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CARBOHYDRATE INTAKE IN CHILDREN

– associations with dietary intakes, growth, serum lipids, and dental health

The STRIP Project

by

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ABSTRACT

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Carbohydrate intake in children – associations with dietary intakes, growth, serum lipids, and dental health. The STRIP Project. The Research Centre of Applied and Preventive Cardiovascular Medicine, the Department of Pediatrics and the Department of Medicine, University of Turku, Turku, Finland. Annales Universitatis Turkuensis, Medica-Odontologica,Turku, Finland, 2011.

This study is part of the STRIP study, which is a long-term, randomized controlled trial, designed to decrease the exposure of children in the intervention group (n=540) to known risk factors of atherosclerosis. The main focus of the intervention was the quality of dietary fat. The control group (n=522) did not receive any individualized counselling. Food consumption was evaluated with food records, and blood samples were drawn and growth was measured regularly for all participating children from 13 months to 9 years. A subsample of 66 children participated in a dental health survey.

The number of studies on children's carbohydrate intake, especially fibre intake, is insufficient. The current international recommendations for fibre intake in children are based on average assumptions and data extrapolated from intakes in adults and intake recommendations for adults. Finnish nutrition recommendations lack strict recommendations for dietary fibre in children. Due to fibre's high bulk volume, excessive dietary fibre is considered to decrease energy density and hence it may have an adverse effect on growth. If fats are reduced from the diet, the low-fat diet may become high in sucrose. Therefore, especially in the STRIP study, it is important to determine the use of fibre and sucrose in children and possible associations with growth and nutrition as well as dental health.

The results of the present study indicate that a high fibre intake does not displace energy or disturb growth in children and that children with high fibre intake have better quality of diet than those with low fibre intake. Additionally, dietary fibre intake associated inversely with serum cholesterol concentration. Other carbohydrates also affected serum lipid levels as well, since total carbohydrates, sucrose, and fructose increased serum triglyceride concentration. Total carbohydrate intake reduced HDL cholesterol concentration only in children with apoE3 or apoE4 phenotype.

Over the period from the 1970s to 1990s dental health of children in Finland has substantially improved, mostly due to improved dental hygiene and the use of fluorine, despite an increase in sucrose intake. However, during the past twenty years improvement in dental health has stopped. The present study showed that high long-term sugar intake increases risk of caries in children. High intake of sugar had also negative effects on the diet of children, because it worsens dietary quality by displacing essential nutrients. Furthermore, the quality of dietary fat was worse in children with high sucrose intake. Interestingly, children's high sucrose intake was not associated with overweight but inversely with growth.

Key Words: carbohydrate, children, cholesterol, dental health, diet, dietary fibre, growth, nutrition, sucrose, sugar

TIIVISTELMÄ

Soile Ruottinen

Hiilihydraattien saanti lapsilla – yhteydet muiden ravintoaineiden saantiin, kasvuun, veren rasva-arvoihin ja hammasterveyteen. STRIP -projekti. Sydäntutkimuskeskus, Lastentautioppi ja Sisätautioppi, Turun yliopisto, Turku. Annales Universitatis Turkuensis, Medica-Odontologica,Turku, 2011.

Tämä väitöskirjatyö on osa pitkäkestoista ja satunnaistettua STRIP -tutkimusta, jonka tavoitteena on vähentää interventioryhmän (n=540) lasten altistumista tunnetuille valtimoiden kovettumataudin riskitekijöille. Intervention painopisteenä on ollut ruokavalion rasvan laatu. Kontrolliryhmän lapset (n=522) eivät ole saaneet erityistä henkilökohtaista ohjausta. Tutkimuslapsilta kerättiin ruokapäiväkirjat, otettiin verinäytteet ja heidät mitattiin sekä punnittiin säännöllisesti 13 kk-9 vuoden iässä. Osa (n=66) tutkimuslapsista osallistui hammasterveyttä kartoittavaan tutkimukseen.

Lasten hiilihydraattien saantia on tutkittu riittämättömästi, varsinkin ravintokuidun osalta. Kansainvälisesti lasten kuidun saantisuositukset ovat perustuneet keskimääräisiin arviointeihin ja aikuisten suosituksiin. Lasten kuidun saannille ei ole suomalaisia suosituksia. Ravintokuidun on ajateltu suuren energiattoman volyyminsa takia pienentävän energian saantia ja sitä kautta voivan vaikuttaa haitallisesti lasten kasvuun. Kun ruokavaliosta vähennetään rasvaa, se saattaa lisätä sokerin käyttöä. Sen takia erityisesti STRIP – tutkimuksessa on tärkeää selvittää lasten kuidun ja sokerin käyttöä ja niiden yhteyksiä kasvu- ja ravintotekijöihin sekä hammasterveyteen.

Tutkimuksessa kävi ilmi, ettei runsaastakaan kuidun saannista ole haittaa lasten kasvulle, ja että kuitu parantaa lasten ruokavalion laatua. Lisäksi havaittiin, että ravintokuitu oli yhteydessä alhaisempiin veren kolesterolipitoisuuksiin. Myös muut hiilihydraatit vaikuttivat veren rasva-arvoihin, sillä kokonaishiilihydraatti, sokeri ja fruktoosi suurensivat seerumin triglyseridiarvoja. Kokonaishiilihydraatin saanti pienensi HDL kolesterolia vain niillä lapsilla, joilla on apoE3- tai apoE4-fenotyyppi.

Lasten hammasterveys on parantunut Suomessa 1970-1990, paranemisen ajateltiin olevan seurausta parantuneesta hammashygieniasta ja fluorin käytöstä, lisääntyneestä sokerin käytöstä huolimatta. Viimeisten 20 vuoden aikana hammasterveyden paraneminen on pysähtynyt. Tässä tutkimuksessa todettiin, että pitkäaikainen runsas sokerin käyttö lisää edelleen riskiä lasten hampaiden reikiintymiselle. Runsaalla sokerin käytöllä oli myös negatiivisia vaikutuksia lasten ruokavalioon, sillä se huononsi ruokavalion laatua vieden tilaa tärkeiltä ravintoaineilta. Myös ruokavalion rasvan laatu on huonompi runsaasti sokeria käyttävillä lapsilla. Lasten runsas sokerin käyttö ei ollut yhteydessä ylipainoon, mutta sokerin käytöllä oli heikentävää vaikutusta lasten pituuskasvuun.

Avainsanat: hammasterveys, hiilihydraatti, kasvu, kolesteroli, lapset, ravintokuitu, ravitsemus, ruokavalio, sakkaroosi, sokeri

CONTENTS

ABBREVIATIONS

AAP	American Academy of Pediatrics
AHF	American Health Foundation
ANOVA	Analysis of variance
ANCOVA	Analysis of covariance
Apo	Apolipoprotein
BMI	Body mass index
CAPC	The Research Centre of Applied and Preventive Cardiovascular Medicine
CATCH	The Child and Adolescent Trial for Cardiovascular Health
CARDIA	The Coronary Artery Risk Development in Young Adults
CHD	Coronary heart disease
CVD	Cardiovascular diseases
CI	Confidence interval
DIPP	The Type 1 Diabetes Prediction and Prevention study
DMFT	Sum of decayed, missing and filled teeth in the permanent teeth
dmft	Sum of decayed, missing and filled teeth in the primary teeth
DONALD	the Dortmund Nutrition and Antropometric Longitudinally Designed Study
DRI	Dietary Reference Intake
E%	Percentage of total daily energy intake
EAR	Estimated Average Requirement
EFSA	European Food Safety Authority
EPIC	European prospective investigation into cancer and nutrition
ESPGHAN	European Society of Paediatric Gastroenterology, Hepatology and Nutrition
FAO	Food and Agriculture Organization of the United Nations
FFQ	Food frequency questionnaire
HDL	High-density lipoprotein
ILSI	International Life Sciences Institute
IoM	U.S. Institute of Medicine of the National Academy of Sciences
LDL	Low-density lipoprotein
Md	Median
MUFA	Monounsaturated fatty acids
NDA	EFSA Panel on Dietetic Products, Nutrition and Allergies
NNR	Nordic Nutrition Recommendations
NMES	non-milk extrinsic sugars
NSP	Non-starch polysaccharides
PUFA	Polyunsaturated fatty acids
RDA	Recommended Dietary Allowance
RM ANCOVA	Repeated measures analysis of covariance
RM ANOVA	Repeated measures analysis of variance
SFA	Saturated fatty acids
SD	Standard deviation
STRIP	Special Turku coronary Risk factor Intervention Project
T1-T5	Tanner stage 1-5
WHO	World Health Organization
VLDL	Very low-density lipoprotein

LIST OF ORIGINAL PUBLICATIONS

The present thesis is based on the following original publications, which are referred to in the text by the Roman numerals (I-IV). Some previously unpublished data are also presented.

I Ruottinen S, Rönnemaa T, Niinikoski H, Lagström H, Saarinen M, Pahkala K, Kaitosaari T, Viikari J, Simell O. Carbohydrate intake, serum lipids and apolipoprotein E phenotype show association in children. Acta Paediatr 2009;98:1667-73.

II Ruottinen S, Lagström HK, Niinikoski H, Rönnemaa T, Saarinen M, Pahkala KA, Hakanen M, Viikari JS, Simell O. Dietary fiber does not displace energy but is associated with decreased serum cholesterol concentrations in healthy children. Am J Clin Nutr 2010; 91:651-61.

III Ruottinen S, Niinikoski H, Lagström H, Rönnemaa T, Hakanen M, Viikari J, Jokinen E, Simell O. High sucrose intake is associated with poor quality of diet and growth between 13 months and 9 years of age: the Special Turku Coronary Risk Factor Intervention Project. Pediatrics 2008;121:e1676-85.

IV Ruottinen S, Karjalainen S, Pienihäkkinen K, Lagström H, Niinikoski H, Salminen M, Rönnemaa T, Simell O. Sucrose intake since infancy and dental health in 10-year-old children. Caries Res 2004;38:142-8.

1 INTRODUCTION

Cardiovascular diseases (CVD) are still the most common cause of death in Finland, accounting for 41% (20235) of all deaths in 2009 (Statistics Finland 2010); coronary artery disease accounted for 22% (11534) of all deaths. Coronary artery disease is considered the most significant national disease in Finland, and its main classic risk factors are high LDL cholesterol, smoking, and high blood pressure. Amongst nutritional reasons behind the high prevalence of this national disease are e.g. excessive intake of saturated fatty acids (SFA) and salt and insufficient intake of dietary fibre.

Several studies have shown that atherosclerotic changes occur in the circulatory system already in childhood (Pesonen 1974, Davies 1990). The extent of the changes is related to serum cholesterol levels even in children (Newman et al. 1986). Males with high serum cholesterol values in their youth have a quadruple risk of developing coronary heart disease (CHD) as adults compared with males that have low values in their youth (Klag et al. 1993). The prevention of CHD should start as early as possible, since atherosclerosis is a process of decades (Davies 1990), and because it may be possible to affect future nutritional habits already in childhood. The first years of life may be critical for the establishment of good eating habits. Diet during infancy and early childhood must not only support normal growth and development but is also the foundation of adult health. High-quality diet with optimal levels of food and nutrients help to maintain optimal health.

At the global level, 40-80 percent of human dietary energy comes from carbohydrates, the primary energy source for the human metabolic system. According to the Finnish nutrition recommendations (National Nutrition Council 2005) and the Nordic Nutrition Recommendations (NNR) (Nordic Council of Ministers 2004), the proportion of carbohydrates should represent 50-60% of the total energy intake from age three onwards. Food carbohydrates are mostly mono-, di-, and polysaccharides. Measured in volumes, the most important absorbing carbohydrate is starch. Rapidly available carbohydrates are decomposed and absorbed in the small intestine into sugar, mainly in the form of glucose, for use by all cells. Non-absorbing carbohydrates, which means dietary fibre or the same kind of substances, are transferred to the large intestine where they provide food for intestinal bacteria.

Carbohydrate intake in Finnish men is slightly below the recommended level. According to the National FINDIET 2007 Survey, the energy intake from carbohydrates is 47.7% for men and 50.5% for women in Finland (Paturi et al. 2008). The average daily intake of dietary fibre for men is 24 g and for women 21 g, while the recommended level is 25 - 35 g. To improve national food intake and to maintain the good public health status in Finland dietary fibre intake should be higher and sucrose intake lower than the present level. The goal is based on the health benefits of fibre. Increasing the use of food containing dietary fibre would significantly remedy the nutritional defects in the Finnish diet, leaving less room for fat and sugar and also increasing fibre intake.

The term *dietary fibre* refers to plant foods that are resistant to digestion by human digestive enzyme or other similar dietary substances. Fibres are functionally important

for the health of children: they are claimed to improve normal bowel function, prevent gastrointestinal disorders, prevent and treat obesity, reduce cholesterol values and lower plasma glucose excursions after meals (Williams 1995). Sufficient fibre intake in adults has also been associated with prevention of cardiovascular diseases, type 2 diabetes and presumably some cancers. High-fibre foods can help to reduce serum cholesterol concentrations by as much as 6 - 23 % (Glassman et al. 1990). A high-fibre diet contains less fat, cholesterol and energy than a low-fibre diet (Nicklas et al. 1995a,b). The current U.S. dietary recommendations for fibre intake in children are based on assumptions and data extrapolated from studies in adults (Edwards and Parett 2003). No specific fibre intake guideline for children is provided in the Finnish nutrition recommendations (National Nutrition Council 2005) or the NNR (Nordic Council of Ministers 2004). According to the Finnish nutrition recommendations, too much fibre in the diet of children under the age of three reduces the energy density of food and the food volume to meet the child's energy needs may become too high. Currently the research data available on dietary fibre intake in children is too limited to allow precise recommendations.

The term *refined sugar* refers to sucrose, fructose, starch-based sweeteners (syrups of glucose-fructose) and other similar sugar products used either alone or added to food during preparation (National Nutrition Council 2005). High sugar content reduces the nutritional density of food. Sugars, such as sucrose, fructose, and other carbohydrates that are absorbed partially already in the mouth are often detrimental to oral health. Since the 1970s, the dental health of Finnish children at pre-school and school age has substantially improved, but during the past twenty years caries has become common again.

Especially for children and for those adults whose energy need is small, sugar intake should be limited to 10 % of the total energy intake (E%) according to the Finnish nutrition recommendations (National Nutrition Council 2005). It is essential to limit high and repeated use of sugar-containing beverages and sweets to prevent obesity and dental damage. From the nutritional point of view, the main reason for limiting the amount of refined sugar is ensuring sufficient intake of essential nutrients and dietary fibre. Recently, high intake of sugar and low intake of fibres have been reported in children (Kranz et al. 2005a,b, Kyttälä et al. 2008). However, dietary data on Finnish children are sparse (Erkkola et al. 2009).

This thesis is a part of the Special Turku coronary Risk factor Intervention Project (STRIP), which is a prospective, randomised, long-term trial designed to decrease the exposure of children to known environmental atherosclerosis risk factors with infancy-onset intervention. The intervention children were counselled to a fat intake of 30 E% - 35 E% for the child before 3 years of age and later the fat content of diet was targeted to 30 E%. Main target from the beginning was to replace part of saturated fat with unsaturated fat. It has been proposed that counselling for low-fat diet may increase sugar intake. Therefore, it was of utmost importance to investigate in this project the quality of carbohydrate, and the amount of sugar and dietary fibre. The aim of the present study is to describe the role of dietary carbohydrates in children's diet, by evaluating the intakes of carbohydrate and its subgroups and investigating associations between carbohydrates and nutrition, growth, serum lipids, and dental health.

2 REVIEW OF THE LITERATURE

2.1 Classification of dietary carbohydrates

Dietary carbohydrates are a diverse group of substances with a range of chemical, physical, and physiological properties (Cummings and Stephen 2007). Primary classifications of carbohydrates are based on chemistry, and since the early 1900s carbohydrates have been classified according to their chain length (Pereira and Liu 2003). In 1977, the term *complex carbohydrates* was first used in the McGovern report (Dietary Goals for the United States in 1977). The term *complex carbohydrates* function was to distinguish sugars (simple sugars) from other carbohydrates and in the report it meant fruits, vegetables, and cereals. At present the term c*omplex carbohydrates* usually refers to starch alone or the combination of all polysaccharides, or it can be defined as either starch or fibre (Griel et al. 2006). However, the term has not been formally defined. *Simple sugars* (*simple carbohydrates*) refer to monosaccharides and disaccharides (Sigman-Grant and Morita 2003, Griel et al. 2006, Cummings and Stephen 2007).

The current primary classification of dietary carbohydrates was proposed at the Joint Food and Agriculture Organization (FAO)/World Health Organization (WHO) Expert Consultation on Carbohydrates in human nutrition convened in Rome in 1997 (FAO/WHO 1998) by a chemical approach. The chemical approach divides carbohydrates into three main groups: sugars, oligosaccharides, and polysaccharides (FAO/WHO 1998) (Figure 1). Polyols (hydrogenated carbohydrates, sugar alcohols) are naturally found in some fruits and are made commercially, and are used as sweeteners. This group can be defined as a subgroup of sugars (FAO/WHO 1998) or as the fourth main group (ILSI 2003).

Sugars consist of monosaccharides with only one monomeric unit (one single sugar): glucose, fructose or galactose; disaccharides with two monomeric units: sucrose, lactose, maltose, trehalose; oligosaccharides which have chains of 3 to 9 glucose or fructose polymers, and polysaccharides having 10 or more monomeric units with starch and dietary fibre being the main components (Wheeler and Pi-Sunyer 2008). Starch is the principal carbohydrate in diets worldwide; it is the storage carbohydrate of plants such as cereals, root vegetables, and legumes and consists of only glucose molecules (Cummings and Stephen 2007).

For estimating the intakes of carbohydrates terminology is important, because it conveys reliable and comparable results. It is possible that the method of carbohydrate determination is not the same in different countries when carbohydrate intake is compared (Cummings and Stephen 2007). Worldwide variations in carbohydrate intake are assumed to be due to differences in food culture and partly due to local methodologies.

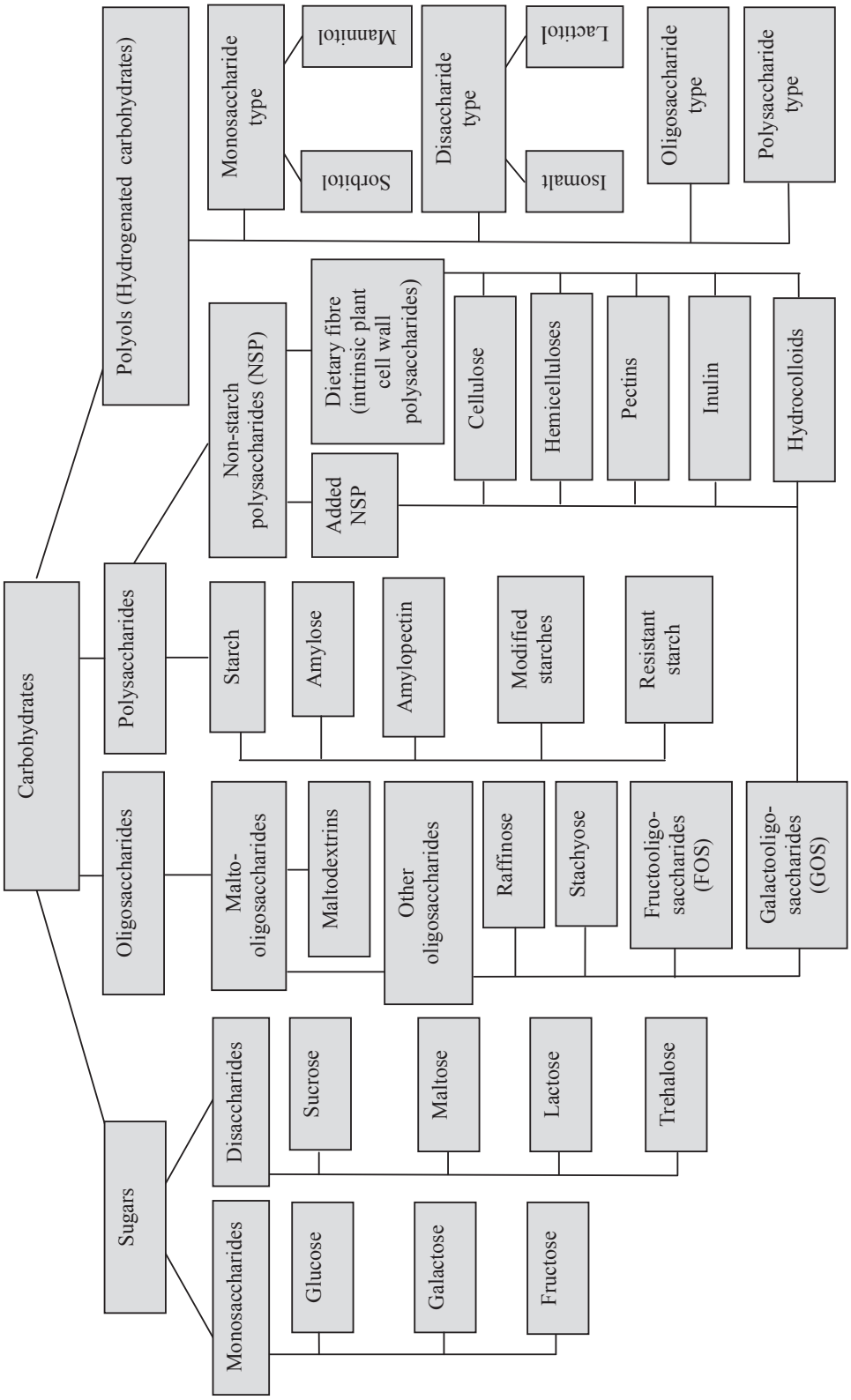


Figure1. Structural classification of main types of carbohydrates (Adapted from Asp 1996, ILSI 2003 and FAO/WHO 1998).

2.1.1 Definition of dietary fibre

The term *dietary fibre* has many different meanings and is not a precise reference to a chemical component of the diet (Cummings and Stephen 2007). Dietary fibre is a complex and heterogeneous class of substances, with a variety of definitions, some based on analytical methods and others on a physiological basis.

The concept "dietary fibre" was first used by Hipsley (1953) to include lignin, cellulose, and hemicelluloses. The original and widely used definition was by Trowell in 1972: "The proportion of food which is derived from the cellular walls of plants which is digested very poorly in human beings". The definition is based on an analytical method that measures plant cell wall and other indigestible plant polysaccharides plus lignin (Aggett et al. 2003).

Since Trowell's definition more definitions has been proposed. Various national and international bodies continue to struggle with the definition (Cummings and Stephen 2007). Definitions are needed for non-digestibility, which varies from person to person and includes many dietary components other than "normal dietary fibre" such as lactose in some people, and polyols and starches in others. The definition of dietary fibre should probably be more clearly linked to health. Therefore it has been proposed that: "dietary fibre consists of intrinsic plant cell wall polysaccharides", which are a good marker of fruits, vegetables, and whole grain (Cummings and Stephen 2007). Plant cell walls have a central role in defining the high nutrient density with respect to vitamins and minerals, among others (Englyst 2007).

Generally, dietary fibre has been classified as *soluble* and *insoluble fibre*, but more recent studies have suggested that other properties of fibre, for example fermentability or viscosity, are more important parameters. The Institute of Medicine (IoM) report (2005) and FAO/WHO report on dietary carbohydrates (FAO/WHO 1998) recommend that the terms *soluble* and *insoluble* fibre should be phased out of use because these divisions are useful neither analytically nor physiologically. Furthermore, according to Buttriss and Stokes (2008): "The soluble/insoluble categorisation has since been recognised to be misleading because some "insoluble" fibres are in fact fermented in the large bowel and solubility in water does not always predict physiological effects." However, there are obvious scientific supports for special health benefits of soluble fibres as well as insoluble fibres. The nomenclature and definitions of NSP (non-starch polysaccharides, dietary fibre) have changed with time, and many of the available studies have used previous definitions, such as soluble and insoluble fibre (WHO 2003). The WHO report on Diet, Nutrition and Prevention of Chronic Diseases (2003) states that the best definition of dietary fibre remains to be established, given the potential health benefits of resistant starch while NSP is defined as "Dietary fibre is a heterogeneous mixture of polysaccharides and lignin that cannot be degraded by the endogenous enzymes of vertebrate animals."

Dietary fibre is defined by the American Association of Cereal Chemists (2001) as follows: "Dietary fiber is the edible parts of plants or analogous carbohydrates that are resistant to digestion and absorption in the human small intestine with complete or

partial fermentation in the large intestine. Dietary fiber includes polysaccharides, oligosaccharides, lignin, and associated plant substances. Dietary fibers promote beneficial physiological effects including laxation, and/or blood cholesterol attenuation, and/or blood glucose attenuation." In the Dietary Guidelines for Americans (2005) dietary fibre is defined as: "Nonstarch polysaccharides and lignin that are not digested by enzymes in the small intestine. Dietary fibre typically refers to nondigestable carbohydrates from plant foods." The IoM (2005) defines dietary fibre as follows: "total fiber is the sum of dietary fiber and functional fiber. Dietary fiber consists of nondigestible carbohydrates and lignin that are intrinsic and intact in plants. Functional fiber consists of isolated, nondigestible carbohydrates that have beneficial physiological effects in humans."

According to Codex alimentarius commission/European Commission (2007): "Dietary fibre means carbohydrate polymers with a degree of polymerisation not lower than three, which are neither digested nor absorbed in the small intestine. A degree of polymerisation not lower than three is intended to exclude mono- and disaccharides. It is not intended to reflect the average degree of polymerisation of a mixture. Dietary fibre consists of one or more of: edible carbohydrate polymers naturally occurring in the food as consumed, carbohydrate polymers, which have been obtained from food raw material by physical, enzymatic or chemical means, synthetic carbohydrate polymers". According to a report by EFSA (European Food Safety Authority) (2010): "Non-starch polysaccharides (NSP) are the main constituents of dietary fibre and include a host of different polymers, highly variable in terms of molecular size and structure, as well as in monomeric composition. Main classes of non-starch polysaccharides are cellulose, hemicelluloses, pectins, and other hydrocolloids." According to Buttriss and Stokes (2008): "Currently, there is no single definition for dietary fibre that is accepted worldwide." This limits the ability to compare fibre intakes across the world.

2.1.2 Definition of sugars and fructose

Terminology for *sugars* differs across the world. The term *sugars* would be used for all monosaccharides (mainly glucose, fructose, and galactose) and the disaccharides (mainly sucrose, and lactose), including sucrose and added sugars (Lineback and Jones 2003, Cummings and Stephen 2007) and the term can refer also to *total sugars*. The term *sugars* can be viewed as an "umbrella term" (Ruxton et al. 1999). According to a recent report by EFSA (2010): "Sugar alcohols (polyols) such as sorbitol, xylitol, mannitol, and lactitol, are usually not included in the term sugars. However, they are partly metabolised and included in "carbohydrates" according to the European legislation". *Total sugars* is defined as the sum of naturally occurring sugar and added sugar in foods and beverages (Sigman-Grant and Morita 2003, Bray et al. 2004, Johnson et al. 2009). Naturally occurring sugars include all sugars occurring naturally in foods and not added in processing, preparation, or at the table.

There are various definitions for *sugar*. *Sugar* can be defined as any free monosaccharide or disaccharide present in a food (Bray et al. 2004) or s*ugar* refers

strictly to sucrose (table sugar) (Lineback and Jones 2003, Sigman-Grant and Morita 2003). Usually *sugar* refers only to sucrose. This has been questioned by Kandelman (1997), who considers that sugar should include not only sucrose but also the other mono- and disaccharides and invert sugar (mixture of glucose and fructose) which are found in many food products and can be fermented by oral micro-organisms to produce acid in the dental plaque.

