Food Starches and Dental Caries

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ABSTRACT: Sucrose and starches are the predominant dietary carbohydrates in modern societies. While the causal relationship between sucrose and dental caries development is indisputable, the relationship between food starch and dental caries continues to be debated and is the topic of this review. The current view of dental caries etiology suggests that in-depth evaluation of the starch-caries relationship requires the consideration of several critical cariogenic determinants: (1) the intensity (*i.e.*, the amount and frequency) of exposure of tooth surfaces to both sugars and starches, (2) the bioavailability of the starches, (3) the nature of the microbial flora of dental plaque, (4) the pH-lowering capacity of dental plaque, and (5) the flow rate of saliva. Studies of caries in animals, human plaque pH response, and enamel/dentin demineralization leave no doubt that processed food starches in modern human diets possess a significant cariogenic potential. However, the available studies with humans do not provide unequivocal data on their actual cariogenicity. In this regard, we found it helpful to distinguish between two types of situations. The first, exemplified by our forebears, people in developing countries, and special subject groups in more modern countries, is characterized by starch consumption in combination with a low sugar intake, an eating frequency which is essentially limited to two or three meals *per* day, and a low-to-negligible caries activity. The second, exemplified by people in the more modern societies, *e.g.*, urban populations, is characterized by starch consumption in combination with significantly increased sugar consumption, an eating frequency of three or more times *per* day, and a significantly elevated caries activity. It is in the first situation that food starches do not appear to be particularly caries-inducive. However, their contribution to caries development in the second situation is uncertain and requires further clarification. Although food starches do not appear to be particularly caries inducive in the first situation, the possibility cannot be excluded that they contribute significantly to caries activity in modern human populations. The commonly used term "dietary starch content" is misleading, since it represents a large array of single manufactured and processed foods of widely varying composition and potential cariogenicity. Hence, increased focus on the cariogenicity of single starchy foods is warranted. Other aspects of starchy foods consumption, deserving greater attention, include the bioavailability of starches in processed foods, their retentive properties, also in relation to sugars present (starches as co-cariogens), their consumption frequency, the effect of hyposalivation on their cariogenicity, and their impact on root caries. The starch-caries issue is a very complex problem, and much remains uncertain. More focused studies are needed. At present, it appears premature to consider or promote food starches in modern diets as safe for teeth.

Key words. Bioavailability, caries, cariogenicity, eating frequency, microbial flora, pH, plaque, salivation, starch, sucrose.

Introduction

Food starches are often regarded as playing only a minor role, if any, in dental caries development. However, a critical re-examination of the literature, including some pertinent reviews (Newbrun, 1967; van Houte, 1980; Sreebny; 1982; Edmondson, 1990; Burt, 1993; Rugg-Gunn, 1993; Kalsbeek and Verrips, 1994; van Houte, 1994; van Palenstein-Helderman *et al.*, 1996), has left us in doubt as to whether this conclusion is justified. One major concern is that the design and interpretation of existing work often does not reflect currently recognized critical features of dental caries causation. A paucity of data exists also regarding issues such as the potentially cariogenic effects of interactions between starches and sugars that occur often simultaneously in modern diets and the cariogenic role of food starches during hyposalivation or in human root caries development.

To facilitate our discussion of the food starch-caries issue, a brief overview of the major elements of dental caries etiology is provided as a guide. Owing to space limitations and the nature of our review, a broad topic with myriad information of varying relevance and quality, no exhaustive detail has been provided. For this reason, too, review articles have frequently been cited as references.

Dental Caries Etiology

As formulated in the past by Keyes and Jordan (1963), caries development requires a triad of indispensable fac-

tors: (1) dietary carbohydrate, (2) dental plaque bacteria, and (3) caries-susceptible teeth. Because of the indispensability of bacteria, caries is considered to be a bacterial infectious disease. The critical bacterial virulence factor consists of the capacity to convert dietary carbohydrates into organic acids. Bacterial acidogenesis in dental plaque can lead, in certain conditions, to demineralization of the underlying tooth surface; for enamel, this means a loss of mainly Ca and P ions from the principally hydroxyapatite structure (for review, van Houte, 1980, 1994; Loesche, 1986; Tanzer, 1989). Demineralization or remineralization of tooth surfaces (see below) is fundamentally governed by the pH of the plaque milieu and, related to this pH, its degree of saturation with respect to tooth minerals (Ca, P). Net tooth mineral loss occurs during undersaturation and tooth mineral gain during saturation conditions.

Dental plaque covers tooth surfaces as a tightly adherent layer that consists of bacteria, occupying most of its volume, and an inter-bacterial matrix. Plaque is generally separated from the tooth surface by a very thin pellicle which is composed of proteins and glycoproteins of mainly salivary origin. The plaque matrix structure originates also mostly from a variety of salivary components, mainly glycoproteins, and contains various amounts of bacterial extracellular polymers, such as fructose polymers and hetero-polysaccharides and, notably, glucans; the latter are formed specifically from sucrose by, particularly, the mutans streptococci (MS). The plaque matrix confers structural integrity upon plaque by means of adhesive interactions between the bacteria and the matrix components. It also contains diffusion pathways for the transport into and out of plaque of bacterial substrates (*e.g.*, sugars), acid or basic bacterial end-products, ions (*e.g.*, Ca, P, F, CO_3), etc. (for review, Gibbons and van Houte, 1973; van Houte, 1980, 1994; Kleinberg *et al.*, 1982).

The link between plaque acidity (pH) *per se* and cariogenicity depends on the extent of the drop in plaque pH, influenced particularly by lactic acid, and its duration. The former depends fundamentally on the innate capacity of plaque bacteria for acidogenesis and on their acid tolerance, *i.e.*, the tolerance of bacterial growth, survival, and acid production in an acidic plaque milieu. Both capacities are known to vary widely among the plaque bacteria. Three groups of organisms appear currently to occupy a high hierarchical position in this respect, *i.e.*, the lactobacilli (L), the MS, and the recently recognized so-called "low-pH" non-mutans streptococci ("low-pH" non-MS). These organisms are cariogenic in animal models, exist in elevated levels in caries-activity-associated human plaque and in caries lesions, and have, in other ways, been implicated in either mostly the progression (L) or the initiation and progression (MS and "low-pH" non-MS) of human caries lesions (for review, van Houte, 1980, 1994; Loesche, 1986; Tanzer, 1989; see also van Houte *et al.*, 1996; van Ruyven *et al.*, 2000).

