# Is Household Smoking a Risk factor for Caries? A Case-Control Study

(Adakah Asap Rokok di Rumah Faktor Risiko untuk Karies? Suatu Kajian Kes

A.M. ZURINA, M. ASMA'\*, R.J. RAJA LATIFAH & Y. NORIAH

#### ABSTRACT

Recent studies suggested that exposure to household smoking (HHS) could be a modifiable risk factor for caries development among children. Majority of the studies were cross sectional in nature. Therefore, a case-control study was designed to test the hypothesis that HHS is a risk factor to caries experience in permanent teeth. Calculation of sample size was based on the ratio of 1 case to 4 controls. Case was defined as a child aged 13-14 years old with caries in at least one second permanent molar and control was defined as a child from the same age and school with no caries second permanent molars. Matching was done for gender and ethnicity. School dental records provided information on oral health status and oral hygiene status. Information on HHS, socio-economic status, child's smoking status and child's oral health practices were obtained from a self administered questionnaire, completed by the children and their parents. The result showed that 55.9% of the case group was exposed to HHS, as compared to 44.1% among the control group. In the final multiple logistic regression model after controlling for important risk factors for caries, children with caries were almost twice as likely to have been exposed to HHS for more than 10 years as compared to children with no caries, (Adjusted OR=1.90 and 95% CI=1.35, 2.60). In addition, children who only received dental care from the school dental service had reduced risk of having dental caries by more than one third (36%) as compared with those who received dental care from school dental service (SDS) as well as had additional dental problem solving visit outside SDS (Adjusted OR=0.64 and 95% CI=0.50, 0.90). It is concluded that exposure to HHS for a long duration (> than 10 years) increase the risk to have caries experience in permanent teeth of children.

Keywords: Children; dental caries; household smoking; risk factor

# ABSTRAK

Kajian terkini menunjukkan bahawa pendedahan kepada asap rokok di rumah (HHS) boleh menjadi faktor risiko yang boleh diubah untuk pembentukan karies dalam kalangan kanak-kanak. Kebanyakan kajian adalah kajian keratan rentas. Oleh itu, satu kajian kes-kawalan telah dirancang untuk menguji hipotesis bahawa HHS adalah faktor risiko kepada karies pada gigi kekal. Pengiraan saiz sampel adalah berdasarkan kepada nisbah satu kes kepada empat kawalan. Kes ditakrifkan sebagai seorang kanak-kanak berumur 13-14 tahun dengan kerosakan gigi di sekurang-kurangnya satu gigi geraham kekal kedua dan kawalan pula ditakrifkan sebagai kanak-kanak yang sama umur (dan dari sekolah yang sama) tanpa karies gigi geraham kekal kedua. Padanan telah dilakukan untuk jantina dan keturunan. Maklumat mengenai status kesihatan mulut dan status kebersihan mulut diambil daripada rekod pergigian sekolah. Maklumat HHS, status sosio-ekonomi, status merokok kanak-kanak dan amalan kesihatan mulut kanak-kanak diperoleh menggunakan borang soal selidik yang diisi oleh anak-anak dan ibu bapa mereka. Hasilnya menunjukkan bahawa 55.9% daripada kes terdedah kepada HHS, berbanding dengan 44.1% dalam kalangan kumpulan kawalan. Dalam model regresi logistik berganda akhir selepas mengawal faktor-faktor risiko yang penting untuk karies, kanak-kanak dalam kumpulan kes hampir dua kali ganda berkemungkinan telah terdedah kepada HHS selama lebih daripada 10 tahun berbanding dengan kanak-kanak tanpa karies, (Adjusted OR=1.90 dan 95% CI=1.35, 2.60). Di samping itu, kanak-kanak yang hanya menerima rawatan pergigian daripada perkhidmatan pergigian sekolah mengurangkan risiko mempunyai karies gigi lebih daripada satu pertiga (36%) berbanding dengan mereka yang menerima rawatan pergigian daripada perkhidmatan pergigian sekolah (SDS) dan rawatan tambahan diluar SDS untuk menyelesaikan masalah pergigian (Adjusted OR=0.64 dan 95% CI=0.50, 0.90). Kesimpulannya, pendedahan kepada HHS untuk tempoh yang panjang (> 10 tahun) meningkatkan risiko untuk mendapat karies gigi kekal dalam kalangan kanak-kanak.

