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CARDIOMETABOLIC HEALTH IN STUDENTS AND YOUNG ADULTS WITH MILD/MODERATE INTELLECTUAL DISABILITIES: RESULTS FROM A LONGITUDINAL FOLLOW-UP STUDY AND A SCHOOL INTERVENTION

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"What gets counted gets noticed."

Fujiura et al., 2010

ABSTRACT

Background

Adults with intellectual disabilities (ID) develop the metabolic syndrome and cardiovascular disease more frequently than individuals without ID. The knowledge about cardiometabolic risk factors in adolescents with mild/moderate ID is scarce.

Aims

The aims were 1) to examine cardiometabolic health among adolescents with ID 2) to study the progress of cardiometabolic risk factors from adolescence to young adulthood among young adults with and without ID 3) to evaluate whether a health-promoting program in an upper secondary school for students with ID could reduce cardiometabolic risk factors 4) to evaluate whether the plate model pattern, inlaid in a specially designed lunch plate, increases vegetable intake.

Material and Methods

Sixty-six adolescents with mild/moderate ID, mean age 18.6y recruited from one upper secondary school for students with ID (year 1-4) were investigated in a cross sectional study (Paper I). Controls were 90 students without ID, mean age 17.8y, recruited from practical and theoretical programs at schools nearby. In the follow-up study five years later 35% (n=23) of the now young adults with ID and 33% (n=30) from the control group were re-investigated (Paper II). Measures were anthropometrics, blood pressure, DXA, fasting blood samples and a submaximal cardiovascular fitness test. The multifactorial school intervention was evaluated on last year students after two years of intervention (n = 11) and compared with their base-line data (Paper I) and with last year students in Paper I (Paper III). The special plate with the plate model inlaid was evaluated in an observational study. The intervention group (n = 27) had eaten on the special plate during school lunches for at least six months. The control group (n=62) was recruited from two other upper secondary schools for students with ID. Food intake was estimated from video recordings and digital photos (Paper IV).

Results

Adolescents with ID had a higher prevalence and severity of cardiometabolic risk factors together with low cardiovascular fitness compared to the control group. At follow-up as young adults (mean age 24.3) 35% were classified as obese and 22% had developed the metabolic syndrome. Those without ID from practical educational programs also developed cardiometabolic risk factors but they did not reach the same level as the group with ID. After two years of school intervention cardiometabolic risk factors had decreased and no one were obese. Evaluation of the special plate showed no difference in vegetable intake between intervention and control group. Eighty-eight percent ate $\geq 37.5\%$ vegetables. The intervention group chose food with a lower fat content and with more carbohydrates, had less plate waste and took fewer portions.

Conclusions

Already during adolescence individuals with ID have more cardiometabolic risk factors than those without ID and as young adults individuals with ID in this study has a cardiometabolic health and cardiovascular fitness similar to the Swedish middle-age population. Actions to promote healthy living habits during school hours including the use of the special plate were promising. This indicates that it is not the ID condition itself but the effects ID has on the living conditions that causes the high cardiometabolic risk. Thus, the results in this thesis show that initiatives especially designed for individuals with ID to promote healthier living habits are required and are likely to be effective.

LÄTTLÄST SAMMANFATTNING

Hjärt- kärl sjukdom är vanligare hos personer som har en utvecklingsstörning.

Personer med utvecklingsstörning blir sjuka tidigare än andra.

Redan hos tonåringar kan man se att fler börjar bli sjuka.

Det kan bero på att det är svårt att veta vad som är nyttigt och bra.

Hjärt- kärl sjukdom får man lättare om man äter för lite frukt och grönsaker.

Hjärt- kärl sjukdom får man lättare om man dricker mycket läsk.

Hjärt- kärl sjukdom får man lättare om man äter mycket sötsaker.

Hjärt- kärl sjukdom får man lättare om man rör på sig för lite.

Det är nyttigt att äta frukt och grönsaker och att röra på sig.

Det är viktigt att göra det lättare att förstå vad som är nyttigt.

Det är viktigt att göra det lättare att välja det som är nyttigt.

Den här avhandlingen visar att den som har en utvecklingsstörning kan leva nyttigt.

Den här avhandlingen visar att skolan kan hjälpa till.

Skolan kan vara en praktikplats i hälsosamma levnadsvanor.

SAMMANFATTNING

Bakgrund

Vuxna personer med utvecklingsstörning har visat sig utveckla det metabola syndromet och hjärt-kärlsjukdom oftare än befolkningen generellt. I vilken grad detta gäller ungdomar med mild/måttlig utvecklingsstörning saknas kunskaper om.

Syfte

1) Att undersöka förekomsten av riskfaktorer för framtida hjärt- kärlsjukdom hos ungdomar med utvecklingsstörning och jämföra med ungdomar utan utvecklingsstörning 2) att studera utvecklingen av sådana riskfaktorer efter övergången från ungdom till vuxen hos personer med och utan utvecklingsstörning 3) att utvärdera om ett hälsofrämjande program på en gymnasiesärskola kan minska riskfaktorerna för framtida hjärt- kärlsjukdom 4) att utvärdera om en specialtillverkad lunchtallrik med tallriksmodellen ingraverad ökar grönsaksintaget.

Material och Metoder

Sextiosex ungdomar med mild/måttlig utvecklingsstörning, medelålder 18,6 år, rekryterades från en gymnasiesärskola (år 1-4) och undersöktes i en tvärsnittsstudie (Artikel I). Kontrollgrupp var 90 ungdomar utan utvecklingsstörning, medelålder 17,8 år, rekryterade från praktiska och teoretiska program på skolor i närheten. I uppföljningsstudien fem år senare deltog 35% (n = 23) av de nu unga vuxna med utvecklingsstörning och 33% (n = 30) från kontrollgruppen (Artikel II). Mätningar som gjordes var kroppsmått, blodtryck, kroppssammansättning, fasteblodprover och ett konditionstest. Den multifaktoriella skolinterventionen utvärderades på sista-års-studenter efter två år av intervention (n = 11) och jämfördes mot deras baslinje data (Artikel I) och med sista-års-studenter i Artikel I (Artikel III). Den speciella lunchtallriken utifrån tallriksmodellen utvärderades i en observationsstudie. Interventionsgruppen (n = 27) hade ätit på specialtallriken under skollunchen i minst sex månader. Kontrollgruppen (n = 62) rekryterades från två andra gymnasiesärskolor. Beräkning av lunchintaget gjordes med hjälp av videoinspelning och digitalbilder (Artikel IV).

Resultat

Riskfaktorer för hjärt- kärlsjukdom var utvecklade både tidigare och i högre grad hos ungdomar med utvecklingsstörning i kombination med en lägre kondition jämfört mot kontrollgruppen. Vid uppföljning som unga vuxna (medelålder 24,3) hade 35% utvecklat fetma och 22% det metabola syndromet. Unga vuxna i kontrollgruppen som tidigare gått på praktiska gymnasieprogram hade också utvecklat riskfaktorer, men nådde inte samma grad som de med en utvecklingsstörning. Efter två år av skolintervention så hade riskfaktorerna minskat och ingen var klassificerad med fetma. Utvärderingen av specialtallriken visade inte på någon skillnad mellan intervention och kontrollgrupp i grönsaksintag. Åttioåtta procent fyllde sin tallrik med $\geq 37,5\%$ grönsaker. Interventionsgruppen valde mat med mindre fett och mer kolhydrater, tog färre portioner och hade mindre matrester.

Slutsatser

Redan under tonåren hade personer med en utvecklingsstörning utvecklat fler riskfaktorer för framtida hjärt- kärlsjukdom än de utan utvecklingsstörning. Som unga vuxna hade de med en utvecklingsstörning i den här studien riskfaktorer och kondition på samma nivå som svenskar i allmänhet har i medelåldern. Åtgärder för att främja hälsosamma levnadsvanor under skoltid inklusive användandet av specialtallriken är lovande. Detta ger en indikation på att det inte är utvecklingsstörningen i sig utan den effekt en utvecklingsstörning har på levnadsvanor, som orsakar den ökade risken för hjärt- kärlsjukdom. Detta tyder på att riktade preventiva interventioner för personer med utvecklingsstörning är effektiva och behövs.

LIST OF PUBLICATIONS

- I. **Flygare Wallén E**, Müllersdorf M, Christensson K, Malm G, Ekblom Ö and Marcus C.
High prevalence of cardio-metabolic risk factors among adolescents with intellectual disability.
Acta Paediatrica 2009; 98: 853-859.
- II. **Flygare Wallén E**, Müllersdorf M, Christensson K and Marcus C.
Progress of cardiometabolic risk factors from adolescence to adulthood in individuals with intellectual disabilities: A five-year follow-up study.
Submitted.
- III. **Flygare Wallén E**, Müllersdorf M, Christensson K and Marcus C.
A school-based intervention, associated with improvements in cardiometabolic risk profiles, in young people with intellectual disabilities.
Journal of Intellectual Disabilities, Volume 17 Number 1 pp 38-50 March 2013.
- IV. **Flygare Wallén E**, Müllersdorf M, Christensson K and Marcus C.
Eating patterns among students with intellectual disabilities after a multifactorial school intervention using the Plate Model.
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LIST OF ABBREVIATIONS

ID	Intellectual Disability
LSS	The law “Act on support and services for persons with certain functional impairments”
WHO	World Health Organization
IQ	Intelligence Quotient
FUB	The Swedish National Association for Persons with Intellectual Disability
ICD	International Classification of Disease
APA	American Psychiatric Association
AAIDD	American Association on Intellectual and Developmental Disabilities
ICF	International Classification on Functioning, Disability and Health
DS	Down Syndrome
NCD	Non-Communicable Diseases
CVD	Cardiovascular Disease
PWS	Prader Willi Syndrome
BMI	Body Mass Index
HDL-C	High Density Lipoprotein Cholesterol
IGT	Impaired Glucose Tolerance
IFG	Impaired Fasting Glucose
AHA	American Heart Association
NHLBI	National Heart Lung and Blood Institute
IASO	International Association of the Study of Obesity
FM	Fat Mass
LBM	Lean Body Mass
WC	Waist Circumference
DXA	Dual-energy x-ray absorptiometry
CVF	Cardiovascular Fitness
HR	Heart Rate
VO ₂ max	Maximal Oxygen Consumption
CRP	C-Reactive Protein
Non-ID	Without intellectual disability
Non-ID-p	Without intellectual disability from practical high school education
Non-ID-t	Without intellectual disability from theoretical high school education
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure
BMD	Bone Mass Density
BMC	Bone Mass Content
A/G	Android/Gynoid Ratio
FFM	Fat Free Mass
HOMA-IR	Homeostasis Model Assessment – Insulin Resistance
RPE	Rate of Perceived Exertion
Non-DS	Intellectual disability without Down syndrome
Hs-CRP	High Sensitive-C-Reactive Protein
WHR	Waist to Hip Ratio
WHtR	Waist to Height Ratio

PERSONAL NOTES

This thesis stems from more than 20 years of experience as a school nurse working with children and adolescents with an intellectual disability (ID). Over the past years, I have come to question the living habits of children and adolescents with an ID. To begin with, many of them are overweight and their daily lives often revolve around coffee breaks. In addition to this, people with an ID are sometimes protected from a physically active life and, to make matters worse, talking about healthy living habit in the nurse's office is bound to fail because learning takes place in everyday hands-on situations for people with an ID.

A Swedish school year consists of 185 days. Most children go to school for 13 years, for a total of 2405 days. Every day a school lunch, snacks, and, in some schools, even breakfast is served to pupils, and each day presents an opportunity to be physically active. If all these days were based on healthy living habits, with staff and other students serving as role models, my idea was that students would in fact become used to what healthy living habit entail. Even if they did not continue with these particular habits, at least some healthy habits would have been formed.

My thesis work is derived from this idea of changing the school environment through scheduled physical activity, staff and students acting as positive role models, healthy meals being cooked on site, and an exclusion of unhealthy choices. There have been additions and some changes along the way, but I sincerely hope that this thesis can lead to further interventions and studies in this field.

1. INTRODUCTION

Living with an intellectual disability (ID) in a country that closed its institutions years ago can be challenging from a living habit and health perspective. This must by no means, however, be interpreted as a wish to get back the institutions. The magnitude and consequences of this challenge, when expressed as development of risk factors for premature cardiovascular disease and cancer, are perhaps unknown to people with ID, as well as their parents, clinicians and, politicians.

1.1 HISTORY

Sweden and Norway are the two countries in the world that have closed all institutions for people with an ID. This was carried out between 1970 and 2005 (1) as a result of the sociopolitical drive for integration and normalization for people with IDs as formulated in the Normalization Principle (2) published by the Swede Bengt Nirje in 1969. The normalization principle involves providing patterns and conditions for people with an ID as close as possible to those of everyone else. To live under equal conditions and with the same opportunities and possibilities as other citizens is a human right for all people in a society (2). The institutions at this time in Sweden functioned like small municipalities (3) with little or no opportunity for people with an ID to participate in mainstream society. This segregated and stigmatized people in contradiction to the full citizenship principle. Closing the institutions then became crucial in order to implement the normalization principle, i.e. allowing people with an ID to live in society like everybody else (1).

The normalization principle has been interpreted differently around the world, and integration by closing the institutions became the most important aspect in Sweden (4). It comprised moving out to group homes and getting all services through community care and the generic service system in the community (1). The responsibility for the care of people with an ID was a state responsibility until 1954 at which time it was then transferred to the counties and then, in 1990, to the municipalities (1).

The Swedish Act concerning Support and Services for Persons with Certain Functional Impairments (SFS, 1993:387) (LSS) (5) is a rights-based law, a law on top of those provided under general social legislation, which, since 1994, gives people with IDs (together with a few other vulnerable groups) more individual rights (1). This law has 10 areas of service and support and stipulates that people with an ID can make requests if they need and want to, and if the application is rejected, there is the possibility to appeal. This law is handled by local authorities in the municipality except for one issue, 'personal counseling', that is still under the authority of the county council.

1.1.1 Health Care and Health Promotion for People With an ID

When the counties were responsible for the housing, health, and well being of people with IDs, there were medical professionals specialized in ID. These professions virtually ceased to exist when all services were transferred to the municipalities and health care to the generic system (6, 7). Health promotion for people with an ID has not received a great deal of attention, even though people with an ID might be in as much need of this as the population at large. This is not only an issue in Sweden. In WHO's atlas 'Global Resources for Persons with Intellectual Disabilities, 2007', only between 30 and 40% of pediatricians, primary health care workers and physicians in the world were reportedly trained to work with people with IDs compared to the percentage of special educators, which is 76% (8). Only 38% of the high-income countries had any epidemiological information on people with an ID compared to 32% in the whole world (8).

1.1.2 Different Perspectives on an ID

During the time when institutions were in use, an ID was considered to be a medical issue; a pathological problem in an individual that required medical care. In the social sciences this is referred to as the medical model (9). Different models have evolved over the years such as, the social model in the UK, which sees the disability as being caused by the environment and is therefore, a social construction. In the US there is a minority perspective that views people with disabilities as a subgroup, a construction of culture, whereas in Sweden there is a relational perspective. This perspective, referred to as the 'Nordic relational model', views people with disabilities as being 'disabled through dynamic relationships of body/mind and the environment' (9).

The fact that an ID is no longer regarded as a medical condition has led in many countries, including Sweden, to the absence of specialized medical professionals (6, 10). The health care system may not be aware of the manifestations of an ID and the drawback here is that people with an ID sometimes face special difficulties concerning their health because of their limited cognitive skills.

1.2 INTELLECTUAL DISABILITY

1.2.1 Terminology

"Intellectual disability" is the preferred term in the European Union (11) as well as in large parts of the world today (8, 12), and is translated into Swedish as "intellektuell funktionsnedsättning". The term mental retardation is still the most commonly used term in the world (8), although less frequently in high-income countries. In these countries this term is viewed as a remnant from the time when an ID was regarded as a medical condition. The term mental retardation is still in use in most of the classification systems, but this is changing. In the UK the term "learning disability" has represented people with an ID, but in many countries (one of them Sweden) "learning disability" refers to people with a learning disability but with an intelligence quotient (IQ) above 70, which is considered a normal IQ. In Australia the term "developmental disability" has been used and in the US the term

“mental retardation” is still used but the term “intellectual disability” is preferred. In Sweden the National Board of Health and Welfare decides what term should be used officially and that term is “utvecklingsstörning”, or with “psyisk” (mental) in front, which translates to “(mental) developmental disorder”. Canada has used the term “developmental disability” meaning only ID (12). Unfortunately the term “developmental disability” in other English-speaking countries includes all developmental conditions (13). “Developmental disability” in the US, for example, is broadly used as an umbrella term for disabilities that share some characteristics, one being that it has been diagnosed before the age of 22. Some examples of what is included in “developmental disabilities” are mental retardation, autism, Asperger’s syndrome, and cerebral palsy. Thus, intelligence can be both lower or above average (IQ 85 – 115). Sensory impairments as deafness and blindness are not included when being a single disability (14). On the other hand, intellectual disability translated to Swedish as “intellektuell funktionsnedsättning” means all intellectual disabilities (cognitive impairments) and not only ID. In Sweden neuropsychiatric disabilities (neuropsykiatriska funktionsnedsättningar) is the umbrella term for diagnoses such as autism, attention deficit hyperactivity disorder, and Asperger’s syndrome or neurodevelopmental disorders that comprise diagnoses such as mental retardation, cerebral palsy, and spina bifida (15). “Utvecklingsstörning” is the word that Swedish people with an ID and members of The Swedish National Association for Persons with Intellectual Disability (FUB) voted for in 2011(16). Thus, the term “intellectual disability” is used in this thesis and when writing scientific literature, but when speaking in Swedish “utvecklingsstörning” is the preferred term.

1.2.2 Classification Systems

There are several manuals or classification systems in use around the world for intellectual disabilities, and the criteria for these are generally similar. Three areas of deficits should be fulfilled: an IQ below or equal to 70, several adaptive problems in the domains social, practical, and conceptual abilities, and the disability must originate before the age of 18.

In Sweden, and world-wide, the WHO’s International Classification of Diseases (ICD-10) (17) classification of mental and behavioral disorders, clinical descriptions, and diagnostic guidelines is the most commonly used (8). The Swedish National Board of Health and Welfare published a new Swedish version in 2011, ICD-10-SE (18). The second most used classification system, in the world and in Sweden (8), is the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Revised (DSM IV-TR) published in 2000 by the American Psychiatric Association (APA) (19). This is used in Sweden, because several diagnoses including an ID, such as autism, are diagnosed by psychiatrists. The APA uses the term mental retardation (DSM Axis I; 317 – 319) as having impairments in at least two of the following adaptive behaviors: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health or safety. A new version of APA’s

classification system, DSM-V with the umbrella term “Neurodevelopmental disorders” and the subclassification “intellectual developmental disorder” is currently being developed (20). All diagnoses according to the APA are converted to ICD-10 by the Swedish National Board of Health and Welfare. A third classification system is the one used in the US by the AAIDD, the American Association of Intellectual and Developmental Disabilities (21). In 1992 the AAIDD (then known as the AAMR) stopped using the classification with levels of an ID such as mild, moderate, severe, and profound ID, but this system is still in use in the other two classification schemes. In Sweden, the ICD-10, is frequently used together with the ICF, the WHO’s International Classification of Functioning, Disability, and Health, in order to clarify areas that are related to health and health function (22). Intellectual disability is thus a functionally defined diagnosis with no exact measures, but instead has criteria that should be fulfilled. The reason for this thorough review of these terms and definitions is the large impact they have on the ability to compare and interpret international data in this field.

1.2.3 Causes of Intellectual Disabilities

In many cases a specific cause for the ID is unknown, and this is the case for about half of the population with an ID and is greater in cases with a mild ID compared to more severe ID (23). The most common genetic cause is Down syndrome (DS) and the most common heritable cause is Fragile X syndrome (23-25).

1.2.4 Population

The prevalence of intellectual disabilities is uncertain, not only because there are few countries that keep records, but also because of the fact that it is a functional diagnosis. It is also a social construction that changes depending on context and time. Most people with an ID are identified between the ages 10 to 16 years, especially those with a mild ID (26). Those having a more severe ID or a specific syndrome are usually diagnosed by a physician at an early age. The reasons for this could be the variability in the assignment of the diagnosis ID. Those with a mild ID are usually not tested until more advanced academic achievements are expected in school. Another reason for testing for IDs is when children are missing certain adaptive skills. It is almost five times as common for children with an ID to have behavioral problems (26).

A prevalence of ID of 0.44% was reported from the Swedish National Board of Health and Welfare in 1979, with a further expected decrease in number, and 23% of those were diagnosed as having a mild ID (4). At that time this was considered the lowest prevalence in the world (4). Reasons discussed for the expected further decrease were a decrease in the incidence of multi-handicapped children; a positive effect from programs of early stimulation and habilitation; a positive effect of systematic training and education (4). Since the 1970s, there are, to my knowledge, seven studies in Sweden reporting the prevalence of ID, with reports of mild ID between 0.4 and 1.4% in children (age groups between 8 and 19 years) and severe ID with an IQ < 50 of between 0.3 and 0.4% (aged 1 to 16 years). These studies were

from populations of 6,000 to 40,000 children (27-31). Gillberg estimated the prevalence of ID to be between 1 and 2.5% (6). In Europe the estimation of people with an ID is around 1% (11). A meta-analysis of population-based studies reported just over 1%, and studies with children and based on psychological assessments showed higher levels (32). The incidence and prevalence of ID in two Finnish birth cohorts were followed for 11.5 years and showed a stable prevalence over 20 years between 1966 and 1986 of 1.1% and an incidence of 1.3% in 1986 (33).

