

MEDICAL OPINION

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Report on the outputs and potential impact of findings contained in the Air Quality Report conducted by the UK Centre for Tobacco and Alcohol Studies, University of Nottingham.

Prepared by:

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I am Professor of Epidemiology at Nottingham University, and an honorary consultant physician in respiratory medicine at Nottingham City Hospital. My qualifications and experience are summarised in the appended curriculum vitae.

This report is prepared on the instruction of The National Offender Management Service (NOMS) to provide clarification and background information on the findings contained in our report on air quality testing in four prisons in England, submitted to NOMS in March 2015. I am asked to clarify the implications of our report in relation to the reasonable probability of injury being caused by exposure to second-hand smoke (also referred to in the literature as passive smoke, or environmental tobacco smoke (ETS)) in the prisons we studied, and to distinguish where possible between inception injuries (those caused directly as a result of exposure to second hand smoke) and exacerbation of existing conditions. I am invited also to identify any additional questions which, in my opinion, would assist NOMS in being able to make an informed assessment of what, if any, reasonable steps it might need to consider taking. I am asked to comment specifically on the following questions (reproduced in bold; responses in indented text):

- 1. In the discussion section of their report at annex A, the authors say “Research by the World Health Organisation (WHO) and others suggests that there is no safe level of exposure to second hand smoke”. We should be grateful for your opinion as to what this actually means. Does it mean that it is not possible to say at what point second-hand smoke might cause injury, as opposed to meaning that even the smallest amount carries with it a reasonable probability of injury?**

- 1.1 This means that even the smallest amount carries with it a reasonable probability of injury. The authors of the WHO report cited as our reference 15, which aims to define safe limits for indoor air pollution, comment as follows:

The group concluded that the WHO guidelines for environmental tobacco smoke (ETS) published in the second edition of Air quality guidelines for Europe (2), stating that there is no evidence for a safe exposure level, are clear and still valid. Therefore, ETS is not included in

the current work. Furthermore, the guidelines for other pollutants should be developed based on the assumption that ETS is eliminated from indoor spaces.¹

- 1.2 The guideline from the second edition of Air quality guidelines for Europe, referred to in the 2010 WHO report at 1.1 above, is as follows: ²

ETS has been found to be carcinogenic in humans and to produce a substantial amount of morbidity and mortality from other serious health effects at levels of 1–10 µg/m³ nicotine (taken as an indicator of ETS). Acute and chronic respiratory health effects on children have been demonstrated in homes with smokers (nicotine 1–10 µg/m³) and even in homes with occasional smoking (0.1–1 µg/m³). There is no evidence for a safe exposure level. The unit risk of cancer associated with lifetime ETS exposure in a home where one person smokes is approximately 1 × 10⁻³.

This guideline refers to levels of ambient nicotine as a marker of second-hand smoke exposure. For logistic reasons we used PM_{2.5} particulates rather than nicotine as a marker of second-hand smoke but this does not matter; the important conclusion above is that serious health effects occur with exposure in the range generated by living in a home with a smoker. This level of exposure will be broadly similar for staff and prisoners in areas of a prison where someone is smoking.

This WHO report quantifies that lifetime risk of cancer in someone who lives with a smoker at one in a thousand.

- 1.3 Our report did not attempt to summarise the extensive scientific evidence on the health effects of second-hand smoke; rather we referred to the above authoritative statements by the World Health Organisation, and also to review [reference 22] reporting a 2010 study which specifically looked for evidence of a threshold of exposure at which second-hand smoke exposure begins to have an adverse effect on the function of cells lining the airways of human lungs. The study concluded

There was no threshold of urine nicotine without a small airway epithelial response, and only slightly above detectable urine cotinine threshold with a small airway epithelium response.³

- 1.4 However there are many other reports reaching similar conclusions, and aside from the World Health Organisation we could also have cited a 2005 report by the Royal College of Physicians of London ⁴, and a 2006 report by the US Surgeon General which states:⁵

This broadly reaching body of evidence on the toxicology of second-hand smoke and on these biologic mechanisms indicates that any exposure to second-hand smoke will increase risk for adverse health outcomes.

- 1.5 It is therefore evident that even the smallest amount of exposure to second-hand smoke carries a reasonable probability of injury.

