified Alternate Healthy Eating Index; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.



#### 4.2.5 OTHER FACTORS

The characteristics and categorization of the factors used as covariates, determinants, effect modifying factors, or as part of the exclusion criteria are detailed in the original articles (I–IV). Table 8 briefly describes the details of the factors (sociodemographic, lifestyle, biomarkers, somatic health, mental health, and psychological) used as determinants (I) or as effect modifying factors (III, IV). The factors that are described in more detail in the preceding chapters are excluded from this table in spite of their use as determinants or effect modifying factors as well.

**Table 8** Characteristics of the background factors used in the substudies.

Variable	Sub-study	Data source	Determination and categorization
<b>Sociodemographic factors</b>			
Sex	I, III, IV	reg	men / women
Age (years)	I, III IV	reg reg	30–39 / 40–49 / 50–59 / 60–69 30–49 / 50–69
Education	I, III, IV	inter	low (did not graduate from upper secondary school or vocational school) / intermediate (graduated from upper secondary school or vocational school) / high (graduated from university or university of applied sciences)
Marital status	I, III	inter	married or cohabiting / divorced or separated / widowed / single
<b>Lifestyle-related factors</b>			
Leisure-time PA	I, III, IV	quest	low (not physically active) / moderate (regular engagement in light PA like walking or cycling) / regular vigorous training (exercising for $\geq 3$ hours/week or training for competitive sports)
Sitting time / day (hours)	I	quest	sex-specific tertiles of average of sitting during weekdays and weekend days
Alcohol consumption (g ethanol/week)	I, III, IV	quest	non-users / moderate users (1–199 g for males or 1–99 g for females) / heavy users ( $\geq 200$ g for males or $\geq 100$ g for females)
Smoking	I, III, IV	inter	never-smokers / former smokers / current smokers
Daily consumption of certain sugary products	I	quest	daily consumption of juices, lemonades, hot chocolate, toffee, liquorice, dried fruit, sweets, hard pastilles or candy without xylitol, chocolate, or filled biscuits: no / yes
Sleep duration (hours/night)	I, IV	quest	$\leq 6$ / 7–8 / $\geq 9$
FFMI (kg/m <sup>2</sup> )	I	health ex	FFM measured with bioimpedance (InBody 3.0, Biospace, Seoul, South-Korea); FFMI calculated as FFM (kg) divided by the square of height (m <sup>2</sup> ): sex-specific quintiles
<b>Biomarkers</b>			
Fs-triglycerides (mmol/l)	I	health ex	concentration determined from frozen ( $-70^{\circ}\text{C}$ ) serum samples (automated enzymatic method, Olympus system reagent, Germany): $<1.7$ / $\geq 1.7$
Fs-HDL (mmol/l)	I	health ex	concentration determined from frozen ( $-70^{\circ}\text{C}$ ) serum samples (enzymatic method, Roche Diagnostics, Mannheim, Germany): $\geq 1.03$ for men or $\geq 1.29$ for women / $<1.03$ for men or $<1.29$ for women
Fs-glucose (mmol/l)	I	health ex	concentration determined from frozen ( $-70^{\circ}\text{C}$ ) serum samples (hexokinase, Olympus System Reagent, Germany): $<5.6$ / $\geq 5.6$

Table 8 continues

Variable	Sub-study	Data source	Determination and categorization
<b>Somatic health</b>			
Elevated blood pressure	I, III, IV	health ex, inter	blood pressure measured twice at two-minute intervals using a standard mercury manometer, (Mercurio 300; Speidel & Keller, Jungingen, Germany); elevated blood pressure determined by systolic blood pressure of $\geq 130$ mmHg, or diastolic blood pressure of $\geq 85$ mmHg (the mean of two blood pressure measurements), or use of antihypertensive medication: no / yes
Metabolic syndrome (MetS)	IV	health ex, inter	definition by the International Diabetes Federation (IDF) (Alberti <i>et al.</i> 2009): no / yes
Severe MetS	IV	health ex, inter	fulfillment of each MetS precondition (according to IDF definition) (Alberti <i>et al.</i> 2009): no / yes
Diabetes	I, III	health ex, inter, quest, reg	case ascertainment (ICD-10, E11) by physicians on the basis of physical status, symptoms, self-reports, medical history, and information on diabetes medication reimbursement: no / yes
Knee or hip osteoarthritis	I, III, IV	health ex	determination by physicians on the basis of physical status, symptoms, and medical history: no / yes
Cancer	III	inter	self-report previous or current cancer: no / yes
Self-rated health	III	inter	good self-rated health (covering good or quite good perceived health) / poor self-rated health (covering average, quite poor or poor perceived health)
	IV	inter	good self-rated health (covering good, quite good or average perceived health) / poor self-rated health (covering quite poor or poor perceived health)
<b>Mental health and psychological factors</b>			
Poor mental health	IV	quest	General Health Questionnaire (GHQ) (Goldberg 1972) score $> 2$ : no / yes
Depressive disorder	I, III	inter	German Composite International Diagnostic Interview (M-CIDI) and DSM-IV diagnostics (Wittchen <i>et al.</i> 1998): no / yes
Anxiety disorder	I, III	inter	German Composite International Diagnostic Interview (M-CIDI) and DSM-IV diagnostics (Wittchen <i>et al.</i> 1998): no / yes
Sense of coherence (SOC)	I, III	quest	Antonovsky's SOC-13 scale (measuring a disposition to consider life as comprehensible, manageable and meaningful) (Antonovsky 1993): sex-specific quartiles from the mean of the questions
Burnout	III	quest	Maslach Burnout inventory (conducted only for those who had worked during the last 12 months) (Maslach <i>et al.</i> 1996): sex-specific quartiles from the weighted sum of the questions
Social support	I, III	quest	a self-assessment scale including questions concerning possibilities to obtain help and support from close ones: sex-specific tertiles
Concerns about one's appearance	I	quest	one item of the self-administered Beck Depression Inventory (BDI) (Beck <i>et al.</i> 1961): does not feel that one looks any worse than used to / concerns about one's appearance
Concerns about one's health	I	quest	one item of the self-administered Beck Depression Inventory (BDI) (Beck <i>et al.</i> 1961): not worried about one's health any more than usual / concerns about one's health

Abbreviations: DSM-IV, the Diagnostic and Statistical Manual of Mental Disorders, 4th revised edition; FFMI, fat free mass index; health ex, health examination; ICD, International Statistical Classification of Diseases and Related Health Problems; IDF, International Diabetes Federation; inter, interview; MetS, metabolic syndrome; quest, questionnaire; PA, physical activity; reg, national health registers; SOC, sense of coherence.

In Substudy I, initially, a larger number of factors were considered as determinants, but due to their nonsignificant associations with dieting variables found during domain-specific scrutiny (e.g. marital status, residential area, number of children, alcohol consumption, depressive disorder, anxiety disorder, and social support received from close ones) or their similarity and high correlation with other determinants (e.g. waist circumference and FMI with BMI), they were excluded from further scrutiny.

#### **4.2.6 STATISTICAL METHODS**

The linear regression model, the logistic regression model, and Cox's proportional hazards model were used, and the strength of association was estimated with model-adjusted means (Lee 1981), odds ratios (OR) and hazard ratios (HR) (Cox 1972), respectively. Significance was tested with F-test in the linear model and with likelihood ratio test in the logistic model and the Cox's model with significance level of  $P < 0.05$ . The statistical models, exposure variables, outcome variables, potential effect modifying variables, and the models for control for confounding factors used in the substudies are detailed in Table 9.

The factors that fulfilled the criteria for confounding were chosen separately in each substudy. Categorization of the confounding factors is described in the original articles I–IV.

Effect modification was studied by including an interaction term between the exposure variable in question and the potential effect modifying factor at issue in the model in question.

All analyses were performed using SAS 9.3 (SAS Institute Inc 2009).

**Table 9** Statistical characteristics of the substudies

	Substudies			
	I	II	III	IV
<b>Statistical model for the main analysis</b>	logistic	logistic	linear	Cox's
<b>Exposure</b>	Potential determinants listed in Table 8	quality of diet determined by the mAHEI, components of the mAHEI	self-report dieting attempts and changes in weight during the previous year	self-report IWL during the previous year
<b>Outcome</b>	self-report dieting attempts and IWL during the previous year	continuous BMI, prevalence of overweight and obesity	continuous change in BMI and WC during the 11-year follow-up	T2D incidence during a 15-year follow-up
<b>Confounding factors</b>				
<b>Model 1</b>	sex and age	sex and age	sex and age	sex and age
<b>Model 2</b>	sex, age, education, BMI, FFMI, leisure-time PA, sitting time, smoking, energy intake, mAHEI, daily consumption of certain sugary products, sleep duration, serum triglycerides, serum HDL cholesterol, blood pressure, T2D, osteoarthritis, SOC, concerns about one's appearance, and concerns about one's health	sex, age, education, marital status, geographical area, leisure time PA, alcohol consumption, smoking, and energy intake	sex, age, education, marital status, leisure-time PA, alcohol consumption, smoking, energy intake, mAHEI, and geographical area	age, sex, WC
<b>Model 3</b>		Model 3 used only when analyzing the associations between the components of mAHEI and overweight or obesity: variables of Model 2 and all components of the mAHEI (listed in Table 7)	variables of Model 2 and the baseline value of the outcome variable in question	age, sex, education, alcohol consumption, leisure time PA, smoking status, mAHEI, energy intake, BMI, and sleep duration
<b>Model 4</b>				variables of Model 3 and WC, blood pressure, serum glucose, serum triglycerides, and serum HDL cholesterol
<b>Effect modifying factors</b>	sex and BMI	dieting attempts	BMI and self-report changes in weight during the previous year; additional potential effect modifiers listed in Table 8	Potential effect modifiers listed in Table 8

Abbreviations: mAHEI, modified Alternate Healthy Eating Index; BMI, body mass index; FFMI, fat free mass index; HDL, high density lipoprotein; IWL, intentional weight loss; PA, physical activity; SOC, sense of coherence; T2D, type 2 diabetes; WC, waist circumference.

## 5 RESULTS

The Results section presents the main findings of Substudies I–IV and some unpublished results from additional analyses.

Table 10 shows the distributions of the selected baseline characteristics in the whole study population and by sex. Table 11 shows the corresponding distributions by two self-report dieting variables used in the substudies: dieting attempts and IWL during the previous year. Table 12 shows the same distributions by BMI categories at baseline. The distributions according to categories of weight change during the previous year (Substudy III) and the distributions of the rest of the background variables used can be found in the respective substudies' tables (Substudy I; Table 1, Substudy II; Tables 2 and 4, Substudy III; Table 1, Substudy IV; Table 1).

### 5.1 CHARACTERISTICS OF THE STUDY POPULATION

Nearly half of the study population consisted of men (Table 10). The mean age was 48 (SD 11) years. One third had a high education, two thirds were married, one fifth regularly took part in vigorous exercise, and one third were current smokers. The mean BMI was 26.8 (SD 4.68) kg/m<sup>2</sup>, a total of 32% of the participants had attempted to diet, and 13% had lost weight intentionally. Most of the factors differed according to sex (Table 10). Women were more often highly educated, but less often married or cohabiting with a spouse. Participating regularly in vigorous exercise was less common among women, but then again women consumed less alcohol and were less often smokers. Women had lower energy intake, and they slept longer. They also had lower BMI and less often MetS, but more often had concerns about their appearance and their health. Moreover, women had more commonly attempted to diet and lost weight intentionally.

Those with dieting attempts and with IWL were more commonly women and had a higher education than those who had not attempted to diet or had not lost weight intentionally (Table 11). Both measures were also associated with lower energy intake, higher diet quality, obesity, MetS, T2D, osteoarthritis, concerns about one's appearance, and concerns about one's health. Of the two dieting measures, only dieting attempts was related to being married or cohabiting, regular vigorous exercise, more seldom being a current smoker, and to a lower SOC score. Conversely, only IWL was associated with a lower age.

**Table 10** Unadjusted means (SD) or prevalences (%) of selected variables in the whole study population and by sex.

Baseline variable	n	The whole population (n=4525)	Men (n=2147)	Women (n=2378)	P for heterogeneity between sexes
<b>Sociodemographic factors</b>					
Sex (% male)	4525	47.4	-	-	-
Age (years)	4525	47.9 (10.7)	47.7 (10.6)	48.0 (10.8)	0.46
High education <sup>1</sup> (%)	4513	32.4	26.6	37.6	<.0001
Married/cohabiting (%)	4514	74.3	76.7	72.2	0.0005
<b>Lifestyle factors</b>					
Regular vigorous exercise <sup>2</sup> (%)	4498	19.1	22.2	16.2	<.0001
Sitting time (min/day)	4363	340 (169)	340 (176)	340 (162)	0.95
Alcohol consumption (g ethanol/week)	4502	84.7 (153)	135 (195)	38.8 (74.8)	<.0001
Current smoker (%)	4510	30.2	35.5	25.3	<.0001
Energy intake (kcal/day)	4221	2304 (791)	2408 (827)	2213 (746)	<.0001
mAHEI score <sup>3</sup> (range 7-35)	4221	21.2 (4.94)	21.1 (4.96)	21.3 (4.91)	0.29
Sleep duration (hours/day)	4224	7.46 (1.04)	7.33 (1.02)	7.57 (1.05)	<.0001
<b>Obesity measures</b>					
BMI (kg/m <sup>2</sup> )	4525	26.8 (4.68)	27.1 (4.12)	26.5 (5.11)	<.0001
WC (cm)	4519	92.1 (13.4)	97.5 (11.5)	87.2 (13.1)	<.0001
FFMI (fat free mass kg/m <sup>2</sup> )	4382	19.3 (2.41)	21.0 (1.90)	17.8 (1.70)	<.0001
<b>Somatic health</b>					
MetS (IDF definition) <sup>4</sup> (%)	4504	38.2	43.0	33.9	<.0001
Type 2 diabetes (%)	4525	3.54	3.87	3.24	0.25
Knee or hip osteoarthritis (%)	4467	4.88	5.21	4.58	0.33
<b>Psychological factors</b>					
SOC (mean score) <sup>5</sup> (range 1-7)	4314	5.48 (0.80)	5.46 (0.81)	5.50 (0.80)	0.08
Concerns about one's appearance (%)	4471	16.6	11.1	21.6	<.0001
Concerns about one's health (%)	4480	30.9	28.1	33.3	0.0002
<b>Exposure measures</b>					
Dieting attempts during the previous year (%)	4525	31.8	24.2	38.6	<.0001
IWL during the previous year <sup>6</sup> (%)	4525	13.0	10.4	15.3	<.0001

Abbreviations: BMI, body mass index; FFM, fat free mass index; IDF, International Diabetes Federation; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; MetS, metabolic syndrome; n, number of participants in respective category; SD, standard deviation; SOC, sense of coherence; WC, waist circumference.

<sup>1</sup> Graduated from university or university of applied sciences.

<sup>2</sup> Exercising for  $\geq 3$  hours/week or training for competitive sports.

<sup>3</sup> (McCullough *et al.* 2002)

<sup>4</sup> (Alberti *et al.* 2009)

<sup>5</sup> (Antonovsky 1993)

<sup>6</sup> Self-report dieting attempts and weight loss during the previous year

**Table 11** Sex- and age-adjusted means (SD) or prevalences (%) of selected variables by self-report dieting attempts and IWL during the previous year (n=4525).