In developed countries, the reported variance in prevalence of mild ID (0.2– 3.7%) was higher than that of severe ID (0.2 – 0.6%)(33). The FUB reports a total of 38,000 children and adults which would equal a prevalence of 0.4% (16).

Sweden has kept no national records since 1994 of people having an ID and uses an administrative definition of ID in the existing records. These records consists of persons that have special support and services in accordance with LSS law and/or attend the special school program for students with ID (5). One is eligible for services according to the LSS act he or she is assessed belonging to one of three groups stated in the law. Group number one are people with intellectual disabilities.

The National Board of Health and Welfare, reported 53,236 children and adults with ID in 2011 whose request for support were granted (34). The majority of these are persons aged 10 to 30 years and included more males than females. This equates to a prevalence of ID of 0.5% in a total population of 9,514 406 (35). In 2011, 19,825 children and adolescents (ages 7 to 20 years) were admitted to special school programs out of a total of 1,257,741 students admitted to all schools. This is a prevalence of 1.6% of all school children having an ID (36). Of these students, 18,125 (ages 7 to 22 years) also received support and services in accordance with LSS. The Swedish records, therefore only cover students in special school programs and/or those getting support according the LSS law.

1.3 NON-COMMUNICABLE DISEASES

Non-communicable diseases (NCDs) are currently the largest cause of death worldwide. Premature death (before the age of 60) due to an NCD occurs in eight percent of men and ten percent of women in high-income countries and these numbers are more than tripled in low-income countries (37). In Sweden, NCDs are estimated to account for 90% of deaths and the most common diseases are, by far, cardiovascular diseases (CVDs) and diabetes. Together these ailments are responsible for 44% of all deaths compared to 25% from cancer and 5% from accidents (37). These diseases are regarded as preventable in most cases and the four main behavioral risk factors for CVD and diabetes are tobacco use, physical inactivity, harmful use of alcohol and an unhealthy diet (37, 38).

1.3.1 Cardiovascular Disease and Type 2 Diabetes

The evidence for the importance of preventing CVD and type 2 diabetes is growing rapidly, and over the past decade more than 150 000 papers on the subject physical activity and health and 200 000 on food and health have been published. The vast

majority of these focus on cardiovascular health (39). In the Swedish National Guidelines for Methods of Preventing Diseases published in 2011, the health care system strongly recommended increasing efforts in four primary prevention fields: tobacco use; hazardous use of alcohol; insufficient physical activity; and unhealthy eating habits (38). The health care system currently offers advanced advice and counseling on these four fields according to these guidelines.

1.3.2 Cardiometabolic Risk Factors

High energy intake in relation to energy expenditure is strongly associated with overweight or obesity that in turn is associated with an increased risk for CVD, type 2 diabetes, and some cancers. But not all who are overweight or obese develop CVD, diabetes type 2, or cancer, and some individuals who are not overweight or obese do develop these diseases. Many different factors contribute to whether the individual will develop obesity or not. Childhood obesity and determinants that have been proven to be associated with obesity can be both modifiable and non-modifiable. Examples of modifiable risk factors are being sedentary or having a low level of physical activity; poor diet; insufficient sleep; poor living conditions, living in an urban or rural environment; intrauterine exposure to high maternal adiposity, and gestational diabetes. The latter is considered modifiable because of results of studies on obese mothers who had had bariatric surgery showing that the offspring tended to become less obese compared to their siblings born before their mother had the surgery. Other modifiable risk factors are the socioeconomic position, which could be modifiable with political and culture changes; some endocrine diseases; epigenetics, with the possibility to modulate gene transcriptions in the developmental environment both prenatally and during childhood (40, 41). Risk factors that are not modifiable and are associated with obesity are; heredity; birth weight, including those born with high birth weight and those born small for gestational age; early age at body mass index (BMI) rebound; ethnic origin; and country of birth (40). In addition, medications frequently used in people with ID are often associated with obesity, including psychotropic drugs, anticonvulsants, and hormonal contraceptives (40, 42).

Obesity is a complex disorder involving many interacting genetic and environmental factors (40). It is highly heritable with a genetic contribution of between 40 and 70% if one has two obese parents (43-46). About 30 specific genetic loci have been found associated to obesity but together these do not explain more than a few percent of the variation in BMI (43). There are some rare specific syndromes related to obesity that have variations in ghrelin, the hunger signal hormone, and leptin, an adiposity signal hormone and both of these have a major influence on the energy balance in the body (40, 47). Some of these syndromes are associated with ID including Prader-Willi Syndrome (PWS) and Bardet-Biedel Syndrome (40).

1.3.2.1 Consequences

The consequences of an increase in adipose tissue is that this contributes to elevated levels of circulating free fatty acids and adipokines. The adipokines contribute to both a proinflammatory state and a prothrombotic state. The increase in free fatty acids leads to an increase in insulin resistance in the muscles and liver and an elevation in plasma glucose. High levels of free fatty acids also increase triglyceride levels in plasma, which lowers the amount of high-density lipoprotein cholesterol (HDL-C). High levels of free fatty acids can also damage β -cells through lipotoxicity, and this also increase glucose levels (48).

Glucose homeostasis is required for the body to remain healthy. Glucose is one of the body's main sources of energy and thus it is important to be able to store it for use between meals and during exercise. Under normal circumstances, many tissues can also use fat or protein as an energy source in addition to glucose, but some organs, such as the brain and red blood cells, can only use glucose and ketone bodies. The essential hormone insulin reduces glucose release from the liver and stimulates glucose uptake in muscle cells and fat cells to be used as a source of energy. Glucose is stored as glycogen in liver and muscle cells. When the blood glucose concentration is low, stress hormones such as adrenalin, glucagon, and cortisol are released. These hormones mobilize glycogen, which is converted to glucose by glycogenolysis. Additional glucose may also be produced from non-carbohydrate precursors, such as triglycerides (glycerol) and protein (amino acids), through the process of gluconeogenesis.

Disrupted glucose and insulin homeostasis are early consequences of obesity and are a step towards pre-diabetes and diabetes. When glucose levels are not regulated properly, many cells and organs suffer and one particularly harmful target during hyperglycemia is suggested to be the endothelial cells (48). Pre-diabetes, defined as impaired glucose tolerance (IGT) and impaired fasting glucose (IFG), increases the risk of developing diabetes and CVD (49). The risk of developing diabetes is higher when both IGT and IFG are impaired. Pre-diabetes is associated with the metabolic syndrome. Obesity is strongly associated with both pre-diabetes and the metabolic syndrome, but the underlying mechanism behind this connection is still unclear and under debate (48). Two mechanism that are probably involved are insulin resistance and systematic inflammation, both of which are effects of obesity. Pre-diabetes only has a minor association with microvascular disease, but can to some extent, predict macrovascular disease. The metabolic syndrome, however, is a much stronger predictor of cardiovascular disease and diabetes type 2 (48).

Obesity is associated with hypertension but this association is not fully understood (48). At middle-age, every 20 mmHG in systolic blood pressure and 10 mmHG in diastolic blood pressure are associated with a two-fold difference in the stroke death rate and a similar death rate due to ischaemic heart disease (50).

1.3.3 Metabolic Syndrome

Metabolic syndrome was first described 80 years ago and has over the years, been described with slightly different components, different cut offs, and under different names such as Syndrome X, The Deadly Quartet and the Insulin Resistance Syndrome (51). The syndrome contains a cluster of risk factors for CVD and type 2 diabetes that occur together more often than by chance alone (52). The core components of the metabolic syndrome are obesity, insulin resistance, dyslipidaemia, and hypertension (51). Recently there has been a debate as to whether or not the metabolic syndrome predicts CVD better than the individual components of the syndrome (53). One problem with evaluating the evidence of the additional benefits of considering the metabolic syndrome as whole is that different variables and different cut offs have been used by different organizations (53). Recently a consensus was come to regarding the definition of the metabolic syndrome by all of the primary actors in the debate including: the International Diabetes Federation (IDF); the American Heart Association /National Heart Lung and Blood Institute (AHA/NHLBI); the International Arteriosclerosis Society; WHO and the International Association of the Study of Obesity (IASO) (52, 53). The metabolic syndrome is diagnosed if three of the five following variables are present: a waist circumference for females ≥ 80 cm and for males ≥ 94 cm (this is the European cut off and, adjustments to national or regional cut offs are used); blood pressure $\geq 130/85$ mm/Hg; fasting glucose ≥ 5.6 mmol/L; fasting triglycerides ≥ 1.7 mmol/L; HDL-C for females < 1.29 mmol/L and for males < 1.03 mmol/L. These cut off levels can be used on adolescents age 16 years and older (54, 55). The cut off levels included in the metabolic syndrome are lower for the blood pressure and fasting glucose variables than when these levels are used to diagnose hypertension ($>140/90$ mmHg) and diabetes (≥ 7.0 mmol/L), respectively (56, 57). The metabolic syndrome usually starts with the onset of obesity and insulin resistance (52).

1.3.4 Body Composition

Body composition includes fat mass (FM), bone mass and lean body mass (LBM). BMI is the simplest measure of body composition, but it does not separate these different components and only considers weight in relation to height. This is why a “normal” BMI can include both unhealthy levels of fat mass and muscle mass and does not give any information about the fat mass location. Measuring waist circumference (WC) gives some information about the location of the adiposity, but not if this adiposity is subcutaneous or visceral located fat, and it gives no information about muscle mass. Other techniques for measuring fat mass are skinfold thickness and bioelectrical impedance. Skinfold thickness measures fat under the skin and is a good measure to detect changes in percentage of fat mass and to predict adiposity a better measure compared to BMI (58). It is less adequate on lean or obese individuals, does not measure muscle mass, and require good technique to get useful results (59). Bioelectrical impedance analysis estimates total body water, both intra- and extra-cellular water, and calculates body composition together with weight, height, age, and sex (60). Despite these measurements, this

techniques has poor accuracy in detecting changes in percentage fat mass (59) and is not recommended for single individual measures (60). This technique is, however, frequently used in published studies. Dual-energy x-ray absorptiometry (DXA) measures body composition in more detail and can provide overall and regional assessments of lean, fat, and bone mass (59). LBM is mostly muscle mass and is associated with the body's metabolism. Skeletal muscle plays an important role in many biochemical processes and is the second largest body composition component after fat mass. It is important in terms of body weight, blood pressure, blood lipids, and blood glucose and thus is an important component to measure.

The DXA measurement does not distinguish the location of the abdominal fat to determine if it is intra-abdominal or not. This is a valuable piece of information because the level of visceral fat is strongly associated with insulin resistance and cardiovascular health. Several mathematical equations have been suggested for calculating these variables, but these have not been used in this thesis (61). Magnetic resonance imaging is a method that separates abdominal fat but is more expensive and more difficult to access, and computed tomography gives significant exposure to ionizing radiation (61).

All body composition parts differ between the sexes after puberty with bone mass and lean body mass content being higher in males individuals and fat mass higher in females of the same BMI (62).

Obesity is traditionally viewed as beneficial to bone health because of its mechanical loading, and many studies report a positive association between being overweight and having higher bone mass density (63). Probably, however, obesity is detrimental to bone health (64) and is reported associated with a higher risk for fractures in both children and adults (65, 66). DXA measures bone in two dimensions, but bone is a three-dimensional structure. This is why DXA is not the optimal method for measuring bone mass, but bone mass is not the primary target in these studies. A recent study on pre-pubertal children reported a positive association between fat mass and bone size after adjustment for LBM, but a negative association when the true volumetric density was assessed by pQCT (peripheral quantitative computed tomography)(67). The cause for the decreased bone mass associated with obesity is still unclear but many explanations are suggested including increased cytokine and leptin levels, reduced adiponectin levels, and reduced calcium absorption (64).

1.3.4.1 BMI

In adults there is an internationally agreed upon standard for classifying BMI: <18.5 represent underweight; 18.5 – 24.99 represents normal weight; 25– 30 is overweight; and ≥ 30 is regarded as obesity (68). The BMI classification is different for children due to many factors that affect weight such as age, sex, puberty, and ethnicity and several different methods are used. Cole's age- and gender- specific cut off points are used in an international classification of overweight and obesity

that correspond to the adult BMI for overweight and obesity and have been adopted by the International Obesity Task Force (IOTF) (69). Many countries compare children against country- specific charts. For example, the US classifies those with a BMI > 95th percentile as obese; > 85th percentile as overweight; and a BMI < 5th percentile as underweight (70).

1.3.5 Cardiovascular Fitness

Physical fitness consists of “a set of attributes that are either health- or skill-related” and cardiovascular fitness (CVF) is one component of the physical fitness. The other components are muscular, metabolic, and morphological fitness (71). In the sub-studies in this thesis only CVF is measured. CVF measures the ability of the body to transport and use oxygen (72) and is a direct measure of a person’s physiological health status. There are several interchangeable names for CVF such as cardiorespiratory fitness, aerobic capacity, aerobic fitness, aerobic work capacity, and maximal oxygen uptake. CVF is a predictor of cardiovascular disease in adults (73). In recent years, several studies have shown that a low CVF is strongly associated with total and abdominal fat in children (74, 75) as well as to other risk factors for CVD (76, 77). Longitudinal studies have shown that CVF affects risk factors present from adolescence to young adulthood (78, 79) and this association is suggested to be independent of physical activity levels (76, 80). Maximal oxygen consumption (VO₂ max) is an objective measure of CVF and it is the maximal amount of oxygen that a person can consume at a certain time (81) p.106).

1.3.5.1 *Measuring Cardio Vascular Fitness*

VO₂ max can be assessed via indirect calorimetry during a maximal exercise test or estimated in a maximal or a sub-maximal test. VO₂ max is expressed in absolute or relative terms. The absolute measure is oxygen uptake/time presented in litres per minute and the relative measure is oxygen uptake in milliliters per bodyweight or LBM in kilograms per minute, thus fitness. Estimation from sub-maximal tests can be made because heart rate (HR) has a linear relationship with oxygen consumption under a certain workload. Specific nomograms have been developed for certain tests (82).

1.3.5.2 *Cardio Vascular Fitness Test for Individuals With ID*

Several fitness tests have been evaluated for validity and reliability in the population with ID with and with or without DS, and most of these have been developed for adolescents and young adults (83-88). CVF tests can be divided into field tests and laboratory test; into maximal tests or sub-maximal tests; and into performance tests or tests that requires a minimum of performance. Most tests validated for people with ID are field tests that are in a large part performance-based such as the shuttle run test and the walk/run test (85-87). All maximum tests, such as maximum capacity bicycle tests and treadmill tests also rely mostly on performance. Sub-maximal tests are bicycle tests or step tests and estimates a maximal heart rate from nomograms.

Performance tests were excluded from our studies, even though more of these are validated for people with ID, with and without DS, such as the 600 yard walk-run, the 16 m shuttle run, and the 20 m shuttle run (83, 85). Tests on treadmills were not an alternative in our studies because this is a laboratory choice. Their usefulness is also affected by the participant's weight and they are more difficult to use in different locations.

We chose the sub-maximal ergometer bicycle test performed according to Åstrand together with the Åstrand and Rhyming nomogram (the Åstrand test)(89, 90), a bicycle test frequently used for measure of CVF, and a field test that does not incorporate performance and does not depend on the participant's weight (91). The Åstrand test is used in many studies both in children (91, 92) and adults (93) without ID. Predicting VO_2 max compared to a direct-measure of maximal VO_2 consumption from a running test has recently been shown to have no mean differences at the group level (mean difference $0.01 \text{ L O}_2 \text{ min}^{-1}$, 95% CI -0.10 to 0.11) in adults (94). Underestimation of CVF in younger children (ages 11 to 12 years) without ID has been reported (92) as well as overestimation in adults with ID (95, 96). The measurement error in a sub-maximal tests is the same for the same individual over time as long as the procedures are standardized. The test has been suggested to be sub-optimal for comparison between individuals but it does have indicative properties on a group level.

1.3.6 Unhealthy Living Habits

1.3.6.1 *Lifestyle and Living Habits*

The term lifestyle is defined by MeSH as “a typical way of life or manner of living characteristics of an individual or group” and in the Swedish public health dictionary says that “since the 70s in social medicine, the term lifestyle has had the meaning of describing personal habits without any reference to society and including the fields smoking, drugs, exercise and food habits” (97). Lifestyle has thus commonly been understood to be synonymous with personal choices and their attendant connotations of right or wrong (98). Today, however, there is a much wider understanding of the determinants of an individual's lifestyle (99-102). In Sweden the term “levnadsvanor” or “living habits” has replaced “lifestyle” to cover the total context defined as “specific behavior in daily life activities that the individual can affect” together with how living habits are affected on a micro and macro level with living conditions and living environment (38). The micro level concerns the individual's specific circumstances such as living, work, education, and material hardship and is often used to grade living conditions. The macro level concerns socioeconomic background and social systems beyond the individual's ability to control (38).

1.3.6.2 *Measures of Unhealthy Living Habits*

Living habits that are followed by the Swedish National Institute of Public Health are the intake of vegetables and fruits, physical activity, sedentary leisure time, weight, gambling habits and the use of tobacco, drugs, and alcohol (103). Other

living habits associated with being less healthy are lack of sleep and too much unhealthy stress, but these were not measured in this thesis nor were alcohol use, drugs use or gambling habits.

1.3.6.3 Social Determinants Associated With Bad Health

Material hardship, education and employment have been known for many years to affect living habits and cardiovascular health (104). Several recent reports (99, 105) have include many more societal issues that affect an individual's cardiovascular health. These issues go further in measuring health than only looking at smoking or eating habits, physical activity habits. Socioeconomic patterns characterized most living habits in the latest public health report on living conditions and health in Sweden (106). Those with short education and poor personal finances tend to have worse living habits compared to those with extensive education and strong finances. Students at practical school programs are at greater health risk compared to those on theoretical programs and people with a disability are more likely to have unhealthy living habits. There is also a link between living environments and living habits. Those who engage in more sedentary leisure time activities report feeling more unsafe compared to those who are physically active 30 minutes per day, and eating habits are affected with the local food supply (106). Conditions during childhood may affect health later in life. Risk factors for health problems were 80% more common in children and adolescents coming from less favorable social circumstances, and an association with delayed cognitive development has recently been reported (107).

Suggestions have also been made that a health related selection operates already in childhood and appears to influence socioeconomic position later in adult life (108).

1.4 PREVALENCE OF CARDIOMETABOLIC RISK FACTORS IN ID

1.4.1 Adults

Adults with an ID has during the last 20 years been reported with more obesity, being less physical active and with more developed cardiometabolic risk factors (42, 109-117) or at least at the same level as the population at large (118-121). Obesity are more common among females with an ID compared to females without an ID (109-111) and also compared to males with an ID (111). Underweight is reported more often in the ID group compared to the general population and more often among males with an ID (110, 111, 115). The most frequent associations with obesity except being female in the group with ID reported is having DS, having autism, not having a cerebral palsy, being younger, being independent or having a milder ID (110, 111).

1.4.2 Adolescents and Children With an ID

A few recent studies on the physical health of adolescents with an ID have been published. These have recruited participants from schools or from clinical institutions and their results have been mixed (122-127). Increased levels of obesity have been reported as early as in pre-school children with developmental delays (128, 129), and obese children have been shown to more often suffer from developmental delays (130).

1.4.3 National Health Survey Data

National health survey data on this target group is lacking from all parts of the world (131, 132). Over the past decade attempts have been made to present a bigger overview of suspected health disparities between people with an ID and the general population in Europe (11). The EU-funded Pomona project developed a set of health indicators specific to people with an ID, investigated these throughout Europe, and concluded that disadvantageous health disparities exist for those with an ID, and that there is serious lack of experimental evidence in several fields (133-137). In addition, in 2006 the Journal of Mental Retardation and Developmental Disabilities Research Reviews published several papers on health disparities among adults with an ID compared to the general population (113, 114, 138, 139). Very little is known about Swedish conditions in adults and nothing is known concerning children or adolescents with an ID (140).

1.5 DOWN SYNDROME AND ALTERED METABOLISM

Individuals with DS have an altered metabolism and seem to be protected against atherosclerosis even though they tend to have high levels of body fat, triglycerides, and the inflammatory marker C-Reactive Protein (CRP) (141). The reason for this is unclear. One suggestion is that the extra copy of chromosome 21 that leads to DS has functions that, in triplicate, alter metabolic function (142). For example, compared to the population at large persons with DS have been reported to have increased levels of oxidative stress and impaired endothelial function (143); a propensity for developing Alzheimer's disease at an earlier age associated with higher total cholesterol levels, however delayed if treated with lipid lowering statins (144); earlier onset of type 1 diabetes together with reduced insulin dependence (145-147); menopause at an earlier age (148); and sleep apnea in children (142, 149). The resting metabolic rate has been suggested to be lower in pre-pubertal children with DS (150) but not in adults with DS (151). Children with DS have been reported to have higher leptin levels in relation to the percent fat mass (152) compared to children without DS.