The plaque matrix constitutes potentially a second determinant of plaque's pH-lowering ability. Its diffusion properties are not constant, since they depend on its varying composition as well as on its volume. Following the initial findings on the link between sucrose-mediated extracellular polymer (glucans) synthesis and the formation of plaque and caries induced by the MS in experimental rodents (for review, Newbrun, 1967; Guggenheim, 1970), glucans have also become implicated in human plaque and caries development (for review, van Houte, 1994). Thus, glucans may promote the initial attachment of MS to the tooth surface (pellicle), thereby increasing the number of MS-infected sites on tooth surfaces. Glucan synthesis can also contribute to more voluminous plaque formation. The latter has been associated with an increased glucan content of the plaque matrix and, possibly, a glucan-enhanced spacing of the bacterial cells in plaque. Several studies have suggested that such increased bacterial cell spacing provides for a larger volume of the matrix diffusion channels which appears associated with a greater exposure of the plaque bacteria to external fermentable carbohydrates and, hence, a greater pH-lowering ability of plaque, particularly in its deeper layers near the tooth surface. Direct evidence indicating an influence of glucans or of other bacterial extracellular polymers on caries in humans is lacking. The same is true for other matrix factors, such as buffering capacity.

The duration of plaque acidity below what several investigators have simplistically termed the "critical pH" is also highly important. From the dietary standpoint, duration can be influenced by the amount and the frequency of carbohydrate consumption as well as by the bioavailability of carbohydrate (see below) in the mouth and in plaque. Since the classic Vipeholm study (Gustafsson *et al.*, 1953), the frequency of carbohydrate consumption, rather than its amount, has been recognized as a major cariogenic determinant (for review, Sreebny, 1982; Kalsbeek and Verrips, 1994). More recent studies suggest that the relationship between food consumption frequency and caries activity has become less easily demonstrable in certain human populations (for review, Burt and Szpunar, 1994).

Demineralized tooth surfaces can become remineralized *in vivo*, *i.e.*, gain Ca, P, and other ions from the plaque milieu and saliva (Backer-Dirks, 1966; for review, Ten Cate, 1994), and caries development appears to reflect a prolonged imbalance between episodes of de- and remineralization with progressive net tooth mineral loss, leading, eventually, to overt tooth-surface cavitation (for review, van Houte, 1980, 1994; Loesche, 1986; Tanzer, 1989). This dynamic de- and remineralization relationship can be seen as reflecting the consumption frequency of carbohydrates which, if increased, would cause an unfavorable shift toward an increased demineralization time and a decreased remineralization time. However, this simplified formulation hides a complex process involving the definition of "eating occasions", *i.e.*, demineralization time during regular meals as opposed to that during the consumption of a snack (Burt *et al.*, 1988; Firestone *et al.*, 1984), and so on.

More recently, the concept of a triad of indispensable caries factors has been succeeded by a broadened

concept in which dynamic interactions among the following factors are central: (1) dietary carbohydrate, (2) the bacterial composition of plaque, (3) the pH-lowering potential of plaque, and (4) the effects of saliva (for review, van Houte, 1980, 1994). According to this concept, increased exposure of plaque to fermentable carbohydrates induces a shift to elevated levels of highly acidogenic, acid-tolerant organisms, *i.e.*, the L, MS, "low-pH" non-MS, and perhaps other "low-pH" organisms, which, in turn, enhances plaque's pH-lowering (cariogenic) potential. A decreased carbohydrate intake induces the opposite effect. The microbial shifts in plaque appear to reflect microbial selection on the basis of prevailing plaque pH, *i.e.*, more acid-tolerant organisms have a growth advantage over less-acid-tolerant organisms during frequent episodes of plaque acidification. Thus, a critical feature of the current concept is the inclusion of a carbohydrate-driven alteration of plaque's microbial composition for the creation of a plaque-pH-lowering potential which suffices for caries induction.

The pH-lowering carbohydrate effect is antagonized by the action of saliva. Saliva helps to raise plaque pH by promoting the intra-oral (and tooth surface) clearance of food carbohydrates and plaque acids, by its acid-neutralizing buffering action (*e.g.*, bicarbonate), and by providing substrates for base production; it also helps to maintain mineral ion concentrations (*e.g.*, Ca, P) at the plaque-pellicle interface (for review, Kleinberg *et al.*, 1982; Kleinberg, 1985). Hence, caries development can be viewed as reflecting, in essence, (1) the net effect of carbohydrate and saliva as opposing external forces on the pH within the microbial ecosystem that is dental plaque, as well as (2) the pH-regulated plaque flora composition as a determinant of plaque's pH-lowering potential. Caries activity, then, can be seen as a function of both (1) and (2).

The dynamic relationship between dietary carbohydrates and plaque's microbial composition and pH-lowering potential is well-documented (for review, van Houte, 1980, 1994; van Palenstein-Helderman *et al.*, 1996). Drastic short- and long-term reduction of consumption of total carbohydrate (sugars and starches) by humans can cause a numerical reduction of members of the highly acidogenic/acid-tolerant segment of the plaque flora (L and MS), whereas increased carbohydrate intake has the opposite effect. However, even prolonged and drastic reduction of carbohydrate intake does not eliminate the L and MS completely. Likewise, plaque's pH-lowering potential can vary widely. It may be low or negligible, with little or no exposure of the teeth to dietary carbohydrates, *e.g.*, during prolonged stomach tube feeding, or very high, with prolonged, intensive carbohydrate exposure of the teeth.

The question concerning the relative influence of sugars and starches in humans on the observed variations of the plaque levels of L and MS and plaque's pH-lowering potential has not been answered satisfactorily so far. Three different situations would appear to be of particular interest: a high-sucrose diet and a low-sucrose diet, both with substantial starch, and a basically starch-only diet; test substrates for pH determinations would be sucrose and starch. With respect to the plaque levels of L and MS, studies with hereditary fructose-intolerant (HFI) subjects indicate that a diet with extremely low sucrose but substantial starch content (Newbrun *et al.*, 1980) is associated with unusually low L and MS plaque levels (Hoover *et al.*, 1980). This relationship is also found for subjects belonging to the "xylitol group" (see "Studies of the Starch-Caries Issue with Humans") in the Turku studies (Scheinin and Mäkinen, 1975). Data obtained with rats (Guggenheim *et al.*, 1966) and Macaque monkeys (Beighton and Hayday, 1984) suggest the same relationship. However, inoculated MS cells established and persisted on the teeth of de-salivated rats fed a high-starch and a high-sucrose diet at high and equal levels (Bowen *et al.*, 1986).

