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RESEARCH ARTICLE

A REVIEW ON MICROBIAL ECOLOGY OF MUTANS STREPTOCOCCI IN HUMAN MOUTH

Dr. Apoorva Mehrotra[1], Dr. Mohammad Iqbal[2], Dr. Waleed Khalil Al Dahlawi[3] and Dr. WalaSaad Al Raddadi[4]

1. Assistant Professor, Department of Conservative Dentistry & Endodontics, Career Postgraduate Institute of Dental Sciences and Hospital, Lucknow, India.
2. Associate Professor and Head, Department of Restorative Dental Sciences, Faculty of Dentistry- Al Baha University, Kingdom of Saudi Arabia.
3. Dentist, Al Hijra Dental Complex, Kingdom of Saudi Arabia.
4. Dentist, Sydalshuhada Primary Health Care Centre, Kingdom of Saudi Arabia.

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Abstract

The streptococci constitute a large and complex group of bacteria that have widely varying characteristics and that under certain conditions are capable of independent pathogenicity. In human mouth, the viridians streptococci are one of the main groups of bacteria and they are the most commonly occurring microorganisms in oral infections including dental caries.

Introduction:-

Streptococcus mutans were named by Clarke in 1924 due to its variable morphology after been isolated due to the fact that they were predominant in many human carious lesions. Clarke also found out that in artificially induced caries these Streptococcus mutans stuck closely to tooth surfaces. Characteristics and features of this cluster of streptococci have been termed as non-motile, catalase negative, gram positive cocci in medium or short chains[1,2]. It shows a number of important properties:

1. Insoluble polysaccharides are synthesized from sucrose.
2. Homo-fermentative lactic acid former.
3. Colonizes/ settles on tooth surfaces.
4. More aciduric than other streptococci.[1]

Taking into consideration the physiological, ecological and morphological characteristics the Streptococcus mutans forms a homogenous group. Based on the physiological and biochemical tests, a classification of strains were named by Facklam (1974), Shklair and Keene (1974, 1976). Studies and research done by Coy Kendall (1971, 1974) and Coykendall et al (1976) states the analysis of guanosine and cytosine content and hybridization studies on the homologies of the DNA isolated from strains of Streptococcus mutans revealed significant differences.[13]Although these cariogenic organisms are phenotypically similar, genetically they are heterogenous and hence divided into 5 genotypes or genospecies. Streptococcus mutans (36 to 38 mol% guanosine and cytosine), Streptococcus rattus (41 to 43 mol % guanosine and cytosine), Streptococcus sobrinus (44 to 45 mol % guanosine and cytosine) and Streptococcus ferus (44 mol % guanosine and cytosine). Streptococcus mutans and Streptococcus sobrinus are the organisms most frequently assosciated with dental caries .[14] For caries initiation Streptococcus mutans has been recognized as the primary cariogenic bacterium while for enhancing and aggregating caries initiation, progression and development. Streptococcus sobrinus is responsible. [3-8]

Corresponding Author:- Dr. Mohammad Iqbal
Address:- Associate Professor and Head, Department of Restorative Dental Sciences, Faculty of Dentistry, Al Baha University, KSA.

Discussion:-

Serological classification of mutans group:

Based upon the serological specificities of Streptococcus mutansBratthall classified them into 5 types known as a, b, c, d, and e. Serotypes f and g where additionally established by Perch et al. Difference in the geographical distribution of the different serotypes or the biotypes of Streptococcus mutans has been revealed by many researchers. It has been stated that serotype d may be more cariogenic than others, since this organism has been isolated more frequently from plaque of caries active persons than from those who are caries free or have little caries. But, Masuda and Tsutsumi stated in their study that serotype c strains were predominant among persons who developed caries.[9-11] (Fig 1)

Initial acquisition of mutans streptococci:

