Association between environmental tobacco smoke and depression among Korean women

Na Hyun Kim,¹ Hyeon Chang Kim,²,³ Joo Young Lee,² Ju-Mi Lee,² Il Suh²

ABSTRACT

Objective: To evaluate the association between environmental tobacco smoke (ETS) exposure and depression among Korean women.

Methods: Between 2008 and 2011, we examined 731 men and 1249 women (aged 39–85 years) for the Korean Genome and Epidemiology Study (KoGES)-Kangwha. Among 1208 never-smoking women, we excluded two women taking antidepressants and five women who did not complete the Beck Depression Inventory (BDI). Therefore, we performed a cross-sectional analysis on 1201 women. ETS exposure was assessed using a self-reported questionnaire, and was classified into three groups: no exposure, occasional exposure and regular exposure. Depression was assessed using the BDI score, which ranged from 0 to 63, and the presence of depression was defined as a BDI score ≥10.

Results: Women exposed to ETS were more likely to have depression than those without ETS exposure (p=0.019). When BDI was analysed as a continuous variable, women exposed to ETS had significantly higher BDI scores after full adjustment (overall exposure: β=1.36, p=0.013; occasional exposure: β=1.15, p=0.063; regular exposure: β=1.90, p=0.039). ETS exposure was significantly associated with depression in a dose–response manner even after adjusting for age, body mass index, menopause, socioeconomic status, lifestyle and prevalent chronic diseases. The adjusted OR for depression (95% CI) was 1.72 (1.25 to 2.37) for overall ETS exposure, 1.56 (1.09 to 2.24) for occasional exposure and 2.19 (1.30 to 3.69) for regular exposure, when compared to no exposure.

Conclusions: Exposure to ETS was associated with depression among middle aged and elderly Korean women.

INTRODUCTION

It has been estimated that there have been more than 20 million premature deaths attributable to direct and indirect tobacco smoke.¹ In the USA, the current annual burden of smoking-attributable mortality is estimated to be about 480,000, with millions more living with smoking-related diseases.³

Secondhand smoke, or environmental tobacco smoke (ETS), is a mixture of two forms of smoke: the sidestream smoke that comes from the lighted end of a cigarette and the mainstream smoke that is exhaled by the smoker.² It is known that exposure to ETS can increase the risk of cardiovascular disease, respiratory illness and cancer in adults.¹ ³ ⁴ In addition, recent studies have reported that ETS can lead to mental health problems, including depression.⁵–¹⁰ Depression is the most common mental illness, with an estimated 350 million people affected. Symptoms of depression are usual mood fluctuations and short-lived emotional responses to challenges. Especially when long-lasting and with moderate or severe intensity, depression may cause an affected person to suffer greatly and function poorly in daily life. At its worst, it can lead to suicide, which results in an estimated 1 million deaths every year.¹¹ Many studies have found that active tobacco smoking can increase the risk of depression.¹²–¹⁵ Recently, exposure to ETS has been associated with depression or depressive symptoms in some studies,⁵–¹⁰ though not in others.¹⁶ ¹⁷

The purpose of the present study was to explore the possible association between ETS and depression in never-smoking Korean women. Increasing evidence supports the hypothesis that exposure to ETS can cause various chronic disorders.¹ ³ ⁴ As these
chronic conditions are closely related to depression.\textsuperscript{18–21} ETS exposure can be associated with depression as well. Additionally, in animal studies, nicotine exposure has been reported to induce a negative mood, lower mobility and dopamine imbalance, all of which are known to increase the risk of depression.\textsuperscript{22–24} Although a positive association between ETS and depression has been reported among Korean adolescents,\textsuperscript{25} such association has not been observed in an adult Korean population. Thus, we hypothesised that ETS exposure would be associated with depression in Korean adults. We limited our analysis to never-smoking women, as the effects of former or current active smoking cannot be properly controlled with statistical adjustment.

