DIETARY INTAKE OF NON-MILK EXTRINSIC SUGARS AND CARIES EXPERIENCE OF 12-13 YEAR OLD SCHOOLCHILDREN

A thesis submitted to the University of Manchester for the degree of
Master of Philosophy
in the Faculty of Medical and Human Sciences

2011

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Abstract

The emergence of clinical care pathways within dentistry has placed renewed emphasis on the caries risk assessment of patients to help predict further disease and guide future prevention and intervention strategies. With dietary intake of non-milk extrinsic sugars (NMES) a key aetiological factor in the development of caries, the predictive level of risk posed by some dietary habits is questioned. Therefore the aim of this study was to investigate the influence of dietary habits on the prevalence of dental caries with adolescent schoolchildren. Dietary risk factors concerning the frequency and timing of NMES consumption including snacking between meals and bedtime consumption were investigated to determine if any factors could be utilized to predict caries risk.

128 subjects, 12-13 years of age, were randomly selected from an observational epidemiological survey for which the caries status and level of material deprivation was available. Caries experience was recorded with the variable DMFT, where the caries threshold was defined as code 4 (dentine involvement) with the International Caries Detection and Assessment System (ICDAS). Subjects underwent a 24 hour dietary recall interview with a dietician focusing on NMES intake; an estimation of which was based on McCance and Widdowson’s food composition tables. Logistic regression and odds ratio estimation was used to determine if any dietary habits predicted caries experience. The level of statistical significance was set at p=0.05.

54 (42%) subjects were found to have caries (ICDAS>4). Chi-square analysis for subject groups with/without caries revealed no significant differences in caries experience between high and low deprivation, consumption of NMES between meals and percentage of total NMES consumed. The consumption of NMES before bedtime revealed a statistically significant difference between the caries/no caries groups (p<0.005). Logistic regression analysis on the caries/no caries groups revealed a predictive odds ratio of 2.26 (1.232, 4.146) for NMES consumption before bedtime that correctly predicted 65% of caries experience (when age of exam included as a variable). However, a small effect size was noted possibly owing to the relatively low caries experience overall and small sample size.

This study suggests that the consumption of NMES before bedtime may be an important predictive risk factor for adolescent caries experience. Further work is necessary to validate the findings of this study.
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Acknowledgements

I would like to express my gratitude to:

• The members of the Dental Health Unit (The University of Manchester), and in particular to: Dr Iain Pretty for his guidance and mentorship throughout my period of study; Mr Mike McGrady for his advice and assistance with the clinical data collection and analysis; Michaela Goodwin for her statistical support and encouragement; and Mrs Nicola Boothman for her administrative support as project manager.

• Mrs Avni Vyas (Clinical Epidemiology Group, University of Manchester) for her assistance with the dietary data collection and analysis.
Chapter 1
1 Literature Review

1.1 Introduction

Dental caries is a disease that is ubiquitous among the world’s population. If left untreated, it can lead to dental pain, infection and eventual loss of a tooth. Although it is debatable whether the process is truly preventable due to the natural activity within dental plaque biofilms, progression of carious lesions can be controlled through various methods (Kidd, 2005). There is much evidence for the role of dietary carbohydrates in the development of caries, and consequently the importance of good dietary habits has been promoted not only by dental professionals but on a wider scale through government public health initiatives and policy. The emergence of clinical care pathways within dentistry has re-emphasised the importance of diet in the overall assessment of oral health and prevention of further disease.

1.2 Dental Caries

1.2.1 Pathogenesis

Dental caries has been defined as the localised destruction of susceptible dental hard tissues by acidic by-products from bacterial fermentation of dietary carbohydrates (Marsh and Martin, 1999). Tooth structure is lost through a process of demineralisation where calcium and phosphate ions diffuse out of enamel in response to low pH levels. The threshold at which this occurs is pH 5.5, commonly referred to as the critical pH. When the pH rises however, this process has the ability to be reversed and in its earliest stages a carious lesion can be arrested and become inactive. Saliva plays an important role in remineralisation due to its supersaturated content of calcium and phosphate at pH 7; additionally fluoride has been shown to enhance remineralisation (Levine, 1991). Dental caries can therefore be considered as a dynamic process of episodic demineralisation and remineralisation as shown in Figure 1-1 (Kidd, 2005). The process of remineralisation is slow however and if conditions favour demineralisation, progression of initial lesions into dentine with bacterial invasion is likely, potentially resulting in pulpal necrosis and subsequent spread of infection into the periapical tissues causing pain (Kidd, 2005).
1.2.2 Aetiology

Caries has a multifactorial aetiology with numerous risk factors implicated in its development, as illustrated in Figure 1-2 (Selwitz et al., 2007). Most variables that have been investigated relate to the classic model of host, microflora and diet. Behavioural/social factors as shown in the outer circle have been found to have profound effect on the risk of developing caries (Fejerskov and Kidd 2003). Additionally, oral environment factors as shown within the inner circle have a major impact on the core factors needed for the initiation/progression of caries.
The multifactorial aetiology of the disease makes it very difficult to accurately predict future caries development. Conditions on which any predictive factor is based would have to remain stable to hold any reliability. Since many of the risk factors implicated in caries development may change over time such as living conditions and oral health behaviours, a person’s risk of developing caries may change in either direction.

In studies comparing multiple predictors, past caries experience is usually the single most powerful predictor of future caries increment (Honkala et al., 1984, Klock and Krasse, 1979, Raitio et al., 1996, Wilson and Ashley, 1989). This is unfortunate in that the disease needs to
inflict its damage before an individual may be identified as high risk. Since past caries experience summarises the cumulative effect of all risk factors, known and unknown, its power in predicting future caries increment is not too surprising.

Studies have explored the effect of combining risk factors through multivariate analysis to determine if this would lead to more accurate prediction. This seems logical since with a multifactorial disease such as dental caries, increasing the number of risk factors under investigation should increase the predictive power of the model. Seppa et al., (1991) combined information from six available predictors: baseline DMFS (as a measure of past caries experience), mutans streptococci score, salivary flow rate, sucrose intake frequency score, tooth-brushing frequency score and social group, to see if in combination the factors would provide a more accurate prediction of approximal 2 years caries increment than considering the factors individually. The study was conducted in Finland with a cohort of 350 adolescents followed longitudinally over 2 years. A logistic regression model was created with the factors as independent variables. The results showed practically all of the predictive power of the model came from the baseline DMFS score and the remaining five predictors did not add anything to the accuracy of the prediction, even when regarded together. This study highlighted the value of past caries experience as a principal predictive risk factor of caries over other core risk factors.

Another notable longitudinal study involving multivariate analysis of caries risk factors was that carried out by North Carolina University (Disney et al., 1992). This comprehensive 3 year longitudinal study involved 4000 adolescents over four different sites, and explored multiple clinical, microbiological and socio-demographic risk factors for caries risk prediction. The results revealed microbiological and socio-demographic factors contributed little to caries risk prediction over the 3 year follow up period. Strong predictors of caries risk were: baseline DMFS scores, pit and fissure morphology and predicted caries risk status (by the examiner at baseline of the future 3 year caries increment). Due to variability seen in individual subjects and different population groups within the study, the authors felt a single, all inclusive caries risk assessment model was an unrealistic goal and recommended instead multiple models focused on readily available clinical factors for different age groups, populations and disease levels.
Results of these studies support the importance of past caries experience as a principal predictive risk factor, although the predictive value of other risk factors in combination with this seem to vary with different populations and disease levels.

1.2.3 The Role of Dental Plaque and Bacteria

The plaque biofilm plays an important role in the caries process, allowing microorganisms a protected environment to develop in close approximation to the tooth surface. A layer of glycoprotein precipitated from saliva, called the plaque pellicle, quickly forms on clean enamel surface and provides a tenacious surface for the anchorage and further attraction of specific types of bacteria. Initially the flora consists of bacteria cocci in form, a large proportion of which are streptococci, but with maturation within a few days a mixed flora develops consisting of cocci, rods and filaments (Kidd and Joyston-Bechal, 1997). Hence plaque is a host to a complex micro-system of micro-organisms resulting in the accumulation of bacterial biofilms, particularly in stagnant sites around the dentition.

The presence of bacteria has been shown to be essential to the development of carious lesions. Orland et al. (1954) demonstrated that caries did not develop in rats reared in germ-free conditions when fed a cariogenic diet. In contrast, caries developed in rats that were fed the same diet but allowed to develop the usual mixed microbial flora. Further experiments by Fitzgerald and Keyes (1960) demonstrated rats would develop caries when infected with specific strains of streptococci and that caries was potentially infectious and transmissible (Keyes, 1960). The use of gnotobiotic rats has also shown mutans streptococci and some strains of lactobacilli and actinomyces were of particular relevance to the development of caries. The artificial nature of the above experiments where animals were inoculated with only one strain of bacteria is in contrast to the human oral environment where multiple strains of bacteria co-exist and compete. Despite these limitations, the body of evidence from various experiments and observations supports dental caries as a specific infection with mutans streptococci (in particular S. mutans and S. sobrinus) as the main pathogens (Loesche, 1986).

Cariogenic bacteria such as mutans streptococci and lactobacilli possess several caries-inducing properties. These characteristics have been outlined by Loesche (1986) and include: the ability to rapidly produce acid from fermentable carbohydrates (acidogenic), thrive under
acid conditions, and the ability to adhere to tooth surfaces aided by the production of extracellular polysaccharides (EPS). EPS consists of glucans and fructans which provide a gelatinous consistency to the biofilm matrix, thereby aiding adherence of bacteria and thickening the plaque layer. The neutralising action of saliva on plaque acid at the tooth surface is thus reduced.

1.2.4 Plaque Acid and the Stephan Curve

The characteristic form of the pH response of plaque after exposure to sugar is illustrated by the Stephan Curve shown in Figure 1-3 (Fejerskov and Kidd, 2008). After a sucrose challenge, there is a rapid drop in plaque pH with the minimum pH occurring within 5-10 minutes. The drop in pH below the critical pH of 5.5 is sufficient for the demineralisation of tooth structure from the loss of calcium and phosphate ions. The gradual return to resting plaque pH values is due to the important role of saliva in providing clearance of the carbohydrate challenge and its alkalinity and buffering power to restore plaque pH towards neutrality. However, it can be seen that repeated and frequent consumption of sugar will depress plaque pH to levels below which demineralisation readily occurs. Stephan curves can be used to quantify the potential cariogenic challenge (i.e. cariogenicity) of a food-stuff by measuring the area delimited by the critical pH and the Stephan curve, as indicated in Figure 1-3.

Figure 1-3: The Stephan curve, demonstrating the effect an oral sucrose rinse has on the plaque pH, from Fejerskov and Kidd (2008).
1.3 Cariogenicity of carbohydrates

1.3.1 The Evidence

There is overwhelming evidence supporting the link between the frequent consumption of fermentable carbohydrates and the prevalence of dental caries. The evidence comes from a variety of sources each with their own advantages and disadvantages (Table 1-1). There is a variation in the quality of these studies and limitations within individual study designs should be borne in mind when interpreting the results. However the strength of the evidence for the cariogenicity of carbohydrates comes from the multiplicity of the studies rather than the power of any individual study (Arens, 1998).

<table>
<thead>
<tr>
<th>STUDY DESIGN</th>
<th>DESCRIPTION</th>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Observational</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(epidemiological) studies</td>
<td>Relationships between disease and possible causative and confounding factors are observed.</td>
<td>Relatively quick data collection and analysis.</td>
<td>Confounding factors may mask the true effect.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Greater access to larger, more diverse populations.</td>
<td></td>
</tr>
<tr>
<td><strong>Interventional studies</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical trials</td>
<td>Diets intentionally altered and effects of this observed.</td>
<td>Clinical trials provide the most reliable form of evidence.</td>
<td>Difficult to carry out; large groups needed to adhere to strict diets for a long time to allow the development of caries to be observed. Ethical considerations.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Animal studies</strong></td>
<td>Dietary intake of animals (commonly rats) strictly controlled under experimental conditions.</td>
<td>Experimental time period short.</td>
<td>Difficulty extrapolating findings to humans.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Conditions can be controlled.</td>
<td></td>
</tr>
<tr>
<td><strong>Enamel slab experiments</strong></td>
<td>Observe the effect of diet on the demineralisation of slabs of enamel which are held in the mouth of human volunteers in a removable plate.</td>
<td>In-vivo: closer to the human situation.</td>
<td>Removable appliance that may be removed by volunteers. Site specific.</td>
</tr>
</tbody>
</table>
The best form of evidence comes from human clinical trials although ethical considerations can limit the magnitude of interventions. Many studies of human populations have adopted a cross-sectional study design with obvious benefits to time and cost. However due to the chronic nature of the caries process a snapshot view such as this will not take into account dietary changes over the years which may be quite significant for some patient groups such as adolescents and adults. Longitudinal studies would be more suitable to explore the relationship of caries and diet over time, but organisational and cost implications limit their widespread use. The multifactorial nature of the pathogenesis of caries can present problems with confounding variables; an obvious example is fluoride status. Confounding variables need to be taken into account to prevent incorrect assumptions of the role of one variable over another.

A large proportion of the evidence linking caries and diet has been derived from epidemiological, intervention, animal and plaque pH studies.

### 1.3.1.1 Epidemiological human studies

There have been many human observational studies that have looked at dietary habits of groups of people over the years and the caries prevalence. Historically, diet was based on the available food sources of raw grains, roots, berries and herbs with almost complete absence of sugar and processed starch that are normally associated with modern day diets. Although archaeological evidence reveals caries did occur in ancient humans, the prevalence was low. This is in contrast to the steady increase in the consumption of sugars over the last 150 years.
with the mass production and refinement of sugar cane, and the corresponding rise in caries prevalence, as illustrated in Figure 1-4 (Rugg-Gunn and Nunn, 1999). Epidemiological studies based on more contemporary isolated populations have also demonstrated increased caries prevalence when traditional non-cariogenic diets have been altered in favour of more modern diets with refined carbohydrates and sugars. An example of this is the remote South Atlantic island inhabitants of Tristan da Cuhna who until the 1930’s had an excellent dental state, with a diet based comprised of potatoes and other vegetables, meat and fish. However, with an increase in imported sugary foods in the 1940’s there had been a striking increase in caries experience (Fisher, 1968).

Figure 1-4: Percentage of dental caries prevalence related to mean sugar consumption in British populations from Iron Age to modern times, from Rugg-Gunn and Nunn (1999), source Corbett and Moore (1976).

