

Influence of preterm birth for child's oral health status

Sandra Žemgulytė, Ingrida Vasiliauskienė*, Eglė Slabšinskienė*, Kristina Saldūnaitė*,
Julija Narbutaitė**

SUMMARY

Objectives. To search the publications related to this topic, subsequently to find out a prevalence of prematurity worldwide and classification of preterm birth, to analyze the both maternal and foetal risk factors related to preterm birth, to depict relationship between prematurity and developmental defects of enamel and development of dental caries.

Material and methods. A review was carried out to identify relevant studies reporting data on risk factors of prematurity, low birth weight and its' relationship between developmental defects of enamel and early childhood caries. The data was extracted from the selected papers.

Results. A total 75 of articles were identified after initial search and 48 publications were enrolled in this review.

Conclusion. Prematurity and low birth weight of child can cause both general and oral health complications and altered infant's development or growth.

Key words: prematurity, low birth weight, developmental defects of enamel, early childhood caries.

INTRODUCTION

The rate of preterm birth ranges from 5% to 18% of new-born babies according to the World Health Organization (WHO) data across 184 countries (1), meanwhile the higher rate of premature birth occurs in the populations with low socioeconomic status. According to the Statistics Lithuania the prevalence of preterm birth of Lithuania was 5.6% in 2017 (2). Medical and technological advances in prenatal and neonatal intensive care have reduced mortality of preterm infants, but those children suffer more severe complications (3, 4). Preterm delivery exposes the foetus to extrauterine life when newborn is poorly equipped, and this exposure may be harmful to the developing brain (5). Furthermore, preterm and low birth weight children are at increased risk of cerebral palsy, seizure disorders, severe mental retardation, psychosocial and behavioral disorders, hearing loss, visual impairment and lower respiratory tract infections (4, 6). The risk of developing disabilities increases with decreasing gestational age and birth weight (7), besides oral abnormalities, such as palatal defects caused by

oral intubation and long-term mechanical ventilation, developmental defects of enamel, reduced dental dimensions, delayed eruption of primary and permanent dentition are reported more frequently among prematurely born children than full-term born infants (8-11).

The aim of this review was to assess the influence of preterm birth for child's oral health status.

MATERIAL AND METHODS

Collection of data, article selection strategy

The search was performed in PubMed, Medline and Wiley online library databases and was limited to human studies and publications written in English. The comprehensive search covered the period from 1994 to November 2014. One investigator carried out the selection and evaluation of articles.

The inclusion criteria covered the follow-up studies, case control studies, cross-sectional surveys and systematic review. The search was conducted using keywords or combinations of the following: prematurity, low birth weight, developmental defects of enamel, early childhood caries.

Data extraction

Full texts of selected publications were analyzed and the following data was extracted: date of

*Clinic for Preventive and Pediatric Dentistry, Lithuanian University of Health Sciences, Kaunas, Lithuania

Address correspondence to Sandra Žemgulytė, Clinic for Preventive and Pediatric Dentistry, Lithuanian University of Health Sciences, Luksos-Daumanto 6, LT-50106 Kaunas, Lithuania.
E-mail: zemgulyte.sandra@gmail.com

publication, authors, setting of the study, type of the study, measured outcomes (etiology, risk factors, and clinical status).

RESULTS

Overall, 75 of articles were identified after initial search and 48 publications were enrolled in this review.

DISCUSSION

Etiology of preterm birth

The etiology of premature birth is multifactorial and may be related to both maternal and foetal disease (12, 13). Maternal risk factors include obesity, smoking, liver disease, drug and alcohol use, medication consumption, maternal malnutrition, pre-eclampsia, placental abruption, chorioamnionitis, multiple births, intrauterine growth restriction, congenital malformations, cervical colonization, unspecified bleeding, maternal age (under 17 and elder than 34) and may result in intrauterine growth retardation and infant being small for gestational age (5, 6, 10, 14, 15).

