DOI: 10.1111/j.1365-263X.2012.01260.x

INTERNATIONAL JOURNAL OF PAEDIATRIC DENTISTRY

REVIEW

A systematic review of risk factors during first year of life for early childhood caries

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International Journal of Paediatric Dentistry 2012;

Background. Early childhood caries (ECC) describes dental caries affecting children aged 0–71 months. Current research suggests ECC has important aetiological bases during the first year of life. Gaps in knowledge about disease progression prevent the effective and early identification of 'at risk' children.

Aim. To conduct a systematic review of research studies focusing on (a) acquisition and colonization of oral bacteria and ECC and (b) risk and/or protective factors in infants aged 0–12 months.

Design. Ovid Medline and Embase databases (1996–2011) were searched for RCT, longitudinal, cross-sectional and qualitative studies. Two investigators undertook a quality assessment for risk of bias.

Results. Inclusion criteria were met for (a) by four papers and for (b) by 13 papers; five papers were rated medium or high quality. Bacterial acquisition/colonization and modifying factor interrelationships were identified, but their role in the caries process was not clarified. Key risk indicators were infant feeding practices (nine papers), maternal circumstances and oral health (6) and infant-related oral health behaviours (4).

Conclusion. This review confirmed that factors occurring during the first year of life affect ECC experience. Despite heterogeneity, findings indicated maternal factors influence bacterial acquisition, whereas colonization was mediated by oral health behaviours and practices and feeding habits.

Introduction

Early childhood caries (ECC) describes dental caries affecting children 71 months of age or younger[1]. Studies in nonindustrialized and industrialized countries have reported caries prevalence in very young children to vary between 28% and 82%[2–6] depending on the population studied. There are few Australian studies reporting caries prevalence in infants and preschool-aged children with most Australian data coming from school-aged children[7, 8] accessing the public school dental services in each state. This data indicate that 47% of 5- to 6-year-old children have cavitated carious lesions, and of these lesions, 80% are active and untreated[9]. A recent Australian study estimated that 45% of hospital 'oral cavity' admissions for children aged <2 years of age were related to dental caries[10]. It is important to understand the natural history of ECC in order to implement effective preventive strategies. Prevention and early intervention are critical as children with ECC may experience pain and infection of dental origin and exhibit poor sleeping patterns, altered eating habits and behaviour, poor self-esteem[11], reduced speech production and communication skills, low body weight and height[12] and failure to thrive[13, 14]. Furthermore, ECC is a strong predictor of dental caries experience in later life[15].

Although ECC is recognized as multifactoral in nature, there are gaps in our knowledge as

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to how the risk factors interrelate and why some children suffer a greater burden of disease than others. In particular, little is known about the infant's oral environment prior to, and during, early tooth eruption. Influences in the first year of life may have an important effect on the health of the primary dentition. Most studies report on ECC once the teeth have erupted into the mouth and there are visible signs of the disease process, or on the interrelationship of bacteria between a mother and her child. For example, a recent systematic review of preschool-aged children and ECC yielded 120 papers, and of these, one paper investigated pre-dentate children only[16].

The present paper describes a systematic literature review addressing ECC during the first year of life. Two independent searches were undertaken to capture as many papers as possible addressing the overall research question: What factors occurring during an infant's first year of life influence the initiation and progression of ECC? The first objective was to address the association between the acquisition of oral cariogenic bacteria and caries outcomes in infants; the second objective was to identify the proposed determinants of ECC during the first year of life.

Materials and methods

Search strategy

Two searches were conducted to identify, describe, quality-appraise and synthesize published studies: first, exploring acquisition and colonization of oral bacteria in children during their first year of life and subsequent development of ECC; second, addressing/exploring the risk and/or protective factors for ECC in children aged up to 12 months. The outcomes of both searches were then synthesized.

The search strategy undertaken is shown (Fig. 1). Separate searches of Medline Ovid SP and Embase Ovid SP electronic databases were undertaken (April 2011), storing results in separate Endnote libraries and deleting duplicates. The first sieving of papers, undertaken by two independent reviewers, examined papers by title and abstract according to inclusion and exclusion criteria as below. The first reviewer (PL) scrutinized all identified papers from both searches, and at least 10% of papers in each search were independently reviewed by a second reviewer (MG or SLB). Where title and/or abstract were unclear, the full text was obtained. All papers meeting the inclusion criteria were retrieved, and full texts were reviewed by two independent reviewers (Search 1: addressing bacterial acquisition and colonization: by PL and MG; Search 2: addressing all proposed determinants of ECC: by PL and SLB), using screening questions of the Critical Appraisal Skills Programme (CASP)[17]. All excluded papers were recorded in a spreadsheet along with the reason for their exclu-

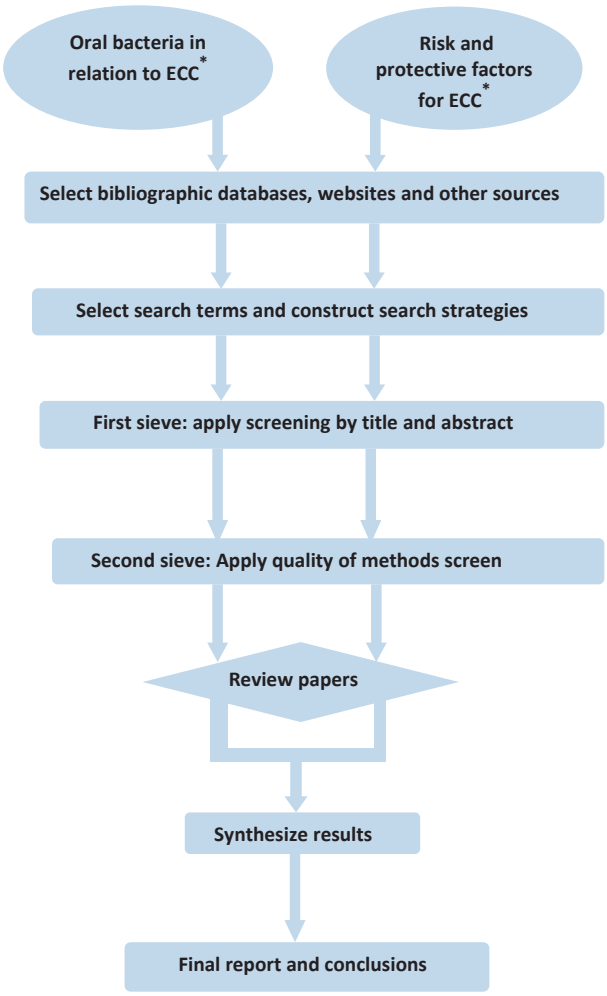

Fig. 1. Flow chart of the search strategy used for two searches.