Baseline variable	Dieting attempts during the previous year		P for heterogeneity	IWL during the previous year		P for heterogeneity
	No (n=3087)	Yes (n=1438)		No (n=3939)	Yes (n=586)	
<b>Sociodemographic factors</b>						
Sex <sup>1</sup> (% male)	52.7	36.1	<.0001	48.9	37.9	<.0001
Age <sup>2</sup> (years)	48.0 (10.9)	47.5 (10.3)	0.14	48.1 (10.8)	46.3 (10.1)	0.0002
High education (%)	31.0	35.2	0.004	31.7	37.0	0.009
Married/cohabiting (%)	73.4	76.3	0.04	74.1	75.7	0.43
<b>Lifestyle factors</b>						
Regular vigorous exercise (%)	18.1	21.0	0.02	18.8	20.7	0.27
Sitting time (min/day)	333 (169)	354 (168)	0.0002	337 (169)	361 (169)	0.002
Alcohol consumption (g/week)	83.7 (165)	86.8 (121)	0.50	84.8 (156)	83.6 (123)	0.84
Current smoker (%)	32.4	26.8	0.0001	30.8	29.7	0.59
Energy intake (kcal/day)	2330 (792)	2250 (784)	0.002	2316 (792)	2229 (778)	0.001
mAHEI score <sup>3</sup> (range 7-35)	20.7 (4.88)	22.3 (4.89)	<.0001	21.0 (4.90)	22.6 (4.98)	<.0001
Sleep duration (hours/day)	7.47 (1.03)	7.44 (1.06)	0.47	7.46 (1.05)	7.43 (0.99)	0.53
<b>Obesity measures</b>						
BMI (kg/m <sup>2</sup> )	25.7 (4.17)	29.1 (4.97)	<.0001	26.5 (4.51)	28.9 (5.35)	<.0001
WC (cm)	89.4 (12.6)	97.8 (14.0)	<.0001	91.3 (13.1)	97.3 (14.6)	<.0001
FFMI (fat free mass kg/m <sup>2</sup> )	19.0 (2.36)	20.1 (2.47)	<.0001	19.2 (2.39)	21.1 (2.52)	<.0001
<b>Somatic health</b>						
MetS (IDF definition) <sup>4</sup> (%)	32.2	51.2	<.0001	36.7	48.6	<.0001
Type 2 diabetes (%)	2.53	5.70	<.0001	3.13	6.24	0.0001
Knee or hip osteoarthritis (%)	4.21	6.32	0.002	4.49	7.51	0.001
<b>Psychological factors</b>						
SOC (mean score) <sup>5</sup> (range 1-7)	5.52 (0.79)	5.40 (0.83)	<.0001	5.48 (0.80)	5.47 (0.81)	0.78
Concerns about one's appearance (%)	14.6	20.9	<.0001	16.2	19.5	0.04
Concerns about one's health (%)	28.0	37.0	<.0001	30.3	34.4	0.05

Abbreviations: BMI, Body mass index; FFM, Fat free mass index; IDF, International Diabetes Federation; IWL, Intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; MetS, Metabolic syndrome; n, Number of participants in respective category; SD, Standard deviation; SOC, Sense of coherence; WC, Waist circumference.

Standard deviations are unadjusted.

<sup>1</sup> Adjusted for age.

<sup>2</sup> Adjusted for sex.

<sup>3</sup> (McCullough *et al.* 2002)

<sup>4</sup> (Alberti *et al.* 2009)

<sup>5</sup> (Antonovsky 1993)

A total of 39.9% of participants had overweight and 21.5% had obesity. The individuals with obesity were older, less educated, physically less active, and less commonly current smokers (Table 12). MetS, T2D, and osteoarthritis were statistically significantly more common in individuals with obesity. Moreover, obesity was related to having a lower SOC score, and having more often concerns about one's appearance, and concerns about one's health. The prevalence of individuals with dieting attempts and IWL

Results

grew in parallel with the BMI categories; of the participants with obesity 54.1% had attempted to diet and 20.4% had had IWL, while among participants with normal weight the corresponding numbers were 14.8% and 7.4%, respectively.

**Table 12** Sex and age adjusted means (SD) or prevalences (%) of selected variables by baseline BMI categories (n=4525).

Baseline variable	BMI (kg/m <sup>2</sup> ) at baseline			P for heterogeneity
	<25 (n=1746)	25–29.9 (n=1805)	≥30 (n=974)	
<b>Sociodemographic factors</b>				
Sex <sup>1</sup> (% male)	39.2	56.1	46.2	<.0001
Age <sup>2</sup> (years)	45.0 (10.4)	48.9 (10.6)	51.1 (10.3)	<.0001
High education (%)	36.1	31.7	26.7	<.0001
Married/cohabiting (%)	72.1	76.2	74.9	0.02
<b>Lifestyle factors</b>				
Regular vigorous exercise (%)	21.1	19.8	14.0	<.0001
Sitting time (min/day)	341 (174)	337 (162)	343 (172)	0.70
Alcohol consumption (g/week)	74.2 (130)	92.2 (168)	89.4 (157)	0.0008
Current smoker (%)	33.0	29.8	27.9	0.01
Energy intake (kcal/day)	2302 (745)	2298 (805)	2320 (843)	0.78
mAHEI score <sup>3</sup> (range 7-35)	21.0 (4.92)	21.3 (4.87)	21.4 (5.04)	0.11
Sleep duration (hours/day)	7.45 (1.01)	7.45 (1.00)	7.50 (1.17)	0.48
<b>Obesity measures</b>				
WC (cm)	81.8 (7.94)	93.4 (7.74)	108 (10.3)	<.0001
FFMI (fat free mass kg/m <sup>2</sup> )	17.8 (1.71)	19.6 (1.88)	21.6 (2.18)	<.0001
<b>Somatic health</b>				
MetS (IDF definition) <sup>4</sup> (%)	11.1	46.4	71.6	<.0001
Type 2 diabetes (%)	2.15	2.86	7.27	<.0001
Knee or hip osteoarthritis (%)	2.76	4.54	9.35	<.0001
<b>Psychological factors</b>				
SOC (mean score) <sup>5</sup> (range 1–7)	5.52 (0.78)	5.50 (0.79)	5.36 (0.86)	<.0001
Concerns about one's appearance (%)	14.5	16.3	20.8	0.0002
Concerns about one's health (%)	25.3	32.4	37.9	<.0001
<b>Exposure measures</b>				
Dieting attempts during the previous year (%)	14.8	36.2	54.1	<.0001
IWL during the previous year (%)	7.37	14.3	20.4	<.0001

Abbreviations: BMI, body mass index; FFMI, fat free mass index; IDF, International Diabetes Federation; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; MetS, metabolic syndrome; n, number of participants in respective category; SD, standard deviation; SOC, sense of coherence; WC, waist circumference.

Standard deviations are unadjusted.

<sup>1</sup> Adjusted for age.

<sup>2</sup> Adjusted for sex.

<sup>3</sup> (McCullough *et al.* 2002)

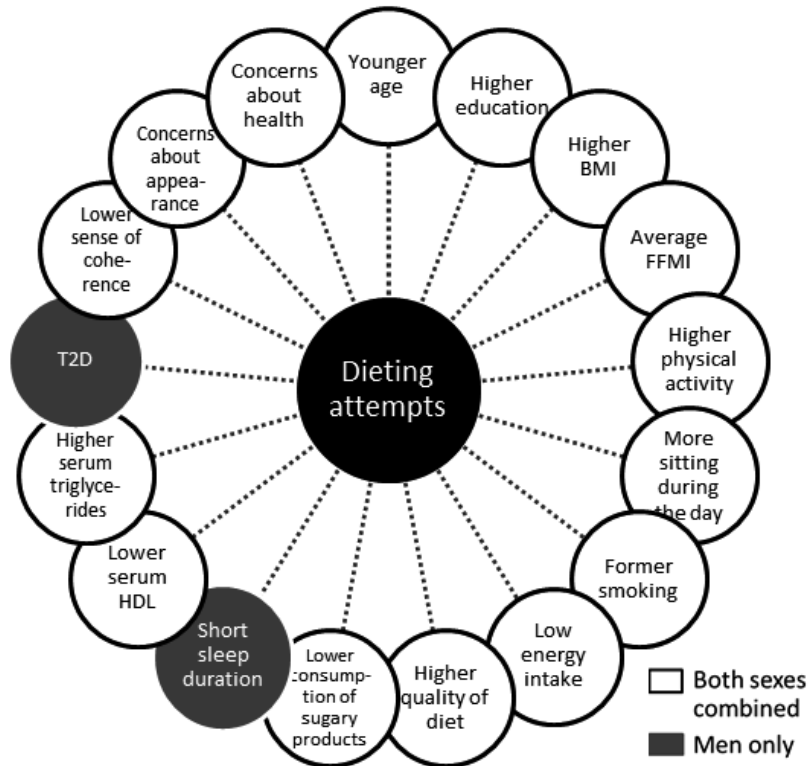
<sup>4</sup> (Alberti *et al.* 2009)

<sup>5</sup> (Antonovsky 1993)



## 5.2 WHO ARE SELF-REPORT DIETERS?

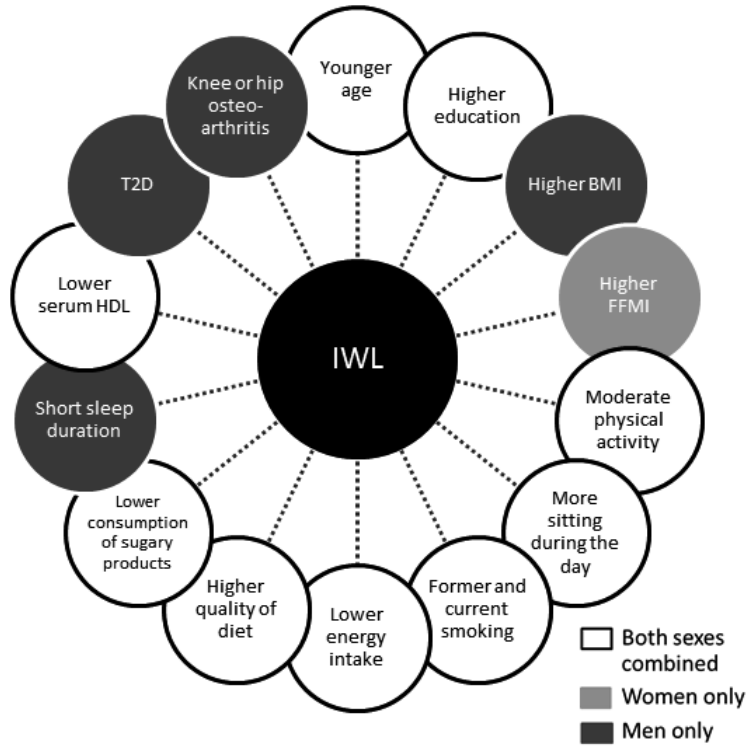
Of women, 39% reported dieting attempts and 15% IWL during the previous year, while the rates among men were 24% and 10%, respectively (Table 10). Dieting attempts and IWL were associated with several sociodemographic, lifestyle-, somatic health-, and mental health-related factors (Figures 5 & 6, Table 13).



**Figure 5** Factors significantly associated with dieting attempts after multivariate adjustment\* among both sexes combined or among men only.

Abbreviations: BMI, body mass index; FFMI, fat free mass index; HDL, high density lipoprotein; mAHEI, modified Alternate Healthy Eating Index; PA, physical activity; SOC, sense of coherence; T2D, type 2 diabetes.

\* Adjusted for sex, age, education, BMI, FFMI, leisure-time PA, sitting time, smoking, energy intake, mAHEI, daily consumption of certain sugary products, sleep duration, serum triglycerides, serum HDL cholesterol, blood pressure, T2D, osteoarthritis, SOC, concerns about one's appearance, and concerns about one's health.



**Figure 6** Factors significantly associated with IWL after multivariate adjusting\* among both sexes combined, among women only, or among men only.

Abbreviations: BMI, body mass index; FFMI, fat free mass index; HDL, high density lipoprotein; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; PA, physical activity; SOC, sense of coherence; T2D, type 2 diabetes.

\* Adjusted for sex, age, education, BMI, FFMI, leisure-time PA, sitting time, smoking, energy intake, mAHEI, daily consumption of certain sugary products, sleep duration, serum triglycerides, serum HDL cholesterol, blood pressure, T2D, osteoarthritis, SOC, concerns about one's appearance, and concerns about one's health.

**Table 13** *Multivariate adjusted<sup>1</sup> self-report dieting attempts and IWL during the previous year by potential determinants (Odds ratios (OR) and 95% confidence intervals).*

Determinants	n	Dieting attempts		IWL	
		OR	95% CI	OR	95% CI
<b>Sociodemographic factors</b>					
Sex	3749				
Women	1969	1		1	
Men	1780	<b>0.42</b>	<b>0.35-0.50</b>	<b>0.62</b>	<b>0.50-0.78</b>
Age (years)	3749				
30-39	1055	1		1	
40-49	1119	0.83	0.68-1.02	<b>0.75</b>	<b>0.58-0.97</b>
50-59	970	<b>0.58</b>	<b>0.46-0.72</b>	<b>0.62</b>	<b>0.46-0.83</b>
60-69	605	<b>0.45</b>	<b>0.34-0.60</b> *	<b>0.42</b>	<b>0.29-0.62</b> *
Education (%)	3749				
Low	1108	1		1	
Intermediate	1366	1.10	0.90-1.35	1.14	0.87-1.50
High	1275	<b>1.39</b>	<b>1.12-1.72</b>	<b>1.40</b>	<b>1.07-1.85</b>
<b>Lifestyle-related factors</b>					
BMI (kg/m <sup>2</sup> )	3749				
<25	1455	1		1	
25-29.9	1517	<b>2.52</b>	<b>1.99-3.19</b>	1.30	0.95-1.78
≥30	777	<b>4.24</b>	<b>3.03-5.93</b> *	1.49	0.97-2.30 *
FFMI quintiles <sup>2</sup> (fat free mass kg/m <sup>2</sup> )	3749				
1st (lowest)	752	1		1	
2nd	745	<b>1.54</b>	<b>1.16-2.05</b>	1.45	0.98-2.15
3rd	754	<b>1.55</b>	<b>1.16-2.08</b>	<b>1.62</b>	<b>1.10-2.40</b>
4th	753	<b>1.64</b>	<b>1.18-2.26</b>	<b>1.95</b>	<b>1.29-2.95</b>
5th	745	1.08	0.72-1.62 *	1.59	0.96-2.64
Leisure-time physical activity	3749				
Low	900	1		1	
Moderate	2097	<b>1.40</b>	<b>1.15-1.70</b>	<b>1.35</b>	<b>1.04-1.74</b>
Regular vigorous training	752	<b>1.65</b>	<b>1.29-2.09</b>	1.31	0.96-1.79
Sitting time tertiles <sup>3</sup> (min/day)	3749				
1st (lowest)	1211	1		1	
2nd	1225	1.06	0.88-1.28	1.18	0.92-1.52
3rd	1313	<b>1.30</b>	<b>1.08-1.57</b>	<b>1.38</b>	<b>1.08-1.77</b>
Smoking	3749				
Never	1860	1		1	
Former smoker	788	<b>1.28</b>	<b>1.05-1.56</b>	<b>1.45</b>	<b>1.13-1.88</b>
Current smoker	1101	0.88	0.73-1.07	1.25	0.98-1.59
Energy intake quintiles <sup>4</sup> (kcal/day)	3749				
1st (lowest)	735	1		1	
2nd	755	0.86	0.68-1.09	0.81	0.60-1.10
3rd	743	<b>0.74</b>	<b>0.58-0.94</b>	0.78	0.57-1.06
4th	758	<b>0.76</b>	<b>0.60-0.96</b>	0.87	0.64-1.18
5th	758	<b>0.60</b>	<b>0.47-0.77</b>	<b>0.66</b>	<b>0.48-0.91</b>
mAHEI quintiles <sup>5</sup>	3749				
1st (lowest)	653	1		1	
2nd	742	<b>1.37</b>	<b>1.05-1.78</b>	1.35	0.94-1.94
3rd	870	<b>1.45</b>	<b>1.13-1.88</b>	1.36	0.96-1.92
4th	704	<b>1.70</b>	<b>1.31-2.23</b>	<b>1.59</b>	<b>1.11-2.27</b>
5th	780	<b>2.26</b>	<b>1.74-2.95</b>	<b>2.21</b>	<b>1.56-3.12</b>
Daily consumption of certain sugary products <sup>6</sup>	3749				
No	2925	1		1	
Yes	824	<b>0.73</b>	<b>0.60-0.89</b>	<b>0.73</b>	<b>0.56-0.96</b>