Individuals with DS are reported obese more frequently compared to the general population as well as to the population with ID in general (153, 154). This occurs also at an earlier age and with a peak in their thirties compared to ten years later in the general population (155, 156). Obesity is more common among females with DS compared to males with DS (141, 155, 157, 158) and they tend to have more abdominal fat mass (159, 160). Other cardiometabolic risk factors reported to differ

compared to the population at large and to the general ID population are lower blood pressure (158, 161, 162), lower LBM (153, 159), lower bone mass density (153, 160, 163) and low incidence of hypertension (121). Glucose and insulin levels have been reported to be both higher and lower (153, 157, 158, 164), and no differences in certain blood lipids have been seen although some studies have reported higher triglyceride levels (141, 164, 165).

People with DS have a low maximal heart rate, diminished work capacity with concomitantly reduced VO_2 max, a reduced heart rate response to exercise, and chronotropic incompetence (166, 167). The explanation for this dysfunction is still unclear. Reduced blood pressure and heart rate response to sympathoexcitatory tasks such as the isometric handgrip exercise indicate reduced sympathetic nervous system activity (168). Alterations in the autonomic nervous system with greater parasympathetic activity at rest has also been found but the group difference disappeared with the onset of exercise and thus could not be responsible for the chronotropic incompetence (166). Reduced total heart rate variability, which indicates a possible autonomic dysfunction, has been reported (169). One suggestion for the reduced heart rate response to orthostatic stress is an impairment in the autonomic cardiac regulation with a greater baroreflex sensitivity when standing (170). However a lower baroreflex sensitivity during the isometric handgrip exercise and at rest when controlling for systolic blood pressure has been reported (171). A heightened sympathetic modulation of heart rate variability has been reported, and this could indicate a poor cardiac responsiveness to changes in autonomic modulation during exercise (172).

This divergence in cardiometabolic risk factors as well as in cardiovascular fitness, and the abnormal physiology in individuals with DS compared to the general population with ID, requires attention.

1.6 HEALTH PROMOTION

The aim of a public health system is to achieve the best possible health for its population and to equally allocated its service (97). Health promotion is the process of enabling people to increase control over their health and its determinants and thereby improve their health. This is a core function of public health services and contributes to the work of tackling communicable and non-communicable diseases and other threats to health.

1.6.1 School and School Health Service

The new Education Act (SFS 2010:800) has strengthened the school's responsibility in terms of disease prevention and health promotion (173) by imposing that schools strive to promote healthy living habits. Education in healthy food and exercise habits is emphasized in particular. The Act also points out that the school should create a healthy environment for children to learn in and to use scientific evidence in achieving this goal.

The school nurses have an important part in fulfilling these responsibilities. In the guidelines for the school health service, it is stated that school nurses are to attend in particular to students with health risks related to living habits (174). This should be a priority area for the work of the school nurses and the school health service should be diligent in its health promotion work.

1.6.2 School Interventions

Several reviews and meta analyses suggest that school interventions are effective (175-180) especially if they combine increased physical activity, healthier food, and health education (177, 179, 180). There is strong evidence for an increased effect of interventions with younger children (178), for longer interventions (181), and for including a general policy (182, 183). Schools are beneficial for health promotion because they provide access to almost all children, but they are at the same time problematic because they are complicated environments in which to perform the experimental studies required to know how effective the intervention has been (184, 185). Some evidence suggests that more vulnerable groups at higher risk for unhealthy living habits benefit more from school intervention of this kind (186, 187). In regards to students with an ID, there are only a few interventions at the school level published and these are only concerned with increased physical activity to increase fitness (188, 189). The design of school interventions have to be more “hands-on” to reach this group, and a comprehensive approach seems necessary (190).

2 AIMS

The overall aims of this thesis were to examine and follow changes in cardiometabolic health of youths with mild/moderate intellectual disabilities and to examine the effect of health-promoting activities in an upper secondary school.

2.1 SPECIFIC AIMS

- To investigate the prevalence and severity of cardiometabolic risk factors and cardiovascular fitness among adolescents with ID and compare them with adolescents without ID (Paper I).
- To examine if there is a progression in cardiometabolic risk factors among young people with and without ID after transition into adulthood (Paper II).
- To investigate whether or not a multifactorial school intervention involving increased physical activity and healthy food during school hours could reduce cardiometabolic risk factors and increase cardiovascular fitness among adolescents with mild/moderate ID (Paper III).
- To evaluate whether a multifactorial school intervention using the Plate Model results in healthy food choices with a recommended intake of $\geq 37.5\%$ vegetables (Paper IV).
- To look at the food-taking behaviour and choices in an independent meal situation (Paper IV).

3 MATERIAL AND METHODS

3.1 STUDY DESIGN AND PARTICIPANTS

Design and participating individuals in Paper I-IV are described in Figure 1. Participants were between 16 and 21 years old (Papers I, III and IV) or between 22 and 26 years old (Paper II).

3.1.1 Recruitment

The participants in Paper I (and consequently Papers II to IV) came from a convenience sample recruited from one upper secondary school for students with ID that was familiar to the author. Participants without ID were recruited from partly the main schools practical programmes and from a theoretical upper secondary school located nearby (Papers I and II). Those from practical (vocational) programmes came from handicraft or food programmes and those from theoretical from natural and social science programmes. In total 249 adolescents were asked to participate in sub-study one: ID $n = 85$; practical programmes (non-ID-p) $n = 74$; and theoretical programmes (non-ID-t) $n = 90$.

In the group with ID five participants refused to participate but 93% of the 71 eligible students participated in the study. In the non-ID-p group, several groups of students were out on work experience and not available and this group also had the highest refusal rate. Forty six percent of the students from the practical programmes participated. In the group from the theoretical programmes, the first 60 that signed up for participation were included. At follow-up five years later, 35% of the participants with an ID agreed to participate along with 33% in the non-ID group. The reasons for exclusion in the ID group were school transition, death, or participation in sub-study three. The reasons for dropout were similar for the ID and non-ID groups except for some in the ID group that did not participate because personnel at the group home did not have time to assist them in participating. Additional reasons for not participating in the follow-up were that they did not want to, did not answer the request, or did not attend scheduled meetings. In the non-ID-t group several of the dropouts wanted to participate but were abroad making participation impossible to arrange.

All but one of the available students agreed to participate in the evaluation of the school intervention in sub-study three. Participants from the intervention school in sub-study four were not included in sub-studies one, two or three. The control group in sub-study four was recruited from two similar upper secondary schools for ID students, one located in the same city and the other in a city nearby.

3.1.2 Participants' Characteristics

All individuals with ID had a mild/moderate ID. They had all been administratively defined with an ID through tests (psychological, pedagogical and sometimes medical test) before being accepted into the special programmes at the upper secondary school for intellectual disability students from which they had been

recruited. One or two participants in each group of ID (except Paper III) had a diagnosis within the autism spectrum – these were not treated specifically. None of the participants had any known obesity syndromes such as PWS.

All participants were ambulatory and no one had any major physical disabilities. They were all able to take instructions required for the measurements included in the studies. Most of the participants in Papers I, III and IV lived at home with their parents and a few lived in group homes and the majority travelled to school by themselves by public transportation. In Paper II most of the participants had moved out from their homes to independent or semi-independent living arrangements or group homes. All participants in Paper I were in Tanner puberty stage IV or V (191, 192). Participants with ID were slightly older compared to the non-ID control group because upper secondary school for students with ID is for four years but is only three years for students without ID. Participants with ID were recruited from years 1 to 4 and participants without ID from years 2 and 3.

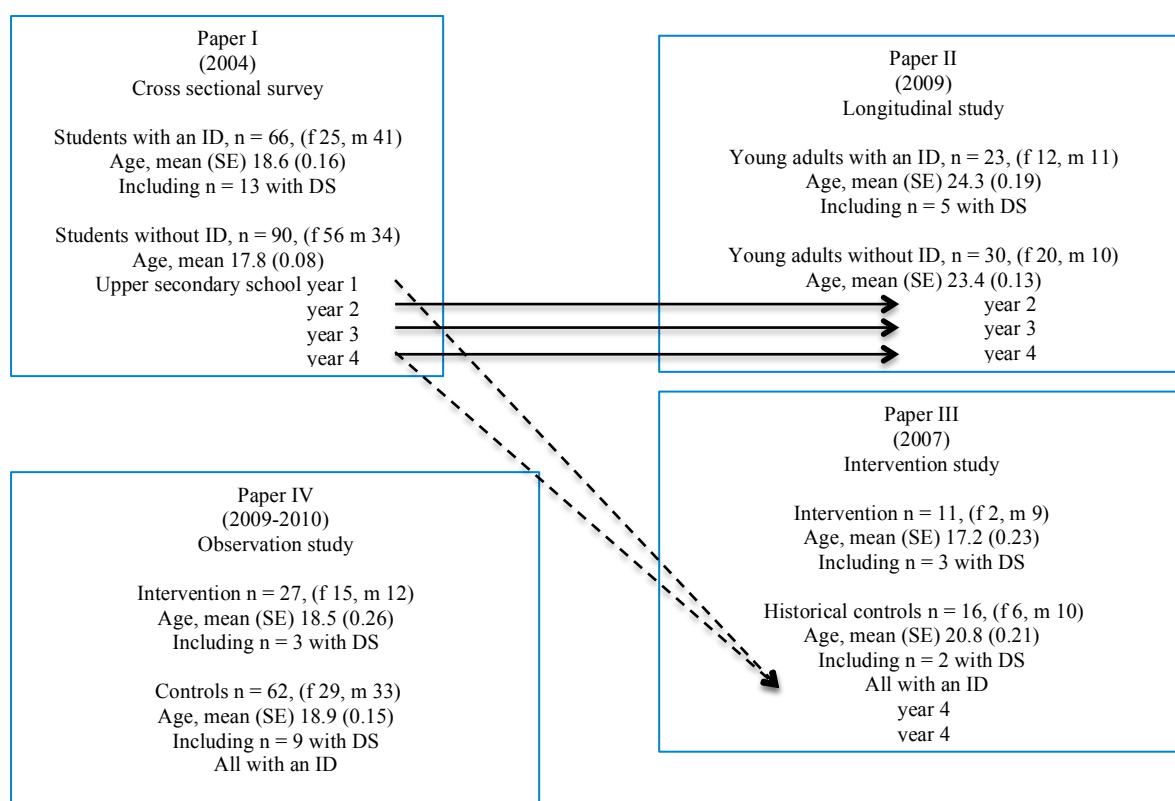


Figure 1. An overview of the design and participants included in Papers I – IV.

f = female; m = male; DS = Down syndrome; ID = intellectual disabilities.

3.2 PROCEDURES

3.2.1 The Cross Sectional Baseline Study (Paper I)

Fasting blood samples were drawn at the participant's schools by a specialist nurse in the morning after fasting from midnight the night before. Cardiovascular fitness tests were performed by personnel at the Swedish School of Sport and Health Science. All participants were familiarized with the cycle ergo meter, the use of the HR telemetric devices, and the Borg scale in advance of the test. The participants with ID received an extra familiarizing session at their school. Body composition measurements were taken by specialized personnel at the Karolinska University Hospital. Height, weight, hip, and WC were measured twice; once in conjunction with the cardiovascular fitness test and then together with blood pressure during the body composition measurement.

3.2.2 The Longitudinal Follow-up Study (Paper II)

Height, weight, WC, blood pressure, fasting blood samples after fasting from midnight, body composition and cardiovascular fitness test were all performed at Karolinska University Hospital by specialized personnel.

3.2.3 School Intervention (Paper III)

“Friskare, Smartare, Gladare”

The ‘Healthier, Smarter, Happier’ intervention used a whole-school strategy consisting of a new school policy in line with the STOPP study (187). The intervention comprised health-promoting changes in the school schedule with an increased number of physical education lessons together with education about food and changes in the available food according to the National Food Administration's recommendations and the Nordic Nutrition Recommendations of 2004 (193). The policy included all students and personnel as well as all school activities and included classes in home economics, classes in cooking, trips outside of school during school time, and coffee breaks.

A health educator was employed as a teacher, partly due to the inclusion of additional physical education but also to provide general support to health-promoting activities. The school lunch was served on specially designed ‘Plate-Model’ plates (194) that served as a pedagogical tool to help the students understand and remember how and what to choose and effectiveness of this tool was evaluated in sub-study four. The school intervention was introduced in the autumn of 2005.

3.2.4 The Special Plate (Paper IV)

The Plate Model was designed to function as a daily reminder of what should be on the plate and in what proportions. The main purpose of the plate was to increase vegetable intake. The specially designed plate is based on the idea of the Plate Model (194, 195) and replaced the regular lunch plate in the cafeteria in 2006. The model plate was 25 cm in diameter and was divided into three sections by a thin line with every section representing a food group.

The plate had a 4-cm edge that contained color pictures of different food categories (green for vegetables; brown for potatoes, rice, pasta and bread; and red for meat, fish, and beans (Figure 2). The idea behind the plate was repeatedly explained to all students by the health education teacher every new school year. The three different food groups were exemplified and the purpose of the different nutrients was described in a simply way. The school staff was encouraged to discuss the purpose of the plate during lunchtime as well as to comment on missing items when the food was taken or being eaten.



Figure 2: A special plate according the Plate Model with decor handmade from templates, manufactured at Gustavsbergs porcelain factory, in Stockholm, Sweden.

3.3 DATA COLLECTION

Data collected in Papers I through IV are presented in Table 1.

Table 1. Overview of data collected

	Paper I	Paper II	Paper III	Paper IV
Height (cm)	x	x	x	x
Weight (kg)	x	x	x	x
Hip circumference (cm)	x	x	x	
Waist circumference (cm)	x	x	x	
Blood pressure (mmHG)	x	x	x	
Fasting insulin (pmo/L)	x	x	x	
Fasting plasma glucose (mmol/L)	x	x	x	
Total cholesterol (mmo/L)	x	x	x	
HDL cholesterol (mmo/L)		x		
LDL cholesterol (mmol/L)		x		
Triglycerides (mmo/L)	x	x	x	
Hs-CRP (mg/L)		x		
Dual photon x-ray absorptiometry	x	x	x	
Cardiovascular fitness test	x	x	x	
The Meal Model picture book				x
Dietist XP				x
Template ($\geq 37.5\%$ vegetables)				x

3.3.1 Measurements

3.3.1.1 Anthropometrics

3.3.1.1.1 Weight and Height

Standing height (to the nearest cm) and body mass (to the nearest 100 g) were measured twice, and a third time in Paper II if there was more than a 2 cm or 1 kg discrepancy in the first two measurements using a SECA (Model 701 with an incorporated stadiometer; Vogel & Halke, Hamburg, Germany) (Papers I, III, and IV) or a Tanita BWB 800S (Tanita, Tokyo, Japan) (Paper II). The mean of the two measurements were used. The participants wore light clothing with empty pockets and no extra sweaters, belts or shoes. BMI was calculated as body mass in kilograms divided by height squared in meters (kg/m^2) and with special cut offs for adolescents.

3.3.1.1.2 Hip and Waist Circumference

Hip circumference in cm was assessed as the largest circumference around the hip (Papers I and II). WC in cm was measured as the horizontal circumference in the standing position between the lowest rib and the iliac crest (Papers I and III). The mean the two closest measures were used for both hip and waist measurements. The waist to hip ratio (WHR) was calculated by dividing the WC by hip circumference (Papers I and II), and the WC to height ratio (WHtR) was calculated by dividing the WC by the height (Paper I).

3.3.1.2 *Blood Pressure*

Seated auscultatory diastolic blood pressure (DBP) and systolic blood pressure (SBP) was measured in the right arm with the participant sitting after having rested for 10 minutes. In Paper I only one measure was taken. In Papers II and III two measurements were taken with a third if there was a difference > 10 mmHG in the first two. The mean of the measures were used.

3.3.1.3 *Body Composition*

Total body composition was measured with the aid of dual photon x-ray absorptiometry (DXA; Lunar Prodigy X-R model 6830, GE Lunar Corporation, Madison, WI, USA) (Papers I, II, and III). Analyses were performed using the Prodigy encore 2004 software version 880001. The total areal bone mineral density (BMD) in g/cm² and bone mineral content (BMC) in grams were measured with the values for the pelvis and spine alone. The z-score was calculated from BMD corrected for age and sex in a group of population controls. Fat mass was measured as the total percentage in the total region and in the trunk region. The ratio of percentages of android and gynoid fat (A/G) was calculated. The android fat mass was determined as the area above the iliac crest that was defined as 20% of the distance from iliac crest to the base of the skull, and the gynoid fat mass was defined as that found in an area 3.5 times the size of the android area and located 1.5 times the android area below the base of the android region. Lean body mass include everything except fat mass and BMC. Fat free mass (FFM) include everything except fat mass. FFM was estimated as: body mass minus total fat mass in kg (FFM = BM – FM). In the estimation of relative VO₂ with LBM instead of body mass in Paper I FFM was used but was incorrect written as LBM in the formula (196).

3.3.1.4 *Biochemistry*

Fasting venous blood samples were collected by a specially trained nurse for total cholesterol (mmol/L), triglycerides (mmol/L), plasma glucose (mmol/L) and fs-insulin (pmol/L) (Papers I and III). In addition HDL cholesterol (mmol/L); LDL cholesterol (LDL-C; mmol/L) and CRP sensitive (Hs-CRP) (mg/L) were collected (Paper II). The homeostasis model assessment of insulin resistance (HOMA-IR) (197) was calculated according the formula of fasting insulin x fasting glucose/22.5. Insulin in pmol/L was converted to μ unit/L by dividing it by 6.945 (198). All insulin and lipid measurements were analysed by the hospital's accredited chemistry laboratory (Clinical Chemical Laboratory at Karolinska University Hospital, Huddinge) using standard laboratory techniques.

3.3.1.5 *Cardiovascular Fitness*

Cardiovascular fitness was estimated by the Åstrand test (90) and was initially used together with the Borg rating of perceived exertion (RPE) scale (199). The Borg scale was used to assess perceived exertion in the legs and chest. The Borg scale was only used in the first data collection but because of problems for the participants regarding deciding on the level of exertion, it was never analysed, thus

was excluded from the test (Paper I). The Åstrand cardiovascular fitness test is a bicycle ergometer test using a standard speed at predefined workloads. If necessary, the workload is increased until a steady state heart rate has been established above 120 beat/min. VO_2max (L/min) was estimated from the measured HR and workload using the nomogram provided by Åstrand and Rhyming (89). Estimated VO_2max was expressed in both absolute terms (L/min) and for relative VO_2max in two different ways (mL/kg/min) and (mL/kg LBM/min). LBM is calculated by subtracting fat mass from body mass (Paper I). The participants were familiarised with the cycle ergo meter, the use of the HR telemetric devices (Accurex plus and Polar T31, Polar Oy., Tampere, Finland) and the Borg scale in advance. Additionally, participants with ID received an extra familiarisation session at their school ahead of the first data collection. All participants received verbal support during the test (85). Tests were performed by personnel at the Swedish School of Sport and Health Sciences (Paper I) and from hired research assistants from the same school (Papers II and III). The bike was calibrated in advance of each test day (Papers I, II and III).

3.3.1.5.1 Estimation of HR in Participants With ID With and Without DS

According Fernhall, maximal HR can be predicted with similar accuracy in people with and without ID, provided DS is accounted for in the equation for the group with ID (85).

3.3.1.5.1.1 The Equation

For individuals with ID: $\text{HR}_{\text{max}} = 189 - 0.59 (\text{age})$ ($R=0.30$, $\text{SEE}=13.8$, $P < 0.01$).

$\text{HR}_{\text{max}} = 210 - 0.56 (\text{age}) - 15.5 (\text{DS})$ ($R=0.57$, $\text{SEE}=11.8$, $P < 0.01$).

DS coded as 2; non-DS coded as 1.

For individuals without ID: $\text{HR}_{\text{max}} = 205 - 0.64 (\text{age})$ ($R=0.52$, $\text{SEE}=9.9$, $P < 0.01$).

The correction for maximal HR was made in two ways in this thesis, manual with intrapolating between the points in the Rhyming table (89)(Paper I) and from a linear regression (Papers II and III). The corrected maximal HR were used to adjust the prediction of maximal oxygen uptake according to Ryhming et al. (90).

The difference in estimate is dependent on the fact that many of the participants had a HR that resulted in an underestimation and gave 4-7% higher levels in Paper I.

3.3.1.6 School Intervention

The multifactorial school-based intervention was evaluated using an evaluative design (200). Pre- and post-data from the intervention group was also compared with data from a viable group, i.e. earlier students attending the same school.

3.3.1.7 Special Plate

The observed lunch meals were distributed over four occasions due to a lack of space; 14, 22, 27 and 26 participants. Thus each participant attended one occasion. The participants were video-recorded while filling their plates but not while eating.

All plates were numbered and the participants had the same number on their chest for identification of portions; faces were not recorded. The plate was photographed before and after all servings.