¹ Page 3 from: World Health Organisation Europe. WHO guidelines for indoor air quality: selected pollutants. WHO Regional Office for Europe: http://www.euro.who.int/_data/assets/pdf_file/0009/128169/e94535.pdf; 2010

² Page 203 from: WHO Regional Publications European Series No.91. Air Quality Guidelines for Europe. World Health Organization Regional Office for Europe: http://www.euro.who.int/_data/assets/pdf_file/0005/74732/E71922.pdf; 2000

³ Strulovici-Barel Y, Omberg L, O'Mahony M, Gordon C, Hollmann C, Tilley AE, Salit J, Mezey J et al. Threshold of biologic responses of the small airway epithelium to low levels of tobacco smoke. *Am J Respir Crit Care Med* 2010;182:1524-1532

⁴ Royal College of Physicians. Going smoke-free: the medical case for clean air in the home, at work and in public places. A report on passive smoking by the Tobacco Advisory Group of the Royal College of Physicians. London: RCP <https://www.rcplondon.ac.uk/sites/default/files/documents/going-smoke-free.pdf>; 2005

⁵ Page 66 from: US Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke : a report of the Surgeon General. U S Department of Health and Human Services, Public Health Service, Coordinating Center for Health Promotion, Prevention, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health: <http://www.surgeongeneral.gov/library/reports/second-hand-smoke/fullreport.pdf>; 2006

2. How does the WHO upper limit guidance of 25µg/m³ as a 24 hour mean relate to the results contained in the report at Annex A which were done over a shorter period of time?

- 2.1. When we undertook to measure second-hand smoke levels in the sample of prisons we made the decision, on the grounds of logistics and costs, to use a proxy measure in the form of PM_{2.5} particulate levels. Second-hand smoke generates high levels of PM_{2.5} in the atmosphere, and this measure is widely accepted and used as a marker of second-hand smoke pollution in environmental studies such as ours. More specific markers of second-hand smoke, such as atmospheric nicotine or tobacco-specific nitrosamines, can be measured in indoor settings but the methods involved are more complex, costly, and less suitable for the prison environment.
- 2.2. However, as stated in our report, PM_{2.5} pollution also arises from other sources, such as cooking, open fires and motor vehicle emissions. Therefore whilst we consider that the great majority of the sometimes high PM_{2.5} levels detailed in our report are attributable to tobacco smoke, some is likely to have arisen from other sources.
- 2.3. We therefore cited the WHO limits on PM_{2.5} levels to provide some context for the levels we observed, but did not intend by doing so to imply that exposure below these levels are safe, since the evidence given under section 1 above indicates that, insofar as the PM_{2.5} are attributable to second-hand smoke, there is no safe level of exposure.
- 2.4. However for context, the WHO conclude in their 2005 global update on particulate pollution ⁶ that:

The risk for various outcomes has been shown to increase with exposure and there is little evidence to suggest a threshold below which no adverse health effects would be anticipated. In fact, the low end of the range of concentrations at which adverse health effects has been demonstrated is not greatly above the background concentration, which for particles smaller than 2.5 µm (PM_{2.5}) has been estimated to be 3–5 µg/m³ in both the United States and western Europe. The epidemiological evidence shows adverse effects of PM following both short-term and long-term exposures.

- 2.5. The WHO report goes on to say that the annual average concentration of 10 µg/m³ was chosen as the long-term guideline value for PM_{2.5} as this level represents the lower end of the range over which significant effects on survival were observed. Specifically, this is stated to be the lowest level at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95% confidence in response to long-term exposure ⁶.
- 2.6. With regard to the 24-hour limit of 25 µg/m³ the report comments that
- (a) the annual average [should] take precedence over the 24-hour average since, at low levels, there is less concern about episodic excursions.
 - (b) Meeting the guideline values for the 24-hour mean will however protect against peaks of pollution that would otherwise lead to substantial excess morbidity or mortality. ⁶

⁶ World Health Organisation. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide Global update 2005. World Health Organisation: http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf; 2006

