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Relationship between the concentration of volatile sulphur compound and periodontal disease severity in Nigerian young adults

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Abstract

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Background:

The aim of this study was to determine the relationship between the concentration of volatile sulphur compounds (VSC) in mouth air and the severity of periodontal diseases in young adults.

Materials and Methods:

A total of 400 subjects were studied. Estimation of periodontal disease severity was done using the basic periodontal examination (BPE) and the baseline measurement of the concentration of VSC in the mouth air of the subjects was done objectively using the Halimeter®.

Result:

The mean concentration of VSC for the group with BPE code 0 was 91.0 ± 5.9 parts per billion (ppb), 156.4 ± 9.4 ppb for BPE code 1, 275.2 ± 38.5 ppb for BPE code 2, 353.5 ± 72.3 ppb for BPE code 3, and 587.0 ± 2.1 ppb for BPE code 4 ($P = 0.001$). Majority (79.0%) of the subjects with BPE code 0 had concentration of VSC <181 ppb. Sixty-two (54.9%) with BPE code 1 had concentration of VSC <181 ppb, 34% with BPE code 2 had concentration of VSC <181 ppb and 42.9% with BPE code 3 had concentration of VSC <181 ppb. Only 6.5% of the subjects with BPE code 0 had VSC concentration >250 ppb, whereas all (100%) of those with BPE code 4 had VSC concentration >250 ppb ($P = 0.001$).

Conclusion:

It was concluded that a relationship exists between the periodontal pocket depth and the concentration of VCS in mouth air of young adults.

Keywords: Periodontal pocket, volatile sulphur compound, young adults

INTRODUCTION

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Volatile sulphur compounds (VSC) that include hydrogen sulphide (H_2S), methyl mercaptan, ethyl mercaptan, dimethyl sulphide, dimethyl disulphide, dimethyl trisulphide, and diethyl sulphide[1] have been implicated in the disruption of oral mucosa contributing to the progression of periodontal disease.[2]

They are highly toxic to tissues even at extremely low concentrations.[3] They alter the morphology and function of fibroblasts-like protein synthesis.[3,4,5,6] They also alter the metabolism of fibronectin.[7]

VSCs induce deleterious changes in non-keratinized epithelium, basement membrane, and underlying lamina propria[8] and speed up the degradation of collagen, delay the healing of existing wound, and affect periodontal cell functions.[8] They also decrease protein or collagen synthesis[4,6] and inhibit cell migration in periodontal ligament cells.[9] Also, VSC interfere with the enzymatic and immunological reactions leading to tissue destruction while showing an increase in the release of interleukin-1 (IL-1) and prostaglandin E2 (PGE2).[5]

They increase the permeability of oral mucosa[2] and also cause its breakdown,[8] leading to bacterial invasion. Although no inflammatory response could be initiated by topical application of lipopolysaccharides (LPS) to healthy gingival, exposure of the gingival tissues to H_2S facilitated penetration of LPS and resulted in inflammation.[10] It has been established that there is a transition of health to gingivitis when the lining epithelium, whose permeability has been enhanced by VSC, is invaded by bacterial antigens such as LPS.[11] The initial gingivitis progresses to periodontitis as both soft and hard tissues are affected. More tissue destruction leads to increase in the concentration of VSC, and this multiplier effect worsens the periodontal disease.

The review of literature revealed that few studies have been done on the relationship between the concentration of VSC in mouth air and periodontal health in Caucasian and Asian populations.[12,13,14,15,16,17,18] They were found in higher concentrations in gases emitted from mouths of patients with periodontal disease than in their healthy counterparts, therefore suggesting a relationship between VSC and periodontal diseases.[19]

This study is important because its results may serve as baseline values for Nigerian young adults, with and without periodontal disease. Also, the establishment of a relationship between VSC concentration and periodontal health may give rise to an objective and easily monitored therapeutic approach for the management of periodontal diseases, which is focused on the reduction of the intraoral bacterial load and the subsequent reduction of the concentration of VSC. This knowledge may also lead to the formulation of another approach that will focus on the conversion of VSC to their nonvolatile substrates. The aim of the study was to determine the relationship between the concentration of VSC in mouth air of young adults and the level of the health of their periodontium.

MATERIALS AND METHODS

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This was a descriptive cross-sectional study carried out on 400 patients who visited the Periodontology Clinic of the University of Benin Teaching Hospital, Benin City between January 2011 and December 2011. Only nonsmoker young adult (aged 20-40 years) with varying degrees of periodontal disease subjects, who gave informed consent were consecutively selected. Subjects with any underlying systemic disease were excluded. Data collected, included the demographic details of the subjects, estimation of their periodontal disease severity, and the level of the concentration of VSC in their mouth air.

The estimation of the periodontal disease severity was done by two calibrated examiners using the basic periodontal examination (BPE),[20,21] a modification of the Community Periodontal Index for Treatment Needs (CPITN).[22] The dentition was divided into sextants. For each sextant, only the highest score was recorded. Any sextant containing only one tooth was recorded as missing and the score for that tooth included in the adjacent sextant. Code 0 was given to the sextant where there were no pockets exceeding 3 mm in depth, no calculus or overhangs of fillings and no bleeding after gentle probing. Code 1 was given to the sextant where there were no pockets exceeding 3 mm in depth and no calculus or overhangs of fillings, but with bleeding after gentle probing. Code 2 was given to the sextant where there were no pockets exceeding 3 mm in depth, but dental calculus or other plaque retention factors were below the gingival margin. Code 3 was given to the sextant where the deepest pocket depth was

>3 mm, but <6 mm. Code 4 was given to the sextant with pocket depth of≥6 mm. Code* was given to a sextant if there was total attachment loss at any site of≥7 mm or if a furcation can be probed.

