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Aroma Release and Delivery Following the Consumption of Beverages

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Processes controlling aroma release and delivery during and after the consumption of a beverage were studied using real-time physiological and aroma release measurements. The key processes were as follows. During swallowing, a portion of the buccal gas phase was transferred first to the throat and then to the nasal passages via the tidal breath flow. This mechanism accounted for the sharp pulse of aroma seen at the beginning of the swallow breath and on subsequent swallows. The persistence effect was due to liquid−air partition from beverage coated on the throat and was dependent on the concentration of volatile compounds in the beverage. Lipid in the beverage caused a decrease in the intensity of volatile compounds on the breath, but the presence of a thickening agent had no effect on persistence.

KEYWORDS: Aftertaste; flavor release; swallowing; aroma; odorant transport; API-MS; MS Nose

INTRODUCTION

Flavor perception of foods occurs when flavor compounds are released from foods and then transported to the appropriate receptors in the mouth and nose. Oral processing is the initial step that releases flavor from food; flavor is then transported to the receptors via processes that are affected by a wide range of physiological and physical factors (*1*, *2*). The proposed sequence of events starts in the mouth with the release of aroma compounds from the food into the mouth liquid phase. Partition from the liquid phase to the gas phase then occurs, and portions of the mouth gas phase are transferred to the throat during swallowing (*3*). Delivery from the throat to the olfactory receptors is achieved by two mechanisms. The first is a rapid, direct transfer of gas-phase aroma compounds to the olfactory receptors caused by the swallowing action. This is sometimes referred to as the "swallow breath". The second involves a slower partition of aroma compounds from the liquid phase, which now lines the throat, into the tidal air stream and subsequent delivery to the olfactory receptors during exhalation (*4*). This second mechanism is thought to be responsible for the persistence of aroma delivery after swallowing. These two processes are responsible for the profiles observed in breath-by-breath profiles (*5*) when a solution is placed in the mouth and then swallowed. A high concentration of aroma is seen on the first breath, followed by a much lower concentration on subsequent breaths, which show an exponential decrease. The first breath concentration is very variable (*5*), presumably because it depends on the exact way the person swallowed the solution (their swallowing action) as well as factors such as different mouth volumes.

Some data to support this sequence of events can be found in the scientific literature, but most attention has focused on the persistence aspect. The origin of the large first breath peak was investigated by monitoring swallowing, aroma release in the nose, and nasal air flow simultaneously. From these measurements the sequence of events was confirmed, and estimates of some physiological parameters such as mouth volume were obtained (*6*). The origins of persistence have also been studied using imaging methods such as videofluoroscopy and magnetic resonance imaging (MRI) (*7*), which showed the formation of a viscous salivary coating on the back of the tongue after swallowing, which was proposed as a potential odorant depot, prolonging the release of odorants into the tidal breath stream. Other studies have reported the development of afterodor following the consumption of wine (*8*). Linforth (*9*) obtained breath-by-breath traces after the consumption of aroma solutions. A QSPR approach was used (*9*) to model the persistence of several volatiles, and hydrophobicity, volatility, ether linkage, and carbonyl count were identified as important factors controlling the persistence. Normand (*4*) also obtained breath-by-breath data but treated it as two regimes—one relating to the initial release of volatile compounds and the second relating to persistence. A consistent model for the initial phase was not achieved, but the second, persistence phase was modeled successfully. Other attempts at modeling the persistence, using a power law function (eq 1), encountered the same problem (*6*).

$$C = C_1 t^{-P} \quad (1)$$

C is the concentration in the breath, C_1 is the concentration in the breath 1 min after the beverage has been consumed, t is the time, and P is the decay exponent.

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The purpose of this paper is to study the relative contributions of the gas and liquid phases to aroma transport and to determine how the composition of the liquid phase can affect aroma persistence in vivo. It is known from other studies that release from such systems depends on the composition of the thin film [e.g., the lipid content (*10*)] or the gas flow rate through the system (*11*). It is also intuitive that the viscosity of the thin film may affect the residence time in the throat and hence the persistence of the aroma release. A model beverage was chosen as it is ideal for studying the two aroma transport mechanisms. A range of volatiles with different hydrophobicities were used along with a lipid emulsion system and a viscosifying agent.

