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# A Study of Risk Factors for Early Childhood Caries in Rural El Salvador

Ву

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Spring 2005

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University of California, Berkeley

Spring 2005

Risk Factors for Early Childhood Caries in Rural El Salvador

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By Veronica Anne Jordan

# **DEDICATIONS**

To Marilyn Jordan, the most amazing mother in the world.

To the mothers and children of El Salvador, may we help one another find health.

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# CHAPTER 1: A Review of the Early Childhood Caries Literature

### I. Introduction

"A child who has poor dental health is not a healthy child, and any efforts to improve child health which do not address oral health are certain to fail" [1].

Dental caries is an infectious disease of the teeth in which organic acids, produced by the metabolism of oral microorganisms, lead to demineralization and destruction of tooth structure. Early Childhood Caries (ECC) is a condition that affects the deciduous dentition (i.e. primary or baby teeth) of children under the age of six. ECC has also been called *nursing caries*, *rampant caries*, *maxillary anterior caries*, and *baby bottle tooth decay*.

The process of ECC begins with colonization and infection by microorganisms usually acquired from the mother's mouth, accumulation of the organism with repeated carbohydrate exposure (e.g. via the nursing bottle or frequent carbohydrate-containing snacks), and finally, demineralization and cavitation of the tooth structure. Primary features of ECC include the involvement of many teeth (the four maxillary incisors are most often affected), rapid lesion development, carious lesions on surfaces generally considered to be low risk for decay (e.g. the facial surfaces of maxillary anterior teeth and the lingual surfaces of the posterior teeth), and the absence of decay of the mandibular incisors [2]. Although it is a widely recognized phenomenon worldwide, there is currently no standard definition for ECC.

Whereas the ECC prevalence in developed countries is estimated to be around 5% [2], reports of ECC in the developing world are significantly higher. A 1990 review titled Caries in the Preschool Child: International Trends summarizes a small number of

studies from countries in Southeast Asia, Africa, and Latin America [3]. Though ECC is not explicitly defined in this paper, the authors report caries rates in young children that range from 30% and 38% in Nigeria and Tanzania, to 79% and 82% in Malaysia and Guatemala, to 93% and 95% in parts of Mexico and China. These are much higher rates than those found in North America and Western Europe [3].

It is common knowledge that children's primary dentition is deciduous— primary teeth are lost within the first 7 or 8 years of life and are replaced with permanent teeth, which are never replaced. It may be argued, therefore, that ECC is not a serious problem and that the focus on dental health should really be on the permanent dentition. This is a huge misconception: children must live with their primary dentition during crucial years of development, and the sequelae of ECC are quite serious and permanently life altering. Complications from ECC include:

- Severe pain: painful caries can lead to difficulty eating, sleeping, difficulty paying attention in school, problems with daily activities that disrupt basic functioning and growth and development.
- Infection/abscesses: local infections in the mouth and gums, as well as an increased risk of systemic infections, including bacteremia, endocarditis, etc.
- Difficulty chewing: pain and infection make it difficult to eat and lead to and/or exacerbate malnutrition.
- Malnutrition: One study implicates ECC as contributing to other major health
  problems in early childhood [4], including failure to thrive. Of children with ECC in
  this study, 8.7% weighed less than 80% of ideal weight, compared to 1.7% of
  controls. The mean age of low weight patients with ECC was significantly greater

than for those at/above ideal weight, indicating that progression of nursing caries may adversely affect growth, or that poverty and malnourishment are related to poor growth *and* caries.

- Difficulty in speech development.
- Low self-esteem/psychiatric concerns: embarrassment, unwillingness to smile, teasing/criticism by peers.
- Children with ECC continue to be at high risk for new lesions as they get older, both in primary and permanent dentition because bacterial colonization occurs not only in the primary dentition but also in the secondary dentition, making children more susceptible to caries in their permanent dentition [5, 6].
- Treatment is expensive and very painful, and, in developing countries, there is no
  affordable or available treatment other than extraction, which can literally leave
  children without teeth for years.
- ECC can have marked generalized effects on a child's growth and development: increased glucocorticoid production in response to pain, decreased growth hormone secretion in response to disturbed sleep patterns, and overall increased metabolic rate during the course of infection may conspire to retard normal growth and development in children with ECC [7].

As in any infectious process of the dentition, the pathogenesis of ECC involves the interactions of three components: microorganism, substrate, and host. Time also plays an important role. Each of these is necessary but not sufficient to cause ECC. This model of ECC (seen in Figure 1) is helpful in framing a basic understanding of the phenomenon

and will be used to begin our discussion. However, in considering the epidemiological patterns of ECC throughout the world and the relevance of the model to our current understanding of the ECC literature—much of which attempts to categorize individual behaviors and/or conditions as risk (or protective) factors—this model simply falls short. Though technically accurate, it fails to recognize the context in which the microorganism, the substrate, and the host exist. Clearly, each of these components is affected by a variety of individual, cultural, social, and structural factors—factors like poverty, education, perceptions of health and disease, language and cultural barriers, health priorities, and even parent-child relationships. These complications are important, and the context matters!

This paper uses the current literature to generate a new model of ECC pathogenesis that considers not only the interactions of organism, substrate and host, but also the influence of health access, health awareness, health-related behaviors and other social and structural factors that lead to ECC. The ultimate goal of creating a more comprehensive model is not to subvert the basic model, but rather to enrich our understanding of the nature of ECC as an adverse health outcome in the developing world and to establish a foundation for the design and content of my own research project in rural El Salvador.

# II. ECC Pathogenesis: Microorganism, Substrate, and Host

<sup>&</sup>quot;While the general etiology of ECC appears similar to that of other types of caries, the predisposing factors are still unclear. The biology of ECC may be modified by several factors unique to young children, related to implantation of cariogenic bacteria, immaturity of the host defense systems, as well as behavioral patterns associated with feeding and oral hygiene in early childhood" [8].

Dental caries in both primary and permanent teeth develop in stages that have been systematically described by researchers: There is an initial loss of minerals from hydroxyapatites of calcium and phosphate (both building blocks of both enamel and dentin). As the loss of tooth structure continues, large microporous areas develop, so the refraction of light through the enamel/dentin changes, leading to the appearance of chalky white or opaque patches in the tooth surfaces. With repeated infusion of acids into enamel and the removal of apatite, the outer tooth surface covering the inner carious demineralization collapses, leading to the formation of a "cavity" [9].

As ECC progresses in its most prevalent pattern, the maxillary incisors develop a band of dull white demineralization along the gum line. White lesions develop into cavities that appear on the necks of the teeth in a brown or black collar. In many advanced cases, the crowns of the four maxillary incisors are completely destroyed, leaving only decayed black root stumps. The classic pattern of ECC described above is often symmetrical and is conventionally correlated with:

- 1) The chronology of primary tooth eruption: maxillary incisors, being some of the first of the primary dentition to erupt will be the first to experience a cariogenic exposure and will also suffer the longest carious attack.
- 2) The duration of the habit that exposes the teeth to fermentable carbohydrates: i.e. nursing, bottle use, candy consumption, etc.
- 3) The muscle patterns of infant sucking: in breast and bottle feeding, the natural or artificial nipple rests against the palate with the tongue extended over the mandibular incisors. The liquid from the bottle/breast bathes all the teeth except the lower incisors, which are physically protected by the tongue. If the liquid contains a

fermentable carbohydrate, the carbohydrate can be metabolized by the microorganisms into organic acids that ultimately demineralize the teeth.

Historically, ECC has been attributed to extended periods of baby bottle use (hence common usage of the term *baby bottle tooth decay*) as well as extended practices of breast-feeding "on demand". Recent research is questioning these hypotheses [7]. This new research will be discussed later in this paper; however, it is important to point out these assumptions here because these etiologic beliefs inform much of the conventional understanding of the progression of ECC that is described.

The specific pathogenesis of ECC has been carefully studied and, as presented above, requires the interactions of three key components: microorganism, substrate, and host.

## A. Microorganism

It has been established that the initiation of ECC requires the colonization of the oral cavity by microorganisms that are not present in infants' mouths at birth [10, 11]. Much work has been done to identify the organisms, understand how they colonize children's mouths, and explain their role in ECC formation. A group of organisms, *Streptococcus mutans* (MS), is the principle bacterial species thought to initiate human dental caries and has been shown to be present in children with ECC; *Lactobacillus* is also present in a disproportionate number of children with ECC [12-15]. MS is frequently isolated in mouths, even from sites with no evidence of caries [16]; however, in one study of breastfed children, the mean counts of MS and lactobacilli were 100-fold higher in plaque samples from children with rampant caries as compared with caries-free children [12].

MS is both acidogenic and aciduric. This means that the organism's own metabolic processes generate an acidic environment and that MS thrives in the extremely acidic conditions it has created for itself. MS adheres to tooth surfaces, generates acid byproducts that penetrate the exterior tooth surface (the enamel), and can survive and continue metabolism at self-imposed low pH conditions [13]. MS has been shown to induce caries in a variety of experimental animals and has been shown to be transmissible between animals [17, 18].

Many studies have shown that colonization with MS must precede cavity formation; however, MS cannot be isolated from predentate surfaces in children because its proliferation and survival in the oral cavity depends on the presence of adherent surfaces [2]. As teeth enter a child's mouth usually around 6 months of age, more surfaces are available for microorganism proliferation [8]. It has also recently been shown that the cariogenic threshold of MS and lactobacilli are lower in younger children and toddlers than older children. What were once considered very low levels of MS have been associated with ECC in very young children [19].

MS is clearly transmitted between humans, and it has been observed that interpersonal transfer occurs within families [20]. In Rogers' 1981 study, 88% of family members shared a common MS bacteriocin typing. In several instances, when a child was infected with an MS type common to that child's family, in no case was that type present in any of the child's school classroom. Another study of 18 Chinese families with colonized 3-year-old children showed that in 4 families, the mother shared the genotype with the child; in 3 families, the father shared the genotype; and in 2 families all members (father, mother and child) had an identical genotype [21]. Though these studies do not

prove direct intrafamilial transmission, the findings clearly support the hypothesis that strains of the organism can be (and often are) transmitted between members of a family.

It has generally been assumed that the mother and/or primary caregiver is most often responsible for direct microorganism transfer through saliva sharing—either via direct feeding, premasticating food for infants, blowing on food to cool it, etc [22, 23]. In addition, indirect transfer of MS has been documented through contaminated objects such as glasses, forks, spoons, toothbrushes, and toothpaste tubes, which have been shown to harbor viable MS for hours [23].

There has been recent interest in the ECC literature in determining whether or not there is a "window of infectivity" (a specific risk period) during which infants' mouths can be colonized by the microorganisms. The majority of studies cited in a recent review suggest that colonization occurs around twelve months of age, within a few months of the emergence of the primary incisors [8]. Others believe that this window of infectivity is closer to 19-31 months of age, coinciding with the emergence of the primary molars [24]. It is important to note that there have been documented cases of children with ECC as young as 10 months [7]. The age of colonization may be particularly important in identifying "high-risk children" because the earlier the colonization, the higher the risk for ECC [25].

Additional evidence for the role of microorganisms in ECC pathogenesis includes the observation that patients on long-term broad spectrum antibiotics have a reduced rate of caries [16]. This has led to a number of studies testing the use of topical antimicrobial therapy in the prevention of ECC [26, 27]. In an interventional study of Puerto Rican children considered to be "high risk for caries", 32% of children in the control group

(who did not receive topical iodine) eventually developed caries, whereas only 8% of the group that received antibiotic treatment developed caries. These findings support the idea that topical application of iodine solution for children at high risk for ECC suppresses MS proliferation and reduces the risk of ECC development in these children.

#### B. Substrate

Dietary sugars and fermentable carbohydrates are the major food element affecting the prevalence and progression of caries. Food and drinks that can cause a decrease in salivary pH to less than 5.5 and demineralization when in contact with microorganisms in the mouth are called *cariogenic*. Foods that are not metabolized by microorganisms but subsequently cause a drop in salivary pH to less than 5.5 within 30 minutes are called *cariostatic*. The primary factors determining the cariogenic, cariostatic and anticariogenic properties of the diet are: food form (liquid, solid or sticky, long-lasting), frequency of consumption (sugar and other fermentable carbohydrates), nutrient composition, potential to stimulate saliva, sequence of food intake, and combinations of food. [28]

Certain cariogenic carbohydrates are actually used by oral microorganisms to form an extra-cellular polysaccharide *glucan*, a sticky plaque matrix that enables microorganisms to adhere to the tooth enamel. These glucan polymers are also thought to enable MS to inhibit diffusion properties of plaque, which means that the sugars are ultimately allowed to diffuse to a deeper layer adjacent to the tooth surface and cause more damage than they otherwise would [29]. Carbohydrates also serve as metabolites in the production of organic acids that physically demineralize the teeth [2]. When sucrose is metabolized to glucose and fructose, it produces acid; intra-oral colonization of MS is highly dependent on a sucrose-induced acidic environment [29]. Thus, in young children, frequent sugar

consumption enables copious acid production by bacteria. Depending on the absolute pH decrease and the length of time of exposure to this decreased pH (note: pH of demineralization is in the range of 5.2-5.5), this acid can actively demineralize the tooth structure.

ECC has long been associated with night-time bottle-feeding, especially bottles filled with fermentable carbohydrates; however, some recent studies are challenging this longheld assumption [30, 31]. According to this theory, sweetened fluid from the bottle stagnates around maxillary incisors, allowing prolonged contact; fermentation of carbohydrates by bacteria results in acid production; this is compounded in a sleeping child by decreased salivary flow (which means decreased buffer activity), causing a great potential for enamel decalcification.

It makes sense that the choice of sweetened liquid (whether administered in bottle or cup) will have a discrete effect on the ECC outcome. Many specific liquids have been studied for their cariogenic properties. These include: sugary fruit juices, sodas, cow and breast milk, and infant formulas [29, 32-36]. Results of a study of dental caries and beverages in young children found that consumption of sweetened carbonated beverages, regular powdered beverages, and to a lesser extent 100% juice was associated with increased caries risk. Associations between different types of sugared beverages and caries experience were not equivalent, and they suggest that this is attributable to the different sugar compositions of the beverages or the different roles these liquids play in the diet [32].

# Sugary fruit juices and carbonated beverages

Not only do fruit juices contain natural fructose but most are also intrinsically acidic. Orange, apple, and lemon juices produce acidic attacks of short duration in which pH decrease is comparable to that caused by a 25% sucrose rinse and decrease in plaque pH from carbonated beverages [37]. One study looking at the effect of sports drinks on dental caries and erosion in rats found that, although sport drinks did not significantly promote caries, they did induce marked erosion on the lingual surface of the rat dentition. Though the formation of caries and erosion are different processes, in both cases, acidic dissolution of tooth material occurs [35]. Another laboratory study suggested that when fruit juices are involved in ECC, acidic erosion rather than cariogenic carbohydrate may be the primary enamel change preceding ECC [2].

In addition to having an intrinsically acidic pH, carbonated beverages invariably contain a sugar-sweetening agent. This makes soda and other carbonated beverages a clear suspect in caries risk. A recent release from the Iowa Fluoride Study, a cohort of 642 children followed since birth showed that those children who developed ECC had significantly higher median intakes of regular soda at 2, 3, 4, and 5 years. The authors assert that, in this population, consumption of regular soda pop is associated with increased caries risk [32]. A recent review article from *Infant Oral Health and Oral Habits* contends that "soda pop" has the highest relative decay potential of all beverages listed. The authors list the cariogenic potential of water as 0, human breast milk as .01, and soda as 1.05 [38]. These numbers support the empiric evidence that sodas possess a dramatic potential for cariogenicity.

### Milk

Milk is a particularly interesting substrate, and researchers have long debated the milk paradox: milk is simultaneously protective and cariogenic, and human studies looking at the carious effects of milk are often equivocal. For example, in the Iowa Fluoride Study mentioned above, milk intake had a neutral association with caries—it was shown to be neither protective nor destructive[32]. Milk's protective qualities are derived from its high calcium and phosphorous contents, which contribute to the remineralization of enamel, as well as its intrinsic proteins (including casein and whey), which are thought to provide protective organic coating on enamel surfaces [2]. Studies have also suggested that milk is less cariogenic than might be assumed because phosphoproteins in milk inhibit enamel dissolution [39, 40].

There is little argument, however, that milk is at least partially cariogenic. There have been clinical reports (although rare) that children who consumed only cow's milk in their bottle or were exclusively breastfed developed ECC [41]. Human milk contains twice the amount of lactose as cow's milk and can produce a greater drop in plaque pH and more enamel demineralization than cow's milk [34]. Other studies have shown that lactose, which is metabolized to glucose and galactose, enhances the oral implantation of cariogenic bacteria in animals, produces caries when fed to animals, and demineralizes tooth enamel when acted upon by bacteria [2]. In humans, acid production in dental plaque has been shown to increase after frequent use of either lactose or milk [42]. In addition, milk may also act as a "vehicle" for more cariogenic substance (e.g. combining milk with formula and/or sugar). Also, formulas containing sucrose instead of lactose may be particularly cariogenic. It has been hypothesized that milk's role in ECC

development requires the exposure to milk to be frequent and prolonged, resulting in pooling and stagnation around the necks of the teeth, especially the maxillary incisors.

### Acid

Prolonged or frequent exposure to acidic substances leads to dental erosion, a chemical dissolution of the dental hard tissues in a process that occurs even in the absence of bacteria [43]. Extrinsic acid sources include acidic foodstuff or drinks as well as use of acidic oral hygiene products and acidic medicines (such as vitamin C or aspirin) [44]. Drinks with a pH less than 4 can lead to demineralization of dental hard tissues and can lead to destruction of teeth depending on the intrinsic strength, coexisting low calcium intake, and "frequency of erosive attacks" [45]. One observation study in Saudi Arabia showed "large erosive lesions" in the deciduous teeth of children who "frequently consume acidic beverages in hot climates" [43]. Protective factors against such erosion include saliva and enamel chemistry. These protective factors are easily influenced by a nutritional intake, fluoride exposure, prenatal development, and other factors.

#### C. Host

The teeth provide an important non-shedding surface for the colonization of oral microorganisms. In most children, the primary maxillary incisors erupt at six months of age; ECC have been reported in children as young as ten months. The structural integrity of the primary dentition (and, therefore, resistance to caries) is dependent on a variety of factors.