Table 13 continues

Determinants	n	Dieting attempts		IWL	
		OR	95% CI	OR	95% CI
Sleep duration (hours/day)	3749				
≤6	491	1		1	
7–8	2866	0.84	0.67-1.05	0.93	0.69-1.25
≥9	392	0.78	0.57-1.07 *	0.94	0.63-1.41 *
<b>Somatic health</b>					
Fs-triglycerides (mmol/l)	3749				
<1.7	2584	1		1	
≥1.7	1165	<b>1.20</b>	<b>1.00-1.44</b>	0.85	0.67-1.07
Fs-HDL (mmol/l)	3749				
≥1.03 in men or ≥1.29 in women	2478	1		1	
<1.03 in men or <1.29 in women	1271	1.18	0.99-1.40	<b>1.33</b>	<b>1.07-1.65</b>
Elevated blood pressure <sup>7</sup>	3749				
No	1691	1		1	
Yes	2058	0.97	0.82-1.16	1.02	0.81-1.27
T2D	3749				
No	3633	1		1	
Yes	116	1.43	0.94-2.19 *	1.60	0.99-2.58
Knee or hip osteoarthritis	3749				
No	3582	1		1	
Yes	167	0.92	0.63-1.34	1.53	0.99-2.38 *
<b>Psychological factors</b>					
SOC quartiles <sup>8</sup>	3749				
1st (highest)	1003	1		1	
2nd	1007	0.99	0.80-1.22	1.00	0.76-1.30
3rd	923	1.07	0.86-1.33	0.83	0.62-1.10
4th (lowest)	816	<b>1.45</b>	<b>1.15-1.82</b>	1.06	0.79-1.42
Concerns about one's appearance	3749				
Does not feel that one looks any worse than used to	3144	1		1	
Concerns about one's appearance	605	<b>1.27</b>	<b>1.03-1.58</b>	1.10	0.84-1.44
Concerns about one's health	3749				
Not worried about one's health any more than usual	2617	1		1	
Concerns about one's health	1132	<b>1.21</b>	<b>1.02-1.43</b>	1.00	0.80-1.25

Abbreviations: BMI, body mass index; CI, confidence interval; FFMI, fat free mass index; Fs-, fasting serum; HDL, high density lipoprotein; mAHEI, modified Alternate Healthy Eating Index; n, number of participants in respective category; OR, odds ratio; SOC, sense of coherence; T2D, type 2 diabetes.

Bolded results are statistically significant.

\* Statistically significant (P<0.05) sex-interaction (see Table 2 and Table 3 in Substudy I)

<sup>1</sup> Model 2 adjusted for all the other variables in the table: sex, age, education, BMI, FFMI, leisure-time physical activity, sitting time, smoking, energy intake, mAHEI, daily consumption of certain sugary products, sleep duration, fs-triglycerides, fs-HDL, elevated blood pressure, T2D, osteoarthritis, SOC, concerns about appearance, concerns about health.

<sup>2</sup> FFMI quintile ranges (fat free mass kg/m<sup>2</sup>): 1st 14.3–19.5 for men, 11.1–16.3 for women; 2nd 19.6–20.5 for men, 16.4–17.2 for women; 3rd 20.6–21.4 for men, 17.3–18.1 for women; 4th 21.5–22.6 for men, 18.2–19.2 for women; 5th 22.7–29.4 for men, 19.3–24.2 for women.

<sup>3</sup> Sitting time tertile ranges (min): 1st 0–236 for men, 0–240 for women; 2nd 237–381 for men, 241–390 for women; 3rd 382–1200 for men, 391–1311 for women.

<sup>4</sup> Energy intake quintile ranges (kcal): 1st 688–1745 for men, 593–1613 for women; 2nd 1746–2097 for men, 1614–1942 for women; 3rd 2098–2467 for men, 1943–2285 for women; 4th 2468–3013 for men, 2286–2692 for women; 5th 3014–6413 for men, 2693–6495 for women.

<sup>5</sup> mAHEI quintile ranges (points): 1st 7–16 for men, 7–16 for women; 2nd 17–19 for men, 17–19 for women; 3rd 20–22 for men, 20–22 for women; 4th 23–25 for men, 23–25 for women; 5th 26–34 for men, 26–35 for women.

<sup>6</sup> Daily consumption of juices, lemonades, hot chocolate, toffee, liquorice, dried fruit, sweets, hard pastilles, or candy without xylitol, chocolate, or filled biscuits.

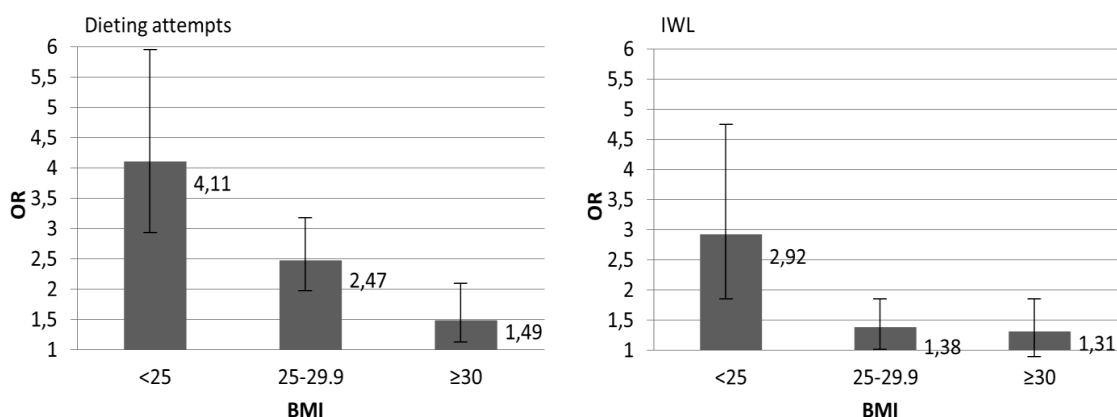
<sup>7</sup> Systolic blood pressure ≥130 mmHg, or diastolic blood pressure ≥85 mmHg, or use of antihypertensive medication.

<sup>8</sup> SOC quartile ranges (score): 1st 1.50–5.00 for men, 2.25–4.83 for women; 2nd 5.01–5.50 for men, 4.84–5.50 for women; 3rd 5.51–6.00 for men, 5.51–6.00 for women; 4th 6.01–7.00 for men, 6.01–7.00 for women.

## 5.2.1 SOCIODEMOGRAPHIC FACTORS

After multivariate adjustments (Model 2), dieting attempts and IWL were substantially more common among women than among men (Table 13). Dieting attempts were inversely associated with age; dieting being less common among individuals aged 60–69 than among individuals aged 30–39. Also IWL had a parallel gradient with younger age. Of the sociodemographic factors, high education associated with both dieting attempts and IWL in comparison to low education. No associations emerged with marital status, the number of children or residential area (data not shown).

Further interaction analyses by BMI revealed a significant interaction with sex, with both dieting attempts (P for BMI interaction < .0001) and IWL (P-value for BMI interaction = 0.01) as an outcome (Substudy I; Table S1). Women with normal weight had fourfold odds of dieting attempts and threefold odds of IWL compared to men (Figure 7). In contrast, in individuals with obesity no such notable differences emerged.



**Figure 7** Multivariate adjusted\* self-report dieting attempts and IWL of women vs. men (ref.) by BMI ( $\text{kg}/\text{m}^2$ ) categories (Odds ratios (OR) and 95% confidence intervals).

Abbreviations: BMI, body mass index; FFMI, fat free mass index; HDL, high density lipoprotein; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; PA, physical activity; OR, odds ratio; SOC, sense of coherence; T2D, type 2 diabetes.

\* Adjusted for sex, age, education, BMI, FFMI, leisure-time PA, sitting time, smoking, energy intake, mAHEI, daily consumption of certain sugary products, sleep duration, serum triglycerides, serum HDL cholesterol, blood pressure, T2D, osteoarthritis, SOC, concerns about one's appearance, and concerns about one's health.

## **5.2.2 LIFESTYLE-RELATED FACTORS**

Dieting attempts and BMI had a notable gradient in both sexes, but especially in men (P-value for sex interaction  $<0.001$ ) (Table 13, Substudy I; Table 2). IWL showed a strong direct association with BMI in men only, whereas in women, no association emerged (Substudy I; Table 3). Dieting attempts were more common among the individuals within the three middle quintiles of the FFMI range than among the individuals in the lowest quintile (Table 13), while IWL was unusual among individuals with the lowest FFMI quintile, and was pronounced among women with the highest FFMI (Substudy I; Table 3).

Both dieting attempts and IWL were more common among individuals who were physically more active during their leisure time than among individuals with low physical activity (Table 13). Conversely, those who sat more during the day had more often had dieting attempts and IWL than those who sat the least. Former smokers had more often attempted to diet than never-smokers, whereas both former and current smoking were related to IWL.

Dieting attempts and IWL were related to having a higher quality diet, lower energy intake and less frequent (than daily) consumption of certain sugary products (Table 13). Moreover, short sleep duration was related to both dieting attempts and IWL in men only (Substudy I; Tables 2 and 3). No association between alcohol consumption and dieting attempts or IWL emerged (data not shown).

In the interaction analyses by BMI and having dieting attempts as an outcome, smoking and BMI showed a tentative interaction (P-value for BMI interaction =0.12), with ex-smokers with obesity having more commonly attempted to diet than never-smokers with obesity. On the contrary, no differences emerged within the other BMI groups (Substudy 1; Table S1). Moreover, among individuals with normal weight or overweight, those with the highest energy intake had attempted to diet less often than those with low energy intake, whereas among individuals with obesity, no such differences existed (P-value for BMI interaction =0.02). In addition, a difference between those who did and did not consume certain sugary products daily only emerged among individuals with overweight. In this subgroup, those who consumed sugary products daily had attempted to diet less often than those with less frequent consumption (P-value for BMI interaction =0.002).

With IWL as an outcome, no significant interactions emerged between BMI and the lifestyle-related factors studied (Substudy I; Table S1). A tentative interaction, however, emerged between BMI and energy intake (P=0.15), according to which low energy intake was only associated with IWL among individuals with normal weight.

### 5.2.3 SOMATIC HEALTH

Individuals with unfavorable values in most of the MetS components, with diagnosed T2D or with knee or hip osteoarthritis had more often attempted to diet, after adjusting for age and sex (Substudy 1; Table 2). Inclusion of all the variables in the model (Model 2), however, attenuated part of the associations into nonsignificant, and only having pathological values in triglycerides remained a significant determinant of dieting attempts in the whole population, mostly due to the accentuated association in women (Table 13, Substudy I; Table 2). In contrast, the association between T2D and dieting attempts remained significant in men only (P-value for sex-interaction =0.02) (Substudy I; Table 2).

The same somatic health factors were associated with IWL after adjusting for sex and age (Substudy 1; Table 3), but in the multivariate model (Model 2), only HDL remained significant in the whole population, principally due to a stronger association in women (Table 13, Substudy I; Table 3). In the multivariate model, T2D and knee or hip osteoarthritis diagnoses were associated with IWL in men only (Substudy I; Table 3).

Of the somatic health indicators, only knee or hip osteoarthritis showed a significant interaction with BMI when having both dieting attempts (P-value for BMI interaction =0.03) and IWL (P-value for BMI interaction =0.01) as an outcome (Substudy I; Table S1). Accordingly, only in individuals with overweight osteoarthritis was directly associated with dieting attempts and IWL.

### 5.2.4 MENTAL HEALTH AND PSYCHOLOGICAL FACTORS

Individuals with low SOC scores had more often attempted to diet, whereas with IWL, SOC showed no association (Table 13). In addition, dieting attempts were related to concerns about one's appearance and one's health. Of the other indicators of mental health and psychological factors considered, neither diagnosed depressive or anxiety disorders nor social support received from close ones were significantly related to dieting attempts (data not shown). None of the mental health or psychological factors were significantly related to IWL.

Generally, BMI and mental health or psychological factors showed no significant interactions when having dieting attempts as an outcome (Substudy 1; Table S1). An indicative interaction, however, appeared between BMI and concerns about one's appearance (P-value for BMI interaction =0.06). Such concerns were related to dieting attempts among individuals with normal weight or overweight only, while among individuals with obesity no association existed. Concordantly, BMI and concerns about one's appearance showed a significant interaction with IWL as an outcome, according to which the concerns were only associated with IWL among those with normal weight (P-value for BMI interaction =0.02).

### **5.3 ROLE OF DIETING ATTEMPTS IN THE ASSOCIATION BETWEEN QUALITY OF DIET AND OBESITY**

Dieters had higher BMI (29.1 vs. 25.7 kg/m<sup>2</sup>) and mAHEI scores (22.3 vs. 20.7) than non-dieters (Table 11, Substudy II; Table 4). A higher mAHEI score was associated with higher education, being married or cohabiting, non-smoking, being physically more active, and having a higher energy intake (Substudy II; Table 3). The distributions of background variables according to categories of dieting attempts and BMI are presented in Table 11 and Table 12.

Those with a higher mAHEI score were at a statistically significantly higher risk of overweight and obesity after adjusting for the multivariate model (Table 14). In interaction analyses between mAHEI and dieting attempts in the prediction of overweight and obesity, non-significant, yet indicative, interactions emerged (P-value for interaction =0.29 for overweight; P-value for interaction =0.16 for obesity). Among individuals with no dieting attempts, quality of diet was non-related to the risk of overweight or obesity, whereas among those with dieting attempts, a higher risk of overweight and obesity could be seen in the highest mAHEI quintile compared to the lowest quintile.

A study on the interactions between mAHEI components and dieting attempts indicated that a high intake of trans fatty acids was related to a lower risk of overweight and obesity in non-dieters (Substudy II; Table 6). Furthermore, in the same subgroup, the highest consumption of fruit and berries was associated with a tentatively lower risk of obesity compared to those with the lowest consumption. In dieters, a higher risk of both overweight and obesity could be seen among those with the highest intake of vegetables compared to those with the lowest intake.

Further interaction analyses compared dieters and non-dieters by mAHEI quintiles in terms of the risk of overweight and obesity. In each quintile, the odds of being overweight or obese were multifold in dieters in comparison to non-dieters (Substudy II; Figure 2). The differences were most notable, however, within the highest mAHEI quintile.



**Table 14** Multivariate adjusted<sup>1</sup> odds ratios (OR) and their 95% confidence intervals (CI) of being overweight (BMI  $\geq 25$  kg/m<sup>2</sup>) and obese (BMI  $\geq 30$  kg/m<sup>2</sup>) between quintiles of sex-specific mAHEI in the whole population and by dieting attempts during the previous year.

mAHEI quintiles <sup>2</sup>	Overweight				Obesity			
	n	N	OR	95% CI	n	N	OR	95% CI
<b>The whole sample</b>	3625	5779			1298	5779		
1	664	1109	1		240	1109	1	
2	705	1145	1.16	0.98-1.39	231	1145	1.01	0.82-1.25
3	803	1309	1.21	1.02-1.43	303	1309	1.29	1.06-1.58
4	690	1069	1.41	1.17-1.70	241	1069	1.28	1.04-1.58
5	763	1147	1.51	1.26-1.82	283	1147	1.48	1.20-1.82
<b>P for trend</b>			<.00001					
<b>No dieting attempts<sup>3</sup></b>	2139	3896			612	3896		
1	470	841	1		148	841	1	
2	449	812	1.08	0.88-1.32	125	812	0.98	0.75-1.28
3	461	881	1.01	0.82-1.23	138	881	1.07	0.82-1.40
4	391	685	1.21	0.97-1.51	111	685	1.15	0.87-1.52
5	368	677	1.03	0.83-1.29	90	677	0.92	0.69-1.24
<b>P for trend</b>			0.49					
<b>Dieting attempts<sup>3</sup></b>	1357	1674			636	1674		
1	168	220	1		80	220	1	
2	234	293	1.20	0.78-1.86	100	293	0.93	0.64-1.37
3	309	384	1.27	0.84-1.93	152	384	1.27	0.89-1.81
4	273	336	1.34	0.87-2.07	120	336	1.06	0.73-1.54
5	373	441	1.67	1.10-2.55	184	441	1.40	0.98-1.98
<b>P for trend</b>			0.13				0.10	
<b>P for interaction of mAHEI and dieting</b>			0.29				0.16	

Abbreviations: BMI, Body mass index; CI, confidence interval; mAHEI, modified Alternate Healthy Eating Index; n, Number of cases in respective category; N, Number of subjects in respective category; OR, Relative Odds; PUFA, Polyunsaturated fatty acids; SAFA, Saturated fatty acids.

