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Where there's smoke ...

Poor air quality is an important contributor to cardiovascular risk

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Air pollution has received much attention in the past year. The Global Burden of Disease Study estimated that 3.2 million deaths a year are attributable to particulate matter in outdoor air, ¹ the International Agency for Research on Cancer classified polluted outdoor air as carcinogenic,² and we witnessed extreme episodes in Beijing and Shanghai. While effects on respiratory health have long been recognised, it is the impacts on cardiovascular disease³ that are responsible for most of the disease burden attributable to air pollution. Two linked papers provide new insight into the role of air pollution on cardiovascular disease and subsequent impacts on population health.^{4 5}

Perhaps nowhere are the health impacts of outdoor air pollution more acutely felt than in China, where air pollution is the fourth most important risk factor for disease burden⁶ and is responsible for 1.2 million deaths each year. In one linked paper, Guo and colleagues assessed the relation between daily pollution levels in Beijing and years of life lost.⁴ The results suggest that air pollution has a substantial impact, but it is not possible from this study to directly assess the magnitude of life shortening attributable to air pollution. Cohort studies conducted in North America and Europe suggest that a $30 \,\mu\text{g/m}^3$ difference in long term exposure to particulate matter <2.5 µm in aerodynamic diameter (PM25; about a third of the interquartile range encountered throughout China) is associated with a reduction in life expectancy of about two years.7 Deriving such estimates for China will require analysis of cohort studies, an important research priority.

The linked paper by Cesaroni and colleagues reports on the relation between long term exposure to air pollution and incidence of myocardial infarction and unstable angina in a meta-analysis of 11 cohort studies from five European countries.⁵ As part of the ESCAPE project, their analysis complements recent reports from this extensive collaboration on associations between chronic exposure to air pollution and natural mortality⁸ and lung cancer.⁹ As one of the largest studies on



Horseman of the apocalypse

cardiac events in relation to air pollution, the finding that this association is not dominated by fatal events suggests that cardiovascular disease events attributable to particulate matter are underestimated in more traditional analyses that consider only deaths. The study is notable for its size. The authors analysed data from more than 100000 people in heterogeneous cohorts with a standardised statistical approach including assignment of exposure at the individual level.

A trigger for acute events, but anything more?

While this study is a noteworthy addition, several important questions remain. For example, the analysis by Cesaroni and colleagues compares only exposure differences within cities caused by local sources of air pollution largely related to traffic.⁵ Comparisons between exposures from sources other than traffic cannot be evaluated. Understanding the relative role of local and regional pollution, specific sources of pollutants, and mixtures has important policy implications. The focus on long term exposures and the observation of an association with increased acute cardiac events suggests that air pollution can be a trigger for acute events, as reported previously.¹⁰ Whether air pollution also encourages the progression of atherosclerosis is still unclear but is the subject of intense investigation.¹¹

The study by Cesaroni and colleagues has specific relevance to the management of air quality in Europe. Particulate air pollution was associated with cardiac events even after they excluded exposures below the 25 μ g/m³ European Union limit value for fine particles $(PM_{2,5})$.⁵ Significant effects were also discernible for exposure levels only slightly above the 10 µg/m³ World Health Organization (WHO) air quality guideline. Nearly 90% of the world's population live in locations where the WHO guidelines are exceeded.¹² Indeed, in the analysis of Guo and colleagues the mean PM_{2.5} concentration over a five year period in Beijing was more than 10 times the WHO guideline value.⁴

The important impact of air pollution on cardiovascular disease highlighted by these two papers supports efforts to meet existing and even more stringent air quality standards to minimise cardiovascular morbidity and mortality. A specific focus on the mitigation of other widely recognised risk factors for cardiac events in areas where poor air quality presents an additional risk might also be warranted. For example, there are currently no focused interventions to enhance smoking cessation in highly polluted areas or to provide more specific guidance as to how to safely achieve targets for aerobic exercise in such areas.

As particulate air pollution can trigger cardiovascular events, there may also be a need for more deliberate assessment of this risk, in conjunction with other traditional risk factors for cardiovascular disease, to ensure that treatments that might prevent events are being used. People with or at risk of cardiovascular disease who live in highly polluted areas might warrant more aggressive use of primary and secondary preventive therapies, including antiplatelet agents, lipid lowering agents, and treatments for hypertension or diabetes, all known to prevent cardiovascular events. Indeed, the relative effectiveness of such approaches in highly polluted compared with cleaner areas is unknown but potentially important to public health.