Birth weight of infant is classified considering the criteria of the World Health Organization: extremely low birth weight (<1000g), very low birth weight (<1500g), low birth weight (<2500g), normal birth weight (equal to or >2500g) (1). Meanwhile, prematurity can be based on gestational age: preterm infants are born in the 33- 36th gestational week, very preterm newborn are born in the 29- 32nd gestational week, and extremely preterm infants are born before the 29th gestational week (10).

Apgar score reflects the neonate conditions immediately after birth, when the respiratory effort, reflex irritability, muscle tone, heart rate and colour are evaluating at 1 minute and 5 minutes after delivery. The results of Apgar score reflect neonatal respiratory distress and need for orotracheal intubation (16). A low Apgar score can be related to enamel hypoplasia, compromised immune development, learning, cognitive and behavioural problems (16, 17).

Development of teeth

Developmental defects of enamel are defined as disturbances in hard tissue matrices and mineralization during odontogenesis process and are classified as either enamel hypoplasia or enamel opacities (demarcated or diffused), based on clinical appearance (18, 19). The amelogenesis of primary teeth starts in the 15th gestational week

and continues until 12 month after birth (second primary molar) (12, 20). Consequently, pre- and post- natal stresses adversely affect ameloblasts and odontoblasts during tooth formation and can result in both types hypoplastic and hypomineralized enamel (10). If alterations develop during the organic matrix formation or secretory stage, the possible clinical appearance is enamel hypoplasia. In addition, when the enamel developing process disturbs later, during the maturation period or enamel calcification, the result will be a hypocalcification and clinical expression is white, yellow or brown opaque or decalcified areas in color, surrounded by enamel of normal appearance (21, 22). Enamel hypoplasia is a quantitative defect, whereas enamel hypomineralization or opacities is a qualitative defect (23). The most common evidence of perinatal stress affecting tooth development is found in virtually every child and sub-clinical appearance is a neonatal line (NNL) (10). The major accumulation of calcium and phosphate occurs during the last semester of pregnancy also the altered calcium homeostasis during perinatal period may interfere the mineralization of enamel in primary teeth (23, 24). Complications of birth, periods of malnutrition, morbidity of infectious diseases also leave their mark on the developing teeth. The various insults occurred during stages of teeth formation can leave mark more than once on teeth surfaces (10), because the dental tissue does not remodel like bone and defects, which will occur during metabolic disturbances even the primary disturbances will not be corrected (8). If the ameloblasts will be damaged during the initial or final maturation phase and the cells will be able to recover and continue their normal function, as a result the demarcated defects will occur (23). Furthermore, preterm children have thinner enamel formed prenatally than full-term children (11).

The secretory phase of permanent incisors and first permanent molars begins in utero and the maturation phase starts after birth (3). Demarcated opacities in these teeth can be observed in very low birth-weight children (9). Results of several studies showed that metabolic derangements, infections and respiratory diseases can affect enamel development between 0 and 2 years of child age (18, 25).

Both types of defects can develop on the same tooth surface separated by normal enamel or adjacent to each other (26). The maxillary teeth are more frequent affected than lower ones (22). These defects can be generalized or localized (18). The generalized defects are caused by systemic illnesses associated with prematurity.

Association between prematurity and developmental defects of enamel (DDE)

The prevalence of DDE ranges from 24.4% to 96% (27, 28).

Considering relationship premature birth and DDE, several studies showed controversial results. Findings of some studies showed, that here was significant relationship between low birth weight and enamel defects of primary dentition in children born preterm (14, 18, 29-31), while results of another study didn't show significant association between premature birth and DDE (18).