Because Mutans streptococci (MS) proliferate and multiply at a rate which exceeds the salivary fluid flow washout rate they can endure freely in the saliva or in a similar environment by establishing a colonies on mucosal surfaces which are adherent. The possibility of MS to colonize in a normal infant's mouth is proved to be unconvincing by the review studies done by Gibbon and Van Houte where they proved that MS had a very weak capacity adhering to the epithelial surfaces.[13]

"Window of infectivity" proposed by Caufield et al. is supported by some clinical researches which demonstrated the initial colonization of MS varied between 7–36 months which is a time period overlapping with the upliftment of the primary teeth.. If the establishment of MS colonies occurs with the mergence of teeth, then many simultaneous windows of infectivity may co-exist, such as duration corresponding with the eruption of incisors and molar i.e. (primary dentition) and first molars (permanent), respectively.[14]

Whereas, Studies by Wan, Law and Seow revealed that there was a substantial increase of colonization of MS assosciated with the advancing age of the offspring, without any distinct windows of infectivity.[15]A longitudinal investigation by Carlsson and co-workers described that in 20% infants 12 months to 16 months old having 6 to 10 deciduous teeth MS were identified.[16]Detection of MS in 22% infants having only the primary incisors was reported by Berkowitz and his team.[17] In a subsequent study they were not found to be present in 16 infants without teeth , but were present in 7% infants (mean age=8.9 months) with 1 to 5 primary incisor teeth and in 29% infants (mean age=13.8 months) with 6 to 8 primary incisors.Similarly, Stiles isolated them in 22% of infants (median age=nearly 14 months) with 6 to 8 deciduous incisors and in 3% of infants (median age=almost 9 months) with 2 to 4 deciduous incisors[18.] Isolating MS from predentate kids or kids with only primary incisors could not be achieved by Catalanotto and colleagues[19]. MS were isolated only once the eruptions of the deciduous molars are seen. Karn and co-workers stated that MS were observed even at 10 months of age. Detectable MS levels were seen in 25% children 12 months old and 60% children were colonized in the 15 months old age group[20].

Occurrence of the first deciduous tooth, and also that monozygotic co-twin should display alike sequence of first tooth emergence and colonization, reflecting underlying mutual genetic influences. 20% children harboring 10 teeth and 40% children harboring 12 teeth showed a cumulative probability of detecting MS in a study conducted on 39 children by Japanese researchers in a Hiroshima day nursery. These observations put together showed that until the later stages of deciduous incisor emergence (i.e. 10-12 months old) the MS are not revealed in the mouths of predentate kids and rarely colonize the oral cavity. Consequently the concept of a non-shedding oral surface required by MS for a persistent oral colonization has become a principle of oral microbial ecology. For the treatment of infants suffering from palatal cleft an acrylic plate is used to obturate the cleft and recommended to be worn until 18 months of age. This hard, non-shedding surface plate is expected to enable MS to an early colonization.

According to Loveren et al, at 18 months of age, the oral cleft lip and palate children wearing an acrylic plate from just after birth were colonized earlier with mutans streptococci and lactobacilli than the non-plate oral cleft children.[21] Similar observation was done by Berkowitz who stated that these organisms were not detected in 91 normal pre-dentate infants, but were detected in 2 of 10 newborns with acrylic cleft palate obturators[17].

The colonization of MS in predentate infants has been demonstrated by more recent clinical investigations. The furrows of the tongue seemed to be a vital ecological niche as demonstrated by Tanner and his team. Wan and colleagues displayed more than 30 percent of pre-dentate infants at the age of 3 months were infested with Streptococcus mutans and over 60 per cent revealed manifestation of the bacteria by the age of 6 months. These above findings suggest that there is a possibility of the presence of MS prior to the first tooth eruption[22].

Early acquisition of mutans streptococci and its role in dental caries:
A few studies have reported that early acquisition of MS is a key risk reason for early childhood caries and future caries experience. A longitudinal assessment for MS and dental caries was performed by Alaluusua and Renken in children aged 2 to 4 years. The most caries detected were in children aged 4 who had harbored MS in their plague at 2 years of age. The dmfs (mean decayed, missing, and filled primary tooth surface) was 10.6 in these children when compared to a dmfs of 3.4 at age 4 (P<.005>) in children where colonization occurred in a later stage[23].

