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PREFACE

The increasing prevalence of obesity among children and adolescents has placed the lifestyle of these age groups on the political agenda. An unhealthy lifestyle during childhood and adolescence, in particular unhealthy eating habits and physical inactivity, increases not only the risk of developing obesity but also, along with smoking, the risk of developing chronic diseases later in life. Habits, both healthy and unhealthy, are established during childhood, and there is good reason to believe that measures taken to influence the lifestyle of children at an early age are more effective than subsequent attempts to change habits already established. This gives rise to several important questions that need to be addressed. At what age should interventions begin? How comprehensive should they be? And which tools should be used to promote the establishment of healthy habits among children and adolescents? This report comprises a scientific review of the instruments used by professionals in the scientific community to influence the lifestyle of children and adolescents in order to reduce the risk of heart disease in adult life. The purpose of the report is to provide a basis for policy-making for children and adolescents with regard to diet, physical activity and smoking. The Danish Heart Foundation will comply with this policy in the coming years.

During the preparation of this report, a group of experts have considered, discussed and commented on the content. The group comprised Professor Bjørn Holstein and lecturer Pernille Due, both from the Institute of Public Health, Professor Lars Bo Andersen, MD, Norwegian University of Sport and Physical Education, and research assistant and specialist Karin Helweg-Larsen from the National Institute of Public Health. The Danish Heart Foundation would like to thank the group for their active contribution to the preparation of this review.

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SUMMARY

It is recognised that atherosclerosis originates in childhood and adolescence in a significant part of the population, and there is good evidence to suggest that the well-known lifestyle factors (diet, physical activity and smoking) and risk factors (obesity, high blood pressure, high cholesterol, poor physical fitness) in children and adolescents, exactly as in adults, are associated with the accelerated development of atherosclerosis and a higher risk of cardiovascular disease. Despite some improvements in certain cardiovascular lifestyle factors (decreased intake of fat and increased intake of fruits and vegetables, and decreased smoking) many children and adolescents continue to eat an unhealthy diet and are physically inactive. Moreover, a considerable number of adolescents begin to smoke. The increasing number of fat children is also an expression of an unhealthy lifestyle with increased cardiovascular risk.

Healthy (and unhealthy) habits and the level of cardiovascular risk factors in children and adolescents track into adult life. Despite the evidence of clear tracking, it is encouraging that most children are not "locked" into unhealthy habits. In fact, there are many more children and adolescents who modify their lifestyle and risk factors than there are children who maintain unhealthy habits into adulthood. An important question that needs to be addressed is when, how and why lifestyle habits are established and maintained. Answers to this question will make it possible to direct interventions more effectively to specific target groups.

There is good scientific evidence for the claim that much behaviour (including eating habits, physical activity and smoking habits) is established already during childhood years and becomes increasingly cemented during adolescence and adulthood. All other things being equal therefore, it can be expected that healthy habits acquired early in life will have a significantly higher chance of being maintained, and in the long term reduce the risk of cardiovascular disease. The family is crucial in this respect, and several studies have established that healthy habits in parents and siblings "rub off" on the child.

A closer examination of behaviour reveals that it is governed by a number of specific determinants. These include exogenous factors, such as availability, marketing and advertising, and pricing policies (which are determined by general attitudes in society) and endogenous factors, such as self-efficacy and will-power (which are generally elements of the upbringing process in the immediate social environment).

A large number of broad-based randomised intervention studies have been conducted, many of them in school settings, and most drawing from theoretical models, with the purpose of changing or preventing unhealthy lifestyles among children and adolescents. Overall the results have been good as long as the interventions were ongoing, but follow-up investigations have most often shown a return to status quo. This stresses the need for sustained broad efforts on a national, local and individual level, which build on several strategies (and which also include changing attitudes in society, for example with regard to the significance of unhealthy fast-food, rapidly increasing television viewing and smoking in public places) in order to maintain or re-establish a healthy lifestyle in children and adolescents and thereby reduce the risk of the early development of cardiovascular disease.

INTRODUCTION

Unhealthy dietary habits, (i.e. a high-calorie diet, high intakes of sugar and "hard" fat and low intakes of fruit and vegetables), physical inactivity and cigarette smoking are common among adults in all industrialised societies. Many public health initiatives have aimed at changing this lifestyle among adults. As a person grows older, however, habits become entrenched and more difficult to change. Health promotion programmes should therefore target the young before their habits become too deeply ingrained. Effective promotion of healthy lifestyles early in life is preferable to attempts to modify lifestyle at a later stage in life, when many will already have developed pathological cardiovascular conditions related to unhealthy lifestyles.

If we assume that the following statements are correct, there is good reason to target prevention to children and adolescents:

1. A significant number of children and adolescents have a lifestyle/engage in behaviour which may increase their risk of developing disease later in life (risk behaviour).
2. Risk behaviour among children and adolescents increases the level of somatic/biological risk factors for disease.
3. The presence of somatic/biological risk factors also increases the risk of children and adolescents developing disease.
4. The incidence of risk behaviour and risk factors persists through childhood and adolescent years and tracks into adulthood.
5. Interventions aiming to change the risk behaviour of children and adolescents reduce risk factors for disease.

This review will focus mainly on behaviour-related lifestyle factors among children and adolescents (<18 years) and describe certain of the factors that are associated with risk-contingent lifestyles = risk behaviour. The lifestyle factors for coronary heart disease related to behaviour will be discussed under three main headings: dietary intake, physical activity and cigarette smoking. Somatic/biological risk factors (sometimes called intermediary risk factors) are here defined as the biological markers believed to be causal factors for the development of coronary heart disease, primarily physical fitness, obesity (abdominal; apple-shape), hypertension (elevated blood pressure) and dyslipidaemia (elevated blood fat content). There are a number of other risk factors for the development of coronary heart disease which are not discussed here.

The review will occasionally consider the importance of the intermediary risk factors for the development of coronary heart disease. Findings are presented from a large number of studies, which may clarify relationships between lifestyle and the development of coronary heart disease, and from studies which assess the importance and possibility of intervening in risk-contingent lifestyles in childhood and adolescence. The literature review is not exhaustive, but the most important findings have been included, the emphasis being on prospective studies in human populations.

ATHEROSCLEROSIS

Atherosclerosis is a general term for a number of chronic degenerative changes in the arteries and is characterised by: reduced elasticity of the artery wall, narrowing of the arteries and a greater or lesser tendency towards plaque formation.

Atherosclerosis develops gradually over a long period of time, from the time the first microscopic changes occur in the artery wall until the clinical manifestations of atherosclerosis appear. The pathogenic mechanisms which cause the characteristic changes (hardening of the arteries) have been extensively researched; yet the exact causes of atherosclerosis are not known.

In particular how atherosclerosis begins is not known, but it is perhaps caused by a dysfunction in the innermost layer of the artery (endothelial layer). This dysfunction results in the penetration of lipoproteins, primarily low density lipoprotein (LDL), into the part of the arterial wall known as the intima, i.e. the layer of the arterial wall lying within the innermost cell layer in the artery (endothelium). Changes develop particularly at sites where the artery divides or for other reasons is under considerable mechanical pressure from the bloodstream. The process is believed to involve oxidation of LDL, which attracts white blood cells (macrophages), and which means that LDL cholesterol is more easily taken up and accumulated in the macrophages. As a result of this accumulation of fat, a small deposit is formed in the artery – a fatty streak. Fatty streaks do not penetrate into the artery, produce no symptoms and are harmless to the extent that they can be reversed – and often are completely reversed. The accumulation of the oxidized LDL cholesterol alters the appearance of the macrophages, hence the term foam cells. The formation of foam cells is believed to be a marker for early atherosclerosis (Steinberg 1997).

The macrophages, and now the smooth muscle cells, absorb more and more cholesterol, which may now also be found deposited outside of the cells. A cap of tissue forms around the lesion, surrounding the mixture of cells, fat and debris. At this stage the lesion is called *atherosclerotic plaque* (fibrous plaque or raised lesion). The atherosclerotic plaque is not reversible, and quite often will even increase in size. This may gradually restrict the passage of blood in the artery so much so that it causes symptoms, for instance angina pectoris to occur. The atherosclerotic plaque can also break and come into direct contact with blood. If this occurs, coagulation will occur – and a thrombus will be formed – which can completely block the blood supply. The presence of macrophages and other inflammatory cells (lymphocytes) in the entire process from the early fatty streaks to advanced, complicated lesions has led many to regard atherosclerosis as an infectious disease similar to other chronic infectious conditions (Ross 1999).

The significance of fatty streaks in the development of atherosclerosis

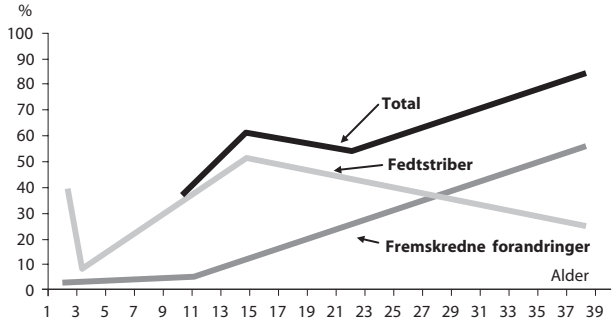
It is unclear whether atherosclerotic plaques originate from fatty streaks or whether plaques occur independently of the presence of fatty streaks. The fact that fatty streaks in children and plaques in adolescents and adults are found at the same sites in the coronary arteries and the aorta may indicate that fatty streaks are an early stage in the development of atherosclerosis (Strong et al. 1999). Morphological studies point in the same direction, and have found a progressive build-up of lesions from the early fatty streaks to plaques related to age and the presence of risk factors (McGill et al. 2000a). The microscopic findings in fatty streaks described above (foam cells and deposits of fat outside cells) also indicate that fatty streaks may be the first step in a process that leads to clinical disease.

However, fatty streaks occur more frequently in aorta than in coronary arteries in children, and are more frequent in girls than in boys, unlike the case of atherosclerosis in adults. Also, fatty streaks occur in children all over the world, regardless of race, sex and diet (Fernandez-Britto et al. 1999; Olson, 2000). These findings do not support the theory that fatty streaks are early atherosclerotic lesions.

Atherosclerosis in children and adolescents

Plaques, and particularly thrombosis, are extremely rare prior to adulthood, but this obviously does not mean that conditions during childhood do not affect the development of plaques and thrombosis later in life. Morphological studies of coronary arteries in children and adolescents have shown that the incidence of fatty streaks gradually increases from 3 to 4 years of age and up through adolescence (Figure 1). An American study, the Pathobiological Determinants of Atherosclerosis in Youth Study (PDAY), found that approx. 60% of adolescents aged 15 to 19 years had visible changes in the coronary arteries, increasing to 70-80% in men and women aged 30 to 34 years (Stary 2000; Strong et al. 1999). These were most often just fatty streaks, particularly among the younger age groups. More progressive lesions begin to appear around puberty, and are present in about a quarter of the 25- to 35-year-olds (Stary 2000; Strong et al. 1999). Similar findings were made by the multinational study, the PathoBiological Determinants of Atherosclerosis in Youth (PBDAY) study, which showed the presence of fatty streaks in the coronary arteries of subjects as young as 5 years of age (Fernadez-Britto et al. 1999).

Figure 1. Percentage of children and adolescents with fatty streaks and more advanced changes dependent on age based on microscopic examinations of coronary arteries. The more advanced changes comprise preatheroma, atheroma and fibroatheroma. Plaque is rare before the age of 40 (modified from Stary 2000).



The first stage in the development of atherosclerosis may occur in the foetus. Pathological studies of children who died before the age of 1 year have shown changes in the coronary arteries compatible with early atherosclerosis (Ikari et al. 1999; Napoli et al. 1999), and studies of humans and animals have established that elevated cholesterol levels in mothers during pregnancy increase the occurrence of foam cells in the coronary arteries of the foetus, and are linked to a faster progression of atherosclerosis early in life (Palinski and Napoli 2002).

Consequences of atherosclerosis

Atherosclerosis when present in the large and medium-sized arteries can reduce or block the supply of blood and oxygen to many organs. It mainly affects, however, the heart, brain and legs. Blockage of the blood supply to the brain can cause stroke and blockage to the legs can cause claudication or gangrene. Blockage of the coronary arteries gives rise to myocardial infarction, and an insufficient supply of oxygen to the heart decreases the heart's ability to pump blood, precipitating heart failure and sometimes arrhythmic disturbances. All of these symptoms are included in the general term ischaemic heart disease.

Conclusion

Atherosclerosis develops over a long period of time, from the time the first microscopic changes occur in the artery wall until the clinical manifestations of atherosclerosis appear, usually in the heart, brain or legs. The origin of atherosclerosis is unclear, in particular the extent to which the presence of fatty streaks in coronary arteries during childhood is a precursor to atherosclerosis later in life. Morphological studies of coronary arteries in children and adolescents have established, however, that the prevalence of fatty streaks gradually increases from 3 to 4 years of age and up through adolescence. Clear atherosclerotic lesions begin to manifest during the teenage years and are present in about a quarter of all 25- to 35-year-olds.

CORRELATION BETWEEN LIFESTYLE, RISK FACTORS AND HEART DISEASE

It was already established about fifty years ago that the likelihood of an individual developing cardiovascular disease could be predicted by assessing relatively few of the individual's characteristics – also called *risk factors* (Table 1) (Misra 2000). Many of these risk factors are interrelated, and several of them have an additive or synergetic effect on the risk.

Table 1. Risk behaviour and somatic/biological (intermediary) risk factors for atherosclerosis and cardiovascular disease.

Risk behaviour

- Smoking
- Inactivity
- Diet composition, e.g. high intake of saturated fat

Intermediary somatic/biological risk factors

- Obesity, especially excess abdominal fat deposits
- Insulin resistance, hyper-insulinaemia and glucose intolerance
- Dyslipidaemia – high total cholesterol level, LDL cholesterol and triglycerid, low HDL cholesterol level, high lipoprotein (a)
- Other factors, e.g. high plasminogen, plasminogen activator inhibitor-1 activity and other factors which increase the risk of thrombosis as well as high C-reactive protein and other inflammatory markers

It is a well-documented fact that a high intake of saturated fat and a low intake of fruit and vegetables as well as dietary fiber, and physical inactivity and smoking are associated with the development of dyslipidaemia, high blood pressure and glucose intolerance. Detection of biological/somatic risk factors and modification of risk behaviour among adults have proven to be quite efficacious for reducing the risk of heart disease (Ammerman et al. 2002; Forrester et al. 1996). This effect can also be expected among children. Three extra

hours of physical education at school was found after eight months to have significantly reduced the systolic and diastolic blood pressure in 9- to 11-year-old school children (Hansen et al. 1991).