Short-term lowering of the sucrose content of the diet also leads to a reduced capacity of plaque to lower the pH, with sucrose (Scheie *et al.*, 1984) or gelatinized starch (Dodds and Edgar, 1986) as the test substrate, or to a reduction of lactic acid production, with cola, beer, or chocolate as the test substrate (Sgan-Cohen *et al.*, 1988). The study by Hess and Graf (1975) also suggests a decreased plaque-pH-lowering capacity for HFI subjects with glucose and, particularly, sucrose as test substrates. However, whether the effect on plaque pH is substrate (sugar)-specific is unclear. For example, in the study by Littleton *et al.* (1967), plaque sampled from tube-fed human subjects exhibited a negligible pH response, not only with sucrose but also with glucose and fructose as test substrates. A study by Lingström *et al.* (1994) (see also "Other Studies") has also shown that very frequent consumption of 4 high-starch foods with low-to-negligible sucrose content during 3 weeks, when superimposed upon the normal diet, increased plaque's pH-lowering capacity to a level equal to that induced by the consumption of 4 high-sucrose foods with the same high frequency. The possibility that the effect of the high-starch foods was related to the residual sugar content cannot be excluded.

Collectively, analysis of the above data suggests that if the sugar content of a high-sugar/high-starch diet of humans were to be reduced or eliminated, a significant reduction of the plaque levels of L and MS and plaque's acidogenic capacity (with sugar and starch as test substrates) would occur. However, it is not clear whether the subsequent elimination of the diet's starch component would lead to a further reduction of both parameters. Solid data on the effects of food starches on the plaque pH in subjects on a starch-only diet are unavailable.

Dietary Carbohydrates

(A) SUGARS

Dietary carbohydrates can be divided into simple sugars and complex carbohydrates, *e.g.*, starches. Among the sugars, sucrose is a major component of the modern diet of Western countries and, increasingly, of developing countries (for review, Sreebny, 1983; Rugg-Gunn *et al.*, 1986; Burt

et al., 1988; Woodward and Walker, 1994). Its consumption far exceeds that of other common dietary sugars (glucose, fructose, lactose), and it appears also to be the most frequently consumed sugar. The increasing use of high-fructose corn syrups (*e.g.*, 42% and 55% HFCS) instead of sucrose in beverages, for example, is of more recent date.

Sucrose, upon entering dental plaque, can be readily fermented by a wide variety of plaque bacteria to organic acids which are responsible for caries formation. Sucrose is also unique among the common dietary sugars by serving as a specific substrate for the bacterial synthesis of extracellular polysaccharides (glucans) that have been implicated in caries causation, as noted earlier. A recent *in vitro* study of interest here has indicated that hydrolysates of starch, formed by salivary α-amylase activity, can stimulate sucrose-mediated glucan synthesis by one of the glucosyltransferase enzymes (GtfB); the properties of the glucans formed, that could affect their putative cariogenic effect, also appeared to be different (Vacca-Smith *et al.*, 1996).

(B) Starches

Starches are also a major component of the human diet. Cereal grains are often a primary source of calories, proteins, minerals, and vitamins (Sreebny, 1983; Bibby, 1990; van Palenstein-Helderman *et al.*, 1996). In the Western diet, starches, particularly wheat, are found in a wide variety of foods and constitute a high percentage of total dietary carbohydrate. For example, in Michigan, USA (Burt and Szpunar, 1994) and the United Kingdom (Rugg-Gunn *et al.*, 1986), starch makes up about 50% of total carbohydrate.

(i) Intra-oral bioavailability of starch

Starches are polymers of glucose that vary in length and branching, *e.g.*, amylose and amylopectin, and that form an extensive network of branched chain structures. Starch molecules are located within starch granules, which vary in size within any plant source and even more between sources and can assume a wide variety of shapes (for review, Lineback, 1984). Different starches also have various amylose and amylopectin contents. For example, cereal starches such as oats, wheat, potato, corn, and rice have an amylose content varying from 17 to 28%, although certain varieties of corn, barley, and rice starch contain no amylose and consist entirely of amylopectin (for review, Lineback, 1984).

During food preparation, starch granules are damaged by heat and mechanical forces and undergo a series of changes. Rupture and eventual disintegration of the granules cause a release of the starch molecules in a process called gelatinization. Gelatinization of each starch type occurs over a relatively narrow range of temperatures, usually 8 to 10°C. However, the temperature ranges vary considerably between starches (Lineback, 1984). Further, the water-starch ratio of the starch preparation, *e.g.*, flour, also affects starch gelatinization. For example, the temperature at which rice starch begins to gelatinize increases from 85.3 to 111.3°C when the water-starch ratio drops from 2.0 to 0.75 (French, 1983; Lund, 1983, 1984). The starches in baked products such as bread are prepared with low levels of water and appear to be only partially gelatinized (Osman, 1967). In bread, damaged granules, gelatinized starch, and amylodextrins occur in mixture and contribute to bread formation and texture. The degrees of gelatinization in bread are 5% for the original flour, 35% for the bread crust, and 70% for the bread center (crumb). Similar values have been reported for cakes and doughnuts.

In general, it is only the gelatinized starches that are susceptible to enzymatic breakdown, *i.e.*, are bioavailable. In the human mouth, starches can be hydrolyzed by salivary and bacterial amylases into maltose, maltotriose, and low-molecular-weight dextrins (Mörmann and Mühlemann, 1981). Starch hydrolysis on the teeth is very rapidly initiated, and entrapped particles of food can accumulate high levels of maltose and maltotriose, which are good substrates for bacterial acid production (Neff, 1967; Imfeld, 1983; Kashket *et al.*, 1994, 1996). Maltose fermentation requires bacterial adaptation under certain conditions (Kashket *et al.*, 1994). In humans, however, plaque bacteria appear to be adapted and can ferment this sugar without delay (Neff, 1967; Imfeld, 1983; Lingström *et al.*, 1993a).

Information on the effects of various starchy foods in the human mouth was first obtained by Swenander Lanke (1957). Clearance times for salivary sugars, derived from the starches, were found to be longer for bread than for potatoes or rice. Systematic evidence for the effects of food processing was obtained subsequently in studies involving the pH response of plaque *in vivo* (Björck *et al.*, 1984a,b). For example, suspensions of white wheat flour or starch were highly acidogenic when boiled, but even more so when extrusion-cooked, *i.e.*, cooked at up to 180°C, with low water content and high mechanical agitation. Further, suspensions of wheat flour that were subjected to increasingly severe processing conditions induced an increasing plaque acidogenicity in the order: steam-flaked < dry-autoclaved < extrusion-cooked < drum-dried < popped wheat (Lingström *et al.*, 1989). A higher acidogenicity was also induced by boiled wheat or rye flour than by the corresponding breads, consistent with the greater degree of gelatinization after boiling. It was also shown that the level of plaque pH drop correlated well with the rate of *in vitro* hydrolysis of these products by alpha-amylase (Björck *et al.*, 1984b).