On the fourth occasion, all servings were weighed. On the same occasion, after finishing the meal, everyone filled out a questionnaire with five questions about their experience of the lunch and how much they felt affected by being filmed and their servings being photographed. Everyone received assistance with reading the questions if needed.

Food quantity was estimated in grams from “Matmallen” a meal model picture book (201) augmented with our own pictures and entered into the Dietist XP software package, version 3.0, 2007 (Kost & Näringsdata, Bromma, Sweden). This dietary programme is linked to the Swedish Food Database (National Food Administration, 2007, Uppsala, Sweden). Dietist XP estimations are based on age, BMI, sex and physical activity level. Visual inspections of the photographs were made to quantify the numbers of food items and food categories and number of servings. An estimate of the proportion of $\geq 37.5\%$ vegetables on the plate was made based on a transparent template.

We used an observational method with video monitoring (Canon Digital Camcorder MV900, Canon INC Oita, Kyushu, Japan) and digital photos (Minolta Dimage Z1 with 3.2 mega pixels and 10x optical zoom, Konica Minolta Canon INC, Osaka, Japan) collected during the experimental lunch buffet at the Department of Occupational Therapist Training kitchen in the hospital. A common and popular Swedish lunch meal was chosen: meatballs, boiled potatoes and a brown sauce. In addition, eight different vegetables and legumes (raw and hot) were served. All food was presented in identical bowls. Water was served with the food. An ordinary white plate with a diameter of 23 centimetres was used for the lunch. The participants were informed in advance about the video recording and its purpose. The instructions they were given was to eat whatever and as much as they wanted.

3.3.1.8 Validation of Method (Paper IV)

Two ways were used to validate the method. Films and photographs from occasion 2 were sent to two external dieticians (not familiar with each other) who independently estimated all of the servings. All portions were weighed at occasion 4 before and after servings on a calibrated digital scale (Attaché, Leifheit/Soehnle, Montréal, Switzerland) at the same time as images of the plate were taken.

3.3.1.9 Analysis of Food-taking Behaviour

Films were analysed according to following questions: “Does the participants appear to stop and think about where to start to take from the buffet?”, “Does the participant take from all bowls, only the closest bowls, or appear to think it over and make a choice?”, “Does the participant say anything that indicates that a decision has been made?” and “How does the participants place the food on the plate?”.

3.4 STATISTICAL ANALYSES AND DATA HANDLING

Statistical methods used in this thesis are presented in Table 2.

Table 2. Statistical methods used in Paper I - IV and in this thesis

	Paper I	Paper II	Paper III	Paper IV	Thesis
Descriptive statistics	x	x	x	x	x
ANOVA, Bonferroni posthoc	x				
Student's paired t-test	x	x	x		x
Cross tabulation, Pearson's chi-square test/Fisher's exact test	x	x	x	x	x
Kappa statistics for agreement between pair	x				
Pearson's correlation	x			x	x
Independent sample's t-test		x	x	x	x
Mann-Whitney U-test				x	
Intraclass correlation coefficient (ICC)				x	
ANCOVA	x	x			x
ANOVA, repeated measures				x	
Continuous metabolic risk score		x			x
Non parametric related samples test					x
Quantitative content analyses				x	

3.4.1 Data Handling

3.4.1.1 Groups Compared in the Four Papers

ID and non-ID; ID non-DS (the group with ID without individuals with DS) and DS and non-ID divided in non-ID-p and non-ID-t (Papers I and II). The intervention group was compared with a comparison group all with ID, with and without individuals with DS (Papers III and IV). Estimation of food was compared between 2, 3 and 4 different estimators (Paper IV).

3.4.1.2 Non Normally Distributed Variables

The distribution of triglycerides, f-insulin, HOMA-IR (Papers I - III), Hs-CRP and HDL-C (Paper II), body mass (in kg), protein content, potatoes in grams, lunch eaten in kcal and vegetables in grams (Paper IV) did not reveal a normal distribution and these factors were log transformed with Ln or with the square root. The result is presented in antilogged and squared values although without SD, since it is not statistically possible to antilog.

3.4.2 Statistics

3.4.2.1 Descriptive Statistics

Descriptive statistics is presented as the means, SE and their corresponding 95% confidence intervals (Paper I) and as the means and SD (Papers II-IV), together with the median and the ranges (Paper IV). Female and male participants were analysed separately to explore differences between sex and the groups ID and non-ID. The T-test for independent samples was used to test for mean differences between two groups and ANOVA was used to test between the means of three independent groups, using a post hoc pair wise comparison (Papers I and II). Paired

sample T-test was used for comparison between independent groups with regard to changes from the baseline to follow-up (Papers I and II) and between two separate weight measures (Paper I). Categorical data was presented in cross tables and comparison between proportions of individuals in various categories from ordinal data was carried out using the Paersons chi-square test for differences while kappa statistics were used for the analysis of agreement between pairs in a number of variables (Papers I-IV). Where there were too many expected cells with < 5 in the Chi-square tests, Fisher's exact test was used instead. The Paerson's correlation was used for estimation of correlations between two variables (Papers I-IV). The Mann-Whitney U test was used where an assumption of approximately normal distributed data could not be made and alternatives such as log transformation failed. A general linear model was used to estimate the within-subject effect for standard deviation SD and further to calculate measures such as measurement error (ME) and repeatability (REP) for the estimated food between 2, 3 and 4 different estimators and presented in grams and for differences in estimated and weight portions (Paper IV). Agreement was estimated using the intraclass correlation coefficient (ICC) measured for variables of estimated and actual kcal intake (Paper IV).

3.4.2.2 *ANCOVA*

Analysis of Covariance was used for analysis of continuous variables with age and sex as covariates, and the Bonferroni post hoc test was used to compare the means between the three groups (Paper I). Data was tested and presented as the difference between the mean change between groups, from modelling of data including the group as fixed and 2004 levels as a covariate in the model (Paper II). Data was also tested and presented as the difference between mean change between groups for the variables of sex; medication; smoking; origin; parents origin; parents education; working included as fixed (Paper II). Because of the small sample these variables were tested one by one. All estimates in the results of the ANCOVA analyses have been adjusted for group, sex and age and in addition with sex as a split file. All estimations are made with and without DS and the non-ID group divided into non-ID-p and non-ID-t are adjusted for group, sex and age and in addition with sex as a split file. All estimations are made with and without DS and the non-ID group divided into non-ID-p and non-ID-t.

3.4.2.3 *Continuous Metabolic Risk Score*

An aggregated risk score was computed from the variables of body fat, blood pressure, total cholesterol, f-insulin, fp-glucose, triglycerides and cardiovascular fitness in ml/kg (Paper II). Body fat was calculated as the mean of BMI, WC and truncal fat mass percentage. Blood pressure was derived as the mean of systolic and diastolic blood pressure. For each variable, a Z-score (SDS score) was derived as the number of SD units in relation to the samples mean, the mean of 2004 and 2009 values, after normalisation of the variables ($Z = (\text{value} - \text{mean})/\text{SD}$). The cardiovascular fitness variable was multiplied by -1 to reverse the order and indicate higher metabolic risk with increasing scores. This sum was then divided by 7 to compile the risk score with units of SD. This derived score only applies to this

population (202, 203). To have an eligible score, missing data was accepted for at most one sub score.

3.4.2.4 Quantitative Content Analysis

Analysis of the four films on behaviour for the selection of food at the buffet table considering choices, in terms of appearance, whether active or more random (Paper IV). Two sided tests were used in all hypothesis testing and analyses and $p < 0.05$ was regarded statistical significant. Statistical analyses were performed using the software program Statistical Package for the Social Sciences (SPSS), version 14.0 (Paper I), 16.0 (Paper II), 19 (Paper III and IV) (SPSS Inc. Chicago, Illinois, USA), today IBM SPSS, IBM Inc.

3.5 ETHICAL APPROVAL

All studies were approved by the Ethical Review Board in Stockholm (DNR 21/03; 2006/570-32; 2008/158-33/3; 2008/1875-32; 2009/772-32).

4 RESULTS

The major findings were that adolescents with ID had a more unfavorable cardiometabolic health compared to non-ID. The level of cardiometabolic risk factors increased during transition to adulthood in the group with ID and for non-ID from earlier practical secondary education. A multifactorial school intervention with a healthy school environment can contribute to healthier food choices and a deceleration of both fat mass gain and loss of cardiovascular fitness. A specific tool such as the special plate for lunch at school might facilitate healthier food choices.

The result is presented in the different measures of cardiometabolic health and the change in levels at follow-up and after intervention based on the results from the sub-studies presented in Papers I, II and III. BMI is presented from the sub-study presented in Paper IV. All analysis in this section, regardless of whether otherwise made in the publications, all data is adjusted for age and sex if not otherwise stated. Furthermore, results from Paper IV where the plate designed is influenced by the plate model, will be presented at the end.

4.1 BODY FAT MEASURES

4.1.1 DXA

The group with ID had higher levels of body fat measured as FM total % or FM trunk % compared to non-ID (Table 3). Females with ID had higher levels compared to non-ID females (Papers I-III). Females had higher levels compared to males in both groups, with one exception, FM trunk % did not differ between sexes in the ID group at follow-up, females vs males 43% vs 33%, $p = 0.064$ (Paper II). Males with ID had higher levels compared to non-ID males with one exceptions regarding FM trunk%: males with ID vs non-ID males, 25% vs 19%, $p = 0.075$ (Paper I) (Figure 3). Males with ID was the only group with a mean ratio above 1, measured as android fat mass divided by gynoid fat mass percent (Figure 3).

4.1.1.1 Non-ID Individuals From Practical Versus Theoretical Programs

Both FM total% and FM trunk %, were higher in the non-ID-p group compared to non-ID-t (28.32 (1.30) vs 23.89 (1.10), $p = 0.011$); (29.16 (SE 1.46) vs 24.05 (SE 1.24), $p = 0.009$) (Paper I).

4.1.1.2 Change Over Time

4.1.1.2.1 After Intervention

There was no increase in fat mass in the intervention group after 2 years of intervention. No additional differences between groups.

4.1.1.2.2 Change at Follow-up Without Intervention

The ID group had increased both FM total% and FM trunk% between 2004 and 2009 (Paper II) and with a larger change than in the non-ID group in FM trunk% (Table 4).

Table 3. Body fat measured in different ways in the sub-studies in Paper I, II, III and IV presented with mean (SE), adjusted for age and sex.

	Paper I and II				Paper III		Historical CG	Paper IV
	Baseline	Follow-up	Baseline	Follow-up	Before intervention	After intervention		
	ID, n = 66	ID, n = 23	Non-ID, n = 90	Non-ID, n = 30	ID, n = 11	ID, n = 11	ID, n = 16	ID, n = 89
BMI kg/m ²	25.0 (0.5)	28.2 (1.1)	22.9 (0.5)*	23.6 (1.0)*	20.5 (1.2)	20.5 (1.3)	23.9 (0.9)*	24.4 (0.5)
WC (cm)	85.1 (1.6)	91.3 (2.6)	78.0 (1.3)*	80.5 (2.4)*	72.8 (3.1)	74.0 (3.5)	80.9 (2.3) ^a	
Fat mass total %	31.0 (1.1)	35.9 (2.1)	25.8 (1.0)*	25.7 (1.8)*	27.3 (3.4)	25.2 (3.3)	30.3 (2.2) ^a	
Fat mass trunk %	31.8 (1.3)	37.9 (2.3)	26.2 (1.1)**	26.8 (2.1)**	26.4 (3.8)	24.3 (3.9)	31.1 (2.6) ^a	
Android fat (A) %		43.5 (1.8)		35.0 (1.6)**				
Gynoid fat (G) %		43.8 (2.7)		24.5 (2.5)**				
A and G ratio (A/G)		1.0 (0.04)		0.9 (0.03)**				

* Significant difference between ID and non-ID groups; between intervention and historical comparison groups.

^a Sex influences the result.

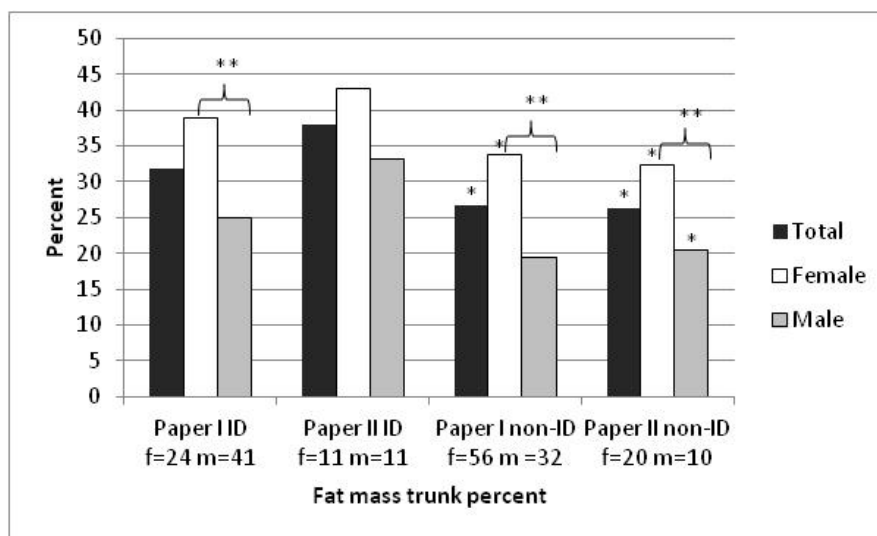


Figure 3. Fat mass trunk percent, adjusted for age and sex

* significant difference between ID and non-ID groups

** significant difference between females and males in the group

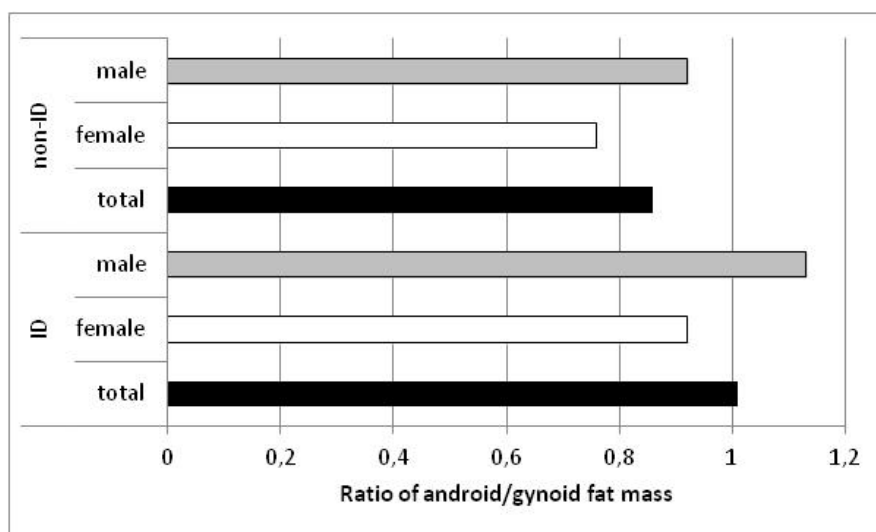


Figure 4. The ratio between android and gynoid located fat mass presented for the ID and non-ID groups in Paper II.

4.1.2 BMI

The group with ID had higher levels of body fat measured as BMI compared to non-ID (Table 3). Females with ID had higher levels compared to non-ID females (Papers I-IV). However, when excluding individuals with DS no differences between the groups were found when measured as BMI (mean (SE) 24.5 (0.59) vs 22.9 (0.46), $p = 0.135$) (Paper I). No difference between males and no differences between females and males in the two groups. Females with ID had higher BMI compared to males with ID (mean (SE) 25.8 (0.78) vs 23.1 (0.77), $p = 0.019$) (Paper IV). Level of ID was not associated with BMI. There were no differences in BMI between individuals with ID on or from individual respectively national programmes (e.g. level of ID), (Papers I, II and, IV). Between 27 – 44% in the groups with ID had developed overweight and between 12 -17% in the non-ID groups (Papers I, II and, IV) (Figure 5). Obesity was present at between 14 - 35% in the groups with ID and among 3-7% in the non-ID groups. The small group that received the school intervention showed 9% overweight and no one was obese after intervention. One participant with DS was found to be underweight in all measurements. Normal weight was most common in the non-ID group.

4.1.2.1 *Non-ID Individuals From Practical Versus Theoretical Programmes*

There was no difference between non-ID individuals from practical vs theoretical programmes (Papers I and II).

4.1.2.2 *Change Over Time*

4.1.2.2.1 After Intervention

There was no increase in fat mass in the intervention group after 2 years of intervention measured as BMI (Table 3). Mean BMI was lower in the intervention group compared to the historical comparison group after the school intervention. No additional differences between groups.

4.1.2.2.2 Change at Follow-up Without Intervention

Both groups, ID and non-ID increased body fat measured as BMI between 2004 and 2009 with no difference in size of change but with different progress between baseline and follow-up between non-ID-p and non-ID-t groups (Figure 6).

4.1.3 Waist Circumference

The group with ID had higher levels of body fat measured as WC compared to non-ID (Table 3). Females with ID had higher levels compared to non-ID females (Papers I-III). No difference between males, and no differences between females and males in the two groups (Papers I and II) (Figure 7).

4.1.3.1 *Non-ID Individuals From Practical Versus Theoretical Programmes*

There was no difference between non-ID individuals from practical vs theoretical programmes (Papers I and II).

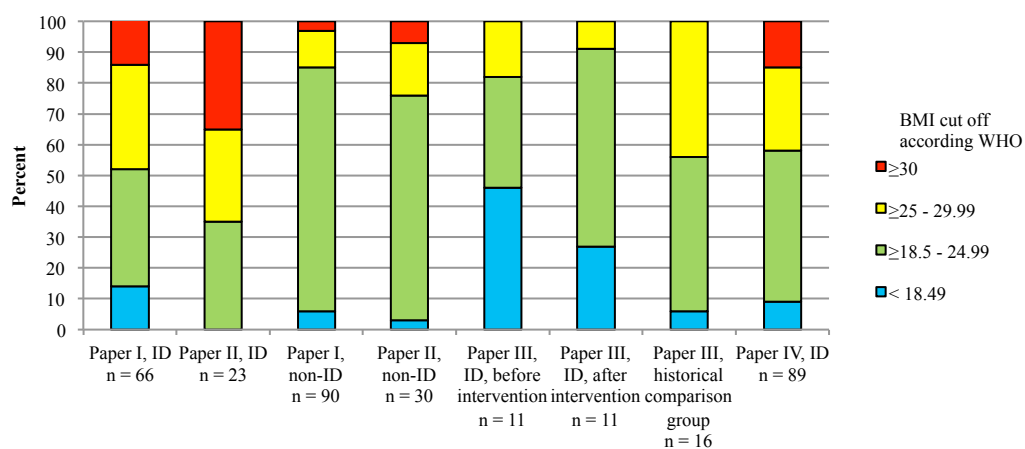


Figure 5. BMI categories for underweight, normal weight, overweight, and obesity presented for all four sub-studies.

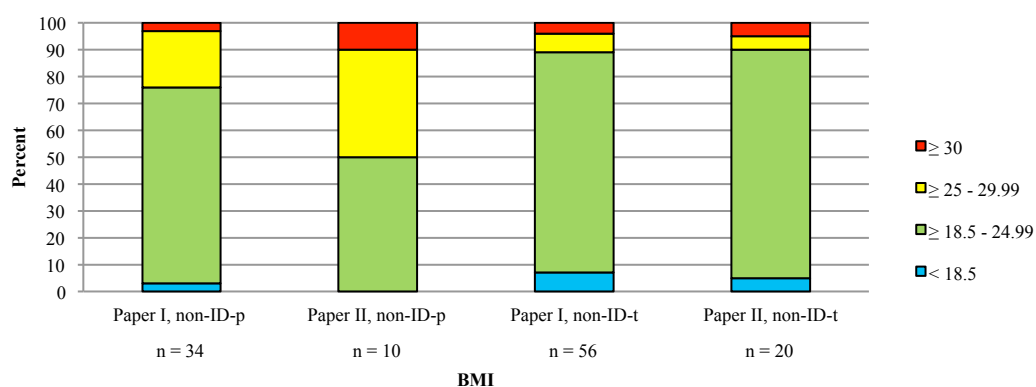


Figure 6. BMI categories for underweight, normal weight, overweight and obesity according WHO divided in students on/from practical respectively theoretical programs.

4.1.3.2 Change Over Time

4.1.3.2.1 After Intervention

There was no increase in fat mass in the intervention group after 2 years of intervention. WC was lower in the male group after intervention, when excluding DS individuals, (mean (SE) 86.22 (2.81) cm vs 75.51 (3.45) cm, $p = 0.034$). No additional differences between groups.

4.1.3.2.2 Change at Follow-up Without Intervention

Both the group with ID and the non-ID group increased WC between 2004 and 2009 with no difference in size of change (Table 4).

