Association between Exhaled Carbon Monoxide and Oral Health Status in Active and Passive Smokers

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Background: *Few studies so far have evaluated the association between oral health status and level of carbon monoxide in the exhaled breath (exhaled CO).*
Objective: *Evaluate the relationship between oral health status, namely dental caries and periodontitis, and exhaled carbon monoxide in active and passive smokers.*
Material and Method: *The analytic cross-sectional study sample comprised of 296 Thai adults, aged 30-72 years, residing in an urban district of Khon Kaen province, Thailand during the year 2007, who received exhaled carbon monoxide measurement, oral examination, and interview. Spearman correlation tests were employed to determine the relation between exhaled carbon monoxide and dental caries. In addition, the relationship between exhaled carbon monoxide and periodontitis was evaluated using Mann-Whitney U Test.*
Results: *The results revealed no relationship between exhaled carbon monoxide and oral health status among passive smokers. Moreover, the relationship between exhaled carbon monoxide and dental caries among active smokers did not exist when education was adjusted.*
Conclusion: *There is no relationship between exhaled carbon monoxide and oral health status in both active and passive smokers.*

Keywords: *Exhaled carbon monoxide, Oral health, Active smokers, Passive smokers*

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Data from the World Health Organization and other sources indicate that smoking is the main source of disease and causes health destruction and death in smokers, since many materials present in cigarettes are poisonous to cells and various systems in the body. Many past studies have confirmed the ill effects of components of cigarettes and the danger to many bodily systems. Inhaling smoke from other peoples' cigarettes from the environment into their bodies (passive smoking) means non-smokers take in components of sidestream and mainstream smoke. They then exhale these components as mainstream smoke again, which can occur in both open and closed environments. The severity of contact with smoke by non-smokers depends on the quantity of cigarette smoke produced, as well as the environment, the lung volume, the frequency of exhaling, age and the physical health of the person[1,2].

In 2001, Nelson published a paper discussing sidestream smoke, which consists of many substances poisonous to the body, such as polycyclic aromatic hydrocarbons (PAHs), aromatic amines, nitrosamines, heavy metals, poisonous gases, pesticide residues, and radioactive elements etc., which tend to have higher proportions than in mainstream smoke. People who receive passive smoking risk death from cancer and heart disease, as well as risks to the reproductive system, the nervous system, DNA and to pregnant women and their fetuses. There tends to be a history of asthma, severe infections and damaged immune systems sensitive to allergy. In addition, higher nicotine levels are found in the blood, in the form of cotinine, which is in the form of an inactive metabolite of nicotine in liquid solution, enabling distribution of nicotine throughout the body. This can increase sensitivity to dental disease in those who smoke. Severe effects of inhaling cigarette smoke in the environment are throat

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irritation and infection. Sidestream smoke causes air pollution and symptoms that can be seen rapidly after contact. It include throat irritation caused by stimulation by excess gas such as carbon monoxide (CO), which is able to attach to hemoglobin as carboxyhemoglobin and reduce the transport of oxygen, causing headache, dizziness, difficulty breathing and unconsciousness, depending on the severity and length of time of contact. Cigarette smoke and carbon dioxide (CO_2) affect the progress of the symptoms of shortage of air, causing dry eyes, dry mouth, dry throat and perhaps red eyes. It has been found that coughing is often found more in children who inhale smoke from the environment, as well as having a history of tonsillectomy, than in children who do not receive smoke from the environment. Apart from this, people who inhale smoke from the environment become less able to smell, since there is a change in the number of nasal mucocilia after receiving cigarette smoke. The immediate response of the body to cigarette smoke can occur in the area of the bronchi as well, where the changes in bronchial resistance of non-smokers affected by cigarette smoke can be measured. In addition, the effect of inhaling cigarette smoke in the environment can damage the membranes of the lungs, and respiratory clearance can be reduced. The breathing passages are reduced in diameter in children with a history of breathing passive smoke. Studies of genetic predisposition have shown effect on immediate symptoms[2].

