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Genetic and environmental influences on body image, disordered eating, and intentional weight loss

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Doctoral dissertation

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Contents

Abstract

Aims

Many previous studies have shown that body size is strongly genetically determined. The rapid global increase in rates of obesity implies that environmental influences must also be important. In concert, disordered eating and eating disorders have become increasingly widespread in Western countries during the second half of the 20th century. However, relatively little is known about genetic and environmental contributions to individual variation in weight control. The aim of this study was to explore the genetic and environmental contributions to breakfast skipping, body weight and shape concerns, and intentional weight loss attempts in a large population sample of young Finnish twins.

Methods

The participants, five birth cohorts of Finnish twins born 1975-79, were followed longitudinally from the age 16 to age 22-27. At baseline, the health habits of the twins were assessed using self-report questionnaires that included questions on weight, height, sociodemographics, various health habits, and breakfast skipping frequency. Simultaneously, similar information was obtained by a behavioral and medical self-report questionnaire from the twins' mothers and fathers. The twins received three follow-up questionnaires at ages 17, 18, and 22-27. The final fourth questionnaire assessed weight, height, intentional weight loss, eating styles, and many other nutrition-related variables. Body shape and weight concerns were measured using the Body Dissatisfaction and Drive for Thinness subscales from the Eating Disorder Inventory. Demographic and behavioral correlates of breakfast skipping, body shape and weight concerns, and intentional weight loss were analyzed using cross-tabulations and univariate and multivariable logistic regression models. The family-based sampling was taken into account in all analyses. Different structural equation modeling strategies were used to estimate genetic and environmental influences on body image and weight control patterns in twins; twin-family modeling was used to explore breakfast eating patterns in twins and their families.

Results

Parental breakfast skipping was the strongest factor associated with adolescent breakfast skipping. In both adults and adolescents, health-compromising factors, such as smoking, infrequent exercise, frequent alcohol use, and a high BMI, were significantly associated with breakfast skipping. Breakfast skipping was associated with low family socioeconomic status in adults and adolescent boys, but not in girls. Additive genetic effects explained 41% (95% confidence interval [CI]: 21-63%) of the variance in breakfast skipping patterns in girls and 66% (95% CI: 47-79%) in boys, and common environmental effects 45% (95% CI: 23-62%) in girls and 14% (95% CI: 5-29%) in boys. Twin-family models confirmed that there are substantial additive genetic and shared environmental influences in addition to individual-

specific environmental effects and gender differences.

With regards to body shape and weight concerns, various psychosomatic symptoms were significantly associated with high Body Dissatisfaction (BD) and Drive for Thinness (DT) in both genders. In women, early onset of puberty, early initiation of sexual activity, multiple sex partners, and lower educational attainment at age 16-17 were statistically significant risk factors of BD experienced in young adulthood. In gender-specific univariate twin models, additive genes accounted for 59% (95% CI: 53-65%) of variance in BD and 51.0% (95% CI: 44-58%) of DT in females, but for none in males.

Individuals engaging in intentional weight loss (IWL) attempted to restrict food intake and avoid fatty and calorie-rich foods, but simultaneously exhibited disordered and unhealthy eating patterns. Snacking and eating in the evening were characteristic of individuals with at least two IWL attempts. Eating in response to visual and emotional cues was very pronounced in women who had engaged in IWL, but much less so in men. IWL was estimated to have a heritability of 38% (95% CI: 19-55%) in men and 66% (95% CI: 55-75%) in women. The overlap in genetic effects on BMI and IWL was 0.38 (95% CI: 0.28-0.47) for men and 0.45 (95% CI: 0.41-0.52) for women, implying that genetic effects that affect BMI are only partially shared with those affecting IWL.

Conclusions

Weight control related issues have general health implications: breakfast skipping was associated with health-compromising behaviors. Body shape and weight concerns were associated with larger body size and multiple psychosomatic symptoms. Intentional weight loss attempts were also associated with larger body size and disordered eating styles, particularly restricting and overeating.

Overall, both genes and environment were important for the eating-related phenotypes studied in Finnish young adult twins. Breakfast skipping in girls and boys, intentional weight loss in women and men, and body shape and weight concerns in women exhibited moderate to strong genetic influences. Body shape and weight concerns exhibited substantial and breakfast skipping modest influences of shared family environment. The genetic influences on intentional weight loss were only partially shared with those affecting BMI.

List of original publications

I Keski-Rahkonen A, Kaprio J, Rissanen A, Virkkunen M, Rose RJ. Breakfast skipping and health-compromising behaviors in adolescents and adults. Eur J Clin Nutr 2003; 57: 842-53.

II Keski-Rahkonen A, Viken RJ, Rissanen A, Kaprio J, Rose RJ. Genetic and environmental factors in breakfast eating patterns. Behavior Genetics 2004; 34:503-514.

III Keski-Rahkonen A, Bulik, CM, Neale BM, Rose RJ, Rissanen A, Kaprio J. Body dissatisfaction and drive for thinness in Finnish young adult twins. International Journal of Eating Disorders (in press).

IV Keski-Rahkonen A, Neale BM, Bulik CM, Pietiläinen K, Rose RJ, Kaprio J, Rissanen A. Intentional weight loss in young adults: sex-specific genetic and environmental effects (submitted).

Abbreviations

A	additive genetic effects
ACE	a structural equation model that consists of three parameters: additive genetic effects, common environmental effects, and non-shared environmental effects
ADE	a structural equation model that consists of three parameters: additive genetic effects, dominance effects, and non-shared environmental effects
BD	body dissatisfaction
BMI	body mass index
χ^2	chi-squared
C	common environmental effects
CI	confidence interval
D	dominance effects
DT	drive for thinness
DZ	dizygotic twins
E	non-shared environmental effects
EDI	Eating Disorder Inventory
GHQ	General Health Questionnaire
h^2	heritability
IWL	intentional weight loss
MZ	monozygotic twins
OR	odds ratio
SE	standard error
SES	socioeconomic status

1. Introduction

> "Food is the new sex."
>
> Anonymous magazine headline, circa 2001

> "Fat can be fatal. Obesity is the great new global health scare.... The danger is baffling because it is paradoxical. For ours is the most diet-conscious era and diet-obsessed culture in the history of the world. We think thin and we get fat."
>
> Felipe Fernandez-Armesto, The Guardian, Sept 14, 2002

The current Western popular culture sends powerful and conflicting messages about food and weight. On one hand, indulgence is encouraged: celebrity chefs, lushly edited coffee-table cookbooks, magazine extras, and food commercials promote the sinful goodness of sugar and fat, backed by multibillion marketing budgets and governments grappling with agricultural overproduction (Schlosser 2002; Knapp 2003; Critser 2003). On the other hand, it is common knowledge that overindulgence results in overweight and obesity, implying serious health risks (Pi-Sunyer 1991) and social stigma (Harris 1983; Turnbull et al. 2000).

Over the past millennia, the relative scarcity of highly nutritious food may have given an evolutionary advantage to individuals with "thrifty genes", ie, the capability of effective long-term energy storage (Neel 1962; Pijl 2003). The very recent and dramatic changes in the living environments of Western countries have turned our societies highly obesogenic. If genes load the gun and the environment pulls the trigger, the question of obesity poses a double-barreled challenge. The genetic propensity for obesity is fed by the environment. The result emerges as a rapidly unfolding obesity epidemic.

In this obesogenic and fat-phobic society, weight control strategies assume paramount importance in people's lives. The market for quick fixes to weight problems is ever expanding, but there are few good long-term weight loss solutions. The discrepancy between ideals and reality causes much psychological distress (Lautenbacher et al. 1992; Rierdan & Koff 1997). Even an ascetic and disciplined attitude toward food is not necessarily safe: restrictive eating attitudes can become severe clinical eating disorders, or encourage disordered eating, such as overeating and unhealthy weight control methods which may increase the risk of obesity.

It is not exactly easy to grow up in this conflicting cultural climate. Moreover, as traits predisposing to obesity are the result of genes, environment, and the interaction of genes and environment, individuals with certain genetic make-ups seem to be more vulnerable to obesogenic environmental influences than others. Thus there is an increased need to explore genetic and environmental contributions to various weight and eating related phenotypes.

This study focuses on adolescents and young adults who are trying to come to terms with different aspects of eating patterns, body image, and weight control. Methods of genetic epidemiology, particularly twin analyses, as well as those of classical epidemiology are implemented in studying genetic, environmental, and behavioral correlates of disordered eating (here exemplified by breakfast skipping), body shape and weight concerns, and intentional weight loss attempts.

2. Literature Review

Overview

The weight regulation mechanisms in humans are highly asymmetrical: various pathways of appetite control render weight loss difficult, but safeguards against weight gain are few (Blundell & Gillett 2001; Flier 2004). This asymmetry makes many individuals in our current environment vulnerable to obesity. The risk of obesity is often addressed by weight control behaviors. Sometimes, however, weight control behaviors exacerbate rather than solve problems: dieting attempts may spiral out of control, resulting in clinical eating disorders, or in overweight.

The genetic and molecular basis of weight control is very poorly understood. Interindividual variation in weight control behaviors is considerable, probably because these behaviors are a result of complex genetic and environmental interplay. Yet a deeper understanding of these behaviors might give us more effective means of combating the obesity epidemic.

In the following three sections, three weight control behaviors, disordered eating (as exemplified by breakfast skipping), body shape and weight concerns, and intentional weight loss attempts, are defined and quantified. Second, existing research into demographic and behavioral correlates of these behaviors is reviewed. Third, what is already known about the genetics of various weight control related behaviors is briefly summarized.

Definitions and prevalences

Disordered eating

The term *disordered eating* emerged in medical and psychological literature in the late 1970s, coinciding with the introduction of diagnostic criteria for bulimia nervosa (Russell 1979). *Disordered eating* was first used to describe dietary chaos and emotional instability experienced during recovery from anorexia nervosa (Palmer 1979). Soon, the term was used more loosely to describe young women, who "…diet at some time and lose more than 3 kg in weight; […] may experience episodes of binge eating and "picking" behavior; […] wish to be thinner irrespective of their current body weight [,] and abuse laxatives or diuretics in order to achieve a fashionably slim Figure (Abraham et al. 1983)." Another early study defined disordered eating as "bingeing, highly restrictive dieting, emotional eating, or purging (Kagan & Squires 1984)."

Although the concept still lacks uniform definition, it is generally used to describe disordered eating behaviors that are more broad and benign than eating disorders defined in ICD-10 and DSM-IV classifications. In contrast to these diagnostic classifications, milder forms of disordered eating are often not considered illnesses worthy of medical attention, although they are relatively common among adolescents and young adults in the general

population. In a national survey conducted in the U.S., 13% of girls and 7% of boys reported disordered eating, defined as self-reported binge-purge cycling (Neumark-Sztainer & Hannan 2000). In a Finnish nationwide school health survey, loosely defined bulimic-type eating behavior was reported by 16.5% of girls and 12.3% of boys, although discrepancies in how the study subjects understood the concept bingeing may possibly have inflated these prevalence estimates (Beglin & Fairburn 1992). The prevalences of these behaviors may be higher still in some particularly vulnerable subgroups, such as dancers (Abraham 1996), athletes (Johnson 1994), vegetarians (Neumark-Sztainer et al. 1997b), and patients with juvenile-onset diabetes (Neumark-Sztainer et al. 1996b; Rydall et al. 1997).

Breakfast skipping

Breakfast skipping, a commonly used weight control practice among adolescents (Neumark-Sztainer & Story 1998) and adults (Levy & Heaton 1993), can also be considered a mild form of disordered eating – it commonly coexists with body shape and weight concerns and other symptoms of subclinical eating disorders (Melve & Baerheim 1994; Pastore et al. 1996). If present in early adolescence, it may constitute a risk factor or an early manifestation of eating disorders (Fernandez-Aranda et al, manuscript in preparation).

Breakfast skipping has been of interest for many researchers in the recent years, perhaps because it is a question often addressed in large-scale population surveys. In scientific literature, there is much more information on breakfast skipping than skipping lunch or dinner, perhaps because a morning meal is more homogenous across cultures and populations than the other main meals of the day. Skipping one of the daily main meals also predisposes to skipping another meal: breakfast and lunch/dinner skipping often coexist (Urho & Hasunen 1999; Sjöberg et al. 2003). Breakfast skipping has been variously defined in different studies: usually it means the omission of the first morning meal, intended to be consumed at home. Sometimes specific time points are also used. As eating habits vary greatly in different countries and cultures, comparisons are often difficult.

Breakfast skipping has become increasingly widespread among children, adolescents and adults in Western countries during past 30 years (Haines et al. 1996; Nicklas et al. 1998; Siega-Riz et al. 1998). Lifestyle changes may play a role in this trend. In Finland, breakfast used to be a substantial, cooked meal (Prättälä & Roos 1999). As majority of women now have full-time careers, there is less time and opportunities for family meals, and the traditional three-meal pattern has changed into a pattern of one or two daily meals (Prättälä & Roos 1999). Breakfasts have become lighter, consisting most often of bread, cheese, coffee or tea, and contributing typically less than 20% of the daily energy intake (Kleemola et al. 1997). In the most recent FINRAVINTO survey, breakfast accounted for 16% of the daily energy intake in adult men and women (Männistö et al. 2003). When adolescents were asked why they skip breakfast, "lack of time" or "not hungry" were the reasons supplied by the majority of them (Shaw 1998; Urho & Hasunen 1999). Many Finnish adolescents may skip breakfast simply because they will have an early school lunch or may purchase a snack whenever it feels convenient.

Prevalences of breakfast skipping in adolescents are detailed in Table 1. In studies conducted in different countries, estimates of breakfast skipping among adolescents have ranged from 3% to 66% (Terre et al. 1990; Neale & Cardon 1992; al Sudairy & Howard 1992; Isralowitz & Trostler 1996; Samuelson et al. 1996; Brugman et al. 1998; Höglund et al. 1998; Shaw 1998; Siega-Riz et al. 1998; Urho & Hasunen 1999; Cavadini et al. 2000; Murata 2000; O'Dea & Caputi 2001; Abalkhail & Shawky 2002). Some of this great variability is due to age and gender effects, but methodological and cultural differences are a more likely source of

Table 1. Prevalence of breakfast skipping in adolescents

Age	N	% breakfast skippers	Study setting	Author	How was breakfast skipping measured?
14-15	7605	20-32%	Göteborg, Sweden	Höglund	"less than 3 days a week"
16-18	not reported, about 250	54%	Missouri, USA	Terre	"usually skip breakfast"
15-18	1513	47-66%	Israel	Isralowitz	not detailed
15-18	5243	35%	Bogalusa, LA, USA	Siega-Riz	"consumption of food, beverage or both between 0500 and 1000", 24-h recall
13-15	876	13%	The Netherlands	Brugman	24h recall
13	699	12%	Australia	Shaw	questionnaire, not detailed
12-15	800	15%	Jeddah City, Saudi Arabia	Abalkhail	questionnaire, not detailed;
16-18	114	20%	Riyadh, Saudi Arabia	al Sudairy	questionnaire, not detailed; males only
15	411	5%	Trollhättan and Uppsala	Samuelson	"less than 5 x/week", 7-day dietary record
16-19	419	3%	Lorraine, France	Michaud	2-day dietary record
13-16	3248	15%	Finland (12 municipalities)	Urho	"24-h recall"
7-14	the boys in Tokyo metro area	4%	Tokyo, Japan	Murata	not detailed
15-19	the boys in Tokyo metro area	18%	Tokyo, Japan	Murata	not detailed
6-12	466	8% girls, 10% boys	New South Wales, Australia	O'Dea	"whether on most days they usually consumed breakfast"
12-19	660	24% girls, 13% boys	New South Wales, Australia	O'Dea	"whether on most days they usually [did not] consume breakfast"

variability. For instance, some studies assess breakfast eating using 24-hour dietary recall; others use questionnaire items that are much more vaguely defined. All studies are liable to self-report bias.

Considerably fewer studies have assessed breakfast skipping in adult populations. A Finnish study published almost a quarter-century ago found that 23% of Finnish adult women and 33% of adult men reported skipping breakfast (Puska & Smolander 1980); in 2003, 17% of Finnish adult women and 21% of adult men reported breakfast skipping (Helakorpi et al. 2003). In a similar large population study, conducted in 1987-98 in the Netherlands, 34% of women and 38% of men skipped breakfast. In contrast, in a recent 24-h dietary recall based population study of American adults (Ma et al. 2003), only 3.6% of participants were classified as breakfast skippers. However, in that study, 18.9% of the breakfasts were eaten away from home, usually on the way to work or school, reflecting perhaps a fast food lifestyle more typical of the U.S. than Northern Europe. In many self-report questionnaire assessments of breakfast eating, a morning meal eaten away from home is either implicitly or explicitly defined as a snack, not breakfast (Gatenby 1997).