sion. If reviewers disagreed on a paper, the protocol required them to discuss to consensus. Included papers were then quality-appraised using the full CASP criteria[17]. Finally, data were extracted onto a spreadsheet for analysis and synthesis. In addition, authors of papers requiring clarification on particular aspects of their study relevant to this review were contacted by email for further information.

Population sample and search terms

Inclusion criteria for the population group in both searches required studies to report on children aged 0–12 months. Where a broader age range was reported in the paper, information relating specifically to 0- to 12-month-olds must have been included for the paper to be included in the present review. Search strings were determined with the assistance of two medical research librarians and modified as necessary for each database (Table 1).

Inclusion and exclusion criteria

Additional to the age limitation, only papers published between 1996 and April 2011 were included. The latter limitation was applied because laboratory methods used to analyse cariogenic bacteria have progressed markedly since the mid-1990s, and it is unlikely that earlier published papers would supplement this review concerning current knowledge (S. Daspher, Personal communication). Papers where participants were physically, intellectually or medically compromised, or had syndromes, were excluded, as were papers other than primary studies (e.g. guidelines, recommendations, conference proceedings, letters or similar).

Additional requirements applied to Search 1 (acquisition and colonization of oral bacteria and ECC) were a cariogenic bacterial assessment must have been undertaken for an infant by the time they were aged 12 months, and these infants must have also had a caries assessment by 18 months of age. The latter requirement was based upon the assumption that caries in newly erupting teeth may take several months to become clinically visible[18, 19], and maxillary anterior teeth do not generally erupt until an infant is about 8–10 months of age.

Quality appraisal

The CASP criteria[17] were selected for this review as an established and accepted appraisal tool allowing for a broad range of study designs to be appraised. The criteria were developed by the Public Health Resource Unit of the UK National Health Service[17] and applied to each study in the present review to enable assessment across three broad areas: study methodology (internal validity)[20]; reporting of results (reliability); and applicability or generalizability (external validity)[20], with particular reference to studies in Victorian/Australian populations. To enable inter-study comparisons, each area was then rated high, medium or low depending on the strength of reporting. An overall rating was then determined based on these outcomes, weighting 'materials and methods' and 'results' sections more heavily than 'applicability'. Working independently, quality appraisal was undertaken by the same two

Table 1. Search strings used for two searches.

Search strings for Search 1 addressing the association between the acquisition of oral cariogenic bacteria and caries outcomes in infants:
exp *Dental Caries/AND *Streptococcus/or *Streptococcus mutans/or *Streptococcus sobrinus/or *Saliva/or *Lactobacillus casei/or exp *Periodontal Diseases/
Search strings for Search 2 addressing all proposed determinants of early childhood caries (ECC):
exp *Dental caries/AND *Streptococcus/or *Streptococcus mutans/or *Streptococcus sobrinus/or *Saliva/or *Lactobacillus casei/or exp *Periodontal Diseases/*Fluorides/or exp *Oral Hygiene/or *Oral health/or exp *Dental Prophylaxis/or (*Diet/or *Diet, Cariogenic) or (*Breast Feeding/or Feeding Methods/or *Bottle Feeding/) or exp *Infant Nutritional Physiological Phenomena/or *Parent–Child Relations/or *Mother–Child Relations/or exp *Mothers/or *Social Conditions/or exp *Socioeconomic Factors/or *Attitude/or exp *Attitude to Health/or *Child Rearing/or *risk/or *risk factors/

reviewers who reviewed the papers for inclusion and exclusion.

Results

Search addressing the acquisition and colonization of cariogenic bacteria and ECC (Search 1)

A total of 68 papers were identified in this search as shown (Fig. 2); two duplicates were excluded leaving 66 papers for review by title and abstract. Both reviewers agreed on all included and excluded papers. The first sieving excluded 62 papers. The main reasons for exclusion were because the papers did not report on children aged 12 months or younger (n = 28); did not report on studies (e.g. the paper was a review, report or guideline etc. n = 12); or were the outside scope of this review (n = 11).

Four papers then remained for quality appraisal and data extraction (Table 2). The study by Wan *et al.*[21] was rated as high overall and that of Teanpaisan *et al.*[22] rated medium. Studies by Lindquist *et al.*[23] and Mohan *et al.*[24] were both rated as low. The studies all differed in design and methods used to collect, assess and report data; these influenced the ability to assess the consistency of findings across the studies and to make strong conclusions. Three studies were longitudinal[21–23] and were rated as high[21], medium[22] and low[23], respectively. The fourth study [24] used a cross-sectional design to determine a relationship between parent-reported infant feeding practices occurring during the previous week and bacterial colonization and caries experience. Wan *et al.*[21] used a cohort from a larger prospective longitudinal study (Table 2). The cohort was selected because there were no reportable levels of *Streptococcus mutans* (*S. mutans*) at the time of initial tooth eruption, thus enabling a comparison regarding the timing of colonization with remaining children in the larger study who were colonized in the pre-dentate stage. Participants were examined, and samples of biofilm (plaque) were collected at 3-month intervals until children reached 2 years of age. Despite the study by Lindquist *et al.*[23] being 7 years in