### **Enamel**

Enamel is composed of carbonated calcium hydroxyapatites, and its integrity is influenced by genetics, nutrition, and acid exposure. Primary teeth enamel is usually less than 0.5 mm in thickness, whereas secondary dentition enamel averages 1.0 mm in thickness. This means that primary dentition is physically more susceptible to a quick caries progression (beginning with enamel demineralization, progressing to cavitation, and culminating with the involvement of dentin). In addition, lack of enamel maturation or presence of developmental structural defects in enamel increases risk of ECC by enhancing plaque retention and increasing MS colonization. Loss of enamel leads to greater susceptibility to teeth demineralization.

Enamel defects in primary dentition are most strongly associated with prenatal, perinatal, postnatal conditions, such as low birth weight, child/mother malnutrition or illness [40, 46, 47]. Even in the United States, prevalence of enamel defects in the primary dentition is relatively common: 13-39% in normal full-term babies and 62% in preterm babies [8, 46]. It is not a huge leap to imagine that children in developing countries, suffering from a combination of poor nutrition and limited access to preventive healthcare, would be more susceptible to enamel defects, a reality that would make them particularly susceptible to ECC [3].

Enamel hypoplasia, defined as a deficiency in enamel formation, may be inherited or can result from acquired environmental factors encountered during the period of enamel formation (*amelogenesis*). Inherited types of enamel hypoplasia are a very small percentage of enamel defects in the general population. Acquired types as a result of environmental factors are more commonly found, and especially high prevalence has been identified in many underdeveloped countries, such as Nigeria, Guatemala and in

Hawaiian children as well [46]. Amelogenesis is very sensitive to a variety of factors, and in the primary dentition, these factors may act prenatally, perinatally and postnatally and be systemic or localized. Important factors include birth trauma, infections (i.e. syphilis and rubella prenatally or measles, chicken pox, scarlet fever postnatally), nutritional disorders (i.e. deficiencies of vitamins A and D), metabolic diseases, birth prematurity and low birth weight, and chemicals (tetracycline, fluoride, chemotherapy) [46].

#### Fluoride

When fluoride is incorporated into enamel, it lowers the critical pH of the tooth surface and protects the tooth against attack immediately after fermentable carbohydrates consumption [43]. With systemic fluoride supplementation a standard in many parts of industrialized countries since the 1960s, significant decreases in ECC prevalence have been reported. When fluoride is started close to birth and continued for 5 or more years, a 30-80% lower caries prevalence has been observed [48] [49] [50]. In addition, in rat studies, fluoride added to sports drinks has been shown to significantly decrease the amounts of caries. This effect is probably due to the ability of the fluoride to reduce enamel dissolution both during and immediately after acid attack [36].

Recently, some researchers have called into question whether or not systemic fluoridation is needed. According to a recent study by Levy, "there is a growing body of literature that children, whether living in a fluoridated or non-fluoridated area, ingest sufficient quantities of fluoride from dentifrice, beverages and food" [50]. It has been suggested that fluoride supplements be prescribed to children in non-fluoridated communities and to those who have been identified as moderate/high risk. In addition,

the American Academy of Pediatrics (AAP) recommends fluoride supplements for infants who are exclusively breastfed because they are not getting fluoride through a water source [51].

#### **Nutrition**

According to the American Dietetic Association, diet and nutrition have a direct influence on the progression of tooth decay, and a preventive dental regimen should include diet counseling and nutritional education [28]. Adequate nutrients are needed pre, peri, and postnatally for normal growth and development of the oral cavity. Anterior maxillary cavities, the major nutrition-related oral disease found in young children, are directly related to the patterns of infant feeding and suckling. Nutrition and specific effects of malnutrition will be discussed in more detail later in this paper.

# **Tooth brushing**

Tooth brushing is a common oral healthcare habit that may help prevent dental caries and periodontal disease; its preventive potential is dependent on many different factors, including frequency and duration of brushing, rinsing habits and brush type [52]. Even though tooth brushing is often taught as a quick and relatively inexpensive intervention for preservation of oral health, the relationship between individual oral hygiene status and caries experience is actually relatively weak. There is only questionable evidence that tooth brushing prevents dental caries [53]. However, there is convincing evidence for the decay-preventing benefit of tooth brushing with a fluoride-containing toothpaste [7, 31]. In other words, tooth brushing appears to be effective for the prevention of tooth decay only in the presence of fluoride-containing toothpaste.

### III. A modified model of ECC

"In industrialized countries, the prevalence, severity, and patterns of caries are thought to relate to determinants that include social and educational background and dietary and oral hygiene practices. These factors may not affect caries development in the same way in developing countries" [1].

By this point, it should be clear that the model presented at the beginning of this paper to demonstrate ECC pathogenesis (Figure 1) is helpful for framing an initial understanding of the etiology of ECC; however, it does not adequately convey the context in which each of its components exists. As an example, we can think for a moment about all the factors that contribute to our notion of *substrate*. In doing so, it becomes obvious that a child's consumption of a sugary snack is not an isolated event. In order to understand the pathogenesis of ECC within a real world context, we cannot disregard the complex structural and behavioral influences that lead to the consumption of this very *substrate*. Let us use fermentable carbohydrate as our prototypical substrate.

Fermentable carbohydrate consumption is not only a function of exposure of a tooth to sucrose, but also whether or not such snacks exist, whether or not they are readily available, how much they cost, the impact of advertising campaigns and marketing promotions, the economic environment in which snacks are being bought and sold, and the presence of traditional snacks and eating patterns of specific cultural groups. On a behavioral level, *substrate* also depends on mothers' or children's view of such snacks—whether or not they are desirable to each individual, if they are seen as healthy or harmful, whether they are purchased, if they are actually being eaten, and how they are integrated into a child's diet, etc. If we do not consider the effect of these factors on the consumption of carbohydrates and the other parts of the etiologic model, then the

research (and any intervention that is derived from the research) will be inherently flawed.

In order to take this context into account, I have created a concept map that may serve as an adapted model for ECC pathogenesis. The model maintains the interactions of substrate, microorganism, and host as important etiological concepts; however, it also includes additional layers of influence—namely, how an important structural factor (low socioeconomic status), creates a "risky" environment for maternal and child behaviors, thereby increasing risk even further, and ultimately leading to the carious interactions of substrate, microorganism, and host. You can refer to the concept map seen in Figure 2 as you read this section.

## A. Socioeconomic Status (SES)

Despite a documented improvement in dental health in the U.S. and other developed countries in the past 50 years [54], the Centers for Disease Control and Prevention (CDC) recently reported dental caries as the most prevalent infectious disease in children in the U.S. [55]. Dental caries is five times more common than asthma and seven times more common than hay fever in North American children [56]. In the developing world, caries rates are increasing [57].

Though ECC may occur in all ethnic and socioeconomic groups, certain subpopulations have been shown to be particularly vulnerable. In the US, these risk groups consist of children 1) who are of poor, 2) whose mothers have low education level, 3) who consume more sugary foods, and 4) who have increased salivary *Strep Mutans* [38]. A fifth risk group is children of recent immigrants [58].

There is a strong relationship between socioeconomic status (SES) and ECC; this association has been shown to be statistically significant across populations around the world and has been documented with remarkable consistency [19, 30, 53, 59-61]. There is no other risk factor that shows such a constant role in ECC as SES. As a result, researchers have begun to consider specific patterns of SES in ECC pathogenesis. For example, children of low SES and high caries rates may be *more* likely to: present with high ECC rates but without fillings or extractions due to lack of access to health care [62], lack dental insurance [19], or lack adult supervision during the day [63]. They also might be *less* likely to have access to fluoridated water [64] or have had regular pediatric check-ups [65]. There are many more possibilities to consider, and it is important to keep in mind how SES will interact with all of the other risk categories discussed below.

SES is inextricably linked to a wide range of risk factors for ECC—regardless of whether we return to the basic model of microorganism, fermentable carbohydrate or host, or whether we consider an adjusted model of maternal and child factors. Low SES is associated with lack of formal education, lack of preventive health care (i.e. prenatal or dental), varying health priorities, limited nutritional access, and more. These associations, in turn, lead to further accumulation of risk, including the incapacity to recognize that ECC is a disease, the failure to prioritize primary dentition as an important health issue, the inability to purchase healthy snacks for one's child, and the rampant spread of an untreated, unrecognized problem. This list could go on; the point is to acknowledge that 1) SES is connected to ECC, 2) the role of SES in ECC involves both structural and behavioral risks, and 3) it is virtually impossible to discuss the behaviors involved in ECC pathogenesis without acknowledging the context in which the behavior is occurring.

In an attempt to dissect the relationship between SES and ECC, some researchers have begun to focus their studies on specific immigrant groups within the U.S. population. Recently, special interest has been taken to studying ECC in Latino immigrants because they have higher than average ECC rates, second only to that of Native Americans [5, 19, 63]. As members of the fifth risk group presented above (immigrants), these high rates are not surprising; in addition, many Latino children fall into one or more of the other risk categories, a fact that might explain some of the especially high rates that have been described for this population.

The only population subgroup in the U.S. with a higher caries prevalence than Latinos are Native Americans. Though most studies of this population are outdated, even today, their prevalence rates are often closer to rates in the developing world [66]. It may be argued that Native Americans have more similar social, economic, and cultural issues to children living in rural villages in Latin America than any other group living in the U.S. A 1985 study of Native American infants ages 12-36 months showed a prevalence of ECC between 53% to 70% [62]. No child had a filled/missing tooth due to caries. The authors suggest that the absence of fillings is because of limited access to dental care; as a result, carious teeth are left to cavitate without any sort of intervention. In a study published in 2004 of an aboriginal community in Canada, investigators found a 52% prevalence of ECC [67].

Immigration also plays an interesting and important role in this discussion. A 1996 study found that significant predictors for ECC were colonization with MS, immigrant background, and candy consumption [68]. According to the authors, children with immigrant background were more susceptible to caries due to "unfavorable dietary

habits, low tooth brushing frequency, and early colonization by bacteria." What does it mean that these immigrants have "unfavorable dietary habits"—is this referring to a traditional diet that makes them more susceptible to caries, or is it lack access to nutritive food in their new country due to economic and cultural barriers? Why do they have a decreased tooth brushing frequency? Is this an issue of cultural conceptions of dental health, a matter of less access to dental equipment and or education, different health or hygiene priorities, or something else? Why earlier colonization? This study does not speak to how much of this risk is related to factors from the native country versus how much of this risk is related to factors of migration.

These disparities bring up an important aspect of the ECC story—one that is often difficult to study—the fact that issues of health and health access are also intricately connected to cultural conceptions of health care and cultural perception of disease. It is difficult to determine in a study like this how barriers such as language and illegal immigrant status might influence a parent's care of his/her child's dental health. And, perhaps even more complicated, to what extent cultural ideas of what is health and what is disease influence the parents' action or inaction. In other words, it is not clear from this study if children had not been taken to the dentist because there was no affordable dentist to take them to, no Spanish-speaking dentist, or simply because their parents did not deem it necessary.

It is also important to recognize that there are many ways in which Latino immigrants in the U.S. and Latino children in their native countries share certain risk factors—including traditional diet, genetic susceptibility, and certain cultural practices and norms. However, these populations depart from one another in important ways as well—for

example, both health care and nutritional access have the potential to be dramatically different in the country of origin and country of migration. These are potentially interesting factors to investigate in future studies.

### **B.** Maternal Factors

#### 1. Poor maternal nutrition

Prenatal tooth development occurs between 34-38 days after fertilization, and calcification occurs during the fourth to six months of pregnancy; therefore, the primary dentition may provide unique information related to in utero development [8, 46]. During this crucial time, developing teeth are clearly affected by maternal nutritional factors, which can lead to enamel defects (in the fetus) that have been associated with caries in the young child [30]. In addition, enamel hypoplasia may be the result of inadequate vitamin intake at various stages of development.

Adequate calcium and phosphorous stores are required during tooth and bone mineralization [46]. As calcium metabolism is directly involved in dental development, it is not surprising that in maternal prenatal conditions with demonstrated disturbed calcium metabolism, enamel hypoplasia is often noted [8]. Prenatal deficiencies of Vitamin D also lead to enamel defects [30]. In fact, in some underdeveloped countries, the high prevalence of linear enamel hypoplasia has been significantly associated with malnutrition; this could be a result of general malnutrition, though deficiencies of Vitamin A and D have been documented, and a relationship between low birth weight and prevalence of hypoplasia [8].

Since a clear foundation exists that the prenatal period is important for primary tooth development, it is surprising that so little research has been conducted to elucidate this relationship. Taking into account the fact that caries have been seen in children as young as 10 months, should ECC be considered a primary problem of prenatal development rather than a problem of early childhood? One 1996 review of ECC concludes with the statement, "A shift in focus to improving prenatal nutrition represents a move to primary prevention for [ECC] . . . perhaps a more appropriate name, which would reflect the stage of development and increased susceptibility would be "pre-eruptive precarious syndrome."

A prospective study focusing on the prenatal period through early childhood might be the most important step to answer this question and might lead to primary prevention for ECC. Such a study would be time consuming and logistically complicated; however, it shifts our framework for understanding ECC and might generate more successful interventions than we currently have. This perspective is often ignored in the current literature, which generally places much more focus on the child's postnatal nutrition, eating, and hygiene habits; however, as we consider the problem, this early influence in the development of ECC is very important to bear in mind.

### 2. Poor maternal dental hygiene

It has generally been assumed that the mother and/or primary caregiver is most often responsible for direct microorganism transfer through saliva sharing—probably as a result of direct feeding, premasticating food for infants, blowing on food to cool it, etc [22, 23]. In addition, indirect transfer of MS has been documented through contaminated

objects such as glasses, forks, spoons, toothbrushes, and toothpaste tubes, which have been shown to harbor viable MS for hours [23].

These findings support the idea that the mother's dental hygiene also has an impact on the evolution of ECC in children, a hypothesis that has been widely supported. For example, in Taiwan, mothers with bad teeth are more likely to have children with nursing caries [65]; in India, the dental status of caregiver was correlated with ECC in the child [59]; and, in New York, researchers found a strong association between child's caries and mother's MS levels and mother's active caries [69]. That being said, it is important to note that in one study of Hispanic Americans in Boston, there was no increased risk of ECC among younger siblings in the presence of affected older siblings [63], a finding that potentially contradicts the other findings if we assume that the mother's poor hygiene would affect all children in the same family equally.

# 3. Maternal Education, Awareness, and Parenting

Another risk factor often considered in ECC research is the formal education of parents, but no clear patterns emerge. In one study of children with severe ECC, the educational level of both parents was high [70]. In another, the higher the father's educational level, the higher the ECC risk [1]. In others, the mother's educational level had no statistically significant relationship with regards to the presence or absence of ECC in their child [1, 62, 63]. In other populations, low educational level was associated with ECC [19, 71-73]. Presumably, this discrepancy is related to the fact that formal educational level does not correspond directly with specific awareness around ECC. In studies looking at what I will deem "ECC awareness," parents who had less awareness of

ECC as a disease, the associated risk factors, or simply did not recognize ECC as "a problem" were more likely to have children with ECC [59, 71, 74].

Frequently attached to the discussion of parental awareness of ECC in the literature is the controversial consideration of how parenting styles correlate with ECC. Though much of this literature is from the early 1980s, it is important to consider here because it seems to be reemerging in more current studies of ECC in developing countries. For example, in one study in which low "awareness of ECC" was correlated with caries prevalence, these same parents also were statistically significantly more reluctant to say "no" to their child, and be unaware of cariogenic potential of sleeping with the bottle [71, 72]. In its discussion section, one 1982 article suggests that "parents should be taught that they can demonstrate love for their children without putting them to bed with a bottle or a honey dipped pacifier" [2, 65]. Another author writes that "nursing caries is the result of improper parenting" and that parents of children with ECC consistently use behavior control interventions with feeding habits that produce carious destruction [2]. This same article hypothesizes that those caretakers who "use food to control behavior" probably employ other behaviors that lead to adverse dental consequences (e.g. sweetened beverages in sippy cup, cookies and candies) to pacify their children [2].

Such assumptions about parenting styles have often been criticized for being patronizing and for oversimplifying the problem by placing blame on individual parents; however, even today, these same judgments are being passed. As an example, in a recent study from Puerto Rico, investigators found that "what the mother did at night to stop the baby from crying" was related to caries risk. The options given to the mothers during the

interviews were: "give babies bottles or nurse vs. hold, rock, talk, or distract the children" [75]. Though such questions are not overtly judgmental, there is an explicit interest in parenting style, and the presentation of the parenting options as "food" vs. "non-food" might easily be interpreted as "poor parenting" vs. "good parenting". In their discussion section, the authors propose behavioral interventions that focus on "managing the crying baby" and "successful parenting". Though such interventions may be worthwhile, these suggestions place much of the blame on individual parents and ignore the myriad of structural and environmental factors that may play an extremely important role in ECC for this community.

Conclusions such as these are not as explicitly judgmental as literature from the early 1980s; however, they do oversimplify the predicament and, in targeting mothers' behaviors, essentially ignore the situation in which these mothers are living in poverty. I encourage the reader to think about how "parenting options" are not the same as "parenting decisions." For example, mothers of low SES in urban New York may have fewer options in terms of providing their children with healthy snacks and preventive care than mothers with more economic means. In much the same vein, mothers in Puerto Rico living in very close quarters may not have the time or resources to make a decision about how to prevent their child from crying in the night.

#### C. Childhood Factors

### 1. Poor childhood nutrition

Calling to mind the basic model for ECC pathogenesis (microorganism, substrate, and host), and considering our discussion of prenatal development, it is not surprising that childhood nutrition arises as an important step in ECC progression. Nutrition clearly plays an important role in both substrate and host components and even has a part in the capabilities of oral microorganisms to be cariogenic. Childhood nutrition is a complicated component of ECC pathogenesis, and for the purposes of this discussion will be broken down into four components: 1) insufficient nutrient intake, 2) fermentable carbohydrate intake, 3) breastfeeding and 4) bottle feeding.

#### a. Insufficient nutrient intake

Review of anesthesia and sedation records of children with nursing caries shows that children with caries weighed significantly less than control children (among children with caries, 8.7% had weights in lowest percentile category, whereas among controls, only 1.7% were lowest weight). This finding, while interesting, does not elucidate whether the caries led to low weight or the low weight led to caries. However, there is evidence that early diet affects brain growth and bone mineralization in pre-term infants and that early diet is a determinant of later dietary habits [33]. This evidence supports the hypothesis that problems with childhood nutrition could lead to inadequate tooth mineralization and that continued poor dietary habits could support the disease progression.