A similar report was made by Kohler et al stating that 89% of kids showed mean dfs score of 5.0 in children with dental caries by 4 years of age who had MS colonization by age 2. Whereas a mean dfs score of 0.3 was exhibited by 25% of kids with dental caries by 4 years of age but not infected by MS at age 2[24]. Grinderfjord et al, for a longitudinal study, evaluated 786 children aged 1 year for caries risk factors which included oral hygiene, dietary habits, fluoride exposure as well as MS infection and were re-examined at 3 and a half years of age for the presence of dental caries. The presence of MS at an year of age was the peak effective prognosticator of caries at 3.5 years of age.[25]

All the above mentioned findings as well as those done by Fujiwara et al and Roeters et al clearly illustrate that the substantial risk factor for future progress of dental caries is an early infection of MS[26,27].

Transmission ofmutans streptococci:
Understanding the factors that affect the initial MS colonization is essential to understanding caries development. The colonization of MS in the oral cavity of infants is believed to be through the transfer of these bacteria from the primary care giver to the child. High MS levels, open lesion and inadequate oral hygiene in the care taker as well as low economic status and frequent snacking by the child are the reasons associated with this transmission. Due to the fact that there is no clarity in the exact mode of transmission, there are suggestions for possible contributing factors like intimate contact, food or utensils been shared, or immunological issues[28].

Thus there has been a great interest in the past several decades to determine by what means children develop colonized with MS and whether this colonization can be postponed or reduced to decrease a child's caries risk.

The first attempt to investigate the possibility of person to person transmission of MS was done by Jordan et al in 1972, since then numerous studies have been performed about MS transmission. The use of serologic typing to differentiate clinical MS isolates was developed in 1970s. Most of the early investigations were cross sectional studies using serologic or bacteriocin characteristics to distinguish MS isolates[28]. In late 1980s and early 1990s, molecular genetics were developed that differentiated MS isolates based on genotypic characteristics of individual DNA samples. These techniques identified diverse genotypes of MS strains within the same serotypes or among MS isolate carrying similar bacteriocins. Therefore, the use of bacteriocins or serologic typing to determine the fidelity of MS transmission from primary care givers to their children could be challenged for sensitivity and lack of accuracy[28].

To overcome these difficulties, molecular typing methods have been developed and become important tools for studying MS transmission and colonization. To date, the most commonly used genetic methods include MS chromosomal DNA finger printing with specific endonuclease enzyme digestion and MS chromosomal DNA ribotyping. Recently, a single polymerase chain reaction (PCR) with randomly selected DNA primers has been sufficient to examine similarity of MS clinical isolates among family members[29].

Application of genotyping suggests that mothers are the primary MS transmission source to children and that saliva may be the principal vehicle by which transmission of MS may occur.

Source for vertical transmission:
There is a possibility of the association of the following factors, in part, like magnitude of the innoculum, occurrence of small dose inoculations, and a lowest infective dose in the successful colonization of maternally spread MS cells. Kohler and Brathall (1978) stated that mothers with high levels of Streptococcus mutans can more easily transmit them to their children. On the other hand mothers having low MS in their salivary concentration were likely to raise uninfected children[24].

According to a study report by Berkowitz et al the frequency of infection in infants whose mothers had greater than 10 colony forming units (cfu) of MS/mL of saliva was 58% whereas it was 9 times less (6%) in infants whose mothers had 10 or more cfu of MS/mL of saliva. These reports suggest that the risk of infants been infected early in their life is due to their mothers with condensed salivary pools of MS[17]. Suhashini, Reddy JS and Hamid studied the association between salivary levels of 50 children with rampant caries in age group 3-6 years and their mothers were compared with 50 caries free children of the same age group and their mothers and found a positive correlation with Streptococcus mutans levels in toddlers with rampant caries and their mothers[30].