The term *added sugars* is a commonly used term in the United States (U.S.). It is defined by the Dietary Guidelines for Americans (2005): "Sugars and syrups that are added to foods during processing or preparation. Added sugars do not include naturally occurring sugars such as those that occur in milk and fruits." In the United Kingdom (UK) the preference is for the national definition to distinguish sugars which are viewed as potentially cariogenic from those which are not (Ruxton et al. 1999). The terms *extrinsic and intrinsic sugars* had their origin in the UK Department of Health committee report in 1989. *Intrinsic (naturally occurring) sugars* refer to sugars that are an integral part of certain unprocessed foodstuffs enclosed in the cell, the most important being whole fruits and vegetables (containing mainly fructose, glucose, and sucrose). Intrinsic sugars are naturally occurring and accompanied by other nutrients (Cummings and Stephen 2007, Johnson et al. 2009). *Extrinsic sugars* are not located within the cellular structure of food, and are mainly found in fruit juice and added to processed foods. Lactose in milk is extrinsic in that it is not found within the cellular structure of food. The definition of the term *non-milk extrinsic sugars* (NMES) includes all extrinsic sugars, which are not from milk, that is excluding lactose, nor contained within plant cell walls (Gibson 1997, Cummings and Stephen 2007). It includes sugars in fruit juices, honey, and table sugar, sugars and syrups used in processing. The reason behind these definitions is that NMES are cariogenic, while intrinsic and milk sugars have been thought to have negligible effects on teeth. In an Australian study Baghurst and co-workers (1989) defined *discretionary sugars* to cover all *refined sugars* that are added at the table (almost exclusively sucrose), and *added refined sugars* were defined as sugars added to food and drinks either in commercial or domestic food preparation. In a study of Australian children's sugar intake (Somerset 2003) the term *refined sugar* referred to a combination of the terms discretionary sugars and added refined sugars during manufacture.

In the World Health Organization report *free sugars* is defined as all monosaccharides and disaccharides added to foods by manufacturer, cook or consumer, and sugars which are naturally present in honey, syrups, and fruit juices (WHO 2003). Whereas according to the same report the term *fermentable carbohydrate* refers to free sugars, glucose polymers, oligosaccharides, and highly refined starches; it excludes non-starch polysaccharides and raw starches (WHO 2003). Kandelman (1997) has also defined *fermentable carbohydrates* as any type of sugars or cooked starches that are digested by the oral bacteria to produce acids and constitute the most important substrate for oral microbial metabolism. According to Englyst et al. (2007) the terms *added sugars, non-milk extrinsic sugars,* and *free sugars* are essentially synonymous with each other.

Fructose is a naturally occurring sugar (monosaccharide) in fruits and vegetables, or added during food processing mainly in food industry as high-fructose corn syrup or

fructose-glucose syrup, for example to beverages and bakery products. Because of sweetness and low costs, high-corn-fructose syrups have commercial application (Bantle 2009). In the mid-1980s, high-fructose corn syrup was adopted by the carbonated-beverage industry and became the predominant sweetener in soft drinks in the U.S. (Bantle 2009). One-third of fructose came from fruits, vegetables, and other natural sources, and two-thirds was added to beverages and foods in the U.S. (Park and Yetley 1993).

2.2 Dietary sources of carbohydrates in children

2.2.1 Sources of total carbohydrate

In the European Prospective Investigation into Cancer and Nutrition (EPIC) adult population sources of carbohydrate were bread, fruit, milk and milk products, sweet buns, cakes and pies, potato, sugar and jam, pasta and rice, vegetables and legumes, crispbread, and fruit and vegetable juices (Wirfält et al. 2002). In Finnish and Swedish children carbohydrate sources were similar as in adults in Europe, i.e. bread and other cereal products, milk products, fruits, sweets and beverages (Garemo et al. 2007b, Kyttälä et al. 2008) whereas in Spain the main carbohydrate sources in children were French fried potatoes, white bread, and biscuits (Royo-Bordonada et al. 2003), and in the U.S. bread, soft drinks, and milk (Subar et al. 1998) (Table 1).

2.2.2 Sources of dietary fibre

Most of the fibre in the diet of young children is obtained from cereals, legumes, and vegetables, some from fruits and fruit juices according to a commentary by the European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) Committee on nutrition (Aggett et al. 2003). In addition, potatoes, bread, breakfast cereals (Saldanha 1995, Gregory and Lowe 2000) are remarkable sources of fibre. Other remarkable but unexpected sources include savoury snacks like potato crisps (Ruxton and Kirk 1996), and the milk group, due to chocolate flavoured milk (Nicklas et al. 1995a,b) since one mug can contain as much as 1.5 g of fibre. The sources of dietary fibre depend on foods are used frequently in the country being studied (Table 1).

2.2.3 Sources of sugar and fructose

The three main food sources in caloric sweetener (all caloric carbohydrate sweeteners, except naturally occurring sugars) in the United States from 1977 to 1996 were soft drinks, desserts, sugar, and jellies (Popkin and Nielsen 2003). The order of added sugar has changed with time; from 1977 to 1978 the main source was desserts, whereas from 1994 to 1996 the main source was soft drinks for American children over 2 years of age and for adults. In children in the Nordic countries, beverages, such as juices and soft drinks, sweetened milk products, sweets and chocolate, and confectioneries are the main sources of sugar (Lyhne and Ovensen 1999, Øverby et al. 2004, Erkkola et al.

2009, Patterson et al. 2010) (Table 1). Other notable sources of sugar in children are desserts and fruits (Gibney et al. 1995, Kranz et al. 2005b). Repeated dietary surveys of English children over three decades indicate that sugar intake levels have remained stable, while the sources of sugars have changed, e.g. soft drinks consumption has multiplied since 1980 (Moynihan 2005).

Fructose sources in the United States were non-alcoholic beverages (54%) and cereal products (20%) across both gender and all age groups (Marriot et al. 2009). For children aged 1-3 years the predominant source of total fructose was fruits and fruit products (38%), and in the age range 4-6 years non-alcoholic beverages (34%) followed by fruits and fruit products (21%). Corresponding results were received from Switzerland, where fruits and fruit juices were the largest source of fructose (30%) at 2-5 years of age, while in older children aged 6-11 years sugar-sweetened beverages (31%) was the main source (Aeberli et al. 2007). In Swiss children aged 6-14 years, 50% of fructose intake came from fruit and vegetables and 32% from sweets and drinks. However, only few studies are available on fructose sources in children.

Table 1. Comparison of main sources of total carbohydrate, fibre, or sugar in ≤ 10-year-old children using a Western-type diet, in nutrition studies with N ≥100.

Author, year, possible data source, country	Method	Age	Gender	N	Total carbohydrate sources	Fibre sources	Sugar sources
Erkkola et al. 2009 Finland	3-day food record	1 y	M+F	455	Cereal products except bread (31%); milk products (16%); Fruits and berries (15%)	Cereal products except bread (41%); Fruits and berries (21%); Meat foods (15%)	
Kyttälä et al. 2008 KTL-National Public Health Institute and University of Tampere		3 y	M+F	471	Cereal products except bread (23%); milk products (21%); beverages (14%)	Cereal products except bread (29%); Breads (23%); Fruits and berries (17%)	Juice drinks; yoghurt and cultured milks; chocolate and confectionery (Added sucrose)
The Diabetes Prediction and Prevention study, DIPP Finland		6 y	M+F	713	Cereal products except bread (22%); milk products (21%); beverages (13%); bread (13%)	Breads (31%); Cereal products except bread (25%); Fruits and berries (15%)	
Garemo et al. 2007a,b Sweden	7-day food record	4 y	M+F	153	Bread; sweets; milk products; fruits	Bread; fruit and vegetables; potatoes	Sweets; milk products
Fox et al. 2006 U.S.	24-hour recall	6-11 mo	M+F	1395	Infant formula; infant grain; 100% juice	Baby food dinners; infant grain; bananas	
		12-24 mo	M+F	1003	Milk; 100% juice; sweetened beverages	Non-infant grains; bananas; bread/rolls/biscuits/bagels	
Royo-Bordonada et al. 2003 Spain	FFQ	6-7 y	M+F	1112	Chips (French fried potatoes); white bread; plain (Marie type) biscuits	Legumes; white bread; bananas	
Subar et al. 1998 U.S.	24-hour recall	2-18 y	M+F	4008	Bread; soft drinks; milk	Bread; ready-to-eat grain; potatoes	
Kranz et al. 2005a U.S.	2-day dietary intake data	2-5 y	M+F	5 437		Fruits; soy and legumes; ready to eat grains	
Smithers et al. 2000	7-day	4-18 y	M+F	1701		Vegetables; potatoes; savoury	Soft drinks; chocolate

Author, year, possible data source, country	Method	Age	Gender	N	Total carbohydrate sources	Fibre sources	Sugar sources
The National Diet and Nutrition Survey, UK	weighed food record					snacks; grains and grain products	confectionery; biscuits, buns, cakes and pastries
Gregory and Lowe 2000 UK		4-18 y				Grains and grain products 39%; vegetables 18%; potatoes 17%	
Hampl et al. 1998 U.S.	24-hour recall and 1-day food record	4-6 y	M+F	603		Breads 12%; vegetables 12%; fruit 10%; grains 9%; potatoes 8%	
Ruxton and Kirk 1996 UK	7-day weighed inventory technique	7-8 y	M+F	136		Potato crisps; bread; ready to eat breakfast grains; chips; fruits	Milk; diluting juices; fresh juices; chocolate; fizzy drinks (Total sugars)
Nicklas et al. 1995a,b Bogalusa Hearth Study America U.S.	24-hour dietary recall method	10 y	M+F	1254		Vegetables, soups; breads, grains; milk group (chocolate flavoured milk products); fruits and fruit juices	
Saldanha 1995 U.S.	24-hour recall and 2-day food record	2-5 y	M+F	567		Bread 15%; breakfast cereals 14%; fruits 13%	
Patterson et al. 2010 The European Youth Heart Study, Sweden	24-hour recall	10 y	M+F	551			Soft drinks; sweets, chocolate; other sweet foods
Kranz et al. 2006 U.S.	2-day dietary intake data	2-5 y	M+F	5 437			Fruits drinks; high-fat desserts; soft drinks

Author, year, possible data source, country	Method	Age	Gender	N	Total carbohydrate sources	Fibre sources	Sugar sources
Øverby et al. 2004 Norway	pre-coded 4-day food record	4-9 y	M+F	1201			Soft drinks; sugar & sweets; milk products
Lyhne and Ovensen 1999 Denmark	7-day food record	4-14 y	M+F	983			Sweets, cakes, table sugar; soft drinks; fruit juices
Farris et al. 1998 The Bogalusa Hearth Study, U.S.	24-hour recall	10 y	M+F	568			Milk; beverages; sweets
Linseisen et al. 1998 Germany	7-day food record	4-9 y	M	294-303			Confectionary and ice-cream; milk products; biscuits, cakes and pastries
		4-9 y	F	270-341			Milk products; confectionary and ice-cream; beverages and preserves
Gibney et al. 1995 Nationwide Food Consumption Survey, U.S.	food record	1-10 y	M+F	402-2788			Fruit and fruit juices; milk products; grain products with pastries
Nicklas et al. 1992 The Bogalusa Heart Study, U.S.	24-hour dietary recall	10 y	M+F	871			Sweets, beverages, desserts
Payne and Belton 1992 UK	7-day weighed food record	2-5 y	M+F	153		Wholemeal/brown bread, fruit, Weetabix/shreddies	Fruit-based uncarbonated soft drink and fruit drink concentrate; fruit juice; milk products

M, male
F, female
FFQ, Food frequency questionnaire

2.3 Carbohydrate intake in children

2.3.1 Total carbohydrate intake

Proportion of carbohydrate intake is approximately half of children's total energy intake. In the age range 1-9 years the mean total carbohydrate intake was 48-55 E% worldwide (Table 2).

2.3.2 Dietary fibre intake

Average fibre intakes for U.S. children and adults are less than half of the recommended levels (Park et al. 2005). Children's fibre intake at the age of 1-2 years is usually approximately 9 g/d (Räsänen et al. 1992, Alaimo et al. 1994, Kyttälä et al. 2008), at 3-5 years 11 g/d (Räsänen et al. 1992, Sepp et al. 2001), and at 9 years 15-17g/d (Räsänen et al. 1991, Øverby et al. 2004, Villa et al. 2007) (Table 2).

2.3.3 Sugar and fructose intake

There has been a large increase in the overall trends in the sugar consumption in the world, since the per capita increment of caloric sweetener in year 2000 was 74 kcal/d more compared with year 1962 (Popkin and Nielsen 2003). The researchers noted that the intake of caloric sweetener had doubled from earlier studies, and they saw a pronounced shift in the world's diet toward increased consumption of caloric sweetener and away from high-fibre foods. Sugar intake can be measured by different sugar definitions, which makes comparison between studies difficult. At 7- 8 years of age total sugar intake has been as high as 26 E% (Ruxton and Kirk 1996), and sucrose intake at 7-10 years approximately 14 E% in some studies (Linseisen et al. 1998, Lyhne and Ovensen 1999). In a study by Pattersson and colleagues (2010) sucrose intake of 10-year-old children was 11 E%, i.e. lower than in most other studies (Table 2). In children aged 3-6 years, sucrose intake in the Nordic countries has been approximately 12 E% (Räsänen et al. 1985, Garemo et al. 2007b, Sepp et al. 2001), while it was 14 E% in a German study (Linseisen et al. 1998). In the U.S., added sugar contributed to 17 E% already at 4 to 5 years of age (Kranz et al. 2005b). In a Finnish study sucrose intake was 12 E%, i.e. more than the recommended upper limit of 10 E %, in children aged only 1-2 years (Räsänen and Ylönen 1992).

There is very little information on children's fructose intake. However, according Vos et al. (2008): "measurement of fructose consumption is important because growing evidence suggest that it may play a role in health outcomes." Median fructose intake of 6-14 year-old Swiss children was 2 g/d (range 0.1-12.2) (Aeberli et al. 2007). In the United States, the mean fructose intake in the age of 2-5 years was 45 g/d and at 6-11 years 54 g/d (Vos et al. 2008). Another U.S. study found similar intakes, with the mean fructose intake being 34 g/d in children 1-3 years old, 43 g/d in the age range 4-6, and 51 g/d in children aged 7-10 years (Marriot et al. 2009).

Table 2. Comparison of mean daily intake of total carbohydrate, fibre or sucrose, and possible other carbohydrates such as starch or fructose in children 1-10 years old consuming Western-type diets.

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Patterson et al. 2010 The European Youth Heart Study, Sweden	24-hour recall	10 y	M+F	551	51 E%		Sucrose 10.6 E%
Buyken et al. 2008 The DONALD study Germany	3-day weighed food record	2 y	M+F	Total 380	49 E%	10 g/d	Added sugar 9.5 E%
		7 y	M+F		52 E%	16 g/d	Added sugar 14 E%
Erkkola et al. 2009 Finland	3-day food record	1 y	M	255	114 g, 55 E%	9 g, 2.3 g/MJ	Sucrose 12 g, 6 E%, starch 57g, 25 E%
			F	312	103 g, 55 E%	8 g, 2.3 g/MJ	Sucrose 11 g, 5 E%, starch 51 g, 24 E%
Kyttälä et al. 2008 KTL-National Public Health Institute and University of Tampere		3 y	M	236	167 g, 53 E%	10 g, 1.8 g/MJ	Sucrose 42 g, 13 E%, starch 74 g, 24 E%
			F	235	158 g, 53 E%	9 g, 1.8 g/MJ	Sucrose 40 g, 14 E%, starch 70 g, 24 E%
The Diabetes Prediction and Prevention study, DIPP Finland		4 y	M	307	180 g, 53 E%	10 g, 1.7 g/MJ	Sucrose 47 g, 14 E%, starch 80 g, 24 E%
			F	247	170 g, 53 E%	9 g, 1.7 g/MJ	Sucrose 44 g, 14 E%, starch 75 g, 23 E%
		6 y	M	364	206 g, 53 E%	11 g, 1.7 g/MJ	Sucrose 52 g, 13 E%, starch 95 g, 24 E%
			F	349	185 g, 53 E%	10 g, 1.7 g/MJ	Sucrose 49 g, 14 E%, starch 83 g, 24 E%
Garemo et al. 2007b Sweden	7-day food record	4 y	M+F	132	190 g, 55 E%	11 g	Sucrose 45 g, 12 E%

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Villa et al. 2007 Estonia and Sweden	24-hour recall	9 y	M+F				
			Estonia	444	53 E%	18 g/d	14 % of total energy came from sugar & sweets & drinks
			Sweden	441	52 E%	15 g/d	8 % of total energy came from sugar & sweets & drinks
Devaney et al. 2004 Feeding infants and toddlers study, U.S.	24-hour recall data	12-24 mo	M+F	998	165 g , 53 E%	8 g/d	
Øverby et al. 2004 Norway	4-day pre-coded food record	4 y	M+F	391	53 E%	12 g	Added sugar 55 g,15 E%
		9 y		810	54 E%	15 g	Added sugar 80 g,17 E%
Alexy et al. 2002b The DONALD Study 1985-2001 Germany	weighed dietary record	2-3 y	M+F	795	49 E%		Added sugar 11 E%
		4-8 y			50 E%		Added sugar 13 E%
Emmett et al. 2002 ALSPAC study England	3-day food record	18 mo	M	563	141 g		Total sugar 78 g, NMES 37 g, starch 62 g
		43 mo	M	488	181 g		Total sugar 92 g, NMES 59 g, starch 87 g
		18 mo	F	463	131 g		Total sugar 72 g, NMES 34g, starch 58 g
		43 mo	F	375	170 g		Total sugar 89 g, NMES 57 g, starch 80 g
		18 mo	M+F	1026	47 E%		NMES 12 E%, starch 21 E%
		43 mo	M+F	863	49 E%		NMES 16 E%, starch 23 E%
Sepp et al. 2001 Sweden	7-day food record	3-5 y	M+F	109	52 E%	11 g	Sucrose 12 E%

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Smithers et al. 2000 U.K.	7-day weighed dietary record	4-18 y	M	Total 1701	52 E%	11 g	NMES 17 E%
			F		51 E%	10 g	NMES 16 E%
Kersting et al. 1998 The DONALD study Germany	3-day weighed food record	1-3 y	M	Total 627	Md 124 g, 49 E%	Md 8g, 1.9 g/MJ, 0.7 g/kg	Added sugars Md 9 E%
		4-6 y	M		Md 170 g, 50 E%	Md 11.6, 1.9 g/MJ, 0.6g/kg	Added sugars Md 14 E%
		7-9 y	M		Md 204 g, 48 E%	Md 14g, 1.7 g/MJ, 0.5g/kg	Added sugars Md 14 E%
		1-3 y	F		Md 114 g, 49 E%	Md 8g, 2.0 g/MJ,0.7g/kg	Added sugars Md 9 E%
		4-6 y	F		Md 152 g, 49 E%	Md 10g, 1.9 g/MJ, 0.6g/kg	Added sugars Md 15 E%
		7-9 y	F		Md 193 g, 49 E%	Md 14g, 1.9 g/MJ, 0.5 g/kg	Added sugars Md 13 E%
Ruxton and Kirk 1996 U.K	7-day weighed inventory technique	7-8 y	M+F	136	50 E%	NSP 8 g	Sugars 26 E%
Ylönen et al. 1996 Finland	3-day food record	1-3 y	M+F	30	168 g, 49 E%	11 g, 8 g/1000kcal 13 g, 8 g/1000kcal	Sucrose 47 g, 14 E%, starch 60 g
		4-7 y		47	220 g, 51 E%		Sucrose 72 g, 17 E%, starch 76 g

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Payne and Belton 1992	7-day	2-5 y	M+F	153			
U.K.	weighed	2 y	M		150 g, 52 E%	9.7 g	Total sugars 85g, 30 E%, starch 60 g
	food record	2 y	F		145 g, 52 E%	8.3 g	Total sugars 88g, 31 E%, starch 53 g
		3 y	M		167 g, 53 E%	11.2 g	Total sugars 91g, 29 E%, starch 72 g
		3 y	F		157 g, 52 E%	9.9 g	Total sugars 90g, 30 E%, starch 63 g
		4 y	M		179 g, 53 E%	11.2 g	Total sugars 98g, 29 E%, starch 77 g
		4 y	F		162 g, 51 E%	10.3 g	Total sugars 91g, 29 E%, starch 67 g
Räsänen and Ylönen 1992 Finland	3-day food record	1-2 y	M+F	46	155 g, 51 E%	10 g	Sucrose 34 g, 12 E%, starch 56 g
			M	23	167 g, 54 E%	11 g	Sucrose 37 g, 12 E%, starch 62 g
			F	23	142 g, 52 E%	9 g	Sucrose 32 g, 12 E%, starch 50 g
Räsänen et al. 1991 The Young Finns study Finland	48-hour recall	9 y	M	119	243 g, 48 E%	18 g	Sucrose 67 g, 14 E%, starch 98 g
			F	109	228 g, 49 E%	16 g	Sucrose 60 g, 12 E%, starch 85 g
Räsänen et al.1985 Finland	48-hour recall	3 y	M	153	195 g		Sucrose 45 g, starch 74 g
		3 y	F	128	176 g 49 E% for 3 years old boys and girls		Sucrose 39 g, starch 64 g Sucrose 12 E% for 3 years old boys and girls
		6 y	M	139	246 g		Sucrose 54 g, starch 103 g
		6 y	F	145	209 g 50 E% for 6 years old boys and girls		Sucrose 45 g, starch 84 g Sucrose 11 E% for 6 years old boys and girls
		9 y	M	162	267 g		Sucrose 53 g, starch 121 g
		9 y	F	154	228 g 48 E% for 9 years old boys and girls		Sucrose 47 g, starch 98 g Sucrose 10 E% for 9 years old boys and girls

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Williams and Strobino The Healthy Start project 2008 U.S.	24-hour recall	3-4 y	M	Total 33		10 g	Sucrose 42 g, 11 E%
		7-10 y	M			12 g	Sucrose 46 g,11 E%
		3-4 y	F			11 g	Sucrose 32 g, 9 E%
		7-10 y	F			11 g	Sucrose 39 g, 10 E%
Kranz et al. 2005a,b U.S.	2-day dietary intake data	2-3 y	M+F	Total 5 437		10 g 7/100kcal	Added sugars 15%
		4-5 y				12 g 7/1000 kcal	Added sugars 17%
Gregory and Lowe 2000 U.K.		4-18 y	M+F			10.5 g (boys 11.2, girls 9.7)	
		4-6 y	M+F			9.1 g	
Nicklas et al. 1995a,b The Bogalusa Hearth Study, U.S.	24-hour recall	10 y	M+F	1254		12 g, 6g/1000 kcal	
Saldanha 1995 U.S.	24-hour recall and 2-day food record	2-5 y	M+F	2894		8.5 g	
		6-11 y	M+F	4692		11.8 g	
Alaimo et al. 1994 NHANES III U.S.	24-hour recall	1-2 y	M+F	1231		8.5 g	
		3-5 y		1547		10.7 g	
		6-11 y		1745		12.5 g	

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Alexy et al. 2006 The DONALD study Germany	3-day dietary record	1 y	M+F	530		10g, 3.1 g/MJ, 13g/1000kcal, 1.1 g/kg	
		1.5-3 y	M+F	653		10g, 2.6g/MJ, 11g/1000kcal, 0.8 g/kg	
		4-8 y	M+F	631		15g, 2.5 g/MJ, 11g/1000kcal, 0.7 g/kg	
Somerset 2003 Australia	24-hour recall	3 y	F	86			Added refined sugar 10.6 E%
		5 y		102			Added refined sugar 12.7 E%
		7 y		81			Added refined sugar 10.5 E%
		9 y		81			Added refined sugar 8.2 E%
		3 y	M	114			Added refined sugar 7.6 E%
		5 y		94			Added refined sugar 10.4 E%
		7 y		97			Added refined sugar 10.0 E%
		9 y		106			Added refined sugar 10.6 E%
Lyhne and Ovensen 1999 Denmark	7-day food record	4-6 y	M	136			Added sugars 13 E%
		7-10 y		185			Added sugars 14 E%
		4-6 y	F	137			Added sugars 14 E%
		7-10 y		191			Added sugars 14 E%
Farris et al. 1998 The Bogalusa Hearth Study, U.S.	24-hour recall	10 y	M+F	568			Sugar 15 E%

Author, year, data source, country	Method	Age	Gender	N	Total carbohydrate	Fibre	Sugar definition, amount of sugar, other carbohydrates
Linseisen et al. 1998 Germany	7-day food record	4-6 y	M	303			Sucrose 61 g, 14 E%
		7-9 y	M	294			Sucrose 67 g, 13 E%
		4-6 y	F	341			Sucrose 56 g, 14 E%
		7-9 y	F	270			Sucrose 65 g, 14 E%
Gibson 1997 U.K.	4-day weighed food record	1.5-4.5 y	M	848			NMES 18.9 E%
			F	827			NMES 18.6 E%
Gibney et al. 1995 Nationwide Food Consumption Survey U.S.	food record	1-10 y	M+F	402-2788			Total sugars minus lactose 75g, 20 E% Fructose 16 g, 4 E%

M, male
F, female
Md, Median
E%, Percentage of total daily energy intake
DONALD, the Dortmund Nutrition and Antropometric Longitudinally Designed Study
NMES, non-milk extrinsic sugars
ALSPAC, The Avon Longitudinal Study of Parents and Children
The Young Finns, The Cardiovascular Risk in Young Finns
NHANES, National Health and Nutrition Examination Survey
NSP, Non-starch polysaccharides

2.4 Dietary factors and cardiovascular diseases

The World Health Organization reports that CVDs are the major cause of death and disability in developed countries (WHO 2003). High concentrations of total and LDL cholesterol, low concentration of HDL cholesterol, other dyslipidemias, hypertension, and smoking are well-established risk factors for CVD (Hubert et al. 1983, Mensink and Katan 1992, Klag et al. 1993). Other possible risk factors for CVD are age, gender, obesity, central distribution of body fat, type 2 diabetes and metabolic syndrome, physical inactivity, and abnormalities in blood clotting factors (Ludwig et al. 1999). Main dietary factors that influence CVD risk include saturated fatty acids, trans fatty acids, cholesterol, unsaturated fatty acids, salt, and fibre. Higher dietary intake of saturated and trans fat are associated with increased risk of coronary heart disease, whereas a higher intake of monosaturated and polyunsaturated fats are associated with decreased risk of CHD (Hu et al. 1997). There is a positive association between total and saturated fat and serum total and LDL cholesterol and an inverse association between polyunsaturated fat and serum total and LDL cholesterol (Wu et al. 2007). These associations are evident also in children, as in the CATCH study increased total and saturated fat associated with increased total cholesterol (Nicklas et al. 2002). High intake of dietary fibre has been associated with lower risk of CVD, mainly because of the ability of fibre to reduce plasma cholesterol concentrations (Marshall et al. 1996, Pietinen et al. 1996, Aller et al. 2004). Excessive salt intake associates with predicted mortality and risk of coronary heart disease (Tuomilehto et al. 2001).

It has been universally documented that when carbohydrates are substituted for saturated fat, a reduction in total and LDL cholesterol is observed (Parks and Hellerstain 2000). An additional consequence of low fat diets (because of higher carbohydrate intake) is their propensity to increase fasting triglycerides and reduce HDL cholesterol (Griel et al. 2006).

2.4.1 Associations between carbohydrates and serum lipids

2.4.1.1 Total carbohydrate and serum lipids

The substitution of dietary carbohydrate for fat has been known to increase plasma triglycerides since the 1950s (Parks and Hellerstein 2000). Total carbohydrate intake associates inversely with total and HDL cholesterol and LDL cholesterol concentration in adults, whereas triglyceride concentration increases with increasing carbohydrate intake (Truswell 1994, Marshall et al. 1996, Ma et al. 2006). However, in the Framingham Offspring study in adults, higher total carbohydrate intake associated with higher triglyceride concentrations and lower HDL cholesterol concentrations but no association was found between total carbohydrate intakes and total or LDL cholesterol values (McKeown et al. 2009). In children, increased carbohydrate intake associated with decreased total (Cowin et al. 2001), HDL (Nicklas et al. 2002), and LDL cholesterol concentrations (Morrison et al. 1980) and increased triglycerides (Morrison et al. 1980, Perry et al. 1997).