<sup>a</sup> mAHEI quintile ranges: 1 (7-16, lowest), 2 (17-19), 3 (20-22), 4 (23-25), 5 (26-35, highest).

<sup>b</sup> Multivariate model: Adjusted for age, sex, education, marital status, geographical area, leisure-time physical activity, alcohol consumption, smoking, and energy intake.

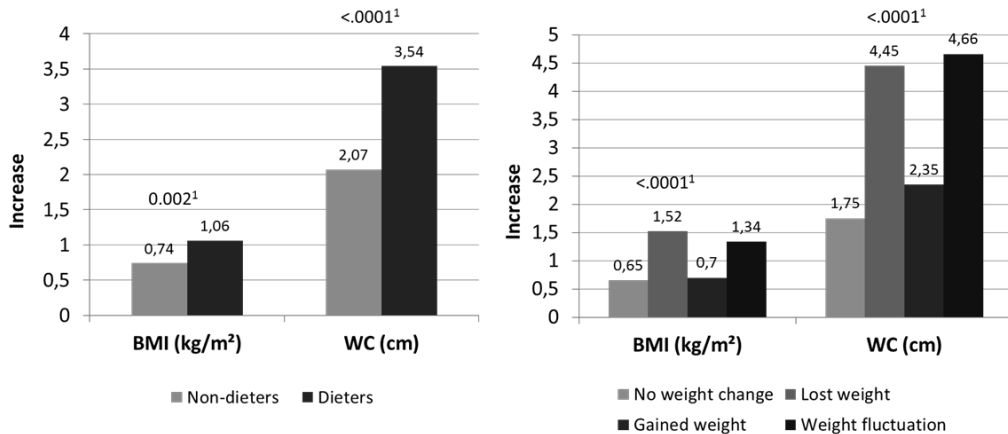
<sup>c</sup> Interaction analysis including an interaction term between mAHEI and dieting attempts.

## 5.4 DIETING ATTEMPTS, PREVIOUS CHANGES IN WEIGHT AND LONG-TERM CHANGES IN BMI AND WAIST CIRCUMFERENCE

Table 11 and the Table 12 present the baseline characteristics by dieting attempts and BMI groups.

BMI (26.5 vs. 27.4 kg/m<sup>2</sup>,  $P < .0001$ ), WC (91.0 vs. 93.6 cm,  $P < .0001$ ), and the proportions of individuals with obesity (19.5 vs. 25.0 %,  $P < .0001$ ) and those with abdominal obesity (35.4 vs. 42.9 %,  $P < .0001$ ) increased during the 11-year follow-up (Substudy III; Table 2). Women; younger individuals; single, divorced or separated individuals; smokers; those with less social support from close ones; and those with poor self-rated health experienced a statistically significantly greater increase in BMI and WC than the individuals in the reference groups (Substudy III; Table S1).

During the 11-year follow-up, BMI and WC increased more among dieters than non-dieters, after controlling for confounding factors (Figure 8, Substudy III; Table 3). Similarly, the increases were more notable among individuals with weight loss or weight fluctuation during the previous year than among those with no previous changes in weight.



**Figure 8** Multivariate adjusted<sup>2</sup> mean increases in BMI (kg/m<sup>2</sup>) and WC (cm) during the 11-year follow-up by dieting attempts and changes in weight during the previous year (n=2644).

Abbreviations: BMI, body mass index; WC, waist circumference.

<sup>1</sup> P-value for heterogeneity.

<sup>2</sup> Adjusted for sex, age, education, marital status, leisure-time physical activity, alcohol consumption, smoking, energy intake, modified Alternate Healthy Eating Index, geographical area and baseline value of the variable in question.

The risk of gaining  $\geq 5\%$  or  $\geq 10\%$  weight during the follow-up was also significantly elevated in dieters and in individuals with previous weight loss or weight fluctuation during the previous year in comparison to individuals in the respective reference groups (Table 15).

**Table 15** *Multivariate adjusted<sup>1</sup> odds ratios (OR) and their 95% confidence intervals (CI) of weight gain of  $\geq 5\%$  and  $\geq 10\%$  during the 11-year follow-up between categories of dieting attempts and weight change during the previous year (n=2644).*

Weight gain during the follow-up	Dieting attempts <sup>2</sup>		Previous weight change <sup>3</sup>			
	OR (95% CI)		OR (95% CI)		Gained n=780	Weight fluctuation n=147
	No (ref.) n=1775	Yes n=869	No (ref.) n=1268	Lost n=449		
$\geq 5\%$	1	<b>1.35</b> (1.11-1.65)	1	<b>2.16</b> (1.69-2.74)	1.20 (0.97-1.49)	<b>1.63</b> (1.11-2.39)
$\geq 10\%$	1	<b>1.59</b> (1.25-2.03)	1	<b>2.13</b> (1.60, 2.83)	1.09 (0.83, 1.42)	<b>2.32</b> (1.52, 3.53)

Abbreviations: BMI, body mass index; CI, confidence interval; mAHEI, modified Alternate Healthy Eating Index; OR, odds ratio; ref., Reference group; WC, waist circumference.

Bolded results are statistically significant.

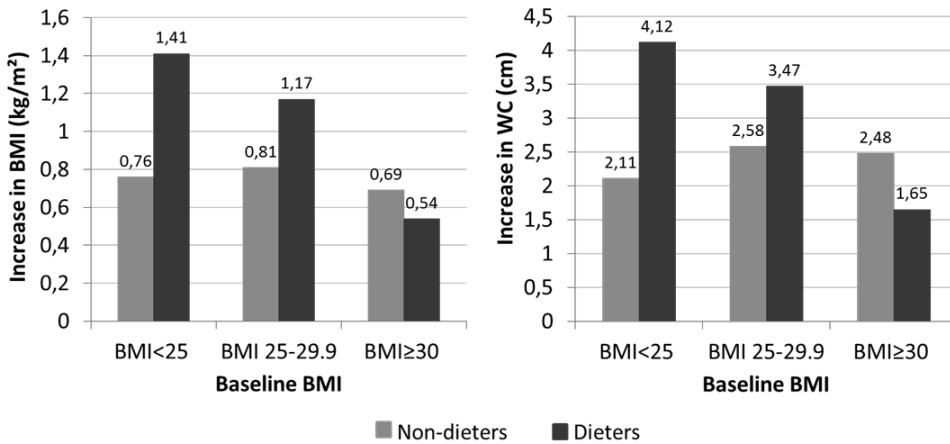
<sup>1</sup> Multivariate model: adjusted for sex, age, education, marital status, leisure-time physical activity, alcohol consumption, smoking, energy intake, mAHEI, geographical area, and baseline value of weight.

<sup>2</sup> Self-report dieting attempts during the previous year.

<sup>3</sup> Self-report weight change during the previous year.

The interaction between dieting attempts and previous changes in weight appeared non-significant in the prediction of subsequent changes in BMI and WC. Notable differences in changes during follow-up, however, could be seen between the non-dieters without previous weight change and dieters who had lost weight. The increases were approximately threefold in the latter subgroup (Substudy III; Table 5).

BMI at baseline proved to modify the association between dieting attempts and subsequent changes in BMI (P-value for BMI interaction =0.01) and WC (P-value for BMI interaction =0.005). BMI and WC increased more among dieters with initial normal weight than among non-dieters with initial normal weight, whereas among individuals with obesity, the differences in the increases between dieters and non-dieters tended to be inverted (Figure 9, Substudy III; Table 4). BMI at baseline and previous changes in weight showed no significant interactions in regard to subsequent changes in BMI and WC (data not shown).



**Figure 9** Multivariate adjusted<sup>1</sup> mean increases in BMI and WC during the 11-year follow-up by interaction of baseline BMI and dieting attempts during the previous year (n=2644).

Abbreviations: BMI, body mass index; WC, waist circumference.

In both analyses, P value for interaction <0.01.

<sup>1</sup> Adjusted for sex, age, education, marital status, leisure-time physical activity, alcohol consumption, smoking, energy intake, modified Alternate Healthy Eating Index and geographical area.

Further interaction analyses were conducted to test whether baseline characteristics (i.e. sociodemographic, lifestyle, somatic health, mental health, psychological, and social factors) modified the association between dieting attempts and subsequent changes in BMI and WC (Substudy III; Table S2). No significant interactions were found, but indicative interactions emerged between dieting and PA in prediction of changes in BMI (P-value for interaction =0.09) and WC (P-value for interaction =0.07). The increases in both BMI and WC tended to be greater in dieters who exercised regularly than in non-dieters who exercised regularly.

## 5.5 INTENTIONAL WEIGHT LOSS AND RISK OF DEVELOPING TYPE 2 DIABETES DURING A FOLLOW-UP

The distributions of baseline characteristics according to IWL categories during the previous year are presented in Table 11. Generally, all the considered potential risk factors of T2D (including sociodemographic, lifestyle-related, metabolic, and somatic health-related factors) significantly predicted the incidence of T2D during the 15-year follow-up after adjustment for sex and age, the only exceptions being energy intake and quality of diet (Substudy IV; Table 2).

IWL was significantly associated with an increased risk of T2D incidence even after comprehensive adjustment for confounding factors (Model 4) (Table 16). The finding was consistent across most of the different follow-up periods (0–15 years, 6–10 years, 11–15 years, and 6–15 years). The results of the first five years, however, attenuated to non-significant after further adjustments.

**Table 16** Hazard ratios (HR) and their 95% confidence intervals (CI) of T2D incidence by IWL during a 15-year follow-up (n=4270).

IWL	n of cases	N at risk	Model 1 <sup>1</sup>	Model 2 <sup>2</sup>	Model 3 <sup>3</sup>	Model 4 <sup>4</sup>
			HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
<b>Total follow-up</b>						
No	328	3712	1	1	1	1
Yes	89	558	<b>2.18 (1.73-2.77)</b>	<b>1.48 (1.16-1.88)</b>	<b>1.58 (1.23-2.03)</b>	<b>1.44 (1.11-1.87)</b>
P-value			<b>&lt;0.001</b>	<b>0.002</b>	<b>&lt;0.001</b>	<b>0.008</b>
<b>0-5-year follow-up</b>						
No	60	3712	1	1	1	1
Yes	18	558	<b>2.39 (1.41-4.06)</b>	1.46 (0.85-2.51)	1.46 (0.83-2.57)	0.94 (0.48-1.83)
P-value			<b>0.003</b>	0.19	0.21	0.84
<b>6-10-year follow-up</b>						
No	147	3587	1	1	1	1
Yes	42	533	<b>2.26 (1.60-3.20)</b>	<b>1.50 (1.05-2.14)</b>	<b>1.68 (1.16-2.41)</b>	<b>1.74 (1.20-2.53)</b>
P-value			<b>&lt;0.001</b>	<b>0.03</b>	<b>0.008</b>	<b>0.005</b>
<b>11-15-year follow-up</b>						
No	121	3354	1	1	1	1
Yes	29	485	<b>1.98 (1.32-2.98)</b>	1.48 (0.97-2.24)	<b>1.56 (1.02-2.39)</b>	<b>1.63 (1.06-2.49)</b>
P-value			<b>0.002</b>	0.08	<b>0.05</b>	<b>0.03</b>
<b>6-15-year follow-up</b>						
No	268	3587	1	1	1	1
Yes	71	533	<b>2.14 (1.64-2.78)</b>	<b>1.49 (1.14-1.96)</b>	<b>1.62 (1.23-2.14)</b>	<b>1.70 (1.28-2.25)</b>
P-value			<b>&lt;0.001</b>	<b>0.005</b>	<b>0.001</b>	<b>&lt;0.001</b>

Abbreviations: BMI, body mass index; CI, confidence interval; HDL, high density lipoprotein; HR, hazard ratio; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; n, number of participants in respective category; T2D, type 2 diabetes.

<sup>1</sup> Adjusted for sex and age.

<sup>2</sup> Adjusted for sex, age, and waist circumference.

<sup>3</sup> Adjusted for sex, age, education, BMI, physical activity, alcohol consumption, smoking, energy intake, mAHEI, and sleep duration.

<sup>4</sup> Adjusted for sex, age, education, BMI, physical activity, alcohol consumption, smoking, energy intake, mAHEI, sleep duration, waist circumference, elevated blood pressure, serum HDL cholesterol, serum triglycerides, and fasting serum glucose.

Examination of the amount of intentionally lost weight showed that it grew in parallel with the risk of incident T2D (Table 17). In a sensitivity study, the IWL variables considering different amounts of lost weight did not differ significantly from the IWL variable used in the primary analyses and considering any loss. When comparing the IWL used and an intentional weight loss of  $\geq 5\%$  when predicting the incidence of T2D (P-value for difference between models = 0.10) the hazard ratios were 1.44 (95%CI 1.11-1.87) and 1.65 (95% CI 1.21-2.25), respectively.

**Table 17** IWL during the previous year defined by different percentages of weight loss during the previous year and risk of T2D incidence during a 15-year follow-up (n=4270).

IWL	Prevalence (%)	HR <sup>1</sup>	95% CI	P for heterogeneity <sup>2</sup>	P for diff. with any loss
Any loss	13.1	<b>1.44</b>	<b>1.11-1.87</b>	<b>0.008</b>	-
$\geq 2\%$	12.3	<b>1.52</b>	<b>1.17-1.97</b>	<b>0.003</b>	0.11
$\geq 5\%$	6.93	<b>1.65</b>	<b>1.21-2.25</b>	<b>0.002</b>	0.10
$\geq 7\%$	4.00	<b>1.65</b>	<b>1.11-2.46</b>	<b>0.02</b>	0.23
$\geq 10\%$	2.01	<b>2.07</b>	<b>1.13-3.78</b>	<b>0.03</b>	0.14
$\geq 15\%$	0.56	<b>3.32</b>	<b>1.35-8.16</b>	<b>0.03</b>	0.08
$\geq 20\%$	0.26	2.13	0.52-8.74	0.35	0.54

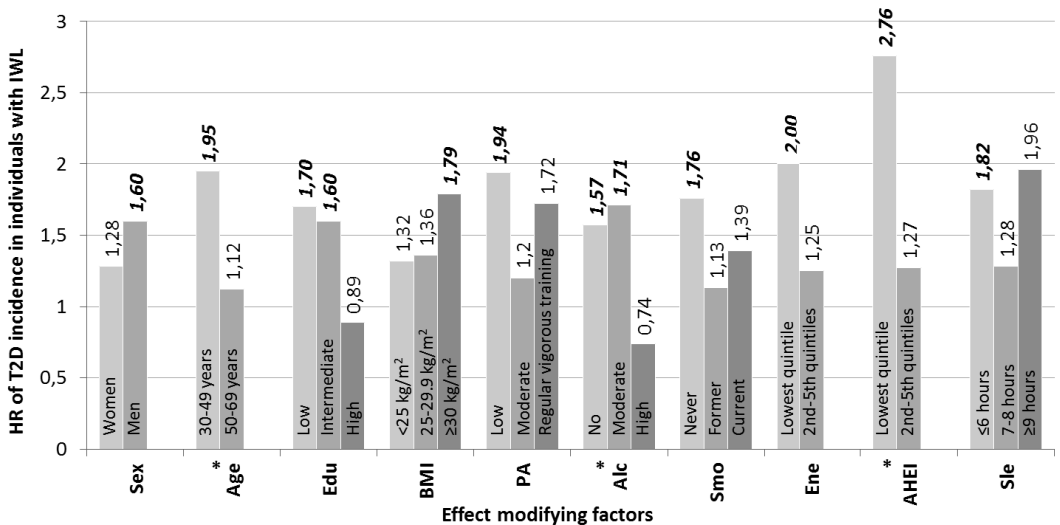
Abbreviations: CI, confidence interval; diff, difference; HR, hazard ratio; HDL, high density lipoprotein; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; T2D, type 2 diabetes.