Evidence of a proportional decline in caries prevalence has also been seen with reductions in the availability of sugar such as during the World Wars, as illustrated in Figure 1-5 (Rugg-Gunn and Nunn, 1999). The caries prevalence level can be seen to rise after the war as consumption of sugar returned to pre-war levels. A longitudinal study of children 6-13 years of age living in an Australian children’s home further supports these observations of the
relationship between sugar consumption and caries prevalence. At the home, the diet was mainly lacto-vegetarian with minimal amounts of sugar and refined flour. There was a low caries prevalence compared with the control group. However, on leaving the home, caries levels soon rose to the same level as the general population (Harris, 1963).

Subjects with the rare hereditary disease fructose intolerance (HFI) have also been studied to investigate the link between diet and caries. Ingestion of fructose or sucrose in this patient group would cause nausea due to the absence of a liver enzyme, hence avoidance of sugary foods. Consequently, these individuals have been found to have low caries levels (Marthaler and Froesch, 1967, Newbrun et al., 1980). Newbrun et al., (1980) also observed the higher consumption of starch within the HFI group not appear to be conducive to caries development.

**Figure 1-5: UK sugar consumption over the last 200 years (Rugg-Gunn and Nunn, 1999).**
1.3.1.2 Interventional human clinical studies

The classic caries study at Sweden’s Vipeholm hospital for the mentally handicapped was carried out between 1946 – 1951 and is one of the few studies where the intentional increase in dietary sugar and the relationship between a variety of sugar intakes and caries increment was recorded (Gustafsson et al., 1954). Modern day ethical committees would not approve such a study to alter diets in a direction likely to increase caries levels, however at the time the study commenced the link between caries and diet had not been fully made. Patients were divided into one control and six experimental groups. For one year the patients received a diet low in sugar, with no sugar between meals. The number of new carious lesions was assessed and it was found to be low. The effect of altering the diet with large sucrose supplements in sticky and non-sticky form, either with or between meals was assessed. The main findings are shown in Figure 1-6. The control group who continued the basic low sugar diet show little increase in caries. There was a marked increase in the caries activity in all groups, except when sugar was taken at mealtimes. The greatest caries activity was associated with those who took sugar between meals in the sticky form of toffees. In fact the caries activity was so great that the 24-toffee group had their supplement withdrawn during the study, which interestingly saw a fall in the caries increment. Despite the questionable ethics and the use of a population that is not representable of modern society, the study yielded important data associating a high caries risk with frequent sugar consumption, particularly in a form that adheres to teeth.
Figure 1-6: The Vipeholm study results showing the DMFT per person in relation to the type and time of eating various sugar-containing products (Gustafsson et al., 1954).

Another important human interventional study was the longitudinal Turku sugar study, which involved the comparison of xylitol, a sugar substitute which is not metabolised acid by plaque bacteria, with sucrose and fructose (Scheinin et al., 1975). The consumption of starch was noted to be high and similar in all three groups. The results showed significant reduction in caries incidence in the xylitol group, indicating the importance of removal of sugar from the diet. This helped identify the benefit of sugar substitution by substances which impart sweetness but lack cariogenicity.
1.3.1.3 Animal experiments

Rats have commonly been used for caries experiments over the years. The necessity for oral contact of food in the development of caries was demonstrated by Kite et al., (1950), who found rats fed a cariogenic diet through a stomach tube developed no caries, whilst the control group which was orally fed developed caries. In the same series of experiments Kite also investigated the effect of removing the major salivary glands on the development of caries and found desalivated rats had more caries, thus demonstrating the important role of saliva in caries prevention. A landmark study by Orland et al., (1954) used germ free rats to prove that cariogenic bacteria are essential to the development of dental caries. Rat experiments by Konig et al., (1968) have also linked high caries rates with frequency of intake of sugar, whilst total sugar consumption was kept the same.

The cariogenicity of starch has also been researched using rat experiments; however the results have provided wide variations. The differences in cariogenicity lie with the level of processing and are discussed in further detail below (section 1.3.2.4).

1.3.1.4 Plaque pH studies

The use of in-dwelling electrodes capable of measuring dental plaque pH was pioneered by Stephan in the 1940’s as a means of assessing the cariogenic challenge posed by various dietary foods (Stephan, 1940). The acidogenic potential of a range of carbohydrates has been demonstrated, with sucrose, glucose, fructose and maltose of a similar acidogenicity, while lactose and galactose are less acidogenic (Kidd and Joyston-Bechal, 1997). It should be remembered however that plaque pH experiments investigate acidogenicity and not cariogenicity, and do not take into account any protective factors present in food or the oral environment, notably salivary flow. Hypersensitivity of the in-dwelling electrodes causing an ‘all or nothing’ response to all carbohydrates are a known limitation of this study method (Edgar, 1985).
1.3.2 Dietary Carbohydrates

Miller’s pioneering work in the 1880’s showed that carbohydrate foods, when incubated with saliva, caused demineralisation of teeth in vitro (Miller, 1883). However, carbohydrates can vary considerably in composition and are not all equally cariogenic. Classification of dietary carbohydrates and their varying cariogenicity is described below.

1.3.2.1 Classification

Carbohydrates can be classified in a variety of ways (Moynihan, 1998). Based on their chain length, they can be split into simple sugars (mono- and di-saccharides) or complex sugars (oligosaccharides, polysaccharides). Alternatively, they can be referred to by food source, for example fruit sugars or milk sugars. In an effort to reduce confusion amongst the general public and health professionals, the UK government committee COMA (Committee on Medical Aspects of Food Policy) have recommended a revised naming system based on where the sugar molecules are located within the food or drink structure (Department of Health, 1989) (Figure 1-7). Intrinsic sugars refer to sugars found inside the cell structure of unprocessed foods, such as whole fruit and vegetables. Extrinsic sugars are divided into milk extrinsic sugars, such as lactose in dairy products, and non-milk extrinsic sugars (NMES) which as the name suggests, refers to all other ‘added sugars’ in the diet to provide sweetness. This classification has the most significance with dental health, with NMES regarded as cariogenic, whilst intrinsic sugars and milk sugars considered as having low or negligible cariogenicity (Department of Health, 1989).

Figure 1-7: The COMA classification of dietary sugars (Department of Health, 1989).
1.3.2.2 Sucrose

The term ‘sugars’ commonly refers to the monosaccharide (glucose, fructose and galactose) and the disaccharides (sucrose, maltose and lactose), while the term ‘sugar’ is used synonymously with sucrose. Simple sugars have a low molecular weight which allows for rapid diffusion into plaque and quick metabolism by homogenous fermentation by plaque bacteria. The high amounts of lactic acid that is produced lowers the pH to a level at which demineralisation of enamel takes place. Sucrose has been labelled as the most cariogenic of the sugars, and perhaps rather more emotively has been referred to as the ‘arch criminal of dental caries’ (Newbrun, 1969). This has been largely based on numerous animal experiments which have demonstrated high caries rates with sucrose diets and its unique ability to produce large quantities of extracellular glucans by mutans streptococci. Glucan acts as a virulence factor for caries by increasing the porosity of plaque, permitting deeper penetration of dietary sugars and greater acid production adjacent to the tooth surface, (Zero, 2004). Some criticism has been placed on the reliability of animal studies in which rats super-infected with streptococcus mutans showed sucrose to have the highest cariogenicity of the sugars. Since this bacterial strain is particularly good at metabolising sucrose, it was not surprising therefore that there were higher scores in the animal groups who received the sucrose diet. Nevertheless, sucrose is highly cariogenic and widely available, and plays a significant role in dental caries.

1.3.2.3 Milk sugars

Lactose is the main sugar in human and bovine milk. Animal studies have shown lactose to be less acidogenic and cariogenic than other sugars (Rugg-Gunn and Nunn, 1999). Milk is not considered to be cariogenic despite the presence of a potentially cariogenic sugar such as lactose. This is mainly due to the protective factors in milk such as the presence of casein and high levels of calcium and phosphorus which promote remineralisation. Studies conducted by Stephan (1940) have found milk to be non-cariogenic and animal studies by Bowen et al., (1991) have supported the non-cariogenicity and caries protective property of milk. Desalivated rats where shown to develop caries when fed lactose in water but developed no caries when given milk or lactose reduced milk.
1.3.2.4 Starch

Starch is a complex carbohydrate (polysaccharide) found in staple foods such as potatoes, rice and wheat. The cariogenicity of starch is considered to be low due to the prolonged process of breakdown by salivary amylase to glucose, maltose and indigestible limit dextrans. The slow rate of this heterogeneous fermentation slows down glycolysis and therefore acid production. Numerous experiments have studied the cariogenicity of starch and the results have been varied. The differences in cariogenicity lie with the level of processing. Raw starches appear to have very low cariogenicity whereas starch molecules which have been heated or put through a mechanical process such as during cooking, have an increased susceptibility to enzymatic breakdown and therefore a higher acidogenic potential (Lingstrom et al., 1993, Lingstrom et al., 2000). Enamel slab experiments have shown cooked starch to be one quarter to a half as cariogenic as sucrose (Koulourides et al., 1976).

Plaque pH studies have highlighted the ability of starch to depress plaque pH to below 5.5 (Pollard, 1995), however the potential cariogenic effect of this are not seen clinically due to the presence of protective factors. Less refined starches have protective factors that may reduce the cariogenicity such as greater oral clearance due to an increased saliva flow from the vigorous chewing of fibre rich foods and the presence of organic phosphates such as phytate which may help protect teeth from dissolution. Plaque pH experiments investigate acidogenicity and not cariogenicity and so would not take into account protective factors which decrease cariogenicity by affecting solubility and not acid production (Rugg-Gunn and Nunn, 1999). Two types of experiments are principally used in plaque pH studies: the sampling method or in-dwelling electrode. The sampling method has tended to indicate that cooked starch, or starchy foods, are less acidogenic than sugar or high sugar foods and that uncooked starches are virtually non-acidogenic (Rugg-Gunn and Nunn, 1999). In-dwelling electrode experiments however have shown starch to lower the pH to below the critical pH. A problem with indwelling glass electrodes experiments is that they tend to give an all-or-nothing response to foods leading to a maximum drop in pH. This has been criticised by Edgar (1985) who considers this experiment type to be ‘hyper-responsive’ and can pose difficulties in the evaluation of the relative cariogenicity of snack foods. The technique is mainly used in verifying the low acidogenicity of sugar substitutes.
Animal studies have shown an increased cariogenic effect when starch is consumed with increased consumption of sugars, and high intake frequency mixtures of cooked starches and sucrose have been demonstrated to be more cariogenic than sucrose alone (Firestone et al., 1982). Prolonged retention of sugars on teeth may result when combined with starches and this may detrimentally shift the plaque ecology towards a more cariogenic status. However, some caution should be taken when using the results of animal experiments as an indication of cariogenicity in humans due to inherent anatomical differences such as tooth morphology, plaque bacterial ecology, salivary flow and composition, and also the level of refinement of the food (usually powdered form in animal experiments) and fluoride exposure (Moynihan and Petersen, 2004).

The most valid estimate of the cariogenicity of starches comes from human observational and interventional studies. Historically, starchy foods have formed the staple diet of humans and consequently there has been very little caries. As previously discussed, restrictions placed on the diets of contemporary populations have supported the low cariogenicity of starches, such as wartime rationing where the increased consumption of starch has been shown to lower the caries rate temporality until the rise of sugar in post-war diets caused an increase in caries rates (Toverud, 1957). In addition, patient groups with medically restricted/absent dietary intakes of sucrose or fructose, such as those who suffer from hereditary fructose intolerance (HFI), have been shown to have low caries levels in the absence of sucrose and fructose despite high compensatory levels of starch in their diet (Newbrun et al., 1980).

1.3.2.5 Novel carbohydrates

1.3.2.5.1 Glucose polymers

In an effort by food manufacturers to control the taste or energy content of foods, glucose polymers have been increasingly added to foods. Glucose syrups and maltodextrins are collectively known as glucose polymers, and consist of short chain saccharides and alpha-limit dextrins. Although evidence of their cariogenicity is limited, animal, plaque pH and in-vitro studies suggest glucose polymers are cariogenic, although to a lesser extent than sucrose (Moynihan et al., 1996, Grenby, 1972, Grenby and Mistry, 2000).
1.3.2.5.2 Oligosaccharides

Oligosaccharides are synthetic, non-digestible medium chain carbohydrates and similar to glucose polymers their use is also increasing. Evidence from plaque pH studies and in-vitro experiments suggests isomalto-oligosaccharides and gluco-oligosaccharides are not as acidogenic as sucrose (Ooshima et al., 1988, Roberts and Hayes, 1980). The more widely available fructo-oligosaccharides however have been shown to have a similar acidogenicity to sucrose (Hartemink et al., 1995).

1.3.2.5.3 High fructose corn syrup

High fructose corn syrup (HFCS) refers to any group of corn syrups which have been subjected to an enzymatic process to convert its glucose to fructose, and then mixed with pure corn syrup (100% glucose) to achieve the desired sweetness. This is similar in composition to invert sugar (50% fructose + 50% glucose) and has a cariogenicity slightly less than sucrose due to a lack of extracellular polysaccharide production (Frostell et al., 1991).

1.3.2.6 Medical sugars

Sugars are often added to children’s medicine to improve palatability. They can be up to 70% concentration and are commonly syrup based. As with the majority of cases, medicine is taken infrequently as a short course of treatment and so would not present significant concern for the development of caries. However, in long term chronic illnesses, frequent exposure to high levels of sugar, which are sometimes taken before bedtime or during sleep when salivary flow is reduced, can dramatically increase the cariogenic potential. In response to this perceived risk, the Department of Health in England COMA report, ‘Weaning and the weaning diet’ (Department of Health, 1994b) recommended all paediatric medications should be sugar free. Maguire et al., (1996) showed a lower prevalence of caries with long term chronically ill children who had sugar medication compared to those who had sugared medication. The elderly also pose another at risk group, and tend to consume more long term medication with an increase time for oral clearance (Maguire and Baqir, 2000).
1.4 Sugar Consumption

1.4.1 Frequency

The frequency of intake of sugars has been shown to play a crucial role in the development of caries. Stephan curves of the pH response of plaque after frequent exposure to sugar throughout the day demonstrate the prolonged time at which the plaque pH can potentially remain at levels below the critical pH for demineralisation of tooth structure Figure 1-8.

Figure 1-8: A Stephan curve illustrating the effect of frequent snacking during a 24 hour period on the pH of dental plaque.

Animal studies carried out by Konig et al., (1968) have shown caries to increase with an increased the frequency of sugar intakes in rats, despite the total sugar intake remaining the same with all groups (Table 1-2). Conversely, Firestone et al., (1984) found less caries developed in rats when the interval between feeds was increased.
Table 1-2: The mean dental caries severity and daily food intake in five groups of rats fed at different frequencies per day, (Konig et al., 1968).

