Therapeutic CPAP improved glycaemic control after 3 months in our subjects with diabetes. Changes in body composition may play a role. Unfortunately, bioelectrical impedance analysis, as used in all studies, has its limitations.[3]

It would be very interesting to know whether there is an effect of CPAP therapy on insulin sensitivity in less obese diabetic subjects as we demonstrated a rapid improvement in insulin sensitivity in our study in the non-diabetic OSAS group in those with a BMI <30 kg/m^2. That this early effect of CPAP may be related to acclimatisation to the conditions of the sleep laboratory and the clamp procedure is questionable as our studies were done under exactly the same conditions and there is no reason to postulate a higher stress sensitivity in leaner patients.

Although we could not measure plasma catecholamines, we were able to re-measure serum cortisol as another marker of sympathetic stimulation in 20 individuals in our study,[1] and could not find significant differences before (mean 19.18 (SD 3.52) μg/dl) and 2 days after (19.35 (3.27) μg/dl) onset of CPAP therapy (p = 0.59).

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Authors' reply

We thank Harsch *et al* for their comments. Their letter highlights the important contribution of obesity in studies of both insulin resistance and obstructive sleep apnoea (OSA). Although obesity underlies both pathologies, it also confounds studies investigating these conditions. The only studies therefore that can determine conclusively the effect of continuous positive airway pressure (CPAP) on improvements in insulin resistance and glycaemia in patients with OSA are double blind randomised controlled trials. We agree that a randomised controlled trial of CPAP in less obese subjects with type 2 diabetes would clarify this area further, but a study of pre-diabetic subjects with insulin resistance would be even more enlightening.

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Mould eradication and asthma

The paper by Burr *et al*[1] on the efficacy of eradicating visible indoor mould on respiratory health in patients with asthma is of great interest, but I think the authors underestimate the clinical relevance of their findings because they overestimate the lack of effect on peak expiratory flow (PEF) variability as an objective assessment of their intervention. The lack of effect on this primary end point in the presence of highly significant effects on medication use and symptoms—even after 12 months—simply illustrates once again that PEF is too insensitive to contribute meaningfully to the interpretation of our therapeutic interventions. The study by Burr *et al*[1] and those of others[2 3] are examples of investigations that demonstrate a lack of efficacy using PEF parameters as primary end points whereas the secondary end points—such as respiratory symptoms—demonstrate efficacy of the interventions. Increased PEF variability is a specific feature of unstable asthma but it is not necessarily a sensitive one. PEF mainly reflects central airway mechanics[4] and is therefore not the optimal monitoring tool because asthma predominantly affects the smaller airways. Hence, PEF may severely underestimate peripheral airway patency. Clinical studies are much more convincing and powerful if sensitive and relevant end points are chosen, and I would strongly advocate using end points that are both relevant and sensitive. This will teach us more and provide more credit for all involved—doctors as well as patients.

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Competing interests: None.

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CORRECTIONS

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The label "OR (odds ratio)" was erroneously introduced into the headings to tables 3 and 4 in the paper by Aldington *et al* (*Thorax* 2007;**62**:1058–63). In table 3, the numbers in the columns refer to the estimates of the difference of the particular measurement of respiratory function between those who do and those who do not smoke tobacco, and those who do and do not smoke cannabis, respectively. The heading in table 4 refers incorrectly to OR for association between tobacco pack years or cannabis joint years and the measurement of respiratory function. The numbers in the columns refer to the change in the particular measurement of respiratory function per unit change of pack years and joint years respectively. The "OR" label should be omitted from these tables.

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We would like to draw readers' attention to a typographical error in the article by Chapman *et al* (*Thorax* 2008;**63**:228–33). In the discussion, the antigen CAGE is referred to as CAGE (DDX58) and should read CAGE (DDX48); however, the corresponding references are correct. The section is given in full below:

"The DEAD-box cancer testis antigen CAGE (DDX48) has previously been shown to be expressed in a number of cancers including gastric, cervical and lung cancer tissue and cell lines, and autoantibodies have been reported to this protein in some but not all of the cancers samples studied.[25]"