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Letter

Quantification of passive smoking using proton-transfer-reaction mass spectrometry

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Abstract

Using acetonitrile as the lead component, quantitative comparison between passive smoking and direct smoking was achieved by performing measurements using proton-transfer-reaction mass spectrometry. Staying for a working day (8 h) in a smoke laden environment, as is typical for pubs where guests are smoking heavily, is equivalent to smoking one to two cigarettes. (Int J Mass Spectrom 178 (1998) L1–L4) © 1998 Elsevier Science B.V.

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Passive smoking has become a highly debated social issue since several countries have taken legal action against smoking in restaurants, aeroplanes and in public buildings. Smoking was banned in order to avoid the discomfort of nonsmokers by inhaling smoke laden air but also arguments were brought forth that inhaling such air is, to some degree, equivalent to smoking cigarettes by the nonsmokers and thus harmful to the health of these passive smokers.

In the present work we have quantified the uptake of compounds present in smoke laden air by two test persons and compared these results with the amounts of compounds absorbed by persons smoking cigarettes directly.

An important prerequisite for this investigation is our earlier observation [1] that the main loss of acetonitrile in the human body occurs via breathing. Therefore this compound accumulates in the body of a smoker to a level where equilibrium is reached between the rate of exhalation and intake by smoking. Together with the other volatile organic compounds (VOCs) present in cigarette smoke, acetonitrile is resorbed via the lungs. It becomes evenly distributed within the body fluids which make up about 70% of total body weight. Equilibrium between the concentration of acetonitrile in the body liquids and acetonitrile in the air within the lungs (for the time when no smoking occurs) is established according to Henry's law, $H_c = c_w$ [mol/L]/c_a [atm] [c_w: molar concen-

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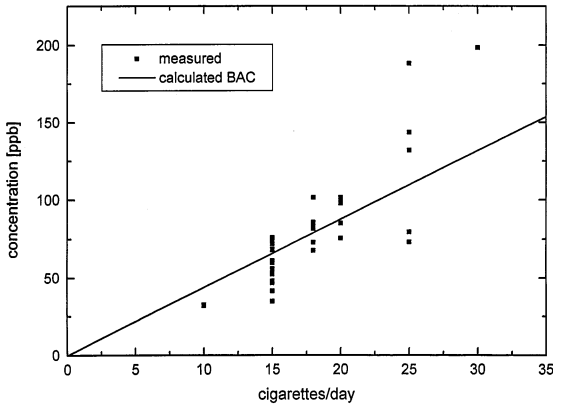

Fig. 1. Measured and calculated breath acetonitrile concentrations as function of the average number of cigarettes smoked per day.

tration of the dissolved compound in the solvent, c_a: partial pressure above the solvent, H_c: Henry's law constant (HLC) in units of mol (L atm)$^{-1}$] so that the concentration of acetonitrile in the breath is a precise measure for its concentration in the body liquids and thus for the total content of acetonitrile in the human body. With the HLC for acetonitrile–water being 19 M/atm at 36 °C [2], we calculate a content of 7.7 × 10^{-7} g acetonitrile/liter(body fluid) × ppbv (acetonitrile in the breath). The body of a smoker (71 kg weight and thus 50 L body fluid) who has a breath acetonitrile concentration (BAC) of 100 ppbv thus contains in total 4.1 mg acetonitrile or 82 μg acetonitrile/L(body fluid). This breath acetonitrile concentration is typical for a person smoking about 25 cigarettes per day. Using the above relation between BAC and acetonitrile concentration in the body fluid, we have calculated the equilibrium BAC as a function of the average number of cigarettes smoked per day [1] and compared these values with measured ones [3]. The good agreement of the two sets of data, shown in Fig. 1, confirms the assumption that acetonitrile is mainly lost by exhalation. Measurements and calculations of the decline of the breath acetonitrile concentrations of smokers who stopped smoking have shown that it takes about a week until these reach levels of a few ppbv, levels typical of nonsmokers [4], again confirming the aforementioned assumption.

This slow elimination of acetonitrile is in strong contrast to the fast metabolism and thus decline in the smoker's breath of nearly all other VOCs present in cigarette smoke. No other compound shows comparable breath concentrations for extended time periods. Benzene, like acetonitrile, is also mainly lost by exhalation, reaching concentrations of about 20–30 ppbv in the breath after smoking a single cigarette, but declines to negligible values within less than an hour [4]. It has a small HLC of 0.08 M/atm [5] therefore the human body is a very inefficient buffer and exhalation eliminates the little amount dissolved in the body fluid within a short period of time.

The exceptional situation of acetonitrile having a long residence time in the body, made it a valuable lead compound for the present investigations on the quantification of passive smoking.

The measurements were performed using a Proton-transfer reaction mass spectrometer (PTR-MS) system which allows for on-line monitoring of the concentrations of VOCs. The system and measuring procedure have been described in detail (see [3] and [6]).

