GROWTH IN SWEDEN

Surveillance of growth patterns and epidemiological monitoring of secular changes in height and weight among children and adolescents

Bo Werner
GROWTH IN SWEDEN
Surveillance of growth patterns and epidemiological monitoring of secular changes in height and weight among children and adolescents

Bo Werner

Stockholm 2007

Copyright © Bo Werner

Cover: Conscripts’ examination
By Axel Petersson, ”Döderhultaren”, 1868–1925
Photograph of wooden sculpture,
By permission of Döderhultarmuseet, Oskarshamn, Sweden

Karolinska Institutet, Department of Public Health Sciences
Division of Social Medicine
SE-171 76 Stockholm, Sweden
Printed in Sweden by Tryckverkstan, Örebro, 2007
ABSTRACT

This thesis investigates the growth patterns of infants, children and adolescents in all of Sweden and the secular change for height and weight for schoolchildren. This thesis is based on the study of two cohorts: First, schoolchildren born 1973 from age 7 y to 18 y, and second children born in 1981 from birth to age 19 y. Cohort 1973 was all children born on the 15\textsuperscript{th} of any month in 1973, 3 579 boys and girls. Longitudinal data were collected from school health records. Missing cases were 4.5\%. Cohort 1981 was all children born on the 15\textsuperscript{th} of any month of 1981, 3 158 boys and girls. Longitudinal data were collected from child health records and school health records. Missing cases were 1.6\%.

Longitudinal data for somatic growth, cohort 1973 from 7 y to 18 y (height and weight), cohort 1981 from birth to 19 y (height and weight), from birth to age 48 mo (head circumference) from two nationally representative sample of children in Sweden, collected from child health records and school health records, can be used for epidemiological monitoring of growth with fewer missing individuals and at lower costs compared with other dedicated studies. Data quality is comparable to similar national surveys.

These studies represent, without selection bias, the current growth situation among children and adolescents, enabling both epidemiological comparisons over time and comparisons with other national surveys.

For the first time in Sweden, and without selection bias, means and distribution of head circumference measurements are documented longitudinally for a nationally representative sample of infants.

The time trend analyses revealed: An increased rate for those born in 1981 compared with those born in 1973 of relative weight reduction episodes was found for both boys and girls. The increase for girls were most pronounced, started from a higher rate and was seen in nearly all body weight categories and in all ages. For boys the reductions increased for all body weight categories in the age interval 7–9 y; otherwise the pattern was much more heterogeneous. Body weight and reduction of BMI were highly correlated in both cohorts, as more of the overweight than the thinner children reduced their BMI. For girls the increase in reduction rate between 1981-born and 1973-born was highest among the thinnest individuals.

From age 7 y to 18 y a strong positive secular change for BMI exists in all ages, and the rate of overweight and obesity is increasing for both boys and girls. Furthermore, obesity was growing more severe.

Keywords: adolescents, birthweight, body mass index, children, epidemiological monitoring, final height, growth, head circumference, height, infants, length, longitudinal study, national study, overweight, obesity, Sweden, secular change, schoolchildren, time trend, weight, weight loss
LIST OF PUBLICATIONS


V Werner B, Bodin L. Obesity in Swedish schoolchildren is increasing in both prevalence and severity. 2007 submitted.

All papers are reprinted with the permission of the respective copyright holders.
To my children, from birth to final height. Moa-Lisa -76: 50–167 cm, Tove -77: 49.5–165 cm and Joel -82: 52–184 cm.

Margit, Signe, Miriam, Elisif, Dagmar.
From Henrik Berg: ”Om barnavård”, Stockholm, 1898

"Pygmies placed on the shoulders of giants see more than the giants themselves."

Robert Burton 1621
CONTENTS

1. BACKGROUND .................................................................................................................. 7
  1.1 Introduction .................................................................................................................. 7
    1.1.1 Growth – nature and nurture ............................................................................... 7
    1.1.2 The description of growth ................................................................................... 10
  1.2 History of human growth ............................................................................................ 12
    1.2.1 The height of man in pre-historical times ......................................................... 13
    1.2.2 The stature of Neolithic and medieval populations .............................................. 14
    1.2.3 What we learn from soldiers ............................................................................. 15
    1.2.4 The first longitudinal studies ............................................................................. 18
  1.3 Secular changes ........................................................................................................... 19
    1.3.1 Birthweight ........................................................................................................ 22
    1.3.2 Final height ......................................................................................................... 23
    1.3.3 Age at menarche ............................................................................................... 24
    1.3.4 Body proportions ............................................................................................. 25
    1.3.5 Time trend of overweight and obesity .............................................................. 27
    1.3.6 Dietary changes in Sweden .............................................................................. 31
  1.4 Growth and depending factors ..................................................................................... 32
    1.4.1 Growth during pregnancy ................................................................................. 33
    1.4.2 Stature from birth to final height ........................................................................ 39
    1.4.3 Weight from birth to adulthood ........................................................................ 50
    1.4.4 Longitudinal aspects of growth and later health .............................................. 60
  1.5 Growth surveys, geographical coverage and design .................................................... 62
    1.5.1 Growth surveys in Sweden .............................................................................. 63
    1.5.2 Growth surveys – international examples ......................................................... 68
    1.5.3 Internationally used reference values ............................................................... 70
  2. AIMS ................................................................................................................................. 71
    2.1 General aim ............................................................................................................. 71
    2.2 Specific aims ........................................................................................................... 71
  3. MATERIAL AND METHODS .......................................................................................... 72
    3.1 Target populations and study populations ............................................................ 72
      3.1.1 Target populations ......................................................................................... 72
      3.1.2 Study populations ......................................................................................... 72
    3.2 Data collection ......................................................................................................... 75
      3.2.1 Cohort 1973 .................................................................................................... 76
      3.2.2 Cohort 1981 .................................................................................................... 76
3.3 Analysis.......................................................................................................................... 77
  3.3.1 Paper I, II and III........................................................................................................ 77
  3.3.2 Paper IV....................................................................................................................... 78
  3.3.3 Paper V......................................................................................................................... 79
3.4 Ethics.................................................................................................................................... 79
4. Results...................................................................................................................................... 80
  4.1 Paper I................................................................................................................................... 80
  4.2 Paper II................................................................................................................................ 80
  4.3 Paper III............................................................................................................................ 80
  4.4 Paper IV............................................................................................................................ 80
  4.5 Paper V................................................................................................................................ 81
5. General discussion.................................................................................................................. 82
  5.1 Epidemiological monitoring............................................................................................. 82
    5.1.1 The representativity of the samples............................................................................. 82
    5.1.2 Selection bias............................................................................................................... 83
    5.1.3 Data quality................................................................................................................ 84
    5.1.4 Comparability............................................................................................................ 84
  5.2 Comparison to other surveys............................................................................................. 85
    5.2.1 In Sweden................................................................................................................ 85
6. General conclusions............................................................................................................... 90
7. Considerations for further investigations.......................................................................... 91
8. Sammanfattning på svenska................................................................................................. 92
9. Acknowledgements............................................................................................................... 93
10. References.......................................................................................................................... 95
11. Papers I – V....................................................................................................................... 133
12. Appendix............................................................................................................................ 189
ABBREVIATIONS, CONCEPTS AND GLOSSARY

*Adipositas rebound* – from a rapid increase of *body mass index*, (BMI), the first years of life the BMI subsequently declines and reaches a minimum by the age of about 6 years, before beginning a gradual increase up to the end of growth. The point of minimum BMI has been called the adiposity rebound

*Adolescent growth spurt* – the rapid and intense increase in the rate of growth in height and weight that occurs during the adolescent stage of the human cycle

*Anthropometry* – the science of measuring the human body (height, weight and proportions)

*Anorexia nervosa* – a disorder that is characterized by low body weight, intense fear of weight gain, and an inaccurate perception of body weight or shape

*Anthropometric measures* – body measures, for example height, weight, relative weight and head circumference

*Auxology* – the science of physical and physiological growth of man (auxein=to grow, to increase)

*Benn index* – an index that in a statistical sense is highly correlated with weight and uncorrelated with height. Benn index is generally formulated weight/height^n, weight divided by height^n, where n is a number calculated from the data to ensure zero correlation between the index and height

*Binge eating* – is a recognized condition featuring episodic uncontrolled consumption, without compensatory activities, such as vomiting or laxative abuse, to avert weight gain

*Body mass index, BMI* – weight (kg) divided by the square of height. Higher BMI scores indicate that an individual has relatively more weight-for-height than a person with lower score. In the general population higher BMI scores usually indicate more body fatness

*Bulimia nervosa* – a disorder that includes both *binge eating*, and compensatory activities

*Catch up growth* – can be defined as a height velocity above the statistical limits of normality for age and/or maturity during a defined period of time, following a transient period of growth inhibition (recovery from disease or re-feeding after short-term starvation)

*Childhood phase* – a stage in the human life cycle that occurs between the end of infancy and the start of the juvenile phase

*Cross-sectional study* – measurement on a single occasion of individuals grouped by age, sex, and sometimes other characteristics

*Dedicated study* – a study performed especially arranged with a controlled situation when measuring according to method and age

*Descriptive* – how it (growth) is at a defined point of time or period

*Distance curve* – a graphic representation of the amount of growth achieved by an individual over time

*Diurnal variation of standing height* – the variation that means that an individual is tallest in the morning after sleep and shortest in the end of the day before sleep

*Early maturer* – an individual with early puberty and maturing

*Eating disorders* – includes *anorexia nervosa*, *bulimia nervosa*, and *binge eating*
**Epidemiology** – the study of the causes and transmission of disease

**Failure to thrive** – low rate of weight gain with no previously known disease explaining the condition

**Final height** – when no more height can be achieved

**Foetus** – stage of prenatal development lasting from the tenth week following conception to birth

**Genotype** – the genetic constitution of an individual

**Growth** – quantitative increase in size or mass

**Growth phase** – a part of growth of an individual that can be defined specifically

**Growth standard** see standard values

**Head circumference** – maximal fronto-occipital circumference or a Frankfurt plane circumference, but both techniques have largely been replaced by simply measuring the maximum head circumference

**Heritability** – an estimate of the relative genetic contribution to the phenotypic expression of a physical or behavioural characteristic. The value of the heritability estimate is a function of genetic factors, environmental factors and the interaction of genetic and environmental factors

**Heterosis** – the mating of individuals born in different geographic regions, which may lead to greater genetic variability and "genetic vitality" in their offspring

**Hominid** – living human beings and their extinct, fossil ancestors

**Infant phase or infancy** – a stage in the life cycle of all mammals. For human beings it lasts from the second month after birth to end of lactation, usually by age 36 months

**Juvenile phase** – a stage in the life cycle that is defined as the time of life when an individual is no longer dependent on parents for survival, and prior to that individual’s sexual maturity

**Kurtosis** – a statistical measure, indicating whether the distribution around the mean is thick-tailed (a higher proportion of subjects is found at the extremes of the distribution, kurtosis >0) or thin-tailed (the opposite case, kurtosis <0)

**Late matures** – individuals that have late *puberty* and maturing. One of the consequences of this is, for boys for example, sometimes growth up to age 25 years

**LMS** – a method by Cole to construct growth curves that summarizes the changing distribution by three curves (L= lambda, M= mu and S= sigma) representing the median, coefficient of variation and skewness, the latter expressed as a Box-Cox power. Using penalized likelihood the three curves can be fitted as cubic splines by non-linear regression, and the extent of smoothing required can be expressed in terms of smoothing parameters or equivalent degrees of freedom

**Longitudinal study** – measurements of the same individual or group of individuals, repeated over time

**Low birthweight** – a birthweight of 2 500 g or less for a neonate of normal gestation length

**Mean** – after adding all variable-values and then dividing the sum with the number of variable-values

**Median value** – the value that divides a distribution of data in two similar big parts. Is the same as *P50*

**Medical Birth Registry, MBR** – a registry that since 1973 includes birth data for all newborns in Sweden

**Menarche** – the first menstrual period

**Migration** – the movement of people from place to place
**Mixed-longitudinal study** – a study that combines a cross-sectional method with a longitudinal. For example 2-y-olds, 4-y-olds and 6-y-olds are followed, longitudinally, 2 years and thus covering age-interval from 2 years to 8 years but it takes only 2 years to perform

**Military Service Conscript Registry, MSCR** – a registry that includes, among other things, growth data collected at military conscription in Sweden

**Neolithic period** – earlier stone age, follows Palaeolithic period, which ended 10 000 years ago

**Neonatal period** – from birth to age 28 days

**Obesity** – BMI >30 for adults or age-related limits suggested, for example, by Cole et al.

**Osteology** – the branch of human anatomy that deals with the study of bones and the skeleton

**Osteologist** – one who is skilled in osteology, an osteologer

**Overweight** – BMI >25 for adults or age-related limits suggested, for example, by Cole et al.

**P50** – the 50th percentile; it is the same as median value

**Palaeolithic period** – later stone age, precedes Neolithic period

**Peak height velocity, PHV** – the maximal height velocity of an individual achieved during the adolescent growth spurt

**Percentile of growth** – a method of ranking growth status for height, weight, etc. of an individual to other members of a sample or population of people. Example: a child at the 75th percentile for height is taller than 75 percent of the other children in the group under consideration

**Phenotype** – the physical or behavioural appearance of an individual, resulting from the interaction of the genotype and the environment during growth and development

**Ponderal index** – weight divided by the cube of height

**Prescriptive** – how it (growth) ought to be

**Prevalence** – the total number of all individuals who have an attribute or disease at a particular time (or during a particular period) divided by the population at risk of having that attribute or disease at this point in time or midway through the period

**Psychosocial short stature** – a type of growth retardation produced by a negative physical and emotional environment for growth

**Puberty** – an event of short duration (days or a few weeks) that marks the reactivation of the central nervous system regulation of sexual development. Puberty occurs at the end of the juvenile stage and is the period that starts with PHV and where menarche of girls puts a relative upper limit for further growth. Usually a girl grows only 6–8 cm after menarche, seldom more than 10 cm and just in extreme case 12 cm

**Reference values** – constructed values as reference to compare individuals with

**Register study** – a study based on data from a registry

**Relative weight** – see further: body mass index, ponderal index, Quetelet index, Benn index

**Standard deviation, SD** – a measure of distribution

**Secular change (secular trend)** – the process that results in a change in the mean size or shape of individuals of a population from one generation to the next

**Sedente** – a person living in her geographic region of birth; a non-migrating individual

**Sexual dimorphism** – differences between the sexes in physical appearance, behavioural performance, and psychological characteristics
**Skewness** – a statistical measure that gives information on the deviation from normality, within each interval. Should be close to zero in the case of an approximately normal distribution within the interval

**Socio-economic status, SES** – an indicator, often defined by measures of occupation and education of the parents or head of household, used as a proxy for the general quality of the environment for growth and development of an individual

**Standard values (growth standard)** – normative values for growth

**Quetelet index** – see body mass index
1. BACKGROUND

1.1 INTRODUCTION

This thesis is aiming to be a part of the description of human somatic growth and to explore why studies of growth mirrors the social and health situation in populations or the state of public health in a society. There is a relation between somatic growth and social conditions both on an individual and a population level.

This thesis is also aiming to be a part of the longitudinal descriptions of health from birth to adulthood, the life course of the first part of life.

1.1.1 Growth – nature and nurture

*To realize the biological potential*

Some quotations about nature-nurture:

"Every character is both genetic and environmental in origin. Let us be quite clear about this. Genotype determines the potentialities of an organism. Environment determines which or how much of those potentialities shall be realised during development" [Thoday 1965].

"The height of man is a response to both nature and nurture. If we leave genetic differences aside changes in living conditions cause dramatic change in average height. The better those conditions are, the taller the average citizen becomes. "Although genes are important determinants of individual height, studies of genetically similar and dissimilar populations under various conditions suggest that differences in average height across most populations are largely attributable to environmental factors.” [Steckel 1995].

Human beings are plastic during development, and a single genotype can produce more than one alternative form of structure or physiological state in response to environmental conditions [Bateson 2001]. In general, plants and animals have a predetermined adult genetic size; this is referred to as their *genotypic size*. However, observation shows that the population’s mean adult size of any species in the natural environment always manifests a significant "growth deficit", demonstrating that no living organism has yet been able to achieve its theoretical genetic size. The actual size is called its *phenotypic size*. This difference between phenotype and genotype is commonly referred to as *nature-nurture* difference: Genotype-Phenotype=Growth deficit.

In fact, in human beings, the long slow historical reduction of this growth deficit is scientifically known as the "secular trend". Growth deficit on a population level is the secular trend, or more correctly the secular change, since "trend" indicates that it is a positive change, which is not always the case.

*The environmental conditions decide*

Even though many puzzles connected with human growth remain, the outlines of the process of growth and its connections with environmental influences are quite clear.
Growth is a measure of nutritional status, of the synergistic relationship between nutritional intake, work effort, and health. Malnourished and unhealthy populations are short, while well-fed and healthy ones are tall. This fact is of enormous importance to historians, because it means that height records offer a way of summing up many of the different influences on human existence that we think of when we speak of the standard of living. Growth statistics can complement measures of real wages in discussing the standard of living within individual societies, and they are also a means of comparing standards of living between different societies within the same genetic group.

Somatic growth is a mirror of conditions in society [Tanner 1987, Bielicki 1986]. To grow from foetus into man is a trajectory in order to attain the biological potential and a process of complicated interactions between protective and risk factors. As Tanner says:

"Provided always that the parental size is known, growth emerges as a prime measure of a child’s physical and mental health. The study of growth emerges also as a powerful tool to monitoring the health and nutrition status of populations, especially in ecological and economical circumstances that are sub-optimal. It can be used for studying the effect of political organisation upon the relative welfare of various social, cultural and ethnic groups that make up a modern state. Thus the study of growth has a very direct bearing upon human welfare. At the same time it provides valuable lessons on the way in which our biological heritage and our technological culture interact. It warns us that all too soon we could be technically capable of creating monstrously specialised or monstrously similar children. It points to our need to be reconciled with our origins, to see ourselves once again as a part of the natural order; not foetus into angel, nor foetus onto monster, but foetus into man.” [Tanner 1989 p.239]

Factors of importance

Growth and development result from the integrated work of four groups of factors: (1) genetic, (2) paragenetic (genetic resonance of the foetus), (3) factors related to lifestyle (work, sleep, etc) and (4) exogenous factors. The latter should be understood as the modifiers of development both natural and cultural (populational and intra-family) [Wolanski 1988].

Children’s height is a sensitive indicator of their physical well-being, and has been recommended as a measure of their nutritional state [WHO 1976]. WHO has in a series of reports addressed this issue [Jeliffe 1966, WHO 1976, Waterlow 1977, WHO 1983, WHO 1986, WHO 1995]. In 1996 de Onis and Habicht stated that anthropometry is the single most portable, universally applicable, inexpensive, and non-invasive method available to assess the proportions, size and, composition of the human body [de Onis 1996]. Associations between height and environmental characteristics have been investigated as possible indications of adverse influences on growth. In such investigations a number of variables including the parental social class, and the employment, the family size [Bielicki 1986], the number of parents at home [Garman 1982, Rona 1986], and the presence of overcrowding [Foster 1983, Rona 1986] have been used to characterize social conditions during childhood [Lissau 1993]. A gradient associating height with social class has been documented [Bielicki 1986], but the existence of a gradient does not in itself indicate a direct influence on growth.
It is necessary to adjust for confounding variables, but this raises a question: what confounding variables should be allowed for in such analyses? More complicated to analyse is the pattern of interactions between all the factors (variables) related to growth as an outcome.

**A proposal for health measures**

On the subject of measures for health and public health, Sally McIntyre says:

"There are a couple of problems with measures and measurements when comparing health of different populations or sub-groups within populations. Mortality, morbidity, health care utilisation, self-reported health have all their obvious limitations and problems." [Macintyre 1988].

She also sets up some criteria for useful measures:

* Dichotomous measures (present/absent) of rare phenomena are less useful than measures which can be used to characterize every individual and be subdivided into a number of categories, i.e. those which are continuously (and preferable nearly normally) distributed.
* We should be seeking for measures that summarize characteristics with substantial consequences for life-chances, not ones that are trivial.
* The measures should not be overly disease specific or organ specific but should have general significance for health and longevity.
* The measures should display social patterning rather than randomly distributed across countries, time and social groups.
* The measures should be acceptable to those on whom they are made, be practicable in field settings, and be reliable.

She concludes by saying: "Measures approximate to these criteria are height and body mass index." [Macintyre 1988].

**Somatic growth indicates health and public health**

Other authors, outside auxology, also argue that somatic growth is seen as an important indicator, both on individual and on population level, for health and public health [Floud 1990, Steckel 1995].

As Eveleth and Tanner put it: "At present we know that factors such as improved nutrition, control of infectious diseases, reduced family size, more widespread health and medical care, as well as population mobility (both geographically to urban areas and socially upwards) appear to be responsible for the increase in growth observed over the past 100 years in industrialized countries and, recently, in some developing ones. On the contrary, deprivations and poor living conditions, stress, etc., could strongly affect the sensitive growing years in a negative way, causing an absence of trend or even a negative tendency.

A child’s growth rate reflects, better than any other single index, his state of health and nutrition; and often indeed his psychological situation also. Similarly, the average value of children’s heights and weights reflect accurately the state of a nation’s public health and the average nutritional status of its citizens, when allowance is made for differences, if any, in genetic potential.” [Eveleth 1990].
Tall is not always best

Studies do not conclude that being tall is in itself a positive benefit. In old-fashioned warfare being big probably meant being a better target. Neither is longevity a simple consequence of increased stature. What the studies show is tallness being a product of good living conditions in a society, meaning good general health, and therefore bodies that on average are more likely to resist time’s ravages for a longer span of time. Another statement is important to do, in this context, namely that bigger does not necessarily mean better. In "Foetus into Man" James Tanner cites from a study of Frisancho and his co-workers from the slum outside Cuzco in the Andes in Peru, that it is the small mothers who have the largest number surviving babies [Tanner 1989, Frisancho 1980]. Small body size can be an advantage under certain circumstances. Malcolm in New Guinea [Malcolm 1970] and Stini in Latin America [Stini 1975] have come to the same conclusion. In an agricultural economy, a small man is more effective than a tall one and therefore he needs to do less work to earn his living.

Genetics matters

That genetics can explain some of the differences in stature between populations are illustrated for example by Bogin: The differences between the Netherlands, the US, Africa Turkana and Japan can be the expression of differences between societies. But the stature of Indians from Latin America and African pygmies has a more genetical explanation [Bogin 1999 p.226]. The African Efe pygmies may be, on average, the shortest people in the world, and their short stature appears to have a strong genetic component [Rimoin 1968, Merimee 1981, Hattori 1996].

A group with Chinese background living in Kingston, Jamaica was studied. All the children had from upper-middle to upper socio-economic status and attended private fee-paying schools. There were no significant differences in height or weight between the European, African and Afro-European groups. However, the Chinese sample was significantly shorter and lighter than the other three groups at almost every age, suggesting a hereditary difference in amount or rate of growth between the Chinese children and the other samples of children. More recent surveys of the growth of Chinese, Japanese, and other Asian children, adolescents, and adults find that they on average remain shorter and lighter than European and African populations living in the same cities or nations. However the differences in mean size have narrowed over the past 30 years [Eveleth 1990]. This is too short a time for genetic change, and indicates that other factors must be influencing the size of members of these populations.

Differences between different populations with various genetic heredity are described for example in "World-wide variation in Human Growth" [Eveleth 1990] and in "Foetus into Man" [Tanner 1989].

1.1.2 The description of growth

"Human growth has always fascinated statisticians, judging by the number of statistics books that use weight or height for examples. The fascination may be due to not only the ready availability and statistically well-behaved nature of the data, but also to the fact that growth is universal: everybody has experienced it." [Cole
The most obvious statistical construct used in the study of human growth is the fiction that growth is a smooth process. Hermanussen et al. has shown that on a sufficiently short time scale the process is anything but smooth, proceeding in fits and starts over periods of weeks [Hermanussen 1988]. Equally, on a scale of months there are important seasonal influences on growth. This is particularly so in the developing countries, where growth rates can vary enormously from one season to another [Maleta 2003], but even in the industrialized world there is clear evidence of seasonality [Marschall 1971, Gelander 1994].

In the 1960s, Tanner, Whitehouse and Takaishi published several different charts based on the heights and weights of British children. They distinguished between charts of attained height and weight, and charts showing the growth in height or weight over a period of a year or so; these two types of chart they termed distance and velocity, respectively, by analogy with a moving object [Tanner 1966].

Reference centile curves are used widely in medical practice as a screening tool. They identify subjects who are unusual, in the sense that their values of some particular measurement, for example height or weight, lie in one or other tail of the reference of the distribution. The need for centile curves, rather than a simple reference range, arises when the measurement is strongly dependent on some covariate, often age, so that the reference range changes with the covariate. The case for making the centile curves smooth is to some extent cosmetic – the centiles are more pleasing to the eye when smoothed appropriately – but there is also the underlying justification that physiologically small changes in the covariate are likely to lead to continuous changes in the measurement, so that the centiles ought to change smoothly. In such cases, fitting discontinuous curves could lead to substantial bias.

Relative weight – a measure with complications

Height and weight is by definition clear. But relative weight is more complicated, especially concerning growing people. The expression of relative weight ought to be an index that in a statistical sense is highly correlated with weight and uncorrelated with height. Benn index is of general form weight/height^n, weight divided by height^n, where n is a number calculated from the data to ensure zero correlation between the index and height [Benn 1971]. By testing this relationship you can see that n is about 3 for a newborn and then goes towards 2 at final height. A mean for n 0–18 y is 2.64 [Cole 1996, Bodin 2007].

The relationship between weight-height indices and the triceps skinfold measure among children age 5–12 y was studied by Michielutte [Michielutte 1984]: This study examines several weight for height indices – Quetelet’s index W/H^2, W/H and Ponderal index (Rohrer’s index) W/H^3 – for their appropriateness in estimating adiposity among young children. Data were obtained for a sample of 1 668 children age 5–12 y residing in Forsyth County, North Carolina. Although W/H^2 was found to be the most useful of these indices, the results suggest that no index, including the triceps skinfold measure, can be considered completely satisfactory in estimating adiposity among children [Michielutte 1984].

A description is not a prescription

Since there is often not an obvious distinction, in the literature, between how growth is and how it should be, it is very important to make the difference between these to
concepts quite clear. Descriptive values show the up-to-date situation for a population, i.e. epidemiological monitoring. No exclusions are made and everybody belonging to the population is included. From a description you can exclude sub-groups with deviating growth patterns in order to get values that, for different purposes, can be referred to in order to reveal deviating growth patterns. This you can call reference values [Sullivan 1991]. If you want to state what is normal you have to have an idea of what ”normal” is, and also what could count as desirable. If you succeed in this you have standard values [Preece 1988]. Now you can compare the growth of an individual or a population with the standard, and conclude if the comparison reveals a normal or un-normal growth-situation for the individual or population of investigation.

1.2 HISTORY OF HUMAN GROWTH

The beginning of history

The earliest documentation of stature of man is from osteological material from our ancestors who lived about 2 million years ago. The earliest written records about human growth date from Mesopotamia, about 3500 BC. Both in Sumerian art and in life there was a relationship between growth and biosocial conditions.

For the investigation of the historical stature of man there are three main types of sources:

1. Archaeological, bone and skeleton material from pre-historical times to 18th century.
2. Historical data from registries or other written documentation, from 18th and 19th century and onwards.
3. Surveys from the middle of 19th century.

Historians and growth

Historians have only recently become familiar with the notion that measurements of height are relevant to the standards of living [Floud in Komlos 1994]. Early work of Emmanuel Le Roy Ladurie in this field was sceptically received and was not followed up [Le Roy Ladurie 1971]. By contrast, human biologists and physical anthropologists have long believed that human growth is responsive to changes in the environment. An early pioneer in the measurement of human growth, Adolphe Quetelet (1796–1874), a Belgian whose work was widely known throughout Europe [Quetelet 1870], analysed the growth of children and adolescents, as did L.R. Villerme’ in France at the same time [Villerme’ 1829]. Their research inspired Edwin Chadwick in England to organize surveys of the height of children in factory districts in the 1830s [Chadwick 1842]. Thereafter, the study of human growth or ”auxology” became a scientific discipline of its own right, and has given rise to numerous surveys of growth throughout the world [Eveleth 1990].

Fogel was not the first historian to make use of anthropometry, since Emanuel le Roy Ladurie had used the records of French conscripts to describe the physical characteristics of Frenchmen in the early nineteenth century [Fogel 1993]. But Fogel’s use of anthropometric material, gathered by himself, Engerman and Coopersmith, represented the first attempt to use such material – in particular the records of the
heights of recruits – for comparative purposes and in the study of economic history and historical demography.

In the light of this knowledge, it is interesting that Karl Marx, presumably aware of the work of Quetelet, Villerme, and Chadwick, used height data in his discussion of the impact of industrialization. He referred to J. von Liebig’s description of the declining health of the French and German populations as shown by the condition and height of their military recruits, and argued that this decline in England made factory legislation necessary [Marx 1954].

1.2.1 The height of man in pre-historical times

Stature of our ancestors – from Homo habilis to Homo sapiens sapiens

Height modifications in the course of human evolution can only be made apparent by means of studying the osseous remains from our ancestors, a difficult task because of the scarcity of data for earlier periods and the methods used for stature estimation.

In spite of the obvious difficulties represented by the methods for height estimation and of the problems regarding anthropological remains [Trotter 1958, Bennike 1985], at present there are enough data about the evolution of this remarkable characteristic [Garralda 1991].

Homo habilis (2.2–1.8 million years ago) showed a high variability with heights between 120 and 150 cm.

By Homo erectus (1.8 million–300 000 years ago) for the first time a clear increase of stature is observed, the variation ranging globally, between 150 and 180 cm. H. erectus show much geographical variation in general appearance and size, since they were the first humans to occupy and adapt to not only the tropical or sub-tropical areas but also the coldest climates of Northern Eurasia.

In relation to the data that exist, H. erectus reflects the “actual human range” regarding stature. The chronological successors, and probably genetic relatives, the earlier forms of H. sapiens, furnishing measurements for height estimation, show similar variation; this is the case for H. sapiens sapiens.