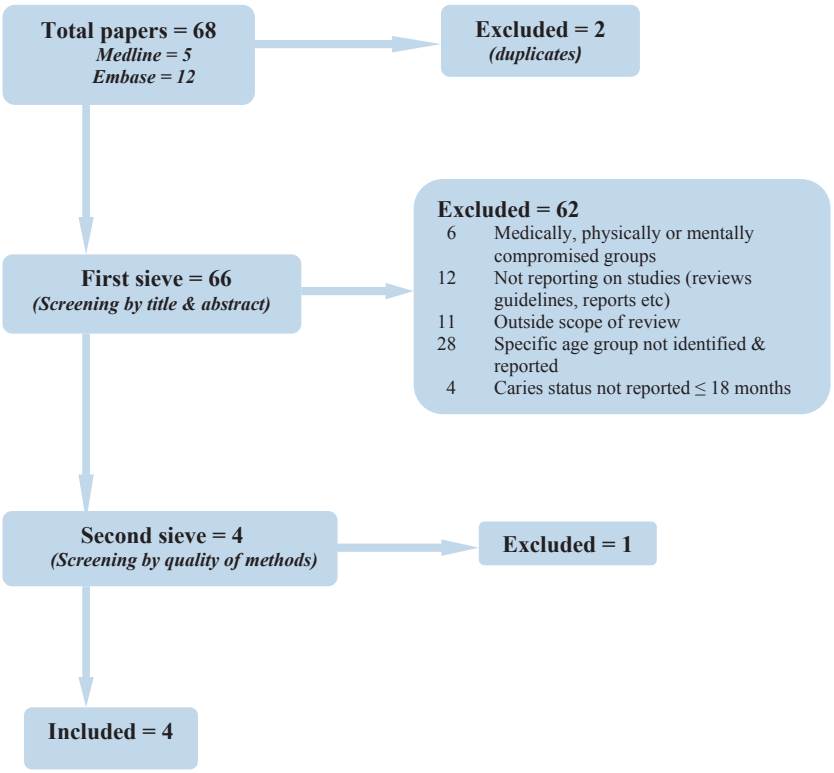

Fig. 2. Flow chart illustrating the outcome of each stage of Search 1 addressing the association between the acquisition of oral cariogenic bacteria and caries outcomes in infants.

Table 2. Study overview and quality appraisal summary and results of Search 1 addressing the association between the acquisition of oral cariogenic bacteria and caries outcomes in infants.

	Quality appraisal						Study overview			
Author and year	Overall rating	Materials and methods	Results	Applicability	Country of study	Population selection	Study design	Study focus	Sample size	Age range infant data
Wan *et al.* 2003[21]	High	High	High	High	Australia	C H RB SEBG with no Sm pre-dentate	Longitudinal cohort (0–24 months)	Infection rate, ages Sm colonization and contributory factors	111 C	6–24 months
Teanpaisan *et al.* 2007 [22]	Medium	Medium	Medium	Low	Thailand	C MIHC H LIF MR	Longitudinal (0 –24 months)*	Relationship bacteria to caries development	*n* = 198 C	9–24 months
Lindquist *et al.* 2004 [23]	Low	Medium	Low	Low	Sweden	PW H with both Sm and Ss	Longitudinal (7 years)	M-C Bacterial transmission and subsequent caries	*n* = 12 M; *n* = 15 C	6 months –7 years
Mohan *et al.* 1998 [24]	Low	Low	Low	Low	US	M C LIF MIHC	Cross-sectional	Risk factors and MS acquisition	*n* = 118 MC	6–24 months

C, child; H, hospital attendees; LIF, low income/socio-demographic families; M, mothers; MR, mainly rural families; MS, Mutans Streptococci; MIHC, mother/infant health care centre attendees; PW, pregnant women; RB, recruited at birth; SEBG, socio-economically balanced population group; Sm, *Streptococcus mutans*; Ss, *Streptococcus sobrinus*; UTR, Unable to report (insufficient detail).

*Part of prospective study aiming to follow children birth – 24 years of age.

duration, it had few participants ($n = 12$ mothers, $n = 15$ children), which restricted reviewers' ability to draw conclusions about the sample population and, in addition, lacked a description of both the sample population group and the population frame. The study by Teanpaisan *et al.*[22] was a large prospective longitudinal study undertaken in Thailand where there is a high prevalence of ECC among the population. This study reported data from 9 months of age and sought to identify a relationship between bacteria and caries development.

All studies varied in their population samples (Table 2). The study by Mohan *et al.*[24] in the United States was undertaken in a low socio-economic group of mothers and their infants. The study by Teanpaisan *et al.*[22] was conducted in Thailand. The study by Lindquist *et al.*[23] was undertaken in Sweden with a small sample of 12 mothers and their 15 children; the sample and sampling frame were not described. Of all the studies, the study by Wan *et al.*[21] rated highest in applicability. It reported a cohort sample representative of the general Australian population.

Different methods were used to collect and measure bacterial samples in each study (Table 3). For example, the sample type and location ranged from Mutans Streptococci (MS) and/or *S. mutans* in saliva, plaque, tongue scrapings or a combination of sites; in addition, techniques used for plating and processing samples varied. This would have resulted in different numbers of colony-forming units (CFU) following cultivation. Methods used to identify and enumerate CFU varied from direct visual techniques to microscopy or colony counters. Only the study by Wan *et al.*[21] used control plates with known bacterial concentrations during the incubation process. The variety of sample collection and analytic methods used for bacterial measures was also apparent in the diagnosis and reporting of dental caries (Table 4). Studies varied from reporting early lesions, to recording frank cavitations only. In one study[24], participant groupings varied, precluding the ability to follow groups of children across time points or assessments.

