



# Completely scoobied: the confusing world of temperature and pollution effects on sleep apnoea

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Four key studies clearly contradictory over the effects of temperature on sleep apnoea and it is not clear why <http://ow.ly/RWg41>

Aficionados of 1970s and 1980s cartoons will have no trouble imagining the look of befuddlement upon our faces reminiscent of Scooby Doo's when presented with a new mystery. Normally in an editorial, one should write a nice, succinct summary of the field providing the reader with a clear but concise understanding of where we are and where we are heading. However, it is just not possible in this case. The evidence is truly befuddling. The four key studies [1–4] are not just inconsistent with respect to the effects of temperature on sleep apnoea, they are clearly contradictory and it is not clear why. So, this will be an editorial that poses more questions than it answers.

The risk factors for sleep apnoea are already well described at both clinical and population levels. Age, sex and obesity all play a role, as do upper airway anatomy/physiology [5] and control of breathing [6, 7], but these traditional risk factors are all measured at an individual level rather than at a household, community or city level. Many conditions, particularly in respiratory medicine, are driven by environmental factors that may exist at a household level (tobacco smoke exposure or solid fuel heating), community level (for instance, urban noise levels [8]) or municipal/city level (pollen counts and heatwaves). Some work has identified neighbourhood as being a risk factor for poor compliance with continuous positive airway pressure therapy [9] but very few investigators have looked at what components of a neighbourhood might increase the risks of sleep apnoea itself.

In this issue of the *European Respiratory Journal*, WEINREICH *et al.* [2] present analyses from the Heinz Nixdorf RECALL study of 1733 community-dwelling participants (aged 50–80 years) across Essen, Bochum and Mulheim in Germany. Using the single-channel version of the ApneaLink device (ResMed, Sydney, Australia) for one night in the participants' own homes, they reported estimates of sleep apnoea prevalence of 23.5% (defined as an apnoea–hypopnoea index equal to or above 15 events per hour of sleep). The data collection period ran from May 2006 to September 2008, allowing them to investigate the influence of 2.5 years of temperature and air pollution (particulate matter less than 10 µm in diameter (PM10) and ozone) on sleep apnoea severity. Across the seasons, an 8.6°C increase in temperature and a 39.5-µg·m<sup>-3</sup> increase in ozone were associated with about a 10% increase in obstructive sleep apnoea

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(OSA) severity. There was some evidence that effects might be stronger in the summertime, although this interaction was not statistically significant. The authors also found no increase in sleep apnoea risk associated with PM<sub>10</sub> levels. In subanalyses, the authors investigated whether the effect was strong enough to significantly affect the diagnosis of sleep apnoea (and therefore potentially treatment decisions) by changing markedly the categories of sleep apnoea diagnosis (none, mild, moderate and severe). Although the effects of temperature and ozone on sleep apnoea severity were statistically significant and potentially important at a public health level, they did not seem to be so strong as to affect clinical decision making at an individual level. Notwithstanding, treating clinicians should be aware that some patients near the cut-offs of diagnostic categories may be pushed into higher or lower severity classifications during abnormal weather conditions.

However, the published literature as a whole seems to present a paradox, particularly obvious when thinking about the universally reported exposure metric of air temperature (table 1). The four key studies each appear to present good-quality science, albeit using different epidemiological study designs. However, they fundamentally disagree on the effects of temperature and air pollution on sleep apnoea. The literature can be summarised as follows: two community-based studies (Germany and USA) reported that increased temperature makes sleep apnoea worse [1, 2] and two clinic-based studies reported that increased temperature ameliorates sleep apnoea [3, 4]. Our difficulty as editorialists is “how do we explain that?”. We could have been tempted to discount the Brazilian retrospective chart review from a single sleep laboratory [3] as being an aberrant observation but the only experimental study in sleep apnoea patients, in Umea, Sweden, seems to report similar results [4]. It is also possible to speculate that the key difference could be that the two community-based studies where participants have not been pre-selected for having sleep apnoea agree, as do the two clinical studies. Maybe patients with sleep apnoea respond differently to community-based participants who have largely non-clinically evident, milder apnoeic severity. We also do not really understand the potential interactive effects of manually modifying bedroom temperature, such as using air conditioning or fans, extra bedding (duvets or blankets), or warmer sleeping clothes.

As epidemiologists, we are trained to look for consistency across studies as a way to protect against the accidental false positives to which studies are prone. Each of the four studies contends that seasonal differences may be important in the manifestation of OSA symptoms, although as indicated, the results for temperature are in contrast with each other. It may be that other seasonal influences are more important, although the findings for ambient ozone and PM (two pollutants that exhibit variation over seasons) are inconsistent as well. Two studies measured the effects of ozone: the current cross-sectional study [2] reported an increasing association, whereas the Brazilian retrospective clinic study did not [3]. The US-based Sleep Heart Health Study’s cross-sectional analyses found an association between summer PM only (and not overall PM) and worse OSA [1], while the Brazilian study [3] did not find any association with PM (although they do not report actual pollutant concentrations). The mean PM<sub>10</sub> concentration of 27.05 µg·m<sup>-3</sup> reported by WEINREICH *et al.* [2] fell within the range of centre means reported in the US study [1]. The PM<sub>10</sub> means were fairly consistent across the four studies so the varying results cannot be explained by substantially varied exposures. Could it be that other seasonal influences interact with or modify the effect of air pollution and temperature? Relative humidity was a significant predictor of sleep apnoea severity in Brazil [3] and in the study by WEINREICH *et al.* [2], relative humidity was retained in all significant multivariate models. The Sleep Heart Health Study did not consider relative humidity and, interestingly, the Swedish laboratory-based study [4] did not measure or control for relative humidity. The Swedish investigators highlighted this as a limitation, noting that humidity could have an influence on the nasal and pharyngeal mucosa, and hence impact on OSA outcomes, but as that was a laboratory-based randomised cross-over experiment, it does not seem likely that relative humidity varied substantially over the study period. Our point here is that none of these studies agrees entirely with any other, although they each represent good-quality scientific methodology.