- 2.7. The 25 $\mu\text{g}/\text{m}^3$ is an arithmetic mean, and on the assumption that second-hand smoke levels are highest during the day, rather than at night when many if not all prisoners are asleep and therefore not smoking, 24-hour average levels are likely to be lower than the average levels we recorded during the day.
- 2.8. However the WHO guidance is clear in stating that the 24-hour limit is intended to prevent exposure to peaks of pollution, such as we recorded in smoking areas of the prison. The more important limit is the annual limit of 10 $\mu\text{g}/\text{m}^3$ which, even assuming no exposure during sleeping hours, is likely to be exceeded in many of the prison settings we studied. Average levels in the prisons are documented in Tables 2-4 of our report for.
- 2.9. The data in our report can therefore be interpreted as demonstrating that indoor levels of particulate pollution in smoking areas of the prisons we studied exceeded the WHO 24 hour limit some of the time, the annual limit much of the time, and the safe limit for second-hand smoke exposure (zero) almost all of the time.
- 3. Assuming that there is no safe level of exposure to second hand smoke, are particular levels of exposure to second hand smoke related to the risk of injury and/or a particular type of injury? If so, we should be very grateful if you could give some examples based on the levels of exposure recorded in the report at Annex A.**
- 3.1. Evidence relevant to this question is summarised in the 2005 RCP report ⁷.
- 3.2. The three most common causes of death from active smoking are lung cancer, chronic obstructive pulmonary disease (COPD) and cardiovascular disease, of which myocardial infarction and stroke are key components. These conditions are all caused by smoking (ie, in the terms of the opening paragraph of this document are inception injuries).
- 3.3. The exposure-response curve for lung cancer is approximately linear, that is, demonstrates that every cigarette smoked adds to the risk of developing and dying from lung cancer ⁸. This increase in risk of cancer is not reversed by stopping smoking ⁹.
- 3.4. Non-smokers who live with smokers typically sustain smoke exposure equivalent to about 1 or 2% that of active smoking ⁷. Meta-analyses indicate that the risk of lung cancer is increased by second-hand smoking by about 25%, and this increase is roughly proportionate to the effect of the higher levels of exposure arising from active smoking.
- 3.5. Evidence on COPD is less extensive than for lung cancer, but indicates that the nature of the exposure-response relation is similar to that for lung cancer, and that second-hand exposure increases risk also by about 25% ⁷.
- 3.6. For cardiovascular disease the exposure-response relation is very different, rising very quickly at very low levels of exposure and then much less steeply with heavier smoking. Thus, while smoking 5 cigarettes a day is associated with a 50% increase in risk, and 20 cigarettes a day an approximate 80% increase in risk, second-hand smoke increases risk by about 30%. One cohort study of British men suggested that non-smokers exposed to high levels of second-hand smoke in the home were at similar risk of cardiovascular

⁷ Royal College of Physicians. Going smoke-free: the medical case for clean air in the home, at work and in public places. A report on passive smoking by the Tobacco Advisory Group of the Royal College of Physicians. London: RCP <https://www.rcplondon.ac.uk/sites/default/files/documents/going-smoke-free.pdf>; 2005

⁸ Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *Br Med J* 2004;328:1519-1533.

⁹ Halpern MT, Gillespie BW, Warner KE. Patterns of absolute risk of lung cancer mortality in former smokers. *J Natl Cancer Inst* 1993;85:457-464.

events to light (around 7/day) smokers ¹⁰.

- 3.7. The risk of cardiovascular disease from second-hand smoke is therefore disproportionately high, and indicates that even low level exposure translates into increased risk. This disproportionate effect of low levels of exposure probably explains why admissions to Scottish hospitals for acute coronary syndrome fell by around 14% more than the background secular trend when Scotland went smoke-free in 2006 ¹¹.
- 3.8. This increased risk of cardiovascular disease arising from low levels of exposure is thought to be mediated by an increase in blood coagulation, which occurs almost immediately after exposure and is probably reversed within a few days of ending exposure.
- 3.9. In sub-questions i-iii I am asked the following:

- i. **For example, all other things being equal, does exposure to 1124µg/m³ necessarily cause a greater likelihood of risk of injury/a particular injury compared to, say, 806µg/m³?**
- ii. **Would, for example, all other things being equal, exposure to 806µg/m³ cause a greater likelihood of risk of injury compared to, say, 121µg/m³?**
- iii. **Do certain levels of exposure increase the probability of particular types of injury? If so, which levels and which types of injury do those levels 'correspond' to?**