<table>
<thead>
<tr>
<th>Group</th>
<th>Eating frequency/day</th>
<th>No. of fissure lesions</th>
<th>Daily food intake (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>0.7</td>
<td>6.0</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>2.2</td>
<td>6.0</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>4.0</td>
<td>6.0</td>
</tr>
<tr>
<td>4</td>
<td>30</td>
<td>4.7</td>
<td>6.0</td>
</tr>
<tr>
<td>5</td>
<td>ad lib</td>
<td>4.2</td>
<td>11.7</td>
</tr>
</tbody>
</table>

The Vipeholm study, as previously mentioned, provided direct evidence of the detrimental effects of increasing the frequency of sugar intakes on a human population (Gustafsson et al., 1954). Patients who ate sugary foods between meals in addition to that provided at mealtimes had a higher caries rate than those patients who only had sugary foods at mealtimes. In fact, the group which consumed the most sugar in-between meals, the 24 toffees a day group, had such a high caries rate that the toffees were eventually withdrawn, which interestingly resulted in a fall in the caries increment.

Holbrook and co-workers have described a threshold effect for caries experience based on the frequency of sugar intakes of children in an area of high caries prevalence in Iceland. They noted a dramatic increase in the level of caries in children with more than 4 sugar intakes a day or greater than 3 sugar intakes between meals (Holbrook et al., 1995) and doubling of the dmfs scores in 4 year-old children who consumed more than 30 sugar intakes a week (greater than 4 sugar intakes per day) (Holbrook et al., 1989).

1.4.2 Bedtime Consumption

When sleeping, salivary flow is considerably reduced and so the potential for unchallenged demineralisation is increased. Levine (2001) highlighted bedtime as the worst time of the day to consume a sugary snack or drink. His research found children who consumed a sugary drink or snack within an hour before bed had four times the mean DMFT score (1.24) than children who consumed neither (0.31). The sample however was not totally representative of the general population as a whole; the majority of children were from low socioeconomic backgrounds and with a high caries experience.
1.4.3 Amount Consumed

The amount of sugar consumed has also been shown to play an important role in the development of caries, with some controversy over the greater importance of frequency of intake or amount consumed. Animal experiments in which rats were fed increasing concentrations of sugars whilst maintaining the same frequency of feeding have demonstrated an increase in the caries rate (Rugg-Gunn and Nunn, 1999). Longitudinal studies on adolescent populations have reported the amount of sugar intake to be more important than the frequency (Rugg-Gunn, 1993, Szpunar et al., 1995, Burt et al., 1988). Figure 1-9 illustrates the slightly higher correlations found between caries increments and total sugar intake as compared to the frequency of intake in a population of 12-14 year old children in the North-East of England (Rugg-Gunn, 1993).

Figure 1-9: A plot of the frequency of intake of confectionary per day against the weight consumed per day, in a population of 12-14 year old children in the North-East of England. Correlation coefficient +0.77. (Rugg-Gunn, 1993)

Due to the closely related nature of frequency and amount of consumption, it is unsurprising of the strong correlation that exists between the two variables. Dietary advice therefore has been directed at cutting down both the frequency of consumption and the amount of sugar consumed (Rugg-Gunn and Nunn, 1999).
1.4.4 At Risk Groups

Some population groups may have an increased risk of developing caries depending on the dietary intake. Factors such as age, medical health and socioeconomic status may influence dietary choices and subsequently the risk of developing caries.

1.4.4.1 Infants and toddlers

Early childhood caries (ECC) describes the development of dental caries in infants and toddlers. Night-time bottle feeding with cariogenic liquids has been implicated as an important risk for its development. A comprehensive review by Winter in 1980 highlighted the detrimental use of night-time comforters and found rampant caries was four times more likely to occur in infants with sugar comforters than those without (Winter, 1980).

1.4.4.2 Children and adolescents

Confectionary and soft drinks provide the main sources of sugar in the diets of adolescents, as shown Table 1-3, (Rugg-Gunn, 1993). The higher prevalence of caries in adolescents has been associated lifestyle factors such as increased snacking, particularly during school-time breaks, with confectionary and soft drinks (Flinck et al., 1999).

<table>
<thead>
<tr>
<th></th>
<th>Non-milk extrinsic</th>
<th>Intrinsic and milk</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>g</td>
<td>%</td>
<td>g</td>
</tr>
<tr>
<td>Confectionary</td>
<td>30</td>
<td>33</td>
<td>1</td>
</tr>
<tr>
<td>Soft Drinks</td>
<td>24</td>
<td>27</td>
<td>0</td>
</tr>
<tr>
<td>Biscuits and cakes</td>
<td>10</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Table sugar</td>
<td>11</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Milk</td>
<td>0</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Sweet puddings</td>
<td>5</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Breakfast cereals</td>
<td>5</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Fruit</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Syrups and preserves</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Other sources</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>All sources</td>
<td>90</td>
<td>100</td>
<td>28</td>
</tr>
</tbody>
</table>
1.4.4.3 Medically compromised

Increased medical problems, particularly within elderly populations, are often related to an increase in caries rate. Some medications can induce low salivary secretion rates; consequently the reduction in the buffering capacity of saliva and lower clearance flow rate can result a higher incidence of caries (Hase et al., 1987). Medical conditions such as gastrointestinal disease, eating disorders and uncontrolled diabetes can cause the pattern of dietary intake to increase in frequency. Patients with conditions such as Crohn’s disease, chronic renal failure, malnutrition or a failure to thrive, will have an increased need for carbohydrate intake. Dietary energy supplements tend to have high levels of sugars and so a greater intake would increase the caries risk. Rooney (1984) found greater caries prevalence in Crohn’s disease patients as compared to matched controls.

The dental health of the elderly in care homes has been found to be poor, with high intakes of sugary foods and limited access to dental care (Simons et al., 1999).

1.4.4.4 Social deprivation

Epidemiological studies have shown a clear link between low socio-economic status and the prevalence of caries. Figure 1-10 illustrates the relationship of DMFT scores in 5 year old children in Scotland to the DEPCAT status (measure of social deprivation based on postcodes); an increase in caries rate can be seen as the social deprivation worsens. The 2006 Expenditure and Food Survey found the intake of NMES in low income groups to be significantly higher than the general population (Office for National Statistics 2008).
Figure 1-10: DMFT levels for 5 year old Scottish school children in relation to DEPCAT scores (a measure of social deprivation), modified from Sweeney et al., (1999).

1.4.5 Caries Epidemiology

There has been a gradual decline in caries prevalence over the last 25 years (Figure 1-11). This has mainly been attributed to the use of fluoridated toothpastes as the single most important factor (Bratthall et al., 1996). The way in which sugar is consumed has also changed and may have also contributed to the decline in caries rate. There has been a shift in the widespread consumption of sugar based confectionary to chocolate based confectionary. Cocoa is not intrinsically cariogenic due to a lack of significant amounts of fermentable carbohydrate; however sugar is often added to it in varying quantities. Animal studies on rats fed common snack foods found solid milk chocolate among the lowest in cariogenicity (Morrissey et al., 1984). Human studies have also supported this finding. The Vipohelm study found no statistical difference between dental caries in the control and chocolate consumption group (Gustafsson et al., 1954). Luke et al. (1999) looked at the retention time of dietary carbohydrates in the oral cavity and found chocolate to have a rapid clearance time, thought to be due to the fat content aiding clearance.
The substitution of sugars with non-sugar sweeteners has also been felt to have contributed to the reduction in caries, particularly in countries such as Switzerland and Finland (Maguire and Rugg-Gunn, 2003). The use of sugars less cariogenic than sucrose such as high fructose corn syrup (HFCS) have also been used in increasing amounts and have now displaced sucrose as the major sweetener in manufactured products in the USA (Rugg-Gunn, 1993).

**Figure 1-11: The decline in the prevalence of dental caries in 12 year old children globally between 1967-1983. From Kidd (2005), source Renson (1986).**

1.4.6 **The Influence of Fluoride on the Caries–Sugar Relationship**

Extensive research supports the anticariogenic effect of fluoride (Mellberg and Chomicki, 1983, Rolla, 1988, van Rijkom et al., 1998). There are several mechanisms through which fluoride acts to reduce demineralisation and enhance remineralisation of tooth structure (Levine, 1991). The main action of fluoride is topical at the enamel surface post-eruptively. Fluoride reduces the susceptibility of enamel to demineralisation by converting hydroxyapatite into a more acid resistant fluoroapatite. Remineralisation of porous
deminerallised enamel lesions in the presence of fluoride causes more a more stable and acid resistant fluoroapatite to form rather than hydroxyapatite. Fluoride also has an effect on the plaque biofilm through the inhibition of the bacterial metabolism of sugar thus reducing acid production (Edgar et al., 1970). Pre-eruptive effects of fluoride on enamel have also been seen. Enamel formed in the presence of fluoride is more acid resistant due to improved crystallinity and increased crystal size. Also, cusps and fissure patterns are more rounded which reduces the depth of plaque retentive areas, although the effect is small.

Many studies which link caries and dietary sugar were conducted in the pre-fluoridation era. Due to the preventive effect of fluoride, its presence particularly in the form of fluoridation, can act as an important confounder. The causal link between dietary sugar and caries has been shown to still remain when efforts have been taken to record and control for use of fluoride and oral hygiene practices in two large longitudinal studies of adolescents (Burt et al., 1988, Rugg-Gunn et al., 1984). Burt and Pai (2001) carried out a systematic review into sugar consumption and caries risk, and explored the effect of fluoride exposure on caries severity. They concluded: (1) where there is good exposure to fluoride, sugars consumption is a moderate risk factor for caries in most people; (2) sugars consumption is likely to be a more powerful indicator for risk of caries in persons who do not have regular exposure to fluoride; (3) with widespread use of fluoride, sugars consumption still has a role to play in the prevention of caries but this role is not as strong as it is without exposure to fluoride.

1.5 Diet Advice

1.5.1 General Diet Advice

There have been many important UK publications concerning the relationship of nutrition and health; notable authoritative reports have been listed in Table 1-4 (Rugg-Gunn and Nunn, 1999). In 1992 the landmark government White Paper ‘The Health of the Nation’ set out a national strategy for the improvement of health and highlighted the vital role of diet and nutrition (Department of Health, 1992a).
Table 1-4: A list of authoritative national nutritional reports published in the UK, modified from Rugg-Gunn and Nunn (1999).

<table>
<thead>
<tr>
<th>Year</th>
<th>Report</th>
<th>Publisher</th>
</tr>
</thead>
<tbody>
<tr>
<td>1988</td>
<td>Present day practice in infant feeding: third report</td>
<td>COMA</td>
</tr>
<tr>
<td>1989</td>
<td>Dietary sugars and human diseases</td>
<td>COMA</td>
</tr>
<tr>
<td>1991</td>
<td>Dietary reference values for food energy and nutrients for the United Kingdom</td>
<td>COMA</td>
</tr>
<tr>
<td>1992</td>
<td>Health of the nation</td>
<td>Department of Health</td>
</tr>
<tr>
<td></td>
<td>The nutrition of elderly people</td>
<td>COMA</td>
</tr>
<tr>
<td>1994</td>
<td>The balance of good health</td>
<td>Health Education Authority</td>
</tr>
<tr>
<td></td>
<td>Weaning and the weaning diet</td>
<td>COMA</td>
</tr>
<tr>
<td></td>
<td>Eat well</td>
<td>Department of Health</td>
</tr>
<tr>
<td>1996</td>
<td>Eat well II</td>
<td>Department of Health</td>
</tr>
<tr>
<td>1997</td>
<td>Healthy diets for infants and young children</td>
<td>Ministry of Agriculture and Fisheries and Food</td>
</tr>
<tr>
<td>1998</td>
<td>Nutritional aspects of the development of cancer</td>
<td>COMA</td>
</tr>
<tr>
<td></td>
<td>Our healthier nation</td>
<td>Department of Health</td>
</tr>
</tbody>
</table>

The Committee on Medical Aspects of Food Policy (COMA) has published several key reports on the nutrition of children and adults. In 1989, COMA made clear the positive relationship of caries experience and the frequent consumption and amount of non-milk extrinsic sugars in the diet (Department of Health, 1989). Subsequently recommendations were made by COMA in 1991 for the daily consumption of NMES not to exceed 60g per day or 10% of the total dietary energy intake (Department of Health, 1991).

The Health Education Authority (HEA) has helped to pass on recommendations of COMA into clear nutritional messages. In 1997, HEA published its ‘Eight guidelines for a healthy diet’ which provided practical nutritional advice for general health, as listed in Table 1-5 (Health Education Authority, 1997). The ‘balance of good health’ programme by the HEA has also helped to translate nutritional requirements into food recommendations through the pictorial representation of a balanced diet shown in Figure 1-12.
Table 1-5: Eight guidelines for a healthy diet, (Health Education Authority, 1997).

1. Enjoy your food
2. Eat a variety of different foods
3. Eat the right amount to be a healthy weight
4. Eat plenty of foods rich in starch or fibre
5. Eat plenty of fruits and vegetables
6. Don’t eat too many foods that contain a lot of fat
7. Don’t have sugary foods and drinks too often
8. If you drink alcohol, drink sensibly

Figure 1-12: ‘The eatwell plate’ (Food Standards Agency, 2007) is an updated version of 'The Balance of Good Health' plate (Food Standards Agency, 2001).

1.5.2 Dental Advice

Dental dietary advice should be given in agreement with general nutritional recommendations for good health (Moynihan, 2002). The message should be clear and consistent with all health care professionals. To facilitate this, a general consensus view of dietary advice for the prevention of caries has been adopted and is shown in Table 1-6, taken from the recently
Table 1-6: Consensus recommendations on dietary advice for the prevention of caries, from the Delivering Better Oral Health: An evidence-based toolkit for prevention (Department of Health and the British Society for the Study of Community Dentistry, 2009).

- The frequency and amount of sugars should be reduced. Consumption of sugary foods should be restricted to mealtimes.
- Limit consumption of foods and drinks with added sugars to a maximum of four times a day.
- Sugars (excluding those naturally present in whole fruit) should provide less than 10% of total energy in the diet or less than 60 g per person per day. Note that for young children this will be around 33 g per day.