Study population

This study is a cross-sectional analysis of baseline data from a community-based prospective cohort known as the Korean Genome and Epidemiology Study (KoGES)-Kangwha, which started in 2006 on Kangwha Island, South Korea. This analysis enrolled participants who attended baseline health examinations between 2008 and 2011, as the BDI questionnaire has only been available since 2008. Among the initial total of 1980 participants, 1208 women were never-smokers. We excluded two women with depression medication and five women who did not complete the Beck Depression Inventory (BDI). A total of 1201 women were enrolled in this study. All participants signed written informed consent forms.

Measurements

All participants were individually interviewed using a standardised questionnaire to obtain information about sociodemographic characteristics, health behaviours, chronic diseases and psychosocial stress. Trained interviewers conducted dialogues according to a predefined protocol and double-checked whether responses were appropriate. The income category was divided into two groups: <1 500 000 won or ≥1 500 000 won, based on the median of data. Occupations were initially classified into 13 categories based on the Korea Standard Classification of Occupations, although they were later re-categorised into two groups, employed and unemployed, which included housewives. Exposure to ETS was measured with two questions: “How many days per week are you exposed to tobacco smoke at home and/or your workplace?” and “How many minutes per day are you exposed to tobacco smoke at home and/or your workplace?” Based on the answers to these questions, ETS exposure status was categorised into three groups: no exposure, occasional exposure (≤4 days/week and/or <30 min/day) and regular exposure (>1 days/week and ≥30 min/day), according to a previous study.\textsuperscript{26} Alcohol consumption was categorised into two groups, current alcohol drinkers and current non-drinkers. Physical exercise was categorised into two groups based on the frequency of leisure time physical activity. Psychosocial stress was divided into two groups: those who had been feeling stress for the last month and those who had not.

Participants’ standing height was measured to the nearest 0.1 cm with an extensometer (DS-102, JENIX, Korea) and body weight was measured to the nearest 0.1 kg with a digital scale (DB-I50, CAS, Korea). Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in metres. Resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured at least twice using an automatic oscilloscopic sphygmomanometer (CareScape Dinamap V100, GE Healthcare). If the first and second measurements differed by ≥10 mm Hg for SBP or DBP, then additional measurements were performed, and the average of the last two measurements was used for the current analysis. Blood samples were collected from the antecubital vein of participants, after at least 8 h of fasting. Collected blood samples were analysed at the central research laboratory for measurements of complete blood count, fasting glucose and insulin, as well as for lipid profiles. Hypertension was defined as elevated blood pressure (SBP ≥140 mm Hg or DBP ≥90 mm Hg) or use of antihypertensive medication. Diabetes mellitus was defined as elevated fasting blood glucose (≥126 mg/dL), elevated glycaed haemoglobin (≥6.5%) or treatment for diabetes. Hypercholesterolaemia was defined as elevated total cholesterol (>200 mg/dL) or use of lipid-lowering medication.

Depressive symptoms were assessed using the BDI questionnaire. The BDI consists of 21 questions for emotional, cognitive, motivational, physiological and other symptoms. Each item contains four statements describing the intensities of symptoms of depression. Each item is rated on a scale from 0 to 3, reflecting how participants have felt over the past week.\textsuperscript{27–29} Thus, the total BDI scores range from 0 to 63, with higher scores representing greater disability. This index has demonstrated acceptable sensitivity and specificity in distinguishing between participants with and without depression, and is considered a valid and reliable measure of depression.\textsuperscript{27 28 30} The presence of depression was defined as a BDI score ≥10, and participants were further classified into three groups according to severity: mild (10–15), moderate (16–23) and severe (24–63).\textsuperscript{27 28 30}

Statistical analysis

General and clinical characteristics were described for a total of 1201 women. Exposure to ETS and other variables were compared between women with depression (BDI score ≥10) and those without depression, and then compared again across the four groups of BDI scores: none, mild, moderate and severe depression. Independent associations between ETS and depression were assessed using serial linear and logistic regression models: (1) adjusted for age, BMI, menopause, household income and employment; (2) adjusted for age, BMI, menopause, household income, employment, alcohol intake, regular exercise, hypertension, diabetes
and hypercholesterolaemia and (3) adjusted for age, BMI, menopause, household income, employment, alcohol intake, regular exercise, hypertension, diabetes, hypercholesterolaemia and psychosocial stress. All statistical analyses were performed using SAS V.9.2 (SAS Inc, Cary, North Carolina, USA). All analyses were two-sided and p values less than 0.05 were regarded as statistically significant.