Systemic responses

The significance of the bioavailability factor has been clearly demonstrated systemically, where different starches or starchy foods have been shown to induce different responses in blood glucose and insulin. Glucose in blood is derived from consumed starches following their breakdown in the small intestine to maltose and glucose by pancreatic alpha-amylase and the subsequent hydrolysis of maltose by maltase. The glycemic index (GI) (the measure of post-prandial blood glucose)

and the serum insulin level, or insulin index (II), have been developed for the assessment or ranking of starchy foods (Jenkins *et al.*, 1981). Several studies have shown that starchy foods exhibit a wide range of index values (Jenkins *et al.*, 1981; O'Dea *et al.*, 1981; Granfeldt and Björck, 1991; Granfeldt *et al.*, 1992; Foster-Powell and Miller, 1995). Thus, the GI values for bakery products and some breakfast cereals are very high; those of vegetables, rice, and spaghetti are lower, and those for fruit and dried legumes are the lowest. Foods such as spaghetti induce relatively slow but prolonged elevations in blood glucose (the so-called "lente" response; Jenkins *et al.*, 1994).

Modern commercial processing methods such as high-temperature extrusion cooking, explosion puffing, toasting, or "instantization" with repeated wetting and drying, render food starches more digestible (bioavailable) than boiling or baking (Brand *et al.*, 1985; Ross *et al.*, 1987). For example, corn flakes and instant rice induce considerably higher GI and HI (hydrolytic index, or the measure of hydrolysis rate by pancreatic amylase *in vitro*) values than do the corresponding non-processed foods, sweet corn and boiled rice. Nevertheless, some foods with incomplete gelatinization (*e.g.*, shortbread biscuits and quick-cooking wheat) can elicit a high GI (Granfeldt *et al.*, 1995). These findings suggest that starch needs to be only partly gelatinized for the indices to be raised. Other factors, such as starch-protein and starch-lipid interactions, or food texture and fiber content, have also been proposed as modifiers of starch bioavailability and contributors to the GI and II of different foods (Björck *et al.*, 1984a,b).

Applications to cariology

Based on the above, it may be concluded that the genetically based make-up of starches and differing cooking and food-processing methods can critically affect the bioavailability, and presumably the cariogenicity, of food starches in the mouth. Variation of the bioavailability of starch in foods is well-recognized in the medical arena and forms the basis for food-ranking (Jenkins *et al.*, 1981; Granfeldt and Björck, 1991; Granfeldt *et al.*, 1992; O'Dea *et al.*, 1981; Foster-Powell and Miller, 1995), nutritional advice for patients with diabetes and other anomalies of sugar metabolism (Thorburn *et al.*, 1986; Jenkins *et al.*, 1988), and has been applied to sports training and research on appetite. Regrettably, variation of starch bioavailability has been largely unrecognized by cariologists.

(ii) Starch consumption frequency and retention

The exposure of plaque bacteria to the starch-derived sugars, glucose and maltose, is influenced not only by bioavailability but also by starch consumption frequency and starch retentiveness. In contrast to the situation for dietary sugars (sucrose), recorded food consumption frequency data for dietary starches are few. Populations that consume primarily "pure" starchy foods with very few sugars, *e.g.*, African peoples (van Palenstein-Helderman *et al.*, 1996), people in New Guinea (Schamschula *et al.*, 1978) or China (Afonsky, 1951), do so only during two or three meals *per* day. In contrast, populations that consume high-sugar/high-starch diets, *e.g.*, Western populations, do so with an increased consumption frequency extending beyond regular meals. Besides "pure" sugary or starchy foods, such diets contain a wide array of foods consisting of a mixture of sugars and starches in widely varying proportions (Martinsson, 1972; Holm *et al.*, 1975; Ismail, 1986; Rugg-Gunn *et al.*, 1987). Starch consumption frequency for North American subjects has been reported to be about 3.0 or 2.5 *per* day for subjects with or without root caries, respectively; that for sugars, including sugar-starch mixtures, was 7.2 or 5.1 *per* day, respectively (Papas *et al.*, 1995). The consumption frequency of pure starches or sugar-starch mixtures *per se* was not given. A similar problem of interpretation of such data exists with other studies, *e.g.*, Hankin *et al.* (1973). Existing evidence indicates a considerable increase of consumption of between-meal starch during the recent decennial (*e.g.*, Bibby, 1990).

Evidence indicating increased stickiness of starches or starchy foods in the human mouth dates back many years (Bibby *et al.*, 1951; Gustafsson *et al.*, 1953; Ludwig and Bibby, 1957; Caldwell, 1970). However, the measurements in these studies were largely of whole-mouth retention and included retention on the soft tissues and in soft-tissue spaces. The influence, if any, of retained food particles in these locations on the caries process is not clear, and hence these studies are of limited value. More recent work has focused specifically on the retention of foods on the teeth (Kashket *et al.*, 1991). Particles of foods containing high levels of starch were found to be retained in larger amounts than foods that contained relatively little starch, but high levels of sucrose and other fermentable sugars. An interesting example of the former was potato chips. These were retained in large amounts and were found to accumulate the starch breakdown products, maltose and maltotriose, within the retained food masses. The significance of retention and starch breakdown on the cariogenic process was illustrated by the finding that potato chips exhibited a high cariogenic potential (Lingström *et al.*, 1997) (see also "Studies of the Starch-Caries Issue with Animals").

Studies of the Starch-Caries Issue *with* Humans

The classic Vipeholm study (Gustafsson *et al.*, 1953), the Hopewood House experiments (Harris, 1963), the Turku studies (Scheinin and Mäkinen, 1975), and the study of HFI individuals (Newbrun *et al.*, 1980) have been cited frequently as indicating that food starches are not cariogenic in humans (*e.g.*, Newbrun, 1982; Beighton and Hayday, 1984; Rugg-Gunn *et al.*, 1987). However, this interpretation has often failed to take into account critical caries factors such as frequency of sugar or starch consumption, plaque-pH-lowering potential, starch bioavailability, or starch-sugar interaction.

In the main part of the Vipeholm study, all subjects belonged to different diet groups and consumed 3 different basic diets. The first was consumed during the

first year, the second during the following 2 years, and the third was consumed during years 4 and 5. All basic diets were eaten only at 3 meals and varied with respect to amounts of total starch, total sugars, and sucrose. A control group consumed all basic diets in unsupplemented form throughout the study. Other diet groups, after a first year of unsupplemented diet, received the basic diet with different supplements for different groups, consisting of sucrose in beverages or food at meals only, caramels or toffees (8/day) either immediately after breakfast and lunch or between meals, and toffees (24/day) at and between meals.

The major finding of this study is the dominant cariogenic effect of food consumption frequency. For example, with food consumption only at meals, all of the basic diets even when supplemented with a high amount of sucrose, induced only little caries. However, supplementation of the basic diet with foods, *i.e.*, caramels or toffees providing the same or less total sucrose, induced a much higher rate of caries when given immediately after breakfast and lunch, or between meals.

