A multifactorial investigation of the ability of oral health care products (OHCPs) to alleviate oral malodour


Abstract

Aim, background: Oral malodour (halitosis) is generally ascribable to oral microbial putrefaction generating malodorous volatile sulphur compounds which predominantly comprise dihydrogen sulphide and methyl mercaptan. This study assesses the relative effectiveness of 6 oral health care products in reducing oral cavity volatile sulphur compound concentrations.

Method: A mixed model 3-factor factorial experimental design involving 6 volunteers, 7 treatment regimens (products I–VI* and water placebo) and 5 time-points (0.00–5.29 h) was undertaken. Electron-donating volatile sulphur compound levels were determined in triplicate using a sulphide monitor (Interscan model 1170) both prior to (0.00 h) and following oral rinsing (20 ml of 5 of the products) or chewing (2 capsules of the remaining product) episodes with each product examined (0.29, 1.29, 2.29 and 5.29 h post-administration).

Results: Results were recorded as peak and steady-state volatile sulphur compound equivalents (ppb). With the exception of one of the products, each oral health care product tested was found to reproducibly reduce volatile sulphur compound concentrations within 20 min of treatment; the mean % decreases in peak (and corresponding steady-state) levels ranging from 3.6 (0.0) to 16.8 (16.4)%.

Subsequently, volatile sulphur compound concentrations returned to their zero-control (baseline) values within 5 h, the rate of this regression being in the reverse of the order observed for the magnitude of the primary 20 min reduction for both peak and steady-state measurements. As expected, the water placebo exerted no influence on oral cavity volatile sulphur compound levels. The most effective oral health care products contained admixtures of chlorite anion and chlorine dioxide (both of these agents have the ability to directly oxidise volatile sulphur compounds to non-malodorous products and the latter is also powerfully cidal towards odouric micro-organisms).

Conclusions: We therefore conclude that oral health care products containing such oxohalogen oxidants may provide a useful therapeutic strategy for the treatment of oral malodour.

Key words: halitosis, volatile sulphur compounds, halimeter, oral health care products, periodontology, oxohalogen oxidants.

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Oral malodour (halitosis, bad breath) is a very frequent human condition which primarily affects the adult population

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(Tonetzich 1977). In general, cases of this aesthetically-unacceptable disorder are attributable to microbial putrefaction occurring in the oral cavity (usually within anaerobic loci) (McNamara et al. 1972, Tonetzich & McBride 1981), a process giving rise to the generation of malodorous volatile sulphur compounds (VSCs) which predominantly comprise dihydrogen sulphide (H₂S) and methyl mercaptan (CH₃SH) (Tonetzich 1977, Kleinberg & Westbay 1990). Periodontal diseases, excessive bacterial colonisation of the tongue,
unclean dentures, erroneous dental restorations and limited salivary flow can result in halitosis of oral aetiology (Kostelec et al. 1984; Morris & Read 1939, Toonzetich 1978, Spouge 1964, Prinz 1936, Tachibana 1959, Yagasaki & Sanada 1992), whilst upper and lower respiratory tract conditions, various systemic diseases, gastrointestinal and neurological diseases, and the therapeutic application of selected drugs are common non-oral aetiologies (Atia & Marshall 1982).

Evaluations of the nature and level of oral malodour require reliable, sensitive, accurate and precise experimental techniques and previously-reported methods of assessment have involved (1) organoleptic (subjective) systems (Tsunoda et al. 1981, Ishikawa et al. 1984), (2) time-consuming and labour-intensive measurement of VSCs via gas chromatography (GC) coupled with flame-photometric detection (Solis-Gaffar et al. 1975), (3) a combination of (1) and (2) above (Schmidt et al. 1978), (4) cryo-osmoscopy (Diening et al. 1939), or (5) the numerous culture of plaque and periodontal pocket exudates in appropriate bacteriological media (Tachibana 1957). To date, however, only a limited amount of experimental data are available on the application and reproducibility of such methods, and parameters such as the menstrual cycle, heterogeneity in oral hygiene control, circadian variation, smoking habits and climate may influence results acquired from such investigations (Rosenberg & McCulloch 1992). Furthermore, subsequent to the evacuation of malodorous gases in the oral cavity, the rate and extent of their restoration to this environment remain speculative.