Few studies have been published on the effect of nutritional status on ECC (or the converse, the effect of ECC on nutritional status). The studies that have been published were all done by one research group studying the relationship between anthropometry and ECC in children in Peru [76, 77]. In their first study, they found that growth-stunted children showed a delayed exfoliation of primary teeth. In addition, children aged 7-9 years with stunted growth showed a significantly higher percentage of carious teeth than did well-nourished children of the same age. With these findings, they conclude that nutritional deficits that lead to chronic malnutrition not only affect tooth exfoliation, but also appear to render the primary teeth more susceptible to caries attack later in life [77]. In essence, poor childhood nutrition (as characterized by stunted development) increases risk for ECC. However, the evidence is not clear, and the question still remains as to whether the caries themselves were partly responsible for the malnutrition.

Follow-up studies in similar populations in Peru found that eruption of primary teeth was delayed in stunted/wasted infants in comparison with healthy infants [78]. At 4 years old, stunted and wasted children had higher mean number of decayed, extracted, and filled primary teeth. In addition, a significantly higher prevalence of caries in the primary and permanent dentitions was observed in stunted and wasted children compared with healthy children [79].

Though these studies all show that a statistically significant relationship exists between caries and nutritional status, no study has been able to answer the question as to which comes first (or if the distinction is even important). Presumably, if a child is chronically malnourished, he/she will not be ingesting sufficient vitamins and minerals (i.e. vitamin D or calcium) to fortify his/her primary dentition, thereby leading to

increased susceptibility to caries formation. However, if 1) a child has prenatal vulnerability to caries and 2) from a very young age has active caries with pain, it is not unreasonable to imagine that both pain and mechanical difficulties (lack of teeth or loose teeth) would make proper nutritional intake difficult. This could thereby lead to poor nutritional status (i.e. stunting or wasting).

The Peruvian studies' authors suggest that the effect of nutritional status on the timing of tooth eruption and caries development is an important confounder that has prevented other researchers from observing a clear effect of malnutrition on ECC experiences. The temporal component of ECC (progression over time) is difficult to control for and might explain the varying age distributions seen in ECC children. This information is also important in considering epidemiological studies of malnourished children with caries, as comparisons of ECC between different countries or even between different regions in the country cannot be made without a nutritional factor (skeletal growth) being taking into account. In addition, infected, carious molars may stay two or three years longer in the mouths of malnourished children than in healthy children, thereby increasing the levels of cariogenic bacteria in the mouth and increasing risk for caries in the permanent dentition.

An important question that remains to be answered is whether or not malnutrition in children actually leads to increased caries. Rat experiments have shown that protein-calorie malnutrition results in increased caries [80] and additional animal models with protein deficiency and specific nutrient deficiencies have been associated with more severe caries in animals. However, the human data is scarce. In a study from Thailand cited in a review of international trends in ECC, there is some speculation about the

possibility of children being undernourished, which might predispose them to linear enamel hypoplasia, which would increase the risk of dental caries; however, diet was not studied in this paper [3]. In a US study, one episode of mild to moderate malnutrition in the first year of life and/or folate deficiency has been associated with increased incidence of caries in primary and permanent dentition [81]. In another review, a study from China is cited in which there is a 45% prevalence of ECC without a clear set of risk factors [7]. The author suggests that other etiologies in developing countries may include: linear hypoplasia of primary teeth, which has been associated with malnutrition. In addition, inadequacies of the host's immunodefenses is suspected to play a role in the acquisition of carious lesions, and this is also closely linked to nutrition [4]. Because ECC is an infectious disease, it makes intuitive sense that the immune system would play an important role in protecting children from the disease.

The discussion of the connections between malnutrition and ECC is one large issue, but immunodefenses and the mechanisms for how a child in a developing country might have a decreased immunity (both via nutritional and non-nutritional routes) could be a separate paper. It is mentioned here for completeness; though the idea is not widely presented in the literature, it originates in a paper from the developing world and points to less frequently considered (but certainly plausible) ways in which children in poverty might be at increased risk for ECC.

### b. Exposure to fermentable carbohydrates

In a 1970 pilot study by Canada's Department of National Health and Welfare to determine the dental needs of Indian children in the Sioux Lookout Zone of Northwestern

Ontario, researchers found very high rates of ECC [82]. Interestingly enough, in their discussion section, the authors attributed the high caries experience to the introduction of "Free Traders", "Indian Cooperatives", and the "Hudson Bay Shops" in Indian settlements that gave recent access to processed foods including candies, confectionaries, and refined carbohydrates. These new commercial venues, combined with minimal fluoride, "[are] probably a main reason for the present poor oral health of Indian children" [82]. These observations, though written over thirty years ago in rural Canada, are very applicable in thinking of the introduction of similar refined sugars in Latin America and the rest of the developing world, especially in recent years. Though the study is old and though it focuses on categorizing the problem as opposed to understanding its etiologies, the discussion points very clearly call attention to what is currently happening with processed fermentable carbohydrates in places like rural El Salvador. Interestingly enough, more recent studies from Malaysia and Kuala Lumpur all consider caries to be increasing and blame the situation on easier access to confectionary and soft drinks [3].

Fermentable carbohydrate availability is one part of the issue; another important component is behavior. Though there is laboratory evidence that increased fermentable carbohydrate intake influences caries, the risk found in human studies is not always clear. For example, one study from Brazil found no significant correlation between type or frequency of cariogenic substances and ECC in children [83]. On the other hand, in Saudi Arabia: two-thirds of children with ECC were given fruit juice/soft drinks from bottle/can at less than 2 years (2 or more per day) and sweet intake two or more times per day [70]. Though there was no control group in this study, the researchers assume this consumption

is significant. In Kerala, India consumption of snacks, and giving snacks as a reward was correlated with ECC [59]; the same was true for a recent Swedish study [73]. Because all of these studies depend on parental report and recall of carbohydrate consumption, there are unquestionable weaknesses that could explain some of the inconsistencies.

Furthermore, because children may be more or less prone to caries upon exposure to sugars, this immeasurable susceptibility may explain some of the discrepancies.

### c. Bottle feeding

Prolonged/night-time bottle-feeding has been suspected as a major cause of ECC for decades. During night-time feedings, the saliva flow is diminished, and demineralization can occur without the cleansing and buffering influences of the saliva. In a Turkish study, bottle feeding at night as well as combined breast and bottle-feeding during the day led to a higher ECC prevalence than breastfeeding alone [1], a finding that places partial blame, yet again, on the bottle. Such high percentages and observations have historically been very persuasive.

In studies from Saudi Arabia, the U.S. and Turkey, night time bottle habits were correlated with ECC [63, 70] [1, 72]. Another argument that is sometimes posed is the age of weaning from the breast or bottle. Studies from Texas and Japan show that later weaning is correlated with increased risk [63, 64].

However, newer studies are questioning these assumptions. In studies from Brazil and Japan, no significant correlation was found between bottle feeding and caries [64, 83]. However, Japanese infants using the bottle had other risky behavioral patterns (i.e. irregular or frequent snacking, lack of tooth brushing, and snacking while playing).

One paper reports that the majority of US toddlers take the bottle to bed (close to 90% of children are bottle-fed at night), but only a relatively low percentage (20%) develop ECC [7]. The discrepancy between the high numbers of toddlers having night-time bottle feedings and the relatively low percentage who actually develop ECC is a heated discussion point. To support a paradigm shift, one author argues that the bottle to bed habit may be inferred as a cause of ECC simply because parents of children with ECC report night-time bottle-feeding, but this may not actually be a causal factor [5]. He (and others) are not suggesting that the bottle plays no role in ECC; however they contend that the baby bottle may not play as important a causal role as has long been assumed. This is still under investigation, and regardless of the final conclusion, such observations raise questions about why some children are particularly susceptible to ECC while others (with very similar circumstances) are not. It also challenges researchers to think outside of the box and consider ECC problem from a wider perspective.

### d. Breast feeding

Another contributing factor for ECC is a phenomenon often referred to as "breastfeeding on demand" or "breastfeeding at will" (recall that another name for ECC is *nursing caries* [41]). Breastfeeding on demand assumes that mothers are not feeding their infants on a timed schedule, but rather when the child cries or asks for food, the mother provides the breast. Evidence that breastfeeding leads to ECC is contradictory and confusing (particularly in light of the paradoxical nature of milk itself). However, it is a particularly relevant theory in thinking about ECC risk in developing countries, where the majority of women breastfeed in this manner.

A 1992 study of breastfed children with ECC concludes that breast feeding also allows the colonization and proliferation of MS and lactobacilli on the teeth of young children and they can occur in young children in the absence of any bottle [12].

In one retrospective study comparing the dietary practices of children with ECC to children without ECC, the children with ECC nursed for an average of 8.3 hours/day compared to 2.2 hours/day for children without caries [84]. Findings such as this support the hypothesis that actual feeding frequency and excessive exposure to breast milk have an important influence on caries development. In addition, mothers sleeping in the same bed as their infants and breastfeeding on demand during the night is considered particularly risky because any time a baby sleeps in the same bed with the mother, there will be an increased night-time feeding frequency.

A recent review cites that one unpublished report surveyed more than 1000 mothers of children breast fed at will for 1-4 years and found only a 5% prevalence of maxillary anterior caries [7]. It is important to note that, as in most fields, many of these "lack of association" findings are not often published—this means that if someone finds that breastfeeding practices are not significant risk factors, such a finding may not even be mentioned in the paper. That being said, some recent studies have shown mixed results. In a recent study from Brazil, there was no significant correlation between reported breast feeding and caries [83] and in Turkey, duration of breast feeding did not significantly relate to the presence of caries [1]. However, two thirds of Saudi Arabian children with ECC had been breastfed [70] and in Japan, researchers found that a high rate of breast feeding at 18 months related to dental disease. In fact, breastfeeding was the most important single predictor of all factors for caries status and caries activity [64].

A question we might as is, what other habits and/or exposures does breastfeeding at will correlate with? These behaviors might be associated closely with lack of other nutritive foods, lack of sugar-containing snacks, pattern forming of more frequent snacking, or even the habit-forming of suckling as soothing.

Perhaps it is because breastfeeding has been recognized widely as being so important for infant nutrition during the first two years of life, that many clinicians and researchers are obviously hesitant to place any blame of ECC on breastfeeding. For example, in the Turkish study mentioned above, the authors are quick to say in their discussion section that breastfeeding is very important for children in developing places like Turkey, and they specifically state their position that the benefits of breast feeding outweigh possible harmful effects [1]. Though their hesitance to challenge breastfeeding practices may be well founded, it is an important question that deserves more systematic study.

## 2. Poor childhood dental hygiene (tooth brushing)

There is general recognition that good dental hygiene probably reduces ECC risk, though it is not clear whether or not it is the mechanical aspect of tooth brushing or the exposure to fluoride in toothpaste that makes tooth brushing important for caries prevention. In Brazil, for example, the only significant finding that correlated with ECC in children 0-3 was presence of biofilm on the children's teeth. In other words, children with ECC were more likely to have less sanitary mouths than children without ECC [83]. In this same vein, in India, the poor oral hygiene status of child and poor cleaning of the child's mouth was correlated with ECC [59].

Some studies show that parental report of brushing of child's teeth before bedtime or brushing with fluoridated toothpaste is significantly associated with less caries [60, 65], though the results are mixed. In fact, in Turkey, the mothers' self-reported brushing habit showed no effect on carious development, and in Puerto Rico the mothers' reported use of fluoridated toothpaste was actually associated with increased rates of ECC [75]. Such findings are puzzling and quite disconcerting since we would assume that toothpaste use would be correlated with lower ECC. It is likely that there is some reporting bias here, though it is difficult to understand clearly what is happening with regards to hygiene and ECC. There are no observational studies that do not depend on parental report. This makes analysis of this issue difficult.

# 3. Limited access to dental care (preventive care, fluoridation, curative care)

Fluoride exposure, either through systemic fluoridation of water, fluoride treatment, fluoride varnish, or toothpaste is clearly an issue affecting the prevalence of ECC in the developing world. The World Health Organization (WHO) states that studies are remarkably consistent in demonstrating substantial reductions in caries prevalence as a result of water fluoridation. Where caries prevalence was high, the percentage of reduction was 40-49% in primary teeth and 50-59% in permanent teeth [85]. In the UK, water fluoridation was shown to reduce dental caries experience more in materially deprived areas than affluent ones, and the authors claim that the introduction of water fluoridation would substantially reduce inequalities in dental health [86]. In Taiwan, the

drinking water is not fluoridated, and researchers found a 95% rate of active caries in children at age seven [65].

In addition, limited access to regular pediatric dental care (both primary and secondary prevention) contributes to the problem. In one Native American study, in which more than half of children had caries, not one child had missing/filled teeth caused by caries—showing how the children had no access to curative dental care [64]. In Taiwan, more children who did not have caries had regular pediatric check ups [65]. In Latino families in Massachusetts, the parents in families without ECC were more likely to have visited a dentist recently [63], and here in California, lack of dental insurance was significantly correlated with ECC [19].

The issues are intimately connected with SES, which was discussed extensively at the beginning of this section, but it is important to address it here once more, if only because once a child has so many risky environmental and behavioral factors piled on their dinner plate, a lack of access to regular pediatric health and dental care clearly adds a not-so-sweet dessert onto an already loaded plate.

#### **IV. Conclusions**

Children should be free from pain; they should be able to sleep comfortably, focus in school, chew food, and have healthy teeth in their mouths during their first six years, a most critical period of human development. These are goals that can never be accomplished if: 1) in utero development is deficient, 2) from the moment the first tooth emerges, the mouth is immediately and constantly under attack, 3) caregivers are not

aware that this is a problem, and 4) the health care system continues to pay such little attention—preventive and/or curative. The primary dentition sets the stage for the rest of each child's life, and ECC is a phenomenon so prevalent worldwide that if it were any other infectious disease, it would be considered an epidemic.

In 1981, the WHO made a goal for global oral health to have 50% of 5-6 year olds in the world totally free from dental caries by the year 2000. They have not yet been successful in meeting this goal [57]. It should be clear from the discussion above that ECC is the result of much more than sugar, more than bacteria, and more than poor hygiene or nutrition. Recurring risk factors also include immigrant status, low SES, low education, lack of awareness, increased carbohydrate availability, lack of fluoride, prolonged use of baby bottles, breastfeeding on demand, and lack of access to dental care.

Though rates are improving in developed nations, they are still unacceptably high in the developing world. Often times, the basic definitions of risk categories are remarkably different in the context of a developing nation. For example, what it means to have a "low education level" or "limited access to healthcare" in Central America is different than what it means in the United States. When the average mother in a rural village has two years of primary schooling and the nearest dentist is only available once a month at a center located two walking hours from home, basic terms like "education" and "access" take on altered meanings. Perhaps the most important complicating factor is that each population is unique with respect to all components of the ECC etiology—ranging from bacteria exposure to diet, habits, susceptibility, and health beliefs. However, even

with all these differences across and among groups, ECC is widespread throughout the developed world!

Many of the studies cited above leave us with more questions than answers: How destructive/protective is breast feeding? What role do sugars and the recent introduction of processed foods play in the developed world? How much of ECC could be prevented with systemic fluoridation? What is the role of genetics? What about prenatal nutrition? Does malnutrition lead to ECC, or does ECC lead to malnutrition? What is the impact of tooth brushing? Are caries different in developing countries than developed ones? Surely we could think of many more, but whatever your favorite question might be, one conclusion is clear: children around the world are at high risk for ECC. This leads us to one more question: What do we do about it?

While simple prevalence studies are important to characterize the extent of the problem, it may be that more in-depth analysis of the health situation of these children would help create a clearer picture of the ECC story. This is our goal in one little corner of the world—rural El Salvador—to look carefully at the individual/behavioral, environmental/structural and traditional/cultural elements that interact with and influence the problem of ECC.

As we move toward that goal, it is helpful to classify the information that has been presented in this paper in a three-tiered format: structural, individual, and cultural elements (See Table 1). In our project in El Salvador, we will address all three of these elements by asking mothers what they think about caries in their children; by talking to teachers and health promoters; by peeking inside stores and into families; and, finally, by thinking about where the current health care system is succeeding and how we might be

able to take advantage of that success to push the definition of health and healthy children just one step further.

# CHAPTER 2: A Study of Risk Factors for Early Childhood Caries in Rural El Salvador

## **Background and significance**

The World Health Organization (WHO), in collaboration with the International Dental Federation, adopted as a global goal for oral health that by the year 2000, 50% of 5 to 6-year-olds should be caries-free [87]. However, dental caries continue to be a major oral health problem around the world, affecting 60-90% of schoolchildren as well as a majority of adults [88]. Caries are most common in developing countries in Asia and Latin America and less prevalent and less severe in most African countries (Figure 1) [89]. Such differences are thought to be related to a higher consumption of refined sugars and insufficient exposure to fluoride in these regions; however, due to changing dietary habits, most experts expect that the incidence of caries will soon increase in many developing countries in Africa as well.

There is currently no agreement on a definition for early childhood caries (ECC) and little data documenting caries in the primary teeth of very young children around the world. Though interest in this field is growing, international trends regarding caries in children ages 1 to 6-years-old remain virtually undescribed.

El Salvador, a part of the AMRO region by the WHO classification system<sup>1</sup>, is a Central American country of over 6.6 million people; it is one of the most densely populated countries in Latin America, with an estimated 298 people per square kilometer. Over 50% of the population is under nineteen years old, and 48% of the population lives

<sup>&</sup>lt;sup>1</sup> WHO member states are grouped into six regions: Africa (AFRO), Americas (AMRO), South-East Asia (SEARO), Europe (EURO), Eastern Mediterranean (EMRO), and Western Pacific (WPRO).

below the poverty line. The country was divided by a brutal and violent civil war from 1979 until 1992 and devastated again by Hurricane Mitch in 1998 and a major earthquake in 2001.

El Salvador's infant mortality rate is estimated to be 26 deaths/1000 live births, which is four times the rate in developed countries but well below the international infant mortality rate, estimated to be 50 deaths/1000 live births [90]. In 2003, WHO reported that over 90% of El Salvadoran children were receiving the BCG vaccine, 88% were receiving DPT, and 99% a measles containing vaccine. In addition, according to the WHO Oral Health Profile Program 2000 estimate, El Salvadoran 12-year-olds have a mean decayed/missing/filled teeth (DMFT) of 1.4, which also falls slightly below the global 2001 DMFT average for 12-year-olds at 1.74 [91]. However, after an extensive search, no information on caries prevalence in younger children (1-to-6-years old) in El Salvador or any other Central American country was found.