The evidence above suggests that the higher the exposure the greater the risk of lung cancer or COPD, but that even very low exposures can trigger acute cardiovascular problems. For particulate pollution in general, the WHO reports indicate that risk increases with exposure, and therefore that exposure to 1124µg/m³ will cause a greater likelihood of risk of injury/a particular injury compared to, say, 806µg/m³; and that exposure to 806µg/m³ will cause a greater likelihood of risk of injury compared to, say, 121µg/m³. However, exposure at all of these levels causes harm. The evidence on the exposure-response curve for smoking and cardiovascular disease also indicates that the risk of acute cardiovascular events could be raised to a relatively similar at all of these levels of exposure.

4. **Is the amount of time that a person is exposed to second hand smoke relevant to assessing the risk of injury and is that the case for every type of likely injury or just some types of likely injuries? For example, all other things being equal (including the level of exposure), is someone exposed to second hand smoke for, say, 2 minutes a day at lesser risk than someone exposed (to that same level) for 5 hours a day? If so, we should be very grateful if you could give some examples based on the report at Annex A.**

- 4.1. For the same reasons outlined above, I would expect the risk of lung cancer and COPD to be higher in someone exposed to the same level of exposure for 5 hours than for 2 minutes, but would expect the increase in cardiovascular risk to be less proportionate and hence relatively similar.

5. **Does the degree of risk of injury (either generally or particular types of injury) to a person vary depending on whether the period of time for which that person is exposed to second hand smoke is continuous or made up of separate periods of shorter exposure, between**

¹⁰ Whincup PH, Gilg JA, Emberson JR, Jarvis MJ, Feyerabend C, Bryant A, Walker M, Cook DG. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. *Br Med J* 2004;329:200-205.

¹¹ Pell JP, Haw S, Cobbe S, Newby DE, Pell AC, Fischbacher C, McConnachie A, Pringle S et al. Smoke-free legislation and hospitalizations for acute coronary syndrome. *N Engl J Med* 2008;359:482-491

which they are not exposed to second hand smoke or to much reduced levels. For example, all other things being equal, is exposure for a continuous period of 2 hours more likely to cause injury compared to 12 separate periods of 10 minutes. If so, we should be very grateful if you could give some examples based on the report at Annex A.

- 5.1. In relation to the risks of lung cancer and COPD, I would expect the cumulative injury from continuous or interval exposure at the same level and totalling the same duration to be exactly the same. This would also be true for cardiovascular disease if the intervals of exposure occurred within a short period (for example, the same day), as the effect of short-term exposure probably lasts for a few days. If comparing two hours of exposure on one day with twelve 10-minute intervals spread over a year, then in relation to total increase in cardiovascular risk the latter would much more sustained with interval exposure.
- 6. Are prisoners and staff with underlying health conditions more at risk of injury from second hand smoke compared to prisoners and staff who are fit and well? If so, which underlying medical conditions give the most cause for concern with respect to probability of risk of injury either generally or in relation to particular types of injury? (Please note that NOMS is taking immediate steps already in relation to staff and prisoners who NOMS knows are pregnant.)**
- 6.1. Second-hand smoke harms everyone. However people with a history of cardiovascular disease are probably at increased risk of acute exacerbation with short-term exposure.
- 6.2. Second-hand smoke also exacerbates, but does not necessarily cause, asthma; and can cause infective exacerbations among individuals with other chronic lung conditions.
- 6.3. Given that NOMS is already taking measures to protect pregnant women I would identify people with cardiovascular disease, asthma and other chronic pulmonary complaints as those at particular risk of injury (exacerbation) from short term exposure. However everyone, whether or not they have these conditions, is harmed by second-hand smoke.
- 7. If possible, we should be grateful if you could provide a view on how NOMS might identify those staff and prisoners who are at greatest risk of exposure to second hand smoke.**
- 7.1. To identify staff or prisoners to prioritise for protection from second hand smoke my personal suggestion would be to select those receiving treatment for, or with a history of, asthma, COPD, other chronic lung disease, angina, myocardial infarction, stroke or transient ischaemic attack. As a second-line and on account of the increased risk of cardiovascular disease that these conditions confer, I would identify those treated for hypertension or diabetes

The opinions I have expressed represent my true and complete professional opinions on the matters to which they refer. I believe that the facts that I have stated in this report are true and that the opinions I have expressed are correct.