4.1.3.3 Waist Circumference as Part of the Metabolic Syndrome

WC as a part of the metabolic syndrome and above cut off as being a risk factor differed between groups and was present in 35% in the group with ID compared to 17% in the non-ID group (Paper I) (Figure 7). The prevalence of a WC above cut-off was at follow-up 70% in the group with ID and 20% in the non-ID group (Paper II). More females compared to males with ID had a WC above cut off (69% vs 31%, $p = 0.027$). No one in the intervention group had, after intervention, a WC above cut off.

4.1.4 Correlation Between Fat Mass Measuring Methods

Total percent FM correlated well between 2004 and 2009 ($r = 0.924$) and compared to other fat mass measurements (at follow-up); FM trunk % ($r = 0.901$); WC ($r = 0.884$); BMI ($r = 0.891$).

Table 4. Change at follow-up in cardiometabolic risk factors and cardiovascular fitness between ID and non-ID groups, presented in means (SD)

	Group with ID		Group without ID		Difference ID - without ID*	
	n = 23		n = 30			
	Mean (SD)	p = value	Mean (SD)	p = value	Mean (SD)	p = value
WC (cm)	+ 4.25 (7.31)	0.011	+ 3.12 (5.30)	0.003	+ 1.13	0.518
Fat mass % total	+ 3.55 (3.28)	0.000	+ 1.39 (5.21)	0.154	+ 2.16	0.066
Fat mass % trunk	+ 4.61 (3.66)	0.000	+ 1.85 (6.20)	0.113	+ 2.76	0.031
Lean body mass (kg)	- 0.003 (1.54)	0.992	+ 1.71 (3.04)	0.005	- 1.71	0.018
Bone mineral density (BMD)(g/cm ²)	+ 0.01 (0.03)	0.236	+ 0.02 (0.04)	0.011	- 0.01	0.264
BMD z-score	- 0.14 (0.68)	0.367	+ 0.14 (0.65)	0.238	- 0.28	0.008
Systolic blood pressure (mmHg)	- 7.21 (10.91)	0.004	- 2.00 (11.48)	0.347	- 5.21	0.338
Diastolic blood pressure (mmHg)	- 5.29(10.34)	0.022 ¹	- 3.22 (11.35)	0.131	- 2.07	0.337
Fasting glucose (mmol/L)	+ 0.55 (1.29)	0.086	+ 0.21 (0.33)	0.001	+ 0.34	0.072
HOMA-IR ²	+ 0.53 (-)	0.023	+0.19 (-)	0.178	+0.34	0.042
Total cholesterol (C)(mmol/L)	+ 0.33 (0.66)	0.051	+ 0.49 (0.53)	0.000	- 0.16	0.743
VO ₂ max (lxmin ⁻¹) ³	- 0.06 (0.51)	0.632	- 0.22 (0.54)	0.031	+ 0.16	0.307
VO ₂ max (mlxkg ⁻¹ xmin ⁻¹) ³	- 3.22 (7.89)	0.112	- 5.91 (9.51)	0.002	+ 2.69	0.033

¹When participants with Down syndrome are excluded diastolic blood pressure is no longer decreased.

²HOMA-IR = (fasting glucose x fasting insulin)/22.5

³The variable is corrected for heart rate according to Fernhall (205).

4.2 OTHER CARDIOMETABOLIC RISK FACTORS

4.2.1 Blood Pressure

Blood pressure (systolic and diastolic) did not differ between groups with no differences between females or males between the groups (Paper I and II) (Table 5). Excluding individuals with DS resulted in higher levels in diastolic blood pressure in the ID group compared to non-ID (ID 75.00 (SE 2.09) mmHg vs non-ID 69.19 (SE 1.66) mmHg, $p = 0.047$) (Paper II). In the non-ID group males had higher both systolic and diastolic blood pressure than females (SBP, mean, (SE) 121.4 (1.66) vs 112.1 (1.28), $p = 0.000$; DBP 78.4 (1.46) vs 68.9 (1.13), $p = 0.000$) (Paper I).

4.2.1.1 Non-ID Individuals From Practical Versus Theoretical Programmes

No difference between non-ID-p and non-ID-t individuals.

4.2.1.2 Change Over Time

4.2.1.2.1 After Intervention

Diastolic blood pressure was lower in the intervention group compared to the historical comparison group when excluding DS individuals (mean (SE) 67.23 (3.74) mmHg vs 76.90 (2.57) mmHg, $p = 0.049$).

4.2.1.2.2 Change at Follow-up Without Intervention

Systolic and diastolic blood pressure decreased in the group with ID between 2004 and 2009 (SBP (mean (SD) diff – 7.21 (10.91), $p = 0.004$; DBP diff – 5.29 (10.34), $p = 0.022$) with attenuation of the decrease of diastolic blood pressure when excluding individuals with DS (Table 4). No difference in change between ID and non-ID groups.

4.2.1.3 Blood Pressure as Part of the Metabolic Syndrome

Blood pressure above cut off was present in both groups and did not differ between groups (Papers I-III) (Figure 8).

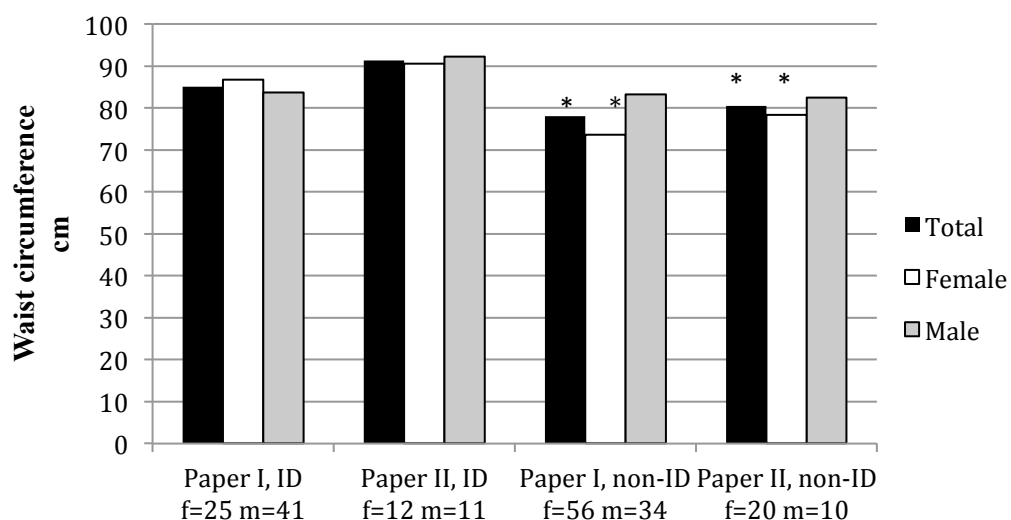


Figure 7. Waist circumference adjusted for age and sex presented in means for the total group and divided in females and males.

* significant difference between ID and non-ID groups

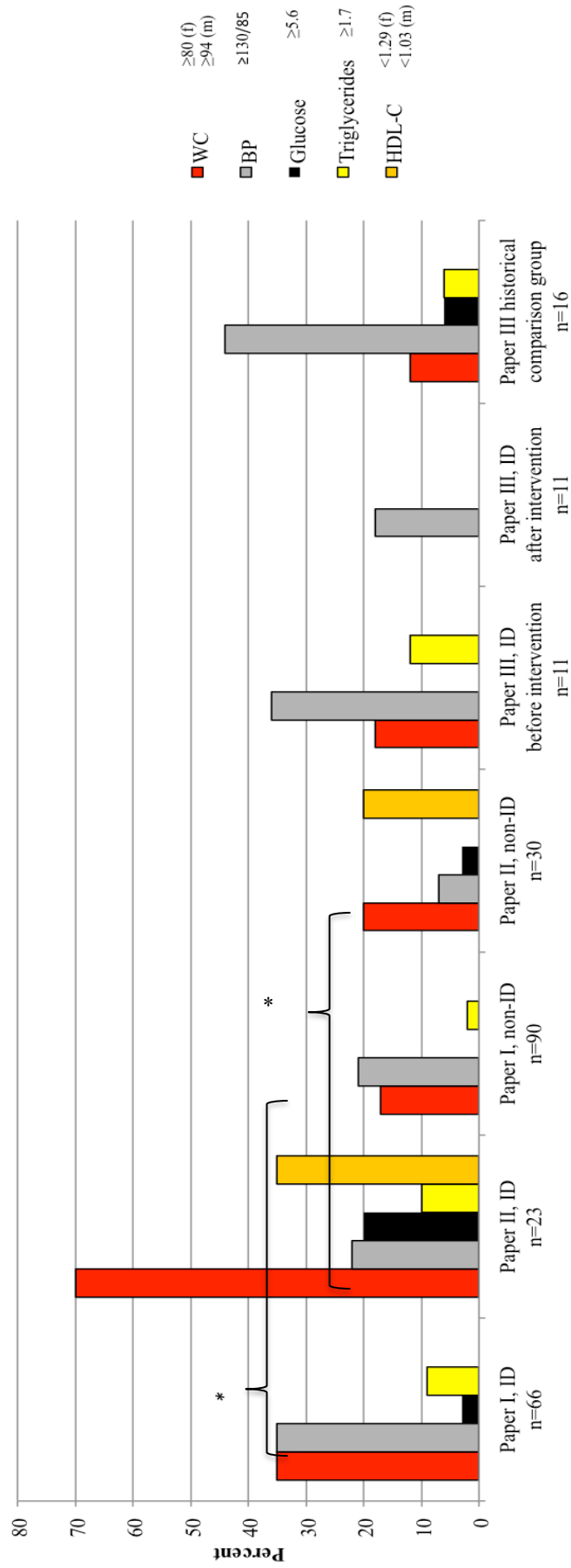


Figure 8. Percent individuals with risk factors above the cut off levels included in the Metabolic syndrome in Papers I – III. HDL-C is not measured in Paper I

* Significant difference between ID and non-ID groups

4.2.2 Glucose metabolism

4.2.2.1 HOMA-IR

HOMA-IR levels were higher in the group with ID compared to non-ID (Table 6). Females in the group with ID had higher HOMA-IR levels compared to non-ID females (Figure 9).

4.2.2.1.1 Non-ID Individuals From Practical Versus Theoretical Programmes

No difference in HOMA-IR between non-ID- p and non-ID-t groups.

4.2.2.2 Change Over Time

4.2.2.2.1 After Intervention

The intervention group had no change in HOMA-IR after intervention or compared to the historical comparison group.

4.2.2.2.2 Change at Follow-up Without Intervention

HOMA-IR levels increased in the ID group between 2004 and 2009 and the size of change was larger than in the non-ID group (Table 4). HOMA-IR above the suggested cut off by Keskin et al (at baseline for adolescents > 3.16 and at follow-up for adults > 2.5) had changed from 15% to 35 % in the group with ID and from 2% to 10% in the non-ID group (not significant) (204).

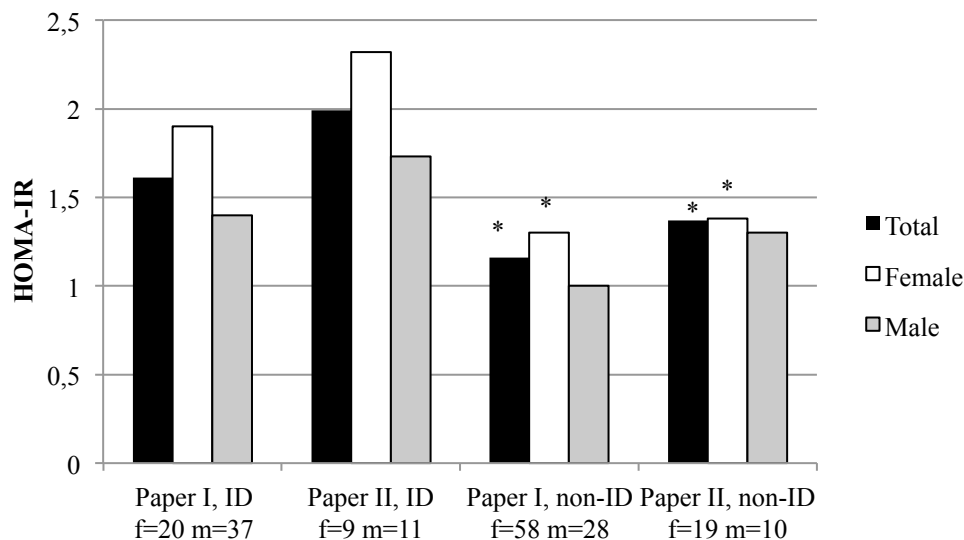


Figure 9. HOMA adjusted for age and sex presented in means for the total group and divided in females and males. Antilogged values presented.

* significant difference between ID and nonID groups.

4.2.2.3 Insulin

Insulin levels were higher in the group with ID individuals compared to the non-ID group (Paper I). Females with ID had higher insulin levels compared to non-ID females and females had higher insulin levels compared to males in both groups (Figure 10).

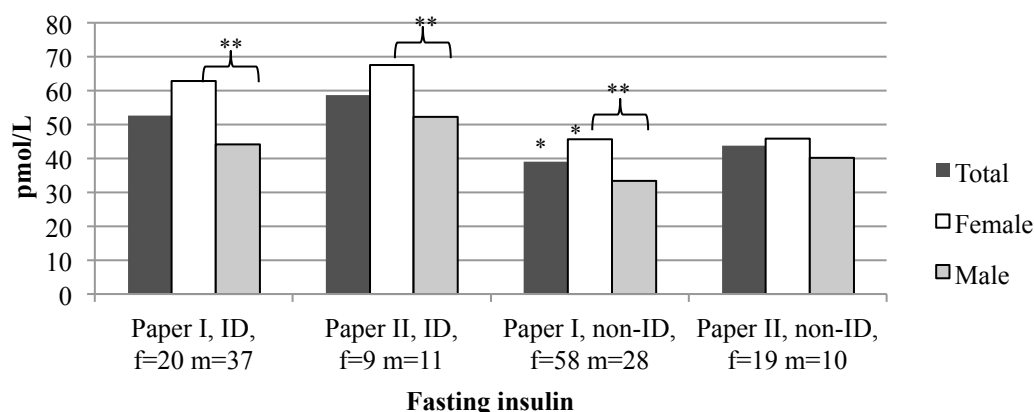


Figure 10. Insulin levels adjusted for age and sex presented in means for the total group and divided in females and males.

* sign difference between ID and non-ID group

** sign difference between sex in the group

4.2.2.4 Non-ID Individuals From Practical Versus Theoretical Programmes

No difference in insulin levels between non-ID- p and non-ID-t groups.

4.2.2.5 Change Over Time

4.2.2.5.1 After Intervention

The intervention group had no change in glucose levels after intervention or compared to the historical comparison group (Table 6).

4.2.2.5.2 Change at Follow-up Without Intervention

No changes in insulin levels in either group.

Table 5. Blood pressure, bone mass and lean body mass presented with means (SE), adjusted for age and sex in Paper I-III

	Paper I and II				Paper III		Historical CG
	Baseline	Follow-up	Baseline	Follow-up	Before	After	
	ID	ID	non-ID	non-ID	ID	ID	ID
	n=66/at DXA 65	n=23/at DXA 22	n=90/ at DXA 88	n=30	n=11	n=11	n=16
SBP (mmHg)	116 (1.4)	111 (2.4)	117 (1.2) ^a	113 (2.2)	113 (2.7) ^{a,c}	110 (4.6)	116 (3.1)
DBP (mmHg)	75 (1.2)	72 (2.0)	74 (1.0) ^a	69 (1.9) ^b	77 (2.7)	67 (3.7)	75 (2.5) ^b
LBM (kg)	43.7 (0.9)	45.0 (1.4)	48.8 (0.8) ^{a,d}	50.2 (1.3) ^a	39.5 (1.7) ^{a,c}	40.7 (2.3)	43.6 (1.6) ^a
BMD (g/cm ²)	1.16 (0.01)	1.19 (0.02)	1.22 (0.01) ^{a,c}	1.24 (0.02)	1.06 (0.03)	1.08 (0.03)	1.17 (0.02) ^b
BMD Z-score ^c	-0.02 (0.16)	0.14 (0.21)	0.57 (0.11) [*]	0.79 (0.14) [*]	-1.31 (0.41)	-0.82 (0.30)	-0.02 (0.22) ^{a,b}

CG = comparison group; SBP = systolic blood pressure; DBP = diastolic blood pressure; LBM = lean body mass; BMD = bone mass density

* significant difference between ID and non-ID group; significant difference between after intervention compared to the CG

^a sex influences the result; ^b the result changes when excluding Down syndrome individuals; ^c age influences the result; ^d interaction effect between sex and group

^cBMD z-score is analyzed with T-test

4.2.2.6 *Glucose*

No differences in fasting glucose between groups (Paper I-II). No difference in glucose levels regardless of including DS individuals or not in the analyses. Non-ID females had lower fasting glucose compared to non-ID males (mean (SE) 4.7 (0.07) mmol/L vs 5.1 (0.10) mmol/L $p = 0.004$) (Paper II).

4.2.2.7 *Non-ID Individuals From Practical Versus Theoretical Programmes*

No difference in glucose levels between non-ID- p and non-ID-t groups.

4.2.2.8 *Change Over Time*

4.2.2.8.1 After Intervention

The intervention group had no change in glucose after intervention or compared to the historical comparison group.

4.2.2.8.2 Change at Follow-up Without Intervention

There was an increase in fasting glucose in the non-ID group (Table 4).

4.2.2.9 *Fasting Glucose as Part of the Metabolic Syndrome*

Glucose as a part of the metabolic syndrome, and above cut off, was present as a risk factor was present in the group with ID (Paper I) and in both groups at follow-up (Figure 8). No differences between groups (Paper I-II). No one in the intervention group had a fasting glucose above cut off neither before or after the school intervention.

4.2.3 Correlation Between Insulin and Fat Mass

FM total percent correlated with fasting insulin in both groups at baseline. Insulin correlated highly with FM total % in the ID group ($r = 0.926$) and not at all in the non-ID group, nor with any of the fat mass measurements (Paper II) (Table 7).

4.2.4 Blood Lipids

4.2.4.1 *Triglycerides*

Triglycerides were higher in the ID group compared to the non-ID, however this was attenuated when excluding individuals with DS ($p = 0.198$) (Table 6) (Paper I). Males with ID had higher triglycerides compared to non-ID males (0.91 mmol/L vs 0.64 mmol/L, $p = 0.006$). No differences in triglyceride levels (Paper II). No differences between females and no differences between sexes in either group.

4.2.4.1.1 After Intervention

Triglyceride did not differ between the intervention group and the historical comparison group.

4.2.4.1.2 Change at Follow-up Without Any Intervention

There were no differences in change in triglyceride levels in the groups or between the ID and non-ID groups.

4.2.4.1.3 Non-ID Individuals From Practical Versus Theoretical Programmes

No differences in triglyceride levels between non-ID-p or non-ID-t groups.

4.2.4.2 Total Cholesterol, HDL-C, LDL-C

No differences in total cholesterol levels between the ID and non-ID groups (Paper I). Females with ID had higher cholesterol levels compared to males with ID (mean (SE) mmol/L 4.3 (0.18) vs 3.8 (0.13) mmol/L, $p = 0.048$ (Paper I). No differences in total cholesterol or HDL/LDL cholesterol levels (Paper II). No differences between females and no differences between sexes in either group.

Table 6. Blood lipids, insulin, glucose and Hs-CRP presented as means (SE) and adjusted for age and sex between groups in Paper I, II and III.

	Paper I and II				Paper III		
	Baseline ID, n = 57	Follow-up ID, n = 20	Baseline non-ID, n = 81	Follow-up non-ID, n = 30	Before intervention ID, n = 8	After intervention ID, n = 9	Historical CG ID, n = 16
Glucose mmol/L	4.8 (0.05)	5.4 (0.19)	4.7 (0.04) ^a	4.9 (0.16)	4.6 (0.09)	4.3 (0.26)	4.7 (0.12)
Insulin (pmol/L) ^b	52.7	58.7	39.0	43.8*	46.9	33.5	33.4
HOMA-IR ^b	1.6	2.0	1.2	1.4*	1.4 (0.22)	0.98 (0.39)	1.0 (0.19)
Triglycerides (mmol/L) ^b	0.8	0.8	0.7*	0.8	0.7	0.6	0.8
Total Cholesterol, (mmol/L)	4.0 (0.11)	4.3 (0.17)	3.9 (0.9) ^a	4.3 (0.14)	4.1 (0.30)	4.1 (0.47)	4.0 (0.23)
HDL-C (mmol/L) ^b		1.3		1.4 ^a			
LDL-C (mmol/L)		2.6 (0.17)		2.5 (0.14)			
Hs-CRP mg/L ^b		1.9		0.9 ^a			

ID = intellectual disabilities; non-ID = without ID; CG = comparison group;

* significant difference between ID and non-ID group; difference between intervention and historical comparison group;

^asex influences the result; ^bthe variable was log-transformed for the statistical analysis. Antilogged values are presented.