Systemic risk factors of developmental defects of enamel

Prematurity and low birth weight is closely related to long-term illness; perinatal infections and the poor growth and developmental delays of children (32, 33). The most common complications are metabolic (such as hyperbilirubinemia), respiratory (perinatal asphyxia, hyaline membrane disease, bronchopulmonary dysplasia, pneumonia), cardiovascular (congestive cardiac failure, patent ductus arteriosus), neurological and nutritional (such as vitamin D, calcium and iron) deficiencies, gastrointestinal problems (gut intolerance, feeding difficulties, necrotizing enterocolitis) (18, 23).

Prematurity is a risk factor of *osteopenia* because 80% of mineral accretion occurs in the 3rd semester of pregnancy (33). Breast milk contain inadequate supply of calcium and phosphorus to enable intrauterine mineral accretion in preterm neonates and metabolic bone disease of prematurity can occur in up 23% of very low birth weight newborns and in 55% of extremely low birthweight ones (34, 35). The immaturity of kidney and liver and inadequate gastrointestinal absorption have influence for low supply of calcium and phosphate as well (36).

Deficiency of vitamin D can affect immune system in the intestine, because that an adequate vitamin D status is needed for the synthesis of antimicrobial peptide cathelicidin (37). Clinical ricket is diagnosed for extremely preterm neonates due to severely impaired calcium and phosphate metabolism (9). In addition, bone accretion is higher, when isocaloric protein- and mineral-enriched postdischarge formula were preferred than a standard term formula or human milk for preterm infants (37).

Iron deficiency is common form of nutritional deficiency in the world. Furthermore, there is the unable maintenance of normal physiological function of tissues for iron deficient child. Schroth et al. revealed that haemoglobin level was significantly lower for children with S-ECC than in caries free

control group (38). Controversially, L. Aine et al. did not find difference in prevalence of enamel defects in both dentition between the preterm children who had feeded breast milk unsupplemented or supplemented with minerals and vitamin D (6).

Finally, maternal habits play a role in developmental defects of enamel formation. It is found a statistically significant association between tobacco use during pregnancy and the prevalence of enamel defects (14).

Local risk factors of developmental defects of enamel

The main reasons of localized dental defects can be laryngoscopy and orotracheal intubation those are required to overcome respiratory distress (39). Palatal grooving and asymmetry, high arched palate and dental crossbite can develop due to the presence of the tracheal tube on the palate (12). Furthermore, there is the lack of protection of bone for primary teeth germs at birth, so the pressure exerted during intubation can cause alterations in dental germs, changes in the path of eruption of teeth. Subsequently, longer duration of intubation can cause greater chance of enamel defects and palatal deformities. The upper left incisor, left maxillary lateral incisor and upper right central incisor of primary dentition are mainly affected by hypoplasia due to the side of the laryngoscope position during orotracheal tube insertion (10, 12, 21, 27, 40). This local trauma to the primary teeth can cause the enamel defects in the underlying permanent teeth (8). The prevalence of DDE was higher among premature infants intubated than not intubated. Subsequently, duration of intubation was related with to the probability of development of DDE (27).

Relationship between prematurity and dental caries

The risk factors of dental caries can be classified with respect to period of tooth development as pre-natal, perinatal and postnatal.

A positive relationship between low birth weight and early childhood caries might be attributed to a complex set of pathways, including biological and environmental factors, such enamel hypoplasia, poor nutrition, eating habits, oral hygiene and socio-economic status (14). Early childhood caries is defined by presence of primary teeth caries for children under 6 years of age (10). The term of Severe Early Childhood Caries is used when dental caries is present in any smooth surface for children under 3 year old and in one or more smooth surfaces of primary maxillary anterior teeth for 3-5 year old

children (28, 41). Several researches proposed the new classification of severe early childhood caries related with these risk factors: hypoplasia- associated severe early childhood caries (HAS-ECC) (10).

Visible plaque is more common among children with low birth weight compared to children with normal birthweight (30, 39). Consequently, the prevalence of dental caries is statistically significantly higher among preterm low birthweight children group than among full-term normal birthweight children (26, 30, 39, 42), while another study didn't find association between preterm birth and the prevalence of dental caries (43).