Kata kunci: Asap rokok di rumah; faktor risiko; kanak-kanak; karies gigi

### INTRODUCTION

Dental caries is considered a public health problem because of its widespread characteristic and its significant effects on the general health of children. Although caries is not a life threatening disease, untreated caries among children can cause toothache and this has a profound effect on their general health and adversely affects their quality of life Sheiham (2006). Nomura et al. (2004)

reported that, dental caries is one of the major pathological causes of dental pain among children. Jaafar and Razak (2002) also reported the primary cause of oro-facial pain among Malay school children was dental caries. Based on the findings of the Malaysian National Oral Health Survey of Adults (NOHSA) 2000, disruption in sleep is the most common factor arising from dental pain and the mean number of school days disrupted was 0.9 per year (Oral Health Division 2004).

Realising the importance of good oral health status, the Ministry of Health Malaysia conducted numerous oral health promotion activities targeting the established risk factors for caries. However, these have not been highly successful, even though there was a decline in the prevalence of dental caries in the last few decades. The complex interplay of multi-factorial aetiology in caries development is the reason for the less than satisfactory outcome from these preventive efforts. There may be other associated direct or indirect factors that are yet to be identified or proven which could explain caries formation in children.

Recently, the role of tobacco smoke and the exposure to HHS in the aetiology of caries among children have been explored. Available evidences suggest that the exposure to HHS could be a modifiable risk indicator for caries in deciduous and permanent teeth among children. These cross sectional studies reported that children who were exposed to HHS have higher chances of having caries in deciduous and permanent teeth as compared with those who were not exposed (Aligne et al. 2003; Ayo-Yusuf et al. 2007; Leroy et al. 2008; Shenkin et al. 2004; Tanaka et al. 2010; Williams et al. 2000). However, for permanent teeth, the results have been inconsistent in associating caries to HHS. A study among the Japanese children, failed to substantiate an association between HHS and dental caries in permanent teeth (Tanaka et al. 2006). However, more recent studies (Ayo-Yusuf et al. 2007; Tanaka et al. 2010) involving larger samples reported significant association between HHS and carious permanent teeth. The findings by Ayo-Yusuf et al. (2007) imply a post natal effect of HHS in the development of caries in permanent teeth because of the association between HHS with carious second permanent molar. This is so because the formation of the second permanent molar begins about 10 months after birth (Ten Cate et al. 2008).

The inconsistencies in the findings of the above mentioned studies create the need to conduct a study which can produce a higher level of evidence than the available studies. Therefore, this study was designed to test the hypothesis that HHS is a risk factor to have caries experience in permanent teeth in the presence of other risk factors such as sugar diet, oral hygiene status, oral health practices and socio-economic factor.

### MATERIALS AND METHODS

### PARTICIPANTS

Study participants were recruited based on school dental records of children attending the school dental service (SDS) in all 12 secondary schools in the District of Klang. Clinical findings from annual oral health examinations undertaken by SDS nurses were recorded in case notes which captured caries experience of each tooth. The clinical diagnosis and recording of caries experience were based on international protocols (World Health Organization 1997) detected solely by visual criteria and clinician elected whether or not to additionally use compressed air and/or a dental probe. A tooth was considered to have caries experience if it was recorded as decayed (D), missing due to caries (M) or filled (F). SDS nurses in the Ministry of Health Malaysia are calibrated annually for detecting caries experience.

A search was conducted on the dental records of 8713 children aged 13-14 years. Any child who was recorded to have caries experience in at least one second permanent molar was considered a case. The second permanent molars in this age group of children were selected for this study to investigate and to establish post natal effect of HHS as a risk factor to caries formation. The second permanent molar starts its formation about 10 months after birth (Ten Cate et al. 2008) and erupts around the age of 11-12 years old (Hussin et al. 2000). Thus, any caries experience observed among the children in this study would be due to the factors that were present post natal and not *in utero*. Children aged 13-14 years would have second permanent molars erupted for the past 1-3 years, making it suitable to establish risk association between HHS and caries.