Table 7. Pearson correlation (r) presented for pair wise correlation between body mass index, waist circumference, percentage body fat and fasting insulin

	Without intellectual disability			With intellectual disability		
	WC	n = 30 FM total %	f-insulin ^a	WC	n = 23 FM total %	f-insulin ^a
BMI	0.926*	0.590*	0.182	0.908*	0.725*	0.739*
WC (cm)		0.392*	0.154		0.667*	0.780*
FM total %			0.204			0.926*

*correlation is significant

^a the variable was log-transformed for the statistical analyses

Table 8. Cardiovascular fitness presented as estimates of aerobic capacity both absolute and relative from body mass and from fat free mass adjusted for sex in participants with ID, with DS and without ID. In addition heart rate and work rate at steady state. Data is presented in means (SE).

	Paper I ID without DS n = 51	Paper II ID without DS n = 14	Paper I IDDS n = 9	Paper II IDDS n = 3	Paper I non-ID n = 88	Paper II non-ID n = 30	Before intervention ID including DS n = 9	After ID including DS n = 9	Historical CG ID including DS n = 14
Heart rate (bpm)	137 (2.0)	141 (3.8)	125 (5.6)*	126 (8.3)	141 (1.4)* ^{ab}	145 (2.7)	131 (5.0)	133 (8.6)	134 (4.8)
Work rate (watt)	75 (3.5)	88 (5.2)	58 (8.0) ^a	80 (11.2)	101 (2.4)* ^a	114 (3.1)* ^a	61 (10.1)	70 (13.0)	79 (7.3) ^a
VO ² (l/min)	2.2 (0.09)	2.3 (0.11)	2.1 (0.26)	2.1 (0.23)	3.0 (0.07)* ^a	2.8 (0.10)* ^a	1.8 (0.19)	2.0 (0.30)	2.3 (0.17) ^a
VO ² (ml/kg/min)	33 (1.5)	31 (1.8)	33 (4.2)	31 (3.9)	44 (1.1)* ^a	39 (1.3)*	32 (4.1)	32 (4.9)	36 (2.7) ^a
FFM ^c (ml/kg/min) BF-FM	47 (1.8)	47 (1.8)	47 (5.0)	40 (3.7)	58 (1.3)*	53 (1.5)* ^a	49 (3.9)	46 (5.2)	50 (2.9)

ID = intellectual disability; DS = Down Syndrome; non-ID = without ID; CG = comparison group; IDDS = ID with DS.

*significant difference between ID and non-ID groups; between ID with and without DS.

^asex influences the result; ^binteraction effect; ^cFFM = body fat minus fat mass

4.2.4.3 *Change Over Time*

4.2.4.3.1 *After Intervention*

Total cholesterol did not differ between the intervention group and the historical comparison group.

4.2.4.3.2 *Change at Follow-up Without Any Intervention*

The group with non-ID increased their total cholesterol, no difference between ID and non-ID group (Table 4).

4.2.4.4 *Non-ID Individuals From Practical Versus Theoretical Programmes*

No differences in any of the measured blood lipids between non-ID-p or non-ID-t groups.

4.2.4.5 *Triglycerides and HDL as Part of the Metabolic Syndrome*

Triglyceride levels above cut off were present in both groups; ID and non-ID (Paper I), and only in the group with ID at follow-up (Paper II). HDL cholesterol levels under cut off were present in both groups, ID and non-ID (Figure 8) (Paper II). No differences between groups in either paper.

4.2.5 *Inflammatory Marker Hs-CRP*

No differences between groups, ID and non-ID, with or without individuals with DS (Table 6). Males in the group with ID had higher Hs-CRP levels compared to males in the non-ID group (mean 2.54 mg/L vs 0.44 mg/L, $p = 0.011$) (Paper II).

4.2.5.1 *Non-ID Individuals From Practical Versus Theoretical Programmes*

Hs-CRP was higher in the non-ID-p group compared to non-ID-t (mean 1.93 mg/L vs 0.51 mg/L, $p = 0.012$) (Paper II).

4.3 **BONE MASS AND LEAN BODY MASS**

The group with ID had lower LBM and z-score BMD (Paper I and II) and lower BMD g/cm² (Paper I) compared to the non-ID group with or without individuals with DS (Table 5). Males with an ID had lower LBM, BMD and z-score BMD compared to non-ID males with no difference between females (Paper I). Z-score BMD was lower in the group with ID (Figure 11) (Paper II). Males in both groups had more LBM compared to females (Paper I and II) (Figure 12) and more bone mass presented both as BMD and z-score BMD compared to females (Paper I). No differences between males in the ID and non-ID group in BMD when adjusting for LBM (Paper II).

4.3.1.1 *Non-ID Individuals From Practical Versus Theoretical Programmes*

No differences between non-ID-p and non-ID-t in LBM or BMD (Paper I and II). The non-ID-p group had lower z-score BMD compared to the non-ID-t group (mean (SE) 0.38 (0.26) vs 0.99 (0.15), $p = 0.041$) (Paper II).

4.3.1.2 Change Over Time

4.3.1.2.1 After Intervention

No difference in LBM between intervention and the historical comparison group. The historical comparison group had higher BMD and Z-score BMD compared to intervention group but this was no longer present when excluding individuals with DS and adjusting for sex ($p = 0.051$) (Table 5).

4.3.1.2.2 Change at Follow-up Without Any Intervention

The non-ID group increased LBM, BMD and BMD z-score levels, and level of change in LBM was larger compared to the ID group (Table 4).

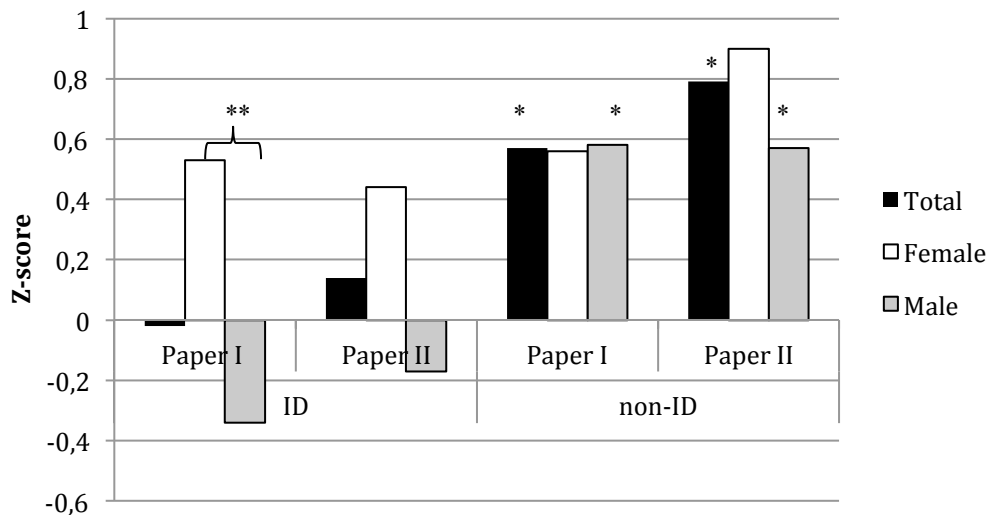


Figure 11. The z-score of bone mass density presented in means for the total group and divided in females and males.

* Significant differences between the ID and non-ID groups.

** Significant difference between sex.

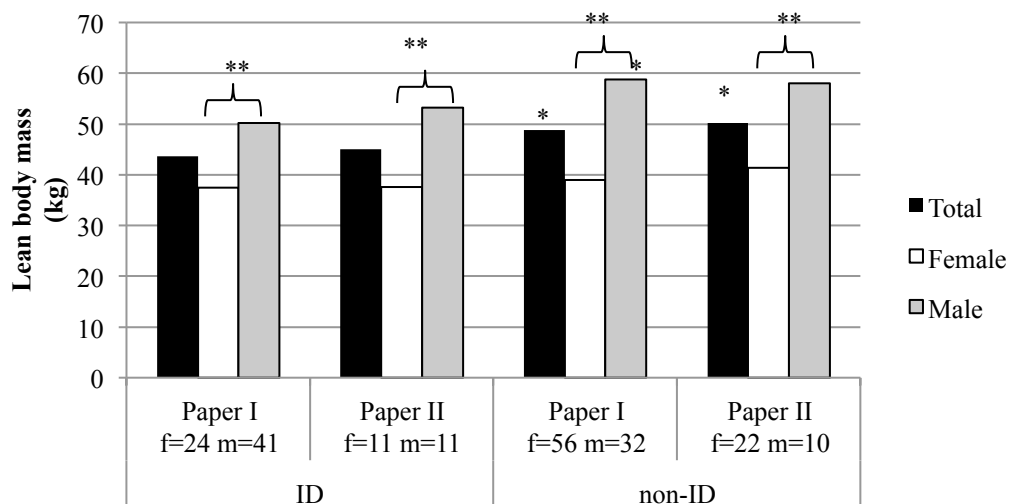


Figure 12. Lean body mass adjusted for age and sex presented in means for the total group and divided in females and males.

* Significant difference between ID and non-ID groups.

** Significant difference between sex.

4.4 CARDIOVASCULAR FITNESS

4.4.1 Absolute and Relative VO₂

Cardiovascular fitness was lower in the ID group compared to the non-ID group both estimated as absolute in litre per minute or in relation to bodyweight (Table 8) (Paper I and II). Participants with DS exhibited levels equal to the group with ID when corrected according Fernhall (205). Both females and males with ID had lower cardiovascular fitness compared to females and males without ID with one exception, between females (Paper II) in absolute VO₂ (Figure 13). Males had higher levels compared to females in both groups (Paper I). No differences between sexes in the group with ID (Paper II) and only in absolute VO₂ in the non-ID group (mean (SE) males 3.1 (0.16) L/min vs females 2.5 (0.12) L/min, $p = 0.012$). Changing body mass to body mass without fat mass in the calculation of fitness e. g. fat-free mass, did not change the difference in fitness (Table 8).

4.4.1.1 Non-ID Individuals From Practical Versus Theoretical Programmes

The non-ID-p group had both lower absolute and relative VO₂ compared to non-ID-t (mean (SE) 2.8 (0.11) L/min vs 3.1 (0.09) L/min, $p = 0.022$ and 40.4 (1.69) ml/kg/min vs 45.6 (1.34) ml/kg/min, $p = 0.017$ (Paper I). Sex influenced the result.

4.4.1.2 Change Over Time

4.4.1.2.1 After Intervention

No differences between intervention and the historical comparison group (Table 8).

4.4.1.2.2 Change at Follow-up Without Any Intervention

The non-ID group had a decrease in fitness both measured as absolute and relative VO₂ and with a larger change compared to the group with ID in relative fitness (Table 4).

4.4.2 Heart and Work Rate in the Bicycle Test

Heart rate level and work rate at steady state during the bicycle test differed between the groups with lower levels in the group with ID compared to non-ID. The group with DS had lower levels compared to ID non-DS with the exception of heart rate that did not differ at follow-up (Paper II) (Table 8).

4.5 METABOLIC SYNDROME

No differences between groups in individuals that had developed the metabolic syndrome (Paper I). The distribution of risk factors in the different groups is presented in Figure 8. Percentage of individuals that had developed at least one metabolic risk factor above cut off included in the metabolic syndrome was higher in the group with ID (Paper I and II).

4.5.1.1 Change Over Time

4.5.1.1.1 After Intervention

No one in the intervention group had developed the metabolic syndrome and no one in the historical comparison group.

4.5.1.1.2 Change at Follow-up Without Any Intervention

There was no difference between ID and non-ID groups in decrease versus increase of any risk factor above cut off (Figure 14) (Paper II). The metabolic syndrome was developed with at least three risk factors above cut off in 22% of the group with ID compared to no one in the non-ID group ($p = 0.012$) (Paper II). No difference with or without DS or between females and males. Excluding HDL-C, 13% had developed the metabolic syndrome in the group with ID.

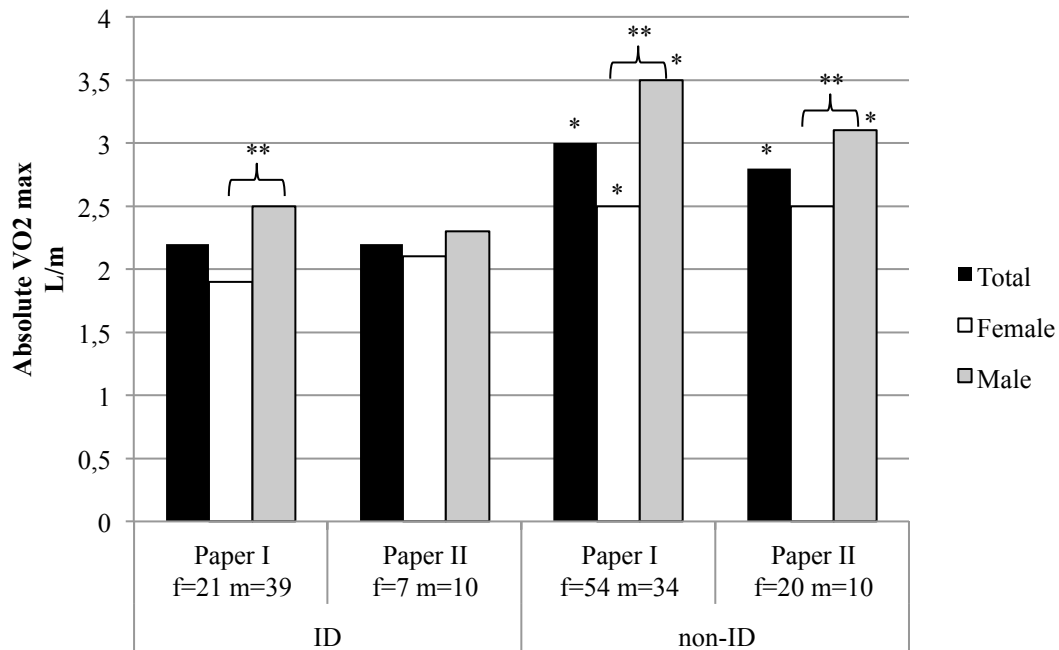


Figure 13. Cardiovascular fitness in litre per minute, adjusted for sex, corrected for heart rate and presented in means for the total group and divided in females and males.

* Significant difference between ID and non-ID group

** Significant difference between sex

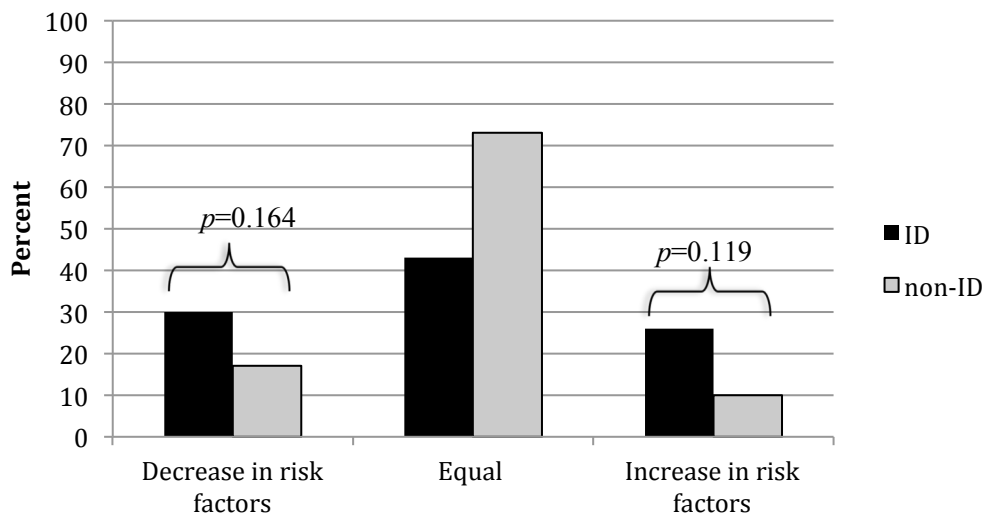


Figure 14. Individuals with a change in risk factors included in the metabolic syndrome between baseline and follow-up, presented in percent.

ID = intellectual disabilities; non-ID without intellectual disabilities.

4.6 AGGREGATED RISK SCORE ON CONTINUOUS VARIABLES

The aggregated risk score was higher in the group with ID compared to the non-ID-t group (Paper I and II) (Figure 15 a+b). The risk score increased between 2004 and 2009 in the ID group (0.27 to 0.59, $p = 0.027$) and the non-ID-p group (-0.57 to 0.08, $p = 0.000$). No risk score was calculated for the intervention group due to the sample being too small.

4.7 DOWN SYNDROME

DS differed compared to non-DS ID with lower systolic blood pressure 108.4 (3.60) mmHG vs 118.2 (1.68) mmHG, $p = 0.016$, lower bone mass density in pelvis 1.11 (0.03) g/cm² vs 1.19 (0.02) g/cm², $p = 0.047$ and being shorter 153.9 (2.36) cm vs 167.5 (1.10) cm, $p = 0.000$ (Paper I). Male individuals with DS had lower BMD z-score compared to males both in the group with and without ID (Paper I). In addition individuals with DS had lower heart- and work-rate in the cardiovascular fitness test (Table 8). Lower SBP and shorter height and with the addition of lower DBP, 61.8 (4.13) mmHG vs 75.6 (2.15) mmHG, $p = 0.008$ at follow-up (Paper II). No other differences. Height (Paper III and IV) and systolic blood pressure (Paper III) differed with lower levels in the DS group. Correlation between BMI and fasting insulin was exceptionally low in the group with DS ($r = 0.27$) (Paper I). Excluding DS from the ID group affected the result in fat mass total %, waist circumference, Hs-CRP and triglyceride levels (Paper I-III).

4.8 ASSOCIATIONS WITH SOCIOECONOMIC STATUS

Socioeconomic status was only measured in the sub-study presented in Paper II. There were no differences between the groups with the exception of a higher proportion in the ID group who had a mother of a non-Swedish origin (Paper II). Higher levels of fat mass trunk percentage, HOMA-IR and a lower BMD z-score were associated with being unemployed or on sick leave. To be on medication was associated with lower BMD z-score. Associations were found between higher levels of total body fat mass percentage and unemployment/ being on sick leave ($p = 0.011$); earlier attendance in a practical programme ($p = 0.032$); having a father without higher education background ($p = 0.040$). The increase in fasting insulin from 2004 to 2009 was associated with being on sick leave or unemployment ($p = 0.010$). Being born outside Europe was associated with a decrease in estimated oxygen uptake in litre per minute ($p = 0.024$).

4.9 EVALUATION OF THE PLATE (PAPER IV)

A total of 89 adolescents with ID participated in the experimental evaluation of the plate, 27 had eaten from the plate at school lunch for at least six months and 62 came from two other schools unfamiliar to the special plate. The majority (88%) of the total group filled their plate with $\geq 37.5\%$ vegetables and mostly with three or four different kinds of vegetables, no differences between groups. Nor did the intake of vegetables differ between groups. The total group had a mean intake of 558 kcal for lunch, which is less than what is recommended as normal, however more male participants (42%) took and ate more for lunch than assess as normal compared to females (20%, $p = 0.027$).

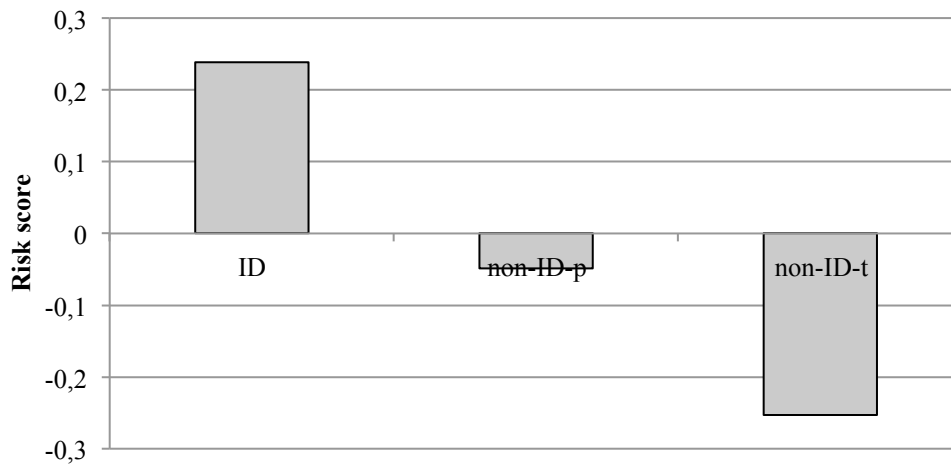


Figure 15 a. An aggregated risk score in sub-study I, comparing the groups ID, non-ID-p and non-ID-t.

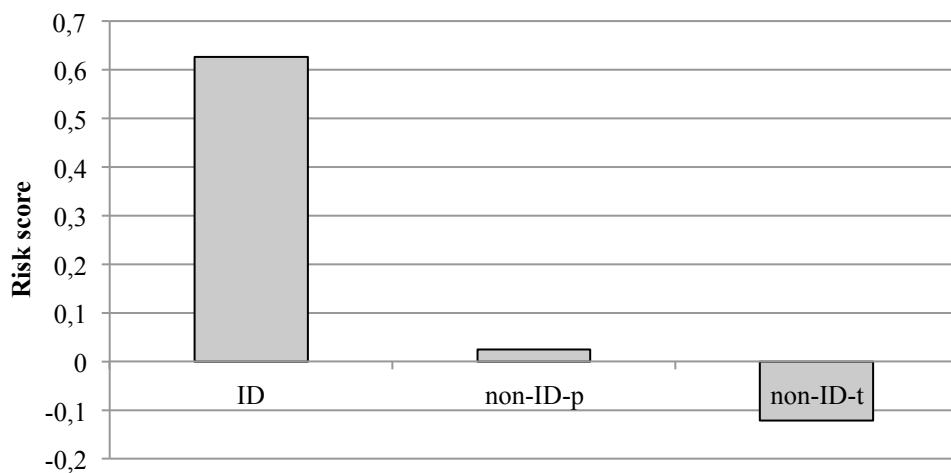


Figure 15 b. An aggregated risk score in sub-study II, comparing the groups ID, non-ID-p and non-ID-t.