The baseline measurement of the concentration of VSC in the mouth air of the subjects was done objectively using the Halimeter®.[23] The Halimeter® reading, based on the method used in similar studies,[24,25] was categorized into three types, as follows: Normal = 0-180 parts per billion (ppb), weak = 181-250 ppb, and strong = >250 ppb.

The data was analyzed using the Statistical Package for Social Sciences (SPSS) version 15.0 for frequency distributions and cross-tabulation. Chi square test was done to test statistical significance. Mann Whitney U test (non-parametric test) was used to compare the mean of the concentration of VSC in the different groups with varying periodontal disease severity. *P* values less than 0.05 was considered statistically significant. Binary logistic regression was done to determine the correlation between independent variables, such as subject socio-demographic factors, on the severity of the periodontal disease. For the purpose of regression analysis, the subjects were grouped into two: without periodontitis (BPE 0-2) and with periodontitis (BPE code 3-4). The protocol for human participation was reviewed and approved by the Ethics and Research Committee of the University of Benin Teaching Hospital.

RESULTS

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Majority (75.5%) of the subjects who participated in this study were <35 years of age. The males:female ratio was 1.1:1 and majority (74.5%) of the subjects had tertiary level of education. Professionals made up 58.3%, while students constituted 21.5% [Table 1].

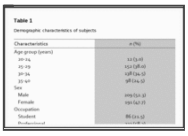

Table 1
Demographic characteristics of subjects

The mean concentration of VSC for the group with BPE code 0 was 91.0 ± 5.9 ppb, 156.4 ± 9.4 ppb for BPE code 1, 275.2 ± 38.5 ppb for BPE code 2, 353.5 ± 72.3 ppb for BPE code 3, and 587.0 ± 2.1 ppb for BPE code 4 [Table 2]. There was a statistically significant difference between the mean concentration of VSC for the group with BPE code 0 and other groups with BPE code 1-4. The group with BPE code 0 elicited statistically significant lower concentration of VSC than the groups with BPE code 1-4 (*P* = 0.001) [Table 2].

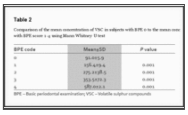

Table 2
Comparison of the mean concentration of VSC in subjects with BPE 0 to the mean concentration of VSC in subjects with BPE score 1-4 using Mann Whitney U test

Majority (79.0%) of the subjects with BPE code 0 had concentration of VSC <181 ppb. Sixty-two (54.9%) with BPE code 1 had concentration of VSC <181 ppb, 34% with BPE code 2 had concentration of VSC <181 ppb, and 42.9% with BPE code 3 had concentration of VSC <181 ppb. Only 6.5% of the subjects with BPE code 0 had VSC concentration >250 ppb, whereas all (100%) of those with BPE code 4 had VSC concentration >250 ppb (*P* = 0.001) [Table 3].

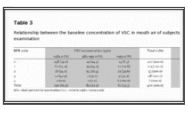

Table 3
Relationship between the baseline concentration of VSC in mouth air of subjects and basic periodontal examination

The binary logistic regression revealed that the strongest predictor of severe periodontal condition was increasing concentration of VSC ($P < 0.05$). Gender had no statistically significant explanatory power in explaining severity of periodontal disease ($P = 0.904$). Level of education also was not statistically significant ($P = 0.375$) [Table 4].

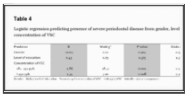

Table 4
Logistic regression predicting presence of severe periodontal disease from gender, level of education, and concentration of VSC

DISCUSSION

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The effect of age on the concentration of VSC is considered controversial. A previous study showed an age-related increase in both organoleptic and VSC measurements,[27] while another demonstrated that age did not contribute to VSC increase.[14]

The concentration of VSC in this study was not gender specific. This supports the finding that the age-related increase in both organoleptic and VSC measurements were similar in both males and females[27] and refutes the general assumption that periodontal disease may be more severe in males.[28]

The mean of the concentration of VSC in the subjects with periodontal pockets >3 mm is almost four times more than that of the subjects with normal periodontal pocket depth. This is similar to the result of a study that reported that the amount of VSC in the breath air of subjects with periodontal involvements was 8 times that of the control.[29] This study therefore suggests that persons with periodontal diseases may have a higher concentration of VSC.

It also appears that the deeper the periodontal pockets, the more is the concentration of VSC. The subjects with deeper periodontal pockets (>6 mm) all had a concentration of VSC >250 ppb. This study is similar to a previous study which reported that VSC in mouth air of the subjects increased with the increase in the number of periodontal pockets and as the depth of periodontal pockets become >3 mm.[30] Other clinical studies demonstrated the elevated VSC levels in periodontally involved pockets.[31,32,33] It is suggested that the deeper periodontal pockets habor more periodonto pathogens that cause more putrefaction.[33] Also, oral hygiene measures instituted by an individual with deeper periodontal pocket may not be optimal, especially when symptomatic. This may lead to poor plaque control, more periodonto pathogens, and, eventually, increase in the concentration of VSC whose production is basically by the action of bacteria on protein in the diet, saliva, and gingival fluid.[34]

CONCLUSION

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It can be concluded that a relationship exists between the periodontal pocket depth and the concentration of VCS in mouth air of young adults since the VCS concentration in the mouth air of young adults with deeper periodontal pockets was higher than that of young adults with shallow or no periodontal pockets.

Footnotes

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Source of Support: Nil

Conflict of Interest: None declared.

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