A control was defined as a child from the same school and of the same age as a case but had no caries experience in any second permanent molars. The exclusion criteria were children with systemic diseases, special needs children and children in residential schools. These criteria were applied equally for both groups.

### SAMPLE SIZE ESTIMATION

Sample size estimation was carried out to provide sample size requirement for a case control study with a ratio of 1 case: 4 control children. The estimation aimed at a power of 0.90 to detect an odds ratio of 2.0 or more for the association between the risk factor differences in case and control groups, with type I error of *p*<0.05. The prevalence of HHS in the Malaysian population was reported around 0.5 (Ministry of Health Malaysia 2003; Sharina et al. 2007) and for the estimation of sample size, the probability of exposure to HHS was taken as 0.4 among controls and 0.6 for cases. This created a minimum requirement of 106 children in the case group and 424 in the control group. Sample size was estimated using the power and sample size calculations of PS program version 3.0 (Dupont & Plummer 2009)

### DATA COLLECTION

Data collection was conducted from October to November 2009. Table 1 shows all the variables studied. Secondary data from the school dental records provided

TABLE 1. Variables investigated

No	Variable Name	Description
1.	Caries experience	A tooth (permanent second molar) visually detected with decay or missing due to decay or filled (WHO 1997)
2.	Household smoking (HHS) status of child : • Presence of regular smokers at home	This measures child's exposure to HHS. A child was considered to be exposed to HHS if he/she had lived with at least one parent or household member who smokes or had smoked for any duration of time since the child was born
	Duration of exposure	• It was calculated based on the differences between ages of the most recent exposure with the age of the initial exposure to smoking in their household. Initially data was collected as continuous variable, and then it was re-categorized into three groups; 'no exposure', 'exposure of 1-10 years' and 'exposure more than 10 years'.
	• Frequency of exposure	<ul> <li>For frequency of exposure to HHS, the child was asked how often he/she was present when their parent/household member smoked per week.</li> </ul>
3.	Household income	The combined gross income of all the family members who live together and contribute to the household expenses. The income was grouped into four groups 'Poor < RM700', 'Low Income Household (LIH) RM700-RM1999', 'Middle Income Household (MIH) & High Income Household (HIH) ≥ RM2000'.
4.	Last dental visit	It refers to last dental care in the past two years (recalled period) besides care received under SDS. If more than one visits in the last 2 years, only the last visit was considered.
	Reason for last dental visit:     Problem solving     Preventive visit     No visit	Reasons for last dental visit:  • having a dental problem (getting treatment for it)  • routine check-ups  • only dental visit under SDS
5.	Child's smoking status	A child is considered a smoker if he has ever smoked cigarettes.
6.	Frequency of tooth brushing	The number of times the child usually brushes his/her teeth in a day
7.	Use of fluoridated toothpaste	The child uses fluoride toothpaste.
8.	Frequency of sugar intake	The number of times sugary food is consumed in a day. Frequency of sugar intake was estimated based on recall of common daily diet (for 24 hours period)
9.	Mother's level of education	The highest level of formal education attained by the mother of the child
10.	Oral hygiene status	The child's oral hygiene status as recorded in the school dental record which was based on the Simplified Oral Hygiene Index (Greene & Vermillion 1964). Based on the plaque score oral hygiene status was group into three: 'Good 0-4', 'Moderate 5-9' or 'Poor 10-18'.

information on child's caries status and oral hygiene status for 2009. The children and their parents responded to self-administered questionnaires to elicit information on HHS, socio-economic status, child's smoking status and child's oral health practices. A child was considered to be exposed to HHS if he/she had lived with at least one parent or household member who smokes or had smoked for any duration of time since the child was born. Frequency of sugar intake was calculated based on recall of common daily diet (for 24 h period). Sugar intake with the frequency of more than 4 times per day was used as the reference point to denote high sugar intake; this was

a suggested limit which can lead to a significant increase to caries risk (Moynihan 2005).