Relationship between DDE and dental caries

Teeth with DDE are more susceptible to caries, insofar as these teeth have retentive areas that facilitate the adhesion and colonisation of cariogenic bacteria, enamel is less mineralized, more porous and acid-soluble (25, 29, 32, 44). Some authors show that strong association exists between enamel hypoplasia and dental caries (41), meanwhile another study reveals that early childhood caries was significantly associated with DDE, especially with diffuse and demarcated opacity, but was not associated with hypoplasia (28).

Several studies investigated the histomorphology in primary teeth. The polarized light microscopy (POLMI) and scanning electron microscope (SEM) were used to examine enamel maturation, subsurface lesions (SSLs). Hypomineralized areas of enamel have an increased porosity with more than 5% pore volume, a less regular orientation of prisms and a difference in the overall appearance of the prisms (22). Preterm children have reduced dental dimensions or reduction of enamel thickness (11). Under SEM examination, a half of preterm incisors without visibly detectable defects can demonstrate surface enamel hypoplasia at the SEM level (11).

Relationship between bacteria and development of dental caries

Several studies investigated if the mode of delivery has the influence for the infant's oral microbiota and development of ECC. Colonization among vaginally born children is higher with mutans streptococcus than C- section born children, the ECC is more prevalent in vaginally born children group (45). The results of other study showed that prevalence of lactobacilli species among vaginally delivered infants was statistically significantly higher. The colonization with *Enterococcus faecalis* was more often among infants delivered by C-section, whereas the oral colonization of both groups was observed without differences (46).

Results of several studies showed that mothers and children demonstrated identical bacteriocin typing pattern of Mutans streptococci. Vertical transmission can be proven out utilized chromosomal DNA patterns or identical plasmids (47). The habitual kissing or pretesting of child food can increase exposure of bacterial transfer (48). The study performed in southern China showed that enamel hypoplasia and low birth weight was significantly associated with *S. mutans* colonization in children (49).

Association between dental caries and behavior factors

Maternal weight, intake of carbohydrates and fats are associated with children's dietary intake and caries experience. Results of several studies showed that prevalence of dental caries in children whose mothers consumed more sugars was 1.5 times higher, and maternal obesity is related with child obesity (49).

Preterm children have more dental behavioral problems causing stress compared with full-born children because of previous medical treatment and repeated painful procedures related with prematurity (37). The increase rate of hyperactivity, difficulties in concentration, below-grade-level performance at school can be observed for these children (10). Teeth with DDE are more sensitive, less esthetic and clinical treatment becomes more complicated because of difficulties to anesthetize these teeth (24).

Preterm children have significantly more dental behavior management problems than full term born children (7).

Association between cerebral palsy and dental caries

Cerebral palsy is common developmental disorder of preterm infants. The prevalence of cerebral palsy increases with more severe prematurity and lower weight at birth and ranges from 8.5% for the extremely preterm to 0.4% for late preterm (4, 5). Several studies showed that prevalence of caries is higher among children with cerebral palsy than other children. The maintaining of adequate oral hygiene can be complicated because of patient's visual, hearing, motor and cognitive impairments (50, 51). Abnormal movements of the tongue and facial muscles, reduced salivary flow can increase the risk of caries development as well (51).

CONCLUSION

Prematurity and low birth weight of infant can cause various both general and oral complications,

besides altered development or growth. Therefore, it is very important to pay attention to supervision of these newborn to prevent or relieve perinatal infections and developmental defects of enamel, dental caries and possible complications.