In the Vipeholm study, neither starches nor sugars, provided at meals, induced significant caries activity. Also, since all foods, consumed at or between meals, consisted of a mixture of starches and sugars, the findings cannot be interpreted with respect to the impact of starch consumption between meals on caries development. Finally, starch-sugar interactions, resulting from prolonged starch retention on the teeth, could also have contributed also to caries development (see section "Dietary Carbohydrates—Starches").

The children at Hopewood House consumed a lacto-vegetarian diet, *i.e.*, many uncooked vegetables, fruits and dairy products, little refined carbohydrate (about 17 grams/day), and starchy foods, *i.e.*, toast, potatoes, rice, corn. The total dietary carbohydrate content was about 240 grams/day. There were 3 regular meals, with milk upon rising and milk or fruit before dinner. The children's caries experience was very low, whereas that of non-institutionalized state school children of similar age, eating a normal diet, was far higher. Data on dietary starch content and plaque-pH-lowering potential were not obtained.

In another study (Newbrun *et al.*, 1980), HFI subjects consumed a diet with little sucrose (2.5 grams/day) and a total carbohydrate content (starches, sucrose, other sugars) of about 160 grams/day, which, as implied by the authors, included a major amount of starch. Although the data do not show whether the subjects ate at regular meals only or also between meals, HFI subjects are known to develop little or no caries (see also Marthaler and Froesch, 1967).

The Vipeholm and Hopewood House studies suggest that food starches are not particularly caries-inducive when eaten during essentially 3 regular meals. In most subjects who consumed 3 meals only, some caries developed, but it is unclear whether this was due to dietary starches, sugars, or both.

The Turku studies involved 3 groups of subjects who consumed a diet prepared with either sucrose, fructose, or the non-fermentable sweetener xylitol. Subjects of the "xylitol group" developed little or no caries, whereas those of both other groups developed considerably more. All diets had a significant starch content (Mäkinen, personal communication), whereas food consumption frequency was not recorded. Also, the subjects of the "xylitol group" accumulated less plaque and exhibited a diminished acidogenic/acid-tolerant segment of the plaque flora (MS and L). Definitive interpretation of these studies with respect to the starch-caries relationship is not possible, due to reports that suggest the possibility of an anti-caries effect of xylitol. Thus, the presence of this sugar alcohol has been associated with a significant reduction of the cariogenic effects of sugar- or starch-containing diets (for review, Mäkinen *et al.*, 1995; Tanzer, 1995; Trahan, 1995).

In other studies (Rugg-Gunn *et al.*, 1987), it has been found that a group of subjects on a high-starch/low-sugar diet (187 grams of starch/day and 87 grams of sugar/day) did not have a significantly lower caries increment (DMFS = 2.8) during two years than did subjects on a low-starch/high-sugar diet (125 grams of starch/day and 143 grams of sugar/day; DMFS = 4.1). The former had a lower eating frequency than the latter (5.7/day *vs.* 7.8/day), but partial correlation analysis failed to indicate a positive relationship between eating frequency and caries increment. This study does not rule out a cariogenic role of starches.

Studies of 12-year-old children from various countries who consume a diet consisting of various types of starches as the primary energy source and little sucrose (< 18 kg/*capita*/year), taken only during two or three main meals, indicate a low-to-negligible caries experience (Sreebny, 1983; van Palenstein-Helderman *et al.*, 1996). The same situation is found in certain populations in China (Afonsky, 1951). By contrast, the caries experience of certain European and North American children of the same age is much higher; these children consume high-starch diets with increased sucrose levels (18 to 45 kg or more/*capita*/year), with an increased frequency. The staple food of people in a primitive society in Papua New Guinea is sago (starch without detectable sugars) (Schamschula *et al.*, 1978). They consume little refined sugar or processed foods. Foods such as fish and vegetables are obtained locally. Caries activity varies markedly between individuals (0-22 DM|F|T) and between villages (0-8.7 DM|F|T). For these people, the consumption of a high-starch diet with negligible sugar, during only two meals *per* day, appears to be linked to plaque with a significant pH-lowering capacity and a moderate-to-high caries activity in some villages (see also Bibby, 1990).

Studies of the Starch-Caries Issue with Animals

The use of experimental animals such as rats or hamsters has allowed for the study of the starch-caries relationship under more controlled conditions. Control of the amount and frequency of food consumption is possible

with programmed feeding (the König-Hoffer apparatus; König *et al.*, 1968; Grenby and Mistry, 1995). The method of Bowen *et al.* (1980) allows for the evaluation of the specific effects of test foods by the administration of basic nutrients by stomach tube and test foods by mouth. Caries development in rodents is greatly influenced by the type of plaque flora, *i.e.*, the indigenous flora only or supplemented with the progeny of single or multiple artificially introduced test strains, and the type of carbohydrate used. Both parameters influence the amount, localization, and acidogenic potential of plaque on the teeth, due to variation of the acidogenic/acid-tolerant and plaque-forming ability among bacteria.

The classic animal model involves MS-free rats which are fed a high-sucrose diet and are inoculated with S. *mutans* cells. The teeth of such rats often exhibit multi-surface plaque deposits and extensive caries; the latter appears to reflect a combination of enhanced plaque formation linked to sucrose-mediated glucan synthesis by S. *mutans*, and the organism's high acidogenicity and acid tolerance. If the dietary sucrose is replaced by glucose or by starch, and glucan synthesis does not occur, development of plaque mass may be impaired, particularly on smooth tooth surfaces. Hence, caries may mainly occur mainly in tooth fissures (for review, van Houte, 1980, 1994). The same is true if bacterial test strains other than S. *mutans* are used that are incapable of glucan synthesis. This variation of plaque mass and plaque localization may hinder data interpretation. For example, in S. *mutans*-inoculated animals fed a starch or a sucrose diet, the plaque mass may be unequal and, hence, may complicate comparison of the cariogenicity of starch and sucrose (Bowen *et al.*, 1980).

Studies of the starch-caries issue before the early 1970s involved mostly rodents with only their indigenous flora. Subsequent work has been done often with animals with a supplemented flora (particularly strains of S. *mutans*, S. *sobrinus*, and A. *viscosus*, singly or in combination) and *ad libitum* feeding, programmed feeding, and the intubation method. The studies have tested a variety of raw starches, variously processed "pure" starches, and starchy foods. Generally, untreated raw starches (*e.g.*, corn, wheat) exhibit negligible cariogenicity (Guggenheim *et al.*, 1966; Grenby, 1966, 1970; Hefti and Schmid, 1979; Grenby and Colley, 1983; Havenaar *et al.*, 1984; Horton *et al.*, 1985). Processing of pure corn, wheat, potato, tapioca, amioca, sorghum starches (*e.g.*, boiling, roll-drying) greatly enhances their cariogenicity and affects various starches differently (Grenby, 1965, 1967; Green and Hartles, 1967; Frostell and Baer, 1971a,b; Schmid *et al.*, 1987). Amylopectin and amylose can also be considerably cariogenic (Frostell and Baer, 1971a; Birkhed *et al.*, 1980).