The intervention and control group differed in distribution of macronutrients at these lunches. The intervention group had a lower fat intake (mean 21(6) % vs 24 (7) %, $p = 0.031$) and a higher carbohydrate intake (57 (7) % vs 53 (8)%, $p = 0.035$) compared to the control group. More individuals in the intervention group were satisfied with one portion (56% vs 32%, $p = 0.039$). Measured in grams, the intervention group had fewer leftovers and no leftovers with vegetables compared to the control group (5 (10) grams vs 25 (43) grams, $p = 0.021$).

4.9.1 Food-taking Behaviour and Choices

Everyone appeared to choose consciously before starting to take food, beginning at different bowls and adjusting the amount from the serving spoon before putting it on their plate. Ninety-two per cent (of the total group) took from all three food-groups and no one took from all the bowls. More individuals who took more than one portion had plate wastes (88%, $p = 0.010$) compared to those without plate

wastes. More of those who took more than one portion had eaten the recommended amount for lunch or more compared to those who took one portion (61% vs 52%, $p = 0.000$).

4.9.2 Results From Validation of the Method

Reliability of the measurement method was assessed in three different ways: between estimated and weighed portion; between assessment from two external dieticians; between external dieticians' assessments and the researcher's assessment. Validity was assessed in one way; the questionnaire with questions concerning how the participants' liked being part of this experimental situation. Measured mean difference between estimated and weighed portion was done for all portions at one of four occasions. The level of agreement was estimated to be ICC = 0.98. The mean difference was 66 grams (SD 80) with a variance of 144 grams in measurement error (ME) and with repeatability (REP) of 204 grams. Comparison with the assessment from another occasion between two external dieticians with earlier experience of estimation of portions showed the level of estimated agreement to be ICC = 0.98. The mean difference was 0.36 grams (SD 35) with a variance of 49 grams in ME and a REP of 69 grams. The level of agreement comparing our own assessment with that of the external dieticians was estimated to be ICC = 0.99. The mean difference was -3.9 grams (SD 24) with a variance in ME of 34 grams and REP of 48 grams.

4.9.2.1 *Participants' Experience*

The participants' experiences from the experimental situation were evaluated on one occasion using a questionnaire. Fifty-four per cent wrote positive comments, 23% felt affected and in total 35% reported on at least one question that they had been affected by the situation.

5 DISCUSSION

5.1 MAIN FINDINGS

This thesis presents the longitudinal alteration of cardiometabolic risk factors and cardiovascular fitness among adolescents with a mild/moderate ID during transition to adulthood. The overall aim was to examine and follow change in cardiometabolic health and to examine the effects of health promoting activities.

The main findings were that individuals with a mild/moderate ID, in adolescents with a marked progress during five years into young adulthood, had a higher prevalence and severity of cardiometabolic risk factors together with low cardiovascular fitness compared to individuals of the same age without ID. At follow-up as young adults, 35% were classified as obese, 20 % showed impaired fasting glycaemia and 22% had developed the metabolic syndrome. The health promoting activities, i.e. actions at school level with daily physical activity, healthy food and use of a special lunch plate showed promising results. Single risk factors included in the metabolic syndrome were only present in 11% in this group after the school intervention with no one having an elevated waist circumference or being obese.

5.2 PREVALENCE OF CARDIOMETABOLIC RISK FACTORS

5.2.1 Overweight and Obesity

This group with ID already had a mean BMI of 25 as adolescents with 34% being overweight and 15% being obese, a noticeably higher prevalence compared to the group without ID. Why are these adolescent overweight to this extent and are they representative of Swedish adolescents with an ID? The sub-sample in paper IV featuring adolescents with ID, involved in the special plate evaluation, was the same age but came from three different schools and had a comparable mean BMI of 24.4. Only one Swedish comparable study was found, just recently published. They report a lower mean BMI, 23.6 (SD 4.9) (206). One of their exclusion criteria was reduced strength in the lower extremities and in addition the participants were recruited from an area with a national upper secondary sport school for students with an ID (206) and these differences might explain the lower BMI. I wanted to compare the level of obesity and cardiometabolic risk factors as well as the discrepancy with the group without ID found in this work with international data. This turned out to be difficult.

Individuals recruited from school appeared to be most similar since population based data is rare (131). Six studies reporting overweight and obesity levels in adolescents with an ID recruited from schools (124-127, 211, 212). There were four from Europe and two from Asia and with contradictory results however with several reasonable explanations for this. Age ranges were different, there was a large proportion of individuals with autism in one study, students with physical disabilities were included, syndromes such as PWS were included and some of the

schools appeared to be institutional-like. Two of the school studies used complementary methods to measure fat mass, one measured WC (211) and one both WC and body fat with bio-impedance (127). Ells found no difference between the prevalence of obesity measured as BMI or WC however Salaun et al did and report the discrepancy in measured body fat with BMI compared to WC or with bio-impedance with lower prevalence of obesity measured with BMI. Thus, none of these studies were really comparable.

BMI is a rough measure of body composition as it does not separate fat from muscle mass and bone mass. BMI levels among people with ID might underestimate the prevalence of adiposity. With lower levels of muscle and bone mass, especially in male individuals, that I report in this sample and suggested by others, (207) result in higher fat percentage for the same BMI.

In some comparisons with the group without ID in this thesis, sub-studies of BMI levels have not differed between groups. At the same time, if measured as WC or fat mass from the DXA measures, level of adiposity has been higher in the group with ID. Thus BMI is not optimal as measure of adiposity (127, 207). Measuring waist circumference and DXA should probably be preferred.

Is there a systematic difference between the sexes in the group with ID in prevalence of overweight and obesity? Being female and having an ID appears to increase the risk for being obese as adolescence. Females with an ID were more obese both compared to males with an ID and compared to females without an ID, consistent with other studies on adolescents (206) but primarily on adults (109-111, 115, 208). However this difference between the sexes looked different at follow-up. Among those who were obese, it was the opposite or equal, females (33%) and males (36%) showed no significant statistical difference. This is more in accordance with the general older population with male individuals being more obese (37). Males with an ID had higher levels of adiposity measured with DXA compared to males without ID as young adults and had narrowed the adiposity levels compared to females with an ID so that the natural difference between sexes in adiposity was gone.

5.2.1.1 Differences in DS

Several studies have suggested the population with DS is more overweight/obese compared to people with other causes of ID (123, 153, 155, 159). This work does not report that any differences between ID with or without DS in any of the body fat measures were prevalent. We saw no significant differences in BMI, and on the contrary, at follow-up, when measured as a fat mass percent total or trunk located, with lower levels in individuals with DS. Probably the DS individuals' shorter height and maybe different body fat placement disturbs the accuracy of BMI levels. However DS have a specific growth chart although this was not used on those included in this work (209). Individuals with DS have been investigated much more prevalently than the group with ID at large. In connection with the creation of the Swedish growth chart for DS a decade ago, 33% of those who were 18 years old

were reported with a BMI > 25 which is a much lower prevalence compared to the 49% in this work. However, those with DS were very few and the 49% is for the total group with an ID (209). However DS has, in several studies, been reported as being associated with high fat mass levels, more frequent in females (154, 159) but also with an earlier peak in increasing fat mass and with a plateau reached in the thirties (155, 156). I think the small sample of individuals with DS makes it impossible to draw any firm conclusions regarding DS specifically in this thesis.

5.2.1.2 The group without ID

The control group without ID had a mean BMI 22.9 with 15% being overweight of whom 3% were obese consistent with the control group in Blomqvist et al (206) as well as reports on the general Swedish population with levels of between 3 -7% in adolescents (210, 211). In less advantaged environments the figures look different and there is also a difference between rural and urban areas (212-214). However both this thesis and Blomqvist et al recruited the age matched control groups from the same area.

The group without ID studying on practical programmes had a higher prevalence of being overweight compared to those on theoretical programmes measured as fat mass with DXA, but not measurable by comparing mean BMI or WC. However having a BMI ≥ 25 was present in 24% in the group non-ID-p and 11% in the group non-ID-t. The effects of social determinants on weight and health is today well known although it is not fully understood what the pathways are (99, 215). This difference between non-ID-p and non-ID-t appears to be step wise with the group with ID in the bottom with 49% having a BMI ≥ 25 .

Females had higher levels of fat mass compared to males consistent with natural differences between the sexes. In our sample without ID, female participants were somewhat more overweight compared to males, however there was not at a significant level and no difference is reported by others (210).

5.2.1.3 Underweight

The adult population with ID has been reported with higher proportions of people being underweight (110, 111, 208). In sub-study one we found 14% were underweight compared to 6% in the group without ID which is in agreement with the observations in adults.

5.2.2 Other Cardiometabolic Risk Factors

Fifteen percent in the group with ID had a degree of insulin resistance as adolescents measured as HOMA-IR which had increased to 35% at follow-up. The group without ID had an increase as well but at a much lower level from 2% to 10%. These findings contribute to the assumption that the high levels of obesity found is affecting the glucose metabolism, maybe already damaging the endothelial cells with increase of the intima media and increasing the risk of future cardiovascular disease as well as type 2 diabetes (48, 216, 217). However others

report central obesity itself as predicting intima media thickening (218). The correlation found between fat mass and insulin support how these variables are associated in these individuals. At this time, no one had a fasting glucose level regarded as pre-diabetes as adolescents supported by Lin et al who report impaired fasting glucose in 0.3% of this age group in a large sample with ID (124).

The value of measuring HOMA-IR instead of only insulin is subject to debate but one big concern is the diverse methods used (219-221). However levels of HOMA-IR in the non-ID group, with normal BMI levels according age, were consistent with earlier reports measured in the same way (222, 223). The suggestion is to yield valuable data when the variable has been logaritmed, as we did, and use this on a healthy population, in accordance with how it was carried out in this work (224). The result suggests a difference between the sexes described by others with females having higher levels, both measured as insulin and HOMA-IR (219). However there were no sex differences in HOMA-IR above the cut off.

Blood pressure mainly did not differ between the ID and non-ID groups but the group with IDs level had a trend with higher levels for all measures. Lin et al report 12% with hypertension and elevated triglycerides higher compared to the general population in the same age group (124). However it is difficult to know if these groups are comparable such as those from data being taken from medical records.

Levels of most measured cardiometabolic risk factors in this work in the group without ID are in agreement with earlier Swedish studies on adolescents with the group with ID being deviant with mostly more unhealthy levels (210, 211). This suggests the control group is representative for comparison.

5.2.2.1 Differences in DS

Individuals with DS were shorter and had lower blood pressure consistent with earlier reports compared to the rest of the group with ID (153, 158, 162). One additional difference was that fat mass percent did not at all correlate with insulin levels in contrast to both the ID and non-ID group. The reason for this might be something concerning the DS group specific atherogenic profile, but this was a small group and needs to be investigated further. In a recent study on variables associated with intima media thickness in individuals with and without DS insulin was an associating variable in the group without DS and in DS instead there were high triglycerides and high Hs-CRP (141).

5.2.3 Bone Mass and Lean Body Mass

Males with an ID had both lower bone mass and lean body mass even when excluding DS (160, 163). Unfortunately I have not controlled for other diagnosis associated with low levels such as Klinefelter's syndrome in this work (225). However, an increased prevalence of osteoporosis has been reported in a pair of studies carried out previously both in females and males with ID, both with and without including DS individuals (226, 227). Body size, pubertal stage, skeletal

maturation as well as ethnicity, gender and age but also genetic, hormonal and environmental factors affect bone mass levels leaving a lot of possible explanations for this result (228). The group with an ID was slightly older compared to the non-ID group so male individuals with ID would have had slightly higher levels compared to non-ID. Only 90% of peak bone mass is expected to be reached at the age of 18 thus would be regarded as under growth in a wider sense > 20 years of age (228). The low level of cardiovascular fitness and high levels of obesity in the group with ID might be the primary reason. However, as these participants are not controlled for medication nor diagnosed it is hard to interpret data (229). Other suggestions for low bone mass in individuals with ID have been insufficient intake of vitamin D (230). This work included a questionnaire with questions concerning food habits from the beginning, however not at a level which made it possible to estimate vitamin D levels. This questionnaire was never analysed due to difficulties with the validity of the answers a recurrent problem in research on this target group (231).

Males in both groups had higher levels of lean body mass and bone mass consistent with natural differences between sexes. There is a strong relationship between lean body mass and bone mass content (62). Lean body mass peak about two years earlier than bone mass content and this happens at the age of 15 to 17 in boys and for girls earlier (62). Muscle mass and bone mass usually continue to increase in males during the transition from adolescence to adulthood (62). There was a very low change in the group with ID from their already low levels compared to the non-ID group and compared to normal development.

5.2.3.1 Differences in DS

It is known that individuals with DS have lower levels and one hypothesis for this has been growth hormone deficiency that includes short stature and low levels of lean body mass. This was recently studied in 10 adult individuals with DS but they showed normal GH secretion compared to an age-matched control group (153).

5.3 CHANGE OVER TIME

5.3.1 Overweight and Obesity

The prevalence of obesity had more than doubled over five years for this thesis group with ID which makes you wonder where it will end. This prevalence of obesity is higher than what is reported for the general population in the US today, which I think is a comparison that clarifies the level presented (37). The US is one of few countries that have some kind of population data including people with an ID and thus some data on changes in weight over the decades (109, 113, 131). In the US National Health Interview Survey between 1985 and 2000, households were interviewed once a year including all family members who were not institutionalised. They report higher levels of obesity in people with an ID compared to the general population and that this difference increases with time. In 1985, the difference in the prevalence of obesity was eight percentage points (ID 19% vs non-ID 11%) and in 2000 14% (ID 35% vs non-ID 21%). This supports the

theory that the environment is important (113). In this thesis, the difference at follow-up was 28% (ID 35% vs non-ID 7%). Swedish data has reported stabilising trends in obesity in the general population but with the exception perhaps of the total population and this I think has to be considered when trying to understand this result (212-214).

Will obesity levels increase in our Swedish population with an ID in the same way as in the US? There is, to my knowledge, no published study on obesity levels for a similar older adult group as the one studied in this thesis and because of the lack of Swedish studies, baseline data from an ongoing study (Bergström et al, unpublished data with great thanks to the authors) is worth mentioning. This is a similar but older sample to mine, recruited from the same county, thus an urban area, including individuals with mild/moderate ID ($n = 130$), 57% females, 84% born in Sweden, individuals with DS included and ambulatory but with 15% having a minor physical disability. Their sample had a mean age of 37.8 (SD 10.8) range 20-66 and a mean BMI of 29.2 (SD 7.1) with females 30.7 (SE 0.90) being heavier compared to males 27.4 (SE 0.79). They report 43% with obesity and 27% as being overweight. However, this sample does not include those with a mild ID who after school, survive on their own without support from such as group housing or service housing and who might be the largest group at risk (208). This level of independency in several studies has been associated with obesity and those excluded in Bergströms et al's sample are probably the most independent in the group with mild/moderate ID (110, 111). However, this indicates an increase by age in our population with ID suggested by others and the increase or decrease over the decades is impossible to know (129, 232, 233). There is an ongoing discussion as to whether the obesogenetic societies we live in today, with so many opportunities for food intake and sedentary leisure time, have affected those susceptible. That the increase in obesity is levelling off results from the fact that those in the populations who are predisposed to being overweight have become overweight and the remaining people are resistant to excessive adiposity (213). One may wonder if the population with ID has reached this level yet, which to some extent must be dependent on the inclusion process in municipalities. Beyond a high prevalence of obesity, measurements of waist circumference and abdominal fat mass percent have increased, causing a further risk of impact on cardiometabolic health.

5.3.1.1 Difference Between Sexes

Almost twice as many females with ID were obese at the baseline compared to males, however five years later, more male individuals were obese (36% vs 33%) and if looking at abdominal adiposity levels measured by DXA male individuals had increased the percentage of fat mass trunk from 25 to 33 percent and reduced the natural difference between males and females to non-significant. When measuring the ratio of android and gynoid fat mass, where males by nature more commonly have a more android body composition, males with an ID now had a ratio > 1 thus more of the unhealthy located android fat mass associated with

increased insulin resistance and an elevated cardiovascular disease risk (234). This pattern of obesity relating to age and sex needs to be studied further.

5.3.1.2 The Group Without an ID

The level of increase in BMI in the total non-ID group is consistent with another recently published study on the same age group in the European Youth Heart Study (235). However, the increase in adiposity was only in the non-ID-p group when separating the group without ID. Half of the individuals from previous practical high school education were overweight or obese, despite the fact that, in the follow-up study, participants from the non-ID-p group who agreed to participate were healthier according to most measured variables compared to those not participating. This development is consistent with the public health authorities' latest reports on social determinants effect on cardiovascular health (99, 106) and in a recent report measurable among children (107).

5.3.2 Other Cardiometabolic Risk Factors

The increase in fat mass together with the increased insulin resistance in the ID group with these two variables correlating strongly, suggests an elevated risk for future cardiovascular disease and type 2 diabetes (79, 217). There was no correlation between fat mass and insulin levels in the non-ID group and with a much weaker correlation at the baseline in both groups. This might be explained by the non-ID groups higher cardiovascular fitness levels as the cardiovascular fitness is negatively associated with HDL-C (236) and HDL-C has a large impact on the glucose metabolism. However there were no differences in mean HDL-C levels between groups and physical activity level was not analysed. When looking at HDL-C using the metabolic syndromes' cut off, 35% in the group with ID had a low HDL-C compared to 20% in the non-ID group.

Autocorier (2009) found a strong correlation in obese adolescents between A/G ratio and insulin resistance measured with HOMA-IR but I did not(234). Instead A/G ratio was positively correlated with Hs-CRP levels ($r = 0.559$, $p = 0.013$) and negatively with HDL-C ($r = -0.514$, $p = 0.024$) in the ID group, not in non-ID.

It was the two groups that increased their abdominal adiposity most during transition to adulthood, males in the ID group and the non-ID-p group, which had highest levels of Hs-CRP. Unfortunately this was not measured at the baseline thus impossible to evaluate any change.

Generally, there were levels in the non-ID group for cardiometabolic risk factors in accordance with Swedish population data on adults (237).

5.3.3 Bone Mass and Lean Body Mass

The lower bone and lean mass levels at the baseline in the group with ID had not improved at follow-up. One reason for this could be the high levels of obesity. Bone mass is dependent on hormones associated with fat mass for its development with

adiponectin negatively associated with fat mass and leptin and insulin positively associated (67).

A question that should be asked is if there is a higher prevalence of osteoporosis in this population in Sweden? This work's presented data with a high prevalence of obesity, low levels of lean and bone mass and low levels of cardiovascular fitness reveals the danger of premature osteoporosis becomes tangible. Recently low levels of volumetric bone mass have shown an association with obesity at the same time as a positive association with bone size and this was adjusted for lean body mass (67).

Certain medication is associated with lower bone mass as mentioned earlier. This was asked for at follow-up and being on medication showed an association with lower bone mass measured as a BMD z-score and both medications that were included in the medication variable are known to be associated with affected bone mass (227). In addition certain diagnoses, not only DS, are associated with abnormal levels such as thyroid hormone, growth hormone and testosterone which in turn might affect bone mass (153, 225). Also the low levels of cardiovascular fitness together with 35% having a low HDL-C are both variables associated with low bone mass (238). All this together raises the need to look for a possible increase in premature osteoporosis in people with an ID.

5.4 CLUSTERING OF THE RISK FACTORS

It appears that females with an ID are obese and with an increasing insulin resistance while males with an ID are obese with a more android located fat mass, together with higher triglycerides and Hs-CRP levels. In addition males with an ID have low levels of bone and lean body mass. Both females and male with an ID have very low cardiovascular fitness. This most certainly positions them at high risk as risk factors obtained during adolescence are suggested to predict subclinical atherosclerosis in adulthood (217, 239).

5.4.1 Metabolic Syndrome

Twenty-two percent, had developed the metabolic syndrome in the group with an ID and this is comparable to prevalence reported in middle age Swedes (240, 241). Despite that 69% of the females with an ID had a WC above the cut off compared with 31% of males, all but one of those who developed the metabolic syndrome were males. This agrees well with the fact that males with an ID had a high A/G ratio and the suggestion that this increases the risk for cardiometabolic disease but is somewhat surprising with the much higher prevalence of an increased waist among females. The fact that more male individuals developed the metabolic syndrome reflects reports from the general population (240, 241). Levels of the different risk factors were consistent except that more people in these older groups had developed high blood pressure and high triglyceride levels (240).