There has been some debate on the potential for a reduction in the consumption of dietary sugars to cause an increase in fat intake; otherwise known as the sugar/fat seesaw (Gibney et al., 1995). Evidence from a repeated cross-sectional study of the dietary intake of adolescents over 20 years did not support this hypothesis and found significant reductions in fat intake was not accompanied by increases in sugar consumption (Rugg-Gunn et al., 2007). Dietary intervention studies have also failed to show an inverse relationship between fat and sugar, and found simultaneous reductions in intake of added sugars and fats (Cole-Hamilton et al., 1986). Nevertheless, it is important that the well-meaning dietary advice for a reduction of NMES should not lead to an increase in the consumption of snacks high in saturated fats.

1.5.3 Dietary Counselling

There are many factors which have an impact on the dietary choices individuals take and so dentists providing dietary advice to should be mindful of this (Watt et al., 2003) (Table 1-7). Advice should be tailored to the personal circumstances of each individual to increase the likelihood of acceptance and behavioural change. A systematic approach to providing dietary advice has been described by Watt et al., (2003) as a six step dietary counselling model derived from evidence-based general dietary guidelines (Roe et al., 1997) and this will be discussed in further detail with relevance to dental dietary advice.
Individual level
• Lack of motivation to change — enjoy taste of sugary foods and drinks
• Lack of confidence to change — previous attempts have failed
• Lack of information — not clear which foods contain sugars
• Lack of skills — unable to prepare and cook healthier foods

Social level
• Peer group pressures — everyone else eats chocolate at coffee time
• Lack of time — too busy to cook
• Family pressures — husband and children will not eat vegetables
• Cultural food beliefs — sugar is needed for energy

Environmental level
• Healthier choices too costly — high costs of healthier snack foods and drinks
• Limited choices available — tuck shops only stocks soft drinks and confectionery
• Advertising pressures — children demand latest gimmick food as seen on television

1.5.3.1 Identification of high risk patients
A comprehensive dental history and examination will help identify patients at a high risk of developing caries. High risk patient groups may include those with chronic medical problems, pre-school children, adolescents, older dentate adults, and individuals from low socio-economic backgrounds. Clinical examination will reveal the extent of current and previous disease levels. Patients deemed to be of a high caries risk should have a diet history carried out to identify any potentially damaging dietary intakes or behaviours. Many are often unaware of the level of sugar they may consume, with ‘hidden sugars’ a common feature in many diets.

1.5.3.2 Diet history
The assessment of existing dietary intake is essential to identify potential cariogenic food sources and habits. From an oral health perspective, important areas of consideration are:
1. The frequency and amount of NMES
2. Whether sugary intakes were taken as snacks between meals
3. Whether any sugary intakes were taken within one hour of bedtime.
Assessment of dietary intake can be accomplished through the use of diet diaries, 24 hour recall interviews or food frequency questionnaires. These methods provide a useful picture of
dietary intake which can then be correlated with the caries status. This then allows the
opportunity for corrective advice to address cariogenicity issues within the diet. Each of these
methods however has the disadvantage of forming a snapshot only of a limited period of time
and cannot be taken as representative of the long term diet (Rugg-Gunn and Nunn, 1999).
Their use is also heavily dependent on the cooperation and motivation of the patient to
provide a truthful record. In view of this diet histories can provide useful tools but should be
interpreted with a degree of caution.

1.5.3.3 Diet diaries

Information is normally collected on all food and drink consumed over a 3-4 day consecutive
period, one day of which must be a weekend. The data recorded should include: the time,
amount and description of the intake, and the use of medication particularly if it is syrup
based. An example of a diet sheet is shown in Figure 1-13.

Figure 1-13: An example of a 4-day diet sheet, from Kidd (2005).

<table>
<thead>
<tr>
<th></th>
<th>THURSDAY</th>
<th></th>
<th>FRIDAY</th>
<th></th>
<th>SATURDAY</th>
<th></th>
<th>SUNDAY</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>BEFORE BREAKFAST</td>
<td></td>
<td>Time</td>
<td>Item</td>
<td>Time</td>
<td>Item</td>
<td>Time</td>
<td>Item</td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td>7:45</td>
<td>Tea. *</td>
<td>Item</td>
<td>7:00</td>
<td>Tea. *</td>
<td>1:00</td>
<td>Tea. *</td>
<td>Item</td>
</tr>
<tr>
<td>Breakfast</td>
<td>9:00</td>
<td>Coffee *</td>
<td>8:45</td>
<td>Coffee *</td>
<td>10:00</td>
<td>Tea. *</td>
<td>2 pieces of tea</td>
<td></td>
</tr>
<tr>
<td>MORNING</td>
<td>10:00</td>
<td>Coffee *</td>
<td>9:30</td>
<td>Coffee *</td>
<td>12:00</td>
<td>Coffee *</td>
<td>10:45</td>
<td>Tea. *</td>
</tr>
<tr>
<td></td>
<td>10:45</td>
<td>Bread and butter</td>
<td>9:45</td>
<td>Coffee *</td>
<td>12:45</td>
<td>Coffee *</td>
<td>11:30</td>
<td>Tea. *</td>
</tr>
<tr>
<td>Mid-day Meal</td>
<td>11:30</td>
<td>Coffee *</td>
<td>1:45</td>
<td>Coffee *</td>
<td>1:45</td>
<td>Cheese &amp; onion, &amp; garlic</td>
<td>1:45</td>
<td>Coffee *</td>
</tr>
<tr>
<td>AFTERNOON</td>
<td>2:00</td>
<td>Coffee *</td>
<td>2:30</td>
<td>Coffee *</td>
<td>3:00</td>
<td>Tea. *</td>
<td>3:30</td>
<td>Tea. *</td>
</tr>
<tr>
<td></td>
<td>2:30</td>
<td>Coffee *</td>
<td>2:30</td>
<td>Coffee *</td>
<td>4:15</td>
<td>Tea. *</td>
<td>3:30</td>
<td></td>
</tr>
<tr>
<td>Evening Meal</td>
<td>7:30</td>
<td>Country hash, Tea. *</td>
<td>7:00</td>
<td>Lasagne, Tea. *</td>
<td>7:00</td>
<td>Spare Ribs, Rice, Tea. *</td>
<td>5:00</td>
<td>Tea. *</td>
</tr>
<tr>
<td>EVENING &amp; NIGHT</td>
<td>9:00</td>
<td>Milk, biscuits, Tea. *</td>
<td>10:00</td>
<td>Tea. *</td>
<td>9:30</td>
<td>Tea. *</td>
<td>8:00</td>
<td>Coffee. *</td>
</tr>
<tr>
<td></td>
<td>10:00</td>
<td>Teas &amp; 2 biscuits, Tea. *</td>
<td>11:00</td>
<td>Tea. *</td>
<td>10:00</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
If used contemporaneously, this method of diet analysis has the benefit of not being dependent on the memory of the subject, however it can be appreciated many individuals may fill out the diaries retrospectively which would present a level of recall bias. The process can be quite burdensome on individuals and motivation should be very high at the outset. The success of diet diaries is dependent on a high level of cooperation to honestly fill out the record over the time period. There is a risk that the diet diary may have significant omissions or modifications to show the subject in a perceived better light or to make the recording process easier. Subjects also need to have adequate literacy skills to fill out the diary appropriately.

1.5.3.4 24 hour recall

This method involves the recording of dietary intake over the last 24 hours. This retrospective analysis will have the potential for recall bias may be less suitable for use with children and the elderly. Rugg-Gunn and Nunn (1999) have questioned the value of 24 hour recall interviews, highlighting potential problems with atypical preceding days. Advantages of this method include the minimal training of the subject to take part in the diet history and the minimal effort required to provide information to the interviewer. Additionally, the reduction in the amount of data collected from a 24 hour recall as compared to a 3-day diet diary would mean cheaper and less time consuming data processing. 24 hour recalls may be more suited to use in large dietary surveys where the average intake of a large population is assessed rather than the assessment of an individual’s diet which can vary from day to day (Gibson, 1998).

1.5.3.5 Food frequency questionnaires

Food frequency questionnaires (FFQ’s) are commonly used in large diet and health epidemiological studies to assess long term dietary intake. They are self-administered dietary questionnaires, comprising of a list of foods/food groups and the frequency of consumption. Assumptions are often made about portion size, although subjects can be asked to provide rough estimates. FFQ’s present a less arduous task for the subject to complete compared to a diet diary, and the ease of analysis due to the structured design of a questionnaire provides beneficial savings in time and cost. However, FFQ’s have been criticised for their measurement error with the accuracy of quantifying absolute intakes lower than for other
methods (Brown, 2006). The depth of dietary assessment will always be limited by what is feasible to include within the questionnaire.

1.5.3.6 Setting goals

In relation to oral health, any goals which are set should be mindful of the overall aim of reducing the frequency and quantity of NMES. Goals should be ‘personable, practical and positive’ (Rugg-Gunn and Nunn, 1999) to ensure maximum acceptance and compliance. A phased approach to dietary changes may sometimes be more suitable with some subjects who have become very used high sugar taste thresholds.

1.5.3.7 Action plan

A useful outcome of the diet history is the creation of an agreed action plan of suggested changes to dietary intake. Barriers for reducing the intake of NMES sugars can be discussed and suggestions made to overcome any issues such as practical advice on alternative ‘safe’ foods. An example of foods of low cariogenicity or possible anti-cariogenic effect is given in Table 1-8 (Moynihan, 2002). The timing of intakes can also be discussed using the diet history as a reference point to help identify periods of the day when sugars are more likely to be consumed.

<table>
<thead>
<tr>
<th>LOW/NO CARIES RISK</th>
<th>POSSIBLE ANTI-CARIOGENIC EFFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bread (sandwiches, toast, pitta bread)</td>
<td>Milk</td>
</tr>
<tr>
<td>Pasta, rice and starchy staple foods</td>
<td>Cheese</td>
</tr>
<tr>
<td>Unsweetened or artificially sweetened yogurt</td>
<td>Peanuts</td>
</tr>
<tr>
<td>Low-sugar breakfast cereals</td>
<td>Sugar-free chewing gum</td>
</tr>
<tr>
<td>Sugar-free confectionery</td>
<td>Fibrous foods (e.g. raw vegetables)</td>
</tr>
<tr>
<td>Fresh fruit (whole and not juices)</td>
<td>Xylitol sweeteners, gum and mints</td>
</tr>
<tr>
<td>Water</td>
<td>Tea (unsweetened)</td>
</tr>
<tr>
<td>Sugar-free drinks</td>
<td></td>
</tr>
</tbody>
</table>
1.5.3.8 Monitor and review

Due to the inherent difficulties with changing dietary habits, progress should be monitored and reviewed on a frequent basis. This then allows the opportunity for further support and feedback on any changes made. The best method of checking progress would be to ask the subject to fill out another diet sheet to see if suggested dietary recommendations have been followed.

1.5.3.9 Refer

Some patients may present with complex dietary requirements such as those with particular medical conditions and these cases may be more suitable under the management of general medical practitioners or dieticians. For example, patients with eating disorders such as anorexia or bulimia often require complex treatment and management with a range of healthcare professionals.

1.5.4 Dietary Advice in the Dental Setting

Dentists have an ethical responsibility to give consideration to the impact of any dietary habits and provide any necessary dietary advice which would encourage the maintenance of good oral health. Preventive interventions recommended by the Oral Health Toolkit include the investigation of diet and assistance in the adoption of good dietary practices (Department of Health and the British Society for the Study of Community Dentistry, 2009). National guidelines have repeatedly included dietary assessment as a factor to consider when assessing caries risk (Faculty of Dental Surgery, 1997, National Institute for Clinical Excellence, 2004). Notably, the NICE guidance on dental recalls advocated risk assessments for all patients and featured the category ‘Dietary Habits’ within its Oral Health Assessment checklist. A recent review of NHS Dentistry by Professor Steele and co-workers (2009) reaffirmed the mainstream role preventive measures such as diet improvement and smoking cessation should hold within dentistry and the importance of wider public health measures to address oral health risks.

A close relationship exists between diet and oral health and the importance of its assessment within the dental health care setting as part of the routine examination process should not be
overlooked. The emergence of clinical care pathways has placed renewed emphasis on the risk assessment of patients to help predict further disease and guide future prevention and intervention strategies. However the success of this risk assessment approach is reliant on the accurate prediction of caries risk and as yet, there is no reliable tool for predicting caries (Hausen, 1997). Reliable predictors are therefore needed, validated by evidence-based research, to assist in the risk assessment of caries.

1.6 Aims of the Study

With dietary intake of NMES a key aetiological factor in the development of caries, the predictive level of caries risk posed by dietary habits such as NMES frequency of intake and timing is questioned. The complexity of accurately recording this dietary variable has previously been discussed and therefore there exists a need to find a simple measure of dietary intake with respect to caries experience that can be used in caries studies and clinical risk assessment to reliably measure this important confounding variable.

The aim of this study was to investigate the influence of dietary intake of NMES on the caries experience of a population of 12-13 year old schoolchildren, and to identify reliable dietary predictive risk factors for caries experience that can be used in clinical risk assessments and within caries research studies.

Specific objectives were to:
- To determine the caries experience of subjects within the study group and the quantity of NMES intake (as a percentage of energy, %NMES), and the frequency of NMES snacks between meals and before bed.
- To determine if any of the dietary variables as listed above can be used to reliably predict caries experience in the study group

The null hypothesis was that dietary variables, namely the quantity of NMES intake and frequency of NMES snacks between meals and before bed, would have no effect on caries experience of the subjects under investigation. The working hypothesis was that the consumption of NMES foods, both in quantity and frequency, would have an effect on the caries experience of the subjects.
Chapter 2
2 Materials and Methods

2.1 Subjects

This cross-sectional observational study involved a convenience sample of 128 healthy male and female subjects aged 12-13 from comprehensive schools in Greater Manchester and Newcastle. The sample was selected based on consent, and logistical considerations such as school access and availability for the planned examinations. All subjects were selected from an observational epidemiological survey for which the caries status and level of material deprivation was available; I had previously participated with this study as a clinical dentist involved with the clinical examination of subjects and data collection.

2.2 Consent

Ethical approval was granted from the University of Manchester Committee on the Ethics of Research on Human Beings (Appendix I). Permission was sought from the Local Education Authority (LEA) and Head Teachers of the selected schools and notification provided to the Local Dental Committee (LDC) (Appendix II).

Parents/guardians of all 12-13 year old children who were expected to be at school during the examination period were notified of the study through a postal information pack and provided with a consent form to give positive consent for the inclusion of their child/children in the study (Appendix III and IV). Written consent was also gained from each subject prior to the examination (Appendix V).