One issue that is not well addressed because of the use of average daily temperature data is what happens with large or sudden fluctuations in temperature from day to night. However, Swedes may mitigate this effect as they typically live in very well insulated houses with indoor temperatures of around 18–22°C (although these data are out of date and the real figure may be higher now) [10]. So, 16°C in summer could be colder than the normal average bedtime temperature for Sweden in winter when the windows are kept shut. How this might explain the findings in Germany, where the authors [2] state that it is very common to sleep with the windows open at night, is not clear to us. One explanation for the divergence in findings could be that effects are heavily dependent on locality, so that temperature has different effects on people living near the arctic, in temperate climates or nearer the equator [11, 12]. The effects of temperature on sleep or sleep apnoea might hinge on what a person is used to; for instance, it is apparently commonly regarded as healthy in Finland to leave babies outside in their prams to nap even in –5°C or lower temperatures [13].

TABLE 1 Studies investigating temperature and other climatic variables on sleep apnoea severity

First author [ref.]	Setting	Study design	Participants	Subjects n	Ozone	Temperature	PM <sub>10</sub>	Notes
<b>WEINREICH [2]</b>	Essen, Bochum and Mulheim, Germany	Cross-sectional analysis of a cohort study Adjusted for relative humidity, season, age, sex, BMI, education, smoking, alcohol, physical activity, and CVD and diabetes outcomes	Community-dwelling adults aged 50–80 years	1733	↑ (8-h mean) Adjusted for RH, PM <sub>10</sub> , age, sex and lifestyle	↑ Adjusted for RH, PM <sub>10</sub> , age, sex and lifestyle	–	Daily means (lag 0), moving averages (lag 0, 1 day and lag 0–2 days) City-wide monitors (2–4).
<b>ZANOBBETTI [1]</b>	7 US urban areas	Cross-sectional analysis of a cohort study	Community-dwelling adults (aged >39 years) who were members of parent cohorts	3030	ND	↑ (overall, short-term temperature) Adjusted for season, mean temperature, age, BMI, sex, education, smoking, coffee, tea and alcohol ↓ Adjusted for sex, age, BMI, neck circumference and RH RH was a significant predictor in multivariate model	↑ (summer) Short-term PM <sub>10</sub> (365-day average) RDI and % time with oxyhaemoglobin desaturation <90% and sleep efficiency – (other seasons or overall)	Several monitors PM <sub>10</sub> averaged over locations for each city Daily mean PM <sub>10</sub> concentration used in analysis
<b>CASSOL [3]</b>	Clinical sleep laboratory in Porto Alegre, Brazil	Retrospective chart review, January 2000–December 2009	Routinely tested sleep apnoea patients	7523	–	↓ (no data on room humidity) Adjusted for BMI, time in supine position/sleep time and sleep stage on awakening	–	Lags of 1, 2, 3 and 4 days tested for environmental variables
<b>VALHAM [4]</b>	Sleep laboratory in Umea, Sweden	Experiment: 3-arm randomised cross-over (3 nights at 16°C, 20°C and 24°C)	Untreated mild–moderate sleep apnoea patients (AHI 10–40 events per h)	40	ND	↓ (no data on room humidity) Adjusted for BMI, time in supine position/sleep time and sleep stage on awakening	ND	

CASSOL *et al.* [3] also reported the following associations with OSA: atmospheric pressure (↑), humidity (↑), carbon monoxide levels (↑), precipitation (–) and sulfur dioxide (–). BMI: body mass index; CVD: cardiovascular disease; AHI: apnoea–hypopnoea index; ↑: increased levels increased the severity of sleep apnoea; ↓: increased levels decreased the risk of sleep apnoea; –: increased levels had no effect on sleep apnoea; ND: not determined; RH: relative humidity; PM<sub>10</sub>: particulate matter less than 10 μm in diameter; RDI: respiratory disturbance index.

In the end, we simply could not find enough consistent data about the effects of temperature and air pollution on OSA to come to clear conclusions (or imagine it with Scooby's particular speech impediment, "Ri ron't row [I don't know]"). The way forward may be to conduct more studies with a mix of observational and experimental methods investigating a range of environments. More observational studies from a range of climates are clearly needed. It is possible that extreme climate or pollution events may be useful here and, thus, studying bush fire effects in Australia or California (characterised by high PM levels) or the effects of pollution in places prone to high pollutant concentrations may be fruitful. Because of the ever-present risk of uncontrolled confounding and the potential for complex effect modification of meteorological factors, it may be worth testing some of these hypotheses under laboratory conditions. Nevertheless, well-conducted community-based studies that are able to measure individual level covariates, and use the same methodology as the current German and extant US studies, are also needed.

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