The justification for lifestyle interventions among children and adolescents (apart from smoking) has, however, long been debated. Among other things, it was not known whether the presence of risk factors – or the modification of risk factors – in children and adolescents had any effect on the development of atherosclerotic disease several decades later. It is only in the past ten years that the relationship between lifestyle, risk factors and the development of atherosclerosis in children and adolescents has become clearer, along with the significance of this relationship for the development of disease later in life (Daniels 2001).

Relationship between lifestyle and risk factors in children

Cross-sectional studies have shown that a high intake of energy primarily from fat, sugar and fast-food and physical inactivity in children and adolescents is associated with overweight (DuRant et al. 1993; Fogelholm et al. 1999; French et al. 2001; Gazzaniga and Burns 1993; Gillis et al. 2002; Nguyen et al. 1996; Trost et al. 2001). Inactivity and unhealthy eating habits are associated with elevated levels of fat in the blood, elevated insulin levels and high blood pressure (Gutin et al. 1990; Raitakari et al. 1997). Cigarette smoking among children has an effect on a number of other risk factors for heart disease similar to the effect it has in adults (Glueck et al. 1981).

Approximately 20-30% of overweight children have elevated systolic or diastolic blood pressure (Figueroa-Colon et al. 1997). A study of over 13,000 Danish secondary-school pupils showed that both a high body mass index (BMI) and a low level of physical fitness increase the risk of elevated blood pressure, and that the correlation between BMI and blood pressure was closer among adolescents with low levels of fitness (Nielsen and Andersen 2003). As for adults, it is especially abdominal fatness, the so-called "apple" shape that is risky for children and adolescents (Daniels et al. 1999; Gillum 1999; van Lenthe et al. 1998).

A high fat mass – and low physical activity and low physical fitness – in children and adolescents is often associated with metabolic changes compatible with the *metabolic syndrome* – elevated blood pressure and abnormal blood lipids (dyslipidaemia) as well as reduced glucose tolerance and elevated insulin (Andersen et al. 2003; Bergstrøm et al. 1996; DuRant et al. 1993; Freedman et al. 1999; Steinberger et al. 1995). These are factors which, when they coexist, significantly increase the risk of heart disease. The syndrome has been observed in children as young as 5 years of age (Young-Hyman et al. 2001).

Recent findings have shown that many overweight children have increased levels of C-reactive protein in their blood (Ford et al. 2001). An increased level of this protein is directly linked to the risk of cardiovascular disease.

Longitudinal studies indicate that lifestyle has an equally important impact on cardiovascular risk factors among children and adolescents as it has among adults (Berkey et al. 2000). A moderate consumption of tobacco, for example, increases LDL cholesterol and decreases HDL cholesterol (Freedman et al. 1986), a high intake of sugar-sweetened soft drinks increases the risk of obesity (Ludwig et al. 2001), and increased physical activity reduces blood pressure (Hofman and Walter 1989). Most longitudinal studies, however, are of relatively short duration, and rarely follow subjects into adulthood.

One longitudinal study conducted in New Zealand showed that the longer the time children and adolescents spent watching television, the higher were their BMI, serum cholesterol and triglyceride levels, and the poorer was their physical fitness at the age of 26 years; the study did not show, however, an association between television viewing and blood pressure levels (Hancox et al. 2004).

A Danish study measured physical fitness and physical activity as well as a number of cardiovascular risk factors (blood pressure, skinfolds, waist circumference, total cholesterol and triglyceride levels and LDL and HDL cholesterol) among 15- to 19-year-old girls and again 8 years later (Hasselstrøm et al. 2002). Fitness and activity while young were not associated with risk factors in adulthood, but changes in physical fitness from childhood to adulthood was a good predictor of most risk factors in adults and of changes in risk factors from childhood to adulthood. The cohort was characterized by considerable changes in levels of physical fitness in the course of the 8 years the study lasted.

In the American Bogulosa Heart Study, overweight during teenage years (13 to 17 years) was associated with higher systolic and diastolic blood pressure, and higher concentrations of total cholesterol, LDL cholesterol, triglyceride, insulin and glucose 12 to 14 years later (Srinivasan et al. 1996). A Finnish study found that the risk of developing metabolic syndrome was four times greater among overweight children (at 7 years of age) who continued to be overweight as adults compared to those who first became overweight as adults (Vanhala et al. 1999).

A Dutch study, the Amsterdam Growth and Health Longitudinal Study (AGHLS), measured physical fitness and physical activity among 13-year-olds (Twisk et al. 2002). After a 20-year tracking period, the study established that good physical fitness in childhood is related to a healthy risk profile among adults (lower fat mass, waist measurement and total cholesterol, though not HDL cholesterol).

However, not all longitudinal studies have found an association between lifestyle and/or overweight in children and high levels of biological intermediary risk factors in adults. The British Newcastle Thousand Families Study thus failed to report an association between overweight among 13-year-olds and the incidence among 50-year-olds of high systolic and diastolic blood pressure, and concentrations of total cholesterol, HDL cholesterol and LDL cholesterol as well as insulin and glucose (Wright et al. 2001).

Relationship between risk factors and atherosclerosis

In an American multi-centre study, the Pathobiological Determinants of Atherosclerosis in Youth (PDAY), post mortal examinations were performed of the coronary arteries and aortas of almost 3,000 individuals aged 15 to 34 years who had died of causes other than disease (accidents, homicide or suicide). The arterial changes observed were related to the incidence of risk markers for cardiovascular disease measured post mortally. The results of this study for all age groups show that atherosclerosis is more frequent, more widespread and more serious when one or more of the classical risk factors are present – high total cholesterol, high LDL cholesterol, low HDL cholesterol, high blood pressure (measured by changes in the renal arteries) overweight and reduced glucose tolerance (McGill et al. 2000b; McGill et al. 2001; McGill et al. 2002). A similar, but somewhat smaller, study conducted under the auspices of the World Health Organization and the World Heart Federation (WHO/WHF PBDAY Study) covering persons aged 5 to 34 years also demonstrated an increased incidence of atherosclerosis associated with the presence of the established risk factors (Sternby et al. 1999).

Another American study, the Bogulosa Heart Study, included a younger age group of 204 individuals who were aged between 2 and 9 years when they died of various causes, mainly traffic accidents or homicide (Berenson et al. 1998). Of the 204 subjects, 93 had earlier been identified as having cardiovascular risk factors while alive. The study showed that BMI, systolic and diastolic blood pressure, serum lipids and lipoproteins (total cholesterol, triglyceride, LDL cholesterol, HDL cholesterol) and smoking were strongly associated with the formation of fatty streaks and atherosclerotic plaque in the coronary arteries and aorta. The presence of several risk factors was associated with more widespread atherosclerosis. Individuals with 0, 1, 2 and 4 risk factors (high BMI and systolic blood pressure as well as high concentrations of triglyceride and LDL cholesterol) had 1.3%, 2.5%, 7.9% and 11% respectively of their arterial walls covered in fatty streaks. The corresponding figures for plaque were 0.6%, 0.7%, 2.4% and 7.2%.

In recent years, a large number of *non-invasive* methods have been used to study early atherosclerosis in children and adolescents, for example ultrasound measurements of the thickness of the artery wall (the width of the innermost (intima) plus the middle (media) layers of the artery; it is mainly the thickness of the wall of the carotid artery that is measured) or measurements of the elasticity of the artery walls.

Children with several of the cardiovascular risk factors, including high total cholesterol and LDL cholesterol and overweight, have thicker arterial walls when adults than children who do not have these risk factors (Davis et al. 2001; Li et al. 2003). The Finnish study, the Cardiovascular Risk in Young Finns Study, which covered more than 2,000 individuals, demonstrated greater artery wall thickness in adults (24-39 years) who when young (12-18 years) had been measured as having higher LDL cholesterol, systolic blood pressure and BMI and who were smokers, even after the data were adjusted for the incidence in adult life of high LDL cholesterol and systolic blood pressure (Raitakari et al. 2003). The Dutch Atherosclerosis Risk in Young Adults Study, which followed adolescents from about the age of 13 until they were 27-30 years old, also showed that BMI in adolescence is related to intima-media thickness, independent of blood pressure in adolescence, lipid levels and blood pressure in adulthood, but not BMI in adulthood (Oren et al. 2003).

Poor physical fitness in adolescence increases the risk of developing atherosclerosis later in life. A Dutch study has demonstrated that 13- to 16-year-old boys who were physically unfit had greater artery wall thickness when 36 years old (Ferreira et al. 2002), and that improvements in fitness levels during the course of the 30-year study reduced arterial stiffness (Ferreira et al. 2003).

Other studies have shown that obesity, reduced HDL cholesterol and elevated blood pressure in childhood and adolescence are associated with a higher incidence of plaque formation (often a late stage in the development of atherosclerosis) in the coronary arteries 15-20 years later (Mahoney et al. 1996). Finally, children, especially overweight children, with high blood pressure have already developed hypertrophy of the heart muscle (Daniels et al. 1998; Hansen et al. 1992), a condition which, in adults, significantly increases the risk of

heart disease, just as reduced elasticity and thickness of the artery wall does.

Relationship between risk factors and coronary heart disease

It is very difficult to perform *prospective studies*, which follow children or adolescents over longer periods in order to assess the long-term effects of lifestyle or risk factors on morbidity and mortality. This is due to the length of time participants must be studied, and also because these studies require extremely large samples of populations. Available data, therefore, are based mainly on retrospective cohort studies, which use already-existing cohorts established for purposes other than the study of coronary heart disease. These cohort data could include, for example, records of the weight and height of children and adolescents which are regularly taken in many western countries at birth, during childhood and during school years. For the same reason, most of the major studies have been on the relationship of overweight in children and adolescents and their later risk of developing coronary heart disease.

Data on college students from Harvard University and the University of Pennsylvania in the US were collected from 1916 to 1950, including smoking habits, participation in sport, height, weight and blood pressure (Paffenberger and Wing 1969). 1,126 of the subjects had died of coronary heart disease 17-51 years later, and these were compared with the remaining 2,292 live subjects. Both cigarette smoking and a lack of participation in sport were associated with a 50% greater risk of mortality, high blood pressure increased the risk by 40% and overweight increased the mortality by 30% in comparison with individuals who did not have these lifestyle and risk factors at the age of 17 years. The presence of multiple factors in one individual increased the risk even further compared to individuals without any or with one factor.

Height/weight data were collected from 717 9- to 13-year-old children in the state of Maryland, USA, and were related to morbidity from atherosclerosis in a study performed on the same subjects 30-40 years later (Abraham et al. 1971). No relationship was established between the degree of overweight in children and the subsequent development of atherosclerotic heart disease or hypertensive arterial disease, neither in data adjusted nor in data not adjusted for adult weight. The study did show, however, that the group of children who were underweight in childhood and who became overweight as adults had an increased risk of hypertensive arterial disease in adult life.

A Swedish study followed a group of overweight children and adolescents aged 2 months to 16 years through 40 years, and showed an excess of morbidity and mortality in the group, mainly caused by cardiovascular disease, in comparison with a Swedish reference group (Mossberg 1989). Morbidity and mortality rates were higher among children who were recorded as becoming overweight after puberty. This could be a result of the greater degree of tracking (see below) of overweight into adult life (DiPietro et al. 1994).

Height/weight data collected from 2,399 English children and adolescents aged 2-14 years, born 1937-1939 (the Carnegie Survey of Family Diet and Health) revealed that a high BMI (>75 percentile) was associated with a doubling of mortality from ischaemic heart disease compared to subjects of average weight in a follow-up study of the cohort 57 years later (Gunnell et al. 1998). In this study, as in the Swedish study described above, there was a stronger association with mortality from overweight among older children compared to younger children.

Morbidity and mortality from overweight in adolescence (13-18 years) were examined in the retrospective Third Harvard Growth Study (Must et al. 1992). 508 individuals were selected from the cohort, of whom half had been overweight and half average weight during adolescence. A total of 181 of the cohort of 508 were still alive at the age of 73 years. A higher rate of morbidity from ischaemic heart disease was reported for men who had been overweight in adolescence compared to those of average weight. The same relationship was found for mortality, assessed on the basis of diagnoses on the death certificates. No relationship was observed among women.

It should be noted that the relevance of findings of cohort studies, which stretch over several decades, might not be the same for the children and adolescents of today. Children who were studied around the time of the Second World War lived under conditions very different from those of today, for example in terms of diet (fast food) and patterns of activity (TV, transport).

Intrauterine nutrition and risk

There is great scientific interest in the importance of nutrition for the unborn child and the risk of developing coronary heart disease (and other lifestyle diseases) later in life. One hypothesis suggests that poor nutrition during pregnancy, at critical periods during prenatal development, can "lock" the genes that control the metabolic and physiological processes in the body (*the programming theory*) (Barker 2002). If the genes that control cholesterol metabolism or regulate blood pressure, for example, are locked at an unfortunate point in

foetal development, there may be an increased risk of coronary heart disease in later adult life.

Numerous studies performed in the past 10-15 years have shown that children with low birth weights, especially if the children are short and thin at birth or have a low weight at the age of 1 years, have an increased risk as adults of overweight, metabolic syndrome and cardiovascular disease (Curhan et al. 1996a; Curhan et al. 1996b; Osmond and Barker 2000; Vestbo et al. 1996). This increased risk can be partly explained by the well-established association between overweight and other biological risk factors (Frankel et al. 1996). Recent studies have shown that low birth weight is not an absolute necessity to increase the risk of heart disease, as inadequate nutrition during pregnancy has been found to have had a permanent and negative effect on the metabolism of the foetus (Roseboom et al. 2000a), and to increase the risk of ischaemic heart disease (Roseboom et al. 2000b), even though the weight of the child was normal at birth.

Irrespective of birth weight, an above-average rate of increase in weight during childhood will increase the risk of metabolic syndrome in adult life (Sinaiko et al. 1999; Steinberger et al. 2001; Srinivasan et al. 2002), and the risk of ischaemic heart disease would seem to be particularly high in men who are thin at birth but who grow rapidly during childhood (Eriksson et al. 2001).

The significance of diet for the programming of genes during foetal life remains unclear, however. It may very well be that factors other than inadequate nutrition are the real explanations for the established associations. It should also be noted that the hypothesis rests on retrospective cohorts, where there is a considerable risk of selection bias. Finally, not all studies have established an association between anthropometric variables at birth and later risks (Matthes et al. 1994).