Body shape and weight concerns

In modern Western societies, high-energy food is abundant, and the necessity for physical activity is decreasing. Body shape ideals for women and men are gravitating towards those that are the most difficult to obtain and maintain: thinness, leanness, and muscularity (Gordon 2000; Morgan 2000). The discrepancy between ideals and reality results in body shape and weight concerns, evident early in childhood (Sands et al. 1997; Schur et al. 2000) and widespread in adolescents and young adults (Moore 1988; Heatherton et al. 1997; Jaeger et al. 2002).

A variety of approaches have been used to measure body shape and weight concerns. Sometimes assessment by a simple question is deemed sufficient. A more indirect approach is to contrast actual body weight and shape to ideal body weight and shape; for instance, an array of silhouettes of varying degrees of muscularity and leanness can be used (Bulik et al. 2001). Of self-report measures, the Eating Disorder Inventory (EDI) (Garner & Olmsted) Body dissatisfaction (BD) and Drive for Thinness (DT) subscales (Table 2) are widely used and well-validated.

The risk of body and weight dissatisfaction increases with increasing body mass, particularly in the presence of strong thinness idealization, teasing and bullying, or if there is external pressure to be thin (Stice & Whitenton 2002; van den Berg et al. 2002). Nevertheless, many people who are not overweight are still dissatisfied with their bodies. In a large (N=19,841) and representative sample of Canadian adults, 32% of women and 10% of men who were at normal weight (BMI 20-24) were still trying to reduce their weight (Green et al. 1997). In a national survey conducted in the US, 47% of women currently trying to lose weight had a body mass index under 25 (Biener & Heaton 1995). Similarly, in a school-based

Table 2. Three subscales of Eating Disorder Inventory 1 (Garner 1991). All items were rated on a 6-point Likert scale (always, usually, often, sometimes, rarely, never).

Drive for Thinness
I eat sweets and carbohydrates without feeling nervous.
I think about dieting.
I feel extremely guilty after overeating.
I am terrified of gaining weight.
I exaggerate or magnify the importance of weight.
I am preoccupied with the desire to be thinner.
If I gain a pound, I worry that I will keep gaining.
Bulimia
I eat when I am upset.
I stuff myself with food.
I have gone on eating binges where I felt that I could not stop.
I think about bingeing (overeating).
I eat moderately in front of others and stuff myself when they are gone.
I have the thought of trying to vomit in order to lose weight.
I eat or drink in secrecy.
Body dissatisfaction
I think that my stomach is too big.
I think that my thighs are too large.
I think that my stomach is just the right size.
I feel satisfied with the shape of my body.
I like the shape of my buttocks.
I think my hips are too big.
I think that my thighs are just the right size.
I think my buttocks are too large.
I think that my hips are just the right size.

survey of Finnish adolescents aged 14-16 (N=60,252) 46% of girls and 34% of boys were not satisfied with their weight; among normal-weight adolescents dissatisfied with their weight, 81% of girls and 48% of boys thought they were overweight (Mikkilä et al. 2003).

Body dissatisfaction is a risk factor for eating disorders and disordered eating, particularly bulimic behavior patterns; the risk is probably mediated through dieting and negative affect (Stice 2001; Stice & Shaw 2003). However, at least in young women, dissatisfaction with body weight and shape is currently so widespread that it is almost normative. Estimates of rates of body dissatisfaction vary with measures used. In a Southern Italian study of students from secondary schools, 58.4% of girls and 19.7% of boys displayed dissatisfaction with regard to their own body (Dalla Grave et al. 1997). Of Norwegian students of comparable age, 37% of girls and 20% of boys reported that they felt "a bit or much too fat" (Borresen & Rosenvinge 2003). Body shape and weight ideals are also strongly culture dependent. In the European context, the most extreme body dissatisfaction is evident in Northern Mediterranean countries, followed by Northern European countries (Jaeger et al. 2002). Levels of body and weight dissatisfaction are intermediate to high in women living in cultures currently undergoing westernization (Abdollahi & Mann 2001; Becker et al. 2002; Tsai et al. 2003; Sarlio-Lähteenkorva et al. 2003). Women in non-western countries demonstrate rather low levels of body dissatisfaction (Jaeger et al. 2002). However, there is some evidence from Finland that although obesity and overweight are becoming increasingly common, the level of weight dissatisfaction in the general population of adolescents is actually decreasing (Kaltiala-Heino et al. 2003a).

Intentional weight loss

Body shape and weight concerns are inextricably associated with weight loss attempts. In this study, we focus on *weight loss*, because it is less vague and easier to quantify than *dieting*. *Intentional weight loss* is often used to make a distinction to *unintentional* weight loss, such as weight loss resulting from malignancies, endocrine abnormalities, or severe depression (French et al. 1995a; Meltzer & Everhart 1996; Williamson 1997).

In an American population study conducted in 1989, approximately 40% of women and 25% of men were trying to lose weight; the average weight loss goal was -14 kg (Williamson et al. 1992); more recently, 44% of women and 29% of men reported attempting to lose weight (Serdula et al. 1999).

Surprisingly little is known about factors that motivate weight loss and means taken to lose weight. In the Weight Loss Practices Survey (Levy & Heaton 1993), conducted among a representative sample of adults in 1992 in the U.S., 33% of women and 20% of men were currently trying to lose weight. Weighing oneself regularly was the most common currently used means of weight loss (reported by 71% of women and 70% of men, respectively), followed by walking (58% and 44%), using diet soft drinks (52% and 45%), taking vitamins and minerals (33% and 26%), counting calories (25% and 17%), and participating in organized weight-loss programs (13% and 5%). Less healthful practices that also reflect the population prevalence of disordered eating in adults included skipping meals (21% and 20%), taking diet pills (14% of women and 7% of men), fasting (6% in both sexes), misuse of laxatives (3% of women and 1% of men), use of dieting devices (1% in both sexes), and vomiting (1% of women, 0% of men). In another and more recent large American study (Neumark-Sztainer et al. 2000), 57% of adult women, 50% of adult men, 44% of adolescent girls, and 37% of adolescent boys reported current weight control behaviors. Disordered eating behaviors included meal skipping (in 19% of women and men, 23% of girls, and 14% of boys currently trying to control their weight), fasting (3% of women and men, 8% of girls, 7% of boys), diet

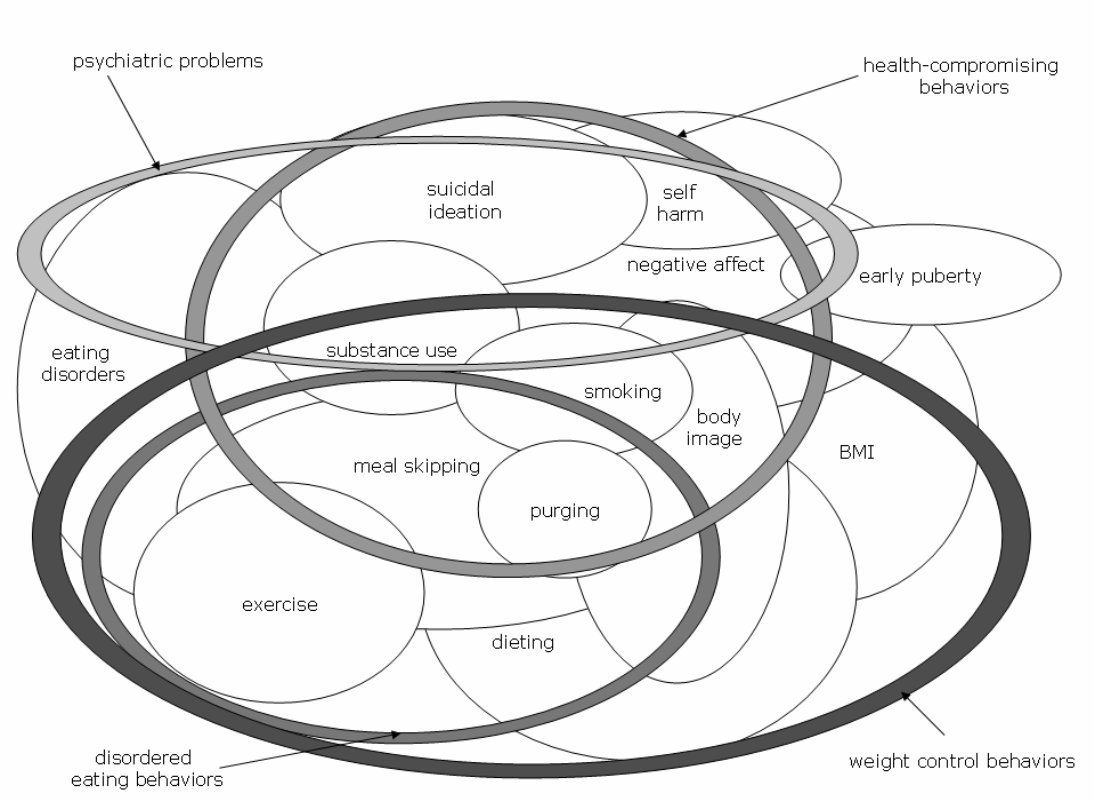
pills or laxatives (7% of women, 3% of men, 4% of girls, 3% of boys), and vomiting (<1% of women and men, 7% of girls, 2% of boys). Thus it seems that a substantial minority of individuals trying to control their weight engage in questionable weight control practices that fall within the boundaries of disordered eating.

Although many people report that health concerns or health improvement are their primary motivation for weight loss, particularly if they are overweight, dieting for appearance reasons in the absence of overweight is very common, as noted above, particularly among young women (Levy & Heaton 1993; Biener & Heaton 1995). Paradoxically, weight loss attempts are rarely successful, and "weight cycling" (repeated weight loss and weight gain) may have harmful consequences (Brownell & Rodin 1994; Weight cycling. National Task Force on the Prevention and Treatment of Obesity 1994). Women also generally have lower BMI goals than men, and normal-weight individuals have lower BMI goals than overweight ones (Anderson et al. 2003).

Weight control behaviours: correlates and demographic determinants

Operating on the hypothesis that weight control behaviors may also have harmful, undesired consequences, numerous studies have explored the relationship of weight control behaviours and health-compromising behaviors. There is considerable common interface particularly between weight control behaviors and psychological and psychiatric problems (Figure 1).

Figure 1. Schematic drawing of associations of weight control behaviors: although weight control is often considered healthful, some psychiatric and psychological problems and health-compromising behaviors, particularly substance use, are sometimes associated with weight control.



Below, a more detailed overview focuses on three core areas of importance for this study, disordered eating (with particular focus on breakfast skipping), body shape and weight concerns, and intentional weight loss.

Disordered eating and peripubertal problems

Obesity in childhood and adolescence and subsequent body dissatisfaction are well-known risk factors of eating disorders and disordered eating (Fairburn et al. 1997; Ackard & Peterson 2001). They are particularly important precursors of bulimic behavior patterns; the risk is probably mediated through dieting and negative affect (Stice 2001; Stice & Shaw 2003). A common thread of these problems seems to be their peripubertal manifestation. Adolescents who experience puberty earlier than their peers seem to be at higher risk for body dissatisfaction, eating disorders, meal skipping, and many psychological problems (Fairburn et al. 1997; Kaltiala-Heino et al. 2001; Sjöberg et al. 2003; Kaltiala-Heino et al. 2003b).

Abnormal eating attitudes and disordered eating have been found to cluster with problem behavior, eg, tobacco, alcohol, substance use, and risk-taking in general (Fisher et al. 1991; Chandy et al. 1994; Chandy et al. 1995; Neumark-Sztainer et al. 1997c; von Ranson et al. 2002). Suicidal attempts, self-harm, delinquency, school problems, sexual abuse, date rape and violence, and high-risk sexual behaviours are also associated with disordered eating (Neumark-Sztainer et al. 1996a; Neumark-Sztainer et al. 1998; Ackard & Peterson 2001; Wonderlich et al. 2001; Ackard & Neumark-Sztainer 2002). In addition to suicidal behaviours, also other psychosomatic symptoms are more common in individuals with disordered eating attitudes (Buddeberg-Fischer et al. 1996). In fact, preoccupation with weight and/or dieting concerns in male and female adolescents is likely to indicate psychological problems (Casper 1998; Neumark-Sztainer & Hannan 2000). In a population sample of 2525 Australian teenagers, 7% of girls and 1% of boys fell into a group of extreme dieters: of them, 62% reported high levels of depression and anxiety; these levels are fully comparable with the psychiatric comorbidity of clinical eating disorders.

Weight dissatisfaction seems to be more prevalent in girls from low SES families, at least in a large (N=60,252) Finnish school-based survey (Mikkilä et al. 2003). Similarly, weight dissatisfaction was associated with more smoking in girls; smoking is perhaps used as a chosen method of weight control. Interestingly, health behaviours seems to be associated more with perceived weight/weight satisfaction than with actual body weight (Mikkilä et al. 2003).

Behavioral and demographic correlates of breakfast skipping

Meal skipping shares the timing of peripubertally initiated disordered eating behaviors: perhaps reflecting differential parental supervision of the morning meal, children in primary school are usually at a lower risk than teenagers (Brugman et al. 1998; Nicklas et al. 1998; Murata 2000; O'Dea & Caputi 2001). Similarly than other disordered eating behaviors, breakfast skipping is associated with health-compromising behaviors, such as smoking (Höglund et al. 1998; Sjöberg et al. 2003) and alcohol and drug use (Isralowitz & Trostler 1996). Conversely, regular breakfast eating has been associated with a health-conscious lifestyle and regular exercise (Cavadini et al. 2000).

Probably the most widely researched health risk associated with breakfast skipping is obesity and related weight control issues. Individuals who skip meals tend to compensate by snacking (Urho & Hasunen 1999; Sjöberg et al. 2003). Meal-skippers seem to end up with less healthful food choices, significantly lower intakes of micronutrients, poorer overall nutrient intake, but higher intakes of sucrose and alcohol compared to the groups with regular meal

intake (Cho et al. 2003; Sjöberg et al. 2003). Given that meal skipping is often associated with a sedentary lifestyle (Terre et al. 1990; Baumert, Jr. et al. 1998; Boutelle et al. 2002), it has been hypothesized that meal skipping, particularly breakfast skipping, may be a risk factor of obesity. Many cross-sectional studies have observed this association (Kaufmann et al. 1975; Wolfe et al. 1994; Nordlund & Jacobson 1999; Boutelle et al. 2002; Cho et al. 2003), but this finding has not been confirmed by longitudinal studies (Berkey et al. 2003).

Some experts have recommended breakfast skipping to obese individuals because it may limit total daily energy intake (Schlundt et al. 1992). Indeed, baseline breakfast eaters lost more weight if they started skipping breakfast rather than continued to have it. Baseline breakfast skippers, however, lost more weight if they started to have regular morning meals (Schlundt et al. 1992). Several other studies have successfully used the introduction of regular breakfasts as a weight loss intervention, hypothesizing that regular meals limit excessive energy intake from impulsive snacks (Mattes 2002; Falkenberg & Hellman 2002; Wyatt et al. 2002). Indeed, regular breakfast eating, rather than breakfast skipping, seems to be characteristic of individuals who can maintain weight loss over long periods of time (Wyatt et al. 2002).

Despite these findings, meal skipping remains a popular weight control strategy (Levy & Heaton 1993; Bellisle et al. 1995; Neumark-Sztainer & Story 1998). Some studies suggest that adolescent girls may be more likely to diet by skipping breakfast or lunch and by decreasing their meal size; boys, on the other hand, tend to limit snacking and high-energy foods in order to lose weight (Brugman et al. 1997; Nowak 1998; Shaw 1998). However, boys who were encouraged to diet by their mothers were at risk for health-compromising eating and dieting behaviors, particularly skipping meals, fasting, and binge-eating (Fulkerson et al. 2002). In other studies, adolescent girls who skipped meals were more likely to diet and expressed greater levels of body shape and weight dissatisfaction (Shaw 1998; Mikkilä et al. 2003). In Norway, 15-19-year-old girls with subclinical eating disorders skipped breakfast more often than healthy control subjects (Melve & Baerheim 1994). Meal skipping is often an early manifestation of an eating disorder, and if present before puberty, a risk factor of anorexia (Fernandez-Aranda, in preparation).