2.3 Inclusion/Exclusion criteria

Schools were approached based on their anticipated cooperation with the study, uptake of free school meals as a measure of social deprivation (Shuttleworth, 1995, Muirhead and Marcenes, 2004) and an expectation of high lifetime residency status for the children. A total of 13 schools were used: 10 from Newcastle and 3 from Manchester. The schools in Newcastle were from fluoridated areas, the Manchester schools were not. Subjects were screened for suitability by the clinical examiner using the screening form as detailed in Appendix VI. The main inclusion/exclusion criteria have been listed in Table 2-1.
Any subjects deemed unsuitable to take part were informed and provided with a rejection letter explaining their unsuitability (Appendix VII).

Table 2-0: Criteria used to identify suitable subjects for the study.

| 1. Children 12-13 years of age |
| 2. Good general health |
| 3. No parental/guardian objection (via postal consent) |
| 4. Cooperative and able to be examined |
| 5. No fixed orthodontic appliances |
| 6. Lifetime resident of the area under examination |

Each recruited subject was then allocated a five-digit ID number; the first two digits specified the school and the next three digits the subject’s individual study number within the school, based on the sequence of their recruitment (Appendix VIII). A study folder was generated for each subject, containing adhesive labels with their unique identifier, data collection sheets, photo log and consent forms.

2.4 Data Collection

Demographic details such as age and postal code of each subject was obtained from the parental consent form. The postal code was used to arrive at a measure of relative deprivation of the participants through the use of the Index of Multiple Deprivation (IMD) (Department of Communities and Local Government, 2007). IMD quintiles were not used due to the low subject numbers and so an IMD score of >30 was chosen to categorise subjects residing in areas of high material and social deprivation.

Two examinations were used to collect the data required for this study, carried out over two school visits. The first visit involved a clinical caries examination led by a dentist and the second visit involved a diet assessment led by a dietician, as detailed below.

2.4.1 Clinical Examination

Under standardised clinical conditions with the use of a dental operatory light and dental unit equipped with compressed air, a caries examination was carried out by a dentally trained clinical examiner (MG) using the ICDAS diagnostic criteria as detailed in Appendix IX. All
teeth present were examined which included the buccal surfaces of incisors and canines, buccal/occlusal surfaces of premolars and the occlusal/buccal/mesial/distal surfaces of molars. The number of decayed, missing and filled permanent teeth (DMFT) were calculated. The variable DMFT was generated with a caries threshold of ICDAS stage 4 (dentine discolouration shadowing through intact enamel) for the decayed teeth component. Missing teeth were noted as those teeth extracted due to caries, unless known to have been extracted for orthodontic reasons (as established from the subject at the time of examination). Missing teeth extracted due to caries were given an ICDAS score of 6 and included for analysis. Filled teeth with clinically visible restorations were also recorded. No radiographic examination was carried out. Teeth with fissure sealants or sealant restorations were considered to be sound. An example of the clinical data capture form is shown in Appendix X.

All subjects were given a 3-day diet diary together with written and verbal instructions for its completion. Importance of recording all food and drink consumption over the 3-day period was stressed, with the inclusion of one weekend day. Subjects were advised to return the diet diaries to the school office on completion.

2.4.2 Dietary Assessment

The diet assessment was conducted by a qualified dietician (AV) over one visit at the subject’s school. All the subjects had returned a diet diary which was used as a prompt to discuss dietary intake and a detailed 24 hour recall interview was then carried out; no comparison was meant between the two methods, merely the diet diary helped facilitate a discussion on dietary intake by the dietician. The 24 hour recall focused on the number of non-milk extrinsic sugar (NMES) snacks consumed between meals in the preceding 24 hours and the number of NMES snacks consumed within an hour of bedtime. NMES were defined as ‘added sugars’ most damaging to dental health, such as sucrose, glucose, fructose. This excluded natural sugars present in milk, fruit and vegetables. Any foods or drinks consumed which contained NMES were recorded as a positive intake. Calculations of total NMES intake were made based on information from McCance and Widdowson food composition tables (Food Standards Agency, 2002). Average portion sizes were used. The variable %NMES was calculated as a percentage of the total calories consumed within the given 24 hour period.
The subject flow through the study on both visit 1 and 2 is summarised below (Figure 2-1 and Figure 2-2).

**Figure 2-1: Visit 1, subject flow through the study**

- Child collected from class by administrator 1
- Explanation of the study and completion of screening form and consent
- Clinical examination (MG)
- Allocation of diet diary and instructions on use
- Completion of examinations, child returns to class

**Figure 2-1: Visit 2, subject flow through the study**

- Child collected from class by administrator
- Explanation of the supplementary diet analysis and consent to take part
- 24 hour recall diet interview (AV)
- Completion of the diet interview, child returns to class
2.5 Data Analysis

Data analysis was performed using SPSS version 16. Initially the population groups from both sites (Manchester and Newcastle) were analysed for statistical differences in caries experience, deprivation and dietary variables (percentage NMES, number of NMES snacks consumed between meals and before bedtime) to determine the possible significance of confounding factors between sites, namely fluoridation. The data from both sites was then combined and dichotomised for caries experience with the threshold for caries set at the ICDAS 4 level. Significant differences in the dietary variables were determined using the Mann Whitney U test. Any variables found to have a significant difference in caries experience were investigated further using logistic regression to determine if any of the dietary variables as listed above (independent variables) predicted caries experience (dependent variable). The level of statistical significance was set at p=0.05.
Chapter 3
3 Results

3.1 Subjects

A convenience sample of 128 subjects (68 boys and 60 girls) was included for statistical analysis in this study. 51% of the subjects were from Newcastle (a fluoridated area), 49% from Manchester (a non-fluoridated area). The mean age at examination was 12.8 years, with 64 % of the subjects residing in areas of high deprivation (IMD>30) (Table 3-1). The difference in deprivation between the Manchester and Newcastle subjects was marginally significant (34.3, 42.8 p=0.05), (Table 3-2).

<table>
<thead>
<tr>
<th>Total</th>
<th>Newcastle</th>
<th>Manchester</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total subjects (%)</td>
<td>128</td>
<td>65 (51%)</td>
</tr>
<tr>
<td>Male (%)</td>
<td>68 (53%)</td>
<td>33 (51%)</td>
</tr>
<tr>
<td>Female (%)</td>
<td>60 (47%)</td>
<td>32 (49%)</td>
</tr>
<tr>
<td>Age at exam – mean (SD)</td>
<td>12.8 (0.45)</td>
<td>12.6 (0.37)</td>
</tr>
<tr>
<td>Deprivation – Low (%)</td>
<td>46 (36%)</td>
<td>28 (43%)</td>
</tr>
<tr>
<td>Deprivation – High (%)</td>
<td>82 (64%)</td>
<td>37 (57%)</td>
</tr>
</tbody>
</table>

Table 3-2: The difference in deprivation status of subjects from Manchester and Newcastle

<table>
<thead>
<tr>
<th>Mean total (SD)</th>
<th>City</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deprivation (IMD)</td>
<td>39.1 (21.1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>34.4 (22.8)</td>
<td>42.8 (18.6)</td>
</tr>
</tbody>
</table>

Based on the Mann-Whitney U test for dietary variables, * marginal significance at the 0.05 level.

All the 128 subjects attended for the 24 recall diet interview and had returned diet diaries, however none were fully completed and so data from them was deemed to be too limited and therefore omitted from the dietary analysis. The diet diaries were used however to facilitate a
discussion of diet at the 24 hour recall interview. Data from the 24 hour recall interview was used to quantify the dietary variables under investigation: NMES consumed between meals, NMES before bed and % NMES intake.

On average, subjects in Manchester and Newcastle showed no significant difference in the amount of NMES they consumed either between meals or before bed (Table 3-3). There was a significant difference in percentage of total NMES consumed, with a greater mean percentage of total NMES consumption in Manchester as compared to Newcastle (19.6%, 16.8% p<0.03). Newcastle subjects had a significantly lower DMFT than Manchester subjects (0.52, 1.48 p<0.001).

Table 3-3: Mean differences in dietary intake of NMES and DMFT (ICDAS 4) in subjects from Manchester and Newcastle

<table>
<thead>
<tr>
<th></th>
<th>Mean (SD)</th>
<th>City</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Newcastle</td>
<td>Manchester</td>
</tr>
<tr>
<td>NMES between meals</td>
<td>1.91</td>
<td>1.86 (1.20)</td>
<td>1.95 (1.44)</td>
</tr>
<tr>
<td>NMES before bed</td>
<td>0.46</td>
<td>0.45 (0.64)</td>
<td>0.48 (0.67)</td>
</tr>
<tr>
<td>%NMES (SD)</td>
<td>18.17</td>
<td>16.78 (7.23)</td>
<td>19.56 (7.31)</td>
</tr>
<tr>
<td>DMFT (ICDAS 4)</td>
<td>1.58</td>
<td>0.52 (1.06)</td>
<td>1.48 (1.86)</td>
</tr>
</tbody>
</table>

Based on the Mann-Whitney U test for dietary variables, * Significant at the 0.05 level

3.2 Caries experience

After combining the subject numbers from Manchester and Newcastle, 42% were found to have caries at the ICDAS >4 threshold (Table 3-4). There was no significant difference in caries experience when looking at high and low deprivation, NMES between meals and percentage of total NMES consumed. The only dietary variable that showed a statistically significant difference when looking at caries experience was NMES before bed.
Table 3-4: Comparisons of dietary intake of NMES and deprivation status with caries experience (DMFT-ICDAS 4)

<table>
<thead>
<tr>
<th></th>
<th>No Caries (ICDAS&lt;4)</th>
<th>Caries (ICDAS&gt;4)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total subjects (%)</td>
<td>74 (58%)</td>
<td>54 (42%)</td>
<td>-</td>
</tr>
<tr>
<td>Low deprivation</td>
<td>30</td>
<td>16</td>
<td>0.204</td>
</tr>
<tr>
<td>High deprivation</td>
<td>44</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>NMES between meals</td>
<td>1.8</td>
<td>2</td>
<td>0.422</td>
</tr>
<tr>
<td>NMES before bed</td>
<td>0.32</td>
<td>0.64</td>
<td>*0.002</td>
</tr>
<tr>
<td>% NMES</td>
<td>17.4</td>
<td>19.2</td>
<td>0.108</td>
</tr>
</tbody>
</table>

Based on the Mann-Whitney U test for dietary variables, * Significant at the 0.05 level

3.3 Predictive factors for caries experience

Logistic regression analysis was used to identify any dietary habits that predicted caries experience in the sample. The dietary factors of NMES between meals, NMES before bed and % NMES were used as independent factors within a logistic regression model. The only factor which emerged as a significant predictor of caries experience was frequency of NMES snacks consumed before bed, with an odds ratio of 2.26 (Table 3-5). This model correctly predicted 58% of caries experience in the children studied, which rose to 65% with the addition of the age at exam variable. A small effect size however was noted.

Table 3-5: Logistic regression analysis of NMES before bed and caries experience (DMFT-ICDAS 4)

<table>
<thead>
<tr>
<th></th>
<th>95% CI for odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SE)</td>
</tr>
<tr>
<td>Included</td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-14.538 (5.836)</td>
</tr>
<tr>
<td>Age at exam</td>
<td>1.040 (0.46)</td>
</tr>
<tr>
<td>NMES before bed</td>
<td>0.815 (0.310)</td>
</tr>
</tbody>
</table>

Note: Dependent variable = caries experience

R2 = 0.031 (Hosmer and Lemeshow), 0.116 (Cox and Snell), 0.156 (Nagelkerke).
3.4 NMES intake

The mean DMFT for subjects who consumed at least one NMES snack before bed was 1.49; this was over double the mean DMFT for subjects who did not consume any NMES snacks before bed, 0.68. There was a significant difference between these two groups (Table 3-6).

Table 3-6: Mean DMFT (ICDAS 4) score of subjects and whether NMES has been consumed before bed

<table>
<thead>
<tr>
<th></th>
<th>Mean total (SD)</th>
<th>NMES snacks before bed</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>None (SD)</td>
<td>1&gt; (SD)</td>
</tr>
<tr>
<td>DMFT (ICDAS 4)</td>
<td>1.91</td>
<td>0.68 (1.42)</td>
<td>1.49 (1.70)</td>
</tr>
</tbody>
</table>

Based on Mann-Whitney U test, * significant at the 0.05 level

Of the 49 subjects who consumed NMES before bed, the majority (83.7%) also had NMES snacks during the day (Table 3-7). However, of the 109 subjects who consumed NMES snacks during the day, 62.4% did not consume NMES before bed. Only 11 subjects (9% of the total sample) did not have NMES snacks during the day or before bed.

Table 3-7: A contingency table of subjects who consumed NMES before bed and between meals

<table>
<thead>
<tr>
<th>NMES snacks between meals</th>
<th>NO</th>
<th>YES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>11</td>
<td>68</td>
<td>79</td>
</tr>
<tr>
<td>% within NMES before bed</td>
<td>13.9%</td>
<td>86.1%</td>
<td>100.0%</td>
</tr>
<tr>
<td>% within NMES snacks</td>
<td>57.9%</td>
<td>62.4%</td>
<td>61.7%</td>
</tr>
<tr>
<td>YES</td>
<td>8</td>
<td>41</td>
<td>49</td>
</tr>
<tr>
<td>% within NMES before bed</td>
<td>16.3%</td>
<td>83.7%</td>
<td>100%</td>
</tr>
<tr>
<td>% within NMES snacks</td>
<td>42.1%</td>
<td>37.6%</td>
<td>38.3%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>19</td>
<td>109</td>
<td>128</td>
</tr>
<tr>
<td>% within NMES before bed</td>
<td>14.8%</td>
<td>85.2%</td>
<td>100.0%</td>
</tr>
<tr>
<td>% within NMES snacks</td>
<td>100.0%</td>
<td>100.0%</td>
<td>100.0%</td>
</tr>
</tbody>
</table>
Chapter 4
4  Discussion and Conclusions

4.1  Discussion

The aim of this study was to investigate if dietary variables, namely the percentage NMES intake and frequency of consumption of NMES snacks between meals and before bed, had any correlation with the caries experience of adolescent children in the sample population. The only factor to emerge with a significant predictive relationship of caries experience was NMES consumption before bed.

Cariogenic dietary habits provide important causative risk factors for caries but due to the multifactorial aetiology of the disease, it can be difficult to single out specific factors to predict a significant proportion of the observed caries experience. It is interesting therefore that this study found that the NMES consumption before bedtime helped predict a high proportion of caries experience in the subjects sampled (58% which rose to 65% with the addition of the age at exam variable) with a predictive odds ratio of 2.26 (1.232, 4.146). However the small effect size of the regression model (0.156 Nagelkerke), compounded by the overall small sample size (n=128, of which only 42% of the subjects had caries), places limitations on the validity of the predictive relationship NMES consumption before bed was shown to have. Caution should be taken in drawing any definite conclusions from these results due several limitations and confounding factors as discussed below.