Results from many studies have shown that constituents of cigarettes are poisonous to cells and cause defects in cells, leading to severe dental disease[3-7]. From studies in the laboratory of Gamal and Bayomy in 2002 on the adhesion of fibroblast cells to the tendons around the root surfaces of teeth in smokers, it was found that there was a significant reduction in the adhesion to samples of teeth roots from smokers smoking less than 19 cigarettes per day and more than 20 per day, when compared with non-smokers and those smoking less than two cigarettes per week. There was no difference between those smoking less than 19 or more than 20 cigarettes per day. Results of the present study showed that smoking causes the adhesion of PDL cells to the surface of tooth roots to reduce, causing an increase in periodontitis[8]. In addition, the study by Cattaneo et al in 2000 agreed with these findings. They tested the effect of cigarettes on the reproduction of cells, adhesion and the fundamental structure of fibroblast cells in humans. They used a transmission electron microscope and the results showed that acrolein and acetaldehyde reduced the reproduction of cells and adhesion of fibroblast cells in the gums. The effect was seen to depend on the amount of acrolein and acetaldehyde. Thus, smoking is a risk factor for periodontitis[9].

There are many chemical constituents of cigarettes which are poisonous to cells in the body, and which can lead to abnormalities and can lead to severe periodontitis. Cotinine, which is an inactive metabolite of nicotine in saliva, affects the distribution of nicotine in the body, which affects the increase in susceptibility to periodontitis in smokers. A study by Eramo et al in 2000 compared the effect of cotinine in the saliva of regular smokers, non-smokers and those who received smoke from the environment, with the sample groups having no visible symptoms of periodontitis at the start of the study. They measured the quantity of cotinine using ELISA and found that they could find higher levels of cotinine in the saliva of the group of smokers, followed by the passive smokers and lastly the non-smokers. The average quantity was 92.3 ± 4.15 ng/ml in the smokers group, 12.9 ± 6.67 ng/ml in the passive smokers group and 5.4 ± 1.22 ng/ml in the non-smokers group. The study found that the concentration of cotinine in the saliva could help to estimate the amount of nicotine received into the body, which is the substance that has an effect on periodontitis, even in the group that received smoke from the environment[10].

Constituents of cigarettes such as nicotine have an effect on the working of the fibroblast cells in the gums. They cause changes in the structure of the cells. They reduce both the adhesion and proliferation of cells. The study by Rota et al in 2001 found that there is an effect on the shape of cells[11]. In addition, Poggia and Rota found in 2001 while studying the effect of acrolein and acetaldehyde on the interior of human gum fibroblast cells by fluorescence microscopy that both substances reduced the adhesion of fibroblast cells while the fibroblast cells changed and arranged themselves differently or had their arrangement disturbed, causing changes in cell shape[12].

Cotinine in saliva also has an effect on the bacteria that cause periodontitis. Sayers et al in 1999 studied chicken embryos infected with the bacteria Prevotella intermedia, Prevotella nigrescens and Porphyromonas gingivalis with Staphylococcus aureus and Escherichia coli as the control group. They found to be a reinforcing effect between cotinine in the saliva and poisons from the bacteria, which cause

periodontitis. This may be an important mechanism making periodontitis more severe and increased invasion[13]. Barbour et al in 1997 reviewed the literature on the effect of smoking on the immune system and the effects on periodontitis. They found that smokers were at higher risk than non-smokers by a factor of 2.5 to 6[14].

Substances in cigarette smoke also affect the work of neutrophils (polymorphonuclear leukocyte-PMN) which are important in the progress of periodontitis. One study showed this connection between cigarette smoking and periodontitis. It wasdone by Numabe et al in 1998 and studied the effect of smoking on the phagocytic function of salivary PMN (SPMN). They separated out salivary PMN to test them before and after they had experienced cigarette smoke in smokers and passive smokers, and found that the proportion of phagocytic cells increased after receiving smoke. In the smokers group they increased from 33.2% to 42.1% and in the passive smokers group they increased from 36.2% to 44.1%. This was a statistically significant increase ($p < 0.01$). This study showed that the phagocytic activity of the SPMN increased after smoking and in passive smokers, which may indicate that constituents of cigarettes such as nicotine stimulate the responses of the body in the mouth[15].

Breathing in cigarette smoke in the environment is another factor that may cause dental caries. A study by Williams et al in 2000 looked at 749 children aged 3 to 4.5 years old on data by the UK National Diet and Nutrition Survey of 1995. Results of the study show that where mothers smoked they could predict having dental caries in the children when using logistic regression with control of other factors[16]. In addition, the work by Aligne et al. in 2003 studied the relationship between dental caries in milk teeth and nicotine in serum using data from the National Health and Nutrition Examination Survey (1988-1994) in a population of 3,531 children aged 4-11 years old, and found that the increase in the amount of nicotine in the serum had a statistically significant relationship with the incidence of dental caries. The study showed that when eliminating other factors that might have influence, such as age, sex, race, and family income etc., it was seen that the Odds Ratio in the group with dental caries was 1.8 (95% CI of 1.2-2.7) and 1.4 (95% CI of 1.1-2.0) in the group with fillings. It was found that children who breathed cigarette smoke in the environment had a risk of dental caries of 27% and of fillings of 14%. The study was able to conclude that there was a relationship between breathing cigarette smoke from the environment and having dental caries. Thus reducing smoke breathed from the environment does not only affect physical health but also promotes the dental health of children[17].