However, despite carbohydrates' partially disadvantageous associations with serum lipids, carbohydrate restriction is not recommended because decreasing carbohydrate increases fat intake, and a low-carbohydrate diet tends to be a high fat diet (Truswell 1994). Total cholesterol is usually little affected by carbohydrates. If carbohydrates replace saturated fat, total cholesterol concentration may be reduced, or mildly elevated, since carbohydrate may produce an increase in VLDL concentration (Truswell 1994). In some circumstances, sucrose and fructose can increase fasting triglycerides more than starch or glucose is able to do (Truswell 1994).

2.4.1.2 Dietary fibre and serum lipids

Dietary fibre has a decreasing effect on total and LDL cholesterol concentrations (Jenkins et al. 1993a,b, Marshall et al. 1996, Wu et al. 2003, Aller et al. 2004, Wu et al. 2007), also in children (Williams 1995, Williams and Strobino 2008). Furthermore, dietary fibre intake has been inversely related with apoB levels (Jenkins et al. 1993a,b).

Soluble fibre, in particular, seems to be able to reduce serum total and LDL cholesterol concentration (IoM 2002). In the early 1960s Keys and co-workers (1961) reported a decrease in serum cholesterol when subjects were fed with pectin. Increased dietary fibre has also been shown to have therapeutic benefits in lowering cholesterol values in childhood (Anderson et al. 2009). It has been reported that adding approximately 6g of soluble fibre (in psyllium-enriched cereal) to children's diets could lower LDL cholesterol by 6% more than a low-saturated fat, low cholesterol diet alone (Williams et al. 1995b), and in adults also very high intakes of foods rich in soluble fibre decrease total and LDL cholesterol even in diets low in SFA and dietary cholesterol. Soluble or viscous fibres affect serum total and LDL cholesterol values by binding bile acids in the small intestine and increasing their excretion in faeces (Kirby 1981).

In adults no differences have been found between triglycerides and HDL cholesterol and dietary fibre (Aller et al. 2004). However, the total cholesterol/HDL cholesterol ratio has been lower with higher dietary fibre intake (Wu et al. 2003). Conversely, in some studies HDL cholesterol has been positively associated with fibre (Ludwig et al. 1999, Wu et al. 2003), while no associations were found in 3-year-old children between the intakes of fibre and total or HDL cholesterol (Cowin et al. 2001). Although there are several studies on the inverse association between dietary fibre and total and LDL cholesterol, there are also studies where no association has been detected, both in adults (Pietinen et al. 1996, Buil-Cosiales et al. 2009) and in children (Cowin et al. 2001).

2.4.1.3 Sugar and serum lipids

A number of studies link high sugar intake with adverse changes in lipoproteins (Archer et al. 1998, Howard and Wylie-Rosett 2000). Especially a diet high in sucrose associates with an elevation of plasma triglyceride concentration in adults (Parks and Hellerstein 2000), and also in children aged 6-19 years sucrose has been associated with increased plasma triglycerides (Morrison et al. 1980).

However, there is evidence which indicates that a high sucrose intake does not affect total or LDL cholesterol concentration. Conversely, it has also been reported that sugar

intake is inversely associated with total cholesterol in 3-year-old boys (Cowin et al. 2001) and decreased total, LDL and HDL cholesterol in the age range 6-19 (Morrison et al. 1980). HDL cholesterol is negatively associated with sugar in adults (Archer et al. 1998). Intake of sucrose has been inversely associated with HDL cholesterol levels in children aged 3-12 years (Kouvalainen et al. 1982, Cowin et al. 2001, Williams and Strobino 2008).

2.4.1.4 Fructose and serum lipids

In several studies fructose has been associated with increased triglyceride concentrations in adults (Hallfrish et al.1983, Reiser et al. 1989, Bantle et al. 2000). Few studies have addressed the fact that fructose increases triglycerides even more than sucrose does. In some studies fructose diets have also associated with increased LDL cholesterol (Hallfrish et al.1983, Reiser et al. 1989), although several studies have shown no adverse effects due to fructose on plasma lipids in adults (Bossetti et al. 1984, Crapo et al. 1984). In children no fructose-feeding studies have been published but there are studies of correlations between fructose intake and plasma lipids (Vos et al. 2008). In a study by Aeberli et al. (2007) in 74 Swiss children aged 6-14 years fructose intake was related to LDL particle size. High intake of fructose caused reduction in LDL particle size, a phenomenon regarded as atherogenic. However, there are only few studies on fructose intake and dyslipidemia in children.

2.4.2 Diet and serum lipids and apolipoprotein E polymorphism

Apolipoprotein E (apoE) gene is polymorphic, with three common alleles, designated apoE ∈2, apoE ∈3, and apoE ∈4. The apoE polymorphism may explain approximately 10% of the interindividual variance in serum total cholesterol concentration (Kamboh et al. 1993). In 13-month-old children apoE phenotype explained 5% of the cholesterol concentration variation (Lapinleimu et al. 1997). It is well known that this apolipoprotein polymorphism associates with serum cholesterol concentration already in childhood, with the carriers of the E2 allele having a lower total and LDL cholesterol concentration and in some studies, a higher HDL cholesterol concentration than those with E3 or E4 (Lehtimäki et al. 1994, Lapinleimu et al. 1997, Isasi et al. 2000). Triglyceride levels do not differ between the apoE phenotypes in children (Lehtimäki et al. 1994).

A positive relation has been found between dietary fat, SFA, and cholesterol and serum total and LDL cholesterol, dependent on apoE genotype, subjects with apoE4 expressing the strongest association (Lehtimäki et al. 1995, Marshall et al. 1996, Masson et al. 2003). However, there are also studies where associations have been in the same direction between the different apoE alleles and with no interactions between the diet and genotype group on blood lipids (Wu et al. 2007).

Although individuals with the E4 allele have the greatest lipid response to changes in dietary fat and cholesterol, they may not be the most responsive to changes in other aspects of the diet (Masson et al. 2003). Accordingly, individuals with the E2 phenotype have shown the greatest responsiveness to a dietary change involving

decreased serum cholesterol levels when dietary fibre intake increases with wheat- or oat-bran supplementation (Jenkins et al. 1993a). Similarly, in the study of Couture and co-workers (2003) individuals with the E2 allele had greater reduction in plasma LDL cholesterol after a high carbohydrate diet than E3 or E4 allele carriers. However, in another study associations between carbohydrate and fibre, and serum total, LDL and HDL cholesterol concentrations were similar in all apoE genotype subgroups (Marshall et al. 1996, Wu et al. 2007). In adults, high sucrose intake has been associated with a high triglyceride concentration only in those CHD patients with apoE2 (Erkkilä et al. 2001). After all, the apoE polymorphism might play only a minor role in modifying the relation between diet and serum lipid concentrations (Marshall et al. 1996).

2.5 Associations between carbohydrates and health

2.5.1 Total carbohydrates, diet quality, and growth

Carbohydrates are an integral part of human diet throughout the world. Mainly a diet with high intake of carbohydrates (except sugar) associates with the high nutritional quality of the diet. In the Framingham Offspring study on adults, total carbohydrate was associated with a lower intake of saturated and polyunsaturated fat, higher total energy, fibre and magnesium (McKeown et al. 2009). In the Bogalusa study on the nutritional quality of a high carbohydrate diet consumed by children, total energy, protein, fat and saturated fatty acid, cholesterol, and vitamins and minerals intakes decreased with increased carbohydrate intake (Nicklas et al. 1996). However, the intake of total sugar increased with increasing carbohydrate intake, for children with high carbohydrate intake (>55E%) received 78 g/d of (total) sugar. The findings from the Bogalusa sample for 10-year-old children (Nicklas et al. 1992) show the importance of the type of carbohydrates in relation to fat; children with a low fat intake had higher carbohydrate intakes than those with higher fat intakes, but the lower fat intake was also associated with a higher intake of sugar.

Total carbohydrates were inversely associated with body weight in the Coronary Artery Risk Development in Young Adults (CARDIA) study (Ludwig et al. 1999) and lower waist circumstance (McKeown et al. 2009). Among 3-year-old boys height has been negatively associated with total carbohydrate (as energy-adjusted) and starch intakes (Cowin et al. 2001).

The associations with health factors of carbohydrates depend on the type of carbohydrate. In case of total carbohydrate, it is important to know which carbohydrates are included, what is the proportion of sucrose, and how much fibre is included.

2.5.2 Dietary fibre, health, growth, and diet quality

High dietary fibre intake provides many health benefits. Possible health benefits of dietary fibre in reducing the risk of CVD were hypothesized already in the 1970s (Trowell 1972). Later it was confirmed that an inverse association exists between the intake of dietary fibre and risk of CHD (Pietinen 1996), with high-fibre-intake persons

having a 29% lower risk for CHD than those with lower fibre intakes (Anderson et al. 2009). This might be explained by the fact that dietary fibre has a decreasing effect on total and LDL cholesterol (Jenkins et al. 1993a,b, Marshall et al. 1996, Wu et al. 2003, Aller et al. 2004, Wu et al. 2007), in adults as well as in children. Besides blood cholesterol, increased fibre intake is also associated with other CHD risk factors, such as lower blood pressure in adults (Sandström et al. 1992, Ludwig et al. 1999, Anderson et al. 2009, Buil-Cosiales et al. 2009). In adults high fibre intake is inversely associated with carotid intima-media thickness (Wu et al. 2003, Buil-Cosiales et al. 2009).

Dietary fibre has been associated with lower waist circumference in adolescents (Liese et al. 2005, Ventura et al. 2008, Du et al. 2010). A positive association has been found between fibre and insulin sensitivity, and fibre has been inversely associated with insulin levels (Ludwig et al. 1999, Liese et al. 2005). A high-fibre diet is associated with improved measures for glucose tolerance and inflammation markers (Ludwig et al.1999, Ma et al. 2008). Fibre is also associated with lower plasma fibrinogen concentration (Luwig et al. 1999). Dietary fibre has been associated inversely with metabolic syndrome risk factors and may associate with lower risk of metabolic syndrome (Ludwig et al.1999, Liese et al. 2005, Ventura et al. 2008). Prospective epidemiological cohort studies have shown that high-fibre diets can reduce diabetes risk (Anderson et al. 2009).

A low-fibre diet is a risk factor for chronic childhood constipation (Morais et al. 1999, Anderson et al. 2009), and it is well known that inadequate intake of dietary fibre is also related to some other gastrointestinal disorders, including irritable bowel syndrome, diverticular disease, and colorectal cancer according to a commentary by the ESPGHAN Committee on nutrition (Aggett et al. 2003). Evidence shows that high intakes of dietary fibre reduce the risk for colorectal and small intestinal cancer (Bingham et al. 2003, Schatzkin et al. 2008) and can be protective against breast cancer (Suzuki et al. 2008). However, the available data are too sparse to draw any other conclusions of fibre's association with cancer risk.

Although dietary fibre has health benefits, there are concerns of possible adverse effects of excessive fibre intake. These might be too low energy intake, faecal energy loss, as well as bioavailability of minerals and nutrients, and poor growth (Williams 1995, Williams et al. 1995a). A diet with high fibre can reduce energy intake in small children because high-fibre foods tend to be bulky and have low energy density according to Williams (1995) and this may lead to poor growth.

Poor growth and nutritional status in children with exceptionally high intake of dietary fibre has been reported (Sanders 1988, Dagnelie et al. 1994). However, high intake of NSP had no adverse effects on growth within one year in 7-8 years old British children (Ruxton et al. 1995). Accordingly, this association between fibre intake and growth needs to be further investigated. Besides, in early childhood, the impact of dietary fibre intake on growth might also be important at the time of the pubertal growth spurt (Edwards and Parett 2003). There are only a few prospective studies on the association between dietary fibre and growth in children. In recent studies, fibre intake at the age of 2 years was not related to the percentage of body fat, and the changes in fibre intake

between the 2-7 years of age did not associate with BMI SD scores (Buyken et al. 2008) or weight changes in 9-14 years old children and adolescents (Berkey et al. 2000).

Epidemiological studies strongly indicate that dietary fibre intake protects against development of obesity in adults (Slavin 2005, Anderson et al. 2009). In the 10-year prospective CARDIA study on changes in cardiovascular disease risk factors in adolescents, Ludwig and co-workers (1999) showed that regardless of fat intake, adolescents eating the most fibre gained less weight over a 10-year period than those eating the least fibre. Fibre intake has been shown to be associated with a decreased risk of overweight in children and adolescents (Hanley et al. 2000, Anderson et al. 2009) and fibre to be associated with decreases in visceral adipose tissue in adolescents (Davis et al. 2009). Inverse association between fibre and BMI has also been found in several studies in adults (Ludwig et al. 1999, Liese et al. 2005, Slavin 2005, Buil-Cosiales et al. 2009, Du et al. 2010). However, there are studies in which this association has not been found (Pietinen et al. 1996, Wu et al. 2003).

There are several explanations why fibre intake facilitates weight control. Dietary fibre may be related to body-weight regulation through a plausible physiologic mechanism (Pereira and Ludwig 2001). Food with high fibre tends to be more satiating due to low energy density and dietary fibre slows down digestion (Pereira and Ludwig 2001, Slavin 2005, Anderson et al. 2009).

Diets that are low in fibre are often also low in some vitamins and minerals and are high in sugars, salt, and fats (Mann and Cummings 2009). A high-fibre diet is generally healthier than a low-fibre diet. Usually subjects with high fibre intake have lower intakes of total fat and saturated fat in adults (Wu et al. 2003), but fibre intake does not seem to differ between fat intake groups in children (Nicklas et al. 1992). A high-fibre diet tends to associate with higher intake of vitamins and minerals than a low-fibre diet in children. In 15-year-old adolescents high fibre intake has been associated with higher intakes of energy-adjusted vitamin A, B6, B12, and C, and niacin, thiamine, riboflavin, folacin, magnesium, iron, zinc, phosphorus and calcium (Nicklas et al. 2000), and in children high dietary fibre intake has been associated with increased energy and energy intakes adjusted for iron, folate, vitamin A, and vitamin C (Kranz et al. 2005a). Because of associations with vitamins and minerals it is obvious that high dietary fibre intake is also associated with more nutrient-dense food intake and increased intake of fruits and cereals (Kranz et al. 2005a).

2.5.3 Sugar and fructose, health, growth and diet quality

Sugar contributes to the energy density of the food consumed (Gibson 1997, Alexy et al. 2002a). A common finding is that added sugars intake (as E%) is positively associated with energy intake (Lewis et al. 1992, Alexy et al. 2002a). Humans have an innate preference for sweet foods (Anderson 1995). Hypotheses have been developed suggesting that sugar is unique among carbohydrates for its effect on appetite because its high hedonic properties seem to overrule the regulatory control. Sugars provide a

strong, sweet taste and at the same time they are an important source of energy (Anderson 1995).

The World Health Organization (2003) has identified evidence suggesting that high intake of sugars-sweetened soft drinks and fruit juices might promote weight gain and obesity. However, studies regarding effects of children's sugar intake on the development of obesity are inconclusive and limited (Hill and Prentice 1995). There are studies which show that high intake of sugar is inversely associated with occurrence of obesity (Hill and Prentice 1995) or absolute BMI (Villa et al. 2007). Studies have also shown that high sugar intake is associated with higher BMI in 4-year-old Norwegian boys (Øverby et al. 2003), and that 4-year-old Swedish children with higher sucrose intake (>15E%) have higher BMI than children with lower sucrose intake (<10E%) (Garemo et al. 2007b). Conversely, the Healthy Star Project in the U.S. showed that in the same age group (3-4-year-old children) sucrose intake was inversely associated with BMI (Williams and Strobino 2008). Furthermore, in a Norwegian study a negative association was observed between consumption of added sugar and BMI among 13-year-old girls (Øverby et al. 2004). However, the positive association between consumption of sugar-sweetened drinks and obesity development is well documented. The effect of increased consumption of sugar-sweetened beverages is associated with increased energy intake and obesity in children aged 12 years (Ludwig et al. 2001).

Children with high sucrose intake are at risk for nutrient dilution due to high intake of foods with low nutrient density, since as the dietary sugar intake rises, densities in the diet of several essential nutrients tend to decline (Lewis et al. 1992, Gibson 1997, Farris et al. 1998, Linseisen et al. 1998, Lyhne and Ovesen 1999, Alexy et al. 2003, Kranz et al. 2005b). Nevertheless, several studies show that a reasonable dietary quality is achievable within a wide range of sugar intakes and that the nutritional quality of high-sugar diets may still be adequate regarding the intake of most vitamins and minerals (Gibson 1997, Farris et al. 1998, Alexy et al. 2002a, Alexy et al. 2003, Kranz et al. 2005b). Studies regarding the effects of children's sugar intake on the displacement of other nutrients are inconclusive (Gibson 1997, Alexy et al. 2002a). Nevertheless, the question of nutrient dilution is important in children (Gibney et al. 1995). Association between sucrose and fat quality has been reported only in a few studies; in the Bogalusa Heart study intake of saturated fat (as g/1000 kcal and E%) decreased with increasing sugar intake (Farris et al. 1998). However, in a Finnish study no difference was seen in saturated + trans fatty acids intake between the quartiles of added sucrose intake (as E%), though children consumed more monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA) and *n*-3 fatty acids in the lowest sucrose intake quartile (Erkkola et al. 2008).

There has been a concern that counselling aiming at a low saturated-fat diet might lead to inappropriately increased sugar intake (Gibney et al. 1995, Ruxton and Kirk 1996), and some studies suggest that an inverse relationship exists between the intake of fat and sugar (Rugg-Gunn et al. 1991, Lewis et al. 1992, Gibson 1993, Hill and Prentice 1995, Gibson 1997, Kersting et al. 1998). Hence, there are differences of opinion about the age at which fat restriction can be safely implemented in early childhood (Gibson

1997). In the Bogalusa Heart Study 10-year-old children with a low fat intake (<30 E%) consumed 20% more total sugar than those with a high fat intake (> 40 E% fat) (Nicklas et al. 1992) and in a study of Gibson (1997) children with lower intake of NMES (as g/MJ) had higher intake of fat (as E%) compared with children on a high NMES intake.

Fructose may increase apoB concentrations and small, dense LDL and postprandial hypertriglyceridemia (Stanhope and Havel 2008b). Besides adverse effects on serum lipids, high intake of fructose can be a risk factor for insulin resistance, obesity, metabolic syndrome, type 2 diabetes, and CVD (Johnson et al. 2007, Stanhope and Havel 2008a,b). In addition, it has been observed that high intakes of fructose and sucrose are related to an elevated colorectal cancer risk (Michaud et al. 2005).

2.5.4 Sugar intake and dental health

The amount of dental decay is measured using the dmft/DMFT score, a count of the number of teeth or surfaces in a person's mouth that are decayed, missing or filled as a result of caries (in primary dentition with small letters and permanent dentition with capital letters) (WHO 2003, Moynihan and Petersen 2004). The DMFT-index has been used worldwide for decades, and it ensures good comparability of studies (Reich 2001).

From the 1970s to 1990s the dental health of children and adults has improved in most industrialised countries (Marthaler 1990, Burt and Szpunar 1994) without similar concomitant changes in sucrose consumption. The decrease in caries prevalence began in the 1960s and 1970s first in the Nordic countries and after that also in the U.K and the Netherlands (Reich 2001). For example, in Finland in 1975 the DMFT score was 7.5, but in 1982 it was 4.0, and in 1997 the DMFT had declined to 1.2 (WHO 2003), while sugar intake was 38 kg/person in 1975, 37 kg/person in 1982, and 34 kg/person in 1997 (Kettunen 1976, Leppälä 1992, MTH-Agricultural Economics Research Institute 2002). The basic concept of sugar consumption as a risk factor of dental caries has been seriously questioned, although a number of epidemiological and experimental studies have established the role of sugar in dental caries development for a long time.

Studies about the association between sugar intake and dental caries before the early 1990s have been summarized by Rugg-Gunn (1993): 9 out of 21 studies found associations between the amount of sugars consumed and caries increment, while the other 12 did not. Later Woodward and Walker (1994) analyzed data from 90 countries amongst 12-year-old children on dental caries and sugar consumption. In the studied 29 industrialised nations no evidence of a relationship between the DMFT score and sugar consumption were found. However, for the whole data, the DMFT score tends to rise with sugar intake, which denotes that in developing countries the association between dental caries and sugar is still distinct (Woodward and Walker 1994). Corresponding findings have been noticed in younger children. In a study on the association between dental caries and sugar intakes in children (1 to 5 years old) no differences were found in NMES intake in young children with or without caries experience (Marshall et al. 2007). However, beverage NMES intake at the age of 3

years predicted caries development in that study. In children who brushed their teeth at least twice a day the consumption of sugar or sugary foods was not associated with caries (Gibson and Williams 1999). Furthermore, children who ate sugary foods more than five times a day had a threefold higher risk of caries compared with the least frequent eaters (Gibson and Williams 1999). Similar results were gained in a Finnish study (Kleemola-Kujala and Räsänen 1982) that showed increased risk of caries with increasing sugar consumption to exist when oral hygiene was simultaneously poor. Gibson and Williams (1999) concluded that regular teeth brushing at least twice a day with a fluoride toothpaste may have greater impact on caries in young children than restricting sugary foods. Llena and Forner (2008) have stated that in the developed countries the clear association between sugar intake and increased caries levels no longer applies. This modification of the relation of sugars to dental caries can be probably interacted by fluorine and improved oral hygiene (Marthaler 1990, Woodward and Walker 1994, Ruxton et al. 1999). However, there are studies in toddlers and small children (Jones et al. 1996) providing support to the positive association between sucrose intake and caries even though the association has been weak in recent cross-sectional (Gibson and Williams 1999) and longitudinal studies of older children (Rugg-Gunn et al. 1984, Burt and Szpunar 1994).

The dental risk of dietary sugars is dependent on the frequency and amount of sugar intake, but the prevalence of caries is also modified by other dietary, social, genetic, and behavioural factors (König and Navia 1995). The general discovery is that parents of children with caries have lower levels of education and income (Marshall 2007). It is important to notice that patterns of dental health behaviour are established in early childhood (Jones et al. 1996).The most important of the other factors than sugar is the lack of regular tooth brushing. Tooth brushing removes the bacterial plaque that causes caries and makes fluoride (in toothpastes) available for teeth. According to authors König and Navia (1995), this explains the marked decrease in caries prevalence during the past 20 years in developed countries, although the consumption of sugars has remained high. However, dental caries remains a major public health problem in the EU and in other developed countries despite the marked declines in caries in the past 30 years (Sheiman 2001, Moynihan and Petersen 2004). Dental caries is common in 12-year-old children in countries with relatively low DMFT scores, since as much as 65% of children have dental caries in their permanent teeth (Sheiman 2001). The decrease in the prevalence of dental caries in industrialized countries may now be halted in younger age groups (Moynihan and Petersen 2004).

There has been a debate about whether the frequency or the amount of sugar is more important in causing caries (Sheiham 2001). However, there is conclusive evidence of a high correlation between the frequency and the amount of sugar intake, and by definition, when sugar intake rises, the frequency increases (Moynihan and Petersen 2004). Accordingly, most of the recent studies have found stronger associations between the frequency than the quantity of sugar intake and caries (Sheiham 2001). It is perceived that an increase in frequency of sugar intake of more than four per day increased the risk of caries (Sheiham 2001). Sheiman (2001) concluded that both the frequency and the total amount of sugar are important in the aetiology of caries.

However, it is hard to distinguish from each other the effect of sucrose frequency versus the total consumption of sucrose, and this makes evaluating sucrose effects on dental caries difficult (Lingström et al. 2003, Moynihan and Petersen 2004). According to the WHO report (2003) "when investigating the association between diet and the development of dental caries it is more appropriate to use a longitudinal study design in which sugars consumption habits over time are related to changes in dental caries incidence."

2.6 Recommendations of carbohydrate intake in children

2.6.1 Total carbohydrate intake recommendations

The Estimated Average Requirement (EAR) is defined as the average daily nutrient intake level estimated to meet the requirement of half of the healthy individuals in a particular life stage and gender group. According to the U.S. Food and Nutrition Board the EAR of carbohydrates is 100 g/d for children (IoM 2005), based mainly on data regarding glucose utilisation by the brain. The Recommended Dietary Allowance (RDA), on the other hand, is defined as the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all healthy individuals in a particular life stage and gender group. Dietary reference intake (DRI) for carbohydrate is set as 130g/day for adults and children, based on the average minimum amount of glucose used by the brain (IoM 2005) and the criterion for carbohydrate adequacy exceeds the minimum amount of glucose required by the brain without depending on fat or protein as an alternative energy source (IoM 2002). The acceptable Macronutrient Distribution Range for carbohydrate is limited to no less than 45% in order to prevent a high intake of fat, which has an upper range of 35% on energy (IoM 2005). The recommendation for carbohydrate in children from the age of 1 upward is 45-65 E%. According to the WHO (2003) the population goal for total carbohydrate intake should be 55-75 E%, after taking into account that consumed as protein and fat, hence the wide range. The joint report issued by FAO and WHO, Carbohydrates in human nutrition (1998) states that in an optimum diet at least 55% of total energy comes from a variety of carbohydrate sources for all ages except for children under the age of two.

The EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA) (EFSA 2010) proposes 45-60 E% as the reference intake range for carbohydrates for all ages except for children under the age of two. According to the NNR (Nordic Council of Ministers 2004), carbohydrates (including dietary fibre) should provide 50-60 E% for children older than 2 and for adolescents and adults, and the population goal is 55 E%, which should be used for planning purposes. Total carbohydrate recommendation for infants aged 6-11 months is 45-60 E% and for toddlers 12-23 months 50-55 E%. Exclusive breastfeeding is recommended for infants during the first 6 months and no recommendation for carbohydrate is given for that age.

The Finnish nutrition recommendations (National Nutrition Council 2005) are quite similar to the NNR. The NNR on total carbohydrate includes fibre, but in the Finnish carbohydrate recommendations total carbohydrate is mentioned without fibre, which

hardly produces problems since the energy content in dietary fibre within the amount used is only marginal. Total carbohydrate recommendation according to the Finnish nutrition recommendations is 50-60 E% (without fibre) for children older than 2 years of age and adolescents and adults, and the population-based recommendation is 55 E%. To toddlers aged 12-23 months the recommended total carbohydrate is 50-55 E% and to infants 6-11 months old 45-60 E%. As in the NNR, the Finnish nutrition recommendations report recommends exclusive breastfeeding for infants during the first 6 months and no recommendation for carbohydrate is given.

2.6.2 Dietary fibre intake recommendations

In the WHO report (2003) the term used for dietary fibre is NSP, which is used also in the U.K. According to the report the recommended intake of fruits and vegetables and consumption of whole grain foods is likely to provide >20 g/d of NSP (> 25 g/d of total dietary fibre) for adults, and this report contains no mention about children and fibre. Similarly, the FAO/WHO report Carbohydrate and human nutrition (1998) gives no specific recommendation for dietary fibre intake in children. The U.K. has no specific recommendation for children. According to the British Nutrition Foundation: "The recommended average daily intake for fibre is 18 g for adults although children need proportionally less. For preschool children, introduction of more fibre should be done gradually. Too much fibre can make a young child's diet so bulky that they become full before they have eaten sufficient food to satisfy their need for essential vitamins, minerals, and energy" (British Nutrition Foundation 2009). Previously Ruxton and colleagues (1995) concluded that the recommendation used for NSP in the U.K., i.e. 8g per 1000 kcal energy, is set too high. They also concluded that for further research on larger sample sizes of children is urgently required to establish the most appropriate fibre recommendations for children and to confirm the findings of the their study, which showed that high intake of NSP did not have any association with growth in 136 British children.

American Academy of Pediatrics' (AAP) Committee on nutrition (1981) declaimed that increased fibre intake in children had two arguments to be considered: the first was that children have small stomach capacity and the caloric density of high-fibre foods is low. Therefore, children would be unable to ingest adequate calories (AAP 1981). The other was the fear that fibre may influence adversely the absorption of certain essential minerals such as calcium, iron, copper, magnesium, phosphorus, and zinc. Although it is believed that mineral deficiencies are unlikely to develop in children on typical Western diets, even with a reasonable increase in their dietary fibre intake, there is little direct evidence to support this argument. In the early 1990s the AAP Committee on Nutrition recommended the 0.5 g/kg of body weight for fibre for children older than 2 years (with an upper limit of 35g/day). Later the "Age plus 5" recommendation was released by the American Health Foundation (AHF), practically recommending that children older than 2 years of age should consume a minimal amount of dietary fibre age plus 5 g/day and a safe range of dietary fibre intake is suggested to be between age plus 5 and age plus 10g/day (Williams et al. 1995a).