The results of the above four studies are summarized (Table 5). Three studies[21, 22, 24] detected cariogenic bacteria in infants before their first birthday; Teanpaisan *et al.*[22] reported MS in 1.78% of the pre-dentate infants ($n = 169$) as young as 3 months and caries in 9-month-old infants. The longitudinal study by Wan *et al.*[21] detected *S. mutans* in 5% of 312 children <1 month of age and 18% at 6 months of age. Further, the study by Mohan *et al.*[24] reported 4/22 children were colonized with MS by 6–9 months of age. The study by Lindquist *et al.*[23] did not detect *S. mutans* until the infants were aged between

Table 3. Bacterial sampling techniques and analyses used in four studies.

Techniques	Mohan *et al.* [24]	Lindquist *et al.* [23]	Wan *et al.* [21]	Teanpaisan *et al.* [22]
Bacteria site	Tongue (moistened)	Plaque (biofilm), saliva and tongue (scraping)	Plaque (biofilm), Tongue	Saliva
Collection Tool	Wooden tongue blade	Edentate: cotton Swab dentate: toothpick	Cotton tips	Wooden tongue depressor
Collection site	Dorsum of tongue	Ridges and tongue scrapings taken with toothpick from dried tongue (as separate samples)	Tongue and tooth surfaces	Into oral cavity
Processing	Pressed onto agar plates of MS selective media and cultivated	Pre-dentate: swabs streaked directly onto MS selective agar plates Dentate: Plaque and tongue scrapings into separate vials of RTF transport medium; serially diluted and cultivated on MS selective media and cultivated	Phosphate buffered saline, diluted and plated on Sm selective media and cultivated. Control plates with known cultures also used	Immediately pressed onto petri dishes and cultivated
Assessment	Visual count by morphology	Morphology	Colony counter	Microscope by morphology

Table 4. Methods used to measure caries experience in four studies.

Methods	Mohan *et al.* [24]	Lindquist *et al.* [23]	Wan *et al.* [21]	Teanpaisan *et al.* [22]
Age assessed	*n* = 22, 6–9 months *n* = 31, 10–13 months *n* = 22, 14–17 months *n* = 34, 18–21 months *n* = 9, 22–24 months	Visually at each sampling session ages: 6 monthly from 6 months of age until 3 years then annually till 7 years	3 monthly	9, 12, 18 and 24 months
Measures used to diagnose caries	Modified Radike criteria: Fissures: probe resists removal or loss of translucency; Smooth surfaces: enamel penetrated or scraped away by probe)	Clinical records and radiographs: After study period: Initial and frank lesions also filled surfaces. Findings compared and joint diagnostic decision made.	WHO criteria: (frank lesions only recorded)*	WHO criteria: d_1 (enamel) d_2 (dentine) d_3 (pulpal involvement)
Examiner	One dentist	Two dentists (records reviewed post hoc): joint diagnostic agreement where necessary	One examiner: intra examiner consistency established	Five dentists: Kappa scores for inter and intra examiner reliability

*From personal contact with researchers.

1.5 and 5 years; 5/15 children had no detectable levels of *S. mutans* during the 7-year study period.

Caries was not detected in infants aged 18 months or less in three studies[21,23,24] (Table 5). The study by Wan *et al.*[21] used cavitated lesions as the criterion for the presence of caries (W. K. Seow, Personal communication), and by the time the children were 24 months of age, of the 111 infants who were colonized with *S. mutans*, caries was found in 8 (9%). The study by Mohan *et al.*[24] used modified Radike criteria and diagnosed caries at ages 20 and 21 months ($n = 3$). Diagnosis of caries in the study by Lindquist *et al.*[23] relied upon interpretation using dental records and radiographs of initial, frank and restored lesions. Although dental assessments were undertaken during the study period, assessment of the dental records did not take place until the end of the study when the authors reported caries in 7/15 children.

In summary, three of the four studies (Table 5) reported the presence of cariogenic oral bacteria in the pre-dentate and very early dentate stages of an infant's life[21,22,24], and one study reported caries occurring soon after tooth eruption in a few children[22]. All four studies reported finding caries in children during the study period, and in every child with diagnosed caries, MS or *S. mutans* was present; however, not all children harbouring these bacteria developed caries during the study period.

The four studies demonstrated a relationship between the acquisition and levels of cariogenic bacteria in an infant and several mediating factors (Table 5). The most significant factors reported by Wan *et al.*[21] that potentially increased a 9-month-old child's exposure to higher levels of bacterial transfer were habitual kissing on the lips or having their food pretested; sharing eating utensils or being exposed to dietary sugars four or more times per day. At 12 months of age, there was a shift to child-related risk factors predominating: being formula-fed on demand; snacking four or more times per day or sharing of food with others; spending more than ten hours per week in a child care facility; and an infant not having their teeth brushed at least twice a day[21]. Maternal influences increasing levels of *S. mutans* in an infant were predominantly family income and mother's education for infants at both 9 months and 12 months[21]. In addition, maternal oral health in relation to her *S. mutans* levels and periodontal pocketing depths was considered important, as was maternal snacking of four or more times per day[21].

The study by Mohan *et al.*[24] found that in children aged 6–24 months (Table 5), colonization increased with the number of teeth present and sweetened beverages in bottles. This study reported on bottle usage in terms of children who consumed either sweetened beverages or plain milk in their bottles or did

Table 5. Results of four studies addressing oral bacteria in relation to ECC.

