IJPSR (2019), Volume 10, Issue 11 **(Review Article)**

INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES AND RESEARCH

Received on 23 February 2019; received in revised form, 07 June 2019; accepted, 14 July 2019; published 01 November 2019

DENTAL CARIES AND *LACTOBACILLUS*: ROLE AND ECOLOGY IN THE ORAL CAVITY

Sonu Singh Ahirwar [1], M. K. Gupta [2] and Sunil K. Snehi [*1]

Department of Microbiology [1], Barkatullah University, Bhopal - 462026, Madhya Pradesh, India.
Swargiya Dadasaheb Kalmegh Smruti Dental Collage & Hospital [2], Nagpur - 441110, Maharashtra, India.

Keywords:

Lactobacillus, Oral cavity, Ecology, Dental caries, Biofilm

Correspondence to Author:
Dr. Sunil Kumar Snehi

Assistant Professor,
Department of Microbiology,
Barkatullah University, Bhopal - 462026, Madhya Pradesh, India.

E-mail: sunilsnehi@gmail.com

ABSTRACT: Dental caries is an infective serious transmittable bacterial disease characterized by a multi-factorial pathology. Main players in the etiology of this disease are cariogenic bacteria, fermentable carbohydrates, a susceptible tooth, the host, and the time. It is still one of the most common disease worldwide. Biological factor such as cariogenic bacteria plays an important role in dental caries initiation, progression as well as tooth decay. Previous studies suggested that the main pathogenic bacteria are *Streptococcus mutans* and *Lactobacillus*. Consequently, *Lactobacillus* is considered the second most cariogenic bacteria of oral flora. It is not the caries initiator but plays an important role in caries progression. Most of the previous studies were quantitative, so till now, researchers not know about the specific species of *Lactobacillus* which are directly associated with dental caries progression. Some species-level identification studies suggested that the most common *Lactobacillus* species are *L. gasseeri, L. fermentum, L. vaginalis* and *L. casei* which were predominantly reported at the most of the oral sites, *i.e.* saliva, tongue, carious lesion, dental plaque, *etc.* and maybe plays the main role in caries progression. The present review spotlights the role, pathogenicity of *Lactobacillus* in dental caries and their ecology in the oral cavity.

INTRODUCTION: Now a day's, the oral diseases such as dental caries and periodontal diseases are still the most prevalent diseases worldwide, and WHO considers these pathologies as one of the most important public health issues. The World Health Organization described that 60–90% of school children, and nearly 100% of adults have dental caries. Dental caries can be prevented if a constant low level of fluoride is maintained in the oral cavity.

QUICK RESPONSE CODE

DOI: 10.13040/IJPSR.0975-8232.10(11).4818-29

This article can be accessed online on www.ijpsr.com

DOI link: http://dx.doi.org/10.13040/IJPSR.0975-8232.10(11).4818-29

Severe periodontal disease, which may result in tooth decay, is found in 15–20% of middle-aged (35-44 years) adults. Globally, about 30% of people aged 65–74 have no natural teeth. Cases of oral diseases in children and adults are more among poor and disadvantaged population groups. Risk factors for oral diseases include an unhealthy diet, tobacco use, abusive alcohol use, poor oral hygiene, and other social determinants [1, 2, 3, 4, 5, 6].

As we know, dental caries is an infective serious transmittable bacterial disease characterized by a multi-factorial pathology. Main players in the etiology of this disease are cariogenic bacteria, fermentable carbohydrates, a susceptible tooth, the host and the time [7, 8, 9]. At present, it is still one of the most common disease worldwide. If good oral hygiene is followed, chances of dental caries will

be less. When the equilibrium is compromised, and when an imbalance appears among the indigenous bacteria, pathologies such as dental caries or periodontitis could occur. However, the presence of nutrients, epithelial debris, and secretions make the mouth a favorable habitat for a great variety of bacteria. The most notorious bacteria for dental cavities are *S. mutans,* and *Lactobacilli* [10, 11, 12], among the wide range of bacteria, occurs in the mouth which produce acids from fermentable carbohydrates and cause demineralization, whereas others contribute to plaque development [13, 14, 15].

Historically, *Lactobacillus* was the first known microorganism associated with dental caries development [16]. They appear during the first years of a child's life [17] and are present in high numbers in saliva, on the dorsum of the tongue, mucous membranes, the hard palate, in dental plaque and, in fewer numbers, on tooth surfaces [18]. During the past few decades, the *Lactobacillus* genus has been subjected to numerous taxonomical changes. Thus, *Lactobacillus* is the group of the bacteria which produces lactic acid after the carbohydrate fermentation. It is gram's positive, rod shape facultative anaerobic, non-spore forming bacilli. It comprises over 150 species which are acid tolerant and characterized by a low G+C content of the genome. The percentage of GC in the DNA is 40% (by either Tm method or Bd method) [19, 20, 21]. It has been abundantly present in the milk and milk production as well as also found in feces, saliva [22, 23], stomach, vagina, gastrointestinal tract, and oral cavity [24, 23].

This review aims to describe the pathogenicity and role of *Lactobacillus* in the formation of dental caries. Moreover, their ecology in the oral cavity and how to get prevent caries initiation.

History of *Lactobacillus* in the Oral Cavity: The oral cavity is the part of the mouth behind the teeth and gums that is bounded above by the hard and soft palates and below by the tongue and the mucous membrane connecting it with the inner part of the mandible. It is not a uniform atmosphere for the resident of microbes but offers numerous distinct niches for microbial growth, such as the tongue [25], teeth, gingiva, hard and soft palates [26]. The history of *Lactobacillus* in the oral cavity started from over a century, a scientist name Lewkowicz (1901), was initially isolated from the oral cavity of the patients of dental caries [16].

For nearly a century, *Lactobacillus* has been generally associated with dental caries [16]. However, the key ecological determinant for the sustained colonization of lactobacilli in the oral cavity seems to be the presence of caries. It is of historical note to mention that this relationship was established by Harrison and Opal (1944) [27], they isolated lactobacilli from 93% of saliva samples and 66% of fecal samples of children with dental caries but not from those who were caries free [11]. The major characteristic of the genera *Lactobacillus* is acid production from fermentable sugars. Rodríguez-Vargas, (1921), had isolated three species of the lactic acid bacteria from dental caries, and he concluded that these species of *Lactobacillus* were responsible for the production of acids that cause tooth decay [28]. Owen OW described that it was the first microorganism which was responsible for dental caries development [29]. The oral cavity is free from microbes at the time of birth; the colonization occurs after some time [30].

Lactobacillus appears during the first years of a child's life, and are present in high numbers in saliva, on the dorsum of the tongue, mucous membranes, the hard palate, in dental plaque and, in fewer numbers, on tooth surfaces [18]. Although, five hundred strains of lactobacilli were isolated from fresh saliva samples of children, of which *L. salivarius* and *L. cellobiosus* are novel. Based on the above observation, Ragosa *et al*., concluded that not only *L. acidophilus* found in the saliva sample but also two or three species were present at a time in each sample [31]. In the decade of 1950s Davis, was tried to classify *Lactobacilli* which were isolated from the human mouth, where total 473 strains were classified into five groups as *L. acidophilus, L. casie, L. plantarum, L. fermenti, L. bravis* [32].

Steinle *et al.,* (1967) investigated the relationship between the carious lesion and dental caries; the agar-replica method was used to determine the relationship between the clinical carious lesion and the localized *Lactobacillus* growth sites. The results of the study suggested that this technique is significant in studying the relation of *Lactobacilli* to carious lesions and, further, that it can be used in

longitudinal studies of dental caries. The technique added the advantage of studying it at their actual points of growth. Their observations suggest that a direct relationship exists between the presence of *Lactobacilli* and the active carious lesion. Thus the concentration of *Lactobacillus* in the oral cavity is 1% of the entire cultivable oral flora [33, 34].