# DATA ANALYSES

Data was analysed using the Statistical Package for Social Science (SPSS) programme version 12. Simple associations were assessed with frequency tables and Pearson's chisquare test ( $\chi^2$ ) for two independent proportions. Finally, to consider joint effects of all risk factors found to be statistically significant at a cut off point of p<0.02 in the simple associations, a multiple logistic regression (MLR)

model was constructed to test the hypothesis. Stepwise forward and backward selection methods were used in the preliminary modelling. No interactions were found between the significant variables. The best model was selected as final model based on model fitness (Hosmer and Lemeshow test) and the value for area under receiver operating characteristic (ROC) curve. Estimates of adjusted odds ratio and accompanying 95% CI are presented for every risk factor.

### ETHICAL CONDUCT OF RESEARCH

This study was reviewed and approved by the Medical Ethics Committee of the Faculty of Dentistry, University of Malaya. Parents and guardians included in this study provided informed and signed consent for their participation.

### **RESULTS**

A total of 1460 children fulfilled the inclusion criteria; 292 in the case group and 1168 in the control group. Among them 232 (79.5%) cases and 967 (82.8%) controls responded to the questionnaires and were included in the analysis.

The distributions of participants in the case and control groups were very similar for the matched variables (gender and ethnicity) (matched variables) as well as mother's education level (Table 2). In terms of household income (Table 2) there was observed differences but it was not statistically significant.

In terms of gender, female participants were 53.9 and 53.1% in cases and controls, respectively. The majority of cases and controls were Malays (72.4 and 71.8%, respectively), followed by Indian (15.5 and 15.9%, respectively) and Chinese (12.1 and 12.3%, respectively).

### UNIVARIATE ANALYSIS

The chi-squared test of significance showed that four variables; namely dental visit, having regular smokers at home, frequency of exposure to HHS and duration of exposure to HHS had p values of less than 0.05 when tested independently as potential risk factors to caries (Table 2).

# MULTIVARIATE ANALYSIS

A cut off point of p<0.02 in univariate analysis was used to select variables to be included in the preliminary modelling. In addition, variables of biological importance in the aetiology of caries were also included. Only the variable on child's oral hygiene status was excluded in the MLR, as at univariate analysis, its association was highly not significant (p=0.979).

The result showed that more than half of the case group (55.9%) had been exposed to HHS, as compared to 44.1% among the control group. The final multiple logistic regression (MLR) model (Table 3) appears to support the hypothesis that HHS is a risk factor to have

caries experience in permanent teeth of children, even after adjusting for potential confounders. The final model demonstrated that exposure to HHS for more than ten years increased the risk of dental caries by almost double as compared to the non-exposed group (OR=1.90 and 95% CI=1.35, 2.60).

In addition to the main findings, this study also found that children who received dental care from the school dental service only had reduced risk of dental caries by more than one third (36%) as compared with those who received dental care from SDS as well as had additional dental problem-solving visit outside SDS (Adjusted OR=0.64 and 95% CI=0.50, 0.90).

### DISCUSSION

The findings of this case-control study is in agreement with a previous cross sectional study by Ayo-Yusoff et al. (2007), who reported that children raised in smoking households were twice more likely to have carious permanent teeth (OR=2.02 and 95% CI=1.22, 3.33). In an earlier study, Tanaka et al. (2006) failed to find an association between caries in permanent teeth, which the authors explained may be due to the crude information obtained on HHS, low statistical power because of the inadequate sample size (n=925) and also the presence of more deciduous teeth in the children studied making it difficult to detect a significant positive association between HHS and carious permanent teeth. However in 2010, Tanaka et al. (2006) repeated the study with a large sample size involving 20703 children. That study showed a significant association, consistent with the finding of this study.