<sup>1</sup> HR of those with respective IWL compared to those without respective IWL. Adjusted for sex, age, education, BMI, physical activity, alcohol consumption, smoking, energy intake, mAHEI, sleep duration, waist circumference, elevated blood pressure, serum HDL cholesterol, serum triglycerides, and fasting serum glucose.

<sup>2</sup> P-value for heterogeneity between those with respective IWL and those without respective IWL.

Analysis of whether various factors modify the prediction of IWL on T2D incidence during a 15-year follow-up revealed statistically significant interactions ( $P < 0.05$ ) between IWL and age, alcohol consumption, and mAHEI. The association between IWL and the risk of T2D was intensified in the younger age group, among non-users or moderate users of alcohol, and among individuals with the lowest diet quality (Figure 10, Substudy IV, Table 4). In addition, in spite of the lack of significant interaction in the respective analyses, an indicatively elevated risk among those with IWL emerged among men, those with low or intermediate education, individuals with obesity, those with low PA, never-smokers, those with the lowest energy intake, and those with short habitual sleep (Figure 10, Substudy IV, Table 4). Further analyses of the potential effect modification of different aspects of health (i.e. severe MetS, mental health, self-perceived health, and osteoarthritis) in

individuals with overweight (BMI $\geq$ 25 kg/m<sup>2</sup>) revealed no significant interactions. Accordingly, the risk of T2D was elevated in all subgroups with IWL, irrespective of health status, except for individuals with osteoarthritis (Substudy IV, Table 5).



**Figure 10** Multivariate adjusted<sup>1</sup> Hazard ratios (HR) of T2D during the 15-year follow-up in individuals with IWL compared to individuals without IWL (reference group) by subgroups of potential effect modifying factors (n=4270).

Abbreviations: Alc, alcohol consumption; BMI, body mass index; Edu, education; Ene, energy intake; HDL, high density lipoprotein; HR, hazard ratio; IWL, intentional weight loss; mAHEI, modified Alternate Healthy Eating Index; PA, leisure time physical activity; Sle, habitual sleep duration; Smo, smoking; T2D, type 2 diabetes; WC, waist circumference.

In subgroups with bolded and italic values, the risk of T2D incidence was statistically significantly larger in individuals with IWL than in individuals without IWL.

\* Significant interaction with IWL.

<sup>1</sup> Adjusted for sex, age, education, BMI, physical activity, alcohol consumption, smoking, energy intake, mAHEI, sleep duration, waist circumference, elevated blood pressure, serum HDL cholesterol, serum triglycerides, fasting serum glucose, and interaction variable in question. Analysis concerning interaction between IWL and BMI not adjusted for WC.

## 6 DISCUSSION

### 6.1 MAIN FINDINGS

The studies presented in this thesis examined how dieting attempts and IWL associate with a comprehensive set of background characteristics, how dieting attempts modify the association between quality of diet and obesity, how dieting attempts and previous changes in weight predict subsequent changes in BMI and WC during an 11-year follow-up, and how IWL is associated with T2D incidence during a 15-year follow-up. The study was conducted using the representative Finnish Health 2000/2011 Surveys, with extensive information on health, wellbeing, functional capacity, and background characteristics.

Previous studies have identified a few factors that are associated with dieting. These include female sex, higher education, healthier reported dietary intake, and higher BMI. Substudy I aimed to provide new, more comprehensive information on this topic and revealed many associations between dieting and several sociodemographic, lifestyle, somatic health and psychological factors.

Observational studies have shown dieting to associate with a healthier diet and obesity. A healthier diet has also shown an inverse association with obesity. These three factors, however, have not been studied simultaneously in a general adult population. Substudy II provided new information on the effect-modifying nature of dieting in the association between quality of diet and obesity. Accordingly, whereas no association emerged between quality of diet and obesity among non-dieters, among dieters, higher quality of diet was tentatively associated with an increased likelihood of having overweight or obesity.

Several previous observational studies have demonstrated a direct association between dieting and subsequent weight gain. The results of Substudy III verified that dieters had a greater increase in BMI and WC than non-dieters, and also provided new information on the factors that modify this association. Dieting was associated with greater weight gain in dieters with initial normal weight in particular.

Only a few observational studies, and all conducted in populations with overweight only, exist on the association between IWL and incidence of T2D. Substudy IV was the first to study this association in a general adult population also comprising individuals with normal weight, and to examine whether several sociodemographic, lifestyle, or health factors modify this association. IWL was associated with an increased risk of developing T2D during the 15-year follow-up.

The results of the substudies are interpreted in the following chapters.



## **6.2 WHO ARE SELF-REPORT DIETERS?**

The results of this thesis indicate that dieting attempts and IWL during the previous year are associated with several factors related to sociodemography, lifestyle, health and psychological issues in a cross-sectional setting.

### **6.2.1 SOCIODEMOGRAPHIC FACTORS**

Women, younger individuals and higher educated individuals had more often attempted to diet and had had IWL than the individuals in the corresponding reference groups, which is in agreement with previous findings (Stephenson *et al.* 1987, Jeffery *et al.* 1991, Paxton *et al.* 1994, Neumark-Sztainer *et al.* 1997, Crawford *et al.* 1998, Serdula *et al.* 1999, Neumark-Sztainer *et al.* 2000, Hjartaker *et al.* 2001, Bendixen *et al.* 2002, Anderson *et al.* 2002, Kruger *et al.* 2004, Keski-Rahkonen *et al.* 2005, Bish *et al.* 2005, Weiss *et al.* 2006, Lee *et al.* 2007, Zapka *et al.* 2009, Yaemsiri *et al.* 2011, Machado *et al.* 2012, Jackson *et al.* 2013, Cai *et al.* 2014, Santos *et al.* 2017, Barebring *et al.* 2018). Sociocultural pressures to be slim are prominent in western societies. Such pressures may cause body dissatisfaction and consequently lead to dieting attempts (Stice & Shaw 2002). Apparently, sociocultural pressures have a greater impact on women and younger individuals (Stice & Shaw 2002). Even though men also experience body image-related concerns, these are most commonly directed towards being muscular (Lavender *et al.* 2017). More highly educated individuals tend to be more health conscious, but alternatively, they may also be more strongly affected by sociocultural pressures to be slim.

### **6.2.2 LIFESTYLE FACTORS**

Of the lifestyle factors, obesity was the most notable predictor of dieting attempts among both sexes, which is in line with the literature (Stephenson *et al.* 1987, Jeffery *et al.* 1991, Williamson *et al.* 1992, Paxton *et al.* 1994, Crawford *et al.* 1998, Korkeila *et al.* 1999, Serdula *et al.* 1999, Wardle *et al.* 2000, Hjartaker *et al.* 2001, Anderson *et al.* 2002, Paeratakul *et al.* 2002, Kruger *et al.* 2004, Provencher *et al.* 2004, Weiss *et al.* 2006, Rideout & Barr 2009, Andreyeva *et al.* 2010, Yaemsiri *et al.* 2011, Machado *et al.* 2012, Jackson *et al.* 2013, Rose *et al.* 2013, Cai *et al.* 2014, Santos *et al.* 2015, Piernas *et al.* 2016, Barebring *et al.* 2018). This association may be explained by health consciousness, but also sociocultural pressures and dissatisfaction with excess weight, pushing individuals with obesity to try to lose weight. Also due to sociocultural pressures, individuals with obesity may report dieting attempts without necessarily having dieted.

Even though the prevalence of dieting attempts grew in parallel with BMI in women, women with normal weight had also attempted to diet relatively often (22% of women with normal weight vs. 61% of women with obesity). In contrast, among men, as expected, dieting attempts were more strongly dependent on BMI (6% of men with normal weight vs. 48% of men with obesity). Previous findings defend this notion (Stephenson *et al.* 1987, Crawford *et al.* 1998, Serdula *et al.* 1999, Kruger *et al.* 2004, Bish *et al.* 2005, Weiss *et al.* 2006), which is also in line with pressure to be slim having a greater impact on women (Stice & Shaw 2002). In western societies, weight and appearance ideals seem to set quite narrow norms for women, which supposedly also encourage women with normal weight to diet. Men, on the other hand, usually appear to start dieting only when affected by obesity.

IWL was associated with higher BMI in men only. Even though women with obesity attempt dieting more often than women with normal weight, in this study, weight status did not seem to make a difference in succeeding in such attempts. Among men, in contrast, a higher BMI appears to be related to better success in dieting attempts (Hansen *et al.* 2018). It seems that men do not start dieting so easily, but that when they do, they take it more seriously.

Apparently, no previous studies have been conducted on the associations between dieting and FFMI. After controlling for potential confounding factors, positioning in the three middle quintiles of FFMI was related to more commonly having attempted to diet. The absence of the association between the highest and the lowest quintile derived from including BMI in the multivariate model. FFMI correlates with BMI (VanItallie *et al.* 1990) because individuals with obesity usually have more supportive tissues and muscle tissue, enabling them to carry their excess fat tissue. However, it appears that in the absence of obesity, a high FFMI, in this case consisting mostly of muscle tissue, is not associated with dieting. Among women, after adjusting for potential confounding factors, including BMI, the prevalence of IWL grew along with the FFMI quintiles. This association was absent between dieting attempts and FFMI, suggesting that women with high FFMI do not attempt dieting more often than women with low FFMI, but that when they do, they more commonly succeed. This suggests that having more muscle tissue and thus presumably higher energy expenditure may aid in weight loss. Alternatively, having more muscle tissue may be a proxy of higher PA.

Individuals who exercised vigorously had attempted to diet more often than individuals with lower PA. This finding is in agreement with most (French *et al.* 1994a, Green *et al.* 1997, Kabeer *et al.* 2001, Bopp *et al.* 2006, Machado *et al.* 2012, Jeffery *et al.* 2013, Barebring *et al.* 2018) but not all (Paeratakul *et al.* 2002) previous studies. PA is often used as a strategy to lose weight (Santos *et al.* 2017). Alternatively, some individuals who participate in regular physical exercise may be athletes who attempt dieting

in order to stay in shape and to gain optimal performance. The possibility of misreported PA cannot be ruled out either (Lichtman *et al.* 1992).

Apparently, the association between dieting and sitting time has thus far been an unexplored study field. According to the results, dieting attempts and IWL were more common among individuals who sat the most than among individuals who sat the least. Obesity, being linked with more sitting time (Pedisic *et al.* 2014), may partly account for this association. However, as BMI was controlled for in the analyses, obesity cannot explain the association entirely and some other factors may lie behind this finding.

The finding that former smokers had attempted to diet and had succeeded in IWL more often than never-smokers is supported by some (Serdula *et al.* 1999, Hjartaker *et al.* 2001, Saarni *et al.* 2007) but not all previous results (Weiss *et al.* 2006). This found association was pronounced among women and among individuals with obesity. Quitting smoking often leads to weight gain (French & Jeffery 1995) which may consequently lead to dieting. Alternatively, it is possible that quitting smoking is part of a holistic lifestyle change, including weight loss.

Our findings on those with dieting attempts and IWL having lower energy intake (Jeffery *et al.* 1991, Neumark-Sztainer *et al.* 2000, Kant 2002, Bleich & Wolfson 2014, Wolfson & Bleich 2015), higher diet quality (or in previous studies, consumption of the components of a healthy diet)(French & Jeffery 1997, Neumark-Sztainer *et al.* 2000, Andreyeva *et al.* 2010, Jeffery *et al.* 2013, Bleich & Wolfson 2014), and less frequent consumption of certain sugary products (French *et al.* 1994a, French & Jeffery 1997, Wardle *et al.* 2000, Jeffery *et al.* 2013, Bleich & Wolfson 2014) are in concordance with those of previous studies. Decrease of energy intake and choosing higher-quality dietary components while avoiding sugary products is an advisable strategy to lose weight, and hence, the associations found presumably derive from using these alterations as weight loss strategies (Santos *et al.* 2017). In addition, underreporting of energy intake and foods considered unhealthy and overreporting of foods considered healthy is common among dieters, and may thus distort the results (Livingstone & Black 2003, Maurer *et al.* 2006, Poslusna *et al.* 2009, Castro-Quezada *et al.* 2015).

An association between short sleep duration ( $\leq 6$  h a night) and dieting attempts and IWL only emerged among men. A Canadian study (Gallant *et al.* 2013) reached similar results, although they concerned both sexes combined. Short sleep duration has been suggested to be a risk factor for obesity and even an obstacle to losing weight (Itani *et al.* 2017, St-Onge 2017). Indeed, it is possible that sleeping inadequately could induce weight gain and consequently, attempts to lose weight.

### 6.2.3 SOMATIC HEALTH

Individuals who had elevated serum triglyceride and lowered serum HDL concentrations had more often dieted than individuals with more favorable corresponding serum values. The associations were indicatively pronounced in women. Previous studies have found high dietary restraint to be associated with higher serum triglycerides (Laessle *et al.* 1989) and dieting attempts to be related to high serum total cholesterol (Paeratakul *et al.* 2002, Yaemsiri *et al.* 2011). Moreover, the results of the PREVIEW lifestyle intervention study indicated that HDL cholesterol concentration decreased statistically significantly after a rapid eight-week weight-loss, and the reduction was greater in women than in men (Christensen *et al.* 2018). In addition, weight cycling, often resulting from failed and repeated weight loss attempts, has been indicated to associate with elevated serum lipids (Montani *et al.* 2015). Consequently, an unfavorable serum lipid profile among dieters may derive from earlier dieting attempts and possible weight fluctuation, in addition to overweight or obesity per se.

T2D was only associated with dieting attempts among men. One observational American study found T2D to be more common among dieters than non-dieters (Paeratakul *et al.* 2002). Conversely, a Canadian study failed to find such an association (Green *et al.* 1997). In addition to T2D, dieting attempts and IWL proved to be more common among men with knee or hip osteoarthritis. No previous studies exist at the population level on this association. It is possible that for men, an actual disease diagnosis may act as a deterrent, driving them to try to lose weight in order to prevent the progression of the disease. Disease diagnosis may have a similar effect on women, but as women already diet more often, the potential association remains non-significant.

### 6.2.4 MENTAL HEALTH AND PSYCHOLOGICAL FACTORS

Low SOC values were associated with dieting attempts. No previous studies exist on this subject. SOC measures disposition to consider life as comprehensible, manageable and meaningful (Antonovsky 1993), and thus, individuals with low SOC values have a relatively poor capacity to cope with everyday life and manage its stressors. Individuals with such poor capacity have been shown to be less successful in achieving health-related lifestyle changes and a better quality of life than those with higher SOC values (Eriksson & Lindström 2006, Eriksson & Lindström 2007).

Appearance and health-related reasons are the most commonly reported motives behind weight loss attempts (Santos *et al.* 2017). Accordingly, in this study, concerns about one's appearance and for one's health were more common among dieters than among non-dieters. Even though the associations between dieting attempts and both concerns were significant in the analyses with both sexes combined, the association between concerns

about one's appearance and dieting attempts was slightly accentuated among women, while the association between concerns about one's health and dieting attempts was slightly accentuated among men. Previous findings on women experiencing more sociocultural pressures related to appearance (Stice & Shaw 2002) support this outcome. Furthermore, the finding on men dieting only when having an actual disease diagnosis or obesity is also in accordance with this tendency.