Recent reports have outlined the applications of a portable industrial H₂S monitor (Halimeter) for determining oral malodour gases (Rosenberg et al. 1991a, b) and highly significant correlations between measurements obtained therefrom and organoleptic ratings made by a total of 7 judges have been demonstrated (Rosenberg et al. 1991a). This electro-chemical VSC detector contains a voltammetric sensor which draws a sample of oral gas across an electrocatalytic sensing electrode operating at a potential of +0.50 V, a value sufficient to ensure the complete oxidation of electron donating thiols such as CH₃SH and H₂S (Redox potentials (Eₚ₅₀) of thiol/disulfide couples generally lie in the −0.20 to −0.40 V range).

Such electrochemical reactions generate an electric current, the magnitude of which is directly proportional to the total chemically-reducing, gaseous VSC concentration. This current is converted to a voltage which in turn is transferred to a meter which gives VSC levels in parts-per-billion (ppb) throughout a range of 0–1000. Measurements made with this novel device have been shown to be more reproducible than those acquired by subjective, organoleptic panel methods and more sensitive to diminishments in oral malodour levels induced by rinsing with oral health-care products (Rosenberg et al. 1991a, 1992). Moreover, this VSC monitor has many advantages over complex GC methods, including substantially lower cost, rapid sample throughput, facile portability, no requirements for the experimental employment of specialist technical staff, and the appropriateness of the manner in which samples are collected.

In this investigation, we present data concerning the relative effectiveness and longevity (up to 5.3 h post-administration) of a total of 6 commonly-utilised, commercially-available OHCPs (I–VII) in suppressing oral malodour (electron-donating VSC levels in air directly sampled from the oral cavity, determined by the portable industrial sulphidihalomonitor described above). The VSC-neutralising capacity of each product is discussed in detail with special reference to their chemical compositions, e.g., those containing agents which are cidal towards odourgenic micro-organisms and/or have the ability to directly oxidise VSCs to non-malodorous products.

Material and Methods

Volatil sulphur compound (VSC) determination

Measurements of total reducing VSCs were made on a portable industrial sulphide monitor (Interscan model 1170, 1.00 p.p.m. full scale) without a scrubber attachment (enabling the combined determination of both H₂S and CH₃SH (Rosenberg et al. 1991a, b). This instrument was zeroed on ambient air and measurements were performed by inserting a 6.5-mm diameter disposable plastic straw approximately 4 cm into the partially opened oral cavity. Volunteers were required to refrain from talking for >5 min prior to measurement and to breathe through their nose during the VSC determination. Results were recorded as both peak (maximum) and steady-state parts-per-billion (ppb) electron-donating VSC equivalents, and each measurement was made in triplicate (repeated triplicate determinations were completed within 6 min). Periodic calibration of the halimeter sensor was conducted by the manufacturer at 4-month intervals, a procedure involving an appropriate H₂S gas concentration reference standard. In this manner, the existing sensitivity of the monitor (output signal/ppb) is measured and on return to our laboratory the instrument calibration control was adjusted accordingly to achieve sensor calibration.

Oral health care products (OHCPs) investigated

The OHCPs tested in this study consisted of products I–VI. Volunteers were required to rinse with each mouthwash for 30 min (I, II, III, IV or V), or chew with 2 capsules of product VI, for a period of 30 s. Each volunteer also rinsed with an equivalent volume of tap water which served as a placebo control.