Irregular meal patterns have traditionally been associated with food insecurity and deprivation. Following the general patterns of SES and food behavior (Roos et al. 1998; Irala-Estevez et al. 2000; Robinson et al. 2004), family SES and educational attainment are clearly linked with breakfast eating patterns. In Western countries, several previous studies have found and association between breakfast skipping and low socioeconomic status (Pastore et al. 1996; Brugman et al. 1998; Höglund et al. 1998; Nordlund & Jacobson 1999; O'Dea & Caputi 2001; Robinson et al. 2004), although this finding is not consistent (Walker et al. 1982; Brugman et al. 1998; Höglund et al. 1998; Nordlund & Jacobson 1999; O'Dea & Caputi 2001). In developing countries and in countries where income inequality is large, poverty has been the most important reason for children and adolescents to skip breakfast (Melnik et al. 1998; Tzimis & Kafatos 2000; Gross et al. 2004). Given that multiple studies link cognitive problems (decreased ability to concentrate and memory problems) to missing breakfast, special school breakfast programs and other community-level interventions have been implemented in many low-income settings (Murphy et al. 1998; Pollitt & Mathews 1998; Powell et al. 1998). However, in Finland and other affluent societies, lack of economic resources in itself does probably not explain breakfast skipping.

Weight control behaviours

As already discussed above, weight control behaviours are associated with disordered eating and body shape and weight concerns. Although it would be natural to assume that weight

control behaviours are triggered by overweight and obesity, they behaviors are actually relatively common among normal-weight individuals, especially women (Strauss 1999; Wardle & Johnson 2002). Maladaptive weight control strategies are particularly common among low SES women: the higher risk of obesity and family exposure to these behaviors places women at increased risk of unhealthy behaviors (Breitkopf & Berenson 2004).

Adolescents' perceptions of direct pressure from their parents to diet has been found to be a significant predictor of dieting, and perceived parental encouragement of autonomy, and self-confidence were associated with less dieting behaviour (Huon & Strong 1998). Adolescent dieters have poorer self esteem than their non-dieting counterparts (Pesa 1999).

Like body shape and weight concerns, weight loss has been linked with depression and low mood. This probably has a physiological basis: extreme starvation has severe psychological consequences, such as depression and anxiety (Keys et al. 1950), and experiments of extreme weight loss have triggered low mood, heightened irritability, difficulties concentrating, and fatigue (Laessle et al. 1996). In some studies, weight cycling (ie, repeated weight gain and weight loss) has been associated with psychological problems (Venditti et al. 1996). Mostly, this link has been refuted, and it may be that the psychological discomfort is mediated rather by binge eating than dieting or by the individuals perception of being a weight cycler rather than actual weight loss (Telch & Agras 1994; Bartlett et al. 1996; Foster et al. 1996; Venditti et al. 1996; Foster et al. 1997; Friedman et al. 1998; Kensinger et al. 1998). Severe binge eating is often associated with severe comorbid psychopathology (Grilo et al. 2001). However, many recent studies confirm that in nonclinical samples, the rates of psychopathology do not appear significantly greater in obese than nonobese individuals (Wadden & Stunkard 1985; Stunkard & Wadden 1992; Fitzgibbon et al. 1993), even though obesity often carries severe social stigma and is associated with a low degree of life satisfaction (Rosmond & Björntorp 1998).

Very little is known about behavioural correlates of weight loss on the population level. A meta-analysis of mostly observational studies of structured weight loss programs in the U.S. revealed that the results were relatively modest at best: five years after completing the programs, the average individual maintained a weight loss of about 3 kg (Anderson et al. 2001). In a Finnish study, only 6% of all overweight individuals lost at least 5% of their weight and maintained this weight loss over nine years (Sarlio-Lähteenkorva et al. 2000).

It seems that only extreme behavioural modifications yield long-term weight maintenance after weight loss: usually this entails continued attention to body weight, low levels of stress, consumption of a low-energy and low-fat diet, and continued physical activity (Klem et al. 1997; Shick et al. 1998; Sarlio-Lähteenkorva et al. 2000). It is also possible that long-term weight-maintainers experience higher levels of anxiety than obese individuals who do not attempt weight loss (Sarlio-Lähteenkorva & Rissanen 1998). Weight-loss maintainers seem to use more cognitive and behavioral strategies to control their weight than weight-regainers or weight-stable controls (McGuire et al. 1999a; Dohm et al. 2001). Factors that seem to predict weight regain include very recent and very large weight losses, higher levels of depression, dietary disinhibition, binge eating, and overall stress (McGuire et al. 1999b; Sarlio-Lähteenkorva et al. 2000).

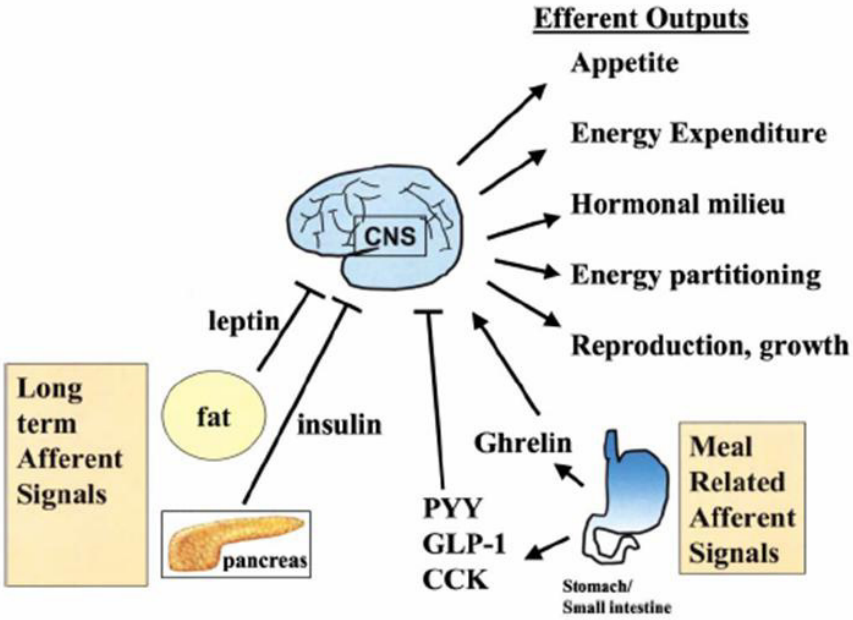
If the long-term success of weight loss is rather pessimistic, it is also unclear whether individuals who self-report dieting actually have a lower energy intake and a higher energy expenditure than their non-dieting counterparts. Although most research in this field has been conducted in experimental studies, a few population studies, all conducted in the U.S., are available. In adolescents, moderate weight control methods seem to be associated with more health-promoting and exercise behaviours than extreme weight control methods (Story et al. 1998). However, in another study, dieting frequency was positively associated with concern about weight and shape, but not with physical activity; energy intake was lower in dieting high

school students than their non-dieting counterparts, but in preadolescents "dieting" did not imply any significant differences in energy intake (Field et al. 1993). Dieting was also associated with unhealthy weight control practices (diet pill, laxative, and vomiting) and psychological changes (higher BD and DT and more frequent alcohol use) (French et al. 1995b). In adults, the association of exercise and dieting varies from one study to another – women dieters may be more likely to exercise than their non-dieting counterparts, but often self-reported dieting makes no difference in energy expenditure (French et al. 1994; Biener & Heaton 1995). Weight loss or weight maintenance attempts were associated with lower percent of energy intake from fat and sweets, more frequent consumption of vegetables and fruits, more frequent self-weighing, and lower tolerance for weight gain prior to taking action (French & Jeffery 1997).

Summary

The great pains and concerns invested in weight control reveal how toxic and obesogenic our current environment is. The huge difficulty of successful weight loss underscores the underlying biologically hardwired asymmetry in weight regulation, which may be compounded by environmental influences, such as socioeconomic disadvantage. Moreover, interindividual variation in weight control behaviors seems great: the ground is not level for different individuals in terms of weight loss. The same amount of energy consumed do not mean the same reasons for different individuals, but the genetic mechanisms responsible for this discrepancy are largely unclear. These issues will be further explored in the next chapter.

Figure 2. Components of the weight regulation system: The brain integrates long-term afferent signals from fat (leptin) and pancreatic beta cells (insulin) with short-term, meal-related afferent signals from the gut, such as inhibitors of feeding (PYY, GLP-1, and CCK), and stimulator of feeding (ghrelin). Efferent outputs regulate appetite, energy expenditure, hormonal milieu, energy partitioning, and the status of reproduction and growth. (Reprinted from Flier, J. S. *Obesity wars: molecular progress confronts an expanding epidemic.* Cell 2004:116, 337-350, with permission from Elsevier.)



Familial aggregation and heritability

Obesity and eating disorders are likely disorders characterized by etiological heterogeneity. The risk of obesity or eating disorders is not equal for all individuals: it is probably based on a complex interplay of individual-specific genetic vulnerability and different environmental exposures. Identical environmental exposures (eg, the same amount of energy consumed) may result in dissimilar outcomes because of genetic variation; conversely, identical genes may also result in dissimilar outcomes in the presence of different environmental influences. Some currently known pathways of weight regulation are briefly introduced in Figure 2. As comparatively little is known about the genetics these pathways, defining eating-related phenotypes and examining them in using twin and family designs is a useful first piece in understanding the relative contributions of genes and environment to weight control behaviors.

Family studies are used to assess whether a trait of interest runs in families. Twin and adoption studies can help to understand the extent to which familial resemblance is caused by genetic influences and what part of the resemblance is environmental in origin. Understanding how genes and environments work together will increase our options to correctly target interventions to modify harmful behavioural patterns, such as obesity and its consequences. This section attempts to review some of the most essential research findings pertaining to genetic influences on eating patterns and energy intake, weight loss and weight change, body shape and weight concerns, and disordered eating.

Studies of familial aggregation of weight control related behaviors have focused on a few specific themes. There are numerous twin and family studies of BMI: all these studies agree that BMI is substantially heritable, with possible age and gender specific effects (for a recent combined analysis from eight twin registries, see Schousboe et al. 2003b; for a review of earlier twin studies of BMI, see Maes et al. 1997).

Genome-wide linkage scans of BMI and obesity are also starting to emerge and have identified chromosomal regions possibly containing predisposing genes, although findings have not been consistent. This is probably due to relatively small sample sizes as well as real differences between populations (Adeyemo et al. 2003; Saar et al. 2003). Some gene mutations giving rise to relatively rare forms of obesity are already known (Farooqi et al. 2003; Branson et al. 2003; Suviolahti et al. 2003; Snyder et al. 2004). A few studies also assess the heritability other measures of body composition, such as truncal and extremity skinfolds, waist circumference, hip, and total body fat %, obtaining estimates that are very much in line with the heritability estimates of BMI (Maes et al. 1997; Rice et al. 1999; Schousboe et al. 2003a).

Genetics of weight change

Little is known about genetic factors that influence weight change. Analyses of genetic influences on weight change, dieting, weight loss, and ultimately permanent weight change are fraught with difficulties. As noted previously, permanent long-term weight loss is difficult to achieve: in a recent Finnish study, only 6% of overweight individuals were able to maintain a 5% loss of body weight over a period nine years or more (Sarlio-Lähteenkorva & Rissanen 1998). Dieting is difficult to operationalize reliably (Neumark-Sztainer et al. 1997a), and weight changes in the general population may have several other plausible explanations, such as pregnancies and diseases, that may be confounded with intentional weight loss attempts. However, a few existing studies have attempted to resolve these difficult issues.

Family analyses conducted in the Quebec Family Study showed that BMI change over 12 years had a heritability of 37%, and change in total subcutaneous fat (as measured by skinfolds) over the same period had a heritability of 16% (Rice et al. 1999). Similarly, extended pedigrees of the Framingham study population revealed that longitudinal changes in body weight change had a heritability of 15%, and body weight change up to age 50 a heritability of 22% (no standard errors reported; Golla et al. 2003), and the heritability of difference between maximum and mean BMI was 23% (95% CI: 9-37%) (Coady et al. 2002). Some susceptibility areas for genes possibly influencing long-term body weight regulation were identified in the long arm of Chromosome 8 (Coady et al. 2002; Golla et al. 2003).

A factor analytic solution of the Eating Attitudes Test (EAT) produced a "dieting" factor with an estimated heritability of 41% (no standard errors were reported) in a volunteer sample of young female British twins (Rutherford et al. 1993), implying that some genetic influences on dieting were evident. Some evidence of genetic influences on weight change was found in a Swedish twin sample (Heitmann et al. 1999). Although the net population weight change over a period of eleven years was positive and fairly consistent across different zygosity groups, no information was given on subgroups of weight trajectories. Thus the study population is probably a genetically heterogenous mixture of individuals who gain weight, lose weight, and maintain their weight, making understanding the construct of weight change and drawing conclusions about it very challenging. Furthermore, the study sample size was relatively small (N=98 MZ and 176 DZ twin pairs).

Another study, conducted in Finland, also attempted to understand genetic influences on weight change utilizing longitudinal twin models (Korkeila et al. 1995). In this study, the phenotype was more clearly defined: the type and directionality of weight change were specified. The analyses suggested that additive genetic influences on BMI remain relatively stable throughout adulthood, but that the influence of genetic effects on a 6-year weight change was modest at best. Twins who cohabited in adulthood were more likely to be similar in regard to weight change than twins who lived separately, suggesting some role of familial environment (Korkeila et al. 1995). In later studies of the same population, it was found that genetic factors may modify the effects of physical activity on weight change, in particularly so that sedentary lifestyles may have an obesity-promoting effect in men with a genetic predisposition (Heitmann et al. 1997), and that genetic and familial factors may contribute to major weight gain after weight loss attempts (Korkeila et al. 1999). Education level also plays a role in weight change (Silventoinen et al. 2004).

Genetics of disordered eating

In contrast to weight change, there is a lot of research on the genetics of disordered eating. Very early in life, overeating eating style seems to be passed on from parents to children (Wardle et al. 2001). In adults, bingeing on food is moderately heritable (Virginia Twin Registry, women: 46%, 95% CI: 33-59%; Norwegian Twin Registry, both women and men: 51%, 95% CI: 43-58%) and self-induced vomiting is highly heritable (72%, 95% CI 55-88%) (Sullivan et al. 1998; Reichborn-Kjennerud et al. 2003). There is substantial genetic overlap between vomiting and bingeing (r= 0.74)(Sullivan et al. 1998), but much more modest overlap between between bingeing and obesity (r=0.34, 95% CI, 0.19-0.50) (Bulik et al. 2003). Overall, about 60% (95% CI 50-68) of the variance in liability to disordered eating is explained by genetic factors (Wade et al. 1999). For clinical eating disorders, heritability estimates are similar or higher (for reviews, see Fairburn et al. 1999; Bulik et al. 2000).

Several relatively recent studies have addressed the familiality and genetics of body shape and weight concerns (Holland et al. 1988; Rutherford et al. 1993; Wade et al. 1998; Klump et

al. 2000; Wade et al. 2001; Wade et al. 2003; Reichborn-Kjennerud et al. 2004). The search for candidate genes affecting eating disorders and weight-regulation has stimulated research in this field, as variability in perceptions of body image may mark endophenotypes (ie, intermediary phenotypes) of eating disorders (Devlin et al. 2002; Gottesman & Gould 2003). Twin studies of body shape and weight concerns are detailed in Table 3. These studies have used different approaches, ranging from standard interviews (EDE: Fairburn & Cooper 1983), and validated questionnaires (EDI: Garner 1991, BAQ: Ben Tovim & Walker 1991) to assessing body image by figural silhouettes (Wade et al. 2001) and to using a DSM-IV criterion of disordered body image ("undue influence of weight on self-evaluation") in a questionnaire form (Reichborn-Kjennerud et al. 2004). To give a different cultural perspective, there is also a recent Japanese twin study of EDI subscales (Kamakura et al. 2003). Regrettably, the authors only report result from subscales that are not directly related to weight and shape concerns (maturity fears, ineffectiveness, interpersonal distrust, interoceptive awareness, and perfectionism).

Considering the heterogeneity of the measures, it is not surprising that estimates of heritability obtained from these studies vary considerably. However, heritability estimates for the subscales of EDI, the most widely used measure of body and weight dissatisfaction, are

Table 3. A summary of twin studies of body image and body shape attitudes (adapted from Wade et al. 2003).