Lactobacillus plate count being used from 1985s for the process of caries detection [35], and shows a positive correlation in carries as caries increases the count also increase with positive caries activity [36]. It is already proved that the number of DMFS and concentration of *Streptococcus mutans* and *Lactobacillus* is correlated to each other if the number of DMFS increases then *S. mutans* and *Lactobacillus* count will also be increased [37]. Previous studies suggested that oral lactobacilli are directly associated with caries development [38].

Modern studies suggested that the younger infants below 10 years also showed a correlation between the presence of *Lactobacillus* in the oral cavity and dental caries [39, 40, 41, 22]. They have regularly isolated from caries active sites and in the almost caries studies [42] so that in the various private dental clinics an assay for *Lactobacillus* count have been used [43]. In the adults, lactobacillus reported in the 100% samples and associated with dental caries [44]. Most recently, three species of *Lactobacillus* are *L. fermentum*, *L. gasseri*, and *L. salivarious* were identified in the most carries active subjects and absent in caries-free subjects [45, 46, 47, 48]. The scenario in the developing countries like India is not different; their presence is reported in most of dental caries-related studies [47, 49, 50, 51, 52].

Ecology of *Lactobacillus* in the Oral Cavity:

***Lactobacillus* in the Saliva of Caries Active Subjects:** Saliva is a biological mixture of fluid which is secreted by salivary glands. It contains 99% of water, and the remaining 1% is electrolytes, proteins, and enzymes. Saliva establish a link between soft and hard tissues of the oral cavity [53, 116, 118]. When oral bacteria are in the planktonic state (for instance; in saliva), they are not considered as the causative agent of dental caries. However, some bacteria in saliva mediate the transmission of pathogenic bacteria intraorally [53, 54]. The genus *Lactobacillus* represents 0.1% of the total salivary flora; however, a correlation also exists between *lactic acid bacteria* of dental plaque and in human saliva. The critical concentration of *Lactobacillus* is 10^5 CFU/ml of saliva, which can cause oral disease such as dental caries in human [18, 52]. So, the total salivary count test uses for detection of dental caries. It is believed that the *Lactobacillus* absent in the oral cavity of newborns and colonization occurred after the few hours of birth when newborns started the breastfeeding. Various species of *Lactobacillus* reported in saliva **Table 1**; the common are *L. acidophilus, L. casei, L. crispatus, L. fermentum, L. gasseri, L. oris, L. paracasei, L. plantarum, L. rhamnosus,* and *L. salivarius* [24, 26, 52, 56, 57, 58, 59, 120].

TABLE 1: *LACTOBACILLUS* SPECIES ISOLATED FROM SALIVA OF CARIES SUBJECTS

Koll-Klais *et al.*, (2005) [26]	Tennpaisan and Dahlen (2006) [56]	Román-Méndez *et al.*, (2009) [60]	Piwat *et al.*, (2010) [22]	Yang *et al.*, (2010) [45]	Anderson *et al.*, (2014) [61]	Zhang *et al.*, (2014) [57]	Wang *et al.*, (2017) [62]
L. rhamnosus	*L. rhamnosus*	*L. brevis*	*L. rhamnosus*	*L. rhamnosus*	*L. salivarius*	*L. rhamnosus*	*L. gasseri*
L. paracasei	*L. casei*	*L. rhamnosus*	*L. paracasei*	*L. salivarius*	*L. gasseri*	*L. salivarius*	*L. fermentum*
L. plantarum	*L. plantarum*	*L. delbrueckii*	*L. salivarius*	*L. gasseri*	*L. rhamnosus*	*L. gasseri*	*L. vaginalis*
L. acidophilus	*L. acidophilus*	*L. acidophilus*	*L. gasseri*	*L. casei*	*L. paracasei*	*L. plantarum*	*L. mucosae*
L. fermentum	*L. fermentum*	*L. plantarum*	*L. oris*	*L. fermentum*	*L. fermentum*	*L. pentosus*	*L. reuteri*
L. salivarius	*L. salivarius*	*L. salivarius*	*L. mucosae*	*L. oris*	*L. oris*	*L. paracasei*	
L. oris				*L. vaginalis*	*L. crispatus*	*L. oris*	
L. delbrueckii					*L. kalixensis*	*L. fermentum*	
L. gasseri					*L. reuteri*	*L. delbrueckii*	
L. crispatus					*L. helveticus*	*L. casei*	
						L. mucosae	

***Lactobacillus* on Tongue:** *Lactobacillus* present in the all most and all sites of the oral cavity of a human. Their presence in the tongue was most studied; various species of lactic acid bacteria were reported abundantly on the soft tissues of tongue in the caries active subjects [63]. Some previous studies have shown that tongue cleaning leads to a healthy oral environment, and also inhibit the growth of cariogenic bacteria as well as removed the other main etiological agents of dental caries and

periodontal diseases [64]. Ahumada *et al.* characterized lactobacilli in the children who had caries; they were identified 31 different species where 78% came from the tongue surface and rest of from the gums. The other author identified two species of *Lactobacillus* on the tongue surface of periodontitis patients, but their study didn't found any co-association between lactobacilli and periodontitis [25, 65]. Some previous study supported that dominant flora of tongue also associated with dental decay **Table 2**, especially *L. gasseri* detected in childhood and adult caries [66], *L. vaginalis* reported in caries-active women, and *L. fermentum* is associated with childhood caries [67, 68].

TABLE 2: *LACTOBACILLUS* SPP. IDENTIFIED FROM THE SURFACE OF TONGUE

Ahumada *et al.*, (1999) [63]	Colloca *et al.*, (2000) [69]	Tanner *et al.*, (2002) [25]	Gizani *et al.*, (2009) [70]	Kanasi *et al.*, (2010) [67]	Asakawa *et al.*, (2018) [68]
L. delbrueckii	*L. acidophilus*	*L. acidophilus*	*L. acidophilus*	*L. fermentum*	*L. fermentum*
L. lactis	*L. delbrueckii*	*L. uli*		*L. vaginalis*	*L. salivarius*
L. bulgaricus	*L. salivarius*			*L. gasseri*	*Lactobacillus spp.*
L. salivarius	*L. agilis*			*L. acidophilus*	*L. vaginalis*
L. acidophilus	*L. brevis*			*L. plantarum*	*L. paracasei*
L. paracasei	*L. casei*				*L. oris*
L. plantarum	*L. rhamnosus*				*L. ultunensis*
L. fermentum	*L. plantarum*				*L. gasseri*
L. rhamnosus	*L. fermentum*				*L. iners*
L. reuteri	*L. paracasei*				*L. reuteri*
					L. pentosus
					L. cripatus

***Lactobacillus* in Dental Plaque:** Dental plaque is a very complex bacterial community which is accumulated on the hard tissue of the oral cavity. Over 500 bacterial species comprise in the plaque formation [71]. It is difficult to give the exact idea of the relationship between *Lactobacillus* and dental plaque, but some study which is highlighted and strongly recommended that *Lactobacillus* is the part of the dental plaque and his presence plays an ideal role in caries development. Corby *et al.*, [72] studied on the microbial risk indicator of early childhood caries and observed that the top *Lactobacillus* species found to be overabundant in the caries-active group were *L. fermentum* and *L. gasseri* which are associated to dental plaque formation as well as caries. Some study gave interesting results because it has been quantitatively less reported in the dental plaque than in saliva [73, 74]. Most of the studies reported *L. fermentum*, *L. gasseri*, *L. casei* and *L. salivarius* in the dental plaque of children and young adults [74, 75]. Thus, these three species of *Lactobacillus* have been also reported in the childhood caries when the plaque samples had cultured anaerobically on the blood agar medium [11, 76]. Some recent studies conformed **Table 3** that the *L. casei* and *L. gasseri* predominant in childhood caries, *L. fermentum* and *L. salivarius* are more frequently found in dental plaque samples in adults [46, 77, 78, 119].