Studies of the effects of pure starches in combination with other carbohydrates are often difficult to interpret with respect to the effect of starch *per se* (König and Grenby, 1965; Green and Hartles, 1966; König, 1967; Ishii *et al.*, 1968). Studies involving dietary ingredients such as skim milk or liver powder, which contain a significant amount of sugar, suffer from the same restrictions (Green and Hartles, 1967; Frostell and Baer, 1971a,b; Horton *et al.*, 1985).

Studies with processed pure starches such as wheat, corn, potato, amioca, tapioca, and arrowroot starches have shown that all exhibit major cariogenic potential that sometimes exceeds that of sucrose (Frostell and Baer, 1971a,b; Shaw and Ivimey, 1972; Firestone *et al.*, 1982; Mundorff and Curzon, 1985). On the other hand, cooked maize and sorghum, both staple foods for black South Africans, have exhibited only a low-to-moderate cariogenic potential compared with a mixture of cooked wheat starch and sucrose (Schmid *et al.*, 1987). Foods with a high-starch/low-sugar (1.5% or less) content, such as cereal, potato chips, French fries, corn chips, pretzels, or bread, can also induce high cariogenicity (Grenby, 1970; McDonald and Stookey, 1977; Bowen *et al.*, 1980; Mundorff and Curzon, 1985; Mundorff *et al.*, 1990).

In Macaque monkeys, little or no caries developed with a high-cooked wheat starch diet with little sucrose; caries activity was greatly enhanced during a subsequent "high-sucrose" diet period (Beighton and Hayday, 1984). Although it was concluded that cooked starch products are virtually non-cariogenic in humans, possible differences with respect to plaque-pH-lowering potential during both diet periods or the increased amount as well as consumption frequency of sucrose as compared with starch were not considered.

Studies with rats indicate the possibility of a caries-enhancing interaction between starches and sugars. In experiments with programmed feeding and intubation together with super-infection with S. *mutans* and *Actinomyces*, Firestone *et al.* (1982, 1984) found that cooked wheat starch was about as cariogenic as sucrose, but that a mixture of the two was more cariogenic. They attributed this effect to starch retention, which prolonged the contact time of the starch-sucrose mixture with the teeth. In studies with humans, Firestone (1982a,b) showed that the pH of the plaque was lower and remained low longer after its exposure to sucrose mixed with a sticky non-fermentable substrate (guar gum, a galactose mannan) than to sucrose alone. Analysis of these data suggests that starch, besides being a source of fermentable sugar for plaque organisms, may also function as a "co-cariogen" and promote the retention of sugar (sucrose) on the teeth (see also "Studies of the Starch-Plaque pH Relationship in Humans").

The function of starch as a "co-cariogen" is supported by other studies. In two separate tests, pure powdered sucrose, provided in 17 meals, induced lower caries scores (tooth fissures) than did chocolate cookies with soft filling (Bowen *et al.*, 1980). In a second, more elaborate, study with this cookie (a starch-sucrose mixture), it was again found to be more cariogenic than pure sucrose (Bowen *et al.*, 1983). Foods such as French fries (0.8% total sugars, 0.5% sucrose) were also found to be more cariogenic than the sucrose controls; the same was true

for sucrose-starch combinations such as cupcakes (50:1 ratio), banana (30:1), raisins (2:1), or French fries (1:15) (Mundorff *et al.*, 1990).

Studies of the Starch-Plaque pH Relationship in Humans

Studies of the effect of starch on the pH response of plaque in humans also have involved raw starches, pure processed starches, and starches in foods. Different plaque pH methods—such as the "sampling" technique, the touch/microtouch technique, and the indwelling/telemetric method—have been used (Harper *et al.*, 1985b; Lingström *et al.*, 1993b). These methods yield pH profiles with different shapes and pH minima (Schachtele and Jensen, 1982; Lingström *et al.*, 1993b). The different studies also vary with respect to the nature of the exposure of plaque to test substrates. Thus, there have been significant variations in the quantities of food administered, concentrations of sugar solution in rinses, times of food consumption or rinsing, degrees of food retention, types of dentition area used for pH determination (*i.e.*, retentive *vs.* non-retentive), etc. Plaques in different subjects or dentition sites, or even in the same sites but at different times, are known to vary greatly in their pH response due to salivary and microbiological factors. A necessary sugar control has been used only infrequently.

Nevertheless, some general conclusions can be drawn which appear to parallel those obtained from the animal studies. Untreated raw starches cause, at best, only a limited plaque pH drop, whereas an enhanced pH drop occurs when starch is subjected to different heat processes which cause gelatinization and further breakdown (Neff, 1967; Graf, 1969; Frostell, 1970, 1972; Birkhed and Skude, 1978; Mörmann-Buchmann, 1979; Mörmann and Mühlemann, 1981; Lingström *et al.*, 1989, 1993a,b). Processed pure wheat, corn, rice, rye, and potato starch can all cause a significant plaque pH drop (Mörch, 1961; Mörmann and Mühlemann, 1981; Imfeld, 1983; Lingström *et al.*, 1989, 1993a,b). Glucose and maltose, both breakdown products from starch, can also cause a large plaque pH drop which is comparable with that of sucrose (Mörch, 1961; Frostell, 1964; Neff, 1967; Imfeld, 1977, 1983).

Several studies have dealt with plaque pH effects induced by a variety of starchy foods (Mörch, 1961; Edgar *et al.*, 1975; Rugg-Gunn *et al.*, 1978; Schachtele and Jensen, 1981, 1984; Jensen and Schachtele, 1983; Bibby *et al.*, 1986; Lingström *et al.*, 1993a,b; Pollard, 1995). Generally, they indicate that foods containing starches and sugars can induce major plaque pH drops. As noted earlier, tests with such complex foods do not provide, with few exceptions, specific data on the effect of starch *per se*. Sugars, even in low concentration, can significantly affect plaque pH (Kleinberg, 1961). Hence, for starchy foods with low sugar content, differentiation between starch-induced acidogenesis and starch-related retention of the sugar with regard to plaque's pH response is difficult. It is of interest here that several studies have shown a different pH response to sugary and starchy foods (Jensen and Schachtele, 1983; Bibby *et al.*, 1986; Park *et al.*, 1990). Sugary foods often induce a low pH minimum and a rapid return of the pH toward neutrality. On the other hand, consistent with a retention effect, starchy foods often induce a prolonged pH drop, with the total time and extent to which the pH remains below the "critical pH" exceeding that of the sugary foods. "Pure" starchy foods—such as boiled potatoes (Mörch, 1961), unsweetened bread (Swenander Lanke, 1957), boiled rice (Pollard, 1995), or potato chips (Edgar *et al.*, 1975; Schachtele and Jensen, 1984; Harper *et al.*, 1985a; Bibby *et al.*, 1986)—containing few or no sugars other than those derived from starch, have also been shown to induce significant pH drops that may be close to those obtained for sucrose.