4.1.1  Study Limitations

4.1.1.1  Sample population

The subjects used in this study were taken as a convenience sample from an epidemiological survey which compared caries experience over two sites which differed in their fluoridation status. The use of a convenience sample and therefore non-randomisation of the subjects inevitably meant there was an increased risk of selection bias to the study sample. Due logistical considerations and restrictions on subject numbers with this school-based study, randomisation of the subjects was not carried out, although with a larger study sample this could be achieved to eliminate the impact of selection bias and test the hypothesis with a more representative population.
The requirement of positive consent placed further restrictions on the sample size. Detrimental consequences of positive consent in school based surveys has been described previously by Dyer et al., (2008), where concern was raised for the potential of response bias if some parents within the community would be more likely to provide a positive response than others and thus leading to an unrepresentative sample.

The caries experience reported in the study was found to be unrepresentative of 12 year old children on a national level. The reported caries experience of the sample population was 42% compared with 33.4% at a national level as reported in a recent Oral Health Survey of 12 year old children 2008-09 (NHS DEP for England, 2010). Due to this difference, the higher caries experience of the sample population places limitations on applying the findings to the wider population of 12 year olds, although the preliminary results may form the basis for further research to validate the findings with a wider, more representative population.

4.1.1.2 Confounders
In order to maximise the sample size used for analysis, the subjects in Manchester and Newcastle were combined and analysed on an individual level. However, notable confounders existed within the two population groups and should be taken into consideration when drawing any conclusions from the results.

On comparing the two populations of Manchester and Newcastle, subjects from Manchester were marginally more deprived which could have acted as a confounding factor; differences in deprivation within population groups are widely known to result in health inequalities; with numerous studies providing evidence for poor dental health in poor socio-economic groups (Attwood et al., 1990, Pitts and Palmer, 1994). However, the method by which deprivation was categorised had its limitations. The Index of Deprivation (Department of Communities and Local Government, 2007) was used as a relative measure of concentrations of deprivation at the small area level. Deprivation statistics are often reported within quintiles to allow an appreciation of a spectrum of deprivation levels. This was not possible within this study due to the low sample size and so deprivation was dichotomised based on an arbitrary figure of 30; subjects with deprivation scores 30 or greater were categorised as residing in a more deprived area. Dichotomising deprivation in this context has the disadvantage of being
imprecise and may lead to over/underestimation of deprivation, whilst providing categories that are too broad to reliably explore the statistical relationship of deprivation and oral health.

The exposure of fluoridation in the Newcastle subjects may have caused the true effect of cariogenic dietary habits on caries experience to be underestimated. The sites were compared initially in the analysis to determine if any differences could be identified that could be explained by the fluoridation status. The caries experience in the Newcastle population was significantly lower than Manchester. It was likely this was due principally to fluoridation, however it was also noted the Manchester subjects had a significantly higher consumption of NMES. This difference in NMES consumption however did not seem to impact on dietary habits of NMES snacks between meals and bedtime consumption of NMES with no significant difference between the two populations.

Caries has a wide reaching multifactorial aetiology and the presence of other factors such as poor oral hygiene may have acted as a confounding factor during this investigation on dietary factors for caries. The oral hygiene of the subjects was not investigated in this study, however future research utilising the preliminary findings could explore the impact of other aetiological factors such as oral hygiene, in combination with NMES intake, through multivariate analysis (see section 4.3).

4.1.1.3 Dietary data
Dietary assessments such as the 24 hour recall interview used in this study rely on self-reported data. The reliability of this is dependent on the subject’s cooperation and motivation to provide a truthful record. There are several potential limitations such as recall bias, embellishment and the possibility of atypical dietary intakes. Cross sectional studies using ‘snap-shot’ dietary assessments such as the 24 hour recall should always be taken with caution when comparing it to DMFT which provides a measure of lifetime caries experience, where the assumption is that diets have not changed over several years previously. A longitudinal study design would help to overcome this limitation of the cross sectional approach, however this would considerably increase the time and expense of the study (Rugg-Gunn et al., 1984).
3-day diet diaries were also used in this study and suffer many of the same limitations. In particular it was noted there was poor cooperation with their use and all the diaries within the convenience sample were returned incomplete/illegible, highlighting a limitation of their use in this age group when cooperation may not be optimum. Due to their incompleteness, their use in this study was found to be limited and the data derived from them was not used in the statistical analysis, however they did help stimulate a discussion of dietary intake at the 24 hour recall assessment between the subject and dietician. Diet diaries when completed appropriately, ideally contemporaneously to limit recall bias, with full cooperation and honesty provide an effective tool in establishing individual dietary intake thereby facilitating tailored dietary advice. Although they can be time consuming and labour intensive to fill out by the participant and to analyse by the investigator, the potential benefit offered by diet diaries to aid tailored dietary advice which is relevant to the individual cannot be ignored.

Although NMES before bed was shown to be predictive of caries experience, it is appreciated bedtime intake may be part of a general pattern of increased NMES intake. The caries experience of the sample might be explained by this overall increased frequency of NMES intake and not just NMES consumed before bed. 84% of subjects who consumed NMES before bed were also found to consume NMES snacks between meals which support this observation. However, the majority of subjects who consumed NMES during the day were found not to consume NMES before bed (62%). With this in mind, the fact that NMES snacks during the day was not found to significantly predictive of caries experience suggests that NMES consumption at night is possibly a more important determinant of caries experience.

4.1.1.4 Clinical examination
The clinical examination used ICDAS as a clinical scoring system to record caries experience within the sample population. For statistical analysis, an ICDAS code 4 threshold was chosen to distinguish subjects with caries, i.e. clinical signs of dentine involvement, from those that did not have signs of dentine involvement. This threshold was chosen since it relates to the D3 threshold of caries dentine involvement traditionally reported in previous caries studies. ICDAS is a novel clinical scoring system and has the benefit of reporting a range of caries severities. This benefit however was not utilised within this study due to the low distribution of caries severity levels which prevented a wider statistical analysis of the relationship dietary
factors had on caries experience at different severities. Further research with a larger sample size may rectify this by increasing the range of severity of caries experience within the sample.

4.1.2 Implications for Clinicians and Policymakers

Diet analysis with regards to caries risk assessment can become a time consuming process and so by identifying key risk factors that can predict caries experience, it is hoped these predictors can allow practitioners to focus their time on relevant questions asked as part of a risk assessment. Although there is some debate over whether a population based approach or risk-based approach to caries prevention should be followed (Milsom and Tickle, 2010, Page et al., 2010), risk-based approaches such as the dental recall intervals as advocated by the National Institute of Clinical Effectiveness (NICE, 2004) are likely to continue due to their assumed advantage of greater cost effectiveness. This places a great deal of importance in precise risk assessment, since if this is incorrect, costly clinical time potentially will be wasted. Reliable predictors are therefore needed for risk assessments to be valid enough to identify high risk patients. Currently, the only accepted accurate predictor of risk is evidence of current or previous caries experience (Hausen, 2008), which is unfortunate in that this predictor of future disease requires prevention strategies to have failed in the first place. Identifying detrimental dietary habits as predictors of caries risk at an early stage is therefore needed to provide an opportunity for children and parents to be educated in their harmful effects and instigate change before irreversible damage is caused.

The predictive relationship of bedtime NMES consumption and caries experience, if sufficiently validated with further research, would be of obvious benefit to caries prevention and health promotion programmes. Within the dental setting, patients can be questioned on their NMES bedtime consumption during a routine diet history interview to ascertain, along with other known risk factors, the predicted caries risk. Identifying at risk patients would then provide an opportunity for focused oral health promotion by the dental team. In broader sense, good dietary habits can also promoted through public health programmes where the detrimental effects of NMES bedtime consumption can be emphasised from both a dental and general health viewpoint.
The limited effectiveness of conventional oral health promotion has been previously described in the literature (Kay and Locker, 1996, Sheiham and Watt, 2000). Although there is evidence for oral health promotion increasing knowledge levels, there is no evidence that changes in knowledge are causally related to changes in behaviour (Kay and Locker, 1996). Ultimately, behavioural change is the real challenge and requires greater participation and commitment from individuals. Halpern and Bates (2004) highlighted this sense of personal responsibility and the need for public behavioural change for the successful outcome of health promotion initiatives. Collaboration between health professionals and agencies to promote universal messages of health promotion is also needed for effective oral health promotion. Publications such as the Oral Health Toolkit (Department of Health and the British Society for the Study of Community Dentistry, 2009) provide clear evidence-based guidance for the promotion of oral health which should help to reduce the chance of conflicting messages to the public. There are various implementation methods for oral health promotion not just limited to the dental practice setting and partnerships between organisations should be encouraged. School based education through classroom teaching or school dental visits can help reinforce positive messages, together with overall promotion of healthy dietary habits on a public health level.

4.1.3 Other Studies

The significant relationship of bedtime consumption of NMES and caries experience reported in this study is consistent with other studies (Levine, 2001, Levine et al., 2007). An additional finding was that the DMFT increment for subjects that consumed at least one sugar snack before bed (1.49) was over twice the DMFT for subjects that did not consume any sugar snacks before bed (0.68). This supports the findings of a previous study into bedtime consumption of sugary snacks (Levine, 2001) which found four times the mean DMFT score (1.24) in children who consumed bedtime sugar snacks compared to those who did not (0.31). Although differences in the study designs are noted such as the criteria for the caries increment in this study involving ICDAS stage 4 and caries at the D3 level in Levine’s study, these thresholds are similar in that dentine involvement is seen in both and does not detract from the similar trend shown in both studies of a positive relationship between caries experience and bedtime NMES consumption.
4.2 Conclusion

Within the limitations of this study, consumption of NMES before bedtime has been shown to provide be an important predictive risk factor for caries experience in adolescent children. The results should be interpreted with a degree of caution due to a low sample size and low caries experience in the sample population. The results however are consistent with previous studies and supports advice promoted by Levine for a ‘sugar free zone’ in the hour before bed as a recommendation for the prevention of caries (Levine, 2001).

4.3 Suggestions for Future Research

The results reported in this study have emphasised the detrimental impact cariogenic dietary habits can have on caries experience, in particular bedtime consumption of NMES. The small sample size however prevents any definitive conclusions to be drawn and therefore further research is needed on a larger scale to validate these preliminary findings.

Socio-economic status has been shown to have an effect on NMES intake (Office for National Statistics 2008) and caries prevalence (Sweeney et al., 1999). It would be interesting to explore this relationship in more detail across the socio-economic groups for specific cariogenic dietary habits such as bedtime NMES consumption. A larger, more representative sample arranged in IMD quintiles would help facilitate this analysis.

This study focused on dietary variables of NMES intake (percentage intake, frequency of snacks between meals and before bed) as risk factors for caries development. Since dental caries has a wide multifactorial aetiology it would be interesting to assess level of risk posed when known risk factors are combined. For example, it is likely other risk factors such as poor oral hygiene would increase the risk of caries experience further when in combination with high NMES intake. Statistical analysis using logistic regression would help to investigate this further.

Future research should consider a longitudinal study design to allow for the impact of changing dietary habits on observed life-long caries experience. This would avoid the limitation placed on the results of the reported cross sectional study which assumes dietary
habits have not changed for several years previously. A longitudinal design however would increase the time and expense of the study and provision would need to be made for this.

Research focusing on the effect of diet on the prevalence of dental caries in schoolchildren provides an opportunity also for dietary habits to be investigated from a wider health perspective. In collaboration with dieticians, total dietary intake could be assessed with regards to healthy eating. It would then be hoped the data could be used to develop preventative strategies aimed at improving not just oral health but the overall general health of schoolchildren with respect to dietary intake. However, as discussed above, behavioural change would be the real challenge and the effectiveness of collaborative efforts across different organisations involved in practice-based and school-based partnerships should be explored further for effectiveness in oral and public health promotion.
Appendices
Appendix I

Ethical approval

Dr Iain A Pretty,  
Dental Health Unit,  
3A Skelton House,  
Manchester Science Park,  
Manchester, M15 6SH

11th May 2007

Dear Iain,

Committee on the Ethics of Research on Human Beings
Pretty, McGrady: International caries detection and assessment system (ICDAS) training and calibration exercise (ref 07051)
Pretty, McGrady: Effect of social deprivation on caries and fluorosis prevalence in fluoridated and non-fluoridated areas (ref 07052)

I write to thank you and Mike McGrady for coming to meet the Committee yesterday and to confirm that the Committee gave ethical approval to the above projects. The Committee did, however, recommend that the information sheet should be in a larger font. It also gave provisional approval for part of the second project (07052) to be undertaken in the Manchester area, should this be necessary. If this should happen I would be grateful if you would let me know.

This approval is effective for a period of five years and if either project continues beyond that period it must be submitted for review. It is the Committee’s practice to warn investigators that they should not depart from the agreed protocol without seeking the approval of the Committee, as any significant deviation could invalidate the insurance arrangements. We also ask that any information sheet should carry a University logo or other indication of where it came from.

Finally, I would be grateful if you could complete and return the attached forms at the end of the project or by May 2008, whichever is earlier. We hope the research goes well.

Yours sincerely,

Timothy Stibbs

Dr T P C Stibbs
Secretary to the Committee
Appendix II
Example of Letter to the LDC (copied to LEA and Head Teachers)

The Dental Health Unit
3A Skelton House
Manchester Science Park
Manchester
M15 6SH

Dr. Iain A Pretty, BDS, MSc, PhD, MFDS RCS(Ed)
Senior Lecturer in Dental Public Health
Postgraduate Research Trainee

Tel: 0161 228 1211
Fax: 0161 222 4700

www.dentalhealthunit.org
iain.pretty@manchester.ac.uk

IP/ims

25-Oct-97

Chair of LDC

Dear Sir or Madam,

Ref: Dental study taking place in the North East and North West

I am writing to you on behalf of a research team based here at the University of Manchester and Newcastle University to tell you about a project that we intend to run within schools in Manchester and Newcastle.

Legislative changes in the UK will shortly enable more areas of the country to assess the feasibility of water fluoridation. Prior to the implementation of such new schemes, one of which may be implemented in Manchester, it is essential that as dental researchers we can develop better techniques to assess the benefits of such a public health measure. As part of the development of strategies to assess such benefits we are testing some new ways of measuring dental caries. This has the support of the Medical Research Council. We have chosen Newcastle and Manchester as the sites for our study because they contain clearly defined areas of fluoridated and non-fluoridated water supplies.