John Britton

28th May 2015

CURRICULUM VITAE: John Richard BRITTON**PRESENT APPOINTMENTS:**

Professor of Epidemiology, University of Nottingham (appointed May 2000)

Director, UK Centre for Tobacco and Alcohol Studies, University of Nottingham (Appointed June 2008)

Honorary consultant in respiratory medicine, Nottingham University Hospitals (since October 1990).

ADDRESS: Clinical Sciences Building, City Hospital, Nottingham NG5 1PB.

DEGREES:	BSc Pharmacology	University of London	1975
	MBBS	University of London (Guys Hospital)	1978
	MRCP	UK	1981
	MD	University of London	1987
	MSc Epidemiology	London School of Hygiene and Tropical Medicine	1988
	FRCP	UK	1994
	FFPH	UK	2003

HONOURS: CBE for services to respiratory medicine, January 2013

PREVIOUS APPOINTMENTS:

Feb 78-July 82 House Physician/Surgeon, Guy's Hospital; Senior House Officer in Paediatrics, A&E, General Medicine at Guy's, London Chest and Southampton Hospitals.

Aug 82-Feb 84 Registrar in General Medicine, Southampton/Basingstoke.

Mar 84-Jan 86 Research Fellow, Respiratory Medicine Unit, University of Nottingham.

Feb 86-Sept 90 Clinical Lecturer, Respiratory Medicine Unit, University of Nottingham.

Oct 87-Sept 88 MRC Training Fellow, London School of Hygiene and Tropical Medicine.

Oct 90-May 00 Senior Lecturer/Reader/Professor of Respiratory Medicine, University of Nottingham

SELECTED CURRENT RESEARCH GRANTS:

1. J Britton, A McNeill, L Bauld, P Hajek, R West, G Hastings, A Amos, P Aveyard, T Coleman, S Lewis, J Leonardi-Bee, M Field, A Gilmore, I Gilmore, J Collin, S Michie, S Casswell, A Brennan, P Meier, S Parrott, M Munafo, C Griffin, R Ashcroft. The UK Centre for Tobacco and Alcohol Studies. UKCRC, Sept 2013-August 2018. £4,600,000 (approx., TBC)

3. J Britton, J De Gruchy, A McNeill, S Lewis, A Venn, T McKeever, I Hari, J Leonardi-Bee, R Murray, T Coleman, A Smyth, C Godfrey, S Parrott, L Jones, R Hubbard, W Sherwood. Smoking: new approaches to the cessation service delivery, prevention of passive smoke exposure in children, and healthcare cost estimation. NIHR Programme Grant, Jan 10 – May 16, £2,002,012

4. T Coleman, P Cansfield, S Sutton, M Ussher, S Cooper, J Thornton, J Britton, L Bauld, K Pickett, J Leonardi-Bee, S Parrott, M Battlemuch, L Tata, A McEwen, E Croghan, C Quinn. Improving the effectiveness and reach of NHS support for smoking cessation in pregnancy. NIHR Programme Grant Jan 11-Dec 16, £1, 909,0605.

5. Szatkowski L, Lewis S, McNeill A, Britton J, Jones L, Bains M, Bauld L, Parrot S, Fairs-Billam T, Letniowski T, Docherty G. Development and evaluation of a novel intervention providing insight into the tobacco industry to prevent the uptake of smoking in school-aged children. NIHR Public Health Research Programme Grant Jan 13-Dec 17. £710,751

SELECTED RECENT PUBLICATIONS:

I have published over 200 peer-reviewed original research papers, with a personal h-index of 71, and over 90 review articles and contributions to books. Selected papers since 2012 include:

1. Parental Smoking and the Risk of Middle Ear Disease in Children. A Systematic Review and Meta-analysis. Jones LL, Hassanien A, Cook DG, Britton J, Leonardi-Bee J. Arch Pediatr. Adolesc Med 2012;166:18-27
2. A randomized trial of nicotine replacement therapy patches in pregnancy. Coleman T, Cooper S, Thornton JG, Grainge MJ, Watts K, Britton J, Lewis S. New England Journal of Medicine 2012;366:808-18