RESULTS
The characteristics of the study participants are summarised in Table 1. The mean age was 58.7 years for all participants, 59.9 years for the group with no ETS exposure and 54.4 years for the group exposed to ETS. Overall, 21.5% (n=258) of non-smoking women were exposed to ETS; within them, 15.8% (n=190) were exposed occasionally and 5.7% (n=68) were exposed regularly. The mean BDI score was 7.9 in total participants, 8.6 in ETS exposure group and 7.7 in no exposure group. However, the difference between the no exposure and exposure groups was not statistically significant (p=0.090). The prevalence of depression was higher in the ETS exposure group (38.4%) than in the no ETS exposure group (30.4%), and this difference was statistically significant (p=0.019).

Table 2 presents the characteristics of the study population according to the presence and severity of depression. Women with depression were more likely to have lower household income (p=0.044), be unemployed or be housewives (p=0.018), report psychosocial stress (p<0.001) and be exposed to ETS (p=0.019) than...
those without depression. When depression was further classified according to severity, a higher severity of depression was associated with lower blood pressure, lower income, unemployment, psychosocial stress and ETS exposure.

Table 3 describes the association between ETS and BDI scores as continuous variables in serial regression models. Compared to those without ETS exposure, women exposed to ETS had significantly higher BDI scores when adjusted for age, BMI, menopause, income and employment ($\beta=1.38$, $p=0.013$). The association was weakened by additional adjustment for psychosocial stress, yet still remained significant ($\beta=1.21$, $p=0.022$). When ETS was divided by the degree of exposure, regular ETS exposure was significantly associated with higher BDI scores ($\beta=1.78$, $p=0.046$), although occasional exposure was not significant ($\beta=0.98$, $p=0.100$). Figure 1 displays the distribution of BDI scores separately for women with no exposure, occasional exposure and regular exposure. Women with regular ETS exposure had higher BDI scores than those with no exposure or occasional exposure.

Table 4 shows the association between ETS and depression using serial logistic models. Compared to
those with no exposure, people with ETS exposure had significantly higher odds for depression, with an OR of 1.70 (95% CI 1.25 to 2.33) after adjusting for age, BMI, menopausal status, income and employment. This association did not change when further adjusting for lifestyle factors, comorbidity and psychosocial stress. There was also a dose–response relationship between ETS exposure and depression. In contrast to the no exposure group, occasional and regular exposure groups showed gradually increased ORs for depression after adjusting for age, BMI, menopausal status, income and employment (occasional: 1.55 (95% CI 1.08 to 2.21); regular: 2.16 (95% CI 1.29 to 3.61)). This dose–response association was robust to further adjustment for lifestyle factors, comorbidity and psychosocial stress. We performed further analysis to assess whether psychosocial stress could modify the association between ETS and depression. Although the association was slightly weaker among women who reported psychosocial stress than among those who did not, dose–response association was observed in both groups (data presented in online supplementary table S1).

**DISCUSSION**

We observed a significant association between ETS exposure and depression among never-smoking Korean women. We also observed that regular ETS exposure might have a higher risk for depression than occasional exposure to ETS. To the best of our knowledge, this is the first report on the association between ETS and depression in a community-dwelling Korean adult population.