Asociación Salvadoreña Pro Salud Rural (Salvadoran Association for Rural Health, ASAPROSAR) is a non-governmental organization headquartered in Santa Ana, El Salvador. Santa Ana is El Salvador's second largest city with a population of 167,000, of which 50% live in rural areas. The region around Santa Ana is very productive, and agricultural products include basic grains, coffee, sugarcane, yucca, potatoes, tobacco, cotton, banana, plantains, fruits, and flowers. ASAPROSAR was founded in 1972 by Dr. Vicky Guzman to provide health services to El Salvadoran people in rural and marginal urban areas in and around Santa Ana. The organization continued to provide health care during the long civil war and in recent years has expanded its definition of community health by adding multidisciplinary development programs. These include rural health

care and education, the "barefoot angels" urban youth program, an eye care clinic, a micro-credit program, sustainable agriculture, and rural *nucleos* (preschools) for children 3-6 years old. The purpose of these *nucleos* is to provide young children with early education, hot meals, and health services (i.e. regular heights/weights, medical brigades). Currently, ASAPROSAR operates eighteen functioning *nucleos* in four regions surrounding Santa Ana. These *nucleos* are the sites of the data collection for this study of ECC.

As discussed at length in Chapter 1, it is generally accepted that interactions among microorganism, substrate, and host are important in the pathogenesis of ECC. A short recap of this discussion will be presented here.

It has been established that the initiation of ECC requires the colonization of the oral cavity by microorganisms that are not present in infants' mouths at birth [10, 11]. Much work has been done to identify the organisms, understand how they colonize children's mouths, and explain their role in ECC formation. A group of organisms, *Streptococcus mutans* (MS), is the principle bacterial species thought to initiate human dental caries and has been shown to be present in children with ECC; *Lactobacillus* is also present in a disproportionate number of children with ECC [12-15]. MS is frequently isolated in mouths, even from sites with no evidence of caries [16]; however, in one study of breast-fed children, the mean counts of MS and lactobacilli were 100-fold higher in plaque samples from children with rampant caries as compared with caries-free children [12].

It is also widely accepted that dietary sugars and fermentable carbohydrates are the major food element affecting the prevalence and progression of caries. The primary

factors determining the cariogenic properties of the diet are food form, frequency of consumption, nutrient composition, potential to stimulate saliva, sequence of food intake, and combinations of food [28]. Certain cariogenic carbohydrates are actually used by oral microorganisms to a sticky plaque matrix that enables microorganisms to adhere to the tooth enamel [29, 34]. Carbohydrates also serve as metabolites in the production of organic acids that physically demineralize the teeth [2]. When sucrose is metabolized to glucose and fructose, it produces acid, and the intra-oral colonization of MS is highly dependent on a sucrose-induced acidic environment [34]. Thus, in young children, frequent sugar consumption enables copious acid production by bacteria. It is not surprising, therefore, that, in many studies, ECC has been correlated with high and frequent carbohydrate intake in children [3, 68, 70].

Tooth brushing is a common oral healthcare habit that may help prevent dental caries and periodontal disease; its preventive potential is dependent on many different factors, including frequency and duration of brushing, rinsing habits and brush type [52]. Though tooth brushing is often taught as a quick and relatively inexpensive intervention for oral health, the relationship between individual oral hygiene status and caries experience is relatively weak. There is only questionable evidence that tooth brushing by itself prevents dental caries [53]. However, there is convincing evidence for the decay-preventing benefit of tooth brushing with a fluoride-containing toothpaste [7, 31]. In other words, tooth brushing appears to be effective for the prevention of tooth decay only in the presence of fluoride-containing toothpaste [92].

Finally, there is some evidence that mothers' and primary caregivers' awareness of caries and the cariogenic potential of certain foods and behaviors is related to caries

prevalence [59, 72, 74, 93]. However, there is currently no literature exploring how this lack of awareness contributes to specific cariogenic behaviors or how the absence of such consciousness in the community and the healthcare system generates a context in which ECC is allowed to proliferate.

For this study population in rural El Salvador, I hypothesized that the endemic nature of ECC is related to:

- o a lack of awareness that ECC is an illness, and
- o failure to consider the importance of early childhood dental health by the health care system, parents, and the community.

This hypothesis does not challenge the current understanding of ECC pathogenesis; rather, it adds a contextual layer: because mothers, communities, and healthcare professionals do not recognize ECC as a serious (yet preventable) illness, early childhood dental health is not prioritized. As such, individual behaviors (i.e. frequent carbohydrate consumption and lack of tooth brushing with a fluoride toothpaste) and structural factors (i.e. lack of early childhood dental healthcare and preventive education during prenatal care) contribute substantially to the high rates of ECC in these communities.

The literature describing the protective and destructive factors that relate to ECC is compelling, and the purpose of this study is not to prove or disprove the assumptions discussed above. Instead, by accepting these premises, I pose this hypothesis with the intention of exploring how and why known risk factors exist (and persist) at current levels in these communities. In addition, the study design itself challenges the traditional manner in which ECC has been approached in the literature. The mixed method case studies encourage us to consider the problem on three distinct levels: 1) individual

behaviors, 2) the social/cultural health beliefs that inform those behaviors, and 3) the environment in which each of these inevitably exist. I am hopeful that this design and unique approach to ECC will ultimately be useful in framing interventions to prevent ECC in the future.

### **Research questions**

- What is the prevalence of ECC in the four regions in which ASAPROSAR works?
- What impact does ECC have on children's lives in these communities?
- o What health behaviors that contribute to (or protect from) ECC are prevalent in this group? Are these behaviors significantly correlated with caries status?
- What is the level of awareness of ECC as an illness in this region?
- o How might a lack of awareness contribute to the problem?
- o Given these circumstances, what might be effective interventions to prevent ECC?

### **Methods**

This study was designed as an explanatory case study, based upon the case study approach described by Yin [94]. It uses both qualitative and quantitative methods for data collection and analysis. The project was developed after a pilot study in El Salvador in 2003 and an extensive literature review on ECC in the developed and developing worlds. Each of the four regions in which ASAPROSAR currently works is considered a case. The regions will be designated Volcan (Vo), Chalchuapa (Ch), Ahuachapan (Ah), and Camones (Ca) throughout the remainder of the paper. In three of the four regions, data collection occurred in more than one rural community. Refer to Figure 2 for a map of El

Salvador, Table 1 for a brief introduction to each of these regions, and Table 2 for a summary of the data collection process. For each case, data collection included:

- Oral examination, height and weight measurements of children (ages 1-7) in one
   or more ASAPROSAR nucleos in the region
- Structured one-on-one interviews with mothers/primary caregivers
- Focus group with additional mothers/primary caregivers
- Focus group with ASAPROSAR health care promoters working in the regions
- Store observation and storeowner interview
- Additional in-depth interviews (mothers, dentist, health care worker)

One hundred and sixty-eight children were included in the study. To be included in the study sample, the children must have lived in one of the four regions in which ASAPROSAR works and have been receiving ASAPROSAR services in the daycare centers or through community outreach workers. Children older than 7 years and/or children with two or more secondary (adult) teeth were excluded from our study sample. Because all of the children in this study were receiving ASAPROSAR services, this was not intended to be a random sample of all children in the region, but rather a sample of children already affiliated with ASAPROSAR. Therefore, our findings will be conservative; the problem is probably worse among children not affiliated with ASAPROSAR. The children's teeth were examined by one of two investigators (Kappa Score for inter-examiner reliability was .97) using a toothbrush and dental mirror for visualization. In addition, all 168 children were weighed and measured.

Seventy primary caregivers of a subset of the 168 children participated in a structured one-on-one interview to evaluate health behaviors, known risk factors for caries

(including feeding habits and oral hygiene habits), and health beliefs regarding ECC. The interview included both open and closed ended questions, and responses were recorded on a survey instrument that had been developed by the principal investigator.

Four focus groups (one in each region) were conducted with a different group of primary caregivers to elaborate on specific health beliefs and practices in the communities, thoughts specifically related to ECC, as well as obstacles to better dental health. The focus groups included five to seven primary caregivers and were conducted at the *nucleos* in each region.

Five focus groups were conducted with ASAPROSAR health care promoters (who also serve as local preschool teachers) to discuss specific health concerns and observed behaviors from the health promoter's perspectives. Each group was comprised of four to seven health promoters, and the actual focus groups occurred on ASAPROSAR grounds in Santa Ana, El Salvador.

An important local resource for these regions is the corner store, which is typically a converted space in a private house, where townspeople may make small purchases of necessary goods. The local store may, in fact, be the families' only regular source for goods outside of what their own land produces. As a means of understanding environmental influences of ECC, four store observations and four storeowner interviews were also conducted as part of the study. The observations were done for 1.5-2 hours in a randomly selected store in each of the regions. During the designated time period, all sales transactions were recorded, along with the age of the patron. In addition, an informal inventory of the store was taken, and storeowners were interviewed on their

health beliefs surrounding fermentable carbohydrate availability, healthy snack options, milk, etc in their communities.

Finally, several in-depth interviews with additional mothers, public health ministry workers, local dentists, and ASAPROSAR-trained health promoters were also conducted. Excerpts of these interviews will be included in the analysis.

All study subjects were recruited with the help of ASAPROSAR health promoters, who live and work locally and were responsible for explaining the purpose of the project and inviting parents with children ages 0-7 to participate. Of those that were approached, no parent, child, storeowner, or health promoter refused to participate in the study; however, parents and children who were invited but did not come to the site where data collection was occurring were not pursued further.

This study was approved by the UC Berkeley Center for the Protection of Human Subjects.

All adult subjects signed an informed consent form that was read out loud to them before any data collection occurred, and everyone received a toothbrush and toothpaste for his/her participation in the study. In the case of the children study subjects, a parent or primary caregiver signed the informed consent form, and both the parent and child received a toothbrush and toothpaste for their collaboration.

All quantitative data for this study were analyzed using SPSS 11.0 software. The anthropometric/nutritional analysis was also done with the help of ANTHRO, a software program for calculating anthropometry that was developed for the Centers for Disease Control (CDC) and Department of Nutrition for Health and Development WHO [95].

The qualitative data were coded inductively by the principal investigator (V. Jordan) using QSR NVivo, a qualitative analysis software program [96]. All of the focus group, store observation, and interview notes were translated and read once by the principal investigator. Through this reading, it was established that the seven themes to be coded were: general health beliefs, primary health concerns, awareness regarding caries in children, motivation for health behaviors, structural factors that influence health attitudes and behaviors, destructive health practices (fermentable carbohydrate intake, bottle use, breast feeding on demand), and protective health practices (hygiene, nutrition, dental, visits). Then the data were read again using the themes presented above. Data reports were generated for each of the seven themes and respective sub-themes using the QSR NVivo Software. Relationships within themes and relationships between the quantitative analysis and the qualitative themes were explored and are reported in the results section.

## Results<sup>2</sup>

### A. Demographics:

The exam study sample consisted of 168 children, with a mean age of 4.8 years (range 1.2-7.7 years). There were 87 girls (52%) and 81 boys (48%). Descriptive statistics for the children in each region are shown in Table 3.

The interview sample consisted of 70 primary caregivers (66 mothers, 2 grandmothers, 1 aunt, and 1 father), ages 18-65, with a mean age of 30.3 years. The mean

<sup>&</sup>lt;sup>2</sup> The results section is organized according to the research questions presented above; however, in interpreting the results, it may be helpful for the reader to consider how the research questions relate to the hypothesis presented in the introduction, as it will be revisited in the discussion section.

period of school attendance for the caregiver was 4.0 (±10) years, which illustrates extremely low education levels. The caregivers reported having an average of 3.3 (±6.7) children and 5.3 (±5.7) people living with them in their home. With regards to living situation and basic services, 79% of those surveyed were living in their own house, 80% had electricity in their house, and only 41% had potable water (4.5% of those interviewed in Vo reported having potable water, whereas 100% in Ca reported having potable water). Descriptive statistics for these primary caregivers according to region can be found in Table 4.

The health promoter group consisted of a total twenty-two ASAPROSAR-trained community health promoters: 20 women (91%) and 2 men (9%), with a mean age of 23 years (range16-36 years). All lived in the communities in which they work. They had a wide range of educational background: some having only primary school education, others currently pursuing their high school degrees. One health promoter identified herself as a trained traditional birth assistant. Many health promoters also identified themselves as mothers.

### **B. Early Childhood Caries:**

Caries prevalence Of the 168 children included in this study, at least one cavity was seen in the primary teeth of 142 children (85%). Fifty-eight children (35%) had caries in all four maxillary incisors; only eight (4.8%) had caries in all four mandibular incisors. Almost two thirds (60%) had one or more carious molars. Three children (1.8%) had caries in all twenty primary teeth.

The mean DMFT for children with caries was 6.52; however, only one child had a cavity that had actually been filled by a dentist. All other DMFT values represent primary teeth that were either decayed or missing, presumably due to decay (not to deciduous loss). Keeping in mind that there are only four mandibular incisors and four mandibular molars, we found that the children had an average of 2.1 carious mandibular incisors and 2.0 carious mandibular molars. The range of caries severity for all 168 children is shown in Figure 3. The prevalence and severity of caries according to ASAPROSAR region are shown in Tables 5 and 6. Analysis using the chi square test showed that differences in caries across the four regions are not statistically significant.

Impact of caries In response to an open-ended interview question about how caries affect children, primary caregivers' reported that the major effects of caries include pain, broken/lost teeth, infection/illness, language acquisition problems, nutritional difficulties, and self-esteem issues—among others. Of note, over half (59%) of mothers reported that caries cause pain for children.

During another part of the interview, 25 mothers (36%) report that their child sometimes has mouth pain, and 8 mothers (11%) report that their child has frequent mouth pain. Fifteen mothers (21%) report that their child sometimes has problems eating due to mouth pain, and 8 mothers (11%) report that their child has frequent problems eating. In addition, 13 mothers (19%) said that their child cries at night with tooth pain. The effects of caries on children's lives can be seen in the Figures 4 through 6, and Table 7.

One woman explained the caries progression from her perspective:

"It (caries) breaks [the teeth] up rapidly, sometimes it hurts, the gums hurt, [and] they bleed" (mother).

Mothers also reported that their children inform them of their pain, both verbally and nonverbally:

"Broken molars hurt, he always tells me that he has pain, and he cries" (mother).

During a focus group discussion, one health promoter explained how pain affects children's health and wellbeing:

"When your teeth hurt, you cannot eat, then you get malnourished, and you don't gain weight. Without teeth, you cannot eat meat, fruit, tortillas, all types of food" (health promoter).

During the individual interviews and the focus groups, not one primary caregiver or health promoter suggested that caries do not affect children; in other words, 100% acknowledged that caries impact their children in some way. Additional excerpts from the survey responses and focus groups regarding how caries impacts children's lives are presented in Box 1.

The anthropometric analysis showed that, of the 168 children examined for this study, 18% were stunted (low height for age), 4.4% were wasted (low weight for height) and 13% were underweight (low weight for age). A breakdown of these findings by age is presented in Table 8. However, there was no association between nutritional status and caries severity. (see Figures 7 and 8). The mean height-for-age Z score for children with caries was -1.14 ( $\pm$  1.1) and for children without caries was -1.07 ( $\pm$ 1.2). The mean weight-for-height Z score for children with caries was -0.95 ( $\pm$  0.92) and for children without caries was -1.20 ( $\pm$ .89). These are not statistically significant differences.

Caries awareness According to the interviews, 93% of primary caregivers interviewed believe that ECC is a serious illness, and 97% believe that ECC impacts their children. These results are shown in Figure 9. We asked primary caregivers to identify and characterize the severity of their child's caries/teeth (as "excellent", "good", "bad", or "horrible"). Of the primary caregivers who said that their child's teeth were "bad", their children had a mean DMFT of 7.8. Of those who said their child's teeth were "good", the children had a mean DMFT of 6.5. However, in three of four regions, the mean DMFT was actually higher for "good" teeth than "bad" teeth. Mothers who thought their children's teeth were "excellent" averaged 3.0 caries. Only one mother characterized her child's teeth as "horrible", and that child had 20 caries. These findings are shown in Figure 10.

One relevant major theme (about caries awareness) elicited during the focus group discussions was why baby teeth are important for development and how children get caries in their primary teeth. Across all four regions, primary caregivers agreed that baby teeth were important for chewing food and eating, and in three of the four cases, they commented on the importance of primary dentition for appearance and for their influence on permanent teeth. See Box 2 for additional cross-site analysis and qualitative excerpts.

Both primary caregivers and health promoters had very clear ideas about what purposes primary teeth serve:

"They are what we begin to eat with, they are the first thing we use to feed ourselves. All the future teeth depend on them. Sometimes people don't value the primary teeth, but they affect how the next teeth are going to be" (mother).

"They only have them for a time, but they are so important for the nutrition of the baby." (health promoter).

There was a small minority who disagreed. One mother said:

"No, [baby teeth] are not important, they are not stable, they fall out" (mother).

In addition to their discussions about baby teeth, focus group members talked about the etiology of caries; they identified certain health habits, eating behaviors, genetics, and the infectious components of caries. In all four regions, the women concluded that lack of tooth brushing was the main reason children develop caries, and in three sites, they discussed how sugary snacks, candy, and gum contribute to caries in their communities.

Other causes mentioned included: baby bottles (Ch, Ah), iron supplements (Vo, Ah), ignorance (Vo), soda (Ca), and nutritional deficiency (Ch).

"Cavities are like a little animal that passes from the mother to the child." (health promoter).

"Not brushing [their teeth] good or consistently enough. Eating a lot of sweets, a lot of chocolate, chewing gum, chewing ice. Sometimes because of iron, the blackness the iron gives them" (mother).

In all four regions, the focus groups agreed that caries stains children's teeth and often turns them black; however, in three regions (Vo, Ah, Ca), the women also believed that iron supplements actually cause caries. In these same sites, women in the focus groups admitted that many women do not give their children iron because it causes caries. See Box 6 for excerpts and cross-site analysis of the effects of iron on children's teeth.

"Giving them iron, how their teeth rot!" (mother)

"[Iron] makes them black, then they go rotting [...] Lots of mothers don't give the iron because it damages their teeth. They don't give it because of the teeth problems" (health promoter).