STATEMENT OF CONFLICTS OF INTEREST

The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

REFERENCES

- World Health Organization (Cited 19 February 2018). Available from: <https://www.who.int/news-room/fact-sheets/detail/preterm-birth>
- Medical data of Births, 2017. Vilnius, 2018 p.7. (http://hi.lt/uploads/pdf/leidiniai/Statistikos/Gimimu/gimimai_2017.pdf)
- Nelson S, Albert JM, Lombardi G, Wishnek S, Asaad G, Kirchner HL, et al. Dental caries and enamel defects in very low birth weight adolescents. *Caries Res* 2010;44:509-518.
- Soeimani F, Zaheri F, Abdi F. Long-term neurodevelopmental outcomes after preterm birth. *Iranian Red Crescent Medical J*. 2014; 16(6): e17965.
- Tronnes H, Wilcox AJ, Lie RT, Marketsad T, Moster D. Risk of cerebral palsy in relation to pregnancy disorders and preterm birth: a national cohort study. *Dev Med Child Neurol* 2014; 56(8): 779-785.
- Burt BA, Pai S. Does Low Birthweight Increase the Risk of Caries? A systematic Review. *J Dental Educ*,2001;65(10): 1024-1027.
- Brogardh-Roth S, Stjernqvist&Lars Matsson K. Dental behavioural management problems and dental caries prevalence in 3- to 6-year-old Swedish children born preterm. *Int J Pediatr Dent* 2008; 18(5): 341-347.
- Aine L, Backstrom MC, Maki R, Kuusela AL, Koivisto AM, Ikonen RS, et al. Enamel defects in primary and permanent teeth of children born prematurely. *J Oral Pathol Med* 2000; 29:403-9.
- Jacobsen PE, Haubek D, Heriksen TB, Ostergaard JR, Poulsen S. Developmental enamel defects in children born preterm: a systematic review. *Eur J Oral Sci* 2014, 122:7-14.
- Caufield PW, Li Y, Bromage TG. Hypoplasia-associated Severe Early Childhood Caries- A Proposed Definition. *J Dent Res* 2012; 91(6):544-550.
- Seow WK, Young WG, Tsang AKL, Daley T. A study of primary dental enamel from preterm and full-term children using light and scanning electron microscopy. *Pediatr Dent* 2005; 27(5):374-379.
- Paulsson L, Bondemark L, Sodefheldt B. A systematic review of the consequences of premature birth on palatal morphology, dental occlusion, tooth-crown dimensions, and tooth maturity and eruption. *Angle Orthod*. 2004; 74(2):269-279.
- Al- Sayagh G, Qasim AA, Al- Rawi BA. The effect of Premature Birth on the Primary Dentition. *Al-Rafidain Dent J* 2008; 8(1):18-22.
- Vello MA, Martinez-Costa C, Catala M, Fons J, Brines J, Guijarro-Martinez R. Prenatal and neonatal risk factors for the development of enamel defects in low birth weight children. *Oral Dis* 2010; 16: 257-262.
- Bogges KA, Edelstein BL. Oral health in women during precenception and pregnancy: implications and for birth outcomes and infant oral health. *Matern Child Health J* 2006; 10:169-174.16.