MATERIALS AND METHODS

Chemicals. All of the volatile compounds (99% purity; isoamyl acetate, acetaldehyde, hexanal, benzaldehyde, ethyl butyrate, ethyl hexanoate, menthone, carvone, 2,5-dimethylpyrazine, and ethanol) were obtained from Firmenich SA (Geneva, Switzerland). Hydroxypropylmethylcellulose (HPMC) was obtained from Dow (Schwalbach, Germany; tradename Methocel).

HPMC Solution Preparation. HPMC solutions were prepared by dispersing 5 and 10 g of HPMC, respectively, in water (400 mL at 80 °C) and allowing the solutions to cool to 4 °C (with constant stirring). Aqueous solutions of the volatiles (500 mg/kg) were prepared with vigorous shaking, using an SF1 flask shaker (Stuart Scientific, Redhill, U.K.). These were then mixed with the two HPMC solutions and distilled water to give 100 mg/kg solutions of each of the volatiles (2,5-dimethylpyrazine and menthone) in 0, 10, or 20 g/kg HPMC. These final solutions were left to mix overnight on a roller bed (Stuart Scientific, SR2).

Emulsion Preparation. A 400 g/kg Neobee (coconut oil) emulsion was supplied by Firmenich SA, which had a symmetrical particle size distribution; 90% of the particles were $<1.0\ \mu$m. Aqueous solutions of the volatiles were prepared (200 mg/kg) with vigorous shaking, using an SF1 flask shaker (Stuart Scientific). These solutions were then mixed with aliquots of the 400 g/kg Neobee emulsion and/or distilled water to give 100 mg/kg solutions of each of the volatiles (2,5-dimethylpyrazine and menthone) that contained either 0, 4, or 20 g/kg Neobee (lipid). These final solutions were left to mix and equilibrate overnight on a roller bed (SR2, Stuart Scientific).

Breath Volatile Measurement. The volatile compounds present in expired air from the nose were measured with an atmospheric pressure chemical ionization mass spectrometer (APCI-MS; MS-Nose, Micromass, Manchester, U.K.). Samples of the breath were drawn at 35 mL/min into the ionization source through a deactivated fused silica tube (1 m × 0.53 mm i.d.) heated at 160 °C, to prevent condensation of the volatile compounds. Compounds entering the source were ionized by a 4 kV positive ion corona pin discharge, and the ions formed were introduced into the high-vacuum region of the mass spectrometer, where they were detected and quantified as described previously (*12*). The volatiles studied were detected at masses corresponding to their protonated molecular ion (MH^+). Concentrations in the gas phase were expressed as nanoliters of volatile per liter of air after calibration of the APCI-MS with solutions of authentic compounds.

Gaseous Delivery of Volatiles into the Mouth (Olfactometer). An olfactometer was constructed to deliver a continuous gaseous concentration (45 μL/L) of isoamyl acetate into the mouth. The actual concentration was verified using the MS-Nose over the time course of the experiment. A stream of nitrogen was purified through charcoal and then passed through a bottle (100 mL; held at 30 °C) containing pure isoamyl acetate dispersed on glass wool. The outflow (5 mL/min) was led through a heated, deactivated fused silica transfer line (50 cm long, 0.53 mm i.d.), which had a disposable mouthpiece attached to the end. Isoamyl acetate from the olfactometer was delivered through the transfer line into the mouths of five panelists, and their nosespace concentrations were monitored continuously using the APCI-MS. During this time period, panelists were instructed to hold the mouthpiece in place between their lips and swallow the mouthspace (i.e., the air in the mouth) as, and when, they felt the need. Prior to isoamyl actetate being delivered, panelists were instructed to swallow to remove excess saliva from the mouth. This ensured that it was mainly air in the mouth that the panelists were swallowing.

Solution Sampling Protocol. The nosespace concentration of isoamyl acetate was monitored by APCI-MS after five panelists consumed a solution of isoamyl acetate (200 mg/kg) three times.

The nosespace concentration of 2,5-dimethylpyrazine and menthone was monitored after five panelists consumed 10 mL of each of the HPMC solutions (0, 10, and 20 g/kg HPMC) and the emulsions (0, 4, and 20 g/kg Neobee) three times. Panelists were instructed to swallow only once, cleanse their mouths between samples with water, and keep their exhalation rate constant.

To determine the influence of beverage volatile concentration on the long-term persistence of volatiles on the breath, the nosespace concentration of menthone and 2,5-dimethylpyrazine was monitored by APCI-MS after four panelists consumed three replicates of each solution (50, 100, and 200 mg/kg solutions of menthone and 2,5-dimethylpyrazine). Again, panelists were instructed to swallow only once, cleanse their mouths between samples, and keep their exhalation rate constant.