The National Academy of Sciences in the United States released the new DRIs for fibre in 2005. The recommendation, based on various sources of evidence, stated that people of all ages should consume 14g of total fibre for every 1000 kcal total energy intake. Dietary reference intake (DRI) recommends 19 g/d as the dietary fibre intake for children aged 1-3 years, 25 g/d for 4-8 years old , and between 9 and 13 years of age separate recommendations for boys and girls (31 g/d and 26 g/d, respectively) (IoM 2005). DRI (2005) has a special note for fibre intake in infancy stating that there are no functional criteria for fibre status that reflect response to dietary intake in infants. Furthermore, since human milk is the optimal source of nourishment for infants as the sole nutritional source for infants during the first 4 to 6 months of life (IoM 1991), and because human milk contains no dietary fibre, adequate intake values for infants 0-6 months of age cannot be given. During the age range 7-12-months the intake of solid foods becomes more significant and dietary fibre intake increases. However, there is currently no theoretical reason to establish an adequate intake for infants 7-12 months of age, either. The estimation of adequate intake of dietary fibre is based on the fact that it aids in laxation and promotes satiety, which may help reduce energy intake and therefore risk of obesity, attenuate blood glucose and cholesterol concentrations, and thus reduce the risk of CHD.

The NDA Panel (EFSA 2010) reports that there is limited evidence to set adequate intakes in dietary fibre for children. However, a fibre intake of 2 g/MJ is considered to be adequate for normal laxation in children from the age of one year. According to the NNR (Nordic Council of Ministers 2004) for adults, the intake of dietary fibre should be 25-35 g/d, i.e. approximately 3g/MJ. The NNR also considers important that appropriate amounts of dietary fibre intake should be obtained from a variety of foods for children as well as adults. According to the NNR, by school age an otherwise balanced diet is likely to provide at least 10 g dietary fibre daily and the intake should then gradually increase to reach the recommended level during adolescence (Nordic Council of Ministers 2004). The Finnish nutrition recommendations report emphasizes that infants should not consume dietary fibre excessively, because an extremely high fibre intake decreases energy density and may increase the bulkiness of the diet (National Nutrition Council 2005). From pre-school age fibre content of diet can be increased step by step so that in adolescence fibre intake is similar to adults.

2.6.3 Sugar intake recommendations

In the WHO report (2003) the population goal for *free sugar*s is < 10 E%. The report suggests that sugar-sweetened beverages should be limited for children to reduce chronic diseases. WHO's report (2003) recommends that the frequency of consumption of foods or drinks containing free sugars should be limited to a maximum of four times per day, because several studies have indicated that caries experience increases markedly when frequency of sugars intake exceeds four times a day (Sheiham 2001). For caries prevention Sheiman (2001) concluded that the current dose-response relationship between caries and extrinsic sugar consumption and data from observational studies suggest that for preschool children sugar intake should be limited to about 30g/d and a maximum of four times per day.

The Dietary reference intakes report in the United States suggests limiting added sugars to no more than 25% of total energy (IoM 2005). Unlike natural sugars, such as lactose in milk and fructose in fruits, added sugars are incorporated into foods and beverages during production and processing. The suggested maximum level is based on trends that show that people whose diets are at this level of added sugars or above are more likely to have poorer intakes of important essential nutrients (IoM 2005).

According to the EFSA NDA Panel (EFSA 2010) the available data are not sufficient to set an upper limit for sugar intake. According to the NNR (Nordic Council of Ministers 2004) the intake of refined sugars should not exceed 10 E% in infants over 6 months and in children. Refined sugars include sucrose, fructose, glucose, starch hydrolysates (glucose syrup, high-fructose syrup) and other isolated sugar preparations such as food components used as such or added during food preparation and manufacturing. According the NNR, to ensure an adequate intake of essential nutrients and dietary fibre, especially in children and adults with low energy intake, a limitation for the intake of refined sugars is necessary. Like the NNR, also Finnish nutrition recommendations report (National Nutrition Council 2005) recommends that refined sugar intake should not exceed 10 E%, especially in children and in adults whose energy requirement is low. After all, there are wide differences in guidelines for sugar intake and many countries do not have recommendations to limit sugar intake (Ruxton et al. 1999).

3 AIMS OF THE STUDY

The purpose of the present study was to investigate children's carbohydrate intake and its associations with other nutrient intakes, serum lipids, growth, and dental health, and to provide background information for nutrition recommendations. The study was put into practice in a longitudinal CHD prevention project introducing a low-saturated-fat diet in early childhood. The detailed aims were:

1. To study the association between carbohydrate intake and serum lipids in children at the ages of 5, 7, and 9 years, and to evaluate the effect of apoE phenotype on this association.

2. To analyze children's dietary fibre intake from 13 months to 9 years and investigate its associations with the intake of energy and other nutrients, food consumption, and serum cholesterol concentrations, and to assess whether fibre intake is associated with growth or weight gain.

3. To analyze children's sucrose intake from 13 months to 9 years, and to investigate and compare the effect of sucrose intake on the intake of energy and other nutrients and food consumption, and to assess whether sucrose intake is associated with growth or weight gain.

4. To evaluate the effects of long-term high or low sucrose intake on dental health at the mean age of 10 years, and to study the effects of sucrose consumption frequency on dental health.

4 SUBJECTS AND METHODS

4.1 The Special Turku coronary Risk factor Intervention Project, STRIP

The STRIP project is a prospective, randomised, long-term trial designed to decrease the exposure of children to known environmental atherosclerosis risk factors with infancy-onset dietary and lifestyle intervention.

For the study, families were recruited from the well-baby clinics of the city of Turku, Finland between March 1990 and June 1992. A total of 1054 volunteer families with 1062 infants (8 twin-pairs) (56.5% of the eligible cohort) were enrolled to this project when the child was five months old. At the age of 7 months the children were randomised to form an intervention group (n=540) or a control group (n=522).

The intervention families received individualised counselling aimed at decreasing the child's intake of SFA and cholesterol and increasing the intake of MUFA and PUFA at an interval of 1-3 months until the infants were 2 years old and at 6-month intervals thereafter. The control families received only general dietary information as delivered at the Finnish well-baby clinics and by school health care.

At every visit to the Research Centre of Applied and Preventive Cardiovascular Medicine (CAPC) of the Turku University the families in the intervention group met a physician, a nutritionist, and a nurse. At each visit, a physician recorded the health history and examined the child including measurements of height and weight. Risk factors for atherosclerosis and the amount of everyday physical activity were discussed, and instructions were provided to encourage the families to change the child's habits towards a healthier lifestyle. The nutritionist had the main responsibility for child-targeted counselling of the intervention families, based on the food records of the child and dietary history of the family. A registered nurse introduced the prevention of the onset of smoking to children 9.5 years and older. The control families met the same team (a physician, a nutritionist, and a nurse) twice yearly from the beginning of the trial, and after the age of 7 years only once a year. The child's nutrition was only superficially discussed with the parents of the control children and they were given no detailed advice.

Non-fasting venous blood samples were taken for serum lipid measurement when the child was 7 and 13 months, and 2, 3, and 4 years old. At the age 5 years and thereafter, fasting blood samples were drawn every other year. All families (parents and other caregivers, e.g. the staff at the concerned day care centres and schools) kept a 3-day food record of the child's food intake at 8 and 13 months. From 2 years of age parents were advised to keep food records 4 consecutive days every 6 months until the age of 7 years (except at the age of 3.5 years). After 7 years intervention families kept food records twice a year and control families once a year.

During the STRIP trial, all study children continued their regular visits to the communal well-baby clinics and school health care for vaccinations, growth and development follow-up, and basic health education.

Some families have discontinued in the study over the years, mainly due to changing residence or lack of time. In intervention and control groups the loss of follow-up was similar. The results of a drop-out analysis until age 10 years showed that total cholesterol concentration and SFA intake were similar in the children who had withdrawn from the study and in those remaining in the study (Raitakari et al. 2005).

4.2 Study design and subjects of the present study

Study I

Association between carbohydrate intake and serum lipids in children, and the influence of apoE phenotype on this association were studied. For this study, all children who had fasting blood samples taken at the age of 5, 7, and 9 and who had simultaneous food records, were included (Figure 2a). The children who had not completed all three food records because of illness or lack of parental time, as well as the children from whom the blood draw was unsuccessful were excluded from the analyses. Children with familial hypercholesterolemia, type 1 diabetes or other disease which might affect serum lipid concentration were also excluded. Altogether, 644 children (337 boys and 307 girls) were included in the study.

Six hundred children had apoE determined and they were further divided into three categories: apoE2 (E2/E2 and E2/E3; girls 27, boys 33; n=60, at age 5 years), apoE3 (E3/E3; girls 149, boys 182; n=331), and apoE4 (E4/E3 and E4/E4; girls 102, boys 93; n=195). Children with phenotype E4/E2 (n=14) were excluded due to their small number, and because E4 and E2 alleles have partially opposing effects on lipid metabolism.

Pubertal status was estimated at age 9 years according to the Tanner staging (Tanner and Whitehouse, 1976). Of the girls, 33 (13%) had entered puberty [one girl at T3 and 32 girls at T2 (scale T1-T5)], whereas all the boys were prepubertal.

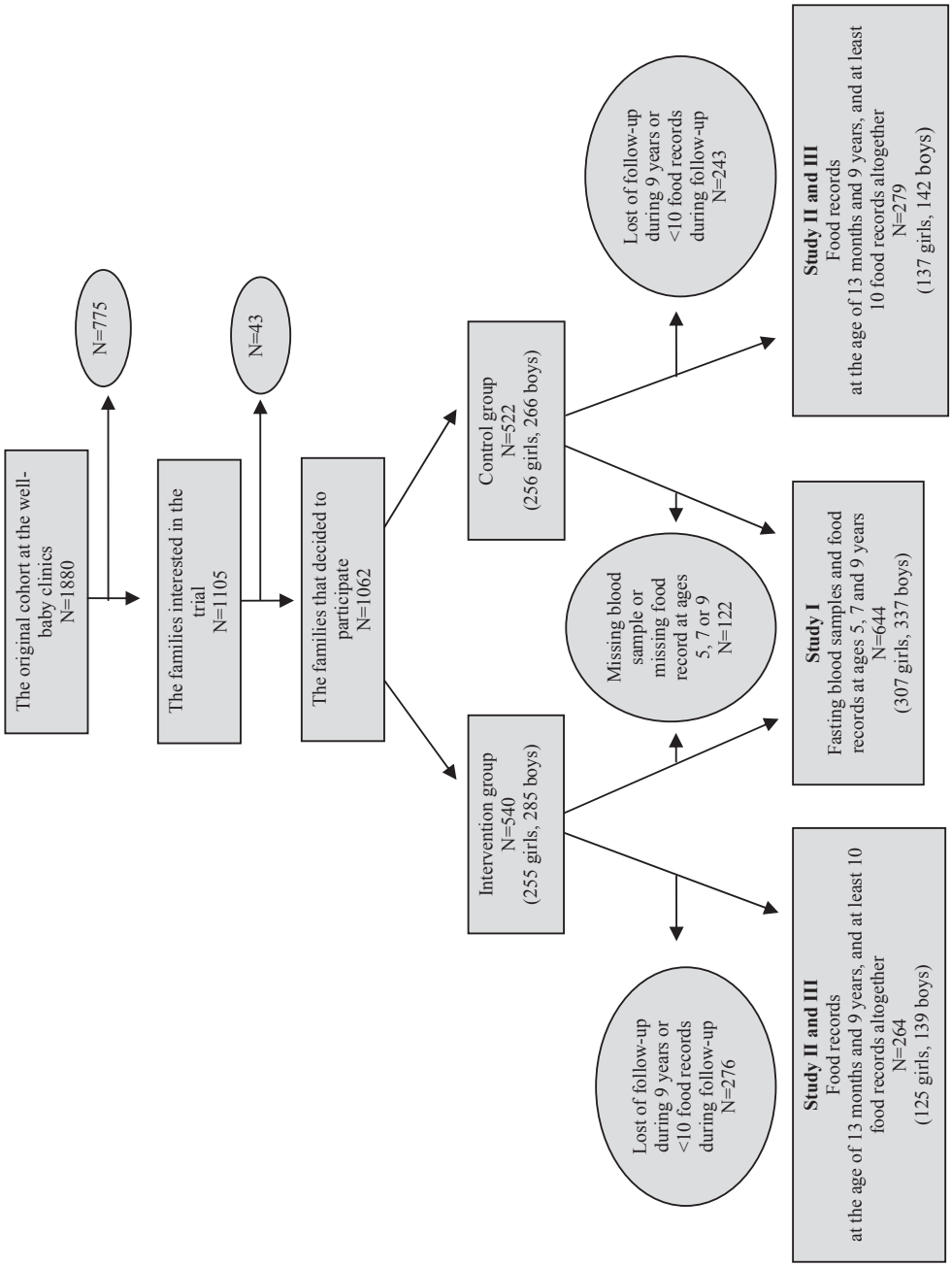


Figure 2a. Flow chart at the beginning of the STRIP study and subjects in studies I-III.

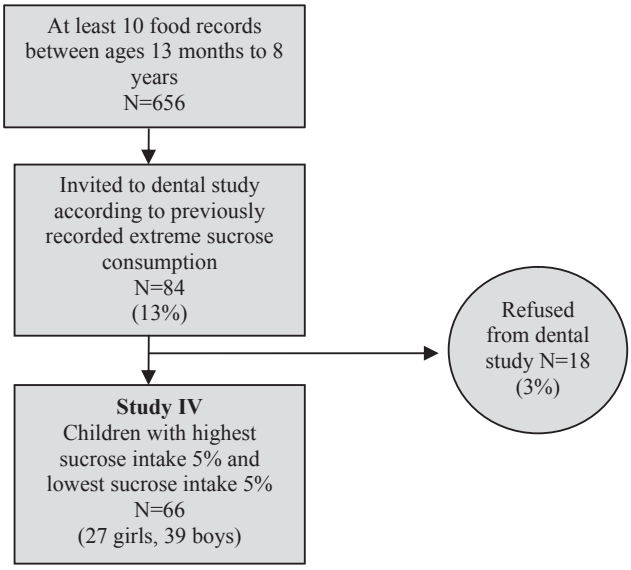


Figure 2b. Flow chart of the subjects in study IV.

Studies II and III

The study group for studies II and III consisted of the 543 children (281 boys and 262 girls) who had returned at least 10 of the requested 15 food records (mean number of returned food records = 13) between the ages of 13 months and 9 years (Figure 2a). Because of illness or lack of parental time, occasional food records were missing between ages 2 and 8 years.

The purpose of study II was to investigate the effects of dietary fibre intake on energy, the intake of other nutrients, food consumption, growth, and serum cholesterol concentrations. For study II, mean dietary fibre intake (as g/MJ) was calculated for every child using all of the available food records. The children were divided into three categories according to energy-adjusted fibre intake: low fibre intake, average fibre intake and high fibre intake children. The division of the children to the groups mentioned above was decided by examining the distribution of fibre intake graphically (Figure 3). The low dietary fibre intake group consisted of children with the lowest mean fibre intake (lowest 10^{th} percentile n=58; 13 intervention children), the high fibre intake group consisted of children with the highest mean fibre intake (highest 10^{th} percentile n=56; 37 intervention children), and the average fibre intake group consisted of the remaining 80% of children (n=429; 214 intervention children). The cut-off points for the low and high fibre intake groups were <1.57g/MJ and >2.36 g/MJ, respectively. The proportions of children in the intervention and control groups differed between the fibre intake groups (Cochran-Mantel-Haenszel method, P<0.001), therefore the STRIP intervention group was included in all further analyses. The number of boys and girls were similar between the fibre intake groups (Cochran-Mantel-Haenszel method, P=0.09). At the child's age of 8 months, food records were also collected (n=464). However, at that age dietary data on e.g. energy intake was incomplete because some of the children were still breast-fed. Thus, at 8 months

dietary fibre intake was expressed only as g/d but not g/MJ. Children were also divided into three weight groups at 9 years of age: thin, normal weight and overweight. Overweight was defined according to the age- and gender specific BMI cut-off points of the International Obesity Task Force BMI for children (Cole et al. 2000), corresponding to the adult BMI ≥25 kg/m^2. Children were classified as thin according to the international age- and gender-specific BMI cut-off points for children (Cole et al. 2007), corresponding to the adult BMI ≤18.5 kg/m^2.

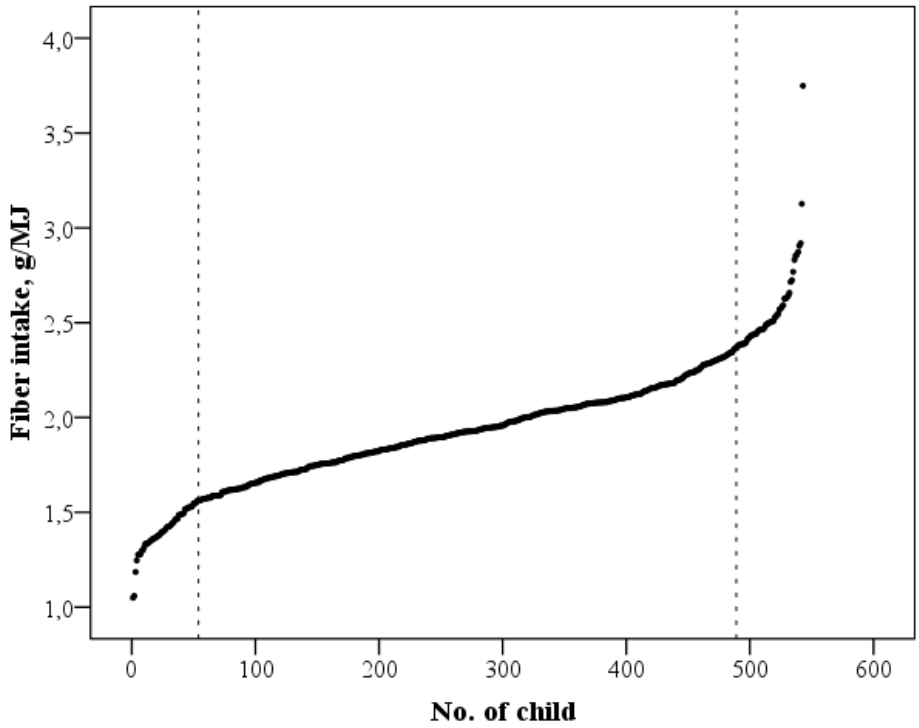


Figure 3. Children arranged according to their mean fibre intake as g/MJ from 13 months to 9 years. The vertical reference lines show the 10th and 90th percentile.

In study III the impact of children's sucrose intake from 13 months to 9 years on intake of essential nutrients, food consumption, and growth and weight gain were studied. For the study III, mean sucrose intake (as E%) was calculated for every child using all of the available food records. For this study, the children were divided into three different sucrose intake-groups (low, average or high). The division of the children to the mentioned groups was decided by examining the distribution of sucrose intake graphically (Figure 4). The low sucrose intake group consisted of children with the lowest mean sucrose intake (10th percentile n=54; 29 boys), the high sucrose intake group consisted of children with the highest mean sucrose intake (10th percentile n=54; boys 35), and the average sucrose-intake group consisted of the remaining 80% of children (n=435; boys 217). The cut-off points for the low and high sucrose intake groups were <6.77 E% and >11.22 E%, respectively. The proportions of intervention and control children and the number of boys and girls did not differ significantly between the sucrose

intake groups (Cochran-Mantel-Haenszel method, P>0.05) and therefore, the intervention and control groups and the genders were combined for the further analysis.

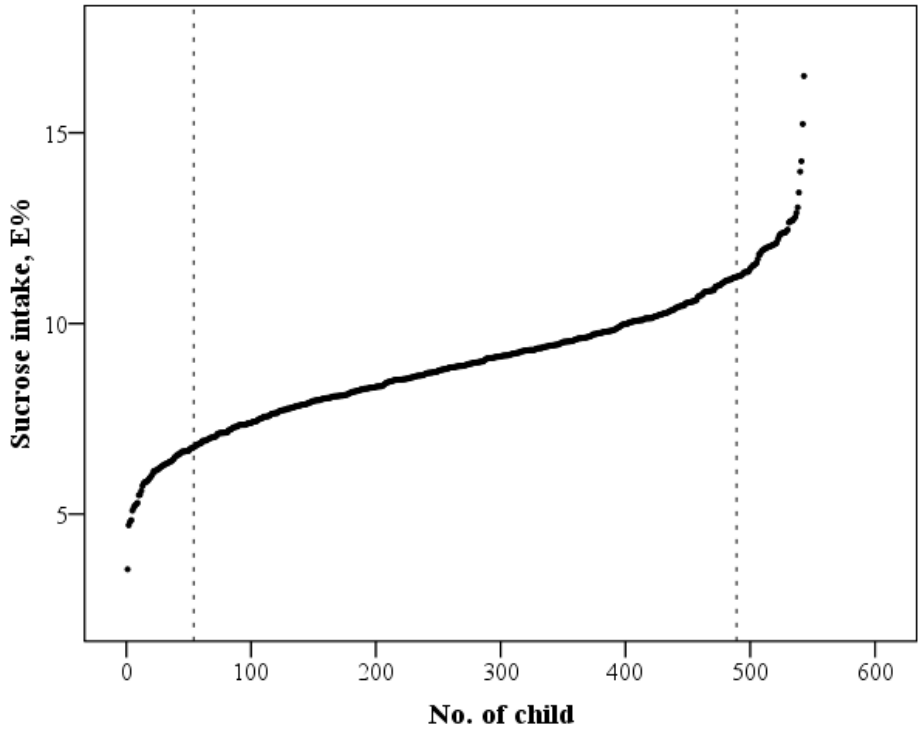


Figure 4. Children arranged according to their mean sucrose intake as percentage of energy from 13 months to 9 years of age. The vertical reference lines show the 10th and 90th percentile.

Study IV

The dental examination of the children was carried out at the mean age of 10 years (range 8 - 11). According to the previously recorded sucrose consumption with food records, 66 children with sucrose intake extremes were selected for this study from the participants of the main project (Figure 2b). The study comprised children with the highest and the lowest 5 percentiles of sucrose intake (n_{high} = 33; 18 boys; n_{low} = 33; 21 boys). To identify these children, each child's mean sucrose intake z-score (standard scores represent deviation around the mean in a distribution with a mean of zero and a standard deviation of 1) was calculated based on all food records available. In these calculations, sucrose intake was expressed as E%. By the age of eight, each child in this study had at least ten food records (of the possible 13) available for analysis. Altogether 84 children were invited to the dental examination, first 66 by letter and 18 more by phone due to refusals. Children with type 1 diabetes or other diseases which might affect sucrose intake were excluded from the study. The proportions of intervention and control children of the main study and the number of boys and girls did not differ significantly between extremely high or low sucrose intake groups.

The three most common reasons for refusing to participate in the dental health study was lack of time (n = 7), numerous other dental visits due to orthodontic treatment (n =

3), and too long a distance to the study location (n = 2). Six families did not name any specific reason. Those who refused (n = 18) did not differ by sucrose intake or by socioeconomic variables from those who participated in the study. The number of refusals was slightly higher in the high sucrose extreme group (n = 11) than in the low sucrose extreme group (n = 7).

4.3 Dietary intervention

The intervention families received individualised counselling aimed at decreasing the child's intake of SFA and cholesterol and increasing the intake of MUFA and PUFA at 1-3 months interval until the infants were 2 years old and at 6-month intervals thereafter. The intervention children were counselled to a fat intake of 30 - 35 E% for the child before 3 years of age and later the fat content of diet was targeted to 30 E%. The main aims of dietary counselling were similar for all intervention families, but the implementation was unique. The main point of the dietary counselling until the child's age of 2 was the type of dietary fat and after that age, attention was also paid to the quantity of fat in the child's diet. The counselling was based on the family's dietary habits and food record analyses.

Between the ages of 7 months and 7 years, counselling was given to parents but focused on the child's diet. However, from age 7.5 years onwards, progressively more information and suggestions were given directly to the child. The child was first met alone without parents, and after that the whole family was informed about the contents of the counselling. The control families received only general dietary information as delivered at the Finnish well-baby clinics and by school health care.

During each visit the parents were asked the type and amount of milk, the fat on bread and the cooking fat the child had used at the day-care centre or at school and at home. In addition, the parents were also asked to inform about possible use of dietary supplements.

4.3.1 Dietary counselling themes of the 7 months to 6-year-old children and their parents

During the first visit when the child was 7 months old, the aim of the dietary intervention was introduced to the families. At the second visit when the child was 8 months old, the families were advised on the differences between saturated and unsaturated fats and on how to use different kinds of vegetable margarine and vegetable oils and differences between margarines were discussed. At the age of 10 months the meal pattern of the child was assessed. Breast milk or infant formula was recommended until the age of 12 months. After that the families in the intervention group were advised to use skim milk and add two to three teaspoons (10 - 15g) of vegetable oil or margarine to the child's diet. At the 13-months visit the food record was used as the basis for dietary counselling and the differences between different types of fats and vegetable oil were discussed again. When the child was 15 months old, balanced and regular diets were discussed. At the age of 18 months, the meaning of dietary fat in children's diet was discussed and it was ensured that

vegetable oils or margarines were being added to the diet. At the age of 21 months, the importance of using different types of unsaturated fats as part of the diet was discussed again and the families were motivated. When the child was 2 years old, the role of sodium in health and the consumption of salt were discussed. When the child was 2.5 years old, a food frequency questionnaire (FFQ) concerning the 20 high-fat food group was conducted to facilitate later counselling, and the nutrient content of cold cuts and cheeses was discussed. At the age of 3 years, the dietary counselling was based on a FFQ and the intake of visible and invisible fats and amounts of fat were discussed. At the age of 3.5, consumption of foods high in SFA was discussed. When the child was 4 years old, the discussion and guidance covered sources of invisible fats. At the age of 4.5 years, regular eating times were discussed. When the child was 5 years old, families were advised on how to reduce the use of salt in food preparation and at the age of 5.5, the theme was revision of quality of fat. When the child was 6 years old, the families received feedback on their food records and the counselling was based on the food records and food choices of the family. At the age of 6.5 years the amount and quality of fat were discussed.

4.3.2 Dietary counselling themes of the 7 to 9-year-old children and their parents

When the child was 7 years old, the topic of the visit was children's food choices outside the home, and a nutrition knowledge test was performed for some of the children. At the age of 7.5, dietary counselling was given for the first time to the child directly, while the parents waited outside the room. After the child's own counselling, the whole family joined in and the child was asked to tell his/her parents and siblings about the counselling. Each time the parents were carefully informed about the tasks the child had performed during the session. Food records were also reviewed. At the age of 7.5 years the topic was to choose from tray foods (plastic food models) the ones that contain or do not contain fat. At the age of 8 years counselling was provided on the use of salt. The amount of salt in different foods was demonstrated using food packages and salt in test tubes. The child did a drawing task. After the child's own counselling the family's salt consumption habits were also discussed. At the age of 8.5 years, fat quality (animal fat and vegetable fat, heart healthy fat and less heart healthy fat) was discussed using pearls and food pictures for visualisation purposes. At the age of 9 years all intervention children performed the nutrition knowledge test including a picture identification test. The test covered the contents of the previous counselling, i.e. the amount and quality of fat and salt.

Between each visit two counselling letters were sent home to the children, one relating to the counselling visit content and the other on food preparation. A more detailed description about counselling has been published by Räsänen (2002).