Even a young dentist who was working in the area doing her rural service year believed that iron causes caries:

"It seems to me that yes, iron has something to do with ruining the teeth, that it damages the enamel. . . Because you see very clearly that when they give the children iron, the teeth get black right away."

There were a few subjects who thought that caries were a natural process, unpreventable. One woman commented on her own experience with caries:

"I lost all my teeth as a young woman, but I never ate sweets. It was natural" (mother).

Additional excerpts from these discussions can be found in Boxes 3.

Though there were a few exceptions, most members of the focus groups agreed that baby teeth are an important part of child development and that early childhood caries negatively impact many aspects of a child's growth. They were also able to identify many of the risk factors that contribute to ECC in their own communities; however, they had some serious misconceptions about iron's role in caries pathogenesis.

Health attitudes and behaviors The prevalence of specific health behaviors known to be related to caries (breast and bottle feeding, carbohydrate and milk intake, and tooth brushing habits) according to region can be found in Tables 9 a, b, and c, respectively. With regards to breast and bottle-feeding, 68 mothers (97%) report breastfeeding their children, with a mean weaning age of 22 months. However, 38 mothers (54%) also reported giving their children a baby bottle, with an average weaning age of 34 months. Mothers reported filling the baby bottles with formula, milk, fresh fruit juices, corn water, artificial juices, soda, coffee, and sugar water.

Members of focus groups in all four regions believed that breast feeding was important for their children and acknowledged that most women in their communities breastfeed "on demand." In two of the four sites (Ch and Vo), the women discussed the potential benefits of breastfeeding on a schedule, and these same two groups expressed concern that excessive breastfeeding may be dangerous. However, the dangers they discussed were not related to oral health, but rather to suckling habits, malnutrition, and throat infections. This cross-case analysis as well as excerpts from the qualitative data can be found in Box 4.

"Breast first, bottle second" (mother).

"Where I live, every time the baby asks [they breastfeed]. When the baby cries, he wants breast. When they need something, there is no way of advising. Moms, how are they supposed to know? Danger? No" (health promoter).

Again, in all four sites, the focus groups recognized that baby bottles can make children sick (i.e. dirty bottles and/or dirty water leading to infections) and can ruin children's teeth; however, only one group (Vo) advocated the importance of good hygiene when using bottles. In addition, the women in one region (Ca) recognized that bottles are a very expensive alternative to breast-feeding. However, two cases (Ch, Vo) acknowledged that sometimes the bottle is helpful and even necessary:

"On one hand, it isn't good to give a baby a bottle instead of the breast, but sometimes the woman doesn't have milk, and she has to give her baby the bottle." (health promoter)

But they also recognized some of the dangers of bottle-feeding:

"It isn't very recommended to give the bottle because it ruins the children's teeth. They don't wash the bottle very well, and it gives infections. It is also very expensive to buy the milk" (mother).

"Sometimes moms leave their child in the hammock with the bottle. Sometimes, bigger kids suck on the bottle of the newborn, they share the bottle [...] The dirtiness of the bottle is passed on to the child" (health promoter).

Many women disclosed giving their child artificial juices, sodas, and coffee (as well as formula and milk) in baby bottles. Several health promoters expressed frustration:

"Only the bottle. They don't take it out of their mouths. Flour, corn, sugar water, juice, artificial juice, coffee in the bottle, they arrive at the preschool." (health promoter)

In addition to high rates of bottle use, primary caregivers report that their children are drinking almost two sodas per week and consuming "sweets" (including gum, cookies, candy, etc) almost daily (6.1 times per week). When asked why they give their children soda and sugary snacks, the women in the four cases had many responses.

Focus groups in all four regions agreed that most mothers give their children sweets and candy because children actively ask for them and that children cry if they are not given sweets. In three groups (Ch, Vo, Ca), the women claimed that giving children sweets was a tradition or habit that was difficult to break. In two (Vo, Ah) the women claimed that candy is often used to keep a child from recognizing his/her own hunger when there is no food in the house. Finally one group alleged that mothers use candy to keep children occupied that they can get work done (Vo). See Box 5 for more results and excerpts of focus group discussions.

Many health promoters said that they give candy to distract or calm an agitated child:

"A mother wants to go out, and the child doesn't want her to go, she has to give him candy so that he stays in the house [...] They pass all the time buying sweets in order to keep them quiet. A stimulus, a gift a prize" (health promoter).

Several mothers explained that mothers give sweets to quench their children's hunger:

"Because they don't have anything to give them to eat" (mother).

Others said that giving their children sweets is simply a habit:

"It is a custom that we give sweets and gums to kids" (mother).

With regards to soda, women from all four regions explained that they frequently give their children soda simply because their children see it in the store and ask for it.

Other themes that varied across sites included: giving soda as a treat (Ch, Vo), soda as a bad habit (Ch), the fact that fathers bring soda from the city after long periods away from home (Ch), the importance of advertising (Ca), and how soda is easier to prepare than fresh juice (Ch). In this vein, one mother said that giving her child soda is a matter of time management:

"Because I don't have time to prepare juice, so I send my child to buy soda" (mother).

Another said that parents cannot deny their children the treats they themselves are indulging in:

"Some moms buy soda for lunch. The family drinks it—how are they going to say no if they are drinking soda?" (health promoter).

Additional cross-site analysis and excerpts about soda intake can be found in Box 5.

These same children are only drinking milk an average of 3.4 times per week, well below the recommended intake for small children. During a focus group, one mother claimed, "It is also very expensive to buy milk" (mother). Other than weekly milk intake,

which was extremely low in Ah, there were no significant differences in these behaviors between regions.

A very high percentage of mothers (97%) reported owning a personal toothbrush and brushing their own teeth at least two times per day (90%). Sixty-one primary caregivers (88%) reported currently having toothpaste at home. Fifty-eight (83%) reported that their child has a toothbrush at home. Forty-eight (69%) stated that that their child brushes his/her teeth at least twice a day, and 45 (68%) said that their child "almost always" uses toothpaste; however, 31 (46%) reported that they never help their young child brush his/her teeth. The focus groups also discussed at what age a child is first able to brush his/her own teeth, and the different groups came up with a wide age range—from twenty months to six years—though most groups acknowledged that younger children may not do as good of a job brushing his/her teeth as an older child.

One mother commented about her child's ability to brush her own teeth:

"There are children who can do it at three years old, but perhaps they cannot do it very well. There are children that get the toothbrush and toothpaste in order to eat it" (mother).

Another said that she models tooth brushing for her child:

"I started brushing my teeth myself so that he could see how it was to be done. I taught him because it is an important thing. I put the toothbrush in, teach them how it has to be" (mother).

With regards to the health behaviors presented and discussed in this section, we found no statistically significant association between any of the behaviors and caries severity. The only statistically significant association with caries severity was the age of child; as would be expected, in all four regions, older children have increased mean

number of caries (p<0.001). This relationship is shown in Figure 11. Additional excerpts from mother and health promoter focus group discussions about ECC health attitudes and behaviors are found in Boxes 4-7.

# Health access: healthcare and food availability

Healthcare: As a way of assessing behaviors related to preventive care and how families are accessing the healthcare system, we asked primary caregivers when was their last dental visit (mean=2.1 years) and how many adult teeth they have had pulled/lost (mean=4.14). In addition, whereas 68 mothers (97%) report that their children's vaccinations are up-to-date, only 12 (17%) report having ever taken their child to the dentist. This information is presented in Table 10.

In addition, primary caregivers' reported use of the medical and dental systems are presented in Figure 12. Nineteen mothers (27%) reported "never going go the dentist," and only 4 mothers (6%) reported going to the dentist for "well-checks". In the focus group discussions, both mothers and health promoters touched upon important structural influences involved in dental health. Mothers and health promoters identified economic hardship, education/awareness, and health access issues as influencing their own health behaviors.

Women in all four regions referred to economic hardship as the number one influence on their dental health behaviors. They also cited lack of cooperation of their children (Vo, Ca), forgetting and/or not having time to go to the dentist (Ch, Vo), not knowing what to do (Vo) and simply not prioritizing dental health (Vo). See Box 8 for additional excerpts

of focus group discussions on influences on dental health behaviors and cross-site analysis.

As mentioned above, financial hardship was the most consistent theme,

"There is no money to buy toothbrushes, it is far to go to the city, takes a whole day, and it is expensive. I have eleven children, it's expensive" (mother).

"Women hardly recognize the benefits to going to the dentist, also there are economic problems, there is no access in rural zones, and taking out the bad teeth is the only treatment. Taking out a tooth costs \$2, filling a tooth costs \$10, and sometimes the fillings you get [...] fall out in two weeks" (health promoter).

Additional excerpts from focus group discussions are shown in Box 8.

The rural dentist (mentioned above) explained that most people do not bring their children to the dentist unless the caries are very advanced:

"The truth is that almost all the cavities that I see in rural areas in children are fourth grade—that is they are very serious and already are affecting even the pulp of the tooth."

She also thought that the solution to such severe dental problems is more education:

"Parents need "more education about hygiene, more education about nutrition, and better general orientation about dental health."

Finally, one trained healthcare provider who works as a nurse's aide for El Salvador's Ministry of Health recognized a lack of services in the area. She commented about how she has seen a dentist come only once to the local schools, and how he only looked quickly into children's mouths, giving them referrals that were never used. She sees it as a combination of responsibility of parents and the system:

"Their teeth are all bad because there is not a program for taking care of the teeth. Plus, the people don't purchase toothpaste or toothbrushes. People just don't pay any attention to dental health."

Fermentable carbohydrate access: During interviews with local storeowners, they reported that their stores generally provide local townspeople with food staples (i.e. eggs, beans, sugar, and rice), cleaning products (i.e. laundry detergent), and snacks and

sodas (i.e. cookies, gum, and Coca Cola). More information about the stores in each region and the products available in these stores is found in Table 11.

Storeowner reports from the four regions vary in terms of what goods were most frequently purchased by adults: sugar, rice and soap (in Ch); sugar, beans, rice and milk (in Vo), "food for preparation" (in Ah), and cookies, soda and juices (in Ca). In all four regions, however, the storeowners independently reported that the products most frequently purchased for (and by) children were: cookies, chips, and candies, as well as artificial juices.

Observations in these stores revealed that 9 of 16 (56%) purchases in Ch, 8 of 13 (62%) in Vo, 2 of 5 (40%) in Ah, and 6 of 11 (55%) in Ca were of sugary snacks/fermentable carbohydrates. These fermentable carbohydrates included bread, cookies, chips/cheetos, gum, mints, lollipops, sodas, frozen artificial juices.

As seen in Table 11, these stores do not regularly sell any fresh vegetables and generally sell very few fresh fruits. Milk costs between \$1.72 and \$2.30 per liter, whereas soda typically costs \$0.50 per liter. In addition, storeowners report that they sell between twelve and 50 liters of soda per week, whereas most sell milk "only rarely" and often do not have milk in stock. In two of the four stores, there were no toothbrushes available; however, all stores had toothpaste (ranging in price from \$0.52 to \$1.00).

#### **Discussion**

ECC is a complex phenomenon that can be difficult to understand due to overlapping etiologies that are often endemic to people living in poverty. It is clear from our study that ECC is rampant in this area of El Salvador—with over 85% of children being

affected and a mean DMFT of 6.5, an especially high number considering that young children have only twenty primary teeth and that effectively *none* of the carious teeth we saw were filled. It is also evident that ECC is having an appreciable impact on children's lives, confirmed by the mothers' poignant reports of very real and serious effects of caries on their children, most notably pain. This pain, together with difficulty eating and problems sleeping undoubtedly contribute to overall poorer health in children who already are at a disadvantage. In addition, these problems can affect children's ability to concentrate in school and lead to serious life-long consequences that extend beyond basic health.

We had expected that children with more severe caries would also have low anthropometry (i.e. an increased frequency of stunting and/or wasting). In this study population, we did find a very high prevalence of stunting (almost 20%) and a relatively high prevalence of wasting (over 4%); however, we found no significant association between anthropometry and caries status. This was somewhat surprising. It is possible that the children with the most *limited* access to food have generally poor nutrition and, therefore, carious teeth; however, children with *better* access to food may actually have more access to fermentable carbohydrates (i.e. soda, candy, etc), and therefore, also have high rates of caries. This may lead to the results we are seeing. It may also be that our sample size and narrow age-range limit our abilities to see statistically significant associations. Finally, it is possible that because the relationship between nutritional status and caries is complicated, any association we might see is being confounded by such factors as age, eating habits, etc.

Primary caregivers and ASAPROSAR health promoters appear to be well aware that ECC is a problem, with almost 100% of mothers agreeing that caries is a disease, and many showing very keen insight as to why primary teeth are important and what causes caries in young children. However, though most mothers are aware that frequent sugar exposure, lack of tooth brushing, and even bacteria all contribute to ECC pathogenesis, subjects report consistently high carbohydrate consumption, infrequent tooth brushing, and saliva sharing. They also report widespread breastfeeding on demand and surprisingly high frequencies of bottle use by children.

It is important to note that a large number of primary caregivers and health promoters in this area believe that iron supplements are a common cause of caries, a notion that is not supported by research. Though iron supplements do stain teeth, they do not cause caries. This is a crucial discussion point that should be addressed immediately by pediatricians and dentists working in the area; many mothers whose children are diagnosed with anemia and prescribed iron supplements by a physician report not giving their children the supplements for fear of causing caries. This means that identified iron-deficient children might be going without, which may further compromise their health. This finding is relevant to our discussion of how caries impact the health of children in these communities and should be considered as an indirect (but extremely important) factor in how ECC negatively influences early childhood health in these communities.

If we consider the total study sample, the primary caregivers' negative opinions of their children's teeth appear to be associated with mean number of caries—that is, children whose teeth are perceived as "bad" have more caries than children whose teeth are perceived as "good". However, if we look at the breakdown per region, then the

relationship between the discernment of the children's teeth as "good" versus "bad" is actually flipped relative to the mean DMFT values. This finding makes it questionable as to whether or not the primary caregivers are really aware of the severity of the ECC problem. Regardless, I argue that the mothers considering their children's teeth to be "good" with a mean number of 6.5 caries is also an important discussion point. Perhaps an average of 6.5 caries is "good" relative to other children in town; however it is certainly not "good" relative to WHO goals for oral health or standards that we would expect in the developed world. It is also important to note that, although mothers agree that ECC is a disease and over 30% describe their children's teeth as "bad" or "horrible," very few have ever taken their child to the dentist.

The four ASAPROSAR regions differ in terms of their geography, population densities, distance to main roads and health services, economic resources, etc; however, our results demonstrate that these four cases are essentially equivalent with regards to ECC prevalence and most behavioral factors thought to be associated with ECC.

Across the four regions, primary caregivers had essentially the same ideas about the purpose of baby teeth and the causes of caries in their children. They also conveyed essentially the same beliefs and practices regarding breast and bottle-feeding (as seen in both the quantitative and qualitative data), as well as similar patterns of soda and fermentable carbohydrate consumption. Certainly, there were individual themes that may be specific to one or more regions (i.e. the idea that fathers' behaviors might plays an important role in caries, which came up only in Ch) as well as some quantitatively significant factors (i.e. very low milk consumption in Ah). However, because the health practices and beliefs in these regions appear so similar and because the caries rates are

also so close, the caries experience can be considered equivalent in all four of these ASAPROSAR regions. This also suggests that potential interventions do not need to be significantly altered when being applied in different regions such as these.

Alternative explanations for the ECC endemic were also considered during analysis; for example, 1) perhaps children in urban sites (or sites closer to urban centers) are at higher risk for caries than those in more rural areas, due to easier access to fermentable carbohydrates, more limited access to homegrown fruits and vegetables, and higher population densities; OR 2) perhaps children in more urban sites are actually at decreased risk for caries because they have generally easier access to healthcare services (both preventive and curative). We looked at these alternatives in light of major differences between the four cases: distance to main highway/urban center (Ca is located right on main highway, twenty minutes from Santa Ana; Ah sites are two hours walking to reach main highway and then still 30 minutes from a small city); the fact that two sites were primarily agricultural and sparsely populated (Vo and Ah) while the other two were more industrial and densely situated (Ch and Ca); and some sites having more direct access to basic health services (i.e. potable water, health care facilities in site). However, we found no differences in caries rates, no differences in health behaviors (i.e. vaccine completion, dental visits, mother's last dental visit); no difference in carbohydrate consumption (cola and sweets); little difference in milk consumption; and no differences in the obstacles the primary caregivers themselves identified as the causes of poor dental health in their communities. Thus, though these alternative explanations cannot be completely ruled out, and though this study was not a designed to compare caries in rural vs. urban parts of El Salvador, the nature of our cases enables us to consider these hypotheses and make some

preliminary conclusions that, in our population, distance to urban centers is not a primary factor in ECC within these communities.

While our study has provided some support for the hypothesis that people in these communities are not completely aware of ECC, there is also some evidence to the contrary (i.e. the overwhelming response from primary caregivers believing that ECC is a serious illness) that leads us to conclude that awareness is simply not enough to exact changes in behaviors. This, I believe, is probably related to the structural and environmental factors that were hinted at in this study (i.e. the expense of buying a tooth brush and toothpaste, travel distance/time to the nearest dentist, and the lack of available healthy snacks in local stores); however, these issues clearly need further exploration. I would also suggest that ECC is just one of many pressing illnesses that these villagers encounter, and because its effects are often surreptitious and slow, it is shoved to the back burner—prioritized behind acute diseases like diarrhea and upper respiratory illnesses.

Why is it, then, that over 97% of children in this study were fully vaccinated, yet only 17% had ever seen a dentist? Of those that had been taken to the dentist, only one child had gone for a well check; the rest were taken because they complained of pain.

Vaccination percentages are often held up as an important health marker for health prevention, and early dental intervention could also reasonably be classified as preventive medicine. There appears, however, to be a difference here in what I will term "preventive behavior". Even though people are aware of the many causes and impacts of caries and that many of its causes are preventable, it has yet to be injected into the preventable illness psyche. Whether this is a simply a consequence of years of intense intervention by the international healthcare system regarding the importance of vaccination, the more

severe threat of death from preventable illness (i.e. measles), a lack of dentists in the area, simply prioritization of scarce resources, or something else is not clear. However, it does present a possibility: people have bought into the idea of preventive medicine. They are taking their children to be vaccinated on a regular basis at predictable intervals. Would it be possible to take advantage of this "vaccination habit" and provide direct dental intervention? Could we integrate preventive dentistry with preventive medicine? What about a program that gives a child a fluoride sealant at the same time he/she is receiving their next vaccine?