With Homo sapiens sapiens (90 000 years ago) living in the Near East, males varied from 179 cm to 186 cm and females from 165 cm to 171.5 cm, and for those living in Europe during the later Palaeolithic age (40 000–10 000 years ago) males varied from 160.2 cm to 189.0 cm and females from 157.9 cm to 164.6 cm [Garralda 1991].

This high variability (sexual, populational) will remain throughout time; the general growth pattern of these ancestors are unknown although we can suppose that long infancy period and pubertal spurt existed; we can suppose that the variability answers partially to an ecosensitivity that was at least as great as nowadays, and partially to differences in genetic pool; in fact the higher stature of H. erectus was also accompanied by brain increase allowing remarkable cultural development, brain sizes between 850 to 900 cm³, better capacity of protection and hunting techniques; for more recent periods the available information confirms, at the same time, the ecosensitivity as well as the individual and populational genetic variation.

For details of cranial variation revealed by skeletal collection from Cambridge see further Lahr [Lahr 1996].
### 1.2.2 The stature of Neolithic and medieval populations

*Man settles down*

Neolithic age is a prehistorical period that followed the Palaeolithic age, which ended about 10 000 years ago. The Neolithic age is characterized by people beginning to settle down, starting to cultivate the land and gathered their livings in villages.

Archaeologists and osteological specialists who have scrutinised skeletons from those times have been able to show that there existed surprisingly tall individuals as far back as the hunting-gathering period. They have also argued that these people lived in a comparatively sound surrounding, enjoying a diet of satisfactory composition [Åkerman 1988, Bogin 1999].

An overview made by Bennike, osteologist and archaeologist, from archaeological populations shows findings on mean height for males and females, see Table 1 [Bennike 1985].

<table>
<thead>
<tr>
<th>Period</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>10000–4000 BC</td>
<td>160</td>
<td>156</td>
</tr>
<tr>
<td>4000–2300 BC</td>
<td>165</td>
<td>153</td>
</tr>
<tr>
<td>2300–1800 BC</td>
<td>177</td>
<td>166</td>
</tr>
<tr>
<td>1800–500 BC</td>
<td>173</td>
<td>167</td>
</tr>
<tr>
<td>0–400 AD</td>
<td>176</td>
<td>167</td>
</tr>
<tr>
<td>800–1050 AD</td>
<td>170</td>
<td>159</td>
</tr>
<tr>
<td>1050–1500 AD</td>
<td>172</td>
<td>162</td>
</tr>
<tr>
<td>1850 AD</td>
<td>165</td>
<td>154</td>
</tr>
</tbody>
</table>

If the terracotta warriors of the Qin dynasty army from 2 200 years ago in Xian, China do represent a selected part of the population and if the heights (169 cm to 196 cm) of the terracotta warriors reflect the heights of human warriors at that time we have here a selected sample that mirrors current stature 200 BC.

*The medieval situation*

"The implication will be that the transition from hunting to agriculture, from mobility to stable settlements, turned out to be more problematic. This fact has been revealed by skeleton material. This means that mankind had to pay a rather high price for the improved security of the agricultural society. As a consequence of a higher density of population we can register a series of epidemic diseases which have tormented rural as well as urban populations ever since. Through history peasants have a height 15 centimetres less than hunters-gatherers. In Sweden the height of man seems to have been rather stable from the Neolithic age to the medieval period" [Åkerman 1988]. But
between medieval populations stature can vary because of locally different social and nutritional conditions [Werdelin 1985].

However, there are exceptions to short height in the Middle Ages in Sweden: from male peasants living 1361 outside the city of Visby we know from a huge bone material that the average height among the peasants was almost 169 centimetres [Hultcrantz 1927]. Around 1900 the average height of men (conscripts) in Sweden just passed 170 cm.

Out of 3 305 skeletons from medieval Lund (at that time belonging to Denmark), between 990 AD and 1536 AD, means for height varied from 160 cm to 163 cm for women, and 171 cm to 175 cm for men [Arcini 1999].

From the village of Holje in Blekinge an archaeological example, from around 1700, where the main part of the village inhabitants died during the last epidemic of plague in Sweden (1710 and 1711) illustrates both height as a measure of living conditions and the different vulnerability of men and women: "Both men and women had an average height that corresponded to the height of the medieval population at that time, that is 159.8 cm for women and 170.6 cm for men […] the men, age between 20 y and 40 y when they died, most of them born between 1680 and 1690, had an average height of 166 cm while the men, age between 40 y and 60+ y had an average height of 171 cm. For the women the corresponding difference was 159 cm, age 20 y to 40 y, and 162 cm in age interval 40 y to 60+ y […] these data agree with the periods of famine we know existed in the 1680s and 1690s" [Arcini 2006]. So, in bad times men seem to be more vulnerable than women, as the average height decreased 5 cm for men compared to only 3 cm for women.

### 1.2.3 What we learn from soldiers

**The height of soldiers makes history**

During the last 100 years, the pattern for human somatic growth has changed in most countries. The somatic maturing has been achieved at an earlier age, while the adult somatic size has increased – an example of the secular change. The ideal method to conclude secular changes is to compare a series of repeated, representative samples from defined populations. These kinds of samples are only available for young men, from military conscription, from the middle of 19th century, and to some extent for slaves in North America [Steckel 1987].

Heights of recruits were recorded in Norway from 1741, and soon afterwards in England from 1755 and in Sweden from 1761. Later, it was common for conscription to be based on regular inspections, of very large sections of the young male population. Thus, records of height, health, and other physical and social characteristics (such as occupation and place of birth) for entire populations of young men have survived.

In Sweden, there is height information gathered since 1776 among soldier-crofter-population that allows us to make reconstructions populations born as early as in the 1740s. Aggregated national statistics about the conscripts can be found from 1840 and onwards, but we have been able to recover local material from certain areas from 1813 [Åkerman 1988]. The ordinary military personnel were on average taller than the conscripts, and they were not included among the conscripts in the muster rolls, but instead measured in a special procedure.
In the 20th century the enlistment of conscripts took place already at the age of 20 y, instead of 21 as was the case in the 19th century (See Table 2); therefore we have to make special arrangement to combine the older material with the modern. It must also be said that during the period 1813–60, persons who were rejected because of illness or physical handicaps are not included in the measurements. After 1860 we have the same information about them as about the accepted individuals. In the very beginning there were not many draft dodgers, but their number increased and reached the level of 6 000–8 000 persons out of about 40 000 every year in the middle of the 1870s. We know that a substantial part of these draft dodgers immigrated to North America and that this contingent tends to be taller than the average population [Åkerman 1988]. All this has to be considered when we make comparisons over time.

1840 we have conscription at age 21 y, and the mean height was 165.1 cm, and 1926 when the conscription was at age 20 y the mean height had increased to 172.5 cm. In Anthropologia Suecia the mean heights are described for all the counties around 1800. The national mean height was at this time 170.9 cm for conscripts [Retzius 1902]. Mean height for Swedish conscripts from 1841–45 (21 y) to 1952 (19 y) has increased with 8.4 cm, see Table 2.
Table 2. Mean height, in cm, at time for conscription for men in Sweden from 1841–45 to 1952. (1953 to 2004 see Table 4 page 17)

<table>
<thead>
<tr>
<th>Age (y) for conscripts</th>
<th>Year of conscription</th>
<th>Height</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>1841–45</td>
<td>167.4</td>
</tr>
<tr>
<td>21</td>
<td>1846–50</td>
<td>167.4</td>
</tr>
<tr>
<td>21</td>
<td>1851–55</td>
<td>167.8</td>
</tr>
<tr>
<td>21</td>
<td>1856–60</td>
<td>168.1</td>
</tr>
<tr>
<td>21</td>
<td>1861–65</td>
<td>168.5</td>
</tr>
<tr>
<td>21</td>
<td>1866–70</td>
<td>169.6</td>
</tr>
<tr>
<td>21</td>
<td>1887–90</td>
<td>169.2</td>
</tr>
<tr>
<td>21</td>
<td>1891–95</td>
<td>169.6</td>
</tr>
<tr>
<td>21</td>
<td>1896–1900</td>
<td>170.1</td>
</tr>
<tr>
<td>21</td>
<td>1901–05</td>
<td>170.8</td>
</tr>
<tr>
<td>21</td>
<td>1906–10</td>
<td>171.6</td>
</tr>
<tr>
<td>21–20</td>
<td>1911–15</td>
<td>172.0</td>
</tr>
<tr>
<td>20</td>
<td>1916–20</td>
<td>171.7</td>
</tr>
<tr>
<td>20</td>
<td>1921–25</td>
<td>172.1</td>
</tr>
<tr>
<td>20</td>
<td>1926–30</td>
<td>173.7</td>
</tr>
<tr>
<td>20</td>
<td>1931–35</td>
<td>173.2</td>
</tr>
<tr>
<td>20</td>
<td>1936–40</td>
<td>174.2</td>
</tr>
<tr>
<td>20</td>
<td>1941–44</td>
<td>174.5</td>
</tr>
<tr>
<td>20</td>
<td>1945</td>
<td>174.7</td>
</tr>
<tr>
<td>20</td>
<td>1946</td>
<td>174.9</td>
</tr>
<tr>
<td>20</td>
<td>1947</td>
<td>174.8</td>
</tr>
<tr>
<td>20</td>
<td>1948</td>
<td>174.9</td>
</tr>
<tr>
<td>20–19</td>
<td>1949</td>
<td>175.0</td>
</tr>
<tr>
<td>19</td>
<td>1950</td>
<td>175.3</td>
</tr>
<tr>
<td>19</td>
<td>1951</td>
<td>175.6</td>
</tr>
<tr>
<td>19</td>
<td>1952</td>
<td>175.8</td>
</tr>
</tbody>
</table>

Source: MSCR

Height differs between social groups (students, peasants, crofters, workers and farm labourers) and the differences are more pronounced than geographical differences or differences between the age cohorts over time. Students were two generations before the mean population as regards to the bodily height development. The mean height of students during the years 1873–74 was 173.2 cm. It was not until the 1930s that this was the mean height of the population in Sweden. Gunnar Sandberg and Herbert Steckel have used more fragmentary information about soldiers-crofters treating the whole period between 1750 and 1850 [Sandberg 1980].

Measuring of conscripts on a large scale was done in the beginning of the 1880s for example in Baden, Germany, by Ammon [Ammon 1894] and in Italy by Livi [Livi 1896].

Comparisons from values at military conscriptions over time have one complication. Mostly conscripts had not reached their final height at examination. The age for final height has continuously been getting lower. A hundred years ago final
height was reached at age 25 y, and today it will be reached below the age of 20 y except for late maturers [Hägg 1991]. See further 1.3.2

1.2.4 The first longitudinal studies

A son and some students were the first to be studied longitudinally

The Count du Montbeillard of France measured the stature of his son every sixth months from the boy's birth in 1759 to his eighteenth birthday. These data are usually considered to constitute the first longitudinal study of human growth. This was thus the first example of a distance curve of growth [Buffon 1777].

Another eighteenth century longitudinal study of growth is that of the students of the Carlschule, conducted between the years 1772 and 1794. The growth data showed that the sons of the nobility were, on average, taller than the sons of the bourgeoisie during the growing years, but that both groups achieved an approximately equal height at 21 y of age. Thus, the sons of the nobility experienced an advancement of the rate of growth [Komlos 1992].

In 1835, Lambert Adolphe Quetelet (1796–1874) published the first statistically complete study of the growth in height and weight of children. Quetelet was the first researcher to make use of the concept of the "normal curve" (today commonly called the normal distribution or "bell-shaped" curve) to describe the distribution of his growth measurements, and he also emphasized the importance of measuring samples of children, rather than individuals, to assess normal variation of growth. The
statistical approach of Quetelet was followed in Europe by Luigi Pagliani (1847–
1932). He began his studies on the size and fitness of Italian military personnel. He
later applied his methods to children and demonstrated that the growth status of
orphaned and abandoned boys, ages 10 to 19 y, improved after they were given care at
a state-run agricultural colony. Pagliani also noted that children from the higher social
classes were taller, and heavier, than poverty-stricken children. Finally, he was taking
longitudinal measurements of the same children. From these Pagliani noted that
menarche almost always followed the peak of the rapid increase in growth that takes
place during puberty [Pagliani 1876].

The early, big studies were cross-sectional

Villerme’ [Villerme’ 1829], Chadwick [Chadwick 1842] and Bowditch [Bowditch
1879], and from Scandinavia Wretlind [Wretlind 1878], Vahl [Vahl 1874–83], Key
[Key 1885], Malling-Hansen [Malling-Hansen 1883], and further Schmidt-Monnard
[Schmidt-Monnard 1895], Boas [Boas 1892], Camerer [Camerer 1893], and Dovertie
[Dovertie 1895] all made surveys on the growth of infants, children or adolescents and
were the pioneers in this field. Bowditch was the first to perform mass investigations
of the development of growth in different ages, and in Boston 13 691 boys and 10 904
girls were measured (height and weight) in different schools.

Boas’ scientific discoveries also include his research into the methodology of
growth studies [Boas 1892, Boas 1930]. Among other things Boas provided the
concept of tempo of growth to understand the difference between early and late
maturing individuals.

1.3 SECULAR CHANGES

To follow changes in populations

The word ”secular” has two meanings: worldly, especially pertaining to the material,
non-spiritual world, and just once in an age, indicating a relatively long span of time
[Bogin 1999 p.244]. The process is aptly named because the factors influencing the
secular trend are related to the material conditions of life, and these conditions do act
on human growth over long spans of time.

History reveals that the height of man, over time, both has increased and decreased.
For example, 8 000 years ago the mean height in Latin America was higher than the
relatively recently noted bottom value for mean height in the 1940s. The difference
was about 7 centimetres [Bogin 1999]. In the beginning of the 19th century, body
height was decreasing in many European countries, linked to the effects of the
industrial revolution [Floud 1990].

Secular changes take place not only within one country or region, but also when
people move between places. For example, it is well known that a positive secular
change is associated with migration from a low socio-economic status (SES) to a
higher SES environment or socio-economic improvement [Garn 1987, Bogin 1988]. A
few classic examples of the secular change in the growth of migrant children include
the work of Boas [Boas 1912, Boas 1940] with European immigrants to the United
States, the work of Shapiro [Shapiro 1939] with Japanese immigrants to Hawaii, the
work of Goldstein [Goldstein 1943] and Lasker [Lasker 1952] with Mexican
immigrants to the United States, and the studies by Greulich [Greulich 1976] of American-born children of Japanese descent. Follow-up studies of these same populations show that, with time, the growth in height of each generation of the children of migrants continues to increase until it converges on that of the host population [Roche 1979].

Investigations from Norway [Udjus 1964] and Japan [Tanner 1982] have shown that the secular trend of body height, which has been ongoing during the 20th century, has been more pronounced for the legs (length of the legs) than for the trunk (sitting height). These changes in the proportions of the body ought to have happened earlier and is a source of error when estimating body height from historical findings of skeletons.

Secular changes between 1880 and 2000 are reported from Poznan, Poland for body height and weight in children and adolescents. The results of measurements obtained in eight subsequent surveys, from 1880 to 2000, were included in the analysis. In general, in the 20th century, children grew taller and heavier and reached final body height and weight more rapidly. The biggest differences in body height and weight in the 20th century, observed at growth spurt, were about 17 cm and 11 kg for boys, and 13 cm and 13 kg for girls. The magnitude of secular changes in body height and weight in the 20th century was not stable. There were periods of increased and decreased intensity of acceleration of physical development (the 1950s and 1970s, and the 1960s and 1980s, respectively), as well as a period of deceleration (the 1940s). In the last decade, the tendency has been towards deceleration in most age groups studied [Krawczynski 2003]. From Zagreb schoolchildren Prebeg reports from secular changes 1951–91 [Prebeg 1995].

But why does the secular trend proceed in some countries but stops in other? To what degree is the secular trend inter-generational; do parents transfer their better health and stature to their children and if so, in what way? Are trends in body-height, weight and tempo synchronized to each other; do they increase or decrease together?

Overviews of secular trend have been done and broaden the perspective [Roche 1979, van Wieringen 1986, Tanner 1987, Hauspie 1997].

Soldiers tell us about secular changes

Information about adult stature in the past is mainly based on data about slaves or on data from conscripts. In some countries, at conscription, the shortest were excluded and thus increased the mean of the examined.

There are data without exclusions in the Netherlands, from 1851 to 1983 [van Wieringen 1986]. For example, a height of 170 cm, in the 76th centile, in 1863 had fallen to the 5th centile in 1983. A height of 180 cm in the 98th centile was in the 43rd centile 120 years later. Besides a minor year-to-year fluctuation and a small decrease around 1890, it was an increasing tendency during the period. There are still positive trends in the 1880s and in the 1890s [Gerver 1994, Fredriks 2000].

From Italian conscript data a decelerating height is seen. Between 1896 and 1900 mean height decreased with 1.9 cm but this is an exception to a continuously positive trend during 1854–1963 [Hermanussen 1995].

Recent data for 18-y-old men at conscription show an increasing trend in mean height between 1960 and 1990 in eleven countries [Schmidt 1995]. The Dutch are tallest (181 cm 1990) and the Portuguese are shortest (170 cm). The trend seems to be stronger in the shortest groups since the change has been 2.4 cm/decade in
Spain against 0.9 cm in Norway. The tall populations in north Europe show signs of decelerating trend during this period. Based on surveys of populations, changes of 1.0 cm are typical for Western Europe in last years while Eastern Europe and Japan have increased with 3.0 cm/decade [Hauspie 1997]. Based in conscription data from different countries, Larnkjaer states: Secular change in adult stature has come to a halt in northern Europe and Italy [Larnkjaer 2006].

Except conscript data there are not much data of adult height, especially not for women from cohorts born before the 1940s. From the 1946 and 1958 cohorts in the UK, data were collected for all, including their parents. In this way data of adult height were collected for people born between 1892 and 1958. For men the tendency was 10.9 mm/decade while for women it was 3.6 mm/decade [Kuh 1991, Wadworth 1997].

Sex-dimorphism, something to consider

The sex-dimorphism in height-tendency, noticed in the study of the 1946 cohort, is striking and has been noticed historically elsewhere [Arcini 2006]. It was bigger before 1940 than after. Among parents born before 1905 the fathers were 6.9% taller than the mothers, and in the 1958-cohort men were 9.3% taller than women. The authors pointed out that, in general, boys’ growth is more affected by the environment, it is more plastic than girls’, so in good times boys accelerate more and in bad times they decelerate [Kuh 1991]. Eveleth and Tanner believe the same [Eveleth 1990]. Anyhow, Kuh et al. do not find the proof for more plasticity for boys convincing and look for other explanations [Kuh 1991].

What is causing the sexual dimorphism in height tendency puts the light on an interesting contradiction. In his famous article about “regression to the mean”, Francis Galton showed that hereditary height made an 8% sex-difference in height when adjusting for the sex-difference of the descendants of the family [Galton 1886], and the same differences are seen in many populations today [Cole 2000]. But Cole like Kuh found that the percentage difference increased by time. Men have either kept on being 8%, or more, taller than women the last 110 years [Cole 2000, Kuh 1991].

Secular changes at different ages

The size of the secular change of children varies with age. Dutch children were at age 1 y shorter in 1997 than in 1965, while at age 8 y respectively 21 y of age they were considerably taller. In Japan, data were published from birth to age 17 y for children between 1940 and 1990, a period of time when dramatic changes in every age took place [Takaishi 1994, Takaishi 1995]. Changes between 1950 and 1990 show small effects both for boys and girls before the age of 2 y (about 10 mm/decade at 2 y), just like the Dutch children. After that the tendency increases with a maximum at 11 y for girls (about 30 mm/decade) and 14 years for boys (about 35 mm/decade) and return to values as adult (10 mm/decade girls, 15 mm/decade boys) like those by the age of 2 y [Cole 2000 p.319]. For boys the curve is still falling at 17 y and probably return (there are data just to 17 y) to the values at 2 y at the age of 20 y.

Consequently, the secular change in childhood seems to be divided in 3 distinct periods: before 2 y with a small tendency; from 2 y to puberty where the increasing tendency is clearly related to the pubertal growth spurt; post-puberty where the tendency decrease and finish like that of the adult. So, when the pubertal spurt is over,
the difference of adult height is the same as was already determined at the age of 2 years. These findings are confirmed by others [Bock 1989, Edlund 1999].

To reveal changes, comparable data are needed

The difficulty to catch secular changes in a valid way increases when using data which is neither representative nor comparable. Questions: Are measurements taken at the same age, and are the measures of good quality? Is there selection bias? But, we are often forced to do comparisons without straight comparability. This is something that has to be taken into consideration. In Sweden there are some unique possibilities, to use the Medical Birth Registry and data from conscription, and also to collect representative samples with few missing cases [Cnattingius 1990].

1.3.1 Birthweight

Birthweight is a central public health measure. The birthweight is the result of the intra-uterine growth and it is a starting point from which the life course in some respects is defined by. I developing countries the mean and distribution of birthweight points out in what direction the development of the country is going.

There is a positive secular change in birthweight in developed countries, including the UK [Alberman 1991] and Sweden. For Sweden, Meeuwisse suggests that if increasing size at birth is positively correlated with fatness in adulthood, this partly underlie the trend of increasing obesity [Meeuwisse 1998].

Looking at the most recent situation in Sweden and the development over the last 30 years, see Table 3, the picture is like this:

Table 3. Percentage of infants in different birthweight intervals 1973–2004 in Sweden

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight in gram</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 500–4 499</td>
<td>47.0</td>
<td>48.3</td>
<td>50.0</td>
<td>50.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 500–5 000</td>
<td>2.4</td>
<td>2.7</td>
<td>3.2</td>
<td>3.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;5 000</td>
<td>0.3</td>
<td>0.3</td>
<td>0.4</td>
<td>0.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;4 500</td>
<td>2.7</td>
<td>2.7</td>
<td>3.0</td>
<td>3.1</td>
<td>3.6</td>
<td>4.0</td>
<td>4.2</td>
<td>4.1</td>
</tr>
</tbody>
</table>

Source: [MBR]

The number of infants with birthweight >5000 g have doubled in 20 years. From 1992 to 2001, mean BMI for newly registered pregnant women, at the prenatal clinics in Sweden, increased from 23.4 to 24.5. During the same period the rate of smoking pregnant women decreased and the rate of maternal diabetes increased.
1.3.2 Final height

At what age we reach final height has varied through time. Udjus showed that the average age for completed growth in the Norwegian male population was 29 y in 1780, 25 y in 1860 and 20 y in 1960 [Udjus 1964]. In the Polish male population born in 1953 the average increment in stature was 2–3 cm between 19–27 years of age [Hulanicka 1983]. Data from the Solna-study by Taranger et al. show that the mean height raises up until age 20 y for boys and up till 18 y for girls [Hägg 1991].

For 18-y-old men in Sweden (see Table 4) we can see that from 1953 to 1962 mean height increased with 1.7 cm, and from 1962 to 2004 with 2.8 cm but from 1994 to 2004 just an increase with 0.7 cm, thus a decelerating increase over time. The development that remains is perhaps that we reach final height at an earlier age. It is possible to predict a future development for a population when looking at the growth of very healthy and social privileged groups.

In the Netherlands, data from the repeated national surveys show that the growing population is extremely tall in all ages, both boys and girls, and the secular change is developing in another way as in for example Scandinavia [Fredriks 2000]. But the exclusions made in the surveyed Dutch population and a huge number of missing cases, especially among older teenagers, make comparisons with other national data hard to evaluate. Exclusion is made for those who is not born in the country or for those having parents not born in the Netherlands.

From the MSCR we get conscription data from 1953 to 2004 in Table 4:

**Table 4. Height (cm) and BMI (kg/m²), means for men at conscription from 1953 to 2004 in Sweden (data from 1841 to 1952 see Table 2 page 11)**

<table>
<thead>
<tr>
<th>Age (y) at conscription</th>
<th>Year of conscription</th>
<th>Height</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>19</td>
<td>1953</td>
<td>175.7</td>
<td>21.4</td>
</tr>
<tr>
<td>18</td>
<td>1955</td>
<td>175.7</td>
<td>20.8</td>
</tr>
<tr>
<td>18</td>
<td>1957</td>
<td>175.8</td>
<td>20.9</td>
</tr>
<tr>
<td>18</td>
<td>1958</td>
<td>175.9</td>
<td>20.9</td>
</tr>
<tr>
<td>18</td>
<td>1960</td>
<td>177.0</td>
<td>20.9</td>
</tr>
<tr>
<td>18</td>
<td>1962</td>
<td>177.4</td>
<td>20.9</td>
</tr>
<tr>
<td>18</td>
<td>1966</td>
<td>177.7</td>
<td>20.9</td>
</tr>
<tr>
<td>18</td>
<td>1970/71</td>
<td>178.3</td>
<td>21.0</td>
</tr>
<tr>
<td>18</td>
<td>1974</td>
<td>178.4</td>
<td>21.3</td>
</tr>
<tr>
<td>18</td>
<td>1978</td>
<td>179.0</td>
<td>21.7</td>
</tr>
<tr>
<td>18</td>
<td>1982</td>
<td>179.1</td>
<td>21.7</td>
</tr>
<tr>
<td>18</td>
<td>1986</td>
<td>179.1</td>
<td>21.9</td>
</tr>
<tr>
<td>18</td>
<td>1990</td>
<td>179.4</td>
<td>22.0</td>
</tr>
<tr>
<td>18</td>
<td>1994</td>
<td>179.5</td>
<td>22.3</td>
</tr>
<tr>
<td>18</td>
<td>1998</td>
<td>179.8</td>
<td>22.5</td>
</tr>
<tr>
<td>18</td>
<td>2002</td>
<td>180.3</td>
<td>22.9</td>
</tr>
<tr>
<td>18</td>
<td>2004</td>
<td>180.2</td>
<td>23.0</td>
</tr>
</tbody>
</table>

Source: [MSCR]
Conscription took place at age 19 y in 1953–1954, but after that at age 18 y. We can here see that mean height for 18-y-old men has increased by 4.5 cm in 50 years, and mean BMI has increased with 2.2 units.

Just like increasing positive secular change of height, an earlier age of final height is a public health measure. Unfortunately, very few investigations of growth have data that cover the age intervals when final height is achieved: on a population level, for females somewhere between 16 y and 20 y and for males somewhere between 18 y and 23 y.

1.3.3 Age at menarche

The decreasing age-trend is decelerating

Age for menarche has decreased sharply during the last 150 years. In for example Norway [Liestöl 1982] and Denmark [Helm 1987] it has decreased with a speed of 12 months/decade. Menarche around age 13 y was achieved for girls born during World War II in Belgium [Wellens 1990], around 1946 in England [Roberts 1994], between 1953 and 1970 in Denmark [Helm 1998], and around 1965 in Poland [Hulanicka 1991]. In Belgium between 1919 and 1967 dropped the 90th percentile of age at menarche faster than the median value while the 10th percentile hardly changed, showing that late menarche has been less frequent during this period [Hauspie 1997]. Thus, over the last 30 years, the age at menarche has not changed though the positive secular change for height has kept on. Hulanicka felt that the turning point for age at menarche depends on a decline in standard of living in Poland after 1978 [Hulanicka 1991], but it might instead be a part of the more general pattern of stabilizing age at menarche over all of Europe after World War II [Cole 2000].

Relation to social factors

Menarche is achieved at about 12.5 years of age for girls of the middle-class from many nations, and at 14 years of age or later for girls of the lower socio-economic classes [Johnston 1974, Eveleth 1990]. The latest median age of menarche on record is 18 years of age for girls from the Bundi tribe of highland New Guinea [Malcolm 1970]. Malnutrition, heavy labour, infectious disease, and living at high altitude are some of the reasons for delayed maturation of this group. Since the 1970s, the living conditions of the Bundi have improved, especially in relation to nutrition and medical care, and the median age of menarche has declined [Worthman 1993].

Physical and physiological stress influences growth and development, including menarche and menstruation [Ruble 1982]. There are many reports that highly competitive female track athletes, who enter training before puberty, reach menarche later than girls in the general population [Malina 1973, Frisch 1980, Merzenich 1993].

Birthweight has decreased, stayed unchanged or increased, not related to height and development of menarche. The lack of synchronizing for height, birthweight and menarche trends tells us that there are different explanations to these phenomena [Cole 2000].
A genetical linkage for menarcheal age is reported by Moisan, who argues that the most convincing relation that exists is between the menarcheal ages for daughter and mother [Moisan 1990]. Two other factors are identified to have relevance for age of menarche: First, the size of a child during childhood. An analysis of 4,000 schoolgirls in Oslo related menarche to age, height and weight, where weight was shown to have a strong relation while height failed to have a significant relation [Liestøl 1995]. Many authors have shown that BMI at age 7 y or BMI before menarche was strongly predictive for later age at menarche [Stark 1989, Wellens 1992, St. George 1994, Cooper 1996, Power 1997]. These observations make it likely with a causal relation between weight and menarche, the so-called ”critical weight-hypothesis” proposed by Frisch et al. [Frisch 1970]. But Ellison did not succeed in finding any evidence of a critical weight or obesity at time for menarche. Instead he found that the velocity of gaining height was a far stronger predictor than weight, showing that the time for menarche is decided by the degree of maturing, not by body size. [Ellison 1982]. Twin studies too are showing that the genetic control of sexual maturing is independent of the genetic system that decides body size [Loesch 1995]. The ”critical weight-hypothesis” is now generally called into question, but in spite of that the has been arisen again and is now linked to the hormone leptin [Clayton 1997]. Furthermore, lack of linkage was found between height and weight and age at menarche during the secular shift in growth of Japanese children [Tsuzaki 1989].

Cooper et al. studied the influence of birthweight, and weight and height at age seven years, on menarcheal age in a national sample of 1,471 girls in England, Scotland and Wales. They found that girls who were heavier at age seven years had menarche at an earlier age. The average age of those in the highest fifth of the distribution of weight at seven years was 7.3 months less than that of those in the lowest fifth of the distribution. In contrast, girls who were heavier at birth had menarche at a later age. The extreme fifth’s differed 2.2 months for age at menarche. They conclude that the observations are consistent with the hypothesis that menarcheal age is linked to programmed patterns of gonadotrophin release established in utero, when the foetal hypothalamus is imprinted, and is subsequently modified by weight gain in childhood [Cooper 1996]. The other factor that affects age of menarche is an environmental factor linked till development of society is shown by Liestøl by studying hospital records from 1860 to 1950 in Norway [Liestøl 1982].