Patient population

This investigation involved 6 male volunteers ranging in age from 21 to 53 years (mean age=standard deviation; 32.3±11.1 years). Prior to the testing period for each commercial oral health care product (5.3 h), each participant was requested to refrain from oral activities (i.e., eating, drinking, tooth brushing, oral rinsing, smoking, etc.) for at least 4 h. Electron-donating VSC levels were determined both prior to (0.00 h) and following oral rinsing/chewing episodes with each OHCP examined (0.29, 1.29 and 2.29 h post administration). The first (baseline) measurement was made at 10.00 am and all participants agreed to avoid their early morning breakfast meal on the days in which they were involved in

* The oral health care products tested here were: I. redhead™ oral rinse; II. Profresh™ oral rinse; III. Oxyfresh™ oral rinse; IV. Listerine Mint™ oral rinse; V. Scope™ oral rinse; VI. Breath Assure™ capsules.

* See previous footnote.
these experiments, together with all of the other oral activities specified above.

Immediately subsequent to the above 2.29 h measurement, volunteers consumed a lunchtime medium spicy Chinese chicken curry meal and a further oral cavity VSC concentration determination was made at the 5.29 h time-point (for every oral-health care product tested, participants consumed equivalent quantities of exactly the same class of foosuff).

Administration of the OHCPs investigated to each of the 6 participants was staggered over time and the "washout" period between each product was at least 3 days. During these "washout" periods, it was ensured that the volunteers resumed their normal oral health care activities.

Experimental design and statistical analysis

The experimental design for this investigation classified as a mixed model, 3-factor system with treatments (OHCPs together with a water placebo) and times at which measurements were made being fixed effects at 7 and 4/5 levels respectively (the latter dependent on the transformation/data-editing system employed prior to statistical analysis), and volunteers (n=6 in total) being a random effect. A mixed model component analysis for experimental peak level or steady-state VSC halitosis data comprising 8 sources of variation (3 fixed effects, 3 1st-order interactions, fundamental error and that between replicate (3) instrument measurements), the nature of these effects (fixed or random) and parameters estimated is shown in Table 1 (Cochran & Cox 1950, Snedecor 1935).

2 transformation procedures were performed on data prior to statistical analysis. Firstly, a logarithmic transformation was employed since, for concentration data, the standard deviation associated with each determination tends to increase proportionally with the measurement's magnitude. Experimental data were then subjected to analysis-of-variance (ANOVA) according to the mixed model delineated in Table 1 (n=630). Secondly, in order to minimise the "between volunteers" component of variance (random effect), and the 1st-order treatments×volunteers and × volunteers' interactions (variance components utilised to test of the main (fixed) effects), each observation was expressed as a proportion (ratio) of the mean of replicates 0.00 h time-point (control) value for each participant prior to statistical analysis according to the model depicted in Table 1 (n=504 for the 0.29–5.30 h time-points only). Subsequent to application of the above ANOVA procedures, further analysis of experimental data involved comparative evaluations of mean logₐ₂₆ or ratio-transformed oral cavity VSC levels and their associated 95% confidence intervals at increasing time-points for each oral health care product administered.

Prior to determining the statistical significance of the relationship existing between the peak and steady-state VSC level determinations (raw data in ppb), the normalities of the complete data sets were examined using the Kolmogorov-Smirnov test. For both the raw peak and steady-state level data, the significance of the $J$ statistic obtained was <1%. Hence, in view of the apparent non-normality of these data sets, a Spearman's rank correlation coefficient ($r_s$) was computed to ascertain the degree of correlation between the peak and steady-state concentration variables.

These deviations from normality also justify the transformations applied to data sets prior to conducting the multifactorial ANOVA described above.

Results

Multifactorial analysis-of-variance (ANOVA) of the log₂₆-transformed steady-state oral cavity VSC level data obtained according to the component analysis outlined in Table 1 revealed that the differences observed between the main effects (treatments (fixed), time-points (fixed) and volunteers (random)) were all statistically significant (Table 2a). However, the 1st-order, 2-factor treatments×time-points interaction component tested was also highly significant ($p<0.0005$), confirming that it is not attributable to sampling variation and that the experimental data acquired demonstrate the individuality of OHCP-induced responses at the different time-points selected for oral cavity VSC determinations, i.e., the contribution of a selected time-point variance component to the VSC level for one of the products tested significantly differs from its contribution under one or more further treatments (or the H₂O placebo test group). Indeed, the multifactorial experimental design utilised here (Table 1) is primarily employed to detect such effects in addition to testing the statistical significance of the main effects, the additivity of which is eliminated by the presence of such interactions.