Study	N	Measure	Additive genetic effects (95% CI)[1]	Common environment (95% CI)[1]	Unique environment (95% CI)[1]
Holland et al, 1988[2]	45	EDI, global score	98	0	2
Rutherford et al, 1993[2]	246	EDI: BD	52	0	48
		EDI: DT	44	0	56
Wade et al, 1998[2]	174	EDE[3]: Weight concern	0	52	48
		EDE[3]: Shape concern	62	0	38
Klump et al, 2000[4]	340	EDI: BD at 11 y	45	6	50
		EDI: DT at 11 y	12	30	58
	301	EDI: BD at 17 y	63	0	37
		EDI: DT at 17 y	54	0	46
Wade et al, 2001[2]	5325	FRS[5]: current body size	54-65	0	35-46
		FRS[5]: desired body size	20-44	0-34	56-80
Wade et al, 2003[2]	1788	BAQ[6]: Feeling fat	53 (47-59)	0	47 (42-53)
		BAQ[6]: Body disparagement	38 (31-44)	0	62 (56-70)
		BAQ[6]: Salience of Weight/Shape	39 (33-46)	0	61 (54-68)
		BAQ[6]: Attractiveness	46 (40-52)	0	54 (48-60)
		BAQ[6]: Lower Body Fatness	52 (45-57)	0	48 (43-55)
Reichborn-Kjennerud, 2004[4]	8045[7]	„Undue influence of weight on self-evaluation"	0	31 (24-38)	69 (68-76)

[1] Given only when CIs or SEs of the parameter estimates were reported in the original article.
[2] Volunteer twin sample
[3] EDE: Eating disorder examination (Fairburn & Cooper 1983)
[4] Population-based twin sample
[5] FRS: Figure rating scale (Stunkard et al. 1983)
[6] BAQ: Body attitudes Questionnaire (Ben Tovim & Walker 1991)
[7] This study sample contains 3443 males and 4602 females; males and females were entered in the models both independently (male and female parameters estimates freely) and by constraining males and females equal. The model fitted the data better using the latter approach, thus parameter estimates reported here are equal for males and females.

very consistent in late adolescence and young adulthood: in a volunteer sample of 492 adult female twins from the United Kingdom (Rutherford et al. 1993), and in the 17-year-old twins from a population study conducted in Minnesota (Klump et al. 2000), EDI subscale scores were moderately heritable. Unfortunately, their relatively small sample sizes, and the absence of confidence intervals limit conclusions that can be drawn (Neale & Miller, 1997). With the exception of the Norwegian study, none of these studies addresses body image in men.

Genetics of food intake

There are far fewer studies of genetics of food intake, perhaps because the phenotype is much more complex (Keller et al. 2002; Keller et al. 2003). The largest one of these studies focused on twins over 50 y using a food frequency questionnaire (van den Bree et al. 1999). Two distinct eating patterns were identified: a preference for food items high in fat, salt, and sugar, and a preference for healthful eating habits. Both of these patterns were highly environmentally modulated, with heritabilities between 15% and 38% for the first pattern, and 33-40% for the latter pattern; clear gender differences were also evident. A Swedish twin study focusing on twins aged 25-59 y found some evidence of genetic influences on the frequency of intake of certain types of food, such as flour and grain products and fruit, but the majority of food intake types seemed to be largely environmentally modulated (Heitmann et al. 1999). In a small volunteer twin sample (110 MZ and 102 DZ twins, both male and female same-sex pairs, and 53 pairs of opposite-sex DZ twins), food intake as recorded in food diaries was examined: 65% of the variance in daily energy intake could be attributed to heritable factors; the heritability of average meal size was also 65% and that of meal frequency 44% (standard errors were not estimated) (de Castro 1993). Daily energy intake was largely genetically independent from body size, and individual meal size was independent from daily intake. In another study from the same sample, the heritability of the daily timing of the meal was 37% (morning meal timing h^2=24%, afternoon meal timing h^2=18%, and evening meal timing h^2=22%) Intake during those meals was estimated to be 47% heritable for morning meals, 64% heritable for afternoon meals, and 58% heritable for evening meals: however, at this age (30-50 years), no shared environmental effects were discernible (de Castro 2001). Another American study, of Minnesota twins reared apart (ranging in age from 18 to 77 y, with a mean of 42 years), found that 32% (95% CI: 10-51%) of the variance in daily energy consumed was attributable to additive genetic effects. The heritability of meal frequency was 33% (95% CI: 11-52%) and that of snack frequency was 30% (95% CI: 9-49%); moderate spousal correlations were also evident (0.30 for total energy consumed, 0.09 for snacking frequency, and 0.40 for meal frequency) (Hur et al. 1998).

Summary

Genes have an important role on weight regulation; however, the specific mechanisms are largely unknown. Although there has been considerable improvement in understanding the genetics of BMI, obesity, eating disorders, and disordered eating, much less is known about the genetics of other weight control behaviours, such as eating behaviors and weight loss. Thus, further research is clearly warranted.

3. Aims of the study

The genetic and molecular basis of weight control behaviors is relatively poorly understood. The complex interplay of genes and environment, great interindividual variation, and various measurement issues make this task even more daunting. Twin and family studies can be used to examine genetic and environmental contributions to weight control related phenotypes and to level the ground for further exploration of genes in this area. In this study, three phenotypes, breakfast skipping, body shape and weight concerns, and intentional weight loss, were examined.

The specific aims were:

1. To describe demographic and behavioural correlates of breakfast skipping, attitudes about body shape and weight, and intentional weight loss (I).
2. To investigate familial patterns of breakfast skipping (II).
3. To estimate genetic and environmental components in the liability in breakfast skipping patterns (II), body shape and weight concerns (III), and intentional weight loss (IV).
4. To assess to what degree the same genetic effects influence both BMI and IWL (IV).

4. Subjects and methods

Participants

To address the aims of this study, the unique twin-family data from the population-based *FinnTwin16* were used. FinnTwin16 comprises five consecutive nationwide birth cohorts of twins born between 1975 and 1979 (Rose et al. 1999; Kaprio et al. 2002). Local ethics committees in Finland and in the U.S. approved the collection of these data. At baseline, a questionnaire was mailed to twins born in 1975 through 1979 within 2 months of their sixteenth birthday. The questionnaire assessed personality, social relationships, general health, health habits, and nutritional matters, including breakfast skipping. Three follow-up assessments were conducted by postal questionnaire at the age of 17 y, 18.5 y, and as young adults. In the fourth wave of data collection, each birth cohort of twins were contacted semiannually between autumn 2000 (1975 cohort) and autumn 2002 (1979 cohort), and the mean age was 24.4 y, range 22-27 yrs. Body dissatisfaction, drive for thinness, and intentional weight loss attempts were assessed in the fourth wave of data collection. Response rates were high (>80%) across all occasions.

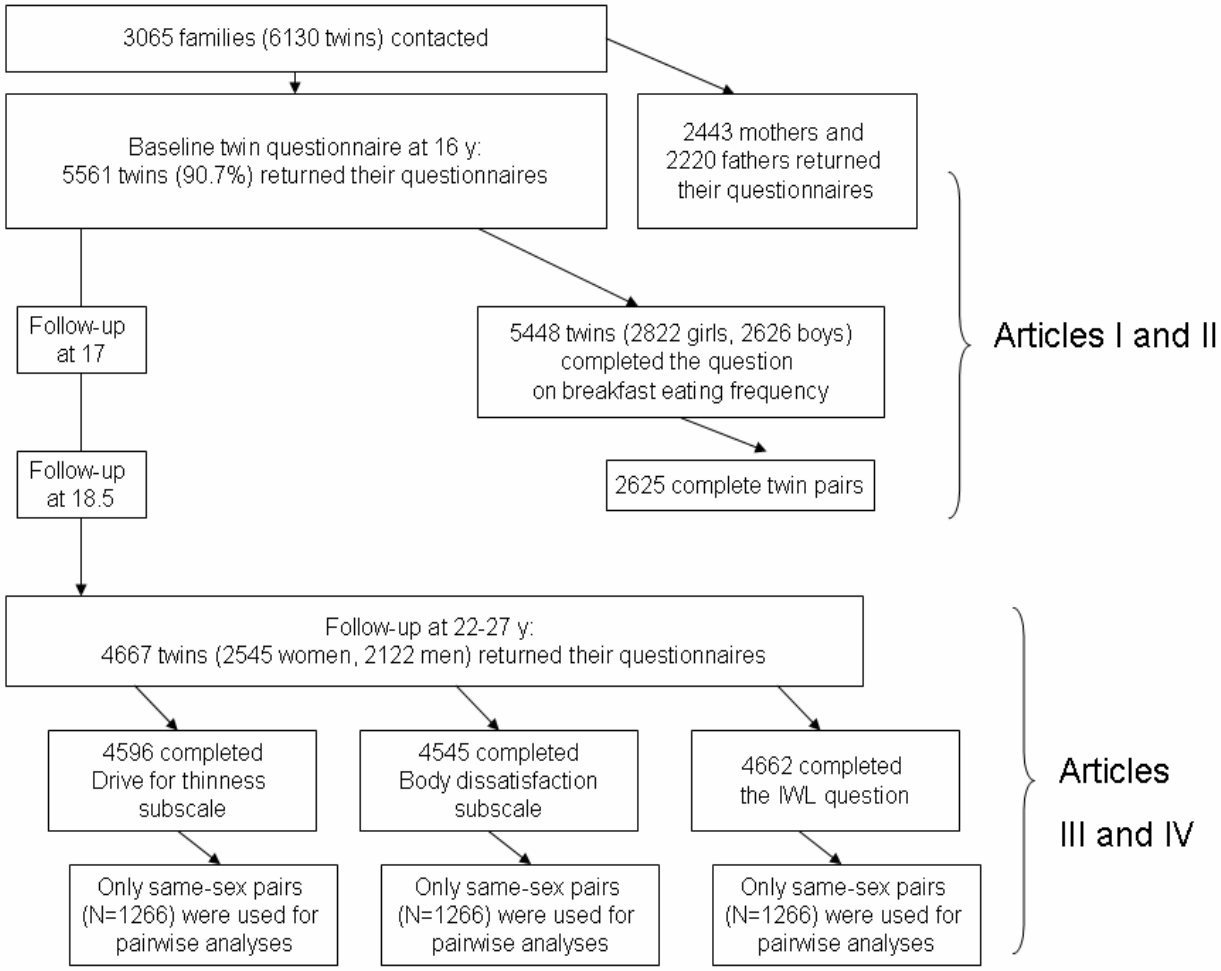
A flowchart depicting different waves of questionnaires and the number of participants in each wave relevant for these studies is presented in Figure 3. A total of 3065 families were contacted. The parents of the twins received their own questionnaires which were mailed at the same time as the baseline twin questionnaires. Of the 6130 twins in these families, 5561 (91%) returned the baseline questionnaire. Individual response rates were 93% for girls, 88% for boys, 84% for fathers and 87% for mothers at baseline.

Information on mothers' and fathers' occupation, SES, lifestyle and health habits (particularly breakfast skipping and weight were used (I, II, III); other variables of interest are detailed below in the Measures section). At the time of the assessment, the age of the mothers ranged from 32.2 to 62 (mean 44.3, SD 4.9) years, and fathers, respectively, from 33.6 to 69.8 (mean 46.5, SD 5.7) years.

At the time when the data for studies III and IV were analyzed, 4667 twins (84% of those taking part in the first wave and 76% of the entire twin sample), 2545 females and 2122 males, had returned their young adult questionnaires. Information on IWL was available from 4662 subjects, BD from 4545 and DT from 4596 subjects. After this, the response rate has further increased still, but we could not include the late responders in these analyses.

Zygosity was determined by standard items included in the baseline questionnaire (Sarna et al. 1978; Sarna & Kaprio 1980) and was in a few cases supplemented with photographs, fingerprints and DNA-marker studies. The twin pairs were classified as monozygotic (MZ), dizygotic (DZ), or unknown zygosity.

Figure 3. Flowchart depicting participation in the FinnTwin16 study



Measures

Breakfast skipping

The frequency of breakfast skipping was assessed by the following question: "How often do you eat breakfast (for example, sandwiches, milk, hot cereal, other similar food) before going to school or going to work?" The three alternative responses were "every morning", "a few times a week", "about once a week or less often".

Body shape and weight concerns

The young adult questionnaire included three subscales of the Eating Disorder Inventory-1 (Garner 1991): drive for thinness (DT), body dissatisfaction (BD), and bulimia. A Finnish version of this instrument has been translated and validated (Charpentier et al., unpublished manuscript). The EDI responses were scored 1 to 6 to ensure a more normal distribution. The DT subscale has 7 items with a coefficient alpha in our sample of 0.87 for females and 0.75

for males. The BD subscale has 8 items with alphas of 0.92 in females and 0.86 in males. For the correlational analysis and twin modelling of male data, DT and BD scores were dichotomised into high vs. low groups, using 75% percentiles as cut-off points (DT, females: 24, males: 14; BD, females: 34, males: 19), because male BD and DT had extremely skewed distributions.

Intentional weight loss

IWL was assessed in our sample using the following question: "How many times during your life have you intentionally lost over 5kg of weight?" The responders in the category "never" formed the no-IWL group. Individuals responding "once" formed the 1-IWL group and those responding "2-4 times" or "5 times or more" formed the 2-IWL group. The last two categories were combined because of a very small number of male respondents in either category. For genetic analyses, IWL was dichotomised to "never" or "once or more" because of the skewed distribution of responses.

Body size

In this study, self-reported weight and height at 16, 17, and 22-27 were used to calculate respective BMIs. At 22-27, the twins also self-measured their waist circumference using a tape measure that was mailed with the questionnaire, together with detailed instructions including a body drawing indicating the site of measurement. The validity of self-reported weight, height, and waist were examined in a subsample of 212 young adult twins. Trained personnel measured the twins' height, weight, and waist circumference using a stadiometer, calibrated beam balance, and a tape measure. The agreement between these standardized measurements and self-report was excellent (correlations of 0.96 for height, 0.94 for weight, 0.88 for waist; Silventoinen et al. 2003); the median time interval between self-report and the standardized measurement was 356 days. In addition to their current weight, the 22-27-year old twins also reported their ideal and maximum weights at adult height.

Dietary restraint / disinhibition

At 22-27y, dietary restraint was assessed with the question: "Which of the following best describes you?" "It's easy for me to eat about the amount I need to." (normal eating, reference category); "I quite often eat more than I actually need." (overeating); "I often try to restrict my eating."; (restrictive eating); "At times, I'm on a strict diet, at others I overeat." (alternating restrictive eating / overeating).

Eating styles

To assess eating styles of the twins, a short 12-item questionnaire was devised: 5 items assessed snacking / grazing styles, 3 health-conscious eating, 2 emotional eating, 1 external eating, and 1 night eating. Exploratory factor analysis was carried out, and the factors largely corresponded to the a priori groupings of items. Individual items will be referred to throughout this paper, but groupings are based on the factor structure. The factor pattern was similar in both genders with the exception night eating, which emerged in women only, was very rare and had a very distinct response pattern, possibly reflecting an underlying eating disorder. Thus, night eating is not included in eating styles explored in this paper.

Education

We measured education level at ages 16, 17, and 22-27. Educational attainment was self-reported. For some analyses, the variable was dichotomised as mandatory school only vs. higher education (vocational school, high school, polytechnic school, or university). The education level in adolescence was taken from the 17 y questionnaire (when 93.0% of twins had finished mandatory education) instead of the 16 y questionnaire (when only 57.5% had finished mandatory education). Education level at 17 is more informative of academic success in mid-adolescence than education level at 16, because further education is voluntary after 16, and the choice of educational paths reflects academic performance.

Other health-related behaviors

The following other variables obtained from the questionnaires at the age of 16 were used in our analyses: smoking status, alcohol use; use of coffee, tea, caffeinated soda, and cocoa; types of milk and bread spread used; frequency of physical exercise, self-perceived physical condition and health, and age of puberty onset (menarche for females, voice break for males).

From the questionnaires filled out at age 17, behavioral disinhibition, experience seeking, and susceptibility to boredom scores as measured by the Sensation Seeking Scale (Zuckerman 1979) were used.

Family SES

Family socioeconomic status (SES) was determined by the occupation of the father or the mother, whichever ranked higher. If only one parent had responded, his or her occupation determined the family's SES. If both parents' occupations were unknown, the family was excluded from the analyses of the effect of SES on breakfast skipping. The parental occupations were divided in seven categories using the Statistics Finland 1989 classification of SES (Sosioekonomisen aseman luokitus 1989); we contrasted the two highest socioeconomic categories (upper-level white-collar workers; independent entrepreneurs and farmers) with the five lower categories (lower-level white-collar workers, blue-collar workers, students, pensioners, and those of unclassified or unknown occupation) to create "higher SES" and "lower SES" categories.

Other parental variables

From parental questionnaires, we obtained data on father's and mother's breakfast skipping and other variables we deemed possibly important for breakfast skipping. The most central variables were parental BMI, smoking, alcohol use, frequency of physical exercise, and highest level of parental education. Smoking and alcohol use variables were dichotomized, primarily to facilitate the assessment of possible interactions. Other information used in the analyses included coffee, and tea use; types of milk and bread spread used; use of vitamin and trace mineral supplements, and of natural and herbal drugs; unemployment in the family; working in shifts, amount of sleep, and feeling tired in the morning.

Statistical methods

General statistical methods

We investigated differences between outcome variables and correlates using cross-tabulations and the Pearson chi-squared test of independence, corrected for clustered sampling within the twin pair (Rao and Scott correction, *svytab* procedure in Stata 8.0) (Rao & Scott 1984). To account for the clustered sampling structure, continuous variables in manuscript III were analyzed using the *svymeans* procedure in Stata 8.0. Odds ratios obtained from univariate, multiple, and polytomous logistic regression models were also used in these analyses (Hosmer & Lemeshow 2000), again correcting for clustered sampling. Twin correlations were assessed using the polychoric correlations function in SAS.