Time for menarche is closely related to maturing and tempo of growth. Early Tanner revealed that menarche appears about 1 year after PHV [Tanner 1962].

1.3.4 Body proportions

*Height does not vary as body proportions do*

Comparisons of stature and body proportion between blacks and whites in the United States provide another example of gene-environment interactions and their affect on growth. Fulwood et al. published data from the first National Health and Nutrition Examination Survey of the United States, which gathered anthropometric data on a nationally representative sample of blacks and white aged 18 to 74 years. When the data are adjusted for differences between the two ethnic groups in income and
education, urban or rural residence, and age, there are no significant differences in average height between black and white men. Nor is there a significant difference in average height between black and white women [Fulwood 1981]. Despite this, the body proportions for the two groups are different. Krogman found that for the same height, blacks living in one American city had shorter trunks and longer extremities than whites, especially the lower leg and forearm [Krogman 1970]. Hamill et al. found that this was also true for a national sample of black and white youths 12 to 17 y old [Hamill 1973].

**Examples of differences in body proportion between populations**

Differences in body proportion are known from other populations. Eveleth & Tanner surveyed studies of boys and girls of European (London), African (Ibadan), Asian (Hong Kong) and Australian Aborigine origin. In proportion to sitting height, the Australians had the longest legs, followed in order, by Africans, Europeans, and Asians. Expressed quantitatively: at a sitting height of 60 cm, for example, London boys have leg lengths averaging 43 cm, Ibadan boys 53 cm and Australian Aborigine boys 61 cm [Eveleth 1976 p.229]. Though unclear, many researchers have assumed that the body proportion differences are explainable only in terms of a genetic model.

**Nurture matters**

On the other hand, work on the growth of Japanese children suggests that environmental factors also may be powerful determinants of body proportion. Kondo & Eto found that between the years 1950 to 1970 the ratio of sitting height to leg length decreased for Japanese schoolchildren, meaning that the children became relatively longer-legged with time [Kondo 1975]. Tanner et al. confirmed this finding by comparing both the rate of growth and the amount of growth for Japanese schoolchildren measured in 1957, 1967 and 1977 [Tanner 1982]. Each successive cohort of children grew faster, and grew larger, than the previous cohort. Between 1957 and 1977, sitting height showed practically no increase, while increased leg length accounted for almost all the difference in height (4.3 cm for boys and 2.7 cm for girls). In 1977, adult Japanese had sitting height to leg length proportions similar to Northern Europeans, whereas 20 years earlier, the two populations were significantly different. The major influences on growth that that changed during the past two decades are improvement in nutrition (especially greater intakes of both protein and energy), health care, and sanitation [Kimura 1984, Takahashi 1984] and all this mirrors the great economic and social improvement in Japan the last decades [Marmot 1989].

Similar findings on the plasticity of body proportions are reported by several researchers who are working in Argentina, Poland and in Mexico [Bolzan 1993, Wolanski 1979, Dickinson 1990, Wolanski 1993, Siniarska 1995].

After 1977, the average height of Japanese men and women continued to increase, but at a slower rate [Takaishi 1995], and both leg length and sitting height seem to have increased at the same rate, at least for young women [Hojo 1981]. Since 1990 there is little evidence for further increase in stature. This means that the body proportions of Japanese and Northern Europeans remain similar, even though the two populations differ in mean stature [Bogin 1999 p.243]. It is difficult to establish a
genetic difference when comparing populations that live on different continents, since important environmental variations that can affect growth are likely to exist.

Another approach to the study of population differences in growth is to compare children and adults of different national or geographic backgrounds living in the same, or very similar, environments. Ashcroft et al. measured the heights and weights of 4- to 17-y-old children and adolescents of European, African, Afro-European descent [Ashcroft 1964, Ashcroft 1966].

1.3.5 Time trend of overweight and obesity

Despite difficulties with measurements and comparability, the trend is obvious

While measurement inconsistencies make it difficult to provide an overview of global prevalence of overweight and obesity in children, studies generally report high and increasing prevalence of overweight and obesity worldwide [WHO 1997, Livingstone 2001, Lobstein 2004]. Current estimates (1998) of childhood obesity prevalence in the USA suggest that 21.5% of African-Americans, 21.8% of Hispanics and 12.3% of non-Hispanic whites are overweight, and overweight increased rapidly between 1986 and 1998 [Strauss 2001]. In Australia, most recent estimates, for the ages 7–12 y, suggest that 16.1–16.9% of boys are overweight and 5.1–6.9% are obese, and for girls 17.4–20.4% are overweight, and 5.7–7.0% are obese [Lazarus 2000]. In the UK, reports suggest that the prevalence of obesity among children of all ages is increasing [Reilly 1999, Reilly 2001, Rudolf 2001]. Data from the National Study for Health and Growth show a dramatic rise in the proportion of overweight primary school children (aged 4–11 y) in the UK between 1984 and 1994 [Chinn 2001]; overweight increased from 5.4% to 9.0% in English boys, from 6.4% to 10.0% in Scottish boys, from 9.3% to 13.5% in English girls, and 10.4% to 15.0% in Scottish girls. Data from a large survey of young children (age 1 mo to 4 y) in England showed a similar rise in the prevalence of overweight [Bundred 2001]. In Denmark an increased prevalence of obesity was identified for birth cohorts from the early 1940s to the mid-1950s and from late 1960s and onwards [Olsen 2006].

What is "normal"?

Developing consistent approaches to the measurements of childhood obesity is a priority issue in this field [Barlow 1998] and international standardized cut-points have been proposed [Cole 2000]. For defining overweight in children, reference values for body mass index (BMI) are available from the US Centers for Disease Control and Prevention (CDC) [Kuczmarski 2000] and the IOTF (International Obesity Task Force) [Cole 2000]. Current expert opinion supports the use of BMI cut-off points for children and adolescents, but experts are at variance as to which centiles should be used for comparison [Bellizzi 1999, Reilly 2002].

Once again – comparability is crucial for epidemiological monitoring

The ongoing worldwide epidemic of overweight and obesity among children and adolescents is a crisis in public health [Lobstein 2004]. The epidemic needs a reliable and valid tool for epidemiological surveillance in order to really catch the
development. It is both important to describe the depth and speed of the time change and to discover when a trend is broken. But, what kind of surveys makes it possible to monitor this epidemic? How is the national situation monitored? It is rare that surveys are, in a statistical sense, representative for larger populations, and they are very seldom nationally representative. Data on body weight is either collected by weighing the individuals in dedicated studies, but more often by simply asking individuals about their body weight. You have to have a national representative study with minimal selection bias and it must be possible to repeat later under similar circumstances in order to study time changes properly. Growth data should be based on measurements. Sometimes you have a time period covered by register data, with national coverage, but just for one single age, as is the case with conscription data, for 18-y-old males, in Sweden [www.pliktverket.se].

The trajectory of obesity is revealed by longitudinal studies

Longitudinal studies of national representative samples make it possible to analyse, both within the cohorts, and between cohorts. Until now, to the best of our knowledge, there is only one single example of such an attempt, but that was a mixed longitudinal study [22]. Examples of growth monitoring by studying large samples do exist in the UK, the Netherlands, China, Finland, Australia, Canada, Norway and Sweden [Hughes 1997, Fredriks 2000, Luo 2002, Kautiainen 2002, Booth 2001, Tremblay 2002, Andersen 2005, Rasmussen 1999].

Comparison of prevalence data on obesity in children and adolescents around the world is difficult because of the lack of standardisation and interpretation of indicators for being overweight or obese in these age groups. There is actually no clear opinion on how to express relative weight or how to consider what a "normal" development in weight in relation to height is. Usually local or national percentile distributions of weight for age are used. These distributions may differ between regions and nations and are also subject to change over time. In addition, different percentiles are used for the definition of being overweight or obese (e.g. 85th, 90th, 95th, and 97th percentiles are used in different countries) [Seidell 1999].

Epidemiological monitoring in Sweden

The situation in Sweden is, up until now, best described by data collected at conscription. Conscript data cover the majority of 18-y-old males, yearly, from year 1944 to 2001, see Tables page 11 and 17. When looking at data from MSCR, Taranger in 1985 asked: Increasing rate of overweight of young men – a medical risk factor? [Taranger 1985]. And later, using the same data source, Rasmussen studied all males born in 1953, 1958, 1963, 1968, and between 1973 and 1977 [Rasmussen 1999]. Of the 503 689 subjects identified in the RTPs, information on BMI was available for 448 732. The increase between 1971 and 1995 was 4.7 kg for body weight, 0.5 cm for height, and 1.38 kg/m² for BMI.

Between 1971 and 1993 muscle power increased by 5.8% among all men. For all birth cohorts the mean muscle power was higher among the overweight individuals than among the conscripts with BMI in the normal range. The increase in muscle power over time was greater among individuals with BMI in the normal range (4.7%) than among individuals who were overweight (2.4%)
Among men born between 1953 and 1963 the prevalence of overweight was 7.2% for those living in large cities, and 9.7% in rural or sparsely populated areas. Among men born between 1975 and 1977 the prevalence of overweight was 13.3% in large cities and 17.5% in rural or sparsely populated areas.

Analyses conducted after exclusion of all young men from immigrant families clearly showed that immigration had no impact on the time trend.

The development of mean BMI among 18-y-old conscripts is illustrated for the years 1962, 1972, 1982, 1992 and 2000 by values 20.9, 21.3, 21.7, 22.1 and 22.7, see further Table page 17 [MSCR]. A shift is seen around 1972. Perhaps this indicates that boys born around 1950 still had a body weight which could be considered as "normal".


**Explanations for the obesity epidemic**

The rising prevalence of obesity in children has resulted in a range of health policy statements, and halting the rising prevalence of overweight and obesity in children is nominated as a public health priority [WHO 1997]. The increasing prevalence of obesity is not under genetic control but rather, related to two major lifestyle factors: the energy of the diet and an increasingly sedentary lifestyle [Jeffery 1987, Beunen 1992, Brownell 1994, Prentice 1995]. Prevention of overweight and obesity must rely on the modification of these two factors, and the efficacy of strategies that seek to alter these factors to cure obesity must be understood.

There are many reports that indicate that the prevalence of obesity and an increasing average BMI, even in a very short period during the last years, is a fact in many countries [Troiano 1995, Mei 1997, Hughes 1997].

Interpretation of these increases in childhood and adolescent obesity rates is difficult. Explanations require unbiased and precise estimates of energy intake and energy expenditure, and these are often unavailable. Small secular shifts in energy balance, which are all well within the margin of error of all available methods of assessment. Such assessments are further complicated by the likelihood that reported energy intake in children is considerably underestimated [Champagne 1998]. In the U.S., however, which is one of those countries in which energy intake over the last decades has, if anything, decreased [Kennedy 1997], there has been a dramatic recent increase in the prevalence of obesity. Some crude evidence suggests that the reduction in energy expenditure in children and adults is the most important determinant of this increase in obesity [Berkowitz 1985]. Several studies have reported low physical activity in obese children compared with their lean counterparts [Harrell 1997, Maffeis 1997], but this may be either a cause or a consequence of their obesity. Prospective studies, however, have also linked sedentary behaviour, such as television viewing, to the development of obesity [Dietz 1985, Robinson 1993, Robinson 1998].
**What comes first?**

Overweight and obesity in childhood are known to have significant impact on both physical and psychosocial health. For example, hyperlipidemia, hypertension and abnormal glucose tolerance occur with increased frequency in obese children and adolescents [Lauer 1975, Steinberger 1995]. In addition, obesity in childhood is known to be an independent risk factor for adult obesity [Whitaker 1997, Parsons 1999]. Guo and Chumlea report that the risk of developing adult obesity (BMI >28) in children older than nine years who are obese (defined as BMI above the 95th percentile for weight), is up to 80% at age 35 y [Guo 1999]. Furthermore, there is evidence of an association between adolescent obesity and increased risks for health in adult life [Must 1992, Power 1997, Must 1999]. The relative risks among men were 1.8 (95 percent confidence interval, 1.2 to 2.7; P=0.004), for mortality from all causes and 2.3 (95 percent confidence interval, 1.4 to 4.1; P=0.002), for mortality from coronary heart disease. It is noteworthy that overweight in adolescence was a more powerful predictor of risk than overweight in adulthood.

**Overviews that make the epidemic situation clear?**

By a summary of cross-sectional, nationally representative school-based surveys in 1997–1998 in 15 countries, with self-reported data, for 13- and 15-y-old boys and girls, the highest prevalence of overweight was found in the United States and the lowest in Lithuania. The highest rate in Europe was found in Greece [Lissau 2004a]. By presenting prevalence data from 21 surveys in Europe, a tendency was revealed for higher prevalence of overweight among children in western and especially southern Europe [Lobstein 2003].

The prevalence of childhood overweight has increased in almost all countries for which data are available. Exceptions are found among school-age children in Russia and to some extent Poland during the 1990s. Exceptions are also found among infant and pre-school children in some lower-income countries. Obesity and overweight has increased more dramatically in economically developed countries and in urbanized populations [Wang 2006]. The prevalence of overweight and obesity among children is rising in the European region, and the annualised rates of increase are themselves increasing [Jackson-Leach 2006].

These two reports by Lobstein and by Lissau [Lobstein 2003, Lissau 2004b] were examined by Lissau, and she concluded that the year of data collection, methods and use of appropriate statistics are of critical importance for the conclusion drawn from comparative epidemiological surveys on the prevalence of overweight [Lissau 2004b].

In the overview by Jackson-Leach [Jackson-Leach 2006] the figures for Sweden for instance is based on the surveys by Ekblom [Ekblom 2003] and Petersen [Petersen 2003]. The first survey have for example decreasing heights (we know that there is a still ongoing, although weak positive secular change for height), over time, for the populations compared that points on some problems in comparability and the second survey just covering the situation in the city of Umeå in the northernmost part of Sweden. This emphasizes the importance of national representative samples when making comparisons over time for revealing national trends in overweight and obesity.
1.3.6 Dietary changes in Sweden

It is hard to catch changes in eating habits on a national level in a scientific meaning. Surveys with both a valid content and without considerable number of missing cases are hard to perform. Anyhow, good surveys exist, though often local, but in 2003 the National Food Administration performed a survey of 4-, 8- and 1-y-olds from all of Sweden.

On the other hand we have data for a longer time period regarding the feeding situation in the first year of life.

Breastfeeding

The rate of breastfeeding in Sweden is possible to follow through official statistics, from 1964 up until now, except for the years 1976–1985, when official statistics are missing. Up until the mid-1930s, most children were born at home. As hospital deliveries increased, the proportion of breastfed children started to decrease. The decrease continued until the beginning of the 1970s, when a strong attitude shift occurred towards emphasizing the social and biological advantages of breastfeeding. This resulted in a strong increase of the breastfeeding frequency up until the mid-1980s. After that there seems to have been stagnation, or even a slight decrease. However, the period of decrease was short. From the beginning of the 1990s we have again seen a continued increase in the number of breastfed children. Currently, Sweden is the leading country among the developed nations with respect to the breastfeeding frequency [Socialstyrelsen Amningsstatistik].

Dietary changes in Västerbotten

The nutrition and health in Swedish children 1930–1980 are described by Persson: In 1930 a nutrition survey was made of 1 675 school children in the county of Västerbotten in northern Sweden [Odin 1934]. In 1967 a second survey was carried out in the same area, covering 1 411 children aged 4, 8 and 13 y [Samuelsson 1971]. A third survey was carried out in 1980 of 572 children in the same age groups. Underweight and iron deficiency anaemia was prevalent in 1930. In the last study the average energy intake had decreased from 100 to 87% of the Recommended Dietary Allowance (the same as Reference Daily Intake). A slight increase in the prevalence of overweight among 13-y-old children was also noted. The fat intake was lower in 1980 than in 1967. The malnutrition problems of 1930 have been eradicated but new nutritional problems, linked to the risk of developing obesity and health problems in adulthood such as coronary heart disease, call for new preventive strategies [Persson 1989]. After these surveys a school based dietary survey in 1989, using seven-day records, was performed in two cohorts, living in Umeå, Västerbotten, aged 14 and 17 y [Bergström 1993]. The study comprised 366 boys and 365 girls. When compared to these previous studies, a striking finding was a decrease in dietary fat intake and an increase in carbohydrate intake. However, the relative intake of saturated fat had not changed (15% of total energy). The dietary change was mainly due to an increased consumption of cereal products.
An example from Uppsala and Trollhättan

In a longitudinal study from 1993 to 1999, 208 adolescents were followed from age 15 y to 21 y in Uppsala and Trollhättan. At 17 and 21 y of age, the adolescents consumed significantly more pasta, vegetables, coffee and tea compared to age 15, while the frequency consumption of fat spread, milk, bread, potatoes, carrots and buns and biscuits decreased. The changes between 15 and 17 were smaller than between age 17 and 21. At age 21, the males decreased their intake of fruit, while the females decreased their intake of meat. No-meat consumers among females increased from 2 to 13%. Milk consumption decreased significantly in both sexes. Breakfast habits did not change: 90% had breakfast five times/week or more [von Post-Skagegård 2002].

National survey of eating habits for 4-, 8- and 11-y-olds in Sweden

In 2003 a national survey was performed that was completed by 590 out of 924 sampled 4-y-olds, 889 out of 1 209 sampled 8-y-olds and 1 016 out of 1 290 sampled 11-y-olds. No large differences were seen in food choice or nutrient intake between children from different socio-economic groups. Children to parents with a university education consumed more fruits and vegetables and had a diet with a slightly higher nutrient density. Children to parents with a non-Swedish background ate more fruit and vegetables but drank less milk. The result showed that the most desired changes in food habits are a lower intake of soft drinks, sweets, crisps, cakes and biscuits and an increase in the intake of fruits and vegetables [National Food Administration 2003].

1.4 GROWTH AND DEPENDING FACTORS

On the one side we have the whole society that influence the population to grow in one way, and on the other we have all studies that examine if there is a relation between growth and one single exposure, trying to take confounding under consideration. Though we want to know about all the interactions important for optimal growth and study growth in a "holistic" way, we almost always have to rely on studying one factor at a time, and the relation to growth. An ecological or interactional perspective developed for example by Magnusson or Bronfenbrenner [Magnusson 1988, Bronfenbrenner 1979] on growth are missing in the literature. Therefore this will be a review in a variable-oriented way.

A review of growth and depending factors, can be presented like this:

a. Growth during pregnancy-birthweight (1.4.1).
b. Height from birth to final height (1.4.2).
c. Weight from birth to adulthood (1.4.3)
d. Relations between a, b and c and later health (1.4.4)

Poor growth is known to have a relation with poverty and misfortune [Tanner 1992]. Obviously, secular changes in height are affected by socio-economic factors as social class [Kuh 1991], income and education [Meyer 1999], family-size [Chinn 1989], urban or countryside [Weber 1995] and region [Padez 1999]. Similar factors affect the tendency in menarcheal age [Laska-Mierzejewska 1982, Bielicki 1986,
Rimpelä 1993, Roberts 1994]. It is well-known that growth is affected by bad housing standard and population density [Foster 1983].

The secular change in final height has its origin during the first two years in life, probably mainly through increasing growth of the lower extremity. This period is when the post-natal growth is as fastest and thus most vulnerable to bad circumstances. In developing countries poor growth during childhood results sometimes in reduced height, and this we know is concentrated to the first 1–2 years of life.

Three causes to reduced growth of the child can be identified: nutrition, infections and mother-child interaction [Waterlow 1994]. Genetical explanations are not that important while children in developing countries who get their basic needs met are growing as well as children in the industrialized world. In terms of secular changes, future generations are coming from the same gene-pool, by definition, and the time-frame is too narrow for the gene-pool to go through any changes. So, genetical explanations are not a very likely part in the explanation of secular changes [Cole 2000].

The pattern of growth in childhood and adolescence is common to all human populations. Growth is most rapid in infancy, slows down during childhood, and then accelerates during puberty (the "adolescent growth spurt") before slowing again until final height is achieved sometime between the late teens and mid-twenties. The timing of particular events, such as the adolescent growth spurt or the achievement of final height, can vary between different genetic groups, but the overall pattern is common to all.

1.4.1 Growth during pregnancy

Growth starts at conception. What factors determine birthweight, and do they have any biological significance for the growth, future health and welfare of the individual? We know now that there is a relationship between birthweight and future health. In a series of studies Barker et al. have investigated the relation between intra-uterine growth and growth early in life and later susceptibility, later health and mortality [Barker 1989, 1992, 1993, 2001, 2002, Forsen 1999, 2000], and other authors have followed [Huxley 2000, Law 2002].

Firstly, there are genetic factors that contribute to birthweight [Johnston 2002], secondly, maternal nutrition determines size [Stephenson 2002], thirdly, there are social influences on birthweight [Drever1997, Spencer 2002].

Birthweight has been observed to relate to, among other factors, socio-economic status [Drever 1997], gestational age [Chamberlain 1975], maternal smoking [Butler 1972, Fox 1990, Nafstad 1997], gestational diabetes [Metzger 1990], and famine [Ravelli 1976].

Genetic factors

Epidemiological studies estimate that environmental influences account for about 25% of the birthweight variance, and genetic influences account for 38–80% of the birthweight variance [Penrose 1954, Magnus 1984, Magnus 1984, James 1998]. There is considerable variability in the estimates of the foetal and parental components of these genetic influences from 18 to 69.4%, and from 3 to 20% variance of birthweight
respectively. Overall there is strong evidence that genetic factors play a significant role in determining birth size.

Birthweight is correlated between half siblings of the same mother but not of the same father [Gluckman 1994] because of the greater contribution of the maternal genotype and environment [Walton 1954].

Familial trends in birthweight have also been observed. There is significant correlation between parental birthweight and birthweight in index cases using multiple regression analysis (mothers 0.19–0.20; fathers 0.12–0.16) [Langhoff-Roos 1987, Magnus 1997]. Other authors have reported evidence of strong familial trends in birth size [Magnus 1984, Carr-Hill 1987, Wang 1995].

Mikulandra reports from a study that the parents (father and/or mother) of male babies are older than those of girls, that pregnancy for male babies lasts longer and that male babies are born heavier than girls. With increased weight, and height and BMI in the father, the birthweight of both male and female babies increases [Mikulandra 2001].

Parental birthweight is a significant and independent predictor of low birthweight in offspring is reported from a Norwegian study. The estimate of the hereditability of birthweight in this study is lower than previously estimated from data within one generation in the Norwegian population [Magnus 2001].


**Maternal nutrition as a determinant**

Maternal nutrition, encompassing maternal dietary intake, circulating concentrations, utero-placental blood flow, and nutrient transfer across the placenta, influences birthweight. Summarized by James et al.: Genetic and environmental contributions (%) to birthweight variation: Genetic factors 38 (maternal genotype 20, foetal genotype 16 and foetal sex 2), environmental factors 62 (general maternal environment 18, immediate maternal environment 6, maternal age and parity 8 and unknown environmental influences 30) [James 1998].

**Deficiency and foetal growth**

The effects of severe macronutrient deficiency depend on the stage of gestation. During the Dutch famine in 1944–45, a 50% reduction in energy intake during the first trimester was associated with increased placental weight but not with change in birthweight. Maternal under-nutrition in late gestation was associated with reduced placental and foetal weights [Lumey 1998].

Embryo transfer and litter reduction experiments similarly show that maternal environment predominantly influences later foetal growth [Snow 1989]. Although macronutrient deficits in later pregnancy would be expected to exert greatest impact on birthweight, the human foetus weighs only 20% of term weight at 24 weeks [James 1998], catch up growth often occurs [Coutant 1998, Fewtrell 2001]. In contrast, the earlier in postnatal life that under-nutrition occurs, the more likely it is to have permanent – programming, that is – effects [Widdowson 1963]. In normal pregnancies of malnourished women, dietary supplementation during late pregnancy increases birthweight [Prentice 1991].
In developed countries, dietary macronutrient or micronutrient deficiency is rarely thought to be responsible for clinically significant impaired foetal growth [Robinson 2000]. Lower birthweight is associated with lower social class, but although it is often assumed that this is nutritional, there are many confounders such as smoking and genetic factors. Recent human pregnancy studies do not confirm the dietary hypothesis [Godfrey 1997, Mathews 1999], but these studies have been criticised [Symonds 2000]. Contemporary studies in Australia, however, indicate that nearly 30% of women who deliver babies with a low birthweight (<2500 g) suffer from eating disorders [Conti 1998]. Experimentally increasing maternal nutrition in sheep enhances birthweight [Symonds 2000].

In developing countries, maternal dietary intake can affect birthweight, and intervention helps. In developed countries, epidemiological studies and experiments using animals indicate that modest reductions in maternal food intake could affect survival at birth and longevity, in the absence of pathological changes in birthweight [Ravelli 1998, Heasman 2000]. It appears to be earlier maternal nutrient restriction that increases placental size [Heasman 1998] and alters the expression of genes regulating the glucocorticoid and renin-angiotensin systems [Whorwood 2001].

Improved long-term nutritional situation and living conditions seems to be the most important prerequisites to counteract low birthweight in developing countries [Andersson 1997]. Muller et al. come to the same conclusion after studying the situation in Papua New Guinea (PNG). Besides improving maternal health, interventions for improving birthweights in PNG should therefore aim at strengthening the economic base of rural populations and promote the cultivation and consumption of high quality foods [Muller 2002].

**Over-nutrition of mother, and foetal growth**

Many researchers have found that heavier women get heavier infants [Langhoff-Roos 1987, Rössner 1990]. For every extra maternal kilogram the birthweight will increase with 15–20 g [Cresswell 1997]. This cannot be accepted as the only or even as a main cause to the observed increase of birthweight over some decades [Meeuwisse 1998]. But Forsum et al. concludes that the relation between the body weight of the mother before and during pregnancy is important for the size of her baby [Forsum 2003]. See further 1.3.1.

**Social influences**

**Obvious relation though social factors are hard to isolate**

Birthweight, like growth, is determined by the complex interplay of genetic and environmental factors. The proportional contribution of these influences is unclear. However, birthweight varies within genetically similar populations [Mackenbach 1992, Elmén 1996, Spencer 1999], suggesting that environmental factors play a significant role. Positive or negative secular changes in birthweight are explained by an environmental influence [Rush 1983, Power 1994, Chike-Obi 1996, Brieger 1997, Bonellie 1997, Meeuwisse 1998, Orskou 2001]. Birthweight also shows a reverse social gradient such as that increasing disadvantage is associated with decreasing birthweight [Mackenbach 1992, Elmén 1996, Spencer 1999].
The effects of social class on birthweight, and its interaction with other maternal factors, were examined in groups of women bearing small-for-dates (SFD), average-for-dates (AFD) and large-for-dates (LFD) babies. The relative risk of a lower social class women having an SFD baby steadily decreased from 1.75 to 1.20 as adjustment was cumulatively made for smoking, hypertension, maternal age and height [Ounsted 1982].

The relationship between social position and birthweight is being studied. A total population of 102,638 single born, first-born infants was included in a Norwegian study. Children of parents with a high education (more than 15 years of schooling) had the highest birthweight. The same tendency appeared for paternal socio-economic status, but the differences were comparatively small. When examining income the pattern was different. The highest maternal income had the highest proportion of low birthweight offspring. When examining parental education jointly, it was found that the mothers’ education had the greatest impact on birthweight. By adjusting for female smoking, the association between maternal education and birthweight was weakened. However, assuming that birthweight is decreased by 200 g from smoking the effect was still significant [Arntzen 1994].

Interventions to increase birthweight in disadvantaged groups have been largely unsuccessful [Paneth 1995], and, although mean birthweight has increased [Alberman 1991, Meeuwisse 1998, Orskou 2001], the rate of change is slow and the gradient remains unchanged.

The social gradient in birthweight probably arises as a result of the accumulation and addition of risk and protective factors over time [Kuh 1997] and across generations [Emanuel 1992], rather than resulting from risk exposures within the index pregnancy. Biological processes during the foetal period are systematically linked to the social circumstances of the mother, but in a different way in the 1920s and in 1985 in Sweden [Vägerö 1999].

Practically all social differences in birthweight are related to the differences in maternal age, parity, height, and smoking habits. If a socio-economic indicator is to be included in the analysis of birthweights (for other reasons like international comparisons), education is recommended [Nordström 1994].

Environmental factors

Environmental factors with a known association to birthweight are nutrition, smoking, maternal ill health, and genital infection. Adult height has a known association with relative nutritional impairment in childhood [Berney 2000], and maternal height is an important determinant of birthweight [Kramer 1987].

Maternal health and birthweight

The association of other factors such as stress [Hoffman 1996] and exposure to some types of work during pregnancy [Homer 1989, Ahlborg 1989] remains unproven, but some authors notice that work during pregnancy appears to reduce the mean foetal weight in both primigravidas and multigravidas, and rest during the last six weeks of pregnancy improves foetal weight significantly [Alegre 1984]. Järvelin et al. stated after analysing a northern Finland cohort that their result taken together with the spatial clustering of birthweights, suggests that there may be important determinants of birthweight that have yet to be identified [Järvelin 1997]. Evidence for an independent
effect of stress is slight, but one study does show stress exerting an effect through increased smoking [Sheehan 1998], while another one does not [Hedegaard 1996].

Other risk factors for low birthweight such as maternal age, although not itself environmental factors, are strongly influenced by the social environment. Severe energy restriction during pregnancy, such as occurs in some developing countries [Achadi 1995] and was noted in the 1945 Dutch Hunger Winter [Hart 1993], reduces birthweight but, randomised controlled trials of nutritional interventions in the index pregnancy have failed to show convincing benefit [Kramer 1998]. Nutrition may exert its effect over a longer period through an effect on maternal growth in childhood [Baird 1980] and possibly through an intergenerational effect [Emanuel 1992].

Maternal ill health has been associated with reduced birthweight [Baird 1974], and genital infection exerts its influence through increasing the risk of preterm delivery [Divers 1993]. It is concluded that hypertensive pregnancies are characterized by lower birthweight and shorter gestational period. However, intra-uterine growth retardation is not a general characteristic of hypertension in pregnancy [Himmelmann 1996].