A previous study analysing the US National Health and Nutrition Examination Survey found that ETS was positively associated with depressive symptoms after adjusting for age, ethnicity, gender, education, cardiovascular...
disease, respiratory disease, diabetes mellitus, hypertension, thyroid disease and cancer. Another study reported that ETS exposure is associated with psychological distress and risk of future psychiatric illness in a Scottish adult population. A recent study also observed that in utero and childhood exposure to ETS was associated with an increased risk of depression in midlife, even after adjusting for direct and indirect exposure to tobacco smoke in adulthood. In the Midlife Development in the US Survey (MIDUS), persistent exposure to ETS across the course of life was associated with an increased risk of depression and panic attacks. Other studies demonstrated that ETS exposure was strongly associated with hyperactivity, conduct disorder and depression, in children and adolescents. On the contrary, ETS was not associated with depression or mental health in the UK Health and Lifestyle Survey (HALS), the Netherlands Study of Depression and Anxiety (NESDA), and the Netherlands Twin Register (NTR). There are several possible explanations for the association of ETS exposure with depression among non-smoking women. First, the secondhand smoke itself can be stressful for non-smokers. Regular exposure to ETS at home and the workplace is a chronic stressor for non-smoking women, and the chronic stress may lead to the development of depressive symptoms. In addition, women exposed to ETS at home might also be exposed to other risk factors of depression as well. Smoking partners are likely to have other adverse health-related behaviours and poor socioeconomic characteristics. Thus, women who have a smoking partner might be more exposed to other risk factors of depression than women who have a non-smoking partner. To indirectly assess the effects of smoking partners on depression, we performed our analysis separately for ETS exposure at home and at the workplace. However, depression was associated with both ETS at home, and ETS at the workplace, and the association was stronger for ETS at the workplace (data are presented in online supplementary table S2). Thus, the partner characteristics were unlikely to be major contributors of the ETS-related depression in our study. Another possible mechanism is the dopamine system, which is known to be related to the risk of depression. Several animal studies support the hypothesis that ETS has an acute and long-term effect on the dopamine system. Another study found that ETS greatly elevated dopamine D1 and D2 receptors in the brains of rats. Furthermore, other studies have provided evidence indicating that exposure to ETS impacts γ-aminobutyric acid b2 receptors (GABAB2), dopamine transporter messenger RNA expression and dopamine receptors. Additionally, an animal study found that nicotine and tobacco particulate matter has an influence on long-term imbalances of dopamine transports. Most importantly, one animal study observed that nicotine exposure induced a negative mood and decreased mobility in rats. Another biological mechanism that may link ETS exposure to depression is chronic inflammation. Several studies have proposed that the activation of inflammatory cytokines plays a role in the development of depression. Cytokines are related to microglia, which may be over-activated in major depression. Additionally, cytokines induce enzyme indoleamine 2,3-dioxygenase (IDO), which limits tryptophan and serotonin transporter and can thus cause depression. The present study has several limitations. First, as a cross-sectional study in which all information was gathered at the same point in time, we cannot establish a temporal relationship between ETS exposure and depression. Second, we measured the degree of ETS and depressive symptoms using an interviewer-assisted questionnaire. The misclassification bias in measuring ETS exposure, if any, is likely to be a non-differential reduction of the association. The BDI has shown high
internal consistency (α=0.88) and test-retest reliability (r=0.60, p<0.001) in previous reports. Thus, our findings are unlikely to be severely distorted by measurement error. Third, objective measurement of participants’ psychosocial stress was unavailable in this study. Psychosocial stress was measured only by asking the participants whether they had been stressed during the last month. Thus, we could not properly investigate whether any psychosocial stress confounded or modified the association between ETS and depression. Lastly, our study population was limited to women from a single rural area; therefore, our findings may not be generalisable to other regions or the male population.

The strength of this study was the analysis of a community-based adult population. Previous studies in the Asian population were conducted at school or occupational settings, thus the target populations were adolescents and working adults. Although our study population was limited to one rural population, there was a wide range of ages, and employed and unemployed women were both included. Cigarette smoking and depression are among the most serious health problems in the Korean population. According to our calculation, 20.7% of depression cases in the current study population were attributed to ETS exposure. This finding implies that a considerable portion of depression cases can be prevented by reducing exposure to ETS, particularly in underlying populations with a high smoking rate.