There are many important questions that were not answered by this research and that could complement the findings. Additional data that would help gain a better understanding of the high rates of ECC in this region include: data on dentist availability, attitudes/behaviors of dentists in the area, how mothers rank ECC relative to other illnesses in the region in terms of severity and importance, cost/benefit analysis of preventive dental care, better fluoride data, observations of interactions between dentists and patients, etc. These are studies that can (and should) be done.

In essence, the quantitative aspects of this study provide us with a relatively clear understanding of individual health behaviors occurring in these communities. These data support our hypothesis that this population has many risk factors and risky behaviors contributing to a very high prevalence of ECC. This information is nicely complemented by the qualitative data, which helps us gain a clearer understanding of some of the social and cultural health beliefs that inform these behaviors. The store observations give us an introduction to one environmental component in the daily lives of El Salvadoran people. However, we clearly need to learn more about the environment in which these behaviors

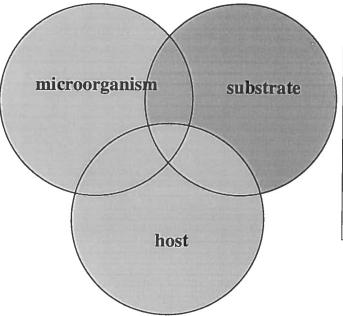
and beliefs are being cultivated. Ultimately, such an understanding may help to explain how risk factors for ECC are perpetuated in these communities, not only in El Salvador, but also in other parts of the developing world. In this vein, perhaps, ECC may come to serve as a new marker for measuring improved health in early childhood—raising the bar and recognizing that healthy teeth means healthier children.

Finally, a broader understanding of the ECC phenomenon might suggest a number of successful interventions—interventions that include not only parental and healthcare provider education but also target earlier, crucial moments in a child's development as well as systemic factors that contribute to ECC and other preventable illnesses. Potential ideas include:

- 1. Systemic water fluoridation.
- 2. Earlier and more thorough prenatal care that includes <u>dental</u> prenatal care: this would not only address the mother's own dental health (to treat mother for virulent MS strains and perhaps cut down on sharing of bacteria with infant), but also place a stronger emphasis on prenatal nutrition for primary dentition formation (i.e. the importance of calcium and vitamin D), especially in early pregnancy.
- 3. Expanded dental access at an earlier age: creating a culture, much like the vaccine culture, in which mothers are accustomed to/believe in the importance of preventive visits to dentist even when their children are very young.
- 4. Taking advantage of immunization contacts: 97% of these children are being seen on a regular basis during their first five years. This is a missed opportunity for education, preventive care, and possible even curative treatment. Vaccine appointments should be more than a simple needle stick.

- 5. Increasing access to potable water: this would be both a way to introduce systemic fluoridation in these communities and a safer way to support use of [free] fresh water and juice instead of cola and artificial juice for children.
- 6. Addressing the exorbitant cost of dental hygiene products and the relatively cheap price of fermentable carbohydrates compared to healthy snacks: toothpaste and milk cost as much in El Salvador as they do in the U.S., sometimes more. As long as cola is \$0.50/L and milk is over \$1.00, people are obviously going to choose soda over milk—they can fill more stomachs with less money. The same is true for chips and candy versus fruit/healthy snacks, which are not even available in these local stores. With the introduction of milk-producing animals (i.e. goats) and sustainable agriculture (i.e. a better variety fruits and vegetables), perhaps these preventive/protective goods can be made available at a cheaper and more accessible rate than the destructive goods.

Figure 1: Basic model for Pathogenesis of ECC



Micro-	Strep Mutans,
organism	Lactobacillus
Substrate	Fermentable
	carbohydrates:
	juices, milk
	(including breast
	milk), carbonated
	beverages, sugary
	snacks
Host	Tooth structure,
	enamel, saliva,
	acid, fluoride, etc

Figure 2: Revised Concept Map for ECC

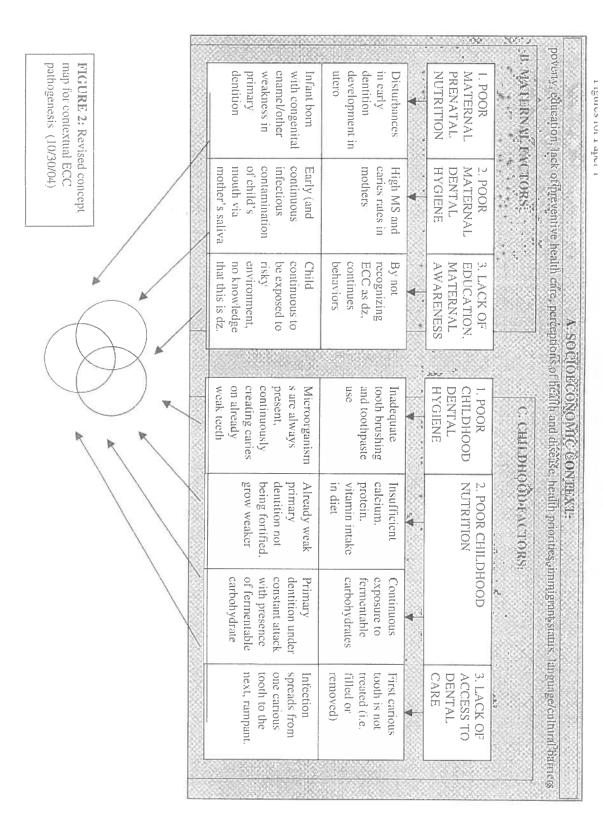
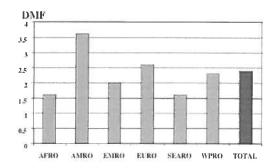


Figure 3: Examples of 3 levels of factors that influence ECC

	Individual behaviors	Structural/environmental components	Cultural/traditional components
Nutrition	<ul> <li>Mother poor prenatal nutrition</li> <li>Child poor perinatal nutrition</li> </ul>	<ul> <li>Limited access to healthy food (due to isolation/money)</li> <li>Low SES</li> <li>Lack of education about nutrition</li> </ul>	Traditional diet may have protective/destructi ve influence
Feeding Practices	<ul> <li>Breastfeeding "on demand"</li> <li>Bottle feeding</li> <li>Frequent fermentable carbohydrate intake</li> <li>High frequency of sweetened liquids</li> <li>Low frequency of milk, fruit, vegetable intake</li> </ul>	<ul> <li>Lack of potable water</li> <li>Increasing numbers of working mothers, lack of availability of "healthy snacks",</li> <li>Introduction of fermentable carbohydrates as primary snack source</li> <li>Advertising</li> <li>Lack of education</li> </ul>	<ul> <li>Traditional eating practices (i.e. corn tortillas with every meal)</li> <li>Cultural norms/tradition support breastfeeding on demand (not based on scheduled breastfeeding)</li> </ul>
Hygiene	<ul> <li>Mother poor personal hygiene (her own lack of tooth brushing)</li> <li>Poor child hygiene</li> </ul>	<ul> <li>Lack of running water</li> <li>No potable water</li> <li>No toothbrush</li> <li>In poor areas, mothers have high MS colonization rates</li> <li>Low SES</li> </ul>	ECC as "not a problem" because it is so prevalent and because teeth fall out
Health Access	<ul> <li>Only goes to clinic for illness</li> <li>Rare visit to dentist</li> </ul>	<ul> <li>No dentist in area</li> <li>Dentist is expensive</li> <li>No fluoride in water</li> <li>No toothpaste (no fluoride)</li> <li>Low SES</li> </ul>	<ul> <li>Seek care for cure not prevention</li> <li>Difficult relationships with medical system</li> </ul>
ECC Awareness	<ul> <li>Mother passing food/objects to children (without washing)</li> <li>Mothers believing that iron causes ECC, not giving child iron supplements</li> <li>Mothers not paying attention to carious teeth</li> </ul>	<ul> <li>Lack of education</li> <li>Lack of preventive health care</li> </ul>	<ul> <li>Perception by parents that ECC is not serious health concern</li> <li>No training for health professionals about ECC</li> <li>Lack of education by health professionals</li> </ul>

Figure 1: WHO Global Oral Health figure (Note that the AMRO region has the highest caries rate of all regions).

Figure. 1 Dental caries experience (DMFT) of 12-year-old children according to WHO region



Source: WHO Global Oral Health Data Bank and WHO Oral Health Country/Area Profile Programme, 2000 Dr. Poul Erik Petersen, World Health Organization

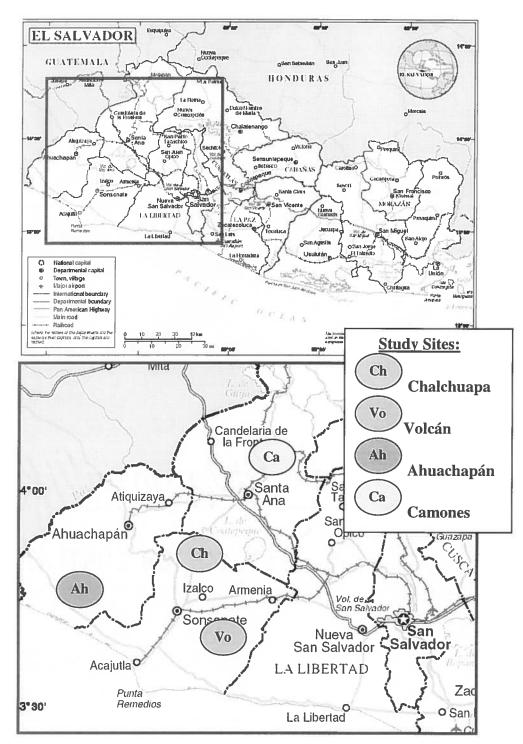
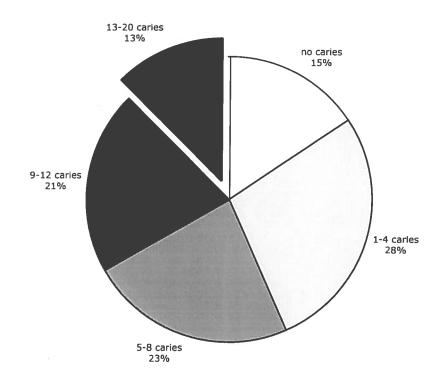
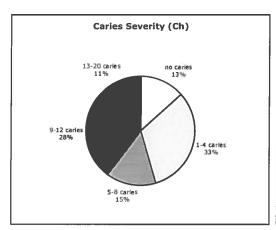
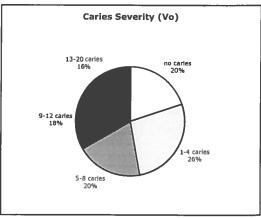


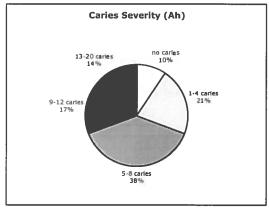
Figure 2: Map of El Salvador with Study Sites

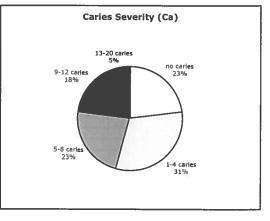
Figure 3: Caries Severity (N=168)

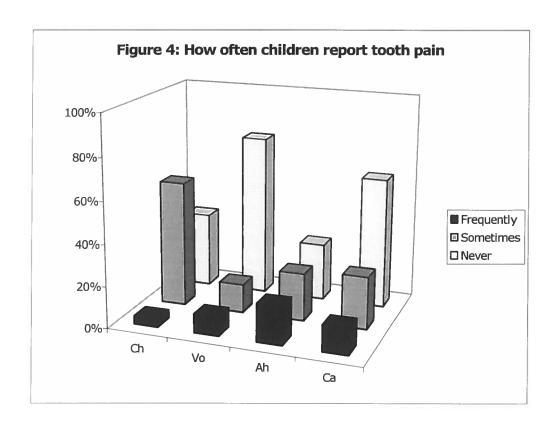


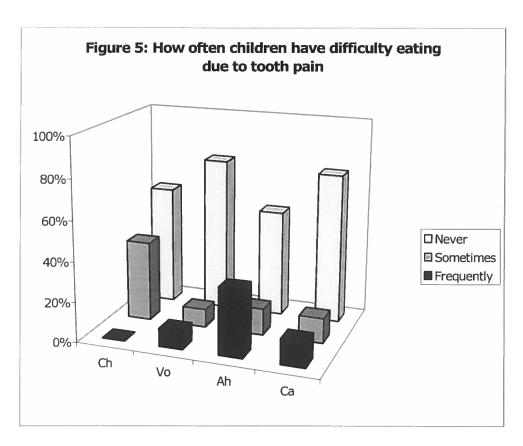


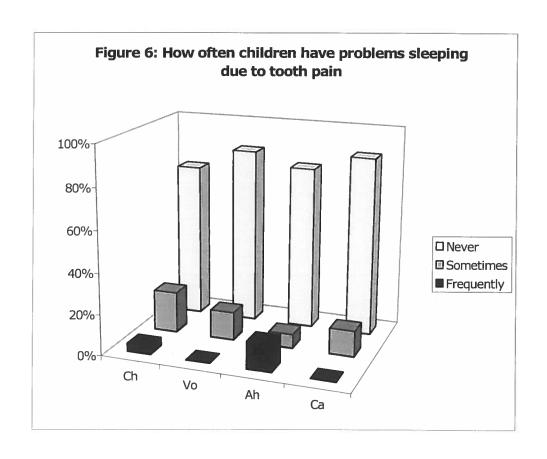


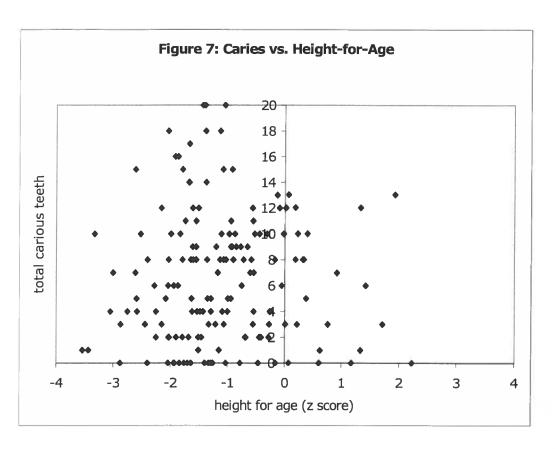


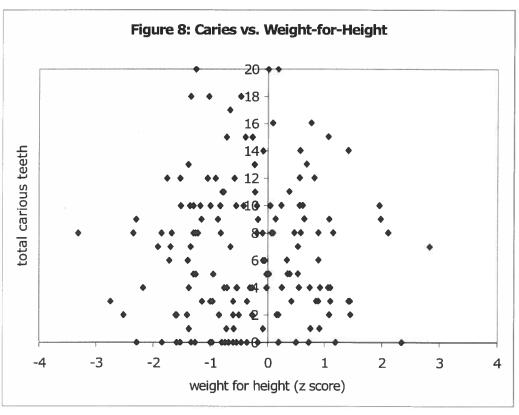


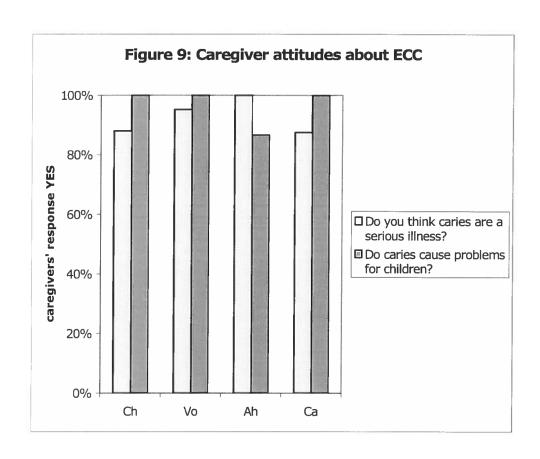


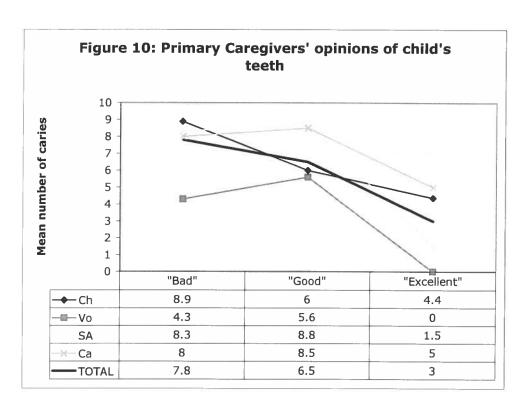












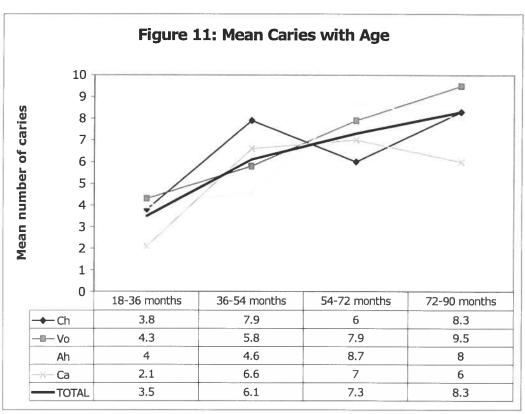
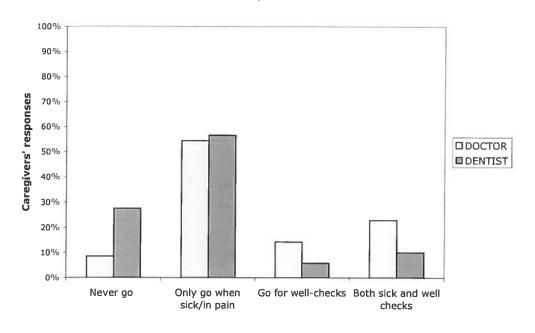


Figure 12: How often primary caregivers go to the doctor/dentist



**Table 1: Introduction to study sites** 

THE REAL PROPERTY.	duction to study sites
<b>Chalchuapa</b> (Ch)	<ul> <li>Rural/semi-rural agricultural region: former sugar cane plantations that were parceled out in last ten to fifteen years, located about one hour from Santa Ana via regular bus service, dirt roads, minimal services.</li> <li>Government-built housing after national highway project that displaced over 300 families: very densely situated 392 two-room houses, one waterspout for entire town. No space for agriculture. Frequent problems with gangs and violence.</li> </ul>
Volcán (Vo)	Rural mountainous region: remote homes tucked away on various levels of the Santa Ana Volcano. Tortuous roads. Most families survive on subsistence farming. Some areas are closer to main highway, and therefore have easier access to services in Santa Ana.
<b>Ahuachapán</b> (Ah)	Rural, isolated coastal region: about 300 total families live in three settlements, located over two hours by foot from the nearest urban center, with no regular transport. Most people survive by subsistence farming (corn and beans) on land that they do not own. The only water source is a small river, which is often dry.
Camones (Ca)	Site of one of Santa Ana's largest garbage dumps: located about 30 minutes by bus to Santa Ana. Single-family houses were built by the El Salvadoran government in 2000. Most people here make their income by scavenging at the nearby dump, which is now government-regulated.