### **6.3 ROLE OF DIETING ATTEMPTS IN THE ASSOCIATION BETWEEN QUALITY OF DIET AND OBESITY**

The quantity of energy intake along with energy expenditure is fundamental in the development of obesity. However, quality of diet also seems to play a role (Mozaffarian *et al.* 2011). In this study, which had a cross-sectional setting, high quality of diet, measured by the mAHEI, modified to suit the Finnish dietary regimen, proved to be associated with a higher risk of overweight and obesity. This is in contrast with most previous findings indicating that high quality of diet measured by different dietary indices is inversely associated with obesity in both cross-sectional and prospective study designs in studies conducted in different countries (Togo *et al.* 2001, Buckland *et al.* 2008, Kanerva *et al.* 2013b, Asghari *et al.* 2017, Kanerva *et al.* 2018). In some of the studies, however, diet and obesity have lacked an association.

Various factors may account for the discrepancy between results. The methodological issues related to the dietary assessment method used (FFQ) have been scrutinized in sections 4.2.4 and 6.6.5. In addition, the different dietary assessment methods used in previous studies (e.g. different FFQs, 24-h dietary recalls and diet records) may have influenced the results (Kipnis *et al.* 2002, Freedman *et al.* 2014). The issues related to the mAHEI have been discussed in section 6.6.5.

In addition to these factors, confounding or effect-modifying factors may also account for the discrepancy. One such factor is dieting status. No previous studies with an adult population, however, have looked into simultaneous associations between quality of diet, obesity and dieting. As dieting has shown to associate directly not only with both the components of a high quality of diet (French & Jeffery 1997, Neumark-Sztainer *et al.* 2000, Andreyeva *et al.* 2010, Jeffery *et al.* 2013, Bleich & Wolfson 2014) and obesity (Stephenson *et al.* 1987, Jeffery *et al.* 1991, Williamson *et al.* 1992, Paxton *et al.* 1994, Crawford *et al.* 1998, Korkeila *et al.* 1999, Serdula *et al.* 1999, Wardle *et al.* 2000, Hjartaker *et al.* 2001, Anderson *et al.* 2002, Paeratakul *et al.* 2002, Kruger *et al.* 2004, Provencher *et al.* 2004, Weiss *et al.* 2006, Rideout & Barr 2009, Andreyeva *et al.* 2010, Yaemsiri *et al.* 2011,

Machado *et al.* 2012, Jackson *et al.* 2013, Rose *et al.* 2013, Cai *et al.* 2014, Santos *et al.* 2015, Piernas *et al.* 2016, Barebring *et al.* 2018) but also with misreporting of dietary intake (Livingstone & Black 2003, Maurer *et al.* 2006, Poslusna *et al.* 2009, Castro-Quezada *et al.* 2015), consideration of the presence of dieting in a study on quality of diet and obesity is justifiable. Indeed, when examining whether dieting modifies the association in question, no association emerged between quality of diet and overweight and obesity among individuals with no dieting attempts during the previous year. Moreover, analysis of the mAHEI components suggested that among non-dieters, a high consumption of fruit and berries was associated with a slightly decreased risk of obesity. This indicates, in line with most of the literature (Togo *et al.* 2001, Buckland *et al.* 2008, Mozaffarian *et al.* 2011, Asghari *et al.* 2017), that in this subgroup, certain healthy dietary components are inversely associated with obesity.

In contrast, among individuals with previous dieting attempts, the risk of overweight and obesity grew in parallel with quality of diet, suggesting that dieting status has a major impact on such an association. Moreover, dieters with the highest consumption of vegetables were at an elevated risk of overweight and obesity.

A few potential explanations could account for the direct associations in dieters in particular. Dieters may have adhered to a healthy diet in order to lose weight. This hypothesis is supported by the lower energy intake observed in dieters (Substudy II; Table 4). As dieters nevertheless had higher BMI, the decision to diet may not have considerably influenced BMI yet. Due to the cross-sectional study design, however, this speculation cannot be confirmed. In addition, as the mAHEI does not take into account intake of, for example, sugary products and refined grains, dieters consuming a healthy diet according to the mAHEI may also, during relapses, eat unhealthy foods outside it. This is further discussed in the methodological considerations in section 6.6.5.

Misreporting of dietary intake, common among dieters and individuals with obesity in particular (Livingstone & Black 2003, Maurer *et al.* 2006, Poslusna *et al.* 2009, Castro-Quezada *et al.* 2015), is probably partly responsible for the association found. Indeed, Rennie *et al.* (2006) suggested that questions on current dieting could be used as tools to identify possible misreporters in dietary surveys. It is possible that sociocultural pressures to be slim may externalize as a need to embellish one's diet and misreport dietary intake. As a consequence, dieters with misreported dietary intake and thus a higher mAHEI score would contribute to forming biased results.

In addition to the validity of dietary intake, the validity of the dieting information is another potential factor that may have affected the results. The same sociocultural pressures could also push individuals with overweight or obesity to report dieting even though not actually having tried to lose weight. Conversely, it is possible that individuals with normal weight

leave dieting attempts unreported. These misreporting patterns would distort the results.

## **6.4 DIETING ATTEMPTS AND LONG-TERM CHANGES IN BMI AND WAIST CIRCUMFERENCE**

Despite the initial purpose of dieting attempts as a means to lose weight, and in individuals with obesity to bring about health benefits (Oster *et al.* 1999, Rueda-Clausen *et al.* 2015, Hruby *et al.* 2016), several observational studies have demonstrated that in the long term, dieting is associated with subsequent weight gain among both adults and adolescents progressing into adulthood (French *et al.* 1994b, Coakley *et al.* 1998, Korkeila *et al.* 1999, Stice *et al.* 1999, Kroke *et al.* 2002, Lowe *et al.* 2006, Viner & Cole 2006, Field *et al.* 2007, Savage *et al.* 2009, Pietiläinen *et al.* 2012, Neumark-Sztainer *et al.* 2012, van Strien *et al.* 2014, Siahpush *et al.* 2015, Brown *et al.* 2016, Kärkkäinen *et al.* 2018). In accordance in this study, dieting attempts during the previous year were associated with a greater increase in BMI and WC as well as a greater risk of gaining  $\geq 5\%$  and  $\geq 10\%$  of weight in comparison to non-dieters during an 11-year follow-up.

In addition, weight loss and weight fluctuation during the previous year predicted a greater increase in the obesity measures used than maintaining one's weight during the previous year. This was in concordance with the majority of the earlier observations. In a large German study on previous changes in weight, recent weight loss and weight cycling were associated with considerable weight gain ( $\geq 2$  kg) during a two-year follow-up (Kroke *et al.* 2002). However, in a review by Mackie *et al.* (2017) only a slight majority of the studies examining the associations between weight cycling and obesity and subsequent weight gain concluded that weight cycling is related to current or future obesity, whereas nearly half of the studies included failed to find a significant association. One potential explanation for the inconsistent results is the unstable weight of the weight cyclers. Even though in the long term their weight may potentially increase, it is possible that during the study, they were in the middle of a weight loss phase, and thus yielded differing results.

The results of the weight loss intervention studies (Franz *et al.* 2007, LeBlanc *et al.* 2018) seem to show that short-term weight loss is achievable and with proper means and support, even maintainable. However, in general populations, the results appear to be inverse. There are a few possible explanations for this discrepancy between observational and intervention studies, including differences in the length of follow-up, the characteristics of the study populations (e.g. BMI), the support received to achieve weight loss and weight maintenance, motivation to lose weight, and the amount of weight lost.

#### **6.4.1 POTENTIAL MECHANISMS EXPLAINING THE DIRECT ASSOCIATION BETWEEN DIETING AND SUBSEQUENT WEIGHT GAIN**

It is possible that dieting as such does not necessarily cause weight gain, but it may represent proneness to gain weight due to other reasons. Lowe (2015), in a review on this subject, concluded that a dieting history would not act as a promotor for weight gain per se, but that weight gain would occur regardless of dieting in this subgroup. It appears that dieters and non-dieters differ according to various sociodemographic, lifestyle- and health-related factors. Differences also exist in regard to changes in BMI and WC according to such background characteristics.

One potential confounding factor not taken into account in this study is genetic susceptibility to gain weight. Two Finnish studies with two different twin datasets have examined the role of genetic predisposition in the association between dieting and weight gain (Korkeila *et al.* 1999, Pietiläinen *et al.* 2012). In a study conducted using the Finnish Twin Cohort, current weight loss attempts at baseline were associated with an increased risk of major weight gain (>10 kg) during six- and 15-year follow-ups (Korkeila *et al.* 1999). In a pair-wise twin analyses, however, the association attenuated to non-significant, which the authors construed as being the result of genetic and familial factors contributing to both weight gain and dieting behavior. Another study utilizing FinnTwin16 study data drew similar conclusions in regard to genetic predisposition, although a finding on monozygotic twins discordant for IWL suggested that dieting could also affect subsequent weight gain independently of genetic factors (Pietiläinen *et al.* 2012).

Thus, dieting may also as such induce later weight gain. Several potential explanations support this theory, including various biological and behavioral anti-weight loss and pro-weight gain mechanisms activated by a decrease in energy intake, FFM and FM (Ochner *et al.* 2013, Sumithran & Proietto 2013, Bosy-Westphal *et al.* 2013, Dulloo & Schutz 2015, Dulloo *et al.* 2015, MacLean *et al.* 2015, Polidori *et al.* 2016, Dulloo *et al.* 2018). Many of such mechanisms further promote weight regain beyond the initial level. These potential pathways have been addressed in more detail in the literature review in section 2.2.6.

#### **6.4.2 FACTORS MODIFYING THE ASSOCIATION BETWEEN DIETING ATTEMPTS AND SUBSEQUENT WEIGHT GAIN**

This study examined whether various background factors modified the associations between dieting attempts and changes in BMI and WC. Increases in BMI and WC during the follow-up were greater among dieters with initial normal weight than among corresponding non-dieters. In contrast, the differences between dieters and non-dieters were not so notable among individuals with initial overweight, and the difference appeared to be



reversed among individuals with initial obesity. Even though prevalence of dieting grows along with BMI (Santos *et al.* 2017), dieting is also relatively common among individuals with normal weight and with underweight (Montani *et al.* 2015). The results found in this study are in agreement with previous studies indicating that dieters with normal weight in particular are susceptible to subsequent weight gain (Korkeila *et al.* 1999, Pietiläinen *et al.* 2012, Dulloo *et al.* 2015) whereas the association appears to be less clear in individuals with initial obesity (Dulloo *et al.* 2012). When individuals with less FM lose weight, weight loss consists of relatively more FFM than FM compared to individuals with higher FM (Forbes 2000, Dulloo *et al.* 2018). This may lead to more unfavorable body composition when regaining the weight and to a greater susceptibility to regain weight beyond the initial level (Dulloo *et al.* 2015). However, an alternative theory has been presented, according to which dieters without initial obesity do not gain weight due to dieting attempts but due to other predispositions to gain weight (Lowe 2015). According to this theory, dieting would only be a response to a tendency to gain weight. Moreover, among individuals with current normal weight but with great weight suppression (i.e. difference between highest lifetime weight, excluding pregnancy, and weight after weight loss) the body's aspiration to revert to the highest previous weight seems to promote weight gain via several pathways, making it difficult to stay at a lower weight (Lowe *et al.* 2013). Even though possible weight suppression was not considered in this study, this could be one reason for the accentuated association in individuals with normal weight.

Of the other potential effect-modifying factors tested, only PA showed an indicative interaction with dieting attempts, according to which the increases in BMI and WC were greater in dieters exercising regularly than in non-dieters exercising regularly. Regular PA is an important part of weight loss and weight maintenance (Goldberg & King 2007, Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013, Bray *et al.* 2016, ), and successful weight maintainers have been shown to have high levels of PA (Melby *et al.* 2017, Paixão *et al.* 2020). However, PA can also increase appetite, and thus negate the energy deficit caused by PA. This effect, however, varies greatly inter-individually (Blundell *et al.* 2015). It is possible that PA is not sufficient to prevent weight gain in the long term if the unfavorable consequences of dieting attempts overrule its impact, or if other lifestyle aspects promote weight gain. Alternatively, it is also possible that dieters overreport their PA (Lichtman *et al.* 1992). Moreover, a high level of PA may have been a temporary part of a dieting regimen, but when dieting has potentially failed (as it often does) the dieter may also have given up on regular PA. In terms of PA and diet, one problem in long-term success in weight loss and weight maintenance is the hurry in which many individuals try to lose weight. Fast weight loss with a strict diet and PA regimen may sound more tempting than slower weight loss with healthy lifestyle changes. Strict weight loss regimens, however, often lead to relapses

or are hard to follow in the long term. Further, relapses may destroy motivation to follow the regimen, and the dieter easily returns to old habits. Moreover, more drastic energy restriction with a faster weight loss rate causes greater loss of FFM (Forbes 2000), which again promotes greater susceptibility to regain weight beyond the initial weight (Dulloo *et al.* 2018).

## **6.5 INTENTIONAL WEIGHT LOSS AND RISK OF DEVELOPING TYPE 2 DIABETES DURING A FOLLOW-UP**

In this study, IWL during the previous year was associated with an increased risk of developing T2D during a 15-year follow-up. The association was relatively consistent and remained significant not only after adjustment for potential confounding factors but also in several subgroups, during different time intervals, and when defining IWL with different percentual cut-off values.

### **6.5.1 RESULTS OF PREVIOUS STUDIES ON ASSOCIATIONS BETWEEN WEIGHT LOSS AND TYPE 2 DIABETES INCIDENCE**

Several intervention studies addressing weight loss as means of preventing T2D have proven successful weight loss to reduce the risk of T2D incidence (Peirson *et al.* 2014, Haw *et al.* 2017, LeBlanc *et al.* 2018). In most of these studies, the differences between the weight lost in the lifestyle intervention groups and the control groups have only been a few kilograms. Despite these small differences, in a meta-analysis by Haw *et al.* (2017), the risk of developing T2D decreased by 39% in the intervention groups compared to the control groups during the active intervention periods. Moreover, intervention studies with long follow-ups after the actual intervention periods have shown the preventive effects of the interventions to last compared to the long-term outcomes of the control groups (Diabetes Prevention Program Research Group *et al.* 2009, Lindström *et al.* 2013).

However, as in most cases, the intervention study participants have had overweight or obesity or have been at an elevated risk of T2D, as the follow-up periods in most of the studies have been relatively short (mostly between 0.5-6.3 years), and as the participants have received support to lose and maintain weight, the results of these studies cannot be directly generalized to the population level. Results of many observational studies with WLW as an exposure, however, have also agreed with the intervention studies (Chan *et al.* 1994, Colditz *et al.* 1995, Resnick *et al.* 2000, Koh-Banerjee *et al.* 2004, Wannamethee *et al.* 2005, Kim *et al.* 2018, Stokes *et al.* 2018), although in some studies, the association has remained absent (Ford *et al.* 1997, Moore *et*

*al.* 2000, Oguma *et al.* 2005, Mishra *et al.* 2007, Waring *et al.* 2010, Nanri *et al.* 2011).

Three studies predicting T2D incidence or T2D-related mortality with IWL, all conducted using extensive (n=approximately 43 000–181 000) American Cancer Society's Cancer Prevention Study data with individuals with overweight or obesity, have shown IWL to be associated with a lowered risk (Williamson *et al.* 1995, Williamson *et al.* 1999, Will *et al.* 2002). Moreover, a study conducted using the San Antonio Heart Study data indicated that self-report dieting predicted a decreased risk of T2D among women (Monterrosa *et al.* 1995). Among men, when BMI was not included in the model, a tentatively elevated risk emerged. This association, however, attenuated to non-existent when BMI was adjusted for in the final model. Hence, apart from this one indicative finding, the results found in this study are rather conflicting compared to the existing literature.