These two new studies can help direct further research to evaluate interventions to improve air quality and other risk factors for cardiovascular disease, with the ultimate goal of reducing the global burden of cardiovascular disease.

Competing interests: MB has collaborated with Pershagen, Hoek, and Brunekreef on topics unrelated to this article. Provenance and peer review: Commissioned; not externally peer reviewed. References are in the version on bmj.com.

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- Research: Lung cancer deaths from indoor radon and the cost effectiveness and potential of policies to reduce them (BMJ 2009;338:a3110)
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- Research: Effect of insulating existing houses on health inequality: cluster randomised study in the community (BMJ 2007;334:460)

Cutting household ventilation to improve energy efficiency

A warning about radon and lung cancer

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If global emissions of greenhouse gases continue on their present trajectory, the Intergovernmental Panel on Climate Change (IPCC) projects that the world may be more than 4°C warmer in 2100 than in 1861-80.¹ To hold warming to less than 2°C on average, the level often cited as the threshold of dangerous climate change, emissions must be reduced radically. For example, the World Bank estimated that global emissions would need to be halved by 2050, and continue falling thereafter, to reach this goal.² The prospect is daunting, but there are many opportunities for intervention. Housing is a good example, as Milner and colleagues point out in the linked paper.³ The sector typically contributes about a guarter of national greenhouse emissions, energy efficiency is often low, and measures such as insulation can improve building performance quickly. We also know that well designed interventions to make homes warmer and safer can reduce energy use and improve health.4

However, energy efficiency measures may damage health if not done well. Milner and colleagues point to one health risk: an increase in indoor radon levels in homes in which ventilation is reduced to control heat loss.

Radon is an inert gas, present in much of the Earth's crust, which rises into buildings through cracks and fissures in the foundations. The radioactive breakdown products of radon are potent carcinogens: the US Environmental Protection Agency estimates that they cause about 21000 deaths a year from lung cancer in the United States.⁵ In the United Kingdom, radon is commonly detected indoors. Although concentrations in most homes are low (fewer than 1% fall above the UK action level of 200 Bq per m³), this does not mean absence of risk: the dose-response relation between radon and lung cancer that best fits the epidemiological data is a straight line with no lower threshold. Because the bulk of the population is at the lower end of the exposure curve, this is where most of the burden of disease occurs. In the UK, 90% of radon attributable lung cancers are estimated to occur in homes with concentrations below 200 Bq per m³.⁶



How to keep the radon in

The national housing energy efficiency strategy for England aims to cut heat loss from homes by reducing uncontrolled ventilation. If this strategy was implemented, what difference would it make to radon levels? Milner and colleagues use mathematical modelling to estimate that indoor concentrations of radon would rise on average by more than 50% if English homes were made airtight enough to meet energy efficiency targets.

A drop in the ocean

They also estimate an extra 4700 life years lost and up to 278 additional deaths from lung cancer annually as a result. Mechanical ventilation and heat recovery systems that extract heat from air before it is passed to the outside would limit the rise in radon levels, but these systems are relatively complex and expensive.

This is the first study that has tried to quantify the risks of energy efficiency measures caused by radon, and several points should be made about the calculations. The long lag between exposure to radon and incidence of lung cancer means the effects of tighter homes are projected to occur 20-30 years in the future. It is impossible to anticipate the changes in all relevant variables so far ahead. For instance, the calculations assume no variation in health status of the population or treatment effectiveness, although other scenarios are also plausible. Moreover, projections about lung cancer are highly sensitive to assumptions about the prevalence of smoking because the risks of radon and tobacco use combine in an additive fashion.⁷ Milner and colleagues' primary model assumes no change in the prevalence of smoking in adults. But in the past 30 years the proportion of British adults who smoke has halved, and if this decline continues the radon effect will also diminish. In their sensitivity analyses, the authors estimate that if the prevalence of smoking halved again (from 21% to 10%) the number of additional deaths from lung cancer attributable to airtight homes would reduce by 44%.