**Table 2: Data Collection** 

	Inter- views with care- givers	Child dental exams, ht/wt	Mother focus groups	Store interviews and observa- tions	Extra data collection
Chalchuapa (Ch)	25	53	1(5)	1(1.5hr)	1. Rural dentist interview 2. In depth interview w/"Irma"
Volcan (Vo)	22	51	1(6)	1(2hr)	1. MSP Auxiliar interview 2. In depth interview w/"Esperan za"
Ahuachapan (Ah)	15	42	1(6)	1 (1.5hr)	In-depth interview w/ "Rubenia"
Camones (Ca)	8	22	1(7)	1 (2hr)	
Total	70	168	4 (24)	4 (7 hr)	
+ 4 mixed focus grou	ips with 2	22 health p	promoters f	rom each of th	nese regions

**Table 3: Children Descriptive Statistics** 

	N	Mean age (years)	Male (%)	Female (%)
Ch	53	5.1 (1.7-7.7)	53	47
Vo	51	4.4 (1.2-7.6)	59	41
Ah	42	5.1 (3.0-7.3)	46	54
Ca	22	4.0 (2.0-6.5)	46	54
Total	168	4.8	48	52

**Table 4: Primary Caregivers Descriptive Statistics** 

	N	Mean Age	Years of education	# of children	# in home	own home (%)	potable water (%)	electricity (%)
Ch	25	31.0	4.6	3.2	4.9	84	60	92
Vo	22	32.5	4.6	3.2	5.6	68	4.5	77
Ah	15	30.1	2.1	4.4	6.1	80	33	60
Ca	8	22.9	4.5	2.3	4.6	88	100	88
Total	70	30.3	4.1	3.3	5.3	79	41	80

**Table 5: Caries Prevalence** 

	N	1 or more teeth	FOUR maxillary incisors	FOUR mandibular incisors	1 or more molars
Ch	53	87% (46)	34% (18)	7.5% (4)	72% (38)
Vo	41	80% (41)	29%( 15)	3.9% (2)	71% (36)
Ah	42	91% (38)	43% (18)	2.4% (1)	81% (34)
Ca	22	77% (17)	32% (7)	4.5% (1)	60% (13)
Total	168	85% (142)	35% (58)	4.8% (8)	60% (121)

Table 6: Mean number of carious teeth per child (DMFT)

	Mean caries (of 20 teeth)	Maxillary incisors (of 4 incisors)	Mandibular incisors (of 4 incisors)	Maxillary molars	Mandibular molars (of 4 molars)
Ch	6.64	2.19	0.42	1.26	1.98
Vo	6.31	1.9	0.2	1.43	1.98
Ah	7.31	2.4	0.31	1.55	2.26
Ca	5.23	2.09	0.27	0.91	1.64
Total	6.52	2.14	0.30	1.34	2.01

**Table 7: Open-ended survey question** 

How do caries impact your children?	%
Pain	59
Damaged/broken teeth	12
Infections/other illness	11
Problems eating	9
Crying	8
Facial swelling	5
Stomach aches	5
Gum problems	3
Emotional upset	3
Bad breath	3
Unable to maintain weight	2
Appearance (ugly)	2

Table 8: Prevalence of Low Anthropometry by Age (measured by z score <-2.0)

Age (in months)	N	Stunted (low ht for age)	Wasted (low wt for ht)	Underweight (low wt for age)
0 to 36	21	10% (2)	10% (2)	38% (8)
36 to 54	37	25% (9)	8% (3)	16% (6)
54 to 72	63	20% (12)	0% (0)	3% (2)
72 to 90	38	16% (6)	5% (2)	13% (5)
TOTAL	159	18% (29)	4% (7)	13% (21)

## Tables 9a-c: Prevalence of known health behaviors related to ECC

9a: Breast and bottle-feeding habits

	N	Mothers who breastfed % (N)	Mean weaning age (months)	Mothers who bottle fed % (N)	Mean age bottle use* (months)
Ch	25	96% (24)	22.6	32% (8)	32.3
Vo	22	96% (21)	24.6	68% (15)	40.2
Ah	15	100% (15)	19.9	67% (10)	31.9
Ca	8	100%(8)	18.0	63% (5)	25.4
Total	70	97% (68)	22.0	54% (38)	34.2

<sup>\*</sup> age at which bottle use was discontinued

9b: Child's mean consumption per week (reported by primary caregiver)

			· · · · · · · · · · · · · · · · · · ·		
	N	Cola	"Sweets"	Milk	
Ch	25	1.8	6.3	4.0	
Vo	22	2.3	5.9	3.8	
Ah	15	0.7	7.7	0.6	
Ca	8	2.5	2.5	6.1	
Total	70	1.8	6.1	3.4	

9c: Tooth brushing habits (as reported by primary caregiver)

		PRIMARY CAREGIVER			CHILD				
	N	Own toothbru sh		Toothpast e in home	Own toothbrush	Brushes at least twice/day	with	Toothpa ste use ("almost always")	
Ch	25	100% (25)	92% (23)	84% (21)	88% (22)	76% (19)	32% (8)	79% (24)	
Vo	22	96 % (21)	91% (20)	91% (19)	77% (17)	68% (15)	50% (11)	52% (11)	
Ah	15	93% (14)	80% (12)	87% (13)	73% (11)	60% (9)	77% (10)	54% (7)	
Ca	8	100% (8)	100% (8)	100% (8)	100% (8)	63% (5)	25% (2)	100% (8)	
Total	70	97% (68)	90% (63)	88% (61)	83% (58)	69% (48)	46% (31)	68% (45)	

Table 10: Health Access and Preventive Health Habits

			Primary	Primary		
	Primary			caregivers		
	caregiver's most			reporting that		
	recent dental visit	caregiver's adult	child's vaccines	child has been to		
	(years)	teeth lost/pulled	are complete	the dentist		
			% (N)	% (N)		
Ch	1.9	3.4	100% (25)	24% (6)		
Vo	2.2	2.8	100% (22)	9% (2)		
Ah	3.0	9.0	93% (14)	27% (4)		
Ca	1.0	0.7	88% (7)	0 (0%)		
Total	2.10	4.14	97% (68)	17% (12)		

**Table 11: Local Store Observations and Interviews** 

	Ch	Vo	Ah	Ca
# of people who	50-60	25	10	25-30
come to store (per day)				
Main products sold at the store	Chips, cookies, bread, chicken, hot dogs, sausage, dry cheese, fresh cheese	meal, corn for chickens, sausages, sodas	sugar, bread, sweets, cookies medicine	Coffee, eggs, margarine, lard, sugar, cheese, rice, beans, soup, sweet bread, French bread
Most frequently sold products (adults)	Sugar, rice, soap	rice, milk	"Food to prepare and cook"	Cookies, soda, juice
Most frequently sold products (children)	Treats, cookies, chips	Diana (corn chips, cheetos, tortillas, potato chips)	Sweets, cookies	Diana products, artificial juice, chocobanana, popsicles
Do you sell fruits and vegetables?	Fruit: No Veg: tomato, onion, chile	Fruit: No Veg: No	oranges	Fruit: watermelon, oranges, bananas Veg: No
How much is milk?	\$0.86/0.5L	\$0.75/130g (powdered milk)	\$2.30/L	\$0.86/0.5 L
How much is soda?	\$0.80/1.5L \$1.00/2L	\$1.00/2L	1	\$0.75/1.5 L \$1.00/2L
How much soda do you sell week?	48 bottles/four days		12 L/week	51 L/week
			each, we sell about 12 per month."	"Yes, \$0.40, it's rare that we sell them, they are way up there high"
Do you sell toothpaste?			\$0.57/4 oz. tube "We sell about	

### BOX 1: The impact of caries on children's lives

# Open-ended survey question (mothers): How do caries affect your children? PAIN

- *Porque a veces agarra dolor, no pueden comer y lloran* Because sometimes pain takes over, they cannot eat, and they cry.
- Muelitas picadas duelen—siempre me dicen que tiene dolor, llora Broken molars hurt, he always tells me that he has pain, he cries
- Se les pican rápido, a veces le duelen, le duelen las encías, le sangran It breaks them up rapidly, sometimes it hurts, the gums hurt, [and] they bleed.
- Ellos lloran a veces no quieren comer porque deuele la muela. They cry sometimes and do not want to eat because their molars hurt.

#### **BROKEN/LOST TEETH**

- Es horrible, pierdan las piezas It is horrible, they lose [their teeth] in pieces.
- Microbios, se pican los dientes Microbes, they break up their teeth.

#### **INFECTION/ILLNESS**

- *Ellos que tienen [caries], caen mas enfermos* Those that have cavities are more sickly.
- Les dan problemas al estómago, mal olor en la boquita, se les pudren demasiado los dientillos It causes stomach problems, bad breath in the mouth, and the teeth rot a lot.

# Focus Groups (health promoters): How do caries affect children? LANGUAGE ACQUISITION

No se completa la palabra, no se entiende. Se critiquen "Ve, no puede hablar."
 Hasta un problema psicológica se puede causar por los dientes. They cannot pronounce the words, and no one can understand them. People criticize, 'look, he cannot talk.' You can even have psychological problems because of having bad teeth.

#### **NUTRITION**

• Cuando los dientes le duelen, no pueden comer—padecen de la desnutrición, falta de aumentar de peso, sin dientes no pueden comer la carne, la fruta, la tortilla, todo el alimento. When your teeth hurt, you cannot eat, then you get malnourished and don't gain weight. Without teeth, you cannot eat meat, fruit, tortillas, all types of food.

#### **SELF-ESTEEM**

- *Un niño que le cayó el diente no quiere sonreir. Los niños se llaman burro.* One little boy, his tooth fell out, and he didn't want to smile. The children call him donkey.
- Afecta hasta ser adultos porque no nos cuidamos los dientes de uno, les da pena.

NOTE: Boxes 2-8 contain qualitative data collected during 8 different focus groups with primary caregivers and health promoters. The plus (+) indicates that the focus group addressed the theme or answered the question presented in the box. If the quote was said by a caregiver, the region where the caregiver lives is in parenthesis following the quotes. If the excerpt was said by a health promoter, it is so noted.

BOX 2: Why are baby teeth important?

What purpose do baby teeth serve?								
	Ch	Vo	Ah	Ca				
Eating/chewing	+	+	+	+				
Appearance	+	+	+					
Influence permanent teeth	+	+		+				
Speaking	1.		+					
Hygiene	+							

- "To eat, to chew, to bite, to speak, to look good, to feel beautiful" (Ah).
- "They are what we begin to eat with, they are the first thing we use to feed ourselves. All the future teeth depend on them. Sometimes people don't value the primary teeth, but they affect how the next teeth are going to be" (Vo).
- "[Children] only have them for a time, but they are so important for the nutrition of the baby. If the teeth are bad, that can be passed along to the mature teeth. you have to learn to take care of your teeth when you are small, forming good habits for all of your life" (health promoter).
- "The [baby teeth] are part of our bodies, and if we are missing them, it is as though an important part of our body is missing as well" (health promoter).

BOX 3: Suggested causes of caries in children

How do children get caries in their primary dentition?						
	Ch	Vo	Ah	Ca		
Lack of tooth brushing	+	+	+	+		
Sugary snacks	_   +	+		+		
Gum, candy	+	+		+		
Baby bottle	+		+			
Iron		+	+			
Ignorance		+				
Soda				+		
Nutritional deficiency	+					

- "Not brushing them good or consistently enough. Eating a lot of sweets, a lot of chocolate, chewing gum, chewing ice. Sometimes because of iron, the blackness the iron gives them" (Vo).
- "Not all teeth are equal" (Ch).
- "My father did not let me eat sweets, nor sugars, but I lost all my molars[...]I think it was lack of calcium" (Ch).
- "Cavities are like a little animal that passes from the mother to the child" (health promoter).
- "Sometimes by not brushing, by eating lots of sweets, sometimes they say because you give them a lot of iron. There are people who say it is genetics, that it comes from your grandparents" (health promoter).
- Dissent: "I lost all my teeth as a young woman, but I never ate sweets. It was natural" (Ch).

### BOX 4: Health Beliefs and Behaviors: BREAST AND BOTTLE FEEDING

Breastfeeding							
	Ch	Vo	Ah	Ca			
is important for children	+	+	+	+			
"on demand" is a common practice in the community	+	+	+	+			
"on a schedule" was discussed	+	+					
excessively is	+	+					

- "Breast first, bottle second."
- "At night, we sleep all together. When they want [the breast], I give it" (Ch).
- "When he asks or cries or puts his hand in his mouth [...] It's
  okay to give them the breast every time they cry [...]if not, the
  bottle" (Vo)
- "There are mothers that give the breast when the child cries, there are mothers that put their babies on schedules" (health promoter).
- "It is more common in rural areas that they breast feed the baby in the night, they sleep together and breast feed all night, the baby wakes up six times or more to take the breast" (health promoter).
- "When they need something, there is no way of advising.

  Moms, how are they supposed to know? Danger? No" (health promoter).

Baby bottles				
	Ch	Vo	Ah	Ca
can make children sick	+	+	+	+
ruin children's teeth	+	+	+	+
can be helpful	+	+		
care must be taken with hygiene		+		
are expensive				+

- "The teeth get eaten up by the rubber of the nipple" (Ch).
- "With the bottle, you have to be more careful to wash it well, boiled water [. . .] If someone has all the possibility to breastfeed their child, better. Those that breast feed do not get sick" (Vo).
- "They say the bottle affects the teeth" (Vo).
- "It isn't very recommended to give the bottle because it ruins the children's teeth. They don't wash the bottle very well, and it gives infections. It is also very expensive to buy milk" (Ca).
- "If they drink too much bottle, it gives them cavities through microbes" (Ah).
- "On one hand, it isn't good to give a baby a bottle instead of the breast, but sometimes, the woman doesn't have milk, and she has to give her baby the bottle" (health promoter)
- "Only the bottle. They don't take it out of their mouths. Flour, corn, sugar water, juice, artificial juice, coffee in the bottle, they arrive at the preschool" (health promoter).
- "Some times moms leave their child in the hammock with the bottle. Sometimes, bigger kids suck on the bottle of the newborn, they share the bottle... The dirtiness of the bottle is passed on to the child" (health promoter).

BOX 5: Health Beliefs and Behaviors: SWEETS and SODA Consumption

Why do you give your children sweets?							
	Ch	Vo	Ah	Ca			
Children ask for them	+	+	+	+			
So children won't cry	+	+	+	+			
Custom/habit/tradition	+	+		+			
Children like them		+		+			
There is nothing to eat (help with hunger)		+	+				
To keep children occupied so that mothers can							
get work done		+					
Their father brings them	+						

- "It is a custom that we give sweets and gums to kids" (Ca).
- "A mother wants to go out, and the child doesn't want her to go, she has to give him candy so that he stays in the house [...] They pass all the time buying sweets in order to keep them quiet. A stimulus, a gift, a prize" (health promoter).
- "Sweets produce parasites" (Ah).
- Sometimes [we give sweets] so they won't cry. 'Take a little candy." Because the moms are doing housework, so that the child stays busy" (health promoter).
- "Because they don't have anything to give them to eat" (Ah).
- "When his father comes home from work, he is the one who brings the sweets" (Ch).
- "Because they think it will satisfy their child's hunger . . . because they are watching the soap operas, so that they keep themselves occupied. . . a lollipop so that they can last a little bit longer. I have seen a boy was hungry, but the lunch wasn't ready yet" (health promoter).

Why do you give your children soda and artificial juices?							
	Ch	Vo	Ah	Ca			
The children see it in the store and ask for it	+	+	+	+			
As a treat	+	+					
Bad habits	+			+			
They see us drinking it	+						
Their father brings it from the city	+						
Advertisements encourage them to ask for it				+			
It is easier than preparing juice	+						

- "Soda is much easier, then later, you don't have to make juice" (health promoter).
- "Soda sucks up your calcium, debilitates your bones, starting in pregnancy" (health promoter).
- "I don't have time to prepare juice, so I send my child to buy soda" (Ch).
- Some moms buy [soda] for lunch. The family drinks it—how are they going to say no if they are drinking soda? (health promoter).
- "Those things that they have in the store we should not buy them because children alone cannot buy those things" (Vo).

Box 6: Health Beliefs and Behaviors: IRON

How do iron supplements affect your children	s teeth	?		
	Ch	Vo	Ah	Ca
Stains them/turns them black	+	+	+	+
Breaks them/causes caries		+	+	+
Discuss need for washing teeth after giving iron	+		+	

- "[Iron] stains [the teeth], makes them black, breaks them. If you give it to them with sugar water, it is much better. Even surer, wash the teeth well after giving it to them" (Ah).
- "Giving them iron, how their teeth rot! Iron causes diarrhea, it makes their teeth all black. In my children, all their teeth fell out because of the iron" (Vo).
- "Because it makes their teeth black and it breaks them" (Ca).
- "Iron is very recommended for children, in the moment, after taking the iron, they have to brush their teeth" (Vo).
- It makes them black, then they go rotting. . . Lots of mothers don't give the iron because it damages their teeth. They don't give it because of the teeth problem" (health promoter).
- "The black only stains, it is not carious. Drink water after taking the iron, so you don't stain the teeth" (health promoter).

#### Box 7: Health Beliefs and Behaviors: SALIVA SHARING

Do mothers share saliva with their babies				
(i.e. blowing				
on food, sharing spoons, premasticating)?				
	Ch	Vo	Ah	Ca
Very common in their community	+	+		
Deny that this occurs in their community			+	+

- "I have broken up candy with my mouth in order to give it to my child, but I do not chew the food because we have bacteria in our mouths that we can pass on" (Ch).
- "Perhaps it is lack of hygiene. Mothers think they are doing it with love and affection. It is common, tradition, the habits of each person" (Vo).
- "Yes they do it! They blow on food, pass it to their mouths, share the spoon, share the plate of food" (health promoter).
- "I visited a place and the woman pre-masticated the food for her child. I
  told her she shouldn't, that bacteria can pass from mouth to mouth. She
  understood me. Lots of women do this. They bite the candy. Maybe
  they aren't telling you because they are embarrassed" (health
  promoter).