Other Studies

In vitro methods—including the prolonged incubation of test substrates with saliva and measurement of acid production (Bibby *et al.*, 1951; Beck and Bibby, 1961), or exposure of harvested human plaque to a solution of the test substrate at 37°C for 30 min (Bibby and Krobicka, 1984)—have shown that acidogenesis from different starches and starchy foods varies widely. The minimum plaque pH values were found to be highest for raw wheat, corn, or potato starches, lower for boiled starches, and lower still for more refined products such as polished brown rice.

An intra-oral method has been developed that follows de- or remineralization of blocks of human enamel or dentin, or of bovine enamel, that are covered by a layer of *S. mutans* cells so as to mimic dental plaque (Brudevold *et al.*, 1985; Kashket *et al.*, 1994). Studies with this model have shown that raw and cooked wheat starch induced negligible and mild demineralization, respectively (Brudevold *et al.*, 1985), whereas boiled gelatinized wheat starch gels (10, 15, or 20% starch) induced significant demineralization and significant pH drops (minimum pH of 4.8-5.0) (Brudevold *et al.*, 1988). In the study by Kashket *et al.* (1994), pH drops as low as pH 4.2-4.3 were obtained with unsweetened cookies prepared in the laboratory, where wheat starch was the sole carbohydrate source as well as sweetened, sucrose-supplemented cookies.

Lingström *et al.* (1994) and Pollard (1995) used slabs of human enamel or dentin that were covered by naturally formed plaque, and demineralization was measured as changes in microhardness or microradiography. The consumption of bread (wheat flour), boiled potato, potato chips, and Cheeze Doodles (maize flour), with sugar contents of 4.6, 0.5, 0.3, and 3.3%, respectively, 12-15 times/day during three weeks increased demineralization of enamel and dentin compared with that during a 21-day control period without the foods. The consumption of four high-sugar, boiled products induced only slightly more demineralization than did the starchy products (Lingström *et al.*, 1994). Pollard (1995) found that starchy foods such as breads, corn flakes, bran flakes, boiled rice, and spaghetti induced significant enamel demineralization following immersion of enamel slabs

that were held in a dental appliance in a test food suspension (3% w/v) 4 times/day for 10 min each time during a five-day test period. The level of demineralization by rice and white bread approximated that of the sucrose solution, whereas that of spaghetti was about one-half that of the sucrose solution. No demineralization was induced by the poorly-bacterium-fermentable sugar alcohol sorbitol.

Other Aspects of the Starch-Caries Issue

(A) Hyposalivation

Saliva functions as a powerful regulator of caries activity, and its interaction with other cariogenic determinants has been well-documented (see "Dental Caries Etiology"). For example, caries activity in the human dentition is lowest in the lower anterior area. Here, the relatively high exposure to saliva appears to be related directly to relatively low plaque proportions of MS, L, and "low-pH" non-MS, and a relatively weak pH-lowering capacity of plaque *in vitro* as well as *in vivo*. In contrast, individuals with highly reduced salivary flow, *e.g.*, in the case of xerostomia ("dry mouth"), exhibit a dramatically increased caries activity. This is accompanied by increased exposure of the teeth to dietary carbohydrates, increased oral levels of MS and L, and an increased plaque-pH-lowering capacity (for review, van Houte, 1980, 1994).

Hyposalivation in humans appears to be increasing, due particularly to the use of medicines such as neuroleptics, tricyclic anti-depressants, and anti-hypertensives (Bertram *et al.*, 1979; von Knörring and Mörnstad, 1981; Parvinen *et al.*, 1984; Risheim *et al.*, 1992). As during xerostomia, hyposalivation could augment the cariogenic potential of sugars as well as starches by causing a decrease of carbohydrate clearance from the teeth, buffering activity, clearance of plaque acids, alkali production, or mineral ion (Ca, P) availability to plaque. In the case of starches, these effects could be opposed by the reduced availability of salivary α-amylase necessary for the breakdown of starches to simple sugars.

Few data exist on the cariogenic effects of food starches during prolonged hyposalivation. In a study with de-salivated hamsters fed a high-corn starch (untreated) diet, also containing 30% powdered whole milk, moderate caries was induced; caries development with a high-sucrose diet occurred much more rapidly and was more extensive (Klapper and Volker, 1953). In a study by Bowen *et al.* (1986), de-salivated rats, super-infected with a strain of MS (*S. sobrinus*) and of *Actinomyces* and fed a base diet with either 56% glucose, sucrose, fructose, or starch (untreated), all developed root caries. The caries scores were highest with sucrose, but were similar for the other sugars and starch. In another study (Lingström and Birkhed, 1993), the salivary flow rate in humans was lowered with methylscopolamine, and the plaque pH was recorded after the administration of a starchy food (potato chips; 0.3% sucrose), boiled starch or sucrose solutions (5% w/v each). Restriction of salivary flow enhanced the pH drop, and under this condition, the pH minima for potato chips (5.64) and boiled starch (5.66) solution were comparable but higher than for the sucrose solution (5.26). The potato chips induced also a prolonged reduced pH drop during salivary restriction as compared with the sucrose solution. This suggests, again, a starch-related retention effect. Sugar-low Cheeze Doodles and sweetened crackers (3.4% and 19% of total sugars, respectively) induced the same effect. Salivary restriction also promoted the oral retention of all three foods. In a study of the intra-oral demineralization of enamel, induced by a variety of foods, demineralization was found to be highest when the stimulated salivary volumes were the lowest (Pollard, 1995).

(B) Root caries

In the above study by Bowen *et al.* (1986), de-salivated rats developed root caries on a starch diet. There is evidence which suggests that root caries develops under less stringent conditions of plaque acidity than does enamel caries. Hence, it is possible that starchy foods may exert their cariogenic effect not only on enamel but also on the roots of teeth. For example, it is well-known that, in rats, moderately acidogenic and acid-tolerant strains of *Actinomyces* can induce significant caries in roots but not in enamel (for review, Jordan, 1986). Studies with roots (cementum, dentin surface) from human teeth that had never been exposed to the oral environment have also indicated that the "critical pH" for root surfaces, *i.e.*, the pH of plaque fluid at which tooth mineral is just in equilibrium with the plaque fluid, is much higher than that for enamel surfaces (Hoppenbrouwers *et al.*, 1987). The authors speculated that this might explain epidemiological data indicating that sugar-consuming populations suffer from enamel as well as root caries, whereas populations that consume starches but little sugar experience only root caries (Hardwick, 1960; Schamschula *et al.*, 1972, 1974; Banting and Courtright, 1975; Moore, 1983; Kitamura *et al.*, 1986; Burt, 1993). Extrapolation from the above studies is complicated by the fact that an anti-caries effect of fluoride probably did not affect the results, whereas, at present, human root surfaces are generally significantly exposed to fluoride in various forms.