The basic principles of twin studies

Twin studies are a natural experiment that allow estimation of genetic and environmental contributions to variation on traits of interest. The basic premise of twin studies is that MZ twins are the product of a single fertilized oocyte, being thus genetically identical. DZ twins are the result of two oocytes and share, on average, 50% of their segregating genes. In quantitative genetics, genetic and environmental factors are assumed to contribute to the phenotype of a given trait. Assuming that MZ and DZ twin environmental variances are equivalent (ie, that both twin types have similar environments with respect to the phenotype being studied), the relative contribution of genetic and environmental influences to trait variation can then be resolved using variance components models. These models specify genetic and environmental sources of phenotypic covariance in MZ and DZ twin pairs and yield estimates of the degree to which trait variation is explained by the genetic and environmental influences.

The understanding of genetic and environmental components can be further refined: genetic contribution can be additive (A), which refer to the additive effect of alleles at a locus and summed over all loci, or non-additive (D, dominant), which refers to intra-locus allelic interactions, summed over all loci. Conversely, the environmental contribution is usually partitioned into shared (C, common) environmental effects and non-shared (E, unique) environmental effects. Common twin or family environment denotes environmental factors that make members of the same family alike; unique environmental influences make members of the same family different from each other. Measurement error is usually estimated in unique environmental effects. C and D effects cannot be simultaneously modeled from twins reared together (Falconer & Mackay 1996): for this reason, classical twin studies estimate variances components using ACE or ADE models and their submodels, but not ADCE models.

Several software packages have been developed to perform maximum likelihood estimation or related function based optimizations of twin and twin-family data. Currently, Mx, Mplus and Lisrel are the most widely used programs for these purposes.

In this study, various twin modelling strategies ranging from the simple and well known to novel and experimental were used. Classical univariate twin models on same-sex twin pairs were the centerpiece of study III. More sophisticated sex-limitation models that included information on opposite-sex twin pairs were used to explore the genetic and environmental influences on breakfast skipping in study II. Bivariate twin models were used both in study III to check the genetic overlap in BD and DT, and in study IV to explore shared genetic and environmental influences between BMI and IWL. Finally, twin-family models, ie, models that

include information from the mothers and fathers to the information obtained from the twins, were used in study II to examine the results obtained from sex-limitation models. Specific details of these modelling strategies are briefly discussed below.

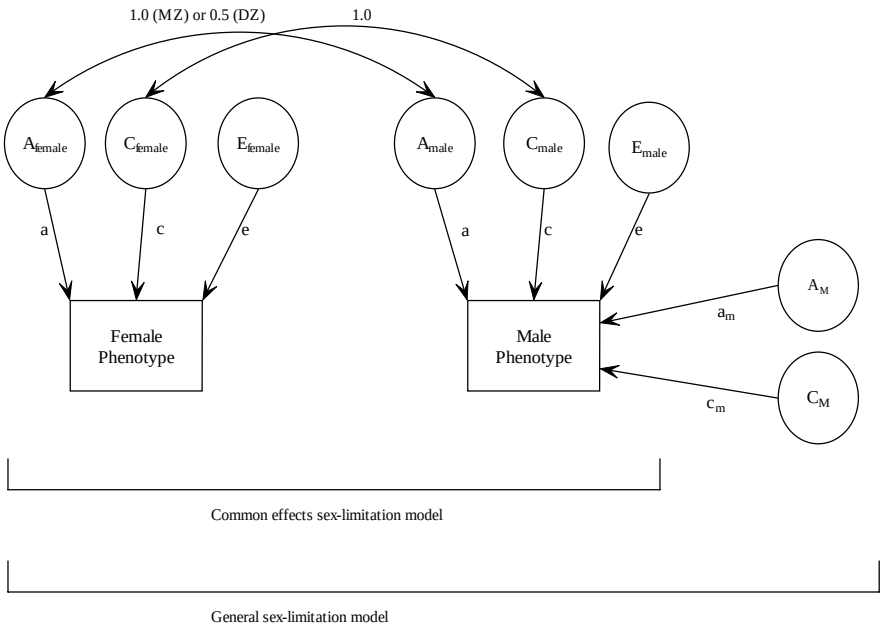
Classic univariate twin models

Classic twin models assess whether a model that consists of additive genetic influences, shared environmental influences, and unique environmental influences (ACE) fits the data better than simpler models that only specify either additive genes and unique environment (AE), shared and unique environment (CE), or unique environment alone (E). Alternatively, ADE and its submodels can be assessed.

In study III, based on twin correlations (Pearson correlations for females, tetrachoric correlations for males, calculated using SAS, version 8.0), ACE and its submodels were deemed most probable explanatory models for body image related variables BD in both genders and DT in men. Pairwise DT correlations in women showed possible dominance effects: both ACE and ADE and submodels were fitted. Model fitting started with the most parsimonious models, AE and CE, and then advanced to ACE/ADE models. However, in this setting the power to detect D effects is relatively low (Falconer & Mackay 1996).

For the classic ACE twin models used in study III, we used the raw data maximum likelihood estimation option in Mx (Neale et al. 2002) that enabled inclusion of unmatched twin pairs (about 10% of our sample). When non-normality was apparent in the distributions, dichotomization was used before model fitting, if simple attempts of transformation (such as log transformation, square and cubic root transformation) did not render a close approximation of the normal distribution. Such dichotomization was necessary for male EDI subscales, where 75% percentile points were used as cut-off points. In females, we used DT and BD scores as a continuous variable, from which variance-covariance matrices were calculated and fitted using Mx. The distributions and variances for EDI subscale scores in each gender were so different that sex-limitation modeling approaches would have proven futile. Instead, we modeled each gender separately, implementing information obtained from same-sex twins only.

Figure 4. Sex-limitation models



Univariate sex-limitation models

In study II, we tested two different models of sex effects on breakfast skipping, first specifying a general sex-limitation model (see Figure 4, previous page) that estimates the magnitude of a sex effect on A, C, and E separately for males and females. Second, we fitted a more restrictive model, the common-effects model that allows the magnitudes of sex effects to vary. The general sex-limitation model allows that sex differences may be caused by different genes (or environmental factors) in males and females; the common-effects model assumes that the same genes and environmental factors influence the breakfast skipping of both boys and girls, but that the magnitude of the influences differs for sexes. Data from same-sex and opposite-sex twin pairs were arranged in 3 x 3 contingency tables; sex-specific threshold values were used. Variances were standardized by constraining the values in males or females to 1. The sex-specific threshold values were 0.6, 1.2 for males and 0.5, 1.1 for females.

Bivariate twin models

Bivariate twin models estimate genetic and environmental contributions to the phenotypic variance in two traits and the covariance between those two traits. However, the model does not determine causality. There are several different ways to model bivariate relationships: Cholesky decompositions were used in this study to assess the relationship of body image related subscales BD and DT (study III) and the relationship of BMI and intentional weight loss (study IV).

Twin-family models

In study II, twin-family models of breakfast skipping were used to confirm and expand the results obtained from sex-limitation twin models. Twin-family models were fitted to data from polychoric correlation matrices and asymptotic covariance matrices based on data from all four family members using weighted least squares estimation. Twin-family models try to explain parent-child behavioral resemblance by specifying biometric expectations to explain the variance-covariance relationship in the data. This model is then fit to the variance-covariance matrix to test whether the model explains the data. First, we tested a model of *social homogamy* (Eaves et al. 1989). A path diagram detailing a social homogamy model is presented in Figure 5. Social homogamy refers to spouse selection that is based on environmental similarities. The spousal resemblance is solely due to this common environment (C) (Reynolds et al. 1996). This might for example be true for people marrying mainly because they belong to the same religious community. According to this model, parents influence their offspring in two ways: through genes and through parental common environment (which brought the parents together in the first place). Parental unique environment (E) is assumed not to transmit to the offspring.

Second, we tested a *phenotypic assortment* model. A path diagram detailing a phenotypic assortment model is presented in Figure 6. Phenotypic assortment refers to the selection of spouses based on observable characteristics (phenotypes) (Neale & Cardon 1992; Neale et al. 1994; Reynolds et al. 1996), that is, traits that can be influenced by either genes, family environment or unique environmental experiences. In contrast to the simplistic social homogamy model, spouse selection is *not* based on any particular component of the phenotype (A, C,or E) alone. Cultural transmission to the offspring originates from the parental phenotype (being thus affected by A, C, and E), and influences the common

environment of the offspring. The model assumes that interdependencies of the model parameters are constant across generations. The phenotypic assortment model also implies that because parents pass both genetic and environmental factors to their offspring, these factors (A and C) are somewhat correlated in the offspring. In the classical twin model, this correlation is assumed to be zero; in the phenotypic assortment model, the magnitude of this correlation can be estimated.

Model fitting and indicators of fit

In these procedures, hierarchically nested models were fitted. Models are usually compared to each other using the principle of parsimony. Models with fewer parameters are preferable if they do not provide a substantially worse fit. To assess how well a model fits the data, several fit statistics can be used. We used the –2 log-likelihood statistic: the likelihood-ratio of alternative hierarchically nested models was calculated by the difference in their χ^2 values. The difference of two such χ^2 goodness-of-fit statistics is itself distributed as a χ^2 statistic with degrees of freedom equal to the difference in degrees of freedom of the two models being compared (Neale & Cardon 1992). If the difference in the χ^2 values of two models was not statistically significant, the principle of parsimony was applied: the model with fewer parameters was preferred. Akaike's information criterion (AIC, calculated as χ^2- 2df) was used as an additional indicator of fit: a model with a lower AIC value is the more parsimonious (Neale & Cardon 1992).

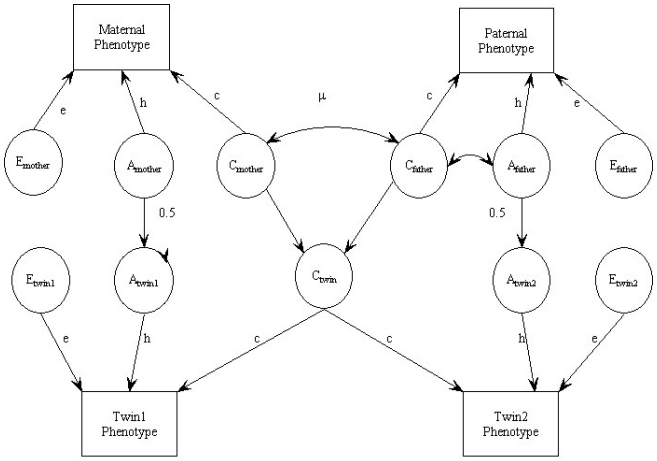


Figure 5.
Social homogamy model

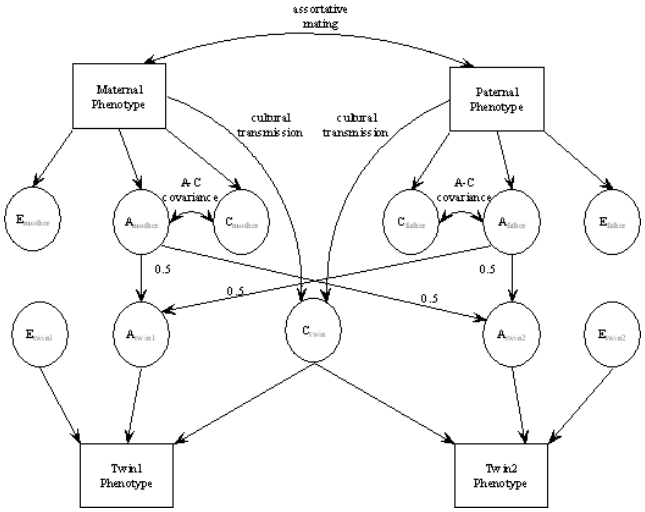


Figure 6.
Phenotypic assortment model

5. Results

Weight control behaviours: correlates and demographics

The first aim of this study was to characterize the phenotypes breakfast skipping, body shape and weight concerns, and intentional weight loss, and to assess some of their demographic determinants and behavioural correlates. Below, each of these three domains is assessed separately.

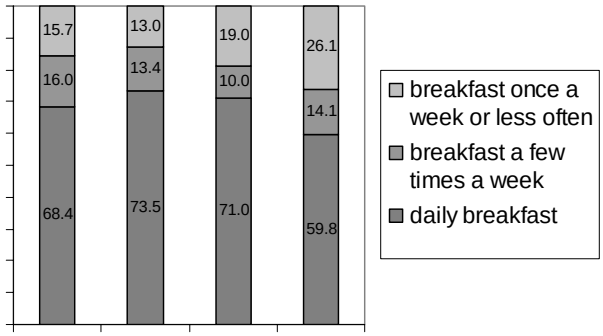


Figure 7. Frequency of breakfast skipping in girls, boys, women, and men of FinnTwin16

Breakfast skipping

As detailed in study I, breakfast skipping was relatively common: 15.7% of girls, 13.0% of boys, 19.0% of mothers and 26.1% of fathers ate breakfast only once a week or less often (see Figure 7). Parental breakfast skipping patterns were the statistically most significant factor associated with adolescent breakfast skipping (Table 4). Low family socioeconomic status was associated with breakfast skipping in adolescent boys, but not in girls (Table 4).

Table 4. Correlates of breakfast skipping in adolescents: the influence of parental factors. In sex-adjusted polytomous logistic regression models, adolescents who skip breakfast were contrasted with adolescents who always eat breakfast

Correlates		Breakfast skippers (vs. eaters) Sex-adjusted odds ratios (95% Confidence Interval[1])	
		Breakfast a few times a week	Breakfast once a week or less often
Parental correlates			
mother's breakfast eating N=4886	every morning (71.0%)	1.0 (reference)	1.0 (reference)
	a few times a week, 10 (10.0%)	1.74 (1.30-2.34)	2.24 (1.65-3.05)
	once a week or less (19.0%)	2.26 (1.82-2.80)	3.57 (2.84-4.49)
father's breakfast eating N=4440	every morning (59.8%)	1.0 (reference)	1.0 (reference)
	a few times a week (14.1%)	1.95 (1.50-2.54)	1.79 (1.30-2.46)
	once a week or less (26.1%)	2.06 (1.66-2.54)	2.93 (2.32-3.70)
family socio-economic status N=4318	upper-level employee (22.6%)	1.0 (reference)	1.0 (reference)
	self-employed, including self-employed farmers (18.8%)	1.03 (0.78-1.36)	1.43 (1.05-1.95)
	lower-level employee (21.7%)	0.91 (0.70-1.18)	1.43 (1.06-1.93)
	manual worker, student, retired, or of unclassified occupation (36.9%)	1.07 (0.85-1.36)	1.55 (1.18-2.03)

In study I, also a second group of correlates, health-compromising behaviors, ie, smoking, infrequent exercise, frequent alcohol use, behavioral disinhibition, and high body mass index (BMI), also had clear cross-sectional associations with breakfast skipping in adolescents (Table 5). Female gender (OR 1.3, 95% CI: 1.1-1.5) and low education level at 17 were also significantly associated with breakfast skipping in adolescents.