There were a number of studies that investigated the effects of HHS exposure and caries in deciduous teeth which showed more consistent findings. All these studies showed that, young children who had been exposed to HHS were at higher risk to carious deciduous teeth (Avsar et al. 2008; Aligne et al. 2003; Leroy et al. 2007; Shenkin et al. 2004; Williams et al. 2000). However, the biological mechanism of the effect of tobacco smoke on dental caries development is not clearly established and is still being studied. Based on the literature reviews, there are a few possible causal pathways that might be considered to explain it. The in vitro studies by Saad (1990) and Yanagita et al. (2008) showed that nicotine has detrimental effects on tooth development which caused hypoplasia or hypomineralization. These observations suggest a possible disruption in the pre-eruptive physiological mechanism of action in tooth development which may cause the tooth to be more prone to caries. Heikkeninen et al. (1994) has suggested that the exposure to nicotine during tooth development may affect the tooth to be more susceptible to caries in the presence of sugar, upon eruption. Nicotine can also affect the composition of the normal flora in the oral cavity by promoting the growth of cariogenic bacteria (Avsar et al. 2008; Lindemeyer et al.

TABLE 2. Univariate analysis to determine the independent role of each variable studied as a risk factor to caries

Independent variables	Case $(n=232)$	Control ( <i>n</i> =967)	$p$ -value $x^2$ test
	n (%)	n (%)	x test
Reason for last dental visit:	<b>72</b> (24.0)	222 (22.4)	
Problem solving	72 (31.0)	223 (23.1)	0.000
Preventive visit	43 (18.5)	192 (19.9)	0.039
Never Visit	117 (50.4)	552 (57.1)	
Household income#:			
• Poor < RM700	46 (19.8)	188 (19.4)	
• LIH (RM700-RM1999)	131 (56.5)	477 (49.3)	0.066
<ul> <li>MIH &amp; HIH &gt; RM2000</li> </ul>	55 (23.7)	302 (31.2)	
Child's smoking status			
• No	216 (93.1)	917 (94.8)	0.301
• Yes	16 (6.90)	50 (5.20)	
Frequency of tooth brushing:	` '	` ,	
	174 (75.0)	755 (70 1)	0.314
<ul> <li>≥ 2 times per day</li> <li>Less than twice per day</li> </ul>	174 (75.0) 58 (25.0)	755 (78.1) 212 (21.9)	0.514
	58 (25.0)	212 (21.9)	
Use of fluoride toothpaste:			
• Yes	197 (84.9)	843 (87.2)	0.361
• No	35 (15.1)	124 (12.8)	
Frequency of sugar intake:			
<ul> <li>Less than 4x per day</li> </ul>	138 (59.5)	605 (62.6)	0.385
• ≥ 4x per day	94 (40.5)	362 (37.4)	
Mother's level of education:			
Primary & none	31 (13.4)	104 (10.8)	
• Secondary	168 (72.4)	730 (75.5)	0.498
Tertiary	33 (14.2)	133 (13.8)	
•	00 (1 <b>.</b> 2)	100 (1010)	
Oral hygiene status:  Good	224 (06-6)	024 (06 6)	0.070
	224 (96.6)	934 (96.6)	0.979
• Moderate	8 (3.40)	33 (3.40)	
Household Smoking Status*	Case (N=227)	Control (N=959)	<i>p</i> -value
5	n (%)	n (%)	$x^2$ test
Presence of regular smoker(s) at home:	. ,	. ,	
• Yes	127 (55.9)	423 (44.1)	0.001
• No	100 (44.1)	536 (55.9)	0.001
	100 (77.1)	550 (55.7)	
Duration of Exposure to HHS (years):	100 (11.1)	504 (55.0)	
No exp to HHS	100 (44.1)	536 (55.9)	0.004
• 1-10 yrs duration of exp to HHS	37 (16.3)	159 (16.6)	0.001
<ul> <li>&gt; 10 yrs duration of exp to HHS</li> </ul>	90 (39.6)	264 (27.5)	
Frequency of Exposure to HHS:			
• Never (with & without smokers)	127 (55.9)	626 (65.3)	
• ≤ Once per week	24 (10.6)	101 (10.5)	0.013
<ul> <li>Once per week</li> </ul>	76 (33.5)	232 (24.2)	

Significant results (p<0.05) presented in bold

1981; Strauss 2001). The group of children in this study, who were exposed to HHS for more than ten years, may have been exposed to HHS at a younger age or since they were born. This is because the second permanent molar starts to form about 10 months after birth (Ten Cate et al. 2008), therefore there is a possibility of pre-eruptive effects on tooth formation of second permanent molar and hence would explain the higher risk for caries.