TABLE 3: LACTOBACILLUS SPP. IDENTIFIED FROM DENTAL PLAQUE OF CARIES SUBJECTS

Corby *et al.*, (2005) [72]	Aas *et al.*, (2008) [74]	Gross *et al.*, (2010) [75]	Tanner *et al.*, (2011) [76]	Mannaa *et al.*, (2013) [77]	Shimada *et al.*, (2015) [46]	Richards *et al.*, (2017) [78]
L. gasseri	*L. casei*	*L. ultunensis*	*L. fermentum*	*L. fermentum*	*L. gasseri*	*L. salivarius*
L. fermentum	*L. gasseri*	*L. casei/ paracasei*	*L. gasseri*	*L. salivarius*	*L. fermentum*	
Lactobacillus spp.	*L. fermentum*	*L. crispatus*	*L. casei/ paracasei*	*L. casei*	*L. salivarius*	
	Lactobacillus sp.	*L. delbrueckii*	*L. oris*		*L. casei*	
L. vaginalis		*L. fermentum*	*L. salivarius*		*L. rhamnosus*	
		L. gasseri			*L. vaginalis*	
		L. oris			*L. brevis*	
		L. plantarum			*L. pentosus*	
		L. rhamnosus				
		L. salivarius				
		L. vaginalis				

***Lactobacillus* in Carious Lesion:** It is still not known that how many microbial species involved in dental caries, scientists have been continued to develop the new techniques for the identification of various microbial populations [79, 80]. Till now, over 500 bacterial species have been identified by culture-dependent and culture-independent methods in the oral cavity [74]. Although, various microorganisms are generally recognized to be associated with disease progression, and some are associated with disease initiation. The association between lactobacilli and dental caries was reported by Goadby in 1899, and experimental evidence was established after the 1950s for the fundamental role of these bacteria in dental caries [18]. They are now considered secondary invaders rather than initiators of the caries process [11, 15, 43, 78, 81].

The studies of *Lactobacillus* in the particular site like carious lesion in the oral cavity not much studied; various authors have only made a simple quantitative analysis of lactobacilli rates [42, 81, 82]. Some site-specific studies **Table 4** also suggested that *Lactobacillus* is the predominant oral flora of carious lesion, and it's directly associated with deep caries and tooth decay. The most common species are *L. casei L. fermentum L. rhamnosus L. gasseri L. crispatus* which are abundantly present in the carious lesion of caries subjects [42, 66, 79, 80, 83, 84, 85].

TABLE 4: *LACTOBACILLUS* SPP. ISOLATED FROM CARIOUS LESION OF CARIES SUBJECTS

Ando and Hoshino 1990 [83]	Becker *et al.*, 2002 [84]	Byun *et al.*, 2004 [42]	Munson *et al.*, 2004 [66]	Chhour *et al.*, 2005 [80]	Obata *et al.*, 2014 [79]	Kianoush *et al.*, 2014 [85]
L. brevis	*L. fermentum*	*L. rhamnosus*	*L. rhamnosus*	*L. pontis*	*L. rhamnosus*	*L. fermentum*
L. casei		*L. paracasei*	*L. casei*	*L. casei*	*L. fermentum*	*L. rhamnosus*
L, catenaforme		*L. fermentum*	*L. gasseri*	*L. rhamnosus*	*L. paracasei*	*L. crispatus*
L. crispatus		*L. delbrueckii*	*L. fermentum*	*L. panis*	*L. gasseri*	
L. minutes		*L. gasseri*	*L. oris*	*L. reuteri*	*L. crispalus*	
L. ptantarum		*L. salivarius*	*L. plantarum*	*L. fermentum*	*L. delbrueckii*	
L. rogosae		*L. crispatus*	*L. vaginalis*	*L. delbrueckii*	*L. oris*	
Lactobacillus spp.		*L. gallinarum*		*L. salivarius*	*Lactobacillus* spp.	
L. paracasei				*L. gasseri*	*L. reuteri*	
					L. vaginalis	
					L. nagelli	
					L. galliranum	

Role of *Lactobacillus* in Dental Caries Formation: Its role in the dental caries formation is not well defined, few previous studies support that the *Lactobacillus* spp. play an important role in dental caries development because their nature to able to survive in the low pH environment. These bacteria use fermentative metabolism and according to these species, their metabolism methods are two types: homo-fermentative and hetero-fermentative. Some species produce lactic acid due to homo-fermentative metabolism, and other produce lactic acid, CO_2, acetic acid or ethanol via using hetero-fermentative mechanisms [21, 81].

The *Lactobacillus* species whatever metabolic mechanism used, it results is only the acidification of the environment. Thus, these bacteria cause pH value decrease less than 4.5 in the oral environment, and they can survive in a pH up to 2.2 [86]. These bacteria have two cariogenic properties, such as adhesion and acid production. Although, the ability of the *Lactobacillus* adhere to the cells culture, and the solid surface is not noteworthy.

The species of *Lactobacillus* have the S-layer protein in their cell wall; the protein has a crystalline structure and responsible to the cell surface hydrophobicity [69]. The exopolysaccharide produced by the *Lactobacillus* species is a key factor in the biofilm formation; numerous studies confirmed that some species of *Lactobacillus* produce exopolysaccharide extensively [65, 87, 88].

Cariogenic Properties and Pathogenicity of *Lactobacillus:* The various microorganisms play their roles in the formation of dental caries. The genera *Lactobacillus* also associated with the dental caries development [46, 81]. If microbes are in the mouth are equilibrium state, dental caries will never develop. The adherence and acid production capacity of *Lactobacillus* is enough for dental

caries development [89, 90]. The cariogenic properties of such *Lactobacillus* are discussed below:

Acid Production: Cariogenic activity of *Lactobacillus* is attributed to the production of lactic acid by the sugar fermentation. Based on carbohydrate metabolism *Lactobacillus* are two types homo-fermentative synthesize only lactic acid whereas hetere-fermentative synthesizes CO_2, acetic acid or ethanol apart from lactic acid. Both the metabolic pattern used by the *Lactobacilli* resulted in an acidification of the environment [91, 92]. It is believed that the *Lactobacillus* is the prime micro-organism in caries development because of their high numbers in carious lesions and their acidogenic and aciduric properties. Various studies have shown that *Lactobacillus* is excellent acid tolerance bacteria, and they have the ability to survive below pH 4.5 [34, 93]. They change the pH of the oral environment neutral to acidic. In the colonization and maturation stage of dental caries formation they get attaches to dental plaque and started to dematerialize the teeth enamel [88, 92, 94].

Cell Surface Hydrophobicity: Attachment of *Lactobacillus* to the tissues or tooth surface is the first step for biofilm formation where the cell surface hydrophobicity greatly modulates the attachment properties of the lactic acid bacteria. In the living systems, the strongest long-range non-covalent interaction is hydrophobic interaction. The hydrophobic interactions are as a major determining factor in microbial adhesion to surfaces [88]. Although, there are several studies concerning the cell surface hydrophobicity of the oral lactobacilli. The S-layer present at the outermost cell surface of some *Lactobacillus* species is responsible for cell surface hydrophobicity [95]. Vadillo-Rodríguez *et al*., studied on the cell surface hydrophobicity of two species of *Lactobacillus*, they concluded that in a low ionic strength suspension, both *Lactobacillus* strains show higher initial deposition rates to hydrophobic glass than to hydrophilic glass, whereas in a high ionic strength suspension no clear influence of cell surface hydrophobicity on adhesion is observed [65, 96, 97, 121].