Box 8: Dental health: external and internal influences

What influences your dental health bel	haviors?			
	Ch	Vo	Ah	Ca
Financial/economic hardship	+	+	+	+
Cooperation of the children		+		+
Forgetting/not having time	+	+		
Not knowing what to do		+		

## Money:

- "There is no money to buy toothbrushes, it is far to go to Ch, takes a whole day, and it is expensive. I have 11 children, it's expensive" (Ch).
- "Not having economic resources in order to give them better food and drink in order to protect their teeth" (Ah).
- "It is also very expensive to buy milk" (Ca).
- "Sometimes, there is simply no toothbrush" (health promoter).
- "There isn't money to buy toothpaste, but they could use salt or baking soda. There is money to buy a rag, but not for a toothbrush" (health promoter).
- "Economic resources affect your dental health" (health promoter).

## Health access:

- "Mothers work, and they leave their kids with people who don't care. There is no time" (Vo).
- "Women hardly recognize the benefits to going to the dentist, also there are economic problems, there is no access in rural zones, and taking out the bad teeth is the only treatment. Taking out a tooth costs \$2, filling a tooth costs \$10, and sometimes the fillings you get. . .fall out in two weeks" (health promoter).
- "When mothers don't get prenatal care, there is poor education in terms of taking care of teeth" (health promoter).
- "Where I work, the mothers don't have the resources. Artificial juice is cheaper [than cola], depends if the juice is nutritive for the children" (health promoter).

## Education/Awareness/Advertising:

- "The lack of attention of the mother and father" (Vo).
- "We have to be aware of what our children are doing and how they are feeling. If there is even a little half cavity, we should take them to the dentist to cure them before the other teeth get contaminated" (Ch).
- Caring for our children's teeth depends on us. It is important to create a habit of brushing your teeth" (Ca).
- "Mothers don't even know what snacks are. Just if they have a little bit
  of extra money, maybe they send [their child] to the store to buy *churros*,
  sweets" (health promoter).
- "The commercials come out on the television. When we go to buy something in the store, they see it, and they want it" (Ca).

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#### **APPENDIX A: Dental Exam Form (Tooth Template)** F Child's initials \_\_\_\_\_ M Date of Birth \_\_\_\_\_ Weight \_\_\_ Height \_\_\_\_\_ RIGHT LEFT С D В E F G Н S R Q P 0 N M L K Child's initials \_\_\_\_\_ F M Date of Birth \_\_\_\_\_ Height\_ Weight\_ RIGHT LEFT С В D F G Н E R 0 T Q P N S M L K Child's initials \_\_\_\_\_ F M

Height _ RIGHT		. 1	Weight	LEFT					
A	В	С	D	Е	F	G	Н	I	J
Т	S	R	Q	P	0	N	М	L	K

Date of Birth \_\_\_\_\_

# APPENDIX B: Primary Caregiver Interview Guide (English and Spanish)

	ECC Primary Caregiver Interview Inmographic information:	nstrume	nt: <u>ENC</u>	GLISH	
1.	Age				
2.	Years of education			_	
3.	How many total children do you have?	?		_	
4.	How many children under 7?			_	
5.	How many people live in your house?				
6.	Where/how does your family get its in	come?			
	Do you have your own house?		Y	N	
8.	Do you have electricity?		Y	N	
9.	Do you have potable water?		Y	N	
10.	Do you cook over fire or with gas?		Fire	Gas	
He	alth Practices:				
11.	When do you go to the doctor?				
	<ul> <li>a) Only when I am sick</li> </ul>				
	b) For check-ups				
	c) When I am sick and for well ch	ecks too	•		
	d) I never go to the doctor				
12.	When do you go to the dentist?				
	a. Only when I have a problem				
	b. For check-ups				
	c. When I have a problem and for	checkup	os too		
	d. I never go to the dentist				
13.	Do you drink milk? Y	N		How often?	/week
14.	Do you drink soda? Y	N		How often?	/week
15.	How do you care for your teeth? (open	-ended)			
15a	. In your opinion, how are your teeth?	Excel	lent, Ok	, Bad, Horrible	
16.	Do you have your own toothbrush?	Y		N	
17.	Do you have toothpaste?	Y		N	
18.	How often do you brush your teeth?	1/day		2/day	3/day
19.	When was the last time you saw the de	ntist?	(	in months)	
	Why did you go? (circle as many as a		\	,	
	a. toothache	11 27			
	b. loose/rotten tooth				
	c. dentures/partials				
	d. routine check-up				
	e. because I was taking my child to th	e dentist	t		
	f. other				
21.	In the past 3 months, have you had any	problen	ns with	your teeth, gum	is, mouth
	a) Pain	F		,, 5	,
	b) Loose teeth				
	c) Bleeding				

d) Inflammation of gums			
e) Other (explain)			
22. How do you make your child sto	op crying during the	day? (open-ended)	
23. How do you make your child sto	op crying at night? (o	open-ended)	
24. What do you do to take care of y	our children's teeth?	? (open-ended)	
The specific questions on children will	be asked for ONE r	andomly selected child < 6	
years old:		-	
25. Initials			
26. Age/Sex		_M_F_/	
27. Did you have signs of PTL with this b	aby?	Y N	
28. How many months pregnant were you	when the baby was	born?	
29. Did you have prenatal care?	·	Y N	
30. If yes, how many visits?		0 1 2 3 4 5 6 7 8 9	
31. Are your child's vaccinations complete	te?	Y N unsure	
32. Did you breastfeed this child?	Y N		
33. Until what age? (in months)			
34. Did this child use a baby bottle?	YN		
35. Until what age? (in months)			
36. How often did/does he/she sleep with	Never, seldom, often, very		
often			
37. What did he/she take in the bottle?	Formula, Water, juice, milk		
coffee, soda, other			
38. If juice, what kind of juice?			
39. How often does your child have:	Coke/soda	/day	
	Candy, gum	/day	
	Milk	/day	
40. Does your child have his/her own toot	thbrush?	Y N	
41. How often does he/she use it?		Never, 1/day, 2/day, 3/day,	
unsure			
42. How often does your child have tooth often	paste?	Never, seldom, often, very	
43. Has your child been to the dentist?		Y N	
44. Has your child ever had to have a toot	h removed?	Y N	
45. How many times?		0 1 2 3 4 5 >5 times	
46. Why did he/she go?			
47. How often does your child cry at nigh	t because his/her mou	uth hurts? Never, seldom, ofte	n.
very often		,,,,,	,
48. How often does he/she have problems	eating b/c his/her me	outh hurts? Never, seldom,	
often, very often	C	,	
49. How often does your child complain of	of pain in mouth/teeth	h? Never, seldom, often	n,
very often	•	, , , , , , , , , , , , , , , , , , , ,	,
50. In your opinion, how are you child's to	eeth?	Excellent, Ok, Bad, Horrible	,
51. How is the general health of your child		Very healthy Somewhat	
healthy Sickly		J 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	

52. Does your child have:		Y			
N	Asthma		Y		
N	Ear infections		Y		
N	Diarrhea		Y		
N	Frequent skin infect	tions	Y		
N	•				
53. How would you describe the temperament of your child? Shy Moderate Outgoing					
Health Beliefs:					
54. Where do you think cavities in baby te		t causes them? (op	en-ended)		
55. Do you think that early childhood cavi	Y	N			
56. Do you think they are a problem for you If so, how do you think they affect you		Y	N		
57. Do you have any home remedies for the		Y	N		

I. Información logística:  1. Edad (de la madre)  2. Nivel de educación (de la madre)  3. ¿Cuántos hijos tiene usted?  4. ¿Cuántos son menores de 7 años?  5. ¿Cuántas personas viven en su casa?  6. Ingresos económicos: como gana (la familia) un sueldo?  7. ¿Viven en su propia casa? si no  8. ¿Tienen luz? si no  9. ¿Tienen agua potable? si no  10. ¿Cocina con leña o gas? Leña Gas  II. Prácticas: Ahora vamos a hacer unas preguntas sobre usted y su salud:  11. ¿Cuándo va usted al doctor? 12. ¿Cuándo va usted al dentista?  1. Solo cuando estoy enferma 1. Solo cuando estoy mal con los dientes  3. Los dos 2. Para chequeos dientes  3. Los dos 2. Para chequeos  4. En verdad, nunca voy al 3. Los dos  doctor 4. En verdad, nunca voy al dentista  13. ¿Usted toma leche? Si No ¿Cada cuanto? (toma leche pura? o solo en café?)  14. ¿Usted toma cola? Si No ¿Cada cuanto? Si No §Cada cuanto?
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cuanto? (toma leche pura? o solo en café?)  14. ¿Usted toma cola? Si No ¿Cada cuanto?  15. ¿Cómo cuida usted a sus dientes?  15ª ¿En su vida, usted tiene cuántos dientes que ha hecho sacar o que han caido?  15b. ¿En su opinion, como están sus dientes? Buenos, Mas o menos, Malos,
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15b. ¿En su opinion, como están sus dientes? Buenos, Mas o menos, Malos,
15b. ¿En su opinion, como están sus dientes? Buenos, Mas o menos, Malos,
Horribles
16. ¿Usted tiene su propio cepillo de dientes? Si No
17. ¿Usted tiene pasta dental en la casa? Si No
18. ¿Cada cuánto cepilla los dientes ud?cada
19. ¿Hace cuánto tiempo que usted se ha ido a ver al dentista?
20. ¿Por qué fue la última vez? a. Dolor de la muela
b. dientes podridos
c. un chequeo/control
d. porque fui con mi hijo/a
e. otra
21. Len los ultimos 5 meses, usteu na tendo argun problema con los dientes. las encias. O
21. ¿En los últimos 3 meses, usted ha tenido algún problema con los dientes, las encías, o la boca, (incluyendo hoy) (puede marcar todos los que aplican)
la boca, (incluyendo hoy) (puede marcar todos los que aplican)  a. Dolor o sensibilidad
la boca, (incluyendo hoy) (puede marcar todos los que aplican)

<ul><li>d. Inflamación en la boca</li><li>e. Otros problemas (explica)</li></ul>				
22. ¿Cuando su niño/a está llorando durante el dia			a calmarlo	?
23. ¿Cuando su niño/a está llorando de noche, que	hace uc	d. para calma	ırlo?	
24. ¿Que hace usted para cuidar a los dientes de lo	s niños	(su hija/hijo	)?	
III. Ahora vamos a hacer preguntas acerca de años)			o mas cerc	ca de 5
25. Nombre Edad	<del>-</del>	años	Sexo:	• м
F Education of F	•	unos	DCXO.	, 141
27. ¿Cuando estaba embarazada con esté bebe, ud meses? Si No	tenía d	olores del pa	rto antes o	de los 9
28. ¿De cuantos meses del embarazo dio a luz?				
29. ¿Recibió controles prenatales cuando estuvo es No	mbaraza	da con este l	bebe?	Si
30. ¿Cuantos controles? 1 2 3	4	5 6	7	8
31. ¿Las vacunas estan completas?	Si	No	No sé	Ş
32. ¿Dió pecho as su bebé?	Si	No		
33. ¿Hasta qué edad (en meses)? 34. ¿Su hijo/a tomó la pacha?	Si	No No		
35. ¿Hasta que edad? (en meses)	51	140		
36. ¿Cada cuanto dormía con la pacha en la boca?	Nunca	a De repente	Frequent	tamente
Casi siempre				
37. ¿Que chupaba en la pacha? agua, jugo, cat	é, cola	., leche, ag	ua azucar	ada,
formula 38. Si jugo, ¿que clase de jugo? Explica:				
50. bi jugo, ¿que clase de jugo. Expilea.				
39. ¿Cada cuánto su hijo consume: Cola				
Caramelos/d	ılces/ch	icle		
Leche	C:			
40. ¿Su hijo/a tiene su propio cepillo de dientes? 41 ¿Cada cuánto lo usa?	Si Nunca	No a 1/d		3/dia
no sé	Tvullet	. 17d	ia Z/Gia	3/dia
41a. ¿Usted le ayuda a su niño/a a cepillarse? Nuncasi siempre	ca	de repente	frequent	tamente
42. Cada cuanto su hijo/a usa pasta dental? Nunc casi siempre	a	de repente	frequent	tamente
43. ¿Su hijo/a ha ido al dentista?	Si	No		

44. ¿Cuantas veces?  45. ¿Por qué fue?		
45. ¿Alguna vez este niño tenía que hacer sacar un diente/una muela?  No	Si	
¿cuantos dientes? 1 2 3 4 5 6 o más		
47. ¿Cada cuanto llora en la noche por dolor de la boca? Nunca, de rep	ente	
frequentamente, casi siempre	, ,	
48. ¿Cada cuanto tiene problemas comiendo por dolor de la boca? Nunca,	de repe	ente.
frequentamente, casi siempre	- v rope	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,
49. ¿Cada cuanto le dice que le duele la boca/muela/diente? Nunca, de rep	ente,	
frequentamente, casi siempre	,	
50. ¿En su opinion, como están los dientes de su hijo/a? Excelentes, Más	o meno	S
buenos, Malos, Horribles		
51. ¿Como está su hijo/a de salud? muy sano más o menos		muy
enfermo		_
52. ¿Su hijo/a sufre de:		
Asma La gripa (frequentamente) Infecciones de la garg	ganta	
Infecciones de los oídos Diarrea Grano	s (infec	ciones
de la piel)		
53. ¿Cómo es la forma de ser de su hijo/a? tímido/callado regula social/muy hablador	r	muy
IV. Creencias:		
54. ¿Qué piensa ud.—de que vienen los caries en los dientes de leche?		
54. ¿Que piensa du.—de que vienen los caries en los dientes de leene:		
55. ¿Usted cree que los caries en los dientes es una enfermedad seria?	Si	no
no sé	G.	
56. ¿ Los caries causan problemas para los niños?	Si	no
no sé		
¿Cómo les affectan a sus hijos?		
57. ¿Usted tiene remedios caseros para cuidar a los dientes de sus hijos? Explica		

## **APPENDIX C: Store Observation Guide**

## Tienda observations

Town:		
	f applicable):	
	er:	
Questions fo	r owner:	
1. What	are the main pro	ducts you sell here?
2. What	is the most popu	alar thing you sell to adults?
3. What	is the most popu	lar thing you sell to kids?
		ole do you think come into your store in a normal day?
		products to sell?
6. Do yo	u sell fresh fruit	here? (if so, what kinds?)
		? (if so, how much is it per liter)
		? (how much per liter?)
		d you estimate you sell per week?
	ouys most of the of gum? One bag	candy, chips, etc? How much is one piece of candy? One
-	-	nes here? How much are they? How often do you sell one
		here? How much is it? How often do you sell it?
		Idren eat more sweets because you own this store?
•	•	•
Things to not	te:	
<ol> <li>Does t</li> </ol>	the store have a	refrigeration system?
2. If so, v	what's in the fric	lge?
3. How r	nany 2-liter bott	les of soda do you see?
	nuch milk do yo	
		of candy (penny candy jars)?
	_	(estimation in meters)
		od goods are packaged/processed? (??)
8. Do yo	u see any toothb	rushes? Toothpaste?
Observations		period of time OR observations):
Time of	Age of patron	Description of what they bought
day		

Time of	Age of patron	What they bought
day		

## Appendix D: Mother Focus Group Guide

## **Questions for mothers:**

- 1. Do you think that teeth are important? Why do you think they are important? What do you use them for?
- 2. Do you think baby teeth are important? Why/why not?
- 3. How do you think children get caries in their teeth?
- 4. What responsibility is it for parents to take care of children's teeth? What age do you think a child is able to brush his/her own teeth all on own without help? What sorts of things should they do? Do you ever brush your children's teeth?
- 5. I have noticed in other villages that when I ask about moms sharing food with their children, people get really shy and tell me they never share food with their children, even though I have seen it happen. Why do they not want to tell me this? How do you share food with your child (premasticating, blowing on it, sharing spoons)
- 6. How do you learn to breastfeed? Who teaches you? Breastfeeding on demand? In general, do your babies sleep with you? Do you feed them in the night?
- 7. What do you think about baby bottles? When, why do you use baby bottles? When you give your child the bottle, do you hold it? Do they? Do they carry it around?
- 8. What about iron supplements—Do you give your children their iron supplements? What do you think they do to the children's teeth?
- 9. Where do you get/buy your food for meals?
- 10. What kinds of things do your children eat during the day between meals? Do your children eat snacks? What is a snack for them? What are their favorite snacks?
- 11. Do you get enough dental care for you and your kids? Why or why not?
- 12. What do you think are the greatest barriers to dental care? How could we help make your teeth better? What would help you most to take care of your children's teeth?

#### Extra questions:

What do you do to make your child stop crying? (do you do different things in the day and in night?)

## Appendix E: Health promoter focus group guide:

## **Questions for Health Promoters**

- 1. What do you think the health priorities are in your village? For adult health? Child's health?
- 2. Do you think that teeth are important? Why do you think they are important? What do you use them for?
- 3. Do you think baby teeth are important? Why/why not?
- 4. How do you think children get caries in their teeth?
- 5. What responsibility is it for parents to take care of children's teeth? What age do you think a child is able to brush his/her own teeth all on own without help? What sorts of things should they do? Do you ever brush your children's teeth?
- 6. I have noticed in other villages that when I ask about moms sharing food with their children, people get really shy and tell me they never share food with their children, even though I have seen it happen. Why do they not want to tell me this? How do you share food with your child (premasticating, blowing on it, sharing spoons)
- 7. Do you talk to women about breastfeeding? Breastfeeding on demand?
- 8. What do you think about baby bottles? Do you talk to women about bottles? When, why do you use baby bottles? When you give your child the bottle, do you hold it? Do they? Do they carry it around?
- 9. Do you think that women give their children iron supplements? Why/why not?
- 10. Do you give moms advice about healthy snacks? What kinds of things do the children eat during the day between meals? Do the children eat snacks? What is a snack for them? What are their favorite snacks?
- 11. What do you think are the greatest barriers to dental care? How could we help make your teeth better?