A similar protocol was used to ascertain the impact of secondary swallows on the breath-by-breath volatile profile, the only difference being that other volatile compounds were used and panelists were instructed to swallow 1 and 2 min after the initial swallow.

The in-mouth persistence of nine volatiles (acetaldehyde, hexanal, benzaldehyde, ethyl butyrate, ethyl hexanoate, menthone, carvone, 2,5-dimethylpyrazine, and ethanol) was studied by exhaling and inhaling through the mouth after the consumption of 10 mL of a solution of volatile compounds. Again, panelists were instructed to swallow only once, cleanse their mouths between samples, and keep their exhalation rate constant.

Calculation of Decay Exponent. The breath-by-breath traces were converted into the plots shown in **Figures 3−5** by taking the maximum intensity of the second and subsequent breath peaks and plotting against time. An exponential curve was fitted to the experimental data and, from this equation, values for the concentration at 1 min (C_1) and the decay exponent (P) were obtained. The rationale for calculating C_1 was to obtain a common parameter for all experiments that was independent of the actual timing of the exhaled breaths.

RESULTS AND DISCUSSION

Volatile Transmission during the First Breath (Swallow Breath) after Consumption. To determine the contribution of the gas and liquid phases to aroma transport, panelists either swallowed gaseous aliquots of isoamyl acetate (dry swallow) or consumed solutions of isoamyl acetate (wet swallow). The resulting concentration of aroma in the exhaled air from the nose was monitored using the APCI-MS technique. The typical traces in **Figure 1** show the breath-by-breath transfer of isoamyl acetate (**Figure 1A**, i; dry swallow) to the nose on the tidal breath flow (measured by monitoring acetone; **Figure 1A**, ii) when administration was by the gaseous route. Transfer of isoamyl acetate via the liquid route is shown in **Figure 1B** (i, wet swallow), with the tidal air flow shown in **Figure 1B** (ii).

Swallowing isoamyl acetate in the gas phase resulted in very sharp initial peaks that had slight shoulders, with little evidence of persistence after swallowing. The acetone trace shows a decrease in signal intensity at the same time as the swallow occurs, confirming that swallowing interrupts the tidal air flow. In comparison, swallowing liquid samples gave initial sharp peaks with broader shoulders and evidence of persistence in subsequent exhalations (**Figure 1B**, i). The interpretation of the results is that the initial sharp peak is due to direct gas-phase transfer of volatiles from mouth to nose, whereas the shoulders on the peaks and the subsequent persistence are due to transfer from the liquid to the gas phase by a slower partition mechanism. The gas-phase delivery is similar to the pulses of volatile

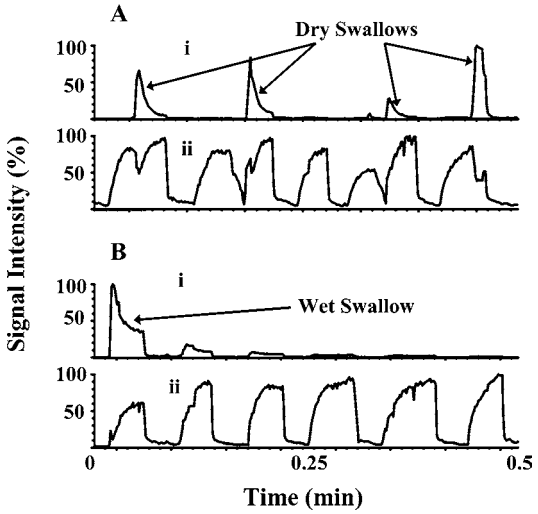

Figure 1. Breath-by-breath profile of isoamyl acetate (i) after it was delivered into the mouth in either the gas phase (**A**) or the liquid phase (**B**) and swallowed. The profile of acetone (ii), which is naturally present on the breath and used as a marker for exhalation, is shown below each of the isoamyl acetate profiles.