4.4 Dietary assessment

4.4.1 Food records (I-IV)

In the STRIP project, all families (parents and other caregivers, including staff at the day-care centres and schools) kept a 3-day food record of the child's food intake at the

age of 8 months and 13 months (Table 3). Later, at the age of 2 years when the daily variation in the child's diet became larger, food records were kept for 4 consecutive days. After 7 years of age, the intervention children kept food records twice a year but the control children only once a year. Written instructions with drawings of food proportions were also distributed. The food records included at least one weekend day and were evenly distributed throughout the year in the intervention and control families. Parents were advised not to change their child's diet at the time of the food recordings. Food records were sent to the centre or brought in at the visit. The records were reviewed by a nutritionist for completeness and accuracy at each follow-up visit and, when appropriate, families were asked to provide further details on incomplete food items or amounts. Booklets with photos of portion sizes of food were used to help estimate portion sizes at the centre visit.

4.4.2 Analysis of nutrient intake and food consumption (I-IV)

Food records were analysed with the Micro Nutrica® program by the same experienced dietary technician. The program was developed at the Research and Development Centre of Social Insurance Institution (Turku, Finland). The program is based on the Food and Nutrient Data Base of Social Insurance Institution and it calculates 66 nutrients of commonly used foods and dishes in Finland (Hakala et al. 1996). From carbohydrates the Micro Nutrica calculates total carbohydrates, starch, sucrose, lactose, fructose, glucose, maltose and dietary fibre (water soluble, water insoluble fibre, cellulose, and lignin). Micro Nutrica uses frequently absorbed carbohydrate, which is a sum of starch, monosaccharides and disaccharides. In Micro Nutrica dietary fibre comprises the edible parts of the plant that cannot be digested or absorbed in the small intestine, and it is based on Finnish analyses (Varo et al. 1984a,b, Plaami 1996) and recent analyses based on data from food industry. In addition, complementary data are obtained from international food composition tables.

Besides carbohydrates, the study covered other energy nutrients, fat quality, and some vitamins and minerals. Food consumption was analyzed for the studies II and III. The data bank is flexible, permitting continuous updating of existing values and the addition of new single or composite foods and includes data on all of the foods commonly consumed by Finnish children. Vitamins and minerals consumed as supplements were not included in the calculations.

4.4.3 Sucrose intake frequency (IV)

Sucrose intake frequency was calculated from a FFQ designed specifically for the study. The form was first tested using a small group of STRIP study children, which were not in the dental health study. Some minor changes were made accordingly. The form included 52 sucrose-containing foods, and in addition to the amount of added sucrose, also the use of xylitol products was asked for. Sucrose containing foods and commonly used sucrose sources in children's diet were selected from food records. Sucrose containing foods were categorised into seven main groups as follows: "Sweetened milk products", "Sugared drinks and juices", "Sweets and chocolates",

"Pastries", "Breakfast cereals", "Desserts", and "Sugared tea or coffee". With the help of the parents, the children indicated the frequency of consumption – per day, week or month, as appropriate – of specific food items and beverages. Portion sizes were described in average servings. All sucrose frequency questionnaires were reviewed concurrently with the dental health examination visit.

4.5 Laboratory methods (I, II)

Non-fasting blood samples for total and HDL cholesterol and apolipoproteins (apo) A-I and B were drawn annually from 13 months to 4 years of age (Table 3) while fasting samples were drawn for all these analyses as well as triglycerides at the ages of 5, 7 and 9 years. Non-HDL cholesterol values were calculated annually for children at the ages 13 months to 9 years, except at the ages 6 and 8. Serum cholesterol concentration was determined by a fully enzymatic cholesterol oxidase-p-aminophenazone method (Merck, Darmstadt, Germany). The Friedewald (1972) formula was used to calculate LDL cholesterol concentration in fasting samples. Apo A-I and B were determined immunoturbidimetrically using ApoA-I and ApoB kits (Orion Diagnostica, Helsinki, Finland) (Riepponen et al. 1987). Serum HDL cholesterol concentration was analysed after precipitation of LDL and VLDL with dextran sulphate 500 000 (Kostner 1976). The interassay (intra-assay) coefficients of variation of total cholesterol and HDL cholesterol were 2.0% (1.5%), and 1.9% (1.2%), respectively. Serum triglyceride concentration was analysed using colorimetric GPO-PAP method (Merck) with an automatic Olympus AU 400 analyser. ApoE phenotypes were determined at the age of 13 months using isoelectric focusing and immunoblotting of delipidated serum (Lehtimäki et al. 1990). All analyses were performed in the laboratory of the Research and Development Unit, Social Insurance Institution (Turku, Finland). The laboratory frequently compared the results of lipid assays of standard samples with those of the WHO reference laboratory (Prague, Czech Republic).

Table 3. Summary of the measured data in study children

	7-8 months	**13 months**	**2 years**	**3 years**	**4 years**	**5 years**	**6 years**	**7 years**	**8 years**	**9 years**
Food records, 3-4 days	*	*	*	*	*	*	*	*	*	*
Growth, height and weight	*	*	*	*	*	*	*	*	*	*
Blood pressure		*	*	*	*	*	*	*	*	*
Serum lipids[1]	*	*	*	*	*	*		*		*
Apolioprotein E phenotype	*									
Dental health subcohort N= 66										*
Socioeconomic status										*

[1] Total and HDL cholesterol, calculated LDL-cholesterol, triglycerides, and apolipoprotein A-1 and B. Fasting samples at the ages of 5, 7 and 9 years.

* Studied/measured at the visit.

4.6 Measurements of height and weight (II, III), pubertal status (I), and blood pressure (II)

Weights of the children were measured (to the nearest 0.01 kg) until the age of 15 months using a baby scale (Seca 725, Hamburg, Germany) and thereafter (to the nearest 0.1 kg) with an electronic scale (S10; Soehnle, Murrhardt, Germany) (Table 3). Recumbent lengths of the children younger than 2 years were measured horizontally using a baby board (Bekvil, Paljerakenne, Helsinki). Thereafter, standing heights were measured to the nearest millimetre with a wall-mounted Harpenden stadiometer (Holtain, Crymych, Great Britain). Weights and heights were plotted on the Finnish growth charts (Sorva et al. 1984). Height, relative height (deviation of height in SD units from the mean height of healthy Finnish children of the same age and gender), weight, and relative weight (deviation of weight in percentages from the mean weight of healthy Finnish children of the same height and gender) (Sorva et al. 1984) were recorded. BMI was calculated as the weight in kilograms divided by the square of the height in metres (kg/m^2).

Tanner's five point rating scale (T1-T5) was used to define pubertal status (Tanner et al. 1976). Sitting blood pressure of children was measured annually from 13 months to 9 years of age using an oscillometric noninvasive blood pressure monitor (Criticon Dinamap 1846 SX) after an appropriate rest of 15 minutes. The accuracy of the device was regularly checked against a mercury sphygmomanometer. Proper cuff size according to the size of the child's right arm was used.

4.7 Socioeconomic status (IV)

The socioeconomic status of the family for study IV – number of children and adults in the family, parental education (basic education and education in the area of nutrition or health care), type of work and typical working hours and the annual income in the family - were collected from parents in the autumn of 1999, at the child's age of 9 years (range 8 to 10 years of age). The separate questionnaire was based on Statistics Finland standards (Statistics Finland 1999).

4.8 Dental examination (IV)

Plaque was scored from the maxillary incisors according to Silness and Löe (1964).
Visual assessment of dental health was carried out at the mean age of 10 years by one experienced paediatric dentist according to the WHO criteria (WHO 1997) as described (Karjalainen et al. 1997). Caries was recorded at the level of cavitation and expressed as dmft+/DMFT scores. Children with a sound, healthy dentition were classified as caries-free. A written summary of the examination was posted to the parents, and children in need of dental care were referred for treatment to the public dental health centre.

Dental radiographs and tooth-brushing habits (IV)

Two intra-oral bitewing radiographs were taken from all but one of the children. One specialised nurse carried out all radiographic procedures. The exposure conditions were 65 kV, 7.5 mA with an exposure time of 0.50 ms. Kodak – Ektaspeed Plus film was used throughout the study and the films were developed automatically. Two experienced specialists, one in cariology and another in paediatric dentistry, examined all radiographs independently in standardised conditions, in random order and blindly to grouping. The two sets of recordings were then compared and in cases of disagreement the radiographs were reviewed and discussed until an agreement was reached. The visual recordings were then completed by the radiographic information.

The children in the dental health study were interviewed for tooth-brushing habits, and for the use of dental floss and mouthwashes. The questionnaires were reviewed at the same visit as the examination of dental health was carried out.

4.9 Statistical analyses

Variables with a skewed distribution were log-transformed for the analyses. The results are expressed as means and standard deviations (SD), skewed distributions with geometric mean and 95% confidence intervals (CI). The level of statistical significance was set at $P< 0.05$. Statistical analyses were performed using the SAS system for Windows®, releases 9.1.3 (I-III), and 8.1 (IV) (SAS Institute, Cary, NC, USA) and the SPSS 11.0 (II), 9.0 (IV) package for Windows® (SPSS, Chicago, IL, USA).

4.9.1 Studies I-III, intakes of carbohydrate and its subgroups

Long-term associations of the data from 13 months to 9 years of total carbohydrate and its subgroups intake were analysed with repeated measures analysis of covariance (RM ANCOVA) models using the backward selection method (exclusion criteria: $P > 0.1$), including gender, STRIP study group and age. No interactions were included due to insufficient degrees of freedom. Comparisons for categorical variables were done with the Cochran-Mantel-Haenszel method.

To detect a possible selection bias, the children selected into studies I or II-III were compared to those who were out ruled due to insufficient data of the measures used as inclusion criteria. Comparisons were done with multivariable repeated measures analysis of variance RM ANOVA using all available data: at the ages 8 mo – 10 y for weight and height, and at the ages 13 mo - 9y (except the ages 6 and 8) for total, HDL and non-HDL cholesterol and 13 mo - 9y for the intakes of energy, fat, SFA, protein, sucrose, and dietary fibre. Gender and STRIP study group were analysed with Fisher's exact tests.

4.9.2 Study I, association between carbohydrate intake and serum lipids and apolipoproteins

All analyses of serum lipids were conducted with multivariable RM ANOVA. In the STRIP study group, gender and age were included in all analyses. The analyses of carbohydrate and lipid associations were also adjusted for BMI, SFA, MUFA, and PUFA. Non-significant covariates were excluded from the final analyses with backward elimination, using exclusion criterion $P > 0.1$. In longitudinal lipid analyses all interactions were included. The effect of each carbohydrate on serum lipids and lipoproteins were analysed with separate models.

In the analyses of the effect of apoE phenotype on the serum lipid concentrations, the interaction between apoE phenotype and gender was included in the model. In case of significant interactions, genders were analysed separately, and pairwise post-hoc comparisons for apoE phenotypes were performed with Tukey-Kramer adjusted T tests. The modifying effect of apoE phenotype on the association of carbohydrates and serum lipids was also analysed. In case of significant interactions of apoE and carbohydrate ($P<0.1$), the analyses were conducted separately among children with the different apoE phenotypes.

4.9.3 Study II, fibre intake and associations with intake of energy and other nutrients, and growth

RM ANCOVA of the data from 13 months to 9 years was used in fibre analyses between fibre intake and energy and in differences between fibre intake groups and longitudinal growth analyses. Differences between food intake groups and fibre intake groups were analysed with RM ANOVA, including the STRIP study group, age and the interaction of these as explanatory variables. The associations of weight gain or height gain in small children, assessed as differences between measured values at 8 months and 2 years, and preceding mean fibre intake (8 months – 2 years) were evaluated with regression models. Magnitudes of the associations are expressed as mean levels (categorical predictors) or regression coefficients (continuous predictors). All regression models were adjusted with gender and the STRIP study group, and RM ANCOVA models also with their interactions with age and main predictor. Non-significant predictors ($P>0.1$) were excluded from all analyses with backward selection. Pearson correlation was used for evaluating the associations between energy, fat, or weight and dietary fibre or sucrose intake in the scatter plots of 5-year-old children. Proportions of Cole weight groups within the three fibre intake groups were analysed with the Cochran-Mantel-Haenszel method.

4.9.4 Study III, sucrose intake and associations with energy, other nutrients and growth

Associations between sucrose intake and energy were analyzed with RM ANOVA, including energy nutrient × age interaction. Long-term associations of sucrose intake with nutrient intake, food group consumption, and growth were analysed with RM

ANCOVA models using a backward selection method (exclusion criteria: P>0.1), including sucrose-intake group, STRIP study group, age, and the interaction of these as explanatory variables. The proportions of children receiving less nutrients than recommended in the NNR (Nordic Council of Ministers 2004) were analysed with the Cochran-Mantel-Haenszel method.

4.9.5 Study IV, sucrose and dental health

Mann-Whitney U test and analysis of variance were used to study differences between the groups. Long-term effects were analysed with RM ANOVA. Spearman's correlation coefficients were calculated between sucrose consumption frequency and caries prevalence.

4.10 Ethics

The main STRIP study was approved by the Joint Commission on Ethics of the Turku University and the Turku University Central Hospital (13/9.11.1989). Informed consent was obtained from all parents.

The dental health study was approved by the Ethics Committee of Turku University and the Turku University Hospital (134/16.5.2000). Informed consent was obtained from all parents and children.

The protocol of the STRIP study is consistent with the principles of the Declaration of Helsinki and is registered at http://www.clinicaltrials.gov (identifier NCT00223600).

5 RESULTS

5.1 Carbohydrate and its subgroup intakes (I-III)

The mean carbohydrate consumption of the children between ages 13 months and 9 years was within the current NNR (Nordic Council of Ministers 2004), i.e. for children older than 2 years 50-60 E% and for children 12 to 23 months old 50-55 E% (Table 4). The mean sucrose intake of the intervention children was close to but did not exceed the current recommended maximum intake of 10 E% (Nordic Council of Ministers 2004 and WHO 2003) (Table 4), whereas in the control boys the mean sucrose intake from the age of 4 years onwards exceeded the current recommendation. Fructose and starch intakes as E% of intervention children were higher than in the control children (Table 4). There were no differences between genders in carbohydrate intakes as E% but carbohydrate and its subgroup intakes expressed as g/d were higher in boys than girls (Table 5). Sucrose intake as g/d was higher in the control children than in the intervention children (Table 5).

Dietary fibre intake in 8-month-old children was 3.9 g/d (SD 2.9) (n=247) in boys and 3.4 g/d (SD 2.6) (n=217) in girls. Dietary fibre intake varied widely (Table 6). Compared with the control group, the intervention children's mean fibre intake was higher in all measured units (as g/d, g/MJ, g/1000kcal, and as g/kg) at every age point. The absolute fibre intake (as g/d and g/kg) was higher in boys than in girls (Table 6).

When all food records (n=4706) between ages 13 months and 9 years were examined, the NNR carbohydrate recommendations were met in 61% of all food records. Sucrose intake was within the NNR and WHO nutrition recommendations (<10 E%) in 64% of all food records. The current dietary fibre recommendations were met very rarely, because less than 1% of all food records met the DRI fibre recommendation (that is, in only 1 % of all food records dietary fibre intake was ≥14g/1000 kcal, on which the DRI recommendation is based), while the AHF's recommendation "age+5 rule" was met by 47% of the food records (n=4163) of children between 2 and 9 years of age. The AAP's Committee on Nutrition recommendation (0.5 g/kg body weight) was met by 62% of the food records (n=4675) of children between the ages of 13 months and 9 years.

Sources of carbohydrate intake

The three main total carbohydrate intake sources in 13 months old children were ready-to-eat baby foods, cereal produts (mostly in the form of porridge and gruel) and milk products (Figure 5 a). In 5-year-old children cereal produts (mostly in the form of porridge and bread), milk products, and fruits and berries were the main carbohydrate intake sources (Figure 5 b). In older children sweets were also notable carbohydrate sources (Figure 5 c).

Dietary fibre intake sources varied between the different age groups: at the age of 13 months the main fibre sources were vegetables, cereal produts other than bread, ready-to-eat baby foods, and fruits and berries (Figure 6 a). In older children bread became

the major fibre intake source whereas a ready-to-eat baby food no more contributed to fibre intake (Figure 6 a-c).

At the age of 13 months the main sucrose sources were sweetened milk products, fruits and berries and juices, and desserts (Figure 7 a). After that age sucrose intake sources were quite similar in all age groups (Figure 7 b, c). Sweetened milk products were the main sucrose source starting from age 3 and other important sources were fruits and berries, juices, pastries and added sugar. In school-aged children soft drinks and sweets were also notable sucrose sources.

Table 4. Mean (SD) dietary carbohydrate and its subgroups intake as percentage of energy (E%) in intervention and control girls and boys from 13 months to 9 years of age.

Child's age Group	**N**	**N**	**Total carbohydrate E%**		**Starch E%**		**Sucrose E%**		**Fructose E%**	
	Girls	Boys	Girls	Boys	Boys	Girls	Girls	Boys	Girls	Boys
13 months										
intervention	125	139	55.7 (5.6)	57.0 (5.3)	21.3 (6.6)	19.7 (6.3)	5.6 (2.7)	5.3 (2.9)	4.0 (2.5)	4.3 (2.7)
control	137	142	55.6 (5.7)	55.1 (4.8)	19.8 (6.1)	19.7 (6.8)	5.2 (2.6)	5.4 (2.5)	4.3 (2.8)	4.0 (2.1)
2 years										
intervention	120	133	52.4 (5.5)	54.0 (4.6)	20.9 (4.7)	19.7 (4.2)	7.8 (2.6)	7.9 (3.1)	4.3 (2.2)	4.7 (2.4)
control	133	137	51.5 (5.1)	51.4 (5.4)	18.7 (4.7)	18.9 (4.7)	8.5 (3.2)	8.4 (3.4)	4.1 (1.8)	4.5 (2.3)
3 years										
intervention	117	134	53.1 (4.5)	53.6 (4.8)	21.3 (3.8)	20.1 (3.9)	9.5 (2.9)	9.2 (3.4)	4.5 (1.9)	4.4 (1.9)
control	126	139	51.2 (5.1)	51.9 (4.7)	19.6 (3.7)	20.3 (4.1)	9.3 (2.9)	9.7 (3.4)	4.1 (1.8)	4.4 (2.0)
4 years										
intervention	119	133	54.0 (4.4)	53.2 (4.9)	21.6 (4.1)	21.4 (3.9)	9.7 (3.0)	9.5 (3.1)	4.5 (1.9)	4.1 (1.9)
control	130	139	52.4 (4.7)	52.0 (4.7)	20.7 (3.9)	21.8 (4.0)	9.6 (3.2)	10.0 (3.3)	3.8 (1.6)	4.1 (1.8)
5 years										
intervention	120	132	53.8 (4.5)	53.6 (4.4)	22.1 (3.5)	21.9 (3.7)	9.8 (2.9)	9.6 (3.0)	4.1 (1.6)	4.1 (1.6)
control	132	137	51.6 (4.9)	52.2 (5.2)	21.1 (3.8)	21.1 (3.5)	9.6 (3.2)	10.3 (3.0)	3.7 (1.6)	3.8 (1.5)
6 years										
intervention	122	131	54.0 (4.3)	53.8 (4.4)	23.2 (3.3)	22.6 (3.5)	9.4 (2.8)	8.9 (2.9)	4.0 (1.7)	4.0 (1.8)
control	128	137	51.8 (4.7)	51.8 (4.3)	21.5 (3.6)	21.9 (3.7)	9.4 (2.7)	10.1 (3.3)	3.6 (1.5)	3.5 (1.3)
7 years										
intervention	117	125	53.4 (4.0)	52.6 (4.3)	22.7 (3.5)	23.0 (3.6)	9.2 (3.1)	8.8 (2.9)	3.8 (1.7)	3.7 (1.6)
control	127	132	52.4 (4.7)	52.6 (4.7)	22.1 (3.6)	22.6 (3.8)	9.6 (2.6)	10.0 (3.1)	3.6 (1.4)	3.8 (1.9)
8 years										
intervention	118	132	53.7 (4.9)	53.3 (4.5)	23.4 (3.9)	23.0 (3.8)	9.5 (2.6)	9.5 (3.5)	3.9 (1.8)	3.6 (1.4)
control	133	137	52.7 (5.3)	51.8 (4.6)	22.0 (3.6)	22.5 (4.1)	9.8 (3.2)	10.1 (3.6)	3.7 (1.7)	3.5 (1.6)
9 years										
intervention	125	139	54.0 (4.8)	53.5 (5.2)	24.0 (3.9)	23.5 (4.1)	9.2 (3.3)	9.4 (3.6)	4.0 (1.7)	3.7 (1.7)
control	137	142	51.8 (5.1)	52.0 (5.5)	22.5 (4.1)	22.3 (4.3)	9.9 (3.6)	10.0 (3.6)	3.4 (1.6)	3.3 (1.8)
P for intervention effect[1]			<0.001		<0.001		0.002		0.003	
P for gender effect[1]			0.82		0.22		0.38		0.78	
P for age effect[1]			<0.001		<0.001		<0.001		<0.001	

[1] Repeated measures analysis of covariance (RM ANCOVA), F-test.

Table 5. Mean (SD) dietary carbohydrate and its subgroups intake as g/d in intervention and control girls and boys from 13 months to 9 years of age.

Child's age Group	N	N	Total carbohydrate g/d		Starch g/d		Sucrose g/d		Fructose g/d	
	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys
13 months										
intervention	125	139	125.7 (24.7)	135.8 (26.6)	45.0 (17.0)	51.4 (19.3)	12.8 (7.5)	12.9 (7.8)	9.1 (5.7)	10.3 (6.4)
control	137	142	129.9 (24.8)	133.8 (23.8)	46.4 (18.2)	48.6 (17.5)	12.1 (6.5)	13.2 (7.0)	10.0 (6.7)	9.8 (5.1)
2 years										
intervention	120	133	140.3 (23.8)	149.8 (26.3)	53.0 (14.0)	58.4 (17.6)	20.7 (7.3)	22.0 (9.8)	11.6 (6.1)	12.8 6.1)
control	133	137	139.2 (24.5)	147.8 (30.0)	51.4 (16.7)	54.0 (16.3)	22.9 (8.9)	24.2 (10.3)	11.2 (5.0)	12.9 (6.4)
3 years										
intervention	117	134	149.7 (24.5)	161.4 (27.5)	56.8 (14.5)	64.2 (15.0)	26.7 (9.0)	27.9 (11.3)	12.6 (5.3)	13.2 (5.8)
control	126	139	152.2 (29.6)	161.1 (31.7)	60.2 (16.7)	61.1 (16.4)	27.6 (10.1)	30.3 (12.5)	12.2 (5.7)	13.6 (6.0)
4 years										
intervention	119	133	167.0 (31.5)	174.6 (30.4)	65.5 (15.1)	70.8 (17.9)	30.2 (11.9)	31.3 (11.6)	14.1 (6.8)	13.5 (6.1)
control	130	139	168.0 (30.3)	179.6 (34.4)	69.8 (17.9)	71.2 (19.1)	30.9 (11.5)	34.5 (12.9)	12.2 (5.4)	14.2 (6.4)
5 years										
intervention	120	132	180.0 (32.6)	190.3 (30.4)	72.7 (16.3)	78.1 (16.7)	33.0 (12.6)	33.8 (11.8)	13.7 (5.8)	14.6 (5.7)
control	132	137	176.9 (28.0)	192.4 (34.5)	72.0 (15.8)	77.6 (18.7)	32.8 (12.0)	37.9 (12.4)	12.6 (5.4)	14.0 (5.9)
6 years										
intervention	122	131	192.7 (33.7)	203.7 (31.3)	80.4 (18.5)	87.5 (18.1)	33.6 (11.9)	33.5 (11.6)	14.3 (6.8)	14.9 (6.7)
control	128	137	181.2 (33.5)	201.3 (34.0)	76.8 (18.7)	83.1 (18.9)	32.6 (10.6)	38.8 (13.0)	12.7 (5.5)	13.7 (5.3)
7 years										
intervention	117	125	195.7 (32.3)	210.4 (33.6)	83.8 (17.4)	90.6 (18.7)	34.1 (13.3)	35.0 (12.5)	14.2 (6.9)	15.0 (7.2)
control	127	132	196.4 (35.8)	215.9 (38.3)	84.1 (19.3)	90.7 (21.4)	35.7 (11.4)	40.9 (14.6)	13.6 (5.8)	15.5 (7.7)
8 years										
intervention	118	132	200.7 (34.2)	216.6 (35.3)	85.5 (18.3)	94.5 (20.1)	35.7 (12.4)	39.0 (17.2)	14.5 (7.5)	14.7 (6.2)
control	133	137	200.9 (34.2)	228.2 (40.2)	85.6 (20.5)	96.4 (22.1)	37.1 (13.3)	45.1 (18.6)	13.8 (6.2)	15.3 (7.6)
9 years										
intervention	125	139	207.0 (41.6)	228.2 (40.6)	89.3 (22.9)	100.9 (20.9)	35.3 (14.9)	40.6 (17.7)	15.2 (7.8)	15.8 (7.9)
control	137	142	204.5 (39.9)	232.8 (43.6)	87.4 (20.6)	100.2 (24.1)	39.6 (16.8)	44.6 (18.2)	13.3 (6.4)	14.5 (8.3)
P for intervention effect[1]			0.62		0.21		<0.001		0.10	
P for gender effect[1]			<0.001		<0.001		<0.001		0.0002	
P for age effect[1]			<0.001		<0.001		<0.001		<0.001	

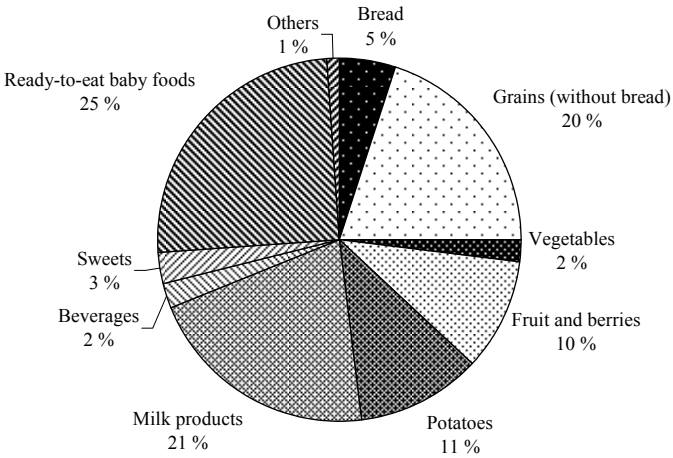
[1] Repeated measures analysis of covariance (RM ANCOVA), F-test.

Table 6. Mean (SD) dietary fibre intake in intervention and control girls and boys from 13 months to 9 years of age.