The investigation was performed in three steps. (1) Three smokers and one nonsmoker, who had given up smoking a few months ago, smoked one to five cigarettes within less than an hour and their breath

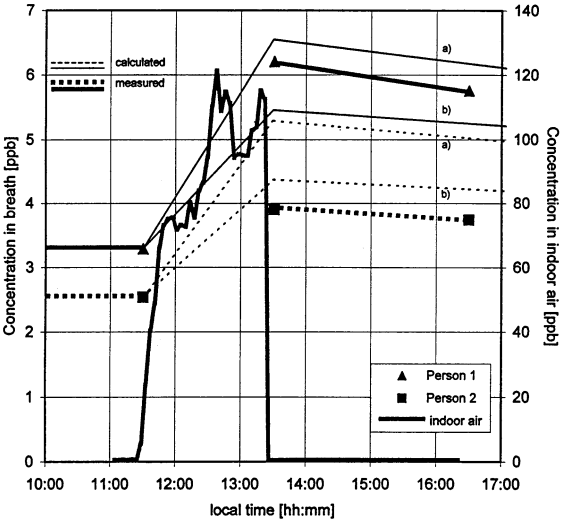

Fig. 2. Concentration of acetonitrile in the breath of two test persons before and after they spent 2 h in the test room, as well as acetonitrile concentration in the test room during the experiment.

Table 1
Concentrations of various compounds in mainstream smoke (diluted by air) and in the air within the test room, respectively

	(1) Concentration mainstream smoke diluted by air (ppb)	(2) Concentration test room (ppb)	Ratio of concentration (1) and (2) [%]
Acetaldehyde	19 724.2	301.5	1.53
Acetone, Propionaldehyde	11 102.3	221.5	2.00
Acroleine	4 709.8	125.7	2.67
Benzene	848.8	28.6	3.37
1,3-Butadiene	2 135.6	92.3	4.32
Dimethylamine	493.6	13.5	2.73
Methylpyridine	196.8	6.0	3.05
n-Nitrosodimethylamine	732.6	47.8	6.53
n-Nitrosomethylethylamine	224.8	6.2	2.75
Pyridine	99.7	4.0	4.02
Acetonitrile	2 975.4	92.6	3.11
Toluene	1 903.8	49.8	2.62
Xylene	873.4	30.7	3.52
Styrene	572.8	8.6	1.51
Propionitrile	806.0	21.0	2.61
Ethyl methyl ketone	4 234.2	53.6	1.27
2-Pentanone	3 473.3	41.2	1.19
Furan	9 015.5	155.4	1.72
2-Methylfurane	2 471.9	46.8	1.90
2,5-Dimethylfurane	2 141.1	49.1	2.29
Phenol	1 421.9	35.3	2.48

acetonitrile concentration was measured before and more than 1 h later. The time span of 1 h is long enough to allow the inhaled acetonitrile to equilibrate within the body. The average increase of BAC per cigarette smoked was 5.0 $\pm$ 0.3 ppbv (reduced to a body weight of about 70 kg). From this we calculate (using Henry's law) an average intake of 200 μg acetonitrile for each cigarette smoked. (2) This increase of the BAC for one directly smoked cigarette was compared with the increase of the BAC in two test persons who spent 2 h in a room (60 m^3) containing air which was strongly contaminated due to the smoke of 30 cigarettes consumed during the experiment by other persons present in the room. The door of the room was opened ten times in order to simulate a situation in a pub where people are smoking heavily. By means of PTR-MS the concentration of acetonitrile and of several other VOCs were monitored on-line in the test room and Fig. 2 represents the results for acetonitrile together with the BACs of the two test persons measured before and after their presence in the room. From the measured ΔBACs of the two test persons, being 2.9 and 1.4 ppbv, we calculate an intake of 106 and 57 μg acetonitrile, respectively. For purposes of consistency we compare these values with the uptake of acetonitrile by the test persons of 80–120 μg, calculated on the basis of the measured acetonitrile concentration in the room and a breath volume of the persons of 5 to 7.5 L min^{-1} [7]. This corresponds to an increase of the acetonitrile concentrations in the breath of the test persons of 2.0–3.0 ppbv, also shown in Fig. 2. The measured BAC of test person 1 falls within the limits of the calculated one, in the case of test person 2, the measured values lie slightly lower, but overall we can state that there is good consistency between the two ways of determining the uptake of acetonitrile.

The measured increase of BAC for the two test persons of 1.4–2.9 ppbv as compared to the increase of BAC due to smoking one cigarette ($\sim$5 ppbv) shows that being in an atmosphere as given in the test room for about 2 h is equivalent to smoking 0.27 to 0.58 cigarettes. This of course is correct only if the relative concentrations of the main components in the

environmental tobacco smoke in the test room are similar to their relative concentrations in the mainstream smoke, i.e. the smoke inhaled by the smoker.

We have therefore carried out a third experiment: (3) The relative concentrations of VOCs in the smoke laden air of the test room were compared with those of VOCs obtained from the mainstream smoke of a smoker which was sampled in a Tedlar test bag and then diluted with pure air in order to avoid too strong contamination of the PTR-MS system. The results presented in Table 1 show that the ratio between the concentration in the mainstream smoke (diluted by air) and the concentration in the test room fall within $\pm 50\%$ for 17 of the 21 components investigated. This finding confirms that the measured increase of BAC in the test persons, due to inhaling the smoke laden air of the test room, can be taken as an equivalent between the effect of direct smoking and passive smoking.

In conclusion, the present investigation shows that passive smoking can be quantified and compared with direct smoking by using acetonitrile as the lead compound. The above data show that a person who remains for a typical working day of 8 h in an environment contaminated as the test room was, suffers passive smoking equivalent to directly smoking one to two cigarettes.

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