Conclusion

Numerous cross-sectional studies have established an association between risk behaviour and risk factors in children and adolescents. These findings have been confirmed in longitudinal studies, which followed children into adulthood. Moreover, there is good scientific evidence to suggest that the presence of risk factors in children and adolescents is related to the earlier development of atherosclerosis and a higher risk of coronary heart disease in adult life. Although the programming theory proposes that risks are developed as early as foetal life, the scientific foundation for this theory remains inconclusive.

DO LIFESTYLE AND RISK FACTORS IN CHILDREN TRACK INTO ADULTHOOD?

The term tracking is used to examine the extent to which an individual's lifestyle and risk factors are stable over time. More precisely, the concept is defined as the degree of stability of a state over time, or as the probability that a person with a certain characteristic at any one time will have the same characteristic at a later time (Twisk et al. 1994). A high degree of stability of a characteristic presents the possibility to predict whether the same characteristic, with a high degree of probability, will be present later in life on the basis of a measurement taken earlier in life or, in other words, the possibility to identify at an early stage individuals at higher risk of disease (if the characteristic is a lifestyle or risk factor) and thus the possibility to target preventive interventions at this early stage.

Generally, tracking is estimated in two ways:

1) by calculating the correlation between 2 (or more) measurements of the same factor in the same individual, or
2) by calculating the number of individuals who, at the second measurement, maintain their relative position within the distribution of values (most often percentiles).

The degree of correlation between 2 measurements of a risk factor depends on a number of factors, including the time interval between measurements, as the correlation will generally be weaker the longer the interval between measurements. But a correlation is in many ways unsatisfactory for the degree of tracking of a risk, as the correlation is an expression of the tracking of the risk factor in the examined interval – and not only extreme values. Moreover, a correlation does not indicate the efficacy of screening for risk factors for coronary heart disease at a young age. This can better be assessed by estimating the probability that an individual who has a high risk (e.g. elevated cholesterol) at first measurement will have a high risk at later measurements (positive predictive value), and by estimating *sensitivity* (the ability to identify high-risk individuals) and *specificity* (the ability to identify low-risk individuals). For the purpose of screening, it is generally necessary to have high values for positive predictive values as well as high sensitivity and specificity.

For many risk factors, there are no fixed limits separating individuals with high risk from those with low risks, which makes it difficult to calculate predictive values, sensitivity and specificity. As a rule of thumb, reasonably high stability is established when the correlation is over 0.6 (which means that a little over a third of the variation in one value can be

explained by knowledge of another value) and with a percentage of more than 3 times the expected random distribution within a certain centile (e.g. over 60% of individuals maintain their position within a quintile).

It should be noted that the strength – or weakness – of an established tracking depends on the measurement uncertainty and day-to-day variation of the value being examined. The lesser the variation, the greater the strength of a correlation over time, all other things being equal. Measurement uncertainty is a problem particularly when measuring food intake and physical activity, while blood pressure and cholesterol levels can display considerable day-to-day variations. More precise tracking can be achieved by repeated measurements (when values display considerable day-to-day variations) or by more exact measurements (when measurement uncertainty is high).

Numerous studies have been conducted to investigate tracking within a time interval of a few years. Here we are concerned only with tracking of lifestyle and risk factors for heart disease from childhood to adulthood, as the main emphasis below is on prospective studies. A problem with interpreting this type of study is the generally considerable, sometimes selective, dropout of subjects over time. Another difficulty, related to self-reported goals for physical activity, is that conceptions of activity levels change over time.

Tracking of lifestyle

Only a few studies have been conducted in order to track *eating habits*. The Dutch study, Amsterdam Growth and Health Longitudinal Study (AGHLS), found low to moderate stability among both sexes for the intake of various nutrients with correlations of between 0.28 and 0.52 over a period of 20 years, from the age of 13 to 33 years (Post et al. 2001). Other studies have established similar, weak correlations for the intake of a number of nutrients over time (Cusatis et al. 2000).

As in the case of diet composition, levels of *physical activity* (assessed on the basis of self-reported data on the frequency and intensity of physical activity in spare time and at school/work) would seem to indicate low to moderate stability (Malina 1996). A Danish study, which followed individuals in the 15 to 19 years range until they reached 23 to 27 years, showed correlations of 0.31 among men and 0.20 among women for physical activity. The Finnish Cardiovascular Risk in Young Finns Study reported that 43% of boys and 57% of girls who were active at the age of 15 years were also active at the age of 21 years, while 54% and 51%, respectively, who were inactive at the age of 15 years continued to be inactive 6 years later (Raitakari et al. 1997). A follow-up investigation 12 years later found only moderate to weak correlations (Telema et al. 1997). In the Dutch AGHLS study, only few of the active or inactive at the age of 13 years were still active or inactive at the age of 21 years, 12% and 24% respectively, and at the age of 27 years 4% and 9% respectively, but when average physical activity was estimated on the basis of a 4-year average (13-16 years), somewhat more were active, 38% of boys and 29% of girls, while 10% of boys and 42% of girls were inactive at the age of 27 years (van Mechelen and Kemper 1995). The correlation was low, 0.34, for physical activity measured as weighted energy consumption (Kemper et al. 1999).

Nicotine dependence develops fast among young smokers. All studies therefore show pronounced tracking of *smoking habits*. The longer adolescents smoke, the less likely they are to quit as adults (Kelder et al. 1994). A Danish study reported that 75% of women and 87% of men who were smokers in their late teenage year were also smokers 8 years later (Andersen and Haraldsdottir 1993). Findings from the Dutch AGHLS study, which assessed results for stability over a period of 14 years, from the age of 13 to 27 years, showed pronounced tracking of smoking behaviour. Adolescents who began smoking between the ages of 13 and 16 years had more than 8 times the risk of being smokers at the age of 27 years compared with the likelihood of their having switched their smoking status from smoker to non-smoker (Twisk et al. 1997). Other studies have established a 16 times greater probability of smoking as an adult for adolescents who smoke regularly, and a double as great a risk for adolescents who experiment with smoking (Chassin et al. 1990).

Stability of eating behaviour and physical activity from childhood to adulthood are thus low to moderate, with correlations of between 0.3 and 0.5. It should be noted that correlations (and relative positions in risk groups) are generally calculated on the basis of 2 individual measurements, which, in the case of lifestyle factors, are imprecise, and which show considerable intra-individual variation. These factors weaken the correlation. The poor stability of diet and physical activity can perhaps suggest a greater possibility of influencing habits in childhood and adolescence. Regular smoking during adolescence appears more likely than eating behaviour and physical activity to track into adult life. Nicotine dependence among adolescent smokers is presumably the most significant factor in explaining why smoking habits, once the experimental stage is passed, are difficult to change (Rojas et al. 1998). There may, however, be other factors, for instance concern about weight, that hold girls, in particular, in their smoking habits (French et al. 1994). Factors that influence whether good or bad habits in relation to diet and physical activity are maintained or discontinued have not been studied.

Tracking of risk factors

Few data are available on correlations over time for various measures of *physical fitness* (e.g. maximal oxygen uptake, muscle strength and endurance), but findings suggest that physical fitness in teenage years is related to some extent to physical fitness and levels of physical activity in adult life (Dennison et al. 1988). In the longest-running study, which followed boys over 17 years, from the age of 13 years, isometric and dynamic tests of muscle function were performed at regular intervals (Beunen et al. 1997). The study showed correlations of 0.3-0.6 for the various tests over the 17 years, with higher correlations for measurements taken at shorter intervals. In the AGHLS study, the correlation was 0.31 for maximal oxygen uptake between measurements taken at regular intervals over a 14-year period, beginning when the adolescents were 13 years old (Twisk et al. 1997).

Numerous studies have examined tracking of *overweight*. A review of all prospective studies published from 1970 to the summer of 1992 concluded that about a third of overweight pre-school children and half of overweight school children will also be overweight as adults, but that the majority of overweight adults (approx. 80%) were not overweight in childhood (Serdula et al. 1993). Since 1992, several well-conducted prospective studies, following the same subjects over a number of years, from childhood to adulthood, have confirmed this correlation (Freedman et al. 2004; Hulens et al. 2001; Kemper et al. 1999; Magarey et al. 2003; Power et al. 1997; Williams 2001).

It has therefore been established that overweight children and adolescents have an increased probability of being overweight as adults, and that the probability increases according to the degree of overweight and age at the onset of overweight in childhood and adolescence. For example, a Danish retrospective study of 429 young men who were severely overweight when they appeared before the draft board (BMI >31) showed that the probability of their being overweight as adults increased exponentially over the entire BMI interval at the ages of 7 and 13 years (Sørensen and Sonne-Holm 1988). One of the best-conducted studies, the American Fels Longitudinal Study (Guo et al. 2002), showed that a fifth of boys <8 years with a high BMI (equivalent to 95 percentile) were overweight in adulthood (aged 35 years), while a third of boys between 8 and 13 years and more than half of boys >13 ended as overweight men. Similarly, the risk among overweight girls increased with age: a third of girls <8 years, more than half the girls between 8 and 13 years and two-thirds of girls >13 years became overweight in adulthood. However, there is little to indicate that an individual who becomes overweight during childhood and who continues to be overweight into adulthood is likely to be more severely overweight than if the individual first becomes overweight in adulthood (Rimm and Rimm 1976).

There appears to be three periods in the lives of children and adolescents during which the risk of developing overweight is greatest (Dietz 1997):

1. *The prenatal period*. Some studies have shown that a high birth weight, as an expression of prenatal growth, is related to higher BMI in adults (Curhan et al. 1996a; Curhan et al. 1996b; Parsons et al. 1999). There is, however, considerable uncertainty about the relationship of birth weight to the development of overweight, as some studies have not been able to demonstrate a relationship (Ravelli et al. 1999), while others have shown the opposite, i.e. that low birth weight is related to higher weight in adults (Frankel et al. 1996, te Velde et al. 2003). Moreover, other studies have found that above-average weight gain in babies can increase the risk of overweight in adults (Charney et al. 1976; Rolland-Cachera et al. 1987).

2. *Children about the age of 6 years*. This is a time in the lives of children when deposits of fat, having been decreasing from about the age of 1 year, again begin to increase (*adiposity rebound*). Early onset of adiposity rebound has been related to increased risk of overweight in adulthood (Rolland-Cachera et al. 1987; Wisemandle et al. 2000). It is not known why some children start adiposity rebound earlier than others. An early rebound has been associated with the hyperplastic form of overweight, but the theory relating to two forms of overweight, the hypertrophic (normal fat cells containing much fat) and hyperplastic (many fat cells containing average amounts of fat) has now been abandoned.

3. Adolescents with high BMI and high fat mass around *puberty* have an increased risk of also beings overweight as adults (Andersen and Haraldsdottir 1993; Guo et al. 2000; Srinivasan et al. 1996; Twisk et al. 1997; Wright et al. 2001). A higher BMI is accompanied by early onset of puberty (Laitinen et al. 2001), and early onset of puberty increases the risk of the individual continuing to be overweight as an adult (Power et al. 1997). This period is characterized by a deposit of a larger amount of body fat and an altered distribution of body fat.

Many factors influence the risk of overweight and may thus influence the degree of weight stability from childhood to adulthood. These factors include: overweight in one or both parents, poor social conditions, low level of physical activity and high energy intake, and fat and sugar intake in childhood and adolescence. The significance of overweight in parents, in

particular, has been well documented. The British 1958 British Cohort Study, for example, established correlations of 0.25 and 0.23 for overweight boys and girls from the age of 7 to 33 years if both parents had average weight, while the correlations rose to 0.46 and 0.54, respectively, if both parents were overweight (Lake et al. 1997). The significance of the other factors for the risk that obesity in children continues into adulthood has been researched less, and findings have been contradictory in many instances (Wang et al. 2000). The relative significance of genetic and lifestyle factors for the risk of overweight in a population and thus their relative significance to tracking has not been fully established either, but each is thought to contribute 50% (Parsons et al. 1999).

Blood pressure levels show only a low to moderate degree of stability from childhood to adulthood. The longest-running study (pooled results from 3 American cohort studies: the Berkeley Growth Study, the Guidance Study and the Oakland Growth Study) monitored blood pressure from the age of 3 to 18 years to ages 30 and 50 years in 211 individuals in a cohort which started with 550 individuals (Nelson et al. 1992). Relatively low, but significant, correlations were found between systolic blood pressure measured when young and at the age of 30 years, but even lower compared to blood pressure at the age of 50 years. Correlations for diastolic pressures were weaker than for systolic pressures. The correlations may have been underestimated, as dropouts were mainly subjects with elevated blood pressure and also because many subjects received treatment aimed at reducing blood pressure. Other studies, in which individuals were followed over a longer or shorter number of years from childhood or adolescence into adulthood, found similar low correlations between the systolic and diastolic levels of blood pressure, varying between 0.2 and 0.5 (Beckett et al. 1992; Lauer et al. 1993; Twisk et al. 1997; Yong et al. 1994).

Two Danish studies have measured the *tracking* of blood pressure from childhood to adulthood. Andersen and Haraldsdottir (1993) found correlations of 0.44 and 0.49 for diastolic and systolic blood pressure, respectively, in men and 0.38 and 0.54 in women, when subjects were measured at the ages of 13 and 21 years. About 50% of the men and 35% of the women who had highest levels of blood pressure at the age of 13 years were in the group with highest levels at the age of 21 years. A somewhat lower tracking was observed in the study of Odense school children (Lambrechtsen et al. 1999). In this study, the blood pressure of children was measured when they were aged 8-10 years and again when they were aged 19-21 years. Correlations in men and women with systolic blood pressure were 0.34 and 0.36, respectively, and 0.22 and 0.12, respectively, with diastolic blood pressure. The chance of remaining in the highest or lowest quartile was about two times greater in the case of diastolic blood pressure and the chance of remaining in the extreme quartiles was 2-3 times greater than the average risk in the case of systolic blood pressure.

The above findings are based on single measurements of blood pressure, and often without adjustment for other factors that influence blood pressure levels (e.g. age, sex, BMI). The correlation between blood pressure measurements over time is weakened by quite pronounced variability in blood pressure in the same individual (within-person). Frequent measurements of blood pressure can compensate to some extent for within-person variations. In prospective studies which measured the blood pressure of the same individual several times during childhood and adulthood, a clearer tracking has been observed, with correlations of over 0.5 (Cook et al. 2000; Gillman et al. 1993). The positive predictive value (the probability of blood pressure being elevated in adulthood if it is elevated in adolescence) of blood pressure is, however, poor. For example, one study showed that fewer than half of 10-year-old boys whose systolic blood pressure was higher than 95 percentile would have a systolic blood pressure higher than 90 percentile at the age of 20 years (positive predictive value). Sensitivity was low at these blood pressure limits (0.17), suggesting that most of the 20-year-olds would not have been diagnosed on the basis of a blood pressure measurement taken 10 years earlier (Gillman et al. 1993).