Table 5. Correlates of breakfast skipping: individual-level factors in adolescents. Adolescents who skip breakfast vs. always eat breakfast: odds ratios of correlated individual-level behaviors and other characteristics, based on sex-adjusted polytomous logistic regression models

Correlates		Breakfast skippers (vs. eaters) Sex-adjusted odds ratios (95% Confidence Interval[1])	
		Breakfast a few times a week	Breakfast once a week or less often
Individual correlates			
BMI, kg/m^2, continuous (N=5403)[2]		1.06 (1.02-1.09)	1.06 (1.02-1.09)
smoking	never (48.7%)	1.0 (reference)	1.0 (reference)
N=5363	past smoker (20.4%)	1.43 (1.15-1.77)	1.56 (1.23-1.96)
	less than once a week (9.4%)	1.52 (1.16-2.00)	1.28 (0.94-1.76)
	once a week or more but less than daily (4.1%)	1.60 (1.07-2.37)	1.47 (0.97-2.24)
	daily (17.4%)	2.28 (1.83-2.85)	4.17 (3.34-5.21)
behavioral disinhibition at 17 y, continuous (N=5334)[3]		1.07 (1.02-1.12)	1.15 (1.10-1.20)
age of puberty onset (years), continuous (N=5279)[4]		1.01 (0.94-1.08)	0.88 (0.82-0.95)
alcohol use	never (23.5%)	1.0 (reference)	1.0 (reference)
N=5425	once a year or less (9.3%)	0.60 (0.40-0.90)	1.33 (0.94-1.87)
	a few times a year (14.4%)	1.09 (0.82-1.46)	1.55 (1.15-2.09)
	once in a few months (13.7%)	1.22 (0.92-1.62)	1.44 (1.05-1.96)
	once a month (11.7%)	1.31 (0.98-1.75)	1.47 (1.07-2.02)
	a few times a month (17.8%)	1.67 (1.29-2.15)	2.13 (1.61-2.81)
	weekly or more (9.7%)	2.03 (1.51-2.73)	2.89 (2.11-3.96)
education at 16 y	senior high or polytechnic school (36.1%)	1.0 (reference)	1.0 (reference)
N=5418	vocational school (19.2%)	1.41 (1.13-1.77)	2.00 (1.56-2.57)
	junior high school (43.0%)	1.16 (0.96-1.41)	1.79 (1.44-2.22)
	not in school (1.7%)	2.92 (1.62-5.25)	4.91 (2.79-8.64)
education at 17 y	senior high school, vocational college or	1.0 (reference)	1.0 (reference)
N=5110	polytechnic school (62.1%)		
	vocational school (30.6%)	1.52 (1.26-1.83)	1.97 (1.62-2.41)
	junior high school (3.4%)	1.60 (0.98-2.61)	3.49 (2.25-5.43)
	not in school (4.0%)	2.23 (1.49-3.34)	3.43 (2.36-4.97)
exercise	daily (15.8%)	1.0 (reference)	1.0 (reference)
N=5428	4-5 times a week (15.2%)	1.36 (1.01-1.84)	0.95 (0.67-1.35)
	2-3 times a week (28.0%)	1.28 (.98-1.69)	1.32 (0.99-1.77)
	about once a week (19.1%)	1.60 (1.20-2.12)	1.36 (1.00-1.85)
	once or twice a month (10.4%)	1.60 (1.15-2.22)	2.33 (1.66-3.28)
	less than once a month (6.6%)	1.72 (1.17-2.52)	4.03 (2.84-5.72)
	never (5.0%)	1.39 (0.90-2.15)	3.75 (2.57-5.47)

[1] Confidence intervals adjusted for intraclass correlation
[2] For 118 BMI values missing at 16 y, BMI values at 17 y were substituted
[3] For 180 disinhibition scores missing at 17 y, disinhibition scores at 18 y were substituted
[4] Menarche for females, voice break for males

In adults examined in study I, the findings were similar for health-compromising factors, although slightly different measures were used: smoking, infrequent exercise, higher BMI, and more frequent alcohol use were associated with breakfast skipping (Table 6). In the sociodemographic domain, adult males, not females, were more likely to skip breakfast. The tendency to have breakfast regularly increased with age, but low education was associated with breakfast skipping.

Body shape and weight concerns

The general characteristics of FinnTwin 16 twins at the 4th wave of assessment (study III) are detailed in Table 7. In average, the level of body shape and weight dissatisfaction, as measured by BD and DT, was higher in women than in men. The association of BD and education level in adult women reflected a greater prevalence of overweight (12.7% vs. 7.1%, $p<0.00001$ in women; 16.9% vs. 11.8%, $p=0.0018$ in men) among individuals with a low level of education.

As detailed in Table 8, larger body size and shape (higher BMI, larger waist circumference, both currently and at the age 16), disordered eating patterns (such as restrictive eating, overeating, and alternating restricting-binging), and psychosomatic symptoms (such as poor self-perceived health, nervousness, sleeping difficulties), depressive mood, dissatisfaction with parents, and feelings of loneliness were associated with BD and DT in both genders in study III. Also, sexual maturation related items were associated with BD, but not with DT: early pubertal onset, early initiation of sexual activity, and multiple sex partners increased BD and late puberty emerged as a protective factor from BD.

Table 6. Correlates of breakfast eating in adults: polytomous logistic regression models adjusted for age and sex.

Correlates		Breakfast skippers vs. eaters Adjusted odds ratios (95% Confidence Interval[1])	
		Breakfast a few times a week	Breakfast once a week or less
family SES	upper-level employee (19.3%)	1.0 (reference)	1.0 (reference)
N=4523	self-employed, including farmers (16.1%)	1.18 (0.84-1.67)	1.69 (1.29-2.21)
	lower-level employee (34.0%)	1.56 (1.16-2.09)	1.75 (1.37-2.22)
	manual worker, student, retired (30.6%)	2.16 (1.63-2.96)	2.66 (2.11-3.35)
smoking	never (40.8%)	1.0 (reference)	1.0 (reference)
N=4602	past smoker (33.7%)	1.11 (0.89-1.39)	1.35 (1.11-1.62)
	current smoker (25.6%)	2.06 (1.63-2.61)	3.53 (2.91-4.28)
alcohol	less than twice a month (41.0%)	1.0 (reference)	1.0 (reference)
use	3-8 times a month (41.5%)	1.19 (0.97-1.47)	1.12 (0.94-1.33)
N=4625	over 8 times a month (17.5%)	0.84 (0.63-1.12)	1.19 (0.96-1.48)
highest	university degree (4.5%)	1.0 (reference)	1.0 (reference)
education	senior high school graduation (17.0%)	1.03 (0.59-1.78)	1.29 (0.80-2.07)
N=4657	vocational school (21.2%)	1.63 (0.96-2.75)	1.99 (1.27-3.12)
	mandatory education only (57.3%)	1.99 (1.21-3.29)	2.54 (1.64-3.92)
BMI, kg/m^2, continuous (N=4608)		1.03 (1.01-1.06)	1.03 (1.01-1.05)
exercise	exercise ≥6 times a month (41.4%)	1.0 (reference)	1.0 (reference)
N=4550	exercise 3-5 times a month (30.1%)	1.14 (0.90-1.45)	1.16 (0.97-1.38)
	exercise 1-2 times a month (24.6%)	1.18 (0.93-1.49)	1.69 (1.40-2.03)
	exercise less than once a month (3.9%)	0.89 (0.50-1.56)	2.83 (2.00-4.00)

[1] Confidence intervals adjusted for clustered sampling.

Intentional weight loss

In study IV, IWL was much more common among women than men (Table 9). Obesity (BMI>30) was equally common in women (3.7%) and men (4.1%) (F=0.39, p=0.53), but IWL was significantly more common among obese women (81.9%) than obese men (69.0%) (F=6.1, p=0.015). Associations of IWL and SES or education were not specifically explored in mean BMIs of individuals with mandatory education only (23.4 kg/m^2) or vocation education this study, but we did find a relationship of BMI and SES that was gender-independent: the (23.5 kg/m^2) were significantly (F=13.7, p<0.00001) higher than those who had completed high school or polytechnic (22.7 kg/m^2), or university (22.1 kg/m^2). Women and men who had lost at least 5kg of weight at least once had significantly higher past, current, maximum, and ideal BMIs and larger waist circumferences than individuals who did not engage in IWL (Table 9). Both women and men who had lost at least 5 kg at least twice had significantly

Table 7. General descriptive statistics of FinnTwin16 twins

	Women	Men
	%	%
Zygosity[1]		
MZ (N)	35.0 (868)	26.3 (540)
Same-sex DZ	30.8 (765)	34.4 (705)
Opposite-sex DZ	31.9 (793)	34.5 (717)
Unknown	2.3 (57)	4.3 (89)
Ongoing education at 17		
Senior high or polytechnic	71.5	53.7
Vocational school	22.0	38.4
Junior high school	2.5	3.7
Not in school	4.0	3.9
Educational attainment at 22-27		
University degree	7.9	4.7
Polytechnic degree	14.8	8.4
Senior high school graduation	41.6	38.3
Vocational college graduation	9.2	7.2
Vocational school or training degree	21.6	35.4
Junior high school graduation only	4.8	6.0
BMI		
Mean (SD)	22.2 (3.5)	23.9 (3.1)
Body image		
Median BD score[2]	26	14
Median DT score[3]	18	11
Intentional weight loss		
Never lost >5kg weight	58.2	75.7
Lost >5kg at once	24.2	14.0
Lost >5kg at least twice	17.6	10.2

[1] Percentages calculated from the proportion of twins still participating at age 22-27 y
[2] The scores range from 8 to 48 when the instrument is scored from 1 to 6. a higher score denoting greater body dissatisfaction.
[3] The scores range from 7 to 42 when the instrument is scored from 1 to 6, a higher score denoting greater drive for thinness

Table 8. Correlates of high body dissatisfaction and high drive for thinness: odds ratios (with 95% CIs) from univariate female and male logistic regression models. Eg, compared to women with low body dissatisfaction, the risk of overeating is 7.9 times higher in women reporting high body dissatisfaction.

	High body dissatisfaction				High drive for thinness			
	women		men		women		men	
Correlate	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Body size and shape								
Current BMI (OR per kg/m²)	1.4	(1.3-1.4)	1.3	(1.2-1.3)	1.2	(1.1-1.2)	1.4	(1.3-1.4)
BMI at 16 (OR per kg/m²)	1.3	(1.3-1.4)	1.3	(1.2-1.3)	1.2	(1.2-1.3)	1.4	(1.3-1.5)
Current waist (OR per cm)	1.1	(1.1-1.1)	1.1	(1.1-1.1)	1.0	(1.0-1.1)	1.1	(1.1-1.1)
Overweight at 16	3.9	(3.0-5.2)	4.3	(3.1-5.8)	2.7	(2.1-3.5)	6.0	(4.3-8.2)
Eating-related variables								
Overeating	7.9	(6.1-10.1)	5.5	(4.3-7.1)	5.7	(4.4-7.4)	5.8	(4.5-7.4)
Restrictive eating	8.1	(6.0-10.9)	16.6	(9.2-29.9)	15.4	(11.4-20.8)	35.7	(17.3-73.7)
Alternating overeating and restrictive eating	15.2	(10.3-22.3)	7.2	(3.2-16.0)	23.3	(15.5-35.1)	11.1	(4.6-26.7)
Intentional weight loss of ≥5kg at least once	4.0	(3.2-4.9)	4.5	(3.6-5.6)	4.0	(3.3-4.9)	6.3	(5.0-7.9)
Self-reported ideal BMI (OR per unit increase)	1.3	(1.3-1.4)	1.2	(1.2-1.3)	1.1	(1.1-1.2)	1.2	(1.2-1.3)
Poor self-perceived physical condition	3.1	(2.5-3.8)	3.0	(2.4-3.7)	1.7	(1.4-2.1)	1.9	(1.5-2.3)
Puberty and sexuality								
Early puberty	1.6	(1.2-2.2)	1.4	(1.0-1.9)	1.3	(1.0-1.7)	1.5	(1.1-2.1)
Late puberty	0.5	(0.4-0.6)	0.8	(0.6-1.1)	0.8	(0.6-1.0)	0.8	(0.6-1.0)
Number of sex partners (OR per each partner)	1.1	(1.1-1.2)	1.0	(0.9-1.1)	1.1	(1.0-1.2)	1.0	(1.0-1.1)
Age at first sexual intercourse	0.9	(0.9-0.9)	1.0	(0.9-1.0)	1.0	(0.9-1.0)	1.0	(1.0-1.1)
Psychosomatic symptoms								
Current unhappiness (vs. current happiness)	2.0	(1.5-2.6)	2.6	(2.0-3.4)	2.6	(2.0-3.4)	1.9	(1.4-2.5)
Current self-perceived poor health	2.1	(1.7-2.7)	2.5	(1.9-3.3)	1.8	(1.5-2.3)	1.7	(1.3-2.3)
Self-perceived poor health at 16	2.3	(1.8-2.9)	1.8	(1.3-2.4)	1.9	(1.5-2.4)	1.4	(1.0-1.9)
Current frequent stomach pain (vs. monthly or less)	1.8	(1.4-2.3)	1.7	(1.1-2.6)	1.9	(1.5-2.4)	1.5	(1.0-2.2)
Frequent stomach pain at 16 (vs. monthly or less)	1.4	(1.0-2.0)	0.7	(0.4-1.3)	1.8	(1.3-2.5)	0.9	(0.5-1.6)
Current headaches (vs. monthly or less)	1.8	(1.5-2.2)	1.6	(1.2-2.2)	1.6	(1.3-1.9)	1.3	(0.9-1.8)
Frequent headaches at 16 (vs. monthly or less)	1.5	(1.2-1.8)	1.4	(1.0-1.8)	1.7	(1.3-2.1)	1.4	(1.0-2.0)
Current nervousness	1.7	(1.4-2.1)	1.8	(1.4-2.2)	2.4	(1.9-3.0)	1.8	(1.4-2.2)
Nervousness at 16	1.3	(1.0-1.6)	1.3	(1.0-1.7)	1.8	(1.4-2.2)	1.3	(1.0-1.7)
Current depressive mood	1.6	(1.3-2.0)	2.0	(1.6-2.6)	2.0	(1.6-2.4)	1.8	(1.4-2.3)
Current sleeping difficulties	2.0	(1.6-2.4)	2.0	(1.6-2.5)	2.1	(1.7-2.5)	1.5	(1.2-1.9)
Sleeping difficulties at 16	1.5	(1.2-1.9)	1.6	(1.2-2.1)	1.3	(1.0-1.6)	1.2	(0.9-1.6)
Current dissatisfaction with partner	1.2	(1.0-1.5)	1.4	(1.1-1.7)	1.4	(1.1-1.7)	1.2	(1.0-1.5)
Current dissatisfaction with mother	1.4	(1.0-1.8)	2.0	(1.8-2.7)	1.4	(1.1-1.9)	2.0	(1.5-2.8)
Current dissatisfaction with father	1.5	(1.3-2.0)	1.8	(1.4-2.3)	1.4	(1.2-1.8)	1.5	(1.2-2.0)
Current frequent feelings of loneliness	1.4	(1.2-1.7)	1.7	(1.6-2.4)	1.6	(1.3-2.0)	1.4	(1.1-1.7)

[1] Confidence intervals adjusted for clustered sampling.

Table 9. Means (95% confidence intervals, adjusted for clustered sampling) of weight-related variables in individuals who have never engaged in intentional weight loss (no-IWL), have lost ≥5kg of weight intentionally once (1-IWL), or at least twice (2-IWL).

	women			Men		
	no-IWL	1-IWL	2-IWL	no-IWL	1-IWL	2-IWL
N	1479	615	448	1605	297	218
%	58.2	24.2	17.6	75.7	14.0	10.3
Age (years)	24.3 (24.3-24.4)	24.3 (24.2-24.4)	24.4 (24.3-24.5)	24.4 (24.3-24.4)	24.3 (24.2-24.4)	24.4 (24.3-24.5)
Current BMI (kg/m^2)	21.2 (21.0-21.3)	22.9 (22.6-23.2)	24.7 (24.3-25.2)	23.2 (23.0-23.3)	25.5 (25.1-25.9)	26.8 (26.2-27.3)
Maximum lifetime BMI (kg/m^2)	22.1 (21.9-22.3)	24.8 (24.5-25.1)	27.2 (26.7-27.6)	24.0 (23.8-24.1)	27.2 (26.8-27.6)	29.0 (28.4-29.5)
Ideal BMI (kg/m^2)	20.4 (20.3-20.5)	21.2 (21.0-21.3)	21.7 (21.5-21.9)	23.2 (23.1-23.3)	24.1 (23.9-24.4)	24.8 (24.5-25.1)
BMI at 16-17y (kg/m^2)	19.5 (19.4-19.6)	20.9 (20.7-21.1)	22.1 (21.8-22.3)	20.0 (19.9-20.1)	21.4 (21.1-21.8)	22.0 (21.6-22.4)
Waist (cm)	72.8 (72.3-73.2)	76.6 (75.7-77.4)	80.2 (79.0-81.5)	83.5 (83.1-83.9)	90.0 (88.8-91.3)	92.2 (90.4-93.9)

higher current, maximum, and ideal BMIs than their counterparts who had engaged in IWL only once (Table 9). As detailed in Table 10, IWL was significantly associated with restricting, overeating, and alternating restricting/overeating (IV). Snacking and eating in the evening were characteristic of individuals with at least 2 IWL attempts. As expected, avoiding fatty foods and calories was significantly more pronounced in individuals who had engaged in IWL than in the no-IWL group.