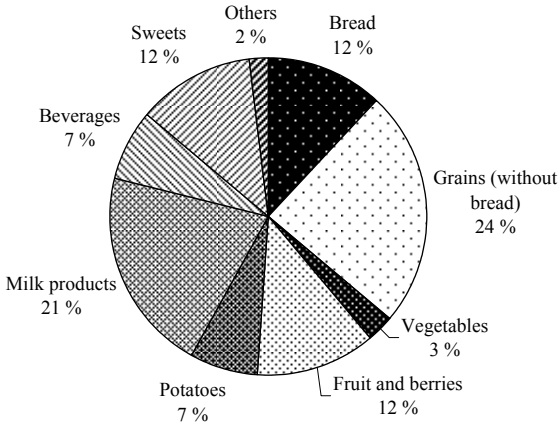
Child's age Group	N	N	Dietary fibre intake g/d		Dietary fibre intake g/MJ		Dietary fibre intake g/1000kcal		Dietary fibre intake g/kg body weight	
	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys
13 months										
intervention	125	139	6.8 (3.1)	7.7 (3.3)	1.7 (0.7)	1.9 (0.7)	7.3 (2.9)	7.9 (3.0)	0.67 (0.3)	0.73 (0.4)
control	137	142	7.0 (3.1)	7.3 (3.1)	1.7 (0.7)	1.7 (0.6)	7.3 (2.9)	7.3 (2.5)	0.71 (0.3)	0.69 (0.3)
2 years										
intervention	120	133	8.6 (2.8)	9.8 (3.2)	1.9 (0.5)	2.1(0.5)	7.8 (2.0)	8.6 (2.3)	0.69 (0.2)	0.76 (0.3)
control	133	137	8.2 (3.0)	8.6 (2.9)	1.8 (0.5)	1.8 (0.5)	7.4 (2.2)	7.3 (2.1)	0.65 (0.2)	0.66 (0.2)
3 years										
intervention	117	134	9.6 (3.0)	10.4 (2.6)	2.0 (0.5)	2.1 (0.5)	8.3 (2.2)	8.6 (1.9)	0.64 (0.2)	0.69 (0.2)
control	126	139	9.6 (2.8)	9.4 (2.9)	1.9 (0.5)	1.8 (0.5)	7.9 (1.9)	7.5 (1.9)	0.65 (0.2)	0.62 (0.2)
4 years										
intervention	119	133	10.7 (2.9)	11.1 (3.1)	2.1 (0.5)	2.0 (0.5)	8.6 (1.9)	8.3 (2.0)	0.62 (0.2)	0.64 (0.2)
control	130	139	10.6 (3.0)	11.0 (3.4)	2.0 (0.5)	1.9 (0.5)	8.2 (2.0)	7.8 (2.0)	0.62 (0.2)	0.64 (0.2)
5 years										
intervention	120	132	11.5 (2.9)	12.4 (3.1)	2.1 (0.5)	2.1 (0.5)	8.6 (2.0)	8.7 (2.0)	0.61 (0.2)	0.65 (0.2)
control	132	137	11.3 (3.3)	11.4 (3.2)	2.0 (0.6)	1.8 (0.5)	8.2 (2.3)	7.7 (1.9)	0.59 (0.2)	0.59 (0.2)
6 years										
intervention	122	131	12.8 (4.1)	13.4 (3.8)	2.1 (0.5)	2.1 (0.5)	8.7 (2.2)	8.7 (2.2)	0.58 (0.2)	0.61 (0.2)
control	128	137	11.9 (3.6)	12.4 (3.1)	2.0 (0.5)	1.9 (0.4)	8.4 (2.0)	7.9 (1.6)	0.54 (0.2)	0.57 (0.1)
7 years										
intervention	117	125	13.0 (3.4)	14.0 (3.7)	2.1 (0.5)	2.1 (0.4)	8.7 (1.9)	8.6 (1.8)	0.54 (0.2)	0.57 (0.2)
control	127	132	12.5 (3.8)	13.3 (3.7)	2.0 (0.5)	1.9 (0.4)	8.2 (2.1)	8.0 (1.8)	0.50 (0.2)	0.55 (0.2)
8 years										
intervention	118	132	12.9 (3.5)	13.5 (4.0)	2.0 (0.5)	2.0 (0.5)	8.5 (1.9)	8.2 (2.1)	0.47 (0.1)	0.50 (0.2)
control	133	137	12.2 (3.7)	13.1 (3.7)	1.9 (0.5	1.8 (0.4)	7.9 (2.2)	7.4 (1.8)	0.44 (0.1)	0.48 (0.1)
9 years										
intervention	125	139	13.3 (3.9)	14.5 (4.1)	2.0 (0.5)	2.1 (0.5)	8.6 (2.1)	8.5 (2.1)	0.43 (0.1)	0.48 (0.2)
control	137	142	12.5 (4.4)	13.3 (3.4)	1.9 (0.6)	1.8 (0.4)	7.9 (2.4)	7.4 (1.8)	0.41 (0.2)	0.44 (0.1)
P for intervention effect[1]			<0.001		<0.001		<0.001		<0.001	
P for gender effect[1]			<0.001		0.27		0.27		<0.001	
P for age effect[1]			<0.001		<0.001		<0.001		<0.001	

[1]Repeated measures analysis of covariance (RM ANCOVA), F-test.

a) 13 mo



b) 5 y



c) 9 y

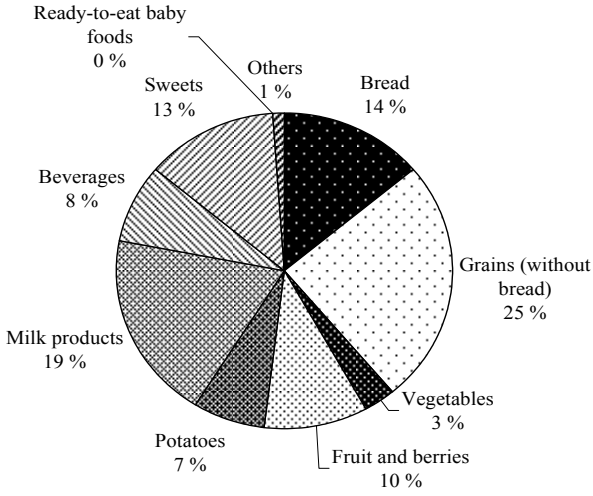
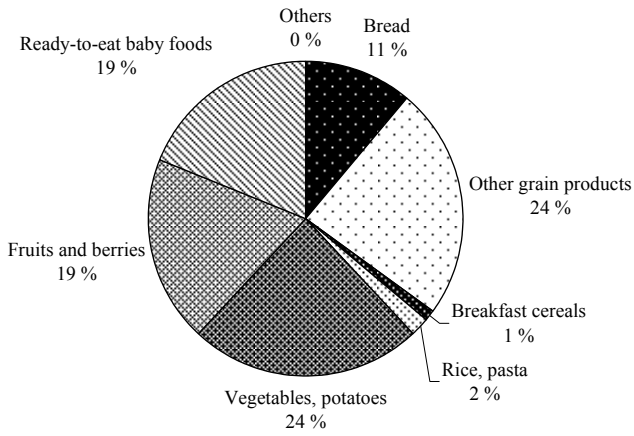
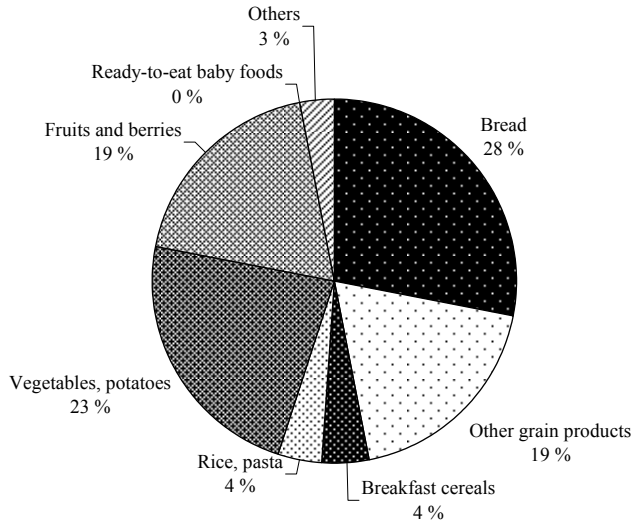


Figure 5. Carbohydrate intake sources at **a)** 13 months, **b)** 5 years and **c)** 9 years old children.

a) 13 mo



b) 5 y



c) 9 y

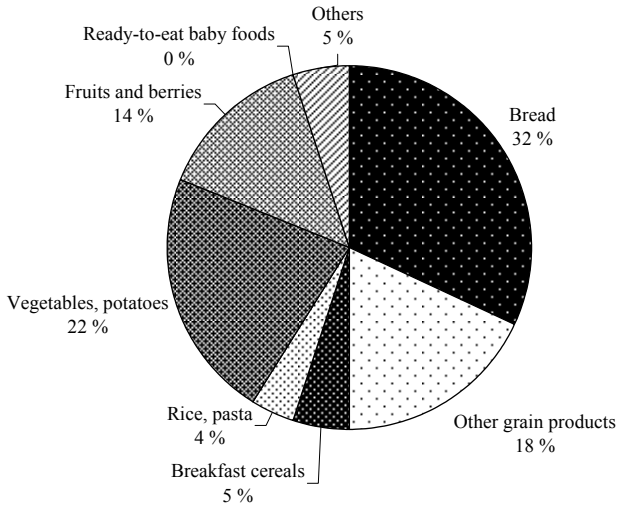
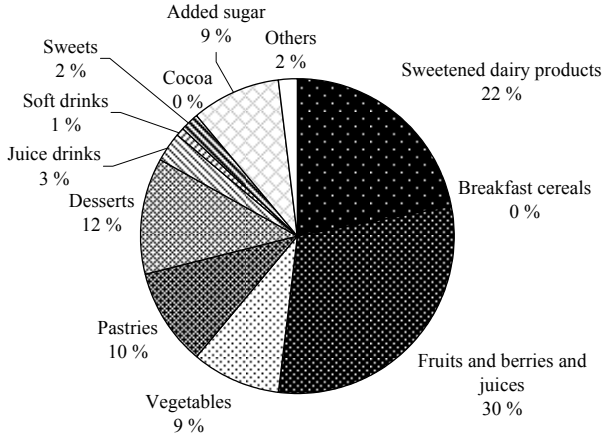
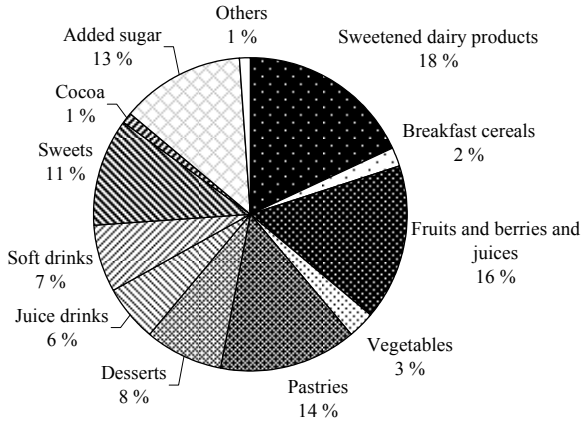


Figure 6. Fibre intake sources at **a)** 13 months, **b)** 5 years and **c)** 9 years old children.

a) 13 mo



b) 5 y



c) 9 y

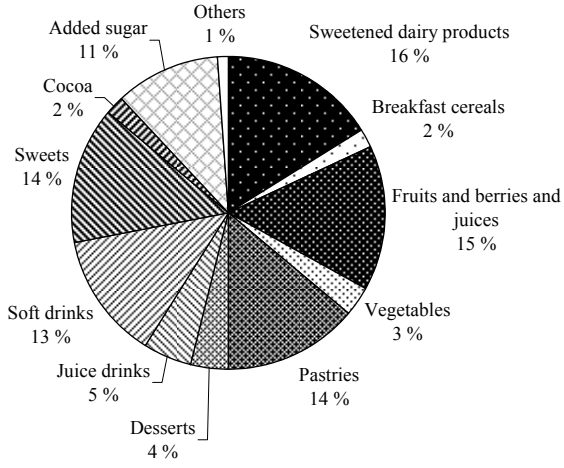


Figure 7. Sucrose intake sources at **a)** 13 months, **b)** 5 years and **c)** 9 years old children.

5.2 Serum lipids and apolipoproteins (I)

Total cholesterol, HDL and LDL cholesterol, apoA-I and apoB concentrations differed between the intervention and control children, often with a gender interaction (Tables 7- 9), but there were no differences between the STRIP study groups in triglycerides (Table 8). Total and HDL cholesterol, and apoA-I concentrations increased with age (Table 7 and 9).

5.2.1 Association between carbohydrates intake and serum lipids (I, II)

At the ages 5, 7, and 9 years total carbohydrate intakes (as E%) associated inversely with HDL concentrations when adjusted for dietary fatty acids SFA, MUFA, and PUFA (Table 10). An increase in total carbohydrate intake by 1 E% decreased HDL cholesterol by 0.006 mmol/L. Increased total carbohydrate and sucrose intakes associated with increased serum triglyceride concentrations when adjusted for dietary fatty acids (Table 10): an increase in total carbohydrate intake by 1 E% increased triglyceride concentration by 0.02 mmol/L and 1 E% increase in sucrose intake increased triglyceride concentration by 0.01 mmol/L. Fructose intake (as E%) was inversely associated with the HDL-cholesterol/total cholesterol ratio when adjusted for dietary fatty acids. ApoB concentration increased with increasing fructose intake when adjusted for dietary fatty acids. Starch intake (as E%) had no effect on serum lipid concentrations after fatty acid adjustments (data not shown).

In children between ages 13 months and 9 years serum total cholesterol concentration decreased as fibre intake increased. An increase in dietary fibre intake by 1 g/d decreased total cholesterol concentration by 0.007 mmol/L and 1 g/MJ by 0.059 mmol/L, whereas 1 g increase in SFA increased total cholesterol by 0.008 mmol/L and 1 E% increment in SFA increased cholesterol by 0.027 mmol/L. Fibre intake as g/d did not associate with HDL, while in longitudinal analyses a one g/MJ increase in dietary fibre intake decreased HDL cholesterol by 0.021 mmol/L. Neither fibre intake as g/MJ or g/d associated with LDL cholesterol at ages 5, 7, and 9 years.

The effect of dietary fibre on serum cholesterol values was stronger with lower SFA intakes (fibre [as g/MJ] main effect β=-0.17, P=0.007, fibre × SFA E% interaction, β=0.012, P=0.013). Non-HDL cholesterol results paralleled serum total cholesterol results (fibre [as g/MJ] main effect β=-0.13, P=0.015, fibre × SFA E% interaction, β=0.010, P=0.022).

Serum total cholesterol concentration and non-HDL cholesterol were lower and fibre intake was higher in STRIP intervention boys than control boys between ages 13 months and 9 years (for total and for non-HDL cholesterol P<0.001). Figure 8 shows non-HDL cholesterol and dietary fibre intake (as g/MJ) association in 13 months to 9 years old children. Although the dietary fibre intakes were higher in the intervention girls than in the control girls (*P*=0.017), no differences were found in serum total or non-HDL cholesterol concentrations between the groups (for total cholesterol P=0.18 and for non-HDL cholesterol P= 0.31) (Figure 8).

5.2.2 The effect of apoE phenotype on the association between carbohydrate intake and serum lipids (I)

The association between total carbohydrate intake and HDL cholesterol tended to be different between the apoE phenotype groups (ApoE × total carbohydrate interaction P=0.099). An increase in total carbohydrate intake as E% associated with lower HDL cholesterol in children with apoE3 or apoE4, but not in children with apoE2 (apoE2: P=0.78, apoE3: P<0.001, and apoE4: P<0.001, at the ages 5, 7, and 9 years). High intake of carbohydrates was associated with total and LDL cholesterol and triglyceride values similarly in all apoE groups at the ages of 5, 7, and 9 years (P>0.1 for interaction).

Table 7. Mean (SD) serum total and HDL cholesterol concentrations in intervention and control girls and boys from 13 months to 9 years of age.

Child's age Group	**N**	**N**	**Total cholesterol (mmol/L)**		**HDL cholesterol (mmol/L)**		**HDL/total cholesterol**	
	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys
13 months[1]								
intervention	107	119	4.25 (0.71)	3.74 (0.63)	0.86 (0.20)	0.88 (0.20)	0.21 (0.05)	0.24 (0.06)
control	115	120	4.38 (0.78)	4.15 (0.77)	0.91 (0.17)	0.92 (0.20)	0.21 (0.05)	0.23 (0.05)
2 years[1]								
intervention	106	125	4.25 (0.68)	3.91 (0.58)	0.97 (0.16)	1.00 (0.22)	0.23 (0.05)	0.26 (0.06)
control	115	121	4.89 (0.75)	4.32 (0.71)	1.02 (0.18)	1.04 (0.19)	0.23 (0.05)	0.25 (0.05)
3 years[1]								
intervention	109	126	4.38 (0.72)	4.05 (0.65)	1.04 (0.20)	1.06 (0.24)	0.24 (0.05)	0.27 (0.07)
control	111	131	4.52 (0.74)	4.41 (0.65)	1.08 (0.21)	1.11 (0.22)	0.24 (0.05)	0.26 (0.06)
4 years[1]								
intervention	109	126	4.36 (0.64)	4.06 (0.64)	1.11 (0.18)	1.12 (0.23)	0.26 (0.04)	0.28 (0.06)
control	121	131	4.40 (0.82)	4.40 (0.68)	1.13 (0.21)	1.16 (0.22)	0.26 (0.06)	0.27 (0.06)
5 years[2]								
intervention	109	125	4.41 (0.62)	4.15 (0.59)	1.17 (0.23)	1.21 (0.27)	0.27 (0.05)	0.29 (0.06)
control	121	126	4.56 (0.75)	4.42 (0.76)	1.22 (0.22)	1.23 (0.26)	0.27 (0.05)	0.28 (0.06)
7 years[2]								
intervention	106	120	4.57 (0.77)	4.16 (0.61)	1.29 (0.20)	1.30 (0.25)	0.29 (0.05)	0.32 (0.06)
control	124	126	4.57 (0.73)	4.42 (0.62)	1.27 (0.22)	1.28 (0.25)	0.28 (0.06)	0.29 (0.06)
9 years[2]								
intervention	115	130	4.56 (0.74)	4.27 (0.57)	1.27 (0.23)	1.35 (0.26)	0.28 (0.05)	0.32 (0.05)
control	131	134	4.72 (0.89)	4.50 (0.75)	1.30 (0.24)	1.33 (0.25)	0.28 (0.06)	0.30 (0.06)
P for intervention effect[3]			<0.001		0.027		0.104	
P for gender effect[3]			<0.001		0.087		<0.001	
P for age effect[3]			<0.001		<0.001		<0.001	

[1] Non-fasting samples at the ages 13 months, 2, 3, and 4 years.
[2] Fasting samples at the ages of 5, 7, and 9 years.
[3] Repeated measures analysis of covariance (RM ANCOVA), F-test.

Table 8. Mean (SD) fasting serum LDL-cholesterol and triglyceride concentrations in 5, 7, and 9 year old intervention and control girls and boys

Child's age Group	**N**	**N**	**LDL cholesterol (mmol/L)**		**Triglycerides[1] (mmol/L)**	
	Girls	Boys	Girls	Boys	Girls	Boys
5 years						
intervention	109	125	2.93 (0.55)	2.65 (0.52)	0.66 (0.62-0.69)	0.60 (0.57-0.64)
control	121	126	3.03 (0.67)	2.90 (0.64)	0.65 (0.62-0.69)	0.59 (0.56-0.63)
7 years						
intervention	106	120	2.96 (0.66)	2.57 (0.52)	0.66 (0.62-0.71)	0.59 (0.55-0.63)
control	124	126	2.99 (0.66)	2.82 (0.54)	0.64 (0.60-0.67)	0.64 (0.60-0.68)
9 years						
intervention	115	130	2.95 (0.65)	2.65 (0.44)	0.70 (0.66-0.74)	0.56 (0.53-0.60)
control	131	134	3.08 (0.78)	2.85 (0.66)	0.70 (0.66-0.74)	0.66 (0.62-0.70)
P for intervention effect[2]			<0.001		0.20	
P for gender effect[2]			<0.001		<0.001	
P for age effect[2]			0.40		0.08	

[1] Geometric mean (95% CI); log-transformed values used in analyses.
[2] Repeated measures analysis of covariance (RM ANCOVA), F-test.

Table 9. Mean (SD) serum apolipoprotein (Apo) concentrations in intervention and control girls and boys from 13 months to 9 years of age.

Child's age Group	**N**	**N**	**ApoA-I (g/L)**		**ApoB (g/L)**	
	Girls	Boys	Girls	Boys	Girls	Boys
13 months[1]						
intervention	107	119	1.06 (0.16)	1.05 (0.16)	0.88 (0.17)	0.75 (0.17)
control	115	120	1.10 (0.15)	1.09 (0.15)	0.89 (0.17)	0.82 (0.18)
2 years[1]						
intervention	106	125	1.08 (0.13)	1.10 (0.15)	0.81 (0.15)	0.73 (0.14)
control	115	121	1.13 (0.14)	1.14 (0.15)	0.84 (0.18)	0.80 (0.19)
3 years[1]						
intervention	109	126	1.12 (0.14)	1.13 (0.17)	0.82 (0.16)	0.74 (0.17)
control	111	131	1.16 (0.15)	1.18 (0.17)	0.82 (0.16)	0.80 (0.17)
4 years[1]						
intervention	109	126	1.18 (0.15)	1.19 (0.18)	0.81 (0.15)	0.72 (0.16)
control	121	131	1.18 (0.16)	1.22 (0.17)	0.81 (0.20)	0.79 (0.19)
5 years[2]						
intervention	109	125	1.25 (0.19)	1.27 (0.22)	0.80 (0.15)	0.70 (0.14)
control	121	126	1.28 (0.18)	1.27 (0.20)	0.80 (0.17)	0.75 (0.17)
7 years[2]						
intervention	106	120	1.31 (0.18)	1.31 (0.21)	0.76 (0.18)	0.66 (0.15)
control	124	126	1.30 (0.19)	1.32 (0.21)	0.75 (0.17)	0.73 (0.16)
9 years[2]						
intervention	115	130	1.44 (0.20)	1.49 (0.23)	0.81 (0.18)	0.69 (0.14)
control	131	134	1.45 (0.21)	1.50 (0.23)	0.82 (0.21)	0.77 (0.19)
P for intervention effect[3]			0.01		0.001	
P for gender effect[3]			0.13		<0.001	
P for age effect[3]			<0.001		<0.001	

[1] Non-fasting samples at the ages of 13 months, 2, 3, and 4 years.
[2] Fasting samples at the ages of 5, 7, and 9 years
[3] Repeated measures analysis of covariance (RM ANCOVA), F-test.

Table 10. Associations of dietary carbohydrate intake with fasting serum lipid and apolipoprotein concentrations in children at ages 5 (n=644), 7 (n=585), and 9 (n=550) years.

	Carbohydrate intake E%		**Sucrose intake E%**		**Fructose intake E%**		**Dietary fibre intake g/d**	
	β[1]	P[2]	β[1]	P[2]	β[1]	P[2]	β[1]	P[2]
Total cholesterol (mmol/L)	-0.0044	0.40	0.0021	0.63	0.015	0.08	-0.0024	0.56
HDL cholesterol (mmol/L)	-0.0058	<0.001	-0.0008	0.60	-0.0021	0.46	0.0001	0.93
HDL/total cholesterol	-0.0004	0.20	-0.0002	0.52	-0.0013	0.027	0.00003	0.91
LDL cholesterol (mmol/L)	-0.0043	0.32	0.0007	0.86	0.013	0.064	-0.0040	0.26
Triglycerides[3] (mmol/L)	0.016	<0.001	0.011	<0.001	0.008	0.12	-0.0008	0.76
ApoA-I (g/L)	0.00007	0.97	0.0005	0.75	0.0000004	1.0	0.0018	0.18
ApoB (g/L)	0.0010	0.36	0.0016	0.09	0.0037	0.047	-0.0014	0.12

[1]β describes net change (mmol/L etc) in serum lipid values per 1 E% (g/d for dietary fibre) change in carbohydrate intake.
[2]RM ANOVA with age, gender, body mass index, and STRIP study group as covariates and adjusted for SFA, MUFA, and PUFA; backward selection.
[3]Log-transformed values used in analyses.

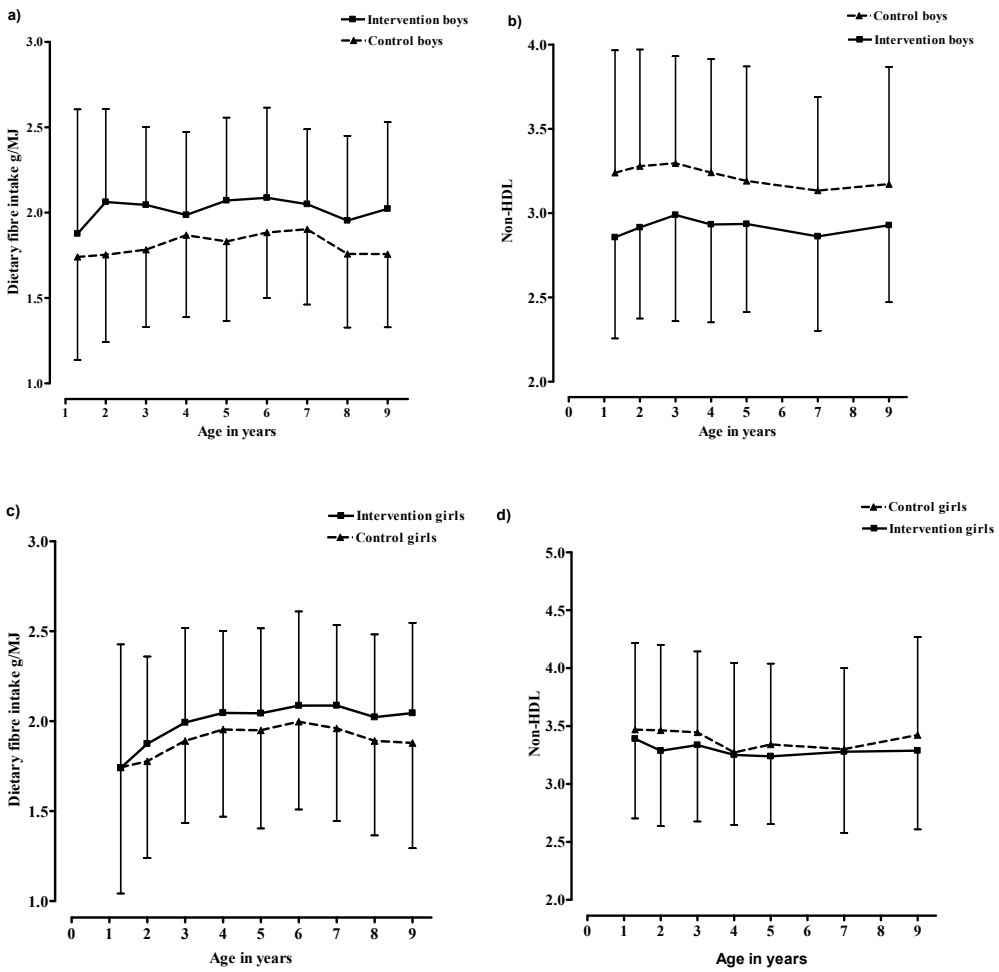


Figure 8. **a)** Dietary fibre intake (as g/MJ) in intervention boys (solid line) and control boys (broken line), intervention effect P<0.001; **b)** serum non-HDL cholesterol in intervention boys (solid line) and control boys (broken line), P<0.001; **c)** dietary fibre intake in intervention girls (solid line) and control girls (broken line), P=0.017; and **d)** serum non-HDL cholesterol in intervention girls (solid line) and control girls (broken line), P=0.31. The data are mean (SD) values, P-values for RM ANCOVA from 13 months to 9 years of age.

5.3 Associations of energy and energy nutrient intakes with fibre intake (II)

Dietary fibre intake associated positively with energy intake, as every 100 kcal increase associated with a 0.7 g higher fibre intake. When adjusted for fat intake, a 1 g increase in dietary fibre intake increased energy intake by 21 kcal in the intervention group and by 23 kcal in the control group (P=0.025 for dietary fibre × group interaction, P<0.001 for fibre main effect). Figure 9 illustrates energy and fibre intake association specifically in 5-year-old children.

Dietary fibre intake associated inversely with fat intake (as E%). When fat intake increased by 1 E%, dietary fibre intake decreased by 0.08 g. Figure 10 illustrates this

association specifically in 5-year-old children. Dietary fibre associated positively with total carbohydrates (as E%). When carbohydrate intake increased by 1 E%, dietary fibre increased by 0.08 g. Sucrose intake was associated with increased fibre intake at the age of 13 months: 1E% increase in sucrose intake increased fibre intake by 0.2 g. However, the effect was the opposite in the older children: between 2 and 9 years of age increasing sucrose intake by 1 E% decreased dietary fibre intake 0.1 - 0.2 g/d depending on the child's age. Increasing protein intake had a decreasing effect to dietary fibre: when protein intake increased by 1E%, dietary fibre decreased by 0.05 g.

5.4 Intakes of energy, nutrients, and food groups in different fibre intake groups (II)

There were no differences in energy intakes between the three fibre intake groups, but fat, SFA, and MUFA intakes were continuously lower in the high dietary fibre intake group than in the other two groups. Furthermore, protein intake was higher and sucrose intake was lower in the high dietary fibre intake group than in the two other groups.