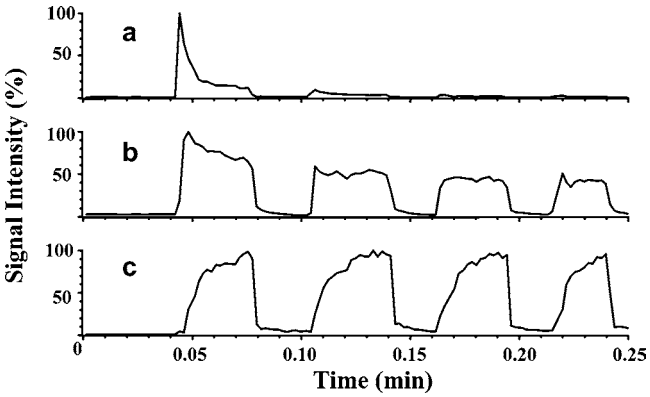

Figure 2. Breath-by-breath profile of ethyl butyrate (**a**) and 2,5-dimethylpyrazine (**b**) after a 100 mg/kg solution of each was consumed. The profile of acetone (**c**), which is naturally present on the breath, was also monitored and used as a marker for exhalation. The maximum signal intensity has been normalized to 100% for each volatile to allow easy comparison of the profiles.

observed during mastication of some foods (*6*). It is interesting that the gas volume transferred to the nose seems to undergo minimal mixing with the exhaled air, and therefore the nasal air flow could be considered to be laminar.

The release of volatiles during the first breath is also affected by the physical properties of the volatiles themselves. **Figure 2** shows that the breath-by-breath release curves of two volatile compounds, ethyl butyrate ($\log P = 1.85$; $K_{aw} = 1.29 \times 10^{-2}$) and 2,5-dimethylpyrazine ($\log P = 1.03$; $K_{aw} = 1.45 \times 10^{-5}$), were different. The pulse of ethyl butyrate delivered to the nose during the first breath was much sharper than that of 2,5-dimethylpyrazine, which had a much larger shoulder, relative to the maximum volatile intensity. These differences can be attributed to the different air−water partition coefficients of the compounds, and the dynamics that affect re-equilibration between volatile compound and air in the throat immediately after the swallow. Linforth (*13*) reported that compounds that have low K_{aw} values (e.g., 2,5-dimethylpyrazine) re-equilibrated much more quickly in vivo than compounds with higher K_{aw} values (ethyl butyrate). The 2,5-dimethylpyrazine concentration in the peak at the start of the exhalation would have been close

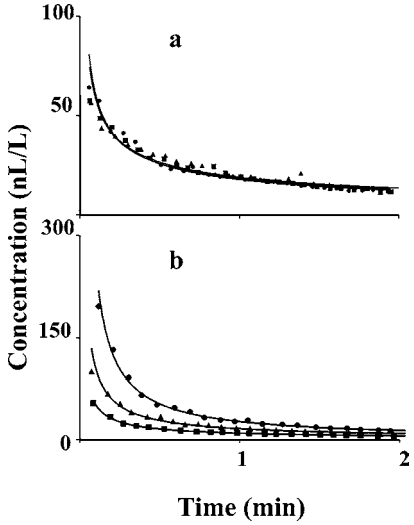

Figure 3. Smoothed breath-by-breath profiles (of one panelist) of 2,5-dimethylpyrazine (**a**) and menthone (**b**) after solutions containing each volatile (100 mg/kg) and different concentrations of lipid, 0 (●), 4 (▲) and 2 g/kg o/w (■), were consumed.

to the headspace concentration at equilibrium. The concentration of the shoulder thereafter was equally close to equilibrium. For ethyl butyrate, not only would the maximum swallow breath concentration be low relative to its headspace (*13*), but the shoulder was much lower and hence further away from its equilibrium headspace concentration.

Effect of Lipid on in Vivo Volatile Release. Figure 3 shows the typical breath-by-breath profiles (for one panelist) of menthone (lipophilic) and 2,5-dimethylpyrazine (hydrophilic) following the consumption of three different samples, each of which contained a different amount of emulsified lipid (0, 4, and 20 g/kg). It was observed that the decay exponent (P) of menthone on the breath was significantly ($P < 0.05$) lower as the amount of lipid in the solution was increased (**Figure 3**; **Table 1**). 2,5-Dimethylpyrazine, on the other hand, was unaffected by the addition of increasing amounts of lipid (**Figure 3**; **Table 1**) with values for P and C_1 remaining constant.

The persistence behavior exhibited by the two compounds was attributed to the different affinities they had for the lipid phase in the emulsion (*10*, *14*). Dynamic headspace studies (*11*) on different esters showed that the lipid concentration had the greatest effect on the most hydrophobic compound (ethyl octanoate); its equilibrium headspace intensity was decreased, but the headspace concentration was least affected by dilution of the headspace.