**CONCLUSIONS**

Exposure to ETS was associated with depression in a dose-response manner among community-dwelling Korean women. Further studies are needed to confirm the causal effects of ETS on the development and aggravation of depression, and to identify the underlying biological mechanisms.

| Variables                      | OR (95% CI) for depression |
|--------------------------------|----------------------------|
|                                | N          | Model 1          | Model 2          | Model 3          |
| ETS                            |            |                  |                  |                  |
| No exposure                    | 943        | 1.000            | 1.000            | 1.000            |
| Exposure (overall)             | 258        | 1.704 (1.246 to 2.331) | 1.723 (1.254 to 2.366) | 1.700 (1.226 to 2.356) |
| Occasional exposure            | 190        | 1.548 (1.083 to 2.213) | 1.562 (1.088 to 2.242) | 1.526 (1.052 to 2.214) |
| Regular exposure               | 68         | 2.158 (1.288 to 3.614) | 2.191 (1.302 to 3.688) | 2.212 (1.295 to 3.778) |
| Age                            |            |                  |                  |                  |
| Per 10 years                   | 1201       | 1.011 (0.993 to 1.030) | 1.010 (0.991 to 1.030) | 1.009 (0.989 to 1.029) |
| Body mass index                |            |                  |                  |                  |
| Per 1 kg/m²                    | 1201       | 0.988 (0.950 to 1.028) | 0.984 (0.944 to 1.026) | 0.991 (0.949 to 1.034) |
| Menopause                      |            |                  |                  |                  |
| Premenopause                   | 254        | 1.000            | 1.000            | 1.000            |
| Postmenopause                  | 945        | 0.963 (0.644 to 1.440) | 0.976 (0.650 to 1.466) | 0.992 (0.654 to 1.505) |
| Income                         |            |                  |                  |                  |
| <1 500 000 won                 | 631        | 1.000            | 1.000            | 1.000            |
| ≥1 500 000 won                 | 470        | 0.807 (0.586 to 1.112) | 0.809 (0.585 to 1.119) | 0.754 (0.541 to 1.052) |
| Employment                     |            |                  |                  |                  |
| No                             | 445        | 1.000            | 1.000            | 1.000            |
| Yes                            | 756        | 0.697 (0.535 to 0.909) | 0.713 (0.546 to 0.932) | 0.661 (0.502 to 0.872) |
| Alcohol intake                 |            |                  |                  |                  |
| No                             | 930        | 1.000            | 1.000            | 1.000            |
| Yes                            | 270        | 1.024 (0.747 to 1.404) | 1.039 (0.752 to 1.437) |                  |
| Regular exercise               |            |                  |                  |                  |
| No                             | 780        | 1.000            | 1.000            | 1.000            |
| Yes                            | 421        | 1.076 (0.818 to 1.415) | 1.038 (0.784 to 1.375) |                  |
| Hypertension                   |            |                  |                  |                  |
| No                             | 765        | 1.000            | 1.000            | 1.000            |
| Yes                            | 436        | 1.142 (0.852 to 1.531) | 1.086 (0.803 to 1.468) |                  |
| Diabetes                       |            |                  |                  |                  |
| No                             | 1023       | 1.000            | 1.000            | 1.000            |
| Yes                            | 176        | 1.087 (0.745 to 1.585) | 1.110 (0.753 to 1.635) |                  |
| Hypercholesterolaemia          |            |                  |                  |                  |
| No                             | 755        | 1.000            | 1.000            | 1.000            |
| Yes                            | 443        | 0.924 (0.700 to 1.220) | 0.895 (0.673 to 1.189) |                  |
| Psychosocial stress            |            |                  |                  |                  |
| No                             | 491        |                  |                  | 1.000            |
| Yes                            | 710        |                  |                  | 2.797 (2.117 to 3.694) |
Contributors NHK contributed to the study concept and design, performed statistical analyses, and drafted and revised the manuscript. HCK contributed to the conception and design, and analysis and interpretation of the data, and revised the article. JYL and J-ML managed KoGES data and supported analysis. IS supervised data analysis and revised the manuscript. All the authors contributed in revising the article critically for important intellectual content, and gave final approval of the version to be published. HCK is responsible for the overall content as the guarantor.