Similarly, multifactorial ANOVA of the ratio-transformed steady-state level data (Table 2b) demonstrated the statistical significance of each of the main effects.

Multifactorial ANOVA of the peak level oral cavity VSC measurements (previously subjected to both log₂₆- and ratio-transformations as described for the steady-state concentrations) also provided much valuable information regarding the statistical significance of the main effect factors and their corresponding first-order treatments×time-points interaction (Table 3). Clearly, for both classes of transformation all 3 main effects and the above 2-factor interactions are highly significant (that attributable to differences between volunteers being extremely so), with the exception of the "between treatments" effect in the ANOVA conducted on the ratio-transformed data where $p<0.10$.

2-dimensional diagrams exhibiting plots of the normalised peak level oral cavity VSC levels (i.e., determinations expressed relative to their mean 0.00 h

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### Table 1. Component analysis of experimental peak level/steady-state halitosis data: mixed model

<table>
<thead>
<tr>
<th>Effects</th>
<th>Nature</th>
<th>Parameters estimated for mixed model</th>
</tr>
</thead>
<tbody>
<tr>
<td>A=treatments</td>
<td>fixed</td>
<td>$\sigma^2 + \sigma_{AB}^2 + \sigma_{BC}^2 + \sigma_{AC}^2$</td>
</tr>
<tr>
<td>B=times</td>
<td>fixed</td>
<td>$\sigma^2 + \sigma_{AB}^2 + \sigma_{BC}^2 + \sigma_{AC}^2$</td>
</tr>
<tr>
<td>C=patients</td>
<td>random</td>
<td>$\sigma^2 + \sigma_{AB}^2$</td>
</tr>
<tr>
<td>AB interaction</td>
<td>random</td>
<td>$\sigma^2 + \sigma_{AC}^2$</td>
</tr>
<tr>
<td>AC interaction</td>
<td>random</td>
<td>$\sigma^2 + \sigma_{BC}^2$</td>
</tr>
<tr>
<td>BC interaction</td>
<td>random</td>
<td>$\sigma^2 + \sigma_{AC}^2$</td>
</tr>
<tr>
<td>error</td>
<td>random</td>
<td>$\sigma^2$</td>
</tr>
<tr>
<td>between replications</td>
<td>random</td>
<td>$\sigma^2$</td>
</tr>
</tbody>
</table>

$\sigma^2$ (no. treatments), $b=4$ or 5 (no. time-points), $c=6$ (no. patients).

3 repeated halimeter measurements made at each time-point.
Table 2. Multifactorial analysis of variance (ANOVA) tables for (a) log transformed steady-state oral cavity VSC concentration data.

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) treatments (A)</td>
<td>6</td>
<td>4.2297</td>
<td>0.7050</td>
<td>2.99</td>
<td>&lt;0.025</td>
</tr>
<tr>
<td>(B) times (B)</td>
<td>4</td>
<td>0.1431</td>
<td>3.33 x 10^{-2}</td>
<td>3.75</td>
<td>&lt;0.025</td>
</tr>
<tr>
<td>(C) patients (C)</td>
<td>5</td>
<td>1.7220</td>
<td>0.3444</td>
<td>120.84</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>A x B interaction</td>
<td>24</td>
<td>0.2586</td>
<td>1.08 x 10^{-2}</td>
<td>3.79</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>A x C interaction</td>
<td>30</td>
<td>7.6476</td>
<td>0.2553</td>
<td>40.69</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>B x C interaction</td>
<td>20</td>
<td>0.1908</td>
<td>0.95 x 10^{-3}</td>
<td>9.54 x 10^{-3}</td>
<td>120.84</td>
</tr>
<tr>
<td>error</td>
<td>120</td>
<td>0.3415</td>
<td>0.0528</td>
<td>9.54 x 10^{-3}</td>
<td>120.84</td>
</tr>
<tr>
<td>total</td>
<td>209</td>
<td>13.8504</td>
<td>1.8 x 10^{-3}</td>
<td>120.84</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>between replicates</td>
<td>629</td>
<td>0.2280</td>
<td>5.43 x 10^{-4}</td>
<td>120.84</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>total</td>
<td>629</td>
<td>14.1783</td>
<td></td>
<td>120.84</td>
<td>&lt;0.0005</td>
</tr>
</tbody>
</table>