Author, year and study design	Results	Risk factors	Level of risk
Mohan *et al.*[24] Cross-sectional (n = 122 infants)	MS: 4 of 22 colonized 6–9 mos Caries: First detected 20 mos	Age: MS colonization more likely increasing infant age	OR = 4.0, CI 95% = 1.2–12.6
		Bottle usage/content: consumption of sweetened beverages *versus* milk or no bottle usage (6–24 mos)	
Wan *et al.*[21] Longitudinal (n = 111 infants)	*S. mutans*: 6 mos = 1% 9 mos = 12% 12 mos = 37% 15 mos = 54% 18 mos = 68%	Consuming pre-tasted foods at 9 mos	OR = 6.4, 95% CI = 2.9–14.5
	Caries: First detected 24 mos = 9 with caries	Sharing eating utensils > 3/day at 9 mos	OR = 4.6, 95% CI = 2.3–9.5
		Total sugar exposures at 9 mos > 3/day	OR = 4.6, 95% CI = 3.0–13.4
		Habitually kissed on lips at 9 mos	OR = 6.4, 95% CI = 3.0–13.4
		Brushing habits $\leq$ 1/day at 12 mos (parental-assisted positive association)	OR = 2.1, 95% CI = 1.2–2.6
		Snacking > 3/day at 12 mos	OR = 5.6, 95% CI = 2.3–9.5
		Formula feeding on demand at 12 mos	OR = 8.9, 95% CI = 1.9–41.6
		Low total income families	9 mos = $P < 0.01$ 12 mos = $P < 0.03$
		Mother's primary education higher risk at 9, 12, 15 mos	
		Maternal Sm levels 10^5 CFU/mL	OR = 2.1–8.5, 95% CI = 1.2–27.6
		Mothers' oral health status: periodontal pocketing (CPI > 2)	OR = 1.6-5.3, 95% CI = 0.3-30.1
		Maternal plaque covering > 50% dentition	OR = 3.8-18.8, 95% CI = 0.9–84.0
Teanpaisan *et al.*[22] Longitudinal (n = 198 infants)	MS: 3 mos = 1.78% 9 mos = 17.75% 12 mos = 28.63% 18 mos = 47.34% Caries: 9 mos = 4.2% 12 mos = 29.9% 18 mos = 83.1%	Bacterial level more important than age at colonization ECC if MS $\geq$ 50 CFU (CFU/1.5 cm^2)	OR = 13.01, 95% CI = 2.89–58.52
Lindquist *et al.*[23] Longitudinal (n = 15 infants)	MS: <1.5 yrs = 0 <7 yrs = 10/15 Caries: 18 mos = 0 <7 yrs = 7/15	Homology of genotypes between mothers-infants found	

MS, Mutans Streptococci; mos, months of age; OR, odds ratio; CI, confidence interval; *S. mutans*, *Streptococcus mutans*; CFU, colony-forming units; CPI, community periodontal index; ECC, early childhood caries; yrs, years of age.

not use a bottle at all. The paper did not report on frequency of consumption, whether night-time bottle-feeding was occurring, or whether the children were breast fed or received a sugar intake from sources other than from the bottle. The study by Teanpaisan *et al.*[22] concluded that the bacterial level was more important than the age of acquisition in subsequent caries experience, and in addition, children who harboured MS before the sample population mean age of 16.7 ± 6.7 months had a higher number of decayed teeth at all ages. However, this was not statistically significant until 24 months of

age. Further, children 12 months of age who had detectable levels of MS had a 13-fold risk of developing caries. This study did not investigate other determinants that may modify bacterial levels and disease process, such as frequency of feeding and bottle contents.

Search addressing all proposed determinants of ECC (Search 2)

This search focused on a broad range of determinants in an effort to identify and describe the risk and/or protective factors that modify cariogenic bacterial acquisition. The stages of the review process are shown (Fig. 3). A total of 313 papers were reviewed by title and abstract; of these, 13 duplicates and a further 285 papers were excluded. The main reasons for exclusion were the papers did not report on children aged 12 months or younger (n = 151), were not studies (e.g. paper was a review, report, guideline etc. n = 65) or were outside the scope of the study aim (n = 33). Full texts were obtained for the remaining 17 papers; of these, two papers were excluded in the second sieve as they did not meet the CASP screening criteria[17]. In addition, two foreign language papers, which, despite seeking the opinions of at least two translators for each, were unable to be translated sufficiently to subject them to a quality appraisal and were therefore also excluded at this point; 13 papers were then quality-appraised.

The quality appraisals of the 13 papers are shown (Table 6). The studies were rated on three criteria (reliability, internal validity, external validity) as high, medium or low, and an overall weighting was then developed. Three papers[21,29,32] were rated overall as high; of these, one was a RCT[29], and two were longitudinal cohort studies[21,32]. Four papers[22,26,30,31] were rated medium, and of these, two were RCTs[26,30], and two were longitudinal cohort studies[22,31]. The remaining six papers[24,25,27,28,33,34] were rated low. Of these, three were RCTs[25,27,28], and three were cross-sectional[24,33,34].

Of the longitudinal cohort studies, the study by Wan *et al.*[21] rated high in all three categories (Table 6). This study of an Australian population was a cohort (n = 111) subset of a larger study (n = 312) and provided