**Smoking mothers and their babies**

The association of smoking with a reduction in birthweight is well established [Butler 1972, Rush 1974, 1983, Kramer 1987, Ahlborg 1991, Wilcox 1993, Nordström 1994, Bjerregaard 1996, Ellard 1996, Groff 1997, Secker-Walker 1997, England 2001]. Brooke et al. conclude that smoking was the most important single factor (5% reduction in corrected birthweight). Passive smoking was not significant (0.5% reduction). After smoking was controlled for, alcohol had an effect only in smokers and the effects of caffeine became non-significant. Only four of the socio-economic and stress factors significantly reduced birthweight and these effects became non-significant after smoking was controlled for [Brooke 1989]. The relation between passive smoke exposure and birthweight is not that convincing [Martin 1986, Rubin 1986].

Recently, Ingvarsson et al. conclude: "Smoking during pregnancy causes symmetrical fetal growth impairment, possible due to decreased oxygen transport to the fetus and decreased concentrations of fetal insulin, insulin-like growth factor I and IGF binding protein 3." [Ingvarsson 2007]

In most investigations, the reduction in birthweight has been found to range between 170 and 235 g, even after adjustment for maternal age, height, weight, weight gain, alcohol consumption and birthweight of previous children [Rush 1974, Davies 1976, Wainright 1983, Secher 1990].

**The first-born babies**

In general terms a woman is more likely to deliver a heavier baby in her second pregnancy than in her first pregnancy [Wilcox 1996]. In Sweden there is an increasing birthweight from the first-born to the third child, and after that birthweight decreases [MBR].
Coffee, any relation?

A significant reduction in birthweight was found to be associated with an average caffeine intake of \( \geq 71 \) mg per day, after adjustment for gestational age, infant sex, and maternal height and weight, but only in infants born to non-smoking mothers [Vlajinac 1997]. Caffeine intake during pregnancy is a risk factor for intra-uterine growth retardation but not for preterm delivery or low birthweight concludes Fortier et al. [Fortier 1993]. As a result, Fenster et al. suggest that heavy caffeine consumption increases the risk for foetal growth retardation [Fenster 1991].

Alcohol, any relation?

Mills noted an effect of alcohol to reduce birthweight [Mills 1984] and Larroque noted a significant reduction in birthweight associated with an average daily alcohol consumption of three drinks or more after gestational age, infant sex, maternal age, parity, weight, and height, and cigarette smoking had been controlled for [Larroque 1993].

Other drugs

Hatch could not detect any effect of marijuana on foetal growth [Hatch 1986]. After a meta-analysis English et al. conclude: There is inadequate evidence that cannabis, at the amount typically consumed by pregnant women, causes low birthweight [English 1997].

Intra-uterine growth retardation

Small-for date babies, many causes

Both the degree and the cause of growth retardation decide the post-natal growth together with the environment. Intra-uterine growth retardation is one of the most frequent types of severe shortness of stature persisting during childhood and adulthood [Job 1991]. By definition, 3% of newborns, either at term or before, are below the statistical limits of normality for length, regardless of the term [Usher 1969], and only part of them will have a postnatal catch-up growth. Intra-uterine growth retardation (IUGR) is a heterogeneous group of disorders, including many malformative or dysmorphic syndromes with or without detectable abnormality of chromosomes and/or genes, a variety of foetal diseases due to maternal conditions (hypertension, drugs, alcohol and other toxins, etc.), to materno-foetal transmission of viruses or other infectious agents, or to anatomical and functional abnormalities of the placenta, uterus, or cord. Such causes may in fact be lacking or not detected, so that many cases of IUGR remain unexplained. The heterogeneity of small-for-date babies followed in neonatology clinics makes a comprehensive appreciation of postnatal events difficult. However, longitudinal studies show that approximately two-thirds of IUGR newborns have some catch-up growing during their first months of life [Tanner 1975, Nilsen 1984, Thierot-Prevost 1988, Fitzhardinge 1989]. Those whose height remains subnormal after 1 year will probably not have a catch-up growth in the following years. And part of those whose height had improved during their first postnatal year will have an insufficient growth velocity during their second to fourth year, so that
they might thereafter return at the third percentile of height or below. No fixed correlation in this evolution of postnatal growth has been demonstrated.

1.4.2 Stature from birth to final height

Gender differences

*Gender differences, and different susceptibility for bad times*

From the first longitudinal study, the study of Montbeillard’s son, we can see the four distinct phases of growth velocity: 1. From birth to 3 years of age, the infant phase, growth decelerates rapidly from its maximum value of 22 cm/year to 6 –7 cm/year. 2. From 3 to about 7 years of age, the childhood phase, growth remains fairly constant. After age 7 years until about 11 years, the juvenile phase, growth decelerates again, at first rather slowly but then much faster. 4. From about 11 to 18 years of age, the adolescence phase, there is a classic adolescent growth spurt, with an acceleration period from about 11 to 14.5 years followed by a deceleration period that continues until 18 years and beyond.

In Europe today men are on average 12–13 cm taller than women. Secular change in this gender difference has been shown [Hall 1978]. Many data suggest that it was a small difference in height between the genders during periods with bad life circumstances. These observations support the opinion that growth of male individuals more easily depends of environmental factors like under-nutrition and diseases than do the growth of female individuals [Eveleth 1990, Kuh 1991, Arcini 2006].

Longitudinal studies, that is, studies of growth of the same individuals from birth to adult age, has shown that gender differences in height are built up by four components of growth [Prader 1984], see Table 4.

**Table 4. Differences between boys and girls in growth**

<table>
<thead>
<tr>
<th>Component</th>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Differences in pre-pubertal growth-velocity, which is faster for boys before birth and during the first year of life.</td>
<td>+1.5 cm</td>
<td></td>
</tr>
<tr>
<td>2. Longer pre-pubertal period of growth for boys, who get their peak velocity in height about 2 years earlier.</td>
<td>+6.5 cm</td>
<td></td>
</tr>
<tr>
<td>3. More intensive peak height velocity during puberty for boys.</td>
<td>+6.0 cm</td>
<td></td>
</tr>
<tr>
<td>4. More growth for girls after the pubertal peak height velocity period</td>
<td>-1.5 cm</td>
<td></td>
</tr>
<tr>
<td><strong>Difference</strong></td>
<td><strong>+12.5 cm</strong></td>
<td></td>
</tr>
</tbody>
</table>

Thus, the result is that in general men are 8% taller than women.

After menarche a girl normally gains 6–8 cm, seldom more than 10 cm [Tanner 1989]. Late maturers among boys can grow up to 25 years of age [Hägg 1991]. Whether you are an early or a late maturer, it is only the legs that show a smaller adult size for early maturers. The shorter growth period is compensated by a higher
prepubertal growth velocity and a higher level in pubertal years. The pubertal peak is a little larger for early maturing boys but not for girls [Gasser 2001].

**Genetic factors**

The old debate between the relative importances of genes versus the environment in human development is largely ignored by most researchers today. In reality, the biological development of the human being is always due to the interaction of both genes and the environment [Bogin 1999]. It is erroneous to consider whether one or the other is more important; genes are inherited and “… everything else is developed” [Tanner 1978].

The human phenotype is the outcome of this interaction. All of the measurable characteristics of the human body, of human behaviour, and of the human mind are phenotypic traits. Although it is possible to describe a human genotype with great precision, such knowledge by itself provides very little information about how a human being will develop without also knowing about the environment with equal precision [Bogin 1999 p.240]. See also 1.1.1 and 1.4.1 (Genetic factors).

**Family-related factors**

Comparative growth of primary schoolchildren from one and two parent families has been studied by Garman: Children from one parent families were shorter than children from two parent families; however, once heights had been adjusted for birthweight, number of siblings, mother’s height, father’s height, and mother’s education this was no longer the case. The higher prevalence of low birthweights and shorter parents that account for the shorter stature of one parent children are factors that cannot be ignored in a consideration of the health and growth of this group of children, and obesity may be a potential health problem among the one parent family children [Garman 1982].

**Socio-economic status (SES)**

*SES and social class have a strong impact on growth*

It is often argued that socio-economic status, SES, is, in reality, only a proxy for better health care, which reduces childhood mortality and morbidity, and results in increased growth. However, the SES effect is more subtle than this. Bielicki & Welon listed four primary factors relating SES and growth: (1) higher SES allows for better nutrition, (2) better health care, (3) reduced physical labour for children, and (4), greater growth-promoting psychological from parents, schools, and peers [Bielicki 1982].

In all societies, social class and SES are powerful influences on human physical and psychological growth and development. Conversely, stature, body composition (fatness and muscularity), and rate of development influence the social, emotional, and economic status of children, youth, and adults. An appreciation of these biosocial interactions between growth and socio-economic status (SES) has been realized only in the past 200 years. Following the studies of children in the Carlschule in the eighteenth and the work of Pagliani in Italy and Bowditch in the United States and Key in Sweden in the nineteenth century, it has been known that children of lower SES are generally smaller and mature less rapidly than children of higher SES.
The National Child Development Study of the UK is a longitudinal study of the influence of SES on growth in height and weight. Data from this study are based on the population of all infants born in England, Scotland, and Wales between March 3 and 9, 1958. Lasker and Mascie-Taylor published the mean height, weight, and BMI of these boys and girls at ages 7, 11, 16, and 23 y, stratified by the social class of the male head of the household (grouped from I to V with I as the upper class). They found that mean body size is significantly related to social class, and declines, generally, from social class I to V at each age. The data for males are paralleled compared to data for females. The statistical impact of the social class effect is achieved by age 7 y, and is then maintained to age 23 y. The notable exception to the stability of size by social class over time is the change in BMI between ages 11 and 16 y. The three lower classes, III, IV, and V, have a significant increase in BMI compared with the social class I, and social class II. The change is due to absolutely faster rate of growth in weight of the lower three classes [Lasker 1989, Lasker 1996]. Lasker and Mascie-Taylor also analysed the association between body size of the boys and girls in the study with the social mobility of their father. They found that the study subjects with fathers who moved up one social class were large for their old social class but small for their new social class. The effect of the size of the child and social mobility of the father was found for the 7-y-olds, but was not statistically significant for 11-or 16-y-olds. Changing of occupation improves the family situation and, at an age most susceptible, improvement leads to better growth [Lasker 1989].

The same situation, as for growth and the relation to SES, is between socio-economic status and maturation [Johnston 1974, Bielicki 1982, Low 1982].

Small or no effect?

In recent years, only three nations, The Netherlands, Norway, and Sweden, as far as it is documented, seem to have eliminated or powerful decreased the differences in growth between children from higher and lower SES levels [Brundlund 1975, Lindgren 1976, Brundtland 1980, Roede 1985, Lindgren 1995]. In Sweden for example, in 1979 Lindgren reported that differences decreased during the era after World War II, but in 1980 she reported that significant differences in height and head circumference were found between 5.5-y-old children of the highest and lowest SES groups. The reason for the reappearance of an SES effect is not completely clear, but it is perhaps an effect of the fact that the gap between rich and poor in Sweden has been widening during the last decades [Lindgren 1994].

Data were analysed for 8 491 representative sample children measured in England and Scotland in 1987 and 1988, and 3 203 inner city children were measured in England in 1987. Height was negatively associated with social class but the association was not significant after allowing for biological variables. A negative gradient of height with size of sib-ship was evident in white children but less so in Afro-Caribbean and Asian children. The individual associations of 11 different environmental characteristics were examined after allowing for biological factors and size of sib-ship. Consistent associations with height included a negative gradient of height with increasing latitude and an association of taller stature with increasing maternal age. A social class gradient in height is accounted for by association with biological factors, particularly the parental heights; environmental attributes are weakly associated with height after allowing for biological factors [Gulliford 1991].
The contents of the social variable and the relation to growth is demonstrated by Söderfeldt and Werner by comparing the relation between the height and weight at age 4 y with three different measures for social class [Söderfeldt 1988].

There can be no doubt that physical abuse and neglect of children leads to short stature and failure to thrive. The correlation between speech and developmental delay and shortness leads to the conclusion that both are manifestations of long-term unhappiness and disruption within the family. The short, abused children will show a recovery of growth with the more radical changes in the home circumstances, and this can be used to gauge the “success” of any intervention [Wales 1991].

*Social mobility and growth*

Empirical data have been gathered on 6 162 19- and 20-y-old Flemish males. The mobility categories have been set up on the basis of the social professional status of the father of the tested subjects and the social professional aspiration level aimed at by the tested subjects themselves.

Statistically significant relations of varying importance have been found between the nature and the degree of social mobility and the vast majority or the anthropological characteristics considered in the present investigation. According to the social professional origin, the aspiration level being kept constant, the former relations disappear almost completely, except for groups originating from agriculturalists [Cliquet 1968].

The present findings may at least partially be considered as being the result of social assortment. Up to a certain level, the social assortment of genotypes should be taken into consideration.

*The hen or the egg – what comes first?*

Scott et al. found that taller women from Aberdeen, Scotland worked at more skilled jobs than shorter women. Furthermore, when taller women married, their husband worked at more skilled jobs than the husbands of shorter women [Scott 1956]. These findings was confirmed by Schreider in a study of British women showing that, regardless of the occupation of the father, taller women tended to marry men with more highly skilled jobs and shorter women tended to marry men performing semi-skilled or unskilled labour [Schreider 1964].

Taller people achieve higher educational status than shorter people, as was shown by Parnell for university students in England and other European countries [Parnell 1954] and by Kimura for Japanese university students [Kimura 1984]. Bielicki and Charzewski found that within families the taller siblings were better educated than shorter siblings. The average difference between the better educated and the less educated brothers was 1.26 cm, a statistically significant difference. The result indicates that tallness leads to a higher level of education [Bielicki 1983]. These findings were confirmed by Susanne [Susanne 1980]. By studying stature, upward social mobility and the nature of statural differences between social classes, Bielicki could confirm the positive effect of social mobility upwards on growth [Bielicki 1992].

Numerous studies, from a diversity of disciplines, find that the taller, non-obese man or woman is given preference in many arenas linked with SES, such as the perception of intelligence, academic performance, and social skills, as well as initial

**Psychosocial factors**

The psychosocial short stature is a well-studied condition that shows how the endocrine system mediates the relationship between psychological factors and physical growth. Psychosocial short stature is a clinical condition of retarded growth stature that cannot be ascribed to an organic problem with the child, but rather to behavioural disturbance and emotional stress in the environment in which the child lives. A diagnosis of psychosocial short stature is confirmed when the child is removed from that environment, and growth is spontaneously restored. Spitz carefully investigated the causes of poor growth experienced by emotionally disturbed infants and children [Spitz 1945].

Growth failure caused by emotional deprivation in children was first reported more than 50 years ago [Talbot 1947] and was subsequently the subject of further reports [Patton 1962, Powell 1967, Powell 1967]. Powell et al. suggested that emotional deprivation of children with psychosocial short stature resulted in growth hormone deficiency secondary to hypopituitarism [Powell 1967, Powell 1967], and that this was reversible many months after the change in environment [Powell 1967, Powell 1973]. It was proposed that the diagnosis of psychogenic growth failure should be based on the child’s response to being separated from the parents, as expressed by an increase in growth velocity [Job 1987, Golkhe 1998]. 18 children diagnosed as having psychosocial short stature, and who had a change in their environment, increased their mean height velocity and attained a stature towards the lower limit of the range of their biological expected final height [Golkhe 2002].

**Life-style factors**

Sleep habits and height at age 5 to 11 y were investigated by Gulliford. Shorter duration of slow wave sleep and lower growth hormone responses have been reported in children with short stature caused by psychosocial deprivation. An investigation was carried out to determine whether lower total sleep duration was associated with shorter stature, and it was investigated in a sample of children taking part in the National Study of Health and Growth. Parental responses to a self-administered questionnaire were used to estimate usual times for going to sleep at night and usual time for waking in the morning for 5 145 children aged 5 to 11 y. After adjusting for the effects of other variables known to be associated with height, it was shown that there was a weak negative association between sleep duration and height. It is concluded that variation in sleep duration between children is unlikely to have an important influence on growth [Gulliford 1990].

43
Nutritional factors

Breastfeeding and growth in infancy (See 1.4.3, page 45)

Dietary factors and growth

Growth and nutrition are closely correlated. Nutritional biochemists have determined that there are 50 essential nutrients required for growth, maintenance, and repair of the body [Guthrie 1995]. The essence to grow and gain weight is the focus for a healthy situation, but since a few decades another focus emerged, namely the growing problem with overweight and obesity.

Nutritional deficiency as a cause of growth failure. Golden lines up what is the requirement of the chondrocyte, in particular, which will be crucial to longitudinal growth. They are likely to require following nutrients for their metabolic machinery: water, nitrogen, sulphur, phosphorus, threonine, lysine, potassium, sodium, magnesium, zinc, oxidisable substrate (energy), carbon skeletons of essential amino acid [Golden 1992].

Under-nutrition is the single most important cause of growth retardation worldwide, resulting from an adaptive response to decreased food availability [Nikens 1976, Torun 1988], however, nutritional dwarfing found in a paediatric endocrine practice in the United States usually does not involve the chronic growth problem of poverty-related malnutrition frequently observed throughout the world. Poor growth and delayed sexual development among suburban middle class adolescents is usually due to inadequate nutrient intake due to psychological causes [Lifshitz 1987, Lifshitz 1992]. Also, poor growth and inadequate nutrition have been seen in relation to organic problems such as chronic inflammatory bowel disease [Kelts 1979, Kirschner 1983, Hildebrand 1994]. Nutritional dwarfing patients usually show a deteriorating linear growth and delayed sexual development that is accompanied by inadequate weight gain. This pattern of growth is similar in patients with any of the different types of nutritional dwarfing syndromes including those of organic nature, such as seen in chronic inflammatory bowel disease [Kelts 1979, Kirschner 1983]. Catch up growth is usually achieved with nutritional rehabilitation [Kelts 1979, Kirschner 1983, Pugliese 1983].

Nutrition is a critical factor. The contents of nutrition are more important than the amount. Animal protein, fat, micronutrients, vitamins, essential fatty acids and amino-acids everything is important [Allen 1994], and to support this Tanner and Takahashi have connected the strong secular trend in height in Japan to the strongly increasing intake of milk since the end of World War II [Tanner 1982, Takahashi 1984, Takaishi 1994].

The milk hypothesis

To what extent can a specific food make an impact on human growth and development? There has been considerable debate in this question, and of all the foods studied so far, milk has generated the most important results.

Orr and Leighton & Clark gave schoolchildren, in several cities in Scotland, an extra pint of milk per day for seven months. Some children received whole milk and some skimmed milk. Both groups increased faster in height and weight than two
control groups of children of the same ages, one group given no supplement and the other given a supplement of biscuits, equalling the milk in total calories. Since the biscuits supplement had no effect on growth, it was concluded that some factor in milk, either whole or skimmed, accelerated growth [Orr 1928, Leighton 1929]. Several other studies afterwards show similar results [Spies 1959, Lampl 1978, Little 1983]. Takahashi made a strong case in favour of what may be called the "milk hypothesis", that posits that increased consumption of milk by infants, children and adolescents is directly related to greater average height of a population [Takahashi 1984]. He found an association between changes in dietary practices, especially milk consumption, and growth in Japan. From 1966 to 1976 the milk consumption rose from 55 to 100 grams per person and day. The height of schoolboys, aged 6 to 17 y, rose by an average of 4.1 cm between 1930 and 1960, a period of relatively great social and economic change, and by an average of 5.3 cm between 1960 and 1975. Although other factors (low rate of disease, reduced family size, society under development) may also have contributed to height increase, the milk was one of the most important reasons for the increase.

Migration

The relation between migration and growth was recognized early

The extent to which migrants differ from non-migrants in their physical characteristics, and the reasons for the differences found, have been the subject of numerous studies, beginning with Livi [Livi 1896] and Ammon [Ammon 1894], both cited by Boas [Boas 1922] and followed by the classical investigations of Boas [Boas 1912] and Shapiro [Shapiro 1939]. Livi and Ammon found, separately, that the children of urban migrants in Italy were taller than rural sedentes (the non-migrating rural population). Livi believed the cause was heterosis, and Ammon believed that the action of natural selection was the explanation. Boas found that neither natural selection nor heterosis could adequately account for changes in growth. Rather these were due to biological plasticity in the face of the new urban environments.

Japanese in Japan and USA

Shapiro’s Japanese migrant study compared the growth of Hawaiian-born of Japanese immigrant parentage, Japan-born Japanese who migrated to Hawaii, and Japanese sedentes living in the same villages from which the migrants originated. The sedentes and the recent immigrants differed in a few anthropometric measurements, some increasing and some decreasing with migration. The largest differences were between the immigrants and the Hawaiian-born. The latter were taller and more linear in body build than their parents or the sedentes. Shapiro argued that, with migration, there were improvements in diet, health care, and socio-economic status and that these conditions, associated with an urban lifestyle, were responsible for the growth changes. For example, Kobliansky and Arensburg have shown that migrants are non-random samples of the populations from which they depart. These authors, in their study of internal migration, conclude: "… an attempt was made to assess the relationship between population mobility and morphology. Samples of stationary urban and village national groups from Europe were compared to similar mobile populations. Statistically significant differences were observed between migrants and the original
stationary population, proving the process of selection. The higher values of length, width, weight stature, etc. are found generally in the mobile population.” [Kobliansky 1977]

Migration and growth are related – but the pattern can change

In fact, a relationship between migration and larger body size, especially greater stature, has been shown frequently [Shapiro 1939, Goldstein 1943, Lasker 1952, Lasker 1961] but also the reverse [Macbeth 1987]. The urban advantage in growth was not always present. Meredith [Meredith 1979], Malina et al. [Malina 1981], and Wolanski [Wolanski 1988] reviewed studies from the period 1870–1920 that showed that rural-living children in the United States and Europe were taller than their urban peers.

By 1930, this pattern was reversed and urban children were consistently taller and heavier than rural children. Other sources of data provide some ideas about changes in the rural and urban environment at the turn of the century [Eveleth 1990, Fogel 1986, Bogin 1988]. For example, prior to 1850, mortality rates for infants, children and adults were higher in the cities than in the rural areas of Europe and the United States. Around the year 1900 the trend reversed, due to improvements in sanitation, water treatment, and food preservation in the city. Urban children benefited from these improvements, as reflected in their greater stature and weight, and more rapid maturation, compared with rural children. All this shows that it is not the shape of the ecological exposure that is important, but the content. In many instances, the motivation for migration is either to improve the life circumstances of an individual or a family, or to maintain standards perceived as deteriorating in the original place of residence [White 1980]. Therefore, much migration is associated with expectations of improved health and nutritional status. Where these expectations are realized, and where migrants are studied at some time after their arrival in the new environment, plasticity rather than initial selectivity may well explain the larger dimensions of migrants [Susanne 1984].

There is certainly evidence in some studies of selectivity of migrants at point of departure with regard to socio-economic and/or demographic variables [Clarke 1984]. Biological selectivity of migrants can therefore be divided into that which is the direct result of some intrinsic propensity for mobility, and that which is the indirect result of an association between biological characteristics and other characteristics related to the decision to move [Macbeth 1984]. In practice these two components cannot easily be disentangled.

Biological selection?

Several studies provide tentative support for biological selection. The study of Steegmann of eighteenth century British military recruits, found that conscripts who migrated from their county of birth were significantly taller (169.1 cm) than recruits living in the county of their birth (167.6). One must remember, though, that he also found that urban-born recruits were shorter than rural-born men. Steegmann pointed out that eighteenth century Britain was a developing country and that urban areas were characterized by food shortages and unhygienic conditions. Thus migration to the city was probably not responsible for an increase in stature; rather taller men living in rural counties were more likely to migrate than shorter men. Whether these were genetically
taller men, or individuals who had experienced better living conditions prior to migration, was not known [Steegmann 1985].

Kaplan found that migration was selective for physical type. Growth differences between migrants and sedentes were found too soon after migration to be due to an environmental change [Kaplan 1954]. Illsley et al. studied those to migrated to and from Aberdeen, Scotland. They found that migrants were, on average, taller than rural or urban sedentes. Migrants had generally better health than sedentes. Migrant women had lower rates of low birthweight and perinatal death for their children. Finally, migrants were generally of higher socio-economic status than sedentes. This study suggests what the true meaning of migrant selection may be that it is more likely to be selection for socio-economic status than biological selection per se [Illsley 1963]. The study by Kobliansky and Arensberg [Kobliansky 1974] confirms this, and so does Mascie-Taylor [Mascie-Taylor 1984]. Boas’ thoughts on human growth response to migration were correct [Boas 1912].

**Greek children in Sweden**

Immigrant children from Greece was studied by Neiderud: They were all second generation immigrants, consisting of all healthy children born in Sweden by Greek parents in the catchment area of a single child welfare centre in Sweden in the southern Helsingborg. The children represented 36 families with 52 children who were born between 1974 and 1980. The parents had emigrated from a rural area in northern Greece in the late 1960s or early 1970s for economic reasons [Neiderud 1992]. Children born between 1974 and 1980 in the two villages of Terpillos and Dipotamos in the northern part of Greece were 66 from 48 families. The mean age for the immigration group was 5.6 years and for the Greek group 5.3 years. The immigrated group was significantly taller than the Greek group but had the identical height-for-age as the Swedish group.

**Refugee children, in Sweden, in the 1980s**

The health of newly resettled refugee children from Chile and the Middle East was studied by Hjern: 62 children from Chile and 43 children from Middle East were studied. They arrived in Sweden during a period of 13 months, from July 1986 until July 1987. They were measured at a first point as soon as possible and then subsequent measurements 5–7 months and 17–19 months after arrival in Sweden. Both refugee groups had a significant catch-up growth in height/age values from 6 to 18 months after arrival in Sweden. Chileans had a significant rise in weight/height values, a rise that was not seen in the Middle East group. After 18 months in Sweden 16 of the Chilean were obese. No child was obese in the Middle East group [Hjern 1990].

**New Americans**

A semi-longitudinal study on growth and development was initiated on immigrant and refugee school-aged children in San Francisco. Anthropometric values (height, weight among others) were collected soon after their arrival in the United States and repeated at 3-month intervals for 1 year. Comparisons for median growth rate indicated that most cohorts exhibited a median growth velocity that was close to, or exceeded, the median for US white children. There was also significant improvement in weight-for-
age. The results indicated that these immigrant and refugee children accelerated their growth markedly in an optimum nutritional environment and were in a period of catch-up growth [Schumacher 1987].

**Geographical, climatical and ecological factors**

**Population density and growth**

The relation of the height of primary schoolchildren to population density was studied by Foster. Shorter stature was found with increasing population density, and this relation remained after allowing for child’s birthweight, mother’s height, father’s height, social class, number of siblings, and additionally a measure of home overcrowding. The results are in contrast to those found elsewhere, and are unlikely to indicate a cause-and-effect relationship between population density and height. The findings cannot be explained either by known differences between urban and rural areas in social characteristics or by plausible hypothesis of genetic heterogeneity [Foster 1983].

**High altitude compared to low altitude**

More than 25 million people live in high altitude regions of the world; that is at altitude of 3 000 meters above sea level or higher. High altitude environments impose a number of stress factors on people, including hypoxia, high solar radiation, cold, low humidity, high winds, and rough terrain with severe limitations of agricultural productivity of the land. Of these, cold temperatures and hypoxia, the lack of sufficient oxygen delivery to the tissues of the body, has been considered to be the most important determinants of growth at high altitude. When statistically adjusted low-altitude infants had a birthweight of 3 415 g compared to high-altitude infants with birthweight of 3 133 g. Birthlength was 49.6 cm compared to 49.0 cm [Baker 1977]. This study and more recent research indicate that problems with the delivery of oxygen to the foetus are part of the reason for the growth delay. One classic study by Frisancho & Baker found that Peruvian children living above 3 000 meters are shorter and lighter, on average, than lowland Peruvian children of the same age [Frisancho 1970]. In contrast, Clegg et al. found that high-altitude-living Ethiopians were taller and heavier than ethnically similar people living at low altitude in Ethiopia. In this case, the lowland population suffered from malaria and intestinal parasites that may have affected their growth. Altitude, meaning hypoxia, was shown to be of secondary importance in this early study [Clegg 1972]. Similar findings are reported by other authors [Frisancho 1975, Mueller 1980]. How arctic populations grow is reported by Jamison [Jamison 1990].

**The sunlight in Stockholm and elsewhere**

Nylin published what was probably the first study of the sunlight effect on growth. He exposed one group of Stockholm boys (n=45) to “sunlamp” treatments (using a lamp that produced both visible and ultraviolet light) during the winter months and compared their growth to a group of boys (n=292) not receiving treatment. During the three-month period of treatment, the experimental group averaged 1.5 cm more growth in height than the control group. During the summer, the control group grew at a faster
rate than the experimental group, so that over the entire year there was no difference between the two groups in total height gain [Nylin 1929].

Two other studies lend support to an association between variation in sunlight and growth rate. Vincent and Dierickx found that healthy children living near the equator in Kinshasa (the Leopoldville), Zaire, grew more rapidly in height in the dry season than in the rainy season. Diet, temperature, humidity, and sunlight variation were considered as possible influences. No evidence in favour of the first three was found [Vincent 1960]. Seasonal variation in growth was also studied by Bogin. 246 healthy children, juveniles, and adolescents of high socio-economic status living in Guatemala City were followed during a 14-month period. About 75 percent of the children grew at a significantly faster rate during the dry season than during the rainy season. Conversely, about 25% grew at their fastest rate during the rainy season. This latter is perhaps an example of that no single exposure works isolated [Bogin 1978].

**Seasonal variation and month of birth**

The cause of seasonal variation in height growth rate is not completely understood; however investigators suggest that seasonal periodicity in sunlight may act on the human endocrine system. A relation between light and growth has been known since 1919, when it was shown that ultraviolet light could cure rickets, a disease of bone growth. Buffon could by scrutinizing the data from the son of Montbeillard conclude among other thing a seasonal variation in rate of growth; the boy grew faster in the summer than in the winter [Buffon 1777].