Johannes et al studied the transmission of Streptococcus mutans in 21 offsprings with (CLAP) cleft lip and or/palate and their mothers based on PCR and suggested that transmission occurred in only one third of population. This indicates that the Spread of Streptococcus mutans from mother to babies with oral cleft is not frequent[31].Kohler et al showed that by decreasing the MS in the saliva of the highly infected mothers it can restrain or delay the setting up of MS in their babies. 11% (3 of 28) babies who had their mothers in the experimental group with their MS reservoirs suppressed by treating active caries lesions and chlorhexidine gel applied topically got infected by the age of 23 months.Whereas at the same age 45% (17 of 38) babies got infected with their mothers in the control group without their MS levels suppressed[24]. Fabiana et al investigated the transmission and genetic identity Streptococcus mutans strains in 17 mother child pairs and confirmed that mother is the main source of infection to the child[32]. (Fig 2)

Source for horizontal transmission:

Despite the fact that mothers are often the major primary caregivers, in many cultures fathers also play an important role in nurturing children. Beyond fathers, other close contacts such as grandparents who are caregivers, siblings and members of a similar age or students in a classroom also could be MS sources. Latest research suggests that horizontal transmission also occurs and vertical transmission is not the only route for the spread of MS in humans. Taking into consideration the socioeconomic variation in the Western culture for the past 20-30 years, this is very crucial. One such example is the daycare center or baby care center or a nanny serving as a new route for acquiring these bacteria[33].

Similar MS genotypes common to father, mother and their child have been reported through many studies. (RedmoEmanuelsson and Wang 1998; van Loveren et al 2000; Hames-Kocabas et al 2008) Horizontal transmission taking place easily in families has been proposed in recent studies.Saarela et al 1993; Kozai et al 1999; Emanuelsson and Thornqvist 2000; RedmoEmanuelsson and Thornqvist 2001; Nie et al 2002; Ersin et al 2004). A study done by Mattos-Graner and team on a group of Barzilian nursery kids aged between 12-30 months strongly advocates the horizontal transmission of MS due to the fact that 29% of the 24 kids with 2 to 5 MS segregates, carried 2 or more matching genotypes. The team had genotyped them by haphazardly primed polymerase chain reaction and limitations fragment length polymorphism analysis[33].

The possibility of horizontal transmission taking place between family members was also reported by Van loveren et al. They reported that when bacteriocin typed isolates of MS were taken from 5 year old children and their parents, the results showed there may be a similarity between the MS of father, mother and child even if the child gets MS after age 5[21]. Another research finding that supports the concept of horizontal transmission taking place between family members is the study done by Emauelson and Wang who genotyped isolates form 18 Chinese families comprising of father, mother and a 3 year old kid with no other sibling. Identical genotypes were found among some of the fathers and their first born which was the main observation of the study. Another key finding was the homology of genotypes between couples[34].

Domejean et al (2010) investigated the likely transmission of mutans streptococci genotypes from child to child in kindergarten in 96 children (ages 5-6 yrs). 6%, i.e. 2 kids who were not siblings from 3 schools respectively shared a matching amplitype of MS exclusive to each duo. 12 kids showed the 19 Sobrinussobrinusamplitypes distinctive to each kid. Matching genotypes of MS present in unrelated kids between 5-6 years of age shows the horizontal transmission of this species[35].

Factors Influencing The Acquisition And Colonization Of Mutans Streptococci In Infants[36]:

Mode of delivery influencing acquisition of mutans Streptococci in infants:

Different elements effect the initial acquisition of Streptococcus mutans in children, including increased maternal levels and caries status, low child birth weight, obturators/ oral appliances for management of cleft palate, early tooth eruptions and decrease salivary Ig A antibody level.

Li et al scrutinized sequences of maternal and perinatal variables and showed a reduced multivariable model. STD infection involvement and family income could be a reason for 35.9% of discrepancy in the time to infection by Streptococcus mutans in the infant. Regarding the use of antibiotics, researchers did not find a substantial correlation amongst exposure to antibiotics and the period to infection by Streptococcus mutans in infants[37]. Amount of this lack of difference might be due to the high rate of antibiotic usage (91.7%) for the management, treatment and or prevention of STDs. Thus the role of antibiotics in 'time to colonization' couldn't be absolutely determined and remains a probable contributor to the difference in Streptococcus mutans acquisition.