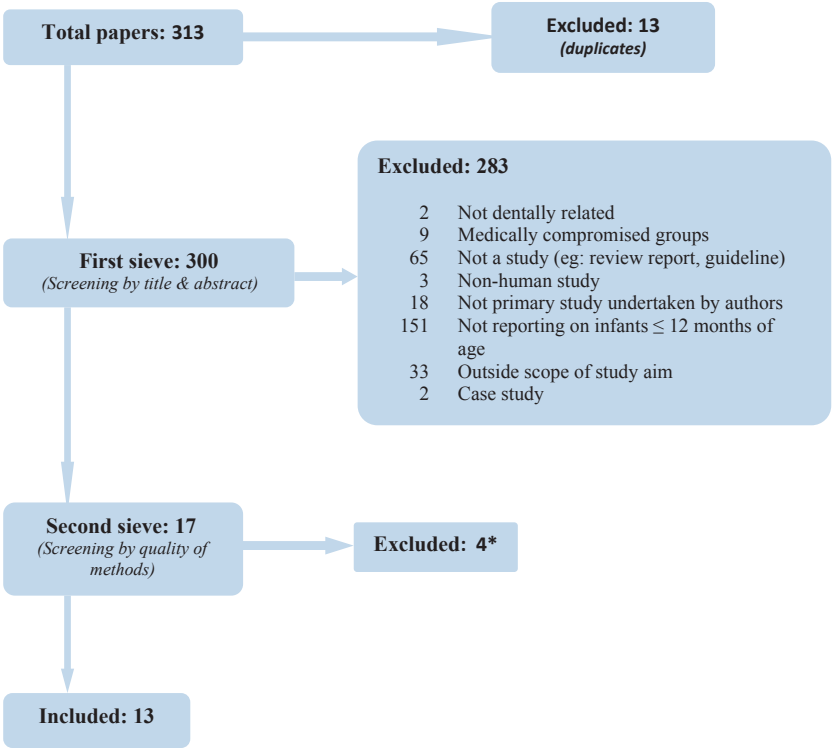

Fig. 3. Flow chart illustrating the outcome of each stage of Search 2 addressing all proposed determinants of early childhood caries.

Table 6. Study overview and quality appraisal summary and results of Search two addressing all proposed determinants of early childhood caries.

	Quality appraisal						Study overview			
Author	**Overall rating**	**Materials and methods**	**Results**	**Applicability**	**Country of study**	**Population selection**	**Study design**	**Study focus**	**Sample size**	**Age range infant data**
Wan *et al.*[21]	High	High	High	High	Australia	HB pre- and full term RP	Longitudinal (>24 months)	Bacterial colonization	*n* = 111 C	B-24 months (3 monthly)
Nakai *et al.*[29]	High	High	High	Medium	Japan	PW MICH >10^5 CFU/mL NVS	RCT (NR approximately 30 months)	Bacterial transmission	*n* = 107 M	0–9 months
Thitasomakul *et al.*[32]	High	High	High	Low	Thailand	NVS	Longitudinal	SES and risk factors	*n* = 495 C	9, 12, 18 months
Dasanayake *et al.*[26]	Medium	Medium	Medium	Medium	USA	PW MIHC; 2.5 × 10^4 CFU/mL	RCT (4 years)	Bacterial transmission	*n* = 75 MC	1, 2, 3 year (birthdays)
Teanpaisan *et al.*[22]	Medium	Medium	Medium	Low	Thailand	RPLIF MIHC/H	Longitudinal (>24 months)	Infant feeding practices	*n* = 1076 C*	6–18 months
Habibian *et al.*[31]	Medium	Medium	Low	Medium	England	AP HB	Longitudinal (NR approximately 30 months)	Infant feeding practices	*n* = 163 C	6, 12, 18 months
Feldens *et al.*[30]	Medium	High	Low	Low	Brazil	M HB LIF	RCT (field) (1 year)	Role of health education	*n* = 500 MC	0–4 years
Mohan *et al.*[24]	Low	Medium	Low	Low	USA	LIF WIC CS	Cross-sectional	Infant feeding practices	*n* = 122 C	6–24 months
Fontana *et al.*[28]	Low	Medium	Low	Low	USA	MICH/Ads 10^5 CFU/mL	RCT (10 months)	Bacterial transmission	*n* = 97 M	0–14 months
Brambilla *et al.*[25]	Low	Low	Low	Low	Italy	PW H with 10^5 CFU/mL; NFD	RCT (30 months)	Bacterial transmission	*n* = 60 MC	6–24 months
Singh *et al.*[33]	Low	Low	Low	Low	Fiji	MIHC NFD	Cross-sectional	Infant feeding practices	*n* = 102 C	6–12,13–24,25–36 months
Franco *et al.*[27]	Low	Low	Low	Low	USA	MICH LIF NFD	RCT (30 months)	Role of counselling	*n* = 132 MC	0–24 months
Qin *et al.*[34]	Low	Low	Low	Low	China	H K C NVS	Cross-sectional	SES and risk factors	*n* = 514 C	<4 years

A, adults; Ads, advertising; AP, affluent population; B, birth; C, child; CFU/mL, colony-forming units per millilitre cariogenic bacteria; H, hospital attendees; HB, hospital birth; K, kindergarten/preschool attendees; LIF, low income/socio-demographic families; M, mothers; MC, mother/parent child pairs; MIHC, mother/infant health care centre attendees; NFD, no further detail; NR, Not reported; NVS, not very specific; PW, pregnant women; RCT, Randomized control trial; RP, representative of broader population group; VCD, vaccination campaign day; WIC, attendees Women Infant Children clinics.
*Not all of these examined dentally and bacterial samples taken.

detailed information on the population, methods used and data validation. The study by Thitasomakul *et al.*[32] also rated as high overall. As it was conducted mainly in rural Thailand, its applicability to Victorian/Australian populations was limited. Studies rated medium were those of Teanpaisan *et al.*[22] and Habibian *et al.*[31] These studies lacked some features of the higher rated studies including recruitment methods, participant attrition, reporting and methods.

Three papers[21, 22, 24] were common to both searches and have been addressed above. The remaining ten papers will now be considered (Table 6). Four studies (all RCTs) investigated bacterial transmission from mother to infant. Of these, two studies, which intervened to reduce maternal bacterial load during pregnancy[25, 29], demonstrated that if the level of maternal MS was decreased prior to tooth emergence, bacterial acquisition in the infants was either delayed[29] or resulted in fewer children infected with MS, than in the control group[25]. In fact, Nakai *et al.*[29] found transmission could be delayed significantly by as much as 8.8 months compared with the control group, and Brambilla *et al.*[25] reported colonization was delayed by 4 months. A third study, by Dasanayake *et al.*[26], which intervened to reduce maternal bacterial load at infant age 6 months, was unable to demonstrate such a finding despite demonstrating significant reduction in maternal bacterial load. The fourth study, by Fontana *et al.*[28], was unable to reduce maternal levels of bacteria, and hence, there was no effect on infant bacterial acquisition.