There are many established evidences to show that excessive dietary sugar is one of the most important factors that contribute to dental caries development (Gustafsson et al. 1954; Harris 1963; Rugg-Gunn 1993). However, the present study did not find any significant association between sugar diet and carious permanent teeth. Burt and Pai (2001) reported that the relationship between sugar consumption and caries is much weaker when the use of

<sup>\* 13</sup> subjects did not respond to these questions.

 $<sup>{}^{\#}\!</sup>LIH\!=Low\ Income\ Household, MIH\!=Middle\ Income\ Household, HIH\!=High\ Income\ Household}$ 

TABLE 3. Multiple logistic regression analysis to determine risk factors to caries of second permanent molars

Independents Variables	Unadjusted OR (95% CI)	Adjusted OR (95%CI))
Duration of Exposure to HHS		
No exp to HHS	1	1
• 1-10 years duration of exp to HHS	1.25 (0.82, 1.90)	1.26 (0.83, 1.91)
• > 10 years duration of exp to HHS	1.83 (1.33, 2.52)	1.90 (1.35, 2.60)
Reason for last dental visit (other than SDS):		
<ul> <li>Problem solving</li> </ul>	1	1
Preventive visit	0.69 (0.45, 1.06)	0.79 (0.51, 1.22)
Never Visit	0.66 (0.47, 0.91)	0.64 (0.50, 0.90)

Multiple Logistic Regression model using stepwise forward (Hosmer-Lemeshow test p-value = 0.851 and area under ROC = 0.59 OR=Odds Ratio, CI= Confidence Interval,

Significant results (p<0.05) presented in bold

fluoride is wide spread. This may explain the finding of this study since all the population in the State of Selangor where the District of Klang is located received fluoridated piped water (OHD 2009).

The existence of SDS is a way to provide equitable access to dental services especially for school children. It is very interesting to find that children who have never visited a dentist (other than in the SDS) have significantly lesser risk to caries as compared with those who made problem solving dental visit. A plausible explanation is children who never made extra dental visit (refers to as 'never visit' group) received adequate care under SDS which provides yearly dental check-up followed by treatment if needed. This yearly dental check-up interval is recommended as good practice point by National Institute for Health and Care Excellence (NICE) for adolescence (NICE 2004). Obviously, those who made problem solving dental visit had untreated dental problems and the reason why the dental problems were not treated under SDS is beyond the scope of this study.

This study also found that mother's education level and household income independently, were not significantly associated with carious permanent teeth. In contrast, many previous studies have shown that an inverse relationship between caries status and SES (Hausen et al. 1982; Mc Nulty 1989; Reisine & Psoter 2001). In Malaysia, the public oral health care services are easily accessible at minimal and affordable cost. The presence of a comprehensive SDS also indirectly educates the children on the importance of their oral health and teaches them the methods of maintaining their oral health. The SDS is a mechanism to ensure the maintenance of good oral health among the school children irrespective of household income and the parental education status. Consequently, it may be difficult to show a socioeconomic difference in caries experience among the younger children in this study because of the number of differences in accessibility to care.

According to Kopec and Esdaile (1990) a case-control study is susceptible to bias. Bias in this study has been controlled or minimised at data collection stage as well as in the statistical analysis. Information bias was minimised by using a structured questionnaires which were distributed and collected in the same way and time for both groups. Despite these limitations, this study provides another piece

of evidence that long term exposure to tobacco smoke is a common risk factor in health. From the oral health perspective, smoking increases risk to develop not only oral malignancy and periodontal disease but also to dental caries; a common oral disease of public health concern. Health education messages should take cognisance of this finding; that HHS is a risk factor to dental caries in permanent dentition. Findings from this study indicate that exposure to HHS be included as a risk factor in a caries assessment tool. However, future research on dose-response effect of HHS on caries will assist to determine the level of risk at different dose of exposure to HHS. In conclusion, the study found that long term exposure to HHS increases the risk to have caries experience in permanent teeth of children.