Concluding Remarks

Modern diets of urban populations contain a wide array of manufactured and processed foods, refined flours, foods consisting of mixtures of starch and sugars, foods with a soft consistency, and so on. The cariogenic impact of the starch component of such diets is derived from a large variety of single foods, each of which contains one or several types of starches in various stages of gelatinization, is consumed at various frequencies, possesses different tendencies for retention on the dentition, and may contain caries-promoting or -inhibitory ingredients. The term "dietary starch", then, refers to a mixture of starch products with apparently widely varying potentials to serve as substrates for bacterial acidogenesis in

plaque and, hence, to induce cariogenesis. The term "dietary starch content", used customarily in connection with the starch-caries issue, does not give due credit to this complexity and may therefore be misleading.

Support for the cariogenicity of food starches in humans can be found in various types of experimental studies. Thus, animal studies involving plaques with significant pH-lowering capacity and a high food consumption frequency have shown that different processed pure starches or starchy foods with negligible sugar content can promote caries development. Studies of demineralization in humans, involving intra-orally-carried, plaque-covered enamel or dentin slabs, have provided a similar picture. Many processed pure starches or starchy foods with little or no sugar can also induce significant plaque pH changes in humans that, in some cases, parallel their cariogenicity in animals (Mundorff and Curzon, 1985). Of course, extrapolation from these studies to the human situation should be done with caution. Even the assessment of the significance of plaque pH differences between sugars and starches in humans *in vivo* is hazardous, because quantitative aspects of the plaque-pH/caries-activity relationship are still unclear.

The above studies indicate also that starch products can be, but frequently are not, as effective as sucrose in inducing enamel caries, enamel demineralization, or plaque pH responses in humans. This relative ineffectiveness appears to be related to the lower bioavailability of starches and the resulting diminished delivery of glucose and maltose to the plaque bacteria. The innate make-up of starches and the widely varying conditions of food processing are likely to be the most important factors that determine bioavailability.

Caries development in humans appears to require a plaque acidity which exceeds critical levels of degree and duration. Since food contact time can be severely limited in the mouth, the bioavailability of food starches assumes major significance in terms of cariogenic impact. Evidence indicates that impaired starch bioavailability, consequently limiting cariogenicity, could be compensated for, in some part, by the enhanced retentiveness of starchy foods. Besides prolonging the presence in plaque of starch-derived simple sugars, starch retentiveness could also prolong the effects of simultaneously-present sugars, *i.e.*, starch may act as a co-cariogen. Increased retention of starch products has been shown in animals as well as directly in humans and is also indicated by the prolonged plaque pH response after the consumption of starchy foods by humans.

Studies with humans which bear on the starch-caries issue are of a varied nature. Their evaluation can be facilitated by the recognition of two distinctly different situations. In the first, there is a combination of a high-starch diet with a low or negligible sugar (sucrose) content, food consumption during two or three meals and little or no food in between, and a low-to-negligible caries activity. The second represents a combination of a high-starch/high-sugar diet, food consumption during and with various frequencies between regular meals, and a moderate to very high caries activity.

Populations that fit the first situation include early primitive man, people in developing countries, and certain special groups in more developed cultures, such as the Hopewood House children and HFI subjects. Historic evidence indicates a specific association between starch consumption and a low level of caries (primarily root caries) for our forebears. However, with respect to their starch component, diets from people in antiquity or present-day developing countries differ dramatically from modern Western diets. The diets of the former consist of only a limited spectrum of simple starch products, *e.g.*, cooked maize, sorghum, millet, and cassava flours in unrefined or slightly refined forms (African children) (van Palenstein-Helderman *et al.*, 1996), or crude polished rice in high amounts (Chinese students) (Martinsson, 1972). Most likely, the starches were less gelatinized and of lower cariogenicity than those consumed in developed countries. The Hopewood House children and HFI subjects represent people with a more modern diet. However, here, a low frequency of food consumption (mostly starches) or the bioavailability of the particular food starches consumed, coupled with a possibly reduced plaque-pH-lowering potential owing to the low level of sugars, could also have contributed to the low caries activity observed.

The second situation differs from the first one not only with respect to dietary sugar content or food consumption frequency but also with respect to factors such as a likely increased bioavailability of starch-derived sugars in modern foods, possible starch-sugar interactions, and a possibly increased plaque-pH-lowering potential. None of the major studies with humans, including the Vipeholm study, sheds much light on the starch-caries issue in this regard. Hence, further research with a focus on the above parameters appears desirable.

In view of the widely varying pH-lowering and cariogenic potentials among starchy foods, increased focus on single foods rather than merely on "the dietary starch content" appears also warranted. Guidelines regarding caries risk for sugary foods should be further supplemented by similar guidelines for "pure" starchy foods as well as for foods consisting of starch-sugar mixtures. Conceivably, the creation of such guidelines might benefit from knowledge obtained in other disciplines, *e.g.*, the use of glycemic indices to evaluate starchy foods for their impact on blood glucose levels (Jenkins *et al.*, 1981).

In view of the guidelines for caries risk, established on the basis of plaque pH in humans (Scientific Consensus Conference on Methods for Assessment of the Cariogenic Potential of Foods; DePaola, 1986), the work by Imfeld (1977) and others suggests that many types of starchy foods, including those with negligible sugar content, can be considered "unfriendly to teeth", *i.e.*, are considered to constitute a caries risk. Since plaque pH is not necessarily a true indicator of a food's cariogenic potential, pH data should be supplemented

with corresponding animal studies. Finally, the significant issue that starches may exert a greater cariogenic effect in the case of hyposalivation (decreased protection of the tooth surface), or on root surfaces (greater caries susceptibility than enamel surfaces), or, we may add, during the progression of coronal caries lesions into the dentin, warrants more attention.

The reviewed evidence suggests that the complex starch-caries issue is far from resolved. Various types of experimental studies indicate that food starches may possess significant cariogenic potential. The studies with humans, unfortunately, have yielded only limited information and appear inconclusive. Further studies of the starch-caries issue should take into account a variety of factors, such as the variation of starch bioavailability and, hence, the different cariogenic potentials of different food starches, the consumption frequency of starchy foods, the potential interaction between food starches and sugars, the pH-lowering potential of plaque, and the cariogenic impact of food starches during reduced salivary flow or on root caries development. Current knowledge suggests that it is premature to consider food starches in modern diets as safe for teeth.

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