Seasonal variation in weight is explained by two categories of exposure. The first category includes population suffering from seasonal food shortages or disease as shown from Gambian studies [Billewicz 1967, Billewicz 1982]. The second category includes healthy, well-nourished populations, and in this case the causes are not so easily discerned. By scrutinizing 29 studies, Bogin [Bogin 1977] found that 22 showed that maximum increments in height and weight do not occur at the same time of the year. The authors of some of these studies speculate that children have a natural, endogenous rhythm for growth in weight that is independent of both the seasonal rhythm of growth in height and of seasonal variation in climate. That height is increasing more during the summer season is reported by many authors [Dahlberg 1931, Broman 1942, Bogin 1978, Gelander 1994], and so do weight [Wretlind 1878, Marschall 1971, Bogin 1978].

A month-of-birth effect on the height of both young people and adults has been reported in a few studies. The validity of this phenomenon is less well established and its causes are not clear. Henneberg and Louw made a cross-sectional study of 1 165 boys and girls, 6 to 18 y old, attending schools in Cape Town, South Africa. The participants in the study were all judged to be healthy and well-nourished. Those individuals born during the months of August to January were found to average about 0.7 cm taller and 800 g heavier than individuals born from February to July. The differences were statistically significant, though small [Henneberg 1990]. Henneberg and Louw made a later study, where they measured 1 522 boys and girls, 6 to 18 y old, living in the poorest region of the Cape Province of South Africa. Most of these participants showed evidence of chronic undernutrition. Even so, a significant month-of-birth effect was evident in the data but the timing was shifted. Individuals born during the months of November to April were taller and heavier than those born during May to October [Henneberg 1993].
Weber et al. discovered a seasonal variation in body height of 507,000 18-y-old Austrian conscripts related to their month of birth with a difference between the highest and lowest mean value of 0.6 cm with the highest value for the month of April [Weber 1998].

**Infections**

What all trained paediatricians recognize is the relation between weak growth and infections. Cole has studied the phenomenon for children younger than 2 years of age. Diarrhoea, infections in lower respiratory tract and measles all have a strong effect on weight gain and reduced weight between 17 and 67 g/day [Cole 1989].

Infections are important in the way they affect and interact with nutrition. Infection can result in gastro-intestinal influence and malabsorption as a result. An infection can also result in more infections by affecting the immune defence [Waterlow 1994].

**The impact of diseases**

Disease acts in the range from stopping growth or severely decreasing growth velocity to having no impact at all on growth. Some examples are given here.

Final height was reduced only in children with Crohn’s disease. Height velocity was significantly decreased, between 6.0 years of age and the onset of puberty in children with ulcerative colitis. In children with Crohn’s disease height velocity was significantly decreased from 3 years to the end of pubertal period. Peak height velocity was assessable in 112 children and it was on average delayed, especially for children with CD [Hildebrand 1994].

Coeliacs with gastrointestinal symptoms treated before adulthood reach a mean height similar to that of the normal population, and their final height is slightly better (3 cm) than that of coeliacs who have not received treatment. This difference appears to occur only in males and might be due to a more rapid evolution of puberty in girls on a gluten free diet [Caacciari 1991]. Growth retardation is a major complication of chronic renal insufficiency in children [Rodriguez-Soriano 1991]. Respiratory conditions, including asthma, and height in primary school are studied in the UK and the impact is presented [Somerville 1993].

**Diurnal variation**

Diurnal variation of standing height results in higher values in the morning and lower values in the evening [Ashizawa 1990, Lampl 1992]. This is important to recognize when measuring children in order to survey them.

1.4.3 **Weight from birth to adulthood**

Trying to conclude the cause of the epidemic of obesity Skelton says: When people ask: ”What is causing this epidemic – genes, diet, exercise, TV, fast food?” the answer is ”Yes – all of them!” The complex interplay of genetic predispositions in an environment geared to making us to eat more and exercise less has led to our recent crisis [Skelton 2004]. Long time ago, before this secular trend started Börjeson told us:
"Under normal conditions, obesity arises almost only when there is a genetic disposition." [Börjeson 1976]

Nature

It is widely acknowledged that genetic factors play a role in obesity, and that fatness is, to some extent ‘heritable’. This topic has formed the subject of several reviews in the last decade [Maes 1997]. The influence of heredity on obesity has been demonstrated in family studies [Court 1977, Merritt 1982]. Especially those by Mossberg [Mossberg 1989, DiPietro 1994] by Shields [Shields 1986] and by Börjeson [Börjeson 1976] and Stunkard et al. [Stunkard 1990] on twins and by Stunkard and Sörensen [Stunkard 1986] by adopted children. From a study in 1999 Stunkard concluded: The result suggest that genetic influences on the body weight of infants may be independent of those that influence BMI in adults, a circumstance that could complicate the search for genetic determinants of obesity [Stunkard 1999].

Data from Kaplowitz et al. suggested that this relationship may be stronger between mothers and their offspring than fathers and offspring, and that the mother-offspring relationship strengthens over time [Kaplowitz 1988]. Whitaker et al. found that parental obesity was a more important predictor of offspring obesity earlier in childhood (<6 y), becoming less important with increasing age of the child [Whitaker 1997]. Data from Lake et al. showed that parental obesity influences tracking if both parents are obese [Lake 1997].


There would seem to be little doubt that the development of fatness has a genetic component. What is far less clear, and is important from a public health perspective, is how much of the variation in fatness is due to the genetic component, and how much to the environment. Also important is the influence of the environment on the genetic component, i.e. gene-environment interactions. But if an individual is susceptible to being fat, the type of exposition factors and the power of them will decide what the result will be.

Nutritional factors

Breastfeeding and growth

More than 25 years ago, Waterlow and Thomson speculated that “growth faltering” around the age of 3 months of breastfed infants was reflecting inadequate energy supply [Waterlow 1979] and so did Duncan et al. [Duncan 1984]. Now we know that this problem, which seemed to be obvious, was not real, and mostly dependent on that an improper norm was used (the old NCHS standard from the 1920s). Among the earliest to address this issue was the Cambridge group that found that though breastfed
Gambian infants were smaller than Cambridge infants, the weight gain patterns by the two groups were remarkable identical expressed as percent of the reference value for weight-for-age. This obvious "faltering" did reflect a tendency of breastfed infants to show an accelerating growth in early months of life. In other words: the pattern described in the international reference and in many other curves did not equal reflect the growth of breastfed infants in industrialized and developing countries [Whitehead 1984]. This discovery has been made by successors [Dewey 1995], who pointed out the necessity of a new reference for infants [de Onis 1997, de Onis 2003]. 1999 a proposal of a new reference was made from Garza [Garza 1999] and from Williams [Williams 2002].

An example is given by Cole. Out of 252 infants were 63 bottle-fed, 69 breast-fed/bottle-fed and 120 breast-fed only for more than 24 weeks. 120 infants that were breast-fed more than 24 weeks, with solid food introduced at an average age of 15 weeks, were followed from birth, being weighed every 4 weeks up to 1 year of age. Those with long breastfeeding were somewhat heavier than the British reference [Freeman 1995, Cole 1998] and crossed the centiles upwards in order to reach +0.3 SDS at age 2 mo but afterwards crossed the centiles downwards -0.2 SDS at age 12 mo [Cole 2002].

The literature on the relationship between early infant feeding and growth shows that after the first 3 or 4 months, breast-fed infants in the developed world are lighter than formula-fed infants with markedly lower adiposity. There is some evidence of a slightly lower rate of linear growth over the first year or so. These differences in weight and length do not appear to persist beyond the first years of life. In the developing world the situation is very different. The growth curves of breast-fed infants of malnourished mothers may falter between the third and sixth month of life. However, the generally poor quality of the supplementary foods offered in the developing world, and the increased risk of diarrhoeal infections, mean that supplementary feeding before the age of 6 months is unlikely to lead to a growth advantage and may well instead lead to growth faltering [Rogers 1997].

Scholtens recently stated that compared with non-breast-fed children, breast-fed children tend to have a lower BMI at about 1 y of age. The association between breastfeeding and BMI between 1 and 7 y of age was negligible, while a high BMI at 1 y of age was strongly associated with high BMI between 1 and 7 y of age.

**Physical activity and inactivity**

"Normal" physical activity is by definition desirable. But an amount of physical activity can be counteracted by an amount of physical inactivity. Physical inactivity could be described as the time spent watching television or the time spent in front of a computer at home [Dietz 1985, Robinson 1993, Gortmaker 1996, Robinson 1996].

For 8- and 13-y-olds in the early 1980s in Sweden Sunnegårdh concluded: In spite of increased sports involvement in Swedish children over the years, a reduction of physical activity was indicated by a tendency towards a higher body fat content despite a lower mean energy intake as compared with such values obtained 10–15 years ago in Swedish children of equal ages [Sunnegårdh 1986]. By studying 2 200 9-y-old boys and girls, Harrell tells us: Children from a higher socio-economic status (SES), especially boys, reported a greater proportion of sedentary activities than lower SES children. Significantly more nonobese than obese children were reported a vigorous (high-intensity) activity as one of their top three activities [Harrell 1997].
There is a relationship between physical inactivity and adiposity in pre-pubertal boys Maffeis has found [Maffeis 1997].

The activity for 52 children, aged 4 to 8 y, was measured during the first 3 days of life, and in this survey, neonatal adiposity was not significantly correlated with parental adiposity, neonatal physical activity, or gender, nor was neonatal activity significantly correlated with adiposity in childhood. Neonatal adiposity did not predict adiposity in childhood. However, in a stepwise multiple regressions, parental adiposity and the children daytime high activity levels were significantly associated with childhood adiposity. The age or gender of the child did not significantly correlate with childhood adiposity. As parental adiposity increased or daytime high activity of a child decreased, the adiposity in a 4- to 8-y-old child was likely to increase [Berkowitz 1985].

Westerståhl et al. compared 16-y-olds in 1974 and 1995 and noticed that although more adolescents participated in leisure-time sports activity in 1995 than in 1974 the BMI increased from 1974 to 1995. One explanation could be that the lifestyle of adolescents between sports training sessions may have become more sedentary [Westerståhl 2003, Westerståhl 2003]. This could fit the finding among conscripts by Rasmussen et al., that among 18-year-old men the muscle-strength has increased at the same time that BMI increased over time [Rasmussen 1999].

**Psychosocial factors**

The classical overview of body size, of infants and children, around the world, in relation to socio-economic status, was made by Meredith [Meredith 1984]. The relation was convincingly shown. This relation is obvious both in developing countries [Arteaga 1982] as in developed [Rolland-Cachera 1986]. In Sweden Mellbin has shown that psychosocial stress and psychosocial problems are related to an increase in relative weight in schoolchildren [Mellbin 1989, Mellbin 1989].

Socio-economic status has a direct influence on weight status in developing countries. Low-weight subjects are encountered in low-income families, and obesity is more frequent in high-income families [Arteaga 1982]. By contrast, in countries with abundant food supplies, weight status and economic status is inversely related [Arteaga 1982], and a lower prevalence of obesity is recorded in higher social classes. The same inverse association is recorded in the study of 18-y-old males in Sweden [Oldenburg 1999]. A large number of lifestyle characteristics, such as eating habits and physical activity, may account for differences between socio-economic groups.

**SES and obesity – not a simple relation**

In a review, Parson showed that the relation between obesity and SES was not clear [Parson 1999].

Sobal and Stunkard have published a comprehensive review about SES and obesity, which includes 144 studies from all over the world, of men, women and/or children [Sobal 1989]. Despite the fact that these studies used a range measure of SES and fatness, and spanned over a period up to 45 y, a strong negative relationship between SES and fatness was obvious in women across the developed world, such as that fatness increased from higher social groups (professional and management) to lower social groups (unskilled and manual). Of 54 studies, 85% demonstrated a negative relationship, 13% showed no relationship, and only 2% a positive relationship.
(greater fatness among higher social groups). Among men and children, however, the relationship was far less consistent. In stark contrast, in developing countries, not a single study found a negative relationship between SES and fatness in women. Indeed, the majority reported among men and women, and in children.

Since 1995, children have been included in the Health Survey. Combined data from 1995 show no pattern in social class and prevalence of overweight in males aged 2–15 y or 16–24 y or females aged 2–15 y. In females aged 16–24 y, prevalence of obesity was higher in manual and non-manual social classes.

The current systematic review examines evidence that low SES in childhood promotes the development of fatness later in life.


The influence of social factors other than social class, such as family size, number of parents at home and child-care, has not been well researched.

Breastfeeding as a protective factor against obesity and a promoting factor for stature

Compared to bottle-fed infants, breast-fed infants in the 1920s and 1930s were taller in childhood and adulthood. As stature is associated with health and life expectancy, the possible long term impact of infant feeding on adult mortality patterns merits further investigation [Martin 2002].

Agras found that breastfeeding only protects against early adiposity to the age of 6 months [Agras 1987] and later he found that predictive factors at 6 years of age were adiposity at birth, with greater adiposity at birth predicting greater fatness at 6 years, parental education, with less education associated with fatness, and a prolonged period of breastfeeding with delayed introduction of solid food [Agras 1990].

Breastfeeding is protecting for obesity at age 6 y [Gunnarsdottir 2003] and up through adolescence [Elliot 1997, Gillman 2001, von Kries 1999] and even up to adulthood that is studied within the Boyd-Orr cohort in the US [Bray 2006, Martin 2002].

The protective effect of breastfeeding on future overweight seems to be explained only partially by decreased maternal feeding restriction [Taveras 2006].

Parsons summarizes the literature

Parsons summarize a review of the literature 1999 as follows:

* Risk factors for obesity included parental fatness, social factors, birthweight, timing or rate of maturation, physical activity, dietary factors and other behavioural or psychological factors.
* Offspring of obese parent(s) were consistently seen to be at risk of fatness, although few studies have looked at this relationship over longer periods of childhood and into adulthood. The relative contributions of genes and inherited lifestyle factors to the parent–child fatness remain largely unknown.

* No clear relationship is reported between socio-economic status (SES) in early life and childhood fatness. However, a strong consistent relationship is observed between low SES in early life and increased fatness in adulthood. Studies investigating SES were generally large but very few considered confounding by parental fatness. Women who change social class (social mobility) show the prevalence of obesity of the class they join, an association that is not present in men. The influence of other social factors such as family size, number of parents at home and child care has been little researched.

* There is good evidence from large and reasonable long-term studies for an apparently clear relationship for increased fatness with higher birthweight, but in studies that attempted to address potential confounding by gestational age, parental fatness, or social group, the relationship was less consistent.

* The relationship between earlier maturation and greater subsequent fatness was investigated in predominantly smaller, but also a few large studies. Again, this relationship appeared to be consistent, but in general, the studies had not investigated whether there was confounding by other factors, including parental fatness, SES, earlier fatness in childhood, or dietary or activity behaviours.

* Studies investigating the role of diet or activity were generally small, and included diverse methods of risk factor measurement. There was almost no evidence of an influence of activity in childhood on later fatness. No clear evidence for an effect of infant feeding on later fatness emerged, but follow-up to adulthood was rare, with only one study measuring fatness after 7 y. Studies investigating diet in childhood were limited and inconclusive. Again, confounding variables were seldom accounted for.

* A few, diverse studies investigated associations between behaviour or psychological factors and fatness, but mechanisms through which energy balance might be influenced were rarely addressed.

The authors, after scrutinizing all the relevant studies, concluded: Many of the risk factors investigated are related, and may operate on the same causal pathways. Inherent problems in defining and measuring these risk factors make controlling for confounding, and attempts to disentangle relationships more difficult. A given risk factor may modify the effect of another, and cumulative effects on the development of obesity are likely, both over time for specific risks, or at any particular time over a range of risk factors. An additional approach to addressing these issues may be to use large samples on which more basic measures of risk factors have been collected. [Parsons 1999].
Prognosis of obesity in infancy and childhood

Studies examining individual fatness development also suggest an early origin of adult obesity [Rolland-Cachera 1984, Prokopec 1993, Whitaker 1997]. Children have a rapid increase in BMI during the first year of life. The BMI subsequently declines and reaches a minimum by the age of 6 y, before beginning of a gradual increase up to the end of growth. The point of minimal BMI has been called the adiposity rebound [Rolland-Cachera 1984, Dietz 1994]. The age at adiposity rebound is associated with BMI development. An early adiposity rebound is associated with an increased risk of adult obesity [Rolland-Cachera 1984, Prokopec 1993], and this was found to be independent of both parental obesity and the child’s BMI at adiposity rebound [Whitaker 1997].

A longitudinal study of nutrition and growth has investigated the early determinants of the age at adiposity [Rolland-Cachera 1995]. The only significant association between nutritional intake at the age of 2 y and age at adiposity rebound was a high percentage of energy provided by protein in the diet, i.e. the higher the percentage of protein, the earlier the adiposity rebound. By the age of 1 y, the infant diet is characterized by a high intake of proteins and a low intake of fat [Roland-Cachera 1999]. To compare with the content of human milk where 7% of the energy is protein and 50% is fat. Childhood obesity is characterized by an increased stature and muscle mass [Valoski 1990].

450 children at age 6 months were followed to the age of 9 years. Weight gain in infancy is also a poor predictor of 9-y-old obesity. Changes from obese to non-obese or lean are often not linear. There is evidence that impending or actual obesity begins at ages 6 to 9 y with some predictability provided as early as age 2 y for girls, age 3 y for boys [Shapiro 1984].

226 children were followed from the first year of life to age 4 y. 23 children were considered obese at some point during the first year of life but only 4 at age 4 y. Only 3 of 23 of the obese children during the first year were still obese at age 4 y. The weight and length of the children obese at 0–1 y of age were significantly increased at age 4 y. Over-nutrition occurred during the first year in 26 infants and the number of obese infants in this group was significantly increased at age 7–12 mo and of overweight children during the first two years of life. At age 4 y, however, none of them were either obese or overweight [Sveger 1978].

27 899 infants were followed from birth to age 7 y and a pattern of rapid weight gain during the first 4 months of life was associated with an increased risk of overweight status at age 7 years, independent of birthweight and weight attained at age 1 year [Stettler 2002].

In a 40-year follow up of 500 overweight children Mossberg found that the degree of obesity in the family and the degree of overweight in puberty were the most important predictors of adult body overweight. Factors of minor importance were birthweight of more than 4 500 g and the age of onset of obesity. Whether an obese infant will stay obese depends very much on the degree of obesity in the family, especially in the mother [Mossberg 1989].

A review of the literature about whether obese children become obese adults you find in Serdula et al. [Serdula 1993, Serdula 1993] and also by Sörensen [Sörensen 1988]. The proportion remaining obese in adulthood is reported to be between 40% and 85% [Lloyd 1961, Court 1977, Taitz 1983].
Dealing with the question whether chubby infants become obese adults and coming to somewhat different results, see further [Charney 1976, Börjeson 1976, Rolland-Cachera 1989, Rolland-Cachera 1990, Valdez 1996].

A large national cohort of children studied from birth to 36 y was used to test the predictive value of childhood obesity for obesity in adult life. Only 21% of obese 36-y-olds had been obese at age 11 y, and even when associated social factors were taken into account the correctly predicted percentage was much lower than the prediction rate achieved using body mass data from age 26 y. At the age of 36 y, 3 754 of the 5 362 members of the study population were contacted, and out of these, 3 322 were successfully interviewed in their homes by specially trained nurses [Braddon 1986].

Early growth and abdominal fatness in adult life was studied by Law. 845 men born between 1920 and 1930, and 239 men born between 1935 and 1943 were followed-up. The tendency to store fat abdominally, which is known to increase the risk of cardiovascular disease and diabetes independently of obesity, may be a persisting response to adverse conditions and growth failure in fetal life and infancy [Law 1992].

Overweight children tend to be taller, have advanced bone age, and mature earlier than non-overweight children, because height gain accelerates or follows shortly upon excessive weight gain [Dietz 1998], but this do not result in higher final height but just final height at an earlier age.

**Weight loss**

*Is the rate of eating disorders increasing, or not?*

Obesity and overweight among children and adolescents are becoming increasingly prevalent around the world [Lobstein 2004], and increased rates of eating disorders are reported for example in the United States and Sweden [Lucas 1994, Edlund 1994, Edlund 1999]. However, in a review of the existing literature, Fombonne found no epidemiologic evidence for an increasing frequency of eating disorders or anorexia [Fombonne 1995, Fombonne 1996]. Fombonne concluded: ”Although based on a limited number of studies, the empirical evidence does not support secular changes in the incidence of bulimic disorders. In keeping with this conservative conclusion, it is noted that high rates of dieting and body dissatisfaction were already reported 30 years ago among adolescent females.” [Fombonne 1996] On the other hand, more recent studies conclude that the rate of eating disorders really is increasing for example in Japan, the United States and in Europe [Nakayama 2001, Kohn 2001, Morande 1999, Martin 1999, Bulik 2006, Steiner 1998].

In Sweden, the risk for anorexia is four times higher among women than men for individuals born between 1935 and 1958 [Bulik 2006]. The prevalence of anorexia defined by DSM-IV [First 1997] criteria is 1.2% for females and 0.3% for males in this population. The rate is higher among those born after 1945 [Bulik 2006].

**Weight concerns and weight loss practices among schoolchildren**

When asking schoolchildren about weight concerns, Edlund et al. [Edlund 1994, Edlund 1999] found that the rate of weight concerns is increasing and that these
concerns are beginning at an earlier age among Swedish children today. Among adolescent girls, 60–70% reported having been on a diet during the previous year [Hill 1993]. Also, dieting behaviour has been reported among preadolescent females [Edlund 1996, Gustavsson-Larsson 1992] and is increasing with age [Edlund 1999].

To estimate the rate of various weight loss practices in U.S. adolescents Serdula et al. [Serdula 1993] surveyed 11 467 high school students in 1989. They found that 44% of the females and 15% of the males reported that they were trying to lose weight. From the same investigation Felts et al. [Felts 1996] concluded that one-quarter of U.S. high school students perceived themselves to be overweight; three-quarters of these students are trying to lose weight. French et al. [French 1995] reported from the U.S. that the frequency of dieting, weight change history, and specific weight loss behaviours in a population-based sample of 1 015 female 9th–12th graders was caused by healthy weight loss behaviours more often than unhealthy weight loss behaviours (healthy behaviours such as exercise, 32.4%; decrease in fat intake, 26.0%; reduction in snack intake, 25.0%; and reduction in kilocalorie intake, 22.4%; and unhealthy behaviours such as fasting, 8.1%; diet pills, 5.4%; and vomiting, 4.4%). Obesity status and restrained eating scores were positively related to more weight loss episodes, pounds lost, and weight fluctuations and to increased use of healthy weight loss methods and weight loss programs.

In Australia Nowak et al. [Nowak 1996] surveyed 791 8th graders from private schools in north Queensland. Only 41% of the girls and 54% of the boys were satisfied with their weight; 52% of the girls and 27% of the boys wanted to lose weight. When surveyed, 35% of the girls and 22% of the boys were trying to lose weight. One of the conclusions from this study is that the drive to lose weight is already apparent among 12–14-y-old adolescents. In another Australian study, McCabe et al. [McCabe 2001] investigated 1 266 adolescents (622 boys, 644 girls) with respect to their body image and body change strategies, as well as the socio-cultural influences on these variables. Girls were found to be less satisfied with their bodies and were more likely to adopt strategies to lose weight, whereas boys were more likely to adopt strategies to increase weight and muscle tone. Respondents with higher body mass index (BMI) had greater body dissatisfaction and more weight loss strategies, but there were no differences between BMI groups in weight gain or strategies to increase muscles. The influences of the media on the desire to alter weight, as well as feedback from parents and both male and female peers, were stronger for girls.

All these surveys deal with questionnaires to adolescents about weight concerns or weight loss practices. They all show that these phenomena are frequent, especially among girls. But, to the best of our knowledge, there are no surveys that longitudinally have collected data of recorded weight loss. Furthermore, we have not found comparable surveys that could catch the factual weight loss situation over time.

**Body image among adolescents**

Bergström et al. [Bergström 2000] report that in Sweden today, many adolescents, especially girls, over-evaluate their body size. Another point of view comes from Kaltiala-Heino et al. [Kaltiala-Heino 2003]. On the basis of surveys that cover a 20-year period in Finland they state: "Even if the adolescent population has gained weight, they are less concerned at being overweight than earlier. It seems that adolescents compare themselves rather to the peers close to them than to ideal models
provided by culture at large”. However, most studies report a stronger desire to be thinner among those with a high BMI [Patton 1990, Hill 1995, Striegel-Moore 1995].

Anthropometry, body composition and body image in dieting and non-dieting 8–16-y-old Swedish girls was studied by Edlund et al.: The subjects participated in a 3 y prospective longitudinal study and were selected randomly after stratification for grades from those scoring in the upper vs. the lower thirds of the Children’s Eating Attitudes Test (ChEAT) score distribution. The ChEAT was completed 6 months before the present study, together with a demographic and dieting questionnaire and a questionnaire for the estimation of body size. In total 43% (n=52) admitted ever dieting (“Dieters”) and 25% (n=30) admitted that they were currently trying to lose weight. The anthropometric and body composition data indicated that ChEAT High-scores and Dieters were somewhat fatter than Low-scorers and Non-dieters, although this pattern was not shown among the 8-y-olds or the 14-y-olds (High-scorers). The mothers of the ChEAT High-scorers were found to be somewhat fatter than the other mothers. A current vs ideal body shape discrepancy was shown for both High-scorers and Dieters, with a larger discrepancy for the Dieters. All groups believed that their parents were aspiring for a leaner body [Edlund 1999].

81 infants were investigated to evaluate the clinical significance of low rate of weight gain. An organic aetiology was found in 28 infants. The remaining infants were analysed with special respect to their psychosocial environment. Growth was assessed by rate of weight gain. Thirty-four infants had subnormal rates. Comparisons were made with 18 infants with low but normal rates and a control group of 72 infants. Several psychosocial risk factors were over-represented in infants with subnormal rate of weight gain such as employment, bad health in fathers, dependence on social welfare etc. No significant differences were found in perinatal factors except for birthweight. The magnitude of the load of adverse factors within the family was measured as a score. A significant negative correlation was found between the score obtained and the rate of weight gain [Kristiansson 1981].

Weights were retrieved from child health records for an annual cohort of 3 418 children, aged 18–30 months, to explore the relationship between deprivation and weight gain. Children from deprived areas were smaller at all ages with a widening gap: by 1 year of age, they were three times as likely as affluent children to be below the third centile for weight [Wright 1994].

**Catch-up growth**

Catch-up growth was introduced as a term by Prader [Prader 1963]. Catch-up growth may be defined as a height velocity above the statistical limits of normality for age and/or maturity during a defined period of time, following a transient period of growth inhibition. One of the first papers in paediatric literature on catch-up growth was published by Bauer [Bauer 1954] where he described the growth pattern of 34 children with nephritic syndrome.

The variability of catch-up growth, not only among suffering from diseases but also among individuals with the same disorder, suggests that influences on catch-up growth are multifactorial [Boersma 1997].
1.4.4 Longitudinal aspects of growth and later health

Every point on the curve of development of an individual contains the possibilities for different development depending on what exposure that exists. If there is, on one point, a decelerated situation, and not too strong impact, then a positive exposure facilitates a catch-up. If the development has been optimal, a positive exposure just maintains that positive development. A point or a period with retardation may lead to an increased susceptibility and with unfavourable exposure in the future lead to disease and premature mortality. More knowledge about this longitudinally relations has been reported the last 15 years.

Poor socio-economic circumstances in early life may lead to biological vulnerability in later life [Wadworth 1997], and adult health behaviours seem to have socio-economic roots early in life [Lynch 1997].

During the last 15 years, however, a considerable body of evidence has accumulated, suggesting that height at maturity is also an important predictor of the probability of dying and of developing chronic diseases at middle and late ages [Waaler 1984, Marmot 1984, John 1988, Forsen 1999]. BMI has similar predictive properties [Heywood 1983, Waaler 1984, Martorell 1985, Payne 1992, Osmani 1992, Srinivasan 1992].

Birthweight and childhood growth and their relations to later health

Given the importance of birthweight for infant, childhood, and adult health [Barker 1992], 150–200 g social gradient in mean birthweight and 30% of births less than 2 500 g attributable to social inequalities [Spencer 1999] is a key public health issue. Reductions in inequalities in infant mortality and many childhood and adult health inequalities, key government health targets are unlikely to be achieved without a narrowing of the social gradient in birthweight.

Paediatricians have long been familiar with the increased risk of mortality and early morbidity of babies born very small or very early. These babies have a greater risk of dying throughout the first year of life [Murphy 1992, Bernstein 2000].

In the last 20 years or so, there has been increasing evidence that size at birth is also associated with later health, particularly with chronic degenerative diseases that are major causes of death in middle and later life. The best documented relations are those between smaller size at birth and higher death rates from coronary heart disease and stroke [Rich-Edwards 1997, Osmond 1993, Stein 1996, Leon 1998, Eriksson 2001]. Smaller size at birth is also related to increased levels of cardiovascular risk factors such as hypertension [Huxley 2000, Curhan 1996 women, Curhan 1996, men, Pharoah 1998], type II diabetes mellitus [Phillips 1998], and hyperlipidemia [Barker 1993]. High birthweight is normally associated with long-term health. However, people with high birthweight have higher death rates from prostate cancer [Ekbom 1996] and possibly breast cancer [Vatten 1996]. They may also be at risk of obesity [Barker 1997, Parsons 1999] but some doubt about this [Allison 1995] and type II diabetes mellitus [Dabelea 1999, Rich-Edwards 1999].

By reviewing the literature since 1956 and 40 years forwards, Law et al. concluded that blood pressure is inversely related to birthweight in children and adults.
The positive results in neonates and the inconsistency in adolescence may be related to the unusual growth dynamics during these phases of growth [Law 1996].

Other reports also show that growth and living conditions in childhood and hypertension and cardio-vascular disease in adult life are related [Arnesen 1985, Blane 1996, Forsen 1999, Barker 2002].

By following 5 914 infants born in 1982 from birth to age 18 y, Victora et al. concluded that height was primarily determined by fetal and infant growth. Weight-related indices, including the fat/lean mass ratio, were more strongly influenced by later growth. No clear critical windows of growth during which absolute tissue masses are programmed could be identified [Victora 2006].