## ACKNOWLEDGEMENTS

The authors would like to thank the Oral Health Division, Ministry of Health and Selangor Dental Division for granting us access to oral health status data of the children involved from school dental record. In addition, the financial support from the Postgraduate Research Fund (vote no: P0108/2009C), University of Malaya is gratefully acknowledged. The funders had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

### REFERENCES

Aligne, C.A., Moss, M.E., Auniger, P. & Weitzman, M. 2003. Association of paediatric dental caries with passive smoking. *JAMA* 289:1258-1264.

Avsar, A., Darka, O., Topaloglu, B. & Bek, Y. 2008. Association of passive smoking with caries and related salivary biomarkers in young children. Archives of Oral Biology 53: 969-974.

Ayo-Yusuf, O.A., Reddy, P.S., Van Wyk, P.J. & Van den Borne, B.W. 2007. Household smoking as a risk indicator for caries in adolescents' permanent teeth. *J. Adolescent Health* 41: 309-311.

Burt, B.A. & Pai, S. 2001. Sugar consumption and caries risk: A systematic review. *J. Dent. Educ.* 65(10): 1017-1023.

Dupont, W.D. & Plummer, W.D. 2009. Power and sample size calculation software. Version 3.0. Department of Biostatistic, Vanderbilt University. http://biostat.mc.vanderbilt.edu.

Gustafsson, B.E., Quensel, C.E., Lanke, L.S., Lundquist, C., Grahnen, H., Bonow, B.E. & Krasse, B. 1954. The

- Vipeholm dental caries study: The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. *Acta Odontologica Scandinavica* 11: 232-364.
- Greene, J.C. & Vermillion, J.R. 2009. Simplified oral hygiene index (OHI-S) 1964; http://www.whocollab.od.mah.se.
- Harris, R.M. 1963. Biology of the children of Hopewood House, Bowral, Australia: Observations on dental caries experience extending over five years (1957-1961). *Journal Dental Research* 42: 1387-1399.
- Hausen, H., Milen, A., Heinonen, O.P. & Paunio, I. 1982. Caries in primary dentition and social class in high and low fluoride areas. *Community Dent. Oral Epidemiol.* 10: 33-36.
- Heikkinen, T., Alvesalo, L., Osborne, R.H. & Tienari, J. 1994.
  Maternal smoking and tooth formation in the foetus. II.
  Tooth crown size in the permanent dentition. *Early Hum. Dev.* 40(1): 73-86.
- Hussin, A.S., Mokhtar, N., Naing, L., Taylor, J.A. & Mahmood, Z. 2000. The timing and sequence of emergence of permanent teeth in Malay schoolchildren in Kota Bharu, Malaysia. Archives of Orofacial Sciences 2: 36-40.
- Jaafar, N. & Razak, I.A. 2002. The prevalence and impact of oro-facial pain among 12-year-old schoolchildren in an epidemiological survey setting. *Annal. Dent. Univ. Malaya* 9: 11-16.
- Kopec, J.A. & Esdaile, J.M. 1990. Bias in case control study: A review. *Journal of Epidemiology and Community Health* 44: 179-186.
- Leroy, R., Hoppenbrouwers, K., Jara, A. & Declerck, D. 2007.
  Parental smoking behaviour and caries experience in preschool children. *Community Dent. Oral Epidemiol*. 36: 249-257.
- Lindermeyer, R.G. 1981. *In vitro* effect of tobacco on the growth of cariogenic streptococci. *JADA* 103: 719-722.
- McNulty, J.A. & Fos, P.J. 1986. The study of caries prevalence in children in a developing country. *J. Dent. Research* 53: 76-82.
- Ministry of Health Malaysia. 2003: *Global Youth Tobacco Survey*. Non Communicable Disease Control Section, Disease Control Division, Ministry of Health Malaysia.
- Moynihan, P.J. 2005. The role of diet and nutrition in the aetiology and prevention of oral disease. *Bull. World Health Organization* 83: 694-699.
- National Institute for Health and Care. 2004. *Dental Recall:* Recall Interval between Routine Dental Examinations. (GC19). London: National Institute for Health and Care Excellence.
- Nomura, L.H., Bastos, J.L.D. & Peres, M.A. 2004. Dental pain prevalence and association with dental caries and socioeconomic status in schoolchildren, Southern Brazil. *Braz. Oral Res.* 18(2): 134-140.
- Oral Health Division, Ministry of Health Malaysia. 2009. *Annual Report: Oral Health Division*. Ministry of Health Malaysia.
- Oral Health Division, Ministry of Health Malaysia. 2004. National Oral Health Survey of Adults (NOHSA) Malaysia 2000. Ministry of Health Malaysia.
- Reisine, S.T. & Psoter, W. 2001. Socioeconomic status and selected behavioural determinants as risk factors for dental caries. *J. Dent. Edu.* 65(10): 1009-1016.
- Rugg-Gunn, A.J. 1993. Nutrition, diet, dental public health. *Community Dental Health* 10(2): 47-56.
- Saad, A.Y. 1990. Postnatal effects of nicotine on incisor development of albino mouse. *Journal Oral Pathological Medicine* 19: 426-429.