Serum lipids and lipoproteins appear to be more stable from childhood to adulthood than blood pressure, eating habits and physical activity (Guo et al. 1993; Lauer et al. 1988; Orchard et al. 1983; Porkka et al. 1994; Twisk et al. 1997 Webber et al. 1991). Findings concur on correlations of 0.5-0.7 for total cholesterol and LDL cholesterol, but somewhat lower for triglyceride and other lipoproteins, and predict that about half of children and adolescents with extreme values will also have extreme values when measured as adults. As for blood pressure, positive predictive value and sensitivity are poor for total cholesterol.

This is illustrated in the American Bogulosa Heart Study which followed over 1,000 children, residents of the town of Bogulosa, Louisiana, aged 5-14 years over a period of 15 years, and found higher correlations for total cholesterol and LDL cholesterol, 0.4-0.6, than for triglyceride, VLDL cholesterol and HDL cholesterol, 0.1-0.4 (Bao et al. 1996). Of those children who had elevated LDL cholesterol and total cholesterol (>80 centile) 40% (twice average) continued to have elevated values 15 years later. The corresponding percentage for triglyceride and VLDL cholesterol was slightly lower. Children with high LDL cholesterol (>3,35 mmol/l) had a 1.6 times greater risk of being overweight in adulthood, 2.4 times greater risk of

Table 2. Correlations for tracking of lifestyle and risk factors from childhood/adolescence to adulthood. The shorter the time interval between measurements, the higher the correlations. Values are approximate.

Lifestyle factors	**Correlation**	**Comments**
Diet	0.3-0.5	Few studies. Depends on diet component
Physical activity	0.3-0.5	Precise measurements give higher values
Smoking	0.8-0.9	Highest values at early uptake of smoking
Kardiovascular risk factors		
Physical fitness	0.3-0.6	Few studies
Overweight	0.2-0.7	Highest values for post-puberty overweight and when parents are overweight
Blood pressure	0.2-0.5	Highest values for systolic blood pressure
Lipids	0.5-0.7	Highest values for total cholesterol and LDL cholesterol. Lower for triglyceride and HDL cholesterol

hypertension, and a 8.3 times greater risk of dyslipidemia in adulthood compared to children with average LDL cholesterol levels.

As stated earlier, one person commonly possesses several cardiovascular risk factors (clusters). In addition to the Bogulosa Heart Study, other studies have found that clusters of cardiovascular risk factors also show tracking, and correlations are stronger than for individual risk factors. A Danish study, for example, showed that the probability of having 2 or 3 risk factors for coronary heart disease at first measurement at 16-19 years and 8 years later at 23-27 years was 6 (95% confidence interval: 2.1-16.9) compared to individuals with 1 or no risk factors (Andersen et al. 2004).

Conclusion

Tracking is a phenomenon which exists for both lifestyle and risk factors for heart disease. It is therefore possible to identify individuals who will have a greater risk of heart disease in adulthood on the basis of the presence of an unhealthy lifestyle and/or high cardiovascular risk factors early in life, and possible therefore to target preventive interventions at this early stage. It should be noted, however, that the estimated predictive values are poor, and that many children and adolescents with normal values will develop abnormal values in adulthood, and, conversely, that many children and adolescents with abnormal values will normalize these values in adulthood. This means, for example, that general screening of children for the presence of risk factors would not be efficacious. Table 2 summarizes the established correlations for tracking of lifestyle and risk factors from childhood to adolescence.

FACTORS DETERMINING LIFESTYLE

For effective prevention to occur it must be possible to identify the factors in everyday life that influence the behavioural choices of children and adolescents, in this context with regard to their diet, physical activity and smoking habits.

In a macro perspective, a range of political, societal, technological, family and developmental factors determine individual lifestyles (French et al. 2001). Political and economic factors (public health policies, the purchasing power of a population, both parents in employment), marketing (advertising of unhealthy food and tobacco, labelling, health campaigns), availability (spread of fast food restaurants, soft-drink vending machines, fitness centres), pricing policies (differentiated rates of sales tax on foods, taxing of tobacco, large portions relatively cheaper) and greater technological development (increased car transport, televisions and computers in all homes, greater supply of different foods) all have a crucial influence on lifestyle and consequently on the risk of disease.

A number of other factors (also called *determinants*; in fact predictive correlations) influence behaviour on the more "micro" level. These determinants can be demographic (e.g. age, sex, social status, education, ethnicity, sometimes called *moderators* of behaviour), and other determinants (sometimes called *mediators* of behaviour) can include factors in the personal sphere (e.g. family-school-peer relations, norms), and personal attributes (e.g. knowledge, skills, attitudes, self-image, self-efficacy, stress). Many of these determinants are inter-related, just as the risk behaviour of adolescents is often inter-related.

Due to the inter-related nature of these determinants, and because it is difficult to accurately define and measure them, their significance, in particular their relative significance to the individual, is also difficult to examine experimentally.

Determinants of behaviour

Certain determinants have particular importance for the behaviour of children and adolescents during particular stages of their development – and are completely irrelevant at other times. This is because the determinants underlying the behaviour of children and adolescents are in constant motion, undergoing constant change.

In general, the behaviour of children and adolescents is driven by three basic needs:

1. to be able to take decisions with regard to one's own self and one's immediate environment,
2. to appear older than one actually is, and to be treated accordingly, and
3. to experiment with actions and push back/stretch boundaries.

In addition, the behaviour of children and adolescents is determined by a number of other underlying behavioural factors, which vary in strength according to the age of the individual (Table 3).

Table 3. Underlying factors determining the behaviour of children and adolescents at various ages

Age (years)	Determinants of behaviour
0-1	Dependence
	Instinctive observation
	Imitation
1-3	Dependence
	Imitation
	Acceptance
4-7	Fantasy and interest
	Acceptance
	Self-assertion
8-11	Solidarity with friends
	World is not black-white
	Emerging independence
12-15	Being with friends and social acceptance
	Independence and rebellion
	Lack of respect for authority

During the first years of its life, a child is totally dependent on others, observes and simply imitates. The age of 1-3 years is when the child's life habits are established, and probably the most important years in the life of the individual. There is a strong need to be able to do things oneself and to be able to decide (independence phase). Children in this phase are still dependent, however, and imitation continues to be a strong determinant of behaviour. Children have absolute belief in everything they are told (acceptance). In the 4-7 years range, children become increasingly interested in cooperation (through play), and their fantasy emerges. During these years they want to immerse themselves in everything, anyhow and everywhere. Children continue to seek acceptance, and the need to assert oneself increases. In the 8-11 years range, social skills are developed, and children develop a stronger sense of solidarity with friends. Children become more extroverted, not as dependent on the home as earlier. Intellectual development increases sharply. Children continue to have strong,

though declining, belief in what they are told, but begin to grasp shades of meaning. Pre-puberty and puberty is the time when the need for independence becomes stronger. Ties to parents and their authority are broken, and the adolescent feels a strong need to be accepted socially in groups that mirror his/her identity.

It is not unusual to observe clear differences in behaviour and lifestyle between boys and girls. These differences increase with age, and become progressively greater from the age of ten and up through adolescence.

PARENTING STYLES

The importance of parenting styles for the health behaviour of children has attracted growing attention from researchers in recent years (Steinberg et al. 1994). Four styles have been identified in this work: authoritative, neglecting, authoritarian and indulgent.

The so-called *authoritative parenting style* seems to provide the best foundation for the establishment of healthy habits in the child. This style is defined as child-rearing where the parents are engaged and sympathetic to the problems that occur in the life of the child, set standards for, and actively and with interest monitor the activities of the child, while also making demands of the child appropriate to his/her development. Numerous studies have shown that authoritative parenting has a positive impact on children's health behaviour whereas the opposite, the *neglecting parenting style* (where parents are uninterested and fail to make demands of the child), has a strongly negative effect on the behaviour of the child (Jackson et al. 1998; Kremers et al. 2003; Schmitz et al. 2002; Simons-Morton 2002). The two other styles, the *authoritarian parenting style* (strong control but little emotional support) and the *indulgent parenting style* (interested and sympathetic but not demanding), have shown mixed results with regard to the health behaviour of the child, though, paradoxically, authoritarian parenting has shown to increase the child's preference for "forbidden" foods (Birch and Fisher 2000; Carper et al. 2000; Fisher and Birch 1999; Fisher et al. 2002).

HEALTH PROMOTION AND DISEASE PREVENTION

Initiatives aimed at promoting health and preventing disease vary in scope from measures targeting individuals and smaller well-defined groups to interventions targeting a whole population. Similarly, interventions can be implemented at different levels, ranging from programmes that (attempt to) directly influence individual behaviour to more indirect, overall interventions, e.g. legislation or other health policy initiatives.

In principle, it is possible to employ two strategies for influencing lifestyle and behaviour: interventions directed at a total population of children and adolescents, considering the whole group at risk of developing coronary heart disease later in life, or more targeted, and often more intensive, initiatives directed at children and adolescents presenting with a particular risk behaviour (individual-based, high-risk strategy).

Population-based strategies are difficult to implement. This is in part due to the fact that, to be effective, they require the coordinated efforts of many actors, including, for example, health workers, schools and other institutions for children and adolescents, central government and local authorities, and the food industry. On the other hand, even small reductions in levels of risk behaviour and therefore in risk factors in the total population considerably reduces the number of people at risk (as the great majority of these are among those who have an average risk).

Starting point for interventions

All children spend a great deal of their time at school, where there are good opportunities to carry out special programmes aimed at modifying behaviour with regard to eating habits, physical activity and smoking behaviour. The *school* has therefore been an obvious place to carry out programmes aimed at the establishment of disease-preventing and health-promoting lifestyles. *Family-based* interventions involve the family and their primary aim is therefore to change the behaviour of the whole family (unlike school-based interventions). Nevertheless, schools are often involved in family-based interventions, solely because schools are where large parts of studies are carried out (instruction, follow-up). The involvement of the whole family in prevention is important not only because behaviour, and thus risk, is often common to a family, but also because it seems unrealistic to attempt to intervene with the child if the whole family does not concurrently modify behaviour.

Models explaining behaviour

Attempts to modify behaviour in children and adolescents rest on different theories and models. Most often, these have been the theory of *reasoned action*, or the derived theory of *planned behaviour*, the theory of *stages of change*, various *social cognitive* theories, most often Bandura's social cognitive theory, and theories of *social influence* (see Glanz et al. 1997 for a review of these theories).

Many earlier studies of behaviour assumed that risk behaviour was caused by insufficient knowledge of risks, and that the provision of information on risks would lead to a change in attitude and a rational choice of a different behaviour (the knowledge-attitude-behaviour model or the *rational model*). An intervention model based solely on the provision of information has proved to be inadequate for (but not irrelevant to) achieving behavioural change. Among other things, the knowledge-attitude-behaviour model does not take into account factors linked to the environment, factors which have a critical influence on the success of interventions aimed at modifying behaviour.

Of the social cognitive models, Bandura's social cognitive theory is widely applied. Bandura's model builds on concepts of *self-efficacy* (the confidence in one's own ability to execute an action) and *outcome efficacy* (the belief that the action will have the desired outcome). The model assumes that behaviour is driven primarily by its consequences (reinforcement). Should a certain behaviour be necessary to achieve social acceptance, the behaviour will act as a positive reinforcer. Intervention will therefore be directed at the motivation underlying the behaviour and point out other behaviour options, by strengthening self-efficacy through modelling (imitation of the behaviour of role models), value clarification and goal setting.

Research in recent years has shown that the model that best explains health-related behaviour is a model of social environmental impacts and social norms. The basic premise for the theory of social influence is that the social environment is a critical factor regulating behaviour. Another important aspect of the model is the emphasis given to subjective norms, i.e. what a person believes other people think. Intervention will therefore be directed at normative methods of instruction and education aimed at correcting misunderstandings (perceptions of what other people think and believe do not always conform to reality) and at improving skills for countering negative environmental influences.

It should be noted, however, that regardless of the model applied, less than a third of behaviour variability can be explained by a model, and often considerably less.

Types of studies

Studies can be divided into two categories: *observational studies*, in which the researcher observes individuals only, and *experimental studies*, in which the researcher allocates subjects to groups meeting different study criteria. In so-called *longitudinal studies*, a group of individuals is followed whose members all possess the presumed attribute, as well as a control group, which does not, and the groups are followed either forward in time (*prospective*; cohort studies) or back in time (*retrospective*; case control studies) in order to observe how often the presumed effect manifests itself.

In *cross-sectional studies* the presumed cause and effect are registered at the same time. For that reason, cross-sectional studies, unlike longitudinal studies, cannot identify the relationship between events over time (the cause should come before the effect), an important, but not sufficient, condition for assessing causality. Longitudinal studies therefore have greater scientific strength than cross-sectional studies, and the emphasis in the following will be placed on longitudinal studies. It should be stressed, however, that it can be difficult, even in longitudinal studies, to capture the dynamic process of risk behaviour, especially when the period between measurements is lengthy (Engels et al. 1999). It is not unusual that less than 10% of the variance in a behaviour can be predicted by explanatory variables.

In health science the only scientifically unimpeachable method for assessing causality is the randomised (or controlled) experiment. The randomised experiment is a study design where subjects are allocated randomly to either active treatment or no treatment (or the best treatment to date). The ideal experiment requires also that neither the investigator nor the subjects are aware of who is receiving the control or the experimental treatment (double-blinded). A number of issues, part ethical, part practical, often mean that these scientifically ideal studies cannot be conducted, as especially blinding, but also randomization, are rarely possible in population-based interventions into lifestyle factors.