Potential explanations for this discrepancy between studies include issues related to the validity and/or reliability of the IWL variable in this study or between-study differences in weight loss variables, the decades of the data collection, the study populations, the lengths of follow-ups, control for confounding, and the definition of T2D. The questions related to the validity and reliability of the IWL variable are discussed in section 6.6.2. Other potential reasons are scrutinized in detail in the appendix of original Article IV. In summary, it can be interpreted that in spite of some methodological differences between studies, none of these differences appears to solely be accounted for the discrepancy. Hence, it seems that the direct association between IWL and the risk of T2D derives from other reasons.

## **6.5.2 POTENTIAL MECHANISMS EXPLAINING THE DIRECT ASSOCIATION BETWEEN INTENTIONAL WEIGHT LOSS AND TYPE 2 DIABETES INCIDENCE**

Despite the inverse associations found in the literature on weight loss and T2D incidence, and despite even a relatively small weight loss with healthy lifestyle changes effectively preventing T2D incidence in intervention studies (Haw *et al.* 2017), long-term weight maintenance appears to be difficult and rare (Melby *et al.* 2017). The direct association between self-report dieting attempts and IWL and subsequent weight gain found in many observational studies is further described in section 2.2.6. Weight gain and obesity, in turn, increase the risk of developing T2D. In addition to the potential linear weight gain after dieting, weight cycling itself may possibly increase the risk of T2D. Suggested potential metabolic pathways by which weight cycling may promote the development of T2D include general weight gain and the accumulation of abdominal obesity (Mackie *et al.* 2017). Abdominal obesity in particular has been indicated to contribute to the development of insulin resistance (Tchernof & Despres 2013). Additionally, weight gain and weight

cycling may induce unfavorable alterations in metabolic factors such as MetS components (Montani *et al.* 2015, Rhee 2017). MetS, in turn, is a considerable risk factor of T2D, increasing the risk fivefold (O'Neill & O'Driscoll 2015). However, in a review by Mackie *et al.* (2017), only a little more than half of the studies found weight cycling to increase the risk of future weight gain or to alter body composition into more unfavorable, and the majority of the studies failed to find an association between weight cycling and impairment in metabolic markers. Despite this uncertainty, the evidence suggesting that weight cycling is associated with T2D occurrence is relatively consistent (Kodama *et al.* 2017, Rhee *et al.* 2018).

In addition, one meta-analysis on weight loss and mortality risk concluded that IWL is associated with increased mortality among initially healthy individuals, but with decreased mortality among unhealthy individuals (Harrington *et al.* 2009). Hence, it is possible that also in this general population the adverse consequences of IWL may reverse the potential beneficial effects of weight loss in the long term (Sørensen *et al.* 2005).

It is possible that self-initiated weight loss is insufficient to reduce the risk of T2D. However, in this study, the magnitude of the risk of T2D appeared to grow in parallel with the amount of intentionally lost weight. This is in disagreement with the findings of some of the previous observational studies suggesting that greater weight loss would result in a greater reduction of risk (Colditz *et al.* 1995, Resnick *et al.* 2000, Koh-Banerjee *et al.* 2004, Kim *et al.* 2018), although not all studies have supported this. The present finding may derive from greater amounts of weight loss during only one year, resulting in greater potential subsequent weight gain or weight cycling, and thus a greater risk of developing T2D.

Alternatively, it is possible that individuals with IWL may initially be at a greater risk of T2D or they may be more health conscious, and thus their T2D may become diagnosed more frequently. In order to understand the findings more thoroughly, the associations were further studied in subgroups of several background variables.

### **6.5.3 FACTORS MODIFYING THE ASSOCIATION BETWEEN INTENTIONAL WEIGHT LOSS AND INCIDENT TYPE 2 DIABETES**

Apparently, no previous studies of general adult populations have comprehensively examined the modifying effects of different sociodemographic, lifestyle- and health-related factors on the association between IWL and T2D incidence.

### ***Health-related factors***

Previous studies found a reduced risk of T2D-related mortality in men and women with IWL among those with pre-existing health conditions (i.e. history of heart disease, stroke, diabetes, or high blood pressure, or reported to have currently experienced severe shortness of breath or severe pain in the chest) but not in men and women with IWL among those without such conditions (Williamson *et al.* 1995, Williamson *et al.* 1999). In the present study, several indicators of poor metabolic health were associated with IWL at baseline and with an increased risk of developing T2D during follow-up. Thus, individuals with IWL may initially be at a greater risk of T2D. Further, it is possible that in this subgroup with a high risk and IWL, weight loss may actually be due to undiagnosed pre-diabetes. However, interaction analyses on IWL and MetS, as well as IWL and different aspects of poor health among individuals with overweight or obesity revealed no statistically significant interactions, but the risk of T2D appeared to be elevated in each subgroup with IWL compared to corresponding subgroups without IWL, regardless of the presence of any health conditions. This finding, combined with the fact that in the primary analyses, inclusion of the MetS components did not notably alter the results during the 15-year follow-up, suggests that it is unlikely that initially poorer health at baseline among those with IWL would entirely be accounted for the direct long-term association. However, as inclusion of the MetS components attenuated the association to become non-existent during the first five years of the follow-up, it appears that the group with IWL still had more individuals with poorer health who developed T2D relatively shortly after baseline. Their impact on the association between IWL and T2D, however, did not seem to persist over a longer period of time.

Of the sociodemographic and lifestyle factors considered, only age, quality of diet, and alcohol consumption showed statistically significant interactions with IWL. In addition, however, several indicative associations emerged.

### ***Sociodemographic factors***

Of the sociodemographic factors, the risk of developing T2D in individuals with IWL seemed to be tentatively pronounced in men, younger individuals, and individuals with a lower educational level. It is possible that younger individuals more often diet more carelessly for appearance-related reasons with more probable subsequent weight regain, while older individuals take IWL more seriously to actively try to improve their health and prevent diseases.

Even though the results of Substudy I indicate that men only start dieting for medical reasons, men may take their IWL less in earnest than women in the long term, resulting in a greater risk of weight regain. Alternatively, the differences in the accumulation of excess fat among men and women may account for this finding. Excess fat has been shown to localize differently in

men and in women, with men being at greater risk of abdominal fat accumulation, which particularly increases the risk of T2D (Tchernof & Despres 2013). The results of Substudy III (Substudy III; Supplementary Table 2) support this phenomenon; women appeared to gain more weight during the 11-year follow-up than men when measured according to BMI. Conversely, in terms of WC, men tended to have a slightly greater increase than women. Thus, weight gain may be more detrimental among men than among women due to the propensity of the accumulation of central adiposity. In addition, men with IWL may initially be at a higher risk of T2D, which is supported by the findings of Substudy I and a few previous studies (Bish *et al.* 2005, Andreyeva *et al.* 2010).

Low educational level has been linked to an elevated incidence of T2D (Maty *et al.* 2005, Lee *et al.* 2011, Kolb & Martin 2017), and overall, low socioeconomic status has been shown to associate with an increased risk of developing obesity (Rissanen *et al.* 1991, Sarlio-Lähteenkorva 2007). In Finland, the rise in T2D incidence during the 1980s and the 1990s occurred predominantly in men with a low educational level (Abouzeid *et al.* 2015). As it has also been suggested that a lack of positive interpersonal and intrapersonal resources act as a pathway between low education and generally poorer health (Matthews & Gallo 2011), it can be further speculated, whether such poor resources could also possibly lead to poorer strategies for trying to lose and maintain weight and to a greater risk of T2D among those with a low education.

### ***Lifestyle factors***

An indicatively accentuated association between IWL and T2D incidence emerged among individuals with lifestyle risk factors of T2D, including those with obesity, low PA, low-quality diet, and short sleep duration. Moreover, the association was also tentatively pronounced among never-smokers, non- and moderate consumers of alcohol, and individuals with the lowest energy intake.

In previous observational studies, dieting has been associated with later weight gain, especially among those with initial normal weight, whereas among those with initial obesity, the growth in obesity measures has been more moderate (Korkeila *et al.* 1999, Pietiläinen *et al.* 2012, Dulloo *et al.* 2015). However, in the present study, the increase in the risk of T2D incidence occurred in dieters with obesity. Initial obesity increases the risk of T2D. Thus, failed IWL, leading to even modest further weight gain or alternatively to weight cycling, inducing fluctuations in metabolic values (Montani *et al.* 2015, Rhee 2017), may be more detrimental in individuals with obesity than in individuals with initial normal weight.

Within the group with the lowest quality of diet, those with IWL had an elevated risk of T2D compared to those without IWL. It can be speculated that IWL, with its subsequent weight regain, combined with low quality of

diet, which adds to the risk of unfavorable changes in metabolic health (Mozaffarian 2017), predisposes people to a greater risk of T2D. Moreover, it is possible that with poor lifestyle habits, weight maintenance after weight loss is more prone to fail (Elfhag & Rossner 2005, Lindström *et al.* 2013, Varkevisser *et al.* 2019, Paixão *et al.* 2020). In accordance, an indicatively elevated risk of T2D also emerged among those with low PA and IWL. Low PA is associated with a greater risk of weight gain (Rissanen *et al.* 1991). In the prevention of T2D, PA works by aiding weight loss and weight maintenance (Swift *et al.* 2018) but also independently by improving insulin sensitivity and other metabolic values (Borghouts & Keizer 2000, Aune *et al.* 2015). Lack of PA may hinder weight loss and weight maintenance, and thus increase the risk of T2D. Some evidence suggests that low PA in particular is associated with increased appetite through low FFM and the body's aspiration to compensate for it via increased energy intake (Dulloo 2017). Those with IWL and short habitual sleep duration had also a tentatively accentuated risk of T2D. Previous studies have indicated that short sleep is associated with an increased risk of T2D (Shan *et al.* 2015, Itani *et al.* 2017). It can be speculated that IWL's potential harmful consequences add to the harmful consequences of short sleep, increasing the risk of T2D.

In addition, a suggestive pronounced association between IWL and T2D emerged in individuals with the lowest energy intake. Even though weight loss requires a negative energy balance, and success in weight maintenance has been associated with lower energy intake (Elfhag & Rossner 2005, Varkevisser *et al.* 2019), too drastic a reduction in energy intake may lead to greater vulnerability to diet relapses and greater loss of FFM, which in turn may lead to faster weight regain and greater fat overshooting (Forbes 2000, Dulloo *et al.* 2018).

The findings on non- and moderate consumers of alcohol and never-smokers with IWL having elevated risk of T2D may derive from IWL increasing the risk by the same mechanisms as in the whole study population. Conversely, among heavy consumers (Knott *et al.* 2015) and smokers (Sliwinska-Mosson & Milnerowicz 2017) the risk is already elevated. Furthermore, it is possible that the implementation of IWL or health consciousness, and thus the frequency of T2D diagnosis, differs between groups according to alcohol consumption and smoking.

Collectively, it is possible that among men, younger individuals, individuals with a lower educational level, individuals with obesity, individuals with unfavorable lifestyle factors, and individuals with potentially too low an energy intake, IWL results in poorer long-term success, and the increase in the risk of T2D is mediated via subsequent weight regain or weight cycling. Conversely weight loss may be more successful among individuals who pursue it using healthier strategies or otherwise more earnestly. Consequently, it is probable that poor execution of IWL is the primary explanation for the results found in this study.

## 6.6 METHODOLOGICAL CONSIDERATIONS

### 6.6.1 STUDY POPULATION AND DESIGN

A general strength of the present study is the large and nationally representative adult population sample used with high participation rate and long follow-up period. Moreover, quality-controlled data collection, including health examinations, interviews and questionnaires enabled the study of versatile research questions, the opportunity to control for confounding, and the study of various interactions. Another strength is the possibility to link the study data with information drawn from extensive national health registers.

Some limitations, however, also exist. First, even though the original Health 2000 sample was nationally representative, and the participation rate was good, the participation rate was somewhat lower in Health 2011, making the study population of Substudy III less representative. It has been shown that individuals who do not participate in health examination surveys have unhealthier lifestyle habits and poorer health than those who participate (Jousilahti *et al.* 2005, Tolonen *et al.* 2017). Accordingly, this phenomenon may have an effect on the results; on the one hand, participants may be more health conscious and thus attempt to diet more often, but on the other hand, they may be at a lower risk of gaining weight or developing T2D. As the objective of this study was not to estimate the representative prevalence of dieting in Finland but to study the associations of self-report dieting more generally at the population level, the sampling weights were not used in this study.

Second, the study design causes limitations. In observational studies, no causal effects can be detected. In Substudies I and II, a cross-sectional study design prevented the examination of chronological associations. In Substudy III, a longitudinal study design with an outcome only at the endpoint was applied, and in Substudy IV, a cohort study design was applied. In these studies, however, lack of repeated measurements prevented the examination of whether dieting behavior or confounding factors changed over time. Third, even though the study data used were extensive, the number of participants in the categories of certain variables (e.g. some somatic and mental disorders) was small. Hence, the results should be interpreted with caution.

Fourth, in spite of careful adjustments for the potential confounding factors conducted in each substudy, the possibility of residual confounding cannot be fully ruled out. Consequently, some associations found may partly derive from unidentified characteristic differences between the groups according to dieting attempts or IWL. Even though an extensive set of potential determinants (Substudy I) and effect-modifying factors (Substudies III and IV) were examined, other important factors may be missing from the scrutiny (e.g. more detailed information on dieting behavior, perceived body size/image, and eating disorders) due to their absence in the data.



Fifth, participants with chronic diseases related to obesity may have affected the results. Individuals with cancer, for instance, may have experienced unintentional weight loss. The inclusion of such individuals in the study population, however, was opted for in order to enable the study of the possible effect modification of the diseases (diabetes, knee or hip osteoarthritis, cancer, depressive disorder, anxiety disorder).

Finally, the possibility of false positive findings cannot be excluded in studies such as these including numerous analyses. A Bonferroni correction was conducted in Substudy I in order to observe this possibility. The correction attenuated part of the associations into non-significant. With the Bonferroni correction, however, the possibility of rejecting true positive findings is increased. Thus, because the findings were plausible when reflecting on the literature, they were chosen to be approved. However, caution must be used in the interpretation of the findings.

## **6.6.2 DIETING AND PREVIOUS CHANGES IN WEIGHT**

The dieting variables used in this thesis involve several factors that deserve discussion. Owing to the dieting variables being self-reported and elicited using only a few questions, and due to the population representing the general adult population, the groups of those with dieting attempts and IWL may have included very heterogeneous dieting. However, the aim of Substudy I was to create an understanding of this issue. Further, simultaneous scrutiny of dieting attempts and IWL (i.e. a little different-natured types of dieting) enabled the examination of whether they would differ in terms of certain potential determinants.

Even though previous studies have found the phrasing of questions concerning dieting to provide different prevalences (French & Jeffery 1994, Neumark-Sztainer *et al.* 1997), previous observational studies have consistently shown self-report dieting attempts or IWL (measured only by one or a few questions, and regardless of the differences in the wording of the questions) to predict weight gain during a follow-up, even after adjusting for potential confounding factors including baseline BMI (French *et al.* 1994b, Coakley *et al.* 1998, Korkeila *et al.* 1999, Stice *et al.* 1999, Kroke *et al.* 2002, Lowe *et al.* 2006, Viner & Cole 2006, Field *et al.* 2007, Savage *et al.* 2009, Pietiläinen *et al.* 2012, Neumark-Sztainer *et al.* 2012, van Strien *et al.* 2014, Siahpush *et al.* 2015, Brown *et al.* 2016, Kärkkäinen *et al.* 2018). This suggests that dieting, elicited using only one or two questions, is a reliable tool to measure tendency toward dieting behavior in general populations.