Abbreviations: df, degrees of freedom; SS, sum of squares; MS, mean square; F, F-variance ratio statistic; p, probability of such an F-value arising purely by chance.

Table 3. Significance of main effects and 1st-order interactions for log transformed steady-state oral cavity VSC data; abbreviations as Table 2.

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Log_{10}-transformed data</th>
<th>Ratio-transformed data</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) treatments (A)</td>
<td>df</td>
<td>&lt;0.0002</td>
</tr>
<tr>
<td>(B) time-points (B)</td>
<td>4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>(C) volunteers (C)</td>
<td>5</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>A x B interaction</td>
<td>24</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

(time point control values: means of 3 replicate determinations) versus time (h) for each participant involved in this investigation for all oral health care products tested clearly indicated the individuality of volunteers' responses to the different products tested in this investigation (data not shown).

Table 1 shows plots of the response of both peak level (maximum) and steady-state oral cavity VSC levels to the therapeutic administration of the six products tested (together with the water placebo) versus time. Each data point represents the mean level arising from summation of VSC concentrations (previously normalised to the zero-control values) across all volunteers and replicated measurements (n=18 for each time-point), and 95% confidence intervals for these mean values are given.

With the exception of product (VI), each OHCP examined was found to produceably reduce VSC concentrations within 20 min of treatment, the mean percentage decreases in peak (and corresponding steady-state) levels being in the order IV > V > III > II > I > 0. (21.9%) > III > IV > V > II > I. Subsequently, VSC concentrations commenced a return to their 0.00 h control (baseline) values, the prior administration of products II-V giving levels at or close to that of the zeroed baseline (i.e., within 5%) at the 2.29 h time-point. Product I, however, effectively retained oral cavity VSC concentrations significantly below the baseline value and only regressed to the 0.00 h control level 3 h subsequent to consumption of the standard lunchtime meal. The rate of this 0.29–2.29 h regression in VSC concentration was in the order IV > V > III > II > I (i.e., the reverse of the order given for the magnitude of the 20 min oral rinse-mediated decrease in oral cavity VSC levels) for both peak and steady-state measurements.

As expected, the water placebo exerted little or no influence on oral cavity VSC levels. However, administration of product VI gave rise to a 6.4 (12.6%) elevation in this parameter at the 0.29 h time-point, values which continued to rise for a 5 h period thereafter.

Interestingly, product II and, to a lesser extent, product IV appear to give rise to an excessive ‘ rebound' in VSC concentrations to values greater than the zeroed baseline subsequent to the meal consumed immediately after the 2.29 h time-point. However, although factors arising from the consumption of a spicy lunchtime meal may transiently elevate the level of halimeter-receptive oral cavity volatile thiols or H2S, these observations are not simply explicable in view of the post-meal data obtained in the H2O placebo test group, and hence further investigations are required to ascertain their significance.

A plot of peak level (maximum) oral cavity VSC levels versus their corresponding steady-state concentrations (ppb) revealed that these parameters were strongly correlated (r = 0.6284, p = 0.0001). The top right-hand corner of this plot exhibited a cluster of data points ascribable to one of the volunteers who registered high peak and steady-state VSC concentrations (when expressed relative to those of the other 5 participants) both before, and at increasing time-points subsequent to treatment with each product. However, removal of this cluster failed to enhance this correlation (r = 0.6093, p = 0.0001), although it should be noted that the normality of each raw data set was improved (i.e., lower values of the Kolmogorov-Smirnov d-statistic were obtained).