By examining all children born from 1977 to 2004 in Denmark (N=1.7 million) Hviid and Melbye found that birth weight was inversely associated with a risk of infectious disease hospitalization; among children aged 0–14 y, the risk of hospitalization increased 9% for each 500 g-reduction in birth weight (increase rate ratio = 1.09, 95% confidence interval: 1.09, 1.11). The effect was found to peak in infancy and to persist until 10 years of age. It was present also in children born at term (37–41 weeks of gestation) [Hviid 2006].

However, Barker’s conclusion that foetal origin of adult disease is a clear fact has been questioned by Lucas [Lucas 1999].

**Final height and its relation to mortality and morbidity**

1993 Robert Fogel stated: "Extensive clinical and epidemiological studies over the past two decades have shown that height at given ages, weight at given ages, and weight-for-height (BMI) are effective predictors of the risk of morbidity and mortality. Until recently, most of the studies have focused on children under age 5 y, using one or more of the anthropometric indicators at these ages to assess risks of morbidity and mortality in early childhood, and it was at these ages that the relevance of anthropometric measures was established most firmly." [Fogel 1993] Many examples of this exist [Sommer 1975, Chen 1980, Billewicz 1982, Kielmann 1983, Martorell 1985].

13 146 persons whose weights and heights were measured between the ages of 15 and 18 y in Hagerstown, Maryland, during the period 1933–1945 were studied. The associations between growth velocities or attained height with mortality tended to be inversely, although not statistically significant. These results are compatible with the existence of positive associations of overweight in school-age children with long-term morbidity and seem to allay fears that harm could come from increased growth rates in childhood. Without jeopardizing growth, the avoidance of overweight in childhood might reduce mortality in the middle age [Nieto 1992].

A study of 3 332 men and women born in 1946 investigated the relation between blood pressure at age 36, and birthweight and BMI in childhood, adolescence and adulthood. Low birthweight and high BMI at age 36 y were independently related to high blood pressure. A reduction in the percentage of low birth-weight babies born in the fourth decade of this century would only have a negligible effect on the incidence of adult hypertension 30–40 years later [Holland 1993]. Cardiovascular disease and diabetes have been shown to be associated with retarded growth during fetal life and infancy [Sveger 1978, Barker 1992].

Within the Nurses’ Health Study, where a prospective cohort of 121 700 US female nurses aged 30–55 in 1976 was examined after a 14 years follow-up and the
conclusion was: The result support the hypothesis that height is inversely related to the risk of coronary heart disease in women [Rich-Edwards 1995].

Observational evidence points towards positive associations of height with risk of some cancers (e.g. prostate, breast, and colon) [Gunnell 2001], and inverse associations with insulin resistance and coronary heart disease [Williams 1997, Davey Smith 2001].

The Norwegian experience about height and mortality

It must be stressed that we are talking about averages, not individuals, but there are clear links between height and health. In Norway, a survey by Hans Waaler showed that tall men and women live significantly longer. For example, women aged 40–44 y, those standing between 145 cm and 149 cm, had a mortality rate double that of women 165 cm to 169 cm tall. Norwegian men aged 55 to 59, who stood 150 cm to 155 cm, had mortality rate twice that of men whose height was 185 cm to 189 cm [Waaler 1984]. See Jousilathi [Jousilathi 2000].

Fogel reproduces a diagram by Waaler and shows that short Norwegian men between 1963 and 1979, aged 40–59 y (by establishing the predictive power of height and BMI with respect to morbidity and mortality) were much more likely to die than tall men. Indeed, the risk of mortality for men with heights of 165 cm was on average 71% greater than that of men who measure 182.5 cm. The Norwegian curve has relative risk that reach a minimum of between 0.6 and 0.7 at a height of about 187.5 cm and a relative risk of about 2 at about 152.5 cm. By comparing this to another result of a survey of US soldiers Fogel concludes: the relative risk of morbidity and mortality depends not on the deviation of height from the current mean but from an ideal mean: the mean associated with full genetic potential [Fogel 1993].

Waaler has also studied the relationship in Norway between BMI and the risk of dying in a sample of 1.7 million individuals. Although the observed values of the BMI ranged between 17 and 39, over 80% of the males at age 40 and older had BMIs within the range 21–29. Within the range 22–28, the curve is fairly flat, with the relative risk of mortality hovering close to 1.0. However, at BMIs of less than 22 and over 28, the risk of dying increases quite sharply as the BMI moves away from its mean value. The BMI curves are much more symmetrical than the height curves indicating that high BMIs are as risky as low ones. The Norwegian survey shows that an adult male with a BMI of 25 who is 164 cm tall runs about 55 percent greater risk of dying than a male who is 183 cm tall and also has a BMI of 25. A BMI of 25 is "ideal" for men in the neighbourhood of 175 cm, but for tall men (greater than 183 cm) the ideal BMI is between 22 and 24, while for short men (under 168 cm) the "ideal" BMI is about 26.

1.5 GROWTH SURVEYS, GEOGRAPHICAL COVERAGE AND DESIGN

It is often unclear if surveys of growth in different countries have a national representative purpose or not. "Reference values" are created and the question of whether it is a description of a local population, and to the best ambition, in spite of that, representative for a country, is seldom declared. It is with this distinction between
surveys based on local populations and based on some type of national representative sample that the following overview shall be read. Longitudinal surveys are very rare.

### 1.5.1 Growth surveys in Sweden

From the first surveys of children’s growth up until today, different attempts have been made to describe the national growth-situation of children and adolescents. Surprisingly, the ambition was highest in the early studies. Key, for example, performed in 1883 an impressing data collection, from a national point of view, with a discussion about the survey’s weakness and what is desirable in a future of geographical and social coverage in order to get a national representative material [Key 1885]. But later, in 1942, Broman et al. are doing something in the lines af whet Key wanted [Broman 1942]. Anyhow, it is not until a study by Lindgren et al. that a national representative sample of data was collected, for children born in 1967, with height and weight data from school health records, longitudinally, from age 7 y to age 16 y [Lindgren 1986].

Table 5. Surveys of growth in Sweden

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Ages</th>
<th>Birth years</th>
<th>Measures</th>
<th>Location and remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wretlind 1878</td>
<td>38</td>
<td>7–16y</td>
<td>1863–67</td>
<td>W</td>
<td>Göteborg, girls</td>
</tr>
<tr>
<td>Petterson 1882</td>
<td>1 675</td>
<td>0–1y</td>
<td>1860s</td>
<td>W</td>
<td>Uppsala, born at hospital</td>
</tr>
<tr>
<td>Key 1885</td>
<td>18 019</td>
<td>7–21y</td>
<td>1862–76</td>
<td>H,W</td>
<td>Stockholm + other places</td>
</tr>
<tr>
<td>Dovertie 1895</td>
<td>485</td>
<td>8–15y</td>
<td>1880–87</td>
<td>H,W</td>
<td>Kristianstad</td>
</tr>
<tr>
<td>Hulcrantz 1896</td>
<td>1 896</td>
<td>21y</td>
<td>1875</td>
<td>H</td>
<td>All of Sweden, conscripts</td>
</tr>
<tr>
<td>Steenhoff 1900</td>
<td>1 331</td>
<td>7–15y</td>
<td>1885–92</td>
<td>H,W</td>
<td>Sundsvall</td>
</tr>
<tr>
<td>Norinder 1907</td>
<td>422</td>
<td>8–15y</td>
<td>1891–98</td>
<td>H,W</td>
<td>Hjortkvarm</td>
</tr>
<tr>
<td>Dovertie 1914</td>
<td>2 313</td>
<td>9–16y</td>
<td>1897–1904</td>
<td>H,W</td>
<td>Jönköping, boys</td>
</tr>
<tr>
<td>Sundell 1917</td>
<td>18 000</td>
<td>7–14y</td>
<td>1902–9</td>
<td>H,W</td>
<td>Stockholm</td>
</tr>
<tr>
<td>Törnell 1920</td>
<td>523</td>
<td>9–16y</td>
<td>1903–10</td>
<td>H,W</td>
<td>Norrälje</td>
</tr>
<tr>
<td>Ljunggren 1925</td>
<td>2 703</td>
<td>7–13y</td>
<td>1911–17</td>
<td>H,W</td>
<td>Skåne</td>
</tr>
<tr>
<td>Dahlberg 1931</td>
<td>962</td>
<td>8–13y</td>
<td>1916–21</td>
<td>H</td>
<td>Uppsala, one year longitudinal</td>
</tr>
<tr>
<td>Ljunggren 1933</td>
<td>63 497</td>
<td>7–14y</td>
<td>1913–20</td>
<td>H,W</td>
<td>All of Sweden</td>
</tr>
<tr>
<td>Broman 1942</td>
<td>8 441</td>
<td>0–18y</td>
<td>1920–38</td>
<td>H,W</td>
<td>Stockholm, Malmö, Östergötland</td>
</tr>
<tr>
<td>Samuelsson 1971</td>
<td>1 401</td>
<td>4,8,13y</td>
<td>1954–63</td>
<td>H,W</td>
<td>Västerbotten</td>
</tr>
<tr>
<td>Ljung 1974</td>
<td>740</td>
<td>10–16y</td>
<td>1955</td>
<td>H,W</td>
<td>All of Sweden, longitudinal</td>
</tr>
<tr>
<td>Sunnegårdh 1986</td>
<td>682</td>
<td>8,13y</td>
<td>1972–67</td>
<td>H,W</td>
<td>Västerbotten, Uppsala, Älvsborg</td>
</tr>
<tr>
<td>Lindgren 1986</td>
<td>2 907</td>
<td>7–16y</td>
<td>1967</td>
<td>H,W</td>
<td>All of Sweden, longitudinal</td>
</tr>
<tr>
<td>Cenerud 1991</td>
<td>10 127</td>
<td>7,10,13y</td>
<td>-33,-43,-53,-63</td>
<td>H,W</td>
<td>Stockholm</td>
</tr>
<tr>
<td>Karlberg 1994</td>
<td>3 650</td>
<td>0–18y</td>
<td>1973–75</td>
<td>H,W</td>
<td>Göteborg, longitudinal</td>
</tr>
<tr>
<td>Aurelius 1994</td>
<td>2 471</td>
<td>0–6y</td>
<td>1980</td>
<td>H,W</td>
<td>Stockholm, longitudinal</td>
</tr>
<tr>
<td>Werner 2006</td>
<td>3 579</td>
<td>7–18y</td>
<td>1973</td>
<td>H,W</td>
<td>All of Sweden, longitudinal</td>
</tr>
<tr>
<td>Werner 2006</td>
<td>3 107</td>
<td>0–19y</td>
<td>1981</td>
<td>H,W</td>
<td>All of Sweden, longitudinal</td>
</tr>
</tbody>
</table>

Measures: H=height, W=weight. h=head circumference
Surveys with locations on different places are marked in **bold**
Local surveys

The pioneer in Sweden is Wretlind (1878) – the first person in Sweden who weighed schoolchildren regularly. He investigated girls in Gothenburg. Without taking height under consideration, he registered mean values for body weight for age 7 y and 8 y in spring and autumn for the three schools, finding a seasonal variation in their weight [Wretlind 1878]:

"För att få en säkrare grund för mitt omdöme om skolgångens inflytande på flickornas helsa och utveckling, än som kan erhållas genom speciella iakttagelser af ofvan antydda art, har jag vid de omnämnda mönstringarna även tagit flickornas kroppsvikt medels en vanlig decimalväg. Jag har nämligen antagit, att stillastående i kroppsvikt eller ännu mer minskningen af densamma vore det bästa beviset för en sjuklig eller åtminstone ganska svag kropp".

E. W. Wretlind 1878.

Weight, during the first year of life, was investigated by Pettersson for children in Uppsala. 1 675 children born in hospital were investigated, thus a strongly selected sample [Pettersson 1882]. Schoolchildren in Kristianstad were surveyed by Dovertie [Dovertie 1895] and in another survey by him in Jönköping, 2 313 boys aged 9–16 y [Dovertie 1914]. In a survey in Sundsvall, with 654 boys and 677 girls, age 8 to 15 y, height was measured without shoes on and weight with clothes on, and because of that body weight was counted minus 1/17 of the weight of body and clothes together. Means for height and weight were presented for schoolchildren, inspired by Hertel in Copenhagen who had done this as a routine already in 1880 [Steenhoff 1900]. In the spring of 1906, 422 schoolchildren were surveyed in Hjortkvarn in the middle of Sweden. These were children from the countryside in Närke [Norinder 1907]. In 1917 Sundell reported from a huge survey of 7–14-y-olds (for example, it included 2 366 10-y-olds) [Sundell 1917]. In Norrtälje, Törnell surveyed 523 9–16-y-olds in 1919 [Törnell 1920]. Another early investigator of weight of infants was Höjer, 0 to 1 y, [Höjer 1926].

Within a study of child health and nutrition in a northern Swedish county medical and anthropometrical examinations were carried out [Samuelsson 1971]. 1 401 children, 4-, 8- and 13-y-olds, were selected from three areas of Västerbotten county that differed from one another geographically and in part socio-economically; namely, one urban area and two rural areas. All the examinations were performed during the autumn of 1967 and thus the children were born in 1954, 1959 and 1963.

Within the so called Solna-study, a prospective longitudinal study within a multi-centre European study, the somatic development of children in a Swedish urban community was studied. At first reported from birth to age 16 y [Karlberg 1976], but then up to age 25 y by Taranger et al. [Hägg 1991]. Data on somatic development were collected for 212 children (122 boys and 90 girls) born between April 1955 and March 1958. 183 of 212 newborns were recruited antenatally by asking every fourth pregnant woman at the antenatal clinic of the community chosen for the study. The other 29 children constituted a pilot group and were the first children selected for the study. At age 16 y, 170 children (84.4%) were still being followed in the study.

They were examined at the following ages: 1 month (4 weeks), 3 months (13 weeks), 6 months (26 weeks), 9 months (39 weeks), 12 months, 18 months, 2 years
and then subsequently once a year. In order that the investigation may be considered to represent true ages the following tolerances regarding time have been aimed at: for the age of 4 weeks ± 2 days; for 3–18 months ± 1 week; for 2–3 years ± 2 weeks and subsequently ± 2 weeks and ± 3 weeks. It is the material from this study that construct the national growth charts used in Child Health care and School Health care all over Sweden between 1973 and 2001.

In the city of Uppsala, Mellbin et al. conducted a study in 1989 [Mellbin 1989, Mellbin 1989]. The study comprises two materials: First, 971 children born in 1963 in the city of Uppsala and for whom weight data were available from the first year of life, and after the first school health examination at the age of 7 y they were followed up at 10, 13 and 15 y. At the last examination 944 children (97%) could still be included in the study. Second, a sample of 5 399 Swedish schoolchildren (all schoolchildren in the municipality of Uppsala born in 1965–67 who were residents in this municipality both in the first and fourth grade were included (2 738 boys and 2 661 girls). The children were then followed up to age 18 y.

By collecting data from school health records for 7-, 10- and 13-y-old children in Stockholm born in 1933, 1943, 1953 and 1963, Cernerud described the development for height and weight over time by using samples of about 25% from these four Stockholm birth cohorts [Cernerud 1991].

The population-based reference values from Göteborg, from birth to 18 y of age, is based on a study population of 5 111 final grade school children who were born in Göteborg, or in the surrounding areas. Data were collected from child health and school health records. Out of this population was 4 488 born in 1974. Information on 319 girls and 304 boys was not collected, since some of them were not willing to or failed to attend the last investigation at school. Exclusion criteria, such as lack of information at birth, multiple births, prematurity and growth disorders were applied in the selection of subjects. 1 849 boys and 1 801 girls remained [Albertsson-Wikland 2002]. Body mass index reference values were reported in 2001 [Karlberg 2001]. This material has been the reference values within Child Health Care and School Health Care since 2001.

Surveys that cover different geographical populations in Sweden

First performed was the classical investigation by Key [Key 1885]. The aim was to shape some reference-values for children’s growth in Sweden, from age 7 y to 21 y. The population who was investigated contained both pupils from State secondary schools, 14 590 boys, (in Stockholm) and girls’ high schools, 2 971 girls, (from different places in Sweden), and from preparatory schools and elementary schools, 458 boys and girls, (in Stockholm). Key pleads the necessity of such investigations in every country, with regard to the influence of race, climate, and social and hygienic conditions on bodily development. An interesting point is that Key was already maintaining the need of investigations in different social classes of the population with different living conditions since the development of the child at different ages can differ quite a lot from one social level to another.
"Längdmåttet har såsom komitén i sitt frågeformulär begärde, tagits, sedan skodonen blivit afdragna. Vid vägningarna hafva ej kläderna fränräknats. I allmänhet, eller åtminstone ofta, torde de väl å gossarna hafva utförts, sedan rockarna eller jackorna blivit aftagna; men bestämda uppgifter härom saknar jag”.

A. Key 1885

Heights for conscripts, at age 21 y, are presented by the professor in anatomy who was very active examining conscript data from late 19th century and forwards [Hultcrantz 1896, Hultcrantz 1927].

Ljunggren made a huge survey of 63 497 schoolchildren aged 7–14 y from different places in Sweden [Ljunggren 1933].


C.A. Ljunggren 1933

Broman et al. had the ambition to establish new Swedish norms for height and weight as a basis for practical hygienic and medical work. Their material, in total 8 441 individuals, was collected during 1938 and 1939: In Stockholm 4 379 individuals (age 0 to 18 y), in Malmö 2 662 individuals (age 1 to 18 y) and in the county of Östergötland 1 400 individuals (age 7 to 14 y). The material was cross-sectional and all the children were measured once standardised. A comparison between the figures for the children from different regions shows small differences. Stockholm comes first both in regards to height and weight and is followed by Malmö. The figures for Östergötland are the lowest. Figures were found to indicate that children born in the autumn have somewhat greater height and weight at birth than children born in the spring [Broman 1942]

Ljung et al. made a study with the ambition to create new standards that should replace the standards made up from Broman’s work for schoolchildren. The material used is the same as for the study of Lindgren below. The sample includes two main groups: One group consists of pairs of twins, the other of one or two classmates to the twins – of the same sex, born in the same month and year (mostly in 1955, a few in 1954) as the twins they were to match. The samples were drawn from urban elementary schools where the possibilities of making uniform arrangements for the different tests and observations were supposed to be better than in rural schools. [Ljung 1974].
In 1985 Persson presented a material with heights and weights of 572 children in ages 4, 8 and 13 y from three communities in Västerbotten, Uppland and Västergötland [Persson 1985].

The aims of a study by Sunnegårdh was to study the height, weight and skinfold thickness in randomly selected 8- and 13-y-old children in Sweden and to compare the results with earlier figures for Swedish children. The study was performed in 1980–81. The investigation comprised 682 children born in 1967 and 1972 in four different regions of Sweden, namely Vilhelmina and Dorotea, Umeå, Uppsala and Tierp, and Vännersborg and Trollhättan. Altogether 789 children were selected at random from a total population of 5 342 children, 8 and 13 y old, by use of the Swedish central population register, and from these 15 children were excluded because of chronic diseases. In addition, 18 children moved from the regions before the investigation was completed. 70 refused to participate for various reasons and one child was inaccessible, whereas 338 8y-olds, and 347 13-y-olds participated. The anthropometric measurements were performed by four paediatricians. Although the children in this study were randomly selected they cannot be regarded as representing all children in Sweden of comparable ages, as the most heavily urbanised areas and the southern part of the country were not included [Sunnegårdh 1986].

The only study in Sweden, before the present studies within this thesis, of a representative national sample was carried out by Lindgren et al. by collecting data for height and weight from school health records for children born in 1967, age 7 to 16 y [W-Lindgren 1986]. By a sampling in many steps the group of children was picked out: First they chose communities all over Sweden out from four factors: number of inhabitants, number of socialist respective rightwing voters, number of occupied in public sector and number of immigrant-pupils. Thus were 29 communities selected and within the communities a number of different classes, argued to represent a mini-Sweden. In every participating school, the school nurse copied the health records for every pupil born the 5th, 10th, 15th, 20th and 25th in each month in 1967. The study was based on records from 1 390 girls and 1 517 boys. Missing number was about 2%. The description was made for mean values, height and weight, for different ages boys and girls, and for social class based on the occupation of the father.

The two materials from Ljung and Lindgren (Ljung 740 children born in 1955 and Lindgren 2 907 children born in 1967) were together presented as the basis for Swedish population reference standards for height, weight, and body mass index attained at 6 to 16 y (girls) or 6 to 19 y (boys) in 1995 [Lindgren 1995]. The proportion of children whose parents were immigrants in the 1967 sample was approximately 10%, in the 1955 sample the proportion was about 2%. By comparing the two samples in the overlapping ages there were no consistent differences between the heights of the two samples, but the earlier sample averaged 3% lower in weight for both girls and boys, with no apparent age trend. All weights from this 1955-born sample were increased by 3% in the subsequent calculations.

They compared this with age-overlapping data from a material of children born in 1980 living in a suburb of Stockholm at age 6 y [Lindgren 1994]. They saw very small differences between the samples in height and weight at age 6 y. The authors argue that there are no differences between national samples over time (55 to 67-born) and no differences between regions.
1.5.2 Growth surveys – international examples

National surveys

Norway
The study group consisted of 3 068 children, 1 479 girls and 1 589 boys, born 1956–68. The children lived in the parishes of Sletteland and Landås where the population may be regarded as representative for an average town in Norway. They were measured at intervals of about one year during the period 1971–74. Height, weight and 10 other anthropometric variables were measured the first year and, for most children, also the second year. In the two subsequent years, height and weight measurements were followed up in many of the children. On 966 of the 3 068 children only height and weight were registered. No children were excluded from the study except those with diseases or malformations affecting their general condition severely [Waaler 1983]. Values from birth to age 4 y are based on data of infants born 1982–84 [Knudtzon 1988]. Norwegian percentile curves are presented by Knudtzon and Waaler [Knudtzon 1988].

Iceland
Height and weight were measured in 5 554 randomly selected Icelandic schoolchildren (2 779 boys and 2 775 girls) at the age of 6 to 16 y. The total number of Icelandic children in the selected groups are 47 583 [Dagbjartsson 1989].

United Kingdom
Cameron [Cameron 2002] examines the base for different growth curves in United Kingdom and discusses in what circumstances they will be adequate.

In 2001, a group reviewed all the growth reference charts in the United Kingdom [Wright 2002] and concluded that the main recommendation is to use the United Kingdom 1990, UK90 [Cole 1995, Cole 1995, Freeman 1995, Cole 1998] for all ages. The Buckler references [Buckler 1997] are suitable for assessing cross sectional height in isolation, but the UK90 should be used where both weight and height are being evaluated. A simple method for assessing centile change based on UK90 is described by Cole [Cole 1998].

The Netherlands
The Netherlands have a strong tradition with their four cross-sectional surveys in 1955, 1965, 1980 and 1996 [de Wijn 1960, van Wieringen 1971, Roede 1985, Fredriks 2000] with nationally representative samples. Roede et al. investigated, cross-sectionally, in 1979–80, a material collected within the Netherlands’ third nation-wide survey of growth [Roede 1985], and in 1997 a fourth nation-wide survey in the Netherlands was carried out [Fredriks 2000]. Within these two Dutch studies the stratified samples were nationally representative of the demographic, geographic, and socio-economic distribution of the population.

To document the distribution of BMI in the Netherlands when obesity was less common, a nationally representative growth survey was made. 41 000 boys and girls age 0–20 y were the subjects [Cole 1999].

A nation-wide growth study performed in the Netherlands in 1979–1980 [Roede 1985]. Length, height and weight were measured in 42 000 healthy 0–19-y-old
individuals of Dutch origin. The stratified sample was nationally representative of the
demographic, geographic and socio-economic distribution of the population. The
measuring was performed during routine medical examination programmes by 200
teams of medical officers and nurses in the child health sector. The survey was cross-
sectional, with a restricted period of data collection to avoid the effect of secular
changes within the survey period. Nearly 46 000 recording forms were received. After
exclusions and the rejection of outliers (children with fewer than two Dutch parents,
infants with birthweights below 2 500 g, and children with pathological conditions),
data on 21 521 males and 20 245 females were available for analysis [Fredriks 2000].
Furthermore, special surveys have been done for children with Moroccan or Turkish

Ireland
A cross-sectional study from Ireland of 3 509 children was carried out on
approximately 100 boys and 100 girls of each year of age from 5 to 19 y. A sample of
schools was selected to include pupils of different socio-economic class, and we
measured 50 children of each sex and year of age, in both rural and urban areas. 227
were excluded because of illness, non-Irish parentage, or inadequate information
[Hoey 1987].

Europe
Fully longitudinal data from the Euro-Growth Study is the base for Euro-Growth
references for length and BMI. Birth dates were between August 1990 and October
1993 for 2 145 children collected in 21 study centres in 11 European countries
[Haschke 2000, van’t Hof 2000].

Cuba
In 1970 the population of Cuba was 8 575 00 of whom 3 900 000 were between 0–20
y of age. To distribute the sample size within the population consideration was given to:

First, the number of children under 20 y by province.
Second, the average number of children under 20 y per household. This average
was different for urban and rural zones. For example, in metropolitan Havana it was
1.6; in urban Oriente province it was 1.8, while Oriente province had 3.1 per family.
Third, the sample was initially stratified by province according to these figures.
The final sample size was 56 000; 28 000 children of each sex. The sample should
enable the investigators to achieve two main objectives: precision (particularly for the
outlying centiles) and representativity. In addition, it should allow comparisons
between different regions of the country and different ethnic and social groups. One
measuring team was assigned to each province, with one in reserve nationally. There
were 9 teams because Havana and Oriente provinces, being more populated, had two
each [Jordan 1975, Jordan 1979].

United States
Several large, expansive long-term studies were initiated in the US. These include Fels
study and this is the only one of these studies still active, which began in 1929 [Roche
1992]. The sample of the Fels study is healthy, well-nourished boys and girls, living in
small urban communities and rural areas of south-western Ohio [Roche 1992]. This
data material was the foundation for the former NCHSC growth charts and was also
used as an international standard in spite of the sample being neither socially nor geographically representative [Hamill 1977, Hamill 1979].

In the US there are Centers for Disease Control and Prevention 2000 Growth Charts for the United States [Kuczmarski 2000, Ogden 2002]. These charts are based on national data collected in a series of 5 surveys between 1963 and 1994.

**Australia**
During 1997, 2 828 females and 2 871 males (age range 5–7 y) were measured when entering primary school by school nurses using a Minimeter, in North Brisbane, Australia (93% of entrants) [Jack 1999].

**China**
Nationwide growth surveys have been performed once every 10 years in China since 1975 (1975, 1985, 1995 and 2005?). In the second survey in 1985, data for almost half a million children and adolescents, aged 7–18 y, were collected [Zhang 1988]. In the second survey the physical growth of 175 290 Chinese children less than 7 y in age was undertaken in the urban and rural areas of ten provinces [Ching 1989]. The third survey was performed in 1995, and from this survey, data for the Beijing population were retrieved to construct the height for age, and weight for age percentile curves [Li 1999].

A survey on the physical growth of Chinese children under 7 y in the urban and suburban rural areas of nine cities of China 1995 was compared with the survey ten years earlier (1985) [Anonymous 1998].

Body mass index reference curves for Chinese children are reported by Leung. [Leung 1998]

**Turkey**
In 1973 growth data were published for the first time in Turkey (Neyzi 1973), but a more recent survey showed body mass index references for Turkish children aged 6 to 18 y. It was constructed by first collecting height and weight measurements biannually for 1 100 boys and 1 019 girls. The children were born in 1975 or later and all children belonged to the highest socio-economic group [Bundak 2006, Neyzi 2006].

### 1.5.3 Internationally used reference values

For establishing a standard definition for child overweight and obesity worldwide, six large nationally representative cross sectional growth studies (Brazil, Great Britain, Hong Kong, the Netherlands, Singapore and the United States) were pooled together in order to get reference values for BMI, IOTF [Cole 2000]

The WHO Multicentre Growth Reference Study (MGRS) (1997–2003) collected primary growth data and related information from 8 440 children from widely differing ethnic backgrounds and cultural settings (Brazil, Ghana, India, Norway, Oman and USA). Eligibility criteria included breastfeeding, no maternal smoking and environments supportive of unconstrained growth. The study combined longitudinal (birth to 24 mo) and cross-sectional (18–71 mo) components [WHO Child Growth Standards 2006]. The construction of the child growth standards are described by Borghi et al. [Borghi 2006].
2. AIMS

2.1 GENERAL AIM

The general aim of this thesis was to explore if it was possible, on a national level, to monitor central measures of public health among infants, children and adolescents, height and weight, by collecting data from health records from all of Sweden.

All children should be included and missing cases should be very few.

A good description of growth could reveal how the situation is, and in what direction the development goes, and be a base for creating references values for different purposes.

2.2 SPECIFIC AIMS

The specific aims, each related to one of the five papers, were:

Paper I: To investigate the suitability of using routine height and weight data from records within a school health service system, for population monitoring of child and adolescent growth on a national level, in order to produce a base for both descriptive and prescriptive values.

Paper II: To describe Swedish children’s growth (i.e. height, weight and body mass index) from birth to age 19 years.

Paper III: To describe the development of head circumference, from birth to age 48 months, of infants in Sweden.

Paper IV: To determine changing patterns over time by comparing the rates of relative weight loss (body mass index, BMI) among boys and girls, from age 7 to 18 years, in two birth cohorts.