Funding This work was supported by the Korea Centers for Disease Control and Prevention (2008-E71004-00, 2009-E71006-00) and the Korean Health Technology R&D Project, Ministry of Health & Welfare, Republic of Korea (HI13C-0715).

Competing interests None declared.

Patient consent Obtained.

Ethics approval The study protocol was approved by the Institutional Review Board of Severance Hospital at Yonsei University College of Medicine.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement No additional data are available.

Open Access This is an Open Access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

REFERENCES

1. U.S. Department of Health and Human Services. Reports of the Surgeon General. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta, GA: Centers for Disease Control and Prevention (US), 2014.

2. U.S. Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Centers for Disease Control and Prevention (US), 2006.

3. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke nearly as large as smoking. Circulation 2005;111:2684–96.

4. Venn A, Britton J. Exposure to secondhand smoke and biomarkers of cardiovascular disease risk in never-smoking adults. Circulation 2007;115:990–5.

5. Hamer M, Stamatakis E, Batty GD. Objectively assessed secondhand smoke exposure and mental health in adults: cross-sectional and prospective evidence from the Scottish Health Survey. Arch Gen Psychiatry 2010;67:855–6.

6. Hamer M, Ford T, Stamatakis E, et al. Objectively measured secondhand smoke exposure and mental health in children: evidence from the Scottish Health Survey. Arch Pediatr Adolesc Med 2011;165:326–31.

7. Bandiera FC, Richardson AK, Lee DJ, et al. Secondhand smoke exposure and mental health among children and adolescents. Arch Pediatr Adolesc Med 2011;165:326–31.

8. Bandiera FC, Ahearn KL, Caban-Martinez AJ, et al. Secondhand smoke exposure and depressive symptoms. Psychosom Med 2010;72:68–72.

9. Elmasry H, Goodwin RD, Terry MB, et al. Early life exposure to cigarette smoke and depressive symptoms among women in midlife. Nicotine Tob Res 2014;16:1298–306.

10. Taha F, Goodwin RD. Secondhand smoke exposure across the life course and the risk of adult-onset depression and anxiety disorder. J Affect Disord 2014;168:367–72.

11. World Health Organization. Depression: a global crisis. World Mental Health Day, 10 October 2012.

12. Boden JM, Ferguson DM, Horwood LJ. Cigarette smoking and depression: tests of causal linkages using a longitudinal birth cohort. Br J Psychiatry 2010;196:440–6.

13. Murphy JM, Horton ND, Monson RR, et al. Cigarette smoking in relation to depression: historical trends from the Stirling County Study. Am J Psychiatry 2003;160:1603–9.

14. Wiesbeck GA, Kuhl HC, Yaldizli O, et al. Tobacco smoking and depression—results from the WHO/ISBRA study. Neuropsychobiology 2008;57:26–31.

15. Flensborg-Madsen T, von Scholten MB, Flachs EM, et al. Tobacco smoking as a risk factor for depression. A 26-year population-based follow-up study. J Psychiatr Res 2011;45:143–9.

16. Bot M, Vink JM, Willemsen G, et al. Exposure to secondhand smoke and depression and anxiety: a report from two studies in the Netherlands. J Psychosom Res 2013;75:431–6.

17. Lam E, Kvaavik E, Hamer M, et al. Association of secondhand smoke exposure with mental health in men and women: cross-sectional and prospective analyses using the U.K. Health and Lifestyle Survey. Eur Psychiatry 2013;28:276–81.

18. Patton SB, Williams JV, Lavorato DH, et al. Major depression as a risk factor for chronic disease incidence: longitudinal analyses in a general population cohort. Gen Hosp Psychiatry 2008;30:407–13.

19. Mezuk B, Eaton WW, Albrecht S, et al. Depression and type 2 diabetes over the lifespan: a meta-analysis. Diabetes Care 2008;31:2385–90.