Our results suggest a similar process occurs in vivo. Menthone is lipophilic and partitions into the lipid phase readily, creating a reservoir for further volatile release. Thus, the volatile concentration above the beverage is lower but resists dilution by the tidal air flow, creating a longer persistence. On the contrary, 2,5-dimethylpyrazine did not interact with the emulsion to any great degree as it is hydrophilic and its persistence is not changed. This confirms that the prolonged release of aroma originates not only from the partitioning behavior of the volatiles with the nasal mucosa but also from residues of beverage remaining in the throat after the initial swallow. If aroma release originated solely from partitioning behavior of volatiles in the nasal passages only, the breath-by-breath profile of menthone would have been unaffected by the lipid content in the beverage.

The lipid content of the model beverage did not affect the concentration of menthone on the breath significantly ($P > 0.05$) when measured 1 min after the consumption of the three

Table 1. In-Nose Volatile Decay Exponent (P) and Concentrations of Volatiles on the Breath at 1 min (C_1) for Menthone and 2,5-Dimethylpyrazine after Aqueous Solutions of the Volatile (100 mg/kg) and Different Amounts of Lipid Were Consumed[a]

	decay exponent (P)			concn in breath (nL/L; C_1)		
panelist	0 g/kg lipid	4 g/kg lipid	20 g/kg lipid	0 g/kg lipid	4 g/kg lipid	20 g/kg lipid
			Menthone			
1	0.73	0.65	0.56	43	39	31
2	0.95	0.96	0.77	83	70	69
3	0.67	0.57	0.55	143	137	89
4	0.93	0.78	0.73	21	16	10
5	0.9	0.83	0.59	43	44	47
mean	0.84a	0.76b	0.64c	67a	61a	49a
SD	0.13	0.15	0.10	48	47	31
			2,5-Dimethylpyrazine			
1	0.49	0.51	0.51	16	17	17
2	0.58	0.55	0.56	56	53	58
3	0.40	0.40	0.45	33	40	34
4	0.59	0.56	0.60	16	16	11
5	0.59	0.63	0.54	16	18	21
mean	0.53a	0.53a	0.53a	28a	29a	28a
SD	0.08	0.09	0.06	18	17	19

[a] Each value is the mean of three replicates. Values with different letters (within the same data set) were found to be statistically different using ANOVA and Fisher's LSD ($P < 0.05$).

different samples (**Table 1**). This is in contrast to in vitro systems, where lipid has a significant effect. The emulsion was expected to decrease headspace concentrations by reducing the menthone concentration in the aqueous phase, as seen in the in vitro headspace analyses. Theoretical air emulsion partition coefficients of menthone above solutions containing different concentrations of lipid (0, 4, and 20 g/kg) were calculated as follows:

$$K_{ae} = \frac{K_{aw}}{\Phi(K_{ow} - 1) + 1}$$

where K_{ae} is the air emulsion partition coefficient, K_{aw} is the air−water partition coefficient, Φ is the lipid fraction, and K_{ow} is the oil water partition coefficient obtained theoretically (Molecular Operating Environment, Chemical Computing Group, Canada). From these K_{ae} values, the headspace concentration of menthone above a 4 g/kg lipid emulsion was estimated to be 130 times less than the concentration above an aqueous solution and 4 times greater than the concentration above a 20 g/kg lipid emulsion. These differences are much greater than those observed (C_1) for menthone in vivo (**Table 1**).

This finding supports the work of Normand et al. (*4*) in which the in vivo partitioning of eight volatiles was modeled and found to vary by a factor of only 5, whereas the same compounds varied by a factor of 500 in vitro. This demonstrates that in vivo release is very different from in vitro monitoring.

Effect of Hydrocolloids on in Vivo Volatile Release. Previous studies on hydrocolloid thickeners (*15*) showed that they had no significant effect on the intensity of aroma release in vivo, although they did affect flavor perception. Viscosity of the sample also had no effect on the concentration of volatiles in the swallow breath or the ratio of the concentration of volatile in the second breath relative to the first (*9*). On the other hand, increased solution viscosity may affect the swallowing mechanics, resulting in a thicker coating in the pharynx, and/or promote interactions between the hydrocolloid thickener and the volatile compounds. This may have implications for the prolonged release of aroma from the coating in the throat.