Measuring the amount of carbon monoxide in the exhaled breath is a simple and convenient way of measuring smoking status. If the amount of carbon monoxide is about 17 ppm this indicates that the person is a smoker, 5 ppm indicates that the person inhales carbon monoxide from the environment, and below 3 ppm is the amount for a non-smoker[18-22]. In the past there have been studies in the Thai population in the Northeast province of Khon Kaen of the amount of carbon monoxide in the exhaled breath and the smoking status[23], but there has not been a study of health status and carbon monoxide levels in the exhaled breath in this population before.

Therefore, the purpose of the present study was set to assess the relationship between oral diseases, being periodontal disease and dental caries, and the level of carbon monoxide in exhaled breath in smokers and non-smokers who receive smoke from the environment living in the municipal area of Khon Kaen in 2007.

Material and Method

Sample

In the present study the sample consisted of volunteers aged 30-72 years old living in Nai Muang, Pralup, Ban Peu and Ban Kor sub-districts, Muang district of Khon Kaen province in 2007. The sample consisted of smokers and non-smokers who received cigarette smoke from the environment. The smokers group had smoked at least one cigarette per day up to present, while the non-smokers who received cigarette smoke from the environment were those whose wife or husband had smoked at least five cigarettes per day for at least one year or those who worked with people who had smoked at least five cigarettes for at least one year. The people in the present study area received information about the present study and were encouraged to take part. Those who volunteered signed a consent form to take part in the present study, which was approved by the Human Research Ethics Committee of Khon Kaen University.

Method of data collection

After signing the study consent form, the sample group members were tested for the amount of carbon monoxide in the exhaled breath. Then they were

given a dental examination of periodontal condition by a single dentist, while a second dentist examined for decayed, missing, and filled teeth as well as need for treatment and prosthetic status. Both dentists who examined for oral health status had previously been trained for assessing oral health indices correctly and had Kappa values for repeatability of at least 80%. Research equipment consisted of an instrument to measure carbon monoxide in the exhaled breath (exhaled CO) with paper tubes, basic data, and smoking status questionnaire, form to record oral health data, instruments for oral examination, and WHO probe to assess periodontal condition.

Indices to measure oral health status

Periodontal indices: The periodontal condition was assessed using the Community Periodontal Index (CPI) with the following criteria:

0 = Healthy gingiva, 1 = Bleeding, 2 = Calculus, 3 = Calculus with bleeding, 4 = Pocket 4-5 mm, 5 = Pocket 6 mm or more, 9 = Cannot be determined, X = Missing sextant/excluded

Indices of dental caries status and treatment need: Dental caries status was measured using decayed, missing, and filled teeth (DMFT) index whereby the criteria were coded as follows:

Dental caries status: 0 = normal tooth without caries (sound tooth), 1 = decayed, 2 = filled with decayed, 3 = filled with no decayed, 4 = missing due to caries, 5 = missing due to other reasons, 6 = sealant, 7 = crown or bridge abutment, 8 = unseen in the oral cavity, 9 = status of teeth not included in the above, 10 = fracture

Need for treatment: 0 = does not need treatment, P = prevention of caries, 1 = one surface filling, 2 = two or more surfaces fillings, 3 = crown, 4 = veneer, 5 = root canal treatment, 6 = extraction, 7 = other treatment (specify type of treatment).

Examination of prosthetic status:

Upper denture status: 0 = no denture, 1 = have denture

Lower denture status: 0 = no denture, 1 = have denture.

Data analysis including descriptive and analytical statistics

Descriptive statistics were used in analyzing the basic information from the sample group, such as sex, age, marital status, religion, educational level, main occupation, average monthly income, level of carbon monoxide in the exhaled breath (exhaled CO) and oral health status, being periodontitis and dental caries (decayed, missing and filled teeth) using percentage, average and standard deviation.