Exopolysaccharide Produced by *Lactobacillus:* It is the extra polymorphic substances produced by some plants, fungi, and bacteria; which are long-chain, linear or branched biopolymers. It is high molecular weight biopolymers consists of repeating units of saccharide and linked by α- and β-glycoside bonds. They are sticky in nature and play an important role in the adhesion to the dental biofilm [65, 78]. So far, various EPS-producing *Lactobacillus* species have been identified such as *L. casei, L. acidophilus, L. brevis, L. curvatus, L. delbrueckii subsp. bulgaricus, L. helveticus, L. rhamnosus, L. plantarum,* and *L. johnsonii* [65, 97, 98].

S- Layer Proteins of *Lactobacillus*: The cell wall is an outer layer structural component of bacteria and essential for many aspects of their life. The S-layer proteins are the outermost cell envelope, which is made of different kinds of proteinaceous substances. Particularly, the adaptations of the microorganisms to adverse environmental conditions occur from the diverse structures of the outermost boundary layers of bacteria. The biological functions of S-layer proteins of *Lactobacillus* are not well known, but they play an important role in cell adhesion to host cell. Thus, *Lactobacillus* can adapt their surface hydrophobicity in environmental changes because they have S-layer proteins in their outer cell wall [97, 99, 100].

Mechanisms of Dental Caries Formation: Dental caries is commonly known as tooth decay. The mechanism of tooth decay is very complex. It is known from over 50 years that dental caries causes by the fermentation of food material, these food fermented by certain acid producing bacteria. Two types of bacteria are involved in tooth decay; which is: *mutans streptococci* and *Lactobacillus* species these both can produce acid during the metabolism of sugar fermentation [15, 74, 94, 97]. When these bacteria produced acid in the dental plaque on tooth surface they readily diffuse in all directions in the mouth. The acid, of course, also diffuses into the pores of enamel or dentine. When the acid diffuses into the tooth, it finds soluble acid mineral, and they begin to dissolve it. In the mouth, this process progresses long enough; the result is a cavity. This process usually takes several months to years for progression to cavitation, the end-point of the disease process known as dental caries [15, 101, 102]. In the mechanism of dental caries, the following steps are involved:

Biofilm Formation Capability of *Lactobacillus*: The biofilm is described by Donlan and Costerton in 2002, according to them biofilm is a microbial-derived sessile community characterized by cells which are irretrievably attached to the solid surface and interface to each other. The biofilm formation and development is affected by various factors, such as bacterial population, surface properties and some environmental factors like pH, availability of nutrients and temperature of the oral environment [103, 104, 105]. In the biofilm formation, so many steps are involved, but four steps are important such as adhesion, colonization, maturation, and dispersion. In the first stage, microbial cells start to attach the tooth surface. In the second step, cells start to colonize. In the third step, colonized microbial populations start to maturation, and in the last step, the mature population starts to disperse in the oral cavity [106, 107]. The detail description of each steps **Fig. 1** of the biofilm formation describe below:

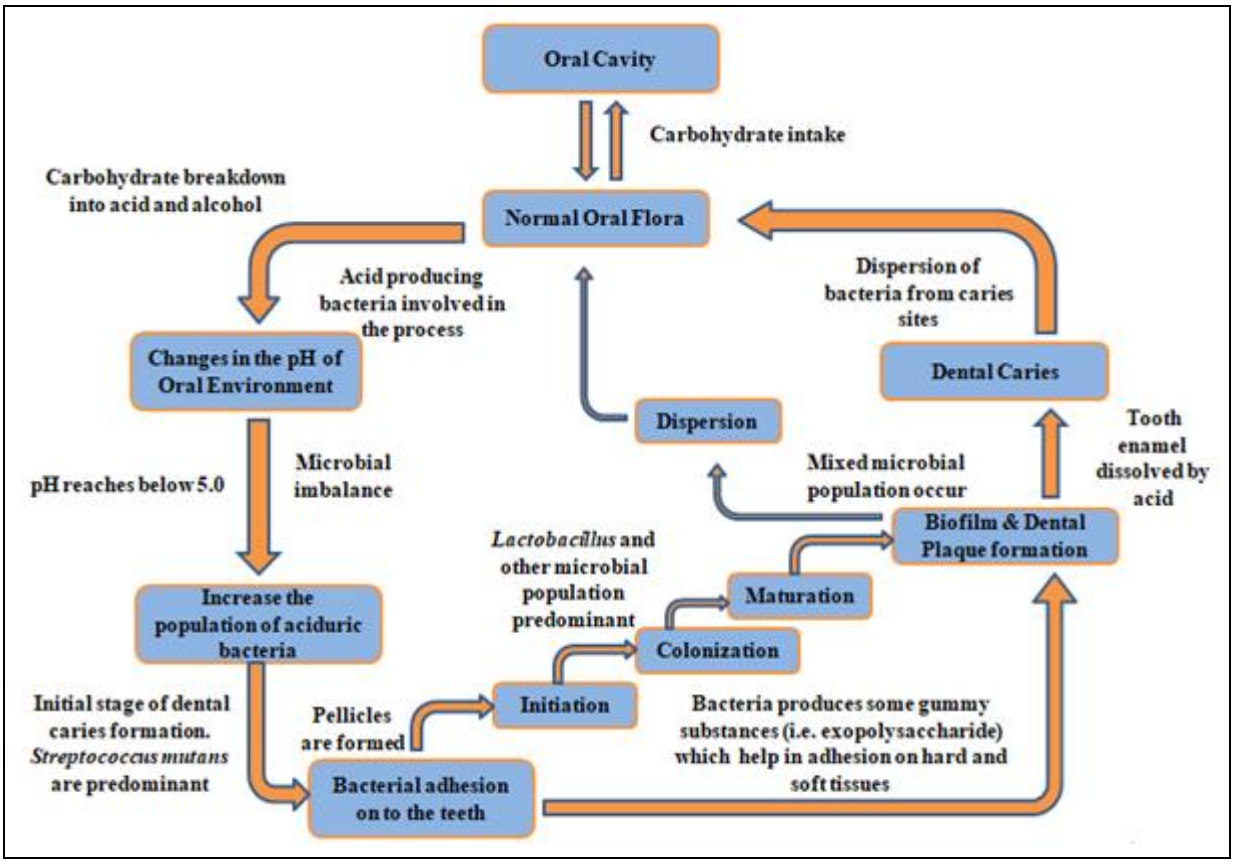

FIG. 1: MECHANISM OF DENTAL CARIES FORMATION

Dental Plaque Formation: The oral cavity is a safe place for microbial flora, where a variety of microorganisms live together. The dental plaque development occurs due to the growth of such bacteria which develops, of course on oral tissues and teeth. Thus, dental plaque is a composite microbial population which is attached on the teeth surface in the oral cavity [71, 104, 105, 107, 108]. When the microorganism starts to adhesion on the surface of teeth is a common condition of plaque formation. Structure of dental plaque varies from the tooth to tooth and location to location, and this is not a uniform structure [70]. When the environments of oral cavity get change, dental plaque structure also changes. Thus, we can say that dental plaque dependent on oral environment and change according to condition on oral environment and adhesion to the microbial population. The dental caries is the result of the adherent deposit of bacteria, and it is by-products [58]. These microbial community works together and always metabolically active. The ecology of dental plaque formation changes due to some external factors such as daily diet and dental care. Normally dental plaque development takes one to two weeks for maturation [105, 109, 110]. In the process of the dental plaque formation, three steps are included such as adhesion, colonization, and biofilm formation, which is described below:

Adhesion: Microbial adhesion to the solid surface is the first step to dental plaque formation. This process starts with the formation of the pellicle of the tooth surface, in the pellicle formation salivary glycoprotein does firstly attach to the surface of the tooth [111]. The pellicle is the proteins- coating film which is derived from salivary glycoproteins. Some planktonic bacteria start to recognize the glycoproteins in the pellicle, and after that begin to attach, the attachment starts within 0-4 hrs. In the initial stage, attachment occurs due to electrostatic attractions and physically attractions, after some time bacterium begins to produce some extra polymorphic substances which are called exopolysaccharides. These exopolysaccharides help in the bacteria stay bound together and attach to the pellicle [104, 106, 110]. The majority of microbes in pellicle are *mutans streptococci* and *Lactobacilli* spp. which are initially attached with the surface of naked tooth [15, 105, 110, 112].