Discussion

In this investigation, the capacity of 6 OHCPs to alleviate oral cavity VSC levels in 6 human volunteers was examined. Of the formulations tested, the three containing the resonance-stabilised free radical species chlorine dioxide (ClO2) in admixture with relatively high levels of chlorite anion (ClO2-) were rated first, second and third (I>II>III oral rinses) in terms of their ability to diminish oral cavity VSC concentrations and hence neutralise oral malodour. However, for the most effec-
the pH value can be <5.0, additional ClO₂⁻ can, in principle, be generated from the high levels of ClO₂⁻ present in oxohalogen-containing oral rinse preparations, a phenomenon potentially facilitating the oral malodour-neutralising actions of such products. The 3 oxohalogen oral rinses evaluated in this investigation contain variable levels of ClO₂⁻ (1–20 p.p.m.=1.48×10⁻⁵–2.96×10⁻⁴ mol·dm⁻³) and products I and II include much higher levels of ClO₂⁻ (e.g., 0.10% w/v) (1.48×10⁻² mol·dm⁻³) for I), i.e., oxidant concentrations sufficient to 'swamp' those of oral cavity electron-donating VSCs during a single oral rinsing episode with a 20 ml volume.

\[
\text{ClO}_2^- + \text{H}^+ \rightarrow \text{HClO}_2 \quad (4)
\]

\[
4\text{HClO}_2 \rightarrow 2\text{Cl}_2\text{O}_3^- + \text{Cl}^- + 2\text{H}^+ + 2\text{H}_2\text{O} \quad (5)
\]

Since both ClO₂⁻ and ClO₂⁻ readily oxidise the amino acid methionine to its corresponding sulphoxide, these oxidants are also likely to effect oxidation of the volatile thioether compound dimethyl sulphide (also a contributor towards oral malodour [Tonzetich 1977]) to a non-malodorous sulphoxide adduct (eq. (6)).

\[
\text{ClO}_2^-/\text{ClO}_2^- + \text{CH}_2\text{SCH}_2 \rightarrow \text{CH}_2\text{S(OCH}_3\text{)_2} \quad (6)
\]

A plausible explanation for the greater effectiveness of product I over product II in suppressing oral cavity VSC levels is that the latter O1HCP contains high levels of xylitol (added as a sweetener), an agent which may act as a significant scavenger of ClO₂⁻ in view of the documented reactivity of carbohydrates with this oxohalogen free radical species (slow reactions which yield carboxylate anion/carboxylic acid functional groups) (Masaenchelien 1979, Becker et al. 1965). However, product I contains only citrate and phosphate buffering agents (species with little or no reactivity towards ClO₂⁻ and ClO₂⁻), and, a detailed multicomponent chemical analysis of this product has revealed the presence of only trace levels of acetate, formate, methanol and ethanol impurities (Lynch et al. 1997).

In view of the enhanced VSC inactivating activity of oxohalogen oxidant-containing oral rinse preparations over those containing conventional bacterial agents (e.g., IV and V), data acquired in this study clearly indicate that much of the benefit of ClO₂⁻ and ClO₂⁻
to the periodontium arises from their ability to directly oxidise electron-donating VSCs.

In addition to the highly significant differences found between each of the treatments, sampling times and patients main effects, multifactorial statistical analysis of the log_{10}-transformed steady-state oral cavity VSC concentration data also revealed that the 1st-order treatments × time-points interaction was highly significant, a phenomenon clearly demonstrating differences in the magnitude and direction of the VSC response curves displayed in Fig. 1. However, for the ratio-transformed steady-state level data, no evidence for the contribution of the treatments × time-points interaction towards the oral cavity VSC level response was obtained. Hence, for steady-state level determinations, it appears that the removal of the 0.00 h time-point data in this case (a consequence of the transformation applied to data prior to analysis) effectively diminishes the contribution of the treatments × time-points interaction to the non-additivity of data examined. As expected, for both transformation classifications differences between volunteers accounted for a high proportion of the oral cavity VSC level variation.