Paper V: To monitor and describe, on a national level, the development of body mass index (BMI), overweight and obesity from age 7 to 18 years of schoolchildren in Sweden during a period of 8 years.
3. MATERIAL AND METHODS

3.1 TARGET POPULATIONS AND STUDY POPULATIONS

3.1.1 Target populations

Birth cohort 1973
The target population is the birth cohort 1973
Living born: 109,663
Average age of the mothers: 26.5 y
Breastfeeding at the age of 2 mo 32%, at the age of 4 mo 14%, at the age of 6 mo 7%

Migration during 1973
Immigration: 29,443 individuals
Emigration: 40,342 individuals

Birth cohort 1981
The target population is the birth cohort 1981
Living born: 94,064
Average age of the mothers: 28.0 y
Breastfeeding data not available (see study population)

Migration during 1981
Immigration: 32,272 individuals
Emigration: 29,440 individuals

3.1.2 Study populations

Cohort 1973
Number with collected data for the sample: 3,579 individuals
Observation period: age 7 y to age 18 y.
Table 6. Individuals born the 15th of every month. Countywise, number of individuals 31.12 1989 and collected

<table>
<thead>
<tr>
<th>County</th>
<th>31.12 1989</th>
<th>Collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stockholm</td>
<td>668</td>
<td>649</td>
</tr>
<tr>
<td>Uppsala</td>
<td>136</td>
<td>127</td>
</tr>
<tr>
<td>Södermanland</td>
<td>114</td>
<td>98</td>
</tr>
<tr>
<td>Östergötland</td>
<td>183</td>
<td>177</td>
</tr>
<tr>
<td>Jönköping</td>
<td>141</td>
<td>129</td>
</tr>
<tr>
<td>Kronoberg</td>
<td>81</td>
<td>77</td>
</tr>
<tr>
<td>Kalmar</td>
<td>107</td>
<td>101</td>
</tr>
<tr>
<td>Gotland</td>
<td>31</td>
<td>30</td>
</tr>
<tr>
<td>Blekinge</td>
<td>76</td>
<td>74</td>
</tr>
<tr>
<td>Kristianstad</td>
<td>137</td>
<td>120</td>
</tr>
<tr>
<td>Malmöhus</td>
<td>328</td>
<td>311</td>
</tr>
<tr>
<td>Halland</td>
<td>131</td>
<td>113</td>
</tr>
<tr>
<td>Göteborg och Bohuslän</td>
<td>275</td>
<td>297</td>
</tr>
<tr>
<td>Älvsborg</td>
<td>211</td>
<td>197</td>
</tr>
<tr>
<td>Skaraborg</td>
<td>124</td>
<td>121</td>
</tr>
<tr>
<td>Värmland</td>
<td>116</td>
<td>113</td>
</tr>
<tr>
<td>Örebro</td>
<td>113</td>
<td>106</td>
</tr>
<tr>
<td>Västmanland</td>
<td>108</td>
<td>104</td>
</tr>
<tr>
<td>Dalarna</td>
<td>125</td>
<td>121</td>
</tr>
<tr>
<td>Gävleborg</td>
<td>133</td>
<td>126</td>
</tr>
<tr>
<td>Västernorrland</td>
<td>113</td>
<td>106</td>
</tr>
<tr>
<td>Jämtland</td>
<td>40</td>
<td>35</td>
</tr>
<tr>
<td>Västerbotten</td>
<td>126</td>
<td>123</td>
</tr>
<tr>
<td>Norrbotten</td>
<td>132</td>
<td>119</td>
</tr>
<tr>
<td><strong>All</strong></td>
<td><strong>3 749</strong></td>
<td><strong>3 579</strong></td>
</tr>
</tbody>
</table>

Breastfeeding data are not available (see target population).
Children born outside Sweden: 225

Diagnosis (from health records) of 32 children with disorders of major impairment for growth:
Status post polio, Cong. dislocation of the hip, Coeliaci 2 children, Right leg shortened, Juvenile chronic arthritis, Diabetes mellitus 2 children, Heart failure op., Growth error 8 children, Growth error and anorexia, Mental retardation and inborn error of metabolism, Myotonicity of m. sternocleidomastoideus, Polyneuropati: Mb Charcot Marie Tooth, Hypothyreosis, Mb Turner, Mb Down, Hereditary angioneurotic syndrome, Antihidotic ectodermal dysplasia, Hashimoto struma, Cerebral palsy (Wheel chair), Pubertas tarda, Scoliosis, Nephrectomy, Dwarfism.

**Cohort 1981**
[Statistics Sweden 1999]
Number with collected data for the sample: 3 107 (1 548 boys/1 559 girls)
Observation period: Birth to age 19 y (height and weight)
Birth to age 48 mo (head circumference)

Table 7. Breastfeeding in percent.

<table>
<thead>
<tr>
<th>Age, in months</th>
<th>Breast-fed Exclusively</th>
<th>Breast-fed and formula-fed</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>42.5</td>
<td>30</td>
</tr>
<tr>
<td>&gt;2</td>
<td>57.5</td>
<td>70</td>
</tr>
<tr>
<td>2–4</td>
<td>20.8</td>
<td>22</td>
</tr>
<tr>
<td>&gt;4</td>
<td>36.7</td>
<td>47.8</td>
</tr>
<tr>
<td>4–6</td>
<td>22.6</td>
<td>22.2</td>
</tr>
<tr>
<td>&gt;6</td>
<td>14.1</td>
<td>25.6</td>
</tr>
</tbody>
</table>

Table 8. Individuals born the 15th of every month. Countywise, number of individuals 31.12 1989 and collected
(5th- and 10th and 20th-born in some counties with low population density, not studied within this thesis)

<table>
<thead>
<tr>
<th>County</th>
<th>31.12 1989</th>
<th>Collected</th>
<th>5th-born</th>
<th>10th- and 20th-born</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stockholm</td>
<td>587</td>
<td>589</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uppsala</td>
<td>114</td>
<td>116</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Södermanland</td>
<td>83</td>
<td>84</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Östergötland</td>
<td>169</td>
<td>166</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jönköping</td>
<td>116</td>
<td>130</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kronoberg</td>
<td>71</td>
<td>75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kalmar</td>
<td>97</td>
<td>104</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gotland</td>
<td>24</td>
<td>25</td>
<td>31</td>
<td>45</td>
</tr>
<tr>
<td>Blekinge</td>
<td>54</td>
<td>51</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>Kristianstad</td>
<td>122</td>
<td>117</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malmöhus</td>
<td>263</td>
<td>289</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Halland</td>
<td>95</td>
<td>94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Göteborg och Bohuslän</td>
<td>247</td>
<td>238</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ålvsborg</td>
<td>176</td>
<td>179</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skaraborg</td>
<td>110</td>
<td>112</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Värmland</td>
<td>104</td>
<td>101</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Örebro</td>
<td>94</td>
<td>101</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Västmanland</td>
<td>90</td>
<td>89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dalarna</td>
<td>114</td>
<td>107</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gävleborg</td>
<td>97</td>
<td>103</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Västerbottens</td>
<td>98</td>
<td>94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jämtland</td>
<td>39</td>
<td>40</td>
<td>51</td>
<td></td>
</tr>
<tr>
<td>Västerbotten</td>
<td>106</td>
<td>112</td>
<td>103</td>
<td></td>
</tr>
<tr>
<td>Norrbotten</td>
<td>88</td>
<td>85</td>
<td>101</td>
<td></td>
</tr>
</tbody>
</table>

All                     3 158     3 107     334     45
Added sample from counties with low population density (not studied within this thesis)

<table>
<thead>
<tr>
<th>County</th>
<th>Available 31.12 1989</th>
<th>Collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Västerbotten</td>
<td>113</td>
<td>103</td>
</tr>
<tr>
<td>Norrbotten</td>
<td>105</td>
<td>101</td>
</tr>
<tr>
<td>Jämtland</td>
<td>54</td>
<td>51</td>
</tr>
<tr>
<td>Blekinge</td>
<td>49</td>
<td>48</td>
</tr>
</tbody>
</table>

Born on the 5th, the 10th, and the 20th of every month

<table>
<thead>
<tr>
<th>County</th>
<th>Available 31.12 1989</th>
<th>Collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gotland</td>
<td>77</td>
<td>76</td>
</tr>
</tbody>
</table>

Immigrated children after 31.12 1989 (not studied within this thesis): 94

Infants with birthweight <2 500 g: 118
Children born outside Sweden: 239
Children with a disorder with major impairment for growth: 24

Diagnosis (from health records) of 24 children with chronic disease of major impairment for growth:
- Many and long stays in hospital
- Growth hormone deficiency 3 children
- Pulmonary hemosiderosis (lungtransplantation)
- Multiple intolerance for food
- Aneurysm op.
- Crippled
- Cerebellar dyspleoi
- Intestinal resection
- Pubertas praecox ?, Mb Down and Heart failure
- Coeliac
- Born with structural scoliosis
- Growth error 4 children
- Pulmonalis stenosis
- Cerebral palsy
- Spinal atrophy of the muscles
- Spinal atrophy
- Disease of long duration
- Juvenile chronic arthritis
- Hypophyseal short in stature

*About head circumference 0–48 mo*

Of the 3 107 children, 123 individuals had immigrated between age 48 mo and 8 y and are thus excluded since they are not represented by any measurements for the age interval investigated. Thus 2 984 individuals could be studied from birth to age 48 mo (see Table 1 in Paper III).

The instructions for measuring head circumference during the period in question are as follows: ”Measuring is done with a measuring-tape, horizontally at the place where the largest head circumference is, read to the closest millimetre.”

[Anvisningar och kommentarer]

### 3.2 DATA COLLECTION

The first wave of data collection for both cohort 1973 and cohort 1981 took place during the autumn in 1989.
3.2.1 Cohort 1973

Almost every Swedish child attends school from age 7 to 19 years and during this time information on growth is recorded on a fairly regular basis by school nurses. Thus, we chose to study a national random sample of records from all children born on the 15th of any month in 1973 and living in Sweden on December 31st 1989. The study design was longitudinal, and for each child measurements of height and weight were recorded with the date that the measurement was obtained. The data collection from school health records, including both public and private schools, was conducted in three waves: the first wave when the children were 16 years old (in grade 9 in compulsory school), and the second when they were 18 years old (one year before Swedish adolescents usually leave secondary school). Finally, in the third wave we looked for records after the adolescents had left school in local community archives.

The nurses also recorded if the child was born outside Sweden or had a chronic disease. A chronic disease involving major growth impairment (as judged by an experienced paediatrician, the author, BW) was present in 32 children. Records were collected for 3 579 of the 3 749 adolescents, bringing the number of missing cases to 4.5%. An example of a page in a school health record is shown in appendix.

A unique study identification code number was appended to school records by school nurses, so that linkage between data collection waves was possible without using the official registration number assigned to all Swedish residents. We anticipated that avoidance of the official registration code was expected to help us to keep the number of non-responders as low as possible. If the adolescent signed a written consent form after age 18 years (completed by approximately 80%), the official 10-digit identification number was used for record linkage. Where school records were incomplete for boys at age 18 years, data from the MSCR were used instead.

In the records, weight was noted in kilograms and height in centimetres with a numerical accuracy of at least ±0.5 (kg/cm). After data entry we plotted height and weight by age for each child to identify registration errors.

3.2.2 Cohort 1981

Several times during their preschool years, almost 100% of Swedish children visit childhood health care centres where they are weighed and measured. These records are then sent to the school nurses when the children enter school. Almost all children stay in school from age 7 to 19 y, and at school they are fairly regularly weighed and measured by school nurses. As all this information is collected in their records, the prerequisites for monitoring children’s growth and selecting suitable samples are met.

Collection of data from the records was done on three occasions in school: in grade 2, in grade 9 and finally in grade 12. Most children are 8, 16 and 19 y old respectively in those grades. For approximately 10% of the children, in particular those who moved or changed schools, records could not be obtained from the school nurses. In many such cases it was possible to find the records, as the records were deposited in the local community archives after the children had left school. Through this combined effort, childhood health records and school health records were collected for 3 107 of the 3 158 children, meaning that missing cases accounted for only 1.6% of the
sampling frame. An example of a page with growth data in a child health record is shown in appendix.

In this investigation, school nurses were asked to mark the record of each child with a unique study identification code, to make it possible to combine the data from the three data collection occasions without using the official 10-digit identification number. If the adolescent signed an informed consent form after age 18 y, then the official 10-digit number could be used. In cases where the school health records were missing values for boys at around age 18 y, and if there was informed consent, data from the MSCR were added.

In the records, all data regarding height and weight were registered by date. Weight was noted in kilograms with a numerical accuracy of at least ± 0.05 from 0 to 6 y and ± 0.5 from age 7 to 19 y, height was noted in centimetres with a numerical accuracy of at least ± 0.5. After data entry, we plotted height and weight by age for each individual in order to identify registration errors. A summary of the data sources is found in Table 1.

3.3 ANALYSIS

3.3.1 Paper I, II and III

Height and weight

For cohort 1973 and 1981 data were analysed using a cross-sectional approach. To do this we had to take into account that height and weight were not measured at the same ages in different children. The adjustment was performed using piecewise linear regression as follows.

For both cohort 1973 and cohort 1981 we centred on the whole numbers from age 7 to 18 years (for cohort 1981 up to 19 years), and defined equal intervals, e.g. 7 years ±180 days. Within each one of these intervals, one randomly chosen observation from each individual (when available) was used to form a linear regression of the outcome (height and weight) on age, measured in days. Thus, observations within each interval were statistically independent in the sense that each individual contributed only one observation. The midpoints of the intervals were from 7 up to 19 years with consecutive steps of one year. A suitable length of the intervals, from the midpoint to the upper or lower limit, was tested with values ranging from 45, 90, and up to 180 days. The final calculation used an interval of 180 days. Linearity within each interval was examined for each chosen length using local regression smoothing [Cleveland 1979], and comparing the smoothed curve with the straight line. Based on this comparison, we accepted the linear function as a satisfactory approach within each interval. Calculations were performed for males and females separately, both with and without exclusions.

The linear regression was used to estimate the mean of the outcome for the centre of each interval. The variability within each interval was derived as the square root of the residual variance of the regression line, which gives a standard deviation. SD, Skewness and kurtosis were calculated using the residuals produced by linear regression. Kurtosis indicates whether the distribution around the mean is thick-tailed.
a higher proportion of subjects are found at the extremes of the distribution, kurtosis >0) or thin-tailed (the opposite case, kurtosis <0).

For cohort 1981 we defined intervals with midpoints at months 1 to 12, and then at every half-year from 1.5 y up to and including 6.0 y. A suitable interval length, from the midpoint to the upper or lower limit, was tested, with values ranging from 7 days (in the first months of life) to 180 days (for age 7 y and older). Within each one of these intervals, one randomly chosen observation for each individual (when available) was used to run a linear regression of the outcome (height and relative weight) on age measured in days.

Head circumference in cohort 1981

We defined intervals with midpoints at various ages from 15 d to 48 mo. In each interval, one randomly chosen observation for each individual (when available) was used to form a linear regression of the outcome on age measured in days. Thus, the observations within each interval were statistically independent, in the sense that each individual contributed only one observation. A suitable interval length, from the midpoint to the upper or lower limit, was tested with values ranging from 7 d at ages 15 and 30 d, 10 d at ages 45 and 60 d, 14 d at ages 75, 90 and 120 d, 21 d at ages 6–8 mo, 30 d at ages 9–17 mo, 60 d at age 18 mo, and then 90 d at ages 24–48 mo. The linear equation assumed to adequately describe the relationship between head circumference and age within each interval was examined using local regression smoothing [Cleveland 1979]; then the smoothed curve was compared with the straight line.

3.3.2 Paper IV

Change in BMI was calculated as the difference in BMI between all adjacent pairs of measurements within each subject. The relative change was obtained by dividing the difference in BMI by the first of the two BMI measurements. BMI reduction was defined as a negative relative change in BMI.

Consecutive multiple BMI reductions within the same subject, with no intervening BMI increase, was defined as one episode of BMI reduction from the first measurement taken before the reduction to the last measurement before BMI increased. The BMI reduction episodes were divided into five categories: no reduction (including increases), <5%, 5–<10%, 10–<20%, and ≥ 20%. A further classification was to dichotomize the reduction as above or below the 5% cut off value.

We categorized the age of the children when the measurement before the reduction was taken into four intervals: 7–9 y, 10–12 y, 13–15 y, and 16–18 y. If an individual had more than one BMI reduction episode during one age interval, only the episode with the largest BMI reduction was considered. We also categorized the BMI at the start of the reduction according to the BMI percentiles calculated from the 1973 cohort, given sex and age: <40th, 40th to <60th, 60th to <70th, 70th to <90th, and ≥90th percentile. This was done because means for BMI increased for cohort 1981 compared to cohort 1973 in all ages for both boys and girls with exception for girls age 17 and age 18 y. The increase in age 7 and 8 y was around 1.5%, and for older ages around 2.5%. If a child was measured more than once during the same age interval but had no

78
BMI reduction we randomly sampled one measurement to assign this child to one of these BMI categories.

Logistic regression was used to analyse the presence (yes/no) of a BMI reduction with the cut off at 5% defined previously. As explanatory variables cohort (born in 1973 or in 1981), sex, age, and BMI before the reduction were used. Age and BMI before the reduction were categorized in the intervals described above. We also tested for statistical interactions between the explanatory variables. The effect parameter is expressed as the odds ratio (OR) and its 95% confidence interval (CI). Because each individual could have more than one BMI reduction episode the CI was adjusted for the intraindividual correlations caused by repeated measurements. This was done by using the cluster option in the logistic regression procedure in the program package STATA [release 9, StataCorp, College Station, Texas, USA].

### 3.3.3 Paper V

Data were analysed with a cross-sectional as well as a longitudinal approach. The cross-sectional approach is more thoroughly described in paper I and in paper II.

In the longitudinal approach the two cohorts were analysed together. We formed a regression model with outcome (height or BMI) as a function of two sets of categorical variables, one for cohort (1973 or 1981) and the other for age in whole years (7, 8, 9 and up to 18 y). Interaction between these two variables was also introduced and tested. The parameters in this model correspond to means for each age category and for each cohort estimated simultaneously using all selected data from the age groups in both cohorts.

The longitudinal model was estimated with a mixed model approach; using the statistical package SAS (Version 8.2, SAS Institute, Cary, NC, US) with covariance structure according to an autoregressive structure of the first order, degrees of freedom according to the Satterthwaite approximation, and restricted maximum likelihood estimation method (REML). We calculated the marginal means at each age and contrasted these means for the two cohorts. Adjustment of p-values in case of several tests was performed. The cross-sectional analysis was done with the statistical package SPSS (Version 13, SPSS Inc, Chicago, IL, US), supplemented with user-defined commands.

### 3.4 ETHICS

These studies were approved by the Ethics Committee of the Örebro County Council, Sweden.
4. RESULTS

4.1 PAPER I

Descriptive statistics for height and weight, from age 7 to 18 y, calculated from data obtained from school health records are summarized and compared with previous Swedish studies. The data include measurements of 3 579 of 3 749 sampled children; 4.5% of individuals are missing, so non-response bias is minimal. The effect of exclusion of children with chronic diseases involving major growth impairment and/or exclusion of children born outside Sweden have a minor impact on the results due to the relatively small number of excluded children.

4.2 PAPER II

The data, from birth to age 19 y, include measurements of 3 107 of 3 158 sampled children; 1.6% of individuals are missing, so non-response bias is minimal. Thus, statistical descriptions and comparisons with similar data sets can be based on data with unusually high national representativeness. Selected subgroups (i.e. individuals born outside Sweden, suffering from chronic disease causing major growth impairment, or with birthweight <2 500 g) deviate in growth pattern, and exclusion of these subgroups increases means and decreases SD for height but only slightly influences summary statistics for weight and body mass index.

4.3 PAPER III

From the sample of 3 158, data for 3 107 infants were collected, making the percentage of missing individuals 1.6%, thus non-response bias is minimized and high national representativeness is achieved. Summary statistics for head circumference are presented. Comparisons are made to international reference materials, and to previous and current Swedish reference curves.

4.4 PAPER IV

An increasing rate, by comparing individuals born in 1973 and in 1981, of relative weight reduction episodes was found for both boys and girls. The increase for girls was most pronounced and started from a higher rate and it was seen in nearly all body weight categories and in all ages. For boys the increase in reductions occurred for all body weight categories only in the age interval 7–9 y, otherwise there is a much more heterogeneous pattern. There is a high correlation between body weight and reduction of BMI, as more individuals with overweight reduces their BMI compared to thinner individuals. For girls, the highest increase in rate is seen among the thinnest individuals.
4.5 PAPER V

From age 7 y to 18 y a strong positive secular change for BMI exists in all ages, and the rate of overweight and obesity is increasing for both boys and girls. Furthermore, obesity is growing more severe.
5. GENERAL DISCUSSION

5.1 EPIDEMIOLOGICAL MONITORING

There are at least four requirements for adequate national epidemiological monitoring. Firstly, the sample ought to be representative of the population. Secondly, there should be few missing subjects and they should not be systematically different from the non-missing subjects. Thirdly, the data ought to be valid and measurements should have acceptable precision. Fourthly, it should be possible to use the material for comparisons over time and therefore collected in a way that can be repeated.

The results indicate that our national samples better assess height, weight and head circumference among infants, children and adolescents than previously employed methods. This conclusion is based on four findings.

5.1.1 The representativity of the samples

Firstly, the sampling frame is by definition nationally representative and furthermore we succeeded in collecting data for almost the whole sample. In practice, the data were tested among 18-years old males through comparison with measurements collected at conscription for all 18-year-old males: the school record data were found to be representative for both cohort 1973 and cohort 1981.

The external validation was made of the cohort 1973 sample for 17–19 year-old males by a comparison with the national MSCR (see Table 4 in Paper I). We performed the calculations using data from MSCR with the same methodology used for the child health records, i.e. age-corrected by piece-wise linear regression of height and weight on age at measurement. For this we required the date of measurement, thus 47,739 measurements were available. For age 18 years, where the majority of the data in MSCR are found, there are only very small differences in mean value and SD between the two data sets. For weight there are practically no differences in skewness and kurtosis, with values of 1.2 and 3.1, respectively, from the school record data and 1.3 and 3.6, respectively, from the MSCR material. For the 19-year olds the difference in height between the two datasets (180.9 cm and 180.1 cm) is just outside the 95% confidence limits, 0.04 to 1.56, just outside zero.

To test the representativity of the cohort 1981 sample, we compared summary statistics of the sample with statistics from two nationwide registries containing height and weight data: 1. The MSCR, which includes measurements for approximately 80% of all males born in 1981 and holding Swedish citizenship at age approximately 18 y. 2. MBR covers all newborns in Sweden.

1. Comparison between data in cohort 1981 and conscript data is shown in Table 5 Paper II. The most measurements are available for individuals around age 18.0 and 18.5 y, both in the present study and in the MSCR. For height in the present study at age 18.0 y after exclusions, the mean is almost the same as in the MSCR (0.6 cm lower in present study without exclusions). At age 18.5 y the present study and the
MSCR have the same means, within 0.1 cm (the present study with exclusions has a 0.7 cm higher mean). For weight, the means in the present study without exclusions and means in the MSCR correspond well at both age 18.0 and 18.5 y (exclusions increase means by 0.6 kg). For both height and weight the registry has less variability as measured by the SD.

2. Comparison with MBR data. See Table 4 Paper II. The comparison was made without exclusions and revealed no differences. In this sense, the present data seem valid or representative of all boys and girls.

5.1.2 Selection bias

Secondly, few subjects were lost from the sample and they can presumably be accounted for, as some children are never measured at school if they only live in Sweden for a short period of time. A sparse pattern of measurements for one individual is sometimes explained by migration, by chronic disease, or disability being treated within the general health care system. This might explain why these individuals never utilise the school health system. Since the data from the subjects with few measurements are available to us, they can either be included or excluded when creating reference data, depending on the purpose.

For boys at age 16.0 y there were no statistically significant differences in height or weight between those for whom measurements from age 19.0 y were available, and those for whom measurements at this age were unavailable. Therefore, we have no evidence of internal selection at age 19.0 y, despite the low number of measurements, much lower than for age 18.0 y and 18.5 y.

Analogous results were obtained for girls in comparing the height at age 14.5 y of those who were and those who were not measured at age 18.5 y. For BMI there was, however, a significant difference at age 16.0 y between those girls who were and those who were not measured for BMI at age 18.5 y. The mean BMI at age 16.0 y for those who, at a higher age, were not measured is 0.5 units higher than for those who were measured at age 18.5 y. One interpretation of this finding is that internally missing cases are selective and probably more likely for overweight or obese subjects. Therefore the mean BMI for girls of age 18.5 y should be considered to be underestimated.

Tests of internal selection bias concerning head circumference was performed. Fewer measurements were made at age 12 mo and above for both boys and girls. To determine whether this fact could influence the analysis, we compared the 10 mo measurements for those who did not have measurements at 12 mo, 18 mo, 24 mo, 36 mo and 48 mo respectively with those who did have measurements at these ages. The differences in the 10 mo means were +0.11 cm, -0.05 cm, +0.03 cm, +0.13 cm and +0.05 cm. We also analysed a possible correlation between head circumference at birth and age at final measurement. The correlation was close to 0, and there was no evidence that those with very low or very high values at birth were observed for a longer period.
5.1.3 Data quality

Thirdly, the quality of data in the present study is acceptable for growth monitoring among school children. Dealing with data not primarily produced for a scientific purpose raises some important questions about data quality. What is the reliability and validity of measurements carried out in schools? Even though school nurses are well trained for measuring height and weight, their actual measuring practice may not be well controlled and the records imprecise [Strandberg 2001]. In their daily work, nurses are most concerned with identifying abnormal growth patterns. Many pupils, especially the older ones, do not want to undress when they are examined, although nurses are instructed to weigh the pupils in form grade 11 in their underwear. However, we believe that measurements are taken with the subject barefoot. At military conscription all males are measured barefoot, wearing only underpants. It is therefore very important to make comparisons at age 18 years between the means in this study and the data from the conscription material. For weight, there is a difference showing that on average, the males in the study set are heavier. One explanation could be that the MSCR has excluded more obese individuals (they do not attend military conscription). Another explanation could be that at military conscription boys are always measured in underpants, but in school there are some individuals who are measured clothed.

In conclusion this representative sample with few missing individuals with well-defined exclusion criteria can be used to create accurate growth charts, both for descriptive and prescriptive purposes. An advantage of using this material is that this is possible at a fraction of the cost required for large dedicated cross-sectional longitudinal studies of growth.

5.1.4 Comparability

Fourthly it is possible to replicate the sampling procedure, facilitating future comparative studies. The collection of this information is cost-efficient compared with material collected for a specific research project. We have shown, in this thesis, the possibility to repeat surveys with high national coverage and minimal selection bias. This makes it possible to do comparisons that have been done in Paper IV and Paper V.

In order to survey secular changes, in the best of all worlds, the contents of time change should be the only exposure. But what impact has the following known circumstances on health, on growth? Cohort 73 versus cohort 81, the size of the birth cohort (109 663 versus 94 445), the average age of the mother (26.5 y versus 28.0 y) and rate of breastfeeding (>6 mo 7% versus 25.6%).
5.2 COMPARISON TO OTHER SURVEYS

5.2.1 In Sweden

Height and weight

We compared data of cohort 1973 with the prospective study by Karlberg (K) [Karlberg 1976], used as national reference material from 1973 to 2000, and with the second reference dataset reported by Albertsson-Wikland (A-W) [Albertsson-Wikland 2002], that was used as national reference material from 2001. Table 5 Paper I shows differences in summary statistics between the school record material and the studies by (K) and (A-W).

Height
The study by (K) showed lower means for height at all ages for both sexes compared with our data. The means for height in (A-W) are higher at every age than those produced using our school records (the amount of increase 0.7 to 1.7 cm). Mean heights for boys in (A-W) at age 18 years (180.4 cm) are higher than means at age 18 years at any time in the MSCR. It has been reported that all cohorts born between 1953 and 1981 represented in the MSCR have means less than or equal to 179.5 cm [Rasmussen 1995 and 2000]. Comparisons for SD, skewness as well as kurtosis in the three studies reveal no substantial differences between them all.

Weight
The study by (K) shows lower means for weight at all ages compared with the present school record material, with a difference for 7-year old males of 1.4 kg, with a difference of 5.2 kg at age 16 years. The differences between the school record material and (A-W) are in both directions and no particular pattern can be found, for both sexes. For variability within age groups, measured by SD, the school record data have, in general, somewhat higher values than the other two data sets. In particular boys show higher values.

The present school record data show skewness similar to the study of (K) at young ages for both sexes, but for adolescents the values in the present school record study are higher. For males of all ages skewness is lower in the present study than in the study of (A-W). For girls there are no differences or only somewhat higher values in our data compared with (A-W). The values that differ most between the three studies are those for kurtosis. For males aged 7 to 12 years, kurtosis is much higher in both our study and (K) compared with (A-W). The differences at ages 13 y to 16 y between (K) and (A-W) are smaller and in both directions. Values in our data for this age group are higher compared with both (K) and (A-W). For females, the values tend to be higher in our study compared with both (K) and (A-W).

However, mean height for males at age 18 years is higher in the reference study from the same time period presented by (A-W). This could be due to exclusion of some individuals in their study, thus introducing potential selection bias. Also, their sampling frame was restricted to the urban area of a big city and urban populations are reported to deviate both in height and weight compared with other areas (urban individuals are taller and less heavy) [Rasmussen 1995]. Moreover, skewness and
Kurtosis for weight were slightly higher in the present study compared with (A-W). Our experience is that exclusion of more obese individuals has a profound effect on skewness, and in particular on kurtosis, and the individuals who failed to attend the last investigation at school (319 females and 304 males) in the (A-W) study might have been more over-weight or obese than the investigated group.

**Head circumference, comparison between cohort 1981 and Swedish surveys**

Figure 2 in paper III presents comparisons, for girls, between findings of the present study and three Swedish reference materials [Karlberg 1976, Lindgren 1994, Albertsson-Wikland 2002].

The means at all ages from the present study are markedly higher than those from the study of Karlberg [Karlberg 1976], higher from age 24 mo to 48 mo compared to the study of Lindgren [Lindgren 1994], and lower at birth but somewhat higher from age 3 mo to 18 mo but similar from age 18 mo to 36 mo compared to the study of Albertsson-Wikland (A-W) [Albertsson-Wikland 2002]. Notably, the reference currently in use in Sweden, based on the study by A-W, states the mean circumference at birth for girls to be 34.9 cm. This is higher than the mean values for the "healthy" population studied by Niklasson [Niklasson 1991], i.e. mean head circumferences for girls of 34.7 cm, and higher than the means in MBR for those born before 1974, i.e. circumference for girls of 34.2. As mentioned earlier, the values from the present study correspond well with data from MBR. The most pronounced differences between our study and the study by (A-W) are skewness and kurtosis for birth. We report skewness of -0.3 for boys and -0.7 for girls, while their study says 2.08 and 2.74, and we report kurtosis of 0.7 and 3.1, while their study states 14.52 and 22.56. These figures from (A-W) are remarkably high and indicate presence of extremes or outliers.