Colonization: After 4-12 hours of adhesion, colonization takes place in the dental plaque formation. Colonization is an important process in plaque formation. In this process, the microbial population attached to the tooth surface and start multiplying. The planktonic bacteria cannot directly colonize on the tooth surface; they have required some receptors site on the cell surface for early colonization and adhesion to the surface. Co-aggregation is a specific cell-to-cell interaction reaction which occurs in oral bacterial colonization and dental plaque formation [104, 106, 110]. Co-aggregation is the most studies microbial interaction mechanism which occurs between distinct bacterial cells. According to the previous study, bacterial colonization starts from the bottom layer of dental biofilm. Early colonizers bind via the complementary pellicles receptors while secondary colonizer binds to previously bounded bacterial cells. *Streptococci, Lactobacilli,* and other anaerobic oral bacteria can co-aggregate with *Fusobacterium* spp, which is the primary colonizer of the tooth surface [15, 104, 113, 114].

Maturation: The next phase of dental plaque formation is maturation; in this phase generates new complex architecture, channels, pores, and the redistribution of bacteria away from the solid surface. The cluster of bacteria forms the mushroom-shaped structure on the tooth surfaces; the shapes depended on the source of nutrients. For the development of mature dental plaque required 8-10 day's period. The mature dental plaque contains numerous gram's positive and gram's negative bacterial species such as *Streptococcus mutans, Streptococcus mitis, Fusobacterium nucleatum, Treponema* sp, *Tannerella forsythensis, P. gingivalis, Aggregatibacter, Actinomycetes, Veillonella* sp. and *Lactobacillus* sp. [15, 90, 104, 115, 117].

Dispersion: Dispersion is the last phase of dental plaque formation; during this phase biofilm of microbes will detach from the colony and quickly returns to a planktonic form, spread out in the oral cavity [107, 114].

CONCLUSION: The present review concluded that a total of 25 species of *Lactobacillus* was reported in the various sites of the oral cavity; these vary with changing the oral sites. Some species dominant on a particular site but they were less reported on the other sites. The most common species which are mostly reported on both the oral sites are *L. gasseri, L. rhamnosus, L. reuteri, L. paracasie, L. plantarum, L. acidophilus, L. oris, L. fermentum, L. casei, L. salivarius, L. brevis,* and *L. vaginalis*. The caries-associated species are *L. gasseri, L. fermentum, L. vaginalis* and *L. casie* which are reported in all most all relevant studies. Where *L. gasseri,* previously detected in childhood and adult caries, *L. fermentum,* detected in childhood caries, *L. vaginalis,* reported in caries-active young women's saliva and *L. casei* reported in childhood and adult both caries. Most of the species were detected by molecular methods from children and adult's saliva, dental plaque, tongue, and carious lesion. In contrast, the *L. rhamnosus, L. acidophilus, L. plantarum,* and *L. reuteri,* which are used in probiotic infant enteric and oral anti-caries therapy, showed negative associations with caries.

Moreover, most of the studies were quantitative in respect of *Lactobacillus* in the oral cavity; the review reported that lactobacillus count higher in the active caries groups while less detected in the caries free groups. So we concluded that, if the sugar consumption is increased in the diet sugar degrading bacteria also increases and produces acid from their metabolic activity, this acid cases demineralization of teeth for his result, dental caries occur.

Thus, *Lactobacillus* does not directly cause dental caries but their secondary metabolic product, *i.e.* lactic acid plays a major role in dental caries formation.

ACKNOWLEDGEMENT: Author would like to thanks University Grant Commission (New Delhi, India) to provide Rajeev Gandhi National Fellowship for financial support (RGNF-2015-17-SC-MAD-20431), the author is also thankful to Prof. Anil Prakash, Head, Department of Microbiology, Barkatullah University Bhopal, India to provide infrastructure and moral support.

CONFLICT OF INTEREST: The authors declare that they have no conflict of interest.

ETHICAL APPROVAL: No ethical approval is required as no animals or humans have been used in the study.

REFERENCES:

1. Petersen PE: The World Oral Health Report 2003: continuous improvement of oral health in the 21st century - the approach of the WHO Global Oral Health Programme. Community Dent Oral Epidemiol 2003; 31: 3-24.
2. Jürgensen N and Petersen PE: Promoting oral health of children through schools: Results from a WHO global survey 2012. Community Dental Health 2013; 30: 204-18.
3. Kassebaum NJ, Bernabé E, Dahiya M, Bhandari B, Murray CJL and Marcenes W: Global burden of untreated caries: a systematic review and metaregression. J Dent Res 2015; 94: 650-58.
4. World Health Organization: Global Oral Health Data Bank. Geneva: WHO, 2016. http://www.mah.se/CAPP/Country-Oral-Health-Profiles
5. Kim J, Choi Y and Park S: Disparities in the experience and treatment of dental caries among children aged 9-18 years: the cross-sectional study of Korean National Health and Nutrition Examination Survey (2012-2013). Int J Equity Health 2016; 15: 88.
6. Alhabdan YA, Albeshr AG, Yenugadhati N and Jradi H: Prevalence of dental caries and associated factors among primary school children: a population-based cross-sectional study in Riyadh, Saudi Arabia. Environ Health Prev Med 2018; 23: 60.
7. Richard JL and Jenkinson HF: Oral Microbiology at a Glance. Wiley-Blackwell, 2010: 3.
8. Arora A, Manohar N and John JR: Factors Associated with Dental Caries in Primary Dentition in a Non-Fluoridated Rural Community of New South Wales, Australia. Int J Environ Res Public Health 2017; 14pii: E1444.
9. Brito AS, Clementino MA, Gomes MC, Barbosa Neves ET, Barbosa Ad, de Medeiros CA, de Aquino MM, Granville-Garcia AF and de Menezes VA: Sociodemographic and behavioral factors associated with dental caries in preschool children: Analysis using a decision tree. J Ind Soc Pedo Prev Dent 2018; 36: 244-49.
10. Laudenbach JM and Simon Z: Common dental and periodontal diseases: evaluation and management. Med Clin Borth Am 2014; 98: 1239-60
11. Caufield PW, Schon CN, Saraithong P, Li Y and Argimon S: Oral Lactobacilli and Dental Caries: A Model for Niche Adaptation in Humans. J Dent Res 2015; 94: 110S-118S
12. Tanner ACR, Kressirer CA, Rothmiller S, Johansson I and Chalmers NI: The Caries Microbiome: Implications for Reversing Dysbiosis. Advances Dent Res 2018; 29: 78-85.
13. Cotter PD and Hill C: Surviving the acid test: responses of gram-positive bacteria to low pH. Microbiol Mol Biol Rev 2003; 67: 429-53.
14. Bowen WH, Burne RA, Wu H and Koo H: Oral Biofilms: Pathogens, Matrix, and Polymicrobial Interactions in Microenvironments. Trends Microbiol 2018; 26: 229-42.
15. Lamont RJ, Koo H and Hajishengallis G: The oral microbiota: dynamic communities and host interactions. Nat Rev Microbiol 2018; 16: 745-59.
16. Lewkowicz X: Erch Med Expt et anat path (Paris) 1901; 13: 633-60.
17. Holgerson PL, Vestman NR and Claesson R: Oral microbial profile discriminates breast-fed from formula-fed infants. J Pediatr Gastroenterol Nutr 2013; 56: 127-36.
18. Badet C and Thebaud NB: Ecology of Lactobacilli in the Oral Cavity: A Review of Literature. Open Microbiol J 2008; 2: 38-48.
19. Hugenholtz P, Goebel BM and Pace NR: Impact of Culture-Independent Studies on the Emerging Phylogenetic View of Bacterial Diversity. J Bacteriol 1998; 180: 4765-74.
20. Claesson MJ, Van Sinderen D and OToole PW: The genus Lactobacillus– a genomic basis for understanding its diversity. FEMS Microbiol Lett 2007; 269: 22-28.
21. Salvetti E, Torriani S and Felis GE: The Genus Lactobacillus: A Taxonomic Update. Probiotics Antimicrob Proteins 2012; 4: 217-26.
22. Piwat S, Teanpaisan R, Thitasomakul S, Thearmontree A and Dahlén G: Lactobacillus species and genotypes associated with dental caries in Thai preschool children. Mol Oral Microbiol 2010; 25: 157-64.
23. Kim YS, Kang SM, Lee ES, Lee JH, Kim BR and Kim BI: Ecological changes in oral microcosm biofilm during maturation. J Biomed Opt 2016; 21: 101409.
24. Aas JA, Paster BJ, Stokes LN, Olsen I and Dewhirst FE: Defining the normal bacterial flora of the oral cavity. J Clin Microbiol 2005; 43: 5721-32.
25. Tanner ACR, Milgrom PM, Kent R Jr, Mokeem SA, Page RC, Liao SIA, Riedy CA and Bruss J: Similarity of the oral microbiota of pre-school children with that of their caregivers in a population-based study. Oral Microbiol Immunol 2002; 17: 379-87.
26. Kõll-Klais P, Mändar R, Leibur E, Marcotte H, Hammarström L and Mikelsaar M: Oral lactobacilli in chronic periodontitis and periodontal health: species composition and antimicrobial activity. Oral Microbiol Immunol 2005; 20: 354-61.
27. Harrison R and Opal Z: Comparative studies on lactobacilli isolated from the mouth and intestine. J Dent Res 1944; 23: 1-22.
28. Rodríguez-Vargas FE: The specific study of the bacteriology of dental cavities. Milit Dent J 1921; 4: 199.
29. Owen OW: A study of bacterial counts (lactobacilli) in saliva related to orthodontic appliances. Amer J Orthodont 1949; 35: 672-78.
30. Rosenblatt R, Steinberg D, Mankuta D and Zini A: Acquired Oral Microflora of Newborns During the First 48 Hours of Life. J Clin Pediatr Dent 2015; 39: 442-46.
31. Rogosa M, Wiseman RF, Mitchell JA, Disraely MN and Beaman AJ: Species differentiation of oral lactobacilli from man including descriptions of lactobacillus salivarius

nov spec and lactobacillus cellobiosus nov spec. J Bacteriol 1953; 65: 681-99.
32. Davis, GHG: The Classification of Lactobacilli from the Human. J Gen Microbiol 1955; 13: 481-93.
33. Steinle CJ, Madonia JV and Bahn AN: Relationship of Lactobacilli to the Carious Lesion. J Dent Res 1967; 46: 191-96.
34. Van-Houte J, Aasenden R and Peebles TC: Clinical Science: Lactobacilli in Human Dental Plaque and Saliva. J Dent Res 1981; 60: 2-5.
35. Crossner CG and Unell L: Salivary Lactobacillus counts as a diagnostic and didactic tool in caries prevention. Community Dent Oral Epidemiol 1986; 14: 156-10.
36. Ramesh K, Kunjappan S, Ramesh M, Shankar S and Reddy S: Comparative evaluation of predictive value of three caries activity tests-snyder, lactobacillus count and cariostat in mixed dentition children with and without caries. J Pharm Bioallied Sci 2013; 5: 63-8.
37. Alaluusua S, Nystrom M, Gronroos L and Peck L: Caries-related microbiological findings in a group of teenagers and their parents. Caries Res 1989; 23: 49-55.
38. Van-Houte J, Jordan HV, Laraway R, Kent R, Soparkar PM and Depaola PF: Association of the Microbial Flora of Dental Plaque and Saliva with Human Root-surface Caries. J Dent Res 1990; 69: 1463-68.
39. Leverett DH, Proskin HM, Featherstone JD, Adair SM, Eisenberg AD, Mundorff-Shrestha SA, Shields CP, Shaffer CL and Billings RJ: Caries risk assessment in a longitudinal discrimination study. JDR 1993; 72: 538-43.
40. Marchant S, Brailsford SR, Twomey AC, Roberts GJ and Beighton D: The predominant microflora of nursing caries lesions. Caries Res 2001; 35: 397-06.
41. Teanpaisan R, Thitasomakul S, Piwat S, Thearmontree A, Pithpornchaiyakul W and Chankanka O: Longitudinal study of the presence of mutans streptococci and lactobacilli in relation to dental caries development in 3-24 month old Thai children. Int Dent J 2007; 57: 445-51.
42. Byun R, Nadkarni MA, Chhour KL, Martin FE, Jacques NA and Hunter N: Quantitative analysis of diverse Lactobacillus species present in advanced dental caries. J Clin Microbiol 2004; 42: 3128-36.
43. Tanzer JM, Livingston J and Thompson AM: The microbiology of primary dental caries in humans. J Dent Educ 2001; 65: 1028-37.
44. Caufield PW, Li Y, Dasanayake A and Saxena D: Diversity of lactobacilli in the oral cavities of young women with dental caries. Caries Res 2007; 41: 2-8.
45. Yang R, Argimon S, Li Y, Gu H, Zhou X and Caufield PW: Determining the genetic diversity of lactobacilli from the oral cavity. J Microbiol Methods 2010; 82: 163-69.
46. Shimada A, Noda M, Matoba Y, Kumagai T, Kozai K and Sugiyama M: Oral lactic acid bacteria related to the occurrence and/or progression of dental caries in Japanese preschool children. Biosci Microbiota Food Health 2015; 34: 29-36.
47. Xu X, He J and Xue J: Oral cavity contains distinct niches with dynamic microbial communities. Environ Microbiol 2015; 17: 699-10.
48. Samaranayake L and Matsubara VH: Normal oral flora & the oral ecosystem. Den Cli North Am 2017; 61: 199-15.
49. Sakeenabi B and Hiremath SS: Dental caries experience and salivary *Streptococcus mutans*, lactobacilli scores, salivary flow rate, and salivary buffering capacity among 6-year-old Indian school children. J Int Soc Prev Community Dent 2011; 1: 45-51.
50. Hebbal MI, Ankola AV and Metgut MC: Association between socioeconomic status, salivary streptococcus mutans, Lactobacilli and dental caries among 12-year-old school children in belgaum City. World J Dent 2011; 2: 316-20.
51. Ravindran S, Huang CC and George A: Extracellular matrix of dental pulp stem cells: applications in pulp tissue engineering using somatic MSCs. Front Physiol 2014; 4: 395.
52. Ahirwar SS, Govind G, Singh V and Gupta MK: Screening, Isolation and Identification of Lactobacillus Species from Dental Caries of Children. Int J Curr Microbiol Appl Sci 2017; 6: 497-03.
53. Guo L: Salivary Biomarkers for Caries Risk Assessment. J Calif Dent Assoc 2013; 41: 107-18.
54. Kaplan JB: Biofilm dispersal: mechanisms, clinical implications, and potential therapeutic uses. J Dent Res 2010; 89: 205-18.
55. Takeshita T, Kageyama S, Furuta M, Tsuboi H, Takeuchi K, Shibata Y, Shimazaki Y, Akifusa S, Ninomiya T, Kiyohara Y and Yamashita Y: Bacterial diversity in saliva and oral health-related conditions: the Hisayama Study. Sci Rep 2016; 6: 22164.
56. Teanpaisan R and Dahlen G: Use of polymerase Chain Reaction techniques and sodium dodecyl sulphate-polyacrylamide gel electrophoresis for differenciation of oral Lactobacillus species. Oral Microbiol Immunol 2006; 21: 79-83.
57. Zhang Y, Liu Y, Ma Q, Song Y, Zhang Q and Wang X: identification of lactobacillus from the saliva of adult patients with caries using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. PLoS ONE 2014; 9: e106185.
58. Yadav K and Prakash S: Dental Caries: A Microbiological Approach. J Clin Infect Dis Pract 2017; 2: 118.
59. Ahirwar SS, Govind G, Singh V and Gupta MK: Comparative study of Lactobacillus species in dental caries active subject. Asian J Sci Tech 2017; 8: 5986-91. https://www.journalajst.com/comparative-study-lactobacillus-species-dental-caries-active-subjects
60. Román-Ménde C, Badet C, Yáñez A, Dominguez ML, Giono S, Richard B, Nancy J and Dorignac G: Identification of oral strains of Lactobacillus species isolated from Mexican and French children. J Dent Oral Hygiene 2009; 1: 9-16.
61. Anderson AC, Sanunu M, Schneider C, Clad A, Karygianni L, Hellwig E and Ahmad AA: Rapid species-level identification of vaginal and oral lactobacilli using MALDI-TOF MS analysis and 16S rDNA sequencing. BMC Microbiol 2014; 14: 312.
62. Wang Y, Zhang J and Chen X: Profiling of oral microbiota in early childhood caries using single-molecule real-time sequencing. Front Microbiol 2017; 8: 2244.
63. Ahumada MC, Bru E, Colloca ME, Lopez ME and Nader-Macias ME: Characterization of lactobacilli isolated from the tongue and gum. Anaerobe 1999; 5: 129-35.
64. Almas K, Al-Sanawi E and Al-Shahrani B: The effect of tongue scraper on mutans streptococci and lactobacilli in patients with caries and periodontal disease. Odontostomatol Trop 2005; 28: 5-10.
65. Ahumada MC, López ME, Colloca ME and NaderMacias ME: Lactobacilli isolation from dental plaque and saliva of a group of patients with caries and characterization of their surface properties. Anaerobe 2001; 7: 71-77.
66. Munson MA, Banerjee TF, Watson TF and Wade WG: Molecular analysis of the microflora associated with dental caries. J Clin Microbiol 2004; 42: 3023-29.
67. Kanasi E, Dewhirst FE, Chalmers NI, Kent R, Moore A, Hughes CV, Pradhan N, Loo CY and Tanner AC: Clonal