Analytical statistics included analysis of the primary relationship between oral health status (decayed, missing, and filled teeth as well as periodontitis) and the level of carbon monoxide in the exhaled breath. Statistics used in for relationship were the Spearman correlation test and testing the differences between means of the level of carbon monoxide in the exhaled breath (exhaled CO) between those with and without periodontal pockets using the Mann-Whitney test since the level of carbon monoxide in the exhaled breath had non-normal distribution. P-value of less than 0.05 was considered of significant.

Control of data quality

Measurement of oral status

Before collecting dental data, the two examiners were trained in measuring every index until they were able to measure correctly according to the principles and standards of the experts on the use of the indices. Then there was adjustment of the clinical assessment standards in the experts' areas of responsibility. The repeatability of the measurements was measured using a Kappa value of more than 80%, with repeated measurements in 10% of the sample that were then tested for Kappa value to make sure that the whole examination had acceptable Kappa higher than 80%.

Data recording

To reduce errors in the data which had much detail, double data entry was performed independently by two people who had been well trained to do it.

Results

The present study had a population of 296 people living in the Nai Muang, Pralub, Ban Kor sub-districts, Muang district of Khon Kaen province, consisting of 123 smokers and 173 non-smokers who received cigarette smoke from the environment. Both groups had a mean age of about 45 years, were mostly married (more than 80%), were Buddhist (99-100%) and mostly completed lower primary education (36-37%). The smokers' group had an average monthly income higher than the group who received cigarette smoke from the environment (7,039 and 6,870 baht respectively). The group of smokers were mostly male

(99%) whereas the non-smokers' group who received smoke from the environment were mostly women (72%). The smokers' group was mostly labourers (33%) whereas the group who received smoke from the environment was mostly agriculturalists (31%). The average level of carbon monoxide in the exhaled breath (exhaled CO) of the smokers' group was 10.62 ppm while that in the group who received cigarette smoke from the environment had a level of carbon monoxide ion the exhaled breath (exhaled CO) of 2.70 ppm. Concerning the oral health status, there were more periodontal pockets found in the smokers' group than in the group that received cigarette smoke from the environment (81% and 57% respectively) while the number of people who had decayed, missing, and filled teeth in the two groups were similar (Table 1).

Among all samples, duration of smoking had some direct correlations with missing teeth and filled teeth. However, no relationship was found between periodontitis (having shallow and/or deep periodontal

Table 1. Basic data and oral health status of the groups of smokers and non-smokers who received cigarette smoke from the environment (sample size 296 people)

Status	Smokers (n = 123)	Non-smokers (n = 173)
Age (mean ± standard deviation in years)	45.51 ± 10.18	44.85 ± 10.47
Income (mean ± standard deviation in baht)	7,039.00 ± 5,181.00	6,870.00 ± 8,155.00
Level of carbon monoxide in the exhaled breath (mean ± standard deviation in ppm)	10.62 ± 6.41	2.70 ± 1.16
Sex		
Male	122 (99.2%)	48 (27.7%)
Female	1 (0.8%)	125 (72.3%)
Marital status		
Married	99 (80.5%)	148 (85.5%)
Single	13 (10.6%)	6 (3.5%)
Widowed	2 (1.6%)	13 (7.5%)
Divorced or separated	9 (7.3%)	6 (3.5%)
Highest education level		
Did not go to school	3 (2.4%)	4 (2.3%)
Lower primary level	45 (36.6%)	63 (36.4%)
Upper primary level	29 (23.6%)	40 (23.1%)
Lower secondary level	16 (13.0%)	19 (11.0%)
Upper secondary level/occupational certificate	13 (10.6%)	24 (13.9%)
Higher occupational certificate/bachelor diploma	3 (2.4%)	5 (2.9%)
Bachelor's degree	13 (10.6%)	16 (9.2%)
Higher than bachelor's degree	1 (0.8%)	2 (1.2%)
Religion		
Buddhist	23 (100%)	171 (98.8%)
Other	10 (0%)	2 (1.2%)
Occupation		
Agriculturalist	34 (27.6%)	54 (31.2%)
Civil servant/state enterprise worker	33 (26.8%)	34 (19.7%)
Labourer	40 (32.5%)	38 (22.0%)
Merchant/private business	10 (8.1%)	22 (12.7%)
Unemployed	4 (3.3%)	15 (8.7%)
Others	2 (1.6%)	10 (5.8%)
People with shallow and deep periodontal pockets		
With	100 (81.3%)	99 (57.2%)
Without	23 (18.7%)	74 (42.8%)
Status of decayed, missing and filled (DMFT) (teeth per person)		
Decayed (mean ± standard deviation)	2.67 (2.65)	2.54 (2.23)
Missing (mean ± standard deviation)	3.72 (4.33)	3.23 (3.22)
Filled (mean ± standard deviation)	0.20 (0.78)	0.43 (1.27)

pocket) and smoking duration, with the mean duration of smoking in the groups without and with periodontitis being 47.5 and 50.5 months, respectively (Table 2).