The conceptual framework

Population-based interventions aimed at promoting health or reducing the risk of disease are depicted in Figure 2. The effect of a particular intervention is often measured on the basis of behaviour or disease markers and assumes therefore that a change in behaviour and markers will lead to an improvement in the health of the population. No (randomised) intervention studies have yet been, or are likely to be, conducted on the effect of a change in behaviour among children and adolescents on the incidence of chronic lifestyle diseases, including heart disease, many years later. As the figure shows, there is also a pronounced and often unclear

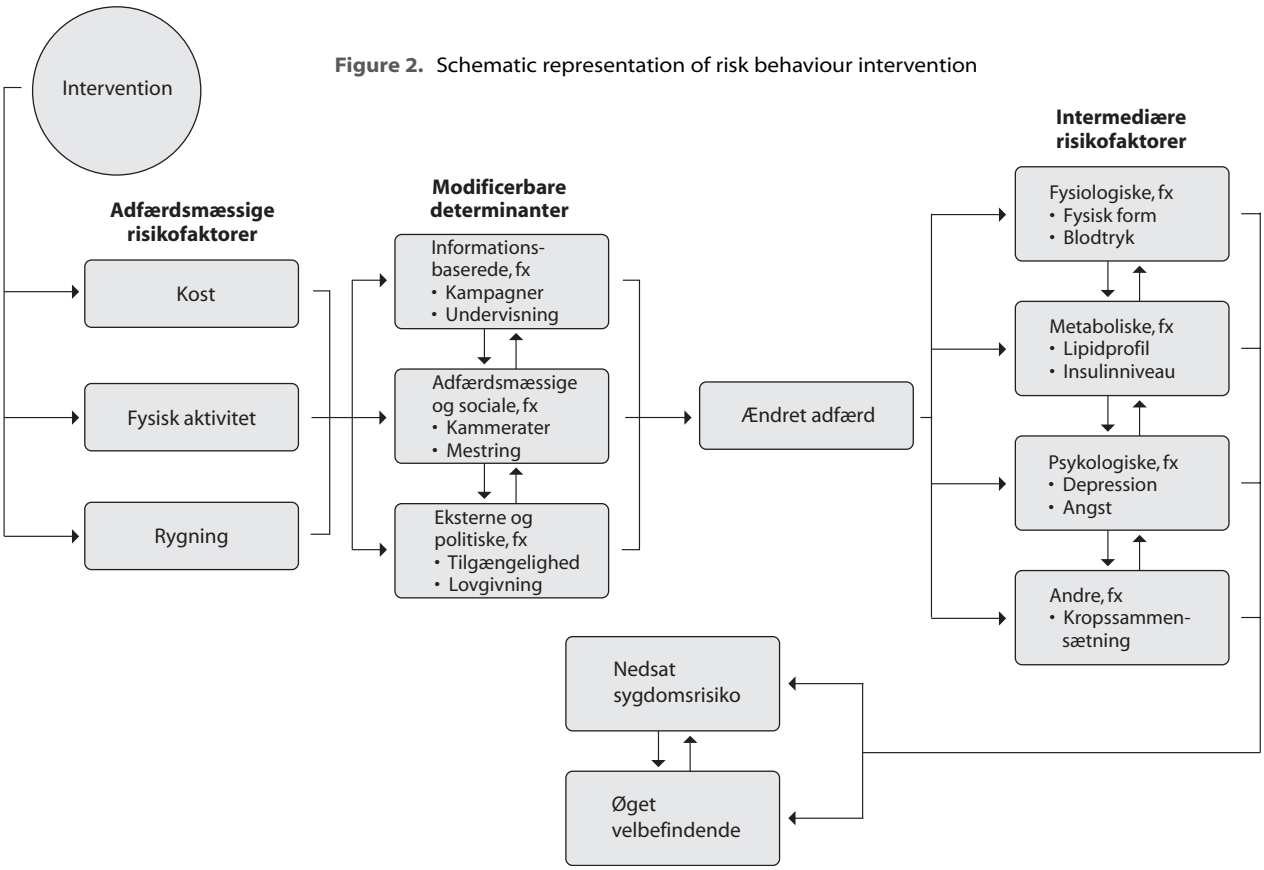


Figure 2. Schematic representation of risk behaviour intervention

interaction within all the stages in the process from change in behaviour to achieved health.

Below are described first the modifiable determinants (mediators of behaviour). Numerous studies have been conducted on the determinants of behaviour. The majority of studies have been cross-sectional, and these will not be considered. Instead, emphasis will be placed on longitudinal studies. For the sake of simplicity, determinants are described under the headings *exogenous* (originating from outside) and *endogenous* (originating from within the individual). Next, controlled intervention studies are discussed, and the emphasis here will be on systematic reviews and meta-analyses. Moderators of behaviour, the non-modifiable determinants, will not be considered at any length.

Finally, it should be mentioned that justifiable doubts are held as to whether randomised experiments are suitable for uncovering the often complicated and diverse problems built into most studies concerning population-based health promotion (Green and Tones 1999). It is argued that comprehensive interventions at several levels (national, local, individual) involving several factors that are important for risk behaviour cannot be conducted in accordance with the stringent principles within bio-medical research.

Diet

Determinants of food intake among young children who are deeply dependent on their parents are obviously very different from determinants governing the eating patterns of adolescents who are attempting to liberate themselves from dependency on adults. Diet composition therefore varies considerably with age.

From birth and onwards, children prefer sweet and salty tastes to sour and bitter (Birch 1999). Small children thus prefer "energy-rich" foods with a high content of sugar (and fat) to "energy-light" foods. This preference is then reinforced because children soon learn to associate the taste impression from energy-rich foods with a feeling of becoming quickly replete (Birch and Fisher 1998).

Another factor which is believed to influence the preferences of (younger) children is an innate *neophobia* ("fear of the new") towards food (Pliner 1994; Pliner and Loewen 1997). This characteristic is believed to be adaptive, and acts to prevent the intake of toxic – often bitter-tasting – foods, especially plants (Cashdan 1998).

Preferences – and aversions to eating specific foods, e.g. vegetables – can be modified and changed at any age, but this is

especially easy if non-preferred foods are introduced at an early stage in children's lives (before children reach the age of 6 years – and the earlier they are introduced, the fewer times children have to be exposed) (Sullivan and Birch 1990). This has been experimentally demonstrated with infants (Birch et al. 1998; Gerrish and Mennella 2001), pre-school children (Sullivan and Birch 1990; Wardle et al. 2003) and school children (Pliner and Stallberg-White 2000).

A study of 3- to 6-year-olds' food choices stresses the importance of seeing the eating habits of children and adolescents as part of the daily routines they develop in relation to their schooling, mobility and social relations to family and peers (Christensen, 2003).

Observational studies

Exogenous determinants

The role of parents, peers and siblings in the food choices of children and adolescents has not been fully elucidated. During childhood, children typically imitate the eating habits of their parents (Fisher et al. 2000; Fisher et al. 2002; Gibson et al. 1998; Oliveria et al. 1992), but the association between the preferred foods of parents and children is weak (Logue et al. 1988; Pliner and Pelchat 1986; Rozin et al. 1987). The influence of the environment on the child's eating habits varies according to age, the influence of the family weakening as the child and adolescent become older (Feunikes et al. 1998; Lau et al. 1990).

Parents and siblings act as role models (*imitation*) and in this way influence the child's dietary habits (Lau et al. 1990). Mothers appear to act as more effective role models for their children than other adults, and older children appear to be more effective role models than younger children (Birch 1999). Peers can be effective role models, and popular cartoon figures have also had this effect (Lowe et al. 2004).

Children's food choices are clearly attributable to many other factors than imitation (Nicklas et al. 2001). Greater *availability* in the home can play a role (Domel et al. 1993; Krebs-Smith et al. 1995). A greater availability in the home of fruit and vegetables increases intake, even the intake of those fruits and vegetables not among the child's favourites (Neumark-Sztainer et al. 2003a). The significance of availability is further illustrated in studies showing that snack vending machines in schools reduce the daily intake of fruit and vegetables among adolescents (Kubik et al. 2003), and the choice of low-fat dishes in school cafeterias reduces pupils' fat intake (Whitaker et al. 1993). The nutritional knowledge of the mother and her attitude to what constitutes a healthy diet also play a role in the eating habits of children (Axelson et al. 1985; Gibson et al. 1998).

Speed and *convenience* are important elements in the food choices of many adolescents. Speed and convenience are provided by fast food restaurants. Frequent fast food meals increase the daily energy and fat intake of adolescents (French et al. 2001). In a recent representative American survey of children and adolescents aged 4-19 years, about a third had eaten fast food on a particular day, and they had a higher intake of energy, fat and sugar and a lower intake of fruit and vegetables than the children and adolescents who had not eaten fast food (Bowman et al. 2004).

The *portion size* of many energy-rich foods and drinks, snacks, sweets and fast food restaurant meals has increased dramatically, especially in the past 10 years, and bigger portion sizes have been accompanied by a higher consumption of sugar-sweetened soft drinks, sweets and ice-cream (Matthiessen et al. 2003). Bigger portion sizes give an increase in intake right down to the age of 5 years (Rolls et al. 2000). It is likely that bigger and relatively cheap portion sizes cause a higher total intake of energy, fat and sugar, particularly among the young. There are, however, no longitudinal studies to support this claim.

The significance of *price level* to adolescents' food intakes is very poorly researched. American studies indicate that price differentiation may be significant. A 50% cut in low-fat snack products from vending machines almost doubled sales (French et al. 2001b), and a corresponding reduction in the price of fresh fruit and vegetables in the school cafeteria led to a 200-400% increase in sales (French 2003).

Most *advertisements* in the media, primarily on television, targeting children and adolescents are for unhealthy foods. There is an established relationship between the number of advertisements for foods that children see on television and the intake of these foods (Coon and Tucker 2002), and on children's attempts to persuade their mothers to buy the food in question (pester power) (Donkin et al. 1993).

Children who spend much time watching *television* have a higher intake of energy, fat and sugar and a lower intake of fruit and vegetables (Boynton-Jarrett et al. 2003; Coon et al. 2001; Müller et al. 2001; Utter et al. 2003; Wake et al. 2003), and a greater risk of overweight (Berkey et al. 2000; Dietz and Gortmaker 1985; Francis et al. 2003; Gortmaker et al. 1996; Müller et al. 2001; Obarzanek et al. 1994). The increased risk of overweight is probably due to a combination of advertisements and/or a situation which in itself encourages a higher intake of energy-rich foods and thus overeating, and to the inactivity associated with television viewing (Robinson 2001), an inactivity that can be more pronounced than if children "aren't doing anything" (Klesges et al. 1993).

Endogenous determinants

The relationship between dietary *knowledge* and food choices among children and adolescents is unclear. A Danish study found a weak association between knowledge (regarding the content of fat, sugar and fiber in food) and the consumption of healthy food among pupils in grades 6 and 8 (Osler and Hansen 1993).

There is a strong relationship between how well children and adolescents like a particular food and intake of the food in question (Neumark-Sztainer et al. 2003a; Resnicow et al. 1997), whereas conceptions of how healthy a food is does not seem to be a good predictor of its intake (Contento et al. 1988; Woodward et al. 1996).

Among adolescents good *self-efficacy* with regard to making healthy food choices is associated with low intakes of fat- and sugar-rich foods (Cusatis and Shannon 1996; Masui et al. 2002), but not with intakes of fruit and vegetables (Resnicow et al. 1997). The latter study found an association between perceived positive consequences of an increased intake of fruit and vegetables and the actual intake (Resnicow et al. 1997).

Families (parents and children) whose *motivation* to eat fruit and vegetables is high (better knowledge of the health effects, greater self-efficacy expectations and expectations regarding the long-term effects of an increased intake of fruit and vegetables) had greater availability of food and vegetables in the home and a greater intake of fruit and vegetables than families with fewer motivational factors (Kratt et al. 2000).

The role *genetic* factors play in food choice is not yet understood, but sensitivity – and reaction – to bitter tastes, particularly the taste of cruciferous vegetables, is genetically determined (Drewnowski and Rock 1995).

Intervention studies

In recent years, the focus of interventions promoting the establishment of healthy eating habits has shifted from mass campaigns aimed at increasing awareness and knowledge to increasing availability and thus to opportunities to choose healthy food, most often in the school setting. French and Stables (2003) have recently reviewed published intervention studies, randomised and non-randomised, aimed at promoting fruit and vegetable consumption among school children. They concluded that a strategy encompassing several different elements, e.g. class-based instruction in behaviour change, the option of choosing more healthy foods in the school cafeteria, including the establishment of a cheap fruit scheme, and the involvement of parents, can increase the consumption of especially fruit by an amount corresponding to 0.2-0.6 portions per day. Only marginal gains in consumption are achieved by focusing in isolation on individual elements in a school-based strategy, e.g. by improving the availability of fruit and vegetables in schools (Perry et al. 2004).

A non-randomised Danish study examined the effect of a *fruit scheme* financed by parents which was set up in 7 public primary schools for children aged 6-10 years (Eriksen et al. 2003). In the 3 intervention schools, 45% of parents entered their children in the scheme. The children's fruit and vegetable consumption was measured at the start of the study and again 5 weeks after the scheme had been established. Only 31% of parents returned information on consumption. Fruit consumption rose significantly (0.35 portion/day) in the intervention schools compared to the control schools. There were no significant differences in the total consumption of fruit and vegetables between the intervention and control schools, indicating that fruit from the school fruit scheme may have replaced consumption of other fruit and vegetables in the course of the day. Worth noting is the observation made that the increased consumption of fruit in the intervention schools was due in part to an increase in fruit consumption among children who were not signed up for the fruit scheme.

The German Kiel Obesity Prevention Study (KOPS) is a school-based intervention mainly to prevent obesity and includes children aged 5-7 years (Müller et al. 2001). The intervention targeted children, parents and teachers and comprised an offer of instruction in diet and nutrition and health promotion building on improved knowledge, self-monitoring, self-image and independence. In addition to this, a family-based intervention programme targeted high-risk children (children whose parents are overweight). The study is scheduled to run for 8 years, but findings from the first year have been published. Compared with the children in the control schools, the children in the intervention schools had smaller increases in fat mass estimated by skinfold measurements and bioelectric impedance.

In another study whose main aim was to prevent obesity, families in New York State, USA, were randomised into groups on energy-reduced diets either with a high content of fruit and vegetable or a reduced content of fat and sugar (Epstein et al. 2001a). To qualify for inclusion in the study, one parent should be overweight, while the child should be normal weight (6-11 years). The intervention targeted the parents, as they were expected to act as role models for the children. The study, which ran for 1 year, comprised increased availability of fruit and vegetables, influencing stimulus control and learning positive reinforcement techniques. The intake of fruit and

vegetables rose among parents and children who were randomised to this intervention group, and a corresponding decrease in fat and sugar was observed in the group randomised to a reduced intake of these nutrients. No weight differences were observed between the two intervention groups. Unfortunately, there was no control group to assess the "spontaneous" weight development.

A Swedish study randomised motivated overweight 10- to 11-year-old children into 3 treatment groups: 1) one group received family therapy (6 sessions spread over 1 year) in addition to conventional treatment (dietary advice provided by a dietician and several control visits), 2) one group received conventional treatment only, and 3) one control group which did not receive advice/treatment (Flodmark et al. 1993). Results were drawn up when the children were aged 14 years, and demonstrated significantly lower skinfold measurements and higher physical fitness in the group that had received family therapy compared to the group treated conventionally (these measurements were not taken in the control group), and there were significantly fewer extremely obese children in the group which received therapy than in the control group.