Of the above four studies, infant caries experience was reported only by Dasanayake *et al.*[26] (Table 6). This study reported the presence of caries in children at 48 months of age; however, owing to the time lapse between study publication and the present review, on personal contact, the author was unable to recall at what ages caries was first detected in the children (A. Dasanayake, Personal communication). Both Fontana *et al.*[28] and Nakai *et al* [29] undertook dental assessments. On personal contact, Fontana reported their study was limited to saliva and biofilm assessments and was not designed to measure short term caries experience (M. Fontana, Personal communication). The study undertaken by Brambilla *et al.*[25] did not include a dental assessment of the infants. In total, eight of the ten studies conducted a dental assessment, with five studies reporting caries experience in children by 18 months of age[27,30,32–34] (M. Qin, Personal communication) [34]. One study[31] found all children studied were caries-free at both 12 and 18 months of age; two further studies either did not report the assessment outcome undertaken at 12 months of age[28] or did not report the assessment age[26].

The main factors identified (Table 6) appearing to increase an infant's risk of early bacterial acquisition, and higher levels of colonization were maternal factors, such as low level of education[32,34]; poor oral health knowledge[34]; and maternal calcium supplementation and milk intake during pregnancy and the first year post-natally[32]. Maternal oral health and bacterial levels were assessed also. The study by Thitasomakul *et al.*[32] found an association between maternal caries levels and bacterial levels in infants. Infant feeding practices were identified as being associated with ECC in some studies and this included night feeding[30,34], and habits of testing and sharing of food and eating utensils[28,34]. The frequency and types of food and liquids introduced, and the age of the infant at the time of their introduction[31–34], were reported as important modifiers in the disease process. Oral hygiene practices, in particular brushing an infant's teeth[32], were another modifying factor identified[21, 31].

Only one study, by Habibian *et al.*[31], undertaken in a high socio-economic group in Southern England, reported toothbrushing had commenced in 90% of infants by 12 months of age, and fluoridated paste was used in 85% of infants (Table 6). Despite the presence of plaque accumulation and risk behaviours occurring such as nonmilk extrinsic sugar (NMES) consumption (which comprised 46%, 60% and 67% of 6-, 12- and 18-month-old infants' mean daily frequency of eating and drinking episodes respectively), all children remained caries-free. This study also reported that infant feeding practices may be established by 6 months of age, with

mean daily frequencies of food and drink consumption not differing significantly at 6, 12 and 18 months of age.

Discussion

The findings of this review have confirmed and identified a range of factors occurring during the first year of an infant's life that impact on early caries experience. Typically infants are totally reliant upon their mothers during this time; hence, there were no studies identified in the search that explored paternal or sibling influences that may affect this age group. As a result, this review is limited to commenting on the outcomes of those studies that explored maternal influences and circumstances as they impact on the infant, rather than those of the broader family.

A synopsis of all the papers addressing risk and protective factors for bacterial levels and ECC is shown (Table 7). In all cases where infant caries experience was reported, bacterial acquisition and colonization was present, and the likelihood of colonization increased with the age of the infant. However, not all children harbouring these bacteria developed caries during the study periods. This feature is consistent with the multifactorial nature of the disease: although cariogenic bacteria are a significant factor in caries development, other factors such as feeding habits and the frequency and/or type of food and liquids consumed by the infant modify disease progression (Table 7).

In addition, factors were identified with the potential to provide a protective influence on the infant oral environment in relation to subsequent early caries experience. Along with reducing maternal bacterial load before the time of infant tooth emergence, which would delay bacterial acquisition, regular infant toothbrushing and the use of fluoridated toothpastes were reported (Table 7). One study also identified that infant feeding habits may be established as early as 6 months of age, and this too may have important implications for risk of caries experience in terms of influencing frequency of dietary intake as well as developing infant preference for particular types of foods.

Bacterial acquisition, colonization and subsequent ECC

The studies demonstrated that infants can be colonized with cariogenic bacteria during the pre-dentate stage, with some children colonized as early as 3 months of age. Further, the studies showed an association between bacterial acquisition and maternal bacterial levels; hence, a vertical pathway for transmission of these bacteria occurs. Notably, in studies where bacterial transmission was investigated, the timing of reducing maternal bacterial levels to achieve a delayed or reduced level of infant bacterial colonization was important.

Influences of the mother and her circumstances

In studies where oral health education was provided to mothers aiming to change attitudes and practices and so reduce risks of poor infant oral health outcomes, it was found that some, but not all, risk behaviours were altered; despite knowledge of some of the risk factors, risk behaviours often continued. It is important then, if oral health promotion programs are to be implemented, to first identify why some health behaviours were adopted and others were not.

In fact, it may well be that there are important determinants affecting a mother's ability to recognize and respond to risk behaviours. Factors not identified in the review that may add further complexity include those identified in the broader social determinants of health, such as maternal cultural beliefs and influences[13, 35, 36], her level of autonomy in decision-making within the family, coping skills and supportive networks[37–40], as well as her past dental experiences, access to personal dental care and related oral health information[41].

Infant feeding, behaviours and practices

From the studies, it was apparent that not only were particular habits or behaviours thought to affect an infant's susceptibility to bacterial acquisition, levels and ECC experience, but the timing and frequency of the

Table 7. Synopsis of papers addressing risk and protective factors for bacterial levels and early childhood caries.

