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## Successful treatment of gut-caused halitosis with a suspension of living non-pathogenic *Escherichia coli* bacteria – a case report

Received: 11 April 2001 / Accepted: 5 July 2001 / Published online: 22 August 2001  
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**Abstract** In up to 90% of cases, severe halitosis is a result of gastrointestinal or orolaryngeal problems. This case study reports on a girl with bad breath caused by increased formation of malodorous intestinal gases (halitosis), which could be successfully treated with a suspension of living non-pathogenic bacteria *Escherichia coli*. **Conclusion:** in unclear cases of bad breath, an increased formation of intestinal gases should also be considered.

**Keywords** Bad breath · Halitosis · Mass spectrometry · Probiotics

### Introduction

Bad breath is a common phenomenon but can be a considerable social problem for the person concerned and their environment. Halitosis is, in up to 90% of cases, of oral origin and would be better named foetor exore. Halitosis, in a narrower sense, is a foul smelling exhalation caused by fatty acids of low molecular weight, ammonia and volatile sulphur compounds in the exhaled air. Such substances can arise from bacterial processes in the bowel which are resorbed into the blood and can be exhaled via the lungs. Unfortunately there is only very little concerning halitosis in the paediatric literature.

### Case report

The patient, a now 9.5-year-old girl, suffered from frequent obstructive bronchitis and later from bronchial asthma in her first years of life. She was therefore treated several times with antibiotics such as ampicillin. Since the age of 5 years she has been suffering from bad breath, which has been the reason for a considerable isolation in kindergarden and primary school. An abnormal diet could be ruled out by history and there was no weight loss according to her normal weight chart making severe chronic disease uncertain.

There were normal findings for gastrointestinal, orolaryngeal and radiological investigations of the thorax, nasal cavity and oesophagus ruling out conditions like caries, parodontitis, pharyngeal or oesophageal diverticula, chronic sinusitis, abscesses, and bronchiectasis, etc. Hydrogen breath tests with fructose, lactose and glucose gave normal results as well as most laboratory data ruling out dyspepsia, diabetes, uraemia and hepatic disease. A ferritin and IgA deficiency could be detected. Stool analysis did not show any sign of intestinal infection.