Children with high dietary fibre intake received more vitamins and minerals than children in the two other fibre intake groups. The children with high fibre intake received more vitamin E ($P<0.001$), vitamin C ($P<0.001$), folic acid ($P<0.001$), vitamin B_1 ($P<0.001$), vitamin B_6 ($P<0.001$), iron ($P<0.001$), zinc ($P<0.001$), potassium ($P<0.001$), and magnesium ($P<0.001$) than children with low dietary fibre intake between ages 13 months and 9 years. There were no differences between dietary fibre intake groups in vitamin B_2 or B_{12} intakes or in calcium intake.

Children with high fibre intake between ages 13 months and 9 years consumed more cereal products, vegetables, and fruits and berries than children in the other fibre intake groups (Table 11). No differences were found between the fibre intake groups in milk products or meat and fish consumption. Children with low fibre intake consumed more sweets, sugar and ready-to-eat-baby foods than children in other fibre intake groups. Sources of dietary fibre intake differed between the fibre intake groups at the age of 13 months. The most common sources of dietary fibre in the high fibre intake group were other cereal products than bread (porridge and gruel) (28%), whereas children with low fibre intake received most dietary fibre (32%) from ready-to-eat baby foods. At 9 years of age there were no differences between the groups in fibre intake sources, bread contributing most to the dietary fibre intake (29-35%) in all fibre intake groups.

5.5 Fibre intake and growth, and blood pressure (II)

Dietary fibre intake between 8 months and 2 years as g/d did not associate with weight gain between ages 8 months and 2 years. Dietary fibre intake had no association with length or height between 8 months and 2 years of age, or in longitudinal growth analyses between ages 13 months and 9 years. Figure 11 illustrates the association between dietary fibre (as g/d) and weight (kg) in 13-month-old children.

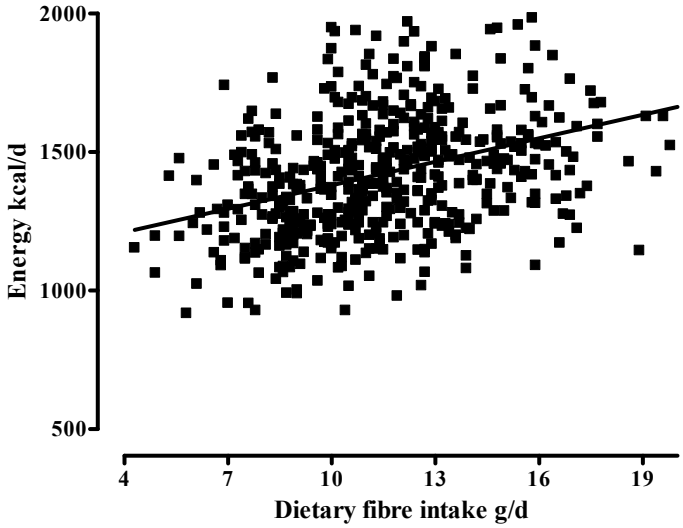


Figure 9. Association of energy intake (kcal) with dietary fibre intake (g/d) in 5-year-old children (n=521). Pearson's correlation coefficient is 0.388 (P<0.01).

Figure 10. Association of fat intake as percentage of total daily energy intake (E%) with dietary fibre intake (g/d) in 5-year-old children (n=521). Pearson's correlation coefficient is -0.241 (P<0.01).

Table 11. Mean (SD) daily food consumption (g/d) of selected food groups by children in groups with lowest (10%), average (80%) or highest (10%) fibre consumption.

Child's age Fibre intake group		Cereal products	Vegetables	Fruits and berries	Spreads	Milk products	Meat and fish	Sweets and added sugar	Ready-to-eat baby foods
13 months									
low	58	42 (26)	98 (75)	62 (61)	6 (6)	441 (248)	41 (32)	4 (6)	437 (312)
average	429	60 (34)	138 (76)	88 (63)	7 (7)	505 (236)	51 (31)	5 (6)	298 (241)
high	56	81 (43)	168 (92)	89 (59)	8 (8)	472 (189)	57 (30)	4 (5)	238 (254)
3 years									
low	57	83 (40)	109 (50)	154 (139)	16 (7)	542 (223)	94 (42)	21 (17)	78 (154)
average	407	100 (35)	141 (60)	152 (97)	18 (7)	543 (176)	94 (38)	19 (13)	22 (57)
high	52	111 (43)	179 (70)	193 (86)	19 (8)	540 (168)	98 (34)	16 (10)	15 (28)
5 years									
low	56	122 (47)	118 (56)	137 (95)	22 (8)	604 (184)	112 (45)	32 (16)	14 (49)
average	411	127 (42)	161 (66)	161 (97)	24 (9)	619 (180)	110 (41)	26 (17)	6 (23)
high	54	139 (40)	195 (94)	209 (98)	23 (10)	607 (190)	99 (35)	21 (15)	3 (9)
7 years									
low	53	132 (41)	156 (65)	124 (98)	28 (10)	662 (217)	125 (42)	34 (22)	4 (10)
average	394	157 (53)	179 (69)	168 (114)	27 (11)	669 (192)	120 (42)	29 (18)	4 (21)
high	54	180 (57)	225 (84)	214 (111)	29 (10)	685 (225)	117 (45)	24 (17)	4 (10)
9 years									
low	58	162 (66)	134 (62)	121 (103)	32 (14)	692 (217)	136 (54)	46 (29)	4 (17)
average	429	172 (58)	179 (75)	174 (126)	28 (11)	663 (231)	135 (52)	36 (27)	3 (12)
high	56	184 (49)	221 (90)	228 (162)	30 (11)	648 (232)	119 (44)	23 (16)	2 (5)
P for group effect [1]		<0.001	<0.001	<0.001	0.013	0.19	0.07	<0.001	<0.001

[1] Repeated measures ANCOVA with STRIP study group, age and STRIP study group × age, F-test.

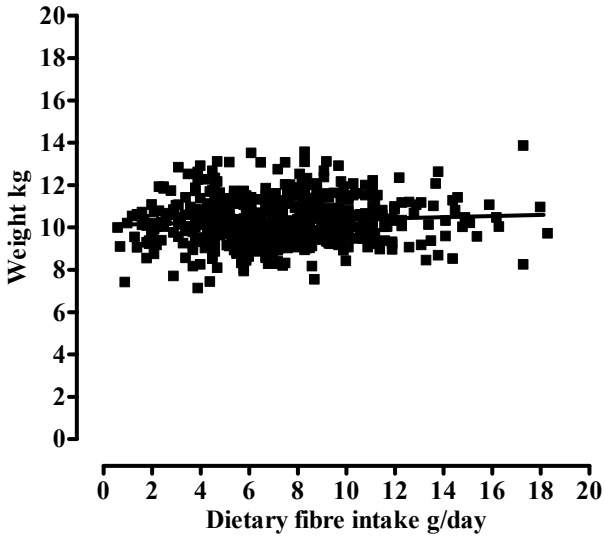


Figure 11. Weight (kg) and dietary fibre intake (g/d) in 13-month-old children (n=543), regression model β= 0.002, P= 0.22.

In longitudinal analyses between ages 13 months and 9 years, weight was similar in all three dietary fibre intake groups (P=0.54) (Table 12). Long-term dietary fibre intakes (as g/MJ or g/d) between ages 13 months and 9 years were similar (P=0.91 for g/MJ, P=0.44 for fibre g/d) in the groups of thin, normal weight, and overweight children at age 9 years. Percentages of 9-year-old children with low, average, and high fibre intake in thin, normal, and overweight groups were similar (Figure 12).

Dietary fibre intake as g/d or as g/MJ did not associate with systolic or diastolic blood pressure of the children between ages 13 months and 9 years, and blood pressures were similar in all three dietary fibre intake groups.

5.6 Associations of energy and energy nutrient intakes with sucrose intake

Sucrose intake associated positively with energy intake, as 1 g increase in sucrose intake increased energy intake by 9 kcal, and when adjusted with fat intake, the increase in energy intake was 5 kcal. Figure 13 specifically illustrates energy and sucrose intakes in 5-year-old children. Sucrose intake associated positively with fat intake in grams, as increase in fat intake by 1 g increased sucrose intake by 0.3 g. However, fat intake as E% had a decreasing effect on sucrose in infants and preschool aged children, as 1 E% increase in fat intake decreased sucrose intake by 0.1 g. This association was the opposite in school-aged children: an increase in protein intake in grams increased sucrose intake, as 1 g increase in protein intake increased sucrose intake by 0.07 g. Protein intake as E% had a decreasing effect to sucrose intake, when protein intake increased by 1 E%, sucrose intake decreased by 2.4 g.

5.7 Intakes of energy, nutrients, and food groups in different sucrose intake groups (III)

There were no differences in energy intake or in total fat intake (as E% or g/d) among the three sucrose-intake groups. However, the children with low sucrose intake received continuously more PUFA and MUFA and less SFA (E%) than the children with high sucrose intake. The children in the low sucrose intake group also received more protein than children in the high sucrose intake group when expressed as E% or as g/d.

The children with low sucrose intake group received more vitamin E ($P<0.001$), niacin ($P<0.001$), calcium ($P=0.002$), iron ($P<0.001$), and zinc ($P<0.001$) than the children with high sucrose intake. An exception was vitamin C, which showed no differences between the groups ($P=0.16$). The children with low sucrose intake also received more dietary fibre than the other children ($P<0.001$). The proportion of children receiving less than the NNR recommended amounts of vitamin B_1 ($P<0.05$), pyridoxine ($P<0.001$), iron ($P<0.001$), zinc ($P<0.001$), magnesium ($P<0.001$), and calcium ($P<0.01$) was constantly higher in the high sucrose intake group than in the other two sucrose-intake groups.

The children in the low sucrose intake group consumed more cereal products, vegetables, milk products, and meat and fish than the other children, whereas the children in the high sucrose intake group consumed more juice drinks, soft drinks, sweets and sweetened milk products (Table 13). The most common sources of sucrose in the low sucrose intake group were fruits and berries, whereas children with high sucrose intake received most of their sucrose from sweetened milk products and soft drinks.

Table 12. Geometric mean (95% confidence interval) weight by children in groups with lowest (10%), average (80%) or highest (10%) dietary fibre consumption.

Child's age **Fibre intake group**		**Weight (kg)** [1]
13 months		
low	58	10.3 (10.0-10.6)
average	429	10.2 (10.1-10.4)
high	56	10.3 (10.0-10.5)
3 years		
low	57	14.9 (14.4-15.4)
average	407	15.0 (14.8-15.2)
high	52	15.4 (14.9-15.9)
5 years		
low	56	18.9 (18.3-19.6)
average	411	19.1 (18.9-19.4)
high	54	19.8 (19.0-20.5)
7 years		
low	53	23.8 (22.9-24.8)
average	394	24.6 (24.2-24.9)
high	54	25.0 (23.9-26.2)
9 years		
low	58	30.3 (28.9-31.7)
average	429	30.8 (30.3-31.3)
high	56	31.0 (29.5-32.6)
P for group effect [2]		P=0.54

[1] Log transformed values used in analysis.
[2] Repeated measures analysis of covariance (RM ANCOVA), with covariates gender and STRIP study group.

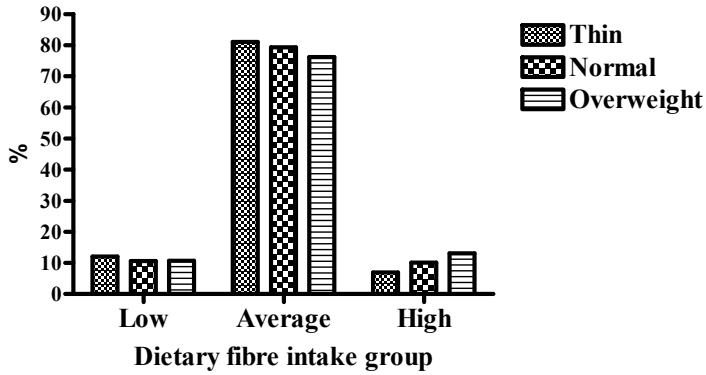


Figure 12. Percentages of 9-year-old children in thin, normal and overweight groups according to Cole et al. (2000, 2007) standards in lowest (10%), average (80%) and highest (10%) fibre intake groups (Cochran-Mantel-Haenszel method P=0.48). Overweight was defined according to the age- and gender-specific BMI cut-off points of the International Obesity Task Force BMI for children (Cole et al. 2000), corresponding to the adult BMI ≥25 kg/m^2. Children were classified as thin according to the international age- and gender-specific BMI cut-off points for children (Cole et al. 2007), corresponding to the adult BMI ≤18.5 kg/m^2. N=538.

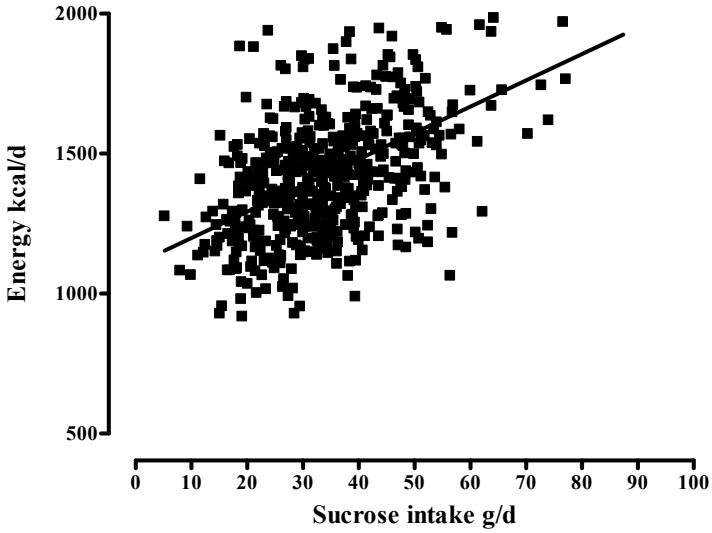


Figure 13. Association of energy intake (kcal) with sucrose intake (g/d) in 5-year-old children (n=521). Pearson's correlation coefficient 0.504 (P<0.01).

Table 13. Mean (SD) daily food consumption (g/d) of selected food groups by children in groups with lowest (10%), average (80%) or highest (10%) sucrose consumption.

Child's age Sucrose intake group	N	Cereal products	Vegetables	Fruits and berries	Spreads	Milk products	Meat and fish	Sweets	Soft drinks
13 months									
low	54	64.1 (39.8)	164.7 (102.5)	59.0 (59.0)	5.9 (5.9)	407.5 (236.7)	45.6 (45.6)	0.3 (1.3)	0 (0)
average	435	60.0 (35.0)	134.1 (76.3)	78.7 (60.1)	7.0 (6.7)	447.8 (227.2)	51.5 (31.3)	0.5 (1.7)	1.2 (9.1)
high	54	58.4 (32.1)	111.8 (59.0)	96.5 (66.0)	8.2 (7.3)	411.1 (238.5)	47.6 (30.4)	1.2 (2.8)	2.6 (8.7)
3 years									
low	52	112.1 (46.9)	161.1 (70.5)	134.6 (106.1)	19.0 (8.9)	509.4 (189.6)	104.6 (49.4)	4.3 (7.1)	8.4 (26.3)
average	415	99.7 (35.4)	134.3 (58.9)	135.9 (96.2)	17.6 (6.7)	459.6 (171.1)	94.1 (37.3)	8.4 (10.2)	22.1 (40.8)
high	49	82.6 (31.5)	93.3 (58.7)	182.5 (123.5)	16.8 (8.9)	380.2 (196.0)	87.9 (29.7)	10.8 (10.4)	43.2 (51.3)
5 years									
low	50	145.1 (44.4)	165.3 (64.5)	125.9 (102.3)	25.6 (10.3)	573.9 (210.2)	118.9 (36.3)	5.3 (7.3)	11.5 (24.3)
average	419	126.5 (42.5)	149.0 (63.6)	146.7 (91.1)	23.2 (8.4)	525.5 (168.2)	109.6 (41.6)	14.4 (15.1)	36.6 (52.2)
high	52	120.5 (37.6)	125.6 (68.5)	169.8 (129.8)	22.7 (9.5)	476.1 (207.9)	96.2 (35.1)	16.1 (13.6)	71.8 (79.5)
7 years									
low	50	182.5 (63.7)	194.2 (86.8)	134.5 (103.9)	28.0 (10.6)	612.6 (210.9)	130.5 (43.3)	10.2 (13.2)	18.9 (34.1)
average	401	156.5 (51.6)	169.9 (64.6)	149.4 (106.6)	27.0 (10.4)	579.5 (195.3)	120.4 (41.7)	15.5 (16.9)	49.9 (71.1)
high	50	138.2 (48.7)	140.7 (68.3)	206.4 (145.2)	27.2 (10.7)	513.5 (212.7)	111.3 (43.9)	21.5 (18.2)	90.5 (100.5)
9 years									
low	54	193.9 (69.2)	186.0 (87.8)	123.4 (120.0)	28.7 (12.4)	644.1 (243.9)	145.8 (54.4)	10.5 (15.9)	30.8 (57.7)
average	435	172.0 (55.4)	168.2 (70.7)	165.8 (130.2)	28.1 (11.0)	571.0 (217.7)	133.7 (52.2)	23.4 (24.2)	81.0 (99.7)
high	54	153.0 (64.2)	137.7 (71.8)	185.9 (137.3)	28.9 (13.4)	465.7 (242.4)	120.5 (44.5)	27.9 (25.2)	127.7 (120.2)
P for interaction between time and groups[1]		0.028	0.715	0.111	0.370	0.049	0.112	<0.001	<0.001
P for group effect [1]		<0.001	<0.001	0.001	0.079	<0.001	0.001	0.001	<0.001

[1] Repeated measures ANOVA including sucrose-intake group, STRIP study group, age, and all their pairwise interactions, F-test.

5.8 Sucrose intake and growth (III)

The children with high sucrose intake were taller than the other two groups of the children during the first 6 years of the study, whereas the children with low sucrose intake were taller than the children in the other two groups between 7 and 9 years of age. The mean height of the children with low sucrose intake increased from 70.0 cm at 7 months to 136.6 cm at 9 years, whereas the respective values were 70.3 cm to 136.0 cm in the children with average sucrose intake and 71.3 cm to 136.4 cm in children with high sucrose intake ($P<0.05$ between groups). Thus, the difference in growth between 7 months and 9 years of age was 1.5 cm between the low sucrose and the high sucrose intake group, low sucrose intake group growing more than high sucrose intake group. A similar pattern was seen in the mean relative heights ($P<0.001$). The children with high sucrose intake weighed more during the first few years of the study, whereas, after 4 years of age their weight was lower than that of the children in the other groups ($P<0.05$). A similar pattern was seen in relative weight ($P<0.001$).

5.9 Sucrose intake and dental health (IV)

The mean sucrose intake of the children with extremely high sucrose intake (5%) (n=33) between ages 2 and 10 years always exceeded the maximum recommended amount of 10 E% (Nordic Council of Ministers 2004, WHO 2003), but mean sucrose intake in the children with extremely low sucrose intake (5%) (n=33) was always under 8 E%. Mean (SD) sucrose intake of the children with extremely high sucrose intake was 7.0 E% (2.9) at 13 months and 11.9 E% (2.8) at the age of 10 years. In the children with extremely low sucrose intake, sucrose intake increased from 3.0 E% (1.8) at 13 months to 7.4 E% (3.5) at 10 years ($P<0.001$ between sucrose intake groups). The highest sucrose intakes between ages 13 months and 9 years were 13.9 E% (3.5) in the children with extremely high sucrose intake at the age of 4 years and 7.4 E% (3.5) in the children with extremely low sucrose intake at the age of 10 years.

According to the frequency questionnaire, the sources of sucrose in the diet were quite similar between the groups at the mean age of ten years. The most frequently used sugar-rich food source was the group of sweetened milk products. Of these, the most frequently used was cocoa milk drinks. Among the group of sugared drinks and juices, the most frequently used group was home-made or commercially available juice concentrates. Among the group of sweets and chocolate, the most frequently used were fruit flavoured sweets. The mean (SD) intake frequency of sugar-rich foods was 5.2 (2.1) times a day in the children with extremely high sucrose intake and 3.8 (2.3) times a day in the children with extremely low sucrose intake. Intake frequency of sucrose and caries prevalence showed a significant correlation in the children with extremely high sucrose intake but not in the children with extremely low sucrose intake (r_s= 0.376; P= 0.031 and r_s= 0.033; P=0.854, respectively).

The mean (SD) dmft-values (sum of decayed, missing and filled teeth in the primary teeth) at the mean age of ten years were 2.7 (3.3) in the children with extremely high sucrose intake, and 1.1 (1.2) in the children with extremely low sucrose intake

(P=0.18). The DMFT (sum of decayed, missing and filled teeth in the permanent teeth) and dmft + DMFT scores were higher in the children with extremely high sucrose intake and lower in the children with extremely low sucrose intake [1.4 (2.0), 3.9 (3.9), and 0.5 (1.1), 1.9 (2.5), P = 0.01, P=0.032, respectively] (Figure 14). The association between sucrose intake and dental health remained similar even though tooth brushing frequency was controlled. There were more children with higher dmft and DMFT scores in the children with extremely high sucrose intake than in the children with extremely low sucrose intake, and more children with dmft score 0 or DMFT score 0 in children with extremely low sucrose intake than in other children (Figure 15 a and b).

Between the sucrose intake groups there were no differences in existence of plaques. Only 12 (36%) children with extremely high sucrose intake and 10 (30%) children with extremely low sucrose intake brushed their teeth twice a day as recommended, and 21 children with extremely high sucrose intake and 23 children with extremely low sucrose intake brushed their teeth only once a day or less frequently. In both groups the children used xylitol products on average once a day (range 0 – 6 times a day). Dental floss (n=7) and mouthwashes (n=4) were used once in a while.

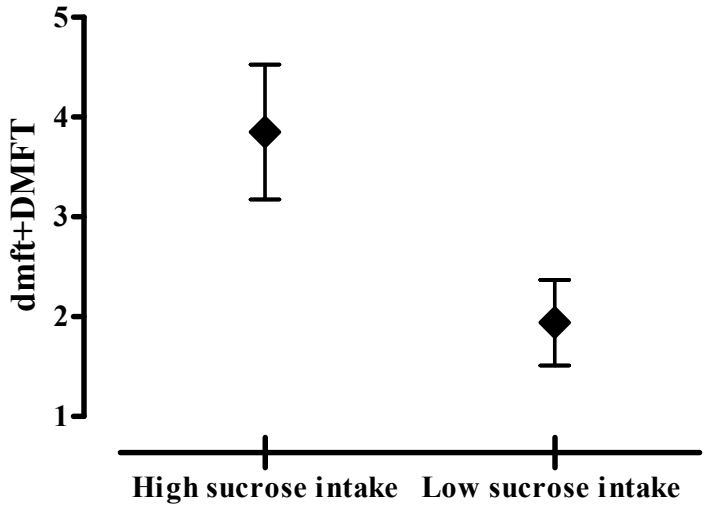
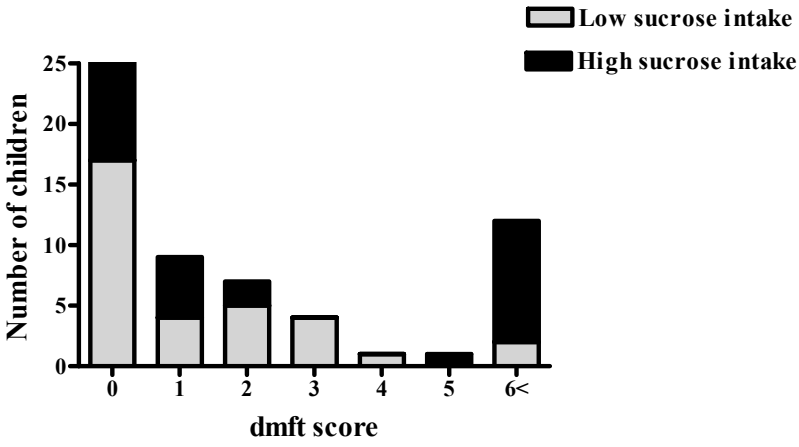


Figure 14. The mean (SD) of decayed, missing and filled teeth in the primary (dmft) and in the permanent teeth (DMFT) in the children with extremely high sucrose intake (5%), and in the children with extremely low sucrose intake (5%) at the mean age of 10-years. (N=65). Mann-Whitney U-test was used for calculation of P value P=0.032.

a)



b)

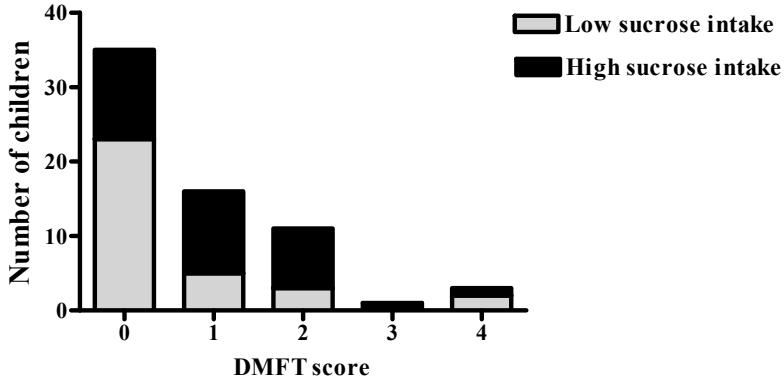


Figure 15. Number of 10-year-old children (n=65) with **a)** dmft (sum of decayed, missing and filled teeth in the primary teeth) and **b)** with DMFT (sum of decayed, missing and filled teeth in the permanent teeth) scores in children with extremely low sucrose intake (5%) and children with extremely high sucrose intake (5%).

6 DISCUSSION

6.1 Study subjects and their representativeness

The representativeness of the study population and the lost follow-up (dropping out) are important factors to be taken into account in a long-term prospective intervention study. It is obvious that children in this study are not entirely representative of the whole Finnish population. The families of the STRIP project volunteered to participate in the study, and thus, they may have had a more positive health attitude than average people in the community. Even if the sample population were somewhat selected in their health attitudes, it only consolidates some of the results gained in this study, such as the unfavourable impacts of sugar and benefits of dietary fibre which are more difficult to detect among positive health attitude subjects.

In long-term studies the loss to follow-up is usually a problem. In this study some families have discontinued participating in the study over the years mainly due to changing residence or lack of time. However, the average adherence to follow-up was good. Attempts were made to exclude the effects of loss to follow-up by selecting the children from the STRIP study who showed maximum adherence in the STRIP study to be included in the substudies (studies I-IV). Study I selected children who had fasting blood samples taken at the age of 5, 7, and 9 and who had kept food records simultaneously. In studies II and III the study subjects consisted of the children who had returned at least 10 of the requested 15 food records. Study IV selected children with sucrose intake extremes (5%) among the participants of the STRIP project according to the previously recorded 9 year-long sucrose consumption. The idea was to maintain the characteristics of the study subjects as appropriate as possible from the point of view of each subproject. Children with familial hypercholesterolemia, type 1 diabetes or other disease which might affect serum lipid concentration or sucrose intake habits were excluded from the studies. Serum sex hormone concentrations during pubertal maturation are known to affect serum cholesterol concentrations (Wennlöf et al. 2005) and puberty can also impact dietary habits. The effect of puberty, as well as many other factors that are potent confounders, e.g. smoking, alcohol use, and lipid lowering medications, were excluded in this study population, since the children were under 10 years of age.

Exclusion of the children from studies I and II-III was mainly due to loss of follow-up prior to age 9 years. Of the children who participated at the 9 y visit, only 7 (1%) were excluded from study I due to missing blood samples and/or food records, and 42 (8%) were excluded from studies II-III due to insufficient food record series. Children excluded from studies I-III and those included did not differ between growth, serum lipid values or nutrients, and no evidence of selection bias was found between the groups. When investigating participants of the main STRIP study and those lost from the follow-up, the drop-out rates have not been influenced, e.g. by the child's weight during the first 10 years (Hakanen 2006). In the intervention and control groups loss of follow-up was equal, the most important interpreter for non-appearance was reluctance for blood samples between ages 8 months and 9 years (Saarinen 2008).