Several intervention studies have been conducted whose main aim was to modify the diet of children and adolescents in order to bring about improvements in blood cholesterol levels. These include a number of smaller randomised intervention studies which showed that the provision of information on a low-fat diet to hypercholesterolemic children and adolescents achieved only moderate and often non-significant falls in concentrations of total and LDL cholesterol in the blood, when the children were followed-up just over a year later (Tershakovec et al. 1998). More effective reductions in blood cholesterol can be achieved if the child's family is involved and engaged (Kuehl et al. 1993), but a lasting reduction in cholesterol probably requires an ongoing contact between patient and therapist.

The best support for this comes from the longest study conducted so far, the American Dietary Intervention Study in Children (DISC). In this study, 663 children of both sexes aged 8-10 years with elevated LDL cholesterol were randomised into an intervention group which received intensive and regular dietary instruction (behaviour modification based on Bandura's "self-efficacy" theory, and later the application of the "stages of change" model and motivational talks) in order to reduce the intake of total fat, saturated fat and cholesterol to accord with American recommendations (National Cholesterol Education Program Step 2 diet) and a control group which received instructional material and a heart-friendly diet (Obarzanek et al. 2001). Subsequent follow-ups, which took place at regular intervals until the oldest children were 18 years, demonstrated good adherence to the diet and a significantly greater fall in total and LDL cholesterol levels during the first years of the study in the intervention group, but no significant differences at follow-ups after 5 and 7 years – a period in which the diets of the two groups also converged. No negative effects of a dietary intervention were observed, as weight, height, age of puberty onset and various blood tests (ferritin and folat levels) were similar in the two groups.

Two further randomised school-based interventions have been conducted among healthy children (Reynolds et al. 2000; Walter et al. 1988), which report fat intake in a way that allows the results to be included in a meta-analysis. Findings from the combined analysis of the 3 studies discussed here show a marginal, but significantly reduced fat intake in the intervention group compared to the control group of 2.19 (95% confidence interval: 1.49-2.89) E% (Ammerman et al. 2002).

The Finnish study, Special Turku coronary Risk factor Intervention Project for children (STRIP), included 1,062 infants aged 7 months, and randomised them into an intervention group (n = 540) in which the family received dietary advice twice a year in order to give the infant a diet corresponding to Nordic nutrition recommendations (fat: 30% of energy intake and a 1:1:1 ratio in the intake of saturated, monounsaturated and polyunsaturated fat), and a control group (n = 522), which did not receive advice (Simell et al. 2000). The 7-year results have recently been published, and showed 7% lower LDL cholesterol and 5% lower total cholesterol levels among boys in the intervention group compared to boys in the control group (Kaitosaari et al. 2003). Moreover, the particle size of LDL was larger in the intervention group (small particle size may be associated with an increased risk of atherosclerosis). No differences were observed among girls, despite the fact that the reduction in saturated fat was the same for boys and girls in the intervention group, and greater than for children in the control group. No side effects of the intervention were observed with regard to the children's height and weight.

Conclusion

Various determinants have been established for dietary intake, food choices and eating habits among children and adolescents. Family and peers do have a certain, though small, effect on the eating habits of children and adolescents. Of more importance are portion sizes and eating out, especially in fast food outlets. Advertising has probably a significant effect on the food choices of children and adolescents, in par-

ticular the increasing marketing of unhealthy foods in television advertisements. Availability has been shown to have great significance for the intake of healthy foods among children and adolescents. The effect of pricing, however, is not well researched and has therefore an uncertain effect on food choices. A number of randomised intervention studies have been conducted, mostly in school settings and drawing on theoretical models, in order to introduce and establish healthy dietary habits among children and adolescents. The results of these studies, which included several elements, have been good as long as the studies have been in progress, but follow-ups have typically shown a convergence in effect measurements between the intervention and control groups. A sustained effort, building on many strategies, is required in order to maintain healthy dietary habits.

Physical activity

The amount and type of physical activity children and adolescents engage in is governed by a range of variables, including biological and developmental factors (e.g. physical fitness, state of health) and environmental factors (e.g. access to sport facilities and equipment, season, weekday/weekend) (Kohl and Hobbs 1998). Few of these factors have been examined in longitudinal studies, nor have other physical and social factors that impact more generally on lifestyle. Cross-sectional studies, for example, show a linear relationship between children's level of physical activity and their level of physical fitness, however not whether physical fitness determines activity levels or whether activity levels determine physical fitness.

A systematic review of approx. 100 studies published from 1970 to 1998 (mainly cross-sectional studies, but a small number of longitudinal studies) and dealing with determinants of physical activity among children and adolescents showed that competence, motive, help and support from parents as well as access and opportunity to participate in physical activity were most often associated with a higher level of physical activity, but there was a large degree of discordance between studies (Sallis et al. 2000).

Observational studies

Exogenous determinants

Numerous studies have reported an association between the exercise habits and levels of physical activity of children and adolescents and the attitudes to exercise and exercise habits of parents, siblings and peers, and the extent to which the immediate environment supports and encourages children to be physically active (Aarnio et al. 1997; Anderssen and Wold 1992; Bungum and Vincent 1997; DiLorenzo et al. 1998; Fogelholm et al. 1999; Gottlieb and Chen 1985; Greendorfer and Lewko 1978; Lau et al. 1990; McGuire et al. 2002; Neumark-Sztainer et al. 2003b; Prochaska et al. 2002; Sallis et al. 1992; Sallis et al. 1999; Vilhjalmsson and Thorlindsson 1998). *Imitation* seems therefore to be an important exogenous determinant of physical activity, and the association with parents' levels of physical activity seems greater the younger the child. It may be that family roles are gender-specific, as a longitudinal study has shown that mothers provide logistical support, while fathers act as role models (Davison et al. 2003). Other role models may also influence levels of physical activity, e.g. professional athletes, but there are no studies to show that this is the case.

It is uncertain whether the inactivity related to *television* viewing is associated with a reduction in the total amount of physical activity engaged in throughout the day, or whether children and adolescents compensate for this while not watching TV (Bungum and Vincent 1997; DuRant et al. 1994; Lindquist et al. 1999; Robinson et al. 1993; Utter et al. 2003; Vilhjalmsson and Thorlindsson 1998). Television viewing, however, reduces the time available for other activities and may be related to total activity in this way. There is reason to believe that metabolic rates in children are lower while they are watching TV than when they are doing nothing at all (Klesges et al. 1993). By contrast, playing video games is an active process that may raise the metabolic rate moderately (Segal and Dietz 1991).

Other factors that increase the percentage of children who are active include participation in *organised sport* (Bungum and Vincent 1997; Telema et al. 1997) and in physical education at school (Gordon-Larsen et al. 2000), and the availability of adult-supervised sports facilities at or close to the school or home with usable and well-maintained equipment, e.g. goalposts, tennis nets (Trost et al. 1997).

Endogenous determinants

Knowledge about the positive health effects of increased physical activity does not seem to be important for behaviour (O'Connell et al. 1985). This is less true for concrete knowledge about how to get physically active (Gottlieb and Chen 1985). *Attitudes* towards the benefit of physical activity have shown only a moderate association with the actual behaviour of adolescents (Desmond et al. 1990; Godin and Shephard 1985; Trost et al. 1997). Confidence in one's own ability (*efficacy expectation*) and *will-power* are, however, strong predictors of physical activity (Godin and Shephard 1985; Greenockle et al. 1990; Reynolds et al. 1990; Trost et al. 1997; Zakarian et al. 1994). *Competence* within sports also seems to be a good predictor of physical activity (Sallis et al. 1999).

Physical activity is associated with the *pleasure* of exercising (Stucky-Ropp and DiLorenzo 1993) as well as physical *well-being* among adolescents (Steptoe and Butler 1996; Vilhjalmson and Thorlindson 1998).

Negative determinants of physical activity – and the reason why teenagers are less active in sports – are explained by lack of time due to homework, paid employment, work in the home and other spare-time commitments (Neumark-Sztainer et al. 2003b), and also, in the case of girls, concerns about their appearance following activity.

Genetic factors also play a (probably minor) role in physical activity (Aarnio et al. 1997; Perusse et al. 1989; Zurlo et al. 1992). The nervous system undergoes rapid development during childhood, and the accompanying development in muscle coordination means that the physiological conditions for many activities will increase. The significance of physical fitness for level of activity is unknown.

Intervention studies

A comprehensive and systematic review of all experimental studies which measured the effect of interventions on physical activity concluded that, on the basis of 12 controlled studies, there was convincing scientific evidence that school-based programmes which included advice on and instruction in behaviour modification and skills training with regard to physical activity raised children's levels of physical activity and improved levels of physical fitness (Kahn et al. 2002). There was not, however, sufficient evidence to support a beneficial effect of class-based instruction and education programmes, including programmes reducing the time spent watching TV or playing video games. Isolated family-based programmes were generally ineffective. The review, which did not specifically assess the effectiveness of interventions in children and adolescents, furthermore concluded that there was good evidence to suggest that strategically-positioned signs at escalators, elevators etc. (point-of-decision prompts), local campaigning and behaviour modification programmes targeting individuals were effective in increasing levels of physical activity.

Another, slightly older, systematic review of randomised and non-randomised intervention studies examining the physical activity in children and adolescents identified 14 school-based interventions and 3 interventions in the local environment (Stone et al. 1998). The majority of the studies was conducted in the US and for their theoretical foundation drew mainly on social cognitive theory. The interventions had a good effect on knowledge and attitudes to physical activity, though the effect on self-reported physical activity varied widely.

The systematic reviews discussed above included studies where increased physical activity was one – of many – of the effects measured. Several of the studies also covered interventions in diet and smoking, and in most intervention studies the main target for the effect was not increased physical activity, but weight change or other risk factors. Several of these studies are discussed below under the respective headings. There are, however, few controlled studies whose main aim was to increase levels of physical activity among children and adolescents.

One of the components in the American Sports, Play, and Active Recreation for Kids (SPARK) study was a school-based programme whose specific aim was to increase physical activity at school and in leisure time (Sallis et al. 1997). With a view to increasing physical activity in physical education (PE) class, 4th grade pupils from 7 schools were randomised into groups taught PE either by specially-trained PE teachers (3 schools) or by grade teachers who had received further training in PE (3 schools). The remaining school acted as a control. PE class in the intervention schools was a minimum of 30 minutes and comprised two parts: the aim of one part was to improve fitness; the other part was devoted to improving motoric skills. The programme also included a sub-programme managed by the pupils themselves aiming at increasing physical activity during leisure time. At the end of the 2-year programme only girls who were taught PE by the specially-trained PE teachers had significant improvements in 2 of 5 dynamic muscle tests compared to the control girls. There was no difference in activity levels during leisure time.

Preschool children in Thailand were randomised into a physical activity programme consisting of 15 minutes of walking and 20 minutes of dancing 3 times per week for 7 months plus the usual PE or into a group receiving PE only (Mo-suwan et al. 1998). At the end of the study, non-significant reductions in the incidence of obesity were recorded, somewhat greater in the intervention group than in the control group. There were no differences among girls with regard to changes in skinfold measurements, which among boys were – contrary to expectations – greater in the intervention group than in the control group.

The project team in the Dutch Amsterdam Growth and Health Longitudinal Study (AGHLS) interviewed and counselled a group of young men and women aged 13 to 33 years on 8 occasions at regular intervals (Kemper et al. 2002). The purpose of counselling was to increase physical activity (and improve dietary habits) assessed on the basis of the results of blood tests, actual physical activity, smoking habits and biological risk factors. The control group had 2 health interviews, one at the start and one at the end of the study, when they

were aged 13 and 33 years, respectively. Contrary to expectations, the control group increased their levels of physical activity during the study period compared to the intervention group.

Conclusion

A number of determinants affect physical activity among children and adolescents. The determinant best researched and supported by most consistent findings is patterns of exercise among parents. Other factors that play a role, and are important in other lifestyle areas, include confidence in one's own abilities and will-power, and participation in organised sport. The availability of sports facilities in the vicinity of school or home also seems to be an important factor. By contrast, it is uncertain whether television viewing in itself affects the average activity level of children and adolescents. Intervention studies have produced mixed results, but school-based programmes aiming at behaviour change and skills training, supplemented by local campaigning, have had the best effect. The greatest effect is likely to be achieved by broad-based multi-component initiatives at national, local and individual levels, but there are no intervention studies to confirm this.

Combined (diet and physical activity) intervention studies

The aim of most intervention studies which have combined dietary change and activity increase has been to reduce the risk of developing obesity or to reduce the degree of obesity. Few randomised trials have been conducted in an attempt to influence other risk factors for coronary heart disease.

Primary aim: healthier behaviour

The Middle-School Physical Activity and Nutrition study (M-SPAN) is a randomised school-based study whose aim was to increase physical activity and reduce the intake of saturated and total fat among pupils in grades 6 to 8 in 24 schools in the US (Sallis et al. 2003). The intervention took place over a 2-year period. Opportunities to increase physical activity during school time were provided, e.g. by increasing the level of activity in PE classes, improved teacher training, increased supervision and better equipment, and a more healthy diet was encouraged, e.g. by increasing the range of low-fat alternatives in school cafeterias and by providing advice to parents in order to promote healthier packed lunches. Pupil-governed health committees were set up and these organised meetings in each intervention school at which all stakeholders formulated health policies for the children's physical activity and diet. Physical activity rose significantly in the intervention schools; however this was a modest increase when compared with the control schools. The increase was observed among boys only, and was spread evenly between increased activity in PE classes and outside of PE classes. There were no differences between the schools in the intake of total fat or saturated fat. According to the authors, a major reason for the lack of effect on fat intake was the difficulty of implementing healthier dietary choice in school cafeterias, which in the US are self-financing and therefore offered the fat-rich dishes the pupils demanded.

Primary aim: reduced blood lipids

The American Child and Adolescent Trial for Cardiovascular Health (CATCH) study included over 5,000 3rd grade pupils in 96 schools in 4 states (Luepker et al. 1996). Schools were randomised for the intervention which comprised several health-promoting efforts in the school setting relating to school food service (to reduce total fat and saturated fat and also reduce the sodium content to the recommended level), PE (a minimum of 40% of PE time to be spent doing moderate to strenuous activity) and instruction on healthy life habits (goal-oriented training in skills to instil healthy dietary habits and encourage physical activity) as well as various measures targeting the family, mainly in the form of suggestions for common activity. The control schools continued with their usual school food service, PE and other instruction in health promotion. After 3 years, the intervention schools showed a considerable reduction in the fat content of school food (from 38.7 E% to 31.9 E%) as well as a reduction in the amount of saturated fat, but no reduction in the sodium content. The total daily intake of fat and saturated fat also showed a significant, though modest reduction. Intensity of physical activity in PE class and in spare time had increased among pupils in the intervention schools. Concentrations of blood cholesterol, which was the main goal of the intervention, fell slightly in both the intervention and the control schools, but there was not a significant difference between groups. There was no difference in BMI.