Table 10. Gender-specific odds ratios (95% confidence intervals, adjusted for clustered sampling) of eating patterns in individuals who have never lost weight intentionally (no-IWL, reference group) vs. individuals who have lost weight intentionally once (1-IWL) or at least twice (2-IWL).

	women		men	
	1-IWL N=615	2-IWL N=448	1-IWL N=297	2-IWL N=218
Restrictive eating / overeating				
Restrictive eating	1.7; 1.4-2.2	3.6; 2.7-4.8	2.5; 1.9-3.4	3.3; 2.3-4.6
Frequent overeating	3.2; 2.3-4.3	8.3; 6.0-11.6	9.5; 5.2-17.5	10.4; 5.4-20.2
Alternating overeating and restricting	6.7; 3.9-11.6	25.4; 14.8-43.6	7.3; 2.1-25.6	34.6; 12.4-96.6
Snacking				
Frequent snacking between meals	1.0; 0.8-1.3	1.7; 1.3-2.1	1.2; 0.9-1.6	1.5; 1.1-2.1
Frequent snacks replace meals	1.4; 1.0-1.8	2.0; 1.5-2.7	1.5; 1.0-2.4	2.5; 1.6-3.8
Highest food consumption in the evening	1.2; 1.0-1.5	1.9; 1.6-2.4	1.2; 0.9-1.5	1.3; 1.0-1.8
Grazing throughout the evening	0.9; 0.7-1.2	1.4; 1.1-1.8	0.7; 0.5-1.1	1.6; 1.1-2.4
Eating while watching TV	1.0; 0.8-1.2	1.3; 1.0-1.6	1.2; 0.9-1.6	1.2; 0.9-1.6
Health-conscious eating				
Maintaining healthy eating patterns	1.4; 1.1-1.8	0.9; 0.7-1.2	1.2; 0.9-1.5	1.0; 0.7-1.3
Avoiding fatty foods	1.6; 1.3-2.0	1.6; 1.3-2.0	1.7; 1.3-2.2	2.0; 1.5-2.7
Avoiding calories	1.9; 1.6-2.3	2.0; 1.7-2.6	2.4; 1.7-3.3	2.9; 2.0-4.1
Psychological aspects of eating				
Visual cues (seeing food or food ads) prompt eating	0.9; 0.5-1.6	2.3; 1.5-3.7	1.0; 0.4-2.6	1.9; 0.8-4.5
Food used as a reward	1.3; 1.0-1.7	2.1; 1.6-2.8	1.4; 1.0-2.0	1.7; 1.1-2.5
Comfort eating	1.9; 1.3-2.7	3.5; 2.5-5.1	1.2; 0.6-2.4	2.1; 1.2-3.9

Genetic and environmental influences on weight control behaviors

These studies (II-IV) also aimed to estimate genetic and environmental liability in breakfast skipping, body shape and weight concerns, and intentional weight loss, to investigate familial patterns of breakfast skipping (II), and to assess overlap of genetic influences on BMI and IWL (IV). Below, each of these three domains is assessed separately.

Breakfast skipping

Estimates of additive genetic, common environmental and unique environmental influences were obtained for breakfast skipping using twin and twin-family models in study II. In the sex-limitation twin models based on information based on twin data only, the sex-specific environmental and genetic components could be removed (Table 11). In the resulting best-fitting common-effects model, variation in breakfast skipping frequency was explained by genetic and environmental factors in boys and girls. Additive genetic effects explained 41% (95% CI: 21-63%) of the variance in breakfast skipping in girls and 66% (95% CI: 47-79%) in boys, and common environmental effects 45% (95% CI: 23-62%) in girls and 14% (95% CI: 5-29%) in boys. The gender differences apparent in the common-effects sex-limitation model were significant: constraining male and female ACE estimates to be equal caused a significant deterioration in model fit ($\Delta\chi^2$=7.25, Δdf=2, p=0.03), implying that common environmental effects in breakfast skipping are more important for females than males. In summary, additive genetic effects account for a larger proportion of variability in breakfast skipping in males than in females, but both sources of variation are needed to account for the data.

Table 11. Sex-limitation models of breakfast skipping patterns

model	components of variance estimates (95% CI)							goodness-of-fit tests			
	additive genetic effects		common environment		unique environment		male-specific component[1]	chi-squared	df	P	AIC[2]
	A		C		E		M				
	♀	♂	♀	♂	♀	♂					
common-effects sex-limitation[3]	0.41 (0.21-0.63)	0.66 (0.47-0.79)	0.45 (0.23-0.62)	0.14 (0.05-0.29)	0.14 (0.11-0.19)	0.20 (0.14-0.28)	-	35.03	32	0.326	-28.97[3]
general sex-limitation with A_M	0.41 (0.22-0.63)	0.23 (0.00-0.77)	0.45 (0.24-0.62)	0.26 (0.06-0.48)	0.14 (0.10-0.19)	0.20 (0.14-0.29)	0.31 (0.00-0.52)	33.94	31	0.328	-28.06
general sex-limitation with C_M	0.41 (0.22-0.63)	0.54 (0.27-0.77)	0.44 (0.24-0.62)	0.15 (0.05-0.30)	0.14 (0.10-0.19)	0.20 (0.14-0.29)	0.11 (0.00-0.29)	33.94	31	0.328	-28.06

[1] Either male-specific additive genetic (A_M) or male-specific common environmental component (C_M)
[2] Akaike Information Criterion
[3] Best-fitting model by the Akaike Information Criterion

Family models were also used in study II to check the validity of modeling results explained above, and to explore assortative mating effects. The overall breakfast eating correlation between parents of the twins was fairly strong, 0.39 (95% CI: 0.34-0.45). The overall mother-daughter breakfast eating correlation was 0.30 (95% CI: 0.25-0.36), mother-son correlation 0.35 (95% CI: 0.29-0.41), father-daughter correlation 0.27 (95% CI: 0.21-0.33), and father-son correlation 0.29 (95% CI: 0.23-0.35). Although mothers appear slightly more like their offspring than fathers do, the difference was not statistically significant.

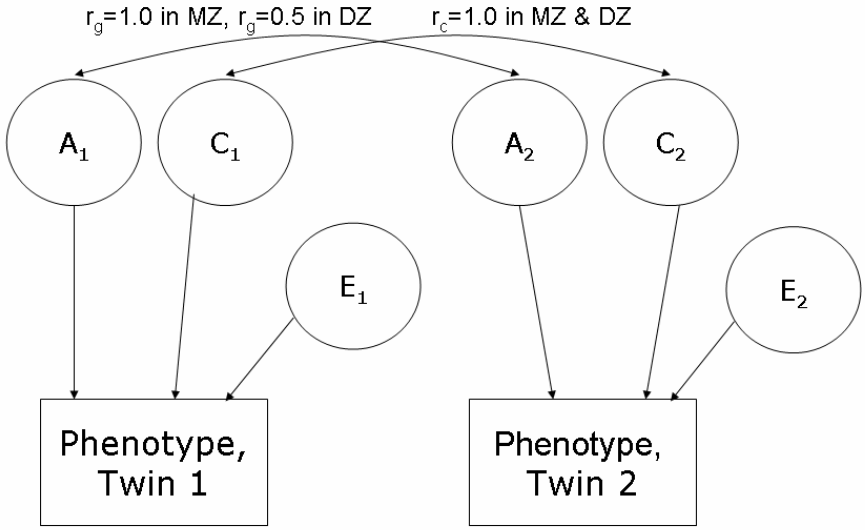
Of twin-family models, we first fit a social homogamy model to the parent and offspring data from the five zygosity groups. Taking the results of the sex-limitation models (Figure 4, p. 31) as a starting point, our baseline model assumed common effects sex limitation in the ACE estimates, but allowed for differing cultural transmission for fathers-sons, fathers-daughters, mothers-sons and mothers-daughters. This model fit the data very well, χ^2=22.33, df=21, p=0.38. Relative to the sex-limitation model based on the twins only (Table 11), estimates of additive genetic effects were lower for both females (0.31) and males (0.47), and estimates of common environmental effects were higher for both females (0.54) and males (0.27). Spousal assortment based on the shared environment was estimated at the upper boundary of 1.0 (95% CI= 0.81-1.0). The individual cultural transmission parameters were small and nonsignificant, and the four parameters could be constrained to an equal estimate (0.16; 95%CI=0.08-0.24) without a significant decrease in fit ($\Delta\chi^2$=0.24, Δdf=3). As in the twin-based sex limitation models, addition of sex-specific genetic ($\Delta\chi^2$=2.65, Δdf=1) or shared environmental ($\Delta\chi^2$=2.66, Δdf=1) parameters did not significantly improve fit, but constraining genetic and environmental estimates to be equal for males and females did result in a significant decrease of fit ($\Delta\chi^2$=6.24, Δdf=2): thus on the family model level as well, the common-effects model provided the best fit.

Second, we fit a phenotypic assortment model to the data to explore whether the assumptions of phenotypic assortment (as described on p. 35-6) provide our data a better fit than the assumption that spouse selection is based on environmental influences shared by the spouses. Our baseline model again assumed common effects sex limitation in the ACE estimates (see Figure 4, p. 34), but allowed for differing cultural transmission for fathers-sons, fathers-daughters, mothers-sons and mothers-daughters. This model fit the data very well (χ^2=15.64, df=21, p=0.79). Addition of sex-specific genetic ($\Delta\chi^2$=3.35, df=1) or shared environmental ($\Delta\chi^2$=3.35, df=1) parameters did not significantly improve fit. As in the social homogamy model, the four cultural transmission parameters could be set equal without a significant decrease in fit ($\Delta\chi^2$=5.16, df=3, p=0.16). This single cultural transmission variable (-0.16; 95% CI=-0.37-0.01) did not quite reach significance. The assortative mating parameter was estimated at 0.40 (95% CI=0.34-0.47). Additive genetic variance components were 0.72 (95% CI=0.46-0.98) for women and 0.63 (95%CI=0.38-0.89) for men. Common environment estimates were 0.29 (95%CI=0.20-0.41) for women and 0.25 (95% CI=0.16-0.36) for men. The gene-environment correlation induced by cultural transmission was -0.19 (95% CI=-0.47-0.01).

Body shape and weight concerns

Study III assessed genetic and environmental contributions to body shape and weight concerns. As detailed in Figure 8 that presents the results of sex-specific univariate twin models of BD and DT, AE models provided the best fit for female body shape and weight concerns: additive genes accounted for 59.4% (95% CI: 53.2-64.7%) of variance in BD and 51.0% (95% CI: 43.7-57.5%) of DT in females. In men, the situation was entirely different: CE models provided the best fit. Thus, according to our models, DT and BD were purely

Figure 8. Comparisons of gender-specific univariate twin models of body dissatisfaction and drive for thinness



	goodness-of-fit tests				heritability	common environment	unique environment
Body dissatisfaction[1]	-2LL	df	$\Delta\chi^2$	p†	a^2	c^2	e^2
Females (continuous)							
ACE (saturated)	11749	1606	-	-	59.4 (42.8-64.7)	0.0 (0.0-14.3)	40.7 (35.3-46.8)
CE	11786	1607	37.1	<0.001	-	44.2 (38.4-49.7)	55.8 (50.3-61.6)
AE*	11749	1607	0	-	59.4 (53.2-64.7)	-	40.7 (35.3-46.8)
Males (dichotomous)							
ACE (saturated)	2133	1222	-	-	7.3 (0.0-15.1)	80.0 (73.1-85.7)	12.7 (10.6-15.5)
CE*	2137	1223	3.6	0.057	-	85.3 (83.2-87.0)	14.7 (13.0-16.8)
AE	2243	1223	109.5	<0.001	88.0 (85.6-89.9)	-	12.0 (10.1-14.4)
Drive for thinness[2]	-2LL	df	$\Delta\chi^2$	p†	a^2	c^2	e^2
Females (continuous)							
ACE (saturated)	10840	1589	-	-	50.1 (37.3-57.5)	0.0 (0.0-10.9)	49.0 (42.5-56.3)
CE	10868	1590	28.0	<0.001	-	36.6 (30.1-42.7)	63.4 (57.3-69.8)
AE*	10840	1590	0	-	51.0 (43.7-57.5)	-	49.0 (42.5-56.3)
ADE (saturated)	10838	1589	-	-	27.0 (0.0-56.1)	25.2 (0.0-57.6)	47.8 (41.3-55.1)
DE	10840	1590	1.7	0.66	-	52.9 (45.8-59.1)	47.1 (40.9-54.2)
AE*	10840	1590	1.5	0.63	51.0 (43.7-57.5)	-	49.0 (42.5-56.3)
Males (dichotomous)							
ACE (saturated)	2084	1207	-	-	1.2 (0.0-9.1)	84.9 (78.9-87.9)	13.0 (10.9-15.5)
CE*	2084	1208	0.3	0.58	-	86.4 (84.4-88.0)	13.6 (12.0-15.6)
AE	2218	1208	134.4	<0.001	87.7 (85.2-89.7)	-	12.3 (10.3-14.8)

† The p value associated with $\Delta\chi 2$ (change in model fit compared to the saturated model).
* Best fitting model.
[1] For females, the C estimate in ACE models is very small, and good model fit is obtained by omitting the C estimate altogether: AE models give female data the best fit. For males, the A estimate in ACE models is very small, and omission of A improves model fit: CE models give male data the best fit.
[2] For females, ADE model fits the data better than ACE according to the –2 Log Likelihood statistic, but AE is the most parsimonious model. For males, the A estimate in the ACE model is very small, and it can be removed altogether without a significant decrease in model fit. CE is the most parsimonious male model.

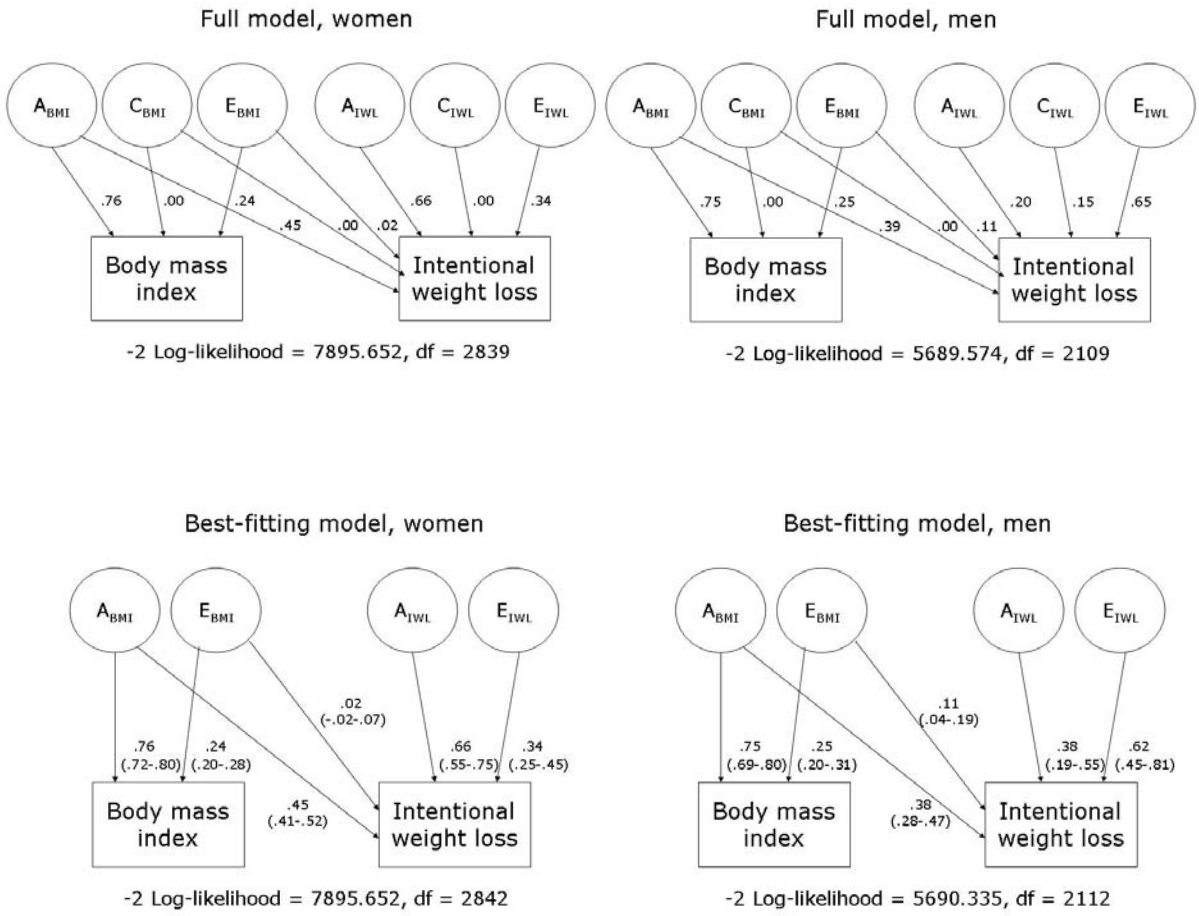
environmentally modulated in men only.

When the genetic and environmental overlap of DT and BD was assessed using bivariate Cholesky decompositions in study III, the genetic effects of DT correlated with the genetic effects of BD (r_g = 0.80, 95% CI: 0.74-0.85) in women, and unique environmental effects of BD and DT had a correlation of 0.59 (95% CI: 0.53-0.65). DT and BD in males were influenced solely by environmental factors in our models, and genetic and environmental correlations of BD and DT in males were not estimated.