Researches have revealed that vaginally delivered babies show a different microbial composition in the intestines compared with babies delivered by (C-section) caesarean section or with the assistance of instruments. By the age of 1 year, babies delivered by (C-section) in Western nations have a reduced ratio of anaerobic to facultative anaerobic bacteria in the gut as matched to vaginally delivered infants, probably demonstrating a less mature microbiota[38]. Furthermore, it was recommended that babies delivered by C-section harbor less proportions of the health-promoting strains of lactobacilli and bifidobacteria. Probable consequences of the delivery means are, however, sparsely investigated, examined and studied with regard to the oral microflora[38].

Li and colleagues have stated in their research of the early colonization of the mouth that it was shown that babies delivered by C-section assimilated the caries-associated pathogen. Some species of streptococci may also be seen in the genitourinary or the vaginal tract[37]. Sautter and Brown collected 65 specimens from vagina normal young women and found the presence of group B streptococci species in 12 %, group D streptococci species in 13 % and Streptococcus not group A, B or D in 7%[39]. Zhou et al also found the presence of Streptococci in healthy Caucasian and black women[40]. Vaginally/ naturally delivered babies come into contact with larger numbers and variations of bacteria from the perineum i.e. vagina and anus previously and with more intensity than do the moderately aseptically delivered C-section born infants indicating a possibility for initial acquisition but it still remains debatable[37].

Host factors affecting oral colonization of M. Streptococci in infants[36]:

Hereditary factors:

Hereditary factors and its role in dental caries is well established, thereby the probability of them being associated with the colonization of MS in kids. It can be hypothesized that genetic factors like HLA genes which modulate the host's immunological responses thereby influence colonization of MS in the oral cavity. Additionally there is a possibility that these genetic factors would control the bacterial colonization by changes in saliva, tooth and mucosal surfaces. To know the comparative contribution of hereditary and environmental factors affecting the oral colonization twin studies are very useful. The relative amount of genetic contribution to trait variation is studied by comparing results of identical or monozygotic twin pairs, who share all their genes, with non-identical or dizygotic twin pairs, who share about half of the segregating genes.

The hypothesis that streptococci salivary levels been controlled by genetic factor but lactobacilli levels been related to environmental factors was first proposed by Goodman et al and later extended by molecular studies done by Acton et al suggesting that MHC genes might play a role in modulating susceptibility for MS colonization levels. While the reported results of Ozawa et al are contradicting. Nearly 52% variation in the salivary MS levels was the result of genetic contribution and the rest variation was supposed to be due to environmental factors as was reported in a recent study conducted on preschool twins[41].

Tooth and mucosal surface factors:

The main adherence surface for MS colonization is the hard non-shedding tissue but recent increasing proofs suggest that even oral mucosa may have a part in MS colonization. According to latest studies it has been reported that in predentate children the oral mucosal surface changes manifested as developmental nodules and oral clefts are providing high retention sites to assist colonization of MS. Hard tissue surface abnormalities and the use of cleft palate obturators may also control the adherence and colonization of MS. Tooth surface irregularities caused by Enamel hypoplasia enhances MS colonization on the surface of the tooth because of increased bacterial adherence, decrease in carbohydrate clearance and plaque retention.

A wide range of inherited and acquired systemic etiologic factors like amelogenesis imperfect (genetic abnormality) to hypo phosphatasia (congenital abnormality of mineralization) to rickets (metabolic defect) and prematurity of birth are a cause of enamel defects. Therefore it is expected that children with these conditions predisposed to enamel defects have a greater risk for early MS colonization[42].

Saliva:
Saliva's protective role against oral bacteria is a well-established fact. Due to the presence of different antimicrobial components in saliva it mediates selective adhesion and colonization of MS on the tooth surface. sIgA (salivary immunuglobin A), mucins, fibronectin, glycoproteins and lysozyme promote MS agglutination and increase bacterial removal[43].