Primary aim: reduction or prevention of overweight

The English Active Programme Promoting Lifestyle Education in Schools (APPLES) study is a randomised intervention study targeting 7- to 11-year-old pupils in Leeds aiming at preventing overweight (primary aim). The intervention comprised class instruction in nutrition, healthy school food and improved opportunities for physical activity through the active involvement of teachers, pupils, parents and catering companies (Sahota et al. 2001a). A slightly higher intake of vegetables was recorded after 1 year compared to the control schools (an increase of one-third of a portion per day), but no effect on BMI (Sahota et al. 2001b).

The APPLES study, as well as 2 other long-term and 3 short-term studies, was included in a systematic Cochrane review of

primary prevention studies (randomised and non-randomised with a control group) (Campbell et al. 2004). The review concluded that it is not possible to generalise results of individual studies, but strategies aiming at reducing inactive lifestyles and increase physical activity have most effect with regard to preventing weight gains among children and adolescents.

A comprehensive review of several, mainly randomised studies of *overweight* children and adolescents concluded that interventions comprising behavioural treatment initiatives to improve diet and increase physical activity were merely moderately successful with regard to achieving permanent weight reduction (Epstein et al. 1998). Certain studies did, however, demonstrate beneficial long-term effects, in particular studies comprising family-based behavioural treatment and which included physical activity.

One study with these characteristics targeted 158 families with overweight children aged 6 to 12 years (Epstein et al. 1994). For 8-12 weeks the children attended weekly treatment meetings at which they received instruction in a low-energy diet, and subsequently monthly meetings for up to 9 months. In addition to the basic treatment, various groups were offered, for example, physical activity and behavioural treatment involving part or all of the family. Weight was measured 10 years later, at which time approx. one third of the children had maintained a weight loss of 20% or more, and 30% had maintained their normal weight.

In another characteristic study, also from the US, overweight children in grades 3 to 5 were offered more PE at school, healthier school food as well as a programme of spare-time activities and information on healthy dietary habits outside of school time (Donelly et al. 1996). After 2 years, it was observed that physical activity at school had gone up and that the composition of school food was healthier compared to a control school, but that these improvements were compensated for by lower levels of physical activity and unhealthier diet during spare time. These findings were supported by the failure to find differences between the intervention schools compared to the control schools in body weight, physical fitness and blood pressure as well as levels of serum cholesterol, insulin and glucose.

The Planet Health study should also be mentioned. The study comprised an interdisciplinary curriculum aimed at reducing fat intake and increasing fruit and vegetable intake, increasing physical activity and limiting time spent watching TV in order to reduce obesity among 12-13-year-old boys and girls (Gortmaker et al. 1999). Over the course of 2 years, the incidence of obesity among girls declined, but not among boys, in the intervention schools compared to the control schools. The effect was attributed mainly to a reduction in the time spent watching TV.

Story (1999) conducted a systematic review of 12 controlled school-based programmes published in the US between 1966 and 1996 whose aim was to reduce weight among overweight children. Almost all of the studies comprised behaviour modification strategies for increasing physical activity and changing dietary habits. The parents of the children were involved in only one third of the studies. The studies, all of which were relatively short-term (the longest ran over a period of 1½ years), showed a modest effect of about 10% weight loss achieved. It is of interest that almost all the studies referred to were conducted prior to 1985. The author suggests that this may be due to a greater awareness of problems of stigmatization associated with participation in studies of this kind.

A Cochrane review included 18 randomised intervention studies with overweight children, all of which lasted over 6 months (Summerbell et al. 2004). Treatments varied, but most included behaviour modification strategies with different levels of family involvement. Many of the studies were small-scale, and included homogenous and motivated groups treated in a hospital setting. It is thus difficult to apply the findings from these studies to the general population of overweight children. The study concluded that there were insufficient data to recommend one particular form of treatment rather than another.

The studies discussed above included reductions in time spent watching TV as one element in more comprehensive interventions. A randomised study looked at the effect of an (isolated) reduction in the time spent watching TV and videos and playing video games on children's weight. The intervention comprised 18 hours of class instruction inspired by social cognitive theory for grades 3 and 4 over a period of 6 months. The intervention reduced the time children spent in front of the TV by 9.6 hours per week compared to the control groups. The intervention children had also statistically significant reductions in BMI, skinfold measurements, weight circumference and waist-hip ratio, and their intake of meals consumed in front of TV was lower compared to the control children (Robinson 1999).

Conclusion

Many studies have included interventions in dietary behaviour as well as in levels of physical activity, the majority aiming at preventing or reducing overweight. Also, most studies were conducted in school settings, and only rarely with the

involvement of children's families. Almost all studies were conducted in the US. Broad-based initiatives and especially perhaps initiatives aiming at reducing inactivity and/or increasing levels of physical activity have had a good effect on the prevention of overweight. Most treatment studies have been conducted in hospital or other medical settings, and their success rate with regard to reducing the degree of overweight has been extremely limited.

Cigarette smoking

The smoking behaviour of adolescents typically passes through several transitions, from the preliminary non-smoking stage to initiation (the first cigarette) and experimentation (tried smoking a couple of times) until the adolescent is a regular smoker and dependent on nicotine (Leventhal and Cleary 1980). Progression through these stages usually takes at least 2 years, and it becomes increasingly difficult to quit the further one proceeds in the process. The risk of becoming nicotine dependent is greater among adolescents than among adults (Kandel and Chen 2000).

Prevention targeting the preliminary stage must be expected to be more effective than interventions targeting the more advanced stages in nicotine dependence. This is supported by studies showing that the earlier adolescents start smoking, the greater the dependency (Chassin et al. 1990), and the less likely they are to quit as adults (Breslau and Peterson 1996; Khuder et al. 1999).

Observational studies

Exogenous determinants

There is convincing evidence for the claim that the smoking habits of friends, (older) siblings and parents are important for the smoking habits of adolescents (Bauman et al. 2001; Boyle et al. 2001; Chassin et al. 1991; de Vries et al. 2003; Distefan et al. 1998; Dusenbury et al. 1992; Jackson and Henriksen 1997; Pederson and Lefcoe 1986; Rajan et al. 2003; Schoefield et al. 2003; Sperber et al. 2001; Swan et al. 1990; van Roosmalen and McDaniel 1989; West et al. 1999; Øygard et al. 1995). The relative importance of smoking habits in the family and among friends has not been fully clarified, but the risk seems to depend more on whether friends or older siblings smoke, than on whether parents smoke (Tyas and Pederson 1998). The initiation phase, in particular, depends on whether smoking occurs in the adolescent's immediate environment (Flay et al. 1994).

A Norwegian study, which followed over 1,200 pupils aged 12 to 19 years for 20 months, showed that parents' smoking habits particularly influenced whether their children established a regular consumption of tobacco, while the smoking habits of friends were important for children's experimentation with smoking (Pedersen and Lavik 1991). A smaller Danish prospective study of 6- to 8-year-old children showed a greater risk of being a daily smoker at the time of the follow-up 13 years later if the mother was a smoker, while factors such as parents' income, education or the smoking habits of the father did not influence smoking behaviour (Osler et al. 1996). Danish adolescents in single-parent families or in two-parent families where one of the parents is a stepparent smoked more often than adolescents living in intact biological families (Griesbach et al. 2003). Pupils in Danish schools smoked more frequently if *teachers* smoked during school time (Poulsen et al. 2002). *Idols* are also important for the smoking habits of adolescents, who, for example, report more often having experimented with smoking if their favourite film star is a smoker (Distefan et al. 1999).

The risk of becoming a smoker is greater if parents (or friends) do not display interest in whether their child smoke or not, an association particularly evident in families where neither of the parents smokes (Andersen et al. 2002; Castrucci et al. 2003; Distefan et al. 1998; Dusenbury et al. 1992; Newman and Ward 1989). A *ban on smoking* in the home or workplace reduces the risk of adolescents starting to smoke (Farkas et al. 2000; Wakefield et al. 2000). Finally, it has been established that a child more rarely starts smoking if parents quit smoking (but not as rarely as when parents have never smoked) (Bricker et al. 2003; Jackson et al. 1997), and, if the adolescent is already a smoker when the parents quit, he/she is also more likely to quit (Farkas et al. 1999).

The effect of tobacco *advertising* on the smoking habits of adolescents has been well documented. Advertising means that more non-smokers (pre-preliminary phase) will be found in the preliminary phase some years later (Pierce et al. 1998), even in families where parenting is authoritative (Pierce et al. 2002). Moreover, there is a strong association between the exposure of young non-smokers to brand-specific cigarette advertising and their subsequent uptake of smoking the brand in question (Pucci and Siegel 1999). Advertising also increases consumption among already-smokers, particularly young smokers (Armstrong et al. 1990), and a tobacco advertising ban, provided it is sufficiently effective, widely applied and consistent, will reduce tobacco consumption (Saffer and Chaloupka 2000).

Children and adolescents are also – increasingly – exposed to "hidden", but no less effective, *marketing* of tobacco through, for example, product placements in films, acquisition of promotional goods ("branded" clothes and other items) and the sponsorship of events (Wakefield et al. 2003). Children who

possess these promotional goods, or who do not object to using such goods, are more likely to become smokers later (Biener and Siegel 2000; Pierce et al. 1998; Sargent et al. 2000).

The frequency of tobacco use in *American films* has risen sharply (Stockwell and Glantz 1997). Smokers are depicted as more positive, more intelligent, romantic and sexually active than non-smokers (McIntosh et al. 1998), and negative messages about smoking feature less often in films targeting young audiences than in films targeting adults (Escamilla et al. 2000). There is a direct relationship between how frequently adolescents view smoking in films, in particular how often their idols smoke in films, and their own smoking habits (Dalton et al. 2003; Sargent et al. 2001; Tickle et al. 2001). Adolescents have more often positive attitudes to smoking when they have seen smoking scenes in films (Pechman and Shih 1999).

A comprehensive review has shown that, if enforced effectively, *legislation* banning the sale of tobacco to adolescents in combination with other initiatives in the local community, including restrictions on smoking in the workplace (school) and in public places is likely to reduce not only the frequency of smoking among adults, but also to reduce smoking uptake among adolescents (Forster and Wolfson 1998). Judging from the effect of tobacco advertising bans that have already been implemented in OECD countries, a comprehensive ban should reduce tobacco consumption by over 5% (Saffer and Chaloupka 2000). Randomised intervention studies have, however, given mixed results (see below). Schools where smoking is forbidden for teachers and pupils, and where the ban is strictly and consistently enforced, have lower smoking uptake among adolescents (Wakefield et al. 2000).

Numerous studies show that *higher prices* for cigarettes lead to a general fall in tobacco consumption (price elasticity among adults is approx. -0.4, i.e. a 10% increase in price will give a 4% reduction in cigarette consumption) (National Cancer Institute 1993). Adolescents' reactions to price increases are less clear, but, due to their lower disposable income, teenagers are more susceptible to changes in price than adults. For that reason, an increase in the price of tobacco must be assumed to have a greater impact on that age group (Ross and Chaloupka 2003).

Endogenous determinants

The personal resources of adolescents associated with *self-efficacy*, for example a high degree of "self-concept", self-esteem and the ability to say no (Barkin et al. 2002; Bonaguro and Bonaguro 1987; Holm et al. 2003; Koval and Pederson 1999; Simon et al. 1995; Young and Werch 1990) and the ability to resist peer pressure (Dielman et al. 1987; Swan et al. 1990), as well as high *competence* (Simons-Morton 2002; Wills et al. 1992), *control* (Dielman et al. 1987; Eiser et al. 1989), *social-isation skills* (Bush and Iannotti 1992), and *distancing* from risk taking (Simon et al. 1995) are all associated with a reduced risk of becoming a smoker.

Curiosity and *rebelliousness* may also be among the factors contributing to adolescents' uptake of smoking (Koval and Pederson 1999).

The teenage years are often characterized by *worry* and *anxiety*, leading many to believe that these feelings linked to low self-efficacy increase the risk of smoking uptake (Bonaguro and Bonaguro 1987; Byrne et al. 1995; Koval and Pederson 1999). Some prospective studies have shown, however, that smoking increases the risk of anxiety symptoms among adolescents (Johnson et al. 2000). Adolescents with depressive symptoms have a higher risk of commencing smoking (Escobedo et al. 1998).

Traumatic experiences during childhood (sexual, psychological and physical abuse; parents' divorce; violence towards the mother; drug or alcohol abuse or mental illness in parents) are associated with an increased risk of smoking (Anda et al. 1999).

Adolescents who start smoking have more frequently positive *attitudes* towards smoking, including the psychological effects of smoking, and it is characteristic that they are more susceptible to tobacco advertising and promotional goods, that they underestimate the health-damaging and habit-forming effects of tobacco, and believe that many more of their friends smoke than is the case (Barkin et al. 2002; Botvin et al. 1992; Castrucci et al. 2002; Chassin et al. 1991; Choi et al. 2002; Romer and Jamieson 2001; Simons-Morton 2002; Virgili et al. 1991). By contrast, greater knowledge of the damaging effects of smoking does not appear to be associated with a reduced risk of smoking (Jensen and Overgaard 1993; Schofield et al. 2003). In order for improved knowledge to reduce the risk of becoming a smoker, it probably needs first to be relevant to the individual, so it can be motivational (be internalised) (Eiser et al. 1989).

Concerns about *weight* is one reason why many teenagers (and betweenagers), especially girls, start smoking. (Tomeo et al. 1999). In two large-scale studies of young girls who were non-smokers at the start of the studies, weight concerns and dieting were associated with higher rates of smoking initiation during the study period of over 1 year (Austin and Gortmaker 2001; French et al. 1994).

It is likely that *genetic* factors are of some consequence during several stages of smoking behaviour (and explain in part the association with the smoking habits of parents and siblings), however appear to be less important than social and other environmental factors (Vink et al. 2003; White et al. 2003). Researchers are looking especially at genetic variation in nicotine metabolism (Pianezza et al. 1998) and receptor affinity (Spitz et al. 1998).