Intentional weight loss

In bivariate twin models (Figure 9) tested in study IV, IWL was estimated to have a heritability of 38% (95% CI: 19-55%) in men and 66% (95% CI: 55-75%) in women. Heritability estimates of BMI were similar in both genders. The genetic covariance of BMI and IWL was 0.38 (95% CI: 0.28-0.47) for men and 0.45 (95% CI: 0.41-0.52) for women, implying that although there is some overlap in genetic factors that influence BMI and IWL, there are also substantial unique genetic influences on each trait. In men, environmental effects influencing BMI and IWL were shared, albeit to a very modest degree (r_g =0.11, 95% CI: 0.04-0.19). In women, the overlap in common environmental effects influencing BMI and IWL was very small (r_c = 0.02, 95% CI: -0.02-0.07).

Figure 9. Variance estimates from gender-specific models of body mass index (BMI) and intentional weight loss (IWL)



6. Discussion

Correlates of weight control behaviors

In this study, weight control behaviors were associated with the study participants' general psychological and physical health. Breakfast skipping was significantly associated with health-compromising behaviors, such as smoking, infrequent exercise, frequent alcohol use, and high BMI in both adults and adolescents. Thus this study confirms similar earlier findings from adolescents (Isralowitz & Trostler 1996; Höglund et al. 1998; Cavadini et al. 2000; Sjöberg et al. 2003) and expands them to adult populations, which have not been studied this extensively previously. In our study, the tendency to eat breakfast regularly increased with age in adults.

Body shape and weight concerns were associated with larger body size and multiple psychosomatic symptoms and with dissatisfaction with close relationships, such as relationship with one's parents, and feelings of loneliness. To my knowledge, these associations have not been reported previously in a population sample of young adults. Body shape and weight concerns were also clearly associated with disordered eating patterns, particularly restrictive eating, overeating, and alternating restricting-binging, confirming many similar previous observations (Grigg et al. 1996; Ackard & Peterson 2001).

Breakfast skipping and body shape and weight concerns are examples of health-related behaviors that become widespread peripubertally (Sjöberg et al. 2003). Late pubertal onset emerged as a protective factor from body dissatisfaction in women: this finding has recently been confirmed by another population study (Slof et al. 2003). Unfortunately, we do not know at which age body and weight dissatisfaction emerged in our sample, although generally they manifest relatively early, before puberty (Schur et al. 2000; Borresen & Rosenvinge 2003). Because our study design was incompletely longitudinal (body image, intentional weight loss, and nutritional variables were only assessed at the fourth wave, although information on BMI and breakfast skipping were available at baseline and the fourth wave), longitudinal evaluations of all variables of interest would be important in establishing the time sequence of these events and determining the direction of causality.

Intentional weight loss attempts were also associated with larger body size and disordered eating styles. Individuals who engaged in intentional weight loss attempted to restrict food intake and avoid fatty and calorie-rich foods, but also reported overeating, snacking, and eating in the evening. These conflicting eating styles often manifest as weight cycling; unfortunately, the amounts of weight lost and regained in each cycle were not assessed in our sample even when the number of intentional weight loss attempts was recorded.

Women in particular reported eating in response to visual and emotional cues. This trend is worrying, but fits well with earlier reports of factors that make weight loss maintenance difficult, particularly stress and anxiety, responsiveness to dietary lapses, and difficulties in implementing behavioural weight control strategies (Sarlio-Lähteenkorva & Rissanen 1998; McGuire et al. 1999a; Sarlio-Lähteenkorva et al. 2000; Dohm et al. 2001).

Our demographic analyses showed female gender was associated with breakfast skipping in adolescents, but not adults, and also with a higher risk of body and weight dissatisfaction and intentional weight loss attempts, confirming that gender differences in food intake and selection usually appear in adolescence (Rolls et al. 1991). In average, men consume more

energy than women do, and there are clear gender differences in eating styles, which may in addition to clear physiological factors indicate that women have been socialized to eat in a more feminine manner and experience more food-related conflict than men do (Rolls et al. 1991).

The parents of low SES families were clearly more likely to skip breakfast than parents of high SES families; however, low family SES was only reflected by boys' breakfast skipping and had no influence on girls. In young adults, no association was found between DT and education level. BD and IWL were more common among those with low education, but this relationship was largely mediated by BMI, which has a clear inverse association with education in the Finnish adult population (Lahti-Koski et al. 2000). Perhaps body shape and weight concerns are pervasive in the society, but individuals with a low education level have a greater risk of unhealthy lifestyles and also a greater tolerance of consequent obesity and weight gain.

Genetic and environmental influences on weight control behaviours

Substantial genetic effects were evident on most weight control behaviors assessed in this study. However, surprisingly, less than half of the genetic influences affecting weight loss attempts were shared with those affecting BMI. Thus, there seem to be considerable genetic influences on eating related phenotypes that are distinct from body size. In women, genetic contributions to weight loss and body shape and weight concerns were relatively large. In young men, body shape and weight concerns were entirely environmentally modulated, and weight loss was substantially less heritable in men than women. In adolescence, examining the phenotype breakfast eating, genetic influences were relatively strong on both girls and boys, particularly when family modeling was implemented. As the direction of the gender difference changed when different modeling strategies were implemented, the clear direction of the gender differences remains unclear.

To our knowledge, the analyses of genetic covariance between weight loss and BMI have not been accomplished previously, although several earlier reports have found small to modest genetic influences on weight change and weight gain in twins and families (Korkeila et al. 1995; Heitmann et al. 1997; Korkeila et al. 1999; Coady et al. 2002; Golla et al. 2003). On the other hand, our study replicates and confirms the heritability estimates of body shape and weight concerns in young adult women, as measured EDI subscales BD and DT and reported by previous authors (Rutherford et al. 1993; Klump et al. 2000), demonstrating that when the measure is clearly defined and well-validated, the gender, age group, and cultural context is broadly similar, heritability estimates can be very consistent indeed. But given our larger samples, we could estimate more precise 95% CIs. Also, given that the populations studied might have varying frequencies of genes that predispose to BD and DT, the consistency of these findings is remarkable.

Perhaps more remarkably, we were able to find substantial shared family environment influences on body shape and weight concerns in men, and smaller but still significant family environmental influences on breakfast skipping in adolescents. The relative paucity of shared environmental influences on BMI-related phenotypes has amazed researchers, because many of the lifestyle changes that have rendered our environment more obesogenic operate on the level of families (eg, cars, televisions, and physical effort saving household appliances are usually shared by the members of a family), leaving them to conclude: "Evidently, what the family has on table must be less important than what individuals take up from the table or leave behind" (Hewitt 1997). Another explanation may be that twin studies often have limited power to detect shared environmental effects, and that adhering to the principle of parsimony often leads to the loss of small shared environmental influences (Hopper 2000; Sullivan &

Eaves 2002). Also, shared environmental influences are probably most relevant and detectable in childhood, and begin to decrease during puberty when adolescents become more independent, particularly in Finnish society. For Finnish twin pairs and matched classmate controls aged 12 years, significant familial and local community effects were observed for a range of phenotypes (Rose et al. 2003).

In our study, breakfast skipping patterns in 16-year-old twins showed strong genetic and shared family environmental influences; individual-specific environment was much less important. Perhaps a part of the explanation lies in the nature of breakfast: it is a meal that even relatively young children can usually prepare unsupervised, although maintaining breakfast foods in the house usually requires parental contribution. This is illustrated by an American school-based study (Terre et al. 1990): 38% of 11-year-old children reported that they prepared their breakfast themselves, 45% had it prepared by their mothers, 7% by fathers, and 3% by siblings or grandparents; the rest either skipped breakfast (8%) or had it at fast-food restaurants (1%). At age 16-18 in the same study, similarly 38% of the adolescents prepared the breakfast themselves, but the proportion of breakfasts prepared by parents had declined to 34% and breakfast prepared by other family members to 1%; breakfast was skipped by 21% and eaten at fast-food restaurants by 5%. It would be of interest to assess whether the changing levels of parental contribution are reflected in breakfast eating and other meal patterns at earlier ages.

Our study on familial breakfast skipping patterns is one of the first to investigate sex-limitation using twin-family models. Beyond the parameter estimates for genetic and environmental effects described above, twin-family models were able to assess some additional features, such as cultural transmission, assortative mating, and the gene-family environment covariance. In our data, twin-family models broadly supported the results obtained using classical twin models: in the phenotypic assortment model, the covariance between genes and shared environment was small and negative, meaning that additive genetic factors and common environmental factors were passed on relatively independently in this sample, and that the classical twin model assumption that A-C correlation is zero was a valid approximation. Although both the social homogamy models and the phenotypic assortment models fit the data well, the overall fit was better and the parameter estimates were more reasonable in the phenotypic assortment model. The somewhat unrealistic results of the social homogamy models were not surprising. In the presence of strong spousal correlations these models expect larger shared family environmental effects than are typically seen in behavior genetic research. However, our data cannot explain whether spousal correlations are the result of cohabitation or assortative mating.

Cultural transmission in breakfast eating was of borderline importance, not quite statistically significant, and negative in direction, implying that the breakfast eating habits of parents (or correlated behaviors) have little direct impact on the breakfast eating of children, or that children tend to behave in opposition to parental example. Another likely possibility is that the cultural transmission estimate is an artifact of the model's assumption that the same genetic influences on breakfast eating are being expressed at the age of the children and the age of the parents. Even though we found no significant effect of parental breakfast eating on child behavior, we found significant effects of the family environment on adolescent breakfast eating for both boys and girls, implying that other types of parental effects may be present. It is possible, for instance, that the parents who encourage regular breakfasts for their children do not follow their own advice, so that children are responding to something other than direct parental modeling.

It is also possible that the crucial environmental influences on this behavior change from generation to generation; the environmental factors influencing the adolescents (even those created by the parents) may differ from those that influence the same behavior in the parents.

The adolescents participating in this study were born in the 1970s and assessed in early 1990s; their parents represent the post-WWII baby boomer generation. Although breakfast foods have not changed as significantly during the latter part of the 20th century than during the earlier part, when Finnish breakfast was still a warm meal, there is ample evidence from many Western countries that eating patterns have become more irregular, convenience foods more common, and shared family meals infrequent as a probable result from women entering the workforce (Samuelson 2000; Briefel & Johnson 2004). The current cultural atmosphere also prefers individualism over collectivism. The body shape ideals have certainly also changed during this period (Barber 1998), perhaps increasing pressure to diet and giving the omission of breakfast new contexts.

Although our twin-family models demonstrated that parental influences have relatively little role on their children's breakfast patterns, this influence was still much stronger than any other influences we were able to detect (eg, sociodemographic factors, or the twin's BMI). Breakfast skipping likely constitutes a marker of decreased health-conscious attitudes. Individuals who skip breakfast may have a higher risk of disordered eating and unhealthy weight control practices. Thus, in school health settings, screening for breakfast and other meal habits may offer a useful way to approach other potentially health-compromising behaviors that are likely to cluster with breakfast skipping.

Strengths and limitations

The strength of this study is that we were able to study several novel phenotypes in a large population-based sample. Its excellent population coverage and high response rate helped to minimize non-response bias. The participants of our study were younger and more homogenous in age than those of many previous studies; thus age x genotype bias was minimized.

In contrast to gender, SES was not as clearly associated with eating and weight related behaviors. Breakfast skipping was associated with low family SES in adults and adolescent boys, but not in girls. In body shape and weight concerns, the role of family SES and personal educational attainment was even more limited. Probably weight-conscious attitudes are so common in all levels of Finnish society that education level and SES make little difference. However, in eating patterns and BMI these influences are still clear, and their role in weight loss and maintenance in young adults should be further studied.

The greatest limitation in our analysis of eating and weight related behaviours was that for many variables of interest, we were limited to a cross-sectional design. This study cannot reveal whether a causal link exists between breakfast skipping and health-compromising factors, eating styles and intentional weight loss, or psychosomatic symptoms and body shape and weight concerns. Nor can it elucidate the direction of causality. Further studies of prospective samples starting at an early age are needed to resolve this issue; we hope that our undertakings can serve as a baseline assessment to prospective evaluations of these traits in these cohorts.

Some further caveats apply to interpreting our results, particularly those pertaining to weight loss and body image. One must be mindful of the gender differences in prevalences and distributions of those traits. To some degree, we failed to take all possible gender differences into account in our analyses. Sources of body shape and weight concerns are likely different in males and females. The measure that we used, the EDI, focuses on core areas of female body and weight dissatisfaction. Thus BD and DT subscales as measured in EDI are clearly not ideal measures of male body shape related attitudes, because domains of core importance such as muscularity and stature are completely ignored (Cohane & Pope, Jr. 2001;

McCabe et al. 2002). We addressed this concern half-way through our raw data collection by adding questions about dissatisfaction with musculature and stature. Preliminary analyses from these items reflect that these male-specific body shape concerns also have clear associations with depressive symptoms and various health-compromising behaviours (Raevuori et al. 2004).

The importance of gender differences was apparent in our genetic analyses. In none of the phenotypes studied here was it reasonable to constrain males and females to be equal. In breakfast skipping, a gender-limitation approach was used. In body shape and weight concerns, the very pronounced gender differences necessitated different modelling strategies for each gender (continuous measures for women, dichotomous for men). Because of this, results from male and female models may not be directly comparable, and loss of power due to dichotomization was reflected in the wide confidence intervals and the relative instability of male parameter estimates. Concerning weight loss, it is not clear whether women and men understood, mentally recorded, and finally reported in their questionnaires weight loss attempts similarly, although our key question was phrased in a gender-neutral way. In addition to different genetic influences, weight-loss behaviours also probably harbour very distinct socially learned gender roles: although many more men than women were overweight in our sample, and the prevalence of obesity was equal across genders, women were much more likely to engage in experiencing body shape and weight dissatisfaction and to attempt weight loss.

The generalizability of results from twin studies to the general population is often questioned. Some caveats apply to interpreting BMI-related measures derived from a twin population. MZ twins are, typically, smaller at birth than DZ twins. In an earlier analysis of this longitudinal sample, the size difference seemed to persist until the end of puberty in males (Pietiläinen et al. 1999). However, at 22-27, the differences in BMI means between male MZ and DZ twins were no longer statistically significant (Schousboe et al, 2003). Other studies of adult Finnish twins have also shown that the BMIs of twins and non-twins are comparable (Rissanen et al. 1988; Korkeila et al. 1991). The prevalences of breakfast eating in our twins and their parents were similar to those obtained from three large non-twin populations from Sweden, the U.S., and Finland (Puska & Smolander 1980; Höglund et al. 1998; Siega-Riz et al. 1998). Also, our measures of body shape and weight concerns, EDI subscale means were in line with age- and gender-specific norms from the Finnish non-twin EDI validation sample (Charpentier et al, unpublished manuscript). All this suggests that conclusions derived from the twin population can be extended to the entire population. However, heritability estimates are always specific to the environment, cohort, and culture studied (Kendler et al. 2000; Kendler 2001). Our results likely apply to adolescent and young adults in Western cultures, but cannot necessarily be extended to other times and places: if cultural influences and environment are different from those addressed in this study, heritability estimates are expected to vary accordingly.

Conclusions

Eating and body image related issues have general health implications: breakfast skipping was associated with health-compromising behaviors. Dissatisfaction with body shape and weight were associated with larger body size and multiple psychosomatic symptoms. Intentional weight loss attempts were also associated with larger body size and potentially disordered eating styles, particularly restricting and overeating.

Young adults, particularly women, who are dissatisfied with their appearances, are often overlooked by health care services. Yet these types of complaints are very common in the

context of school health services. Perhaps in this population, brief routine screenings for symptoms of anxiety and depression and substance use might serve as a warranted early intervention. As health-compromising behaviors exhibit moderate clustering, multifaceted health promotion efforts addressing also other health-compromising factors, such as tobacco and alcohol use, need to be considered. General education programs about healthful eating patterns and healthy weight control methods should be implemented in schools. Because long-term solutions to overweight and obesity are still difficult to come by, preventive efforts should receive increased attention and funding. Currently, prevention is the best solution to obesity.

Overall, both genes and environment were important for the eating-related phenotypes studied in Finnish young adult twins. Breakfast skipping in girls and boys, intentional weight loss in women and men, and body shape and weight concerns in women exhibited moderate to substantial genetic influences. Body shape and weight concerns exhibited substantial and breakfast skipping modest influences of shared family environment. The genetic influences on intentional weight loss were only partially shared with those affecting BMI. This means that for all of the phenotypes studied, there is clearly room for intervention in modifying our living environment to a healthier, less obesogenic direction. This task is daunting and likely to involve all aspects of society from zoning planners and legislators to health educators and school and workplace cafeteria staff. Parents of children and teenagers have a particular role in this effort. However, it is a task well worth undertaking.

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