- Sharina, D., Zulkifli, A. & Nyi, N.N. 2007. Second hand smoke exposure at home and respiratory symptoms among primary school children in Kota Bharu, Kelantan. *Malaysian Journal of Public Health Medicine* 7(2): 59-63.
- Sheiham, A. 2006. Dental caries affects body weight, growth & quality of life in preschool children. *British Dental Journal* 201: 625-626.
- Shenkin, J.D., Broffitt, B., Levy, S.M. & Warren, J.J. 2004. The association between environmental tobacco smoke and primary tooth caries. *J. Public Health Dentistry* 64: 184-186.
- Strauss, R.S. 2001. Environmental tobacco smoke and serum vitamin C levels in children. *Paediatrics* 107: 540-542.
- Tanaka, K., Miyake, Y., Arakawa, M., Sasaki, S. & Ohya, Y. 2010. Household smoking and dental caries in schoolchildren: Ryukyus Child Health Study. *BMC Public Health* 10(1): 335.
- Tanaka, K., Hanioka, T., Miyake, Y., Ojima, M. & Aoyama, H. 2006. Association of smoking in household and dental caries in Japan. J. Public Health Den. 66: 279-281.
- Ten Cate, A.R., Sharpe, P.T., Roy, S. & Nanci, A. 2008. Chapter 5: Development of the tooth and its supporting tissues. In *Ten Cate's: Oral Histology, Development, Structure and Function* Edited by Antonio Nanci. Mosby: Elsevier. pp. 79-99.
- William, S.A., Kwan, S.Y.L. & Parson, S. 2000. Parental smoking practices and caries experience in preschool children. *Caries Research* 34: 117-122.
- World Health Organization (WHO). 1997: Oral *Health Surveys*, *Basic Methods*. 4th ed. Geneva: World Health Organization.
- Yanagita, M., Kashiwagi, Y., Kobayashi, R., Tomedo, M., Shimabukuro, Y. & Murakami, S. 2008. Nicotine inhibits mineralization of human dental pulp cells. *Journal of Endodontic* 34(9): 1061-1065.

A.M. Zurina Ministry of Health Blok E1, E3, E6, E7 & E10, Kompleks E Pusat Pentadbiran Kerajaan Persekutuan 62590 Putrajaya Malaysia

R.J. Raja Latifah International Islamic University Malaysia, Jalan Sungai Pusu 53100 Gombak, Selangor Malaysia

Y. Noriah Faculty of Dentistry Universiti Kebangsaan Malaysia (Kampus KL) Jalan Raja Muda Abd. Aziz 50300 Kuala Lumpur Malaysia

M. Asma'\*

Department of Community Oral Health and Clinical Prevention Faculty of Dentistry, University of Malaya 50603 Kuala Lumpur Malaysia

\*Corresponding author; email: asmar@um.edu.my

Received: 20 September 2013 Accepted: 7 April 2014