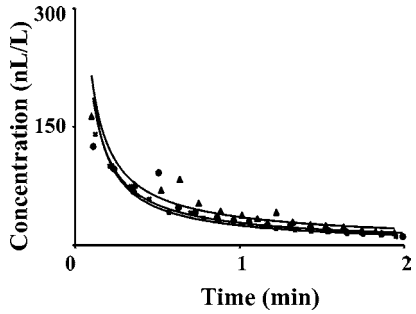

Figure 4. Smoothed breath-by-breath profile of menthone (of one panelist) after solutions containing menthone (100 mg/kg) and different concentrations of hydroxypropylmethylcellulose (HPMC) were consumed: 0 (■), 10 (●), and 20 g/kg (▲).

Table 2. In-Nose Volatile Decay Exponent (P) and Concentrations of Volatiles on the Breath (after 1 min; C_1) after Aqueous Solutions Containing Menthone (100 mg/kg) and Different Amounts of HPMC Were Consumed[a]

	decay exponent (P)			concn in breath (nL/L; C_1)		
panelist	0 g/kg HPMC	1 g/kg HPMC	2 g/kg HPMC	0 g/kg HPMC	1 g/kg HPMC	2 g/kg HPMC
1	0.73	0.70	0.54	34	43	61
2	0.89	0.84	0.83	79	76	64
3	0.91	0.80	0.79	23	19	15
4	0.96	0.92	0.73	14	10	5
5	0.80	0.91	0.98	42	27	30
mean	0.86a	0.83a	0.78a	38a	35a	35a
SD	0.09	0.09	0.16	25	26	27

[a] Each value is the mean of three replicates. Values with different letters (within the same data set) were found to be statistically different using ANOVA and Fisher's LSD ($P < 0.05$).

This was tested by delivering three solutions, containing different amounts of HPMC (0, 10, and 20 g/kg) but the same amount of aroma, to panelists and monitoring the in-nose concentration of menthone after consumption (typical trace is shown in **Figure 4**). The HPMC levels were chosen to represent a fluid that flowed easily (10 g/kg) and a very viscous solution (20 g/kg) that was almost gel-like in consistency. All of the release profiles showed similar behavior, suggesting that HPMC had no significant effect on the long-term persistence of menthone on the breath. **Table 2** shows that the values of P and C_1 for the five panelists were not significantly different ($P > 0.05$) as HPMC content varied, confirming the visual impression in **Figure 4**. This study was repeated using 2,5-dimethylpyrazine but, again, there were no significant effects ($P > 0.05$) on the decay rate of 2,5-dimethylpyrazine or the concentration in the breath (1 min after consumption) as HPMC concentration increased (data not shown).

Interactions between HPMC and menthone or 2,5-dimethylpyrazine were not observed, and HPMC had no significant effect on the longer term persistence, that is, the second breath onward. From this study (albeit with a limited data set) HPMC concentration has no effect on the release of volatiles post-swallowing. This confirms previous work on the effect of HPMC on first-breath concentration and the concentration ratio between the first and second breaths, where HPMC concentration had no effect (*9*).

Effect of Volatile Concentration. Given the results above, it was interesting to consider how volatile concentration in the

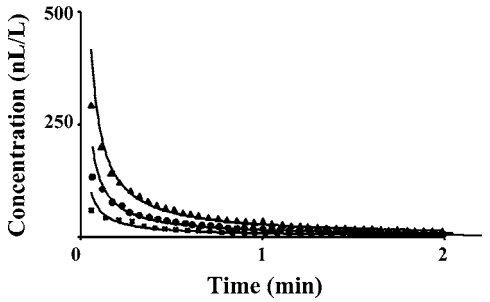

Figure 5. Smoothed breath-by-breath profiles of menthone (for one panelist) after solutions containing different concentrations of menthone were consumed: 50 (■), 100 (●), and 200 mg/kg (▲).

Table 3. In-Nose Volatile Decay Exponents for Four Panelists and Concentrations of Volatiles on the Breath 1 min (C_1) after Aqueous Solutions Containing Different Concentrations of Menthone (50, 100 and 200 mg/kg) Were Consumed[a]

	decay exponent (P)			concn in breath (nL/L; C_1)		
panelist	50 mg/kg menthone	100 mg/kg menthone	200 mg/kg menthone	50 mg/kg menthone	100 mg/kg menthone	200 mg/kg menthone
1	0.75	0.82	0.88	9	15	32
2	0.78	0.87	0.98	6	8	16
3	0.78	0.92	0.92	13	20	32
4	0.66	0.73	0.63	15	37	66
mean	0.74a	0.83b	0.85b	11a	20b	37c
SD	0.06	0.08	0.15	4	12	21

[a] Each value is the mean of three replicates. Values with different letters (within the same data set) were found to be statistically different using ANOVA and Fisher's LSD ($P < 0.05$).

samples might affect breath concentration and release. **Figure 5** shows the breath-by-breath profiles of menthone for one panelist as they consumed three separate solutions containing different concentrations of menthone (50, 100, and 200 mg/kg).