As the information for the dieting variables was only collected at baseline, the continuity of dieting remains unknown; some non-dieters may have started dieting, some dieters may have succeeded in maintaining their weight, some dieters may have failed and regained the weight or just stopped attempting to diet, and others may have ended up with repeated dieting

attempts and weight cycling. Previous studies, however, have indicated that dieting is seldom only a one-time effort, but that dieting behavior seems to be something that lasts (French & Jeffery 1997, Montani *et al.* 2015). In a study measuring the associations between 10-year changes in dieting behavior and 10-year changes in BMI, almost 70% of women but only approximately 40% of men who dieted at baseline were still dieting at a 10-year measurement point (Neumark-Sztainer *et al.* 2012).

The weight fluctuation variable used in Substudy III imitates actual weight cycling variables used in other studies. However, as it contains only self-report information on weight loss and weight gain during the previous year, its character remains rather different from the weight cycling variables with more exact information on longer period of time and more weight cycles. However, as the long-term weight change outcome of the weight fluctuation variable is quite similar to that of most weight cycling studies (Mackie *et al.* 2017), it can be speculated that this weight fluctuation variable is a relatively good proxy of other weight cycling variables.

In Substudies I and IV, in which the IWL variable was used, the primary analyses were conducted by including all the individuals with IWL, regardless of the amount of lost weight in the group in question, making the range of self-reported weight loss relatively broad (in Substudy I: range 1–38 kg, mean 5.43 (SD 4.21) kg). This procedure, however, was consistent with many previous studies using self-report IWL variables (Williamson *et al.* 1995, Williamson *et al.* 1999, Will *et al.* 2002, Gregg *et al.* 2003). In additional analyses, however, IWL variables concerning different amounts of lost weight were utilized. The IWL used, and an IWL of 5% or more, had relatively good overall agreement, the intraclass correlation coefficient (ICC), estimated as kappa, being 0.66 (95% CI 0.63-0.70). Weight loss of 5% or more is often recommended as a target amount of weight loss in clinical practice (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2013, Williamson *et al.* 2015), and perceived as sufficient for prevention and improvement of several conditions, including T2D (Ryan & Yockey 2017). In a sensitivity study, the IWL used did not statistically differ from the intentional weight loss of 5% or more when predicting T2D incidence (P for difference between models=0.10) (Table 17). Thus, the IWL variable including all intentional weight losers was opted for use in the primary analyses. These results suggest that the amount of weight loss does not play a very meaningful role in the prediction of subsequent T2D incidence at the population level, and that the IWL used is an adequate measure to represent IWL in a general population.

### **6.6.3 WEIGHT CHANGE DURING FOLLOW-UP AND BODY COMPOSITION**

The strengths of the present study are the valid and reliable BMI and WC measurements, conducted by trained nurses according to standardized protocols and yielding comparable results in both Health 2000 and Health 2011 (Heistaro 2008, Lundqvist & Mäki-Opas 2016, Aromaa *et al.* 2019). Weight and height were also self-reported. In Substudies I and III, only information on measured weight and height was used to calculate BMI, whereas in Substudies II and IV, also self-report information was utilized. Even though previous findings suggest that self-reported weight often tends to be underestimated, especially among individuals with overweight or obesity (Connor Gorber *et al.* 2007, Maukonen *et al.* 2018), the proportion of those with self-reported weight in this study was small (4.7% in Substudy II; 3.1% in Substudy IV), and the degree of underestimations has usually appeared to be low (Connor Gorber *et al.* 2007). Hence, this factor presumably had at most only a minor effect on the results.

In Substudy I, FFMI was used as a potential determinant of dieting attempts and IWL. Even though the bioimpedance devices were validated, and thus presumably produced reliable body composition values, and the participants were asked not to eat for at least four hours before the health examination (Heistaro 2008), it is possible that for individuals with extreme BMI values or abnormal hydration in particular, the FFMI values were inaccurate (Kyle *et al.* 2004).

### **6.6.4 INCIDENT TYPE 2 DIABETES CASE ASCERTAINMENT**

In Substudy IV, the incident diabetes variable, drawn from the national health registers (the Drug Reimbursement Register, the Finnish Hospital Discharge Register, the National Causes of Deaths Register) and linked to the study data using unique social security codes, is considered valid and reliable (Heliövaara *et al.* 1984). However, the incident diabetes cases identified using ICD-10 codes E10-E14 also cover other types of incident diabetes (including T1D and other types of diabetes but excluding gestational diabetes). As incidence of the other types of diabetes in adulthood is rare (Working group set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society 2018, World Health Organization 2019), it is presumable that nearly all of the incident cases were T2D.

Another potential weakness related to the incident T2D variable, however, may derive from the case identification being based on registers only; thus, T2D cases with lifestyle treatment only, or individuals with undiagnosed T2D were categorized as non-cases. This potentially produced conservative estimates in the analyses. In Health 2000, of the individuals with diabetes, more than one fifth of men and more than one third of women did not use diabetes medication (Aromaa & Koskinen 2004). This suggests that also a

large proportion of the new incident cases probably used lifestyle treatment only and remained among the non-cases in this study.

### 6.6.5 DIETARY VARIABLES

Habitual diet was assessed using a validated and regularly updated self-administered semi-quantitative FFQ (Männistö *et al.* 1996, Paalanen *et al.* 2006, Kaartinen *et al.* 2012). FFQ is a generally used tool to assess long-term dietary intake in epidemiological studies, and the information it provides is suitable to classify participants according to their dietary intake (Willett 2013). Furthermore, FFQ appears to be an adequate tool for assessing quality of diet in order to form diet indices (Benitez-Arciniega *et al.* 2011, van Lee *et al.* 2014). The use of the dietary index method enables the study on the associations between the whole diet and specific outcomes.

The AHEI was chosen to represent quality of diet due to its long history deriving from the US dietary guidelines (Kennedy *et al.* 1996) and the Healthy Eating Index (HEI) (Kennedy *et al.* 1995), and due to its plausible associations with reduced risk of CVDs, CHD, stroke, T2D, cancer, and obesity (McCullough *et al.* 2002, Akbaraly *et al.* 2011, Chiuve *et al.* 2012, Boggs *et al.* 2013, George *et al.* 2014, Fung *et al.* 2015, Cheung *et al.* 2018, Neuenschwander *et al.* 2019). The AHEI has further been modified into a slightly different version, the AHEI-10 (Chiuve *et al.* 2012), but owing to the long nature of this project, the mAHEI was opted for in each substudy to have a consistent variable representing quality of diet.

For the mAHEI used in the present study, some minor modifications were made to adjust the original AHEI index to suit the Finnish dietary regimen. These alterations probably do not notably affect the results. Despite these minor modifications, generally, the cultural differences between countries may pose a challenge for the use of a dietary index designed in another country. Thus, in the future in Finland, it could be advisable to use a dietary index designed on the basis of a healthy local diet. One such index is, for instance, the BSDS (Kanerva *et al.* 2014b).

Another weakness in regard to the mAHEI is the lack of some components (e.g. sugary products and refined grains), which possibly reduces its suitability for the study of obesity. Individuals with a high mAHEI score may also eat unhealthy foods outside the mAHEI. The result of the energy intake growing along with the mAHEI quintiles (Substudy II; Table 3) supports this hypothesis. As the AHEI, however, had yielded plausible results when predicting obesity (Boggs *et al.* 2013, Fung *et al.* 2015) and chronic diseases (McCullough *et al.* 2002, Chiuve *et al.* 2012), it was chosen to represent quality of diet in this thesis. In Finland, the AHEI had not been used as a measure of quality of diet before the beginning of this thesis project, but later, its modifications have shown an association with a decreased risk of airway obstruction (Vasankari *et al.* 2019) and with higher

serum 25-hydroxyvitamin D concentrations (Jääskeläinen et al. 2013), but have lacked an association with the incidence of Parkinson's disease (Sääksjärvi et al. 2013).

## 6.7 PUBLIC HEALTH IMPORTANCE OF THE FINDINGS

Dieting attempts and IWL are common at the population level. Even though such behaviors are more common among individuals with obesity, real weight loss needs, and possibly at an elevated risk of developing NCDs, they are not limited solely to them, but individuals with normal weight also report these behaviors. Intervention studies including participants with obesity have indicated that weight loss is possible with adequate lifestyle changes, and that among high-risk individuals, successful weight loss reduces the risk of T2D for even a decade, despite some weight regain (Lindström *et al.* 2013, Haw *et al.* 2017). At the population level, however, weight loss often results in increasing weight gain. Moreover, the long-term outcomes of dieting among individuals with normal weight in particular appear to be adverse. Thus, research on dieting at the population level is warranted.

The substudies of this thesis were among the first to estimate various associations of self-report dieting in a general adult population sample while simultaneously taking into consideration a vast set of potential factors that modify the associations. The results imply that information on self-report dieting attempts or IWL can be used to identify individuals struggling with a tendency to gain weight, who are possibly prone to future weight gain, and further, at a potentially elevated risk of future T2D. This proxy also helps identify individuals with normal weight, which is essential for the initial prevention of weight gain. Support and tools for weight maintenance and for preventing further weight gain through healthy lifestyle changes should be targeted at dieters with normal weight or overweight. As weight loss generally tends to be ineffective in the long term, and obesity poses a substantial burden not only on public and individual health but also on the public economy, actions targeted at the maintenance of normal weight are called for.

Information on the determinants of dieters can be used as confounding factors in research on dieting attempts and the incidence of chronic diseases. This information can also be utilized in weight-loss-supportive measures by identifying the characteristics associated with dieters with obesity or alternatively by identifying individuals with obesity who abstain from weight loss efforts but would benefit from them. Moreover, identifying the characteristics associated with dieters with normal weight can help in the planning of preventive measures against unnecessary dieting and possible future weight gain.

Previous findings have shown that the misreporting of dietary intake is common among dieters and in individuals with obesity. In addition to this, knowledge that dieting attempts may modify the association between quality of diet and obesity enables taking this into account when planning and monitoring weight loss regimens in clinical practice.

Generally, the findings of this thesis call for an emphasis on the initial prevention of weight gain, the prevention of unnecessary weight loss efforts, support for learning a healthy lifestyle to facilitate weight maintenance and weight loss among individuals with obesity and in clinical settings, long-term support and follow-up after the actual weight loss period to enhance the likelihood of sustained weight loss and to prevent weight regain. In addition, a change in the obesogenic environment is urgently needed in order to fight against the increase in obesity and NCD rates.

## **6.8 IMPLICATIONS FOR FUTURE EPIDEMIOLOGICAL RESEARCH**

The substudies of this thesis increased the knowledge on dieting at the population level, which, despite its commonness, has been a relatively unstudied field. The literature from intervention studies concerning lifestyle interventions and how they predict T2D incidence is relatively comprehensive, determinants of successful weight loss have been explored in various studies, and observational studies have quite consistently detected an association between self-report dieting and subsequent weight gain. In spite of this research, the determinants of dieting in a general population, as well as whether self-report IWL predicts T2D incidence in the long term at the population level, have thus far lacked comprehensive research.

As the substudies were among the first to look into these topics, replication of the studies in other population-based datasets is needed. Further cohort studies with large enough samples to adequately enable the estimation of effect modification are also needed. Moreover, studies using repeated measurements of dieting and changes in weight are warranted. Studies with more detailed information on dieting attempts, IWL, weight loss strategies and motives, attitudes towards weight, and eating behavior in each BMI subgroup are also called for in order to make the meaning of dieting clearer and to be able to possibly categorize individuals according to different types of dieting at the population level.

Whether self-report dieting is associated with the incidence of other chronic diseases also requires research. Furthermore, an even wider range of the potential determinants of dieting attempts and IWL should be examined. Such potential determinants include different measures of PA, different healthy and unhealthy diet components, and various psychological factors.

Furthermore, whether the new discovered determinants or dieting strategies and motives modify the associations between dieting variables and subsequent weight gain and the incidence of chronic diseases should be tested. Studies on how long-term weight loss success and lifestyle changes, achieved in lifestyle intervention studies, could be transferred to the population level are also needed.

## **7 SUMMARY AND CONCLUSIONS**

The present thesis provided new information on the associations between self-report dieting and several factors in a general adult population. It also disclosed that dieting modifies the association between quality of diet and obesity. It appears that at the population level, dieting attempts and IWL are associated with unfavorable long-term outcomes, i.e. larger increase in obesity measures and a higher risk of developing T2D. However, whether dieting per se induces these consequences or whether dieting represents susceptibility to developing such conditions for other reasons remains unresolved. A more detailed summary of the conclusions is presented as follows:

1. Dieting attempts and IWL were associated with several factors cross-sectionally. Of women, 39% reported dieting attempts and 15% reported IWL during the previous year, while the corresponding values among men were 24% and 10%, respectively. In addition to female sex, dieting was associated with younger age, higher education, higher BMI, healthy lifestyle factors, former smoking, unfavorable serum lipid concentrations, a poorer sense of coherence, concerns about one's appearance, and concerns about one's health.

Moreover, women had attempted to diet regardless of their weight, while among men, the prevalence of dieting grew strongly in parallel with BMI. Further, diabetes and knee or hip osteoarthritis diagnoses, as well as short habitual sleep, were associated with dieting only among men. It appears that men do not attempt dieting until they have actual health-related reasons, whereas medical reasons are not so strongly associated with dieting in women. These factors related to dieting should be taken into account when studying the associations between self-report dieting and health-related outcomes.

2. Dieting modified the association between quality of diet and obesity. Quality of diet was directly associated with overweight and obesity in the whole sample. In a subgroup of non-dieters, no association appeared, but among dieters, however, a tentatively positive association emerged. As dieting seems to modify the association between quality of diet and obesity, in order to avoid biased results, dieting status should be taken into account when studying the associations between dietary factors and obesity, and further, potentially when studying the associations between dietary factors and the incidence of chronic diseases.



3. Dieting was associated with subsequent unfavorable changes in BMI and WC. During an 11-year follow-up, BMI and WC increased more among dieters than non-dieters and more among those with previous weight loss or weight fluctuation than those with stable weight. This suggests that in the long term, at the population level, self-implemented dieting attempts are susceptible to failure, and if temporary weight loss occurs, maintenance of this lost weight is likely to fail.

The increase in BMI and WC was greater in dieters with initial normal weight than in non-dieters with normal weight. In contrast, among individuals with initial obesity, no differences between dieters and non-dieters emerged. This suggests that among individuals with obesity, dieting does not inevitably increase the risk of greater weight gain, but among individuals with initial normal weight, however, unnecessary dieting attempts may predispose to a greater risk of future weight gain, or alternatively, represent susceptibility to future weight gain due to other reasons.

4. IWL was associated with an elevated risk of incident T2D during a 15-year follow-up. The positive association found may be due to that IWL often fails at the population level in the long term. Failed IWL may result in weight regain beyond the initial level, in repeated weight loss attempts inducing weight cycling, and in unfavorable changes in metabolic health indicators. Alternatively, the possibility that individuals with IWL may initially be at a greater risk of T2D cannot be fully excluded.

Moreover, the risk was indicatively pronounced in certain subgroups including men, younger individuals, individuals with a lower educational level, individuals with obesity, individuals with low PA, non- and moderate alcohol consumers, never-smokers, individuals with low reported energy intake, individuals with poor quality of diet, and individuals with short sleep duration. The risk being accentuated in individuals with many unhealthy lifestyle factors implies that IWL conducted with an unwholesome lifestyle in particular is prone to fail, and thus lead to an increased risk of T2D.

5. In general, despite the unfavorable associations found in the substudies of this thesis, among individuals with severe obesity or other medical needs for weight loss, dieting should not be avoided, and the benefits of successful weight loss should not be forgotten. Instead, the importance of long-term lifestyle changes (including healthy diet, enough PA and enough sleep) to accomplish weight loss and sustained weight maintenance should be emphasized. As failed weight loss, however, seems to be associated with disadvantageous consequences, unnecessary dieting in the absence of real medical reasons for it, should be avoided.

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