In Table 3, in both the smokers' group and the group of non-smokers receiving cigarette smoke from the environment, there was no correlation found between having decayed, missing, and filled teeth and exhaled CO. There was only a relationship between having periodontal pockets and exhaled CO, with the mean value of the level of carbon monoxide in the exhaled breath (exhaled CO) in the groups without and with periodontal pockets of 124.7 and 162.7 ppm, respectively (Table 3). When considering separately the relationship between oral health status and exhaled CO, in the smokers' group and the group who received smoke from the environment (Table 4), it was found that the smokers' group had only status of filled teeth, which had some correlation with the exhaled CO, while having decayed teeth, filled teeth or periodontitis had no correlation with amount of carbon monoxide in the exhaled breath (exhaled CO). However, when controlling the relationship between filled teeth and exhaled CO with only the level of education variable, the inverse relationship between the two variables disappeared. Therefore, the spurious relationship between exhaled CO and filled teeth might occur as a result of having level of education as a confounding factor.

In addition, Table 4 shows that decayed, missing, and filled teeth as well as periodontitis status had no correlation with exhaled CO in the group who received cigarette smoke from the environment.

Conclusion

Results of the present study show that the level of carbon monoxide in exhaled breath in smokers is higher than that in the non-smokers group. Moreover, duration of smoking did demonstrate some direct connections with missing teeth and filled teeth while no relationship between smoking duration with periodontitis was found.

However, in the present study, no correlation was confirmed between level of carbon monoxide in the exhaled breath and dental caries status (decayed, missing, and filled teeth) or having periodontal pockets in both the group of smokers and the group of

Table 2. Correlation between oral health status and smoking duration among all samples (n = 99)

Oral health status	Level of correlation	p-value
Decayed teeth	0.095	0.351
Missing teeth	0.287	0.004[b]
Filled teeth	0.248	0.013[b]
Periodontitis (group without/with periodontal pockets)	-47.5/50.5	0.696

[b] Spearman correlation test; $p < 0.05$ (2-tailed test)

Table 3. Correlation between oral health status and level of carbon monoxide in the exhaled breath among all samples (n = 296)

Oral health status	Level of correlation	p-value
Decayed teeth	0.016	0.743
Missing teeth	0.019	0.782
Filled teeth	-0.049	0.394
Periodontitis (group without/with periodontal pockets)	124.7/162.7	<0.001[b]

[b] Mann-Whitney U test; $p < 0.05$ (2-tailed test)

Table 4. Correlation between oral health status and level of carbon monoxide in the exhaled breath in smokers and non-smokers who receive cigarette smoke from the environment groups

Oral health status	Level of correlation	p-value
Smokers group (n = 123)		
Decayed teeth	0.061	0.547
Missing teeth	0.059	0.563
Filled teeth	-0.228	0.023[b]
Filled teeth (controlled by level of education)	-0.0809	0.426
Periodontitis (group without/with periodontal pockets)	41.4 / 52.4	0.155
Non-smokers who receive cigarette smoke from the environment group (n = 173)		
Decayed teeth	0.047	0.569
Missing teeth	0.057	0.493
Filled teeth	0.079	0.339
Periodontitis (group without/with periodontal pockets)	73.1 / 75.6	0.709

[b] Spearman correlation test; $p < 0.05$ (2-tailed test)

non-smokers who received cigarette smoke from the environment.

Discussion

The findings that the level of carbon monoxide in the exhaled breath in smokers is higher than that in the non-smokers group agree well with the results of several studies, which may support that the level of carbon monoxide in the exhaled breath might be used as an indicator of smoking status[18-22]. Moreover, that duration of smoking did demonstrate some direct connections with missing teeth and filled teeth is in agreement with previous research[24-27], but the result did not confirm past studies as no relationship between smoking duration with periodontitis existed[3-9]. However, the findings were not comparable in that no correlation was found between exhaled CO and dental caries status, including decayed, missing, and filled teeth or having periodontal pockets both in the group of smokers and the group of non-smokers who received cigarette smoke from the environment, while months of smoking showed some correlations with missing teeth and filled teeth, but not with periodontitis. Previous studies have reported quite clear correlations between smoking and periodontitis[3-9] and between smoking and dental caries[24-27]. This might indicate that the level of carbon monoxide in the exhaled breath might not be a suitable indicator to measure smoking status in the present study, or the present study might have too small a sample size to capture the association between exhaled CO and dental caries status. For example, in order to capture such small correlations ($r < 0.05$) between level of carbon monoxide in the exhaled breath and decayed teeth, missing teeth, and filled teeth, the required sample size would be larger than 3,134 people, given the two-sided alpha 0.05 and power of test 0.80[28].