Analysis of the Microbiota of Severe Early Childhood Caries. Caries Res 2010; 44: 485-97.
68. Asakawa M, Takeshita T, Furuta M, Kageyama S, Takeuchi K, Hata J, Ninomiya T and Yamashita Y: Tongue microbiota and oral health status in community-dwelling elderly adults. mSphere 2018; 3: e00332-18.
69. Colloca ME, Ahumada MC, Lopez ME and Nader-Macıas ME: Surface properties of lactobacilli isolated from healthy subjects. Oral Dis 2000; 6: 227-33.
70. Gizani S, Papaioannou W, Haffajee AD, Kavvadia K, Quirynen M and Papagiannoulis L: Distribution of selected cariogenic bacteria in five different intra-oral habitats in young children. Int J Paediatr Dent 2009; 19: 193-00.
71. Rosan B and Lamont RJ: Dental plaque formation. Microbes Infect 2000; 2: 1599-07.
72. Corby PM, Lyons-Weiler J, Bretz WA, Aas JA, Boumenna T, Gross J, Corby AL, Junior HM, Wevant RJ and Paster BJ: Microbial risk indicators of early childhood caries. J Clin Microbiol 2005; 43: 5753-59.
73. Motisuki C, Lima LM, Spolidorio DM and Santos-Pinto L: Influence of sample type and collection method on Streptococcus mutans and Lactobacillus spp. counts in the oral cavity. Arch Oral Biol 2005; 50: 341-45.
74. Aas JA, Griffen AL, Dardis SR, Lee AM, Olsen I, Dewhirst FE, Leys EJ and Paster BJ: Bacteria of Dental Caries in Primary and Permanent Teeth in Children and Young Adults. J Clin Microbiol 2008; 46: 1407-17.
75. Gross EL, Leys EJ, Gasparovich SR, Firestone ND, Schwartzbaum JA and Janie's DA: Bacterial 16S sequence analysis of severe caries in young permanent teeth. J Clin Microbiol 2010; 48: 4121-28.
76. Tanner AC, Mathney JM, Kent RL, Chalmers NI, Hughes CV, Loo CY, Pradhan N, Kanasi E, Hwang J and Dahlan MA: Cultivable anaerobic microbiota of severe early childhood caries. J Clin Microbiol 2011; 49: 1464–74.
77. Mannaa A, Carlén A, Campus G and Lingström P: Supragingival plaque microbial analysis in reflection to caries experience. BMC Oral Health 2013; 13: 5.
78. Richards VP, Alvarez AJ, Luce AR, Bedenbaugh M, Mitchell ML, Burne RA and Nascimento MM: Microbiomes of sitespecific dental plaques from children with different caries status. Infect Immun 2017; 85: e00106-17.
79. Obata J, Takeshita T, Shibata Y, Yamanaka W and Unemori M: Identification of the Microbiota in Carious Dentin Lesions Using 16S rRNA Gene Sequencing. PLoS ONE 2014; 9: e103712.
80. Chhour KL, Nadkarni MA, Byun R, Martin FE, Jacques NA and Hunter N: Molecular analysis of microbial diversity in advanced caries. J Clin Microb 2005; 43: 843.
81. Jiang S, Gao X, Jin L and Lo EC: Salivary Microbiome Diversity in Caries-Free and Caries-Affected Children. Int J Mol Sci 2016; 17: 1978.
82. Mitrakul K, Chanvitan S and Jeamset A: Quantitative analysis of *S. mutan*, *Lactobacillus* and *Bifidobacterium* found in initial and mature plaques in Thai children with early childhood caries. Eur Arch Paediatr Dent 2017; 18: 251-61.
83. Ando N and Hoshino E: Predominant obligate anaerobes invading the deep layers of root canal dentin. Int Endod J 1990; 23: 20-27.
84. Becker MR, Paster BJ, Leys EJ, Moeschberger ML, Kenyon SG, Galvin JL, Boches SK, Dewhirst FE and Griffen AL: Molecular analysis of bacterial species associated with childhood caries. J Clin Microbiol 2002; 40: 1001-09.
85. Kianoush N, Adler CJ, Nguyen K-AT, Browne GV, Simonian M and Hunter N: Bacterial Profile of Dentine Caries and the Impact of pH on Bacterial Population Diversity. PLoS ONE 2014; 9: e92940.
86. Badet MC, Richard B and Dorignac G: An *in-vitro* study of the pH-lowering potential of salivary lactobacilli associated with dental caries. J Appl Microbiol 2001; 90: 1015-18.
87. De Vuyst L and Degeest B: Heteropolysaccharides from lactic acid bacteria. FEMS Microbiol Rev 1999; 23: 153-77.
88. Ahumada MC, Bru E, Colloca ME, López ME and Nader-Macías ME: Evaluation and comparison of lactobacilli characteristics in the mouths of patients with or without cavities. J Oral Sci 2003; 45: 1-9.
89. Schwendicke F. Dörfer C. Kneist S. Meyer-Lueckel H and Paris S: Cariogenic Effects of Probiotic Lactobacillus rhamnosus GG in a Dental Biofilm Model. Caries Res 2014; 48: 186-92.
90. Hemadi AS, Huang R, Zhou Y and Zou J: Salivary proteins and microbiota as biomarkers for early childhood caries risk assessment. Int J Oral Sci 2017; 9: e1.
91. Succi M, Tremonte P, Reale A, Sorrentino E, Grazia L, Pacifico S and Coppola R: Bile salt and acid tolerance of Lactobacillus rhamnosus strains isolated from Parmigiano Reggiano cheese. FEMS Microbiol Lett 2005; 244: 129-37
92. Senneby A, Davies JR, Svensäter G and Neilands J: Acid tolerance properties of dental biofilms *in-vivo*. BMC Microbiol 2017; 17: 165.
93. Hassanzadazar H, Ehsani Ali, Mardani K and Hesari J: Investigation of antibacterial, acid and bile tolerance properties of lactobacilli isolated from Koozeh cheese. Vet Res Forum 2012; 3: 181-85.
94. Van-Houte J, Lopman J and Kent R: The final pH of bacteria comprising the predominant flora on sound and carious human root and enamel surfaces. J Dent Res 1996; 75: 1008-14.
95. Bevilacqua A, Petruzzi L, Speranza B, Campaniello D, Sinigaglia M and Corbo MR: Changes of the cell surface hydrophobicity of Lactobacillus acidophilus La-5 in response to pH, temperature and inulin. Inter J Food Sci Technol 2017; 53: 1262-68.
96. Vadillo-Rodriguez V, Busscher HJ, van der Mei HC, de Vries J and Norde W: Role of Lactobacillus cell surface hydrophobicity as probed by AFM in adhesion to surfaces at low and high ionic strength. Colloids Surf B Biointerfaces 2005; 41: 33-41.
97. Chenicheri S, R U, Ramachandran R, Thomas V and Wood A: Insight into oral biofilm: primary, secondary and residual caries and phyto-challenged solutions. Open Dent J 2017; 11: 312-33.
98. Oleksy M and Klewicka E: Exopolysaccharides produced by Lactobacillus spp: Biosynthesis and applications. Crit Rev Food Sci Nutr 2018; 58: 450-62.
99. Mobili P, Gerbino E, Tymczyszyn EE and Gómez-Zavaglia A: S-layers in lactobacilli: structural characteristics and putative role in surface and probiotic properties of whole bacteria. In: Méndez-Vilas, A. (Ed.), Current Research Technology and Education Topics in Applied Microbiology and Microbial Biotechnology Formatex Research Center, Spain 2010: 1224-43.
100. Hynonen U and Palva A: Lactobacillus surface layer proteins: structure, function and applications. App Microbiol Biotechnol 2013; 97: 5225-43.
101. Yadav K and Prakash S: Dental Caries: A Review. Asian J Biomed Pharm Sci 2016; 6: 1-07.