The concentration of menthone in the breath, 1 min after consumption (C_1), was found to be affected significantly ($P < 0.05$) by the initial concentration of the volatile in the beverage (**Figure 5**; **Table 3**). Doubling the concentration in the beverage (from 50 to 100 to 200 mg/kg) resulted in a similar increase in the breath volatile concentration. This demonstrates an approximate linear relationship between beverage volatile concentration of menthone and breath concentration of menthone, 1 min after consumption. The same effect was observed when samples containing 2,5-dimethylpyrazine were consumed (data not shown).

Looking at the breath decay rates for menthone, the consumption of either 100 or 200 mg/kg menthone solutions showed no significant difference ($P > 0.05$; **Table 3**). However, a significant difference in the decay rates of menthone at 100 and 200 mg/kg and a 50 mg/kg solution was observed (**Table 3**). Examining each individual panelist's data shows that, at 50 mg/kg, the decay rate is always significantly lower, suggesting some sort of change in the behavior of volatiles at lower concentrations.

Effect of Secondary Swallows on the Volatile Profile. After the consumption of a beverage, small amounts of liquid remain in the mouth, which are subsequently swallowed, along with saliva, as and when the individual feels the need. Aroma release originating from these secondary swallows contributes to the volatile profile, which is likely to affect the perceived aroma, particularly if the volatile is not very persistent and is virtually absent on the breath prior to the secondary swallow.

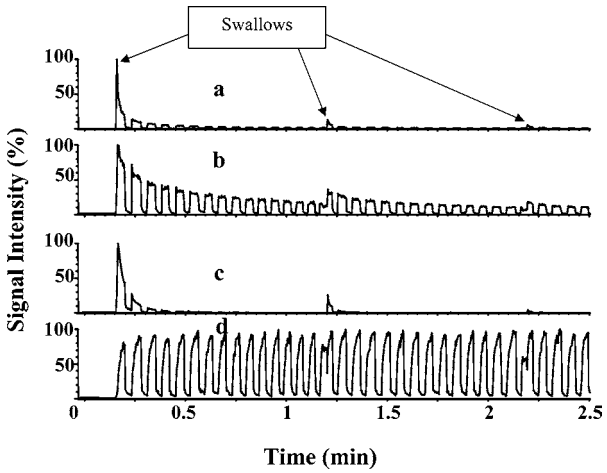

Figure 6. Breath-by-breath profiles of ethyl hexanoate (**a**), ethanol (**b**), and acetaldehyde (**c**) after a panelist consumed a solution containing these three volatiles. The panelist was instructed to perform secondary swallowing actions 1 and 2 min after the initial swallow and to avoid swallowing in between. The profile of acetone (**d**) was monitored and used as a marker for exhalations.

Figure 6 shows the breath-by-breath profiles of three volatiles (ethyl hexanoate, ethanol, and acetaldehyde) that were consumed in one solution. Panelists (five) were instructed to swallow 1 and 2 min after the initial swallow, avoiding swallowing in between.

The intensity of the pulses of volatiles that were associated with the secondary swallowing events were less intense than those observed during the first breath (after consumption), presumably due to dilution of the volatiles in-mouth by saliva, which reduces the gas-phase concentration in the mouth (mouthspace). All three of the volatile profiles showed an increase in the breath volatile concentration immediately after the secondary swallowing events. It was evident that the increase in the breath volatile concentration varied, depending on the physical properties of the volatile. The increase in the breath volatile concentration (relative to the previous exhalation) observed for ethyl hexanoate and acetaldehyde was much greater than that seen for ethanol, a difference that is associated with the extent to which the volatiles persist on the breath. There was already a significant concentration of ethanol in the breath prior to the swallow (**Figure 6**), which reduces the impact of subsequent swallows on the breath-by-breath volatile profile. This is in contrast to ethyl hexanoate and acetaldehyde, which did not persist on the breath to any great extent (i.e., only trace amounts on the breath prior to swallowing). This resulted in a larger relative increase in their breath volatile concentrations and probably a greater impact on volatile perception.