High levels of ethanol represent a source of interference with Halimeter VSC measurements and hence determinations made subsequent to oral rinsing episodes with OHCPs incorporating this alcohol as a solvent for the purpose of retaining active ingredients therein in the solution phase should also be interpreted with some degree of caution. However, with the exception of product VI, we found that products containing excessive, interfering levels of such agents gave rise to a rapid, substantial elevation in Halimeter readings subsequent to oral rinsing episodes and such products were therefore excluded from the investigation. Indeed, this phenomenon served as an exclusion criterion for the incorporation of oral health care products into the trial, and was not observed directly after oral rinsing episodes with all the formulations rigorously tested and reported in this study.

Similarly, we also found that cigarette smoke markedly interferes with Halimeter determinations and hence any smokers included in the investigation refrained from smoking for a period of at least 4 h prior to the commencement of oral cavity VSC measurements.

The finding that administration of product VI resulted in an increase in oral cavity VSC levels at the 0.29 h time-point could be explained by the previous observation that the edible vegetable oils present in this product interfere with the accurate determination of VSCs by the Halimeter employed in this study (Rosenberg et al. 1991b).

Although additional disadvantages of the portable VSC monitor such as lack of chemical specificity (i.e., an inability to distinguish between individual oral cavity VSCs, in contrast to established gas chromatographic methods) and the possibility that further gaseous electron-donating agents may also respond to the electrocatalytic sensing electrode, it is the authors’ view that the advantages associated with the employment of this device far outweigh the above limitations or potential sources of error.

Currently, there is much evidence available to suggest that VSCs play an important pathogenic role in promoting the periodontal breakdown process. Indeed, as might be expected from its ability to participate in redox and thiol-disulphide interchange reactions, CH_SH influences enzymic and immunologic activities in manners that give rise to periodontal tissue destruction (Brunette 1996) and also enhances the permeability of oral mucosa, the latter being a process which facilitates the penetration of hazardous components such as endotoxins into tissues (Ng & Tonzecht 1984). At concentrations similar to those generated in periodontal pockets, CH_SH have the capacity to exert effects concerning the activity and integrity of cells; e.g., this VSC can modify cell shape and cytoskeletal patterns, diminish cell proliferation and migration, alters collagen metabolism and interferes with protein synthesis (Brunette 1996). Moreover, VSCs have been shown to retard wound healing processes (Yangski 1995). Hence, an effective oxidative consumption of such deleterious agents may suppress the disease’s progression. Indeed, the results obtained in this investigation indicate that periodic administration of oxohalogen-containing oral rinse formulations (e.g., at 2-3 h intervals) could exert a protective role against periodontal diseases in addition to their roles in neutralising oral malodour, although detailed clinical investigations will be required to test this hypothesis.

Offensive breath odours predominantly arise in the mouth, a major source being a putrefying post-nasal drip located at the tongue dorsum. However, periodontal health, diminished levels of oral hygiene and the accumulation of mouth debris are also important contributors to oral malodour (Tonzecht & Kestenbaum 1969, Tonzecht 1973, Kostelec et al. 1981). Residues of inherently malodorous foodstuffs remaining in the mouth can also be responsible, as can odouriferous food components which penetrate the systemic circulation subsequent to ingestion and consequently contaminate oral cavity air via blood-gas exchange in the pulmonary alveoli (e.g., aldehyde sulphide present in garlic). Nonetheless, such extrinsic sources of oral malodour are dietary-dependent, discontinuous and contribute only minimally to the process, whereas the bacterially-mediated catalysis of endogenous precursors (specifically cysteine and methionine) serves as the predominant source of breath odours commonly perceived to be offensive. Intriguingly, the actions of L-cysteine desulphhydrase in gram-negative anaerobes such as Fusobacterium spp. and Bacteroides spp. on the VSC precursor L-cysteine generates H:S, ammonia (NH_3) and pyruvate via an α,β-elimination reaction (Claesson et al. 1990), and the latter α-keto acid anion, readily detectable in human saliva and certain dentin by the application of high resolution proton (1H) nuclear magnetic resonance (NMR) spectroscopy (Lynch et al. 1997), may partially reflect the nature and level of VSC generation by such micro-organisms. Similarly, CH_S, NH_3 and α-ketobutyrate arise from an α,β-elimination of L-methionine catalysed by the enzyme L-methionine-lyase in the above bacteria, and salivary α-ketobutyrate effectively serves as a marker of such microbial activity (Claesson et al. 1990).