102. Featherstone JD: Dental caries: a dynamic disease process. Aust Dent J 2008; 53: 286-91.
103. Donlan RM and Costerton JW: Biofilms: Survival Mechanisms of Clinically Relevant Microorganisms. Clin Microbiol Rev 2002; 15: 167-93.
104. Heller D, Helmerhorst EJ, Gower AC, Siqueira WL, Paster BJ and Oppenheim FG: Microbial diversity in the early in vivoformed dental biofilm. Appl Environ Microbiol 2016; 82: 1881-88.
105. Yu OY, Zhao IS, Mei ML, Lo EC and Chu CH: Dental biofilm and laboratory microbial culture models for cariology research. Dent J 2017; 5: e21.
106. Huang R, Li M and Gregory R: Bacterial interactions in dental biofilm. Virulence 2011; 2: 435-44.
107. Santos AL, Galdino AC, Mello TP, Ramos LS, Branquinha MH, Bolognese AM: Columbano Neto J and Roudbary M: What are the advantages of living in a community? A microbial biofilm perspective! Mem Inst Oswaldo Cruz 2018; 113: e180212.
108. Listgarten MA: The structure of dental plaque. Periodontology 2000 1994; 5: 52-65.
109. Scheie AA: Mechanisms of dental plaque formation. Adv Dent Res 1994; 8: 246-53.
110. Marsh PD: Dental plaque as a biofilm and a microbial community – implications for health and disease. BMC Oral health 2006; 6: 429-53.
111. Nogueria RD, Silva CB, Lepri CP, Palma-Dibb RG and Geraldo-Martins VR: Evaluation of Surface Roughness and Bacterial Adhesion on Tooth Enamel Irradiated With High Intensity Lasers. Braz Dent J 2017; 28: 24-29.
112. Nishikawara F, Katsumura S, Ando A, Tamki Y, Nakamura Y, Sato K, Nomura Y and Hanada N: Correlation of cariogenic bacteria and dental caries in adults. J Oral Sci 2006; 48: 157-64.
113. Hojo K, Nagaoka S, Ohshima T and Maeda N: Bacterial interactions in dental biofilm development. J Dent Res 2009; 88: 982-90.
114. Kolenbrander P, Palmer R Jr, Periasamy S and Jakubovics N: Oral multispecies biofilm development and the key role of cell-cell distance. Nat Rev Microbiol 2010; 8: 471–80
115. Wolff MS and Larson C: The cariogenic dental biofilm: good, bad or just something to control? Braz Oral Res 2009; 23: 31-38.
116. Sicca C, Bobbio E, Quartuccio N, Giovanni N and Cistaro A: Prevention of dental caries: A review of effective treatments. J Clin Exp Dent 2016; 8: e604-e610.
117. Peedikayil FC: Antibiotics: Use and misuse in pediatric Dentistry. J Indian Soc Pedod Prev Dent 2011; 29: 282-87
118. Lee Y: Diagnosis and Prevention Strategies for Dental Caries. J Lifestyle Med 2013; 3: 107-09.
119. Gizani S, Papaioannou W, Haffajee AD, Kavvadia K, Quirynen M and Papagiannoulis L: Distribution of selected cariogenic bacteria in five different intra-oral habitats in young children. Int J Paediatr Dent 2009; 19: 193-00.
120. Chokshi A, Mahesh P, Sharada P, Chokshi K, Anupriya S and Ashwini BK: A correlative study of the levels of salivary *Streptococcus mutans, lactobacilli* and *Actinomyces* with dental caries experience in subjects with mixed and permanent dentition. J Oral Maxillofac Pathol 2016; 20: 25-28.
121. Bevilacqua A, Petruzzi L, Speranza B, Campaniello D, Sinigaglia M and Corbo MR: Changes of the cell surface hydrophobicity of Lactobacillus acidophilus La-5 in response to pH, temperature and inulin. Inter J Food Sci Technol 2017; 53: 1262-68.

How to cite this article:
Ahirwar SS, Gupta MK and Snehi SK: Dental caries and lactobacillus: role and ecology in the oral cavity. Int J Pharm Sci & Res 2019; 10(11): 4818-29. doi: 10.13040/IJPSR.0975-8232.10(11).4818-29.