Milner and colleagues do not attempt a comprehensive assessment of the health consequences of reduced ventilation. Airtight homes may increase levels of other indoor pollutants, such as second hand smoke or emissions from gas cookers and heaters, as well as potentially increasing the spread of airborne infections. Better indoor climate control has benefits too. A New Zealand trial found that children with asthma living in warmer homes had fewer episodes of wheezing, fewer visits to the doctor, and fewer days off school.⁴ A full assessment of the benefits and risks would also include the damage averted by slowing climate change. The risks of unmitigated global warming are uncertain, but potentially large. For instance, one study estimates that European "mega-heatwaves," such as the 2003 event that caused about 70000 excess deaths, will become 5-10 times more common in the next 40 years.8

Milner and colleagues' study reminds us that large scale interventions may have unintended harmful consequences. But it also points to opportunities for reducing the risks of climate change in ways that minimise risks to health and may improve it. In the UK, more effective insulation of homes could save energy and warm houses without compromising the flow of clean air. In other countries the win-win possibilities include safer heating, cleaner cooking, and low energy cooling. Everywhere housing can and should contribute to smart growth strategies that improve access to services and facilities, increase the opportunities for physical activity, improve air quality indoors and out, and reduce dependence on fossil fuels.9 Competing interests: None declared.

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Video abstracts

The latest in a series of initiatives to increase the accessibility and visibility of BMJ research

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The *BMJ* continues to explore new ways of disseminating the results of research. For several months now, we have been inviting authors of research articles to submit video abstracts for publication alongside their papers. Authors have been filming short videos that summarise their studies, which are published on bmj.com with their articles and on our multimedia (www.bmj. com/multimedia) and YouTube (www.youtube. com/user/BMJmedia) channels.

Video abstracts enable authors to explain their research findings in person, increasing the reach and understanding of their work. Authors can present the background to their research question, explain why it is important, and discuss their findings, often using animation and infographics. As the social web becomes an increasingly important medium for conversations about research, video abstracts offer a format that is friendly to blogs, Twitter, and Facebook. Studies are therefore made accessible to a wider audience and can be easily shared. We hope that some of the debate that these videos provoke will take place on bmj.com, where "rapid responses" (electronic letters to the editor) have been thriving since their introduction 15 years ago.¹

How have authors been using video abstracts? At their simplest, video abstracts can be as straightforward as a researcher giving an account

of his or her study directly to the camera. However, adding "bells and whistles" of additional audio and visual material can make for a more engaging video

abstract. So far, we have posted videos that cut through jargon to explain the wider relevance of a research question,² videos that use animation to show how the research was conducted,³ and even ones that show musical depictions of migraines.⁴ Our resources for authors give further guidance for filming and submitting a video abstract.⁵

Video abstracts continue the *BMJ* tradition of innovation in sharing research. We are committed to publishing research that can influence clinical practice and health policy, and we want



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All research is published online with full open access.⁶ Since 1998, the full text of every *BMJ* research article has been available to anyone with an internet connection, anywhere in the world, at no charge, from its day of publication.

Five years ago we introduced *BMJ* pico—a one page abridged format for all research papers featured in the print journal.⁷ *BMJ* picos aim to provide an accessible and concise summary of the study design and key findings.⁸ With subheadings including "what is known and what this paper adds," "main results and the

> role of chance," and "bias, confounding, and other reasons for caution," *BMJ* picos serve as an evidence based medicine tool, highlighting areas that will help

readers appraise studies.9

Adding "bells and whistles"

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engaging video abstract...

We actively press release many of our research articles and work hard with our authors to ensure that these releases are as accurate and informative as possible. Work by Dartmouth researchers Schwartz and colleagues has shown that the quality of press releases affects how well science is reported in the media.¹⁰

We are keen to embrace new and emerging technologies to bring research to our readers. In

1995, we were the first general medical journal to have a substantial presence on the internet (www. bmj.com/about-bmj), and in 2011 the first to provide an electronic print edition on the iPad.¹¹

If we build it will they come?

Are these electronic means of communication having an impact on the dissemination of research published in the BMJ? Traditionally, the impact factor has been the only way to measure a journal's influence, but it's an incomplete and inaccurate measure, counting only formal citations.¹² To measure the influence of a paper beyond academia, mentions in social media and news media need to be taken into account, and one tool that attempts to do this is Altmetric.¹³ In future, journals will probably be ranked by a combination of informal interactions and traditional citation. In the meantime, we are happy that Altmetric ranks the BMJ relatively highly, when compared with the other general medical journals,14 and that the journal's impact factor is at an all time high.

An increasing array of tools is now available to improve our communication and understanding of science. The *BMJ* continues to develop new ways to share research, and we welcome your feedback on our efforts and further suggestions for the future.