Influence	Factor	Positive associations	Author
Maternal influences primarily associated with bacterial acquisition	Maternal *Streptococcus mutans* levels	Bacteria 6 months $P = 0.002$	Brambilla *et al.*[25]
		Bacteria by 8.8 months	Nakai *et al.*[29]
	Mother's caries $\geq$ 4 teeth	Bacteria OR 2.1	Wan *et al.*[21]
		Caries $P = 0.05$	Thitasomakul *et al.*[32]
	Pre-tasting foods	Bacteria 9 months OR = 6.4	Wan *et al.*[21]
		Caries $P < 0.001$	Qin *et al.*[34]
	Sharing eating utensils	Bacteria 9 months OR = 4.6	Wan *et al.*[21]
		Bacteria $P = 0.009$	Fontana *et al.*[28]
	Low income	Caries 9 and 18 months	Thitasomakul *et al.*[32]
	Mother's education (primary level)	Bacteria 9 and 12 months OR = 2.1	Wan *et al.*[21]
		Caries $P = 0.023$	Qin *et al.*[34]
		Caries 12 and 18 months $P < 0.05$	Thitasomakul *et al.*[32]
	Mother's education	Caries 22% RR 0.78; 95% CI 0.50–0.92	Feldens *et al.*[30]
Behaviours and habits primarily associated with bacterial colonization	Sweetened beverages	Caries OR = 4	Mohan *et al.*[24]
		Caries < 5 months (1.2 ± 2.8 ds)	Thitasomakul *et al.*[32]
		Caries $P < 0.001$	Qin *et al.*[34]
	Total sugar exposures	Caries $P < 0.0001$	Habibian *et al.*[31]
		Bacteria 9 months OR = 4.6	Wan *et al.*[21]
		Caries $P < 0.001$	Qin *et al.*[34]
	Brushing habits	Caries	Habibian *et al.*[31]
		Bacteria 12 months OR = 5.6	Wan *et al.*[21]
		Caries 9 and 18 months	Thitasomakul *et al.*[32]
	Snacking	Caries 6 months	Habibian *et al.*[31]
		Bacteria 12 months OR = 5.6	Wan *et al.*[21]
		Caries	Singh *et al.*[33]
		Caries 9 and 18 months	Thitasomakul *et al.*[32]
		Caries	Feldens *et al.*[30]
	Night feeding	Caries $P < 0.001$	Qin *et al.*[34]
	Colonization more likely with increasing age	Bacteria	Mohan *et al.*[24]
		Caries	Teanpaisan *et al.*[22]
		Bacteria	Fontana *et al.*[28]

DS, Decayed Surfaces.

habit must also be primary considerations. Some habits were shown to be more likely to pose a greater risk for poor oral health outcomes depending upon the developmental stage of the infant, whereas other habits seemed to pose an increased risk for poor oral health outcomes regardless of the child's age.

Consumption of NMES is an example from several studies examining the link between feeding practices and either bacterial levels or caries experience. Testing/tasting of foods before feeding to an infant and sharing of eating utensils during a meal were identified as risk factors for higher bacterial levels particularly in younger children. The introduction of commercial cereals at 3 months and vegetables into the diet by 6 months of age was suggested to be protective in nature. One study found that habits developed in relation to an infant's diet by 6 months of age affected dietary

behaviours at both 12 and 18 months, suggesting that establishing healthy eating patterns early could well contribute to reducing risk of caries experience. This supports the findings of Gussy *et al.*[42] who found that the frequency of dietary intake in preschool-aged children was more important than the amount, and Mattos-Graner *et al.*[43] who reported salty foods introduced to infants younger than 7 months of age showed a lower prevalence of caries compared with infants who were not introduced to these foods by 7 months.

As not all children exposed to these risk behaviours necessarily develop elevated bacterial levels and/or caries, we presume that factors such as the level of the bacterial acquisition and subsequent colonization must rely upon the amount of bacteria transferred during the behaviour, and how often this occurs, to determine the level of risk. In addition, there was some evidence that regular toothbrushing and use of fluoridated toothpaste are protective, despite risk behaviours occurring. It is uncertain from the two studies reporting this finding whether it is regular toothbrushing or the use of fluoridated toothpaste *per se* that reduces the risk of caries, or whether other factors more closely linked to the higher socio-demographic populations contribute.

Recommendations

This systematic review of the literature identifying risk factors during the first year of life makes the following recommendations:

- Mothers with high levels of cariogenic bacteria must be identified during the prenatal period, and their bacterial levels reduced prior to infant tooth eruption in an effort to delay and/or reduce the levels in their infants. Ongoing social networks must be developed to support new mothers in identifying and minimizing risk behaviours affecting their children.
- The interrelationships between cariogenic bacteria, mediating factors occurring during the first year of life and subsequent caries outcomes require further clarification to identify and quantify key predictors. This would enable the provision of effective support mechanisms for health educators, practitioners and parents, to reduce infant caries experience.
- Consistency among researchers to measure oral health outcomes (principally caries) in infants and toddlers is needed to enable a more accurate knowledge base of caries onset, incidence and prevalence.

Conclusion

Caries prevention commencing before and continuing into the early dentate period is necessary. This review has confirmed current thought that ECC has important causative factors in the first year of life. Cariogenic bacteria were shown to be a significant risk factor for ECC. Maternal factors were shown to influence bacterial acquisition but, although influences modifying bacterial colonization were identified, a relationship with subsequent caries development was not clarified. This was due primarily to the complex nature of the disease and infant age; however, factors such as study design and/or techniques used to measure bacteria and subsequent caries experience limited study outcomes. Further exploration is required to better understand the complex nature of ECC, both the factors affecting its initiation as well as its progression, if infants and toddlers are to be spared the effects of this common and sometimes debilitating disease.

Why this paper is important to paediatric dentists

- Bacteria present in the predentate stage play a significant role in early caries experience
- Pregnancy and the neonatal period are the important times to identify 'at risk' children.
- Early maternal intervention can reduce the likelihood of ECC.

Acknowledgements

The authors acknowledge with gratitude the scientific expertise contributed by Associate Professor Stuart Dashper and Emeritus Professor Louise Brearley-Messer; the research librarian support of Ms Poh Chua and Ms

Cathy Gatt; the research assistance of Dr Shalika Hegde, and the support of Professor Hanny Calache. This study was supported by National Health and Medical Research council Research Scholarship No: 56718 and The Jack Brockoff Foundation.

Conflict of interest

The authors have declared no conflict of interest.

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