Adherence of Stertococcusmutans is prevented by the other proteases and galactosides present in saliva which destroy the surface protein antigens of S.mutans.However, the neutralization of acid resulting from MS fermentation of substrate due to the oral clearance by salivary flow and buffering capacity influence MS colonization. At night oral clearance is believed to be low because of the low salivary flow rate which provides a good condition for enhancing MS colonization and plaque growth[36].

Congenital abnormalities, surgical resection of salivary glands, compromised salivary gland function due to radiotherapy or medication like salbutamol leads to a compromised salivary flow in children who then become prone to be at a higher risk for early MS colonization[44].

Immunological factors:
It is still uncertain as to what role do immunological factors play in MS colonization and dental caries whereas immunological factors do have a significant role in general infections. Proofs suggest that children are orally immunocompetent with certain immune factors at birth or soon after with an increase in antibodies levels against MS with age. The colonization and pathogenicity of MS are mostly influenced by immune factors like sIgA or serum and IgG, IgA, and IgM. Inhibition of the adherence of MS to saliva coated hydroxyapatite and epithelial surfaces as well as neutralization of MS enzymes and virulence factors are caused by both sIgA and IgG. MS colonization may be reduced by enhanced lactoferrin, peroxidase and lysozyme activities in saliva which are enhanced by IgA[45].

Phagocytosis and killing of MS as well as other oral microbes by complement activation or opsonisation is enhanced by IgG antibodies found in gingival cervical fluid[43]. In the early days of primary immune response, IgM antibodies that agglutinate MS and fix complement are vital. But still in MS colonization and dental caries the role that immunoglobulins play is unclear. Separate studies have revealed the presence of high anti MS immunoglobulins associated with and without caries.

Feeding habits and sugar exposure:
Increased MS levels and caries in children are due to persistent bottle feeding with drinks containing sugar, sweetened fluids fed at night, and snacks taken at short intervals. The main cariogenic sugar identified is Sucrose which is metabolized by MS producing plaque dextrans crucial for its adherence and colonization in the oral cavity. In young children fruit juices, sweet solids and drinks which can be easily metabolized by MS into acids demineralizing the tooth structure, are a possible source of sucrose. [The acidity of plaque is increased and establishment and dominance of aciduric MS is due to the high occurrence of sugar in solids as well as liquids[46].

Early colonization of MS in children is strongly related with regular intake of snacks containing sugar which has been strongly proved by the longitudinal studies conducted by Wan et al, Law and Seow[15,22]. As a matter of fact many kids are fed with sweet solids so as to encourage them to take solid food and this leads to higher risks of MS colonization in kids who are started on solids earlier. In Vivo investigations of the cariogenicity of human breast milk and bovine milk is not extensive but it is generally believed that both are negligibly cariogenic.

Bowen and Lawrence's recent animal study suggested that human breast milk at high frequency exposures had greater cariogenicity than bovine milk, may be due to the higher lactose concentration in human breast milk (7%) when compared to bovine milk (4%)[47].MS colonization can be theoretically prevented and reduced by the presence of sIgA, enzymes, lactoferrin, interferon and lysozymes in human breast milk, but clinically their effect on MS colonization is still unclear.

Marta and Garcia Godoy determined the relationship between early colonization and feeding habits in infants of 15-20 months and found that the percentage of MS colonization was relatively high (46%) in children who used a nursing bottle, often with sugar content (cereal was added in 83% of children, who were still bottle fed), although no child presented caries at the time. A relation was also found between MS colonization and the absence of breastfeeding or, on the other hand, the excessive prolongation of natural or artificial feeding[48].

Erickson estimated the caries-related risk associated with 26 infant formulas and whole milk and found that some infant formulas support significant bacterial growth and may dissolve enamel mineral content[49].