- Salustiano EMA, Campos JADB, Ibiñ SM, Ruando R, Zugaib M. Low Apgar scores at 5 minutes in a low risk population: maternal and obstetrical factors and postnatal outcome. *Rev Assoc Med Bras* 2012; 58(5): 587-593.
- Sanders AE, Slade GD. Apgar score and dental caries risk in the primary dentition of five year olds. *Austral Dent J* 2010; 55: 260-267.
- Correa-Faria P, Martins-Junior PA, Vieira-Andrade RG, Oliveira- Ferreira F, Marques LS & Ramos-Jorge ML. Developmental defects of enamel in primary teeth: prevalence and associated factors. *Int J Pediatr Dent* 2013; 23:173-179.18.
- Ruiz LA, Maya RR, Perlatt D'Alpino PH, Atta MT, Svizero NR. Prevalence of enamel defects in permanent teeth of patients with complete cleft lip and palate. *Cleft Palate Craniofac J* 2013; 50(4):394-9.
- Masumo R, Bardsen A, Astrom AN. Developmental defects of enamel in primary teeth and as-sociation with early life of course events: a study of 6-36 month old children in Manyara, Tan-zania. *BMC Oral Health* 2013, 13:21.
- Ferrini FRO, Marba STM, Gavião MBD. Oral conditions in very low and extremely low birth weight children. *J Dent Child* 2008; 75(3):235-42.
- Rythen M, Noren JG, Sabel N, Steiniger F, Niklasson A, Ann Hellstrom A et al. Morphological aspects of dental hard tissues in primary teeth from preterm infants. *Int J Pediatr Dent* 2008; 18:397-406.
- Nelson S, Albert JM, Geng C, Curtan S, Lang K, Miadich S. Increased enamel hypoplasia and very low birthweight infants. *J Dent Res* 2013; 92(9):788-794.
- Robles MJ, Ruiz M, Bravo-Perez M, Gonzalez E, Maria-Angustias Penalver. Prevalence of enamel defects in primary and permanent teeth in a group of schoolchildren from Granada (Spain). *Med Oral Patol Oral Cir Bucal*. 2013; 1;18(2):187-193.
- Faria PC, Martins-Junior PA, Vieira-Andrade RG, Marques LS, Ramos- Jorge ML. Perinatal factors associated with developmental defects of enamel in primary teeth: a case control study. *Bras Oral Res* 2013; 27(4):363-8.
- Rajshekar SA, Laxminarayan N. Comparison of primary dentition caries experience in pre-term low birth-weight and full-term birth-weight children aged one to six years. *J Indian Soc Pedo-dont Prev Dent* 2011; 2(29):128-134.
- Melo NSFO, Vieira Cavalcante da Silva RPG, Lima AAS. The neonatal intubation causes de-fects in primary teeth of premature infants. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2014; 158(4):605-12.
- Faria PC, Paixao-Goncalves S, Paiva SM, Pordeus IA, Marques LS, Ramos-Jorge ML. Association between developmental defects of enamel and early childhood caries: a cross-sectional study. *Int J Pediatr Dent* 2014; 25(2):103-9.
- Cruvnel VRN, Gravina DBL, Azevedo TDPL, Siqueira de Rezende C, Bezerra ACB, Toledo OA. Prevalence of enamel defects and associated risk factors in both dentitions in preterm and full term born children. *J Appl Oral Sci* 2012; 20(3):310-7.