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Long term outcomes for women treated for cervical precancer

Cervical cancer risk increases with age and looks worse for women treated more recently

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Although the risk of cervical cancer after treatment for screen detected cervical precancer is low compared with non-treated women, the incidence of invasive cervical cancer is still significantly higher than in the general population.¹ These findings are confirmed by Strander and colleagues in a trend analysis that linked data from pathology, cancer, and cause of death registries that have covered the whole Swedish population for more than half a century.² The authors report that the risk of developing or dying from cervical or vaginal cancer in women with a history of treatment for CIN3 (cervical intraepithelial neoplasia grade 3) is two to three times higher than in the general population. Furthermore the increase in risk among women treated for CIN3 rises significantly with older age and more recent year of treatment.

These results agree with previous data suggesting that the rates of residual or recurrent high grade CIN after treatment are higher for older than for younger women.^{3 4} Endocervical precancerous lesions, a predisposing factor for recurrence, are more common in older women. The lower recurrence rates in younger women that are independent of the completeness of excision suggest that age specific immunity may also contribute to the ultimate cure of cervical precancer.⁴

Worse outcomes with less aggressive treatment

It is worrying that Strander and colleagues found that women who received local treatment more recently were at greater risk of developing cervical and vaginal cancer.² The authors suggest that the use of less aggressive treatments in the two most recent decades may have adversely affected oncological outcomes. The trend in treatment was driven by an increasing awareness that extensive procedures are associated with poor reproductive outcomes. Recent meta-analyses of reports published since the end of the 1970s and registry based cohort studies have shown that pregnant women with a history of excisional treatment of CIN have a greater risk of premature delivery, particularly if the excised cones were large.⁵⁻⁷ Researchers from Norway have also described a parallel trend between less aggressive treatment for cervical precancer and a lower risk of preterm delivery.⁸

The study population comprising more than three million woman years of follow-up after treatment gave the current trend analysis enough power to identify significant differences between different subgroups of women. Further analysis of the Swedish data on compliance with followup could provide important information on the possible reasons for treatment failure. The suggestion of reduced therapeutic effectiveness

over time might also be partly explained by the decreased use of hysterectomy over the past two decades. A separate analysis of cervical and vaginal cancer rates, adjusted for rates of hysterectomy and for trends in the dimensions of excised cones, would help interpret the observed period effect.

Research is needed to identify accurate biomarkers that predict a woman's future risk of cancer. A recent review concluded that testing for DNA from human papillomavirus helps to identify early treatment failure (recur-

rence within two years of treatment for cervical precancer), with higher sensitivity and similar specificity to follow-up cytology or histological assessment of the section margins.⁹ However, longer term data are limited. A cohort study from the Netherlands assessed the predictive value of combined cytological and virological follow-up for 10 years after treatment for cervical precancer.¹⁰ The overall cumulative incidence of recurrent CIN2 or worse was 17%, and that for CIN3 or worse was 9%. In women with two negative tests (cytology and high risk human papillomavirus DNA) at six and 24 months post-treatment, the risk of these outcomes was similar to that in women who tested negative for cervical precan-

cer at baseline screening. Further cohort studies with long term follow-up are needed to confirm these results and to generate more evidence on the safety of different follow-up protocols for women treated for cervical precancer.

Currently, colposcopists who treat women with high grade CIN lesions must choose between complete excision to obtain free margins or a more prudent approach, especially if a further pregnancy is desired.¹¹ Published and aggregated data still leave considerable room for doubt about the magnitude of the association between the extent of treatment and risk of later preterm delivery. Divergent findings may be explained by variability in therapeutic practices, particularly the size of the cone excised.¹² The COSPCC study—a meta-analysis of individual patient data—should allow more precise

> measurement of the obstetric and oncological safety associated with different treatment options, while accounting for patient and lesion characteristics. The study should also provide more detailed evidence on how to balance treatment decisions.

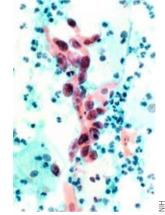
> However, Strander and colleagues' study makes it clear that women who have been treated for a high grade intraepithelial cervical lesion, particularly those aged 50 years or more, require careful surveillance, and that measures

should be taken to assure full compliance with follow-up. The data also underline the need for better standardisation and quality assurance in colposcopic practice to achieve an optimal balance between risk of cancer and obstetric safety.

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A role for age specific immunity?