Systemic antibiotics:

It is suggested that MS colonization may be removed or lessened by the use of long term antibiotic treatment in kids even though the degree of protection from a single course is unknown. Whereas antibiotic effect of the drugs is usually confused by the caries promoting effect of sucrose found in some drugs and effects of enamel hypoplasia that may be the outcome of systemic infections forcing medication[50]. Dasanayake et al presented a report, which could be explained by these factors, showing higher MS occurrence and higher risk of caries in kids who were administered sucrose containing drugs regularly[14].

Fig 1:- Summary of basic differences within the 'mutans group' of streptococci:[12]

Species	mutans			rattus	sobrinus			cricetus	ferus
Serovar	c	e	f	b	d	g	h	a	C
Genotype mol %: guanosine + cytosine	36-38			42-43	44-45			43-44	44
Fermentation: Mannitol	+	+	+	+	+	+	+	+	+
Sorbitol	+	+	+	+	+/-	+/-	+/-	+	+
Raffinose	+	+	+	+	-	-	-	+	_
Melibiose	+	+/-	+	+	-	-	-	+	Not Determined

Fig 2:Studies showing relationship between Mutans Streptococci (MS) levels in mothers and their children: [14,17,22,23,24,]

Paper	Country	Mother–Child Pairs	Child's Age	Outcome
Kohler et al (1978)	Sweden	36	4-5 Years	75% of children whose mothers has high MS were colonized 25% of children whose mothers had low MS were colonized
Berkowitz et al (1981)	USA	156	8-18 Months	58% of children whose mothers had high MS were colonized 12% of children whose mothers has low MS were colonized
Brown et al (1985)	Australia	24	<6 years	Mothers and children's MS levels were significantly associated 91% of children whose mothers had high MS were colonized 15% of children whose mothers had low MS were colonized
Brown et al (1985)	Australia	112	<2 years	Mothers and children's MS levels were significantly associated 79% of children whose mothers had high MS were colonized 52% of children whose mothers had low MS were colonized
Paper	Country	Mother–Child Pairs	Child's Age	Outcome
Aatonen et al (1988)	Finland	50	4-6 years	Mothers and children's MS levels were significantly correlated (r=0.48)
Caufield et al (1988)	USA	87	2-5 years	Mothers and children's MS levels were significantly colonized 68% of children whose mothers had MS above the median had colonization levels above the median 40% of children whose mothers has MS below the median had colonization levels above the median
Caufield et al (1993)	USA	46	0-5 years	Mothers and children's MS levels were not significantly associated
Aatonen et al (1994)	Finland	55	6 years	Mothers and children's levels were not significantly associated
Roeters et al (1995)	Holland	252	2-5 years	Correlation between mothers and children's MS levels were low and did not exceed r=0.22

Tanner et al (2002)	Spain	156	6-36 months	Caregivers and children's MS level were significantly associated (81% of caregivers were mothers) 93% of children whose caregivers had MS were colonized 54% of children whose caregivers had no MS were colonized Children were 13 times more likely to be colonized if the care giver had MS
Thorild et al (2002)[36]	Sweden	200	18 months- 3 years	Mothers and children's MS levels were significantly associated 18 month olds 40% of children whose mothers had high MS were colonized 13% of children whose mothers had low MS were colonized 3 year olds 52% of children whose mothers had high MS were colonized 15% of children whose mothers had low MS were colonized. Children were more likely to be colonized of their mother had high MS (odds ratio=5.28)
Iedjosasong Ko et al (2002)	Japan	39	0-5 years	Mothers and children's MS levels were not significant
Wan et al (2003)	Australia	111	0-2 years	Mothers and children's MS levels were significantly associated Children whose mothers had high MS had 2.1-8.5 odds ratio of being colonized
Li et al (2005)	USA	156	0-4 years	Mothers and children's levels were not significant except the age of colonization
Stephen et al (2009)	USA	27	18 months- 6 years	Mothers and childrens MS levels were significantly associated Maternal transmission occurred in 41% of mother child pairs MS genotypes did not match maternal strains were identified in the majority of children (74%) within the S-ECC population

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