Intervention studies

Numerous studies have been conducted to examine the immediate effect of various smoking cessation programmes with a view to reducing the incidence of smoking among adolescents. Findings indicate that school-based interventions, in particular interventions applying the social-influence model, result in lower numbers of adolescent smokers, more adolescents postponing their smoking initiation and changes in attitudes to smoking (Bruvold 1993; Center for Disease Control 1998; DuRant and Smith 1999; Institute of Medicine 1994).

In studies that follow adolescents over longer periods, up to 4-5 years, the difference between the numbers of smokers in the intervention and control groups appears to be considerably diminished (Tyas and Pederson 1998). Repeated or ongoing initiatives are therefore necessary to maintain the positive short-term effect. A comprehensive review of the literature from 1994 to the beginning of 2000 concluded that intervention studies had had mixed results, and that school-based interventions were not sufficient in themselves. In order to have relevant and long-lasting impacts on the smoking behaviour of adolescents, interventions need to be combined with community-based initiatives involving the family, and include a wide spectrum of social and political initiatives (Lantz et al. 2000).

This conclusion is supported by conclusions from two systematic Cochrane reviews, which examined the effect of interventions carried out in school settings and interventions in the immediate environment (e.g. a ban on the sale of tobacco, a smoking ban in public places, the use of the media in information campaigns and the involvement of schools and sports clubs).

In all, 76 randomised and controlled studies were published, whose aim was to change behaviour in school settings in order to prevent adolescents taking up smoking (Thomas 2004). Of the 76 studies, only 16 were of sufficiently high scientific quality for inclusion, and 15 of these used social influence as the intervention strategy. 8 of these 15 studies demonstrated a positive, though limited and short-term, effect on the incidence of smoking among adolescents, while 7 studies failed to demonstrate an effect.

It is remarkable, however, that the largest, longest-running and best-conducted study in a school setting, the American Hutchinson Smoking Prevention Project, failed to show a difference in the incidence of smoking between the intervention and control schools (Peterson et al. 2000). The study included 40 school districts in the state of Washington, where over 8,000 pupils were randomised into schools following the usual programme or schools offering an intensive programme building on social influence for children in grades 3 to 10 and delivered by (specially-trained) teachers. 10 and 12 years after the start of the intervention, an examination of the incidence of smoking showed a remarkable similarity between the numbers of smokers in the control and intervention schools, regardless of how results were presented, e.g. smoking frequency, number of cigarettes, age at smoking initiation, stage in development of smoking habit, smokers in family. The study concluded that programmes targeting schools only are not sufficient, but should probably include additional initiatives in the family and local community.

The other Cochrane review dealt with intervention studies whose main aim was to effect changes in the local community in order to reduce the incidence of smoking among adolescents (Sowden et al. 2004). 63 intervention studies were identified, of which 17 were of sufficiently high quality to meet the inclusion criteria. In the majority of studies, 13 studies, the incidence of smoking was compared with the incidence in a control population, and 2 studies reported a significantly lower incidence of smoking among adolescents. Of 3 school-based interventions, 1 study showed a significant reduction in smoking. The authors concluded that interventions of this kind have only a limited effect on reducing the risk of adolescent uptake of smoking.

Several wide-reaching multimedia campaigns (TV, radio, newspapers, Internet), often in combination with school-based programmes, have achieved some success (Sowden and Arblaster 2004; Wakefield et al. 2003). It is uncertain which forms of mass-media campaigns are most effective, and to some extent this is culturally determined, but campaigns playing on fear (scare tactics; fear appeals) in descriptions of health consequences have been most effective (Hill et al. 1998; Witte 1992).

A controlled study in Massachusetts, USA, showed that stricter enforcement (control, fines) of a ban on the sale of tobacco products to adolescents (<18 years) is not sufficient to reduce the incidence of smoking. A sales ban did have an effect on retailers who increasingly acted in accordance with

legislation and more frequently refused to sell tobacco products to adolescents. However, it remained easy for adolescents to purchase tobacco products, and smoking habits remained unchanged (Rigotti et al. 1997). Another controlled study, conducted in Minnesota, USA, recorded a reduction in tobacco sales and a decline in the number of adolescent smokers in local areas where legislation banning tobacco sales to adolescents was strictly enforced, compared to control areas (Blaine et al. 1997).

Other studies have examined the effect of various programmes aimed at encouraging young smokers to quit. Sussman et al. (1999) report on 17 experimental studies with this purpose. Most studies did not include a control group, and were typically school-based, drew on very different theories and were relatively short-term. About 20% had quit smoking at the end of the intervention, an incidence that had fallen to 13% 6 months later – compared to a 10% "spontaneous" cessation.

Two other reviews have been published of studies whose aim was to encourage young smokers to quit, and both report a very limited success rate of an average of about 10% cessation in both the intervention and control groups (Garrison et al. 2003; Moolchan et al. 2003). The studies had typically a short follow-up period (<1 year), included selected populations (adolescents wishing to quit) and a high dropout rate. The effect of a reduction in tobacco consumption (instead of total cessation) on smoking habits among adults is not known.

It should be mentioned that there was only 1 randomised study among the school-based studies reviewed (Adelman et al. 2001). This study randomised subjects into a group of adolescents wishing to quit smoking who received 8 hours of individual and group-based intervention, over a period of 6 months, based on the principles of *stages of change* theory. The control group received only a leaflet encouraging them to quit smoking. At the end of the intervention, there were significantly more adolescents in the intervention group than in the control group who had quit smoking (59% vs. 17%). The number of non-smokers in the intervention group fell by 50%, however, during the course of the following 20 weeks.

A low dropout rate was achieved in a randomised study which included adolescents who had been caught smoking during school hours, and who risked disciplinary action or expulsion, and were therefore presumably motivated to quit smoking (Robinson et al. 2003). The intervention group was offered 2 months of individual therapy (based on the principles of social influence) followed by regular controls, and the control group received only a leaflet containing advice and encouragement on smoking cessation. After 1 year, a minor and similar fall was observed in the number of smokers in both the intervention and the control groups.

Conclusion

The longer smoking initiation is postponed, the greater is the likelihood of smoking cessation in adulthood – and the lesser the risk of adolescent uptake of smoking. Extensive research – though still relatively few longitudinal studies – has identified a number of determinants which, to greater or lesser extents, govern different stages in the smoking behaviour of adolescents. The behaviour and attitude of friends, family and society towards smoking have a decisive effect on whether adolescents take up smoking. Bans on the marketing and sale of cigarettes to children and adolescents as well as higher prices all bring down the incidence of smoking. Adolescents' personal attributes, e.g. self-efficacy, are also very important. Comprehensive programmes to limit adolescent smoking in several American states have resulted in considerable reductions in tobacco consumption. Strategies included media-borne information, legislation banning smoking in schools and public institutions, bans on the sale of tobacco to adolescents and high price increases on tobacco products in combination with a wide range of local initiatives, for example the establishment of smoking policies at workplaces and more comprehensive school-based programmes as well as the establishment of smoking cessation programmes for individual citizens (Wakefield and Chaloupka 2000).

It should be noted that the majority of this research was conducted in the US, and it is uncertain whether results can be generalised to apply to countries with other "smoking cultures".

Broader-based intervention studies

Two Nordic studies have been conducted which included interventions in risk behaviour (smoking, diet and physical inactivity) at different levels. Even though these were not randomised studies, they deserve a mention in this context.

The Finnish North Karelia Youth Project took place between 1978 and 1980. This non-randomised study included 7-grade pupils and comprised 3 "arms": 1) a small intensive school-based intervention; 2) a broader-based intervention in local schools; and 3) a control group (Vartiainen et al. 1991). The intervention included training in resisting peer pressure (smoking) and instruction in healthy eating habits delivered by trained project workers and school teachers, as well as changes in school meals (less fat, increased polyunsaturated fat, less salt), health checks and health talks with the school

nurse, and information via the mass media. After 2 years there were fewer smokers among the adolescents in the intervention schools than among those in the control schools, a difference which was still present in a follow-up investigation 10 years later, but not 15 years later – a convergence that was explained by a general decline in Finnish smoking habits, which was greater in the earlier control group than in the intervention group (Vartiainen et al. 1998). Dietary changes were only maintained for a shorter period, but were more pronounced in the intervention schools than in the control schools. Despite this, there were no significant differences in blood cholesterol or blood pressure between the schools at the end of the programmes.

The Oslo Youth Study was a similar non-randomised school-based study whose aim was to demonstrate that health promotion among young people could achieve reductions in smoking frequency, improved eating habits and higher levels of physical activity. The study was conducted between 1979 and 1981. Pupils in grades 5-7 at 3 schools in Oslo, Norway, received a multiple intervention programme, and the effect on risk behaviour and risk factors was compared with 3 control schools, who received the usual information. A sharp increase in smoking among adolescents in both the intervention and control schools was observed in a follow-up investigation 10 years later (from about 10% to about 50% who reported smoking weekly), without any great differences in smoking habits between the intervention and control schools, but significantly more young men who had attended the intervention schools, and who were non-smokers at the start of the study, continued to be non-smokers 10 years later, compared to non-smokers in the control schools (58% vs. 44%), while there was no differences in the numbers of women who were non-smokers Klepp et al. 1993). The total cholesterol and HDL cholesterol levels of about 490 pupils were measured at the start of the study and 2 years later. Compared to the control group, a net reduction in total cholesterol of 4% was recorded among girls and boys (both significant). There were no differences in HDL cholesterol.

Safety of intervention programmes

It is possible that an (excessive) focus on one particular healthy eating pattern can interfere with adolescents' food choices, eating patterns and body image. It has been feared that an increased focus on healthy eating habits could push young girls at risk of eating disorders into an eating disorder. There are, however, no studies indicating that dietary information, as it is normally delivered, aiming at limiting the consumption of sugar and fat in a population, has any effect on the incidence of eating disorders among children and adolescents. On the contrary, weight loss among overweight children and adolescents is accompanied by generally improved psychological functions (Epstein et al. 2001). The large-scale intervention studies discussed above have failed to show any increase in the prevalence of eating disorders.

Reduced growth is a well-known complication of a low intake of energy. In the western world, extreme forms of diets have led to growth inhibition, e.g. macrobiotic diets (van Staveren and Dagnelie 1988), or uncontrolled low-energy diets among children with hypercholesterolemia (Lifshitz and Moses 1989) or obesity (Pugliese et al. 1987). There has therefore been some concern that an energy- and/or fat-reducing diet, corresponding to 20-30 E% fat, for children and adolescents could result in insufficient food intake and reduced growth, particularly if recommendations on fat reduction were extended to the general population. A low-fat intake among children does not reduce growth and is nutritionally more efficacious than a diet with a normal or high fat content (Clauss and Kwiterovich 2002; Oberzanek et al. 1997; Sanchez-Bayle and Soriano-Guillén 2003). Neither has any relationship been found between energy intake and anthropometric variables within the usual intake intervals in the population (Boulton and Magaray 1995; Epstein et al. 1990; Shea et al. 1993).

OVERALL CONCLUSIONS

Atherosclerosis develops over a very long time, from the time the first microscopic changes occur in the artery wall until the manifestation of clinical symptoms, most frequently in the form of an occlusion in the arteries supplying the heart, brain or leg. Demonstrable atherosclerotic lesions can already be observed during the teenage years, and are present in about a quarter of 25- to 35-year-olds. There is a clear relationship between risk behaviour and cardiovascular risk factors in children and adolescents and the early development of atherosclerosis and the later risk of coronary heart disease in adults.

The lifestyle factors that are of particular importance for the risk of atherosclerosis and coronary heart disease in children and adolescents are the composition of the diet, the degree of physical activity and the incidence of smoking.

Both lifestyle and cardiovascular risk factors track from childhood and adolescence into adulthood. This makes it possible to identify individuals who are more likely to have a higher risk of heart disease as adults, on the basis of the presence of an unhealthy lifestyle and/or high level of cardiovascular risk factors early in life. The predictive values are weak, however, weaker for lifestyle than for risk factors. This means that a large number of children will have changed their lifestyle and risk factors as adults. Initiatives to improve health behaviour should therefore be directed at all children and adolescents, and general screening of children for the presence of risk factors would not therefore be appropriate.

Several determinants have been identified that appear to influence food choices, physical activity and smoking. Determinants have different degrees of influence, depending on, for example, age and sex, and possibly also on a large number of social and cultural factors. Many are common determinants, but that does not mean that an intervention initiative aiming at increasing the intake of fruit and vegetables through, for example, improved self-efficacy, will automatically increase physical activity, and vice versa. There is scientific evidence to suggest that changes made simultaneously in several lifestyle factors have an additive or synergetic effect on cardiovascular risk factors. For this reason, interventions should perhaps to a greater extent include diet and physical activity and smoking.

Family and friends are determinants of the dietary intake, food choices and eating habits of children and adolescents, but so are, to an increasing extent, portion sizes, and eating out, especially in fast food outlets. Advertising also influences the food choices of children and adolescents. Availability is crucial to the intake of healthy food among children and adolescents. The effect of pricing has not been examined adequately. The exercise patterns of parents seem to influence greatly whether children are physically active. But other factors seem also to be important, e.g. self-efficacy, will-power, participation in organised sport and the presence of sports facilities in the vicinity of home or school. It is not clear whether television viewing, on the other hand, influences children's levels of daily physical activity. With regard to smoking, research has identified a number of determinants that come into play, to varying degrees, at different stages in the smoking behaviour of adolescents. The behaviour and attitude of friends, family and society towards smoking have a decisive effect on whether adolescents take up smoking. Banning the marketing and sale of cigarettes to children and adolescents and higher prices combine to reduce the incidence of smoking. The personal attributes of the adolescent, e.g. good self-efficacy, are also very important.

A number of intervention trials have been conducted in "the field", building on different theoretical models. Trials have most often been carried out in school settings, and included the family and local community less often. Generally, these initiatives have shown that it is possible to bring about appropriate changes in particular lifestyles as long as the intervention is ongoing, but follow-up investigations have typically shown gradual congruence in effect measures between the intervention and control groups. A sustained effort is thus required in order to maintain healthier lifestyles.

Comprehensive, ongoing and multi-component programmes conducted in the US to limit adolescent smoking have resulted in considerable reductions in tobacco consumption. Strategies included media-borne information, legislation banning smoking in schools and public institutions, bans on the sale of tobacco to adolescents and high price increases on tobacco products in combination with a wide range of local initiatives, for example the establishment of smoking policies at workplaces and more comprehensive school-based programmes as well as the establishment of smoking cessation programmes for individual citizens. Similar findings do not exist for the areas of diet or exercise.

It appears that the greatest effect is achieved through broad-based, multi-component efforts at national, local and individual levels. It should be noted that the majority of this research was conducted in the US, and it is uncertain whether results can be generalised to apply to countries with other lifestyle cultures.

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