In older studies, published two decades ago, prevalence of cardiometabolic risk factors and the metabolic syndrome among older adults with ID do not report any differences compared to the general population (118, 242). One explanation could be the more protected living circumstances for adults with ID living at institutions. This is in agreement with reports from individuals with ID who today are institutionalised (243). Draheim et. al presented levels of cardiometabolic risk factors in adult groups with ID living more independently and they showed higher levels of cardiometabolic risk factors compared to the general population, consistent with the finding in this thesis (114, 158). A recent study on the aging (> 50) Dutch population with ID reported obesity in 26% which is higher compared to the general population (117)

5.4.2 Continuous Aggregated Cardiometabolic Risk Score

Another way to look at the clustering of different variables predicting cardiometabolic disease is to look at the different variables on their continuous level, make a z-score and compare each individual's score against its group as previously suggested (202). When the result is presented in this way, both the group with ID and the non-ID group from practical education had increased their risk scores between the baseline and follow-up. The step wise pattern between the group with ID, the non-ID from practical high school educations and the non-ID from theoretical educations could indicate that the difference reflect an association with social determinants and not only the fact that one group has an ID. The risk score for 2004 respectively 2009 suggests that the group from practical education had decreased their risk score but this is just a result of that those with less risk score were the ones participating in the follow-up study.

5.4.3 Cardiovascular Fitness

This thesis reports poor cardiovascular fitness in both female and male individuals with ID regardless if measured with or without fat mass or in absolute or relative terms. This is consistent with several studies on this target group in similar age (85, 244-246) but there are contrasting results (247).

There was no difference between females and males with ID at follow-up, thus the natural higher fitness level in the male group was not there. This is in agreement with the high prevalence of the metabolic syndrome in males with ID. The group with an ID with a mean of 30 ml/kg/min in females and males 31.2 ml/kg/min indicates that individuals with ID have a very low fitness (90). Compared to a healthy sample from the general population (mean age 42) estimated for cardiovascular fitness with the same test had levels for females of 34 ml/kg/min and males 35.2 ml/kg/min (248). Again, these young individuals with ID are comparable to levels in the middle-age Swedish general population.

There are studies presenting data on interventions that has increased CVF in adolescents with an ID however most have not succeeded and the reasons for these are probably plenty (249). There are studies reporting on several factors that need to be considered for a successful intervention (186, 250-254). Specific effort must

perhaps be made in terms of motivation (254). One way to do this could maybe be to measure heart rate during training which could fulfil two purposes; one that the training is with sufficient intensity the other that it could maybe be used as a “hands-on” tool to explain the level of effort the training requires. During the fitness testes, we noticed that several of the participants were unfamiliar with the level of exertion that the sub-maximal test required.

It is discussed whether there are physiological differences in people with an ID that hinder them reaching cardiovascular fitness levels comparable to the general population. Van de Vliet with colleagues examined the physical fitness profile of high-performance athletes with an ID and compared to population data but also to age-matched physical education students. They found similar or lower levels in several physical fitness measures but then it was cardio ascular fitness and muscle strength the group with ID had lower levels (255).

The association between level of ID and motor coordination has been discussed but with contradictory results (244, 256). Adolescents with ID have been found that in general score lower compared to age matched controls in balance and muscle performance tests (206). Maybe there are difficulties for children with ID to be physical active without adapted support understanding their specific difficulties.

Individuals from former practical education had lower cardiovascular fitness compared to those from theoretical programmes. Similar results are reported 15 years ago (257) and it is also reported that students on practical programmes are less physical active compared to those on theoretical programmes (258).

The strong association between physical activity and cardiovascular fitness to cardiovascular health, maybe independent each other, and of obesity, speaks in favour of more physical activity options for persons with ID (79, 259-261).

5.5 WHY THIS HIGH ADIPOSITY PREVALENCE?

What can the cause for the increased adiposity among ID individuals be? All individuals in this thesis represent otherwise healthy adolescents with an ID as recruited from schools and not from a clinical setting which maybe could have resulted in a skewed sample such as presented by others (208, 209). Reasons for the difference in adiposity levels between adolescents with and without ID probably originate from many different sources. This thesis presents very low levels of cardiovascular fitness together with low levels of muscle mass in the group with ID which probably is one contributing factor for this difference. Several studies have reported adults with ID being sedentary and having unhealthy eating habits (115, 262-265). However, there might also be a difference between ID and non-ID groups in heredity. The large heredity factor of being obese of up to 70% with two obese parents might be different distributed between groups (43, 46). Children with an ID might have been more exposed to unfavourable pre and post natal conditions (40, 266) or because of their ID be at increased risk to epigenetic effects from unhealthy

living habits, perhaps at particularly bad time points (40, 267). Difference in prevalence of obesity in children with an ID compared to children in general has been reported from as early as three years olds (129). Children and adolescents with an ID may live under more unfortunately socioeconomic circumstances suggested by some (268-270). Another cause could be that the lower IQ level results in specific difficulties in today's society (271).

The disability ID itself, with cognitive problems such as waiting for things, planning to do something later in time or perhaps central capacity, to be able to motivate oneself and keep motivated might be one additional explanation (186, 254). Most genetic abnormalities associated with obesity are found in genes expressed in the CNS. Thus, the underlying disease that causes ID may also increase the risk to become obese. I think all these factors contribute to the difference found. However, and most important to stress, is that our intervention which is discussed in detail below shows that these individuals with ID, with a minor support from society can markedly reduce their cardiometabolic risk factors. This indicates that the differences between individuals with and without ID primarily are due to the fact that adolescents with ID are more vulnerable in an obesogenic society.

5.6 SCHOOL INTERVENTION

Changes solely in the school environment might contribute to a reduction of cardiometabolic risk factors and preserve cardiovascular fitness in adolescents with ID, giving schools an important opportunity. Those students that finished school after two years of intervention had a mean BMI of 20.5, no one was obese and with a small increase in bone and muscle mass however this was not at a significant level. Only 11% had any cardiometabolic risk factor when finishing upper secondary school compared to 64% in the comparison group.

What in the "whole of school" intervention that might have contributed to the promising results is uncertain. Maybe the increased scheduled physical activity that enabled students to achieve recommended levels of physical activity as primary suggested by others (188, 189). Getting the physical activity at school might be even more important for adolescents with ID according a recent Swedish report, reporting that adolescents with ID in high school were less physical active during leisure time compared to adolescents in general (272). However, the increased physical activity needs to be thought about. Students with an ID have been reported being less physical active during physical education at school compared to peers from several reasons such as loosing 50% of the time waiting , unqualified teachers, unwritten policies hindering physical activity (273, 274). The intervention school had well suited teachers. The increased scheduled physical activity in the intervention school was applied by the complementary health educator. Regarding the low levels of muscle and bone mass reported in this thesis together with reports on increased risk for fractures reported for people with ID, schools probably have a great opportunity to do good (75, 227).

We could not measure any increase in cardiovascular fitness and this may be explained by too low intensity in the exercise. The intervention only added hours with physical activity without guidelines governing the intensity of the physical activity. However we don't know if the physical activity increased. It is a well-known fact that physical activity is difficult to improve, both in the Bunkeflo project and STOPP (187), both projects focused on children without ID and failed to do so despite scheduled physical activity. However other benefits could be seen as increased motor skills and school performance (275) but were not measured in thesis.

Other possible beneficial factors could be the removal of unhealthy choices as sodas, candy, ice cream and bakeries as in the STOPP project (187). In addition the "hands-on" education with the special plate discussed below, the school trips with practical experience in the right context or all peers and personnel serving as role models during all school hours probably contribute as important factors. The context is suggested to be important for people with ID (276) as well as the interventions being comprehensive (277). Parents of children with an ID who are overweight are suggested to be less aware of themselves being an important role model for their child compared to other parents (278). The present school intervention was applied to all, personnel and students, making adults role models in that specific context such as with "no cookie to the coffee" for the personnel either. For many adolescents with an ID, school is the place where they meet friends and important others, and they usually spend the whole day at school through all of their school years with the bylaw rights to after school care before and after the school day (5). This means a lot of hours influenced by the living habits available at their school. In addition school interventions have been suggested to be more beneficial for more disadvantaged children such as this target (179, 187).

Maybe the intervention effect came from the increase of fruit and vegetables at school (183, 187). Individuals with an ID often appear to feel comfort within familiar habits and changing habits when already established is difficult (279) thus school years are important shaping habits for adult life.

5.6.1 The Special Plate

The special plate aimed to clarify proportions, support the memory and increase food awareness beyond the increase the vegetable intake. The result suggests that a greater awareness was achieved concerning food and food intake. The intervention group had fewer leftovers and more were satisfied with one serving. They also choose food lower in fat and with more carbohydrates which might result from a greater awareness but this need to be studied further.

The overriding result from the observational occasions was the rich vegetable intake among most of the participants (88%) regardless of being the intervention or control group. A possible reason for this positive observation is the situation and environment that were present. Earlier research has suggested that adolescents most

often choose food either according to taste and convenience or based on situational limitations and for individuals with less preference for vegetables more abilities might be needed (280, 281). The occasion was designed to be different from their usual school lunch pattern for both intervention and control participants, with all bowls being the same size and everything placed in an unusual order. More than half of the bowls were with vegetables which is an unusual large proportion which might have increased the ability to choose vegetables (280). To increase the availabilities is one way to facilitate healthier choices at school (281).

Another reason for the rich vegetable intake could be the place in the serving line the different food groups had. One hypothesis could be that because the meat balls were first in line and if they were the most desirable maybe there was time to consider other food groups after having conquered them. Thus, this is the opposite to what usually, but not scientifically based, is suggested, i.e., that vegetables should be served first. This might be what contributed to both the participants' healthier choices at the evaluation of the plate and the intervention groups stalled weight gain. The special plate was discussed repeatedly during the school years, both at the beginning of every new semester according to the intervention but there were also indications of spontaneous occasion as both personnel and students ate daily on the plate. It is likely that many students knew what a healthy choice was but needed support to make that healthy choice (281). The school intervention included guidelines recommending one serving for lunch and if more food was wanted, vegetables were suggested. This might be the reason for those choosing one portion. We found that none of those satisfied with one serving exceeded the recommended amount for lunch, although some of these servings were overfilled. This supports the one portion rule as suggested by others (282).

The food taking behaviour was unambiguously perceived as the students being much aware of their choices during the observed lunch, both the intervention and control groups. No one took from both bowls with the same content. Many of the participants talked loudly as for remembering rules given to them earlier as "eight meatballs – I take eight meatballs" and several started at different places passing the first bowl but coming back to it later. Earlier research has shown the possibility to change food habits at school by introducing new food as well as increasing information around the food (283, 284).

Sweden together with Finland is one of few countries that have a history of municipally funded hot school lunch at all schools (281). The special plate in the lunch canteen has probably increased the students awareness of that there are different food groups and that it is healthy to include them all on the plate. Every school meal is an educational opportunity and with the large advantage to reach all children it should not be missed from a health promotion perspective. Frequently repeated health education is probably more important for adolescents with ID than for non-ID and more simple educational tools such as the "plate-model" plate should be developed and tested.

5.7 PUBLIC HEALTH AND SCHOOL

This group of young people needs a higher awareness from the society. The group with ID does fulfill many of the criteria's to accommodate to the part of the population with a low socioeconomic position. They have a low level of education, material hardship as adults are scarce depending on the comparatively low income from the social insurance system and usually they do have few opportunities to advance on the labor market. All this qualify this group to belong to the high risk group for cardiovascular diseases that are associated with social determinants (106). Two fields that public health rises are the low vegetable intake and low levels of physical activity. The results in this thesis indicate that these are areas that can be altered in the school environment. The cause of the high cardiovascular disease risk is most probably that an obesogenic environment is more difficult to handle for individuals with an ID as discussed before.

Regarding the elevated risk for premature cardiovascular disease in the population with ID that this work suggest raises the question whether or not the health care system is aware. A recent report from Taiwan report an association with the appearance of nurses and supporting health environments with healthier levels in cardiometabolic risk factors in their institutions (285). In the UK the specialised disability nurse has been kept through community integration of people with ID and this is a likely reason for many published studies on health and health care (233, 286). In Europe except the UK the Netherlands is one of few countries that looked at cardiometabolic health for the population with ID (116, 287, 288) and the reason for that is probably them being the only country in the world with a professorship in medicine for the intellectual disabled.

5.8 DISCUSSION OF METHOD

5.8.1 Recruitment of Participants With ID

Research on people with ID is made on a broad area of themes and between 1000 and 2000 articles have been published each year during the last three decades; however only a small percentage of these are on physical health, physical activity or health promotion (289). Recruitment of participants with ID to research is challenging as research per se is based on voluntarily participation. Many studies have small samples. Trustees, personnel and parents sometimes act as gate keepers to protect individuals from participating in research or consider it too time consuming (290). There are no established best practices today but a personal recruitment strategy similar to what was carried out for the studies in this thesis work is suggested to yield best results, but this needs to develop (291, 292).

Recruitment to the base line study was easy as it was a convenience sample and I was familiar to the participants. A control group to the school intervention turned out to be impossible to achieve maybe due to gate keepers. Several participants with ID were lost to the follow-up study because of time constraints of personnel.

5.8.2 Measurements

The decrease in blood pressure reported at follow-up together with the deterioration in other measures makes the measurement passable. The procedure was equal at the different measures. The device is checked on with no faulty found. It is a manual device and all measures have been made by the presenting author. One explanation could be the “white coat syndrome” but not likely. Another explanation could be that they were all in the final of upper secondary school years.

DXA measures were without controlling for the participants bladders being empty, nor for physical activity at the day of measuring and or for when last eating. This might have affected the result. However this concerns both the ID and the non-ID group.

Anthropometric measures as well as blood pressure were only measured once at base line. It is preferable to measure several times which was carried out in Paper II and III using the mean of the two closest. This in particularly when measuring WC (293).

Biochemistry did not include HDL-C and Hs-CRP at the baseline, resulting in no possibility to measure change.

For the measure of cardiovascular fitness the sub-maximal Åstrand test was chosen because it is a test with a minimum of performance and thus could minimise differences between groups. This despite other submaximal tests are validated for and used on people with ID such as six minutes walk test, shuttle run test and treadmill test. Two recent studies have shown a positive association between IQ and performance in physical fitness tests (87, 294). This strengthens our wish to choose a test with a minimum of performance. Thus we used a submax bicycle test. This test is the same to use for a person who is professional in biking as one who does not usually bike. Another reason for choosing that test was the expectation that participants with ID might be more overweight compared to participants without ID. The bicycle test reduces that effect since the participants do not need to carry their weight. We used a bicycle that was speed independent which made it a lot more simple for the participants to keep up pedalling which been mentioned as a problem with tests on bicycle (84).

We used the equation for estimating maximum heart rate suggested by Fernhall (205) and used by others (189). The participants in this work differed in mean steady state heart rate between groups consistent with those reported by Fernhall. This difference was not significant at follow-up between ID without DS and non-ID but probably depending on the smaller group. According Fernhall, in particular at group level, this equation is valid to use.

Generally the bike test worked out very well. Most participants easily pedalled up to steady state level in heart rate and went on pedalling until the test was finished. There were however some disadvantages. The group with ID had more often a heart rate that was beyond or above the levels for heart rate in the table, 120 -170 which is the range used for estimation of VO_2 max in the Åstrand test. This concerned primarily participants with an ID of whom most had too low a heart rate. Data could not be included for a few individuals, most of them with DS (all with ID). The reason was fatigue or because of levels in heart rate and/or watt impossible to combine in the nomogram.

At the first cardiovascular fitness test the Borg scale was used for perceived exertion (RPE) in the legs and chest. However, it was too complicated for the participants with ID to concentrate and decide what level to choose so it was removed. It was probably too abstract and the participants preferably choose very high or very low levels or as it seemed randomly.

Physical activity level was measured with an accelerometer in sub-study II; however this data has not yet been analysed and is not included in this thesis work. In sub-study I a questionnaire was included with questions concerning food habits, leisure time activities, friends and physical activity habits. Because of doubts over the group with ID's understanding of the questions the results were withdrawn from this study.

5.8.3 School Intervention

The school intervention was implemented according to the plan, however more than one year delayed given that there were very few to evaluate the effect on. In addition the proposed control group retired resulting in a different design. It was unfortunate that the increased physical activity was not increased with a certain intensity level. This could be the reason for no measurable increase in cardiovascular fitness. The one hour a week in the gym that was included is not measured in muscle strength this would have been good to do.

The observational situation used to measure content on the plate and behaviour was conjunct with several problems. In the design we had a control group with participants without ID but we failed with the recruitment. This resulted in no opportunity to control for a different behaviour in a group without ID. We should have measured the intervention group once before introducing the special plate. We included the questionnaire concerning the participants' experience being observed at the last occasion which would have been good to use on all occasions. We needed to validate our estimations from photos and films and at the last occasion we added weighing the plates with and without food at the same time as the photo was taken. The instructions that we gave before the lunch to the participants were "eat whatever and as much as you want" which might have increased their intake. Instead we should have said "eat as you usually do" (295). This might have affected

their food intake. Replicated occasions would have perhaps given another result (282) and given the opportunity to place the food bowls in different order to control for that side effect as well as measure if the behaviour differed between occasions.

The video-recording was totally essential to be able to estimate food content. A large amount of the portions were overfilled and food was placed on top. The analysis of food taking behaviour was valuable. The consciousness and for some, a loud conversation with themselves, before making a food choice, was only possible to detect from the film. We did not use a tripod for the camera which would have been better to gain an exact size of all plates. The template used to estimate the visual amount of vegetables on the plate (> 37.5%) worked well. When estimating the food content we used the Meal model book “Matmallen” (201) together with own pictures. This was time consuming but with an acceptable accuracy according to the validation both between dieticians and weighed portions.

5.9 LIMITATIONS AND STRENGTHS

This thesis work is on a small sample and might have given type-2 error and because of that lack to prove results. The follow-up study included only 34% of the total sample making it difficult to analyse and present sexes and the DS group separately. The same applies to the small sample in paper III. The school intervention was delayed and finally only one school year was possible to evaluate on giving a particularly small sample. The design could have been different. A randomised controlled trial design including several upper secondary schools and randomise some to intervention and some not. Or a similar control school without any intervention to compare against. Baseline data on the students at the intervention school before the plate was introduced and the observation study replicated. This would have strengthened the evidence of the results. In spite of these deficiencies the intervention has contributed to valuable insights. The strength of this thesis is that it has followed cardiometabolic health over five important years from adolescence to young adulthood in a group with ID together with a comparison group. It is also that data collection was made with objective methods.

5. CONCLUSIONS AND CLINICAL IMPLICATIONS

- The prevalence of cardiometabolic risk factors and cardiovascular fitness was high in adolescents with mild/moderate ID.
- Five years later the cardiometabolic risk factors had increased together with continuing low levels in cardiovascular fitness in young adults with ID.
- The metabolic syndrome was developed in more than one fifth of the individuals with ID as young adults.
- Obesity was developed in 35% and impaired glucose tolerance was present in 20% in the individuals with ID as young adults.
- Young adults from practical high school educations had an increase in cardiometabolic risk factors comparable with the group with ID but they did not reach the same level. Young adults from earlier theoretical education had low cardiometabolic risk.
- Two years of school intervention aimed to induce healthy living habits for adolescents with ID resulted in a reduced increase in adiposity. This indicates that it is not the ID condition in itself but the effects ID has on the living conditions that cause the differences between individuals with and without ID.
- A specially designed lunch plate with the proportions of food according to the Plate Model inlaid might facilitate healthier food choices.

The difference in level and degree of cardiometabolic risk factors and the fully developed metabolic syndrome in this young group with ID compared to those without ID means an additional imposition on an already marginalised group. The results from the school intervention concerning physical activity and healthy food show that it is possible with limited but systematic actions by the society to at least delay the unwanted development in cardiometabolic risk factors. More research is needed on larger groups to confirm the results in this thesis. The individuals with ID require other pedagogic tools than non-ID adolescents to be able to adopt healthy living habits. Methods and materials specifically developed for individuals with ID are needed both for use in schools and primary care. It is of utmost importance to enhance the awareness of the increased cardiometabolic risk in people with ID for parents, child health care, school health care systems, primary care and hospital care. Given that people with an ID themselves can learn the importance of healthy living habits and thereby in the next step as adults be able to make their own choices.

Contributions from decision-makers would be to include people with ID in public health surveys and to include the specific needs of this group which constitute approximately 1% of our population in our health care system. This is one important step in our national goals toward “everybody’s right to an equal health”.

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A late evening on a night train to the ski resort Duved in northern Sweden, I presented an idea how to foster healthy living habits for my students with ID to my best friends, Lotta Carlheim Müller and Marie Kierkegaard. This was twelve years ago. I showed them my plans to apply for money in order to introduce healthy living habits at my work. They said; this is enough for a dissertation! I laughed and thought what a strange way to respond.

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This turned out to be a fantastic experience. We ate on the special Plate Model plate and all prominent visitors were impressed with the achievements of the school. My former colleagues expressed their satisfaction about the situation today compared to before.

For a newcomer, the academic research world is a world of good and bad and not the least full of new unexpected experiences. I have noticed cases where an academic career changes people in a surprising way.

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