Children in your area will be asked to participate in a brief dental examination and have a number of photographs taken — all of which will show only their teeth. The study has been approved by the University of Manchester Ethics Committee and all individuals taking part will be provided with information sheets and will sign an informed consent form. I have attached these to this letter for your information.

As part of this examination we are required to inform the child and their parents of any dental treatment that is deemed necessary. In reality this will relate to grossly carious teeth and signs of sepsis. This could result in an increase in attendance of regular child patients or an increase in requests for new patient exams for dentists within your LDC area. I have attached to this letter a copy of the form that will be returned the child’s parents.

Do please contact me if you have any further questions.

Kindest regards

IAIN A PRETTY

GILL DAVIES

MICHAEL MCCRADY

Address Details

Dr Iain A Pretty
Senior Lecturer
School of Dentistry
University of Manchester
Skelton House
Manchester, M15 6SH

Dr Gill Davies
Senior Dental Officer
Specialist in Dental Public Health
Department of Dental Public Health
Maidenhead House
Maidenhead Road West
Manchester, M21 7RL

Mr Michael McGrady
Senior Research Fellow
School of Dentistry
University of Manchester
Skelton House
Manchester, M15 6SH
Appendix III
Parent Information Sheet

Oral Health Care Study – Parent’s Information Sheet

Dear Parent or Guardian

I am writing to you to ask for your consent (agreement) for your child to be involved in a clinical research study that we are conducting in conjunction with the University of Newcastle and the Department of Health. Before you can agree for your child to take part, I would like to give you more information about the background to the study. I also suggest that you read the information provided to your child to ensure you are happy that it reflects what we aim to do.

Dental Decay
Dental caries, or decay, is still a major problem within the UK. A number of measures have reduced the amount of decay within certain parts of the population and the most important of these are fluoride in both water supplies and toothpaste. Dental decay often requires a visit to the dentist and the placement of a filling. This will often mean time off school and work for both child and parent. Untreated dental decay can lead to painful toothache which may ultimately require a tooth to be extracted.

Water fluoridation
Many areas of the UK still do not benefit from water fluoridation. Recent changes in the law will enable more local governments to implement water fluoridation if they choose. However, in order for this to take place we will need to have improved methods of assessing the value of adding fluoride to the water. Our research group is interested in developing new ways of looking at tooth decay that will help in evaluating new water fluoridation programmes.

Why is my child being asked to participate?
We are running this study in two areas, one in the North East, the other in the North West. One area is water fluoridated, the other is not. By looking at these different areas we will be able to see the differences that water fluoridation makes to the oral health of the children living in these regions. Your child has not been individually selected – we are asking the parents or guardians of all children aged 11 and 12 in this school if they would like to participate. Your child will be part of a group of approximately 2000 we aim to assess. Participation in this study is entirely voluntary. You or your child may choose to withdraw from the study at any time and you do not have to give us a reason.

What will happen to my child if they participate in the study?
If you, and your child, agree to take part in the study they will be contacted at school. We will do a number of different examinations. The first will be a very standard dental check up where a dentist will look at all of your child’s teeth. Next, a few pictures will be taken. The first of these will be done with a very small camera in the mouth and the next two pictures will be taken outside of the mouth; looking only at the front teeth. One of these will be with a
standard camera and the other with a special camera which uses blue light. It will be necessary to dry the teeth using some compressed air, just like at a regular dentist’s surgery. None of the photographs will show your child’s face – they will only be of their teeth. We would expect that all of the examinations will take no more than 30 minutes. All of the examinations are done without any probing of the teeth; just with either a mirror or a camera and are completely painless. At any stage your child can decide that they do not want to continue with the study. After the examinations are done your child will be asked to complete a very simple survey on the amount of toothpaste that they use and the size of their toothbrush. After this your child will go back to their classroom. We will ask them to complete a food diary over a 3 day period at home with your supervision and bring the completed diary back into school. We will look at the images we have taken after the study has finished.

For some of the children in the study (a group of approximately 50 children) we will conduct a more detailed questionnaire on their diet history. We will write to you separately about this if your child is selected.

The results of this study may help us to identify children who may be suitable for future studies, if your child is suitable we may contact you again. You and your child would not be under any obligation to take part in any future studies.

Where will all these photographs be kept and who has access to them?
We will store all the photographs and clinical information on computers that are secured with a password. None of the information that we collect will be linked to your child, i.e. the information will be anonymous. The only people to have access to the information are the scientists involved in the study. We hope to publish our results in a scientific journal. No information concerning your child will be included. We will provide a copy of the results to your child’s school if you would like to see them. The study may be monitored by external bodies such as the ethics committee or regulatory authorities. Once again, your child will not be identified during this process.

What if my child has something wrong with their teeth?
If the examining dentist notices something in your child’s mouth that they feel needs treating they will write to you and let you know. You can take this letter to a dentist and they will be able to advise you further.

What benefit is there to me or my child?
This study aims to understand better the link between the reduction in tooth decay and water fluoridation. You and your child will be helping us to develop more information on this important issue. Your child may not gain a direct benefit by taking part in this study but they will receive a full dental examination and this may help identify any problems that they have. Once your child has returned the completed food diary they will be given a toothbrush and toothpaste to thank them for their help.

What risks are there to my child taking part?
There are no risks to your child taking part in this study. All of the procedures and equipment being used have a safe history of use and have been used in studies before. In the unlikely event that something does go wrong and your child is harmed during the research and this is due to someone’s negligence then you may have grounds for a legal action for compensation.
against the Local Education Authority or the University of Manchester, but you may have to pay your legal costs.

The University of Manchester has cover for no fault compensation for bodily injury, mental injury or death where the injury resulted from a trial or procedure you received as part of the trial. This would be subject to policy terms and conditions. Any payment would be without legal commitment. (Please ask if you wish more information on this). The University would not be bound to pay this compensation where the injury resulted from a drug or procedure outside the trial protocol or the protocol was not followed.

Who is paying for this study?
The University of Manchester is the study sponsor and the funding is sourced from the Department of Health.

Who has reviewed this study?
This research has been looked at by an independent group of people called a research ethics committee to protect the safety, wellbeing, rights and dignity of you and your child. This study has been reviewed and given a favourable opinion by the University of Manchester Committee on the Ethics of Research on Human Beings.

Who is running this study, what if I have a question?
The study is being undertaken by researchers at the Universities of Manchester and Newcastle. In Manchester Dr Iain Pretty is the principle investigator, and in Newcastle, Dr Anne Maguire is leading the team. You will find their contact details at the end of this letter. If you have any questions regarding the study please feel free to contact us. For Newcastle, please contact Debra Howe. You can contact her by email (Debora.Howe@anewcastle-pct.nhs.uk) or by telephone (0191 219 5217). For Manchester, please contact Michael McGrady You can contact him by email (Michael.Mcgrady@manchester.ac.uk) or by telephone (0161 2261211).

What do I do now?
Please complete the consent form that is attached to this letter. This is very important, even if you do not wish your child to take part. It would be very helpful to us if you could send the form back to your school.

I would like to thank you for reading this information sheet and considering your child’s inclusion in the study. Do please contact Debra Howe or Michael McGrady if you need any more information.

Yours Sincerely

[Signature]

Dr Iain Pretty
Senior Lecturer
School of Dentistry
University of Manchester
Skelton House
Manchester, M15 6SH

Dr Anne Maguire
Senior Lecturer
Dental School
Newcastle University
Framlington Place
Newcastle, NE2 4BW

Mr Michael McGrady
Senior Research Fellow
School of Dentistry
University of Manchester
Skelton House
Manchester, M15 6SH

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<tr>
<td>Dr Iain Pretty</td>
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<tr>
<td>Senior Lecturer</td>
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<td>School of Dentistry</td>
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Appendix IV
Child Information Sheet

Dear Student

I am writing to ask you if you would like to take part in a research project (study) that I and some scientists from Newcastle University are running in Newcastle and Manchester. I have also written to your parents or guardian. The first thing we will do is to make sure your parents have said it is ok for you to take part.

1. We would like you to help us look at tooth decay and white patches on teeth in school children about your age. We will look at 1000 children from Newcastle and 1000 children from Manchester

2. We will ask you to use a computer screen to show us how big your toothbrush is, how much toothpaste you use, and how you rinse your mouth when you brush your teeth

3. We will look in your mouth like a normal check-up at the dentist. We will use a special camera to take pictures of your teeth – which you will be able to see on a computer screen

4. After we have taken pictures inside your mouth we will take more pictures with a different camera. These pictures will be of only your front teeth using a special camera with a frame to support your head in the right place
5. When we have finished taking pictures you can go back to class. Taking all of the pictures will last about 30 minutes. We will give you a food and drink diary to take home. This diary is for you to write down everything you have to eat and drink. It helps us to know the types of food that you eat and drink and how it might affect your teeth.

6. You can ask your parents to help you fill in your diary at home. Remember to write down everything you have to eat and drink on each of the days!

7. When you have finished your food diary you can bring it back to school. We will give you a toothbrush and toothpaste to say thank you for helping us.

If you have any questions please ask us, if you do not want to take part, you do not have to. It is up to you!

Thanks again!

Iain Pretty

Address Details

Dr Iain Pretty
Senior Lecturer
School of Dentistry
University of Manchester
Skelton House
Manchester, M15 6SH

Dr Anne Maguire
Senior Lecturer
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Mr Michael Mclnroy
Senior Research Fellow
School of Dentistry
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Skelton House
Manchester, M15 6SH
Appendix V
Informed Consent Form

Oral Health Care Study

CONSENT FORM

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<th>Adhesive label to contain the following information:</th>
<th>Signature of Investigator once consent obtained:</th>
</tr>
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<tbody>
<tr>
<td>Initials, Class, School &amp; ID</td>
<td>Date:__________________________________________</td>
</tr>
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</table>

If you want your child to take part in this study please tick this box, complete the information in the box below this one, sign and date it before returning it to your child's class teacher.

If you do not want your child to take part in this study please sign this box and return the form to your child’s class teacher.

<table>
<thead>
<tr>
<th>Contact details</th>
<th>Telephone number (evening)</th>
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<tr>
<td>Address</td>
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<tr>
<td>POSTCODE:</td>
<td></td>
</tr>
<tr>
<td>Telephone number (day)</td>
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Has your child always lived at this address or in this neighbourhood? If “yes”, please tick box A on the right. If the answer is “no” how long have they lived in this area? ______ years _______ months

Is your child entitled to receive free school meals? If “yes” please tick box B on the right.

Please tick box C if your child has ever taken fluoride tablets, drops or any other form of fluoride supplement. If you tick Box C please indicate below when, and what fluoride supplements were taken

At what age did you start brushing your child’s teeth? ______ years _______ months

Did you use adult toothpaste or a children’s toothpaste?

How many times a day did you typically brush their teeth? ______

Please list any allergies that your child has in the box below e.g. Latex Allergy

I have read the explanation of this study in the accompanying letter with this consent form and I agree that my child can take part in the dental study. I understand that all information collected during the study will be kept confidentially but will be made available to the Study Sponsor or Governmental Agencies. I understand that in the unlikely event that something does go wrong I may have grounds for a legal action for compensation against the Local Education Authority or the University of Manchester and any payment would be without legal commitment. I also understand that I have the right to withdraw my child from the study at any time without affecting the future dental treatment rights of my child.

__________________________________________ ______________________
Signed by parent Date Signed by child Date
(Comments)

Please return this page of this letter to your child’s class teacher
Appendix VI
Screening Form

SCREENING FORM

Adhesive label to contain the following information:
Initials, Class, School & ID

1. Is subject in good health? (list medications, allergies, in comments section) Yes No
2. Is subject 11 or 12 years old? Yes No
3. Does subject have sufficient teeth (all four permanent molars) available for examination? Yes No
4. Is the subject a life-time resident of the area? Yes No
5. Is the subject free from bonded orthodontic appliances? Yes No
6. Does the subject have fully erupted maxillary central incisors? Yes No
7. Has the parent or guardian reported on the history of use of fluoride supplements? Yes No
8. Has parent/guardian signed an Informed Consent Form? Yes No

If any answer to the questions above is No the subject is ineligible for study and the subject should be given a rejection notification slip and Question 9 completed (No). If all answers are Yes the subject is eligible to take part in the study and Question 9 should be completed Yes.

9. IS SUBJECT ELIGIBLE FOR ENTRY INTO STUDY? Yes No

If no has “rejection notification” been sent to parent Yes No

10. Has the child agreed to take part in the study and have they provided consent to do so? Yes No

COMMENTS

Signature of screener

Date
Appendix VII
Example of Rejection Information

Oral Health Study

Dear parent,

Your child had a dental examination today as part of the Oral Health Study. Unfortunately we were unable to include them in the study for the reason indicated below.

Thank you for agreeing to help us with this study.

If you require any further information please contact

Mr Mike McGrady (Study Dentist) on telephone 0161 226 1211
Appendix VIII
Study Subject Label

These labels will be prepared on a sheet of 10 prior to attendance at each school. Every child who has returned a consent form will have a sheet of labels generated.

Labels will be placed on each child as an identifier and they will be handed the remainder of the labels to pass to each examiner as they continue through the examination sequence.

Adhesive label to contain the following information:
Initials, Class, School & ID

SJ
MORPETH PRIMARY SCHOOL
CLASS 2A
SUBJECT: 12013
SJ12013

SUBJECT: SJ12013
SJ12013

These sheets will comply with GCP guidelines and the Data Protection Act 1998 for subject anonymity. Any used labels will be returned to the principle investigator and will be shredded.
Appendix IX
Diagnostic Criteria for ICDAS

Coronal Primary Caries Codes

Sound tooth surface: Code 0
There should be no evidence of caries (either no or questionable change in enamel translucency after prolonged air drying (suggested drying time 5 seconds)). Surfaces with developmental defects such as enamel hypoplasias; fluorosis; tooth wear (attrition, abrasion and erosion); and extrinsic or intrinsic stains will be recorded as sound. The examiner should also score as sound a surface with multiple stained fissures if such condition is seen in other pits and fissures, a condition which is consistent with non-curious habits (e.g., frequent tea drinking). Table 1 provides a useful guide for differential diagnosis for various opacities versus other opacities.

First visual change in enamel: Code 1
Code 1 is assigned for the following pits and fissures:
When seen wet there is no evidence of any change in colour attributable to carious activity, but after prolonged air drying (approximately 5 seconds is suggested to adequately dehydrate a carious lesion in enamel) a carious opacity or discolouration (white or brown lesion) is visible that is not consistent with the clinical appearance of sound enamel.

OR

When there is a change of colour due to caries which is not consistent with the clinical appearance of sound enamel and is limited to the confines of the pit and fissure area (whether seen wet or dry). The appearance of these carious areas is not consistent with that of stained pits and fissures as defined in code 0.