The differences between the former Swedish reference values and those of this study are large, most likely due to the equipment used to measure the head circumference. In the study of Karlberg [Karlberg 1976] a narrow (0.6 cm) metal tape was used with firm tension applied when measuring to minimize the influence of hair and subcutaneous tissue; this produced lower values than those measured with a wider, plastic–linen tape, commonly rather loosely applied. Comparison with the current Swedish reference is interesting, because its data and those of the present study are comparable in one important respect: both datasets are based on measurements collected from existing records, so the measurement routines and techniques and the documentation methods are likely to be similar. Thus the differences could probably be explained as either due to different sampling frames (national versus local), different selection criteria (none versus some specified criteria), or a mixture of both these factors. In the current Swedish reference material just 37.7% of the population (1 381 out of 3 650) is represented with values for head circumference at birth. Reasons for this are not presented which makes it hard to evaluate the result of a comparison.

**Comparison of head circumference in the present study with other reference materials**

Figure 1 in paper III presents comparisons, for boys, between findings of the present study and reference materials from other parts of the world, including both pooled and

The Dutch study [Fredriks 2000] has approximately 0.5 lower values at all ages than the present study. Euro-Growth [Haschke 2000] values are lower at all ages, a small difference at low ages and more markedly from age 12 mo and up. The values from the CDC charts are higher from birth to age 9 mo, but from age 12 mo to 48 mo the values are more than 1.0 cm lower. The CDC charts for age 10 mo and older have lower values than those from all other materials, except the Chinese [Zhang 1988] and Cuban data [Jordan 1975]. The values from both Cuba and China are much lower from age 3 mo and up than those from the present study. Comparison not found in the figure show that the Norwegian reference [Knudtzon 1988] has higher values at all ages except at age 48 mo than the present study. The agreement between values at ages 36 and 48 mo from the present study and those from UK90 [Cole 1998] is good, but not in the case of birth head circumference, which is 1.0 cm higher in UK90.

**Height and weight, comparison between cohort 1981 and the current Swedish reference values**

For the present study the percentage differences from the reference values are shown in Figure 3 Paper II both without and with exclusions, i.e. for children with birthweight <2 500 g, those suffering from chronic disease of major impairment for growth and those born outside Sweden. These subgroups, among others, are excluded in the current Swedish reference material. For both boys and girls, height in the present study is somewhat lower before age 4 y, and then again somewhat lower from age 7 y up to age 18 y for values calculated without exclusions. Exclusions increase the differences for height, so from age 5 y to age 13 y the values in the present study are somewhat higher but from age 14 y to 18 y they are almost the same as in the current Swedish reference.

The major differences appear when comparing BMI values. The big difference at age 3 mo might depend on a miscalculation in the current Swedish reference material [He 2000]. We reached this conclusion by recalculating the BMI for 3 mo by using the means for height and weight at age 3 mo (0.25 y) from the same material [Albertsson-Wikland 2002], which could explain the difference of 6–7 percent. For both boys and girls the BMI values follow the same pattern. From about age 6 y and up to age 18 y for boys and from age 6 y up to 17 y for girls the differences show 1 to 5% higher values in the present study.

**Comparison between cohort 1981 and other nationwide descriptive studies**

We have compared the present study with some of the descriptive studies previously mentioned, although these studies do not always cover the same birth years. The studies in question are from Sweden [Lindgren 1986], UK [Hughes 1997], the Netherlands [Roede 2000, Fredriks 2000], China [Zhang 1988], and Cuba [Jordan 1975]. The differences in findings between these studies and the present study with regard to males are presented in Figure 1 Paper II (height) and Figure 2 Paper II (BMI). Means for height are presented, but not for BMI, with its more skewed distribution; median values, when available, are presented for some studies. Heights and BMI values found in the present study were higher than those found in the earlier Swedish study, and also higher than those found in the survey from UK;
heights found in the present study were lower and BMI was higher than in the Dutch survey, heights from age 1.5 y were higher than in the Chinese study, and heights were much higher than in the Cuban study.

**A comparison between present study, cohort 1981, and WHO Child Growth Standards**

The MGRS (1997–2003) was a population-based study covering the cities of Davis, California, USA; Muscat, Oman; Oslo, Norway; and Pelotas, Brazil; and selected affluent neighbourhoods of Accra, Ghana, and South Delhi, India. Longitudinal data from birth to 24 mo and cross-sectional study of infants aged 18 to 71 mo. Individual inclusion criteria were: the absence of health or environmental constraints on growth, mothers willing to follow MGRS feeding recommendations (i.e. exclusive or predominant breastfeeding for at least 4 mo; introduction of complementary foods by the age of 6 mo; partial breastfeeding continued for at least 12 mo), no maternal smoking before and after delivery, single term birth, and absence of significant morbidity [WHO 2006 p.56–65].

**Table 9. Comparison between cohort 1981, boys+girls, with exclusions and WHO Child Growth Standards, boys+girls, pooled values and highest mean length for a single country within the pool.**

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>Cohort 1981</th>
<th>n</th>
<th>WHO Pooled</th>
<th>n</th>
<th>WHO Max.mean</th>
<th>n</th>
<th>Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>50.6</td>
<td>2711</td>
<td>49.5</td>
<td>1742</td>
<td>50.4</td>
<td>300</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>67.1</td>
<td>2023</td>
<td>66.7</td>
<td>1648</td>
<td>67.9</td>
<td>286</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>75.5</td>
<td>1106</td>
<td>75.0</td>
<td>1594</td>
<td>75.5</td>
<td>272</td>
<td>No</td>
</tr>
<tr>
<td>18</td>
<td>82.4</td>
<td>2150</td>
<td>81.8</td>
<td>1535</td>
<td>82.4</td>
<td>285</td>
<td>Bra</td>
</tr>
<tr>
<td>24</td>
<td>87.4</td>
<td>849</td>
<td>87.4</td>
<td>1524</td>
<td>88.3</td>
<td>280</td>
<td>Bra</td>
</tr>
<tr>
<td>48</td>
<td>104.1</td>
<td>2009</td>
<td>103.5</td>
<td>478</td>
<td>104.9</td>
<td>71</td>
<td>Bra</td>
</tr>
<tr>
<td>60</td>
<td>111.7</td>
<td>376</td>
<td>110.3</td>
<td>465</td>
<td>112.5</td>
<td>76</td>
<td>Gha</td>
</tr>
</tbody>
</table>

No=Norway, Bra=Brazil, Gha=Ghana

Unfortunately, MGRS data are not presented for boys and girls separately. The comparison reveals that means for cohort 1981 after exclusions are very close to those of the country with highest means for ages 0 to 18 mo. The same value for cohort 81 as the pooled value at age 24 mo (Brazil is 0.9 to 1.1 cm higher). At age 48 mo cohort 81 has 0.9 cm higher value than pooled value and 0.7 cm lower than Brazil. At age 60 it is hard to compare because of low numbers. For cohort 1981 there is possible to make comparison with those who were breast-fed according to the inclusion criteria made by MGRS. The result of these analyses is not published yet but shows a positive impact for gaining length for those who are breast-fed.
General remarks

It is hard to compare apples with pears. When there is a national representative study (Cuba, China) to compare with, it is either from another time or a cross-sectional survey (The Netherlands, China), or within Sweden, selective samples or local samples. Anyhow, the comparisons with socially selective and pooled material (WHO), you can get an idea of the differences and to what extent they can be explained. But the question about comparability remains.
6. GENERAL CONCLUSIONS

Paper I: Longitudinal data for somatic growth (height and weight) from age 7 to 18 years from a nationally representative sample of children in Sweden, collected from school health records, can be used for epidemiological monitoring of growth with fewer missing individuals and at lower costs compared with other dedicated studies. Data quality is comparable to similar national surveys. The data are suitable for descriptive analysis of growth and other forms of observational study.

Paper II: This study represents, without selection bias, the current growth situation among children and adolescents, enabling both epidemiological comparisons over time and comparisons with other national surveys.

Paper III: For the first time in Sweden, and without selection bias, means and distribution of head circumference measurements are documented longitudinally for a nationally representative sample of infants.

Paper IV: No previous studies have shown, to the best of our knowledge, an increasing rate of weight loss, among schoolchildren, but some have shown an increasing rate of weight concerns. However, in this study, a significant increase was found, for both males and females, most marked for females. In the lowest age interval the increase was strongest. The increasing rate is the result of an addition of weight loss both from those who are overweight, and those who are normal, or underweight individuals.

Paper V: National representative longitudinal data for BMI from two cohorts, where non-response bias are minimized, made it possible to monitor and reveal a nation-wide strong positive secular change for BMI in Sweden, over a period of 8 year for individuals aged 7 y to 18 y.

Summary conclusion

In order to monitor changes in growth patterns, among children and adolescents in a huge population, it is important to have data that are representative at several main points. With this study we have achieved representativity at several main points, and we can conclude that there still is a visible positive secular change for height both for boys and girls in Sweden today, as well as a marked positive change in BMI for both boys and girls from age 7 to 18 y. At the same time it is an increasing rate of weight loss especially among girls.

Multiple comparisons with other surveys reveal that it is hard to achieve comparability and those rare surveys that are national representative are old and therefore not easy to compare with.

It is possible to monitor a national situation by collected data from child health records and school health records and more cost-effective than to perform a dedicated study with the aim to present a national situation for growth.
7. CONSIDERATIONS FOR FURTHER INVESTIGATIONS

The materials within this thesis are further analysed, ongoing or planned as follows:

2. From description to prescription. In a suitable way depending on purposes transform the description of cohort 1981 to growth standards (height, weight and head circumference).


4. Analyse the growth pattern of the exclusively breast-fed children.

5. Analyse the impact of breastfeeding on both height and weight longitudinally.

6. Analyse growth and migration within Sweden and to Sweden.

7. Analyse healthy or unhealthy behaviour among those who lose weight.

8. Look further into aspects of parametric analysis of longitudinal growth data with irregular spacing in time [Lundholm 2007].


Gruppen med barn födda 1973 utgörs av alla födda den 15:e i varje månad (15-födda), 3 579 pojkar och flickor. Uppgifter om längd och vikt hämtades från skolhälsovårdsjournaler. Uppgifter saknas för 4.5%.

Gruppen med barn födda 1981 utgörs av alla 15-födda, 3 158 pojkar och flickor. Uppgifter om längd, vikt och huvudomfång hämtades ur barnhälsovårdsjournaler samt dessutom längd- och viktuppgifter ur skolhälsovårdsjournaler. Uppgifter saknas för 1.6%.

Longitudinella data för längd och vikt från födelsen till 19 års ålder samt huvudomfångsuppgifter från födelsen till 4 års ålder från nationellt representativa urval av barn insamlade på detta sätt kan användas till epidemiologisk bevakning av tillväxtförhållanden med marginella bortfallsproblem, jämförbar kvalitet och till en lägre kostnad jämfört med studier baserade på speciellt vägda och mätta barn och ungdomar.

De nu aktuella studierna representerar med ett minimalt bortfall den aktuella tillväxtsituationen och möjliggör jämförelser över tid och jämförelser med andra nationella tillväxtundersökningar.

Analyser av förändringar över tid (8 år) visar att en ökad frekvens av relativa viktnedgångar kan konstateras både för pojkar och för flickor. Ökningen är mest uttalad för flickor, en ökning som sker från en högre frekvens än för pojkar. För flickor ses ökningen i nästan alla kroppsviktskategorier och i alla åldrar, 7 till 18 år. För pojkar ökar viktnedgångsfrekenensen i alla kroppsviktskategorier i åldern 7 till 9 år, men det är en i övrigt mer heterogen bild.

Kroppsvikt och relativ viktnedgång var starkt korrelerad i bägge kohorterna då fler av de överviktiga än av de smala barnen och ungdomarna minskade sin relativa vikt. För flickor var ökningen av viktminskning starkast bland de smalaste individerna.

Från 7 års ålder och upp till 18 år existerar en stark positiv sekulär förändring i body mass index (BMI) i alla åldrar, och frekvensen övervikt och obesitas ökar både för pojkar och för flickor. Dessutom blir obesitas mer allvarlig.
9. ACKNOWLEDGEMENTS

From the very first steps taken in this thesis until the end 19 years later, there are many to whom I am very grateful. Some have been important since they were there at the very beginning, some have been important during the whole trajectory and some have been important in the process of reaching a final solution. Some have always been important. I thank you all.

From the national ambition in 1988, started and inspired by Gösta Samuelsson, I was included in the reference and project group for realizing this project: To create a data base for somatic growth for preschool ages (cohort born in 1981) and in school ages (cohort 1973) with the double aim to make a description of growth in Sweden and to construct a base for creating "normal" values for growth. Since the project group came to an end in 1990, I have been aware of "the loneliness of the long distance runner". I have been surfing on the work of Annika Strandell and Karin Grundberg and also the work of Gunilla Westin-Lindgren.

Bengt-Erik Ginsburg from the National Board of Health and Welfare was a strong support. When the main institution for the project, the Skolöverstyrelse, was reconstructed, it was a major setback. But the show had to go on. Through a very kind support by Örebro County Council, by supplying resources within the Department of Public Health and Community Medicine (Samhällsmedicinska enheten, SME) and within the Division of Child Health Care (Barnhälsovårdsenheten, BHVE), I did manage to keep on collecting data and register it and finally succeeded in fulfilling this project. Many thanks to Sven Larsson, Lena Kördel, Margareta Zander, Peggie Lönnern and most of all to Lars Ekholm (SME) and Leif Ekholm (BHVE). By giving our group both moral and financial support EpC Socialstyrelsen, through Anders Ericson and Petra Otterblad-Olausson, acted as door openers in the long process of data collection. My bosses, during the main part of this period within Örebro County Council, were Ulf Marcusson and Peter Baeckström. I am also very grateful to two very important persons within the County council, Raul Björk and Mats Lindstrøm, in order to maintain the perspective of monitoring public health and make scientific knowledge a foundation for wise political decisions.

Lennart Bodin, my friend on the 19 years’ journey and my co-author. He is not just a friend but a giant of statistics. Which is fortunate, since I am but a pygmy of the above mentioned, possessing only rudimentary skills in data-processing and statistical analysis. Without the co-operation of Lennart this thesis would never have been finished.

Lars Ekholm, my close friend who has been involved in the logistics of all my data collections throughout the years, and finally I succeeded in bringing one project to an end. With a never-ending patience he has kept in order all the quarter of a million data-points that are collected in this project.

Leif Svanstrøm, the head of Division of Social Medicine at Karolinska Institutet. From 1982, when I first became a doctoral student, he has supported and encouraged me.
Gösta Samuelsson, one of the initiators of a national data collection of height and weight. A member of the society of auxologists and a strong supporter of our project. I thank him with all my heart.

Bengt Erik Ginsburg, one of the initiators of the project, at that time representing the National Board of Health and Welfare. He has been supporting our group far beyond his responsibility.

Annika Strandell, former head of the Skolöverstyrelse and the one who was mainly responsible for the first data collection wave. Together with Bengt Erik she was responsible for the start of this project. I miss the Skolöverstyrelse, and I miss working with Annika in a longitudinal perspective.

John Taranger, friend and an auxological guru. Left the active auxiological science after reporting from the Solna-study. Fortunately he kept on being a very skilled auxological speaking partner for me.

Anders Ericson, the late head of EpC at Socialstyrelsen. In the turbulent world of national epidemiological surveillance of growth in Sweden, he was a strong support, as long as he was among us, to our project though the years. We have received financial support by EpC.

Sven Bremberg, my tutor. He came to my rescue when my former tutor moved to Denmark three years ago.

Anders Magnuson, statistician and co-author in paper IV. We have tested models and analysis of this paper during one year and re-written the paper hundreds of times. Thank you for your patience with me.

Finn Diderichsen, an old friend, and though he was not in this field of paediatric auxological he helped me as a tutor until he left for Denmark.

Sven Larsson, at that time when data collection started in 1989 as the head of Department of Public Health and Community Medicine in Örebro, let resources within the department work with the project. I am deeply thankful.

Peggie Lönggren, registered the first half of the amount of data with a remarkable precision, during 1989 and 1992.

Tove Werner, registered data in the end of data collection.

Moa-Lisa Björk, translating and helpful in the processing from a premature manuscript to a readable book.

Joel Björk-Werner, registered data in the end of the project.

Tove, Moa and Joel all belong to my loving growing people.

All school nurses, the heroes in every day life helping us to collect data from school health records all over the country.

All responsible working in the archives in the communities and County councils.

Petra Otterblad Olausson, for helping with MBR data.

Pliktverket in Karlstad, for help with conscript data.

Karin Grundberg, together with Annika Starndell responsible for the first data collection wave

Staffan Mjönes, member of the first planning group.

Gunilla Westin-Lindgren, member of the first planning group.

Örebro County Council, financial support and helping me to work with this project.
10. REFERENCES


Anonymous. A survey on the physical growth of children under 7 years in the urban and suburban rural areas of nine cities of China in 1995. Chung-Hua I Hsueh Tsai Chih (Chinese Medical Journal) 1998;78:187-91


Arnesen E, Forsdahl A. The Tromso heart study: cormary risk factors and their association with living conditions during childhood. J Epidemiol Community Health 1985;39(3):210-4


Ashcroft MT, Heneage P, Lovell HA. Heights and weights of Jamaican schoolchildren of various ethnic groups. Am J Phys Anthropol 1966;24:35-44


Barker DJP, Martyn CN, Osmond C, Hales CN, Fall CHD. Growth in utero and serum cholesterol concentrations in adult life. BMJ 1993;307:1524-7


Bateson P. Fetal experience and good adult design. Int J Epidemiol 2001;30:928-34


Blomquist HK, Bergström E. Obesity in 4-year-old children more prevalent in girls and in municipalities with a low socioeconomic level. Acta Paediatr 2007;96:113-6
Boas F. The growth of children, II. Science 1892;19:281-2
Boas F. Changes in the bodily form of descendants of immigrants. Am Anthropol 1912;14:530-63
Bodin L. Personnel communication
Brieger GM, Rogers MS, Rushton AW, Mongeli M. Are Hong Kong babies getting bigger? Int J Gynaecol Obstet 1997;57(3):267-71


Camerer W. Untersuchungen uber Massenwachstum und Längenwachstum der Kinder. Jahrbuch fur Kinderheilkunde 1893;18:254-64


Chinn S, Rona RT. Prevalence and trends in overweight and obesity in three cross sectional studies of British children, 1974-94. BMJ 2001; 322: 24-6


Cliquet RL. Social mobility and the anthropological structure of populations. Hum Biol 1968; 40: 17-43


Department of Health. Health inequalities: Health Secretary announces new plans to improve health in poorest areas. 28th February, 2001 URL: http://www.doh.gov.uk/healthinequalities/press.htm


Dietz WH, Jr., Hartung R. Changes in height velocity of obese preadolescents during weight reduction. AJDC 1985;139:705-7


Dovertie Hygiae 1895;57:254

Dovertie 1914 (Cited in Törnell 1920)


Dössing J. Gennemsnitsvæder for vægt-højde-alder for drenga og piger i skole-åldern. (Means for weight-height-age relations for boys and girls of school age). Ugeskr Laeger 1950;112:1171-81 (In Danish)


Fogel RW. New sources and new techniques for the study of secular trends in nutritional status, health, mortality, and the process of aging. Historical Methods 1993;26:5-43


Forsum E, Boström K, Eriksson B, Olin-Skoglund S. Kvinnans vikt före och under graviditet har betydelse för barne. (A woman’s weight before and during pregnancy is of importance to her infant). Läkartidningen 2003;100:3954-8 (In Swedish)


Fredriks AM, van Buuren S, Jeurissen SER, Dekker FW, Verloove-Vanhorick SP, Wit JM. Height, weight, body mass index and pubertal development references for children of Turkish origin in the Netherlands. Eur J Ped 2003;162:788-93


Fredriks AM, van Buuren S, Hira Sing RA, Wit JM, Verloove-Vanhorick SP. Alarming prevalences of overweight and obesity for children of Turkish, Moroccan and Dutch origin in the Netherlands according to international standards. Acta Paediatr 2005;94:496-8


Frisch RE, Revelle R. Height and weight at menarche and a hypothesis of critical body weights and adolescent events. Science 1970;169:397-9


Goldstein MS. Demographic and Bodily Changes in Descendants of Mexican Immigrants. Austin: Institute of Latin-American Studies,1943

Gunarsdottir I, Thorsdottir I. Relationship between growth and feeding in infancy and body mass index at the age of 6 years. Int J Obes 2003;27:1523-7
Hall RL. Sexual dimorphism for size in seven nineteenth century Northwest Coast populations. Hum Biol 1978;50:159-71
Hensley W, Cooper R. Height and occupational success: a review and critique. Psychological Reports 1987;60:843-9
Hermanussen M, Burmeister J, Burkhardt V. Stature and stature distribution in recent West German and historic samples of Italian and Dutch conscripts. Am J Hum Biol 1995;7:507-13


Hill AJ. Pre-adolescent dieting: implications for eating disorders. Int Rev Psychiatry 1993;5:87-100


Hultcrantz JV. Uber die Körperlänge der schwedischen Wehrpflichtigen. Centralblatt fur Anthropologie 1896 årg.1 nr 4


Johnston FE. Control of age at menarche. Hum Biol 1974;46:159-71


Jordan JR. Desarrollo Humano en Cuba. Instituto de la Infancia. La Habana Cuba: Editorial Cientifico-Tecnica; 1979 (In Spanish with translation in English))


Kaplan HI, Kaplan HS. The psychosomatic concept of obesity. J Nerv Ment Dis 1957;125:181-201


Key A. Redogörelse för den hygieniska undersökningen. Bilaga E till Läroverkskomitens underdåniga utlåtande och förslag angående organisationen af rikets allmänna läroverk och dermed sammanhängande frågor. (The School Committee’s humble report and proposals for the organisation of the Secondary Schools and thereto related questions). Stockholm,1885 (In Swedish)


Kjerrulf H. Om s.k. harmonisk vikt och dess praktiska betydelse. Svenska Läkartidningen 1924;46:1125-33 (In Swedish)
Kjerrulf H. Resultat från mätningar och vägningar februari 1927 i Adolf Fredriks folkskola, Stockholm. Svenska Läkartidningen 1927;24:323-5 (In Swedish)


Larnkjaer A, et al. Secular trend in adult stature has come to a halt in northern Europe and Italy. Acta Paediatr 2006;95:754-5


Laska-Mierzejewska T, Milicer H, Piechaczek H. Age at menarche and its secular trend in urban and rural girls in Poland. Ann Hum Biol 1982;9:227-33

Lasker GW. Environmental growth factors and selective migration. Hum Biol 1952;24:262-89


Lindgren G, Aurelius G, Tanner J, Healy M. Standards for height, weight and head circumference from one month to six years based on Stockholm children born in 1980 Acta Paediatr 1994;83:360-6

Lindgren G, Strandell A, Cole T, Healy M, Tanner J. Swedish population reference standards for height, weight, and body mass index attained at 6 to 16 years (girls) or 19 years (boys). Acta Paediatr 1995;84:1019-28
Lissau I, Sørensen TI. School difficulties in childhood and risk of overweight and obesity in young adulthood: a ten year prospective population study. Int J Obes 1993;17:169-75


Liu JP, Baker J, Perkins AS, et al. Mice carrying null mutations of the genes encoding insulin-like growth factor I (Igf-I) and type 1 IGF receptor (Igf1r). Cell 1993;75:59-72


Ljunggren CA. Bidrag till kännedom om svenska folkskolebarns standardvikt. Nord Hyg Tidskr 1925;6:241-56 (In Swedish)


Loh ES. The economic effects of physical appearance. Social Science Quarterly 1993;74:420-38


Macbeth HM, Boyce AJ. Anthropometric variation between migrants and non-migrants: Orkney Islands, Scotland. Ann Hum Biol 1987;14:405-14

McCabe MP, Ricciardelli LA. Parent, peer, and media influences on body image and strategies to both increase and decrease body size among adolescent boys and girls. Adolescence 2001;36:225-40


Marmot MG, Smith GD. Why are the Japanese living longer? BMJ 1989;299:1547-51
Marschall WA. Evaluation of growth rate in height over periods of less than one year. Arch Dis Child 1971;46:414-20
MBR, The Swedish Medical Birth Register, National Board of Health and Welfare, Sweden (Medicinsk födelseregister EPC Socialstyrelsen) URL: www.sos.se/epc/fodelse/mfr
Meeuwisse G, Otterblad Olausson P. Allt större andel nyfödda väger över fyra kilo. (Increasing birthweights in the Nordic countries; a growing proportion of neonates weight over four kg) Läkartidningen 1998;95(48):5488-92 (In Swedish)
Mellbin T, Vuille J-C. Further evidence of an association between psychosocial problems and increase in relative weight between 7 and 10 years of age. Acta Paediatr Scand 1989;78:576-80
Meredith HV. Comparative findings on body size of children and youths living at urban centers and in rural areas. Growth 1979;43:95-104
Meredith HV. Body size of infants and children around the world in relation to socio-economic status. Advances in child development and behaviour 1984;18:81-145
MSCR, The Swedish Military Conscript Registry, Piktverket Karlstad, Sweden (Mönstringsregistret, Piktverket Karlstad) URL: www.piktverket.se
Norinder I. Bidrag till kännedomen om folkskolebarns kroppsutveckling och hälsotillstånd. (Contribution to the knowledge of body development and state of health of elementary schoolchildren). Hygiae 1907;69:1199-1205 (In Swedish)


Orr JB. Milk consumption and the growth of schoolchildren. Lancet 1928;1:202-3


Penrose LS. Some recent trends in human genetics. Caryologia 1954;Suppl.6:521-30


Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? BMJ 1995;311:437-9


Quetelet A. Sur l’homme et Anthropométrie. Brussel, 1870


Rogers IS, Emmett PM, Golding J. The growth and nutritional status of the breast-fed infant. Early Hum Dev 1997;49;S157- 74


Rolland-Cachera MF, Bellisle F. No correlation between adiposity and food intake: why are working class children fatter? Am J Clin Nutr 1986;44:779-87


Rona RJ, Chinn S. School meals, school milk and height of primary schoolchildren in the eighties. J Epidemiol Community Health 1989;43:66-71


Sandberg LG, Steckel RH. Soldier, soldier what made you grow so tall? Economy and History 1980;23-81


Shapiro HL. Migration and environment. A study of the physical characteristics of the Japanese immigrants to Hawaii and the effects of environment on their descendants. New York: Oxford University Press, 1939
Socialstyrelsen Amningsstatistik. (Official statistics of breast-feeding in Sweden). Available athttp://www.socialstyrelsen.se (In Swedish with summary in English)
Spitz RA. Hospitalism: an inquiry into the genesis of psychiatric conditions in early childhood. Psychoanal Study Child 1945;1:53-74
Steegmann AT, Jr. 18th century British military staure: growth cessation, selective recruiting, secular trends, nutrition at birth, cold and occupation. Hum Biol 1985;57:77-95
Steenhoff G. Bidrag till kännedomen om vår folkskolehygien. Hygiae1900;62:145-65
Strandberg U, Bergfors G, Blomquist HK. Även erfarna distriktssköterskor får olika mätvärden. (Measurements of height, weight and head circumference in child health services. Even experienced nurses obtain divergent results). Lakartidningen 2001;98:2330-3 (In Swedish)
Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body mass index of twins who have been reared apart. N Engl J Med 1990;322:1483-7
Susanne C. Interrelations between some social and familial factors and stature and weight of young Belgian male adults. Hum Biol 1980;52:701-9

Sveger T. Does overnutrition or obesity during the first year affect weight at age four? Acta Paediatr Scand 1978;67:465-7

Symonds ME, Budge H, Stephenson T. Limitations of models used to examine the influence of nutrition during pregnancy and adult disease. Arch Dis Child 2000;83:215-9


Tanner JM. Foetus into Man: Physical growth from conception to maturity. Ware: Castlemead Publications,1989
Thoday JM. Geneticism and environmentalism, in Meade JE and Parkes AS. 1965.
Udjus LG. Anthropometrical changes in Norwegian men in the twentieth century. The Anatomical Institute, Anthropological Department, University of Oslo and the Armed Forces Medical Services. Oslo: Universitetsforlaget,1964
Vahl M. Mitteilungen ueber das Gewicht nicht erwachsener Mädeln (1874-1883). Vorsteher der Erziehungs-Anstalt zu Jägerspis. Copenhagen, 1884 (fördrag vid läkarkongress i Köpenhamn 1884)


Wadsworth ME, Kuh DJ. Childhood influences on adult health: a review of recent work from the British 1946 national birth cohort study, the MRC National Survey of Health and Development. Paediatr Perinat Epidemiol 1997;11(1):2-20


Wainwright RL. Change in observed birth weight association with change in maternal cigarette smoking. Am J Epidemiol 1983;117:668-75


Werdelin L. The stature of some medieval Swedish populations. Fornvännen 1985;2:133-41


Werner B, Bodin L. Obesity in Swedish schoolchildren is increasing in both prevalence and severity. 2007 submitted


Whitehead RG, Paul AA. Growth charts and the assessment of infant feeding practices in the western world and in developing countries. Early Hum Dev 1984;9:187-207


Wolanski N. Parent-offspring similarity in body size and proportions. Studies in Human Ecology 1979;3:7-26


Wolanski N, Dickinson F, Siniarska A. Biological traits and living conditions of Maya Indian and non-Maya girls from Merida, Mexico. Int J Anthropol 1993;8:233-46


WHO Child Growth Standards. Acta Paediatr 2006; 95 Suppl 450


Wright CM, Parker L, Lamont D, Craft AW. Implications of childhood obesity for adult health: findings from thousand families cohort study. BMJ 2001; 323: 1280-4

Wretlind EW. Iakttagelser rörande helsotillståndet i några af Göteborgs flickskolor. (Observations of the health situation in some of the schools for girls in Gothenburg). Eira 1878; 2(24): 745-64 (In Swedish)


Åkerman S, Högberg U, Danielsson M. Height, health and nutrition in early modern Sweden. In Brändstöm A, Tedebrand LG. Society, health and population. During the Demographic Transition, 1988