Since most studies in the past that reported the relationship between oral health status and exhaled CO did not account for potential confounding factors, further study should include more samples and information regarding the variables that might influence the relationship between oral health status and exhaled CO so that the power of study as well as the external validity of the study would be enhanced.

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Potential conflicts of interest

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ความสัมพันธ์ระหว่างระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกและสภาวะสุขภาพช่องปากในผู้ที่สูบบุหรี่และผู้ที่ไม่สูบบุหรี่แต่ได้รับควันบุหรี่จากสิ่งแวดล้อม

สุภาภรณ์ ฉัตรชัยวิวัฒนา, อมรรัตน์ รัตนสิริ

ภูมิหลัง: *การศึกษาที่เกี่ยวข้องกับความสัมพันธ์ระหว่างสภาวะสุขภาพช่องปากกับระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกยังมีไม่มาก และผลการศึกษายังไม่มีข้อสรุปที่ชัดเจน*

วัตถุประสงค์: *การศึกษาครั้งนี้มีวัตถุประสงค์เพื่อศึกษาความสัมพันธ์ระหว่างสภาวะสุขภาพช่องปากหลัก ๆ อันได้แก่โรคฟันผุและโรคปริทันต์ กับระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกที่วัดได้ในกลุ่มของผู้ที่สูบบุหรี่และผู้ที่ไม่สูบบุหรี่แต่ได้รับควันบุหรี่จากสิ่งแวดล้อม*

การออกแบบการศึกษา: *เป็นการศึกษาภาคตัดขวางเชิงวิเคราะห์*

วัสดุและวิธีการ: *กลุ่มประชากรศึกษาประกอบด้วย ประชากรวัยผู้ใหญ่ชายและหญิง มีอายุ 30-72 ปี อาศัยอยู่ในเขตอำเภอเมือง จังหวัดขอนแก่นในช่วงปี พ.ศ. 2550 กลุ่มตัวอย่างจำนวน 296 คน ได้รับการวัดระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออก ตรวจช่องปาก และสัมภาษณ์ การวิเคราะห์ผลการศึกษากระทำโดยการพรรณนา และการวิเคราะห์ความสัมพันธ์ระหว่างตัวแปร โดยใช้สถิติสหสัมพันธ์ สเปียร์แมน (Spearman Correlation Test) ในการทดสอบระหว่างระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกกับสภาวะฟันผุ ถอน อุด และทดสอบความแตกต่างของค่าเฉลี่ยระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกระหว่างกลุ่มที่มีและไม่มีกระเป๋าปริทันต์ โดยใช้สถิติแมนน์วิทนีย์ (Mann-Whitney U Test)*

ผลการศึกษา: *ผลการศึกษาพบความสัมพันธ์เชิงผกผันระหว่างสภาวะฟันอุดกับระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกเฉพาะในกลุ่มผู้ที่สูบบุหรี่ แต่ความสัมพันธ์ดังกล่าวหายไป เมื่อทำการควบคุมด้วยตัวแปรระดับการศึกษาเพียงตัวเดียว*

อภิปรายผลการศึกษา: *ระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกอาจไม่เหมาะสมที่จะใช้เป็นตัวแทนสภาวะการสูบบุหรี่ในการศึกษานี้ อย่างไรก็ตาม เนื่องจากการศึกษานี้ยังอยู่ในวงแคบ จึงควรมีการศึกษาในวงกว้าง และมีการรวบรวมข้อมูลเกี่ยวกับตัวแปรที่เกี่ยวข้องมากขึ้น เพื่อเพิ่มอำนาจการทดสอบและขยายผลการนำไปใช้*

สรุป: *ไม่พบความสัมพันธ์ระหว่างระดับก๊าซคาร์บอนมอนอกไซด์ในลมหายใจออกกับสภาวะสุขภาพช่องปากทั้งในกลุ่มผู้ที่สูบบุหรี่และในกลุ่มผู้ที่ได้รับควันบุหรี่จากสิ่งแวดล้อม*