In conclusion, OHCPs containing admixtures of ClO_2 and ClO_2- (both of which have the ability to directly oxidise VSCs to non-malodorous products and the latter is also powerfullyidal towards odourigenic micro-organisms) give rise to small but significant reductions in oral cavity VSC levels, and the application of such products may provide a useful therapeutic approach to the management of oral malodour.

However, it should be noted that since organoleptic assessments were not performed in this investigation, the overall effectiveness of these oxohalogen-
Zusammenfassung

Eine multifaktorielle Untersuchung zur Linde-
zung der Mundgeruchs durch orale Gesund-
heitsprodukte (OHCPS).

Der schlechte Mundgeruch (Halitose) wird allgemein der Zersetzung der oralen Mund-
flora zugeschrieben, wobei schlecht riechende
flüchtige Schwefelbestandteile mit vorherr-
schenden Dihydrosulfophenyl- und Methyl-
mercaptanbestandteilen entstehen. Diese Stu-
die überprüft die relative Effektivität von 6
oralen Gesundheitsprodukten bei der Reduk-
tion der Konzentration von flüchtigen Schwefel-
bestandteilen der Mundhöhle. Ein gemisch-
tes Modell mit einem 3-Faktoren experiment-
tellem Design, was 6 Probanden, 7 Behandlungsgruppe (Produkte 1-6) und Wäs-
erstrahlung und 5 Zeitpunkte (0.00-5.29 h) enthielt, wurde genutzt. Elektrolytische be-
stimme flüchtige Schwefelbestandteile wur-
den dreifach unter Nutzung eines Sulphido-
monitor (Intercam Modell 1700) bestimmt so-
wohl vor (0.00 h) als auch nach der oralen Spülung (20 ml von einem der 5 Produkte)
der Kauen (2 Kapseln mit dem enthaltenen Produkt) mit jedem zu prozidenten Produkt
(0.29, 1.29, 5.29 h nach der Anwen-
dung). Die Ergebnisse wurden als Spitzens
und als Geruchswert (Aquavol) der flüchtigen
Schwefelbestandteile aufzeichnet (ppb).

Mit Ausnahme von einem der Produkte redu-
zerte jedes der getesteten Produkte die flüch-
tigen Schwefelbestandteile innerhalb von 20
Minuten der Behandlung reproduzierbar. Der
mittlere relative Abfall in den Spitzens
korrespondiert im Geruchswert schwankte von 3.8 (0.0) bis zu 16.8 (16.4)%

Ausschließlich ging die Konzentration der flüchtigen Schwefelbestandteile zu ihren Null-
kontrollwerten (Blasenl) innerhalb von 5 h
zurück. Die Art dieser Regression war entge-
gesetzt mit der Regressionsrate, die für das Aus-
maß der Reduktion in den ersten 20 Minuten
sowie für die Spitzes als aus der Geruchswert-
zeugmassenmessungen beobachtet wurde. Wie
erwartet hatte der Wasserspülzahn keinen Ein-
fluß auf die oralen flüchtigen Schwefelbe-
standteil-Konzentrationen. Die effektivsten
oralen Gesundheitsprodukte enthielten Mi-
schungen von Chloramphenicol und Chloram-
phicol (beide aus diesen Arzneien haben die Ei-
genschaft zur direkten Oxidation der flüchtig-
en Schwefelbestandteile zu nicht schlecht
reichenden Produkten und das letztere ist

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