Distinct visual change in enamel: Code 2
The tooth must be viewed wet. Where wet there is a (a) carious opacity (white spot lesion) and/or (b) brown carious discolouration which is wider than the natural fissure/fossa that is not consistent with the clinical appearance of sound enamel. (Note: the lesion must still be visible when dry).

Localized enamel breakdown with no visible dentine or underlying shadow: Code 3
The tooth viewed wet may have a clear carious opacity (white spot lesion) and/or brown carious discolouration which is wider than the natural fissure/fossa that is not consistent with the clinical appearance of sound enamel. Once dried for approximately 5 seconds there is carious loss of tooth structure at the entrance to or within the pit or fissure/fossa. This will be seen visually as evidence of demineralization (opaque (white), brown or dark brown walls) at the entrance to or within the fissure or pit, and although the pit or fissure may appear substantially and unnaturally wider than normal, the dentine is NOT visible in the walls or base of the cavity discontinuity.

If in doubt, or to confirm the visual assessment, the WHO/OP/PSR probe can be used gently across a tooth surface to confirm the presence of a cavity apparently confined to the enamel. This is achieved by sliding the ball along the suspect pit or fissure and a limited discontinuity is detected if the ball drops into the surface of the enamel cavity/discontinuity.

Underlying dark shadow from dentine with or without localized enamel breakdown: Code 4
This lesion appears as a shadow of discoloured dentine visible through an apparently intact enamel surface which may or may not show signs of localized breakdown (loss of continuity) of the surface that is not showing the dentine. The shadow appearance is often seen more easily when the tooth is wet. The darkened area is an intrinsic shadow which may appear as grey, blue or brown in colour. The shadow must clearly represent caries that started on the tooth surface being evaluated. If in the opinion of the examiner, the carious lesion started on an adjacent surface and there is no evidence of any caries on the surface being scored than the surface should be coded "0".

Code 3 and 4, histologically may vary in depth with one being deeper than the other and vice versa. This will depend on the population and properties of the enamel. For example more translucent and thinner enamel
primary teeth may allow the undermining discolouration of the dentine to be seen before localized breakdown of enamel. However, in most cases Code 4 is likely to be deeper into dentine than Code 3.

**Distinct cavity with visible dentine: Code 5**
Cavitation in opaque or discoloured enamel exposing the dentine beneath.

The tooth viewed wet may have darkening of the dentine visible through the enamel. Once dried for 5 seconds there is visual evidence of loss of tooth structure at the entrance to or within the pit or fissure - frank cavitation. There is visual evidence of demineralization (opaque (white), brown or dark brown walls) at the entrance to or within the pit or fissure and in the examiner judgment dentine is exposed.

The WHO/CPI/PDR probe can be used to confirm the presence of a cavity apparently in dentine. This is achieved by sliding the ball end along the suspect pit or fissure and a dentine cavity is detected if the ball enters the opening of the cavity and in the opinion of the examiner the base is in dentine. (In pits or fissures the thickness of the enamel is between 0.5 and 1.0 mm. Note the deep pulpal dentine should not be probed).

**Extensive distinct cavity with visible dentine: Code 6**
Obvious loss of tooth structure, the cavity is both deep and wide and dentine is clearly visible on the walls and at the base. An extensive cavity involves at least half of a tooth surface or possibly reaching the pulp.

**Smooth surface (mesial and distal)**
This requires visual inspection from the occlusal, buccal and lingual directions.

**Sound tooth surface: Code 0**
There should be no evidence of caries (either no or questionable change in enamel translucency after prolonged air drying (suggested drying time 5 seconds)). Surfaces with developmental defects such as enamel hypoplasias; fluorosis; tooth wear (attrition, abrasion and erosion), and extrinsic or intrinsic stains will be recorded as sound.

**First visual change in enamel: Code 1**
When seen wet there is no evidence of any change in colour attributable to carious activity, but after prolonged air drying a carious opacity (white or brown lesion) is visible that is not consistent with the clinical appearance of sound enamel. This will be seen from the buccal or lingual surface.

**Distinct visual change in enamel when viewed wet: Code 2**
There is a carious opacity or discolouration (white or brown lesion) that is not consistent with the clinical appearance of sound enamel. (Note: the lesion is still visible when dry). This lesion may be seen directly when viewed from the buccal or lingual direction. In addition, when viewed from the occlusal direction, this opacity or discolouration may be seen as a shadow confined to enamel, seen through the marginal ridge.

**Initial breakdown in enamel due to caries with no visible dentine: Code 3**
Once dried for approximately 5 seconds there is distinct loss of enamel integrity, viewed from the buccal or lingual direction.

If in doubt, or to confirm the visual assessment, the CPI probe can be used gently across the surface to confirm the loss of surface integrity.

**Underlying dark shadow from dentine with or without localized enamel breakdown: Code 4**
The lesion appears as a shadow of discoloured dentine visible through the enamel surface beyond the white or brown spot lesion, which may or may not show signs of localized breakdown. This appearance is often seen more easily when the tooth is wet and is a darkening and intrinsic shadow which may be grey, blue or brown in colour.

This lesion appears as a shadow of discoloured dentine visible through an apparently intact marginal ridge, buccal or lingual walls of enamel. This appearance is often seen more easily when the tooth is wet. The darkened area is an intrinsic shadow which may appear as grey, blue or brown in colour.

**Distinct cavity with visible dentine: Code 5.**
Cavitation in opaque or discoloured enamel (white or brown) with exposed dentine in the examiner's judgment.
If in doubt, or to confirm the visual assessment, the CPI probe can be used to confirm the presence of a cavity apparently in dentine. This is achieved by sliding the ball end along the surface and a dentine cavity is detected if the ball enters the opening of the cavity and in the opinion of the examiner the base is in dentine.

**Extensive distinct cavity with visible dentine: Code 6**

Obvious loss of tooth structure, the extensive cavity may be deep or wide and dentine is clearly visible on both the walls and at the base. The marginal ridge may or may not be present. An extensive cavity involves at least half of a tooth surface or possibly reaching the pulp.

**Free smooth surface (buccal and lingual and direct examination of mesial and distal surfaces (with no adjacent teeth)**

**Sound tooth surface: Code 0**

There should be no evidence of caries (either no or questionable change in enamel translucency after prolonged air drying (approximately 5 seconds)). Surfaces with developmental defects such as enamel hypoplasias, fluorosis, tooth wear (attrition, abrasion and erosion), and extrinsic or intrinsic stains will be recorded as sound.

**First visual change in enamel: Code 1**

When seen wet there is no evidence of any change in colour attributable to carious activity, but after prolonged air drying a carious opacity is visible that is not consistent with the clinical appearance of sound enamel.

**Distinct visual change in enamel when viewed wet: Code 2**

There is a carious opacity or discolouration that is not consistent with the clinical appearance of sound enamel. (Note: the lesion is still visible when dry). The lesion is located in close proximity (in touch or within 1 mm) of the gingival margin.

**Localized enamel breakdown due to caries with no visible dentine: Code 3**

Once dried for 5 seconds there is carious loss of surface integrity without visible dentine.

If in doubt, or to confirm the visual assessment, the CPI probe can be used with N0 digital pressure to confirm the loss of surface integrity.

**Underlying dark shadow from dentine with or without localized enamel breakdown: Code 4**

The lesion appears as a shadow of discoloured dentine visible through the enamel surface beyond the white or brown spot lesion, which may or may not show signs of localized breakdown. This appearance is often seen more easily when the tooth is wet and is a darkening and intrinsic shadow which may be grey, blue or brown in colour.

**Distinct cavity with visible dentine: Code 5**

Cavitation in opaque or discoloured enamel exposing the dentine beneath.

If in doubt, or to confirm the visual assessment, the CPI probe can be used with N0 digital pressure to confirm the presence of a cavity apparently in dentine. This is achieved by sliding the ball end along the surface and a dentine cavity is detected if the ball enters the opening of the cavity and in the opinion of the examiner the base is in dentine.

**Extensive distinct cavity with visible dentine: Code 6**

Obvious loss of tooth structure, the cavity is both deep and wide and dentine is clearly visible on the walls and at the base. An extensive cavity involves at least half of a tooth surface or possibly reaching the pulp.
The following are supplemental tooth and surface codes for use in conjunction with the ICPS system. Each will be placed on laminated cards available for use during the clinical examination.

### Table 1 Tooth Status Codes

<table>
<thead>
<tr>
<th>Permanent Tooth Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>U</td>
<td>Unerupted or congenitally absent. No part of the crown is visible in the mouth.</td>
</tr>
<tr>
<td>P</td>
<td>Partially erupted. Deformed surface is insufficiently erupted to be reliably inspected or in the case of an anterior tooth, less than half the tooth is erupted.</td>
</tr>
<tr>
<td>K</td>
<td>Extracted apparently as a result of caries.</td>
</tr>
<tr>
<td>Y</td>
<td>Tooth extracted for orthodontic reasons.</td>
</tr>
<tr>
<td>Z</td>
<td>Tooth avulsed or damaged as a result of trauma (includes crowns or veneers due to trauma).</td>
</tr>
<tr>
<td>W</td>
<td>Tooth crowned apparently as a result of caries.</td>
</tr>
<tr>
<td>R</td>
<td>Roots present apparently resulting from dental caries.</td>
</tr>
</tbody>
</table>

### Table 2 Tooth Surface Codes

<table>
<thead>
<tr>
<th>Surface Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>Restored (either permanent or temporary).</td>
</tr>
<tr>
<td>A</td>
<td>Access cavity or restoration not at site of origin of caries.</td>
</tr>
<tr>
<td>N</td>
<td>Surface unable to be scored. Reasons may include the tooth is not adequately erupted, site is bleeding, has calculus etc.</td>
</tr>
<tr>
<td>S</td>
<td>Sealant.</td>
</tr>
<tr>
<td>T</td>
<td>Traumatized loss of surface tissue not associated with caries (may apply to restored and unrestored surfaces).</td>
</tr>
<tr>
<td>H</td>
<td>Hypoplastic, Abnormal tooth anatomy making surface difficult to inspect adequately.</td>
</tr>
</tbody>
</table>

**Caries codes (CCPS):**

<table>
<thead>
<tr>
<th>Caries Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Sound (stained fissure +/-)</td>
</tr>
<tr>
<td>1</td>
<td>White spot only seen when dry, brown discoloration but not wider than fissure.</td>
</tr>
<tr>
<td>2</td>
<td>White opacity seen when wet, brown discoloration wider than fissure.</td>
</tr>
<tr>
<td>3</td>
<td>Enamel breakdown without dentine visible.</td>
</tr>
<tr>
<td>4</td>
<td>Dentine discoloration shadowing through intact enamel.</td>
</tr>
<tr>
<td>5</td>
<td>Caries.</td>
</tr>
<tr>
<td>6</td>
<td>Extensive cavitation involving at least 1/3 of the surface.</td>
</tr>
<tr>
<td>7</td>
<td>Restored Use Code &quot;F&quot; instead for these data recording.</td>
</tr>
</tbody>
</table>
Appendix X
Clinical Data Collection Form
Appendix XI
Diet Diaries

Food Diary
Oral Health Study

Adhesive label to contain the following information:
Initials, Class, School and ID

Record Days
1.) ......................................
2.) ......................................
3.) ......................................
Instructions For Completing Food Diary

① Please remember to carry this diary with you everywhere for 3 days.

② Write down EVERYTHING you eat and drink.

③ Give as much detail as possible stating – the name of the food, brand, flavour, cooking method, and the time eaten.

④ Give the amount of the food eaten:

- Drinks as glasses, cups or mugs.
- Breakfast cereals as bowlfuls or tablespoons
- Bread as slices (large or small loaf, thick/thin slice)
- Vegetables as tablespoons, portion size or number e.g. peas – 2 tablespoons, 1 medium carrot
- Rice/pasta as tablespoons or portion sizes (small, medium, large)
- Most packaging will often include the amount or weight

⑤ If you make a dish, please give the recipe and the amount that you ate.

⑥ If you make a mistake, don’t worry! Draw a line through and write it again e.g. banana apple

⑦ Finally write down your bedtime.

⑧ You can always ask for help in filling in your diary.

The next page is given as an example on how to fill in the diary.
# Example of completed diary page

**DAY:**  Monday  
**DATE:** 3rd Dec 2009

<table>
<thead>
<tr>
<th>Time</th>
<th>Food / Drink</th>
<th>Cooking Method</th>
<th>Amount Eaten</th>
<th>Office Use Only</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.30AM</td>
<td>Kellogg's Cornflakes with semi-skimmed milk and 1 spoon sugar</td>
<td></td>
<td>1 large bowlful</td>
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<tr>
<td>8.00AM</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>8.30AM</td>
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</tr>
<tr>
<td>9.00AM</td>
<td>Walkers Cheese and Onion Flavour Crisps, Can of Pepsi cola sugared</td>
<td></td>
<td>1 x 25g packet</td>
<td></td>
</tr>
<tr>
<td>9.30AM</td>
<td></td>
<td></td>
<td>1 x 330ml can</td>
<td></td>
</tr>
<tr>
<td>10.00AM</td>
<td>banana, apple</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>10.30AM</td>
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<td>small</td>
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<tr>
<td>11.00AM</td>
<td></td>
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<tr>
<td>11.30AM</td>
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<tr>
<td>12.00PM</td>
<td>Sausages (pork and beef) Mashed potato Semi skimmed milk</td>
<td>Grilled, Boiled</td>
<td>2 medium sized 1 tablespoon</td>
<td></td>
</tr>
<tr>
<td>12.30PM</td>
<td>Mors Bar</td>
<td></td>
<td>62g</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>Food / Drink</td>
<td>Cooking Method</td>
<td>Amount Eaten</td>
<td>Office Use Only</td>
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<td>Food / Drink</td>
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</table>
DAY: _____________________  DATE: _____________________

If there are any times that you eat and drink outside the limits of the food and drink diary please complete the boxes below in the same manner you would with the main diary.

<table>
<thead>
<tr>
<th>Time</th>
<th>Food / Drink</th>
<th>Cooking Method</th>
<th>Amount Eaten</th>
<th>Office Use Only</th>
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</table>
References


DEPARTMENT OF HEALTH (1994a) *Eat well! An action plan from the Nutrition Task Force to achieve the Health of the Nation targets on diet and nutrition*, London, Health Publications Unit.


DEPARTMENT OF HEALTH (1996) *Eat well II. A progress report from the Nutrition Task Force on the action plan to achieve the Health of the Nation targets on diet and nutrition*, London, Health Publications Unit.