### Methods

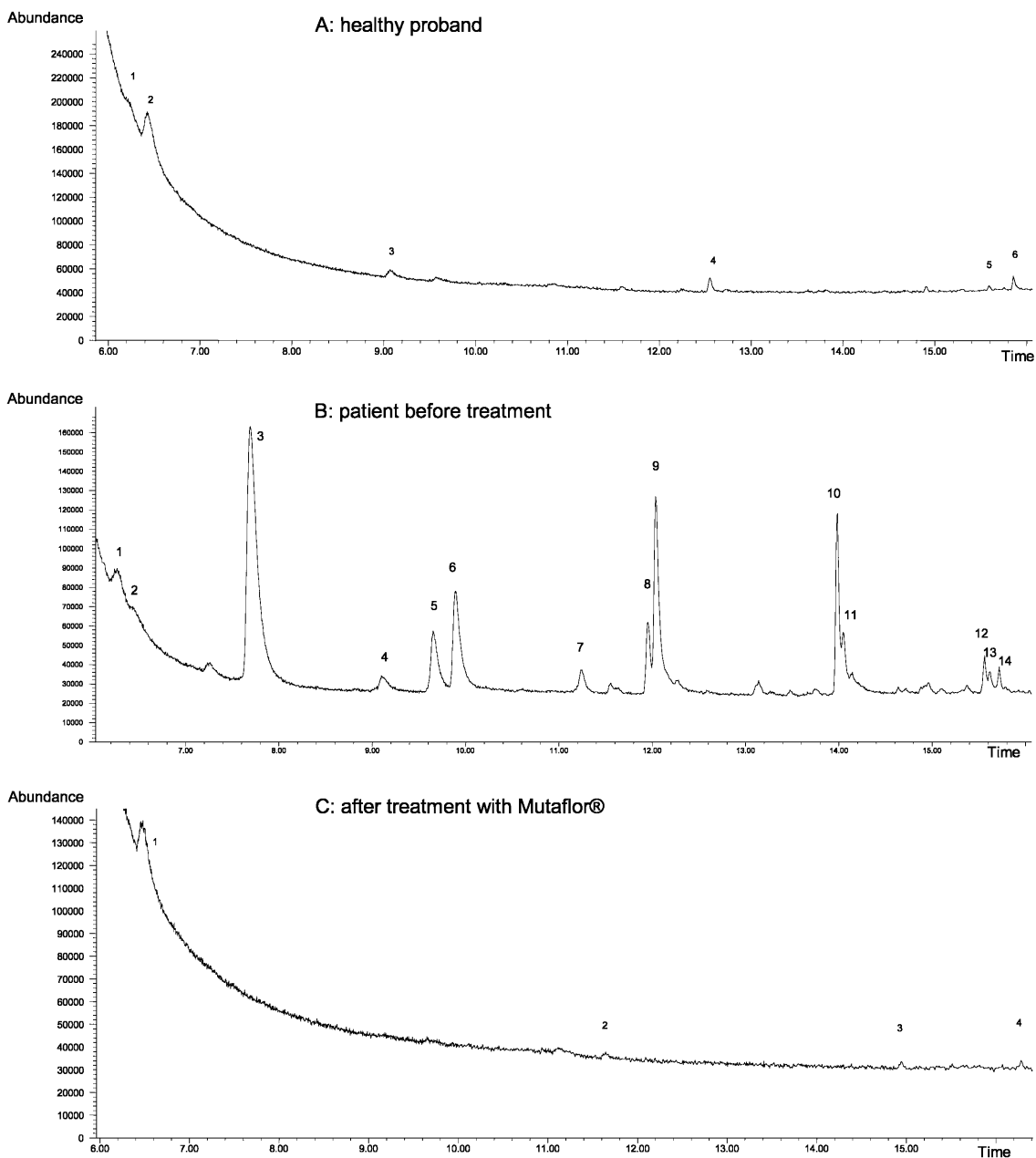
#### Breath gas analysis

The breath gas was accumulated in a gas-sample collector. This apparatus consists of a 0.5 l gas-sample pipette (Gebrüder Schmidt OHG, Suhl, Germany) with two inlet/outlet gas valves and a special fitted septum cupped hole. Solid-phase micro-extraction was found to be the best technique for detection of residues of organic compounds in breath gas. The patient and the control persons were required to blow out the 0.5 l gas-sample pipette filled with double distilled water through a short tube.

To extract the analytical sample, a 75- $\mu$ m-thick carboxen-polydimethylsiloxan resin coated fibre (Supelco, Bellefonte, Pa.), conditioned prior to use according to the manufacturer's instructions, was inserted through the septum cupped hole for 60 min at room temperature (24°C), allowing the organic compounds to adsorb onto the fibre coating. After the period of adsorption, desorption was carried out by exposing the fibre directly to the injector of a gas chromatograph (Model 5890 Series II plus, Hewlett-Packard) via a standard rubber septum. During the whole chromatographic run, the fibre remained in the injector so that the adsorbed organic compounds were thermally desorbed at 270°C. The compounds were separated and identified by gas chromatography/mass spectrometry. A 60 m $\times$ 0.25 mm SPB<sup>M</sup>-5 fused silica column, film thickness 0.25  $\mu$ m, polarity corresponding to HP-5MS or DB-5MS, (Supelco, Bellefonte, Pa.) was used with helium as

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**Fig. 1** **A** Breath gas analysis of a healthy proband. The following compounds were identified: acetone (1), 2-methyl-1,3-butadiene (2), 1-butanol (3), butyl acetate (4), alpha-pinene (5), and hexyl acetate (6). **B** Breath gas analysis of the patient before treatment: acetone (1), 2-methyl-1,3-butadiene (2), 2-butanone (3), 1-butanol (4), 3-pentanone (5), 2-pentanone (6), 3-methyl-1,2-pentanone (7), 3-hexanone (8), 2-hexanone (9), 3-heptanone (10), 2-heptanone (11), 3-octanone (12), 2-octanone (13), and pentamethylheptane (14). **C** Breath gas analysis of the patient after treatment with Mutaflor: 2-methyl-1,3-butadiene (1), toluene (2), beta-ocimene (3) and limonene (4).

carrier gas under electronic pressure control. Injection was splitless with a split ratio of 1:10 at 270°C with a liner of 4 mm ID (990 µl) filled with special glass wool. The oven program was initially 6 min at 40°C ramping at 15°C/min to 300°C, which was maintained for 15 min. The mass spectrometer was a Hewlett Packard Model 5972 Mass Selective Detector operated in electron impact mode with a scan rate of 1/s and a mass transfer line

temperature of 280°C. The AMU program was 10 min/14–150 AMU, 10–15 min/14–300 AMU and 15 min-final time/14–550 AMU. Tuning was done in Maximum Sensitivity Auto Tune mode. The user's private mass spectral data library and the commercial libraries NIST and WILEY (PBM search) were used to identify the compounds in the breath gas. A background fibre chromatogram was run each day prior to sample analysis.

The patient and six healthy probands were submitted to this procedure. All investigated individuals had eaten a meal of fruit, chocolate and biscuits before investigation.

## Results

The six healthy test subjects showed nearly identical breath gas curves (Fig.1). In contrast, the curve of the patient showed more peaks and higher concentrations of ketones with a low molecular weight (Fig.1). After

treatment with a suspension of live non-pathogenic bacteria (*Escherichia coli* strain Nissle 1917, Mutaflor, Ardeypharm, Germany), 2 ml daily for almost 3 months, the breath gas analysis showed a result comparable to that of the healthy test subjects (Fig.1). Clinically, the bad breath of our patient had disappeared.

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## Discussion

Halitosis, in a narrower sense, can be caused by pulmonary diseases (e.g. bronchiectasis, abscesses), metabolic disturbances (e.g. uraemia, diabetic or hepatic coma) and an increased formation of malodorous intestinal gases (e.g. fatty acids with low molecular weight, ammonia, methylmercaptan, indoles, skatoles, hydrogen sulphide) [1,2]. The formation of these gases can arise from a disturbed digestion of nutrients due to altered intestinal transit time or an abnormal intestinal bacterial flora. In addition, the composition of nutrition influences the type of bowel gases produced [3,4].

Provided that organic reasons for the bad breath have been excluded, as for instance intestinal stasis, the following therapeutic possibilities exist to treat halitosis of gastrointestinal origin: (1) change of nutrition, if the ratio of proteins, carbohydrates and lipids is inadequate, (2) change of bacterial flora by administering lactulose, live non-pathogenic bacterial strains or (3), as a last

resort, short-term antibiotic treatment (e.g. with vancomycin or neomycin). Since our patient was given a normal diet rich in fibre, our first step in therapy was the administration of live non-pathogenic *E. coli* bacteria, which was successful. The long-term effect remains to be observed but during a follow-up of now 4 years, the breath smell has remained inconspicuous. The concept of this probiotic therapy is to re-colonise the intestine with normal or other intestinal bacteria and therefore suppress those which break down nutrients resulting in the formation of foul-smelling gases. In this context, antibiotics are used to produce better starting conditions for the probiotics by reducing the existing bacterial flora.

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