The profile of acetone was consistently affected by the swallowing action (**Figure 6**). At the point of swallowing there was a sharp decrease in the concentration of acetone, lasting for a fraction of a second. This is attributed to the moment when the vocal folds close to allow the bolus (liquid in this case) to pass through the pharynx.

Six more volatiles were included in the study (carvone, menthone, 2,5-dimethylpyrazine, benzaldehyde, hexanal, and ethyl butyrate) to give a range of compounds with different physical characteristics. The extent to which secondary swallows increased the in-nose breath volatile concentration, relative to the concentration in the breath prior to the swallow, varied considerably among volatiles; there was a factor of 200 difference between 2,5-dimethylpyrazine and ethyl hexanoate

Table 4. In-Mouth Volatile Decay Exponents (*P*) and Effect of Secondary Swallows on the Volatile Profile after Consumption of Solutions Containing a Range of Volatiles[a]

	effect of secondary swallows		
volatile	% increase relative to last exhalation	% of swallow breath	in-mouth decay exponent (*P*)
2,5-dimethylpyrazine	197	49	0.55
ethanol	213	49	0.60
carvone	236	24	0.69
methone	527	19	1.04
acetaldehyde	577	56	0.78
hexanal	1382	45	1.67
benzaldehyde	2371	19	1.68
ethyl butyrate	2581	21	1.60
ethyl hexanoate	3876	26	1.84

[a] Each value is the mean of 15 determinations (five panelists, three replicates each).

(**Table 4**). It was the esters and aldehydes that gave large increases in the breath volatile concentration, whereas the more persistent compounds, such as ethanol, 2,5-dimethylpyrazine, carvone, and menthone, resulted in smaller, and probably less significant, increases.

The differences observed between the breath concentrations, relative to the swallow breath, were similar, only a factor of 2.5 difference. The persistent compounds (ethanol and 2,5-dimethylpyrazine) were present at a concentration of ~50% of the swallow breath, whereas the esters, along with carvone and menthone, were present at ~20%. The compounds that are poor at re-equilibrating would have been expected to benefit from the long equilibration time in the mouth, but this was not the case; all of the volatiles behaved similarly. Degradation of volatiles in the mouth has been reported by Buettner (*17*), which might explain this observation; however, two of the three aldehydes (acetaldehyde and hexanal), which would have been expected to have been attacked readily, were unaffected. Thus, the persistence of the compounds in the mouth was studied further to examine the processes occurring in the mouth.

In-Mouth Persistence of Volatiles. The decaying concentration of volatiles in the mouthspace was modeled using eq 1. A good correlation (R^2 values >0.95) was found between the experimental data and the theoretical curves for all nine compounds tested (ethyl hexanoate, ethyl butyrate, benzaldehyde, acetaldehyde, hexanal, carvone, menthone, 2,5-dimethylpyrazine, and ethanol). **Table 4** (last column) shows the in-mouth decay exponents of the compounds. 2,5-Dimethylpyrazine was the most persistent compound in the mouth, and the esters and aldehydes were the least persistent, following the trend of hydrophobicity in these compounds. The same trends were observed when the in-nose decay of five volatiles was investigated (*6*). The similar behaviors observed in the mouth and in the nose are probably due to the conditions used in the experiment. Subjects were instructed to continually inhale and exhale through their mouths; thus, the mouth environment becomes very dynamic and similar to the nose environment. Unfortunately, the persistence of the esters and aldehydes could not be followed in the nose, as they decayed so quickly that they were barely detectable after three or four breaths, making comparisons with their in-mouth decay rates impossible. The amount of solution remaining in the mouth, after swallowing, is likely to be much greater than that in the throat, and therefore the gas-phase concentrations are higher and can be monitored. Once the volatiles have been transferred to the upper airways, the potential for them to interact and disperse results in a decreased gas-phase concentration, which prevented the more hydrophobic compounds from being followed over any reasonable time in nose.

Conclusion. The profile of volatile compounds on the first breath after swallowing is due to direct gas-phase transfer as well as some partition effect. The relative importance of the two mechanisms depends on the properties of the K_{aw} value of the compounds. The lipid content of the beverage affects the persistence of the lipophilic compounds as it changes the effective air−liquid partition coefficient. Hydrocolloids had no effect on either aroma transport mechanism, but the volatile content of the sample did directly affect the amount of aroma delivered to the nose. Secondary swallows transferred volatile compounds from the mouth to the throat, followed by renewed persistence; the magnitude of the effect was again a function of the compound's physical properties. In-mouth persistence also followed an exponential decay.

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