30. Schüler IM, Haberstroh S, Dawczynski K, Lehmann T, Heinrich-Weltzien R. Dental Caries and Developmental Defects of Enamel in the Primary Dentition of Preterm Infants: Case-Control Observational Study. *Caries Res.* 2018;52(1-2):22-31.
31. Wagner Y. Developmental defects of enamel in primary teeth - findings of a regional German birth cohort study. *BMC Oral Health.* 2016 ;17(1):10.
32. Elias dos Santos Junior V, Brasileiro de Sousa RM, Oliveira MC, Franca de Caldas Junior A, Rosenblatt A. Early childhood caries and its relationship with perinatal, socioeconomic and nutritional risk: a cross-sectional study. *BMC Oral Health* 2014, 14:47.
33. Dokos C, Tsakalidis C, Tragiannidis A, Rallis D. Inside the "fragile" infant: pathophysiology, molecular background, risk factors and investigation of neonatal osteopenia. *Clinical Cases in Mineral and Bone Metabolism* 2013; 10(2): 86-100.
34. Rythen M, Niklasson A, Hellstrom A, Hakeberg M, Robertson A. Risk indicators for poor oral health in adolescents born extremely preterm. *Swed Dent* 2012; 36(3):115-124.
35. Figueras-Aloy J, Alvarez-Dominguez E, Perez-Fernandez JM, Moretones-Sunol G, Vidal-Sicart S, Botet-Mussons F. Metabolic bone diseases and bone mineral density in very preterm infants. *J Pediatr* 2014; 164(3):499-504.
36. Seow WK. A study of the development of the permanent dentition in very low birthweight children. *Pediatr Dent* 1996; 18(5):379-384.
37. Lagemaat M, Rotteveel J, Schaafsma A, Weissenbruch MM, Lafeber HN. Higher vitamin D intake in preterm infants fed an isocaloric, protein- and mineral-enriched postdischarge formula is associated with increased bone accretion. *J. Nutr* 2013; 143:1439-1444.
38. Schroth RJ, Levi J, Kliewer E, Friel J, Moffatt MEK. Association between iron status, iron deficiency anaemia, and severe early childhood caries: a case-control study. *BMC Pediatr* 2013, 13:22.
39. Masumo R, Birungi N, Asgeir Bardsen A, Fadnes LT, Astrom AN. Impact of low birthweight on early childhood caries in 6-36 month old infants in Uganda: A cross-sectional study. *Acta Odont Scand* 2014; 72: 312-320.
40. Cortines AAO, Corrêa-Faria P, Paulsson L, Costa PS, Costa LR. Developmental defects of enamel in the deciduous incisors of infants born preterm: Prospective cohort. *Oral Dis.* 2019;25(2):543-549.
41. Pascoe L, Seow WK. Enamel hypoplasia and dental caries in Australian Aboriginal children: prevalence and correlation between the two diseases. *Pediatr Dent* 1994; 16(3):193-9.
42. Alshehhi A, Al Halabi M, Hussein I, Salami A, Hassan A, Kowash M. Enamel defects and caries prevalence in preterm children aged 5-10 years in Dubai. *Libyan J Med.* 2020;15(1):1705633.
43. Tanaka K, Miyake Y. Low birth weight, preterm or small-for-gestational-age are not associated with dental caries in young Japanese children. *BMC Oral Health* 2014, 14:38.
44. Targino AGR, Rosenblatt A, Oliveira AF, Chaves AMB, Santos VE. The relationship of enamel defects and caries: a cohort study. *Oral Dis* 2011; 17:420-426.
45. Pattanaporn K, Saraithong P, Khongkhuntian S, Aleksejuniene J, Laohapensang P, Chhun N et al. Mode of delivery, mutans streptococci colonization, and early childhood caries in three-to five-year-old Thai children. *Community Dent Oral Epidemiol* 2013; 41; 212-223.
46. Barfod MN, Magnusson K, Lexner MO, Blomqvist S, Dahlen G, Twetman S. Oral microflora in infants delivered vaginally and by caesarean section. *Int J Paediatr Dent* 2011; 21:401- 406.
47. Colak H, Dulgergil CT, Dalli M, Hamidi MM. Early childhood caries update: A review of causes, diagnoses, and treatments. *J Nat Sci Biol Med.* 2013; 4(1): 29-38.
48. Leong PM, Gussy MG, Barrow SYL, Sanigorski AS, Waters E. A systematic review of risk factors during first year of life for early childhood caries. *Int J Paediatr Dent* 2013; 23:235-250.
49. Zhou Y, Yang JY, Zhi QH, Tao Y, Qiu RM, Lin HC. Factors associated with colonization of *Streptococcus mutans* in 8- to 32-month-old children: a cohort study. *Aust Dent J* 2013; 58: 507-513.
50. Roberto LL, Machado MG, Resende VLS, Castilho LS, Nogueira MH, Abreu G. Factors associated with dental caries in primary dentition of children with cerebral palsy. *Bra Oral Res.* 2012; 26(5):471-7.
51. Liu Z, Yu D, Luo W, Yang J, Lu J, Gao S et al. Impact of oral health behaviours on dental caries in children with intellectual disabilities in Guangzhou, China. *Int J Environ Res Public Health* 2014; 11(10):11015-11027.

Received: 16 11 2016

Accepted for publishing: 24 12 2019