6.2 Methods

The present study has considerable strengths, including its prospective nature, repeated measurements of dietary data, height and weight as well as lipids and the ability to adjust potential confounders except physical activity. Due to the lack of data on physical activity, we were not able to study the impact of physical activity on serum lipids or dietary intake data. However, physical fitness has not been shown to contribute to the serum lipoproteins in 12-year-old children (Perry et al. 1997) and leisure-time physical activity was not associated with the intake of energy nutrients or fibre in the 13-year-old children in the STRIP study (Pahkala et al. 2010).

Food records and their limitations

Limitations of study methods are mainly addressed to the use of food records, which are prone to errors. Food records represent a self-reported method, which always has its limitations, but the comprehensive work performed in the STRIP study probably minimises possible biases. Families were carefully instructed to keep food records. Written instructions with drawings of food proportions were sent to the families. The food records were kept on consecutive days and included at least one weekend day and were evenly distributed throughout the year. Parents were advised not to change their child's diet at the time of the food recordings. The records were reviewed by a nutritionist for completeness and accuracy at each follow-up visit and, when appropriate, families were asked to provide further details on incomplete food items or amounts. A picture book of food portions was used to help estimate portion sizes at the visit to the centre. Food records were analysed with the Micro Nutrica® program by the same experienced dietary technician. She clarified possible ambiguities in the food records and updated the nutrient composition of foods data, contacted representatives of the food industry to ask for nutrient values data of new products, and entered the exact nutrient composition on school catering menus and servings (questionnaires were sent to school kitchens). Also when the children were under school-age, day-care centres were asked to collaborate with filling the food records and provide further details on incomplete food items.

Although several problems have been associated with food records, the primary concerns relate to the underreporting of foods for children by their parents and specifically, conscious underreporting consumption of fat and sugar containing foods known to be socially unacceptable (Eck et al. 1989). However, Lillegaar and Andersen (2005) concluded that, in 9-year-old children, neither underreporters nor acceptable reporters showed a systematic misreporting related to unhealthy foods or macronutrients. Furthermore, carbohydrate or added sugar intake as E% were not significantly different between underreporters or acceptable reporters (Lillegaard et al. 2007). The level of estimated underreporting rises significantly with age (Pikholz et al. 2004) and previous studies suggest that underreporting is not a problem in young children (Livingstone et al. 1992, Rennie et al. 2005). Eck and colleagues report (1989) that parents have been reasonably accurate in recalling dietary intake of their children aged 4.0-9.5 years.

Huybrechts and colleagues (2008) have shown that four days has been required to ensure reliable results for carbohydrate or simple carbohydrate intake in children aged 2.5-6.5 and four days to be sufficient also for most micronutrients to ensure young children's dietary intake. Similar results were found in an earlier study by Nelson and colleagues (1989) who reported that four days was required to ensure reliable results for total sugars in young children. However, for dietary fibre the number of days is more: according to Huybrechts and colleagues (2008) six days for children aged 2.5-6.5 years to ensure reliable results, and according to Nelson and co-workers (1989) four days for children aged 1-4 years and 6-8 days for children in the age 5-17 years. This study used three-day food records for children younger than 2 years old and four-day food records for over 2 years old children. The carbohydrate and sucrose intake results in the present study were fairly congruent with previous studies. However, definite results for dietary fibre should readily require 2 days more in food records.

6.3 Carbohydrate definitions, growth, and diet quality

6.3.1 Total carbohydrate

One problem in carbohydrate research is that the carbohydrate intake usually correlates inversely with the intake of other macronutrients and therefore, it can be difficult to determine which macronutrient is genuinely associated with the research focus. This concerns fat, in particular, because an increase in carbohydrate consumption is tightly associated with a decrease in fat intake (Garemo et al. 2006). Consequently, the results on the relation between dietary carbohydrates and serum lipids or carbohydrates' relation with growth or diet are not unambiguous. It is also difficult to distinguish the effect of total carbohydrate from the effects of carbohydrate subgroups, e.g. starch, dietary fibre, sucrose, and fructose, because the subgroups show a strong connection. To avoid these problems in this study, carbohydrates' association with serum lipids were also adjusted with fat or fatty acids. Dietary carbohydrates correlate also with total energy, so energy adjusted units were also used in this study, e.g. carbohydrate as E%, sucrose as E% and fibre as g/MJ.

6.3.2 Sugar

Sugar can be defined in many ways. Many of the terms, such as "*sugar*", "*sugars"*, "*refined sugar*", "*refined sugars*", "*total available sugars*", *"added sugar"* are used in publications about intakes, making intake comparisons between studies very difficult (Ruxton et al. 1999, Cummings and Stephen 2007). In this study, we used the term *sucrose*, which does not distinguish added or natural sugar. Food records were analyzed with the Micro Nutrica® program, in which nutrient composition data are based on food composition tables, and naturally occurring sugars are not distinguished from sugars added during food manufacturing, although there is no data on how natural intrinsic sugars and added sugars differ in (chemical analysis or) the human physiological metabolism. When estimating intakes of sugars over time, it must be taken into account that definitions have changed and differ between countries, changes

occur in food composition and dietary intake methods, and that there are acknowledged increases in the underreporting of intake (Sigman-Grant and Morita 2003).

In nutrition counselling, when considering sugar intake, total sugar consumption must be taken into account. Total sugar includes also fructose-glucose syrup used by food-industry. Therefore, the term “refined sugar" would be good to adopt into practice, because it refers to sucrose, fructose, starch-based sweeteners (syrups of glucose-fructose) and other similar sugar products used either alone or added to food during preparation (National Nutrition Council 2005).

Some previous studies have shown that a low fat diet might lead to high intake of sugar (Nicklas et al. 1992). However, there is also evidence that a high-sugar diet might lead to high intake of fat (Emmet and Heaton 1995). However, in the present study no differences were found in energy or in total fat intake (as E% or g/d) among the low, average, or high sucrose intake groups. Associations between sugar and fat usually correlate with each other, and much of added sugar in the diet comes from foods that are also high in fat e.g. pastries (Kranz et al. 2006). In the present study, sucrose intake associated positively with fat intake in grams, as increase in fat intake by 10 g increased sucrose intake by 3 g in children aged 1-9 years. Furthermore, the children with low sucrose intake had better quality of fat than children with high sucrose intake. The children in the low sucrose intake group received continuously more PUFA and MUFA and less SFA (E%) than the children with high sucrose intake. In the STRIP study, the intervention children repeatedly received counselling aiming at decreasing saturated fat and cholesterol intake, which might have led also to the lower intake of sugar, regarding foods which are high in fat (especially saturated fatty acids) and high in sugar. A Swedish study by Garemo and colleagues (2007b) has shown that only part of the sucrose in children’s diet come from sweets, and the rest is hidden in products traditionally seen as healthy, such as milk products. In the present study, the main sources of sucrose were also healthy food items, such as milk products, fruits, and berries.

Despite concerns about the adverse effect of sugar on obesity (Ruxton et al. 1999), as sugars provided an increasing proportion of the total food energy, the BMI tended to fall in children (Gibson 1993, Ruxton et al. 1999, Williams and Strobino 2008) as well as in adults, for a weak negative association was found between sugar and the BMI (Gibson 1996). In the present study also an age-dependent association was observed between growth and sucrose intake, because between the ages of 1 and 3 years, children with high sucrose intake weighed more than the children with average or low sucrose intake, but the opposite was seen in children older than 4. The relative gender and height-adjusted weights of the children with high sucrose intake decreased with age, but the differences in energy or total fat intake failed to explain differences in growth. Instead, sucrose was substituted with protein in the diet, because the children with low sucrose intake received more protein than the children with high sucrose intake. Differences in protein intake between children with high and low sucrose intake can be explained with the findings of Garemo and colleagues (2007a) and Kyttälä and colleagues (2008), who found that no variation existed in the energy intake between different days during week although the children had the highest sucrose intake on

Fridays or weekends, indicating that the children compensated a high sucrose intake by consuming less regular food represented by the protein content of food. According Kyttälä et al. (2008) nutrient density was higher on weekdays.

Interesting findings have been obtained in the DONALD study, where added sugar intake at the age of 2 years or changes in intakes between 2 and 7 years had no associations with BMI or percentage of body fat (Buyken et al. 2008). However, models not adjusted with other nutrients suggested an association of higher intakes of added sugar with lower BMI. This association was attributable to the fact that a lower protein intake was principally associated with lower BMI. In several studies, including the present study, higher intake of sugar has been associated with lower intake of protein in children and in adults (Rugg-Gunn et al. 1991, Garemo et al. 2006, Buyken et al. 2008, Welsh et al. 2010). In the Norwegian study by Øverby and colleagues (2004), high added sugar intake was associated with higher BMI in 4-year-old boys and a negative association was observed between consumption of added sugar and BMI among 13-year-old girls. Nevertheless, no correlations with sugar intake have been found for height, height percentile, weight, or weight percentile in children (Payne and Belton 1992) or for BMI or waist circumference in adults (Welsh et al. 2010).

The results from different studies, as well as the present study, show that a high proportion of sucrose in children's diet have had mainly an unfavourable impact on the intake of recommended foods and nutrients in children (Øverby et al. 2004, Erkkola et al. 2009). However, intakes of most nutrients appear adequate regardless of the level of sucrose intake (Gibson 1993, Erkkola et al. 2009) or the intake has been lower than recommendations only in the highest sugar intake group (Øverby et al. 2004). In some studies, nutrient intake has even risen with increasing sugar intake, which has reflected higher consumption of food in general among high sucrose intake children (Rugg-Gunn et al. 1991, Gibson 1993). Under the light of the results from this and previous studies, the negative association between health and high intake of sugars is still not conclusively established. Furthermore, more conclusive evidence is needed to confirm whether high intake of sugars has a dilution effect on many essential micronutrients and which level of decreasing nutrient density can be considered to have a significant nutritional impact on the diet. Significant differences exist in nutrient intakes between children with low or high sucrose intake, but the impact on health depends on how large the differences in the intakes are in mg (or other units). Knowing the proportions of high- and low-sugar-intake children who reach nutrient recommendations would provide indicative evidence. Food intake differences in, for example, the use of vegetables and cereal products are also relevant in evaluating whether high sugar intake is harmful. Also the way that sugar is used is meaningful; in beverages and in sweets or as sweeteners of milk products or in breakfast cereals (Ruxton 2003). These products, while representing a significant source of sugar, contain a wide range of micronutrients, which makes evaluating the nutrition dilution effect more complicated. However, the present study confirms not only the nutrition dilution effect and changes in diet habits caused by high intake of sugar, but also the adverse effects on children's growth.

6.3.3 Fibre

In this study, dietary fibre was defined as non-digestible carbohydrates in cereals, fruits, berries, and vegetables. While some studies have relied on a wider definition of fibre, comparing the results from this study as it comes to fibre with other studies is somewhat limited. The current definition in the U.S. by the Institute of Medicine (2005) suggests that the term total fibre is the sum of dietary fibre and functional fibre. In this study, we were not able to investigate functional fibre (*i.e.*, added fibre to foods). Comparing fibre intake studies and recommendations on fibre are complicated because the declared fibre contents of some foods differ between countries according to the analytic methods used, and there is lack of universally agreed definitions according to a commentary by the ESPGHAN Committee on nutrition (Aggett et al. 2003).

Payne and Belton (1992) found no evidence to suggest that a high fibre intake would reduce energy intake. Instead, their results indicated that children with high energy intakes tend to have higher fibre intakes (Payne and Belton 1992, Sepp et al. 2001, Kranz et al. 2005a), which was found also in the present study and even when adjusted for fat intake. The present study also confirmed that fibre intake has no association with weight or height. It is of utmost importance to note that in the STRIP study children, a high fibre intake did not disturb children's growth. In this study, fibre intake did not displace energy in the age range 13 months-9 years or disturb growth in the age range 8 months-9 years. Association between dietary fibre and growth were studied from many perspectives, separately in young children 8 months-2 years, in longitudinal analyses in children 1-9 years in different fibre intake groups and in different weight groups. However, earlier data on association between poor growth and dietary fibre intake were unconvincing and reported in children with an exceptionally high dietary fibre intake, such as in vegans (Sanders 1988) and in children on a macrobiotic diet (Dagnelie et al. 1994).

In adults, high-fibre diets may protect against obesity, because fibre consumption has been inversely associated with weight gain (Ludwig et al. 1999) and fibre intake correlates inversely with BMI (Nicklas et al. 2000, Kranz et al. 2005a). Fibre consumption has been associated with a reduced risk of overweight also in children, but this assumption was not supported in this study.

In this research the children with high dietary fibre intake consumed foods that were more nutrient dense and thus, had better dietary quality and higher intake of vitamins and minerals than the children with low or average fibre intake. Similar study results have also been found in other studies (Hampl et al. 1998, Nicklas et al. 2000).

6.4 Carbohydrates and serum lipids

In line with previous studies on children, in the present study the total carbohydrate intake associated inversely with HDL cholesterol concentration (Mahley et al. 2001, Nicklas et al. 2002, Slyper et al. 2005) and positively with triglyceride concentrations (Morrison et al. 1980, Perry et al.1997). However, the carbohydrates effect to HDL and

triglycerides were quite small in the present study, as a one E% increase in energy intake from carbohydrates decreased HDL cholesterol by 0.006 mmol/L and increased triglyceride by 0.016 mmol/L. The latter finding is almost identical to an international study in which 8- or 9-year-old boys' serum triglyceride concentration increased by 0.01 mmol/L per one percent increase in energy from carbohydrate (West et al. 1990). In the present study sucrose intake and serum triglyceride concentrations showed moderate associations, as a one E% increase in sucrose intake increased triglyceride concentrations by 0.01 mmol/L. This has also been shown previously in adults (Parks and Hellerstein 2000, Welsh et al. 2010) and in children (Morrison et al. 1980). An interesting finding in the present study was that an increase in fructose intake was associated with a decrease in HDL/total cholesterol ratio and an increase in apoB concentrations. In a previous study in 6- to 14-year-old Swiss children there was no association between fructose consumption and HDL cholesterol, total cholesterol or triglycerides, but one significant result was found: a higher fructose intake was associated with smaller LDL particles (Aeberli et al. 2007). However, in that study the median fructose consumption was only 2 g/d (range 0.1-12.3g/d), whereas the median fructose consumption in the present study was a magnitude higher, the median being 13 g/d (range 1.6-70.4 g/d). In an American study in children and adolescents, inverse correlations existed between HDL cholesterol and fructose (Slyper et al. 2005) and high amounts of dietary fructose may contribute to the development of dyslipidemias in adults (Reiser et al. 1989, Bantle et al. 2000, Stanhope and Havel, 2008a). In the present study, all results concerning the association between carbohydrate and serum lipids were adjusted for SFA, MUFA, and PUFA to exclude the strong connection between carbohydrates and fat in diet. However, the mechanism through which the "dysmetabolic" effects of carbohydrates occur is not completely understood (Johnson et al. 2009, Welsh et al. 2010). Possible explanation can be that these effects could be mediated by fructose, which has been shown to increase de novo lipogenesis in liver, hepatic triglyceride synthesis, and secretion of VLDL (Parks 2001, Welsh et al. 2010).

Furthermore, in this study dietary fibre intake associated inversely with serum total and non-HDL cholesterol concentration in longitudinal analyses in children from 13 months to 9 years. In the STRIP study, part of the cholesterol lowering intervention effect might be explained by changes in dietary fibre intake, as fibre intake associated inversely with serum cholesterol concentration even when adjusted for saturated fat intake. The beneficial role of dietary fibre is also supported by different effects of the STRIP intervention on cholesterol levels in boys and girls: girls in the intervention and control groups had similar cholesterol values whereas intervention boys had markedly lower cholesterol values than control boys. In both genders saturated fat intake was similarly lower in intervention groups (Niinikoski et al. 2007), but dietary fibre intake differences between intervention and control children were clearly greater in boys than in girls. Additionally, in the present study dietary fibre lowed serum cholesterol concentration more effectively when saturated fatty acid intake is low.

Besides total cholesterol, dietary fibre seems to reduce serum LDL cholesterol concentration (Williams 1995, Brown et al. 1999, Aller et al. 2004, Wu et al. 2007). In this study dietary fibre did not decrease LDL cholesterol statistically significantly. One

possible explanation for this is that LDL cholesterol values were measured only at three age points, whereas total cholesterol was measured seven times. Thus, the statistical power for LDL cholesterol was much lower than for total cholesterol. Non-HDL cholesterol, often used to substitute LDL cholesterol as an atherogenic lipid variable and which was measured at seven age points, showed a significant inverse association with dietary fibre intake. Dietary fibre as g/d was not associated with HDL cholesterol, but dietary fibre as g/MJ associated inversely with HDL cholesterol, as has been previously reported also by Marckmann et al. (1994) and Sandström et al. (1992). However, when adjusted with saturated fatty acid intake, dietary fibre as g/MJ had no effect on HDL cholesterol. Quantitatively, approximately two thirds of the decreasing effect of dietary fibre (as g/MJ) on total cholesterol comes from the effect on LDL cholesterol and one third comes from HDL cholesterol. Thus the overall effect of increasing dietary fibre intake on cholesterol metabolism seems to be advantageous. In line with previous studies, this study showed no effect of dietary fibre intake on serum triglyceride concentrations (Brown et al. 1999).

These lipid changes provide additional support for the dietary counselling of low saturated fat diets, which is usually high in dietary fibre and, therefore, also high in micronutrients. It is convenient to recommend an increase on sources of dietary fibre that include cereal products, fruits, berries, and vegetables, which are all rich sources of vitamins and minerals but contain low amounts of fat and sugar. This is supported by the findings by Turley and colleagues (1998) in adults in a study on the effect of low fat, high carbohydrate diet on serum lipids. They showed that when saturated fat was replaced with carbohydrate from cereal products, vegetables, legumes, and fruit, it reduced total and LDL cholesterol with only a minor effect on HDL cholesterol and triglycerides.

This study indicated that a high total carbohydrate intake associates with decreased HDL cholesterol values in children with apoE3 or apoE4, but not in children with apoE2. Further, adults with the E2 allele showed the greatest responsiveness to a dietary change involving increased dietary fibre intake that decreased serum cholesterol levels (Jenkins et al. 1993a), or a greater triglyceride response to high sucrose intakes than adults with the E3 or E4 allele (Erkkilä et al. 2001). There are also studies where no interactions between the diet and the apoE genotype group on serum lipids were found (Marshall et al. 1996, Wu et al. 2007). However, despite a number of intervention and association studies, there is controversy as to whether the association of apoE phenotypes with serum lipids is related to dietary factors (Marshall et al. 1996, Masson et al. 2003, Wu et al. 2007). Inconsistencies in results have been attributed to differences in the study design and small sample sizes leading to lack of statistical power, particularly in the E4 groups (Masson et al. 2003, Masson et al. 2005). In this study, the clinical significance of the association between apoE phenotype and HDL-cholesterol response to dietary carbohydrate is probably modest, because the apoE2 carriers who showed no such association were only a small group. The vast majority of Finnish children are E3 or E4 carriers, who in the present study expressed the association. Thus, dietary interventions aiming at avoiding excessive intake of carbohydrate could well be similar for children with different apoE phenotypes.

The mechanism underlying the differential impact of the apoE polymorphism on lipoprotein response to specific dietary interventions is not known (Couture et al. 2003). However, ApoE is found in many lipoproteins, and it has several functions (Mahley 1988). It is also a component of HDL_2 and HDL_3 lipoproteins, which may explain why apoE is related to HDL cholesterol concentrations in general and why HDL cholesterol response to dietary carbohydrate intake varies across apoE phenotypes. After all, this result is compatible with the idea that gene - environment interaction factors play a role in the regulation of HDL metabolism in children.

6.5 Sugar consumption and dental health

It is important to analyse dental health in children counselled to consume a low fat diet. The accepted role of sucrose intake as a factor affecting dental health has varied over time, but the common understanding is stated in the WHO report, Recommendations for preventing chronic diseases (WHO 2003): "Sugars are undoubtedly the most important dietary factor in the development of dental caries". However, this association is stronger in developing countries where association between dental caries and sugar is still more distinct than in industrialised countries, where several studies have shown that no relationship exists between sugar consumption and caries (Woodward and Walker 1994, Ruxton et al. 1999, Llena and Forner 2008). However, in the present study children with extremely high sucrose intake had, on average, twice as many teeth with past or present caries compared with children with extremely low sucrose intake. This finding was as expected, but the differences were more marked than had been assumed. There were no differences in tooth-brushing habits or in the use of xylitol between the two groups of children. Since these caries preventive methods were used evenly in both groups, sucrose consumption remains one of the most likely reasons for the different level of dental health in the two groups of children. Rugg-Gunn (1984) has previously shown a difference in caries increment between 12-year-old subjects with two sucrose-intake extreme groups. The present results are consistent with those of Rugg-Gunn (1984), showing that the caries risk is increased in children with high sucrose consumption.

Several studies have indicated that caries experience increases as the frequency of consumption of sugar increases (Kleemola-Kujala and Räsänen 1982, Gibson and Williams 1999, Llena and Forner 2008). However, the present results in children with extreme amounts of sucrose intake showed no clear differences in the intake frequency between the groups and no association with dental health. A weak correlation between the intake frequency and caries experience was found only in the high sucrose intake group. Further, the report by WHO (2003) states that the importance of frequency versus the total amount of sugars is difficult to evaluate as the two variables are hard to distinguish from each other. The report also emphasises that cross-sectional studies should be interpreted with caution because dental caries develop over time and therefore, simultaneous measurements of disease levels and diet may not give a true reflection of the role of diet in the development of the dental disease (WHO 2003). In this study food consumption was prospectively recorded from infancy to 9 years of age before dental examination.

The dental caries results in this study were in line with other studies of the same area (province of Turku); in a study by Mattila and co-workers (2008) on 10 year old children 45% were caries free and 40% had dmft+DMFT 1-4 and 15% had dmft+DMFT ≥ 5. In the children with extremely high sucrose intake 27% of the children were caries free, poor dental health (dmft+DMFT ≥ 5) was found in 36% of the children, while in the children with extremely low sucrose intake 45% of the children were caries free and 18% had poor dental health.

In a Swedish study (Grindefjord et al. 1996) risk for caries development increased longitudinally in children from 1 to 3.5 years of age, and the time period of exposure to unfavourable dietary habits was shown to have an impact on caries progression. In a Finnish study, at the age of 1.5 years 42% of the children had sugar added to their meals and 60% of 3-year-olds ate sweets more often than once a week (Paunio et al. 1993). Poor dental health at the age of 10 years was associated with frequent consumption of sweets at the age of 3 years (Mattila et al 2005). In the present study, in the extremely high sucrose intake group the mean sucrose intake of the children, already from 2 years of age, always exceeded the maximum recommended amount of 10 E% (Nordic Council of Ministers 2004, WHO 2003). It is well accepted that dietary habits developed during childhood can carry into adulthood. Therefore, physiological risk factors (such as high cholesterol levels, obesity etc.) for CVD are likely tend to track from childhood to adulthood and high intakes of sucrose or low intakes of fibre can influence future risk of chronic diseases, including CVD, type 2 diabetes, dental diseases, etc. Therefore, it is important to adopt good dietary habits already in childhood.

7 CONCLUSIONS

This study was part of a long-term CHD prevention trial introducing a low-saturated-fat diet in early childhood. The current study shows a 9-year follow-up result on carbohydrates, dietary fibre and sucrose consumption within a cohort of children in the city of Turku and surroundings. The main aims were to examine carbohydrate, sucrose, and dietary fibre intakes, as well as the associations between carbohydrates and other nutrient intakes, serum lipids, growth, and dental health. This study showed that total carbohydrate, sucrose, and starch intake had relatively small and diverse effects on serum lipid and lipoprotein concentrations in children. However, dietary fibre intake was significantly inversely associated with serum total and non-HDL cholesterol concentration in longitudinal analyses. The children from 13 months to 9 years with low sucrose intake or with high dietary fibre intake consumed foods that were more nutrient dense and thus had better dietary quality than the children with high or average sucrose or low fibre intake. Specifically:

- when carbohydrate and sucrose intakes meet the current recommendations, marked decreases in total or HDL cholesterol concentrations are unlikely to occur. However, total carbohydrate and sucrose intakes and serum triglyceride concentration showed moderate associations, as an increase in sucrose intake increased triglyceride concentration.
- children with the apoE3 or E4 phenotype but not those with E2 phenotype showed reduction in HDL cholesterol with increasing carbohydrate intake indicating that genetic and environmental factors interact with children's lipoprotein metabolism. However, the impact of apoE polymorphism was small and does not imply exclusion of children with any apoE subgroup from dietary counselling.
- serum total and non-HDL cholesterol values correlated inversely with dietary fibre intake, indicating that part of the cholesterol-lowering intervention effect in the STRIP project may be explained by dietary fibre.
- high fibre intake did not displace energy in children aged from 13 months to 9 years or disturb growth in children from 8 months to 9 years. Further, fibre consumption was not associated with a reduced risk of excess weight gain. Children with high fibre intake had higher intake of vitamins and minerals than children with low or average fibre intake.
- children with low sucrose intake had higher intake of protein, vitamins and minerals, and their fat quality was better than in children with high or average sucrose intake. Moreover, children with low sucrose intake grew slightly but significantly faster than those with higher sucrose intake. However, it is necessary to conduct larger studies to find out the role of sucrose in children's growth.
- restriction of children's sugar consumption still has a role to play in the prevention of caries, because this study shows that the habit of excessive daily sucrose intake starts early in childhood and a persistently high sucrose intake increases the risk of dental caries in children.

8 CLINICAL IMPLICATIONS

The quality of carbohydrates is important in children's nutrition and special emphasis should be paid on intake of dietary fibre and that of sugar. According to the present study, a high intake of dietary fibre does not adversely effect children's growth or displace energy in their diet. High dietary fibre intake is recommended, as it associated with increases in the intakes of vitamins and minerals and with lower serum cholesterol concentrations, which decreases the risk for CVD in later life. High dietary fibre intake also associates with lower intake of fat, SFA, and sucrose. Consequently, the use of cereal products, vegetables, fruits, and berries is recommended for children. It should be taken into consideration in nutrition counselling that low fibre intake associates with high consumption of sweets, sugar and ready-to-eat baby foods.

However, these results do not justify adding a specific dietary fibre intake recommendation for Finnish children. The children in this study were possibly not entirely representative of the whole Finnish population. The study did not address fibre intake in the prevention or treatment of constipation. Moreover, possible mineral imbalance was outside of the scope of the study. However, it is believed that mineral deficiencies are unlikely to develop in children on typical Western diets. It should also be decided, which unit to use (g/kg, g/MJ, or g/d) for children's fibre intake recommendation.

Decreasing sugar intake in children's nutrition is recommended, because a persistently high sucrose intake increases the risk of dental caries in children. Dental hygiene is also important to prevent caries. This study showed that children's tooth brushing habits are clearly insufficient as only 33% of children brushed their teeth twice a day as recommended. High sugar intake might also have adverse effects on growth. Children with low sugar intake get more protein, vitamins and minerals and their dietary fat quality was better than in children with average or high sugar intake. Accordingly, excessive consumption of products containing high amounts of sugar (e.g. sweets, soft drinks, and biscuits and other pastries) should be avoided in children of all ages. In the present study sweetened milk products were the main sucrose source in children aged 3 years and older, and also home-made or commercially available juice concentrates were remarkable sources of sucrose. Thus, children should use water to quench thirst and skimmed milk with meals. Limiting the use of sugar must be started in early childhood. Sucrose consumption is high already in 2-year-old toddlers and it increases with age.

In conclusion, food habits are established early in life, and new intervention strategies towards favourable dietary habits should be identified. It is important to introduce a nutritionally high quality diet with adequate amounts of dietary fibre, without excessive amounts of sucrose, to support normal growth and development, and to help maintain optimal health and prevent future cardiovascular diseases. Parents are mostly responsible for their child's diet. Therefore, well-baby clinics should support parents by providing nutrition education and counselling for children and the entire family. Nutrition education should also be included in school health care, and teaching and cooperation between school health care staff and parents is recommended.

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In Paimio, January 2011

Soile Ruottinen

"Knowing is not enough; we must apply.

Willing is not enough; we must do."

—Goethe—

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