20. Goodwin RD, Jacobi F, Thefeld W. Mental disorders and asthma in the community. Arch Gen Psychiatry 2003;60:1125–30.

21. Lu L, Mackay DF, Newby DE, et al. Association between salivary cotinine and cardiovascular biomarkers among nonsmokers and current smokers: cross-sectional study of 10,381 participants. Eur J Vasc Endovasc Surg 2014;48:703–10.

22. Dailly E, Chenu F, Renard CE, et al. Dopamine, depression and antidepressants. Fundam Clin Pharmacol 2004;18:801–7.

23. Inatkez SD, Warren BL, Parise EM, et al. Nicotine exposure during adolescence induces a depression-like state in adulthood. Neuropsychopharmacology 2008;34:1609–24.

24. Danielson K, Pult F, Truman P, et al. The effects of nicotine and tobacco particulate matter on dopamine uptake in the rat brain. Synapse 2014;68:45–51.

25. Lee KJ. Current smoking and secondhand smoke exposure and depression among Korean adolescents: analysis of a national cross-sectional survey. BMJ Open 2014;4:e003734.

26. Panagiotakos DB, Pitsavos C, Chrysohou C, et al. Effect of exposure to secondhand smoke on markers of inflammation: the ATTICA study. Am J Med 2004;116:145–50.

27. Jo SA, Park MH, Jo I, et al. Usefulness of Beck Depression Inventory (BDI) in the Korean elderly population. Int J Geriatr Psychiatry 2007;22:195–5.

28. Lee HY, Song JY. A study of the reliability and the validity of the BDI, SDS, and MMPI-D scales. Korean J Clin Psychol 1991;10:98–113.

29. Beck AT, Steer RA, Carbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. Clin Psychol Rev 1988;8:77–100.

30. Beck A, Steer R, Brown G. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation, 1996.

31. Hammen C. Stress and depression. Annu Rev Clin Psychol 2005;1:293–319.

32. Lee JY, Li S, Park MS, et al. Dopamine D1 and D2 receptor mRNA up-regulation in the caudate-putamen and nucleus accumens of rat brains by smoking. Prog Neuropsychopharmacol Biol Psychiatry 2002;26:1095–104.

33. Li S, Kim KY, Kim JH, et al. Chronic nicotine and smoking treatment increases dopamine transporter mRNA expression in the rat midbrain. Neurosci Lett 2004;363:29–32.

34. Adams T, Wan E, Wei Y, et al. Secondhand smoking is associated with vascular inflammation. Chest 2015 [epub ahead of print]. doi:10.1378/chest.14-2045.

35. Jeffers BJ, Lowe GD, Welsh P, et al. Secondhand smoke (SHS) exposure is associated with circulating markers of inflammation and endothelial function in adult men and women. Atherosclerosis 2010;208:550–6.

36. Dowlati Y, Herrmann N, Swardfager W, et al. A meta-analysis of endotoxins in major depression. Biol Psychiatry 2010;67:446–57.

37. Eisenberger NI, Berkman ET, Inagaki TK, et al. Inflammation-induced anhedonia: endotoxin reduces ventral striatum responses to reward. Biol Psychiatry 2010;68:748–54.

38. Pasco JA, Nicholson GC, Williams LJ, et al. Association of high-sensitivity C-reactive protein with de novo major depression. Br J Psychiatry 2010;197:372–7.

39. Dantzer R, O’Connor JC, Lawson MA, et al. Association of inflammation-associated depression: from serotonin to kynurenine. Psychoneuroendocrinology 2011;36:26–36.

40. Schroeter ML, Abdul-Khaliq H, Krebs M, et al. Serum markers support disease-specific glial pathology in major depression. J Affect Disord 2008;111:271–80.

41. Lam TH, Stewart SM, Ho SY, et al. Depressive symptoms and smoking among Hong Kong Chinese adolescents. Addiction 2005;100:1003–11.

42. Nakata A, Takahashi M, Ikeda T, et al. Active and passive smoking and depression among Japanese workers. Prev Med 2008;46:451–6.