3. Prenatal and Passive Smoke Exposure and Incidence of Asthma and Wheeze: Systematic Review and Meta Analysis. Burke H, Leonardi-Bee J, Hashim A, Pine-Abata H, Chen Y, Cook D, Britton J, McKeever T. *Pediatrics* 2012;129:735-744.
4. Systematic identification and treatment of smokers by hospital based cessation practitioners in a secondary care setting: cluster randomised controlled trial. Murray RL, Leonardi-Bee J, Marsh J, Jayes L, Li J, Parrott S, Britton J. *Br Med J* 2013;347: f4004.
5. Tobacco smoking and disability progression in multiple sclerosis: United Kingdom cohort study. Manouchehrinia A, Tench CR, Maxted J, Bibani RH, Britton J, Constantinescu C. *Brain* 2013;136:2298-2304.
6. Who receives prescriptions for smoking cessation medications? An association rule mining analysis using a large primary care database. Huang Y, Britton J, Hubbard R, Lewis S. *Tobacco Control* 2013;22:274-279.
7. Tobacco display and brand communication at the point of sale: Implications for adolescent smoking behaviour. Spanopoulos D, Britton J, McNeill A, Ratschen E, Szatkowski L. *Tobacco Control* 2014;23:64-69.
8. Patients' and healthcare professionals' views on a specialist smoking cessation service in a UK hospital: a qualitative study. Bains M, Britton J, Marsh J, Jayes L, Murray R. *Tobacco Induced Diseases* 2014;12:2.
9. Tobacco imagery on prime time UK television. Lyons A, McNeill A, Britton J. *Tobacco Control* 2014;23:257-263
10. Pictures don't lie, seeing is believing": Exploring attitudes to the introduction of pictorial warnings on cigarette packs in Ghana. Singh A, Owusu-Dabo E, Britton J, Munafò M, Jones LL *Nicotine and Tobacco Research* 2014;16:1613-9
11. Alcohol imagery on popularly viewed television in the UK. Lyons A, McNeill A, Britton J. *J Public Health* 2014;36:423-34
13. Prevalence of smoking among patients treated in NHS hospitals in England in 2010/2011: a national audit. Szatkowski L, Murray R, Hubbard R, Agrawal S, Huang Y, Britton J. *Thorax* 2014;doi: 10.1136/thoraxjnl-2014-206285.
14. Exposure to point-of-sale displays and changes in susceptibility to smoking: findings from a cohort study of school students. Bogdanovica I, Szatkowski L, McNeill A, Spanopoulos D, Britton J. *Addiction* 2015; DOI: 10.1111/add.12826
15. Adolescents' Exposure to Tobacco and Alcohol Content in YouTube Music Videos. Cranwell J, Murray R, Lewis S, Leonardi-Bee J, Dockrell M, Britton J. *Addiction* DOI: 10.1111/add.12835
16. Economic cost of smoking in people with mental disorders in the UK. Q Wu, L Szatkowski, J Britton, S Parrott. *Tobacco Control* doi:10.1136/tobaccocontrol-2013-051464
17. Parental smoking and child poverty in the UK: An analysis of national survey data. Belvin C, Britton J, Holmes J, Langley T. *BMC Public Health* (In press)
18. What is the quality of smoking cessation advice in guidelines of tobacco related diseases? Bogdanovica I, Agrawal S, Gregory B, Britton J, Leonardi-Bee J on behalf of the Tobacco Advisory Group of the Royal College of Physicians. *Clinical Medicine* (In press)
19. Healthcare interventions to promote and assist tobacco cessation: a review of efficacy, effectiveness and affordability for use in national guideline development. West R, Raw M, McNeill A, Stead L, Aveyard P, Britton J, Stapleton J, McRobbie H, Pokhrel S, Lester-George A, Borland R. *Addiction* (In press)
20. The relation between cigarette price and hand rolling tobacco consumption in the United Kingdom: an ecological study. Rothwell L, Britton J, Bogdanovica I. *BMJ Open* (in press)